HIV, VIRAL HEPATITIS & STIs
A GUIDE FOR PRIMARY CARE

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# CONTENTS 2014

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## INTRODUCTION TO HIV, VIRAL HEPATITIS AND STIs

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## CHAPTER 1

**HIV, HBV, HCV and STIs: similarities and differences**

Mark Danta, Lynne Wray

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PREFACE

The 2013 ASHM evaluation of the resource *HIV, viral hepatitis and STIs: a guide for primary care* strongly indicated the need for this valuable resource to remain a hard copy. The first edition of this widely used teaching and reference guide was first produced in 2001, with a second in 2004 and a third edition in 2008. While an online searchable PDF version would be an asset for busy practitioners, the hard copy allows for easier access by those in regional and remote settings and by those working in demanding, time poor work environments with limited computer access and internet availability. Clinicians who test for HIV and viral hepatitis and those who provide non-specialist care for patients with these infections make up the intended audience for this monograph, as well as primary care practitioners who see patients with STIs and those who screen for STIs in their day-to-day work. Physicians, medical students, nurses, allied health professionals, as well as individuals with a specific interest in these conditions, will continue to find this resource valuable.

As the 2014 process was a review only, major structural changes have been minimal. The most significant change has been the separation of hepatitis C and hepatitis B content into separate chapters under the section on management in the primary care setting. Changes to hepatitis C treatment have been significant and our knowledge of how to test and treat people living with hepatitis B in Australia has progressed; these advances are reflected in the rewriting of these chapters.

Many organisations and individuals have contributed to the production of this new edition and previous editions of this resource. On behalf of ASHM, we would like to acknowledge the input of the Gastroenterological Society of Australia (GESA) and the Australian Liver Association (ALA), The Australasian Sexual Health Alliance (ASHA), the Australian College of Rural and Remote Medicine (ACCRM), the Australasian Society for Infectious Diseases (ASID), the Australian Federation of AIDS Organisations (AFAO), the National Association of People Living with HIV Australia (NAPWHA), Australian Injecting & Illicit Drug Users League (AVIL), Australasian Sexual Health and HIV Nurses Association (ASHHNA), the Australian Indigenous Doctors Association (AIDA) and the Australasian Hepatology Association (AHA).

We would like to acknowledge the significant work within a short timeframe of our lead reviewers and writers, Lynne Wray, Siobhan Bourke, Anna McNulty, Iryna Zablostka, Eric Khong, Catriona Ooi, Darren Russell, Gail Matthews, David Orth, Vanessa Towell, Simone Strasser and Indraveer Chatterjee.

In addition, members of the review team deserve our recognition, namely Mark Danta, Ian Denham, Jack Wallace, Paul Haber, Phillip Keen, John McAllister, Andrew Carr, Ingrid van Beek, Mary Burns, Gary Rogers, Deborah Couldwell, Greg Dore, Wendy Cheng, Michael Burke, Paul Harvey, John Patten, Michael Burke, Seamus Duffy, Nicole Allard, Vanessa Towell, Tracey Cabrie, David Youds, Anna McNulty, Marianne Martinello, Astrid Greenup and Anna Roberts.

Authors and reviewers showed considerable commitment to continuing medical education through their dedicated approach to ensuring each chapter accurately reflects the current issues and trends within the viral hepatitis, HIV and STIs sector. Thanks to the many other individuals who contributed ideas and information to the project.

We would also like to acknowledge the contribution of the Expert Reference Group, who provided editorial oversight in the review process of this fourth edition: Michael Burke, Tracey Cabrie, Benjamin Cowie, Gregory Dore, Seamus Duffy, Gail Matthews, Ronald McCoy, Anna McNulty, Catriona Ooi, Simone Strasser, David Youds and Iryna Zablotska.

The 2014 edition of *HIV, viral hepatitis and STIs – a guide for primary care* was funded by the Commonwealth Department of Health.

Edwina Wright
ASHM President
Melbourne, July 2014
INTRODUCTION TO HIV, VIRAL HEPATITIS AND STIs
CHAPTER 1 HIV, HBV, HCV AND STIs: SIMILARITIES AND DIFFERENCES

2014 REVIEW

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Introduction

The three major blood-borne viruses, human immunodeficiency virus (HIV), hepatitis B virus (HBV) and hepatitis C virus (HCV), are members of different virus families but have one thing in common: their major mode of transmission is via blood or bodily fluids. Sexually transmitted infections (STIs) are a diverse group of infections caused by widely differing micro-organisms (viruses, protozoa, bacteria, yeasts, ectoparasites and even a nematode), whose common characteristic is that they are transmitted from person to person by sexual contact such as deep kissing, vaginal sex, anal sex, oral sex, oro-anal sex or close intimate physical contact.

Table 1.1 provides a list, although not exhaustive, of the causative agents and their accompanying infections which are capable of being sexually transmitted (i.e. sexually transmissible infections). The distinction between the terms ‘sexually transmitted’ and ‘sexually transmissible’ is a fine one and there is little consensus about the correct usage — in this monograph the terms will be used interchangeably, with ‘sexually transmitted’ being favoured.

Some infections (e.g. gonorrhoea, chlamydia and syphilis) are readily recognisable as being STIs while others (e.g. hepatitis A and the enteric infections) are only sexually transmitted under certain circumstances, namely where sexual activity facilitates oro-anal transmission (Table 1.1). The three major blood-borne viruses mentioned above are all capable of sexual transmission so can also be categorised as STIs, with HIV and HBV very readily sexually transmitted, but HCV only rarely sexually transmitted (see below). Despite their diversity, STIs share two other common characteristics which justify considering them as a group:

- Similar behavioural characteristics lead to people contracting, and being at risk of, STIs, so control strategies are similar for all of them.
- Most STIs, in their early stages, are asymptomatic or so mildly symptomatic as to be easily overlooked, yet are infectious, screening at-risk people is essential for population management.

KEY POINTS

- Human immunodeficiency virus (HIV), hepatitis B virus (HBV) and hepatitis C virus (HCV) are distinct viruses with different epidemiological profiles, modes of transmission, natural histories and treatments.
- All three viruses lead to chronic infection in many individuals with the viral infection and are characterised by hypermutability and quasi species.
- The microbiological and virological agents that cause STIs are highly diverse, having specific epidemiological profiles, varied modes of sexual transmission, different natural histories and individual treatment modalities.
- HIV is transmitted through sexual contact, blood-to-blood contact and mother-to-child transmission. Without treatment, most individuals with HIV develop severe immune deficiency within 10 years. Combination antiretroviral therapy has transformed the course of the disease, extending the life expectancy of individuals with HIV by many years.
- STIs have a complex synergistic relationship with HIV. Most STIs play an enhancing role in the acquisition and transmission of HIV, while HIV may alter the natural history and response to treatment of some STIs.
- HBV is transmitted through mucous membrane contact (including unprotected sexual contact), blood-to-blood contact, mother-to-child transmission and intrafamilial transmission.
- A safe and effective vaccine against HBV is available. The age at which the person became infected is crucial in determining the natural history of HBV. Chronic active hepatitis B may progress to cirrhosis and hepatocellular carcinoma, which is mitigated with effective therapy. For people who develop chronic active hepatitis B, treatment is effective.
- HCV is transmitted primarily through blood-to-blood contact. The sharing of equipment during injecting drug use is the most common mode of transmission in Australia. The majority (75%) of individuals exposed to HCV develop chronic HCV infection. Some individuals with chronic HCV infection will develop symptoms such as fatigue and nausea. A small proportion of individuals will progress to liver failure or hepatocellular carcinoma. New oral direct acting antiviral (DAA) therapies are highly effective at eradicating HCV infection.
- Early diagnosis and treatment of non-viral STIs are effective interventions for the population control of STIs generally, while suppressive antiviral therapy for genital herpes decreases onward transmission of this virus and may indirectly assist in reducing the spread of HIV.
### TABLE 1.1 Sexually transmissible infections (i.e. those capable of sexual transmission)

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<tr>
<th>Sexually transmissible infection</th>
<th>Causative micro-organism</th>
<th>Mode of sexual transmission</th>
<th>Groups most commonly affected</th>
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<tbody>
<tr>
<td><strong>BACTERIA</strong></td>
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<td></td>
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<tr>
<td>bacterial vaginosis</td>
<td>Gardnerella vaginalis,</td>
<td>unknown</td>
<td>WSW, but also any sexually</td>
</tr>
<tr>
<td>(probably NOT a true STI)</td>
<td>Atopobium vaginae,</td>
<td></td>
<td>active woman</td>
</tr>
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<td></td>
<td>Mobiluncus sp and other</td>
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<td></td>
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<td></td>
<td>anaerobic bacteria</td>
<td></td>
<td></td>
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<tr>
<td>chancroid</td>
<td>Haemophilus ducreyi</td>
<td>genital skin-to-skin and</td>
<td>individuals who have</td>
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<td></td>
<td></td>
<td>mm-to-mm contact</td>
<td>unprotected sex in</td>
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<td></td>
<td></td>
<td></td>
<td>endemic areas</td>
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<tr>
<td>donovanosis</td>
<td>Klebsiella granulomatis</td>
<td>uncertain</td>
<td>remote Indigenous</td>
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<td></td>
<td></td>
<td></td>
<td>communities in Australia</td>
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<tr>
<td>enteric infections</td>
<td>Campylobacter spp</td>
<td>oral faecal contamination</td>
<td>mostly MSM</td>
</tr>
<tr>
<td></td>
<td></td>
<td>during sex</td>
<td></td>
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<tr>
<td></td>
<td>Shigella spp</td>
<td>oral faecal contamination</td>
<td>mostly MSM</td>
</tr>
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<td></td>
<td></td>
<td>during sex</td>
<td></td>
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<tr>
<td></td>
<td>Salmonella spp</td>
<td>oral faecal contamination</td>
<td>mostly MSM</td>
</tr>
<tr>
<td></td>
<td></td>
<td>during sex</td>
<td></td>
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<tr>
<td></td>
<td>Yersinia enterocolitica</td>
<td>oral faecal contamination</td>
<td>mostly MSM</td>
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<td></td>
<td></td>
<td>during sex</td>
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<tr>
<td>chlamydia infection</td>
<td>Chlamydia trachomatis</td>
<td>genital, rectal and</td>
<td>all sexually active people</td>
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<td></td>
<td></td>
<td>oropharyngeal mm-to-mm</td>
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<td></td>
<td>serovars D-K</td>
<td>contact</td>
<td></td>
</tr>
<tr>
<td>lymphogranuloma venereum (LGV)</td>
<td>Chlamydia trachomatis</td>
<td>genital and rectal skin-to-</td>
<td>MSM and individuals who</td>
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<tr>
<td></td>
<td></td>
<td>skin and mm-to-mm contact</td>
<td>have unprotected sex in</td>
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<tr>
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<td>serovars L1-L3</td>
<td></td>
<td>endemic areas</td>
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<tr>
<td>mycoplasma infection</td>
<td>Mycoplasma genitalium</td>
<td>genital mm-to-mm contact</td>
<td>probably all sexually active</td>
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<td></td>
<td></td>
<td></td>
<td>people</td>
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<tr>
<td></td>
<td>Mycoplasma hominis</td>
<td>role uncertain</td>
<td>role in genital infection</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>uncertain</td>
</tr>
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<td>neisseria infection (gonorrhoea)</td>
<td>Neisseria gonorrhoeae</td>
<td>genital, rectal and</td>
<td>all sexually active people</td>
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<td></td>
<td></td>
<td>oropharyngeal mm-to-mm</td>
<td></td>
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<td></td>
<td></td>
<td>contact</td>
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<tr>
<td>urethritis, pharyngeal colonization</td>
<td>Neisseria meningitidis</td>
<td>oropharyngeal mm-to-urethral mm (rarely)</td>
<td>mostly, but not exclusively, MSM</td>
</tr>
<tr>
<td>ureaplasma infection</td>
<td>Ureaplasma urealyticum</td>
<td>genital mm-to-mm contact</td>
<td>probably all sexually active</td>
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<tr>
<td></td>
<td>(some subtypes)</td>
<td></td>
<td>people</td>
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<tr>
<td>syphilis</td>
<td>Treponema pallidum</td>
<td>genital, rectal and</td>
<td>all sexually active people</td>
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<td></td>
<td></td>
<td>oropharyngeal mm-to-mm</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>and skin-to-skin contact</td>
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<td><strong>ECTOPARASITES</strong></td>
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<tr>
<td>pubic lice</td>
<td>Pthirus pubis</td>
<td>close body contact, sharing</td>
<td>everyone</td>
</tr>
<tr>
<td>scabies</td>
<td>Sarcoptes scabiei</td>
<td>a bed</td>
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<td><strong>NEMATODES</strong></td>
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<tr>
<td>thread worms</td>
<td>Enterobius vermicularis</td>
<td>oral faecal contamination</td>
<td>predominantly MSM</td>
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<td>during sex</td>
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<td><strong>PROTOZOA</strong></td>
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<td>enteric infections</td>
<td>Entamoeba spp</td>
<td>oral faecal contamination</td>
<td>MSM</td>
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<td></td>
<td></td>
<td>during sex</td>
<td></td>
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<tr>
<td>Giardia duodenalis</td>
<td></td>
<td>oral faecal contamination</td>
<td>MSM</td>
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<td></td>
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<td>during sex</td>
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</tbody>
</table>
This chapter describes the blood-borne viral and sexually transmitted micro-organisms, specifically focusing on their biology, transmission, pathogenesis and natural history. It also provides an introduction to the principles of therapy and discusses the effects of therapy on the natural history of each of these infections. This chapter will discuss only the following STIs as representative of the group as a whole. These six infections are included because of their serious long-term sequelae or because they are common in Australia and New Zealand:

- Genital chlamydial infection (including lymphogranuloma venereum [LGV])
- Genital herpes (herpes simplex virus, HSV)
- Genital warts (human papillomavirus, HPV)
- Gonorrhoea (*Neisseria gonorrhoeae*)
- Syphilis (*Treponema pallidum*)
- Trichomoniasis (*Trichomonas vaginalis*)

In addition, there will be a brief discussion about bacterial vaginosis because it is very common. Bacterial vaginosis may not be a true STI as it can occur in celibate women on rare occasions and treating the sexual partners of women with bacterial vaginosis has no effect on recurrence rates in index patients.

<table>
<thead>
<tr>
<th>Sexually transmissible infection</th>
<th>Causative micro-organism</th>
<th>Mode of sexual transmission</th>
<th>Groups most commonly affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>trichomoniass</td>
<td><em>Trichomonas vaginalis</em></td>
<td>mm-to-mm contact during penovaginal sex</td>
<td>heterosexually active people</td>
</tr>
<tr>
<td><strong>VIRUSES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>adenoviral urethritis</td>
<td><em>Adenoviruses</em></td>
<td>genital and oropharyngeal mm-to-mm contact</td>
<td>all sexually active people</td>
</tr>
<tr>
<td>cytomegalovirus infection</td>
<td><em>Cytomegalovirus (CMV)</em></td>
<td>oral mm-to-mm contact, saliva exchange</td>
<td>all sexually active people</td>
</tr>
<tr>
<td>infectious mononucleosis</td>
<td><em>Epstein-Barr virus (EBV)</em></td>
<td>oral mm-to-mm contact, saliva exchange</td>
<td>all sexually active people</td>
</tr>
<tr>
<td>genital herpes</td>
<td><em>Herpes simplex virus types 1 and 2 (HSV-1 and HSV-2)</em></td>
<td>genital, rectal and oropharyngeal skin-to-skin and mm-to-mm contact</td>
<td>all sexually active people</td>
</tr>
<tr>
<td>genital human papillomavirus infection (genital warts and squamous intraepithelial lesions - SIL)</td>
<td><em>Human papillomavirus (HPV)</em> (many types, but especially 6 and 11 for genital warts, and 16 and 18 for SIL)</td>
<td>genital, rectal, mouth and oropharyngeal skin-to-skin and mm-to-mm contact and oral faecal contamination during sex</td>
<td>all sexually active people</td>
</tr>
<tr>
<td>hepatitis A</td>
<td><em>Hepatitis A virus (HAV)</em></td>
<td>oral faecal contamination during sex</td>
<td>predominantly MSM</td>
</tr>
<tr>
<td>hepatitis B</td>
<td><em>Hepatitis B virus (HBV)</em></td>
<td>exchange of body fluids during sex</td>
<td>all sexually active people</td>
</tr>
<tr>
<td>hepatitis C (rare)</td>
<td><em>Hepatitis C virus (HCV)</em></td>
<td>blood exchange during sex</td>
<td>potentially all sexually active people, but rare*</td>
</tr>
<tr>
<td>human immunodeficiency virus infection</td>
<td><em>Human immunodeficiency virus types 1 and 2 (HIV-1 and HIV-2)</em></td>
<td>exchange of body fluids during sex</td>
<td>all sexually active people</td>
</tr>
<tr>
<td>Kaposi’s sarcoma (KS)</td>
<td><em>Human herpes virus 8 (HHV-8)</em></td>
<td>uncertain, probably exchange of body fluids</td>
<td>predominantly MSM</td>
</tr>
<tr>
<td>molluscum contagiosum</td>
<td><em>Molluscum contagiosum</em> (pox) virus (MCV)</td>
<td>direct skin-to-skin contact</td>
<td>all sexually active people</td>
</tr>
<tr>
<td><strong>YEASTS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>candidiasis</td>
<td><em>Candida spp</em> (ubiquitous commensals, only incidentally sexually transmitted)</td>
<td>genital mm-to-mm contact</td>
<td>all sexually active people</td>
</tr>
</tbody>
</table>

* Increased sexual transmission of HCV in HIV-positive MSM

mm = mucous membrane
MSM = men who have sex with men
WSW = women who have sex with women
**Biology (virology and microbiology)**

**HIV**

The manifestations of HIV were first apparent in the early 1980s when an epidemic of unexplained cases of immunodeficiency was reported in the western world. Evidence suggested the cause to be a transmissible agent, and in 1984 the agent was confirmed to be a retrovirus now known as human immunodeficiency virus (HIV). Human infection may date back to the early part of the twentieth century and the virus may have originally been transmitted zoonotically to humans from primates in Africa.

HIV is a single-stranded ribonucleic acid (RNA) virus. It has an outer envelope that surrounds two copies of single-stranded RNA as well as a number of viral proteins. From its outer envelope protrudes the 120 glycoprotein (gp 120). The HIV replication cycle commences when gp 120 attaches to the CD4 receptor and the chemokine co-receptor CCR5. (These receptors are expressed on the surface of the CD4 lymphocyte, the cell that HIV predominantly infects). Attachment precipitates the fusion of the membranes of virus and cell via the HIV envelope 41 glycoprotein (gp 41), allowing the virus to enter the cell. The RNA then undergoes reverse transcription, a process whereby RNA is converted to deoxyribonucleic acid (DNA) using the viral-encoded reverse transcriptase. The resulting viral DNA, called the provirus, migrates to the nucleus and integrates into the host chromosome.

The provirus acts as a template to allow production of messenger RNA to produce the components of new virus particles, including the RNA genome of new virions. The viral proteins are processed and cleaved by another virus-specific enzyme known as HIV protease. Viral proteins and RNA are then assembled and bud from the cell membrane, forming mature HIV particles that can infect other cells. Some of the CD4 cells are irreparably damaged by HIV infection. Premature cell death of damaged CD4 cells in part contributes to the immunosuppression characteristic of advanced HIV disease.

**HBV**

HBV is a non-cytopathic virus and contains a partially double-stranded DNA genome. This virus predominantly infects hepatocytes and belongs to the hepadnavirus family. HBV has an outer envelope containing hepatitis B surface antigen (HBsAg) and a core containing hepatitis B core antigen (HBcAg). Excess HBsAg is produced as sub-viral particles which circulate in the blood and permit serological diagnosis of HBV. The core contains the genomic DNA as well as the viral-encoded DNA polymerase, which is detected in liver tissue. HBV also produces hepatitis B e antigen or HBeAg, which is secreted into the blood and is detected by serological assay. The presence of circulating HBeAg and serum HBV DNA is indicative of ongoing viral replication and increased infectivity. Resolution of HBV infection is accompanied by clearance of HBeAg and HBsAg and seroconversion to anti-HBe-positivity (anti-HBe+) and anti-HBs-positivity (anti-HBs+).

Soon after entering the hepatocyte, the genomic DNA is converted in the nucleus to a form known as supercoiled or covalently closed circular (ccc) DNA. This serves as a template to yield two types of RNA: a pregenomic RNA that ultimately undergoes reverse transcription to yield DNA for progeny virus and messenger RNA for structural proteins. The former is assembled into mature virions that are then released from the cell.

In long-term, chronic infection, HBV DNA may integrate into the host cell genome but integration is typically incomplete and a full life cycle cannot occur from these integrated sequences. Viral integration does play a role in the development of hepatocellular carcinoma, especially in the setting of cirrhosis. Supercoiled HBV DNA in the liver cell nucleus is long-lived and resistant to all current antiviral therapies, resulting in lifelong chronic infection.

**HCV**

HCV is a single-stranded, enveloped RNA virus belonging to the flavivirus family. It causes most cases of what was previously known as non-A, non-B hepatitis. HCV was discovered when infected serum was injected into a number of chimpanzees, whose sera were then used to identify a clone that reacted with an infected serum panel from patients with non-A, non-B hepatitis. This finding ultimately formed the basis of the first antibody test for detection of HCV. The virus has only recently been cultivated in cell culture systems.

The hepatitis C virus is composed of envelope glycoproteins in a bilayer that contain the core proteins and RNA. Following entry into the cell, the host cell ribosomes translate the HCV RNA into a large polypeptide, which is cleaved and processed by both host cellular and virus-specific (NS-2 and NS-3) enzymes. The synthesis of the new viral RNA occurs in a structured replication complex that includes: nonstructural (NS), NS4A, (protease complex), NS4B, NSSA NSSB (polymerase). The viral polymerase/replicase (NS5B) copies the viral RNA in the cytoplasm and, as soon as a pool of progeny RNA molecules and core proteins is present, assembly of the nucleocapsids occurs. Mature HCV virions then develop and bud through the plasma membrane. The new direct acting antiviral (DA) therapies are specifically focused on the nonstructural NS3/4A protease, NS4B and NS5B polymerase to prevent viral replication.

**Chlamydia trachomatis**

*Chlamydia trachomatis* is a common human pathogen divided into 15 different serovars. Serovars A, B, Ba and C cause trachoma; serovars D to K cause genital (and
sometimes conjunctival) infection; serovars L1-L3 are associated with lymphogranuloma venereum (LGV) and tend to be more virulent and invasive showing a predilection for lymphatic vessels and tissue. C. trachomatis is a bacterium, but an obligate intracellular one, so it can only be isolated and grown in suitable host cells. There are two main structures in the life cycle of C. trachomatis: the elementary body (EB) and the reticulate body (RB). The EB is a rigid-walled structure packed with DNA and is the infectious particle. It infects a potential host cell by adhering to its surface.

The EBs enter the cell by endocytosis and soon begin the second phase of their life cycle as metabolically active reticulate bodies. The RBs use adenosine triphosphate (ATP) derived from the host cell to replicate by binary fission, each producing several hundred progeny. The RBs become larger and form inclusions in the cytoplasm of infected cells which can be detected by staining (e.g. with iodine). A microscopist can see these intracytoplasmic inclusions in some infected cells draped around the nucleus like a cloak; the word ‘chlamys’ is Greek for ‘cloak’, giving the micro-organism its name. After about 20 hours some of the RBs undergo reorganisation to form new EBs that, with cell lysis, burst out of the old cell ready to infect other susceptible cells. The whole life cycle takes about 72 hours. Chlamydial disease confined to epithelial surfaces tends to produce only a mild immune response, whereas more serious sequelae (e.g. salpingitis) and systemic disease can only be isolated and grown in suitable host cells. The EBs use adenosine triphosphate (ATP) derived from the host cell to replicate by binary fission, each producing several hundred progeny. The RBs become larger and form inclusions in the cytoplasm of infected cells which can be detected by staining (e.g. with iodine). A microscopist can see these intracytoplasmic inclusions in some infected cells draped around the nucleus like a cloak; the word ‘chlamys’ is Greek for ‘cloak’, giving the micro-organism its name. After about 20 hours some of the RBs undergo reorganisation to form new EBs that, with cell lysis, burst out of the old cell ready to infect other susceptible cells. The whole life cycle takes about 72 hours. Chlamydial disease confined to epithelial surfaces tends to produce only a mild immune response, whereas more serious sequelae (e.g. salpingitis) and systemic disease such as LGV, elicit a vigorous antibody reaction.4

Herpes simplex virus--types 1 and 2
(HSV-1 and HSV-2)

The herpes simplex viruses are double-stranded DNA viruses, members of the human herpesvirus family and are exceptionally successful human pathogens. Like the varicella-zoster virus, HSV-1 and HSV-2 are neurotropic viruses but have the ability to cause infection in many other cell types. HSV-1 and HSV-2 are widely prevalent and tend to cause only mild and self-limited disease. A characteristic which they share with other members of the human herpesvirus family is the ability to establish latent infection, so that they are able to persist throughout the life of the host. During latency, the genome of the invading virus is maintained in stable form in the infected neural cell with no production of progeny virus for variable periods of time and no apparent cytotoxic effects. Periodically, reactivation of virus replication occurs with virus migrating back down axons to surface sites. The clinical severity of herpes simplex infections and the host’s capacity to control viral replication depends very much on cell-mediated immunity, although humoral immune mechanisms also play an important part5

Human papillomavirus (HPV)
The human papillomaviruses are small DNA viruses which induce proliferation of epithelial cells with the production of papillomas. More than 35 HPV types infect genital skin and mucous membrane. So far HPV has not been grown in tissue culture and typing is dependent on detection of the genome by molecular cloning and sequencing. Genital HPV types are divided into high risk and low risk depending on their potential to promote the development of squamous cell cancers in infected cells. Types 6 and 11 are low-risk types as they are rarely associated with cancers and tend to cause typical genital warts. Types 16, 18, 31, 33, 35 and 45 are high-risk types. About 50% of invasive squamous cell cancers of the cervix carry the HPV DNA of type 16. HPV has a highly significant role in the development of ano-genital cancers whether they be cervical, vulval, penile or anal cancers.5 Genital HPV is ubiquitous in the community; an American study published in 2006 found that in women aged 18 to 25 years, the overall HPV frequency of detection was 26.9% and there was detectable high-risk HPV DNA in 20%, while another study from a very broad cross-section of the population in the USA found that the prevalence of HPV DNA in young women aged 14 to 24 years was 33.8%.6 Infection usually occurs in adolescence and young adulthood soon after the beginning of sexual activity. Because of the asymptomatic nature of much HPV genital infection and the lack of any specific antiviral treatment, up until now control of high-risk HPV type infection in the community has depended entirely on cervical cancer screening and follow-up with surgical ablation of high grade squamous intraepithelial lesions (SIL). The development and licensing in Australia of two prophylactic vaccines (Gardasil and Cervarix) for girls and young women against the commonest high-risk and low-risk genital HPV types is therefore a considerable step forward, although it should be noted that only one of the vaccines (Gardasil) is active against HPV types causing genital warts9

Australian immunisation programs now recommend Gardasil vaccination for all boys and girls at age 11-13 years. Vaccination programs for young women began in April 2007 and there is already evidence of lower rates of genital warts in the vaccinated cohort with reductions of 85.3% in 15-24 year old females and 70.6% in men from the same age group. Reductions in rates of cervical and anal cancer are also expected to fall in the vaccinated groups with time.10

Neisseria gonorrhoeae

Neisseria gonorrhoeae, or the gonococcus, the causative agent of gonorrhoea, is perhaps the best known sexually transmitted agent and has caused considerable morbidity in human beings since the
earliest recorded history. Gonococci are gram-negative bacteria which characteristically grow in pairs as diplococci. Under a light microscope they are indistinguishable from meningococci and indeed meningococci, on occasions, have been demonstrated to cause urethritis – hence the need for accurate microbiological identification of urethral isolates. Like C. trachomatis, gonococci have a predilection for the mucous membrane surfaces of the urethra, endocervical canal, rectum, pharynx and conjunctiva. Sequelae of untreated infections can be serious and severe. Some uncommon strains of gonococci cause little inflammatory response on mucosal surfaces but have the ability to invade, leading to bacteraemia and more systemic disease.

Gonococci possess surface molecules called pili which are largely responsible for adhesion to mucosal surfaces and also for invasion into the submucosa. Pili also serve as targets for host defences but have an amazing ability to undergo swift antigenic change. This accounts for the almost complete absence of acquired natural immunity against attacks of mucosal gonorrhoea. A person can be successfully treated for gonococcal urethritis or cervicitis today and if exposed to infection again tomorrow will be completely susceptible to re-infection. A great deal is known now about the pathogenicity of N. gonorrhoeae and associated host-bacterial interactions. Despite all this accumulated knowledge, gonorrhoea remains a considerable problem around the world. There has been a notable lack of progress towards vaccine development and the gonococcus has an extraordinary capacity to acquire resistance to antibiotics very rapidly. This ability continues to present a formidable challenge. Medical science continues to be only one step ahead of this doughty Darwinian survivor.11

**Treponema pallidum**

*Treponema pallidum* is the bacterial agent which causes syphilis, another well-known and once feared STI. *T. pallidum* is a spirochaetal organism related to *Borrelia* and *Leptospira*. It is a long, thin, tightly coiled bacterium, just beyond the resolution of the light microscope, although it can be demonstrated in a microscope with a dark field condenser. Here, in a wet preparation, it distinguishes itself from other spirochaetes by its regular tight spirals and its characteristic motility. Both the corkscrew shape and the mobility of the organism play important roles in its invasion and dissemination. The body mounts an immune response against invading treponemes, both humoral and cell mediated, and many of the unique clinical features of syphilis are due to the immune response. Bacteria are able to establish latency in lymphatic and splenic tissue and during this period of latency, which may last for many years in untreated patients, the person with the infection will be resistant to reinfection from a new challenge with *T. pallidum*.12

**Bacterial vaginosis**

Bacterial vaginosis is a common, complex clinical syndrome of which the characteristic feature is an alteration in the normal vaginal flora. Normal lactobacilli are absent or greatly reduced and large numbers of gram-variable, small, mostly anaerobic micro-organisms including *Gardnerella vaginalis*, *Atopobium vaginae*, *Mobiluncus sp*, and *Prevotella sp* replace them. Some of these organisms are highly motile and tend to cluster around shed epithelial cells in vaginal fluid. Microscopists describe such cells as ‘clue cells’ and they are a hallmark of bacterial vaginosis. The normal acidic milieu of the vagina is lost with the pH rising to 7 or above. The cause of this curious condition is still unknown and while it has some features in common with other STIs, namely strong association with sexual activity, the lack of any similar condition or conjunction of micro-organisms in males, whether symptomatic or not, makes its classification as an STI suspect in our present state of ignorance. However, the condition is common in women who have sex with women (WSW) and in this group bacterial vaginosis certainly seems to be acting like an STI.14 Bacterial vaginosis is also rarely seen before the onset of penetrative vaginal sex which further supports the view that it may behave as an STI. In countries with high prevalence of HIV infection in the heterosexual population, bacterial vaginosis is also associated with increased risk of both transmission and acquisition of HIV infection.

**Quasispecies and hypermutability of blood-borne viruses**

The replicate enzymes of all three blood-borne viruses, the HIV reverse transcriptase, the HBV DNA polymerase and the HCV RNA polymerase, are hypermutable. Mutation, particularly under immunological and therapeutic pressure, leads to the presence in a given individual of a number of closely related, but genetically distinct, viral variants known as quasispecies. The emergence of quasispecies is the likely reason why infection with these viruses results in chronic infection in most individuals despite a host immune response. Each one of the virus-specific enzymes previously discussed is the focus of intense research to develop potent and
selective inhibitors of key viral functions, which could result in significant gains in managing the health of people with persistent infection of these viruses.\textsuperscript{15}

**Transmission**

While each blood-borne virus has distinct transmission patterns, HIV, HBV and HCV can all be transmitted parenterally through the sharing of injecting equipment, needle-stick injuries or piercing and tattooing with contaminated equipment. On the other hand, efficiency of sexual transmission differs markedly between viruses. STIs are by definition transmitted through sexual contact but the precise mode of transmission varies from infection to infection. Different sexual activities favour the transmission of different sexually transmissible agents: individuals don’t acquire pubic lice and gonorrhoea in quite the same way (see Table 1.1).

**HIV**

HIV is predominantly transmitted sexually, with efficiency being greatest through receptive anal intercourse. In Australia, transmission is most commonly seen in homosexual men, whereas in developing countries, especially in Africa, HIV is predominantly acquired through vaginal intercourse. Transmission through injecting drug use is uncommon in Australia, accounting for 4% of HIV cases, but is particularly prevalent in parts of Europe and Asia (including countries of the former Soviet Union) and the USA. Transmission by blood products largely occurred before the introduction of antibody screening in 1985 in Australia and was responsible for the high incidence of HIV among multiply-transfused people, such as those with haemophilia. It is now exceedingly rare in countries where blood is screened. Transmission by needle-stick injury occurs in 0.3% of exposures from individuals with HIV infection. Perinatal transmission occurs in 20–45% of infants born to mothers with HIV infection, but this rate can be reduced to 1–2% with the administration of antiretroviral therapy during pregnancy, labour and after delivery, and other interventions, such as caesarean section and avoidance of breast-feeding.\textsuperscript{16} In Australia there have been 34,029 new diagnoses of HIV infection, but this rate may be higher in women who have HIV infection is usually advocated, although its role in reducing perinatal transmission in women with HIV/HCV co-infection or high levels of viraemia. Elective caesarean section in women with HIV/HCV co-infection is usually advocated, although its role in reducing perinatal transmission in women with HCV mono-infection is unclear and is generally not recommended as routine in this context.\textsuperscript{21, 22}

**HBV**

Most HBV cases result from perinatal transmission, which accounts for high prevalence in people from endemic countries, particularly China and South East Asian and Pacific nations. Transmission is effectively prevented by HBV vaccination and administration of hepatitis B immunoglobulin (HBIG) to newborns of hepatitis B surface antigen positive women, but such programs are not currently available in many developing countries where most cases occur.

Among adults, HBV transmission is predominantly via sexual contact and injecting drug use. In Australia in 2012, the overall prevalence rate of HBV infection was 1.02% with an estimated 218,000 living with HBV infection.\textsuperscript{18} The risk of transmission by percutaneous exposure such as a needle-stick injury is approximately 30% if the person with HBV infection has replicative disease (defined as HBV DNA detectable by hybridisation assay or HBsAg positive and HBeAg positive), compared with 3% for those with HBV infection with non-replicative disease (that is, people without HBeAg or HBV DNA but with HBsAg).\textsuperscript{3}

**HCV**

HCV transmission is predominantly parenteral. Hepatitis C transmission in Australia remains predominately among people with a recent history of injecting drug use. In 2012, an estimated 230,000 people were living with chronic hepatitis C infection nationally, including 58,000 with moderate to severe liver disease.\textsuperscript{19} Among particular immigrant populations, poor infection control practices during procedures such as vaccination (European and Asian) and chemoprophylaxis programs for schistosomiasis (Egyptian) may have been responsible for many cases. The role of sexual transmission is still controversial. If sexual transmission of HCV does occur, it is at a very low level that makes it inappropriate to recommend routine safe sex practices among long-term monogamous couples.

Sexual transmission is likely to be more efficient, however, where there is HIV co-infection and high HCV viral load.\textsuperscript{20} Risk of sexual transmission may also be increased when blood is present in the genital tract, such as during menstruation. Perinatal transmission occurs in approximately 5% of deliveries, although this rate may be higher in women who have HIV co-infection or high levels of viraemia. Elective caesarean section in women with HIV/HCV co-infection is usually advocated, although its role in reducing perinatal transmission in women with HCV mono-infection is unclear and is generally not recommended as routine in this context.\textsuperscript{21, 22}

**Chlamydia trachomatis**

*Chlamydia trachomatis* tends to infect columnar epithelium rather than squamous epithelium. Direct mucous membrane-to-mucous membrane contact facilitates transmission and chlamydial elementary bodies in infected genital secretions and discharges readily seed uninfected mucous membrane and cause infection in columnar cells. During birth, transmission occurs from a mother with cervical chlamydial infection to the child very efficiently (overall risk is 50–75%).
Indirect transmission of chlamydial infection by fomites appears to be extremely uncommon. Lymphogranuloma venereum (LGV) serovars are transmitted similarly by direct surface-to-surface contact or contamination of susceptible genital surfaces by contaminated secretions. An LGV outbreak in 2010 in men who have sex with men (MSM) appeared to occur predominantly via anal intercourse with multiple partners, fisting and use of contaminated sex toys. For all practical purposes, transmission of the genital serovars of C. trachomatis is sexual and vertical only, and conjunctival infection in the adult results from auto-inoculation with infected secretions from genitals to eye by the patient’s own fingers.23

HSV-1 and HSV-2

Sexual transmission is a highly significant method of transmission of these viruses.24 However, most people with oral and labial cold sores acquire infection with HSV-1 in childhood usually by being kissed on or near the mouth by family or relatives. When they grow up and become sexually active, they can pass HSV-1 on to various anatomical sites in one or other of their sexual partners by kissing or by oro-vulval, oro-genital or oro-anal sex. A significant proportion of ano-genital sexual partners by kissing or by oro-vulval, oro-genital or oro-anal sex. A significant proportion of ano-genital herpes is due to HSV-1 infection (30% or more in many studies).25

All that is required for transmission of HSV-1 or HSV-2 is for the virus from a person with the infection to come in contact with a mucosal surface (vaginal, cervical, rectal, pharyngeal, buccal, labial, conjunctival) or a slightly abraded skin surface anywhere on the body of a susceptible person.

People never exposed to either virus are most susceptible and may develop a severe primary attack when infection is acquired; those with antibodies to one or other HSV, demonstrating previous exposure, are still capable of obtaining an infection by the alternative virus but have some degree of protection and may suffer a less severe infection. The two viruses exhibit different tropism for anatomic sites; HSV-1 can infect both oral and genital sites but tends to thrive better in the mouth area, in that it reactivates more commonly there with viral shedding and sometimes with clinically obvious recurrences; HSV-2 can also infect both oral and genital sites but thrives better in the genital region.

However, surprisingly, a study published in 2006 of men seropositive for HSV-2 (about half of whom were also HIV positive), showed that 40% of the men shed HSV-2 from both genital and oral sites. Oral shedding was always asymptomatic, it usually occurred at the same time as genital shedding and it occurred more commonly in HIV-positive men.26

All these facts explain why HSV infection is so common in sexually active people and why control of the spread of infection in communities presents such a challenge. Transmission rates can be reduced with careful and consistent condom use and suppressive therapy with antiviral drugs for those who suffer frequent recurrences. Only an effective vaccine will make a significant impact on the problem of herpes simplex virus infection at a population level.

All forms of sexual contact can lead to transmission of HSV-1 or HSV-2 and mother-to-child transmission can occur at the time of birth. Neonatally acquired herpes can be a devastating and life-threatening disease. Fortunately mothers who are already have the herpes infection provide their own antibodies transplacentally which substantially protect the infant from infection, even if HSV is shed by the mother around the time of delivery. However, if a pregnant woman acquires the infection in the ano-genital region with HSV-1 or HSV-2 in the last trimester of pregnancy and fails to develop significant HSV antibody levels (as shown by testing before delivery), the baby is at significant risk of acquiring neonatal herpes. In this situation obstetricians recommend a caesarean section birth.4

Human papillomavirus

Sexual contact of all types accounts for all genital HPV infection. This includes the low-risk HPV types which result in the growth of genital warts and the high-risk HPV types that are associated with ano-genital cancers. Genital HPV is a sexually transmitted infection demonstrated by the now well established fact that women without a present or prior sex partner tend to have a very low yield of HPV DNA in cervico-vaginal secretions, women with only one sex partner have a slightly higher yield and women with a history of more than one partner have a substantially higher yield.

Each time a person acquires a new sex partner, that person’s risk of acquiring genital HPV increases considerably. The consequence is that most sexually active adults have acquired one or more of the plethora of genital HPV types by the time they reach their fourth decade. Direct skin-to-skin, skin-to-mucous membrane and mucous membrane-to-mucous membrane contact is all that is required for transmission to occur. HPV infection with one or more genital types of HPV does occur around the mouth and lips as a result of oral sex but is only a problem in an immunosuppressed patient. Similarly mother-to-child transmission of HPV at the time of birth sometimes does occur resulting in genital or laryngeal infection in the infant, but fortunately these infections rarely cause clinical problems.6 Laryngeal papillomatosis, while extremely rare, can be a very significant clinical problem in young children.
Neisseria gonorrhoeae

The gonococcus is highly infectious but is a fragile organism outside the human body, being poorly resistant to environmental changes such as heat and drying. Transmission is therefore almost exclusively by sexual contact or from mother to infant at the time of birth. Transmission by fomites is extremely unlikely. Gonorrhoea transmission is fairly efficient, with a prevalence of infection of 50–90% in female sexual contacts of a man with urethral gonorrhoea.

Transmission is by direct mucous membrane-to-mucous membrane contact or via infected genital secretions on a susceptible mucosal surface. Transmission from male to female via vaginal sex is slightly more efficient than from female to male and, similarly, transmission from the male partner to the receptive partner in anal sex is more efficient than vice versa. The pharyngeal mucosa is readily infected from an infected urethral meatus via oral sex while transmission from infected pharynx to urethra is less common but well documented, especially among MSM. Adult gonococcal conjunctivitis (a sight-threatening infection) is almost always acquired by auto-inoculation via the person’s own fingers from his or her infected genitals, while neonatal conjunctivitis is acquired by direct inoculation of the baby’s conjunctivae during transit through the infected maternal endocervical canal.

Treponema pallidum

Syphilis is only acquired by sexual contact or by transplacental transmission of Treponema pallidum, i.e. from a mother with the infection to her foetus in utero. An old study suggested that the risk of acquiring syphilis from an infectious partner was about 30% per sexual exposure. A woman with syphilis infection has a potential risk of transmitting syphilis transplacentally during many years of untreated infection (8–10 years at least, although the risk decreases with every passing year). However, people with syphilis seem only able to transmit it to sexual partners during the first 2 years of untreated infection. This suggests that for sexual transmission to occur, moist mucosal or cutaneous lesions (i.e. those that appear in primary and secondary syphilis) must be present, so that active treponemes on those surfaces of a person with the infection have an opportunity of reaching and penetrating moist mucosal or cutaneous surfaces of the sexual partner. Infectious lesions include the primary chancre and all the mucosal manifestations of secondary syphilis, e.g. snail track ulcers, condylomata lata, mucous patches and split papules. Even microscopic and non-clinically obvious mucosal lesions, as may occur in the vagina, the mouth, under the foreskin and perianally, in early latent syphilis, are infectious to sexual partners and probably account for most of the transmission that occurs in areas where syphilis is endemic.

Trichomonas vaginalis

Trichomonas vaginalis is transmitted almost exclusively by sexual contact. There has been a long debate about the possibility of transmission by contaminated fomites such as face cloths, towels and toilet seats. While it is true that the organism is harder than T. pallidum and N. gonorrhoeae and may survive 45 minutes or so outside the body, epidemiological evidence to support non venereal transmission is slim. T. vaginalis is not known to infect rectal mucosa, the conjunctiva or the pharynx. In this respect it differs from the gonococcus and C. trachomatis. Vaginal intercourse appears to be the main way trichomoniass is spread, with oral sex and anal sex having no part to play.

Bacterial vaginosis

The aetiology of this condition is unknown. Recent studies support sexual transmission of agents (known and unknown) plays a part in its aetiology.

Natural history

HIV

Following inoculation with HIV, there is a period of high-level viraemia associated with a reduction in the CD4 cell count. A host immune response then develops, partially controlling viral replication, but is unable to clear HIV from the body. A substantial proportion of patients (proportions in recent reports range from 50–92%) suffer a mononucleosis-like seroconversion illness characterised by fever, pharyngitis, lymphadenopathy, rash, splenomegaly and aseptic meningitis. Other patients with HIV infection are asymptomatic or suffer a more non-specific illness. These acute-phase effects then resolve as the immune system mounts an antiviral response that causes the viral load to decrease markedly. Simultaneously, there is a rebound increase in CD4 cell count to near baseline levels and the patient enters a period of clinical latency, although very high levels of viral replication continue, especially in the lymphoid compartment. The plasma HIV RNA plateaus to a constant level of viraemia known as the virological set point. If left untreated, the patient experiences a gradual decline in CD4 cell count, with a median loss of 80 cells per year. Progression to AIDS (the development of opportunistic infections or specific malignancies) occurs a median of 10 years after initial infection with HIV. At this time the CD4 cell count has usually fallen below 200 cells/µL and the patient is severely immunocompromised (see chapter 10, Figure 10.2).

HBV

HBV, by contrast, is almost exclusively an immune-mediated disease. The outcome of infection is largely determined by the age at which infection is acquired, which relates to the maturity of the immune response.
In endemic countries where infection occurs during birth (perinatal infection) or in early childhood (early horizontal infection), over 90% of HBV transmissions will become chronic (as defined by a persistence of HBsAg for more than six months), and clinical acute hepatitis rarely occurs. If, however, an individual is infected acquires the infection as an adult, chronic infection will occur in less than 5% of people, although almost half will manifest clinical features of acute hepatitis.

Occasionally, under the pressure of immune-mediated flares, HBV mutants are selected. These so-called precore (or HBeAg-negative) mutants fail to secrete HBeAg protein but still replicate, as evidenced by detectable HBV DNA in serum and elevated serum aminotransferase levels. HBeAg-negative infection is particularly prevalent in certain geographical areas, such as around the Mediterranean basin and in South East and northern Asia. In Australia, migrants from these regions frequently have infection with such variants.1

HCV

Unlike HBV, the immune response generated in adults who have newly acquired the hepatitis C infection is usually inadequate to effectively control viral replication. As a consequence, the majority of acute infections progress to chronic infection, defined as a positive HCV RNA in serum 6 months after the estimated date of infection (Chapter 11). The proportion of people estimated to clear acute hepatitis C varies between 25% and 40%, and clearance occurs more frequently in patients who are symptomatic or who become jaundiced. Models based on large longitudinal community-based cohorts estimate the risk of progression to cirrhosis to be 7% at 20 years and 20% at 40 years of infection.2 Estimates of hepatitis C-related mortality are 1% at 20 years and 4% at 40 years.29 Despite this, an increasing burden of advanced liver disease is anticipated within Australia. Currently, HCV is the most common indication for liver transplantation in Australia.

Factors associated with an accelerated risk of progression include older age at infection, male gender, heavy alcohol intake, co-infection with HBV and HIV and possibly obesity, linked to the presence of steatosis (fatty liver) on biopsy. The risk of liver failure in people with compensated cirrhosis is around 4–5% per year and the risk of hepatoma around 1–3% per year in Australia.

People who have chronic hepatitis C and normal liver function tests generally have very low rates of fibrosis progression. At present the majority of these patients are not routinely offered HCV therapy and are treated only in the context of clinical trials, as many new and more effective treatments are on the horizon.

Co-infection with HIV, HBV or HCV

Multiple blood-borne viral infections in the same individual can markedly alter the natural history of disease. For example, HBV has no adverse effect on HIV or the development of AIDS, but HIV does influence HBV and can be associated with accelerated development of cirrhosis and liver failure. The exact mechanism(s) of the pathogenesis of this co-infection are presently unknown but are probably due to virological (higher HBV viral load in co-infection) and host immunological (dysregulated immune responses) factors.

Individuals with HIV and HCV co-infection have higher HCV viral loads and a more rapid course to end-stage liver disease. This has been demonstrated by the correlation between declining CD4 cell counts and the increasing percentage of HCV-related hospital admissions and deaths among people with HIV and HCV co-infection.29

Chlamydia trachomatis

The natural history of genital chlamydia infection varies depending on whether infection is caused by the D to K serovars, or the L1 to L3 serovars.

D to K serovars

Primary sites of genital infection with D to K serovars of C. trachomatis in adults are mucosal surfaces lined by columnar epithelium, hence urethritis, endocervicitis, proctitis and pharyngitis can result, depending on the type of sexual activity. Most of these infections are mild and it is more likely that people with the infection remain asymptomatic for a considerable time rather than developing obvious symptoms and signs. In infants following mother-to-child transmission, primary sites of infection are the naso-pharynx, the conjunctivae and, more rarely, the vagina or urethra.

Genital D to K chlamydia infections can spread from their site of original infection. In women, cervical infection tends to spread upwards through the endometrium causing a mild endometritis with onward spread to the mucosal lining of the fallopian tubes with resulting salpingitis.

Infection can spread from the surface of the fallopian tubes into the surrounding peritoneum and supporting ligaments resulting in pelvic inflammatory disease (PID). Sometimes transcoelomic spread can result in perihepatitis (the Fitz-Hugh-Curtis syndrome). In men, ascending infection can result in epididymo-orchitis. In adults, both PID and epididymo-orchitis caused by C. trachomatis tend to be milder than similar gonococcal disease, but the potential for long-term damage in women (pelvic sepsis, tubo-ovarian abscess, infertility and increased risk of ectopic pregnancy) is equivalent in both infections: chlamydia PID is a more silent and insidious infection than the gonococcal variety. In infants, naso-pharyngeal infection is often a precursor to the development of pneumonitis.29
L1–L3 serovars (LGV)

Until quite recently lymphogranuloma venereum (LGV) remained an uncommon infection mostly seen in tropical and sub-tropical resource-poor countries. It was exceedingly rare in Australia, New Zealand and other Western countries. Tropical LGV has three stages:
- A primary ulcer on the genitals, usually of short-term duration
- A secondary stage characterised by systemic symptoms, inguinal lymphadenitis and sometimes a moderately severe proctitis
- A third stage with chronic sequelae: bubo formation often with rupture and discharging inguinal sinuses, lymphatic obstruction with genital elephantiasis and rectal stricture and fistula formation.

In 2003 and 2004 the first cases of rectal infection with an L2 serovar in MSM were identified in the Netherlands and subsequently more cases have been detected in most other Western countries with significant populations of homosexually active men, including in major Australian cities.

The vast majority of these cases have been characterised by moderately severe to severe rectal proctitis with systemic symptoms (fever and malaise) and little or (more often) no involvement of inguinal lymph nodes.31

Herpes simplex virus – types 1 and 2 (HSV-I and HSV-2)

Both HSV-1 and HSV-2 can cause genital herpetic infection. The most common scenario for genital infection with either of the herpes simplex viruses is for a person, during a sexual contact, to acquire the virus on a genital mucous membrane or cutaneous surface with either extremely mild symptoms or no symptoms at all marking the event. In the case of a person with no previous exposure to HSV-1 or HSV-2, and where a large dose of virus is acquired, within a few days of acquisition painful vesicles or blisters develop which rapidly break down to form shallow tender ulcerations. At the same time, draining lymph nodes become enlarged and tender and there may be systemic symptoms of fever and malaise. This is called primary genital herpes infection. It is distressing and uncomfortable in both men and women and may last up to 3 weeks before spontaneously remitting. There is a third group of people whose first contact with genital HSV (the initial attack) lies somewhere between the extremes of the severe primary outbreak and the entirely asymptomatic group.

The virus establishes latent infection in sensory nerve ganglia in the vicinity of the spinal cord and periodically reactivates with migration down the nerve fibres and intermittent release of infectious virions onto the surface—this is called viral shedding and is the cause of most onward transmission of HSV. All people with genital HSV infection undergo the same pattern of recurrent reactivation of latent virus and intermittent reappearance of infectious virions at a surface site. For some people, recognisable symptoms of blistering and ulceration accompany this reactivation; for others asymptomatic reactivation is the rule. Genital infection with HSV-2 is more likely to result in symptomatic recurrences than genital infection with HSV-1.

Neonatal HSV infection can manifest as lesions localised to the skin, eyes or mouth; an encephalitis; or a severe disseminated life-threatening infection.1

Human papillomavirus

There are still substantial gaps in knowledge about the natural history of genital HPV infection. Most infections are acquired in adolescence and early adult life and HPV infection shares characteristics with other STIs, namely: it is more common in those who commence sexual activity early, those who have frequent partner changes or multiple partners; and those whose partner has or has had frequent partner changes. However, few sexually active people avoid acquiring one or more of the genital HPV types during their lifetime. Studies in MSM and anal HPV infection show that MSM can continue to contract multiple HPV infections throughout life whereas in the female genital tract cervical infections with persistent carriage of oncogenic HPV strains appear to peak in the early 20s and then decline over 35 with resolution of infections in the majority of women unless there are changes in sexual partners. Persistent carriage of oncogenic HPV infection is what leads to cervical cancer. The outcomes of ano-genital infection with HPV include:
- Invisible infection where the only indication that infection has occurred is the presence of HPV DNA in epithelial cells, as detected by an appropriate test
- Cytological signs of infection as seen in a cytological smear or in material taken by biopsy (e.g. at colposcopy). Such cytological changes are in the form of low-grade or high-grade squamous intraepithelial lesions (SIL)
- Typical exophytic warts.

In general, HPV infections tend to resolve over time in immunocompetent people presumably reflecting increasing immune control over the virus, although local immune mechanisms in epithelium are still poorly understood. Genital HPV infection may persist for many years despite apparent complete clinical resolution and it is not uncommon for people who become immunosuppressed later in life to develop recurrences of ano-genital warts which had troubled them in their early adult life. In time many exophytic warts disappear even without treatment and most
low-grade and even high-grade SIL regress. However, a small percentage of SIL do develop into ano-genital cancers. This is much more likely to occur if the HPV types causing the lesions are high-risk types, the most common being types 16 and 18. On the other hand, exophytic warts caused by HPV types 6 or 11 appear to have virtually no potential to develop into cancer.

HPV vaccination will cause significant changes in the natural history of HPV infection with both reduction in incidence of new infections and in progression to malignant disease over time.

**Neisseria gonorrhoeae**

*Neisseria gonorrhoeae* targets exactly the same columnar cells in the mucous membrane of urethra, endocervix, rectum, pharynx and conjunctiva as does *Chlamydia trachomatis*. Within a few days the infection elicits a vigorous local immune response with the production of cytokines and the influx of large numbers of polymorphonuclear lymphocytes. Thus, most strains of gonorrhoea tend to produce visible signs of inflammation, i.e. meatitis, urethritis and cervicitis, although it is only the infection in the male urethra which usually results in early detectable symptoms of dysuria and purulent discharge. Infection at other sites is much less likely to cause readily recognisable symptoms, at least in the first few weeks. A small percentage of men seem to acquire urethral gonorrhoea asymptomatically, probably reflecting infection with less virulent strains or strains less well equipped to elicit a mucosal immune response; some of these strains are more likely to cause epididymo-orchitis than clinically obvious mucosal infection.

Untreated, *N. gonorrhoeae* invades the submucosa sometimes causing submucosal abscesses, spreads into adjoining glandular structures such as Bartholin’s, Skene’s and Littré’s glands with the potential for further abscess formation. A mild lymphadenitis in draining lymph nodes often accompanies acute infection. Gonorrhoea initiates an inexorable ascending infection to the fallopian tubes, surrounding ligaments and adjoining organs in women, causing an acute pelvic inflammatory disease. Less commonly, infection spreads to the epididymis and testis in men, causing an acute epididymo-orchitis. The infection eventually resolves, but, in the absence of early treatment, healing occurs with damaging scar tissue formation and fibrosis. In the urethra and the fallopian tubes such scarring can permanently interfere with normal function.

A small number of gonococcal strains has the potential to invade the blood stream causing bacteremia with systemic symptoms and disseminated skin and joint manifestations (disseminated gonococcal infection). In the neonate with infection from its mother at birth, gonorrhoea characteristically produces an acute sight-threatening conjunctivitis which is recognised 2 or 3 days after birth.

**Treponema pallidum**

The natural history of syphilis in an adult is divided into three stages—primary, secondary and tertiary.

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**FIGURE 1.3 Syphilis serology**

Source: By kind permission of B. Donovan

RPR: rapid plasma reagin  
TPHA: Treponema pallidum haemagglutination test  
FTA (Abs): fluorescent treponemal antibody absorption test  
EIA: enzyme immunoassay
A person without clinical signs or symptoms of syphilis, but having positive syphilis serology and no history of having been treated for syphilis, is said to have latent syphilis. By convention in Australia and the UK, primary and secondary syphilis and the first 2 years of latent infection are called early syphilis (i.e. the period during which syphilis is infectious by sexual contact), while tertiary, cardiovascular, neurosyphilis and latent infection beyond 2 years is called late syphilis. In the USA early syphilis refers only to the first 12 months of infection. Table 1.2 and Figure 1.3 describe the stages of syphilis in adults with a guide to accompanying serology results.

Primary syphilis (the chancre) is a self-limiting condition, with ulceration healing within a few weeks in untreated patients. Secondary syphilis is also self-limiting with clinical manifestations resolving over several weeks, although, in at least 25% of untreated people, relapses of secondary syphilis continue to occur over the first 2 years after infection. Tertiary syphilis, cardiovascular and neurosyphilis occur at a variable period of time after infection, from as short as 1 year through to 40 years later. Historical studies done on untreated patients indicate that only about 30% of those with syphilis develop these late manifestations of disease. In the other 70%, immune responses manage to control the infection. Co-infection with HIV may alter the natural history of syphilis.27

Table 1.2 Stages of syphilis in adults

<table>
<thead>
<tr>
<th>INFECTIONOUS</th>
<th>NON INFECTIOUS (except vertically)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EARLY (&lt;2 years)</td>
<td>LATE (&gt;2 years)</td>
</tr>
<tr>
<td>Early latent (asymptomatic)</td>
<td>Late latent (asymptomatic)</td>
</tr>
<tr>
<td>RPR: 1:8 or greater</td>
<td>Very variable, usually 1:4 or less – sometimes becomes non-reactive eventually</td>
</tr>
<tr>
<td>SPECIFIC TEST: Reactive</td>
<td>Reactive</td>
</tr>
</tbody>
</table>

| EARLY (<2 years) | LATE (>2 years) |
| Primary (chancre) | Tertiary (skin lesions, gummata) |
| May be non-reactive, but then increasing titre with time | Usually less than 1:16 |
| SPECIFIC TEST: Reactive (except very early in infection) | Reactive |

| EARLY (<2 years) | LATE (>2 years) |
| Secondary (rash, mucous membrane lesions, alopecia, lymphadenopathy) | Cardiovascular (aortitis) |
| 1:8 or greater (i.e. 1:16, 1:32, 1:64) | Usually less than 1:16 |
| SPECIFIC TEST: Reactive | Reactive |

| EARLY (<2 years) | LATE (>2 years) |
| Specific test | Neurosyphilis (may be asymptomatic – only abnormal CSF being demonstrated) |
| RPR: rapid plasma reagin | 1:8 or greater |
| TPHA: Treponema pallidum haemagglutination test | Reactive |
| TP-PA: Treponema pallidum particle agglutination test | |
| FTA (Abs): fluorescent treponemal antibody absorption test | |
| EIA: enzyme immunoassay | |

A person without clinical signs or symptoms of syphilis, but having positive syphilis serology and no history of having been treated for syphilis, is said to have latent syphilis. By convention in Australia and the UK, primary and secondary syphilis and the first 2 years of latent infection are called early syphilis (i.e. the period during which syphilis is infectious by sexual contact), while tertiary, cardiovascular, neurosyphilis and latent infection beyond 2 years is called late syphilis. In the USA early syphilis refers only to the first 12 months of infection. Table 1.2 and Figure 1.3 describe the stages of syphilis in adults with a guide to accompanying serology results.

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Trichomonas vaginalis

Trichomonal infection occurs in the vaginal epithelium and lining of the urethra of men and women. It can also infect the cervix and acute trichomonal inflammation at this site causes the clinical appearance called strawberry cervix. Sometimes infection spreads to associated glands (e.g. Bartholin’s and Skene’s). The organism has been isolated from the prostate gland and from epididymal aspirates, but its role in prostatitis and epididymitis is uncertain; if it does occur it is rare. Trichomonal infection in pregnancy has been associated with an increased risk of preterm delivery, preterm rupture of membranes and maternal puerperal infection.13

Bacterial vaginosis

The natural history of bacterial vaginosis remains largely a mystery. There is an association with pelvic inflammatory disease but the significance of this association is uncertain. The presence of bacterial vaginosis in pregnancy (both symptomatic and asymptomatic) may lead to low birth weight in babies, premature delivery and post-partum endometritis but the results of studies of therapeutic interventions against bacterial vaginosis in early pregnancy have been surprisingly variable. There is no clear consensus on how best to manage bacterial vaginosis in pregnancy as yet, but some experts recommend screening and treatment of high-risk mothers (especially those with a previous history of premature delivery).14

HIV and STIs – co-infection and the cofactor effect

There is a complex interaction between HIV and STIs. Very early in the HIV epidemic, studies in sub-Saharan Africa showed that STIs causing ano-genital ulcerative disease (GUD) substantially increased the risk of people acquiring HIV.15 This finding was not surprising, as any breach in genital skin or mucous membrane was likely to increase
ease of entry for HIV. Subsequently, studies showed it was possible to recover HIV from genital ulcers (including herpetic ulcers) in HIV-positive people. In other words, the presence of GUD made it more likely that HIV-positive people could transmit the infection to a sexual partner. Successfully treating the STI responsible for GUD stopped the shedding of HIV and decreased the risk of HIV transmission. The synergy between HIV and genital HSV-2 infection is especially worrying because HSV-2 is the most common cause of genital ulceration around the world and both symptomatic and asymptomatic shedding of HSV-2 occur relatively frequently in patients with the infection. Studies have shown that HSV-2 sero-positivity itself is a risk factor in both the acquisition and transmission of HIV. While suppressive therapy with antiviral drugs (e.g. aciclovir, valaciclovir, famciclovir) may decrease the risk of transmission of HSV-2 and so decrease the risk of transmission of HIV in patients with co-infection, routine use of these drugs in every HSV-2 sero-positive person is not a realistic option globally.

HIV shedding also substantially increases from genital sites during infection with other STIs. Gonococcal urethritis and cervicitis in men and women with HIV infection lead to substantially higher HIV viral loads in genital secretions than when people do not have gonorrhoea. Appropriate treatment for gonorrhoea causes a precipitate fall in such high HIV viral loads. Any STI associated with local inflammation increases the risk of acquiring HIV for a person without infection and enhances the risk of passing on HIV from a person with the infection to sexual partners. Even in bacterial vaginosis, a condition not characterised by local inflammation, but where the vaginal alkalinity is raised, there is an enhanced risk of a woman acquiring HIV infection, perhaps because the usual protective acidic environment of the vagina is lost.

In addition, HIV alters the natural history of many STIs (especially syphilis and genital herpes, but also HPV) and syphilis and herpes appear to have an influence on the natural history of HIV. In summary, the interaction of STIs with HIV is a synergistic one which considerably enhances the transmission of HIV in populations and cumulatively increases the burden of morbidity and mortality of all STIs around the world.

Therapy

HIV

The course of HIV has been drastically altered by the introduction of highly active antiretroviral therapy (HAART) or combination antiretroviral therapy (cART). This therapy usually consists of a combination of at least three drugs from two or three of the different classes of antiretroviral drugs: the nucleoside analogue reverse transcriptase inhibitors (NRTIs), the non-nucleoside reverse transcriptase inhibitors (NNRTIs), protease inhibitors (PIs), integrase inhibitors (II) and entry inhibitors. A combination of three agents, usually two NRTIs combined with either an NNRTI or PI or II, is administered when the CD4 cell count falls below a certain threshold. Although the optimal time to commence therapy has not been established, Australian and international guidelines recommend that treatment should be considered when the CD4 cell count falls below 500 cells/μL, or when the HIV viral load is above 55,000 copies/mL. Individualised decisions should take into account the patient’s readiness to start therapy, the baseline CD4 cell count and HIV RNA level and the potential risks and benefits of treatment. In 2014 current Australian treatment guidelines recommend access to HAART at any level of CD4 count and timing is dependent on the patient’s needs and also tied to the HIV prevention strategy which aims to reduce new infections by a combination of prevention, early diagnosis and early treatment which renders people less infectious when HIV viral load remains undetectable on HAART. cART is very potent in reducing viral load and delaying drug resistance, and has resulted in a dramatic reduction in mortality and increased life expectancy in people with HIV infection. This success has meant that HIV infection is becoming a chronic manageable disease for many people in many industrialised countries. Immune-based therapies, such as therapeutic vaccination, are also under investigation.

The aim of therapy for HIV infection is to sustain an undetectable viral load, which is achievable in approximately 80% of patients, and to produce immune reconstitution. Immunological benefit may be modest (CD4 cell counts frequently remain below normal levels) but may still occur in those who fail to achieve full virological suppression.

Many of the antiretroviral drugs have significant side effects and some have complex dosing schedules, making adherence (a major determinant of the development of resistance) an issue for concern. However, fixed-dose combination drugs are now available and most of the newer antiretroviral agents are administered once or twice daily, making adherence to combination regimens easier. Long-term survival of these people also has unmasked chronic drug toxicities, particularly metabolic problems such as lipodystrophy and lipoatrophy, hyperlipidaemia, insulin resistance and hepatic mitochondrial toxicity.

HBV

The goal of chronic HBV treatment is to prevent progressive liver disease and the development of liver failure and hepatocellular carcinoma (HCC). This is achieved through suppression of viral replication and a reduction of hepatic inflammation, as viral eradication with loss of HBsAg is rare. This has been shown to reduce or even reverse the complications of
Treatment is usually indicated in the immune clearance and immune-escape phases of infection with elevated HBV DNA and evidence of hepatitis with persistently raised alanine aminotransferase (ALT) or fibrosis or inflammation on liver biopsy or Fibroscan. Response to treatment can be assessed biochemically (ALT), virologically (HBV DNA and serology) or histologically (liver fibrosis and inflammation). Currently, a number of different agents are available, including immunomodulators (pegylated [PEG] interferon-alpha) and antiviral nucleos(t)ide analogues (including entecavir and tenofovir). In HBeAg-positive people, the aim of treatment is HBeAg seroconversion which is associated with a durable suppression of HBV DNA off treatment in 50–90% of people. Treatment with PEG interferon-alpha for a 12-month finite course is associated with HBeAg seroconversion rates of around 30%. As interferon is an immunomodulator, it is not associated with viral resistance. It does have significant systemic side effects, however, which often means that patients prefer the oral nucleos(t)ide analogues.

Lamivudine was the first nucleoside analogue found to effectively reduce HBV DNA, with HBeAg seroconversion rates of 17–32%, usually in those with raised ALT. However, at 4 years, over 60% develop viral resistance to lamivudine. The new HBV nucleos(t)ide analogues are entecavir and tenofovir. These are highly potent antivirals with undetectable HBV DNA rates of 70% and low to negligible viral resistance. The HBeAg seroconversion rates are in the order of 20%. HBV resistance has been estimated to be approximately 1.2% at 5 years with entecavir, but is significantly higher (up to 50%) in those with previous lamivudine resistance. Tenofovir has no resistance at 3 years in treatment-naive patients. The treatment of HBeAg-negative infection is problematic and therapy is probably needed lifelong to achieve viral suppression. Combination therapy may offer a future strategy for some patients if studies demonstrate it to be effective; interesting new data are emerging of sequential therapy with nucleos(t)ide analogues and PEG interferon as effective therapy for HBV. Finally, development of end-stage liver disease may mandate liver transplantation, the outcomes of which have been significantly improved with the use of these antiviral therapies and HBV immunoglobulin to prevent graft re-infection.

HCV

Similar to HBV, HCV is treated to prevent the development of cirrhosis, which is associated with hepatocellular failure and hepatocellular cancer. However, unlike HBV, the aim of treatment is viral eradication. The current standard of care for HCV is pegylated interferon alpha and ribavirin with or without the protease inhibitors telaprevir or boceprevir, depending on the HCV genotype. With 6 months of therapy sustained virological response (SVR) rates, which equate to cure, are approximately 80%. Once SVR has been achieved, it is highly durable, with almost all patients (more than 95%) remaining clear of the virus with extended follow-up.

There are currently significant and profound changes occurring in the treatment of HCV. New and highly potent direct acting antiviral (DAA) agents are soon to become available. These agents are inhibitors of the HCV protease and polymerase. Initially, they will be used in combination with interferon but there are now randomised controlled data revealing SVR rates in the order of 95% with 12 weeks of all oral therapy, with potential to shorten this treatment duration to 8 weeks in some individuals. Even the previously difficult-to-treat individuals with failed therapy or cirrhosis appear to respond well. These regimens are associated with minimal side effects. However, a major obstacle to the implementation of these therapies is their cost, which will have to be borne by the government.

An example of this treatment revolution is sofosbuvir, an HCV polymerase inhibitor that was registered with the Australian Therapeutic Good Administration (TGA) in 2014. It is used in combination with ribavirin for genotype 2 for 12 weeks and genotype 3 for 24 weeks with SVRs of 93% and 84%, respectively. For genotype 1 and 4, sofosbuvir is added to PEG interferon and ribavirin and given for 12 weeks with SVRs of 90 and 96% respectively.

End-stage liver disease due to HCV is now the most common indication for liver transplantation in Australia. Graft re-infection is almost universal, although disease progression is still relatively slow in most cases.

STIs

The general principles of therapy for STIs are:

- To cure patients of their infection, if possible; if not, to relieve symptoms and to stop progression of disease
- To render individuals non-infectious as soon as possible to prevent ongoing transmission in the community
- To treat all sexual partners.

Sexual health and public health physicians favour simple, single-dose treatments, capable of being taken immediately. These are obviously ideal principles rarely met in practice, but they need to be kept at the forefront of the minds of all practitioners treating patients with STIs. Except for the viral STIs, simple, single-dose treatments now exist for almost all the common uncomplicated STIs. The development of azithromycin in the mid-1990s truly revolutionised therapy for genital chlamydia infection, as prior to that time, treatment for chlamydia depended on doxycycline which had to be given for a minimum
of 7 days. Many patients failed to complete the course, failed to be cured and so remained potentially infectious.

Antiviral therapies are readily available in Australasia for genital herpes and, although not curative, they will relieve the symptoms of troublesome outbreaks (especially primary attacks) and substantially reduce viral shedding, thus reducing the risk of further transmission. Ongoing clinical trials are currently assessing the public health effectiveness and practical utility of this chemotherapeutic intervention in high-risk groups (e.g. people with HIV infection and men who have sex with men (MSM) with herpes).54

There is no antiviral therapy for HPV infection and none in development, clearance of clinical manifestations of this virus being mediated by the immune response in immune competent people. HPV vaccination prior to onset of sexual activity now offers a prevention strategy for HPV infection.

Public health objectives inevitably link closely with treatment goals in STI management. Where effective treatment is available, the aim is to treat all sexual partners of patients diagnosed with an STI and, where there is no effective treatment, the lesser aim is to provide information, education and counselling for sexual partners. Contact tracing (partner notification) meets these aims. Because of the complex and synergistic interactions between STIs and HIV, especially in communities at high risk for both types of infection, clinicians must make a new commitment to contact tracing. ASHM’s Contact Tracing Manual discusses this important aspect of STI management.55

Prevention

There is an effective and safe vaccine for HBV which is provided universally for babies and adolescents in Australia through the National Immunisation Program. It is important to offer vaccination to high-risk patients who have not been previously immunised. Unfortunately, technical difficulties associated with vaccine development suggest that effective vaccines for HIV and HCV are at least 5 to 10 years away.

There is also an effective and safe vaccine for hepatitis A virus. To prevent infection through sexual transmission of this virus, clinicians should encourage vaccination in all individuals who engage in sexual activities where any degree of faecal contamination of fingers or mouth could occur. This category includes all MSM.

There are two effective and safe vaccines against HPV licensed for use in Australia. There are several differences between them. Gardasil provides protection against HPV types 6, 11, 16 and 18 and is recommended for use in young women aged between 9 and 26 years. There has been a funded vaccination program for school girls since 2007 and boys since 2013. Cervarix, the other HPV vaccine, is designed to protect against infection with HPV types 16 and 18, but also has activity against types 31 and 45. It is licensed for women and girls aged between 10 and 45 years in Australia. Health practitioners should be offering HPV vaccine to all young men and women, preferably before they commence sexual activity. If Gardasil is used, it should protect young men and women from the common genital wart viruses and the most common high-risk types of HPV. It should not be a substitute for regular Papanicolaou smears as other high-risk HPV types exist and circulate in the community. Changes to the cervical screening program recommendations will occur as the vaccine cohort are screened over time.

Prevention strategies for the blood-borne viruses based on public health behaviour modification and harm minimisation approaches have been effective in Australia and elsewhere and remain the foundation of prevention for individuals at risk of these viral infections. All people should be given clear messages about the risks of STIs, the asymptomatic nature of most early STIs in men and women, the enhancing effects of STIs on the risk of acquiring HIV, the need for regular sexual health check-ups for those with multiple sexual partners or frequent changes of partner, and the reliability of male (or if preferred, female) condoms in substantially reducing the risk of transmitting and acquiring almost all the STIs.

References

RISK ASSESSMENT
AND DIAGNOSIS
CHAPTER 2 BLOOD-BORNE VIRUSES AND STIs: MIGHT THIS PATIENT BE POSITIVE? EPIDEMIOLOGY AND TRANSMISSION

2014 REVIEW

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Introduction

Early diagnosis is important for all treatable conditions. Early identification of sexually transmitted infections (STIs) and blood-borne viral infections in particular can facilitate both treatment and prevention. Therapy for human immunodeficiency virus (HIV) infection can postpone immune damage and thereby prevent development of opportunistic infections and malignancies; treatment of treatable STIs prevents sequelae and reduces transmission, while improved therapies for hepatitis B virus (HBV) and hepatitis C virus (HCV) can potentially clear the virus and improve clinical outcomes in some individuals. In addition to providing the benefits of treatment, early diagnosis, accompanied by relevant education, can help to reduce the rate of ongoing transmission of STIs, HIV, HBV and HCV.

Diagnosis of each of these infections generally requires simple tests. However, indications for testing are frequently overlooked and opportunities for early diagnosis are missed. The decision to test should be based on a detailed history of risk behaviour as well as a physical examination of the patient. It should always be borne in mind that people may prefer to conceal a history of risk taking especially when it concerns sex, drugs or both. Consequently, a low threshold for testing should be maintained. Individuals with a blood-borne virus infection who do not report high-risk behaviours are more likely to present with advanced disease. Late presentation has been associated with poor clinical outcomes, particularly in relation to HBV and HIV.

An understanding of the epidemiology and transmission of blood-borne viruses and other STIs, along with a detailed behavioural history, will help the clinician make an accurate assessment of the likely risk of infection, and guide appropriate testing.

Blood-borne viruses (HIV/HBV/HCV): prevalence and risk factors for transmission

Although HIV, HBV and HCV are all blood-borne viruses, the efficiency of transmission in different settings varies enormously (Table 2.1). Transmission will depend on many factors, including the infectivity of the source (e.g. the viral load of HIV, HBV or HCV) and the type of exposure.

The clearest example of the differences in transmissibility of these three viruses is sexual contact. Unprotected anal or vaginal sex with a person who has the infection carries a high risk of transmission for both HIV and HBV but a very low risk of transmission for HCV (Table 2.1).

KEY POINTS

- HIV and HBV are transmitted through sexual contact, as well as blood-to-blood contact and from mother to child. HCV is transmitted by blood-to-blood contact.
- STIs are transmitted through various forms of sexual contact including oral sexual activities.
- In Australia, the prevalence of HIV, HBV and HCV is high in particular groups. However, risk exposure, rather than group membership, should be the basis for risk assessment, particularly in the context of HCV and HIV, and HBV acquired in Australia.
- In Australia and New Zealand, the prevalence of genital chlamydial infection is high in young sexually active people. Most early infection is asymptomatic so screening for chlamydia in primary care practice is vital. Annual chlamydia screening is recommended for sexually active men and women under the age of 30.
- For STIs other than chlamydia the decision to test should be based on an assessment of risk as well as physical examination. Some people may prefer not to reveal a history of risk behaviour, and a low threshold for testing should be maintained.
- People with a blood-borne virus infection who do not report high-risk behaviours are more likely to present late and to suffer poor clinical outcomes.
TABLE 2.1 Risk of HIV, HBV and HCV transmission (from a known positive source)

<table>
<thead>
<tr>
<th></th>
<th>HIV</th>
<th>HBV*</th>
<th>HCV*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sexual contact</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unprotected anal (receptive)</td>
<td>very high</td>
<td>very high</td>
<td>very low</td>
</tr>
<tr>
<td>Unprotected anal (insertive)</td>
<td>high</td>
<td>very high</td>
<td>very low</td>
</tr>
<tr>
<td>Unprotected vaginal</td>
<td>high</td>
<td>very high</td>
<td>very low</td>
</tr>
<tr>
<td>Unprotected oral (cunnilingus and fellatio, receptive and insertive)</td>
<td>very low</td>
<td>low-moderate</td>
<td>negligible</td>
</tr>
<tr>
<td><strong>Mother to child (perinatal)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No intervention</td>
<td>20-45%</td>
<td>30-90%</td>
<td>5%</td>
</tr>
<tr>
<td>With intervention</td>
<td>&lt;5%</td>
<td>&lt;5%</td>
<td>N/A</td>
</tr>
<tr>
<td><strong>Occupational exposure (needle-stick)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sharing injecting equipment among people who inject drugs</td>
<td>very high</td>
<td>very high</td>
<td>extremely high</td>
</tr>
<tr>
<td>Unsterile tattooing and piercing</td>
<td>high</td>
<td>very high</td>
<td>very high</td>
</tr>
<tr>
<td>Unsterile medical and other procedures</td>
<td>high</td>
<td>high</td>
<td>high</td>
</tr>
</tbody>
</table>

a. Refers to chronic hepatitis B (HBsAg+), with higher risk where source is HBeAg+ and/or a detectable HBV DNA level.
b. Refers to chronic hepatitis C (HCV RNA+).
c. Higher risk may be associated with certain practices or circumstances where there is the possibility of blood-to-blood contact (e.g. traumatic sexual practices, sex during menstruation) or high HCV viral load (e.g. HIV co-infection).
d. Some evidence of higher risk for male-to-female than female-to-male transmission.
e. Higher risk (15–20%) in presence of HIV/HCV co-infection, related to higher HCV viral load.
f. Proven interventions include antiretroviral therapy, caesarean section and avoidance of breastfeeding.
g. Intervention includes HBV immunoglobulin and vaccination.
h. There is no currently proven intervention for perinatal HCV transmission.
i. Some evidence of HCV transmission when sharing injecting equipment other than needles (e.g. spoons, tourniquets).

There are also differences in perinatal transmissibility of HIV, HCV and HBV. HCV has a relatively low efficiency of transmission in the perinatal setting; only 5% of infants born to women with HCV will acquire the infection, with factors such as maternal viral load and duration of labour affecting risk of transmission. Without intervention, mother-to-child transmission of HIV and HBV is common. In the absence of prophylaxis, rates of mother-to-child transmission of HBV are very high, particularly from HBeAg-positive mothers with high viral load (more than 85% transmission). Thus, routine HBSAg testing is recommended in all pregnant women, and both passive (hepatitis B immunoglobulin) and active (hepatitis B vaccination) interventions are given to the baby within 12 to 24 hours of birth.

This strategy is thought to be over 95% effective in preventing neonatal infection. The rate of HIV mother-to-child transmission without intervention is 25%. However, proven interventions can reduce the risk of perinatal transmission of HIV to 1–2%1 and HBV to less than 5%3,4

In contrast to the lower efficiency of HCV transmission through sexual contact, HCV is more efficiently transmitted than HIV or HBV through blood-to-blood contact where injecting equipment (including swabs, spoons, water, tourniquets, needles and syringes) is shared.6

The likelihood of transmission after a specific exposure is also related to the risk of infection in the source. Although transmission of blood-borne viruses is associated with certain risk behaviours, prevalence rates are higher in specific groups in Australia: HIV in men who have sex with men (MSM); HBV and HCV in people who inject drugs; HBV in Indigenous Australians and Asian-born populations; and all three viruses in people with haemophilia treated with clotting factor replacement therapy prior to 1990 (Table 2.2). The low, although increasing, prevalence of HIV in people other than MSM in Australia accounts for the relatively low risk of HIV infection after unprotected heterosexual exposure and sexual assault.

The prevalence of HCV is very high among persons who have ever injected drugs, and use of injecting equipment that has been contaminated with HCV-infected blood carries a very high risk of transmission. Consequently, infection is common after even a small number of exposures, such as the occasional sharing of injecting equipment.

**The global HIV epidemic and its implications for Australia**

Outside Australia, the patterns of HIV transmission are diverse. Many countries in Europe and North America are seeing the HIV epidemic move into
TABLE 2.2 Seroprevalence estimates for HIV, HBV and HCV in Australia

<table>
<thead>
<tr>
<th></th>
<th>HIV (%)</th>
<th>HBV (%)</th>
<th>HCV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>People who inject drugs</td>
<td>1-2%</td>
<td>40-50%</td>
<td>50-60%</td>
</tr>
<tr>
<td>Sexual orientation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Homosexual/bisexual men</td>
<td>5-10%</td>
<td>40-50%</td>
<td>5-7%</td>
</tr>
<tr>
<td>Homosexual/bisexual women</td>
<td>&lt;1</td>
<td>2-5%</td>
<td>2-5%</td>
</tr>
<tr>
<td>Heterosexual men</td>
<td>&lt;1</td>
<td>1-2</td>
<td>1-2</td>
</tr>
<tr>
<td>Heterosexual women</td>
<td>&lt;1</td>
<td>1-2</td>
<td>1</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indigenous Australian</td>
<td>&lt;1%</td>
<td>20-30%</td>
<td>2-5%</td>
</tr>
<tr>
<td>Asian</td>
<td>&lt;1%</td>
<td>20-50%</td>
<td>2-5%</td>
</tr>
<tr>
<td>Other</td>
<td>&lt;1%</td>
<td>1-2</td>
<td>1-2</td>
</tr>
<tr>
<td>Health care workers</td>
<td>&lt;1%</td>
<td>1-2</td>
<td>1-2</td>
</tr>
<tr>
<td>Recipients of blood productsa</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>People with clotting disordersb</td>
<td>20-30</td>
<td>50-60%</td>
<td>0-80%</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>1-2</td>
<td>2-5</td>
</tr>
</tbody>
</table>

a. Some of these estimates are based on limited data, and should be considered as guides to levels of infection rather than true prevalence values.
b. Based on prevalence of anti-HBc, indicating previous exposure. Approximately 95% of people exposed to HBV as adolescents and adults clear HBV infection (HBsAg- and anti-HBs+) and are immune to re-infection.
c. Based on HCV antibody prevalence from the Australian Needle and Syringe Program National Data Report 2009-2013.
d. Prevalence of chronic hepatitis B (HBsAg+) estimated to be 2-3%.
e. Based on self-reported HIV status among gay men at gay community fair days in Australian capitals.
f. Higher prevalence estimates than heterosexual groups due to higher prevalence of injecting drug use.
g. Despite higher rates of other STIs, HIV prevalence is similar among Indigenous and non-Indigenous Australians.
h. The majority of transmission occurs during the perinatal period or early childhood, therefore the estimate for chronic hepatitis B (5–10%) is higher than for other high-risk groups (2–3% for people who inject drugs, homosexual men).
i. Higher estimate due to increased prevalence of injecting drug use and incarceration.
j. Higher estimate due to probable increased exposure through non-sterile medical, dental and other skin penetration procedures in non-Australian born Asians. Higher estimated prevalence in people born in other selected, high prevalence countries (e.g. Italy, Egypt).
k. Although cases of occupational transmission of blood-borne viruses have been reported, including 14 cases of HIV, prevalence of HIV, HBV and HCV is estimated to be similar to the general population.
l. In Australia, screening for HIV was introduced in the early 1970s, HIV in 1985, and HCV in 1990.
m. Includes people with haemophilia A, haemophilia B, and von Willebrand’s disease. In general, prevalence rates increase with severity of clotting disorder and age (due to introduction of screening).

Other STIs: prevalence, transmission and who to test

Alongside prevalence and modes of transmission, this section contains a check-list which provides a rough guide for testing for specific STIs. For sexually active MSM, national guidelines recommend at least annual testing for chlamydia, gonorrhoea, syphilis, HIV, and an initial screen and vaccination if necessary for hepatitis A and B. See Chapter 8 for testing methods and appropriate anatomical testing sites.

Genital chlamydial infection

Prevalence

The rate of chlamydia infections reported in the population has been steadily rising over the last decades. Increased testing and the availability of nucleic acid amplification tests (NAATs) such as polymerase chain reaction (PCR) that are easier to perform and have greater sensitivity than the
culture of the organism could account for some of the increase, but it is not thought to be the sole reason. We are not winning the battle against genital chlamydial infection. The common form of genital chlamydial infection is due to *Chlamydia trachomatis* serovars D to K causing urethral, cervical and rectal infections. Chlamydia was the most frequently reported notifiable condition in Australia in 2012 with 82,707 diagnoses. The population rate of diagnosis of chlamydia in 2012 was 355 per 100,000 population. This is a disease of younger people aged 15 to 29 years (more sexual activity, more partners), but is most commonly diagnosed in the 20 to 29 year age groups and in women more often than in men.

**Transmission**

Lymphogranuloma venereum (LGV) is caused by *C. trachomatis* serovars L1, L2 and L3. It is very uncommon in Australia, however, in recent years, there has been a small cluster of cases in very sexually active MSM. It is prudent, therefore, to consider the possibility of LGV in an MSM patient who has symptomatic chlamydial proctitis or where *C. trachomatis* is detected by NAAT on routine rectal screening in an MSM (see Chapter 13). Clinicians will need to discuss with their local laboratory the issue of specific testing for LGV, should the need arise.

**Who to test**

- Women who are pregnant – especially if the pregnancy is unplanned or unwanted (or if having a termination of pregnancy)
- Sexually active patient under 30 years – screen at least annually
- Patient who has had a change of partner
- Patient who has multiple partners
- Patient reporting unprotected sexual intercourse
- Patient diagnosed with another STI
- Aboriginal or Torres Strait Islander patient, if not screened in the past 12 months
- Think of testing for chlamydia in all MSM as it is a common ano-genital infection in homosexually active men. LGV strains of chlamydia (L1 to L3) can cause a moderate-to-severe proctitis in MSM. Clinicians will need to discuss with their local laboratory the issue of specific testing for LGV should the need arise
- Patients who have experienced a sexual assault
- Sex workers (required as part of issuing a certificate); especially street sex workers if requested.

**Genital herpes**

**Prevalence**

Genital herpes is not a notifiable disease. The general population prevalence in Australasia is 12–20%. It is higher in MSM and sex workers. The majority of people with genital herpes are unaware that they have the infection. They have either subclinical infection, i.e. they have mild recurring symptoms that they do not recognise as herpes, or they have truly asymptomatic infection.

**Transmission**

HSV-2 normally replicates in sacral ganglia. The cause of genital herpes infection has changed, with approximately 30% or more of genital herpes diagnosed today caused by HSV-1 (the virus commonly associated with oral herpes). This is thought to be due to less childhood acquisition of facial HSV-1, thus more adolescents and young adults are susceptible to acquisition from oral sex.

**Who to test**

- Symptomatic patients: nucleic acid amplification test (NAAT) testing from any suspicious lesions
- HIV-positive patients: type specific serology (see Chapter 8)
- Some specific clinical situations: type specific serology, e.g. discordant couples, especially if heterosexual – female with no history of herpes, male with a past history of genital herpes and couple are wanting a pregnancy.

**Genital warts and human papillomavirus (HPV)**

**Prevalence**

Human papillomavirus (HPV) infection is so common in the community that acquiring one of the many genital types of HPV is virtually synonymous with having sex. The HPV vaccination program has been running since 2007 in the National Immunisation Program. Originally starting with females only, the program now includes both males and females, at school age. This vaccination program is significantly changing the landscape of HPV infection and the clinical scenarios it presents: warts, dysplasia and carcinoma.

**Transmission**

Most people will come in contact with HPV at some point in their life: the rate of HPV in the population has been previously quoted as over 93%. As young people are naive to the infection they commonly are found to have it when first becoming sexually active. Most people develop natural immunity against the infection and so clear it. The vaccine also creates
this immunity so young people who are vaccinated, then exposed to the HPV types in the vaccine (6,11,16 and 18), are prevented from getting the infection and its sequela.

There are many types of HPV with approximately 50 types site-specific to the genitals. The HPV types 6 and 11 are the viruses responsible for the majority of genital warts (90%). Before the introduction of the vaccine, 10-15% of adults acquired genital warts causing much anxiety and stress. This proportion has significantly changed since the introduction of the vaccine. HPV is mostly asymptomatic, an important fact to explain to patients when they are concerned about where their infection might have come. There are approximately 15 types of HPV which are associated with dysplastic changes in the genital region, especially at the transformation zones of the cervix and the anal canal. Types 16 and 18 cause 70–80% of cervical cancer in Australia and are implicated in the development of other squamous carcinoma such as anal, vaginal, penile and throat. With the HPV vaccine included in the National Immunisation Program, the majority of genital warts and HPV-related cervical cancer should be prevented. Changes are already evident for the incidence of genital warts;27 but as cervical cancer is very slow to develop, it will be 15-20 years before we see a reduction in cervical cancer although there is now evidence of a reduction in CIN 2 and 3 in young women following the introduction of the HPV vaccine.28

In 2014 it was announced that the National Cervical Screening Program Renewal would review the existing policy and consider new evidence since the introduction of the HPV vaccine but that changed would not be adopted until 2016. It is likely a HPV test will be introduced as the triage test for the screening program. For further information see: http://www.cancerscreening.gov.au

Who to test

- Clinical diagnosis only: there is no screening available for genital warts
- Cervical screening is important in all women (see NHMRC guidelines): yearly for HIV-positive women.
- HPV DNA testing is recommended by the NHMRC and funded through the Medicare Benefits Schedule (MBS) as a test of cure following treatment of high-grade squamous epithelial lesions (SIL) of the cervix. Changes to the National Cervical Screening Program to be introduced in 2016 will most likely use HPV testing as the triage test in the cervical screening program.

Gonorrhoea

Prevalence and transmission

Gonorrhoea has been slowly increasing over the last decades. It is mainly seen in MSM, Aboriginal and Torres Strait Islander people, overseas people particularly from South East Asia, and in recently returned travellers who have had sex (either heterosexual or homosexual) with a local person in a country where gonorrhoea is endemic. The overall rate of infection in Australia in 2012 was 58.9 per 100,000. Since 2007 the rate has increased to 84.3 and 36.1 among males and females, respectively, in 2012. The rate of gonorrhoea in men has been consistently a little more than double that in women over the past 5 years, a reflection of the substantial contribution MSM make to gonococcal infection rates in Australia.29 The majority of gonorrhoea diagnosed is urethral and rectal.29

Who to test

- MSM: this group is at substantially higher risk of gonorrhoea than the rest of the general Australian community
- Patient with a history of sex with someone recently arrived from a high-prevalence country (e.g. India, South East Asia)
- Patient recently arrived from a high-prevalence country (e.g. India, South East Asia)
- Aboriginal and/or Torres Strait Islander patient, if not screened in the past 12 months
- Sex workers (required as part of issuing a certificate), especially street sex workers.

Syphilis

Prevalence and transmission

Syphilis is another infection that has been steadily on the rise. Syphilis has for a long time been prevalent in Australia’s Indigenous population. In 2012, the rate of diagnosis of infectious syphilis increased among males from 6.1 in 2008 to 6.7 in 2012; increased rates occurred in Queensland, Victoria and New South Wales and declining rates were reported in Western Australia and the Northern Territory. The rate of infectious syphilis diagnosis in the Aboriginal and Torres Strait Islander population resident in the Northern Territory declined from 11.8 in 2008 to 61.4 in 2012. The rate of diagnosis of infectious syphilis in the non-Indigenous population of the Northern Territory was stable at around 5.4 per 100 000 population in 2008–2012.30
Due to the chronicity of untreated infection and the often long latent periods characteristic of syphilis, this is an infection which can be diagnosed in any age. Indeed, it remains in the differential diagnosis for dementia.8, 29

Who to test
- Men who have Sex with Men (MSM)
- HIV-positive patients
- Sex workers
- Indigenous patients
- Pregnant patients or those planning a pregnancy.

Trichomoniasis

Prevalence and transmission
Trichomoniasis is not a notifiable infection in any state in Australia. Infection due to Trichomonas vaginalis is most prevalent in the Indigenous population or in returned travellers from higher prevalence countries. Infection is often asymptomatic in both sexes. Symptomatic infection may occur in women but is very rare in men. It is still essential to treat both partners when a woman is diagnosed with trichomonal infection.8,20,22,29

Who to test
- Sex workers
- Women with a vaginal discharge
- Indigenous women
- Patients with a history of sex with someone from a high-prevalence country
- Patients recently returned from a high-prevalence country if they had unprotected sexual intercourse with a local person in that country.

HIV and the sexual health context
A person diagnosed with an STI is likely to be at increased risk of HIV infection. An STI can be a marker of recent or past risk and genital inflammation itself may have put the individual at higher risk of HIV infection. A full assessment of a person with an STI includes HIV, HBV and often HCV antibody testing.

HIV risk should be considered in all patients who present with an STI. Although the diagnosis of a heterosexually acquired STI is unlikely to be accompanied by HIV infection in Australia, the presence of an STI calls at least for careful clinical assessment of the actual risk with the informed cooperation of the person. There is a medical and legal imperative to fully investigate any patient diagnosed with an STI or blood-borne viral infection (Chapter 15). Failure to diagnose an STI can lead to ongoing transmission of the infection as well as clinical progression.

More than 20 years into the HIV epidemic, there is some evidence that consistent use of condoms is becoming less common among gay men in Australia.12 Since the mid 1990s, surveys have reported increasing levels of unprotected anal intercourse with casual partners among MSM, and surveillance data reveal increasing rates of gonorrhoea and syphilis in these populations.5 Rates of diagnosis of HIV infection have shown regional differences but overall continue to increase, and transmission occurs primarily through sexual contact between men.8 Regular testing for gonorrhoea and other STIs in MSM who have casual sexual partners should be a routine part of clinical care. All gay and bisexual men should be assessed for HAV and HBV immunity and vaccinated if necessary. As well, clinicians looking after people living with HIV should recommend regular STI screening for their sexually active patients.

In addition to triggering consideration of HIV and HBV infection, the presence of an STI provides the primary care clinician with the opportunity to take a sexual history and promote safer sex practices (Chapter 3).

Prevention strategies
There are several proven means of reducing the efficiency of transmission of STIs, HIV, HBV and HCV (Table 2.3). The use of condoms for anal or vaginal sex and the use of clean injecting equipment remain the most effective means of prevention of transmission of HIV (Chapter 3). Similarly the use of condoms for anal and vaginal sex will significantly reduce the risk of most STIs. Because STIs may be transmitted through oral sex and because condoms are not 100% effective, however, clinicians should recommend regular sexual health check-ups for their sexually active patients, including screening for common STIs (see Chapter 8).

The use of sterile injecting equipment is the most effective means of preventing HCV transmission. Other interventions such as post-exposure prophylaxis may also have a role in prevention, particularly for HIV (Chapter 14). Antiretroviral therapy, caesarean section and avoidance of breast-feeding have reduced the risk of perinatal transmission of HIV to 1–2%.4 Antiretroviral therapy is the most effective means of reducing vertical transmission in Australia and in resource poor settings. Antenatal testing of women for common STIs and STIs with significant infant morbidity (e.g. syphilis) is an important measure for reducing risk of vertical transmission. HBV vaccination
is safe and extremely effective. Nevertheless, many people at risk of infection remain unvaccinated in Australia. The HPV vaccine is now available on the National Immunisation Program Schedule for all adolescents aged between 12 and 13 years. The search is currently underway for effective HIV and HCV vaccines, but these may be many years away. A vaccine against herpes simplex virus (HSV) infection is urgently needed because of the synergistic effect of this infection on HIV transmission. Development of vaccines against other common STIs has unfortunately never been accorded high priority, no doubt a reflection of the ongoing stigmatised nature of these conditions.

**Summary**

STIs, HIV, HBV and HCV are different and distinct infections in terms of epidemiology and risk factors for transmission, although there are some similarities in the modes of transmission. The recommendation to test for common and significant STIs, HIV, HBV and HCV should be based on reported risk factors for transmission or the presence of clinical signs. A low threshold for testing is advised due to the reluctance of some people to disclose risk behaviours or their failure to identify risks.

**Table 2.3 Factors associated with increased or decreased transmission of HIV, HBV, HCV**

<table>
<thead>
<tr>
<th></th>
<th>Increased transmission</th>
<th>Decreased transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV</td>
<td>High viral load in index case</td>
<td>Low viral load (possibly through therapy)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Post-exposure prophylaxis (antiretroviral therapy)</td>
</tr>
<tr>
<td>Sexual</td>
<td>Sexually transmitted infections in either partner (includes STIs and non-infectious vaginal inflammation)</td>
<td>Condoms and safe sexual practices</td>
</tr>
<tr>
<td></td>
<td>Genital inflammation</td>
<td>Treatment of sexually transmitted infections</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occupational</td>
<td>Deep penetrating injury</td>
<td>Universal (standard) precautions</td>
</tr>
<tr>
<td></td>
<td>Hollow-bore needle</td>
<td></td>
</tr>
<tr>
<td>Perinatal</td>
<td>Vaginal delivery</td>
<td>Antiretroviral therapy</td>
</tr>
<tr>
<td></td>
<td>Breast feeding</td>
<td>Caesarean section</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bottle feeding</td>
</tr>
<tr>
<td>HBV</td>
<td>Unvaccinated status</td>
<td>Vaccination</td>
</tr>
<tr>
<td></td>
<td>HBeAg+ or HBV DNA+ in index</td>
<td>Post-exposure prophylaxis (immunoglobulin and vaccination)</td>
</tr>
<tr>
<td>HCV</td>
<td>HCV RNA+ index case</td>
<td>Negligible risk of transmission if source HCV RNA-negative</td>
</tr>
<tr>
<td></td>
<td>High HCV viral load in index case</td>
<td>Use of sterile, unused, injecting equipment in a safe environment</td>
</tr>
</tbody>
</table>
CHAPTER 3 TALKING WITH THE PATIENT: RISK ASSESSMENT AND HISTORY-TAKING

2014 REVIEW

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**Introduction**

This chapter has two purposes. It is designed to provide a framework for health-care workers in a generalist setting to conduct a brief and focused risk assessment in order to trigger screening for blood-borne viruses and sexually transmissible infections and targeted health promotion where appropriate. In addition, it is designed to provide guidance on further relevant history-taking in those who are diagnosed with HIV, viral hepatitis, or an STI. Further information regarding management and counselling post diagnosis is also provided in Chapters 10, 11 and 12.

Rapport, trust, and effective communication are vital components of a therapeutic relationship and contribute significantly to a clinician’s ability to take a comprehensive history, particularly in the context of the sensitive issues around human immunodeficiency virus (HIV), sexually transmitted infections (STIs) and hepatitis virus infections. A sexual and drug-use history is required to identify specific risk factors and behaviours regarding exposure to HIV, STIs, and hepatitis viruses, to establish a diagnosis and to provide a setting for targeted prevention and harm reduction messages and strategies.

**General issues**

Taking a sexual and drug-use history helps to ascertain the patient’s risk of exposure to blood-borne viruses and STIs.

The physical environment in which the consultation takes place needs to be conducive to private discussion and adequate time must be set aside. If one appointment is not sufficient, allowance must be made for further discussion when the patient returns for his or her test results or follow-up. A significant proportion of people with hepatitis B in Australia will have English as a second language, and the use of interpreters, either on-site or from the Translating and Interpreting Service, is recommended when necessary.

Key elements of effective communication are active listening and being interested, non-judgmental and observant. Taking notice of the patient’s unspoken cues and reflecting together on the most salient points may assist communication. Reflection, by summarising the speaker’s words, is a technique that gives the patient the opportunity to correct any misunderstandings and allows the clinician to check that the patient is understanding.

Sexual and drug-use history-taking begins with a discussion of general issues and progressing to more detailed and specific questioning regarding risk behaviours.

**TABLE 3.1 Factors that will assist effective communication during sexual and drug-use history-taking include:**

- a comfortable and inviting space with adequate time
- privacy and the absence of interruptions
- assurance and explanation of confidentiality
- a non-judgemental attitude
- an openness to alternative lifestyles and a willingness to learn
- a willingness to discuss sexual and drug-use behaviour in detail
- listening carefully to the patient
- a focus on the goals of the interview
- attention to the cultural appropriateness of sexual history-taking which may require consideration, particularly with regard to the gender and cultural background of the clinician.

**KEY POINTS**

- Reflect on any specific barriers you may encounter when conducting a risk assessment which may be a barrier to effective communication
- Start the discussion around sexual or drug use history by normalising the discussion, using a hook and incorporating into existing discussion
- A brief risk assessment should be conducted whenever the opportunity presents itself, either via patient request for STI and blood-borne virus screening, or if opportunistic testing moments arise
- Opportunistic testing is dependent on keeping an open mind about the possibility of infection and maintaining a low threshold of suspicion prompting testing
- Work alongside the patient to identify risk behaviours and ways to minimise the risk of infection
- A more detailed history may be required if an infection is diagnosed
- Ensure confidentiality and obtain consent
- Understand your role in contact tracing
Communication style and language will vary depending on the clinician and the patient. The clinician is advised to use language with which he or she feels comfortable and familiar, and that takes account of the language and concepts used by individual patients. If the clinician doesn’t understand a word or phrase or idea the patient has used, clarification should be sought. This helps to develop trust and a sense of engagement between the clinician and patient, as well as ensuring accuracy.

The clinician should assure clients that confidentiality will apply to all information obtained in the context of clinical service delivery. Confidentiality issues may be especially important for adolescents and those living in smaller communities. While reassuring the patient, explain briefly the limitations to confidentiality in your jurisdiction, such as the requirement to report individuals who deliberately and repeatedly put others at risk of HIV infection, or to notify authorities where there is evidence of child abuse (see Chapter 15).

A major barrier to effective communication is awkwardness and embarrassment on the part of the patient or the clinician when discussing sexual practices or recreational and injecting drug use. In particular, a clinician with a long-standing relationship with a patient may feel unable or uncomfortable in broaching certain topics or have difficulty raising sexual matters with patients of the opposite gender, a different sexual orientation or age group. Lack of training, time constraints and limited knowledge of cultural and lifestyle issues can result in a reluctance by the clinician to persevere with these interviews. A simple lack of practice may also impede a clinician in successfully taking a sexual and drug-use history. For issues that challenge the values or beliefs of the clinician, discussion with a colleague may help to familiarise him or her with unusual or challenging language or concepts. Sometimes a referral to another clinician or service may be an appropriate course of action.

Patients are often reluctant to report behaviour that is stigmatised and they may feel unable to discuss their behaviour with friends or family. Clinicians should also be mindful that violence or the fear of violence is stigmatised and they may feel unable to discuss this with the patient or the clinician when discussing sexual practices or recreational and injecting drug use. In particular, a clinician with a long-standing relationship with a patient may feel unable or uncomfortable in broaching certain topics or have difficulty raising sexual matters with patients of the opposite gender, a different sexual orientation or age group. Lack of training, time constraints and limited knowledge of cultural and lifestyle issues can result in a reluctance by the clinician to persevere with these interviews. A simple lack of practice may also impede a clinician in successfully taking a sexual and drug-use history. For issues that challenge the values or beliefs of the clinician, discussion with a colleague may help to familiarise him or her with unusual or challenging language or concepts. Sometimes a referral to another clinician or service may be an appropriate course of action.

It is useful to consider strategies for managing conversations that become awkward or difficult. Breakdown in communication is very common and may result in a change of topic. If the interview is progressing poorly, it may be helpful for the clinician to consider his or her own responses to the content of the discussion. It is vital to be aware of the cues the patient is giving and to try to ensure his or her needs are met by the consultation. Empathy, humour and digression may help to dissipate anxiety. Clarifying or redirecting statements, such as ‘Could I ask another question about HIV?’ can help to structure the interview.

**CASE STUDY 1 Risk assessment: non-disclosure of high-risk sexual activity**

A 29-year-old garage mechanic visits his GP complaining of a purulent urethral discharge. He seems open, personable and readily admits to having many sexual partners in the past—mostly casual ‘one-night stands’—with whom he usually used condoms. However, at the time of presentation, he has been with his current girlfriend for over a year and the couple no longer use condoms. The man reports that while his girlfriend was away last weekend, he went to a nightclub and met a woman with whom he had sex. He was very drunk and is unsure whether a condom was used. He tested negative to an HIV antibody test 2 years ago in another city.

He denies any same-sex partners or injecting drug use.

The GP conducts a screening for STIs, including urethral swabs, and suggests blood tests for HIV, syphilis and HBV. The patient seems a bit resistant to the idea at first, but then agrees. He accepts the treatment with ceftriaxone intramuscular together with oral azithromycin and agrees to return in one week for his results.

The man’s urethral swab culture is positive for *Neisseria gonorrhoeae*, as expected, but his HIV antibody test is also positive. All other tests are negative. He is shocked at the news and admits that he did not tell the full truth on his previous visit; in fact, most of his casual partners have been male. He reports both insertive and receptive anal sex without condoms and says he is most likely to seek casual sex when he has been drinking heavily.

**Risk assessment**

**Purpose of risk assessment**

The clinician needs to consider the purpose of conducting the risk assessment before beginning an encounter with each patient. Common reasons for conducting the risk assessment may include:

- STI or blood-borne virus screening
- investigation of genital symptoms
- investigation of more generalised symptoms suggestive of HIV infection, an STI or hepatitis viruses
- determining what tests should be offered for someone who’s a contact of an STI or blood-borne virus.
A complete risk assessment can appear daunting and a time-consuming process. However it can be tailored to each situation and may only involve a couple of questions, particularly when the patient is well known to the service or clinician. More detailed questioning may be needed in specific situations such as if an adolescent admits that he or she is sexually active or when the patient does not disclose risk behaviour. If a test result is positive, further questioning of the patient and exploration of risk is warranted.

**Risk assessment for blood-borne viruses and STI screening**

A person may have presented requesting a screen or a clinician may want to offer screening on an opportunistic basis. Because of increased prevalence and risk of complications, the RACGP red book recommends all young people under 30 years of age have a chlamydia test once per year.2,3

In those who ask for a screen, it’s always worth checking what has prompted this request. Patient request may be prompted by a recent exposure or a particular infection (thus the importance of window periods for detection of the various infections), or patients may be concerned that their partner may have had sex with others. Many patients have symptoms that they don’t volunteer unless specifically asked. If a person has requested a screen it can be assumed that he or she would expect some further questioning about risk (Table 3.2).

A discussion around condom usage is particularly relevant in STI and blood-borne virus prevention. It is recommended however that all tests be requested for both men and women regardless of their reported condom usage. The brevity of the risk assessment will depend on the patient’s willingness to answer questions about his or her risk, especially if he or she has not presented for this reason. It can be useful to normalise the offer for blood-borne virus and STI screening, for example ‘I offer all young people STI screening once a year’. The screening guidelines for men who have sex with men may also be used to normalise the offer of screening (Figure 3.1).4

Instances in which to offer opportunistic testing include:

- any visit related to contraception or pregnancy, particularly in patients under 30 years of age
- any visit for cervical screening
- a request for a general check-up by a young person.

In these instances it is useful to know firstly if a person is sexually active and, if so, questions such as ‘have you had a change in sexual partner since your last visit’ may be useful to trigger opportunistic testing in a woman presenting for a repeat prescription for oral contraception for example or even asking ‘would you like a chlamydia test?’

Patients may be scared to provide accurate information regarding behaviours that have placed them at risk of exposure to blood-borne viruses and STIs for several reasons including:

**TABLE 3.2 A more detailed risk assessment – useful questions**

<table>
<thead>
<tr>
<th>Sexual practice</th>
<th>Condom usage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you consider that you might be at risk of HIV or another sexually transmitted infection? (Detail what activities may put someone at risk if necessary.)</td>
<td>Do you or your partner/s use condoms? Always, or how often?</td>
</tr>
<tr>
<td>Do you have any concerns about HIV or other STIs (e.g. chlamydia, herpes)?</td>
<td>Can you describe the situations in which you don’t use condoms?</td>
</tr>
<tr>
<td>When did you have sex last? (An important question for women if there is any chance of pregnancy)</td>
<td>Do you have any problems using condoms?</td>
</tr>
<tr>
<td>Who was it with – someone you knew well, or a casual contact?</td>
<td></td>
</tr>
<tr>
<td>How many sexual partners have you had over the last 3 months?</td>
<td>Have you ever had any tattoos? If yes was that here in Australia or overseas?</td>
</tr>
<tr>
<td>Are you currently in a relationship? (it’s also important not to assume that this is the person with whom the patient has sex).</td>
<td>Have you ever been in gaol?</td>
</tr>
<tr>
<td>Have you had any other sexual partners?</td>
<td></td>
</tr>
<tr>
<td>Are your sexual partner’s male, female or both?</td>
<td>How many standard drinks do you have in a week?</td>
</tr>
<tr>
<td>What types of sexual activity do you engage in with your partner?Vaginal?Oral?Anal? (Clarification may be needed)</td>
<td>Do you use any recreational drugs? (If yes seek further details)</td>
</tr>
<tr>
<td>Have you ever had an STI (e.g. chlamydia, herpes, gonorrhoea) or check-up for STIs?</td>
<td>Have you ever injected drugs? (If yes, then further details regarding sharing of equipment and when the person last injected should be sought – see Appendix 4)</td>
</tr>
<tr>
<td>In the past year were you ever paid for sex?</td>
<td>When did you last inject?</td>
</tr>
<tr>
<td></td>
<td>Have you shared needles or other injecting equipment?</td>
</tr>
</tbody>
</table>
**FIGURE 3.1** Australian sexually transmissible infection and HIV testing guidelines for asymptomatic men who have sex with men 2014

### Australian Sexually Transmitted Infection & HIV Testing Guidelines 2014

**FOR ASYMPTOMATIC MEN WHO HAVE SEX WITH MEN**

Men who have sex with men (MSM) in Australia are disproportionately and increasingly affected by sexually transmissible infections (STIs) including HIV. This has been attributed, in part, to changes in sexual behaviour such as reduction in condom use for anal intercourse in recent years. Many STIs do not lead to symptomatic presentations, therefore regular STI testing will identify a large number of infections which would otherwise remain undiagnosed and untreated. The term "men who have sex with men" is simply a behavioural descriptor and is not considered a sexual identity, although most MSM in Australia identify as gay.

These guidelines have been developed to encourage regular STI screening of MSM, including those with HIV, who do not have symptoms of STIs. The recommendations include STI testing at anatomical sites other than the location of any symptoms which may have prompted a clinical consultation.

After behavioural risk assessment and appropriate pre test discussion, **all of the STI tests** listed should be offered to:

- **All men who have had any type of sex with another man in the previous year** → **At least once a year**

- **All MSM who fall into one or more categories listed below:**
  - any unprotected anal sex
  - more than 10 sexual partners in six months
  - participate in group sex
  - use recreational drugs during sex
  - are HIV-positive:
    - syphilis serology: at each occasion of CD4/VIQ monitoring
    - chlamydia/gonorrhoea testing: consider at each occasion of CD4/VIQ monitoring
  → **Up to 4 times a year**

<table>
<thead>
<tr>
<th>SITE SPECIMEN</th>
<th>STI</th>
<th>TECHNOLOGY</th>
<th>COMMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharyngeal swab</td>
<td>Chlamydia &amp; gonorrhoea</td>
<td>NAAT</td>
<td>Self-collected or clinician-collected</td>
</tr>
<tr>
<td>Anorectal swab</td>
<td>Chlamydia &amp; gonorrhoea</td>
<td>NAAT</td>
<td>Self-collected or clinician-collected</td>
</tr>
<tr>
<td>First void urine*</td>
<td>Chlamydia</td>
<td>NAAT</td>
<td>Alternative: self-collected or clinician-collected penile meatal swab</td>
</tr>
<tr>
<td>Serology</td>
<td>Syphilis</td>
<td>EIA*</td>
<td>If HIV negative</td>
</tr>
<tr>
<td>Serology</td>
<td>HIV</td>
<td>EIA*</td>
<td>Test if not vaccinated. Vaccinate if antibody negative</td>
</tr>
<tr>
<td>Serology</td>
<td>Hepatitis A</td>
<td>HAV IgG EIA*</td>
<td>Test if not vaccinated. Vaccinate if antibody negative</td>
</tr>
<tr>
<td>Serology</td>
<td>Hepatitis B</td>
<td>HIV core antibody, surface Antigen EIA*</td>
<td>Test if not vaccinated. Vaccinate if no history or documentation of full vaccination course</td>
</tr>
<tr>
<td>Serology</td>
<td>Hepatitis C</td>
<td>HCV IgG EIA*</td>
<td>Only in HIV-positive or if history of injecting drug use</td>
</tr>
</tbody>
</table>

---

* VLQ: Viral load
* NAAT: nucleic acid amplification test
* EIA: Enzyme-Linked ImmunoSorbent Assay
* PCR: Polymerase Chain Reaction
* *: First void specimen is a first part of the urine stream. Not first urine of the day and not void after arousal.
* HCV: Hepatitis C virus
FIGURE 3.1 continued…

**RATIONALE FOR KEY STI TESTING RECOMMENDATIONS**

**Use of Neisseria gonorrhoeae (NG) and Chlamydia trachomatis (CT) NAATs.** NAATs are now widely used in Australia when testing urine, urethral, anorectal and pharyngeal samples for NG and CT. Where possible, collect NG culture swab before treatment of NAAT confirmed NG to assess antibiotic sensitivity of the NG isolate.

**Anal NG and CT infections.** All MSM should be offered anorectal swabs even if they do not report receptive anal sex. Receptive anal sexual practices such as receptive fellatio, toy insertion or oral-anal sex are risk factors for anal NG and CT, even in men who use condoms for receptive anal intercourse. Anal STIs are also independent risk factors for HIV in HIV-negative MSM, so identification and treatment of anal STIs by regular testing is likely to reduce the risk of HIV acquisition.

**Pharyngeal NG and CT infections mostly occur without concurrent anogenital infection, are asymptomatic, and can be the source of anogenital infections among MSM.** Compared with pharyngeal NG, pharyngeal CT is relatively rare among Australian MSM. However, recent studies overseas have identified a higher prevalence of pharyngeal CT among MSM than previously reported and pharyngeal CT is likely to be long-lasting in the absence of treatment. Testing MSM for both pharyngeal infections is therefore recommended.

**Self-collected samples (urethral, pharyngeal and anorectal swabs) are acceptable and effective at detecting NG and CT using NAATs.**

**Repeat testing.** Repeat testing at 3 months after NG and CT infections is recommended to detect reinfection.

**Testing in HIV positive MSM.** MSM with HIV account for up to 50% of infectious syphilis notifications and mathematical modelling indicates 3-monthly syphilis testing of these MSM could significantly impact on syphilis control efforts within Australia. HIV-positive MSM are also at particularly high risk of anal NG & CT, thus more frequent STI testing should be encouraged in this group. Due to evidence of Hepatitis C (HCV) sexual transmission among HIV positive MSM, all asymptomatic HIV positive MSM should have annual HCV testing.

**Testing reminders.** SMS and email reminders have been shown to increase detection of STIs as well as increasing retreating rates among MSM, both, after an STI diagnosis, and as reminders for regular testing. Therefore STI/HIV testing reminders are recommended for MSM.

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(Used with permission from Templeton DJ, Read P, Varma R, et al.)
• experience of discrimination within the health system and from health-care workers on the basis of drug use or sexual behaviour
• non-acceptance of his or her own behaviours and an inability to discuss these behaviours with any other person, even a health professional
• the desire to disassociate from past risk behaviours
• cultural shame and language barriers
• fear that confidentiality will be breached.

People may not have participated in high-risk behaviour at all. They may simply have had an unprotected heterosexual encounter (which has transmitted HIV) with someone whose own previous high-risk behaviour is unrecognised. Many women with HIV infection fall into this category. Making a diagnosis in these situations is dependent on retaining an open mind about the possibility of infection and a low threshold of suspicion prompting testing. Another reason for not reporting high-risk behaviour may be other psychological issues.

Clinical assessment: might this patient have a blood-borne virus or an STI?

Many patients with STIs or with chronic HIV, HBV or HCV infection are asymptomatic. The diagnosis relies on the clinician retaining an index of suspicion in all clinical situations, an assessment of risk and opportunistic testing.

Patients with acute HIV, hepatitis C virus (HCV), hepatitis B virus (HBV) hepatitis A virus (HAV), disseminated gonococcal infection, primary herpes infection and secondary syphilis may present with systemic symptoms (Chapters 4 and 5). HIV, secondary syphilis or hepatitis virus infections should be considered in any patient with a febrile illness, particularly if there is a possibility of recent exposure to one of these pathogens. When symptoms of chronic infection with blood-borne viruses occur, they are often non-specific (e.g. fatigue, myalgia and fevers).

Symptoms and signs of moderately advanced HIV infection include weight loss, chronic diarrhoea, fevers, lymphadenopathy, oral candidiasis, seborrhoeic dermatitis, herpes zoster, frequent or severe recurrent oral or genital herpes and oral hairy leukoplakia (Chapter 6).

Symptoms and signs of early chronic HBV and HCV infection are more non-specific and include intermittent or chronic fatigue, abdominal discomfort and headaches. Most patients have no symptoms until advanced liver disease ensues. Symptoms and signs of more advanced chronic hepatitis virus infection include the exanthemata of chronic liver disease (palmar erythema, spider naevi), while decompensated cirrhosis (liver failure) is associated with the development of ascites, splenomegaly and abdominal venous distension (Chapters 6 and 7). Some early STIs are usually asymptomatic, while symptoms and signs of complications of STIs vary depending on the clinical condition (Chapter 13: Primary care management of STIs).

A more detailed drug-use history

Recreational drug use and recent or current injecting drug use indicate that a more detailed drug history may be required to determine ongoing risk of blood-borne virus transmission, particularly in those who inject. In addition, a more detailed history allows the clinician to assess the individual’s motivation to change and his or her current drug use. It is also an opportunity to promote safer injecting practices if ongoing use is likely. If testing for a blood-borne virus is positive then a more detailed injecting history also helps determine the duration of infection and potentially helps identify others who may have been exposed.

It is important that the clinician ask specific questions around injecting behaviour as part of a broader discussion about injecting drug use.

• Whether and when any needles or other drug injecting equipment (such as swabs, waters or filters) were shared
• The types of drugs injected
• The frequency of drug use
• The duration of drug use
• The most recent occasion of use
• Whether the patient is drug dependent
• Any complications from drug use.

Interviews about drug use should be informed by a basic knowledge of common injecting drug-use equipment and practice, and the potential for HIV and HCV transmission at all stages of the injecting
process. Chapter 16 provides details of some relevant referral and information services for people who inject drugs.

Drugs that may be injected include performance-enhancing substances such as steroids, as well as amphetamines, ecstasy, benzodiazepines and opiates such as heroin. Frequency and duration of use vary considerably; many people are recreational drug users but may be suffering psychosocial consequences of their drug use. Alcohol, methamphetamine use, other stimulants or nitrous oxide can increase sexual risk-taking behaviour so this aspect should be explored. Using recreational drugs during sex is associated with higher rates of STI and HIV transmission in men who have sex with men and is an indication for more frequent screening (Table 3.3).

Health promotion about harm or risk reduction with regard to injecting drug use requires the clinician to have knowledge of safe procedures and information about local services, such as needle and syringe programs and harm reduction information (Chapter 16 and Appendix 4). There will be times when it is appropriate to discuss whether the patient wishes to reduce or cease drug use, and whether he or she would like a referral to an appropriate treatment service. It is important that the patient leads this discussion, rather than the clinician pressuring the patient. Providing information on non-injecting routes of drug administration, such as snorting and swallowing drugs, will reduce the risks of blood-borne virus transmission. If the patient is drinking alcohol at hazardous or harmful levels, the healthcare provider should discuss strategies such as alternating alcoholic drinks with either water or a soft drink. Patients and clinicians can access a range of agencies and resources providing information, advice and support for minimising harmful substance use (Chapter 16).

A more detailed sexual history

If the brief risk assessment has identified areas of concern then a more detailed sexual history should be taken. This allows the clinician to explore an individual’s sexual behaviour in more detail and work with the individual to identify risk reduction strategies. A more detailed sexual history may be necessary if an STI or HIV is diagnosed or if a brief sexual history taking identifies any specific areas of concern. The clinician may assist the patient to identify any sexual partners who will need to be contacted regarding their possible exposure to an STI or HIV and to develop specific risk reduction strategies. For more detail on biomedical prevention particularly for gay men see Chapter 14.

Sexual orientation or identity does not always equate with a particular behaviour; therefore information about sexual practices, condom use and the risk behaviours of sexual partners is more specific and useful than the patient’s stated sexual orientation and marital or partnership status. Common and often incorrect assumptions are related to heterosexuality, monogamy and preferred sexual practice. A clinician’s familiarity with sexual terminology allows patients to feel comfortable, to open up and be open about their sexual behaviour. It instils confidence that their clinician is interested, current and happy to discuss all issues openly.

The clinician should ascertain whether vaginal or anal penetration has taken place. Questions about anal sex should be asked particularly of men who have sex with men, it should be determined whether penetration was receptive, insertive or both, e.g. was the patient a ‘top’, a ‘bottom’ or both? Oral sex confers a lower risk of HIV transmission, but several STIs including HSV, syphilis, gonorrhoea and chlamydia are readily transmitted by oral sex, which may take the form of oro-genital (fellatio), oro-vulval (cunnilingus) and oro-anal (rimming/anilingus) sex.

**FIGURE 3.2**

<table>
<thead>
<tr>
<th>Brief Sexual History</th>
</tr>
</thead>
<tbody>
<tr>
<td>“I’d like to ask you some questions about your sexual activity so we can decide what tests to do, is that OK?”</td>
</tr>
<tr>
<td>Are you currently in a relationship?</td>
</tr>
<tr>
<td>In the last 3 months, how many sexual partners have you had? How many partners have you had in the past 12 months?</td>
</tr>
<tr>
<td>Were these casual or regular partners?</td>
</tr>
<tr>
<td>Were your sexual partners male, female or both?</td>
</tr>
<tr>
<td>When was the last time you had vaginal sex/oral sex/anal sex without a condom?</td>
</tr>
<tr>
<td>In the past year were you ever paid for sex?</td>
</tr>
<tr>
<td>Have you previously been diagnosed with an STI?</td>
</tr>
<tr>
<td>Is there anything else that is concerning you?</td>
</tr>
</tbody>
</table>

(Reproduced with permission by NSW STI Programs Unit 2014)

**CASE STUDY 2 Sexual health context: an STI indicates the need for HIV testing**

A young, openly gay man in a regional city presents to a GP with a 4-day history of a very painful anus, which he assumes to be haemorrhoids, as he has suffered them previously. He says he has never had anal sex. On examination, there are extensive perianal ulcers and his GP takes swabs for herpes, gonorrhoea and chlamydia.

The patient is appalled that he could have an STI, and he has never had an STI. He reports a negative HIV antibody test about 2 years ago and he has been vaccinated successfully against HBV. He averages about three different sexual partners a month that he meets on the internet and has never injected. Upon further questioning, the patient reports that he and his most recent partner had done ‘just about everything two guys can do, short of fucking’. When questioned, he agrees that there had been some oral-anal contact both ways. The GP suggests pharyngeal and anal swabs and raises the issue of HIV testing. The patient readily agrees. The anal swab returns positive for herpes simplex virus (HSV) type 1 but cultures for gonorrhoea and the HIV antibody test are negative.
Clinicians should keep the possibility of sex work in mind and also, with male patients, the possibility that they are clients of sex workers. Questions about these matters need to be asked sensitively and only when a very good rapport has been established with the patient.

When discussing sexual practices it is important that the clinician and patient understand each other. The clinician may seek to maximise understanding through specific questioning, explanation and clarification. “Have you been sexually active?” may be taken to mean only vaginal or anal penetrative sex, so it may be appropriate to indicate that the question also relates to oral or other sexual activity. Specific questions such as ‘Do you ever have oral sex, where you suck on his penis?’ may be useful in establishing the level of risk. Questions relating to condom usage (Table 3.2) form part of risk assessment and provide an opportunity to discuss effective safer sex practices. In addition, discussion may address other safer sex measures.

Every primary care clinician will have in his or her practice some men who have sex with men (MSM) and some women who have sex with women (WSW) even if those patients do not always, or even often, engage in same-sex sexual activity. These patients have particular sexual health needs and clinicians should gain some basic knowledge about homosexually active men and women—it is counter-productive to reveal ignorance about lifestyle and sexual practices when trying to take a sexual history.

WSW are at low risk for STIs generally, however a significant percentage of WSW do have sex with men throughout their lives and therefore may be exposed to transmission of STIs (including high-risk human papillomavirus [HPV] types like 16 and 18) from male partners. These women should be encouraged to participate in the cervical screening program. WSW who have sex with MSM may be at high risk of exposure to HIV and STIs. For all patients including WSW, oral sexual practices and sharing sex toys will lead to STI transmission risk.

Prevention and harm reduction messages

Opportunities for educating about harm reduction and safer sex often arise during an assessment of risk. The primary care clinician may take these opportunities to ensure the patient understands the risks of sexual activity and drug use, as well as safer practices (Table 3.4). Many people will be well informed about safer practices but may not adhere to them all the time. Occasions of risk-taking can be identified and explored. It is important that the patient feels he or she can discuss episodes of unsafe behaviour without being judged or lectured. Gaining an understanding of the patient’s perspective and responding to his or her emotions will help in facilitating behavioural change. Common themes in a discussion of risk-taking may include negotiating safer sex with partners, drug and alcohol consumption, or apathy and depression.
The clinician may engage the patient in generating his or her own solutions to unsafe practices. Questions such as ‘Has this happened before?’, ‘What did you think or do on that occasion?’ ‘What happened then?’ and ‘How did you feel?’ may assist the patient to identify and avoid particular situations and reinforce safer practices. Acknowledgment of the difficulties a person may face in trying to adopt or negotiate safer sex or safer injecting may facilitate a more productive discussion. Consideration should be given to the difficulty in challenging entrenched cultural norms or conventional ideas such as ‘men are in charge of condoms’ or ‘he looked young and healthy, so he couldn’t have HIV’.

Encouraging patients to have sexual health check-ups can be a harm reduction strategy in itself. However, safer sex practices which allow unprotected oral sex do not always protect patients from common STIs like chlamydia, gonorrhoea and genital herpes. For MSM, those who change partners frequently, those with multiple partners and those having unprotected anal sex it is sensible to recommend regular check-ups (Figure 3.1). For others, the recommendation should be that a sexual health check-up should follow the break-up of a relationship, a casual encounter or the commencement of a new sexual partnership.

Clinicians need to think about members of special groups in their practice, learn about such groups and tailor their preventive and harm reduction messages so they are appropriate and relevant for their special needs. Such groups include MSM, WSW, adolescents and young adults, Indigenous Australians, physically and intellectually handicapped people and people from culturally and linguistically diverse communities.

Being able to show empathy and to convey an understanding of the patient’s situation is an essential component of the clinician’s expertise. However in some situations, the clinician may decide to refer the patient to another service or clinician, a community group or a specialist counsellor or educator (Chapter 16). Having written information can be useful in ensuring that a patient can have information to take away. This information is available through Hepatitis Councils, peer-based injecting drug-user groups and State and Territory AIDS Councils. Table 3.4 provides a check-list of general tips on safe sex and harm reduction education.

### Cross-cultural issues

There is potential for misunderstanding and communication breakdown when talking to patients about sexual practice and drug use in a culturally and linguistically diverse country such as Australia. Use of professional interpreters rather than family members will aid communication. For clinicians who work with a significant number of patients from a particular ethnic or cultural group, it can be useful to learn about relevant attitudes and practices prevalent in that cultural group.

Alternatively, the clinician can ask the patient whether the line of questioning is appropriate. In situations where it is difficult to consult with a patient of the opposite sex, arrangements should be made for the patient to see another clinician if possible.

### Patients with disabilities or psychiatric problems

People of all ages and abilities may be sexually active. Some individuals, such as people with intellectual or physical disabilities, may have particular problems accessing information and harm reduction and safe sex measures, such as condoms. They may also have particular difficulty negotiating safer sex. Ensuring adequate knowledge and support for people with disabilities or psychiatric problems may require involvement with family and carers, and consideration of issues specific to the patient’s particular situation.

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**Table 3.4 Tips on safe sex and harm reduction messages**

- Check the patient’s base-line knowledge about HIV, STIs, HBV, HCV. Correct misconceptions and, if necessary, provide basic information
- Check the patient’s understanding about sexual and drug-use behaviours that carry risk of transmission
- Explore safer sex and safer using options (such as non-injecting techniques) specific to the patient’s needs
- Discuss where new fits can be obtained and the correct method of cleaning fits (Appendix 4)
- Discuss circumstances in which unsafe practice has taken place or is likely to occur
- Discuss the link between alcohol and other drug use and unsafe sex
- Encourage regular sexual health check-ups except for those in stable monogamous relationships
Summary

A brief risk assessment should be conducted whenever the opportunity presents itself either via patient request for STI and blood-borne virus screening or if opportunistic testing moments arise. A more detailed sexual and drug-use history-taking may be conducted in those who are diagnosed with an STI or blood-borne virus or who the clinician identifies are at higher risk of acquiring or transmitting an infection. Clear and non-judgmental communication facilitates accurate history-taking and appropriate management. Impediments to history-taking may be overcome by application of good communication techniques, consideration of the patient’s particular needs, consultation with colleagues and a willingness to learn about alternative lifestyles. However, if impediments persist, referral to another clinician or service is recommended.

References


Bibliography


CHAPTER 4 EXPOSURE AND ACUTE HIV INFECTION

2014 REVIEW

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Introduction

Early diagnosis, monitoring and treatment of patients with recently acquired human immunodeficiency virus (HIV) infection may alter the long-term course of HIV disease. Knowledge of the clinical signs and symptoms of primary HIV infection, as well as the serological and virological markers, enables early HIV diagnosis by clinicians and provides patients with timely options for intervention choices, as well as opportunities for receiving appropriate referral, support and education on prevention of transmission. Antiretroviral treatment of HIV diagnosed during primary infection provides an opportunity to limit transmission at a time when most patients are highly infectious.

Pathogenesis of acute HIV infection

Knowledge of the pathogenesis of primary HIV infection in adults helps the clinician to understand HIV-related pathology testing. Within 12–24 hours of exposure, cells at the site of a mucosal infection are infected with HIV. Forty-eight hours after exposure, HIV has spread to regional lymph nodes where rapid replication occurs within immune cells, primarily CD4 cells. Cells in the gut become infected as well as those of the central nervous system and the skin. Over the next 5–40 days, the host immune response to massive HIV viraemia results in the production of antibodies and a cytotoxic T-cell response mounted by CD8+ T-lymphocytes. The T-helper CD4 cells control the cytotoxic response but also are infected by HIV. Early in the course of infection, memory CD4+ cells are selectively depleted from the circulation; as disease advances, CD4+ cells of both the naive and memory phenotype are lost. The characteristic depletion of lymphocytes in HIV disease appears to result from factors such as cellular destruction, diminished cellular production, and cellular sequestration in lymphoid tissue. The number of circulating CD4+ lymphocytes is widely used as a measure of global immune competence and provides a predictor of the immediate risk for opportunistic illnesses. The changes in immune response to HIV can be observed by monitoring CD4 and CD8 cell counts in the peripheral blood.

The flu-like symptoms of primary HIV infection are thought to be caused by the release of cytokines during the process of infection and immune response. As a result of the immune response, the blood concentration of the virus (the viral load) falls and new CD4 cells are produced by the bone marrow via the thymus. For reasons that are unclear, the cytotoxic CD8 cell and antibody responses are not able to clear or completely control HIV replication, as occurs with some, but not all viral infections.

Detecting primary HIV infection

Acute retroviral syndrome

Familiarity with the range of presentations associated with primary HIV infection (also called acute retroviral infection or seroconversion illness) enables the early diagnosis and management of HIV infection. Clinical suspicion of acute HIV infection should be followed by a thorough risk assessment (Chapter 3). As the symptoms and signs of acute HIV infection are similar to those of many common infections, the presence of HIV infection is more likely when a recent high-risk exposure has been reported.

**KEY POINTS**

- Early diagnosis of HIV disease has significant potential benefits and the likelihood of ongoing transmission may be reduced through implementation of safe sex and risk reduction strategies including early antiretroviral therapy (ART).
- Acute HIV infection may be difficult to distinguish from other acute viral illnesses. Clinical features that should alert the clinician to the possibility of acute HIV infection in the presence of a mild-to-severe flu-like illness include recent sexually transmissible infection, sudden onset, a ‘glandular fever-like’ illness, meningeval involvement, rash, prominent gastrointestinal symptoms and transient neurological symptoms.
- Symptoms of primary HIV infection can usually be managed in the primary care setting by the general practitioner. Decisions about antiretroviral therapy need to be made in conjunction with an experienced HIV clinician.
- While newly diagnosed patients may require ongoing specialist services from a range of providers, the general practitioner remains an important source of initial and continued information and support.
Table 4.1 Symptoms and signs of primary HIV infection

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Frequency (%)</th>
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<tbody>
<tr>
<td><strong>Generalised</strong></td>
<td></td>
</tr>
<tr>
<td>Fever</td>
<td>&gt;80</td>
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<tr>
<td>Lethargy and general malaise</td>
<td>&gt;70</td>
</tr>
<tr>
<td>Myalgia and arthralgia</td>
<td>50-70</td>
</tr>
<tr>
<td>Lymphadenopathy</td>
<td>40-70</td>
</tr>
<tr>
<td>Night sweats</td>
<td>50</td>
</tr>
<tr>
<td><strong>Gastrointestinal</strong></td>
<td></td>
</tr>
<tr>
<td>Pharyngitis</td>
<td>50-70</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>30</td>
</tr>
<tr>
<td>Oral ulcers</td>
<td>10-30</td>
</tr>
<tr>
<td><strong>Neurological</strong></td>
<td></td>
</tr>
<tr>
<td>Headache</td>
<td>40-70</td>
</tr>
<tr>
<td>Aseptic meningitis</td>
<td></td>
</tr>
<tr>
<td>Transient reversible neurological signs (neuropathies, Guillain-Barré syndrome)</td>
<td>Rare</td>
</tr>
<tr>
<td><strong>Skin</strong></td>
<td></td>
</tr>
<tr>
<td>Rash</td>
<td>40-80</td>
</tr>
<tr>
<td>Genital ulcers</td>
<td>5-15</td>
</tr>
<tr>
<td><strong>Initial laboratory finding</strong></td>
<td></td>
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<tr>
<td>Thrombocytopenia</td>
<td>45</td>
</tr>
<tr>
<td>Leukopenia</td>
<td>40</td>
</tr>
<tr>
<td>Raised liver enzymes</td>
<td>20</td>
</tr>
<tr>
<td><strong>Diseases caused by transient immunosuppression</strong></td>
<td></td>
</tr>
<tr>
<td>Oral/oesophageal candidiasis</td>
<td>Rare</td>
</tr>
<tr>
<td>Gut infections</td>
<td></td>
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<tr>
<td>Pneumocystis jiroveci pneumonia (PCP)</td>
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</table>

Signs and symptoms

Signs and symptoms of acute HIV infection can present as early as 3 days or as late as 10 weeks following infection. Most commonly they occur at 10–14 days. The onset of symptoms often coincides with the appearance of HIV antibodies although the patient may be HIV antibody negative (ELISA) for up to 3 weeks after onset of symptoms. The duration of the illness is most commonly 4 to 14 days but may be longer. Approximately 50–90% of patients report signs or symptoms suggestive of primary HIV infection at the time of seroconversion. Patients who experience symptomatic primary HIV infection appear to have more rapidly progressive HIV disease than those who do not.

The frequency of symptoms varies and severity ranges from very mild to very severe (Table 4.1). No single symptom distinguishes acute HIV infection from other acute viral illnesses. However, there are some factors that should alert the clinician to the possibility of acute HIV infection in the presence of a flu-like illness such as:

- Epstein-Barr seronegative ‘glandular fever-like’ illness
- ‘Flu-like’ symptoms outside usual influenza season (e.g. myalgia, arthralgia, headache, malaise)
- Illness of sudden onset
- Fever for more than 3 days
- Maculopapular rash
- Meningeal involvement
- Transient neurological syndromes (e.g. Guillain-Barré syndrome, neuropathies)
- Recent evidence of sexually transmissible infections or genital ulcers
- Recent high-risk exposure

Recent risk exposure

Patients reporting recent risk exposure should be thoroughly assessed and monitored for HIV infection. The possibility of HIV infection can be an emotionally difficult time for the patient. It is important and required that patients are provided with a full pre-test discussion in preparation for the possibility of a positive diagnosis, as well as provided with the required information about HIV infection (Chapter 9).

Patients reporting an occupational HIV exposure in their workplace (nurses, doctors, dentists, dental nurses, chiropodists, ambulance officers, police officers, and others) or non-occupational HIV risk event (such as unprotected anal or vaginal intercourse; shared injecting equipment or other exposure to potentially infectious body fluids where the source is known or
**TABLE 4.2 Pathology tests for HIV screening and diagnosis of primary HIV infection**

<table>
<thead>
<tr>
<th>Screening test</th>
<th>Test type</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>4th Generation Combo enzyme immunoassay (EIA)</td>
<td>Immunoassay</td>
<td>82% of Australian laboratories use this test as the standard HIV antibody screening test. This is a combined p24 antigen plus HIV antibody test; it will become positive before a test using HIV antibody alone in acute infection.</td>
</tr>
<tr>
<td>3rd Generation HIV antibodies EIA</td>
<td>Immunoassay</td>
<td>EIA may take up to 3 weeks to become positive after onset of clinical signs and symptoms.</td>
</tr>
<tr>
<td>Rapid HIV</td>
<td>Immunochromatography</td>
<td>Most rapid HIV tests test for HIV antibodies only. One rapid HIV test (Determine HIV-1/2 Ag/Ab Combo) also tests for p24 antigen but its performance does not match laboratory antigen tests. Rapid HIV tests are suitable for screening when used outside the context of acute HIV infection. Rapid HIV tests require approximately 7-14 days longer than 4th generation EIAs to detect HIV antibodies. Rapid HIV tests are only suitable for screening; reactive rapid HIV test results must be confirmed by laboratory testing. 82% of Australian labs use this test as the standard HIV antibody screening test. This is a combined p24 antigen plus HIV antibody test and so it will become positive before a test using HIV antibody alone in acute infection.</td>
</tr>
<tr>
<td><strong>Supplementary test</strong></td>
<td></td>
<td></td>
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<tr>
<td>Proviral HIV DNA</td>
<td>(Qualitative) reverse transcriptase polymerase chain reaction (RT PCR)</td>
<td>Sometimes ordered when acute HIV infection is suspected. This test detects viral sequences and can be positive up to 8 days before HIV specific antibodies appear. False negative results have been reported in testing of non-B subtype infections. This test is not TGA licensed.</td>
</tr>
<tr>
<td>P24 antigen</td>
<td>Immunoassay</td>
<td>May become positive within a few days of symptoms and be absent after 2 weeks.</td>
</tr>
<tr>
<td>HIV RNA (viral load)</td>
<td>Reverse transcriptase polymerase chain reaction (RT PCR) Quantitative PCR</td>
<td>May become positive within a few days. However, the quantitative viral load assay is generally not recommended to diagnose acute HIV infection due to a reported low false-positive rate in the acute setting (usually indicated by low viral levels).</td>
</tr>
<tr>
<td><strong>Confirmatory test</strong></td>
<td></td>
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<tr>
<td>HIV antibodies (Western Blot)</td>
<td></td>
<td>Used to confirm an HIV diagnosis following a reactive or indeterminate result on a screening assay. Western Blot may take up to 3 weeks to become positive after onset of clinical signs and symptoms. During acute HIV infection Western Blot tests may return indeterminate results. Retesting after a short period (determined in consultation with a laboratory or specialist centre) to confirm an HIV diagnosis is recommended.</td>
</tr>
</tbody>
</table>

Note: Other tests may be indicated and should be performed in conjunction with specialist centres and laboratories.

TGA: Therapeutic Goods Administration (Last updated 1 May 1 2014; last reviewed 1 May 2014)

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likely to have HIV infection) and the exposure has occurred within the previous 72 hours should be considered for HIV post-exposure prophylaxis (PEP) (see Chapter 14). PEP is a 28-day course of anti-HIV drugs that may significantly reduce the risk of HIV infection from a given HIV exposure event. Following assessment, all individuals with high-risk exposures should be referred to an approved antiretroviral prescriber, an HIV or sexual health centre or the emergency department of a hospital that can provide PEP. PEP efficacy is time dependent, therefore, it is essential that assessment and referral be made as early as possible within the 72-hour period since exposure (Chapter 14).

Potential exposure to HIV often indicates a risk of hepatitis B virus (HBV) and hepatitis C virus (HCV) infection as well as a risk of other sexually transmitted
infections (STIs). Consequently, investigations for STIs prevalent in the community, HBV and HCV should be considered in the context of acute HIV infection (Chapters 7 and 8).

**Investigations**

When risk assessment or clinical presentation indicates the possibility of acute HIV infection, laboratory testing ensures correct diagnosis. HIV antibody tests (HIV ELISA and Western Blot) may be negative or equivocal up to 3 weeks after the start of primary HIV illness (Table 4.2). Internationally, a number of rapid HIV antibody tests have become available. These tests are suitable for screening, but positive results must be confirmed by laboratory testing and retesting is strongly recommended in patients with an HIV risk event in the 12 weeks preceding the test. In the context of acute HIV infection, the use of rapid HIV tests is not appropriate for diagnostic purposes as these tests are insensitive when antibody titres are low. However, HIV viraemia appears in the blood about 10 days after infection and so allows for direct detection of virus particles or proteins (antigens) in the absence of antibodies. If available, tests for viral antigens included in later generations of HIV antibody and antigen assays may facilitate early diagnosis of HIV infection during the window period.

It should be noted that the molecular tests currently available in Australia (listed in Table 4.2) do not have approval from the Therapeutic Goods Authority (TGA) which is required for their use in the primary diagnosis of HIV infection. Molecular tests should therefore be considered confirmatory (of an indeterminate serology result) when used in this setting.

Interpreting test results with regard to acute HIV infection can be confusing and, if necessary, clinicians are advised to seek guidance from their pathology laboratory or the National Serology Reference Laboratory (Chapter 16).

**Management of acute HIV infection and recent exposure**

**Acute HIV infection**

People with primary HIV infection can usually be managed in the community by their own general practitioner (GP), with the support of either an HIV-experienced GP or a hospital-based specialist. Most of the physical symptoms of primary HIV infection are treatable with simple analgesics and anti-emetics. Occasionally hospital admission may be required for rehydration or management of rare manifestations such as encephalitis or Guillain-Barré syndrome (Table 4.3).

**TABLE 4.3 Management of primary HIV infection checklist**

- Referral to an HIV-experienced GP and/or a hospital-based clinician
- Support for the primary care clinician from an HIV-experienced GP and/or a hospital-based clinician
- Physical symptom relief such as analgesia for headache, myalgia and arthralgia, and anti-emetics for nausea
- Appropriate treatment for opportunistic infections
- Psychosocial support of the patient by the clinician and referral to an experienced mental health professional as appropriate
- Early and frequent follow-up

**Very early treatment with antiretroviral therapy – a controversy**

Treatment of HIV infection during the early stages of chronic infection (the ‘hit HIV hard and early’ concept) has been revisited. Some HIV clinicians treat primary HIV infection with combination antiretroviral medications after one or more of the confirmatory tests have returned positive. The rationale for treatment during this phase of HIV disease is to minimise immune system damage, to lower the viral replication ‘set point’ (Chapter 1) and to minimise viral dissemination throughout the body.

It has been argued that the early immune response to HIV may require the ongoing presence of HIV antigens and that disturbing this response may be harmful. In addition, short-term and long-term side effects of therapy can be considerable (Chapter 10). The SPARTAC study (an international trial comparing three different strategies of intervention in patients with recent HIV infection to determine whether early treatment for a limited duration delays damage to the immune system and prolongs time to initiation of long-term antiretroviral therapy) has recently published its findings suggesting that ‘very early treatment of HIV infection’, shortly after the virus was acquired, could avert some of the damage done to the patient’s immune system. These findings also suggest that, if this protective effect was sustained, then the inexorable downhill course of the infection could be attenuated.16 The study has found no evidence of adverse effects of ART interruption on the clinical outcome, and no evidence that treatment within the first 6 months of infection led to the virus becoming resistant to the drugs or that coming off the treatment course led to unexpected deaths or damage to the immune system. Conversely, the study found no direct clinical benefit of treatment at this very early stage. According to some international antiretroviral treatment guidelines and the ASHM commentary, ‘all decisions to start ART should be made by the individuals with HIV, in
consultation with their health-care providers and on the basis that they are fully informed and supported in their decision-making. Clinicians inexperienced in the management of HIV infection need to contact an HIV-experienced GP or a hospital centre to discuss further management.

If the patient proceeds with treatment during primary infection, HIV-inexperienced clinicians are encouraged to maintain contact with their patients as part of the treatment team, especially as newly diagnosed people with the infection require considerable information and support from a trusted and accessible source.

Contact tracing

Contact tracing of people who may have been sexually exposed (or exposed through the sharing of injecting equipment) to the patient with primary HIV infection within the 12 weeks preceding the onset of symptoms or the serological diagnosis is important for further case identification and the reduction of onward transmission. The clinician should also enquire about recent blood, sperm or egg donation.

Discussion with the patient regarding contact tracing should be sensitively raised soon after diagnosis. The notification of contacts requires patient consent. While of great importance, contact tracing is not usually the immediate priority; support and engagement with appropriate care take precedence in managing a patient diagnosed with primary HIV infection. An exception to this general principal is when HIV negative (or HIV status unknown) sexual or injecting partners may have been exposed in the previous 72 hours. In this instance, timely post-exposure prophylaxis (see Chapter 14) may prevent HIV acquisition and should be prioritised.

People with HIV can face significant discrimination, including from members of the health-care sector, and rejection by family, friends and sexual partners. Where HIV infection may have resulted from illegal or stigmatised activities, fear of disclosure or prosecution may also be of concern. In addition, there are implications for employment, insurance and immigration. People with newly diagnosed HIV are often very concerned about confidentiality, and contact tracing can be difficult. Modes of contact tracing exist that can help to preserve patient confidentiality and inexperienced clinicians are advised to seek assistance from an HIV or sexual health clinic.

Where a patient is unwilling to proceed with contact tracing, and a clinician has concerns that another person may be at risk of infection, referral of the matter to relevant public health authorities must be considered (see Chapter 15, in particular Notification of Third Parties). State or territory public health guidelines are also useful.

A comprehensive guide to contact tracing for HIV (among other sexually transmitted infections) is available at: http://www.ashm.org.au//images/Publications/Monographs/ctm/ctm_2010.pdf

Public health notification

HIV is a notifiable infection. In most states and territories notification is undertaken by the diagnosing clinicians and pathology laboratories, although there are differences in legislative and regulatory requirements (Chapter 15).

Supporting newly diagnosed patients

The ongoing psychological adjustment of patients to HIV infection can be affected by the nature of early consultations with their doctor after diagnosis. In particular, having a long consultation when the HIV diagnosis is given has been positively correlated with better long-term adjustment, as have the quality of information given and the attitude of the person giving the diagnosis.

Newly diagnosed patients have major issues to face and adjustments to make during early consultations. For example, patients may suddenly confront their mortality or have concerns about future income and relationships with partners, family and friends.

Newly diagnosed patients may also have concerns about how to avoid putting others at risk of HIV infection, and how to manage disclosure of their HIV status to current and future sexual partners, family, work colleagues and others. Patients may also fear discrimination from others on the basis of their HIV status. There are numerous information, peer support and social support programs offered by community based HIV organisations to assist newly diagnosed patients adjust to and help manage HIV infection (see the box below for links to more information and supporting resources for clinicians and patients).

Patients with children often have concerns about how their children will deal with the diagnosis and whether they will be able to continue to provide for the children materially and emotionally. For women of childbearing age, there may be fears and concerns about how HIV affects their future reproductive life. Simple acceptance, in the face of perceptions of social stigma and discrimination, may be the most valuable support a clinician can offer in early consultations. Patients may also need help in deciding whether to disclose their HIV status and, if so, to whom.

Emotional support and acceptance can also assist the person to make beneficial alterations to his or her lifestyle, such as changes to diet and exercise, reduced drug and alcohol use and practicing safer sex.
Severe flu or HIV seroconversion illness?
John is a 39-year-old engineer who presents to his general practitioner, Dr Lewis, with a flu-like illness in April. He has been unwell for a week with muscle aches and pains, fever, headache and retro-orbital pain, particularly upon lateral gaze. He has spent the last 4 days on the couch at home and has noticed that his urine is very dark.

Dr Lewis considers a differential diagnosis of HIV seroconversion illness and conducts a risk assessment. "I need to ask some sensitive questions. Nowadays we need to ask people about risk behaviours for HIV when they present with an unusual flu-like illness. Have you done anything in the past few weeks that might worry you or might put you at risk for HIV? What I mean is, any unprotected sex or sharing needles?"

John relates that he recently started a relationship with Sam and that they have been having sex without condoms for 4 months. They intended to have HIV tests but hadn’t got around to it. While John was HIV-negative when tested last November, he is unsure when Sam was last tested. John has been vaccinated against hepatitis A and B and reports never using needles.

Given his high-risk activity for HIV transmission, Dr Lewis suggests HIV testing to John: "While lots of other common viruses cause symptoms like this, we should consider testing for HIV infection. The first illness that some people get when they have HIV infection can look like flu. Following pre-test counselling, John consents to testing for HIV and HCV. Although rapid HIV testing is available at Dr Lewis’ practice, Dr Lewis suspects that John may be seroconverting to HIV so does not order a rapid HIV test, due to the longer period following HIV exposure before a rapid HIV test is likely to detect HIV antibodies. Instead, he orders a Combo EIA test. Three days later, the laboratory rings Dr Lewis about John’s test results:

The results of the tests are as follows:

Results I:
- HIV antibody/antigen test – indeterminate
- Liver enzymes – slightly elevated
- Hepatitis C antibodies – negative

Given the indeterminate result of the Combo EIA test, the laboratory requests a repeat sample as soon as possible. Dr Lewis now requests an HIV proviral DNA test and Western Blot as well. The following results arrive from the laboratory this time:

Results II:
- HIV antibody/antigen test – negative
- HIV p24 antigen detected
- Western Blot test indeterminate
- HIV proviral DNA test – positive

John’s tests confirm a clinical diagnosis of HIV primary infection. He is referred to a general practitioner experienced in the management of HIV infection after indicating that he would prefer to see a community-based HIV clinician. After lengthy discussion about treatment options, the HIV-experienced clinician and John decide to go ahead with antiretroviral treatment.

Dr Lewis continues regular follow-up with John to address his ongoing medical and psychosocial needs following the HIV diagnosis. In addition to assistance in taking medications, John raises relationship and sexuality issues. Dr Lewis refers him to the local AIDS Council for support and offers written resources for HIV-positive people.

Support services and the role of the clinician
In addition to the support that clinicians can offer, patients should be referred to other agencies for information, counselling and support as appropriate (see box with further resources and Chapter 16). Research has identified the importance of contact with HIV-positive communities in helping newly diagnosed patients come to terms with their status and continue with their lives.

However, while acknowledging that specialist counselling may best meet the psychosocial needs of patients, clinicians must recognise that they may be the first and most important source of this support and information in their patients’ lives. This is especially true during the early stages of HIV infection.

Maintaining contact with the patient after the initial diagnosis, as either the key HIV-treating clinician or as a partner in care, helps to support the patient through the many difficulties that may lie ahead.

Further resources for clinicians and people newly diagnosed with HIV
- Further resources for clinicians
  - www.ashm.org.au/making_a_new_HIV_diagnosis
- Further resources for people newly diagnosed with HIV
  - National Association of People with HIV Australia resources
    - www.napwha.org.au/living-hiv/just-diagnosed
  - Australian Federation of AIDS Organisations (AFAO) ‘Next Steps’ booklet (information and advice for people newly diagnosed with HIV)
    - http://tiny.cc/nextsteps
    - (treatment guidelines in this resource not current)
  - HIV&AIDS The Basics – a safe guide forgay men
    - http://www.hivpozgaysex.org.au

Other sexually transmitted infections
Primary HIV infection often indicates an additional risk of HBV and HCV infection as well as other STIs. Consequently, investigations for STIs prevalent in the community, HBV and HCV should be considered in the context of acute HIV infection.

The detection and treatment of STIs is very important in its own right, but their treatment may also reduce the risk of onward HIV transmission (Chapter 13). For patients who present with high-risk HIV exposure, Australian non-occupational post-exposure prophylaxis guidelines recommend baseline testing for chlamydia, gonorrhoea (Chapter 8), hepatitis B and syphilis with repeat syphilis testing at 3 months. The same STI tests are appropriate after any sexual risk exposure including sexual assault (also refer to national and state guidelines regarding management of sexual
assault). The use of post-exposure prophylactic antibiotics against STIs like chlamydia is not recommended after risk exposure, although they may be indicated after some types of sexual assault.

**Summary**

The primary care clinician has a key role in identifying cases of primary HIV infection and facilitating the clinical investigations, support, contact tracing and management of people with newly acquired infection. Following diagnosis of primary HIV infection, referral to an HIV-experienced clinician is recommended for consideration of antiretroviral therapy. Assistance with contact tracing is readily available from Sexual Health and HIV clinics. To reduce the risk of infection after a high-risk exposure to HIV, post-exposure prophylaxis may be taken within 72 hours of the exposure. Reported exposure provides an opportunity to review risk behaviours, safer sex practices, harm minimisation strategies, assessment for other STIs, and to evaluate whether antiretroviral prophylaxis is appropriate (Chapter 14). Provision of information and psychosocial support are key elements of management following a possible HIV exposure or diagnosis with primary HIV infection.

**References**


2. Kelleher AD, Zaunders JJ. Decimated or missing in action: the primary care clinician has a key role in identifying cases of primary HIV infection and facilitating the clinical investigations, support, contact tracing and management of people with newly acquired infection. Following diagnosis of primary HIV infection, referral to an HIV-experienced clinician is recommended for consideration of antiretroviral therapy. Assistance with contact tracing is readily available from Sexual Health and HIV clinics. To reduce the risk of infection after a high-risk exposure to HIV, post-exposure prophylaxis may be taken within 72 hours of the exposure. Reported exposure provides an opportunity to review risk behaviours, safer sex practices, harm minimisation strategies, assessment for other STIs, and to evaluate whether antiretroviral prophylaxis is appropriate (Chapter 14). Provision of information and psychosocial support are key elements of management following a possible HIV exposure or diagnosis with primary HIV infection.


CHAPTER 5 EXPOSURE AND ACUTE VIRAL HEPATITIS

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<table>
<thead>
<tr>
<th>Name</th>
<th>Position and Affiliation</th>
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<tbody>
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<td>Infectious Diseases Physician, Department of Immunology and Infectious Diseases, St Vincent’s Hospital; and Senior Lecturer, The Kirby Institute, UNSW Australia, Sydney NSW</td>
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<tr>
<th>Name</th>
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<tbody>
<tr>
<td>Jeffrey Post</td>
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</tbody>
</table>
**KEY POINTS**

- The hepatotropic viruses (HAV, HBV, HCV) cause most cases of acute hepatitis, although other infectious agents and drugs need to be considered. Acute HCV infection is often unrecognised due to its asymptomatic or non-specific presentation and imprecise diagnostic testing.

- Primary care clinicians should make a definitive diagnosis where possible, and refer patients with unclear diagnoses or rare, treatable conditions. Patients should be monitored for acute liver failure and hospitalised if signs are detected.

- Primary care clinicians play a critical role in the prevention of hepatitis virus infection. Interventions such as education, vaccination, contact tracing, post-exposure prophylaxis and public health notification are critical to the control of epidemics and prevention of disease in individuals at high risk along with harm reduction strategies such as needle and syringe programs.

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**Acute hepatitis**

**Epidemiology**

Acute viral hepatitis can be caused by the hepatitis viruses, A, B, C, D or E, although D and E are rare in Australia. In 2008 and 2012, 276 and 165 cases of hepatitis A virus (HAV) infection and 262 and 193 cases of newly acquired hepatitis B virus (HBV) infection, respectively, were reported. Fortunately, the rate of diagnosis of newly acquired HBV infection has been in decline for more than a decade. The reported number of diagnoses of newly acquired hepatitis C virus (HCV) infection increased from 365 to 466 in 2008-2012, but it should be acknowledged that many cases are asymptomatic and go undiagnosed. HBV and HCV transmission occurs predominantly among people with a recent history of injecting drug use.

Drug-induced liver injury (DILI), particularly paracetamol toxicity, should be considered in all cases of sudden liver enzyme derangement. Additionally other viral infections (including Epstein-Barr virus [EBV] and cytomegalovirus [CMV]) and alcoholic hepatitis may cause a sudden rise in liver function tests although typically these are less severe and accompanied by other symptoms or a relevant history. Aetiologies of chronic liver disease may present clinically as acute hepatitis, including autoimmune hepatitis, Wilson’s disease and chronic HBV with an acute flare.

**Outcomes of acute hepatitis**

Less than 1% of all cases of acute hepatitis virus infection with jaundice develop acute liver failure. In children, infection with HAV is largely asymptomatic or causes a mild self-limiting illness; in adults, symptomatic infection is more common, but only one-third develop jaundice. Chronic infection does not occur with HAV in contrast to infection with HBV and HCV (Table 5.1). Infants and children with HBV infection are likely to develop chronic infection (up to 90%) as opposed to adults (<5%).

After acute infection with HCV, approximately 75% of adults will develop chronic infection. Chronic HBV and HCV infection may progress to cirrhosis, liver failure and hepatocellular carcinoma. Extra-hepatic manifestations in chronic HBV and HCV infection are frequent and include (but are not limited to) mixed cryoglobulinaemia, glomerulonephritis and dermatological disease in HCV and glomerulonephritis and polyarteritis nodosa in HBV.

**Symptoms and signs of acute hepatitis**

In the pre-icteric phase, patients with acute viral hepatitis often have non-specific symptoms such as myalgia, vomiting, fatigue, malaise, headache and diarrhoea with right upper quadrant discomfort. A self-limiting illness resembling acute serum sickness (with maculopapular rash, diffuse arthralgia) due to immune complex formation occurs in 10% of patients with acute HBV and 5-10% of those with acute HCV.

Physical examination before the development of jaundice is usually unremarkable; hepatomegaly (10%), splenomegaly (5%) and lymphadenopathy (5%) may be present. In general, patients should not have signs of chronic liver disease (but remember acute super-infection, i.e. acute HCV or hepatitis D virus [HDV] infection in a patient with chronic HBV infection). Acute liver failure is rare. The primary features are encephalopathy, jaundice and coagulopathy.

**Incubation periods**

The average time from exposure to the development of symptoms varies for the three major viruses:

- **HAV** – 3 weeks (range 2–7 weeks)
- **HBV** – 10 weeks (range 4–26 weeks)
- **HCV** – 7 weeks (range 2–21 weeks)
TABLE 5.1 Outcomes of acute viral hepatitis

<table>
<thead>
<tr>
<th>Hepatitis A virus</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Approximately 0.1% of patients with HAV develop acute liver failure with most (60%) recovering spontaneously; of those who develop acute liver failure in the developed world, liver transplantation occurs in 25-30% and death without transplantation in 10-15%</td>
<td></td>
</tr>
<tr>
<td>Chronic hepatitis does not occur following HAV infection. Atypical manifestations may occur, including prolonged cholestatic hepatitis in &lt;5% and relapsing hepatitis in up to 20%</td>
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<tr>
<td>Life-long immunity occurs after infection</td>
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<tr>
<td><strong>Hepatitis B virus</strong></td>
<td></td>
</tr>
<tr>
<td>Less than 1% of cases develop acute liver failure. For patients with HBV-related acute liver failure in the developed world, 25% demonstrate spontaneous recovery, 50% require liver transplantation and 25% die without transplantation</td>
<td></td>
</tr>
<tr>
<td>90% of infants with infection acquired at birth develop chronic hepatitis; less than 5% of adults with acute HBV infection develop chronic hepatitis</td>
<td></td>
</tr>
<tr>
<td>Up to 90% of infants born to HBeAg-positive mothers who do not receive any form of prophylaxis develop HBV infection; administration of hepatitis B immunoglobulin (HBIG) at birth followed by routine HBV vaccination in the first 6 months of life has reduced transmission rates to 5-10%</td>
<td></td>
</tr>
<tr>
<td>Those who go on to develop chronic infection are at risk of cirrhosis, hepatocellular carcinoma and liver failure</td>
<td></td>
</tr>
<tr>
<td>Those with chronic infection have persistent HBsAg and are infectious to others</td>
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<tr>
<td>Those who clear infection have life-long immunity, maintain anti-HBc, but may not preserve anti-HBs</td>
<td></td>
</tr>
<tr>
<td><strong>Hepatitis C virus</strong></td>
<td></td>
</tr>
<tr>
<td>Acute HCV infection is often unrecognised due to its asymptomatic or non-specific presentation</td>
<td></td>
</tr>
<tr>
<td>Acute liver failure is rare, but may occur in persons with pre-existing liver disease such as chronic HBV infection</td>
<td></td>
</tr>
<tr>
<td>Approximately 75% of adults with acute HCV infection develop chronic HCV infection</td>
<td></td>
</tr>
<tr>
<td>5% of infants born to women with HCV infection develop HCV infection</td>
<td></td>
</tr>
<tr>
<td>Spontaneous clearance usually occurs within the first 6-12 months and is confirmed by two undetectable HCV RNA tests at least 1 month apart</td>
<td></td>
</tr>
<tr>
<td>Treatment in the acute and early chronic stages of HCV infection is often highly successful</td>
<td></td>
</tr>
<tr>
<td>Those who go on to develop chronic infection are at risk of cirrhosis, hepatocellular carcinoma and liver failure</td>
<td></td>
</tr>
<tr>
<td>If infection resolves and the virus is cleared (either spontaneously or with treatment), the person is NOT immune and can be re-acquire the infection. After resolution of infection, antibodies persist for a variable amount of time (20 years in some cases).</td>
<td></td>
</tr>
</tbody>
</table>

**Diagnostic approach**

The diagnosis of acute viral hepatitis relies predominantly on serological testing, although other features are important to consider. The history should elicit:

- Symptoms consistent with acute hepatitis
- Symptoms suggestive of an alternative diagnosis (e.g. EBV)
- Epidemiological clues (Table 5.2 and Chapter 2)
- Alcohol and drug use (including illicit drugs, over-the-counter and prescription medications and complementary therapies)
- Travel and vaccination history
- Family history of liver disease

An awareness of relevant epidemiology is useful (i.e. an outbreak of HAV in 2009 – 564 cases).

Examination should include evaluation for fever, jaundice, rash, drug injection sites, tattoos, body piercings, arthritis, tender hepatomegaly, splenomegaly and hepatic encephalopathy (altered mental state, asterixis, fetor hepaticus). A general physical examination should be performed.

**Non-serological investigations**

Basic investigations should include liver enzymes, full blood count and coagulation profile. Specific results can assist in establishing the cause of acute hepatitis. For example:

- In viral hepatitis, the alanine aminotransferase (ALT) is usually 10–100 times the upper limit of normal (ULN) with the aspartate aminotransferase (AST)/ALT ratio less than one.
- In alcoholic hepatitis, the ALT is generally 2–10 times ULN with the AST/ALT ratio greater than 1.5; bilirubin is usually elevated.
- In drug-induced liver injury, a mixed profile may be seen with raised hepatic (AST and ALT) and cholestatic (alkaline phosphatase and gamma glutamyltransferase [GGT]) markers; for example, in paracetamol overdose, liver function tests (LFTs) peak at 72-96 hours after ingestion with severe hepatotoxicity manifest by ALT and AST >1000 – 10,000 IU/mL with AST>ALT.
• Atypical lymphocytosis may suggest a viral aetiology and thrombocytopenia may indicate acute alcohol exposure or the presence of chronic liver disease with portal hypertension.
• The coagulation profile may reveal a prolonged prothrombin time or international normalised ratio (INR) suggestive of liver failure.

Serological investigations
All serological investigations should be undertaken after appropriate pre-test discussion with the patient and the results given in conjunction with post-test discussion (see Case Study 1 and Chapter 9). Specific serological investigations are indicated in Figure 5.1. If the diagnosis is unclear, the initial serological investigations may be repeated after 1–2 weeks with HCV RNA polymerase chain reaction (PCR) testing included. Investigation for EBV infection and less common causes of hepatitis can be undertaken at this time. If the diagnosis is still unclear, specialist referral is indicated.

Key considerations when testing for acute viral hepatitis

**HAV**
In the context of acute HAV infection, anti-HAV IgM is invariably present, being both sensitive and specific. False negative results are rare.

**HBV**
Acute HBV infection is best detected by testing for HBsAg and anti-HBc IgM. However, it should be recognised that anti-HBc IgM (usually at low titre) may also become positive in the setting of a flare of chronic HBV. Anti-HBc IgG and anti-HBs appear later in the course of the illness. HBV DNA PCR is not routinely used as a diagnostic tool in acute HBV infection. In patients with HBV infection, hepatitis D virus (delta or HDV) should also be considered, particularly in a patient with chronic HBV who develops a new episode of acute hepatitis or if the disease is severe. Anti-HDV IgG and IgM testing is available at a limited number of laboratories.

**HCV**
In acute HCV infection, HCV antibody may be present at the onset of clinically apparent hepatitis or may develop in the following weeks. HCV antibodies are usually present within 3 months of exposure but may take up to a year to develop especially in HIV-positive individuals. As HCV antibody may be non-reactive or equivocal in recently acquired infection or immunocompromised hosts, HCV RNA PCR should be performed to detect viraemia; HCV RNA should be detectable 2-14 days after exposure.

**Supportive therapy**
Most cases of acute viral hepatitis do not require hospitalisation. Hospital assessment is recommended for patients who are unable to maintain an adequate fluid intake; have an ALT greater than 1000 IU/L; a progressive rise in bilirubin (>60 mmol/L); and/or coagulopathy (INR >1.3). The most ominous biochemical signs are falling ALT with rising bilirubin and INR as these signs indicate severe liver injury with significant loss of hepatocytes. Altered mental status (suggesting encephalopathy) is a sinister sign in acute hepatitis.

All non-essential medications should be ceased during acute hepatitis. Analgesics are generally not required and aspirin, opioids and sedatives should be avoided. Small doses of paracetamol may be used for the management of constitutional symptoms. Patients should be advised to avoid alcohol. If the cause of hepatitis is unclear, a careful medical review should be undertaken and potential hepatotoxins should be ceased. Small meals may be easier for the patient to tolerate.

**TABLE 5.2 Clues to diagnosis – epidemiological and exposure risks**

<table>
<thead>
<tr>
<th>Clues to diagnosis – epidemiological and exposure risks</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Knowledge of current epidemiology, e.g. HAV cluster, sexual (non-injecting) HCV transmission in HIV-positive men</td>
</tr>
<tr>
<td>• Contact with a case of acute or chronic hepatitis</td>
</tr>
<tr>
<td>• Travel to endemic area without vaccination or passive prophylaxis – HAV, HBV, yellow fever</td>
</tr>
<tr>
<td>• Travel to endemic areas – HAV, HBV, HEV, dengue fever, leptospirosis</td>
</tr>
<tr>
<td>• Unprotected penetrative sex – HBV (and HCV if HIV-positive MSM)</td>
</tr>
<tr>
<td>• Unprotected oro-anal sex – HAV</td>
</tr>
<tr>
<td>• Occupation, e.g. sewerage workers, child-care workers – HAV</td>
</tr>
<tr>
<td>• Occupation, e.g. health-care workers – HAV, HBV, HCV</td>
</tr>
<tr>
<td>• Injecting drug use – HAV, HBV, HCV</td>
</tr>
<tr>
<td>• Alcohol consumption</td>
</tr>
<tr>
<td>• Family history – HBV, Wilson’s disease, alpha1-antitrypsin deficiency</td>
</tr>
<tr>
<td>• Country of birth – HAV, HBV, HCV</td>
</tr>
<tr>
<td>• Tattoos and body piercings – HBV, HCV</td>
</tr>
<tr>
<td>• Blood transfusion, medical and dental procedures – HBV, HCV</td>
</tr>
<tr>
<td>• Needle-stick injury or other significant occupational exposure – HBV, HCV</td>
</tr>
<tr>
<td>• History of imprisonment – HCV</td>
</tr>
</tbody>
</table>
Specific therapy

HAV

There is little role for specific agents in the management of acute HAV infection. With prolonged cholestasis, a short course of corticosteroids has been suggested to hasten resolution of pruritus and lower serum bilirubin levels, but is of unproven benefit. Cholestyramine can be administered if pruritus is bothersome.

HBV

In the case of acute HBV, more than 95% of adults will recover spontaneously and seroconvert to anti-HBs without antiviral therapy. In the setting of fulminant or protracted severe acute HBV, the use of a nucleos(t)ide analogue (tenofovir or entecavir as for chronic HBV) should be considered under expert guidance, along with assessment for liver transplantation. The distinction between severe acute HBV infection and reactivation of chronic hepatitis B may be difficult. However, nucleos(t)ide analogue treatment is appropriate in both cases (Chapters 1 and 12).

HCV

Treatment of recently acquired HCV with interferon-based regimens results in far greater rates of sustained virological response (SVR) than in chronic HCV infection. SVR rates are typically in the region of 75-80% even with genotype 1 infection compared to 30-40% in chronic HCV. Treatment duration can also be shortened. Current international guidelines recommend administration of pegylated-interferon (with or without ribavirin) for 12-24 weeks. As approximately 25% of people with HCV infection will spontaneously clear the virus within the first 12 – 16 weeks of infection, monitoring in the early phase of infection before initiating treatment is important. Factors associated with a greater chance of spontaneous clearance include jaundice, female sex, genotype 1 infection and favourable IL28B status.

The management of recently acquired HCV infection is the focus of ongoing clinical trials, particularly in the emerging era of directly-acting antiviral therapy, and referral to a specialist for further advice should be considered for all patients.

Section 100 of the Pharmaceutical Benefits Scheme (PBS) does not currently fund treatment for acute HCV infection.

Clinical monitoring

Liver function tests should be performed once or twice per week in addition to an assessment of coagulation profile and clinical status.

While rare (<1% of cases of HAV and HBV), acute liver failure is the most serious complication of viral hepatitis and refers to the development of severe acute liver injury with encephalopathy and impaired synthetic function (INR ≥1.5) in a patient without cirrhosis or pre-existing liver disease. HCV alone does not appear to be a significant cause of acute liver failure in the absence of co-infection with HBV. In viral hepatitis, acute liver failure results from massive, immune-mediated hepatocyte necrosis. Risk factors for the development of acute liver failure are not fully understood, but older age and concomitant liver disease have been implicated. Death may occur even when the liver has begun to regenerate.

The initial symptoms of acute liver failure are non-specific, and include malaise, nausea, vomiting, anorexia and right upper quadrant pain. As liver failure progresses, jaundice and subtle mental state changes (e.g. lethargy, difficulty sleeping) develop, followed by confusion and coma. The patient may go from healthy to moribund in 2–10 days. Supportive laboratory findings include prolonged prothrombin time (PT)/INR, elevated serum ALT/AST, elevated bilirubin, hypoglycaemia and elevated serum creatinine.

The management of acute liver failure begins with the recognition that patients with coagulopathy or encephalopathy may die. Due to the potential for rapid clinical deterioration and the need for close monitoring, patients with acute liver failure are best managed in hospital. Liver transplantation may be required in a small proportion of cases. Referral to a liver transplant unit is indicated where:

- the patient is in a remote hospital
- there is any evidence of encephalopathy
- there is worsening coagulopathy.

It is recommended that patients are followed for a minimum of 6 months following acute hepatitis to determine if chronic HCV and HBV infection develop. For HCV, repeatedly normal ALT and an undetectable HCV RNA on two occasions more than 4 weeks apart indicate viral clearance. Figure 5.1 provides details of HBV follow-up.

Contact tracing

Contact tracing of individuals who may have been exposed during the infectious period of acute hepatitis should be undertaken to enable preventive measures to be implemented. Discussion with the patient regarding contact tracing is appropriate. The clinician may ask the patient to consider recent blood-to-blood or sexual contacts as well as recent blood donations and clinical procedures. With regard to HAV, more proactive household and occupational contact tracing may be warranted. It is recommended that primary care practitioners keep up to date with the relevant state or territory guidelines.
**FIGURE 5.1** Diagnostic and management algorithm for cases of acute hepatitis

**Diagnosis**
- Case of Acute Hepatitis
  - History and examination including epidemiological and other clinical clues
  - Drug history: Over the counter and prescription medication, illicit drugs, toxins
  - Test for anti-HAV IgM, anti-HBc IgM, HBsAg, anti-HCV Ab, and others as suggested by history

**Management**
- Notification
- Contact tracing
- Notification. Contact tracing. Testing, post exposure prophylaxis. Education and prevention
- Test for anti-HAV IgM, anti-HBc IgM, HBsAg, anti-HCV Ab, and others as suggested by history

**Prevention**
- Paracetamol overdose. Seek expert advice. Refer to hospital for management
- Cease any non-essential drugs that may cause hepatitis
- Acute HAV
- Acute HBV (or flare of chronic HBV)
- Consider testing for acute HDV
- Acute HCV (if compatible clinical illness or documented seroconversion) OR Chronic HCV
- Repeat initial serology. Test for EBV IgM and IgG.
- Re-assess history, symptoms and signs. Other diagnostic tests

**Diagnosis**
- Acute HAV
- Acute HBV (or flare of chronic HBV)
- Consider testing for acute HDV
- Acute HCV (if compatible clinical illness or documented seroconversion) OR Chronic HCV
- Repeat initial serology. Test for EBV IgM and IgG.
- Re-assess history, symptoms and signs. Other diagnostic tests

**Management**
- Monitor for acute liver failure (and refer to hospital as appropriate)
- Assess index for need for other vaccinations, testing, prevention and education strategies
- Follow for at least 6 months to determine if chronic infection develops. Monitor ALT, HBsAg, and anti-HBs
- Follow for at least 6 months to determine if chronic infection develops. Monitor ALT, HBsAg, and anti-HBs
- If chronic infection develops, assess as discussed in chapter 7 and 11.
Public health unit notification
Cases of acute hepatitis are notifiable by doctors and diagnostic laboratories. Public health units coordinate the response to outbreaks of acute hepatitis and can provide advice on the appropriateness of post-exposure prophylaxis for suspected contacts.

Opportunistic diagnosis and prevention strategies
An episode of acute hepatitis should lead to risk assessment and testing for other transmissible infections with similar routes of transmission (Chapters 1–3). The opportunity for promoting preventive and harm reduction measures, such as HAV/HBV vaccination, and use of sterile injecting equipment among those who inject drugs should be taken. Referral to Hepatitis Australia may be useful for ongoing supportive education in several languages.

Specialist and hospital referral
Referral to hospital is appropriate when the primary care clinician determines that an individual has severe hepatitis or acute liver failure. Specialist referral is recommended:
- Where the clinician is unable to make a definitive diagnosis
- Where multiple diagnoses appear to co-exist
- For consideration of antiviral therapy in acute hepatitis C and B (if fulminant or protracted severe)
- Where other, treatable conditions have been diagnosed.

Work
Persons with HAV infection are infectious for up to a week after the onset of jaundice and should not work. Workers in high-risk areas, for example food handlers and child-care workers, may require extended leave. Given that cases in high-risk workers will usually be followed up by the local public health unit, advice should be sought from the relevant state or territory health authority (Chapter 15). Persons with acute HBV or HCV infection do not need to be excluded from work if they are clinically well, unless they are health-care workers who perform exposure-prone procedures (Chapter 14). Further information may be obtained from relevant state and territory health departments or medical registration boards (Chapter 15).

Post-exposure management
The management of a person potentially exposed to viral hepatitis will vary according to the nature of the exposure, the available information about the source of the exposure, knowledge of the exposed person’s immunity to viral hepatitis and the time

CASE STUDY 1 Acute HCV infection
Mark, a 45 year-old man with well-controlled HIV infection (CD4 count 825 cells/µL, HIV viral load undetectable), presents for routine 6-monthly review. The clinician notes that Mark’s liver function tests are abnormal with elevated ALT (60 U/L [normal range: 0-30 U/L]) and AST (49 U/L [normal range: 0-30 U/L]).

Mark states that he has been well since last review, but that his regular male partner had a severe flu-like illness 6 weeks ago. He reports intermittent injecting drug use (crystal meth, 3-4 times per year) and unprotected anal intercourse with his regular partner and other HIV-positive casual male partners. He is compliant with his antiretroviral regimen (raltegravir, tenofovir, emtricitabine) and takes no other prescribed or over-the-counter medications. He consumes 3-4 standard drinks per day.

Investigations are ordered to determine the aetiology of the deranged liver function tests and follow-up is arranged.

On review 1 week later, Mark says that he has been unwell with abdominal discomfort, anorexia, headache and myalgia. Examination is significant for jaundice and right upper quadrant tenderness. There are no stigmata of chronic liver disease and no evidence of ascites or encephalopathy. Repeat liver function tests reveal hyperbilirubinaemia (conjugated bilirubin 78 µmol/L [normal range: 0-18 µmol/L]) and further elevation in ALT (900 U/L) and AST (635 U/L). Serum tests demonstrate that Mark is immune to HAV and HBV (HBs Ab >1000) and has evidence of previously treated syphilis (syphilis Ab reactive, RPR non-reactive). Anti HCV-Ab is non-reactive.

Given the clinical suspicion, further diagnostic tests are requested. HCV RNA is detectable at 47 859 911 (7.7 log10) IU/mL and Mark is confirmed to have acute genotype 1a HCV infection.

Education is provided regarding modes of transmission and prognosis (including development of chronic HCV infection and chance of spontaneous clearance). Safe injecting practices are discussed and referral to appropriate services is offered. Mark is surprised to discover that his regular male partner had a severe flu-like illness 6 weeks ago. He reports intermittent injecting drug use (crystal meth, 3-4 times per year) and unprotected anal intercourse with his regular partner and other HIV-positive casual male partners. He is compliant with his antiretroviral regimen (raltegravir, tenofovir, emtricitabine) and takes no other prescribed or over-the-counter medications. He consumes 3-4 standard drinks per day.

In consultation with a specialist, liver function tests and HCV viral load are monitored with marked fluctuation noted over the subsequent months (see chart). Despite his symptomatic presentation, he does not demonstrate spontaneous clearance and HCV RNA remains detectable over 6 months of follow-up. Consideration is given to treatment of recently acquired HCV.
that has elapsed since the exposure. Exposed individuals may self-present for assessment or may be detected after contact tracing. As well as an assessment of the current exposure, an assessment of future or ongoing risk should be made and preventive strategies put into place. In cases of workplace exposure to viral hepatitis or potentially infected bodily fluids, appropriate documentation should be completed for worker’s compensation purposes.

Exposure to HIV as well as viral hepatitis should be considered following contact with blood or bodily fluids. See Chapter 4 for a discussion of HIV post-exposure prophylaxis.

**Source status**

Details of the source’s clinical status should be obtained where possible. Cases of clinically apparent acute hepatitis represent the most straightforward category but cases of exposure to bodily fluids from people without acute hepatitis may be encountered. An assessment should be made of risk factors for blood-borne virus infections in the source. If the source is available and willing, testing for viral hepatitis and HIV should be conducted with full pre-test and post-test discussion.

In cases where the source has a history of HBV infection, an urgent assessment of HBsAg status will guide decisions regarding infectivity and hence recommendations regarding post-exposure prophylaxis. Knowledge of the source’s HCV status does not change the immediate management, as post-exposure prophylaxis is not currently available. However, the infectivity of a source who is repeatedly negative for HCV RNA in serum is probably negligible.

**Exposed person’s immunity**

After exposure to HAV, no specific tests of immunity are undertaken. Prophylaxis is given to all close contacts.

After exposure to HBV, an urgent assessment of the exposed person’s immunity is required. This entails a history of previous HBV infection or immunisation and response to vaccination. If the history is unclear, or response to previous immunisation is unknown, then tests to ascertain immunity to HBV may be undertaken if the results can be obtained rapidly. Administration of HBIG should not be delayed beyond 72 hours. Check anti-HBc (as a marker of previous infection) and anti-HBs (if assessing response to immunisation). If such tests are not available within this time frame, the person should be assumed to be non-immune.

Post-exposure prophylaxis

**TABLE 5.3 Post-exposure prophylaxis (PEP)**

<table>
<thead>
<tr>
<th>Hepatitis A virus</th>
<th>Hepatitis B virus</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>At-risk population</strong></td>
<td>Non-immune person in contact with HBsAg positive individual via:</td>
</tr>
</tbody>
</table>
| Close contacts (i.e. household, sexual) of people with HAV infection 2 weeks before and 1 week after onset of jaundice | • heterosexual or homosexual sex  
• people who injects drugs (PWID)  
• mother-to-child exposure  
• occupational exposure |
| **Management strategy** | 1. Hepatitis B immunoglobulin (HBIG) | 2. Hepatitis B vaccination |
| Monovalent hepatitis A vaccine | • <12 h from birth  
• <72 h after percutaneous, ocular, mucous membrane exposure | |
| *See text if <12 months, chronic liver disease, immunocompromised, contraindication to vaccine* | • <14 days after sexual exposure  
• Dose: adult 400 IU, child 100 IU | |

**HAV**

Post-exposure prophylaxis (PEP) should be offered to close contacts of people with HAV infection, including household and sexual contacts who have had contact with the index case in the 2 weeks before, or up to 1 week after the onset of jaundice (Table 5.3). Traditionally, PEP for HAV has consisted of parenteral administration of normal human immunoglobulin (NHIG). More recent evidence suggests that monovalent hepatitis A vaccine has similar efficacy to NHIG in immunocompetent people aged over 1 year and is recommended in this group. NHIG is reserved for the following contacts: <12 months of age, individuals with chronic liver disease, immunocompromised hosts (including HIV-positive individuals), those with a contraindication to the vaccine.

If the patient is a food handler, all other non-immune food handlers at his or her place of work should receive PEP. Where the patient is associated with a day-care or preschool facility (attendee child, staff member or household contact of either) and there is any concern about the possibility of further transmission, PEP should be offered to non-immune children and staff in the relevant age groups or classes at the facility. PEP is not indicated for contacts of sporadic cases in the school or work setting.
HBV
Individuals who are HBsAg-positive (HBsAg+) should be considered infectious. Non-immune individuals with a definite HBV exposure through heterosexual or homosexual sex, sharing of injecting equipment, mother-to-child exposure or occupational exposure (percutaneous, ocular, mucous membrane) should be given HBIG as soon as possible (<12 hours after birth; <72 hours after percutaneous/ocular/mucous membrane exposure; and < 14 days after sexual contact). The dose of HBIG is 400 IU for adults and 100 IU for children. Concomitantly, HBV vaccination should be administered into a separate (deltoid muscle) injection site and a full course completed.

HCV
No post-exposure prophylaxis against HCV infection is currently available.

Post-exposure follow-up
After exposure to HAV, no specific serological testing is required. Clinical follow-up is sufficient.

For HBV and HCV, the aim of initial follow-up is to detect the development of acute and chronic infection. Serological follow-up after exposure to HBV and HCV should occur at 1, 3 and 6 months as both infections can have prolonged incubation periods. Detection of HCV RNA by qualitative PCR is currently funded by the government such that a single test can be undertaken for the diagnosis of acute HCV infection. Additional testing may be performed at the expense of the patient. Most cases are viraemic at 4 weeks, although some may only have transient viraemia that clears before this time. A single negative HCV RNA result does not exclude HCV infection and full serological follow-up represents the current gold standard of diagnosis.

Psychosocial issues
In managing patients who report potential exposure to viral hepatitis or patients who present with symptoms of acute viral hepatitis, a range of psychosocial issues may be addressed in a timely and sensitive manner. For example, risk behaviours may be explored and appropriate referral to community support or counselling services offered (Chapter 16).

The anxieties and concerns of the patient regarding transmission to sexual partners and family should be addressed by discussing modes of transmission and preventive strategies (Case study 1; Chapters 2 and 3). Describing potential health outcomes, as well as the process of determining infection status, may also assist the patient.

Prevention
Prevention of perinatal transmission
To prevent perinatal transmission of HBV infection, newborn babies of mothers with HBV infection should receive passive and active immunisation with HBIG and HBV vaccination, commencing within 12 hours of birth. While this strategy prevents most mother-to-child transmission, it may not be effective in a proportion of newborns born to highly viraemic (serum HBV DNA >7 log10 IU/mL) mothers (mostly HBeAg-positive), who carry a risk of vertical HBV transmission approaching 10% despite HBIG and vaccination. In this setting, consult with a specialist and consider the addition of a nucleos(t)ide analogue (NA) (lamivudine, tenofovir [pregnancy category B]) in the third trimester. If NA therapy is given only for the prevention of perinatal transmission, it may be discontinued within 3 months after delivery.

There are no effective strategies to prevent perinatal transmission of HCV, although avoidance of invasive foetal monitoring may be worthwhile. Potential benefits of caesarean section have not been demonstrated. Breastfeeding is safe unless blood is present in the milk e.g. when the nipples are cracked and bleeding.

Table 5.4 People for whom hepatitis A vaccine is recommended
(This vaccine is provided free under the National Immunisation Program for Aboriginal and Torres Strait Islander infants living in areas of higher risk [Queensland, Northern Territory, Western Australia and South Australia])

- Aboriginal and Torres Strait Islander children residing in the NT, Qld, SA and WA
- Travellers (≥1 year of age) to endemic areas
- People who live or work in rural and remote Indigenous communities
- Staff working in early childhood education and care
- Persons with developmental disabilities and their carers
- Health-care workers who provide care for substantial populations of Indigenous children
- Plumbers, sewerage workers or others in regular contact with untreated sewage
- Men who have sex with men
- People who inject drugs, including inmates of correctional facilities
- People with chronic liver disease, including post-liver transplantation
- People with chronic HBV or HCV infection or HBV/HCV co-infection
- Sex industry workers
TABLE 5.5 People for whom hepatitis B vaccination is recommended \(^{13}\)

<table>
<thead>
<tr>
<th>People for whom hepatitis B vaccination is recommended</th>
<th>People for whom hepatitis B vaccination is recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Infants and young children</td>
<td>• People with chronic liver disease and/ or HCV infection</td>
</tr>
<tr>
<td>• Young people aged 10 to 13 who have never received a primary course of HBV vaccine</td>
<td>• People with clotting disorders who require multiple blood product administration</td>
</tr>
<tr>
<td>• Solid organ and haematopoietic stem cell transplant recipients</td>
<td>• Health-care workers with direct patient or human tissue contact</td>
</tr>
<tr>
<td>• Household or other close contacts of people with HBV</td>
<td>• Inmates and staff of correctional facilities. People no longer in a custodial setting but who commenced but did not complete the vaccine course while in custody</td>
</tr>
<tr>
<td>• Sexual contacts of people with HBV (NB, these people should also be offered HBIG as post-exposure prophylaxis)</td>
<td>• People with developmental disabilities and staff involved in their care</td>
</tr>
<tr>
<td>• Men who have sex with men</td>
<td>• Embalmers, funeral workers</td>
</tr>
<tr>
<td>• People who inject drugs or are on opioid substitution therapy</td>
<td>• Travellers to hepatitis B endemic areas</td>
</tr>
<tr>
<td>• Haemodialysis patients and patients with severely impaired renal function in whom dialysis is anticipated</td>
<td>• Police, members of the armed forces, emergency services staff</td>
</tr>
<tr>
<td>• People with HIV infection or impaired immunity</td>
<td>• People adopting HBsAg+ children</td>
</tr>
<tr>
<td>• Tattooists and body piercers</td>
<td>• Sex industry workers</td>
</tr>
<tr>
<td>• Migrants from hepatitis B endemic countries</td>
<td>• Aboriginal and Torres Strait Islander people</td>
</tr>
</tbody>
</table>

Immunisation

HAV vaccination is recommended for populations at high risk (Table 5.4) \(^{13}\) Testing for immunity before immunisation is recommended for people born before 1950, for those who spent their childhood in endemic regions (China, south-east Asia, Pacific islands) and for those who report a previous diagnosis of hepatitis. The recommended schedule is an initial dose with a booster dose 6–12 months later.

HBV vaccine is provided free through the National Immunisation Program to all infants (at birth, 2 months, 4 months and 6 or 12 months). Vaccination of adolescents 10–13 years of age is recommended for all those in this age group who have not already received a primary course of hepatitis B vaccine. Refer to your state or territory health authority for further information about free HBV vaccine for this age group.

HBV vaccination is also recommended for populations at high risk of HBV (Table 5.5) and in many states and territories this is available for free. Refer to relevant states and territories to identify who has access to free HBV vaccination.

Vaccination is safe for people with HIV infection and other immunocompromised people, although protection may be less effective than the protective immunity produced in immunocompetent individuals, and as such four double doses, comprising two injections of the standard adult dose (using Engerix-B) on each occasion, at 0, 1, 2 and 6 months, is recommended. \(^{16}\) For people with HIV infection vaccination is ideally given when the CD4 count is greater than 350 cells/μL. \(^{17}\) However, in patients who present to care at a lower CD4 count, vaccination should not be deferred. \(^{17}\)

Serological confirmation of post-vaccination immunity is not required after routine child and adolescent vaccination but is recommended for some high-risk people, namely those with HIV infection. \(^{13}\) In this setting, anti-HBs titres should be obtained 1 month after completion of the vaccine series. Booster doses are not recommended in immunocompetent people but may be required for those with impaired immunity, in particular those with HIV infection or renal failure. The timing of administration of booster doses should be decided by regular monitoring of anti-HBs levels at 6-12 month intervals. \(^{14}\)

A combined vaccine for HAV and HBV is available and should be considered for individuals at risk of both infections (Table 5.6).

TABLE 5.6 People for whom hepatitis A and B vaccination is recommended \(^{13}\)

<table>
<thead>
<tr>
<th>People for whom hepatitis A and B vaccination is recommended</th>
<th>People for whom hepatitis A and B vaccination is recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>• People with HCV infection</td>
<td>• Travellers to, and expatriates living in, moderately to highly endemic areas for hepatitis A and B</td>
</tr>
<tr>
<td>• Men who have sex with men, sex industry workers, people who inject drugs, inmates of correctional facilities</td>
<td>• People who attend or work at facilities for people with developmental disabilities</td>
</tr>
<tr>
<td>• Health-care workers</td>
<td>• People with chronic liver disease</td>
</tr>
<tr>
<td>• Liver transplant recipients or solid organ transplant recipients who have chronic liver disease</td>
<td></td>
</tr>
</tbody>
</table>
There is no vaccine for HCV at this time.


**Education and harm reduction**

Education about risk reduction and harm reduction methods may lower the incidence of viral hepatitis in at-risk individuals. Chapter 3 discusses prevention and harm reduction messages and strategies.

Concurrent assessment for drug treatment programs may be considered for those who inject drugs. Referral to injecting drug user groups (such as the Australian Injecting and Illicit Drug Users League or a local equivalent) for education and support may be considered (Chapter 16).

Travellers require accurate advice and appropriate vaccination or passive immunisation before travelling to endemic areas.

Hand-washing is important to prevent transmission of HAV.

**Summary**

The primary care clinician has a key role in identifying cases of acute hepatitis and facilitating the clinical monitoring and management of individuals with infection. Specialist referral is advised if signs of acute liver failure develop or if the diagnosis is unclear. Following a possible exposure to viral hepatitis or a diagnosis of acute viral hepatitis, prevention measures and harm reduction strategies should be fully explored to reduce ongoing transmission.

**References**

CHAPTER 6 ASSESSMENT OF THE PATIENT WITH HIV DISEASE

2014 REVIEW

<table>
<thead>
<tr>
<th>Name</th>
<th>Position</th>
</tr>
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<tbody>
<tr>
<td>Catriona Ooi</td>
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2008 EDITION

<table>
<thead>
<tr>
<th>Name</th>
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<tbody>
<tr>
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</tr>
</tbody>
</table>
**Introduction**

Since the mid-1990s, the clinical manifestations of chronic human immunodeficiency virus (HIV) infection have changed dramatically among people with access to combination antiretroviral therapy. This chapter covers the classical signs and symptoms of unmodified HIV disease that can provide a basis for an initial clinical diagnosis. It also discusses the clinical issues seen in the large proportion of people with HIV infection who are now taking combination antiretroviral therapy.

Acquired immune deficiency syndrome was characterised in the early 1980s before HIV had been identified. The Centers for Disease Control (CDC) in the USA listed a group of secondary conditions suggesting immunodeficiency that had been identified in clusters. The original case definition of AIDS has been modified with time but remains a list of conditions, now rare in the Australian setting, seen predominantly in people who present late with untreated HIV infection and severe immunodeficiency (Table 6.1). AIDS remains notifiable in Australia, but its prognostic significance is less important in treated populations than other markers such as CD4 cell count and viral load (Chapter 10).

With long-term effective treatment, people with recently acquired infection can expect a near-normal life-span, free from complications related to immunodeficiency. Timely diagnosis of HIV infection is vital to avoid the poor outcomes associated with late presentation.

**When should HIV be considered in the differential diagnosis?**

The focus of this chapter is on specific clinical illnesses, laboratory abnormalities and aberrant responses to therapeutic interventions that may indicate HIV infection as a differential diagnosis to the astute clinician (see Table 6.2). Whenever HIV testing is recommended, there should be an awareness of the psychosocial impact of testing and an appropriate pre-test discussion should be undertaken (Chapter 9).

HIV infection may be considered in people who report established exposure risks (e.g. men who have sex with men) during general health assessments; however patients will not always disclose private behaviours. Additionally, heterosexual contact as the only risk behaviour identified in newly diagnosed people is no longer unusual. Except for rare occasions such as a legal order or in emergency settings, testing for HIV requires Informed consent.

HIV testing is a recommended component of antenatal screening and is routine for sexually transmissible infections (STIs). HIV antibody screening is performed with blood, tissue and organ donation, prior to military service and may be requested for some visas and work permits. Consideration should be given to HIV infection among other risks or immunosuppression prior to live viral vaccinations, at consideration of transplantation and when prescribing immuno-suppressant medications. The assessment of HIV risk and subsequent counselling and management of the patient are detailed in Chapters 3 and 9.

**Immune activation symptoms – primary infection**

The acute retroviral syndrome characteristic of primary HIV infection (described as seroconversion illness) includes features of immune activation, such as fever, night sweats, myalgia, arthralgia and lymphadenopathy (Chapter 4).

**Clinical latency**

The long phase of clinical latency that follows primary HIV infection conceals substantial virological and immunological activity. For most untreated people with HIV infection there is a gradual decrease in CD4

### KEY POINTS

- Clinical diagnosis of HIV infection is dependent upon consideration of HIV as a possible aetiology in particular clinical presentations.
- Chronic symptoms of immune activation (e.g. lymphadenopathy, night sweats, fever) may indicate HIV infection. Persistent oral or skin conditions may indicate mild, HIV-related immune deficiency.
- Laboratory markers such as thrombocytopenia, neutropenia and lymphopenia may suggest HIV infection.
- With combination antiretroviral therapy (ART), classical AIDS-defining illnesses are uncommon in Australia. These conditions are now most common in untreated patients with advanced HIV disease.
- Combination ART has altered the course of clinical HIV disease. Treated patients are now more likely to experience morbidity as a consequence of concomitant non-HIV-related chronic conditions.
- The importance of ART in reducing community risk of ongoing HIV transmission has driven recommendations for early diagnosis and treatment.
cell numbers over a period of 5–10 years, when clinical HIV disease becomes apparent. A small but significant proportion of people with HIV (elite controllers or long-term non progressors) still have low viral loads and near normal immune function despite HIV infection for over 20 years.

**Mild immunodeficiency**

Some infectious agents can become symptomatic relatively early in untreated HIV infection (Tables 6.1–6.3). Most of these are other chronic viral infections and the appearance of clinical disease in people with HIV infection is usually due to re-activation of latent virus rather than new infection.

**Shingles**

An episode of classical herpes zoster can often occur early in the course of chronic HIV infection, particularly after another illness such as a respiratory infection. It can be managed effectively using acyclovir, valaciclovir or famciclovir. Admission to hospital for intravenous acyclovir may be warranted for those with severe pain or multi-dermatomal or disseminated herpes zoster.

**Herpes simplex**

Oro-facial and ano-genital herpes simplex virus (HSV) recurrences may occur more frequently in people with HIV infection. These may be extensive and persistent. In people with advanced disease, the ulcers may coalesce to form large, extremely painful lesions, and when continuously present for more than a month constitute an AIDS diagnosis. The advent of effective treatment for both HIV and HSV means that severely symptomatic chronic herpes is now rare. Recurrent or persistent herpes may be a sign of HIV infection in undiagnosed patients and may be a trigger for risk assessment, further physical examination and testing. Genital HSV infection is a risk factor for both acquisition and transmission of HIV, and although episodic and suppressive antiviral therapy has been shown to reduce HIV viraemia, it does not translate into reduced HIV transmission. Nevertheless, HSV suppressive treatment should be considered for those with recurrent infection.

**Kaposi’s sarcoma**

This malignancy is associated with human herpesvirus type 8 (HHV-8) which appears to be sexually transmitted. In Australia, Kaposi’s sarcoma is highly suggestive of HIV infection.

Kaposi’s sarcoma most commonly manifests as purple, nodular lesions on the skin or oral mucosa (Figure 6.1) but can occur in visceral organs such as the lungs and the gastrointestinal system. Unpleasant or unsightly local tumours are amenable to local cryotherapy, intralesional chemotherapy or radiotherapy. For progressive disseminated disease, systemic chemotherapy is often beneficial but the mainstay of management involves restoration of immune function by controlling HIV replication through antiretroviral therapy.

**FIGURE 6.1 Kaposi’s sarcoma**

![Kaposi's sarcoma image](image)

**TABLE 6.1 AIDS indicator diseases**

<table>
<thead>
<tr>
<th>Disease</th>
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</thead>
<tbody>
<tr>
<td><strong>Candidiasis (oesophagus)</strong></td>
</tr>
<tr>
<td><strong>Cryptococcosis (invasive)</strong></td>
</tr>
<tr>
<td><strong>Cervical carcinoma (invasive)</strong></td>
</tr>
<tr>
<td><strong>Cryptosporidiosis with diarrhoea &gt; 1 month</strong></td>
</tr>
<tr>
<td><strong>Cytomegalovirus of retina, brain, spinal cord, gastrointestinal tract</strong></td>
</tr>
<tr>
<td><strong>Herpes simplex mucocutaneous ulcer &gt; 1 month</strong></td>
</tr>
<tr>
<td><strong>HIV-associated dementia, disabling cognitive ± motor dysfunction</strong></td>
</tr>
<tr>
<td><strong>HIV-associated wasting loss &gt;10% body weight plus diarrhoea, weakness and fever &gt; 30 days</strong></td>
</tr>
<tr>
<td><strong>Isosporiasis with diarrhoea &gt; 1 month</strong></td>
</tr>
<tr>
<td><strong>Kaposi’s sarcoma</strong></td>
</tr>
<tr>
<td><strong>Lymphoma, brain or non-Hodgkin’s (B-cell or immunoblastic)</strong></td>
</tr>
<tr>
<td><strong>Mycobacterium avium complex or kansasii (disseminated)</strong></td>
</tr>
<tr>
<td><strong>Mycobacterium tuberculosis disseminated or pulmonary</strong></td>
</tr>
<tr>
<td><strong>Pneumocystis jiroveci pneumonia</strong></td>
</tr>
<tr>
<td><strong>Pneumonia (recurrent bacterial)</strong></td>
</tr>
<tr>
<td><strong>Progressive multifocal leukoencephalopathy</strong></td>
</tr>
<tr>
<td><strong>Salmonella septicaemia (non-typhoidal, recurrent)</strong></td>
</tr>
<tr>
<td><strong>Toxoplasmosis (brain)</strong></td>
</tr>
</tbody>
</table>

* Requires HIV diagnosis.

**Ano-genital warts and squamous dysplasia**

Ano-genital warts are common in people with HIV infection and usually represent re-activation of a previous viral infection of the skin with the human papillomavirus (HPV). In patients without an HIV diagnosis, ano-genital warts, especially recurrent warts, indicate the need for HIV risk assessment, STI testing and further examination.
Anal or genital warts in the presence of HIV infection may be conservatively managed, particularly if the person is considering starting HIV antiretroviral therapy. Warts may regress spontaneously. Standard methods of treatment are usually adequate (Chapter 13). When warts are treated surgically, tissue should be sent for histopathological examination. Squamous dysplasia is often seen and requires close follow-up. Squamous carcinoma of the anal canal is more common in people with HIV and is related to infection with high risk HPV types;16 and 18.7 Cervical carcinoma is significantly more prevalent in women with HIV and is also related to HPV infection. Papanicolaou smear cytology is recommended annually, with management of abnormalities undertaken according to the usual approach.

**Molluscum contagiosum**

Molluscum contagiosum is caused by a poxvirus. Diagnosed clinically, these nodular lesions with a central punctum commonly occur on the face, neck or ano-genital area. Although common in HIV-negative people, persistent appearance of lesions in adults should prompt testing for HIV and STIs. In HIV infection, incidence and severity of lesions relate to the degree of immunosuppression. Differential diagnosis in the patient with HIV infection includes cutaneous cryptococcal infection and, in people from South East Asia, infection with *Penicillium marneffei*. Lesions commonly regress with immune recovery due to antiretroviral therapy or may be controlled with local therapy.

**Dermatoses**

Rashes are common in people with HIV infection at any level of immune function. Persistent, new or unusual skin conditions may be the first symptom of HIV infection. The clinician should be alert to the possibility of HIV infection and undertake a full risk assessment and physical examination if extensive, atypical or persistent rash is encountered.

The most common form of rash associated with HIV infection is seborrhoeic dermatitis (Figure 6.2). It occurs at the classical sites of scalp, ears, eyebrows, chest, axillae, groin and feet. Standard treatment with steroid creams or topical ketoconazole is often effective but recurrence is usual. This condition generally improves when antiretroviral treatment is initiated. Dermatophyte infections are also very common and can sometimes be difficult to differentiate from seborrhoea. These infections can be extensive, particularly on the feet, and secondary bacterial infection is common. Misdiagnosis of a dermatophyte infection leads to ineffective treatment with steroid creams that may in turn modify the clinical appearance of the condition.

**TABLE 6.2** Alarm bells suggestive of HIV infection

<table>
<thead>
<tr>
<th>Clinical conditions where HIV should be considered</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral candidiasis</td>
</tr>
<tr>
<td>Atypical mononucleosis syndrome (not EBV- or CMV-related)</td>
</tr>
<tr>
<td>Aseptic meningitis with severe systemic symptoms</td>
</tr>
<tr>
<td>Difficult to manage psoriasis or other dermatoses</td>
</tr>
<tr>
<td>Tuberculosis</td>
</tr>
<tr>
<td>Non-Hodgkin’s lymphoma</td>
</tr>
<tr>
<td>Cerebral space-occupying lesions</td>
</tr>
<tr>
<td>Persistent lymphadenopathy and symptoms of immune activation</td>
</tr>
<tr>
<td>Chronic vaginal candidiasis</td>
</tr>
<tr>
<td>Laboratory abnormalities where HIV should be considered</td>
</tr>
<tr>
<td>Thrombocytopenia, neutropenia, lymphopenia without cause</td>
</tr>
<tr>
<td>Anergy unexplained</td>
</tr>
<tr>
<td>Hypergammaglobulinemia new or unexplained</td>
</tr>
<tr>
<td>Therapeutic responses where HIV should be considered</td>
</tr>
<tr>
<td>Pneumonia unresponsive to standard therapy</td>
</tr>
<tr>
<td>Recurrent antibiotic-associated rash</td>
</tr>
</tbody>
</table>

EBV: Epstein-Barr virus
CMV: cytomegalovirus

**TABLE 6.3** Febrile syndromes in people with HIV infection

<table>
<thead>
<tr>
<th>Differential diagnosis of undifferentiated fever in the patient with HIV infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current CD4 cell count &lt; 200 cells/µl</td>
</tr>
<tr>
<td>• Disseminated <em>Mycobacterium avium</em> complex</td>
</tr>
<tr>
<td>• <em>Pneumocystis jiroveci</em> pneumonia</td>
</tr>
<tr>
<td>• Cryptococcal infection</td>
</tr>
<tr>
<td>• Cytomegalovirus infection</td>
</tr>
<tr>
<td>• Toxoplasmosis</td>
</tr>
<tr>
<td>• Less common infections, e.g. Histoplasma, Bartonella</td>
</tr>
<tr>
<td>Current CD4 cell count ≥ 200 cells/µl</td>
</tr>
<tr>
<td>• Bacterial infections, e.g. pneumonia, septicaemia</td>
</tr>
<tr>
<td>• Drug fever</td>
</tr>
<tr>
<td>• Tuberculosis</td>
</tr>
<tr>
<td>• Disseminated <em>Salmonella, Campylobacter</em> infection</td>
</tr>
<tr>
<td>• Fever associated with malignancy, e.g. lymphoma</td>
</tr>
</tbody>
</table>

Other puzzling rashes are seen in people with HIV infection. Early skin biopsy may be a useful guide when response to therapy is inadequate. Eosinophilic pustular folliculitis is one such pruritic papular condition that commonly occurs on the upper arms and chest for which phototherapy has induced response in many patients. Antiretroviral therapy often leads to resolution of dermatoses.

Psoriasis occurs in people with HIV infection with the typical erythematous scaly lesions occurring over elbows, hands and feet. The guttate form is also common. Pre-existing psoriasis can be exacerbated by HIV infection and newly diagnosed psoriasis has also been described. Immune recovery has been shown to improve these psoriatic lesions.
Oral hairy leukoplakia is commonly seen prior to serious HIV-related opportunistic infections. It manifests as distinctive white areas on the lateral margins of the tongue that cannot be rubbed off with gauze (unlike candidiasis). Its aetiology remains unclear, although one theory is that oral hairy leukoplakia is a manifestation of mucosal EBV infection. The condition is almost pathognomonic of HIV infection and should prompt HIV testing.

Oropharyngeal candidiasis becomes more common in HIV disease when immunosuppression occurs. Often it has the classical appearance of cheesy removable plaques; occasionally, it is more subtle with an area of slightly furry reddening, particularly on the palate. Candidiasis is much less common with effective antiretroviral therapy. Microscopy or culture will confirm the diagnosis. Treatment is only required when the condition is symptomatic, and topical amphotericin lozenges will often be effective for mild disease. If the disease is more severe and persistent, a course of oral fluconazole will usually control it for a period.

Aphthous mouth ulcers appear to be more common and more persistent in people with HIV than among the HIV-negative population. These ulcers may be quite large and are painful. When simple measures are ineffective, topical steroids appear to be beneficial in a proportion of patients.

Periodontal disease is common among people with HIV infection and occurs regardless of smoking. Regular preventive dental care is advised for all HIV-positive individuals. A particularly aggressive form of gum disease known as acute necrotising ulcerative gingivitis is seen in advanced HIV infection. Skilled dental care is required to prevent the loss of otherwise healthy teeth. Severe gingivitis may prompt HIV testing.

Hepatitis co-infection

As other HIV-related opportunistic infections are prevented or controlled, liver disease secondary to co-infection with hepatitis B (HBV) or hepatitis C (HCV) has become more prominent. Co-infection may complicate treatment of HIV. In the initial immune reconstitution phase, flares of HBV or HCV may be associated with increased hepatic transaminase levels, requiring close monitoring of liver function.8

The presence of HIV leads to more aggressive HCV disease and higher HCV viral load. Drug interactions between HIV antiretrovirals and agents used in HCV treatment require careful coordination of treatment regimens for both infections. Close monitoring of liver function tests and markers of HCV infection, as well as avoidance of other hepatotoxins such as alcohol are recommended (Chapters 10 and 11).

Some HIV antiretroviral medications also treat HBV. People with HIV-HBV co-infection should be treated with two agents active against HBV as part of their HIV treatment regimen. Once commenced, antiretroviral treatment should be continued without interruption in these patients due to risk of hepatitis relapse.

Patients with chronic hepatitis, whether due to HBV, HCV or other causes, such as non-alcoholic steatohepatitis, are at risk of developing cirrhosis, and ultimately, hepatocellular carcinoma.
abdominal symptoms. Its symptoms merge with those of advanced HIV itself and a high index of suspicion is required. MAC is an important differential diagnosis of non-specific fever in people with HIV infection (Table 6.3). The diagnosis of MAC is confirmed by culture of a relevant sample or blood collected in special media. However, the organism is slow to grow, so treatment with a combination of anti-mycobacterial drugs is often commenced presumptively. In those with epidemiological risk factors, tuberculosis should be considered as a differential diagnosis and presumptively treated until tuberculosis is excluded.

Upon treatment, significant clinical improvement is usually seen and maintenance therapy is continued until marked and sustained immune recovery is achieved with antiretroviral treatment. Effective prophylactic regimens for MAC are available. Azithromycin given as a single dose of 1200 mg weekly is most widely used and is usually commenced when the CD4 cell count is consistently below 50 cells/µL.

**Diarrhoeal diseases**

Diarrhoea is a common problem in people with HIV infection.

Among patients with known HIV infection, diarrhoea is often related to the adverse effects of antiretroviral medication, particularly some protease inhibitors. When advanced immunodeficiency is present (CD4 cell count below 100 cells/µL), opportunistic infections due to Cryptosporidium and Microsporidium should be considered. Stool examination for standard and sexually acquired pathogens is recommended if no obvious cause for persistent diarrhoea is found in a person with HIV infection. It is also important to request testing specifically for parasites, such as Microsporidium species, as this requires special processing of the specimen. Colonoscopy and mucosal biopsy may reveal CMV colitis in people with very severe immunosuppression.

Advanced HIV disease is associated with diarrhoea and, if no specific cause is found after a full diagnostic assessment, anti-diarrhoeal agents such as loperamide may be effective. The prolonged use of quite high doses is not uncommon. Bulking agents such as psyllium husk may also be useful.

**Immune reconstitution inflammatory syndrome (IRIS)**

In untreated people with advanced HIV infection (CD4 cell count usually below 50-100 cells/µL), marked immunodeficiency inhibits the inflammatory response that would normally occur to a variety of infectious agents such as CMV, HCV and MAC. When treatment reduces HIV viral load, there is rapid restoration of the ability to mount inflammatory reactions. Consequently, infectious agents that remained asymptomatic during extreme immunodeficiency are met with a marked inflammatory response, and clinical disease becomes apparent where few signs or symptoms were evident previously (Figure 6.3). Alternatively, conditions already evident such as Kaposi’s sarcoma, genital warts or dermatoses may temporarily worsen. This phenomenon has been named immune reconstitution inflammatory syndrome (IRIS), and was first described in Australia. An immune reconstitution illness is usually transient because the inflammatory effect is ultimately successful at combating the infectious agent. However, immune reconstitution illnesses can be clinically significant while present. In the case of CMV retinitis, vision can be permanently impaired by an episode of intense inflammation during immune reconstitution.

Where possible, in the severely immunocompromised, appropriate screening should be undertaken prior to commencing treatment. When a patient presents with new symptoms soon after starting antiretroviral therapy, immune reconstitution should be considered as a possible cause and appropriate referral and investigation is advised.

**Figure 6.3 Immune reconstitution and Mycobacterium avium complex (MAC)**

**Non-Hodgkin’s lymphoma**

People with HIV infection have a 250- to 650-fold increased risk of AIDS-related lymphoma over the background population, with lymphoma occurring most frequently in people with CD4 cell counts below 100 cells/µL. Eighty-five percent of all AIDS-related lymphomas are systemic non-Hodgkin’s lymphoma, 15% are primary central nervous system lymphoma, while primary effusion lymphomas occur uncommonly. Almost all AIDS-related lymphomas are high-grade diffuse large B-cell (immunoblastic variant) or Burkitt’s-like lymphomas. EBV has a clear pathogenetic role in primary central nervous system lymphoma, a probable role in systemic non-Hodgkin’s lymphoma, and also may be involved in primary effusion
lymphoma where HHV-8 is also implicated in disease pathogenesis. Isolated enlarged lymph nodes, systemic febrile illnesses and focal neurological abnormalities are among the common presentations. Referral to specialists in oncology and HIV-related malignancies is recommended. Chemotherapy, radiotherapy and combination antiretroviral treatment provide the usual basis of therapy.

**Neurological conditions**

HIV can directly affect neurocognitive functioning, and the term HIV-associated neurocognitive disorder (HAND) encompasses a spectrum from mild impairment to dementia. Features include problems with memory, executive function and psychomotor slowing. Although severe forms of this disorder are much less common in the era of effective antiretroviral treatment, the overall prevalence of HAND, including asymptomatic neurocognitive impairment, has not reduced. Increased risk of HAND is associated with low nadir CD4 cell count and HCV infection. Diagnosis requires exclusion of comorbidities including central nervous system (CNS) infections, malignancy and cerebrovascular disease. CNS penetration of individual antiretroviral drugs should be considered for all patients with HIV. At this time there is limited evidence that optimising the regimen for maximum CNS penetration is associated with better neurocognitive functioning.10 Space-occupying lesions of the brain also are relatively common in untreated people with advanced HIV infection. The most likely diagnoses are primary lymphoma of the brain and abscess resulting from reactivation of toxoplasmosis. Early diagnosis is important as toxoplasma abscesses respond to appropriate antibiotic therapy.

Other neurological conditions that were common in the days before combination antiretroviral therapy are cryptococcal meningitis and progressive multifocal leukoencephalopathy. Referral to an infectious diseases physician is recommended when a neurological condition is suspected in a patient with HIV infection.

**Non-HIV-defining cancers**

As the incidence of AIDS-defining cancers such as non-Hodgkin's lymphoma and Kaposi's sarcoma among people with HIV infection has fallen since the introduction of effective antiretroviral treatment, rates of other cancers not traditionally associated with HIV have increased.11 The increased rate of lung cancer among people with HIV infection is at least partly attributable to smoking rates. However, the similarity in pattern of increased cancer risk between people with HIV and immunosuppressed transplant recipients is evidence that immune deficiency drives an increased risk of malignancy in both groups.12

**Body composition changes**

Weight loss and preferential loss of lean body tissue is characteristic of progressive HIV infection and was common in people with AIDS in the 1980s and early 1990s. Most bodily changes in people with HIV infection now appear to be related to treatment. Lipodystrophy or loss of facial and peripheral fat can be striking in people taking antiretroviral therapy, creating a distinctive and easily identifiable appearance (Figure 6.4). Patients may complain of varicose veins when their healthy leg veins become more obvious as the surrounding subcutaneous tissue is lost. This syndrome is typically associated with prolonged exposure to some nucleoside analogue reverse transcriptase inhibitor (NRTI) drugs.13 Newer agents are less likely to result in changes to fat distribution. Facial lipodystrophy due to antiretroviral therapy can be effectively treated by a cosmetic surgeon with dermal injections of poly-L-lactic acid.14 A proportion of people with HIV infection on therapy also develop accumulation of fat in the abdomen and sometimes the buffalo hump over the lower neck posteriorly. Protease inhibitors are associated with marked dyslipidaemia in a high proportion of patients and also may be involved in this fat accumulation.

**Psychosocial issues**

In cases where clinical signs and symptoms lead to a HIV diagnosis, consideration should be given to the management of psychosocial concerns as well as the clinical manifestations of the infection. Post-test discussion and psychosocial follow-up are fundamental following a positive HIV result and issues for assessment and discussion may include relationships, family, sex, work and disclosure (Chapter 9). Psychosocial issues may have a profound effect on ability to adhere to antiretroviral therapy, essential for long-term treatment success. People with HIV infection are living longer and HIV-associated disease is now comparatively rare in Australia. Despite simpler, more tolerable treatment, long-term adherence to therapies, treatment toxicities and prevention and management of comorbid diseases of ageing still remain challenges (Chapter 10).

**Conclusion**

Although the rate of HIV infection in Australia is relatively low, the primary care clinician may give consideration to HIV infection in relation to a range of conditions, particularly when present in young and otherwise healthy individuals. In the age of combination antiretroviral therapy, early diagnosis of HIV infection leads to improved health and extended life-span in the patient.
All people with HIV infection are advised to regularly consult their general practitioner, who are ideally placed to detect adverse developments at an early stage and to facilitate optimal therapy. Chapter 10 addresses management of the patient with HIV infection, particularly in regard to antiretroviral therapy, psychosocial management, and support and referral.

**FIGURE 6.4** Body composition changes

**References**


CHAPTER 7 ASSESSMENT OF THE PATIENT WITH CHRONIC VIRAL HEPATITIS

2014 REVIEW

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Introduction

Acute infection with hepatitis B virus (HBV) or hepatitis C virus (HCV) can result in chronic hepatitis if the infection persists for more than 6 months. The rate of spontaneous clearance varies according to the virus, the age at onset of infection and other factors.

Spontaneous clearance of HCV generally occurs during the first 6 months of infection in approximately a quarter of people, with the remainder developing chronic HCV infection. Although hepatic fibrosis occurs in most people, the condition is often asymptomatic for an extended period of time. Symptoms arise with the development of complications of advanced liver disease, but non-specific symptoms and impaired quality of life are common among those with earlier stages of liver disease. Cirrhosis occurs in an estimated 20–30% of people who develop chronic HCV infection, 15–40 years after the original infection. Among those who develop cirrhosis, liver failure occurs in 20–30% and hepatocellular carcinoma (HCC) develops in 10–20% over 10 years. Estimates of disease progression in hepatitis C are outlined in Figure 7.1.

The natural history of HBV infection is primarily determined by the age of the person at the onset of infection. When acquired at birth or during early childhood, the risk of developing chronic infection is high, with only 2% of infants spontaneously clearing the virus within 3 years of infection and 15% clearing the virus within 20 years. Among people with perinatally acquired HBV, 40–50% of males and 15% of females die from the liver-related causes.

In the case of adult-acquired HBV infection, however, the situation is reversed, with spontaneous clearance being the rule. Acute liver failure occurs rarely, and only 3–5% of adults with acute infection progress to chronic HBV infection. In many cases, chronic HBV infection does not result in symptoms or long-term problems, although 20–30% of people will progress to cirrhosis.

These differences in outcome between perinatal and adult-acquired infection are outlined in Figure 7.2. Of those with compensated cirrhosis, 20–30% will develop liver failure (decompensated cirrhosis) and 10–20% will develop HCC over the next 10 years. Survival rates are high among those with compensated cirrhosis but much lower among those with liver failure (85% versus 25% at 5 years).

Symptoms and signs of chronic viral hepatitis

Chronic viral hepatitis is frequently hidden due to the asymptomatic nature of liver disease in a large proportion of people and the slowness or absence of progression to advanced liver disease. The absence of symptoms and abnormal clinical signs, therefore, does not exclude significant liver disease. However, early diagnosis and treatment may improve prognosis and, where appropriate, patients should be offered treatment options.

Although there is a great deal of overlap, symptoms and signs of chronic viral hepatitis can be divided into those associated with:

- early or slowly progressive liver disease
- progressive liver disease
- advanced liver disease complications
- extra-hepatic manifestations.

In this classification, early or slowly progressive liver disease includes people with chronic hepatitis C who progress slowly and may have fibrosis.

Progressive liver disease covers people with ongoing hepatic inflammation and fibrosis development, often resulting in cirrhosis or, in the case of chronic HBV infection, those who have clinical evidence of progressive disease such as hepatitis flares but retain adequate liver function (e.g. compensated cirrhosis).

Advanced liver disease complications includes people who have developed clinical liver failure (decompensated cirrhosis, e.g. hepatic encephalopathy and failure of}

**KEY POINTS**

- The presence of significant liver disease in patients may not be apparent from symptoms or clinical examination. Conversely, multiple symptoms in chronic hepatitis infection do not necessarily indicate significant liver disease.
- There is a poor correlation between biochemical and virological markers of chronic viral hepatitis and symptoms and signs.
- Progressive liver disease in chronic hepatitis B may involve marked acceleration and hepatic flares, whereas disease progression is gradual and generally asymptomatic in chronic hepatitis C.
- The presence and severity of liver fibrosis in chronic hepatitis B and C influences therapeutic decisions and is also a key prognostic factor.
synthetic function with increases in International Normalised Ratio (INR), portal hypertension (e.g. ascites, oesophageal varices) and HCC.

Extra-hepatic manifestations refers to a broad range of clinical conditions associated with either chronic hepatitis B or chronic hepatitis C.

Clearly these groups are not mutually exclusive. For example, it is possible to have progressive liver disease and extra-hepatic manifestations of chronic hepatitis. In addition, there may be little clinical distinction between early or slowly progressive disease and progressive disease. A long asymptomatic phase followed by signs associated with cirrhosis or decompensation is not uncommon.

**Early or slowly progressive liver disease**

Symptoms of chronic viral hepatitis associated with early and slowly progressive liver disease are generally non-specific. Individuals frequently complain of tiredness, anorexia, nausea, intolerance to fatty foods and abdominal discomfort, particularly in the right upper quadrant region. Others report general feelings of being unwell but are unable to elaborate further. Fevers and night sweats can also occur.

A number of recent studies have shown that people with chronic HCV infection score poorly on many quality-of-life parameters, including a range of physical and psychological measures of wellbeing. Again, these impairments are relatively non-specific, and include reductions in general health perception, mental health, physical function, social function and vitality. These measures may also be impaired in many people with chronic hepatitis B. Successful clearance of HCV through antiviral therapy has been shown to improve quality-of-life scores.

The major feature of the symptomatology of early or slowly progressive liver disease in chronic viral hepatitis is its highly variable nature. For many people, this stage of liver disease, which may be the only stage they experience, is completely asymptomatic. On the other hand, many people have considerable symptoms despite the presence of mild liver disease or the absence of biochemical evidence of liver inflammation (normal alanine aminotransferase [ALT] and aspartate aminotransferase [AST] levels). In fact, in chronic hepatitis C there is little correlation between the ALT level and presence of symptoms. Furthermore, the stage of liver disease (prior to liver failure) and the viral load in chronic hepatitis C have a poor association with the extent of symptoms.

People with early or slowly progressive liver disease generally have few clinical signs associated with their chronic viral hepatitis. The most common clinical examination reveals either no abnormal findings or mild hepatomegaly. Presence of peripheral stigmata of chronic liver disease, such as multiple spider naevi and palmar erythema, would generally indicate cirrhosis.

**Progressive liver disease**

Although the majority of people with chronic viral hepatitis will not develop advanced liver disease complications, many will eventually have progressive liver disease. The symptoms covered above may also be present in progressive liver disease.

In chronic hepatitis B, particularly in the case of perinatal or early childhood infection, a prolonged asymptomatic period (immune tolerance phase) is followed by a more symptomatic period (reactivation-clearance phase) in which flares of clinical hepatitis may occur as the body's immune system attempts to clear infection. These flares are generally milder than an acute hepatitis B clinical presentation. However, they often consist of similar symptoms and signs. These include lethargy, nausea, anorexia, food intolerance, abdominal discomfort and jaundice. These clinical flares in chronic hepatitis B are closely associated with biochemical evidence of increased hepatic inflammation. Marked elevations of ALT and AST together with increased serum bilirubin levels are often seen. A small proportion of people each year in this reactivation-clearance phase will seroconvert, initially from the hepatitis B e-antigen positive (HBeAg+) immune clearance phase to...
HBeAg-negative immune control phase (generally with development of anti-HBe) (Figure 7.3) In some cases there is subsequent loss of hepatitis B surface antigen (HBsAg). People with frequent flares who have not seroconverted may experience faster disease progression and are at high risk of cirrhosis and HCC. In addition, people who have entered the clearance phase and seroconverted to anti-HBe can have reactivation of disease at a later date with the emergence of the so called pre-core mutants, which are immune escape HBV mutations. This stage of hepatitis B is characterised by negative HBeAg but abnormal liver function tests (LFTs) and elevated HBV DNA. This form of chronic hepatitis B is also associated with more aggressive disease. All patients with chronic hepatitis B (HBsAg positive), particularly with abnormal LFTs or elevated HBV DNA (> 10^4 IU/mL) should be referred for specialist review and consideration of therapeutic intervention.

In chronic hepatitis C, clinical hepatitis flares are rare and people often progress to cirrhosis without development of significant symptoms. Before

**FIGURE 7.3** Phases of chronic hepatitis B infection (adapted from: Decision-making in HBV. ASHM. 2013)

**FIGURE 7.4** Spider naevi in chronic hepatitis

**FIGURE 7.5** Decompensated cirrhosis secondary to hepatitis C
either chronic hepatitis B or hepatitis C.

Factors associated with progressive liver disease in chronic hepatitis C are listed in Table 7.1. Peripheral stigmata of chronic liver disease, such as spider naevi (Figure 7.4), nail changes and palmar erythema, may develop if there is progression to cirrhosis. However, a completely normal clinical examination may also be found in the presence of cirrhosis related to either chronic hepatitis B or hepatitis C.

Advanced liver disease complications

Advanced liver disease complications of both chronic hepatitis B and chronic hepatitis C consist of liver failure (decompensated cirrhosis) (Figure 7.5), often in association with signs of portal hypertension such as refractory ascites and variceal bleeding and HCC. In chronic hepatitis C, HCC is rarely seen without underlying severe fibrosis or cirrhosis. In contrast, as HBV itself is oncogenic, HCC can develop in people with chronic hepatitis B without significant liver fibrosis.

Symptoms and signs of liver failure are the same for chronic HBV and HCV, and are similar to symptoms and signs associated with other causes of decompensated cirrhosis. Consistent with the underlying decrease in synthetic function ( hypoalbuminaemia and coagulopathy), early symptoms of liver failure may include ankle swelling, mild abdominal swelling and easy bruising. Increasing lethargy is generally also a feature. Clinical examination may reveal some peripheral stigmata of chronic liver disease, as well as some evidence of either peripheral oedema or ascites. Later signs may include jaundice, which indicates a poor prognosis in the presence of liver failure, loss of hair and gynaecomastia. Clinical evidence of portal hypertension may include abdominal venous distension, splenomegaly and ascites. Patients who have ascites may develop spontaneous bacterial peritonitis (SBP). Patients with unexplained fever or encephalopathy should raise the suspicion of SBP and they should be referred for diagnostic paracentesis. In addition, the presence of peripheral neuropathy and cerebellar ataxia may suggest alcohol as a contributing cause of liver disease.

A history of haematemesis in a person with other evidence of advanced liver disease suggests the presence of oesophageal varices related to underlying portal hypertension. Hepatic encephalopathy also may be present in advanced liver disease and may be subclinical in early stages. A history of reversal of diurnal sleep patterns, forgetfulness or inappropriate behaviour may signal the onset of early hepatic encephalopathy. Presence of either hepatic encephalopathy or oesophageal varices indicates a poor prognosis.

Table 7.2 summarises the different signs and symptoms related to stages of liver disease in chronic hepatitis B and C.

**Extra-hepatic manifestations**

Extra-hepatic manifestations, although uncommon, represent clinically important aspects of hepatitis B and C. Specific treatment can be directed towards these conditions.

Dermatological presentations include porphyria cutanea tarda (PCT), lichen planus and vasculitic rashes associated with cryoglobulinaemia. These presentations should alert the clinician to the possibility of chronic viral hepatitis. In patients with PCT, which is typically associated with chronic hepatitis C, lesions which are exacerbated by exposure to the sun occur on the dorsum of the hands and forearms. Ferritin levels are often mildly elevated in PCT and these patients respond very well to venesection.

Rheumatological manifestations include arthropathy, Sjogren’s syndrome and polyarteritis nodosa. A high serum globulin level, often associated with positive antinuclear antibody (ANA) and rheumatoid factor, may indicate the presence of cryoglobulinemia, which may be associated with systemic complications such as glomerulonephritis and vasculitis.

### TABLE 7.1 Factors associated with progression to advanced liver disease in chronic hepatitis C

<table>
<thead>
<tr>
<th>Factor</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at acquisition of infection (&gt; 40 years)</td>
<td></td>
</tr>
<tr>
<td>Heavy alcohol intake (&gt; 40 grams/day)</td>
<td></td>
</tr>
<tr>
<td>Male gender</td>
<td></td>
</tr>
<tr>
<td>Longer duration of infection / older age</td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
</tr>
<tr>
<td>Co-infection with HIV and/or chronic hepatitis B</td>
<td></td>
</tr>
<tr>
<td>Regular marijuana use</td>
<td></td>
</tr>
</tbody>
</table>

Note: There is no evidence for an association between HCV viral load and risk of disease progression.

Haematological abnormalities include thrombocytopenia and leukopenia. Thrombocytopenia is most commonly related to hypersplenism and portal hypertension, but may also be due to drug therapy, or be immune mediated. Neurological complications may be related to cryoglobulinemia and present with mononeuritis of cranial or peripheral nerves. Thyroid disease may be subclinical. A variety of thyroid diseases have been described in association with chronic viral hepatitis and interferon-based therapy. Patients who test positive for ANA are more prone to developing thyroid disorders, particularly when treated with interferon. These thyroid disorders, however, are generally reversible.
Assessment of the presence and stage of disease

An assessment of the presence and stage of disease often requires a step-wise investigation of serological, virological, biochemical, ultrasonographic and histological markers of viral hepatitis and liver disease. More recently, non-invasive assessment of fibrosis has been introduced to improve the feasibility and acceptability of liver disease staging. In addition, clinical examination may provide some indication of the stage of disease, particularly when advanced liver disease is present. The results of these investigations may determine prioritisation for antiviral treatment, which is funded under Section 100 of the Pharmaceutical Benefits Scheme. Refer to the National HCV Testing Policy 2012 (first review May 2013) that can be found at: http://testingportal.ashm.org.au/hcv and the National HBV Testing Policy 2012 (first review February 2014) found at: http://testingportal.ashm.org.au/hbv.

Serological markers

In hepatitis C, a positive HCV antibody result indicates prior or current infection but does not distinguish between these two conditions. Detection of HCV RNA is required to make the diagnosis of current HCV infection.

In hepatitis B, serological testing provides useful information on the presence of active infection. To determine hepatitis B status, all three tests (HBsAg, anti-HBc and anti-HBs) should be ordered. All three tests are Medicare rebatable simultaneously. HBsAg is a marker of current infection. It may disappear following acute infection or persist in a person who has HBV infection. Anti-HBs appears following the disappearance of HBsAg, and is a marker of both naturally acquired and vaccine-induced immunity. The presence of anti-hepatitis B core (Hbc) IgM generally indicates recent infection since it usually appears following acute infection and disappears within a year followed by the development of anti-Hbc IgG. Occasionally, anti-Hbc IgM may be positive during hepatic flares in people with chronic hepatitis B. Anti-Hbc IgG can persist indefinitely following an infection, and signifies exposure to HBV.

Most people exposed to HBV as adolescents or adults clear the infection and will test anti-Hbc positive (anti-Hbc+) and HBsAg negative. HBeAg is a marker of viral replication and hence infectivity, although HBV DNA is more accurate measure of infectivity than HBeAg. Anti-HBe generally develops as HBeAg disappears, signalling resolution of acute infection or cessation of replication. More complete clearance of HBV infection is indicated by development of anti-HBs. Refer to Table 7.4 for a summary of serological and virological markers of acute and chronic hepatitis.

Virological tests

HCV RNA testing by polymerase chain reaction (PCR) indicates the presence (qualitative) or level (quantitative or viral load) of HCV RNA. A qualitative HCV RNA test generally distinguishes between a person who has chronic hepatitis C and a person

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**TABLE 7.2 Symptoms and signs of chronic viral hepatitis by stage of disease**

<table>
<thead>
<tr>
<th></th>
<th><strong>Chronic hepatitis B</strong></th>
<th><strong>Chronic hepatitis C</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Early and slowly progressive liver disease</strong></td>
<td>Generally none</td>
<td>Often none Hepatomegaly</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Often none Lethargy Anorexia Nausea Abdominal discomfort Intolerance to alcohol and fatty foods</td>
</tr>
<tr>
<td><strong>Progressive liver disease</strong></td>
<td>Often episodic Hepatic flares</td>
<td>Hepatomegaly</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Often none Lethargy Anorexia Nausea Abdominal discomfort Intolerance to alcohol and fatty foods</td>
</tr>
<tr>
<td><strong>Advanced liver disease</strong></td>
<td>Increasing lethargy Fluid retention Bruising Prolonged bleeding</td>
<td>Peripheral stigmata of CLD* Gynaecomastia Ascites/ oedema Splenomegaly Distended abdominal veins Bruising Hepatic encephalopathy Jaundice (poor prognostic sign)</td>
</tr>
<tr>
<td><strong>CLD – chronic liver disease</strong></td>
<td>Increasing lethargy Fluid retention Bruising Prolonged bleeding</td>
<td>Peripheral stigmata of CLD* Gynaecomastia Ascites/oedema Splenomegaly Distended abdominal veins Bruising Hepatic encephalopathy Jaundice (poor prognostic sign)</td>
</tr>
</tbody>
</table>
who has cleared HCV either spontaneously or during treatment. People who have cleared HCV will continue to test positive for the anti-HCV antibody but will be negative for HCV RNA. Thus, if symptoms and signs of active infection are present in a person with normal serum ALT levels who is anti-HCV antibody positive and HCV RNA negative, a cause other than hepatitis C should be sought. On the other hand, the vast majority of people with elevated serum ALT levels, who test positive for HCV antibody, particularly in the presence of a risk factor for infection, have active infection (viraemia). In these people, HCV RNA will be positive. A quantitative HCV RNA or viral load test does not provide information on the stage of disease as there is little or no correlation between the HCV viral load and the extent of hepatic fibrosis or risk of disease progression (in distinct contrast to the situation with HIV). However, HCV viral load has had prognostic value with regard to response to interferon-based antiviral therapy, and the HCV genotype is even more predictive of response. HCV viral genotyping has been essential in determining the likely response and optimal duration of antiviral treatment. However, with new HCV treatments, in particular highly effective interferon-free direct-acting antiviral (DAAs) regimens, viral load has limited predictive value.

HBV DNA is a marker of active replication and can be assessed quantitatively to monitor response to antiviral treatment. The vast majority of people who are HBeAg+ will be positive on HBV DNA testing. In the setting of hepatic inflammation (elevated ALT), the HBV DNA level correlates with risk of progression to cirrhosis and hepatocellular carcinoma. Active viral replication associated with raised ALT, particularly in the presence of hepatic fibrosis, is an indication for treatment.

**Liver function profile**

The serum ALT level may give an indication of hepatic inflammation although levels may be normal despite progressive liver disease. Nevertheless, people with either chronic hepatitis B or chronic hepatitis C who have consistently normal ALT levels are at low risk of progression to cirrhosis. Although people with abnormal ALT levels are at increased risk of progressive liver disease, the level of ALT in chronic hepatitis C is a relatively poor predictor of disease stage or disease progression. In contrast, in chronic hepatitis B recurrently high ALT levels generally indicate more active underlying disease and risk of disease progression. An inverted AST/ALT ratio (higher AST than ALT) may indicate underlying cirrhosis in either chronic HBV or HCV infection.

Albumin level (along with the prothrombin time) gives an indication of the synthetic function of the liver. Hypoalbuminaemia and increased INR indicate decompensated cirrhosis. Evidence from a cohort of people with chronic hepatitis C demonstrated that one of the strongest prognostic measures was albumin level, with higher rates of progression to advanced liver disease complications among people with levels below 35 g/L, particularly if less than 30 g/L.

**Liver imaging**

Abdominal ultrasound is used to assess the liver and biliary tree, as other causes of right upper quadrant pain, such as gallstones, often need to be excluded. In addition, abdominal ultrasound helps to screen for HCC (particularly if cirrhosis is present) and to assess for small amounts of ascites where doubt exists. However, a normal ultrasound does not exclude cirrhosis and this investigation is probably unnecessary in a person with no clinical evidence of chronic liver disease.

**Table 7.3 Extrahepatic manifestations of chronic hepatitis**

<table>
<thead>
<tr>
<th>Haematological</th>
<th>Cryoglobulinaemia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>Renal</td>
<td>Granulocytopenia</td>
</tr>
<tr>
<td>Rheumatological</td>
<td>Polyarteritis nodosa</td>
</tr>
<tr>
<td>Dermatological</td>
<td>Lichen planus</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Porphyria cutanea tarda</td>
</tr>
<tr>
<td>Neurological</td>
<td>Mononeuritis</td>
</tr>
<tr>
<td>Peripheral neuropathy</td>
<td></td>
</tr>
</tbody>
</table>

**Non-invasive measure of fibrosis in chronic hepatitis**

The presence and severity of liver fibrosis in chronic hepatitis B and C influences therapeutic decisions and is also a prognostic factor. A number of non-invasive methods are currently available to evaluate liver fibrosis, as an alternative to liver biopsy. Serum biomarkers are useful in assessing the severity of fibrosis. The AST/ALT ratio and the AST-to-platelet ratio index (APRI) may be helpful. A number of commercial panels of fibrosis markers (hepascore®, Fibrotest®) have also been validated in evaluating liver fibrosis.

Hepatic elastography (Fibroscan®) uses low frequency ultrasound waves to measure liver stiffness, and subsequently degree of fibrosis. It is a simple, fast and accurate technique. A vibrating piston generates a shear wave which passes through the skin and liver. The ultrasound detects propagation of the shear wave through the liver and velocity is measured. The shear wave velocity is directly related to liver stiffness, with higher velocity corresponding...
Liver biopsy

Percutaneous liver biopsy for HCV was previously performed in the majority of patients undergoing assessment for antiviral therapy and was required under Section 100 guidelines. However, a liver biopsy is no longer mandatory and is generally being replaced by non-invasive methods of disease staging for pre-treatment assessment, and ongoing disease monitoring. Liver biopsy staging of disease can still be an important tool in determining prognosis and guiding therapeutic decisions in selected patients.

In patients with chronic hepatitis B, liver biopsy is also no longer mandatory but remains a valuable investigation in selected patients as fibrosis progression is far less predictable.

Patients are often puzzled because of the lack of correlation between their symptoms, their blood tests and the serious consequences that can be associated with viral hepatitis. It is important to stress that the absence of symptoms, signs and abnormal ALT levels does not exclude significant liver damage.

A summary of the investigations used in chronic viral hepatitis is provided in Table 7.4.

Clinical examination

Physical examination of patients with suspected or confirmed viral hepatitis consists of general inspection as well as attention to specific signs of chronic liver disease and associated systemic disorders. Examination should include:

- General appearance and mental health assessment
- Peripheral examination of the hands (for palmar erythema, Dupuytren’s contracture, leukonychia, skin lesions)
- Examination of the arms or trunk (for abnormal bruising, spider naevi, loss of hair and gynaecomastia)
- Inspection for jaundice, anaemia and parotid enlargement
- Inspection of the abdomen (for evidence of collateral circulation, herniae, hepatomegaly, splenomegaly and ascites)
- Signs of fever or encephalopathy
- Peripheral neuropathy and cerebellar ataxia (which suggest alcohol as a cause of liver disease)
- A history of reversal of diurnal sleep patterns, forgetfulness or inappropriate behaviour, which may signal the onset of early hepatic encephalopathy.
TABLE 7.4 Investigations in chronic hepatitis

<table>
<thead>
<tr>
<th>INVESTIGATION</th>
<th>REASON</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCV antibody (anti-HCV)</td>
<td>Exposure to HCV</td>
</tr>
<tr>
<td>Qualitative HCV RNA</td>
<td>Detects presence of HCV</td>
</tr>
<tr>
<td>Quantitative HCV RNA viral load</td>
<td>Provides quantitative HCV viral load measurement</td>
</tr>
<tr>
<td>HCV genotype</td>
<td>Predicts response and optimal duration of treatment</td>
</tr>
<tr>
<td>HBsAg</td>
<td>Indication of natural hepatitis B infection. Occurs with acute infection and may disappear or persist indefinitely. Marker of ongoing infection</td>
</tr>
<tr>
<td>Anti-HBs</td>
<td>Indication of immunity to hepatitis B (from natural infection or vaccination)</td>
</tr>
<tr>
<td>Anti-HBc</td>
<td>Previous exposure to hepatitis B virus</td>
</tr>
<tr>
<td>Anti-HBc IgM</td>
<td>Marker of recent exposure to hepatitis B virus. Does not persist more than a year following acute infection IgM may also be positive later in disease</td>
</tr>
<tr>
<td>HBeAg</td>
<td>Indication of hepatitis B viral replication and high infectivity. Useful serological marker in the investigation of a person who is found to be HBsAg+</td>
</tr>
<tr>
<td>Anti-HBe</td>
<td>Indication of hepatitis B viral clearance and occurs following loss of HBeAg. May also occur in the presence of pre-core mutant disease (immune escape) in association with abnormal ALT and elevated HBV DNA</td>
</tr>
<tr>
<td>HBV DNA</td>
<td>Indication of viral replication. Quantitative level may help to predict response to antiviral treatment (higher levels associated with poorer outcome) and monitor response to treatment. Useful serological marker in the investigation of a person who is found to be HBsAg+</td>
</tr>
<tr>
<td>ALT</td>
<td>Detection of abnormal ALT suggests antiviral treatment should be considered</td>
</tr>
<tr>
<td>Albumin</td>
<td>Indication of synthetic liver function, i.e. low albumin indicates liver failure</td>
</tr>
<tr>
<td>FBC</td>
<td>Platelet counts may be low due to the progression of fibrosis or portal hypertension</td>
</tr>
<tr>
<td>INR</td>
<td>Indication of synthetic function</td>
</tr>
<tr>
<td>HAV, HBV, HDV and HIV serology</td>
<td>To determine need for vaccination to prevent super-infection with hepatitis A virus and hepatitis B virus. Presence of HIV and hepatitis D virus alters prognosis</td>
</tr>
<tr>
<td>Thyroid function tests</td>
<td>To exclude associated thyroid disorder and as a baseline investigation prior to interferon treatment (which can cause toxicity)</td>
</tr>
<tr>
<td>Ferritin</td>
<td>To exclude haemochromatosis (may reflect severity of liver disease)</td>
</tr>
<tr>
<td>U&amp;E and creatinine</td>
<td>Baseline prior to treatment. To exclude possible renal involvement, i.e. glomerulonephritis</td>
</tr>
<tr>
<td>Alpha-fetoprotein</td>
<td>Baseline investigation for hepatocellular carcinoma in conjunction with abdominal ultrasound</td>
</tr>
<tr>
<td>Caeruloplasmin</td>
<td>To exclude Wilson’s disease</td>
</tr>
<tr>
<td>Alpha-1-antitrypsin</td>
<td>To exclude alpha-1-antitrypsin deficiency</td>
</tr>
<tr>
<td>ANA, SMA, LKM</td>
<td>To exclude autoimmune disease</td>
</tr>
<tr>
<td>Abdominal ultrasound</td>
<td>To assess liver and biliary tree and to screen for hepatocellular carcinoma. Can also be useful to detect small amounts of ascites</td>
</tr>
<tr>
<td>Non-invasive tests for liver fibrosis</td>
<td>Serum biomarkers e.g. AST/ALT ratio, AST-to-platelet ratio index (APRI); commercial panels of fibrosis markers (Hepascore®, Fibrotest®); Transient elastography (Fibroscan®)</td>
</tr>
<tr>
<td>Liver biopsy</td>
<td>May be required to assess severity of disease or exclude other causes or co-existing liver disease</td>
</tr>
</tbody>
</table>

Summary

Chronic hepatitis C and chronic hepatitis B are generally asymptomatic and therefore frequently hidden to both the patient and the clinician. Since a history of risk behaviour is often not disclosed to doctors, a reason to offer testing and diagnosis may not present itself. When symptoms do occur, they are largely non-specific and common symptoms that may be the result of a myriad of diseases. Consequently, the diagnosis of HCV or HBV infection can be easily missed. Being alert to the possibility of chronic viral hepatitis as a cause of many clinical presentations will allow early diagnosis and the offer of treatment. Blood tests and ultrasound imaging help to assess hepatic function and the presence of complications and other associated disease that may be critical to decisions about prognosis and treatment. However, a lack of symptoms and signs and normal ALT levels does not exclude progressive damage in chronic hepatitis. Liver biopsy is seldom required in patients
with chronic viral hepatitis, largely due to change in s100 criteria and the use of non-invasive methods to assess fibrosis.

Many patients who are aware that they may have put themselves at risk of contracting HBV or HCV are reluctant to seek a diagnosis, not only because of fear of prejudice and hesitancy in facing a potential serious illness, but also because they are pessimistic about treatment outcomes. It is essential that clinicians present optimism, since in recent years there have been substantial gains in outcomes following treatment. Support groups such as state and territory Hepatitis C Councils can be helpful in providing additional resources to help present a more optimistic view and give patients a better sense of control over this chronic condition.

References

CHAPTER 8 ASSESSMENT OF THE PATIENT AND TESTING FOR STIs

2014 REVIEW

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Introduction

This chapter will emphasise testing for asymptomatic sexually transmitted infections (STIs) and its importance both for good patient management and good public health. While it is clear that patients with genital symptoms and who are sexually active need to be tested for STIs, it is important to remember that a large number of STIs are asymptomatic and that if health professionals and patients are not aware of this there is a considerable risk of continuing spread of infection in the community. Chlamydia is perhaps the best example of this situation where there is a continuing rise in annual diagnoses throughout the Australian community. Until there is a heightened awareness that chlamydia can be completely silent, the prevalence of infections will not decrease as a much higher participation in screening by people who have had unprotected sexual contacts is needed. Current screening rates in primary care of the 15-29 age group, which are most affected by chlamydia are less than 10%. Screening in primary care is an essential part of achieving STI control; the importance of screening has been recognised by the RACGP in its recommendations in the Red Book that all sexually active people under 30 years should be offered chlamydia testing annually as this is the age range for the highest incidence of infections. Screening not only will detect asymptomatic infection but in combination with effective treatment and contact tracing should also lead to a declining pool of unrecognised carriers of infection. This chapter will discuss how testing can be undertaken in a non-invasive way in community settings and achieve accurate results and the appropriate treatment, contact tracing and follow-up of patients with STIs. Common presenting symptoms and signs of STIs and the specific infections likely to cause such clinical presentations will be reviewed briefly.

Testing

STIs are usually asymptomatic before they declare themselves clinically and during that time people are infectious to their sexual partners. These people with silent or asymptomatic infection, if having ongoing sexual contacts, are transmitting infections to others. Thus, testing for infection in those at risk, and rapid treatment of those found to have an infection (or in some cases, testing presumptively) and contact tracing their sexual partners are the only practical ways to have any significant impact on preventing morbidity and interrupting ongoing transmission to others.

To achieve prevention of transmission and morbidity, three factors are necessary: clinicians to be attuned to offering STI screening to sexually active patients; suitable tests for use in asymptomatic people; and effective treatments. There are well accepted criteria determining the suitability of testing for diseases. They require knowledge of: the disease (or infection); the performance of the test; and the patient population (Tables 8.1).

It is known that no test is perfect, but clinicians often accept pathology results uncritically. Table 8.1 provides criteria for STI screening. Genital chlamydia infections perhaps come closest to the ideal screening situation. Chlamydia poses a threat to public health and can cause significant morbidity in women (pelvic inflammatory disease [PID], infertility, enhanced risk of ectopic pregnancy, chronic pelvic pain). If the target population is those 15 to 29 years old, and with the highest prevalence of infections being in 15-19 year olds, the prevalence of infection in most countries (including Australia and New Zealand) is sufficiently high to justify widespread screening and the risk of some false positives will be minimised. Both males and females are often asymptomatic for reasonably extended periods.

KEY POINTS

- Testing for STIs in those at risk and rapid treatment of those found with infection are practical ways to have a significant impact on the prevalence of both STIs and HIV in communities.
- In practice three factors are necessary: offering screening to those at risk, suitable screening tests and effective treatments for each STI.
- Opportunistic screening means a clinician takes any opportunity which presents itself to screen patients for STIs.
- No laboratory test is perfect: in low-prevalence populations for any infection, false positives can occur with any test no matter how good its overall performance.
- Appropriate information about STIs must always precede screening tests. Patients must be helped to recognise their potential risk of having an STI, why testing for a particular STI is relevant for their situation and how the clinician proposes to manage a positive test result.
- Testing patients for common STIs and testing specific populations at risk of less common, but significant STIs (like syphilis and HIV) is now best practice.
of time, and uncomplicated chlamydia is readily treated. Treatment involves a highly effective oral single dose antibiotic.

Nucleic acid amplification tests (NAATs) are highly sensitive and specific for *Chlamydia trachomatis* and can be performed with reliable and reproducible results on a range of specimens such as first pass urine (FPU), self-collected urethral or vaginal swab and clinician-collected endocervical swab. The ability to perform non-invasive tests, the simplicity of the test for the clinician and the easy interpretation of results all make it an outstanding test for use in community settings. A new population-based screening for STIs is coming with the advent of point of care testing (POCT) which enables tests to be done in the field and to provide patients with results within a time frame of minutes to a few hours. To be useful in the developing world, POCT will need to be done in the field and to provide patients with results all make it an outstanding test for use in community settings. A new population-based screening for STIs is coming with the advent of point of care testing (POCT) which enables tests to be done in the field and to provide patients with results within a time frame of minutes to a few hours. To be useful in the developing world, POCT will need to be done in the field and to provide patients with results within a time frame of minutes to a few hours.

**TABLE 8.1** Criteria for suitable testing for STIs (adapted from WHO Guidelines)²

<table>
<thead>
<tr>
<th>The infection</th>
<th>The test</th>
<th>The patient population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poses a threat to public health</td>
<td>Good sensitivity allowing detection of asymptomatic disease</td>
<td>Infection sufficiently prevalent to reduce false positive rate</td>
</tr>
<tr>
<td>Has significant sequelae (morbidity or mortality) for the individual</td>
<td>Good specificity reducing false positives to a minimum</td>
<td>Effective treatment available and appropriate facilities and</td>
</tr>
<tr>
<td></td>
<td>Well accepted by the patient population</td>
<td>personnel to administer it</td>
</tr>
<tr>
<td></td>
<td>Simple to perform and simple to interpret result</td>
<td>Patients willing to accept treatment, follow-up and</td>
</tr>
<tr>
<td></td>
<td>Cost effective</td>
<td>further assessment if necessary</td>
</tr>
<tr>
<td>Is present in the population screened with reasonable probability</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Can be detected while patient is still asymptomatic with a reasonable chance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>that significant damage has not occurred</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Is amenable to treatment</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

New Zealand communities), screening individuals for these infections is clinically justified. NAATs for *Neisseria gonorrhoeae* and *Trichomonas vaginalis* on FPU specimens in males or females, or self-collected vaginal swabs in women are non-invasive tests and are acceptable to most individuals.²,³

Unfortunately a NAAT for *T. vaginalis* is not yet commercially available but many reference laboratories offer in-house NAAT testing for *Trichomonas*. Of course, microscopy and culture for *T. vaginalis* can be used instead, but FPU is not an appropriate specimen for this purpose and sensitivity and specificity on vaginal swabs subjected to microscopy is inferior to NAA testing as the organism does not survive long in transport media. Microscopy is also not readily available in a POCT setting and culture is limited by survival of the organism during transport to the laboratory testing site.

HIV and syphilis testing fulfil many of the criteria for testing in asymptomatic individuals; excellent serological tests are available for both infections. The HIV antibody test, now accompanied in Australia by a test for HIV p24 antigen, is perhaps the most ideal test ever available to clinicians with its extremely high sensitivity and specificity. False positives can still occur in very low-prevalence populations but these can be clarified by further Western blot testing. The advent of the *Treponema pallidum* antibody test has overcome the poor sensitivity of the rapid plasma reagin (RPR) test in very early and late syphilis. A positive *Treponema pallidum* antibody test can then be verified by using both the RPR and a specific test (enzyme immunoassay [EIA] or *Treponema pallidum* particle agglutination [TPPA]) for testing asymptomatic individuals.⁴

**TABLE 8.2** Patients to consider for testing in primary care

| 15–29 year olds are a priority for testing (RACGP Red Book)² | Individuals who have more than one partner Recent                     |
|-------------------------------------------------------------|-------------------------------------------------|------------------------------------------------------------------|
| People living with HIV, those with a previous history of STIs| People who are concerned about their partner's behaviour               |
| Aboriginal and/or Torres Strait Islander patients, particularly those aged 15-29 | Pregnant women                                                                 |
| Male and female sex workers and their regular partners       | Patients who have recently changed partners                                          |
| People who inject drugs                                      | Men who are clients of sex workers                                                    |
| Men who have sex with men (MSM)                             | Patients who report unprotected sex or inconsistent condom use with partners         |
| Travellers who have had sex overseas in higher prevalence countries | Patients who have a history of being incarcerated                                      |
In general terms, testing asymptomatic people for the viral infections human papillomavirus (HPV) and herpes simplex virus (HSV) is impractical.

**Testing for STIs in primary care**

STI testing in asymptomatic individuals happens in two situations. One is termed *population screening* and occurs where testing takes place in a given population with a known high STI prevalence (e.g. an Indigenous community or a specific community of men who have sex with men (MSM)) as part of a public health strategy. The other situation is relevant to primary care practice and is termed opportunistic testing, which implies the clinician takes any opportunity which presents itself to test asymptomatic high-risk patients for STIs.5,6

It is relatively easy to set out the tests that should constitute a standard sexual health check in an asymptomatic person. Table 8.2 details a suitable check-list as a guideline. Further information can be gained from the Australian STI Management Guidelines7 and the STIGMA Australian sexually transmissible infection and HIV Testing Guidelines for Asymptomatic Men Who Have Sex with Men 2014.8 As most primary care services are now computerised, it is possible to enhance rates of opportunistic testing by setting reminders and recalls to prompt clinicians to offer STI screening to patients in recognised risk groups.

In a primary care setting it would be best to consider offering opportunistic testing for STIs to all sexually active patients. The RACGP Red Book recommends an annual offer of chlamydia testing to all men and women aged 15-29 years and also recommends regular screening for MSM (Chapter 3).1

**Tests to use and the rationale for using them**

**PCR (NAAT) testing for chlamydia and gonorrhoea**

The tests suggested for asymptomatic testing of a range of patients are set out in Table 8.3. The Table provides only a guideline and clinicians must decide on specific tests needed for their individual patients given their sexual histories and the local known prevalence of specific STIs. Testing for chlamydia is a must in almost everybody with any STI risk at all. In general terms, all the NAATs are equally good whether ligase chain reaction (LCR), polymerase chain reaction (PCR), strand displacement amplification (SDA) or transcription mediated assay (TMA). Their use depends on the local laboratory. NAAT tests are validated for chlamydia and gonorrhoea on FPUs, urethral swabs, cervical swabs and self-collected vaginal swabs, pharyngeal and rectal swabs.

In low-prevalence populations for any infection, false positives can occur with any test no matter how good its overall performance. To avoid false positives entirely, test specificity must be 100% which is seldom, if ever, attainable.4 NAATs have good specificity for chlamydia but slightly lower specificity for gonorrhoea; however, their specificity is not 100% for either infection. This fact is of great practical importance, especially when testing for gonorrhoea in most big cities in Australia and New Zealand where the prevalence of gonorrhoea is currently low in the general population. When the prevalence of gonorrhoea is low in the local population and a NAAT for gonorrhoea shows an unexpected positive in a patient at low risk for STIs, the clinician should regard the result with some caution; as it may prove to be a false positive. Many laboratories automatically perform a further confirmatory test (e.g. a different NAAT) when the initial test gives a positive result, but it is good practice for clinicians to adopt a policy of sending off a further gonorrhoea culture themselves on unexpected positive NAAT results. However it is also wise to offer immediate treatment and to discuss sexual contacts being screened and treated while confirmatory test results are awaited. Although this may lead to overtreatment in some patients it also avoids the risk of failure to adequately treat patients and their contacts if they fail to return for follow-up. NAATs for gonorrhoea are now validated for throat and rectal swabs. Ideally gonococcal cultures should always be collected as well to enable monitoring of antibiotic sensitivity in this infection. Although NAATs for chlamydia still remain invalidated for rectal swabs, their widespread and increasing use in this situation indicates general acceptance.3 It is acceptable to take rectal swabs blind in asymptomatic patients (i.e. without use of an anoscope or proctoscope) or allow the patient to take their own swab. Useful diagrams to show patients how to collect swabs can be found in Chapter 13 (and obtained from the NSW Sexually Transmissible Infections Programs Unit (STIPU) website at http://stipu.nsw.gov.au). Routine testing for pharyngeal chlamydia infections is not thought to be cost effective at this time but is now being recommended as part of STI screening in MSM and should also be considered when screening female sex workers who offer unprotected oral sex.

**Syphilis testing**

In low-prevalence populations, enzyme immunoassay (EIA) is a good test for syphilis as it is highly sensitive; however, in moderate-to-high prevalence populations, many EIA positive results will indicate old, previously treated disease. It is more useful to use the RPR test combined with a specific test (EIA, Treponema pallidum haemagglutination assay (TPHA), TPPA) in those populations as the titre will give some indication of recent infection (RPR 1/16 or greater). This does of
### TABLE 8.3 STI Testing Tool


![STI Testing Tool](image)

### Getting started with an STI discussion

**Bringing the subject up opportunityistically**

*Do you know if you have any other sexual activities that could have increased your risk of other infections?*

*What do you think could be the reasons for the increase?*

*Have you ever been diagnosed with an STI?*

*Who else do you think might have been affected?*

**Using a ‘book’**

*Have you heard of the STI Testing Tool?*

*Do you know where and when it can be obtained?*

**As part of a reproductive health consultation**

*Have you considered discussing contraception with your partner?*

*Are you planning to use condoms?*

**Because the patient requests a ‘checkout’ for STIs**

*Have you considered all aspects of your sexual health?*

*Have you discussed all possible outcomes?*

**Brief Sexual History**

*What did you do last night?*

*Did you use condoms?*

*What did you do last weekend?*

*Did you use any contraception?*

**Other risk behaviours**

*Have you had any other sexual partners since the last check-up?*

*How many?*

*Did you use any protection?*

*What did you use?*

*Have you been diagnosed with an STI?*

*What was the outcome?*

*Is there anything else you want to discuss?*

**Consent**

*Do you consent to the testing?*

*If yes, what other tests do you consent to?*

*If no, what are the implications?*

**Contact tracing**

*Have you been exposed to any other partner?*

*What were the circumstances?*

*What was the outcome?*

*What other steps were taken?*

**Support**

*Where can you find more information?*

*Who can you contact?*

*What resources are available to you?*
course rely on patients remembering that they have been treated for syphilis in the past when the clinician discusses screening with them. In the laboratory a positive syphilis EIA test result will trigger further confirmatory tests being done and should not lead to any delay in diagnosis of new infections and will confirm past treated infections.

PCR testing is now replacing dark-field microscopy examination and has a place in the diagnosis of early infections, especially when clinical suspicion is high and initial serology is negative (see Chapter 13).

**Hepatitis A testing**

Testing for hepatitis A virus (HAV) antibodies in MSM is a sensible measure so that those who are not immune can be offered vaccination. HAV is transmitted by the faecal-oral route and there have been mini-epidemics of hepatitis A in communities of urban gay men over the past two decades.

**Hepatitis B testing**

There is debate about the best screening test for hepatitis B. The purpose of testing for hepatitis B virus (HBV) is twofold: to diagnose chronic HBV infection and to offer vaccination for those not previously exposed to HBV. A positive HBcAb is sensitive and specific and will indicate any exposure for HBV. To differentiate chronic HBV infection in those who are HBcAb positive, a further HBsAg test can then be requested. Most laboratories will automatically do surface antigen and antibody testing if the core antibody is positive. Presence of HBsAb will indicate successful vaccination in those unsure of their vaccination history (as is often the case). In general terms, people who are HBcAb positive do not require HBV vaccination.

**HIV testing**

HIV testing is listed in the Table 8.3 as an optional extra for all except MSM. Although the prevalence of HIV is low in Australasia in all groups except MSM, it is the STI with the most serious consequences for the person with HIV infection. Most patients who are being tested for other STIs would wish to have HIV testing, and clinicians should ensure that all patients who are diagnosed with an STI are offered HIV testing in line with national HIV testing guidelines (see case study).

HIV testing in pregnant women (see Table 8.2) is obviously an important issue because if the clinician is aware that an antenatal patient has HIV infection, appropriate management and antiretroviral therapy can substantially reduce the risk of the baby acquiring the infection. HIV testing in pregnancy should be offered to all pregnant women at first antenatal visit as part of their routine serological testing. Those who decline should have access to further information to ensure that they have made an appropriate decision.

Testing should always be with informed consent of the patient. On a global level, a small but significant number of HIV-positive diagnoses have been missed during the antenatal period because HIV testing has been offered only to those with a clear history of HIV risk behaviour. Even in Australia and New Zealand the potential for missing HIV infections in the antenatal period does exist, so all pregnant women should be offered HIV testing.

**CASE STUDY**

Brianna was an 18-year-old woman living in a regional city in Northern Australia when she presented to her general practitioner with mild tonsillitis. In general discussion, the clinician established that Brianna was sexually active; she had had sex with three different young men over the past 4 months and had only used condoms with one of them (‘because he was a one night stand’). Until 4 months ago, she had been in a regular relationship for 3 years with her first sexual partner before he had to leave for study in Sydney. She had never had a Pap smear, nor been vaccinated for hepatitis B. On examination, she had mildly inflamed tonsils which her GP thought was probably viral; however, she arranged for a throat swab for culture and sensitivity and first passed specimen of urine (FPU) for NAAT for chlamydia as Brianna agreed an STI screen was a good idea. Dr Helen gave Brianna some literature about STIs and the HIV test and arranged for her to return in a week for a Pap smear. Brianna said she would think about a blood test for further STI screening.

Brianna returned next week to be told that she had a positive chlamydia test on her FPU and, surprisingly, her throat swab had grown Neisseria gonorrhoeae. Her doctor prescribed her a stat dose of oral azithromycin and ceftriaxone by intramuscular injection, and they had a longer chat about doing an HIV and syphilis test as well as her Pap smear. Brianna decided to have a blood test and, serology for syphilis, hepatitis B and HIV were requested.

Brianna’s HIV test proved positive both on EIA and Western blot testing. Subsequently, contact tracing in collaboration with the local Sexual Health Clinic led to one of Brianna’s recent partners being found to be HIV positive, possibly as a result of unprotected sex with several women while he lived for a year in Thailand. The other recent partner with whom she had unprotected sex had been treated for gonorrhoea by his GP 2 months before, but was HIV negative.

**TABLE 8.4 STI syndromes**

<table>
<thead>
<tr>
<th>Symptom</th>
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<tbody>
<tr>
<td>Urethral discharge</td>
</tr>
<tr>
<td>Vaginal discharge</td>
</tr>
<tr>
<td>Ano-genital ulcer disease</td>
</tr>
<tr>
<td>Ano-genital lumps and bumps</td>
</tr>
<tr>
<td>Ano-rectal syndromes</td>
</tr>
<tr>
<td>Pelvic pain syndrome in women</td>
</tr>
<tr>
<td>Scrotal swelling</td>
</tr>
<tr>
<td>Skin rash – genit or generalised</td>
</tr>
</tbody>
</table>

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Serological testing for genital HSV infection

Type specific HSV antibody tests are now available to differentiate between HSV-1 and HSV-2 antibodies. They are highly sensitive EIA tests but are not quite so highly specific; false positives can only be excluded by doing further, much more expensive, Western blot testing. They give no indication of the anatomical site of infection: while one can make a reasonably accurate assumption that most HSV-2 antibody-positive people will have genital infection, the same does not apply to HSV-1 antibody-positive people – they may have either genital or oral infection. There are some clinical situations where HSV serology might guide management (e.g. in a pregnant woman whose partner has a known history of herpes), but in a testing situation it has little place at the present time, except in MSM.10

How often to test?

Patients who have had their first sexual health screening often ask when they should have their next check-up and how often they should have them. The patient’s pattern of sexual behaviour and the frequency of any unprotected sexual exposures are important factors to take into account. Where people have a regular partner or partners or a number of casual contacts, or fairly regular partner change (e.g. some MSM), establishing a regular 3-monthly or 6-monthly attendance for a sexual health check-up seems sensible and justified. The more common situation is where people have consecutive monogamous relationships, a sexual health check when a relationship ends, or at the start of a new relationship is a safe option. Some people attend as a couple soon after commencing a new relationship so they are both tested for STIs at the same time before deciding to cease condom use. If couples do attend together the clinician should try to clarify what their expectations are regarding access to each other’s test results and to document their responses in the clinical file for future reference. It is always better to see patients individually so that an accurate sexual history can be taken and to give the results to each person as an individual. If patients want their doctor to inform them of each other’s results it is best for them to attend together. The local sexual health service or public health unit can advise clinicians on how to approach patients with a positive result for an STI when the patient does not wish to disclose to his or her partner or is reluctant to cooperate with the clinician for contact tracing of previous sexual partners.11

Patients also need to be aware of the appropriate time for testing: clinicians need to explain window periods during which it may not be possible to completely exclude an infection. It is also important to ensure that patients know exactly what is being tested for and that, in the case particularly of herpes and wart virus infections, it is not possible to make a diagnosis unless clinical signs are present.

Symptoms and signs of STIs

Although mostly asymptomatic, STIs can eventually cause symptoms and signs. There are several classical syndromes which group together the main symptoms and signs of STIs. When considering diagnosis and management of STIs in primary care it is more helpful to think in terms of these syndromes rather than about each individual STI because patients tend to present with a syndrome rather than with one STI; this is called syndromic management. In resource-poor settings there are various algorithms developed for the management of each syndrome which have proved extremely useful in the provision of rapid, mostly effective treatment even when exact diagnosis of the individual STI (or STIs) responsible for the syndrome is impossible. The major drawback of the syndromic approach is that overtreatment for infections that are not in fact present often occur. In resource-rich nations like Australia and New Zealand, a syndromic approach combined with appropriate judicious testing will combine the best of both worlds — rapid effective treatment of the presenting syndrome accompanied by exact diagnosis of the precise STI (see Chapter 13 for the management of syndromes). Refer to Table 8.4 for a brief description of STI syndromes.

Urethral discharge

A discharge from the urethra is almost always abnormal even if clear, mucoid or intermittent. The only exceptions are the scant discharge resulting from frequent milking or squeezing the urethra to check if a discharge is present in the overanxious patient, and the typical mucoid discharge which can occur while on the toilet as a result of straining to open the bowels in a constipated patient. Urethral discharge can occur in females but is hardly ever noticeable.

Vaginal discharge

There are two main problems in trying to interpret vaginal discharge. Is the discharge of which the patient complains physiological or pathological? If the discharge is deemed to be abnormal, it is important to know where it is coming from — the urethra, the vagina, the cervical canal or the endometrial lining of the uterus. Even with excellent history-taking and careful examination, the answers to these questions are often not readily apparent. This fact explains why the algorithm for syndromic management of vaginal discharge in resource-poor settings is the least helpful of all the algorithms for genital syndromes. In Australia and New Zealand there are easily accessible and reliable tests to help sort out vaginal discharge, but sometimes the true cause of the patient’s complaint still proves elusive.

Ano-genital ulcer disease (GUD)

There are two main problems in trying to interpret ano-genital ulcer discharge. Is the discharge of which the patient complains physiological or pathological? If the discharge is deemed to be abnormal, it is important to know where it is coming from — the urethra, the vagina, the cervical canal or the endometrial lining of the uterus. Even with excellent history-taking and careful examination, the answers to these questions are often not readily apparent. This fact explains why the algorithm for syndromic management of vaginal discharge in resource-poor settings is the least helpful of all the algorithms for genital syndromes. In Australia and New Zealand there are easily accessible and reliable tests to help sort out vaginal discharge, but sometimes the true cause of the patient’s complaint still proves elusive.

Refer to Table 8.4 for a brief description of STI syndromes.
fact, attempts to self-treat by patients using antiseptics, insecticides, detergents and over soaping often result in more persistent ulceration which may perplex the unwary clinician. STIs associated with genital ulceration are HSV, syphilis (the primary chancre and the mucous membrane lesions seen in secondary disease), chancroid, lymphogranuloma venereum (LGV) and donovanosis. Chancroid, LGV and donovanosis are virtually never seen in primary care in Australia and New Zealand, with the following provisos: chancroid has been diagnosed in Australia and New Zealand in recently returned (i.e. in the past week) travellers from endemic areas (South East Asia); there is a current outbreak of LGV in some highly sexually active groups of MSM, but it has been as proctitis rather than as genital ulceration that LGV has revealed itself in this highly specific situation; donovanosis still occurs (but extremely rarely now) in remote Indigenous communities in northern and central Australia and in Papua New Guinea. Where patients have scratched their scabetic genital lesions excessively, traumatic ulceration sometimes results but the complaint of overwhelming local itching makes the diagnosis easy. In primary care practice in Australia and New Zealand genital herpes is far and away the major cause of genital ulceration, with syphilis being a rare cause except in populations with a higher than average prevalence for syphilis (Indigenous communities and MSM).

Ano-genital warts

The warty lumps and bumps characteristic of HPV Infection (usually associated with types 6 and 11) and molluscum contagiosum are the major infectious causes of lumps and bumps in the ano-genital region. Lumps and nodules due to sexually transmitted scabies also occur, with the characteristic itching assisting with the diagnosis. Other lumps and bumps are almost invariably non-infectious and are not due to an STI. Many are normal variants (such as pearly penile papules, Fordyce spots and sebaceous glands); some represent minor skin pathology (such as sebaceous cysts and seborrhoec keratoses). Rarely, neoplastic lesions may present initially as nodules or papules. Clinicians should consult larger sexual health or dermatology texts to familiarise themselves with these genital lesions which can cause enormous concern, especially in young patients.

Ano-rectal syndromes

Ano-rectal syndromes are a rather heterogeneous and somewhat artificial group of symptoms and signs of STIs which predominantly affect the peri-anal area, the anus, the ano-rectal junction, the rectal mucosa and more rarely the gastrointestinal tract. It’s an anatomical syndrome more than anything else. As such, virtually all the STIs and some enteric infections not usually regarded as sexually transmitted (such as shigellosis, salmonellosis, hepatitis A and amoebiasis) can be included. Most ano-rectal syndromes result from infections transmitted during various anal sexual activities (peno-anal, oro-anal, fingers, toys, fists in the anus) and are therefore seen most often in MSM, but any patients, men or women, engaging in receptive anal sexual practices can of course have an infection, and oro-anal insertive patients may acquire gastrointestinal infections from the anal area of a sexual partner. Predominant symptoms of ano-rectal syndromes are perianal itch, anal or more deep-seated rectal pain, anal discharge (often noted as mucopurulent material on the surface of bowel motions), diarrhoea and, rarely, rectal bleeding. The key to diagnosing ano-rectal STI syndromes is to recognise their sexual connection and to ask patients about anal sexual activities. In acute primary herpes infections, the anal canal and rectal mucosa may be grossly inflamed, ulcerated and may even bleed. A relevant sexual history will allow the clinician to perform the correct investigations to determine the correct diagnosis and to allow appropriate management and contact tracing to occur.

Pelvic pain syndromes

Pelvic pain in women can be acute or chronic. Acute (i.e. recent onset) pelvic pain has a variety of different causes such as PID, ectopic pregnancy, endometriosis, ovarian cyst, urinary tract infection, appendicitis and lower bowel disorders. Both PID and ectopic pregnancy require early diagnosis and appropriate intervention, vital for prevention of further morbidity and even mortality. A high index of suspicion for PID in any sexually active woman, and the ability to eliminate ectopic pregnancy as a cause of symptoms before anything else in any woman of child-bearing age are prerequisite skills for primary care clinicians. It’s important to appreciate how subtle chlamydia PID can be in its early stages—almost asymptomatic infection may be the rule rather than the exception, yet irreversible damage to fallopian tubes may result.

Scrotal swelling

Scrotal swelling may be painless or painful. Any scrotal swelling in young men (under 35 years of age) must be taken seriously because of the greater risk of testicular neoplasms in this age group. Acute onset painful swelling in young men may be due to torsion or epididymo-orchitis or, much more rarely, a tumour. Differentiation between torsion and epididymo-orchitis is sometimes extremely difficult. The key point is not to miss a diagnosis of torsion. Missing a diagnosis of epididymo-orchitis with resultant delay in treatment is not the disaster that missing the diagnosis of testicular torsion becomes. Taking a good history (including a sexual history), being familiar with the rudiments of scrotal anatomy, performing a careful examination and arranging a quick ultrasound scan will save most potential disasters from happening.
Rash: genital and more generalised

Genital rashes are mostly not the result of a genital STI. The exceptions are:

- The rash due to scratching because of pubic lice (crabs) or scabies
- The rash due to local candidal infection (balanitis or vulvo-vaginitis)
- The more severe vulval and intertriginous rash sometimes associated with profuse discharge in severe vaginal trichomonal infection
- The episodic non-specific rash that occurs with atypical recurrent genital herpes.

Other genital rashes and skin conditions are associated with dermatological conditions like lichen sclerosus, lichen planus and genital psoriasis and are beyond the scope of this monograph.

There are only three generalised skin rashes associated with STIs which a clinician should be aware of:

- The rash of primary syphilis
- The rash of secondary syphilis
- The rash of disseminated gonococcal infection.

The first two rashes share some characteristics: they both tend to be non-itchy; both may involve the palms and soles; both may be accompanied by systemic symptoms (fever and malaise); and both tend to be erythematous maculopapular rashes. The rash of primary HIV is of shorter duration and likely to be less clinically obvious than the rash of secondary syphilis, but is often accompanied by acute aphthous type ulcers in the mouth and sometimes on genital mucosa. Secondary syphilitic rashes are more variable and can mimic other skin conditions such as psoriasis and pustular acne. These rashes are markers for the most highly infectious periods of HIV and syphilitic infection and so thinking of and testing for HIV and syphilis in the absence of other symptoms is a sensible initial approach. The rash of disseminated gonococcal infection is a more severe vulval and intertriginous rash that occurs with secondary syphilis, but is often accompanied by acute aphthous type ulcers in the mouth and sometimes on genital mucosa. Secondary syphilitic rashes are often associated with profuse discharge in severe vaginal trichomonal infection.

Conclusion

The ideal time to diagnose an STI is before it manifests itself clinically or to pre-empt this by educating patients as to when to consider and request STI testing. Testing patients for the presence of common STIs and perhaps less common but significant STIs like syphilis and HIV is best practice. Every clinician in primary care should be confident to be opportunistic about STI testing by raising the topic of STI screening with patients, performing a risk assessment with the patient and offering testing appropriate to each patient’s situation. It is one area of primary care medicine which can make a real difference to the health of individual patients and the wider community.

References

CHAPTER 9 TALKING ABOUT TESTING

2014 REVIEW

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Introduction

Obtaining informed consent is an integral part of testing for human immunodeficiency virus (HIV), hepatitis B virus (HBV), hepatitis C virus (HCV) and sexually transmitted infections (STIs). In addition, providing information and support around the testing procedure, minimising the personal impact of diagnosis, changing health-related behaviour, reducing anxiety of the person being tested, and preparing the person for the possibility of treatment if tests are positive may all be important.

Introduction

Obtaining informed consent is an integral part of testing for human immunodeficiency virus (HIV), hepatitis B virus (HBV), hepatitis C virus (HCV) and sexually transmitted infections (STIs). In addition, providing information and support around the testing procedure, minimising the personal impact of diagnosis, changing health-related behaviour, reducing anxiety of the person being tested, and preparing the person for the possibility of treatment if tests are positive may all be important.

Links to national testing policies

National HIV Testing Policy 2011

National Hepatitis C Testing Policy 2012

National Hepatitis B Testing Policy 2012

Changes to terminology

The 2006 National HIV Testing Policy recommended that the term ‘pre- and post-test discussion’ replace ‘pre- and post-test counselling’. The 2011 National HIV Testing Policy talks about the need to obtain informed consent for testing.1

Formal counselling, a term that was used in the past, is sometimes required in the management of a person who has tested positive, or in the situation where a person who tested negative is continuing to participate in high-risk behaviours for HIV. This counselling is usually specialised and requires referral to an appropriate service or practitioner.

The context of testing

HIV

Testing for HIV antibody has been available in Australia since October 1984. At that time, acquired immune deficiency syndrome (AIDS) was associated with high morbidity and mortality and an HIV diagnosis was highly stigmatised due to its association with marginalised social groups. HIV antibody testing was promoted primarily as a tool to enhance education and prevention initiatives. Since the mid-1990s, HIV treatment advances have reduced the number of AIDS-related diseases, AIDS notifications and AIDS-related deaths in Australia.2

HCV

Testing for HCV antibody has been available since 1990. As with HIV, HCV infection is stigmatised due to the association with injecting drug use. During the process of obtaining informed consent, questions may be asked about a history of injecting drug use that may be an unwanted reminder of a past phase of a person’s life and may be resisted. Discussion of past or current injecting drug use needs to be approached especially carefully. A patient may hold a self-stigmatised view of themselves; they may also be especially wary of discussing illicit behaviours with authority figures. Care should be taken to gain the trust of the patient; as much as possible taking a non-judgmental approach to illicit and injecting drug use. However, a discussion of previous or present drug use provides an opportunity to educate the person about HCV transmission and the natural history of the disease. As with HIV, the benefits of testing include interventions and treatments to improve clinical outcomes and the facilitation of measures to prevent transmission. Additionally, improved HCV testing and dramatic advances in hepatitis C treatment have similarly changed the medical context of HCV diagnostic testing.

KEY POINTS

- Informed consent is required for HIV, HCV and HBV testing, except for rare occasions when a legal order is made for compulsory testing or in emergency settings.
- The person performing the test should use his or her clinical judgment in securing informed consent.
- Securing informed consent should be based on the clinician’s understanding of the context in which the test is being performed, taking into regard the reasons for testing and an assessment of the person’s capacity to understand the testing and the consequences of a result.
- The decision on how a negative test result is conveyed (e.g., in person, by phone or text messaging) should be based on clinical judgement by the person responsible for conveying the result.
- A positive result should always be provided in person except in extenuating circumstances.
- People should have access to culturally appropriate information in their preferred language, supported by access to a free, professional interpreter when their primary health staff are not familiar with their preferred language (TIS, the Telephone Interpreter Service is recommended by the Multicultural HIV and Hepatitis Service).
Development of improved testing technology, including point-of-care tests, will assist in simplifying the testing process for individuals, including addressing improved access and acceptability for priority populations. These may prove particularly useful in settings commonly used by people who inject drugs. Testing strategies and models will need to be developed and reviewed to allow new testing technologies to be included as they become available.3

HBV

Testing for HBV surface antigen (HBsAG) has been available since 1971. Long-term management of HBV infection has improved due to the introduction of effective antiviral treatment and immunisation.

The availability of HBV vaccination enables clinicians to take an active role in case-finding, leading to lower rates of transmission and identification of people with chronic HBV infection who may be suitable for treatment. Widespread community ignorance about the long-term complications of chronic HBV infection (Chapters 5, 7 and 12) still exists, and patients need to be appropriately educated.

When stigma and discrimination occur in relation to chronic hepatitis B it is believed to be primarily related to people’s poor understanding of HBV prevention and transmission. Many people with HBV are from specific communities; cultural and linguistically diverse (CALD) communities, Indigenous Australians, people who inject drugs and gay and bisexual men. These people are often marginalised and can already be affected by stigma and discrimination on the basis of race, illicit drug use and sexuality. These broader dynamics may negatively impact on clinical management of patients with HBV infection.3

Testing for other STIs is generally easily done and opportunistic screening in at-risk but asymptomatic people is a valuable part of best practice in primary care medicine (see Chapter 8).

Reasons for testing

HIV, HBV and HCV antibody testing is indicated in the following circumstances:

- Patient request
- Identification of blood-borne virus clinical symptoms or signs (Chapters 4, 5, 6, and 7)
- Identification of blood-borne virus risk factors in the patient history (Chapter 3)
- Health-care workers who perform exposure-prone procedures – wanting to monitor their blood-borne virus status
- Part of a routine HIV or HBV screening process, e.g. pregnancy
- Presentation for post-exposure prophylaxis (PEP) after occupational or non-occupational exposure to HIV or HBV
- Diagnosis of another STI. People with an STI infection are at increased risk of acquiring HIV and should be offered testing
- To guide vaccination of people who remain susceptible to HBV infection.

Risk factors from the patient history which would indicate blood-borne virus testing include:1

- History of injecting drug use
- Conducting exposure-prone procedures for health-care workers
- Having recently travelled overseas; travellers may be at risk of blood-borne virus through unprotected sex, injecting drugs and medical procedures
- MSM sexual contact. This is the most common mode of HIV transmission in Australia2 and unprotected anal male-male sex is a clear indication for HIV testing, as well as testing for other blood-borne viruses
- Being the sexual partner of a person with HIV infection
- Incarceration is an independent risk factor for HCV infection
- Children born to mothers with HIV, HCV or HIV
- Being from a country or region with a high blood-borne virus prevalence, e.g. the Caribbean, Sub-Saharan Africa, South East Asia, Pacifica and Papua New Guinea (see Chapter 2)
- Reporting a reactive result on a point of care test (POCT), whether in a clinic setting, an outreach service, home-based testing or from an unlicensed test
- Being a recipient of organs, tissues, blood or blood products before February 1990.

Testing may relate to antenatal testing, pre-surgical testing (this is not routinely recommended), military requirements, correctional services, blood, organ and sperm donation, and immigration or insurance requirements. Regardless of the reason for testing, informed consent is important.

Patients who request testing may not reveal all aspects of their risk of infection. In some situations, the clinician may assess the risk of infection as low but the patient’s actual risk of infection may be high. For this reason, all patients requesting testing should be tested. Some patients, for example young people, may attend hoping to arrange an HIV, HBV or HCV test but are unable to state this request directly. In such cases, a request for a check-up or blood tests may prompt questioning by the clinician to elicit specific concerns (Case study 1).
CASE STUDY 1 An adolescent may request testing indirectly

Indirect requests for testing
Mary is a 16-year-old girl who presents for a check-up and reports feeling sick. Upon history and examination she is well but the clinician decides to perform a full blood count and iron studies. While the blood is being taken, Mary asks, ‘By the way, doctor, does this test for AIDS?’ Subsequent assessment indicates that Mary has had unprotected vaginal sex and is concerned about STIs. The clinician performs HIV pre-test discussion and conducts a full STI screen including an HIV test. A follow-up appointment is arranged and information provided about the local youth service which provides targeted health information.

Legal requirements
The Medicare Benefits Schedule (MBS) stipulates that a practitioner requesting an HIV test must ensure that a patient undergoing an HIV test has given informed consent and has received appropriate discussion. Some states and territories have specific legal regulations relating to pre-test and post-test discussion for HIV and viral hepatitis, which may be used as a guide for minimum standards of care. Clinicians should contact relevant state or territory health departments for details.

Chapter 15 contains further discussion of legal responsibilities and highlights the need for full documentation of recommendations, counselling and follow-up undertaken by the clinician.

Obtaining informed consent
During this process, the discussion should be appropriate to the gender, culture, behaviour and literacy level of the person being tested and to their intellectual capacity. In addition, the discussion should be tailored to the condition being tested for. Information may be exchanged and concerns explored. That is, the discussion for HIV, for a high-risk man who has sex with men (MSM) in a major city will differ from the discussion for a pregnant, Indigenous woman undergoing testing for HBV in a remote area of Australia.

A number of issues may be discussed depending on the person concerned and his or her risks. In situations where HIV, HCV and HBV testing is undertaken, unless a person opts-out, measures should be taken to ensure that those who choose testing are free of any form of real or perceived coercion to be tested.

Informed consent aims to ensure that the person being tested agrees to be tested and that he or she understands:
• the testing procedure
• the reasons for testing
• the personal implications.

Health literacy may be low among some marginalised groups, and those from some CALD communities. A tool that is increasingly promoted to ensure patient uptake and retention of health information is teach-back or check-back. Patients are asked to explain back to the health professional in their own words what they were just told.

Furthermore, the informed consent process can be used to ensure the patient is able to take advantage of the health information gained through having the tests done, irrespective of the results.

History-taking and risk assessment
A non-judgmental approach is essential to facilitate honest answers to highly personal questions. Consideration of actual risk practices, rather than making assumptions based on the patient’s perceived membership of a particular risk group, is the accurate way to perform a risk assessment. Chapter 3 addresses sexual and drug-use history-taking in detail, and Chapters 3 discusses risk assessment.

Issues to cover
Table 9.1 lists topics to be addressed during a discussion before testing. As mentioned previously, this discussion may be very brief or may take some time, depending on the person being tested and the situation. In particular, the following key points should be discussed regarding blood-borne virus testing:

TABLE 9.1 Summary of pre-test discussion

<table>
<thead>
<tr>
<th>Reason for testing and risk assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Timing of risk and option of post-exposure prophylaxis (PEP)</td>
</tr>
<tr>
<td>Need for other STI and blood-borne virus testing</td>
</tr>
<tr>
<td>History of testing</td>
</tr>
<tr>
<td>Confidentiality and privacy issues around testing</td>
</tr>
<tr>
<td>Ensuring there is informed consent for the test</td>
</tr>
<tr>
<td>Natural history and transmission information (if appropriate)</td>
</tr>
<tr>
<td>Prevention of transmission and risk reduction through behaviour change</td>
</tr>
<tr>
<td>Implication of a positive or indeterminate test result, including availability of treatment</td>
</tr>
<tr>
<td>Implications of a negative test result</td>
</tr>
<tr>
<td>Explanation of the window period</td>
</tr>
<tr>
<td>General psychological assessment and assessment of social supports in the event of a positive result</td>
</tr>
<tr>
<td>Logistics of the test: time taken for results to become available and the need to return for results</td>
</tr>
</tbody>
</table>

Confidentiality
Advising the person of the measures the service or practice takes to protect personal information, including results, as well as public health notification requirements (Chapter 15). Patients who do not wish
to disclose their name or Medicare number should have access to coded testing (e.g. using the first two letters of surname and first two letters of given name plus date of birth).

**Medical consequences of infection**

Provide information about the natural history and modes of transmission for HIV, HBV or HCV (Chapters 1 and 2 and Appendix 1–3).

**Information about prevention**

Discuss the relative risks of transmission of HIV, HBV and HCV associated with various practices. Explore the person’s ability to practise safer sex or safe injecting (Chapter 3).

**The implications of a positive result**

Inform the patient what the results of a positive screening test mean (care needs to be taken with regard to HBV testing):

- **HIV**: EIA or Ag/Ab or Western blot tests returning a positive result suggest viral infection has occurred.
- **HCV**: anti-HCV test returning a positive result suggests viral infection has occurred.
- **HBV**: anti-HBc IgM or HBsAg tests returning a positive result suggest viral infection has occurred. Anti-HBs and anti-HBc (total) tests returning a positive result suggest a natural HBV immunity or induced immunity via vaccination in the case of isolated anti-HBs being positive.

Discuss the implications of chronic infection for sexual relationships, the existence of treatments and the emotional and social supports that people with an infection can access. For HIV, modern treatments mean that reaching an undetectable viral load is generally easily achievable, and sexual transmission is then very unlikely. For HCV, current treatments are usually curative, though not always easy to take, but newer treatments will be substantially freer of side effects. The benefits of HBV immunisation for household members and sexual partners may be relevant. Some people may be reluctant to test even when the availability of treatments has been explained to them. They may believe that it will be impossible to keep results private and they may hold well-founded fears of discrimination, social exclusion or personal violence that may follow disclosure of HIV or viral hepatitis infection.

**Implications of an indeterminate result**

Prepare the patient for the possibility of an indeterminate result and the need to re-test.

**The window period**

Explain this concept and its possible implications. The window period is usually defined as the period after which it is certain that the person being tested will not seroconvert following a given exposure. The true window periods for HIV and HCV antibody tests have improved greatly over the years.

In Australia, the currently used HIV antibody tests (highly sensitive in themselves) are combined with an HIV antigen test and so can demonstrate reactivity as early as 2 to 3 weeks after the infecting event. The currently licensed POCT has testing for antibody and antigen, also, and its sensitivity in early HIV is similar to laboratory-based testing. Some other POCTs do not detect antigen and may take a little longer to become reactive.

For HIV, 3 months is still usually quoted as the window period, although in practice in Australia, this is rarely the case – a figure of 6 weeks is often used by experienced clinicians. The window period will be determined by the type of test used. More advanced HIV tests (see above) can detect infection sooner than others; however not all jurisdictions currently use the more advanced technology. It is important that a practitioner delivering a test result is aware of what test is being used and how soon after infection it can detect infection. If he or she does not have that information then a window period of 3 months should be used. It is important to explain that someone who has recently acquired HIV is highly infectious during the window period.

For HBV, HBsAg is usually detected 4-6 weeks after exposure.

For HCV, HCV-RNA is usually detected 2 weeks after exposure.

**The implications of a negative result**

Explain that the absence of antibodies (the negative result) means either the person does not have the infection or that he or she is in the window period of infection, before the development of antibodies (see above).

A negative anti-HBc (total) test suggests the need for vaccination to protect against future infection if vaccination has not already been undertaken.

**Coping with a positive result**

Previous ways of coping with crises may indicate how the person will cope with a positive test result. People with a history of depression or other psychiatric issues and those without self-perceived social supports are especially vulnerable following a positive diagnosis.

Assess the patient’s psychiatric history and risk of suicide or self-harm, and identify appropriate interventions in the event of a positive diagnosis. In cases where high-risk practices or clinical features are suggestive of infection, in-depth discussion of these issues may form the basis of a future management plan.
Referral
The need for assistance from other agencies may arise during the pre-test discussion and clinicians need to have a low threshold for referral to specialist agencies. For example, when assessing patients with a history of injecting drug use, issues related to homelessness, poverty or drug and alcohol dependence may become apparent and referral may be indicated (Chapter 16).

Summary
While pre-test discussion may seem time consuming, practice ensures that time is used efficiently within the primary care context. Clinicians will often develop their own style for discussing HIV and viral hepatitis, tailoring information and language to the needs of individual patients. Many of the issues listed above may not be relevant to every patient each time he or she presents for testing, but assumptions regarding the patient’s level of knowledge should be avoided. While the process may seem unnecessary in low-risk patients, some discussion ensures that prevention measures are in place, the patient is prepared for his or her test results, and the clinician’s ethical and legal obligations are met. Such discussions may also be valuable in informing the broader community about recent changes in the diagnosis and management of these viruses.

Post-test discussion
The results of blood-borne virus test results may be given in a variety of ways – via phone, e-mail, text messaging or in person. It is important to establish with people the manner in which they wish to receive their results, taking into consideration the risk history and ability to cope with an indeterminate or positive test result.

Further testing for other STIs and blood-borne viruses should be recommended as appropriate.

Giving a positive result
Positive results should always be given in person, except in very specific circumstances. Key points to be discussed in relation to a positive HIV, HCV, HBV test include (Table 9.2):

Assess patient readiness to receive the result
The person may be asked whether he or she has thought about the likely test result and its implications.

State the result clearly
Some people confuse a positive result with a good result. Ensure that the actual result is understood.

Seek consent to repeat the test for confirmation
Mistakes in labelling at the surgery or in the laboratory are rare but they still do occur. It is important not to raise the patient’s hopes too much over this issue, however.

Avoid information overload
Give the patient time to process and react to the information. Listen and respond to his or her needs.

Reinforce commitment to health care
The primary care clinician may reassure the patient that he or she will continue to be a partner in the patient’s health care without discrimination.

Enlist available supports
Help plan the person’s next 24–48 hours. Support from a community based organisation such as a hepatitis organisation or group for people living with HIV infection may be very helpful. Arrange a follow-up appointment during the next 2 days and offer an after-hours phone contact number.

Discuss disclosure
After a positive result, the patient may experience an urge to tell many people. The balance between disclosure and privacy can be difficult, and the clinician may caution the patient about widely disclosing his or her positive status during the first few days after diagnosis, due to the possibility of negative responses from some people.

Supply written material
Supplying written material gives the person something to read outside of the consultation, reinforcing key messages that may not have been heard in the context of the shock of receiving a positive result. Information may address the medical and social consequences of HIV, HBV or HCV infection and provide details about local support services, including telephone information and support lines, AIDS Councils or Hepatitis organisations (Chapter 16). The ASHM website (www.ashm.org.au) provides patient fact sheets including support services.

Reinforce prevention message including information about modes of transmission
This discussion may form the basis of starting the contact tracing process. It is also a good opportunity to discuss treatments and their role in reducing the risks of transmission.

Managing a positive result
Much of the initial management of a new blood-borne virus diagnosis is psychosocial. Offering the patient the opportunity to return at any time to discuss concerns may help him or her adjust to the diagnosis.

Chapters 10, 11, 12 and 13 discuss the initial and ongoing assessment, monitoring and management of patients with HIV, viral hepatitis, and STIs.
Clinicians inexperienced in managing patients with blood-borne virus infections should collaborate with more experienced general practitioners and relevant specialists and specialist centres (Chapter 16 and the ASHM Directory available through the ASHM website).

**TABLE 9.2 Summary of post-test discussion: giving a positive result**

<table>
<thead>
<tr>
<th>First post-test consultation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Establish rapport and assess readiness for the result</td>
</tr>
<tr>
<td>Give positive test result</td>
</tr>
<tr>
<td>Avoid information overload</td>
</tr>
<tr>
<td>Listen and respond to needs (the patient may be overwhelmed and hear little after being told the positive result)</td>
</tr>
<tr>
<td>Discuss immediate implications along with the availability of modern and highly effective treatments</td>
</tr>
<tr>
<td>Review immediate plans and support</td>
</tr>
<tr>
<td>Reassess support requirements and available services</td>
</tr>
<tr>
<td>Arrange other tests and the next appointment</td>
</tr>
<tr>
<td>Begin contact tracing process and discuss options available to facilitate this</td>
</tr>
</tbody>
</table>

**Subsequent consultations**

| Treatment options and benefits, diet and exercise |
| Effect of diagnosis on relationships and prevention information |
| Issues of disclosure |
| Assessment of contact tracing process and difficulties encountered |
| Access to life insurance may be affected |
| Workplace implications |
| Impact of other issues (e.g., drug use, poverty, homelessness) on ability to access health care and treatments |
| Referral for on-going counselling, social worker, medical specialist as appropriate |

**Giving a negative result**

**Inform the patient of the result**

Tell the patient that he or she does not have the infection. If appropriate, discuss the window period and make an appointment for re-testing.

**Educate the patient about ongoing risk-taking**

Review safer sex and safe injecting practices. Discuss the role of drugs and alcohol in risk taking, as well as how and where to access condoms and clean injecting equipment. Offer referral to local services as appropriate (Chapter 16).

**Offer vaccination**

Hepatitis A and hepatitis B vaccination may be offered, plus HPV vaccines in young women or men who may not have been vaccinated.

**Address attitudinal barriers**

A negative result leaves time to explore important issues that may impact on infection risk. For example, a negative result after a high-risk encounter may reinforce a sense of invincibility among young people, especially young men. Such responses need to be addressed.

**TABLE 9.3 Summary of post-test discussion: giving a negative result**

| Explain the negative test result and the window period (if relevant) |
| Reinforce education regarding safe behaviours |
| Consider vaccination – for hepatitis B, hepatitis A (if indicated), and, for women aged between 9 and 26, human papillomavirus (HPV) |
| Further discuss anxiety or risk behaviours |
| Discuss testing for other STIs |

**Indeterminate results**

Occasionally, an equivocal or indeterminate result from blood-borne virus testing may occur. This result can be a source of great uncertainty and anxiety for the patient. Clinicians may need to consult pathology laboratory staff or the National Serology Reference Laboratory for specialist advice in interpreting indeterminate results. Specific tests for each blood-borne virus have different types of equivocal results and differing rates of false positivity. In the case of HIV antibody testing, a positive ELISA and a single band on Western blot analysis constitutes an indeterminate result.

A patient with an indeterminate result who has reported a recent high-risk exposure is regarded as being in the window period of infection and may require considerable support during this time to deal with the uncertainty. Further tests for viral antigens may be indicated to test for the presence of infection and should be performed in consultation with a specialist clinician. If reactivity in HIV or HCV antibody tests does not progress over approximately 2 weeks it is unlikely that a person is seroconverting.

The result is likely to remain indeterminate due to the presence of non-specific reactivity in the test. Thus a clinician can take a second sample soon after the first to determine the progression. However, to be sure to address absolutely the fears of the person being tested or the health-care worker’s doubts, test results at approximately 12 weeks for HIV and 6 months for HCV should be obtained.
In populations of low seroprevalence of blood-borne viral infections, indeterminate results may be false positives. Factors such as pregnancy, past blood transfusions, intercurrent viral infections, autoimmune diseases and malignancies may play a role in equivocal results. Upon re-testing at approximately 2 weeks, a second indeterminate result is regarded as confirmation of negative status. Note: specialist assistance in these situations is available from pathology laboratory staff and via ASHM.

One of the more confusing HBV serology results is when the patient is anti-HBs negative and HBsAg negative, but anti-HBc positive, known as isolated core antibody positive. This result can have a number of possible interpretations: 5

- distant resolved HBV infection – the most common interpretation, particularly in people born in HBV endemic areas
- false-positive result – more common in people with a low risk of past HBV infection
- resolving acute HBV infection – in the period between HBsAg loss and detectable anti-HBs development
- passive transfer of maternal anti-HBc – in children up to 3 years of age
- occult HBV infection – a rare situation where active HBV infection occurs in the absence of detectable HBV infection. This can be determined by detecting HBV DNA in serum.

Note: this test is not Medicare rebatable in the absence of HBsAg. This should be considered if there is clinical or biochemical evidence of active liver disease.

Special considerations

Point of care testing

POCT for HIV has been licensed in Australia since 2013 and is available in some states and territories. Worldwide there are many brands of test, with some testing for antibody alone, and others testing for antigen and antibody. Most use a finger-prick of blood, but others are designed for use with venous blood samples, saliva or mouth fluid. When using POCTs it is important that the clinician is aware that a reactive result may occur almost before the eyes of the clinician and the person being tested, if the test is sited where it can be seen. For such reasons it is often wise to place the test out of sight while it is being conducted. A reactive result obviously has implications for the testing process and the process of gaining informed consent, which must still be done. The clinician needs to be prepared for the possibility of a reactive result and to have the necessary time and expertise to deliver such a result and to then perform venesection to obtain blood for confirmatory testing.

HBsAg POCTs are available and may be useful in some settings (e.g. testing in remote communities and where there are barriers to accessing traditional health care). However, HBsAg POCTs are known to have a lower analytical sensitivity compared to standard laboratory immunoassays and may be unable to detect low levels of HBsAg. POCTs must comply with the regulatory framework for in vitro diagnostic medical devices (IVDs) and be included in the Australian Register of Therapeutic Goods. A positive or negative POCT should be confirmed by standard HBV testing in a National Association of Testing Authorities Australia (NATA) certified diagnostic laboratory.

The availability of combination antiretroviral therapy (cART) in the contemporary Australian setting has dramatically changed the medical context of HIV antibody testing; an HIV diagnosis now opens up the possibility of appropriate treatment and improved prognosis, and of greatly reducing the risk of transmission. However, despite treatment advances and changes in social perceptions, HIV infection remains a stigmatised condition, and all people who are tested should give informed consent for the process.

Aboriginal and Torres Strait Islander People

The rates of HIV diagnosis per capita in the Indigenous and non-Indigenous populations are similar but there is evidence that Indigenous people are more likely to be diagnosed later in the course of the infection, and therefore have a higher advanced HIV disease diagnosis rate, and a substantially greater proportion is attributed to injecting drug use (13% compared with 2% in the non-Indigenous population) between 2008 – 2012. Higher prevalence of ulcerative and non-ulcerative STIs in this population may contribute to HIV transmission and STI testing should be offered.

The rates of HCV and HBV diagnosis per capita in Indigenous populations are elevated and suggest special attention be given to screening and monitoring for these viruses. This is important given the emergence of more effective treatments for both hepatitis B and hepatitis C, and the availability of HBV vaccine.

The primary objective of the Third National Aboriginal and Torres Strait Islander Blood Borne Viruses and Sexually Transmissible Infections strategy 2010-2013 is to improve access to testing and medical care for HIV, blood-borne viruses and STIs among Aboriginal and Torres Strait Islander people.

Facilitating this goal may involve:

- Understanding differing epidemiology of blood-borne virus and STI in different local settings, for instance, higher rates of infection through heterosexual contact and intravenous drug use.
- Addressing local and cultural issues, such as stigma and shame, associated with HIV, blood-borne virus and STI testing and diagnosis. Routine screening
through antenatal clinics, adult health checks and community STI screening interventions may help reduce the stigma around testing.

- Local systems and policy to ensure confidentiality around STI and blood-borne virus testing.
- Specific programs to facilitate testing through collaboration and partnerships between Indigenous organisations and groups and specialist sexual health and blood-borne virus services. Local input to ensure the relevance and appropriateness of programs aimed at different subgroups, e.g. youth, MSM, sex workers and people with a history of incarceration.
- Pre- and post-test discussion may need to incorporate local patterns of transmission and modes of disease prevention. Education around the potential for blood-to-blood transmission in traditional ceremonial practices may be particularly relevant in some Indigenous settings and discussion should incorporate this information in an appropriate manner.
- Pre- and post-test educational resources such as videos or online resources in Indigenous languages or plain English may assist to ensure informed consent and aid blood-borne virus and STI prevention education.
- Antenatal testing. As heterosexual transmission of HIV occurs in many Indigenous settings, antenatal testing may provide an important opportunity to inform, educate and test Aboriginal and Torres Strait Islander women for HIV. Increased incidence of hepatitis B suggests the need for thorough HBV screening.
- Consideration of the need for an interpreter. However, an interpreter may be closely connected with the patient’s family and may create a fear regarding a possible breach of confidentiality. Indigenous health workers may provide the ability to offer more culturally appropriate information and to enable the Aboriginal or Torres Strait Islander person being tested to engage more in the testing process.
- Testing for other STIs and blood-borne viruses. If HIV is detected, Aboriginal and Torres Strait Islander people should also be tested for human T-lymphotropic virus type I (HTLV-1) where appropriate as this is more common in this population and may alter disease progression and management.19

Other cross cultural issues

Culture, language, literacy level, gender and age will affect how a person accepts and understands HIV, HBV and HCV testing, but this should not interfere with provision of pre-test and post-test discussion.

Language barriers may be overcome by the use of an interpreter and language education resources such as leaflets, videos and multimedia.

HIV, HBV or HCV phobia

Occasionally the clinician will encounter a person whose fear of infection with HIV or viral hepatitis is out of proportion with the actual risk of infection. Such people, sometimes referred to as the ‘worried well’, may repeatedly request HIV or HCV tests after encounters that carry very low or no risk of transmission. Often these people are helped by emotional support or a discussion of the encounter and the provision of factual information about the risk of transmission. This may not be adequate for some people who may have co-existing psychiatric morbidity, such as undiagnosed obsessive compulsive disorder, and may need referral for specialist counselling or psychiatric assessment. Unfortunately, most will not avail themselves of the opportunity for further counselling or treatment, and such patients can become a significant drain on the time and resources of a service. Asking advice from an experienced clinical can be helpful in such circumstances.

Testing and pregnant women

Why test pregnant women?

The risk of perinatal transmission of HIV and HBV can be significantly reduced with appropriate clinical care and interventions.

The basis for offering pregnant women HIV testing is the ability to prevent mother-to-child transmission. Several studies published in the mid-1990s demonstrated that azidothymidine (zidovudine) monotherapy reduced mother-to-child transmission from 25% to 8%.10-12 The use of combination therapy plus planned caesarean delivery and bottle-feeding has reduced HIV transmission to less than 2%.13-16 Mother-to-child transmission of HIV has fallen dramatically in countries where antiretroviral therapy is available to pregnant women.17

Interventions to prevent HBV infection are well established and reference to the National Health and Medical Research Council’s Immunisation Handbook is advised.18

Considerable anxiety and guilt may be associated with diagnosis during pregnancy. Special attention should be paid to the psychosocial aspects of receiving a positive test result during pregnancy. Discussion should include an assessment of the negative effects of diagnosis (e.g. discrimination, domestic violence, psychological difficulties) and should provide information on how to minimise these.
The clinician should evaluate a pregnant woman with HIV or HBV infection to determine her need for psychological and social services. Specialist counsellors or midwives with training in this area may be engaged during this process. The implications of the test result for both mother and child should be reiterated, as should treatment options and measures for preventing perinatal transmission so the woman can make informed decisions regarding her options.

### HIV testing during pregnancy

The 2011 National HIV Testing Policy recommends all pregnant women should be routinely offered HIV testing.1 Pregnancy is a time when women are in contact with clinicians, and it provides an opportunity for detection of previously undiagnosed infections. Previous policy suggested HIV testing in pregnancy if a risk assessment suggested possible HIV risk. However, many women diagnosed with HIV do not acknowledge risk factors, and therefore standard risk assessment may be inadequate to test and detect women with HIV infection.19-21 Because prevention of mother-to-child transmission of HIV is highly effective if HIV is diagnosed antenatally, routine testing with informed consent is now the standard of care.

### HBV testing during pregnancy

Women contemplating pregnancy or seeking antenatal care should be made aware of the benefits of diagnosis of HBV infection and management, and prevention strategies available to protect the infant from infection (see Antenatal Testing and Blood-Borne Viruses (BBVs). ASHM).22 The Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG) guidelines state that all pregnant women should be routinely screened for HBsAg and HBsAb, regardless of previous testing or vaccination.22 If a woman is identified as HBsAg positive, further testing (HBeAg and HBV DNA) should be performed to determine the risk of transmission to the infant and the degree of infectivity in general, to inform clinical decision making.6 Transmission from mother-to-child will not occur if the mother has spontaneously cleared the HCV infection, so all pregnant women who test positive for anti-HCV antibodies should be offered qualitative HCV RNA testing to determine if they are still viraemic. Infants born to anti-HCV-positive mothers will have passively acquired antibodies. It is recommended that HCV RNA be tested at 8 weeks and again 4 to 6 weeks later to confirm ongoing infection and to exclude transient viraemia which can occur in infants. If the test returns positive on both occasions, the child should be referred to a paediatric gastroenterology or infectious diseases unit for 6-monthly monitoring of liver function (this may require travel to a major centre where this service is available). All children born to anti-HCV-positive mothers should have antibody testing at 18 months of age because in rare instances transmission occurs from mothers with low and/or fluctuating HCV RNA levels and who test negative at the time of delivery.

### Summary

Gaining informed consent for HIV and viral hepatitis testing (as well as for other STIs) provides the clinician with the opportunity to review and reinforce prevention and risk reduction messages. It also protects patient autonomy by ensuring informed consent regarding testing and helps prepare patients for positive test results. The benefits of early diagnosis, in terms of access to treatments and improved disease outcomes, should be highlighted when recommending testing. In the context of a positive result, post-test discussion and referral for counselling deals primarily with psychosocial issues, prevention of further transmission, contact tracing and information about the benefits of ongoing monitoring and early treatment. Testing may also be an opportunity to engage with marginalised people – it may represent an opportunity to draw such people into the health-care system, or, at worst, not to alienate them further. ASHM can provide information and education resources on pre- and post-test discussion.

### References


24. HBsAG testing in Australia (blood screening) http://www.redcrossblood.org/hospitals/infectious-disease-testing

MANAGEMENT IN THE PRIMARY CARE SETTING
CHAPTER 10 PRIMARY CARE
MANAGEMENT OF HIV DISEASE

2014 REVIEW

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**Introduction**

The chapter aims to provide the human immunodeficiency virus (HIV) non-specialist clinician with an update on the management of HIV disease and to describe the role of the primary care clinician in the shared management of patients with HIV infection. When managing patients with HIV disease, non-prescribing primary care professionals work in conjunction with a clinician, either a general practitioner or physician, who is able to prescribe antiretroviral drugs under Section 100 (s100) of the Pharmaceutical Benefits Scheme (PBS), as well as other services and agencies.

The general practitioner is essential in contributing to a team-based approach to optimise patient care in all chronic conditions including HIV. Based on earlier models of antenatal shared care, there is now a model of shared care for HIV (Figure 1). The general practitioner is able to collaborate with available specialist care. If an s100 prescriber, he or she may be the primary prescriber of HIV medications as well as monitoring and screening the patient, or the general practitioner may be monitoring and screening the patient for HIV as well as non-HIV conditions. General practitioners may undertake training to enhance their clinical skills in this area or to become s100 prescribers (http://www.ashm.org.au/default2.asp?active_page_id=134). Information regarding support for any clinician involved in HIV care can be found at: http://www.ashm.org.au/default.asp?active_page_id=1

Management of chronic HIV infection is not unlike that of other chronic illness such as diabetes, ischaemic heart disease and viral hepatitis. Ideally, patients are active participants in their health management and require an understanding of the HIV infection, management and treatments. The HIV clinician has a role in facilitating the patient’s understanding of these aspects, empowering self-management skills and facilitating the health literacy of the patient.

The quality of the doctor-patient relationship is central to the successful long-term management of HIV.

Optimal patient engagement and management may be assisted by:
- adequate assessment of the patient’s ethnic identity, gender, philosophical framework and sexual identity.

**KEY POINTS**
- HIV remains a complex and chronic disease and its management continues to evolve.
- Antiretroviral therapy is recommended for all individuals with HIV infection to reduce the risk of disease progression and decrease the risk of transmission.
- Commencement of therapy needs to be negotiated by patient and doctor.
- Antiretroviral therapy (ART) should be continuous and life long.
- Patients with CD4 counts > 500 cells/μL have a life expectancy approaching those who are HIV negative (if adherent to ART).
- The central biomedical goal of treatment is complete suppression of HIV replication to a level below detection.
- The primary care clinician has a role in supporting treatment adherence with patients who are taking antiretroviral therapy, as well as monitoring for adverse events and drug interactions and harm minimization with regards to onward transmission.
- The general practitioner has the specific role of coordinating all aspects of the patient’s care similar to other chronic disease states.
- In the era of life-long therapy for patients with HIV, all clinicians involved in their care need to remain focused on general health maintenance, psychosocial issues and screening for comorbidities.
• negotiation of mutually understood goals for therapeutic intervention within a long term, comprehensive management plan to achieve ongoing viral suppression, maintenance of immune function, prevention of onward transmission of HIV infection within a mutually agreed timeframe.

• development of a management plan that facilitates adequate and thorough ongoing assessment and management of HIV, within an annual cycle of care, that considers all aspects of the patient’s health needs and comorbidities.

• durable record keeping.

• provision of information and referral specific to the needs of the individual.

This chapter explores the natural history of the HIV infection, psychosocial assessment and medical assessment, treatment, monitoring the patient in a primary care setting and then managing the issues related to health maintenance and HIV’s impact on sexual and reproductive health.

Natural history

Following acute infection with HIV (Chapter 4), there is a stage of clinical stability where immunological and virological markers remain relatively stable. During this period, homeostasis exists between the amount of HIV produced and cleared each day, and the number of CD4 T-lymphocytes (CD4 cells) produced and destroyed each day. Subsequently clinical stability may continue despite deterioration in laboratory markers as the immune system progressively fails to replenish the CD4 pool (Chapter 1). At this time, the amount of HIV measurable in the plasma (the viral load) may increase as the number of CD4 cells falls. Constitutional symptoms (lethargy, fatigue, diarrhoea, weight loss and night sweats) may occur in the presence of a high viral load at any stage of the disease. Early symptoms of immune deficiency begin to appear when the CD4 count falls below normal levels (Figure 10.2; Chapter 1). As the CD4 count decreases to levels below 200 cells/μL, the patient is at greater risk of opportunistic infections and acquired immune deficiency syndrome (AIDS)-related malignancies. The average time to progression to AIDS is about 8 years but progression rates vary widely at the individual level. Determinants of the rate of disease progression include age and virological and host factors.

HIV infection is dynamic and when assessing the patient it is important to consider that HIV-related illness can occur at higher CD4 counts than those quoted above. HIV can be described as a chronic inflammatory condition demonstrated by elevated inflammatory markers: C-reactive protein (CRP), fibrinogen, interleukin 6 (IL-6), and tumour necrosis factor (TNF). Risk markers include: low CD4 nadir, long duration of untreated HIV and persistently high viral load, persistence of CD4/CD8 below 20%, and low haemoglobin count. The chronic inflammatory state of HIV may contribute to the increased cardiovascular disease, metabolic disease, musculoskeletal pain, and frailty, seen in patients with HIV.3

Inflammatory markers are higher in patients not on cART, however evidence shows that inflammation persists in patients on cART with CD4≥500.

An understanding of the natural history, the dynamic nature of HIV disease and data from randomised clinical trials provides the basis for the psychosocial and medical assessments that will inform ongoing management and treatment decisions.

**FIGURE 10.2 HIV Natural history**

<table>
<thead>
<tr>
<th>CD4 cell count (cells/μL)</th>
<th>TIME</th>
</tr>
</thead>
<tbody>
<tr>
<td>1000</td>
<td>10 weeks</td>
</tr>
<tr>
<td>500</td>
<td>5 years</td>
</tr>
<tr>
<td>200</td>
<td>10 years</td>
</tr>
<tr>
<td>Fever</td>
<td>PCP, KS</td>
</tr>
<tr>
<td>Myalgia</td>
<td>MAC, CMV</td>
</tr>
<tr>
<td>Rash</td>
<td>Toxoplasmosis</td>
</tr>
<tr>
<td>Pharyngitis</td>
<td>Cryptosporidium</td>
</tr>
<tr>
<td>Adenopathy</td>
<td>Lymphoma (NHL)</td>
</tr>
<tr>
<td>Mouth ulcers</td>
<td></td>
</tr>
<tr>
<td>Asymptomatic</td>
<td></td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td></td>
</tr>
<tr>
<td>Minor infections</td>
<td></td>
</tr>
<tr>
<td>Skin conditions</td>
<td></td>
</tr>
<tr>
<td>Fever</td>
<td></td>
</tr>
<tr>
<td>Weight loss</td>
<td></td>
</tr>
<tr>
<td>TB</td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td></td>
</tr>
</tbody>
</table>

The various stages of HIV infection depicting the development of different opportunistic infections with advanced immunodeficiency and the impact of antiretroviral therapy on CD4 cell count recovery.
psychosocial assessment

To be effective, the HIV clinician needs to have a sophisticated appreciation of the patient’s psychosocial status and context. People living with HIV come from a diverse mix of cultures, sexualities, social situations and genders. Successful management of HIV infection is shaped by varying degrees of stigma the patient attaches to HIV.

During the initial consultations a comprehensive psychosocial assessment should be conducted. General discussion should include the impact of HIV on the patient and the availability of social support systems. The patient’s priorities with regard to HIV disease, and his or her knowledge of HIV, need to be established before the discussion of treatment options and transmission prevention. Consideration of whether the patient’s regular partner is at risk of HIV infection should be discussed.

In addition to psychosocial issues related directly to HIV infection, the social stigma and marginalisation experienced by many of these groups compounds the psychological social and emotional impact of HIV infection.

Aboriginal and Torres Strait Islander, Maori and Cultural and Linguistically Diverse Communities

When working collaboratively with Aboriginal and Torres Strait Islander people the importance of respect for culture, local knowledge and community is essential. The purpose of the Anwernekenhe National Aboriginal and Torres Strait Islander HIV/AIDS Alliance (ANA) is to ensure Aboriginal and Torres Strait Islander communities are mobilised to protect themselves from infection with HIV and support their community (see http://ana.org.au/).

In New Zealand, INA provides an indigenous knowledge base and Māori world view services for Māori Indigenous and Pasifika peoples living with HIV (see http://www.ina.maori.nz/about-ina---hiv.html).

Clinicians working in the HIV and the viral hepatitis sector are encouraged to use the various multicultural HIV services to assist when managing culturally and linguistically diverse communities.

This is based on the principle that understanding culture and language is vital if individuals and communities from CALD backgrounds are to access health appropriately and equitably. Community participation and cultural input are central to better health for people from culturally diverse backgrounds (see http://www.mhahs.org.au/index.php).

Social, emotional and educational support is available through a variety of non-governmental organisations (NGOs), HIV-positive people’s groups and government agencies. Access to these groups differ around Australia. Not all CALD groups and groups of diverse sexualities and genders will be catered for. Clinicians may find themselves to be the primary support person for a variety of individuals (including MSM).

Key relationships and support systems are pivotal to the wellbeing of the patient. Assessment of family relationships and social support should be conducted. Explore whether family members are aware of the patient’s sexuality and HIV status, if this is the situation. Friends may also provide an invaluable support network.

It may be appropriate to discuss the patient’s financial situation and costs required so that these do not impede treatment initiation, follow-up or monitoring for adverse events.

Explore the patient relationship, and their disclosure of HIV infection. Patients who lack supportive and trusting relationships will be isolated and vulnerable, and referral to supportive services or relationship counselling may be appropriate (Chapter 16).

Use of drugs and alcohol should be explored in a non-judgmental way. Offering referral for assistance and specialised treatment may be appropriate. An accurate drug and alcohol assessment is essential before the commencement of antiretroviral therapy (ART) due to the possibility of significant drug interactions and poor adherence to ART.

Issues of family and children may be of particular concern. Fears about onward transmission, stigmatisation or future care may influence a couple’s decision whether to have children; education and discussion are advised as today these fears are generally unwarranted (Chapter 14).

Studies indicate a high prevalence of major depression and dysthymia among people with HIV infection; with particularly high rates among patients with fewer social supports and lower income. Depressive symptoms may affect the person’s ability to maintain safe sexual practice or may lead to suicidal ideation. The presence of mental health issues may negatively impact adherence to ART, poor diet/ reduced exercise, social withdrawal, and increased risk taking.

Recent acquisition of HIV may suggest the presence of mental illness such as depression, mood disorders or post-traumatic stress disorder. Drug and alcohol use or dependence on its own or interacting with mental illness may have played a similar role.

The primary care clinician should screen for mental health issues during the initial assessment and then opportunistically during the time the patient is in primary care. Mental health assessment should be considered when developing the GP Management Plan (discussed below) every 1-2 years. If screening suggests an issue then another consultation should be arranged to undertake a structured assessment and consideration for developing a Mental Health Plan that allows referral to a psychiatrist or a psychologist for further intervention.
### Table 10.1 Psychosocial assessment and management issues

<table>
<thead>
<tr>
<th>Self-esteem, self-awareness, identity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vulnerability and resilience</td>
</tr>
<tr>
<td>Family, social relationships, supports and connectedness to community</td>
</tr>
<tr>
<td>Individual cultural and linguistic diversity and the patient's gender and sexuality</td>
</tr>
<tr>
<td>Access to responsive health care and assistance</td>
</tr>
<tr>
<td>Ability to assert and articulate needs</td>
</tr>
<tr>
<td>Level of acceptance of HIV diagnosis</td>
</tr>
<tr>
<td>Presence of mental illness, depression, anxiety, mood disorders</td>
</tr>
<tr>
<td>Drug and alcohol use and dependency</td>
</tr>
<tr>
<td>Issues around pregnancy and parenthood for men and women</td>
</tr>
<tr>
<td>Financial and employment status and stability</td>
</tr>
<tr>
<td>Housing affordability and stability</td>
</tr>
<tr>
<td>Psychosocial assessment is a process that may take many consultations and is ongoing.</td>
</tr>
</tbody>
</table>

### Table 10.2 Checklist: initial assessment of the patient with HIV infection

| General assessment including medical history and social history which includes family history, relationship status, drug and alcohol history, smoking history, sexual history and risk assessment |
| Assessment of partner risk and contact tracing                                                      |
| Psychosocial and mental health assessment, focusing on the impact of the diagnosis and coping skills, pre-morbid psychosocial issues, and specific effects relating to stigmatisation and discrimination, reproductive intent and associated issues |
| Physical examination including weight, cardiovascular status, oral and dental health, skin, pelvic and ano-genital examination, and general systems examination |
| Sexual health review — screening for gonorrhoea and chlamydia (Chapter 8), syphilis serology, and hepatitis A, B, and C (HAV IgG, HBsAg, anti HBs, anti HBC, HCV Ab; and qualitative HCV RNA if CD4 < 200 cells/μL). Consider pregnancy testing. |
| Cervical pap smear                                                                                     |
| Consider screening in all patients for anal dysplasia and cancer (but the optimal mode of screening for anal dysplasia and cancer remains unclear), other than visual inspection of the anal canal and the perineum with biopsy of abnormal tissue. |
| Screens for infections such as toxoplasmosis (Toxoplasma IgG), CMV (CMV IgG), tuberculosis (Mantoux or QuantiFERON-TB Gold test and chest X-ray). |
| Blood tests including HIV RNA (viral load), CD4 cell count, CD4 cell percentage, fasting lipids and glucose, liver function tests, creatinine and electrolytes, full blood count (FBC), Vitamin D, eGFR, urine analysis, protein-to-creatinine ratio in spot urine samples |
| HIV resistance genotyping at entry into care, regardless of whether antiretroviral therapy will be initiated immediately |
| HLA-B*5701 testing                                                                                     |
| Vaccination history needs to be noted and future vaccinations discussed. The patient should be offered HBV and HAV vaccination in the absence of established immunity or infection. Live vaccination should be avoided in patients who are immunosuppressed or not currently taking antiretroviral therapy. Both Fluvax and Pneumovax are recommended in the NHMRC guidelines. |

### Case Study 1: Mpendwa

Mpendwa is a 36-year-old student from East Africa. She presents seeking treatment for a problem of vaginal discharge. Her English is limited. While her chlamydia urine PCR test is positive, unfortunately her HIV test is also positive. Her viral load is 40,000 copies/mL and her CD4 is 270 cells/μL. After discussion, she agrees to start antiretroviral therapy.

### Issues to consider

Communication: would you arrange an interpreter? How would you do this?

Disclosure and confidentiality: Mpendwa is at risk of significant stigma and discrimination, especially in a small ethnic community.

Beliefs and understanding: People from CALD backgrounds are likely to hold a range of contested understandings about HIV.

Access to care and services: How can Mpendwa afford to purchase antiretroviral therapy as she is not on Medicare?

Patient individual concerns: Is Mpendwa considering pregnancy or is she already pregnant? Will this influence her choice of antiretroviral therapy?

### Available resources

The local interpreter service.


Multicultural HIV and Hepatitis Service.


Local government services may be able to help with supply of medication.

DHHS Guidelines for the use of Antiretroviral Agents in HIV-1-Infected Adults and Adolescents: Considerations for Antiretroviral Use in Special Patient Populations - HIV-Infected Women (last updated 2/12/2013)

Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents. Chapter 6. Considerations for antiretroviral use in special patient populations. Women. Consult with a HIV clinician or a 100 prescriber.

Patients with significant immunosuppression may develop organic brain disease and consideration of mental health implications in this context is warranted. In addition, depressive, neuropsychological side effects may be caused by antiretroviral agents, most commonly efavirenz. The development of symptomatic and asymptomatic HIV-associated neurocognitive disorder (HAND) in patients with good immune function on long-term ART is noted and specialist referral ought to be considered.

### Medical assessment

Initial medical assessment involves the establishment of the stage of HIV disease and assessment of potential comorbidities. Baseline testing (Table 10.2) and a thorough clinical examination should be conducted in...
the context of a HIV diagnosis; the initial assessment may take place over several weeks or months and psychosocial issues may take priority at this time. Chapter 9 addresses how to deliver a positive result to a patient and the importance of early follow-up.

**CD4 cell count**

If HIV infection is untreated, progressive immune damage will occur, expressed as a loss of CD4 cells at an average rate of 60-80 cells/μL per year. Some patients may have a more rapid course, while others will remain stable for longer. There are levels of immune deficiency that are associated with greater risk of HIV-related conditions and opportunistic illnesses (Figure 10.2). For example, when the CD4 count falls to between 200 and 500 cells/μL, oral hairy leukoplaikia, skin conditions such as seborrhoeic dermatitis and psoriasis, recurrent varicella-zoster virus infection (shingles), and bacterial pneumonia may occur. CD4 counts below 200 cells/μL are associated with an increased risk of Pneumocystis jiroveci pneumonia (formerly PCP), cerebral toxoplasmosis, oesophageal candidiasis, Kaposi’s sarcoma and cryptosporidiosis. Advanced immunodeficiency occurs at CD4 cell counts below 50 cells/μL, at which stage the individual is at risk of cytomegalovirus (CMV) retinitis, disseminated mycobacterium avium complex (MAC) infection, cryptosporidiosis and microsporidiosis, primary central nervous system lymphoma and HIV-associated dementia and neurocognitive disorder and non-Hodgkin’s lymphoma. Opportunistic infections are discussed in greater detail in Chapter 6.

The CD4 count is calculated by the percentage of CD4 cells in the lymphocyte component of the white cell count. The total number of CD4 cells will vary according to the white cell count and lymphocyte count. It is important to assess changes in the total CD4 count in the context of the percentage of CD4 cells and the variability of the lymphocyte number secondary to intercurrent illness. A single measurement may be misleading because factors such as intercurrent infection, vaccination, menstrual cycle and the time of day blood is taken can affect results. Consequently, evaluation should focus on the trend in CD4 cell levels rather than a single result. A number of tests need to be performed over a period of time to provide an accurate picture of the patient’s immune function. These test results enable the clinician to form an assessment of the course of a person’s HIV disease and the rate of disease progression.

**Viral load**

The plasma viral load (the amount of HIV RNA in the plasma) is a measure of the balance between the amount of virus produced each day and the amount of virus cleared by the immune system. Viral load is measured by reverse transcriptase polymerase chain reaction (RT PCR). The laboratory will give results in both log number of copies/mL and absolute number of copies/mL. A significant change in viral load is an increase or decrease of greater than 0.5 log. Changes of less than 0.3 log are considered to be within the variability of the laboratory test performance. The lower limit of detection of the assay is currently 20 copies/mL, and viral loads below this level are reported as undetectable; however this does not mean that there is no HIV present.

Viral load is used to assist in making the decision to initiate ART (along with the presence or absence of symptoms and the level of CD4 cells), to monitor the response to ART and to identify treatment failure.

**Treatment**

A comprehensive assessment may require a number of consultations. A management plan with clear goals should be negotiated with the patient. The inevitability

<table>
<thead>
<tr>
<th>Table 10.3 Commonly used tests in HIV management</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Test</strong></td>
</tr>
<tr>
<td><strong>Comment</strong></td>
</tr>
<tr>
<td>HIV antibody test</td>
</tr>
<tr>
<td>Standard screening test, usually a combination HIV antibody / antigen test</td>
</tr>
<tr>
<td>HIV point of care test (HIV rapid test)</td>
</tr>
<tr>
<td>Saliva or blood test that can be performed at the clinic with a result in 10 to 20 minutes. Reactive tests are not diagnostic and must be confirmed with conventional laboratory testing</td>
</tr>
<tr>
<td>HIV Western blot</td>
</tr>
<tr>
<td>Confirmatory diagnostic test for HIV infection. Detects HIV antibodies</td>
</tr>
<tr>
<td>HIV p24 antigen</td>
</tr>
<tr>
<td>High during HIV primary infection</td>
</tr>
<tr>
<td>CD4 lymphocyte count</td>
</tr>
<tr>
<td>Marker of immune function:</td>
</tr>
<tr>
<td>Normal: &gt; 500 cells/μL</td>
</tr>
<tr>
<td>Immune deficiency: &lt; 500 cells/μL</td>
</tr>
<tr>
<td>Severe immune deficiency: &lt; 200 cells/μL</td>
</tr>
<tr>
<td>HIV RNA (viral load)</td>
</tr>
<tr>
<td>Marker of HIV in plasma, expressed as HIV copies/mL; high levels before treatment; should be very low or undetectable on treatment</td>
</tr>
<tr>
<td>HIV genotypic drug resistance assay</td>
</tr>
<tr>
<td>Test performed before treatment and if resistance to ART is suspected</td>
</tr>
</tbody>
</table>
and benefits of commencing ART should be broached very early. Immediate commencement of ART should be considered if the patient is judged to be in the early stages of HIV seroconversion (Chapter 4).

The goal of treatment is suppression of HIV replication to a level below detection. For treatment to be effective, the patient should achieve near to 100% adherence. HIV viraemia is associated with increased morbidity and onward transmission of risk to others.

Once commenced, ideally, antiretroviral therapy is lifelong. Treatment interruptions are associated with significant risks of increased morbidity and mortality. Adequate adherence to antiretroviral therapy is vital to avoid and delay drug resistance.

CD4 level can influence the timing and urgency of ARV initiation. If the CD4 count is low (< 200 cells/μL) then consider referring the patient to an HIV physician in a hospital-based practice. Discuss with a sexual health physician or HIV physician to ensure appropriate opportunistic infection prophylaxis, surveillance and intervention. The risk of the patient developing IRIS (Immune Reconstitution Inflammatory Syndrome) after immediate commencement of ART should be considered when the CD4 count at this time is very low. If the CD4 count is < 500 cells/μL then consider immediate commencement of antiretroviral therapy. Treatment should be comprehensively discussed and a plan put into effect, cognisant of all of the findings.

A more robust CD4 cell level may indicate more time for an open discussion around the timing of antiretroviral therapy initiation and opportunity to deal with significant psychosocial, mental health and drug and alcohol issues within the community setting, however treatment should be considered in all patients to mitigate the risks of transmission.

**The benefits of HIV treatments**

The natural history of HIV infection has been altered significantly by the use of potent combination antiretroviral therapy. Evidence suggests that the life expectancy of patients with HIV infection with CD4 counts > 500 cells/μL approaches the life expectancy of the HIV-negative population.

In recently published trials involving sero-discordant couples, reduction in viral load has been shown to significantly reduce the risk of onward transmission of the virus, irrespective of risk category or sexual positioning.

Maximal and durable viral suppression:

- preserves CD4 T-cell numbers
- delays or prevents viral resistance
- decreases inflammation and immune activation thought to contribute to higher rates of cardiovascular and other end-organ damage
- reduces perinatal and behaviour-associated onward transmission of HIV

**Initiating antiretroviral therapy**

**ART is recommended for all individuals with HIV infection to reduce the risk of disease progression.**

Currently there is strong evidence that initiating ART once an individual’s CD4 count has fallen below 350 cells/μL, or when an individual has developed HIV-associated illness, reduces morbidity and mortality. There is moderate evidence from observational studies that initiating therapy once the CD4 count has fallen below 500 cells/μL is associated with reduced morbidity and mortality.

There is limited evidence regarding the balance between the benefits and risks for commencing ART in an individual with a CD4 count > 500 cells/μL but treatment should be considered if the patient desires to commence ART, if there are concerns about onward transmission of HIV and if the patient is immunologically vulnerable (over 50 years of age, demonstrated rapid CD4 decline, and the presence of significant comorbidities such as symptomatic HIV-associated neurocognitive disorder, tuberculosis and chronic viral hepatitis).

The most appropriate regimen is based upon assessment of the simplest available, tolerable and effective ART regimen, according to current prescribing guidelines. The patient’s wishes, context, lifestyle, comorbidities and other medication ought to influence significantly the choice of the initial antiretroviral regimen. In women, antiretroviral regimen selection ought to consider reproductive intent and contraceptive use. Follow-up visits are planned to review the virological impact of antiretroviral treatment as well as to monitor toxicity and adherence. Patients should be warned about common side effects and the nature of significant reactions to medication (Table 10.4). Reviews should occur regularly after treatment initiation.

The key biological marker of successful ongoing response to therapy remains the viral load. Failure to achieve undetectable viral load after commencement of ART or a significant rise in viral load after achieving viral undetectability requires consideration of the following factors.
**TABLE 10.4 Monitoring and preventive activities**

<table>
<thead>
<tr>
<th>Feature</th>
<th>Monitoring /preventive activity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HIV specific</strong></td>
<td></td>
</tr>
<tr>
<td>History, examination</td>
<td>Every 3 to 6 months</td>
</tr>
<tr>
<td>CD4, HIV viral load</td>
<td>Every 3 to 6 months</td>
</tr>
<tr>
<td>Drug interactions</td>
<td>Check all current medicines at every visit</td>
</tr>
<tr>
<td>Adherence</td>
<td>Assess adherence at every visit</td>
</tr>
<tr>
<td>Side effects</td>
<td>Review side effects at every visit</td>
</tr>
<tr>
<td><strong>Co-infections and vaccinations</strong></td>
<td></td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>Vaccination (two-dose schedule) if at risk</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>Vaccination (three-dose schedule) all patients, unless immune or chronic infection</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>Check at baseline, annual check if at risk</td>
</tr>
<tr>
<td>Influenza</td>
<td>Annual vaccination</td>
</tr>
<tr>
<td>Pneumococcal</td>
<td>Vaccination, revaccination complex – see guidelines</td>
</tr>
<tr>
<td>Tetanus/pertussis</td>
<td>Vaccination every 10 years</td>
</tr>
<tr>
<td>Sexually transmitted infections</td>
<td>Screening depends on risk group – for MSM, 3 to 12 monthly screens</td>
</tr>
<tr>
<td>Human papilloma virus disease</td>
<td>Vaccination (three-dose schedule; not funded for adults)</td>
</tr>
<tr>
<td>Opportunistic infections</td>
<td>Antibiotic prophylaxis if CD4 &lt; 200 cells/μL, complex regimen – see guidelines</td>
</tr>
<tr>
<td><strong>Lifestyle</strong></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>Opportunistic screening and counselling</td>
</tr>
<tr>
<td>Weight</td>
<td>Check weight, BMI, waist circumference every 6 to 12 months</td>
</tr>
<tr>
<td>Nutrition</td>
<td>Review every 6 to 12 months</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Review every 6 to 12 months</td>
</tr>
<tr>
<td>Alcohol intake</td>
<td>Review every 6 to 12 months</td>
</tr>
<tr>
<td><strong>Cardiovascular/metabolic</strong></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular risk calculation</td>
<td>Calculate absolute risk every 2 years</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Check every 6 to 24 months</td>
</tr>
<tr>
<td>Lipids</td>
<td>Check fasting every year</td>
</tr>
<tr>
<td>Glucose</td>
<td>Check fasting every year</td>
</tr>
<tr>
<td>Renal</td>
<td>Check eGFR every 3 to 6 months if taking ART, every 6 to 12 months if no ART. Perform urinalysis at baseline and every 6 to 12 months if taking ART</td>
</tr>
<tr>
<td>Liver</td>
<td>LFT every 3 to 6 months if taking ART, every 6 to 12 months if no ART</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>Assess for risk factors every 12 months in women &gt; 45 years, men &gt; 50 years; consider BMD measurement</td>
</tr>
<tr>
<td><strong>Cancer screening</strong></td>
<td></td>
</tr>
<tr>
<td>Cervical (female)</td>
<td>Pap smear every year</td>
</tr>
<tr>
<td>Breast (female)</td>
<td>Mammogram every 2 years if aged 50 to 69 years</td>
</tr>
<tr>
<td>Colon</td>
<td>FOBT every 2 years if aged 50 to 75 years, or colonoscopy every 5 years if high risk – see guidelines</td>
</tr>
<tr>
<td><strong>Psychosocial</strong></td>
<td></td>
</tr>
<tr>
<td>Depression, mental illness</td>
<td>Opportunistic screening</td>
</tr>
<tr>
<td>Drug use</td>
<td>Opportunistic screening</td>
</tr>
<tr>
<td>Sexual/reproductive</td>
<td>Opportunistic screening</td>
</tr>
<tr>
<td>Housing, financial situation,</td>
<td>Opportunistic screening</td>
</tr>
<tr>
<td>social support</td>
<td></td>
</tr>
<tr>
<td><strong>Other prevention</strong></td>
<td></td>
</tr>
<tr>
<td>Dental health</td>
<td>Referral for regular dental care</td>
</tr>
<tr>
<td>Glaucoma /vision</td>
<td>Referral if at risk (e.g. family history) or low CD4</td>
</tr>
</tbody>
</table>

ART = antiretroviral therapy; BMD = bone mineral density; BMI = body mass index; eGFR = estimated glomerular filtration rate; FOBT = faecal occult blood test; LFT = liver function tests; MSM = men who have sex with men

Adapted from Baker D, Pell C, Donovan B.18
The patient may show poor adherence; changes to the treatment regimen may be required to improve tolerability or convenience.19

Drug levels may be too low to suppress HIV replication due to drug interactions or poor absorption, requiring dose adjustments or a change in regimen.17

Resistance to the antiretroviral drugs may have developed and resistance assays may be conducted by the antiretroviral prescriber. These tests must be interpreted in the context of the patient’s current and past antiretroviral history.30

Case Study 2

Alex

Alex is a 26-year-old man who acquired HIV through sex with men and women; he lives in Melbourne and works as a barman. He was diagnosed 3 years ago following a sexual health screen which also confirmed secondary syphilis. He was started on Truvada (tenofovir and emtricitabine) one tablet once daily and raltegravir 400 mg twice daily. At baseline his CD4 count was 325 cells/μL (23%) and his HIV viral load was 22,000 (log 4.34) copies/mL. He has no symptoms related to HIV and he feels that his health has improved to the extent that he has enrolled for college to study hospitality management. Blood tests have been consistently good; CD4 500 cells/µL and viral load < 50 (log 1.7) copies/mL until the last few months where he has had the following results:

4 weeks ago: CD4 510 cells/µL HIV viral load 107 (log 2.02) copies/mL
Repeat test today: CD4 504 cells/µL HIV viral load 64 (log 1.81) copies/mL

Issues to consider

- Are the viral load results suggestive of virological failure or is this a ‘blip’?
- Is adherence optimal? If not, why not? (Consider factors such as finances, drug and alcohol use)
- Is the treatment schedule optimal?
- Is there intercurrent illness?
- Consider possible drug interactions with prescribed or over-the-counter medication.

Available resources

- Consult with the s100 prescriber

Treatment regimens

Combination regimens are derived from six classes of antiviral drugs: nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs), non-nucleoside reverse transcriptase inhibitors (NNRTIs), protease inhibitors (PIs), fusion inhibitors (FIs), CCR5 antagonists, and integrase strand transfer inhibitors (INSTIs).

Standard treatment comprises a combination of three or four drugs (Tables 10.5 and 10.6). Generally a combination includes drugs from at least two different drug classes. Current preferred regimens include: two NRTIs and an NNRTI; two NRTIs and an INSTI; two NRTIs and a ritonavir-boosted PI.11 Consideration of reproductive intent, pregnancy status and contraceptive use in women should be given when selecting a regimen.10

Monitoring

Following the commencement of medication and the patient is stable (adequate compliance, tolerating regime, virus undetectable) a plan for ongoing routine care can be established. Patients will benefit from regular assessment of clinical state, immune function, viral load, sexual health, screening for the presence of co-morbidities and monitoring psychosocial wellbeing and healthy lifestyle. It is useful to develop this process as an annual cycle of care so that these issues can be addressed in a systematic and achievable way. Similar to managing other chronic diseases (such as diabetes and chronic kidney disease) tools available in general practice such as the GP Management Plan allow time and resources to facilitate this strategy.

Review needs will vary, more frequency if immunocompromised, initiating or switching ART. In general, the untreated immune-competent patient (CD4 cells > 500 cells/µL) and the patient on therapy with stable virological control and immune function should be reviewed every 3-6 months. More regular review is advised if adherence is uncertain, the patient’s

Table 10.5 Recommended first-line regimens for HIV treatment (Australia 2014)

<table>
<thead>
<tr>
<th>First-line treatment (Trade name)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Efavirenz/tenofovir/ emtricitabine (Atripla)</td>
<td>Co-formulated as a single pill; avoid in pregnancy</td>
</tr>
<tr>
<td>Atazanavir (Reyataz); ritonavir (Norvir); emtricitabine/tenofovir (Truvada)</td>
<td>Three tablets daily with food; avoid with proton pump inhibitors</td>
</tr>
<tr>
<td>Raltegravir (Isentress); emtricitabine/tenofovir (Truvada)</td>
<td>Three daily, raltegravir is closed twice daily</td>
</tr>
</tbody>
</table>

Adapted from Baker D, Pell C, Donovan B.18
Table 10.6. Antiretroviral agents available in Australia (February 2014)

<table>
<thead>
<tr>
<th>Class</th>
<th>Generic</th>
<th>Trade name</th>
</tr>
</thead>
<tbody>
<tr>
<td>NRTI/NtRTI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abacavir</td>
<td>Ziagen</td>
<td></td>
</tr>
<tr>
<td>Zidovudine</td>
<td>Retrovir</td>
<td></td>
</tr>
<tr>
<td>Stavudine</td>
<td>Zerit</td>
<td></td>
</tr>
<tr>
<td>Lamivudine</td>
<td>3TC</td>
<td></td>
</tr>
<tr>
<td>Didanosine</td>
<td>Videx</td>
<td></td>
</tr>
<tr>
<td>Emtricitabine</td>
<td>Emtriva</td>
<td></td>
</tr>
<tr>
<td>Tenofovir</td>
<td>Viread</td>
<td></td>
</tr>
<tr>
<td>Lamivudine/zidovudine</td>
<td>Combivir</td>
<td></td>
</tr>
<tr>
<td>Abacavir/lamivudine</td>
<td>Kivexa</td>
<td></td>
</tr>
<tr>
<td>Emtricitabine/tenofovir</td>
<td>Truvada</td>
<td></td>
</tr>
<tr>
<td>Abacavir/lamivudine/zidovudine</td>
<td>Trizivir</td>
<td></td>
</tr>
<tr>
<td>NNRTI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nevirapine</td>
<td>Viramune</td>
<td></td>
</tr>
<tr>
<td>Efavirenz</td>
<td>Stocrin</td>
<td></td>
</tr>
<tr>
<td>Etravirine</td>
<td>Intelence</td>
<td></td>
</tr>
<tr>
<td>Rilpivirine</td>
<td>Edurant</td>
<td></td>
</tr>
<tr>
<td>Protease inhibitors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saquinavir</td>
<td>Invirase</td>
<td></td>
</tr>
<tr>
<td>Indinavir</td>
<td>Crixivan</td>
<td></td>
</tr>
<tr>
<td>Ritonavir</td>
<td>Norvir</td>
<td></td>
</tr>
<tr>
<td>Atazanavir</td>
<td>Reyataz</td>
<td></td>
</tr>
<tr>
<td>Lopinavir/ritonavir</td>
<td>Kaletra</td>
<td></td>
</tr>
<tr>
<td>Fosamprenavir</td>
<td>Telzir</td>
<td></td>
</tr>
<tr>
<td>Tipranavir</td>
<td>Aptivus</td>
<td></td>
</tr>
<tr>
<td>Darunavir</td>
<td>Prezista</td>
<td></td>
</tr>
<tr>
<td>Entry inhibitors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enfuvirtide</td>
<td>Fuzeon</td>
<td></td>
</tr>
<tr>
<td>Maraviroc</td>
<td>Celsentri</td>
<td></td>
</tr>
<tr>
<td>Integrase inhibitors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Raltegravir</td>
<td>Isentress</td>
<td></td>
</tr>
<tr>
<td>Dolutegravir</td>
<td>Tivicay</td>
<td></td>
</tr>
<tr>
<td>Combination of classes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tenofovir/emtricitabine/efavirenz</td>
<td>Atripla</td>
<td></td>
</tr>
<tr>
<td>Tenofovir/emtricitabine/ritpivirine</td>
<td>Eviplera</td>
<td></td>
</tr>
<tr>
<td>Tenofovir/emtricitabine/elvitegravir/cobicistat</td>
<td>Striibild</td>
<td></td>
</tr>
</tbody>
</table>

NRTI = nucleoside reverse transcriptase inhibitor; NtRTI = nucleotide reverse transcriptase inhibitor; NNRTI = non-nucleoside reverse transcriptase inhibitor

Adapted from Baker D, Pell C, Donovan B.18

Circumstances lack stability or significant comorbidities are present.

Regular screening for common comorbidities is essential. Most of these comorbidities are not specific to HIV and include cardiovascular disease, type 2 diabetes, chronic kidney disease, osteoporosis, cervical cancer,10 basal cell cancer, anal cancer (see below), and non-Hodgkin’s lymphoma,32 but they occur earlier and more commonly in the setting of chronic HIV infection.33-36 Some co-morbidities are linked with prolonged immunosuppression, ongoing inflammation, and longevity of infection associated with HIV infection.33-36 These may be associated with HIV therapies (Table 10.6) or the usual behavioural factors such as smoking and activity levels. Comorbidities usually occur earlier in the HIV-positive person’s life than would otherwise be expected. Screening and treatment are usually no different than that for HIV-negative individuals, though there are often special factors to be considered in the HIV-positive group, e.g. screening for osteoporosis earlier,36,38 and annual cervical cancer screening.10 If these comorbidities are diagnosed the HIV prescriber should be informed and involved in decisions regarding appropriate interventions.

There are a number of models of care for the clinical management of HIV infection from the USA, the UK and Europe. Online access to Models of Care can be obtained by consulting the ASHM website at: http://www.ashm.org.au/default2.asp?active_page_id=182

Anal cancer, a relatively rare cancer, is seen 40-fold in MSM and 120-fold in HIV-positive MSM, especially in those who smoke and with low CD4 cell counts. As with cervical cancer,10 anal cancer is related to infection
by human papillomavirus (HPV) types 16 and 18. It is not listed as an AIDS-defining cancer, although cervical cancer is. Like cervical cancer, early detection confers a treatment outcome benefit. The early diagnosis requires routine examination by visual inspection, digital rectal examination (DRE) and proctoscopy (with biopsy of suspicious lesions). This examination is recommended at least annually and more often in patients reporting symptoms.\textsuperscript{12,13} Similar examination is recommended at least annually and proctoscopy (with biopsy of suspicious lesions). This diagnosis requires routine examination by visual confers a treatment outcome benefit. The early diagnosis of cervical cancer is. Like cervical cancer, early detection is not listed as an AIDS-defining cancer, although infection by human papillomavirus (HPV) types 16 and 18. It is not listed as an AIDS-defining cancer, although cervical cancer is. Like cervical cancer, early detection confers a treatment outcome benefit. The early diagnosis requires routine examination by visual inspection, digital rectal examination (DRE) and proctoscopy (with biopsy of suspicious lesions). This examination is recommended at least annually and more often in patients reporting symptoms.\textsuperscript{12,13} Similar examination of women with HIV should be considered as part of routine annual cervical testing. As in women, routine HPV vaccination of young males is expected to reduce the incidence of anal cancer in the next generation of MSM.\textsuperscript{13}

CASE STUDY 3 Richard

Richard is a 68-year-old married business man who was diagnosed HIV positive by his general practitioner 8 years ago. His baseline CD4 count was 750 cells/μL (25%) but he has had a steady decline over the last few years. He is sexually active with other men. He has a number of concurrent medical conditions and he has regular appointments with his general practitioner. His concurrent medical conditions all predate HIV acquisition and they are: rheumatoid arthritis on methotrexate and NSAIDS; diabetes on metformin and gliclazide; total hip replacement; anxiety. His baseline investigations reveal:

- CD4: 360 cells/μL; HIV viral load: 25,000 copies/mL; HIV genotype: never done
- HLA-B5701 gene test: negative
- eGFR =80, U/A + protein + blood
- Hepatitis A,B,C, chlamydia, gonorrhoea all negative; syphilis = EIA positive, RPR positive;
- Chest X-ray: normal
- Hb 88 LFT Normal

Issues to consider

- Significant immunosuppression – will benefit from initiating ART immediately
- Complex concurrent medical and poly-pharmacy conditions require communication between involved clinicians
- Requires treatment of early infectious syphilis
- Sexual health and risk behaviour

Available resources

- Consult with s100 HIV prescriber
- ART initiation guidelines - Australian Commentary DHHS guidelines. September 2013\textsuperscript{18}
- Liverpool University Drug Interaction website - www.hiv-druginteractions.org

Women with HIV

There are significant numbers of women in Australia from a variety of social and cultural groups with HIV. In some areas of Australia there are no specific support services for women. For women with HIV infection, issues of family and children may be of particular concern. Fears about onward transmission, stigmatisation, or future care may influence the decision to have children, and specific education and discussion are warranted. Reproductive intent, contraceptive use and pregnancy status will influence the appropriate choice of antiretroviral therapy. Screening for anal dysplasia and cancer for women should be performed at appropriate intervals. Pelvic examination is a routine part of initial and regular assessment of women with HIV. Pregnancy testing should be considered with a variety of presentations. (See Tables 10.4 and 10.7)

Cancer of the cervix is an AIDS-defining illness.

Consider consulting the following guidelines as needed. Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents. Chapter 6. Considerations for antiretroviral use in special patient populations. Women. Bethesda: Department of Health and Human Services (DHHS), 12 February 2013.\textsuperscript{19}


A team-based approach to HIV care provides a better more integrated pathway of treatment and care for those with HIV infection and those affected. There are many different services that may be accessed to better support HIV patients and their partners, families and friends. Pharmacist can assist with home medicines reviews. Psychologists and counsellors can support the patient by mental health care plans. Dieticians, psychologists, counsellors, Aboriginal support workers, Maori and Pasifika support workers and social workers all have potentially essential contributions to make to best care of people living with HIV. Registered
**TABLE 10.7 Common interactions between antiretroviral drugs and other drugs***

<table>
<thead>
<tr>
<th>Drug class</th>
<th>Interaction with antiretroviral drug</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiarrhythmic, e.g. amiodarone</td>
<td>↑ with PI, cobicistat</td>
<td>Avoid co-administration</td>
</tr>
<tr>
<td>Antihypertensive: calcium channel blockers beta-blockers</td>
<td>↑ with PI, cobicistat; ↓ with NNRTI ↑ with cobicistat</td>
<td>Dose adjustment Dose adjustment</td>
</tr>
<tr>
<td>Anticonvulsants: carbamazepine, phenobarbital, phenytoin</td>
<td>↓ dolutegravir, elvitegravir, etravirine, lopinavir, rilpivirine</td>
<td>Avoid co-administration; take care when administering with other PIs</td>
</tr>
<tr>
<td>Acid-lowering drugs: proton pump inhibitors H2-receptor antagonists antacids</td>
<td>↓ atazanavir, rilpivirine ↓ atazanavir, dolutegravir, elvitegravir, rilpivirine</td>
<td>Avoid co-administration Complex dosing, see guidelines Take at different times</td>
</tr>
<tr>
<td>Antidepressants, most</td>
<td>↑ with PIs, cobicistat</td>
<td>Start with low dose and titrate carefully</td>
</tr>
<tr>
<td>Benzodiazepines: midazolam, triazolam</td>
<td>↑ with most ART</td>
<td>Avoid</td>
</tr>
<tr>
<td>Bronchodilator: salmeterol</td>
<td>↑ with PIs, cobicistat</td>
<td>Avoid co-administration</td>
</tr>
<tr>
<td>Corticosteroids: inhaled or intranasal fluticasone, budesonide</td>
<td>↑↑ With ritonavir, cobicistat. Can result in adrenal insufficiency, including Cushing's syndrome</td>
<td>Avoid co-administration; use beclomethasone instead</td>
</tr>
<tr>
<td>Corticosteroids: systemic, including articular injections</td>
<td>Complex, risk of ↓ ART and ↑ corticosteroid</td>
<td>Use with care, prednisone is safest</td>
</tr>
<tr>
<td>Gout: colchicine</td>
<td>↑ with PI, cobicistat</td>
<td>Reduce colchicine dose</td>
</tr>
<tr>
<td>Hormonal contraceptives</td>
<td>↓ with PI, NNRTI, cobicistat</td>
<td>Complex – consult guidelines</td>
</tr>
<tr>
<td>Lipid lowering: simvastatin, lovastatin atorvastatin, rosuvastatin</td>
<td>↑↑ with PIs, cobicistat ↑ with PIs cobicistat</td>
<td>Avoid, use other statins Use low dose</td>
</tr>
<tr>
<td>Migraine: ergotamine</td>
<td>↑↑ with most ART</td>
<td>Avoid</td>
</tr>
<tr>
<td>Narcotics: methadone buprenorphine</td>
<td>↓ with nevirapine, efavirenz ↑ with atazanavir (un-boosted)</td>
<td>May need methadone dose increase Avoid co-administration</td>
</tr>
<tr>
<td>Phosphodiesterase type 5 (PDE5) inhibitors e.g. sildenafil</td>
<td>↑ with PI, cobicistat</td>
<td>Start with low dose of PDE5 inhibitor</td>
</tr>
<tr>
<td>Stimulants such as MDMA (ecstasy)</td>
<td>↑ with ritonavir, cobicistat</td>
<td>Warn patients of the risk</td>
</tr>
<tr>
<td>St John's wort</td>
<td>↓ level of many ART</td>
<td>Avoid</td>
</tr>
</tbody>
</table>

* * Does not include interactions with other antiretroviral drugs or more specialised drugs such as those used to treat tuberculosis or hepatitis C

Adapted from Baker D, Pell C, Donovan B.18

Nurses can assist as part of community care teams and within primary and tertiary care services. Registered Nurses are particularly valuable within general practices and can help the general practitioner to coordinate all of these services over a 12-month cycle (Table 10.4) within general practice management plans. New opportunities such as electronic health records and health pathways may improve patient outcomes.

**Summary**

Given that people living with HIV will require lifelong therapy, and that as they age, these people will develop the usual age-related morbidities, the involvement of primary care clinicians in their care is crucial. While monitoring of HIV infection should focus on the stability of clinical, immunological or virological markers, the patient benefits from monitoring for a
range of non HIV-specific morbidities. More than ever, generalist and HIV specialist clinicians need to coordinate their management and monitoring to ensure the best health outcomes for people living with HIV. Agreement around treatment goals, monitoring patterns, and coordination of care will best be facilitated by regular communication between clinicians and the use of tools such as an annual cycle of care.

The key to successful management of HIV infection is empowerment of the central figure in this process - the patient. An effective therapeutic relationship grounded in an appreciation of the patient’s spiritual, cultural and social context, based on honesty, expertise and respect will deliver the best outcomes.

References

1. ASHM, NSW shared care update powerpoint presentation. February 2014


CHAPTER 11 PRIMARY CARE MANAGEMENT OF CHRONIC HEPATITIS C

2014 REVIEW

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Introduction

A primary care role in the management of chronic hepatitis C involves the provision of information, support and referral as well as initial and ongoing clinical assessment and monitoring. The primary care clinician may undertake tasks such as specific diagnosis and initial assessment of the severity of disease, counselling the patient about the current understanding of the disease process and potential complications, as well as general issues of diet, mental health, lifestyle, transmission and vaccination.1,2 With recent advances in the treatment of hepatitis C, the primary care clinician has an important role in presenting the patient with specific treatment options and potential side effects.3 This chapter focuses on the primary care management of chronic hepatitis C.

Initial assessment

Patients with positive anti-HCV should be evaluated for the presence of viraemia with an HCV RNA (qualitative) test, as some may have cleared infection spontaneously. The HCV RNA test is rebatable under Medicare for this indication. Patients found to be HCV RNA negative should be reassured that while they have probably been exposed to HCV in the past, they have apparently cleared infection. It is recommended that such patients have their liver enzymes and HCV RNA rechecked one year after initial evaluation; if HCV RNA remains negative and liver enzymes are normal, no further follow-up is necessary. Patients with viraemia should be assessed further.

A detailed history should include an estimation of the duration of exposure, age at infection and whether there are important contributing factors to hepatic fibrosis. These factors may include a history of significant alcohol consumption, viral co-infection and presence of obesity, insulin resistance and diabetes, which are risk factors for non-alcoholic fatty liver disease and may also influence response to antiviral therapy. Concomitantly, the patient should be evaluated for ongoing risks, such as injecting drug use and excessive alcohol consumption.

Initial assessment of a patient with hepatitis C should address whether or not the patient has active or inactive disease, as well as his or her likelihood of having significant fibrosis. Patients are more likely to have significant fibrosis if they have had a long duration of infection (>20 years), acquired infection at an older age, have a history of significant alcohol use (which may be remote), or have been obese or have insulin resistance or diabetes. Chapter 7 discusses virological markers, liver function tests, liver imaging, liver biopsy and other investigations including non invasive methods to assess fibrosis, which form the basis of this initial assessment.

Patients should have liver enzymes monitored every 2 to 3 months for several months to establish whether enzymes are persistently abnormal, persistently normal or fluctuating. It should be kept in mind that while patients with persistent elevation of alanine aminotransferase (ALT) levels are at higher risk of significant liver damage and disease progression, even patients with normal liver enzymes may be at risk of progressive disease.

Table 11.1 Interpretation of HCV specific investigations

<table>
<thead>
<tr>
<th>Marker</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>anti-HCV</td>
<td>Current or past infection. Does not provide immunity against re-infection</td>
</tr>
<tr>
<td>HCV RNA (qualitative)</td>
<td>Circulating virus* indicating current infection</td>
</tr>
<tr>
<td>HCV RNA (quantitative)</td>
<td>Level of circulating virus (represented in log value or IU/mL)</td>
</tr>
<tr>
<td>HCV genotype</td>
<td>Influences treatment type and duration</td>
</tr>
<tr>
<td>Interleukin (IL)-28B polymorphism</td>
<td>Predictor of treatment response to interferon-based therapy</td>
</tr>
</tbody>
</table>

*May be useful in high-risk HCV antibody-negative patients

Patients with detectable HCV RNA, regardless of whether the ALT level is normal or abnormal, may be considered for antiviral therapy; however, if they choose not to be treated at the time they should be followed every 6 to 12 months. Regular follow-up of

KEY POINTS

- Antiviral treatment should be considered for all patients with chronic hepatitis C.
- All patients with chronic hepatitis C should be assessed for severity of liver disease.
- Comorbidities, such as alcohol consumption, obesity, diabetes and viral co-infection are important factors in disease progression and should be addressed in all patients. Effective antiviral therapy is available for many patients with chronic hepatitis C.
- Depending on viral genotype and other factors, 30–80% of patients have a sustained response to currently approved hepatitis C treatments.
- New treatments will soon be available offering improved response rates and tolerability.
patients with hepatitis C allows for monitoring of flares of activity, assessment of disease progression, discussion of current therapies and an opportunity to encourage minimisation of concomitant risk factors for fibrosis. See Table 11.1 for a summary of serological and virological markers of HCV infection.

**Ongoing monitoring of patients with chronic hepatitis C**

The aims of follow-up in patients with chronic hepatitis C are to:

- Reinforce the need for lifestyle changes
- Decide which patients are appropriate for antiviral therapy
- Determine appropriate timing of referral to a specialist
- Monitor patients with cirrhosis for complications such as hepatic decompensation and hepatocellular carcinoma (HCC).

For patients with chronic hepatitis C, ongoing monitoring is recommended every 6 to 12 months, unless there are specific reasons for more frequent monitoring (e.g. encouraging behaviour change). Tests to be conducted may include:

- Liver enzymes
- Full blood count (to monitor platelet count in particular)
- Blood glucose (there is an increased incidence of diabetes in patients with chronic HCV infection)
- Prothrombin time or international normalised ratio (INR) in patients with cirrhosis
- Six monthly liver ultrasound to screen for HCC in patients with cirrhosis
- Six monthly serum alpha-fetoprotein (AFP) to screen for HCC in patients with cirrhosis
- Consideration of referral to a specialist unit for annual transient elastography (see below).

**Assessment for antiviral therapy**

Previously in Australia, antiviral therapy was funded only for patients with significant hepatic fibrosis. However, with increasing data to support the efficacy of antiviral therapy, it is now available to any adult with compensated liver disease irrespective of ALT level or stage of hepatic fibrosis. Pegylated interferon alfa-2b plus ribavirin is also approved for children and adolescents weighing more than 27 kg.

Any patient with chronic hepatitis C should be advised of the potential benefits of antiviral therapy, and much of the assessment should be related to appropriate timing of therapy. Patients with documented or suspected advanced fibrosis (severe fibrosis, cirrhosis), or high risk of disease progression should be encouraged to consider therapy as soon as practicable. For other patients, timing of treatment can be based on other lifestyle issues such as work, social circumstance, control of substance abuse and desire for pregnancy.

The anticipated new era of treatment for chronic hepatitis C will include shorter more effective treatment regimens that can eliminate the need for interferon therapy. In patients with contraindications to the use of interferon, deferral of treatment may be appropriate.

When evaluating current disease severity and risk of progression to fibrosis and cirrhosis, clinical examination should be conducted (Chapter 7) and the investigations listed above should be performed. The finding of an elevated ALT level indicates the presence of...
necroinflammatory activity but is not predictive of cirrhosis or significant fibrosis. Elevated bilirubin, prolonged INR, hypoaalbuminaemia or thrombocytopenia all suggest the presence of cirrhosis with some degree of hepatic decompensation and portal hypertension. However, patients with well compensated cirrhosis due to hepatitis C may have a completely normal bilirubin, INR, serum albumin level and platelet count for many years. Hepatic ultrasound may show features of cirrhosis or fatty infiltration but is commonly normal.

While liver biopsy remains the gold standard for confirming or excluding significant fibrosis, it is less frequently used in the setting of pretreatment assessment for HCV since the advent of non-invasive fibrosis assessment tools. One such tool is transient elastography (Fibroscan®) which is based on the principle of elastic shear-wave propagation through the liver, with the velocity of the shear wave correlating with liver stiffness, and thus fibrosis. Fibroscan can be performed in an outpatient clinic setting, and is complete within 5 to 10 minutes. There are limitations to performance of transient elastography, namely the presence of ascites, right heart failure, extrahepatic cholestasis or obesity, although the use of a different probe (XL) enables deeper penetration at a lower frequency and improved outcomes in obese patients. The interpretation of the acquired elastography score (expressed in kPa) does depend on the aetiology of the underlying condition as well as the degree of hepatic necroinflammation, reflected by ALT.

Further non-invasive fibrosis assessment tools include the use of serum biomarkers, including simple tests such as AST/ALT ratio and platelet count, and panels of fibrosis markers, such as Hepascore (often used in South Australia and Western Australia).

There are circumstances when liver biopsy remains necessary to further assess the underlying stage of chronic liver injury, such as conflicting biochemical, imaging and elastography results, or consideration of concomitant aetiologies. Liver biopsy is a relatively safe procedure and is usually performed as a day-stay procedure, under ultrasound guidance using local anaesthetic only. Patients commonly experience some minor abdominal discomfort and right shoulder tip pain but severe pain is unusual. There is a small risk of significant bleeding (1:300) and death (1:10,000).

There are several systems in use for recording the degree of fibrosis in a liver biopsy. Most of these systems use a scoring system ranging from 0 (no fibrosis) to 4 (definite cirrhosis). The finding of minimal disease activity and no fibrosis (stage 0) or mild fibrosis (stage 1) suggests a low likelihood of disease progression. Consequently, the patient may be reassured, and therapy deferred. A similar recommendation can be made on the basis of hepatic elastography (< 7.5 kPa = F0/1) and treatment may be avoided.

Commencement of antiviral therapy should be considered for all willing patients with compensated chronic liver disease related to HCV, providing there are no contraindications. Consideration of duration of infection is also important in the assessment of disease severity, rate of progression and need for treatment.

Viral genotype affects the length and type of treatment and predicted response. It is useful to check genotype early in the course of HCV assessment and before specialist referral, although it is not essential.

In determining whether a patient is appropriate for antiviral treatment, the primary care clinician may also consider the patient's social support and whether he or she is likely to adhere to treatment. The current dependence on interferon in treatment regimes in Australia has a major influence on decisions regarding patient suitability and willingness for treatment. In the coming years, interferon-free therapies are likely to make antiviral treatment accessible to many more patients.

Local hepatitis C councils or drug user groups may provide information and peer support for people considering treatment (Chapter 16).

**Shared care and referral**

The primary care clinician has an important role in assessing which patients with chronic hepatitis C should be referred for specialist review. Such patients include those who wish to undergo antiviral treatment, those with persistently elevated ALT levels, those with clinical or laboratory features suggestive of cirrhosis, and those who request specialist evaluation. Liver clinics usually offer additional services that may be of benefit to patients. Such services include clinical nurse consultants, psychologists, psychiatrists, social workers and dieticians.

Table 11.2 outlines investigations to conduct before referral. Referral to a liver clinic or hepatitis specialist can be made at any time. Ongoing support and management of the patient on treatment may be conducted by primary care clinicians and specialists in a shared-care setting.

**TABLE 11.2 Pre-referral investigation check-list**

<table>
<thead>
<tr>
<th>Investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver enzymes (usually 2-3 tests are conducted over 6 months)</td>
</tr>
<tr>
<td>HCV serology (anti-HCV)</td>
</tr>
<tr>
<td>HBV serology (HBsAg, anti-HBs, anti-HBc)</td>
</tr>
<tr>
<td>HIV serology</td>
</tr>
<tr>
<td>FBC, electrolytes, creatinine, bilirubin, coagulation studies (INR or APTT)</td>
</tr>
<tr>
<td>Thyroid function tests</td>
</tr>
<tr>
<td>Fasting blood sugar levels and lipids</td>
</tr>
<tr>
<td>Alpha-fetoprotein</td>
</tr>
<tr>
<td>Liver ultrasound</td>
</tr>
</tbody>
</table>

FBC: full blood count  
INR: international normalised ratio  
APTT: activated partial thromboplastin time
General management issues for patients with hepatitis C

Discussion about routes of viral transmission

Patients with hepatitis C will commonly be concerned about the risks of transmitting the infection to others. Issues regarding blood-borne transmission, sexual transmission and mother-to-child transmission should all be discussed.

Hepatitis C is transmitted primarily through blood-to-blood contact. The sharing of grooming tools that can cause skin abrasion (such as razors, toothbrushes and tweezers) should be avoided. Injecting drug users must be encouraged to use sterile water, needles and syringes, as well as new injecting equipment such as spoons, filters and tourniquets each time they inject (Chapter 3 and Appendix 4).

Patients may be concerned about sexual transmission of HCV. There appears to be a very low risk of sexual transmission of HCV between heterosexual partners. In contrast, recent increases in acute hepatitis C among HIV-positive men who have sex with men (MSM) have been reported from a number of countries. These cases have been predominantly associated with sexual (per mucosal) transmission rather than injecting drug use and may relate to both biological factors (e.g. higher HCV viral loads in HIV co-infection) and sexual behaviour (e.g. group sex) or behavioural factors.

There is a risk of transmitting HCV from mother to child, although the risk is low (approximately 5%). This risk is significantly higher if the mother has HIV-HCV co-infection. Currently, there is no indication for elective caesarean section in mothers with HCV infection. There is some evidence that prolonged rupture of membranes and use of invasive foetal monitoring may increase the risk of mother-to-child transmission of HCV, and decisions about intervention may need to be made on a case-by-case basis. Breastfeeding is not generally considered to present an additional risk of HCV transmission. However, breastfeeding should be suspended if the nipples are cracked or if the baby has cuts in or outside the mouth.

Risk of transmission and infection are discussed in detail in Chapter 5. Communication of safer sex and safe injecting messages is covered in Chapter 3 and Appendixes 3 and 4.

Lifestyle issues

The possibility of lifestyle modification needs to be discussed with the patient, particularly in relation to alcohol consumption and recreational drug use, as well as weight loss and physical activity.

Alcohol intake should be minimal. There is strong evidence that heavy alcohol consumption (> 50 g/day) leads to accelerated disease progression and a poorer response to current treatments. Australian guidelines recommend that people with viral hepatitis should drink alcohol infrequently or at low levels, and should consider not drinking at all. Specific strategies are set out in Table 11.3. People with cirrhosis should be encouraged to stop drinking alcohol completely as ongoing alcohol is a major determinant of disease progression and complications.

TABLE 11.3 How to reduce alcohol consumption

<table>
<thead>
<tr>
<th>Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plan at least two alcohol-free days per week</td>
</tr>
<tr>
<td>Switch to low alcohol or alcohol-free drinks</td>
</tr>
<tr>
<td>Avoid situations where there will be pressure to drink, e.g. rounds at the pub</td>
</tr>
<tr>
<td>Alternate non-alcoholic and alcoholic drinks</td>
</tr>
<tr>
<td>Drink a daily maximum of two drinks</td>
</tr>
</tbody>
</table>

Some particular issues arise in regard to people who inject drugs. Ongoing injecting drug use, particularly frequent use, is associated with significant risk of drug-related harm including infectious complications and drug overdose. Thus, HCV treatment assessment needs to be undertaken within the context of assessment of other health and social issues related to ongoing injecting. Referral to drug treatment services and support groups may be appropriate. Many people who inject drugs, particularly those with less frequent use or those who are stable injectors, or on stable drug dependency treatment programs, will be suitable for HCV treatment. All people who inject drugs should be counselled regarding the risk of HCV re-infection after successful viral clearance if injecting risk behaviour continue. With the introduction of short-duration, interferon-free antiviral treatments in the future, HCV treatment for people who inject drugs should become increasingly feasible, and could provide both individual and broader benefits (through HCV treatment as prevention).

Nutrition

There is considerable, unsubstantiated dietary information and advice directed at people with chronic viral hepatitis. For most people with hepatitis C, dietary recommendations are the same as for the general population (encouraging: grilled rather than fried food; lean meats and fish; reduced-fat products; wholesome bread; vegetables and fruit; pasta; minimisation of fat for spreading and cooking). Restrictive diets are not recommended in the absence of clear indications. People with advanced liver disease, or other conditions such as coeliac disease or diabetes, may be referred to a specialist dietitian for further advice.

Overweight or obese patients should be advised of a gradual weight-reduction program, particularly as there is increasing evidence of interaction between HCV, obesity and type 2 diabetes in accelerating...
progression to fibrosis, as well as affecting response to treatment. Those who may have fatty liver need to avoid a precipitous drop in weight as this can induce deterioration in liver function.

Many people with hepatitis C report nausea and intolerance to certain foods and drinks. Referral to a diettian may be appropriate to ensure the patient is consuming necessary vitamins and minerals.

Patients with advanced liver disease who develop protein-calorie malnutrition should be seen by a specialist diettian. Such patients often require protein supplementation, and should be encouraged to eat high-energy foods frequently throughout the day. Patients with advanced liver disease should not be subjected to protein restriction as their protein requirements are high.

Fatigue and other symptoms
People with chronic hepatitis C may report fatigue, mental slowing, malaise, headache, rash and aching muscles and joints. Recently, improvement of neurocognitive function following achievement of viral eradication has been suggested as a potential additional benefit of successful antiviral therapy.9 Consideration should be given to specific food and drinks that may be triggering symptoms, as well as to work, family or other commitments, which may exacerbate stress and fatigue. Patients may benefit from planning rest periods during the day or incorporating light-to-moderate exercise into their routines to reduce fatigue.

Vaccination
Chronic co-infection with more than one hepatitis virus may be associated with more severe liver disease. Superinfection with hepatitis A virus (HAV) or hepatitis B virus (HBV) in a patient with chronic hepatitis C may precipitate the development of acute liver failure. In the long term, patients with HBV and HCV co-infection tend to be more likely to progress to cirrhosis and to develop HCC. Thus, HAV and HBV vaccination should be offered to all patients with chronic hepatitis C (Chapter 5).

Psychosocial support
Patients may experience social isolation, anxiety or discrimination related to infection with viral hepatitis, which may be compounded by physical symptoms. The primary care clinician can begin by listening to the patient and demonstrating sensitivity to linguistic and cultural differences, which may affect an individual’s response to viral hepatitis. Provision of verbal and written information relating to transmission or disease natural history may allay fears (Chapter 16). Referral to counselling or support services may be indicated for patients with complex emotional, family and relationship or disclosure issues. All patients should be made aware of services such as counselling and support groups, telephone helplines and community organisations.

Information about services is available from any teaching hospital liver clinic or the local hepatitis C council (Chapter 16).

Complementary therapies
There is little evidence that complementary therapies including herbal medicines have a significant antiviral effect despite many patients reporting some symptomatic improvement and the ability of some agents to induce a fall in ALT.10,11 Most preparations are safe but some have reported hepatotoxicity and should be avoided (e.g. mistletoe, valerian, heliotropium, kombucha tea). Close monitoring of liver biochemistry is recommended at the commencement of any herbal medicine.

Antiviral therapy for hepatitis C
Aims of treatment
There are a number of aims of antiviral therapy in chronic viral hepatitis. These include:
• Eradication of infection
• Prevention of disease progression
• Improvement in liver histology
• Improvement of symptoms
• Decrease transmission risk
• Reduction in risk of liver cancer
• Improved survival

All these aims can be achieved in a significant proportion of patients with hepatitis C with currently available therapies.

Antiviral therapy
The major aim of treatment is to achieve viral eradication. In hepatitis C, viral eradication is defined by the achievement of a sustained virological response (SVR); that is, negative HCV RNA by a sensitive qualitative test 6 months after the completion of therapy. The specific antiviral treatment protocol is currently determined by the hepatitis C genotype (1–6) a person has, the presence or absence of cirrhosis and whether there have been previous attempts at treatment.

Until recently, the standard of care for all HCV genotypes was a combination of once-weekly subcutaneously administered pegylated interferon plus twice-daily oral ribavirin. The addition of an oral NS3 protease inhibitor, boceprevir or telaprevir, to dual combination therapy has substantially improved antiviral treatment for genotype 1, which traditionally has been the most difficult genotype to eradicate.
Therefore the current standard of care for HCV genotype 1 is triple therapy with a protease inhibitor, pegylated interferon and ribavirin. For the remaining genotypes the most effective, available therapy continues to be pegylated interferon and ribavirin, for 6 to 12 months. These treatment combinations are available in Australia under Section 100 (s100) of the Pharmaceutical Benefits Scheme (PBS). New treatment regimens are emerging rapidly however, and more effective and less toxic direct-acting antivirals (DAA), either in combination with pegylated interferon and ribavirin, or as interferon-free; all oral combinations will emerge as the standard of care in coming years. The European Association for the Study of the Liver (EASL) has produced online recommendations for HCV treatment in the next few years, which take into account that availability and access to new therapies will vary markedly around the world for some years to come.

At present, the most significant pretreatment predictor of treatment response to current therapies is the HCV genotype. The expected SVR rate in patients with genotype 2 or 3 infection is 70-80% after six months of combination pegylated interferon and ribavirin, while traditionally genotypes 1 and 4 have had lower SVR rates of 40 to 50%. The addition of a protease inhibitor to pegylated interferon and ribavirin in patients with HCV genotype 1 has enhanced SVR rates for treatment naive patients to as high as 79%. While HCV genotype is the most powerful pretreatment predictor of response, other favourable predictors of SVR include low viral load, minimal hepatic fibrosis, female gender, age (younger than 40 years), low to normal body weight and the CC interleukin (IL)-28B polymorphism.

The speed that HCV RNA becomes undetectable on treatment is a major factor in predicting SVR. By monitoring on-treatment response, patients can be counselled as to their likelihood of viral eradication. Patients who have undetectable HCV RNA at week 4 (termed a rapid virological response) have approximately a 90% chance of SVR regardless of the regimen used and may have their treatment duration shortened (unless they have cirrhosis or previously failed to respond to antiviral treatment). Stopping rules are used to curtail treatment in those with a very low likelihood of achieving an SVR and serve to prevent ongoing exposure to treatment-related side effects, emergence of antiviral resistance (an issue with protease inhibitors) and to reduce the cost of treatment.

**Table 11.4:** Treatment considerations with boceprevir and telaprevir

<table>
<thead>
<tr>
<th></th>
<th>Boceprevir</th>
<th>Telaprevir</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tablet size</strong></td>
<td>200 mg</td>
<td>375 mg</td>
</tr>
<tr>
<td><strong>Dosing</strong></td>
<td>800 mg three times a day with meal</td>
<td>750 mg three times a day with meal</td>
</tr>
<tr>
<td><strong>Treatment strategy</strong></td>
<td>4 weeks lead in with PegIFN/ribavirin prior to commencing triple therapy</td>
<td>Commence directly with triple therapy and continue for 12 weeks, then PegIFN/ribavirin</td>
</tr>
<tr>
<td><strong>HCV RNA monitoring time points</strong></td>
<td>Pretreatment, week 8, 12 and 24</td>
<td>Pretreatment, week 4, 12 and 24</td>
</tr>
<tr>
<td><strong>Total treatment duration</strong></td>
<td>If RNA undetected week 8 and 24 continue to week 28</td>
<td>If RNA undetected week 8 and 12, continue to week 28</td>
</tr>
<tr>
<td><strong>Treatment naive</strong>*</td>
<td>If RNA detected week 8 and undetected week 24, continue to week 48</td>
<td>If RNA detected (≤ 1000 IU/mL) week 4 and undetected week 12, continue to week 48</td>
</tr>
<tr>
<td><strong>Treatment experienced</strong>*</td>
<td>If RNA undetected week 8, 12 and 24, continue to week 36</td>
<td>If RNA undetected week 8 and 12, continue to week 48</td>
</tr>
<tr>
<td><strong>Patients with cirrhosis</strong></td>
<td>If detected week 8 and undetected week 12 and 24, continue to week 48</td>
<td>If RNA detected (≤ 1000 IU/mL) week 4, and undetected week 12, continue to week 48</td>
</tr>
<tr>
<td><strong>Stopping rule</strong></td>
<td>Stop all therapy if RNA detected at week 12 or 24</td>
<td>Stop all therapy if HCV RNA &gt;1000 IU/mL at week 4 or detected at week 12 or 24</td>
</tr>
<tr>
<td><strong>Anticipated proportion of treatment naive patients qualifying for shortened duration of therapy</strong></td>
<td>44% (28 weeks)</td>
<td>58% (24 weeks)</td>
</tr>
<tr>
<td><strong>Adverse Events more frequent with protease inhibitor</strong></td>
<td>Anaemia, dysgeusia (altered taste)</td>
<td>Anaemia, skin rash, perianal symptoms, nausea, diarrhoea</td>
</tr>
<tr>
<td><strong>Drug interactions (particularly drugs metabolised by CYP3A4)</strong>*</td>
<td>Frequent</td>
<td>Frequent</td>
</tr>
</tbody>
</table>

* in patients without cirrhosis; includes previous relapsers to treatment
PegIFN: pegylated interferon
For patients with HCV genotype 1, the antiviral treatment regimen varies according to which direct acting antiviral agent is used in combination with pegylated interferon and ribavirin. Boceprevir is given as 800 mg (4 tablets) three times a day commencing after a 4-week lead-in period with pegylated interferon and ribavirin alone. Treatment with triple therapy is continued for a further 24 or 44 weeks. The shorter duration regimen is for non-cirrhotic patients who are either treatment naïve or relapsed following a previous course of therapy, and have undetectable HCV RNA at week 8 of treatment. Telaprevir, 2250 mg daily in two or three divided doses, is given as triple therapy for the first 12 weeks of treatment, and then combination pegylated interferon and ribavirin is continued to 24 or 48 weeks according to similar criteria. Specific information regarding boceprevir and telaprevir treatment regimens is provided in Table 11.4.

Despite recent advancements in HCV treatment, there are significant numbers of patients with hepatitis C who respond poorly to therapies or have contraindications to therapy. Decisions about therapy in these individuals are made on a case-by-case basis by the specialist. These include patients who have previously failed therapy, those with HCV and HBV co-infection, HCV and HIV co-infection, chronic renal failure, cryoglobulinaemia and with HCV recurrence after liver transplantation.

**HIV and HCV co-infection**

HIV and HCV co-infection is associated with higher HCV viral load and an accelerated rate of HCV disease progression. There is no fundamental difference in the management of HCV in the presence of HIV although certain antiretroviral agents (e.g. didanosine, stavudine, zidovudine) are contraindicated and should be avoided if possible. Patients with HIV and HCV co-infection who either have stable CD4 cell counts on antiretroviral therapy, or who do not require antiretroviral therapy may be considered for combination pegylated interferon plus ribavirin, with the addition of a protease inhibitor for genotype 1. Telaprevir is preferred to boceprevir, due to fewer drug-drug interactions with antiretroviral therapy. However, clinically important interactions with telaprevir are present, including with efavirenz (dose of telaprevir needs to be increased to 3375 mg per day). Although response rates in this patient group now appear similar to those with HCV mono-infection, management of such patients may be difficult, particularly in patients already taking multiple medications, and therapy should be carried out by a specialist experienced in this area.

**Who should or should not be treated for hepatitis C?**

We are currently at major cross-roads in the treatment of hepatitis C. The next generation of direct-acting antiviral agents (simeprevir, sofosbuvir and daclatasvir) will soon be available, initially in combination with pegylated interferon and ribavirin, but later without the requirement for interferon and possibly ribavirin. Data from phase 3 trials of even newer treatment regimens indicate SVR rates over 90-95% in patients with chronic HCV, regardless of genotype, the presence of cirrhosis, prior treatment response, obesity and ethnicity, all previously predictors of poor response.

Many patients should probably wait for new therapies, rather than go through a course of currently available treatment, which may be associated with significant side effects and inferior treatment outcomes. The decision to defer therapy should be based on an individual’s likelihood of disease progression, the benefits of early viral eradication, the likely treatment response and the anticipated timelines for the availability of new therapies.

Patients who probably should not wait for new therapies are those with a high likelihood of disease progression, and who are likely to respond to current therapies, including:

- Patients with advanced fibrosis or cirrhosis
- Patients who are treatment naïve or relapsers (i.e. became HCV RNA undetected on treatment but became viraemic again once treatment was ceased) before therapy

Other patients may have medical or personal reasons to undergo currently available treatments, and should be supported in their treatment choices. Given the likelihood of significant side effects, decisions about whether to treat and when to treat are often difficult. When discussing therapy with a patient, issues and commitments such as work, study, relationships, substance abuse and pregnancy should be considered. Likelihood of adherence to a complex regimen involving a large burden of tablets, frequent dosing, multiple blood tests and side effects should also be taken into consideration.

Patients with decompensated cirrhosis should not undergo current interferon-based treatments. Treatment with triple-therapy is particularly hazardous, with an increased potential for adverse events, including severe anaemia, sepsis, hepatic decompensation and death. A combination of a pretreatment serum albumin < 35 g/L and platelet count < 100 x10⁹/L is a major risk for treatment and is regarded as a contraindication. Several interferon-free regimens are currently being assessed in patients with decompensated liver disease and appear safe and effective, providing the potential for salvage of patients despite overt liver failure, and possibly preventing death or the need for liver transplantation.

Major contraindications to current therapies include:

- Decompensated cirrhosis
- Major psychiatric conditions, particularly severe depression

**Major psychiatric conditions, particularly severe depression**

Patients who probably should not wait for new therapies are those with a high likelihood of disease progression, and who are likely to respond to current therapies, including:

- Patients with advanced fibrosis or cirrhosis
- Patients who are treatment naïve or relapsers (i.e. became HCV RNA undetected on treatment but became viraemic again once treatment was ceased) before therapy

Other patients may have medical or personal reasons to undergo currently available treatments, and should be supported in their treatment choices. Given the likelihood of significant side effects, decisions about whether to treat and when to treat are often difficult. When discussing therapy with a patient, issues and commitments such as work, study, relationships, substance abuse and pregnancy should be considered. Likelihood of adherence to a complex regimen involving a large burden of tablets, frequent dosing, multiple blood tests and side effects should also be taken into consideration.

Patients with decompensated cirrhosis should not undergo current interferon-based treatments. Treatment with triple-therapy is particularly hazardous, with an increased potential for adverse events, including severe anaemia, sepsis, hepatic decompensation and death. A combination of a pretreatment serum albumin < 35 g/L and platelet count < 100 x10⁹/L is a major risk for treatment and is regarded as a contraindication. Several interferon-free regimens are currently being assessed in patients with decompensated liver disease and appear safe and effective, providing the potential for salvage of patients despite overt liver failure, and possibly preventing death or the need for liver transplantation.

Major contraindications to current therapies include:

- Decompensated cirrhosis
- Major psychiatric conditions, particularly severe depression

**Major psychiatric conditions, particularly severe depression**
• Autoimmune disease
• Significant cardiac disease
• Pregnancy (ribavirin is a teratogen – patients and their partners must avoid pregnancy during therapy and for 6 months after cessation of treatment due to the possibility of birth defects)

Although interferon is contraindicated in people with depression it may be used safely in patients with controlled depression and anxiety disorders or controlled seizure disorders. If the patient is being treated by a psychiatrist or neurologist, discussion with the specialist is recommended before the initiation of interferon therapy.

Many liver clinics are involved in clinical trials of new therapies for hepatitis C. Spaces in clinical trials are limited, but treatment within a clinical trial offers access to new treatments many years before they will be available for routine use in Australia. It should be noted however, that not all treatment strategies evaluated within clinical trials are successful, and some may not proceed to approval and marketing.

**Side effects of Interferon-based antiviral treatment**

Side effects are common but do not usually require discontinuation of treatment. However, patients do require significant support and encouragement throughout treatment. Adverse effects of interferon therapy include flu-like symptoms, irritability, weight loss, insomnia, vomiting, depression and anxiety, mild hair loss, rash, cough, myelosuppression and induction of autoimmunity, particularly thyroid disease.

Ribavirin treatment always induces a degree of intravascular haemolysis, which results in a fall in haemoglobin in most patients. This anaemia may result in tiredness, shortness of breath and precipitation of myocardial ischaemia in at-risk patients. Anaemia may need to be managed with ribavirin dose reduction, or blood transfusions and erythropoietin.

Additional side effects of the protease inhibitors, boceprevir and telaprevir, include anaemia, gastrointestinal symptoms and rashes. Both agents are associated with increased rates of anaemia. Boceprevir is associated with altered taste (dysgeusia). Telaprevir can cause nausea and diarrhoea, as well as anorectal problems, including haemorrhoids, fissures and pruritus. Telaprevir is also associated with the development of rash in up to 50% of patients. The rash is usually of mild or moderate severity, however it has the potential for systemic complications such as drug rash with eosinophilia and systemic symptoms (DRESS) and Stevens-Johnson syndrome. Patient education, and the implementation of a rash management plan with early use of moisturisers and topical corticosteroid creams, are effective and allow completion of the full course of treatment in most patients.

Boceprevir and telaprevir have the potential for significant drug-drug interactions as they are extensively metabolised in the liver and are potent substrate and inhibitors of CYP3A4. Caution is necessary for any changes in prescribed, over-the-counter and complementary medications prior to commencement and during therapy. It is critically important that medications are not started or stopped without first checking with the hepatitis treatment team or discussing the potential for drug interactions with a pharmacist. Once the protease inhibitor is stopped, previous medication doses can then be resumed.

A further important consideration with the use of protease inhibitors is the potential for the development of antiviral drug resistance. It is essential that protease inhibitors are taken as prescribed without dose changes, nor ceased without specialist consultation.

Given the wide range and potential seriousness of side effects, patients must be closely monitored during therapy. Currently, most treatment is provided through public hospitals and patients have ready access to nurse specialists to advise and support them through therapy. In general, patients on therapy are seen once a week for the first month, and then each month until the end of treatment, with blood counts and biochemistry evaluated at each visit. Patients on protease inhibitors may require more frequent assessments. Dose modification guidelines (only for pegylated interferon and ribavirin) are followed when side effects or laboratory changes require intervention.

**Changing models of care**

While most treatment is based in public hospitals at present, there is an important trend towards treatment in the community. This shift will involve primary care clinicians taking a greater role in the support and monitoring of patients on therapy. Many hospitals have put together shared-care programs with specific information and guidelines about management during therapy. Specialist hepatitis treatment nurses can also facilitate community-based treatment, particularly in regional centres with limited access to specialist services. In most jurisdictions, a number of general practitioners and community-based non-specialist doctors have been approved to prescribe antiviral therapy for the maintenance treatment of hepatitis C. Specific criteria for authorisation to prescribe maintenance drug treatment for HCV may differ across jurisdictions. Generally, accredited community prescribers must participate in shared care with a treating specialist affiliated with a recognised hospital-based viral hepatitis treatment facility. To ensure the highest chance of achieving viral eradication, it is important to support patients through a complete course of therapy regardless of the treatment model.
Liver transplantation in viral hepatitis

Chronic hepatitis C is the leading indication for liver transplantation in Australia. Patients should be referred to a transplant unit when they develop signs of hepatic decompensation, such as ascites, encephalopathy, bacterial infections (particularly spontaneous bacterial peritonitis), muscle wasting or worsening fatigue. It is best to try to identify subtle signs of impending liver failure (Chapters 5 and 7), so that early referral can be made. Liver transplantation is also indicated in some patients with HCC. Detailed management of end-stage liver disease is beyond the scope of this publication.

New antiviral therapies in development offer the potential for safe treatment of patients before transplantation to prevent re-infection of the transplanted liver, as well as treatment of patients with post-transplant HCV recurrence.

Summary

Chronic hepatitis C poses challenges of monitoring of underlying liver disease, selection for treatment and care during treatment. It is important that a patient’s concerns be addressed by the provision of information about the disease and access to counselling and psychosocial support. The primary care clinician has a vital role in the assessment and monitoring of patients with chronic viral hepatitis. Shared care is the preferred model of care for patients with chronic viral hepatitis and effective communication between GPs, specialists and referral centres is required for optimal patient management.

References

3. Schiff ER, Hoofnagle JH, editors. Update on viral hepatitis. GPs, specialists and referral centres is required for optimal patient management.
CHAPTER 12 PRIMARY CARE MANAGEMENT OF CHRONIC HEPATITIS B

2014 AUTHORS

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**Introduction**

Management of chronic hepatitis B in the primary care setting involves identification of who to test and appropriate diagnosis, as well as initial and ongoing clinical assessment and monitoring. The primary care clinician should be involved in the provision of information, support and referral, counselling the patient about the current understanding of the disease process and potential complications, as well as general issues of diet, mental health, lifestyle, transmission and vaccination.1,2 The primary care clinician has an important role in presenting the patient with specific management options, including the need for lifelong monitoring and the possibility of antiviral therapy and surveillance for hepatocellular carcinoma if indicated.1,2

**Initial assessment**

The primary care clinician is usually the first point of contact for a person at risk of chronic hepatitis B. Ordering the appropriate hepatitis B virus (HBV) serology tests is essential to make an appropriate diagnosis (HBsAg, anti-HBc, and anti-HBs; see Chapter 7).

The natural history of HBV infection varies under the influence of multiple factors, including age at acquisition of infection, mode of transmission and viral genotype. The aims of the initial assessment of a patient with chronic hepatitis B are:

- to provide information and support to the patient in order to facilitate understanding of his or her diagnosis and ongoing health needs
- to assess the phase of disease and the severity of underlying liver disease.

The assessment should start with a thorough clinical history and physical examination. As many people living with chronic hepatitis B in Australia are from non-English speaking backgrounds, an interpreter should be used to provide a clear explanation following diagnosis and throughout the initial assessment, if required (e.g. Telephone Interpreting Services (TIS): 1300 131450). Use of culturally appropriate material and resources can also help to increase understanding. An awareness of health literacy issues should guide the clinician to convey information in a way the patient will understand, enabling patients to make informed choices.

**History taking should include:**

- addressing patient concerns about the infection
- identifying household and sexual contacts and children who might require further testing
- assessing risk factors for acquisition of chronic hepatitis B, including country of birth, a family history of chronic hepatitis B, and a family history of hepatocellular carcinoma
- assessing host or viral factors that are associated with an increased risk of progression to advanced liver disease, including older age (related to a longer duration of infection), heavy alcohol consumption, cigarette smoking and co-infection with other viruses, e.g. hepatitis C virus (HCV), hepatitis D virus (HDV), and human immunodeficiency virus (HIV).3

**KEY POINTS**

- Opportunistic testing of people at risk will reduce the number of people with chronic hepatitis B who are undiagnosed and subsequently reduce the mortality and morbidity caused by hepatitis B.
- Hepatitis B is vaccine preventable. Test and vaccinate people susceptible to infection, such as family members, and household and sexual contacts of people living with hepatitis B.
- Without appropriate intervention, up to 25% of people with chronic hepatitis B from infancy develop cirrhosis or hepatocellular carcinoma (HCC).
- There is no such thing as a healthy carrier, so do not use this term – follow-up and life-long monitoring are essential for every patient with chronic hepatitis B, as it is a dynamic disease. Patients move between phases and must be regularly re-evaluated to determine which phase they are in; they should be assessed for liver damage and the need for antiviral therapy.
- Antiviral therapy for chronic hepatitis B is available and can prevent cirrhosis and HCC. Antiviral therapy is generally targeted at patients in the immune clearance (phase 2) and the immune escape (phase 4) phases.
- Primary care management of chronic hepatitis B includes lifelong monitoring, HCC surveillance where indicated, education and counselling, psychosocial support and dietary and lifestyle advice.
Additional tests are required to quantify viral replication and the presence of ongoing liver damage following diagnosis (Table 12.1). These tests allow for appropriate information to be conveyed to the patient about the phase of infection (see natural history below) and the need for consideration of antiviral treatment. Ideally all patients should have an assessment of liver fibrosis, which is increasingly being done via non-invasive tests such as transient elastography (FibroScan®) available at some specialist liver clinics. Full viral serology and other investigations to be conducted during the initial testing are discussed in detail in Chapter 7.

**Natural history**

An understanding of the natural history of chronic hepatitis B assists with patient assessment and management. There are four phases of infection that are defined by the degree of viral replication and the patient’s immune response to the virus. Determining the phase of chronic hepatitis B requires three primary tests: HBV viral load (HBV DNA), alanine aminotransferase (ALT) and hepatitis B e antigen/antibody (HBeAg/anti-HBe) status (Figure 12.1).

In phase 1 (immune tolerance) viral replication is high but there is minimal ongoing liver damage. In phase 2 (immune clearance) viral replication remains high and there is evidence of liver damage (raised ALT) as the patient’s immune response causes inflammation and liver cell damage. In phase 3 (immune control) viral replication is controlled, again with no ongoing active liver damage – although significant liver fibrosis may be present following phase 2. Patients in the immune control phase were previously described as ‘healthy carriers’ but this term is no longer used, as a substantial proportion of these patients progress to phase 4 (immune escape), characterised by recurrent viral replication and immune-mediated liver damage (Table 12.2). As active viral replication and ongoing liver damage occur in phases 2 and 4, it is typically patients in these phases who require closer follow-up and consideration for antiviral therapy.

**Ongoing monitoring of patients with chronic hepatitis B**

All patients with chronic hepatitis B should be actively managed, regardless of the apparent phase of infection at initial evaluation. Regular review is required to assess for changes in disease activity and consideration of the need for referral and antiviral therapy. The frequency of monitoring should be determined based on the patient’s age, phase of hepatitis B infection, family history and other factors such as comorbidities; however, a minimum frequency of annual review is recommended (Table 12.2).

**TABLE 12.2 How to monitor based on phase of disease**

<table>
<thead>
<tr>
<th>Phase</th>
<th>Immune tolerance</th>
<th>Immune clearance or escape</th>
<th>Immune control</th>
<th>Immune escape</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Every 6–12 months</td>
<td>LFTs, HBV DNA*</td>
<td>HBeAg/anti-HBe</td>
<td>Every 6–12 months</td>
<td>HBsAg / anti-HBs every 1–2 years (to assess for seroconversion)</td>
</tr>
<tr>
<td>2. Require consideration of treatment (see section Antiviral therapy – hepatitis B below)</td>
<td>Phase 3. Immune control</td>
<td>On treatment Regular monitoring according to treatment regimen (see section Antiviral therapy – hepatitis B below)</td>
<td>HBV DNA only Medicare rebatable once a year in patients not on treatment, 4 times a year on antiviral treatment</td>
<td></td>
</tr>
</tbody>
</table>

* LFT: liver function test

**TABLE 12.1 Tests to be ordered for initial assessment of chronic hepatitis B**

<table>
<thead>
<tr>
<th>Test</th>
<th>Why the result is important</th>
</tr>
</thead>
<tbody>
<tr>
<td>HBeAg / Anti-HBe</td>
<td>Quantify replication, identify phase of infection, prognostication, consideration of treatment</td>
</tr>
<tr>
<td>HBV DNA</td>
<td>Co-infection, vaccination</td>
</tr>
<tr>
<td>HAV, HCV, HDV, + HIV serology</td>
<td>Alcohol, drug use, diabetes</td>
</tr>
<tr>
<td>Evaluation for comorbidities</td>
<td>Necroinflammatory activity, synthetic function</td>
</tr>
<tr>
<td>LFTs</td>
<td>May indicate cirrhosis if thrombocytopenia is found</td>
</tr>
<tr>
<td>FBC</td>
<td>Synthetic function</td>
</tr>
<tr>
<td>Alpha fetoprotein</td>
<td>Hepatocellular carcinoma (HCC)</td>
</tr>
<tr>
<td>Abdominal ultrasound and portal doppler</td>
<td>HCC and evidence of cirrhosis portal hypertension</td>
</tr>
</tbody>
</table>

HAV: hepatitis A virus; HCV: hepatitis C virus; HDV: hepatitis D virus; HIV: human immunodeficiency virus; LFT: liver function test; FBC: full blood count; PT: prothrombin time; INR: international normalised ratio
Liver biopsy and transient elastography
Non-invasive measures of hepatic fibrosis are now available, the most commonly used being transient elastography (e.g. FibroScan®). This uses ultrasound elastography to measure liver stiffness. Shear waves are generated and measured in kPa which correlate with the fibrosis score as determined by biopsy. Readings are given that can accurately place the patient in different stages of fibrosis. While there is no longer a requirement for liver biopsy to begin treatment, there is still place for liver biopsy in the assessment of some patients. For example, patients with intermediate FibroScan® scores, other suspected liver pathology or unexplained elevated ALT, or where there is clinical concern about the degree of underlying fibrosis – e.g. clinical signs of chronic liver disease, or low platelet count.

Hepatocellular carcinoma surveillance
Chronic HBV infection is the leading cause of HCC globally and is responsible for an increasing burden of HCC related deaths in Australia. Unlike hepatitis C, in hepatitis B infection a substantial proportion of HCC occurs in the absence of cirrhosis. Given that patients with early HCC typically have no specific symptoms, timely detection is difficult. Onset of symptoms such as weight loss, lethargy, jaundice or localised pain or a palpable mass in the upper abdomen may indicate advanced HCC. Late diagnosis is often the reason for very short average survival time following diagnosis. HCC is the fastest increasing cause of cancer mortality in Australia. Six-monthly surveillance for those at increased risk can identify small HCC lesions that may be amenable to curative treatment. Six-monthly ultrasound tests – either alone or combined with serum alpha fetoprotein (AFP) testing – are recommended for the following patients with chronic hepatitis B:

- Asian males ≥ 40 years
- Asian females ≥ 50 years
- Africans ≥ 20 years
- Aboriginal and Torres Strait Islander people > 50 years
- All patients with cirrhosis
- Patients with a family history of HCC

If the screening test is abnormal, a four-phase computed tomography (CT) or contrast-magnetic resonance imaging (MRI) scan is recommended for diagnosis.

General management issues for patients with hepatitis B

Discussion about routes of viral transmission
Patients with chronic hepatitis B are commonly concerned about the risks of transmitting the infection to others including their children and family members. Issues regarding sexual transmission, mother-to-child transmission, blood-borne transmission

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**Figure 12.1 Natural history of chronic hepatitis B, the four phases of infection and their relevance to treatment decisions**

<table>
<thead>
<tr>
<th>IMMUNE TOLERANCE</th>
<th>IMMUNE CLEARANCE</th>
<th>IMMUNE CONTROL</th>
<th>IMMUNE ESCAPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>High HBV DNA, normal LFTs, HBeAg positive</td>
<td>High HBV DNA, abnormal LFTs, HBeAg positive</td>
<td>Low HBV DNA, normal LFTs, HBeAg neg, anti-HBe pos</td>
<td>High HBV DNA, abnormal LFTs, HBeAg neg, anti-HBe pos</td>
</tr>
<tr>
<td>Monitor every 6-12 months</td>
<td>At risk of progression to cirrhosis and HCC therefore should be referred for consideration of treatment</td>
<td>Monitor every 6-12 months</td>
<td>At risk of progression to cirrhosis and HCC therefore should be referred for consideration of treatment</td>
</tr>
</tbody>
</table>
and other concerns should all be discussed. Being clear about activities that carry negligible risk like food sharing, touching and kissing is important to dispel myths and prevent stigma, discrimination and sometimes self-imposed social isolation.

In Australia, the majority of people with chronic hepatitis B were born overseas in countries with high or intermediate prevalence and contracted the infection at birth or as infants. Sometimes patients are confused regarding their mode of acquisition. Discussing the fact that HBV transmission early in life is common for those born in endemic areas (or for their Australian-born children in the pre-vaccination era) can help to resolve these concerns.

With universal vaccination of neonates against hepatitis B from May 2000, together with administration of hepatitis B immune globulin (HBIG) at birth to infants of mothers with chronic hepatitis B infection, there is now a very low incidence of vertically acquired HBV in Australia. Most cases of infection that are newly acquired occur through unsafe injecting drug use or sexual transmission.

People with chronic hepatitis B should be advised to protect sexual partners by practising safer sex, and ensuring long-term partners are vaccinated with serological testing to ensure immunity. Vaccination is available free to household and sexual contacts in almost all Australian states and territories, and general practices can order HBV vaccine from state and territory health departments. For further advice on vaccination refer to the Australian Immunisation Handbook.9

Another recommendation to prevent HBV transmission is to avoid the sharing of grooming tools that can cause abrasion (such as razors, toothbrushes and tweezers). People who inject drugs should be encouraged to use sterile water, needles and syringes, as well as new injecting equipment such as spoons, filters and tourniquets each time they inject (see Chapter 3 and Appendix 4).

Risk of transmission and infection are discussed in detail in Chapter 2. Communication of safer sex and safe injecting messages is covered in Chapter 3 and Appendixes 1-3.

**Notification**

Notification of a diagnosis of chronic hepatitis B by the clinician is required in most states and territories. Information collected by the clinician includes risk factor information and is more complete than automatically generated laboratory notifications. Please refer to state or territory health departments or the Cancer Council hepatitis B website at: http://www.hepbhelp.org.au.

**Vaccination to prevent hepatitis A**

Infection with HAV in a patient with chronic hepatitis B may precipitate acute liver failure, particularly in the setting of pre-existing advanced liver disease. HAV serology testing should be part of the initial assessment of a patient. HAV vaccination should be offered to all non-immune chronic hepatitis B patients (Chapter 5).

**Vaccination to prevent influenza and pneumonia**

Patients with advanced liver disease or cirrhosis should receive influenza vaccination and pneumococcal vaccination as recommended in the Australian Immunisation Handbook.9

**Pregnancy**

Women often receive the diagnosis of chronic hepatitis B during pregnancy via routine antenatal screening. Diagnosis in this context can cause considerable distress and it is essential pregnant women receive adequate counselling and appropriate referral. All women with chronic hepatitis B contemplating pregnancy should seek advice regarding management of their chronic hepatitis B during pregnancy. Management during pregnancy should be conducted together with a specialist hepatitis service and involves:

**For the mother**

- Determination of the phase of infection, with the HBV DNA viral load also important for determining the risk of HBV transmission to the infant.
- For women with a high viral load during pregnancy (e.g. over 10,000,000 IU/mL) consideration of antiviral therapy to reduce transmission should be made in conjunction with a specialist.10-13
- It is important the woman is enrolled in ongoing care after pregnancy, and that other family members (and household contacts) are offered testing and vaccination.

**For the infant**

- Concurrent administration of the birth dose of hepatitis B vaccine plus HBIG 100 IU in separate thighs – ideally within 12 hours of birth – is essential, as is completing the course of hepatitis B vaccine in the first 6 months of life.6
- Babies born to women with chronic hepatitis B should have serological confirmation at 9-12 months of age (or 3 months after the last HBV vaccination) that vaccination was successful (HBsAg negative and anti-HBs >10 IU/L).

**Breastfeeding**

Risk of transmission via breast milk is negligible – especially in the setting of infant vaccination – and women with chronic hepatitis B should be encouraged to breastfeed if this is their choice.

**Children**

Children with hepatitis B are usually in the early stages of infection and complications are rare. Liver cancer and liver damage can however occur in childhood and monitoring is advised at least annually.

**Lifestyle considerations**

The possibility of lifestyle modification needs to be discussed with patients, particularly in relation to cigarette smoking, alcohol consumption and healthy diet, exercise and weight.
Cigarette smokers should be encouraged to quit. Alcohol intake should be minimal. Excessive alcohol consumption (> 50 grams a day) increases the risk of progressive liver damage. A drink containing 10 grams of alcohol, such as a can of medium-light beer (3.5% alcohol), a 100 mL glass of wine or a shot (30 mL) of spirits, is regarded as a standard drink. A can of regular beer (4.9% alcohol) equals 1.5 standard drinks (15 grams alcohol). A bottle of wine (9.5–13% alcohol) equals about seven to eight standard drinks (70–80 grams alcohol). Australian guidelines recommend that people with viral hepatitis should drink alcohol infrequently or at low levels, and should consider not drinking at all. Specific strategies to reduce alcohol intake are set out in Table 12.3. People with cirrhosis should be advised, and supported to stop drinking alcohol completely.

**TABLE 12.3 How to reduce alcohol consumption**

| Plan at least two alcohol-free days per week |
| Switch to low alcohol or alcohol-free drinks |
| Avoid situations where there will be pressure to drink, e.g. rounds at the pub |
| Alternate non-alcoholic and alcoholic drinks |
| Drink a daily maximum of two drinks |

People with chronic hepatitis B who inject drugs need support, advice and access to drug dependency treatment programs as well as ongoing care for their hepatitis B infection. If patients employ unsafe injection practices they are at risk of overdose, other health risks and the acquisition of other infections including hepatitis C, hepatitis D and HIV that are associated with worse outcomes in the context of chronic hepatitis B.

**Nutrition**

There are considerable, unsubstantiated dietary information and advice directed at people with chronic viral hepatitis. A well-balanced diet is recommended. For most people with hepatitis B, dietary recommendations are the same as for the general population (encouraging: grilled rather than fried food; lean meats and fish; reduced-fat products; wholemeal bread; vegetables and fruit; pasta; minimisation of fat for spreading and cooking). People with advanced liver disease, or other conditions such as coeliac disease or diabetes may be referred to a specialist dietitian for further advice.

Overweight or obese patients should be advised of a gradual weight-reduction program, particularly as there is increasing evidence of interaction between chronic hepatitis B and non-alcoholic steatohepatitis (NASH) worsening fibrosis. Those who may have fatty liver need to avoid a precipitous drop in weight as this can induce deterioration in liver function.

Patients with advanced liver disease who develop protein-calorie malnutrition should be referred to a specialist dietitian. Such patients often require protein supplementation, and should be encouraged to eat high-energy foods frequently throughout the day. Very few, if any, patients with advanced liver disease should be subjected to protein restriction. This is a change from the previous doctrine that all patients with hepatic encephalopathy should be protein restricted.

**Psychosocial support**

Patients may experience social isolation, anxiety or discrimination related to infection with viral hepatitis, which may be compounded by physical symptoms. The primary care clinician can begin by listening to the patient and demonstrating sensitivity to linguistic and cultural differences, which may affect an individual’s response to viral hepatitis. Provision of verbal and written information relating to transmission or disease natural history may allay fears (Chapter 16). Referral to counselling or support services may be indicated for patients with complex emotional, family and relationship or disclosure issues. All patients should be made aware of services such as counselling and support groups, telephone helplines and community organisations.

Information about services is available from Hepatitis C Councils or multicultural health support organisations (Chapter 16).

**Complementary therapies**

There is little evidence that herbal medicines have any antiviral effect despite many patients reporting some symptomatic improvement and the reduction in ALT observed with some agents. Many preparations are safe but some have reported hepatotoxicity and should be avoided (e.g. mistletoe, valerian, heliotropium, kombucha tea, black cohosh). Close monitoring of liver biochemistry is recommended at the start of any herbal medicine.

**Immunosuppression and chronic hepatitis B**

**Immunosuppression** in a person with chronic hepatitis B infection can lead to potentially fatal flares of liver disease, particularly in the setting of cancer chemotherapy. All patients undergoing chemotherapy or immunosuppression should be tested for hepatitis B. Any patient with chronic hepatitis B undergoing significant immunosuppression needs a complete assessment and consideration of antiviral therapy. In addition to chemotherapy, prolonged glucocorticoid therapy can precipitate a flare, as can agents used in the treatment of autoimmune conditions, including methotrexate and many biological agents including rituximab and infliximab. Short courses of low-dose oral steroids are unlikely to lead to reactivation of chronic hepatitis B.
Antiviral therapy for hepatitis B

Aims of treatment
The decision to prescribe specific treatment for chronic hepatitis B is based on the patient’s viral load and evidence of past or ongoing liver damage. The goal of therapy is to prevent, halt or even reverse the progression of liver injury toward cirrhosis, liver decomposition and liver cancer which are the major causes of death in older patients with hepatitis B infection.

There are a number of aims of therapy, including:
- Limit liver damage due to immune-mediated inflammation and fibrosis
- Achieve sustained suppression of viral replication
- Reduce risk of progression to cirrhosis and HCC
- Reduce morbidity and mortality
- Minimise toxicity, minimise resistance, maximise adherence

These aims can be achieved in a significant proportion of patients with chronic hepatitis B with currently available therapies.

Who should be treated?
In patients with chronic HBV infection, antiviral therapy is generally indicated for:
- Patients in phase 2 and 4 of infection with persistently elevated ALT
- Patients with evidence of cirrhosis and detectable HBV DNA regardless of viral load or ALT
- Patients undergoing immunosuppression
- Pregnant women with high HBV viral load to avert mother-to-child transmission (see section on Pregnancy above)

Oral therapy
Entecavir and tenofovir are the first-line oral antiviral therapies available for patients with chronic hepatitis B. Both treatments are taken as single daily tablets; they are potent, well tolerated with few side effects and have a high barrier to the development of viral resistance. The duration of treatment is generally considered to be indefinite in HBeAg-negative patients (phase 4). HBsAg loss (being a definitive treatment endpoint) is rare, and ongoing viral suppression is the main aim of treatment. Persistent viral suppression in turn leads to decreased immune mediated damage, reduced progressive fibrosis (and often leads to regression in existing fibrosis), and has been shown to significantly reduce the risk of liver cancer.19,20

There is no evidence of difference in efficacy between these two agents for treatment in naive patients, but tenofovir is often recommended for women with child-bearing potential due to greater available evidence for safety in pregnancy. Tenofovir is rarely associated with renal toxicity and electrolyte abnormalities; renal function should be assessed at baseline and during routine monitoring, along with phosphate levels.

Other treatments including lamivudine, adefovir and telbivudine are not first-line agents as they have lower barriers to the development of resistance. For patients previously treated with these agents who have developed resistance, cross-resistance with entecavir and (less commonly) tenofovir is observed; specialist advice is recommended when making treatment decisions in this setting (Table 12.4).

Interferon
Pegylated interferon alfa is an alternative therapeutic option which aims to reduce viral replication through modulating the immune response to HBV in those with chronic infection. It is more commonly used in HBeAg-positive patients with the objective of eliciting HBeAg seroconversion and sustained suppression of viral replication. Pegylated interferon is also associated with a higher probability of HBsAg seroconversion during or after treatment, although this result remains uncommon. Treatment is via self-administered subcutaneous injections weekly for 48 weeks. Common side effects include flu-like symptoms, anorexia and fatigue; psychiatric complications (including irritability and depression) are also observed, particularly in patients with a history of mental illness. Rarely, autoimmune manifestations are observed.

Pegylated interferon is sometimes preferred by patients not wishing to consider potentially indefinite oral therapy, and women considering pregnancy due to the defined treatment course. Interferon is contraindicated in pregnancy, in patients with autoimmune diseases, and in patients with decompensated liver disease. It is rarely used in the setting of cirrhosis given the possibility of decomposition and the availability of alternative oral antivirals.
### TABLE 12.4 Treatments for hepatitis B: Current standard of care and Pharmaceutical Benefits Scheme listed indications

<table>
<thead>
<tr>
<th>Patient to be considered for therapy</th>
<th>PBS listed indications</th>
<th>First-line therapies</th>
<th>PBS streamlined code*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HBeAg-positive patient</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Persistent (at least 3-6 months) elevated ALT\(^a\)  
HBV DNA >20,000 IU/mL.       |                         |                      |                      |
| **HBeAg-negative patient**         |                        |                      |                      |
| HBV DNA >2,000 IU/mL WITH         |                        |                      |                      |
| Persistent (at least 3-6 months) elevated ALT\(^a\)  
OR evidence of accumulated liver damage such as fibrosis or moderate-to-severe inflammation |                         |                      |                      |
| **Patient with advanced fibrosis or cirrhosis,** irrespective of ALT (e.g. Scheuer score of 3 or 4 on biopsy or FibroScan > ~10 kPa) |                         |                      |                      |
| Cirrhosis and detectable HBV DNA   |                        |                      |                      |
| Failed HBV therapy and has cirrhosis and detectable HBV DNA |                         |                      |                      |
| Failed HBV therapy and has evidence of treatment failure\(^b\) |                         |                      |                      |
| Failed lamivudine and has cirrhosis and detectable HBV DNA |                         |                      |                      |
| Failed lamivudine and has evidence of treatment failure\(^b\) |                         |                      |                      |

Note: Elevated ALT
Elevated serum ALT varies between guidelines, but would usually be considered as > 2 x ULN
ALT ULN for men > 30
ALT ULN for women > 19

(1) Greater than 20,000 IU/mL (100,000 copies/mL) if HBeAg positive, or greater than 2000 IU/mL (10,000 copies/mL) if HBeAg negative
(2) As determined by: (a) confirmed elevated serum ALT; or (b) liver biopsy
(3) (a) Repeatedly elevated serum ALT levels while on concurrent antiviral therapy for 6 months or more duration in conjunction with documented chronic hepatitis B infection; or (b) repeatedly elevated HBV DNA levels 1 log greater than the nadir value or failure to achieve a 1 log reduction in HBV DNA within 3 months, while on previous antiviral therapy except in patients with evidence of poor compliance.
All persons with Child’s class B or C cirrhosis (ascites, variceal bleeding, encephalopathy, albumin < 30 g/L, bilirubin > 30 μmol/L) should have their treatment discussed with a transplant unit before initiating therapy.

* PBS streamlined codes – GP HBV s100 prescribers accredited to prescribe by their state or territory through the public hospital system can use streamlined codes. The streamlined authority process is designed to reduce the administrative burden on prescribers, as they don’t need to seek prior telephone or written approval from the Department of Human Services (DHS) or the Department of Veterans’ Affairs (DVA) to prescribe some PBS Authority required items. To prescribe a streamlined authority item, a prescriber is required to include a ‘streamlined authority code’ on the authority prescription. For information on GP prescribing see www.ashm.org.au

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**Liver transplantation in viral hepatitis**

Chronic viral hepatitis is the leading indication for liver transplantation in Australia.

Patients should be referred to a liver transplant service when they develop signs of hepatic decompensation, such as coagulopathy, ascites and hepatic encephalopathy. Subtle signs of impending liver failure (Chapters 5 and 7) should be identified promptly so that early referral can be made. Liver transplantation is also indicated in some patients with HCC. Management of advanced liver disease should be coordinated by a specialist hepatologist.

**Liver cancer**

All eligible patients should be enrolled in 6-monthly HCC surveillance (see hepatocellular carcinoma surveillance section above). If a patient is identified on surveillance as having a lesion suspicious for HCC he or she should be referred to a multidisciplinary liver cancer service. For HCC identified at an early stage, curative treatment is available, emphasising the importance of early detection and prompt referral for further assessment.

Liver biopsy should not be routinely ordered to characterise lesions suspicious for HCC; non-histological diagnosis is increasingly the rule and complications of biopsy (including seeding the biopsy tract with malignant cells) must be considered.
Summary

The primary care clinician has a vital role in the assessment and regular monitoring of patients with chronic hepatitis B. Chronic hepatitis B is a dynamic disease and clinicians must regularly re-evaluate patients to determine which phase of the infection they are in, assess for liver damage and determine the need for antiviral therapy. It is important that a patient's concerns be addressed through the provision of clear information, access to counselling and psychosocial support. Shared care is the preferred model of care for patients with hepatitis B and effective communication between GPs, specialists, hepatology nurses and referral centres is required for optimal patient management.

References

CHAPTER 13 PRIMARY CARE MANAGEMENT OF STIs

2014 REVIEW

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2008 EDITION

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**Introduction**

Sexually transmitted infections (STIs) are a diverse group of infections whose most common feature is sexual transmission. This group embraces the blood-borne viruses – hepatitis B virus (HBV), hepatitis C virus (HCV) (very rarely) and human immunodeficiency virus (HIV) – as well as other well-known STIs such as gonorrhoea, syphilis and genital chlamydia infection.

This chapter aims to provide an update on the management of STIs for primary care practitioners and other specialist clinicians who may be unfamiliar with STIs, and to describe the role of the primary care clinician in the diagnosis and treatment of patients with STIs.

In the first part of the chapter the emphasis will be on general principles of management. The second part of the chapter will look at the individual management of the eight STI syndromes outlined in Chapter 8.

**The challenge of managing patients with STI infection**

There are many challenges in the management of patients with STIs or patients who are at risk of STIs. Misconceptions about STIs are rife in the community and patients are generally less well informed about common STIs than they are about HIV. Only a minority know how common genital chlamydia infection is in the community, that most infections are asymptomatic, that it is associated with pelvic inflammatory disease (PID) and later infertility in women. Providing simple information about STIs and their potential for harm is a key role for the primary care clinician. Management of STIs is affected by the stigma attached to these infections and the fear or anxiety patients may have about the necessary investigations, the efficacy of treatments and exactly what the diagnosis means to them, their regular sexual partners and other current or future partners. Clinicians must be sensitive to these issues and must be prepared to discuss them frankly.

The doctor-patient relationship is central to the successful management of STIs. Establishing, from the first visit an open rapport is a major task for the primary care clinician. Empathy, honesty, sensitivity to the patient’s feelings and needs, accessibility, a firm and practical commitment to confidentiality and the privacy of the patient, and medical knowledge and expertise are essential qualities for the doctor to successfully manage a patient with an STI. In addition the clinician needs to show that she or he is comfortable talking about sex and is able to deal helpfully with patients with possible STIs.

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**KEY POINTS**

- STIs are generally capable of rapid assessment and prompt, effective treatment.
- Some STIs (e.g. chlamydia, herpes simplex, human papillomavirus) are among the most common infections encountered in everyday practice, so clinicians need to be familiar with their management and know how to talk with and inform their patients about these conditions.
- Although single-dose treatment is favoured for STIs, there are some situations (pelvic inflammatory disease [PID], epididymo-orchitis, proctitis) where more prolonged treatment is necessary. If the patient is regularly missing medication, treatment failure may result.
- STIs and fear of STIs introduce a range of psychosocial and sexuality questions in patients; the clinician has a vital role in providing an opportunity for patients to talk about and explore their concerns and clarify misconceptions.
- Contact tracing (partner notification) is an integral part of the treatment of almost all STIs. All treating clinicians have a clear responsibility to help achieve successful outcomes in contact tracing.
- Most ano-genital ulceration in Australia and New Zealand is due to genital herpes until proven otherwise, but clinicians should always ask themselves ‘could this be syphilis?’ particularly in men who have sex with men with genital lesions or skin rashes.
- In a patient with pelvic pain, having first excluded pregnancy, if the sexual history is suggestive, the patient should be treated immediately for STI-related PID, pending results of tests.
- Think of primary HIV infection in any patient with a flu-like illness diagnosed with a recently acquired STI.
- Not all ano-rectal symptomatology is surgical or routinely medical – ask yourself, particularly in gay men, “could this be an STI?”
**Natural history**

When managing and treating STIs, it is useful to understand the natural history of the specific STI you are dealing with; this is especially important where no curative treatments are available – as is the case with the viral STIs.

**Co-infection with HIV**

HIV infection is an STI, and people with an STI, or at risk of STIs, are at some risk of HIV, although the precise level of risk will depend on the type of sexual activities, the sex of the patient’s partner or partners and the prevalence of HIV infection in the region, city or country where the patient’s sexual contact(s) took place.

The interaction between HIV and other STIs is complex and cumulative (see Chapter 1). HIV, on the one hand, interferes with the clinical manifestations and severity of some STIs and, on the other hand, some STIs increase the viral load of HIV in bodily fluids, enhance HIV viral shedding from genital sites and can alter the natural history of HIV infection.1

People living with HIV today are generally well and continue to be sexually active. Their treating clinicians would advise them to adopt safer sex practices to avoid contracting other STIs and to prevent ongoing transmission of HIV. Standard safe sex practices include the use of condoms for all penetrative vaginal and anal sex. Unprotected oral sexual activities may occur. Several STIs are transmitted by oral sex, so all clinicians advise regular screening of people with HIV infection for other STIs and appropriate guidelines are available (see Chapters 4 and 10).2,3

Primary HIV infection (the seroconversion illness) in Australia and New Zealand occurs commonly but patients may not relate the symptoms to an unsafe sexual contact (see Chapter 4). Unsafe sexual contacts sometimes result in the patient contracting another STI in addition to HIV. The STI may be symptomatic or the patient may be diagnosed with an STI after undergoing a routine opportunistic screen on the clinician’s recommendation (see Chapter 8). In either case, primary HIV infection can present first in primary care practices (including emergency departments and sexual health clinics)4 as often the reason the patient attends is for intercurrent flu-like illness (see Chapter 8). The take-home message is: think of HIV testing in any patient diagnosed with a recently acquired STI, as well as in any patient with a flu-like illness (Case study 1).

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**CASE STUDY 1**

Lara is a 22-year-old university student and lives in an inner city suburb of Sydney. About 6 months ago she formed a relationship with a 25-year-old male student from Cambodia. Lara has had an Implanon contraceptive implant in situ for 1 year and has been amenorrhoeic. Lara initially noticed mild pelvic pain of 10 days duration 3 months ago and has had some irregular vaginal bleeding over the past month. Her doctor suspected pelvic inflammatory disease (PID) and commenced appropriate treatment immediately, with good initial resolution of symptoms. A cervical swab showed a positive PCR test for chlamydia; all other STI tests, including HIV antibody, were negative. Her boyfriend attended the Student Health Service (SHS) and also had a positive PCR test for chlamydia on a first passed specimen of urine (FPU). The doctor at the SHS treated him with azithromycin. Ten days into her treatment for PID Lara woke up one morning with nausea, vomiting, fever, headache and painful mouth ulcers. She had developed a mild macular erythematous rash on her chest and abdomen. Her boyfriend took her to the local emergency department and the doctor who assessed her admitted her to the gynaecology ward, as she still had mild tenderness in her lower abdomen.

The presumptive diagnosis was an exacerbation of PID with a possible allergic reaction to either doxycycline or metronidazole. Her treatment was changed to an intravenous regimen of clindamycin plus gentamycin. Over the course of the next 2 weeks she slowly improved. Now, 3 months later, she returns to her usual clinician for follow-up STI tests. Her clinician is concerned to see that Lara has lost 7 kg in weight since her last visit and is even more concerned when the laboratory rings to say Lara’s HIV antibody test is now positive.

**Physical examination**

When patients have symptoms consistent with an STI, clinicians should examine the ano-genital region as well as any other relevant areas (mouth and throat, lymph nodes, abdomen and pelvis) to avoid the possibility of missing clinical signs of infection. The clinician should explain to the patient why a genital examination is necessary, what it involves and, most importantly, what measures she or he proposes will minimise embarrassment and maintain privacy and also the option of a chaperone should the patient so wish. If a speculum or a proctoscopic examination is required, the clinician should show first-time patients the equipment which will be used and also assist the patient to assume a comfortable position for the examination. The aim is to perform the ano-genital examination smoothly, painlessly and with minimal psychological discomfort. It is an art, so it takes practice.
FIGURE 13.1: Self-collection of STI samples
Taking tests

Patients who are asymptomatic can self-collect swabs and this is now the preferred method of screening. Self-collected specimens can include first passed urine specimen (FPS), high vaginal swabs (HVS) and blind ano-rectal swabs (Figure 1). Throat swabs are collected by clinician to ensure that the oropharynx rather than the roof of the mouth or the tongue is swabbed. In patients with symptoms, clinicians should always examine the patient. If the patient declines, although not ideal practice, he or she can self-collect specimens of discharge, or swab a genital lesion so that the opportunity to make a diagnosis is not lost (Figure 1). Some clinicians may prefer to refer some clients to a pathology laboratory for ano-genital testing. This is acceptable if the clinician knows that the local laboratory staff are experienced and appropriately trained in specimen collection.

Serological testing

Serological tests are available for combined HIV antibody/antigen, syphilis (specific antibody tests as a screening test or a non-specific test, the rapid plasmin reagen test [RPR] in patients with a past history of syphilis), herpes (type specific tests for antibody to herpes simplex virus [HSV]-1 and HSV-2), hepatitis A IgG antibody, hepatitis B (surface antigen, surface antibody and core antibody) and hepatitis C (antibody). Syphilis and HIV are relatively rare infections in the Australian and New Zealand general populations, but both are serious infections and early diagnosis is important for the individual and for public health. For this reason, after appropriate pre-test discussion, it is good practice to encourage all patients at risk for STIs to have a serological test for HIV and syphilis. Syphilis is still endemic in Indigenous communities, in neighbouring countries in Asia and around the Pacific rim, and among men who have sex with men (MSM). HIV is a definite risk for MSM in both Australia and New Zealand, so the recommendation for HIV and syphilis testing is even stronger in MSM and Indigenous patients, as well as in travellers who have had sexual contacts in countries where syphilis and HIV are prevalent. In general, screening for HSV serologically is not helpful for individual patient care.

On a first visit, testing for previous exposure to HBV (generally HBcAb, and HBsAb if the patient believes he or she has been vaccinated already) and, in the case of MSM, to HAV IgG is a prelude to offering vaccination if testing shows the patient is not immune.

Making a brief psychosocial assessment

The clinician must take into account psychosocial as well as biomedical factors. For example, the diagnosis of potentially long-term genital infections like genital herpes is more likely to have a significant psychological, social and emotional effect on the patient than the diagnosis of an infection like gonorrhoea or chlamydia, which are easily cured. While individuals vary greatly in their response to an STI diagnosis, there remains a strong undercurrent of stigma in the community around STIs. Patients may feel disproportionate shame and loss of self-worth even when they have a very minor STI (e.g. pubic lice). It is clear that the diagnosis of an STI may bring underlying conflicts and varying degrees of guilt about sex and sexuality to the surface for the first time. In these situations the treatment of the STI may constitute only a small part of the necessary overall management of the patient.

It’s essential, therefore, that the clinician make a brief assessment, at the time of the initial consultation, of the psychological strengths and weaknesses of the patient and his or her psychosocial situation. Consideration of the following issues (many of which are identical to the ones suggested in Chapter 10 in the primary care management of patients with HIV) may be helpful:

- Self-esteem and self-worth
- Perception of stigma associated with STIs
- Family relationships and supports
- Past or present sexual abuse or risk
- Sexuality and patient’s comfort with it
- Compulsive sexual behaviour
- Sexual relationships and related issues of disclosure and safe sex
- Depression and emotional issues (e.g. anger, denial, anxiety, obsessional ideas)
- Drug and alcohol use, especially associated with sex
- Issues concerning pregnancy and motherhood for women
- Beliefs about pharmaceutical drugs (antibiotics in particular)
- Network of friends who may be supportive

An underlying theme which is likely to be of most concern in the patient’s mind if he or she has a regular sexual partner is: how will this STI affect my relationship sexually or emotionally and how will I tell partner/s about this infection. Exploring and helping the patient deal with these issues are very important parts of STI management.

Making a diagnosis and giving treatment

Presumptive diagnosis

It’s generally true that STIs, in their early stages, are simple infections that lend themselves to presumptive diagnosis on the basis of the history of symptoms, the sexual history, the clinician’s knowledge of local STI prevalence and the examination. When patients present with symptoms consistent with one of the STI
syndromes, clinicians should perform appropriate tests, and aim to prescribe treatment for the patient immediately. The rationale for this approach is to relieve symptoms and render the patient non-infectious as soon as possible. It is also important that the patient abstain from any sexual contact until the results are known. One disadvantage of this approach is that some patients will be overtreated, however this is a small price to pay for benefiting the public health and the patient. On the patient’s return visit for results, the clinician is able to give the confirmed diagnosis, discuss whether any further treatment is necessary and discuss contact tracing.

Giving treatment and considering contact tracing

Effective single-dose therapy exists for uncomplicated gonorrhoea, uncomplicated genital chlamydia infection, trichomoniasis, early syphilis and vulvovaginal candidiasis. Treatment for scabies and pubic lice is essentially single-dose topical treatment, although one follow-up treatment several days later may be optimal therapy. Clinicians should use single-dose therapies wherever possible, and should remember that patient management is not complete until contact tracing is addressed and the patient’s sexual partners have also been screened and treated.

Therapy for viral STIs is much less satisfactory and providing a general standardised treatment approach for viral infections is impractical, so for more details readers should consult the management outline below for specific STI syndromes and also national STI treatment guidelines.

Contact tracing (partner notification) is part of the management of almost all STIs. It should be dealt with at the time of initial treatment of the index patient. A good sexual history at the time of testing or screening will determine who needs to be contact traced and also enables the discussion as to how contact tracing can be done. It is the diagnosing clinician’s responsibility to initiate contact tracing. Involving patients in shared responsibility for the management of their sexual partners improves outcomes. Clinicians should use single-dose therapies wherever possible.

Avoiding sex before and during the treatment period is essential. Advising abstinence from sex for 5-7 days after treatment is good practice and allows time for results to be fully assessed and any additional infections detected before sexual activity resumes. With viral STIs, patients need more detailed advice about reducing risk of passing on their infection to others.

Adherence to treatment regimens

Medication must be taken as advised to be effective in the long term. If the patient is not adherent or is taking other complementary medicine which may affect the metabolism of antimicrobial drugs, treatment failure may result.

Vaccination

In non-immune individuals, clinicians should offer hepatitis B vaccine to all patients at risk of STIs, and hepatitis A vaccine to all MSM. Human papillomavirus (HPV) vaccines are available for use in men and women — readers should check the 10th edition of the Australian Immunisation Handbook for recommendations for their use.

Health promotion

Clinicians have a pivotal role in promoting sexual health. Health promotion for STIs consists of giving information about the diagnosed infection (or the likely diagnosis), tips on future prevention, education about STIs in general, including HIV infection, and providing printed resources about STIs, including online addresses of reliable and medically accurate websites (see Chapter 16).

Information giving

In plain, simple, appropriate and language tailored to each patient, the clinician should inform the patient of the diagnosis, or the likely diagnosis. The clinician should explain some facts about the infection, how common it is, how it is transmitted, what the complications may be and what treatment options are available. The clinician should offer simply written brochures, if possible in the patient’s most easily understood language. Brochures are always an additional resource, and not a substitute for an interactive and informative conversation between clinician and patient.

Tips on prevention

It is important that people diagnosed with an STI have a clear understanding of STI transmission so that they know how to reduce the risk of passing their infection on to others, and, if the STI is not curable, how to avoid contracting the same or another STI in future.

• Avoid sexual intercourse and you’ll never catch an STI – true, but impractical in the long term for 99.9% of the sexually active population. In the short term it may have merit.

• Get vaccinated against hepatitis A, hepatitis B and HPV

• Use a condom every time for all penetrative vaginal and anal sex

• Recommend the use of condoms and dental dams during all forms of oral sex for sex workers and others who have multiple sex partners

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• Get vaccinated against hepatitis A, hepatitis B and HPV

• Use a condom every time for all penetrative vaginal and anal sex

• Recommend the use of condoms and dental dams during all forms of oral sex for sex workers and others who have multiple sex partners
• Have a regular STI check-up – STIs are often asymptomatic but are amenable to simple easy treatment
• Talk with your partner about sex; communicate; be honest and open; look at the problem of STIs together and try to negotiate safer behaviours which work for you
• Offer advice to young patients on how to maintain genital hygiene without irritating the skin with too much soap.

Education – imparting general knowledge about STIs
The clinician will need to provide each patient with information about all the STIs relevant to that patient’s situation. The gender of the patient’s partners, the patient’s pattern of sexual behaviour, her or his willingness to use protection, as well as the local prevalence of STIs, will provide some guide. Generally, in Australia and New Zealand, exclusively heterosexual, urban patients need detailed information about HPV, chlamydia, genital herpes and bacterial vaginosis because they are common and brief information about HIV, gonorrhoea and syphilis because of the real threat they pose to health. MSM need detailed information about all common STIs as well as hepatitis A and B. Indigenous heterosexual patients need information about all common STIs which are prevalent in their local communities. Women who have sex with women (WSW) need the same information as heterosexual women, with clear explanation that any sexual contact with men places them at the same STI risk as exclusively heterosexual women.

Resources for health-care professionals and people with, or at risk of, STIs
There is a wide range of resources available to support clinicians and patients. All state and territory health departments have printed information about all STIs, as do major sexual health clinics (see Chapter 16 for links to online information). ASHM distributes the Contact Tracing Manual which includes a good summary of all the STIs and has a website providing information for clinicians and patients which is regularly updated (http://www.ashm.org.au).

Management of specific STI syndromes
1. Urethral discharge

Description and causes
Urethral discharge denotes the existence of urethritis. A gram-stained smear of urethral discharge in urethritis will show > 4 leucocytes on microscopy. For all practical purposes, any discharge from the urethra is abnormal and signals the possible presence of an STI. Thick purulent and profuse discharge with some dysuria is often due to gonorrhoea. Thin, scant, clear or mildly mucopurulent discharge with slight urethral irritation is often due to chlamydia. A similar discharge with marked dysuria can occur in a primary herpetic urethritis. Trichomonas vaginalis mostly causes entirely asymptomatic urethritis but sometimes initiates a very slight mucopurulent discharge. While the appearance of the discharge may be useful in taking a syndromic approach to management appropriate tests need to be performed to confirm the organisms involved.

Some other sexually transmissible microbial agents cause urethritis (Table 13.1), of which Mycoplasma genitalium is probably the most significant, but there are no commercial laboratory tests available as yet for this bacterium however some public laboratories offer testing with in-house nucleic acid amplification tests (NAATs). At least half of the urethritis seen in practice is due to chlamydia or gonococcus. It is also important to recognise that urethritis can be caused by organisms in the mouth and throat such as cytomegalovirus (CMV), adenovirus and streptococcus. Most urethritis responds to antichlamydial treatment or gets better on its own. If it fails to do so, the clinician should reassess the patient carefully.

**TABLE 13.1 STI causes of urethral discharge (in order of frequency)**

<table>
<thead>
<tr>
<th>STI caused by</th>
<th>Description and causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlamydia trachomatis (serovars D–K)</td>
<td>Adenoviruses</td>
</tr>
<tr>
<td>Neisseria gonorrhoeae</td>
<td>Herpes simplex virus types 1 or 2</td>
</tr>
<tr>
<td>Mycoplasma genitalium</td>
<td>Neisseria meningitidis</td>
</tr>
<tr>
<td>Ureaplasma urealyticum</td>
<td>Enteric bacteria</td>
</tr>
<tr>
<td>Trichomonas vaginalis</td>
<td></td>
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</tbody>
</table>

Differential diagnosis
There is no non-STI differential diagnosis for urethral discharge, except perhaps the very rare situation where a gastrointestinal infection triggers urethritis as part of the triad of urethritis, conjunctivitis and reactive arthritis (formerly known as Reiter’s syndrome).

Diagnostic tests
The patient should produce an FPU specimen for NAATs for chlamydia (and for gonorrhoea in areas where it is prevalent). If gonorrhoea is suspected and a urethral discharge is present, some discharge should be collected for culture and antibiotic sensitivity testing on a cotton or Dacron swab. There is rarely a need to insert swabs into the urethral meatus (Table 13.2). The clinician should only consider additional tests such as trichomoniasis, HSV, adenoviruses and Mycoplasma genitalium if symptoms fail to respond to initial treatment.
### TABLE 13.2 Diagnostic tests for STIs

<table>
<thead>
<tr>
<th>Ano-rectal junction</th>
<th>Blood culture</th>
<th>Cervix</th>
<th>Endocervical canal</th>
<th>Faeces</th>
<th>High vaginal swab (HVS)</th>
<th>Joint aspirate</th>
<th>Rash (ano-genital)</th>
<th>Rectal mucosa</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Anal cytology for people living with HIV and MSM (if available – currently only in specialist centres)</td>
<td>• For culture and sensitivity for Neisseria gonorrhoeae</td>
<td>• Sample from ectocervix and endocervical canal for cytology (Papanicolaou smear) (in accordance with NHMRC guidelines – usually two years after sexual debut and then regularly every two years)</td>
<td>• Swab smeared onto a slide for gram-stain microscopy for inflammatory cells and diplococci</td>
<td>• Stool samples (X2) for microscopy for leucocytes, red cells, ova, cysts and parasites (including concentrate microscopy and permanent stains and Cryptosporidium/Giardia antigen test), plus culture and sensitivity (X1)</td>
<td>• Swab smeared onto a slide for gram-stain microscopy for number of leucocytes, presence of clue cells (bacterial vaginosis), spores and hyphae (candidiasis)</td>
<td>• Sample of aspirate for microscopy, culture and sensitivity for Neisseria gonorrhoeae</td>
<td>• Swab from affected area (e.g. vulva, perianal area or under the foreskin) smeared onto a slide for gram-stain microscopy for spores and hyphae (candidiasis)</td>
<td>• Swab collected by direct vision smeared onto a slide for gram-stain microscopy for inflammatory cells and diplococci (not a useful test if swab has been taken blind)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Swab in transport medium for culture and sensitivity for gonorrhoea</td>
<td>• Swab for NAAT for chlamydia</td>
<td>• Swab for NAAT for trichomoniasis (if available)</td>
<td>• Swab in transport medium for culture and sensitivity (for candidiasis and for bacteria; if no NAAT test for trichomoniasis, a wet preparation can be made from this swab to look for motile trichomonads)</td>
<td></td>
<td>• Swab from affected area in transport medium for culture and sensitivity (for candidiasis and for bacteria)</td>
<td>• Swab for NAAT for chlamydia (preferably by direct vision, otherwise blind) – routine tests are for D–K serovars; some specialist laboratories are able to test for L1–L3 serovars</td>
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<tr>
<td></td>
<td></td>
<td>• Swab for NAAT for herpes simplex (only if there is a relevant sexual history, other evidence of herpes externally, or if cervix looks ulcerated)</td>
<td></td>
<td>• HVS swab for NAAT for trichomoniasis if vulval rash (if no NAAT test available, send swab in transport medium, a wet preparation can be made from this swab to look for motile trichomonads and culture is also possible)</td>
<td></td>
<td></td>
<td>• HVS swab for NAAT for trichomoniasis if vulval rash (if no NAAT test available, send swab in transport medium, a wet preparation can be made from this swab to look for motile trichomonads and culture is also possible)</td>
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<td></td>
<td>• Microscopy for scabies mite or pubic louse (if appropriate)</td>
<td>• Microscopy for scabies mite or pubic louse (if appropriate)</td>
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<td>• Microscopy for scabies mite or pubic louse (if appropriate)</td>
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<td></td>
<td></td>
<td></td>
<td>• Punch biopsy for histology if diagnosis is uncertain</td>
<td>• Punch biopsy for histology if diagnosis is uncertain</td>
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### Initial treatment of urethral discharge

Patients with urethral discharge should be treated on the initial visit after tests have been collected. Azithromycin 1 g orally as a single dose is the recommended initial treatment. In addition, a single dose of ceftriaxone 500 mg intramuscularly should be given in the following situations:

- Where a partner is known, or likely to have a gonococcal infection
- In areas and communities where gonorrhoea is highly prevalent (e.g. remote Indigenous communities)
- When the patient has recently had sex with a local person in an overseas country where gonorrhoea is highly prevalent (e.g. South East Asia)
- When the patient gives a history of male-to-male sex (oral or anal)

- Contact tracing and treatment of sexual partners should occur when test results have confirmed diagnosis

See Table 13.3 for treatments for specific STIs. For urethral discharge, treatments are for:

- Gonorrhoea
- Chlamydia
- *Mycoplasma genitalium*
- Trichomoniasis
- Herpes simplex

Consult the *National management guidelines for sexually transmissible infections* and Figures 13.2 and 13.3 and Table 13.2.2,3,9
2. Vaginal discharge

**Description and causes**

There is a normal physiological discharge from the vagina which varies in character and consistency during the course of the menstrual cycle.

An abnormal vaginal discharge may come:

- from the endometrial lining associated with endometritis – this is uncommon except after childbirth or following some gynaecological procedures (insertion of an intrauterine device [IUD], dilatation and curettage [D & C], termination of pregnancy).
- from the cervical canal – gonorrhoea and chlamydia are the most common infective causes of discharge but herpetic and trichomonal cervicitis are also possible causes. *Mycoplasma genitalium* can cause cervicitis and should be considered if chlamydia and gonorrhea tests are negative. There needs to be a significant volume of purulent or mucopurulent exudate from the cervix before a noticeable change is detected in the appearance of the vaginal discharge.

From the vagina itself where the three common causes are trichomoniasis, candidiasis or bacterial vaginosis; bacterial vaginosis is by far the commonest cause. Herpetic vaginal ulceration may also cause discharge.

#### TABLE 13.3 Treatments for specific STIs

<table>
<thead>
<tr>
<th>Condition</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bacterial vaginosis</strong></td>
<td>Tinidazole 2 g orally as a single dose</td>
</tr>
<tr>
<td></td>
<td>Metronidazole 400 mg twice a day for 7 days is superior to tinidazole as a single dose; metronidazole is preferred in pregnancy (equally effective)</td>
</tr>
<tr>
<td><strong>Herpes</strong></td>
<td>Initial therapy, herpetic urethritis and cervicitis or a moderately severe outbreak:</td>
</tr>
<tr>
<td></td>
<td>Valaciclovir 500–1000 mg twice a day orally for 5–10 days or aciclovir 400 mg three times a day orally for 5–10 days; equally effective Suppressive therapy (continuous):</td>
</tr>
<tr>
<td></td>
<td>• Valaciclovir 500 mg orally daily or famiclovir</td>
</tr>
<tr>
<td></td>
<td>• 250 twice a day orally; equally effective</td>
</tr>
<tr>
<td><strong>Candidiasis</strong></td>
<td>Fluconazole 150 mg orally as a single dose (don't use in pregnancy); and/or a topical vaginal anti-candidal preparation for vulvovaginitis and a topical anti-candidal cream for balanoposthitis. Recurrent vulvovaginal candidiasis may need treatment for longer periods</td>
</tr>
<tr>
<td><strong>IGV – <em>Chlamydia trachomatis</em> serovars I–I3</strong></td>
<td>Doxycycline 100 mg twice a day orally for a minimum of 21 days</td>
</tr>
<tr>
<td><strong>Chancroid</strong></td>
<td>Ceftriaxone 500 mg intramuscularly as a single dose</td>
</tr>
<tr>
<td><strong>Pubic lice:</strong></td>
<td>Benzylbenzoate 25% lotion – apply to all affected hairy areas at bed time. Avoid direct contact with scrotum. Wash off next morning. Repeat in 5 days</td>
</tr>
<tr>
<td><strong>Chlamydia – <em>Chlamydia trachomatis</em> serovars D–K</strong></td>
<td>Azithromycin 1 g orally as a single dose; Alternative: Doxycycline 100 mg twice a day orally for 7 days (don’t use in pregnancy); single dose treatment should always be used in preference to this regimen</td>
</tr>
<tr>
<td><strong>Scabies</strong></td>
<td>Permethrin 5% cream – apply in a single application at bed timetopically to whole body except head, Wash off next morning</td>
</tr>
<tr>
<td><strong>Donovanosis</strong></td>
<td>Azithromycin 1 g orally as a single dose. Repeat at weekly intervals for 4–6 weeks</td>
</tr>
<tr>
<td><strong>Syphilis (primary or secondary)</strong></td>
<td>Benzathine penicillin 1.8 g (2.4 million international units) intramuscularly as a single dose – this means 2 injections of 0.9 g in each buttock OR Procaíne penicillin 1.5 g intramuscularly daily for 10 days (both equally effective and safe in pregnancy) Alternative: Doxycycline 100 mg twice a day orally for 14 days (do not use in pregnancy) NB: If patient is a pregnant woman allergic to penicillin, consult a sexual health physician for advice</td>
</tr>
<tr>
<td><strong>Enteritis</strong></td>
<td>Treat appropriately for the specific agent isolated i.e. for giardiasis, amoebiasis, shigellosis</td>
</tr>
<tr>
<td><strong>Trichomoniasis</strong></td>
<td>Tinidazole 2 g orally as a single dose or metronidazole 2 g orally as a single dose (metronidazole preferred in pregnancy); equally effective</td>
</tr>
</tbody>
</table>

*NB: If patient is a pregnant woman allergic to penicillin, consult a sexual health physician for advice.*
More than one condition might cause an abnormal vaginal discharge (Table 13.4). Discharge associated with gonococcal cervicitis may be frankly purulent; discharge caused by trichomoniasis may be heavy green and frothy; discharge due to candidiasis may be like cottage cheese accompanied by vulval erythema, oedema and itching; and discharge due to bacterial vaginosis is usually thin, white-grey and slightly frothy.

**Table 13.4** STI causes of vaginal discharge (in order of frequency)

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial vaginosis</td>
</tr>
<tr>
<td>Vaginal candidiasis</td>
</tr>
<tr>
<td>Chlamydia trachomatis serovars D–K</td>
</tr>
<tr>
<td>Herpes simplex virus types 1 and 2</td>
</tr>
<tr>
<td>Neisseria gonorrhoeae</td>
</tr>
<tr>
<td>Trichomonas vaginalis</td>
</tr>
<tr>
<td>Mycoplasma genitalium</td>
</tr>
</tbody>
</table>

**Differential diagnosis**

Apart from STIs and bacterial vaginosis, other causes of abnormal vaginal discharge are:

- Leucorrhoea (increase in quantity of normal vaginal discharge) – may be associated with hormonal changes, use of the contraceptive pill, hormone replacement therapy
- Other infections – e.g. group B streptococcal infection (although also considered normal flora) (discharge then often resembles the discharge seen with *Trichomonas vaginalis*, *Staphylococcus aureus* (toxic shock syndrome)
- Retained foreign bodies (such as condoms and tampons) resulting often in a foul smelling discharge
- Neoplastic disease (carcinoma of endometrium, cervix, urethra, vagina) – these can cause a blood-stained discharge

**Diagnostic tests**

In the presence of an abnormal vaginal discharge, the clinician should undertake an internal examination using a vaginal speculum and take tests at the same time.

During the internal examination, the clinician should take high vaginal swabs (HVS) for bacterial vaginosis, trichomoniasis and candidiasis and swabs from the endocervical canal for gonorrhoea and chlamydia.

In cases of recurrent candidiasis, request culture on an HVS as it is useful to identify the species of Candida and its sensitivity to antifungal agents. A cervical test for herpes can be taken if there is evidence of herpes externally or if the cervix looks ulcerated. Although not ideal practice, if the patient declines an internal examination self-collected vaginal swabs can be sent for testing for bacterial vaginosis, trichomoniasis and candidiasis and for PCR for chlamydia and gonorrhoea. An FPU specimen is the least useful specimen for detection of chlamydia and gonorrhoea in women with vaginal symptoms. Throat swab and anal swab for PCR should also be considered depending on the patient’s sexual practices. The clinician should also arrange other serological tests as appropriate (See Figure 13.2 and 13.3).

For Initial treatment of vaginal discharge, refer to Table 13.3, the National management guidelines for sexually transmissible infections and the categories of drugs for use in pregnancy.

3. **Ano-genital ulcer disease (GUD)**

**Description and causes**

Ulceration may be accompanied by inguinal lymphadenitis; palpation of both groin areas is an integral part of the examination of all GUD. Trauma is a frequent cause of short-lived genital ulceration, but a number of STIs can present as ulceration in the ano-genital region. These include genital herpes, primary and secondary syphilis, lymphogranuloma venereum (LGV) (due to *Chlamydia trachomatis* serovars L1–L3), chancroid (due to *Haemophilus ducreyi*) and donovanosis (due to *Klebsiella granulomatis*). GUD can be painful (herpes and chancroid) or painless (syphilis, LGV and donovanosis). In Australia and New Zealand, LGV, chancroid and donovanosis are rare. There are two exceptions: LGV is an infection seen now in Australasia in some MSM but in this group, LGV mostly manifests as an acute proctitis rather than as GUD; donovanosis still occurs (but now rarely) in remote Indigenous communities in northern and central Australia. As a rule of thumb, all GUD in Australia and New Zealand is due to genital herpes until proven otherwise, but clinicians should always ask themselves: ‘could this GUD be syphilis?’

**Differential diagnosis**

Other causes of ano-genital ulceration are:

- Trauma
- Scratched scabies lesions on the genitals
- Anal fissures and fistulae
- Herpes varicella zoster lesions involving the ano-genital region
- Neoplastic lesions: precancerous lesions (Bowen’s disease), squamous cell carcinoma, basal cell carcinoma
### STI Testing Tool

**Who?**
- A sexually active young person under 25 years.
- An asymptomatic person of any age requesting a "STI check-up".
- A man who has sex with men (MSM).
- A nurse midwife.
- A person with symptoms.

**Why?**
- This population is at higher risk for Chlamydia.
- This population is at higher risk for Gonorrhoea.
- This population is at higher risk for Chlamydia and Gonorrhoea.
- This population is at higher risk for Chlamydia, Gonorrhoea, THP, HSV, HIV.
- This population is at higher risk for Chlamydia, Gonorrhoea, THP, HSV, HIV and HCV.

**Which?**
- Chlamydia (CHL)
- Gonorrhoea (GON)
- HSV
- HIV
- HPV
- Syphilis (TPH)

**How?**
- First test is on CHL.
- Consider vaccination for HIV & HPV.
- Self-collected vaginal swab (OR)
- Self-collected rectal swab (OR)
- Condom-based rectal swab.
- Blood.

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### Getting started with an STI discussion

**Bringing the patient up opportunistically**
- "Mr Jackson, I’ve noticed you haven’t had an STI check-up before. Would you like to have one while you’re here or find out more about Chlamydia?"

**Using a ‘hook’**
- "Have you heard about HIV or HPV recently? They protect against infections that can be sexually transmitted, perhaps we could discuss these while you’re here?"

**As part of a reproductive health consultation**
- "Since you’re here today for a pap smear, I’d like to talk about contraception; could we also talk about some other aspects of women’s health such as an STI check-up?"

**Because the patient requests a ‘checkup’ for STIs**
- "It’s a good idea to talk about your sexual activity now and consider what tests your partner(s) might also need to do, too."

**Brief Sexual History**
- "Let’s talk about your most recent sexual activity. Who are you thinking about?"
- "Are you currently in a relationship?"
- "How long have you been together?"
- "How many sexual partners have you had?"
- "How many partners have you had in the past 12 months?"
- "Sexual activity with a girl friend or boyfriend?"
- "Was this the first time you had vaginal sex?"
- "Were you or your partner(s) under the influence of alcohol or drugs?"
- "Have you or your partner(s) been diagnosed with an STI?"
- "Is there anything else you’re concerned about?"

**Other risk behaviours**
- "Have you had any injecting drug use?"
- "Have you been treated for an STI before?"
- "Have you ever been in care?"

**Consent**
- "I think we need to talk about Chlamydia."

**Contact tracing**
- "I’ll call for more information."

**Support**
- "If you need support, there are various services available to you."

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*Figures 13.2 GP STI Testing Tool*
Reproduced with permission by NSW STI Programs
FIGURE 13.3 Australian sexually transmissible infection and HIV testing guidelines for asymptomatic men who have sex with men 2014

(Used with permission from Templeton DJ, Read P, Varma R, et al.)
### Rationale for Key STI Testing Recommendations

**Use of *Neisseria gonorrhoeae* (NG) and *Chlamydia trachomatis* (CT) NAATs.** NAATs are now widely used in Australia when testing urine, urethral, anorectal and pharyngeal samples for NG and CT. Where possible, collect NG culture swabs before treatment of NAAT-confirmed NG to assess antibiotic sensitivity of the NG isolate.

**Anal NG and CT infections.** All MSM should be offered anorectal swabs even if they do not report receptive anal sex. Receptive anal sexual practices such as receptive fingering, toy insertion or oral-anal sex are risk factors for anal NG and CT, even in men who use condoms for receptive anal intercourse. Anal STIs are also independent risk factors for HIV in HIV-negative MSM, so identification and treatment of anal STIs by regular testing is likely to reduce the risk of HIV acquisition.

**Pharyngeal NG and CT infections.** Most occur without concurrent anogenital infection, are asymptomatic, and can be the source of anogenital infections among MSM. Compared with pharyngeal NG, pharyngeal CT is relatively rare among Australian MSM. However, recent studies overseas have identified a higher prevalence of pharyngeal CT among MSM than previously reported and pharyngeal CT is likely to be long-lasting in the absence of treatment. Testing MSM for both pharyngeal infections is therefore recommended.

**Self-collected samples** (urine, urethral meatal, pharyngeal and anorectal swabs) are acceptable and effective at detecting NG and CT using NAATs.

Repeat testing. Repeat testing at 3 months after NG and CT infections is recommended to detect reinfection.

Testing in HIV positive MSM, MSM with HIV account for up to 50% of infectious syphilis notifications and mathematical modelling indicates 3-monthly syphilis testing of these MSM could significantly impact on syphilis control efforts within Australia. HIV-positive MSM are also at particularly high risk of anal NG & CT, thus more frequent STI testing should be encouraged in this group. Due to evidence of Hepatitis C (HCV) sexual transmission among HIV positive MSM, all asymptomatic HIV positive MSM should have annual HCV testing.

Testing reminders. SMS and email reminders have been shown to increase detection of STIs as well as increasing retesting rates among MSM, both, after an STI diagnosis, and as reminders for regular testing. Therefore STI/HIV testing reminders are recommended for MSM.

### Rationale for Exclusions from STI Testing Recommendations

**Urethral NG** is extremely rare among Australian MSM tested in the absence of urethral symptoms. Although most commercially available NAAT for Chlamydia are dual assays which also test for NG, current evidence does not support a recommendation to test asymptomatic Australian MSM for urethral NG.

**Lymphogranuloma venereum (LGV).** In Australia and the majority of overseas settings, a substantial reservoir of asymptomatic LGV has not been identified. Therefore routine LGV typing of asymptomatic chlamydia infections among MSM is not currently justified.

**Hepatitis C virus** in HIV negative MSM who have never injected drugs is rare, therefore routine testing is not recommended for this group.

**Herpes simplex virus (HSV) type-specific serology.** Anogenital HSV-1 and -2 infections are highly prevalent in MSM, and HSV-2 increases the risk of acquiring and transmitting HIV. However, serological HSV diagnosis has not been shown to result in behaviour change or reduce onward HSV transmission, and HSV-2 therapy has not been shown to reduce the risk of HIV acquisition. Therefore testing asymptomatic MSM is not recommended.

**Human papillomavirus (HPV).** Most MSM, especially those with HIV, are infected with one or more types of HPV. The utility of anogenital sampling for HPV DNA or serological testing among MSM has not been established. Prospective Australian studies which include assessing the utility of anal HPV testing to predict risk of anal pre-cancerous lesions are ongoing and are expected to guide future recommendations. Testing asymptomatic MSM is not currently recommended.

Anogenital *Mycoplasmata genitalium* is uncommon in asymptomatic Australian MSM. Evidence for a role of *M. genitalium* in ascending male genital infections is lacking and there is no evidence that *M. genitalium* colonizes or infects the pharynx of MSM. Further studies are required to understand the contribution of *M. genitalium* to anogenital clinical syndromes and its impact on HIV acquisition among MSM, before consideration is given to routine testing in MSM.

**Trichomonas vaginalis** rarely colonizes the pharynx or anogenital mucosa of MSM, even in settings where the heterosexual community prevalence of *T. vaginalis* is substantial. Testing for *T. vaginalis* among asymptomatic Australian MSM is therefore not recommended.

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**STI Information for MSM and resources to inform practice:** [www.thedramadownunder.info](http://www.thedramadownunder.info)

**Australian STI Management Guidelines:** [www.sti.guidelines.org.au](http://www.sti.guidelines.org.au)

**Date of Issue:** APR 2014
**Diagnostic tests**

The recommended tests for any ulceration in the ano-genital region are swabs of the lesion for HSV (NAAT) and syphilis (NAAT). Few laboratories now perform dark field microscopy for syphilis. Some laboratories offer a ‘multiplex’ NAAT for GUD which tests for HSV, *Haemophilus ducreyi* and *Treponema pallidum* (and in some parts of remote Australia, for *Klebsiella granulomatis*) on the one swab. Clinicians should use these tests if they are available and if syphilis is a reasonable possibility. If, from the history, there are grounds for believing GUD may be due to chancroid, LGV or donovanosis, the clinician should discuss the case with the local laboratory and a sexual health physician.

Serological tests for syphilis (treponemal antibody test and / or RPR) should be arranged for any patient with ano-genital ulceration.

The clinician should arrange other serological tests as appropriate, after discussion with the patient (HIV, HAV, HBV, HCV), a throat swab for gonorrhoea NAAT, rectal swabs for NAAT for chlamydia and gonorrhoea. See Tables 13.1 and 13.4.

**Initial treatment of ano-genital ulcer disease**

HSV-1 or HSV-2 causes most GUD either on the penis, the vulva, around the introitus or perianally. Primary or initial outbreaks of herpes are painful. Both local and systemic analgesics as well as frequent bathing with luke-warm saline are helpful adjuncts to more specific treatment.

Early treatment with an antiviral agent relieves symptoms, decreases risk of transmission to sexual partners and reduces the length of the outbreak. Clinicians should treat at once on clinical suspicion if lesions are causing the patient discomfort. Recommended initial treatment, pending results of NAAT, is:

Valaciclovir 500–1000 mg orally twice a day or 5–10 days. Aciclovir 400 mg three times a day for 5-10 days is also effective and inexpensive.

Consider treatment for early syphilis (see Table 13.3) in the following situations:

• In communities where syphilis is highly prevalent

• Where the GUD is a single painless indurated ulcer with enlarged non-tender inguinal nodes

• In MSM who have GUD which is not typical of herpes

Patients treated for syphilis require a repeat RPR test at 3 months, 6 months, 12 months and 24 months to check RPR titre is falling. Patients diagnosed with ano-genital herpes may require referral for ongoing counselling.

**TABLE 13.5 Follow-up after treatment for STIs**

<table>
<thead>
<tr>
<th>Clinicians should ask patients to return for follow-up in 7–14 days so they can:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Give the results of tests, so that the patient knows what STIs are present</td>
</tr>
<tr>
<td>Check the response to initial treatment has been successful; in the case of ano-genital warts, mostly further treatments will be necessary</td>
</tr>
<tr>
<td>Check adherence to medication (if necessary)</td>
</tr>
<tr>
<td>Check whether sexual contacts have been contacted and treated appropriately</td>
</tr>
<tr>
<td>Provide further information and education; essential in the case of both ano-genital herpes and syphilis and often with HPV infection</td>
</tr>
<tr>
<td>Arrange further visits to check progress (LGV proctitis, enteritis) or to arrange further serology, e.g. syphilis (if necessary)</td>
</tr>
<tr>
<td>Arrange for a further STI screen in 3 months</td>
</tr>
</tbody>
</table>

**4. Ano-genital lumps and bumps**

**Description and causes**

There are only two STIs causing lumps and bumps in the ano-genital region (apart from the lumps or nodules characteristic of scabies when burrows occur in the soft skin of the penis or labia; itch is still the major symptom in this infestation). The two STIs are genital warts (due to HPV, usually types 6 and 11) and molluscum contagiosum (due to the *Molluscipoxvirus*). Molluscum contagiosum have a characteristic smooth round igloo shape often with central indentation. They have a yellowish waxy colour. They favour the skin of the supra-pubic region, the inner thighs, shaft of the penis and hair-bearing areas of the vulva. Warts can occur anywhere at all on the skin and mucous membrane of the ano-genital region, especially under the foreskin, on the inner surfaces of the labia minora, in the fourchette, and perianally. Warts vary greatly in morphology, ranging from small flat plaques to filiform lesions to large highly keratinised papules. Warts can also vary in colour and degree of pigmentation. There may be only one or two warts or large warty colonies.

**Differential diagnosis of ano-genital lumps and bumps**

Non-STI causes of lumps and bumps are usually anatomical variants such as pearly penile papules, Fordyce spots, Tyson’s glands, lymphochoes and angiokeratomas. The reader should consult a sexual health or dermatology text to familiarise themselves with these common lesions. In the perianal area, the condylomata lata of secondary syphilis, haemorrhoids,
thrombosed external piles, perianal abscesses and sentinel piles are all part of the differential diagnosis (Figures 13.4 and 13.5).

**Diagnostic tests**

Clinicians mostly diagnose ano-genital warts and molluscum contagiosum purely on their characteristic appearance. When lesions look atypical, or fail to respond to usual forms of treatment, it is wise to do an excision or punch biopsy for histological diagnosis.

**Initial treatment for ano-genital lumps and bumps**

Heavy growths of warts in places like the introitus or around the anus become irritated, smell offensive and can be extremely difficult to live with. Heterosexual men may feel embarrassed if they have developed extensive perianal warts without ever having had receptive anal intercourse (Case study 2).

Treatment of warts and molluscum contagiosum is partly for cosmetic reasons, to remove the unsightly and uncomfortable lesions for the patient’s psychological wellbeing, as well as the possibility that treatment will stimulate local immune defences which may reduce the likelihood of recurrence and the length of time during which the patient remains infectious to sexual partners. No matter what form of treatment is used, there is a recurrence rate of about 40%.

Warts tend to be less responsive to treatment in people who are immunosuppressed (e.g. in people with HIV infection). With all patients with warts, start treatment as soon as possible.

**Treatments for specific STIs causing ano-genital lumps and bumps**

**Ano-genital warts**

These are the available treatments for ano-genital warts:

- **Excision**: simple surgery under local anaesthesia (general anaesthesia is rarely needed); safe in pregnancy
- **Ablative, using various modalities**: cryotherapy using liquid nitrogen, CO2 slush, or a cryotip and nitrous oxide (usually with local anaesthetic cream such as xilocaine or emla); diathermy under local or general anaesthesia; laser therapy under local or general anaesthesia; and application of trichloracetic acid (only suitable for single small warts); all safe in pregnancy
- **Antimitotic agents**: podophyllotoxin cream or paint (lends itself to patient application according to manufacturer’s instructions); 5-fluorouracil cream (should only be applied by the clinician and should be washed off after 4 hours); neither treatment to be used in pregnancy
- **Immunomodulatory agent**: imiquimod cream (apply three times weekly at bed time, wash off in the morning, for up to 8–12 weeks); not to be used in pregnancy

No treatment is entirely satisfactory or completely effective; recurrences can occur after any treatment. Quitting smoking is a very important part of treatment if warts are proving recalcitrant. However, except in the severely immunosuppressed patient (HIV, and sometimes in pregnancy), with patience and persistence all warts will regress eventually.
Mollusca contagiosa

There is generally a better early success rate treating mollusca than treating warts. These are the available treatments:

- Deroofing the individual lesion accompanied by squeezing out the firm cheese-like contents (ideal if there are only a few lesions; it’s easy to teach patients to do this themselves)
- Ablative therapies as for warts (all work fairly well for mollusca)

Treatment can be a problem in the severely immunosuppressed, when mollusca can sometimes grow quite large and remain fairly unresponsive to usual treatments.

5. Ano-rectal syndromes

Description and causes

An ano-rectal syndrome is present when a patient reports anal symptoms (i.e. itch, pain, discomfort or irritation) or disturbed bowel function and there’s a possibility the symptoms are related to anal sexual activities. Many other conditions, both medical and surgical, can also cause such symptoms. Many STIs affecting the ano-rectum are asymptomatic. Here are some possible sexually transmissible agents associated with STI-related ano-rectal syndromes, in order of their frequency in practice:

- HSV types 1 or 2 – causes ulceration and proctitis, often asymptomatic, but can cause anal pain and constipation, anal discharge and sometimes bleeding, especially in an initial outbreak
- Chlamydia trachomatis serovars D–K – generally asymptomatic, causes mild proctitis, so sometimes causes anal discomfort and irritation
- HPV all genital types – generally asymptomatic, types 6 and 11 may cause warts
- Neisseria gonorrhoeae – often asymptomatic; causes a moderate proctitis; astute patients may notice purulent discharge around their bowel motions; can cause anal discomfort or mild pain
- Treponema pallidum – often asymptomatic but anus may be site of a painless primary chancre (ulcer), and ano-rectal mucosa may be affected by snail-track ulcers in secondary syphilis. Condylomata lata (flat warty or skin tag-like moist excrescences around the anus) also occur in secondary syphilis
- Chlamydia trachomatis serovars L1–L3 (LGV) usually causes a moderate to severe proctitis characterised by deep anal pain, increased frequency of bowel action and passage of mucopurulent discharge, plus systemic symptoms
- Enteric micro-organisms – there are a variety of which the commonest are Shigella sp, Giardia duodenalis, Entamoeba histolytica and HAV. HAV causes pale stools and hepatitis; all other enteric agents cause diarrhoea of varying severity. In immunosuppressed patients with HIV, a number of other enteric infections may occur (cryptosporidiosis, microsporidiosis, Mycobacterium avium complex (MAC) complex), not usually sexually acquired (Chapter 10)

Differential diagnosis

Here is a list of some non-STI causes of symptoms which mimic the STI ano-rectal syndrome:

Medical

- Enteric infections acquired in conventional rather than sexual modes
- Crohn’s disease
- Ulcerative colitis

Surgical

- Traumatic lesions of anus and rectum
- Retained foreign bodies (dildoes)
- Ano-rectal benign and malignant neoplasms
- Fissures, fistulae, thrombosed external piles and haemorrhoids

The take-home message is that not all ano-rectal symptomatology is surgical or routinely medical. Ask yourself, and not just in gay men: ‘could this be an STI?’

Diagnostic tests

The patient with the ano-rectal syndrome needs a careful examination of the anal and perianal area. Ideally tests can be taken at the same time.

If the clinician suspects an enteric infection because the predominant symptom is diarrhoea or loose bowel actions, then the patient should collect the usual stool specimens for microscopy for ova, cysts and parasites (OCP) and faecal culture. Current guidelines recommend two stool OCP exams (including concentrate microscopy and permanent stains and Cryptosporidium and Giardia antigen test) plus one faecal culture. If results are negative, it is recommended to test again over 1 week later if symptoms persist.

If proctitis is suspected, the rectal mucosa should be viewed directly through an anoscope or proctoscope; sometimes this is not possible because of anal pain. The clinician should examine for traumatic lesions and the possibility of a retained foreign body. The recommended tests are swabs from lesions or ulcers for HSV and syphilis NAAT; swabs from the rectal mucosa (preferably collected through a proctoscope by direct vision unless too painful) for gonococcal
culture and NAAT, HSV NAAT and chlamydia NAAT. If chlamydia is detected all isolates from the rectum are checked for LGV serovars. Anyone with an STI-related ano-rectal syndrome may be at risk for syphilis, HIV and HAV. The clinician should arrange other serological tests as appropriate after discussion with the patient (HIV, HBV, HCV). The clinician should take a throat swab if the patient has a history of performing fellatio and there is a high local prevalence of gonorrhoea.

**Initial treatment of ano-rectal syndromes**

Refer to the National management guidelines for sexually transmissible infections. After tests have been taken, the clinician must provide treatment to relieve the patient’s symptoms.

If there is a proctitis (with mild-to-moderate pain) and an inflamed-looking mucosa with or without obvious purulent material lying on the mucosal surface (as seen by proctoscopy):

- Treat for gonorrhoea and chlamydia (D–K serovars)
- If there is severe proctitis (considerable pain) and very inflamed mucosa with systemic symptoms:
  - Treat for gonorrhoea
  - Treat for an initial outbreak of herpes
  - Treat for chlamydia with an extended course of antibiotics in case LGV is isolated.
- If there is enteritis (diarrhoea):
  - Treat symptomatically as for any other enteric infection with fluid replacement and loperamide pending results of tests

**Treatments for specific STIs causing ano-rectal syndromes**

See Table 13.3 for a list of treatments for specific STIs. Also refer to the National management guidelines for sexually transmissible infections.

Clinicians should ask patients who are unwell, as may be the case with gonococcal, herpetic and LGV proctitis, to return in 3 days for review.

**6. Pelvic pain syndrome in women**

**Description and causes**

Pelvic pain in women is a common presenting symptom. Urinary tract infections, gastrointestinal conditions, as well as a variety of gynaecological conditions can present with either acute or chronic pelvic pain. A number of sexually transmissible agents (Chlamydia trachomatis serovars D–K and Neisseria gonorrhoeae mostly, but probably also Mycoplasmas) may be responsible. After a period of silent cervical infection, these STIs can ascend, often around the time of menstruation, through the dometrium causing infection initially in the mucosa of the fallopian tubes (salpingitis), with subsequent spread through the wall of the tube causing infection in surrounding structures—ligaments, serosa and ovaries (PID). Pelvic pain due to PID varies greatly in severity, ranging from asymptomatic to extremely mild (chlamydial PID) to quite severe, accompanied by systemic symptoms (gonococcal PID). Initially, STI-related salpingitis and PID is an infection caused by one or two STIs; however, once the cervical barrier has been breached, very quickly other micro-organisms ascend from the vagina in the wake of the STIs, causing a mixed aerobic and anaerobic infection.

**Differential diagnosis**

The differential diagnosis of female pelvic pain is too broad for this monograph. The main diagnosis not to miss is an ectopic pregnancy. For this purpose, the menstrual history is vital, including the date of onset of the last normal period, plus details of any intermenstrual bleeding or spotting or post-coital bleeding. A good sexual history is also essential, including the precise date of the last unprotected sex with a male partner. It is relatively simple to diagnose non-STI causes of PID, e.g. history of recent delivery and post-partum endometritis, gynaecological procedures or surgery, and PID secondary to an acute appendicitis. To assist accurate diagnosis, here are some common characteristics of STI-related PID, and some common clinical features which would suggest another diagnosis:

- Common characteristics of STI-related PID:
  - Recent risk factors for contracting an STI
  - Patient under 29 years of age
  - Past history of STIs
  - Gradual onset of pain
  - Ill-defined pain: few or no systemic symptoms
  - Deep dyspareunia
  - Abnormal vaginal discharge, irregular menstrual bleeding, postcoital bleeding
  - No associated gastrointestinal tract symptoms
  - No urinary tract symptoms or only very mild ones (slight dysuria)
  - Tender lower abdomen on palpation with adnexal or cervical motion tenderness on bimanual examination

- Clinical features suggesting another diagnosis:
  - Sexual history non-contributory, or indicates little or no risk
  - History of recent child-birth or gynaecological procedure
  - Pregnancy
• Sudden onset of pain – ruptured ectopic pregnancy or torsion of an ovarian cyst
• Recent missed period – ectopic pregnancy
• No indication of cervical infection – no discharge
• No dyspareunia
• Possible gastrointestinal tract symptoms (anorexia, constipation, diarrhoea, flatulence, nausea, vomiting)
• Possible urinary tract symptoms (marked dysuria and frequency)
• Lower abdomen tender or non-tender on palpation, but bimanual pelvic examination non-contributory and no cervical motion or adnexal tenderness elicited

The take-home message is:
• Once the clinician has excluded pregnancy, if the sexual history is suggestive, treat as STI-related PID pending results of tests and response to therapy

Diagnostic tests
The clinician should organise the following tests before and during the course of the examination:
• Pregnancy test (a urine pregnancy test may remain negative for up to 21 days after an episode of unprotected sexual intercourse)
• HVS or cervical swab for gonorrhoea culture and NAAT and chlamydia NAAT
• Midstream specimen of urine (MSSU)
• HVS – microscopy on HVS is sometimes useful if it shows a greater than normal number of leukocytes (indicating a cervicitis) or the presence of many clue cells (bacterial vaginosis) as PID is more likely in the presence of cervicitis or bacterial vaginosis

Recommended blood tests are:
• A baseline full blood examination especially for the white cell count
• Human chorionic gonadotropin (hCG) if urine test is negative or equivocal and within 3 weeks of the last unprotected sexual intercourse

Other test:
• Pelvic ultrasound examination, if any question remains of possible ectopic pregnancy

CASE STUDY 2

Andy is a 27-year-old trawler fisherman who lives in Weipa on the Gulf. He presents to the local GP with extensive perianal warts, but is otherwise asymptomatic. He is highly embarrassed and stresses to the doctor that he has never engaged in any anal sexual activities. He believes he noticed a wart on the shaft of his penis about a year ago which he succeeded in scratching off he felt very upset for some time. He has had sex with about eight different girls since that time, all very casual, usually on a Saturday night after a few drinks at the local pub. He admits he is a bit of a binge drinker. He says he uses condoms about 60% of the time. In the last 2 months, however, the anal problem has put him off sex. His general health is good although he smokes 20 cigarettes a day which he has done since age 15. He says he does not use drugs other than ‘a few bongs’ when out on the fishing trawler. He thinks he has been vaccinated against hepatitis B.

Physical examination confirms large cauliflower-like growths of warts completely surrounding his anal opening, but is otherwise unremarkable. He agrees readily to an STI screen. The clinician finds that Andy has a positive PCR test for chlamydia on an FPU. He has good levels of HBsAb, a negative syphilis EIA, a negative HIV antibody test but his hepatitis C serology is positive. The clinician treats his chlamydia but is unsure how best to manage Andy’s extensive perianal warts and hepatitis C, so he consults the regional Sexual Health Clinic in Cairns.

Initial treatment of STI-related pelvic pain syndrome in women
All health practitioners encountering patients with possible STI-related PID should have a low threshold for treatment. Once the clinician is satisfied that they have excluded ectopic pregnancy, initiating treatment for PID will not have adverse consequences even if PID is not the cause of the pain. Trial of treatment is a reasonable course of action, as non-STI causes for pelvic pain will fail to respond, while STI-related PID will respond quickly and well to the following suggested regimen:
• Azithromycin 1g orally as a single dose immediately
• Doxycycline 100 mg orally twice daily (not to be used in pregnancy D) for 14 days
• Metronidazole 400 mg orally twice daily for 14 days
• Ceftriaxone 500mg intramuscularly as a single dose (safe in pregnancy). Refer to the National
Treat or arrange treatment for male sexual partner(s) with azithromycin. Review in 3 days to observe effect of treatment and to ensure male sexual partner(s) has (have) been seen and treated. If treatment is proving ineffective and tests fail to confirm an STI, the clinician should initiate further tests to establish the diagnosis (e.g. ultrasound scan, computed tomography [CT] scan, laparoscopy).

**Ongoing treatment for specific STIs causing pelvic pain syndrome in women**

On the third day if the patient has improved, continue doxycycline and metronidazole (as above) to a total of 14 days.

**Review test results**

When the patient is having difficulty adhering to the treatment, or if patient is pregnant (a rare event as PID is uncommon in pregnancy) a suggested alternative (with a limited evidence base) is: repeat 1 g azithromycin on day 7.

Refer to the National management guidelines for sexually transmissible infections for full details.2

**7. Scrotal swelling**

**Description and causes**

There are many abnormal swellings boys and men discover in the scrotum over the course of a lifetime. They are a source of much anxiety but most are totally benign (varicoceles, epididymal cysts). Generally, worried patients can be reassured about their scrotal swellings. There are three main situations where this is not the case:

- Torsion of the testis (sudden onset, acutely painful)
- Testicular cancer (gradual onset, usually not painful; rock hard on palpation)
- Epididymo-orchitis (gradual onset, slowly increasing pain)

Epididymo-orchitis is an STI-related scrotal swelling in young men.10 In older men who have varying degrees of prostatomegaly, epididymo-orchitis may occur secondary to a low-grade bladder infection. However, STI-related epididymo-orchitis can occur in men of all ages. STI-related epididymo-orchitis follows urethritis whether symptomatic, or more commonly asymptomatic, and in theory any of the STI causes of urethritis can also cause epididymo-orchitis (see Table 13.1). In practice, gonorrhoea and chlamydia (serovars D–K) are the most common aetiologies. Epididymo-orchitis is an infection in the epididymis which spreads, if untreated, to involve the testis itself. The epididymis is tender, enlarged and firm or hard on palpation. In later disease, the epididymis and testis may become difficult to define from each other, the whole becoming a knobbly tender mass.

**Differential diagnosis**

- Scrotal swellings may be:
- Infective
- STI-related (Neisseria gonorrhoeae, Chlamydia trachomatis D–K, other STIs)
- secondary to bladder neck infections (Pseudomonas sp, coliforms)
- secondary to a sexually acquired coliform urinary tract infection due to unprotected insertive anal intercourse
- Neoplastic
- Developmental (cysts, hydrocoele, varicocele),
- Traumatic
- Due to torsion (itself really due to a developmental abnormality)

**Diagnostic tests**

In any patient under 35 years old with a significant scrotal swelling, clinicians must first exclude torsion (unlikely past 25 years of age), and testicular cancer. If the swelling is of sudden onset, is very painful and very tender on palpation, the clinician must arrange, as a matter of urgency, a Doppler ultrasound scan and/or a surgical opinion. If the swelling is firm and hard on the surface or in the body of the testis and is clearly differentiated from the epididymis, which feels normal on palpation, a cancer is likely and again an ultrasound scan should be done as soon as possible.

In all other situations and with a suggestive sexual history, look for the presence of urethral discharge. If a urethral discharge is present the clinician should collect swabs of the discharge. If not, as is the more common scenario in epididymo-orchitis, FPU can be used instead. The recommended tests are for gonorrhoea, trichomoniassis (if NAAT available), chlamydia, Mycoplasma genitalium, MSSU, serological tests as appropriate after discussion with the patient (HIV, HAV, HBV, HCV, syphilis) and rectal swabs (culture and sensitivity for gonorrhoea and chlamydia NAAT if appropriate, as well as throat swab for NAAT for gonorrhoea if relevant (see Table 13.2 for details of recommended tests).

In men who have engaged in unprotected insertive anal intercourse, a sexually acquired coliform urinary tract infection (UTI) can lead to epididymo-orchitis, so MSSU for microscopy, culture and sensitivity is an essential investigation to ensure this possibility is not overlooked.
**Initial treatment of STI-related scrotal swelling**

All health practitioners encountering patients with possible STI-related scrotal swelling should have a low threshold for treatment. Once the clinician is satisfied that she or he has excluded other significant causes of scrotal swelling (torsion and tumour) and is satisfied arrangements for follow-up are in place (see below), initiating treatment for epididymo-orchitis will not have adverse consequences even if STI-related epididymo-orchitis eventually proves not the cause of the painful swelling. STI-related epididymo-orchitis will respond quickly and well to the following suggested regimen:

- Azithromycin 1 g orally as a single dose immediately
- Doxycycline 100 mg orally twice daily for 14 days.

Remember, strains of gonorrhoea which have a predilection for the epididymis rarely cause symptomatic urethritis. Gonococcal epididymo-orchitis is still possible in the absence of a profuse purulent urethral discharge. If gonorrhoea is even a small possibility, and in MSM, add to the above regimen:

- Ceftriaxone 500 mg intramuscularly as a single dose.

Review in 3 days to observe the effect of treatment and to ensure sexual partner(s) has (have) been seen and treated. If treatment is proving ineffective and tests fail to confirm an STI, antibiotic therapy can be changed in the light of MSSU results and the clinician can initiate further tests to establish the diagnosis (e.g. blood cultures, MSSU for mycobacteria).

**Ongoing treatment for specific STIs causing scrotal swelling**

On the third day if the patient has improved, continue doxycycline to a total of 14 days.

If other possible causative STI micro-organisms are isolated on laboratory tests (e.g. M. genitalium) and the patient is failing to respond to the above regimen, clinicians should consult a sexual health physician for further advice.

Follow-up: Table 13.5 and Figures 13.2 and 13.3

Clinicians should ask patients to return for follow-up in 3 days as outlined above. The clinician should also see the patient again on day 7 and day 14 to review progress.

8. STI-related skin rashes: genital

**Description and causes**

Most skin rashes affecting the genitals are not STI related. Readers should consult dermatology or sexual health texts. There is a discussion of HIV-related skin rashes in Chapter 6. Specific STIs may cause the following genital skin rashes:

- Candidiasis – vulvitis with erythema and oedema accompanied by itch. Usually associated with vaginitis and cottage-cheese like discharge; and balanoposthitis – inflammation of the glans penis and undersurface of foreskin.
- Trichomoniasis – vulvitis due to the irritation caused by a profuse frothy offensive discharge.
- Recurrent outbreak of genital herpes – recurrent attacks of herpes are often very atypical and may masquerade as a non-specific genital rash or fissuring.
- Scabies – burrows and nodules on the soft genital skin of penile shaft, glans penis and vulva. Buttocks and natal cleft are often involved. Rash is intensely itchy, so often accompanied by excoriation. There is accompanying generalised skin irritation and itching with further burrows around wrist area and between fingers.
- Pubic lice – itchy rash and excoriation in pubic region accompanied by obvious pubic lice clinging to coarse body and pubic hair.
- Fixed drug eruption – included because often indirectly STI related. This is a sharply demarcated erythematous circular lesion, sometimes weeping, classically on the glans penis due to a localised hypersensitivity reaction to a drug such as doxycycline.

**Differential diagnosis**

There is a wide differential diagnosis of genital rashes but all the above STI-related skin rashes are fairly easily recognised clinically or can easily be confirmed by performing appropriate tests. Common non-STI causes for genital skin rashes are:

- Psoriasis
- Lichen planus
- Lichen sclerosus (including balanitis xerotica obliterans on the penis)
- Eczema
- Contact dermatitis

**Diagnostic tests**

Patients who present with STI-related genital skin rashes may have been at risk for other more significant STIs. Clinicians should consider doing screening for other STIs as appropriate. To make the diagnosis of the specific rash, take swabs from affected areas for tests for candidiasis and herpes. To check for trichomoniasis, an HVS is best. A fixed-drug eruption has a characteristic appearance and history of drug exposure, but if uncertain do a punch biopsy and send for histology.
Initial and specific treatment for STI-related skin rashes: genital

Specific treatment can be prescribed immediately on recognition of the aetiology of the rash. See the National management guidelines for sexually transmissible infections for treatments for specific STIs. For genital skin rashes these are treatments for:

- Vulvo-vaginal candidiasis
- Trichomoniasis
- Recurrent herpes
- Scabies
- Pubic lice

For fixed-drug eruption, stop the offending drug (e.g. doxycycline) and the rash will subside over 3 or 4 days.

9. STI-related skin rashes: generalised

Description and causes

Generalised skin rashes have too many causes to discuss in this monograph. The decline in immune function associated with HIV infection leads to well characterised skin conditions and infections (see Chapter 6). In primary care practice, there are three significant generalised skin rashes of relevance to STIs which an astute clinician should try to exclude. These are:

- The rash of secondary syphilis: a generalised mainly macular erythematous rash, seen well on the trunk, but may also involve the palms and the soles. It too is non-itchy. It can be accompanied by fever, malaise, headache and lymphadenopathy or may have little or no systemic symptoms.

- The rash of primary HIV infection (Chapter 4): a generalised mainly macular erythematous rash, seen well on the trunk, but may also involve the palms and the soles. It too is non-itchy. It is usually accompanied by fever, malaise, headache and lymphadenopathy. Primary HIV infection is very frequently mistaken for infectious mononucleosis.16

- The rash of disseminated gonococcal infection: seen on distal portions of the extremities as macules, papules, pustules, petechiae or ecchymoses, usually less than 30 in number. They are usually accompanied by joint involvement with arthralgias, tenosynovitis and sometimes frank arthritis. There is often accompanying fever and malaise, although this may be quite mild.16

Differential diagnosis

The differential diagnoses are many and require a thorough knowledge of dermatology.

Diagnostic tests

Testing for secondary syphilis requires a treponemal antibody test for screening. If positive the laboratory will then perform confirmatory tests including an RPR (Treponema pallidum particle agglutination [TPPA], fluorescent treponemal antibody absorbed serology tests [FTA ABS]). In almost all cases the RPR will be 1/16 or greater.

In primary HIV infection, current tests in Australia will almost always be positive. There remains a short window period in very early infection where HIV tests may be negative and the diagnosis may be missed. A negative HIV antibody test does NOT exclude the diagnosis (for further discussion of this issue, see Chapter 4).

Accurate diagnosis of disseminated gonococcal infection (DGI) is not so easy. The mainstay of testing is to send swabs for culture and sensitivity from all possible sites of exposure (as judged by the sexual history)—an FPU for NAAT is quite adequate for urethral gonorrhoea if there is no urethral discharge. Gonococcal strains causing DGI are often asymptomatic at mucosal sites of infection. In addition, sending blood cultures for Neisseria gonorrhoeae, although they are only positive in about 40% of cases, and an aspirate for microscopy, and culture and sensitivity, from any joint effusion (if possible) will assist in confirming the diagnosis.

In addition to tests outlined above, in all three situations clinicians should screen for other STIs in accordance with the sexual history (see Table 13.2).

Initial treatment of STI-related skin rashes: generalised

In these three conditions, thinking of the diagnosis and initiating the appropriate tests is a major contribution to patient management and public health. In the case of syphilis and disseminated gonococcal infection, once tests have been taken, no harm is done in initiating treatment immediately on suspicion in order to render the patient asymptomatic and non-infectious as rapidly as possible.

In all three situations, clinicians must initiate discussions with patients about contact tracing in order to ensure that their sexual partner(s) are also seen, checked and treated. Refer to ASHM contact tracing guidelines.6
Specific treatment for STI-related skin rashes

Secondary syphilis

(See Table 13.3)
Treat or arrange treatment for male or female sexual partner(s) as soon as possible.

Disseminated gonococcal infection

- Ceftriaxone 1 g intramuscularly or intravenously daily for 7 days
- Azithromycin 1 g as a single dose

If the patient is allergic to cephalosporins and the gonococcus is resistant to ciprofloxacin, the clinician should seek the advice of a sexual health or infectious diseases physician.

Test and treat, or arrange treatment for, male or female sexual partner(s) with single dose ceftriaxone and azithromycin.

Primary HIV infection

(See Chapter 4)
Contact tracing of all sexual contacts over the past 3-6 months.

Follow-up: Figure 13.2 and 13.3. See the National management guidelines for sexually transmissible infections for treatments for specific STIs.²

Summary

STIs have a wide variety of clinical manifestations, and thinking about them in terms of the eight most commonly encountered syndromes may help clinicians achieve better management. Management of STIs in primary care is highly appropriate. Opportunistic testing by GPs for asymptomatic STIs will also reduce the burden of disease in the community.

When the STI is unusual or complicated, fails to respond to recommended treatment, or when there are difficult sexuality or other sexual health issues involved or where contact tracing is beyond the capacity of the primary care clinician, patient should be referred to a specialist sexual health physician or clinic. In addition to specific therapy, psychosocial management, safer sex education, provision of information and referral when necessary are key features of the primary care of STIs.

References

PROFESSIONAL ISSUES
CHAPTER 14 BIOMEDICAL PREVENTION OF HIV

2014 AUTHORS

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Introduction

This chapter aims to provide the general practitioner with up-to-date information about two biomedical human immunodeficiency virus (HIV) prevention approaches which are based on the prophylactic use of antiretroviral drugs following recent (post-exposure prophylaxis (PEP)) or ongoing (pre-exposure-prophylaxis (PrEP)) high-risk exposure to HIV. It provides an understanding of the rationale for both strategies. It also refers to the recent literature reviews and sources of detailed information about both prevention approaches, as well as to the guidance for their use.

The most effective way of preventing HIV infection is preventing exposure to HIV. However, the use of antiretroviral drugs as prophylaxis for HIV infection might be beneficial in two specific sets of circumstances: following unanticipated, known or suspected, exposure to HIV and as a shield against infection during a period of ongoing high-risk sexual or injecting drug use behaviour.

The history of antiretroviral prophylaxis of HIV goes back to the early days of antiretroviral therapy, when the practice of giving health-care workers AZT (zidovudine) to prevent HIV seroconversion after an occupational exposure dramatically decreased the incidence of HIV acquisition.1 Later the same treatment (28 days of antiretroviral drug therapy) was found to reduce the risk of seroconversion in individuals who were exposed to HIV outside of occupational settings, namely through unprotected sex with a high-risk anonymous partner or one who had HIV infection, or through incidents of injecting drug use or sharing injecting equipment.7,8 All of the evidence for human efficacy of antiretroviral prophylaxis has been gathered from a case-control study of occupational HIV exposure, studies of the highly successful prevention of mother-to-child transmission of HIV and PrEP.1,4,5,9,10 The World Health Organization (WHO) has issued guidance on HIV prevention using each of these strategies: PEP, prevention of mother-to-child transmission and PrEP.1,4,5,9

PEP or PrEP?

PEP and PrEP are often confused. PEP has been in use since 1996 and consists of a 28-day course of two or three antiretroviral agents that must be administered within the first 72 hours after exposure to HIV. Its efficacy depends on how soon after the exposure it commences but is close to 100%.7,8 PEP is intended for infrequent, one-time exposure – not for regular use. For people who are potentially exposed to HIV on a regular basis, PrEP, taken consistently every day

KEY POINTS

- HIV is a preventable infection, with a number of effective behavioural and biomedical prevention options.
- PEP and PrEP are two biomedical approaches; both are based on the use of antiretroviral agents for prevention.
- PEP is a 28-day course of two or three antiretroviral agents that must be administered within the first 72 hours after exposure to HIV and it is intended for rare, one-time exposures – not for regular use.
- PrEP is a more suitable option for eligible people who are exposed to HIV on a regular basis; it is meant to be used consistently, as a pill taken every day, and to be used with other prevention options such as condoms.
- Clinicians should carefully evaluate their patients as to their eligibility for PEP or PrEP, provide detailed information about their correct use and support patients along the course of medication use.
- Patients benefit most from a combination of biomedical and behavioural approaches to risk reduction.
alongside other prevention measures such as condoms, is a more suitable option. It may also be considered as one of the options to prevent HIV transmission in serodiscordant couples trying to conceive.

**Post-exposure prophylaxis of HIV infection**

In Australia, antiretroviral therapy for HIV infection is listed under Section 100 (s100) of the Pharmaceutical Benefits Scheme and can only be prescribed by approved clinicians. In addition, none of the individual antiretroviral drugs are licensed for use as post-exposure prophylaxis and hence must be funded by state-based services. Each state determines how PEP is made available and this is usually through hospital emergency departments and public sexual health clinics. Community s100 prescribers can assess cases and write scripts for PEP, but the drugs are dispensed from hospital pharmacies.

**Risk assessment for PEP**

Responding appropriately to a possible HIV exposure requires assessment of the likelihood of HIV infection in the source, risk associated with the exposure, collection of appropriate baseline tests, referral to a PEP prescriber, and ongoing support for the patient.

Assessment should address whether the source is known to have HIV infection. If the source is available and willing, testing for HIV and viral hepatitis should be conducted urgently; however, the often anonymous nature of an exposure can make that impossible. If the source discloses an HIV-positive serostatus, and is contactable, he or she should be asked for consent to access his or her treatment details as it is important to know treatment status and HIV viral load. If the source cannot be contacted or tested for any reason, the seroprevalence data in Table 14.1 may help to determine the need for PEP. If exposure to HIV happened overseas, seroprevalence for individual countries can be found at www.unaids.org/en/dataanalysis/datoools/aidsinfo/. The risk of HIV transmission through a single exposure is determined by the nature of the exposure with its estimated risk per exposure, the risk that the source is HIV positive if their status is unknown, and factors associated with the source in particular HIV viral load in a source known to be HIV infected. These settings may be non-occupational or occupational.

Highest risk is defined as sexual exposure with a person with HIV infection via receptive anal or vaginal intercourse without a condom, particularly when ejaculation into the receptive partner occurs, or exposure to HIV-infected blood via injecting equipment where percutaneous exposure has occurred with a used hollow needle (see Table 14.2). For sexual exposures, it is assumed that similar risk is incurred for exposures without a condom and those when a condom fails.

For percutaneous, occupational exposures, the National Needlestick Injury Hotline can provide advice to healthcare workers regarding the level of risk. For non-occupational HIV exposures, state-based hotlines can provide assessment and referral for members of the public who think they may have been exposed to HIV.

**Needlestick Injury Hotlines**

<table>
<thead>
<tr>
<th>Type of exposure with known HIV positive source</th>
<th>Estimated risk of HIV transmission/ exposure*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Receptive anal intercourse (RAI) – ejaculation</td>
<td>1/70</td>
</tr>
<tr>
<td>– withdrawal</td>
<td>1/155</td>
</tr>
<tr>
<td>Contaminated injecting equipment</td>
<td>1/125</td>
</tr>
<tr>
<td>Insertive anal intercourse (IAI) uncircumcised</td>
<td>1/160</td>
</tr>
<tr>
<td>Insertive anal intercourse (IAI) circumcised</td>
<td>1/900</td>
</tr>
<tr>
<td>Receptive vaginal intercourse (RVI)</td>
<td>1/1250*</td>
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<tr>
<td>Insertive vaginal intercourse (IVI)</td>
<td>1/2500*</td>
</tr>
<tr>
<td>Receptive or insertive oral intercourse</td>
<td>Unable to estimate risk – extremely low</td>
</tr>
<tr>
<td>Needlestick injury (NSI) or other sharps exposure</td>
<td>1/440</td>
</tr>
<tr>
<td>Mucous membrane and non-intact skin exposure</td>
<td>&lt; 1/1000</td>
</tr>
</tbody>
</table>

* These estimates are based on prospective studies, not cross-sectional data or figures derived from modelling.

Adapted with permission from the national guidelines Post-exposure prophylaxis after non-occupational and occupational exposure to HIV**
Implementation of PEP

For details of the clinical and laboratory assessment and follow-up of PEP patients (which would be done by the specialist services or s100 prescribers) see the national PEP guidelines.14

HIV antibody testing is conducted for all individuals presenting for PEP, and HIV testing should be repeated at 4 to 6 weeks and 3 months after exposure. At baseline, PEP patients should also be assessed for the possibility of hepatitis B exposure (non-immune individuals are offered immunisation), screened for STIs (particularly chlamydia, gonorrhoea and syphilis), and, where indicated, screened and followed up for hepatitis C. Women of child-bearing age should be offered pregnancy testing.

As to prescribed antiretroviral treatment for PEP (see national PEP guidelines),14 drugs should be chosen based on the information about the treatment history, viral load and resistance patterns of the source case and the medical history of the exposed individual. Usually the treatment involves two or three drugs, but there is no direct evidence to support the greater or lesser efficacy of three over two drug preventive regimens. When prescribing PEP course, possible benefit should be considered from the increased numbers and classes of drugs for HIV treatment against potential side effects, toxicity, adherence and cost effectiveness of adding a third drug.

The possibility of exposure to HIV causes anxiety and concern. As well as ongoing support from their regular primary care support team, patients may need considerable extra support at this time. Patients may require referral to an experienced counsellor and / or a drug and alcohol service.

Pre-exposure prophylaxis of HIV infection

The effectiveness of antiretroviral medications as pre-exposure prophylaxis of HIV (PrEP) has now been established by four clinical trials conducted in homosexual men (iPrEx), heterosexual men and women (Partners PrEP and TDF2),15,16 and injecting drug users (Bangkok Tenofovir study).17 The daily oral pill containing tenofovir disoproxil fumarate (TDF) and emtricitabine (FTC) is considered safe and effective to reduce the risk of HIV infection in high-risk adults who are able to take the medication correctly and consistently.18 None of the clinical trials has reported any significant increase in behavioural disinhibition, while all trials promoted condom use and safer sex practices. Based on this evidence, the TDF/FTC pill (marketed as TRUVADA® by Gilead Sciences Inc.)20 has been approved by the US Food and Drug Administration as PrEP. Clinical guidance for PrEP prescribers has been issued by the US Centres for Disease Control and Prevention (CDC) for all three population groups: homosexual men, high-risk heterosexual men and women, and injecting drug users.5 The US PrEP guidelines for clinicians are available on the CDC website: http://www.cdc.gov/hiv/pdf/PrEPguidelines2014.pdf

Published evidence from clinical trials clearly suggests a wide gap between the average risk reduction provided by antiretroviral medication and the adherence-adjusted efficacy.5 On average, Truvada efficacy levels were moderate and ranged from 42% among homosexual men to 72% among heterosexuals.4,15 In the same studies, adherence-adjusted efficacy, measured by TDF detection in blood, rose significantly to 92% in homosexual men and 84% in heterosexuals. On the other hand, the FEM-PrEP and VOICE trials, where participating women were not able to adhere to the same medication, were stopped when it was determined there was a very low chance of achieving a result (futility).22,23 The observed gap between average and adherence-adjusted levels of protection appears to vary not only across studies, but also across countries and research sites. This is best illustrated by the iPrEx study, which found better adherence to PrEP among homosexual men in the US as opposed to the study sites in other countries.4 The iPrEx study and its open-label extension demonstrated that high levels of adherence can be expected among self-selected, motivated PrEP users and among those who are better informed about PrEP and HIV prevention.

<table>
<thead>
<tr>
<th>Community group</th>
<th>HIV seroprevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Homosexual men (MSM – men who have sex with men)</td>
<td></td>
</tr>
<tr>
<td>• ACT</td>
<td>4.2</td>
</tr>
<tr>
<td>• Adelaide</td>
<td>5.4</td>
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<tr>
<td>• Brisbane</td>
<td>8.8</td>
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<tr>
<td>• Melbourne</td>
<td>8.1</td>
</tr>
<tr>
<td>• Perth</td>
<td>4.5</td>
</tr>
<tr>
<td>• Sydney</td>
<td>11.8</td>
</tr>
<tr>
<td>Actual seroprevalence may be higher than reported seroprevalence</td>
<td></td>
</tr>
<tr>
<td>Injecting drug users in Australia</td>
<td></td>
</tr>
<tr>
<td>• homosexual</td>
<td>29.2</td>
</tr>
<tr>
<td>• all others</td>
<td>1.0</td>
</tr>
<tr>
<td>Heterosexuals in Australia</td>
<td></td>
</tr>
<tr>
<td>• blood donors (% donations)</td>
<td>0.0004</td>
</tr>
<tr>
<td>• STI clinic attendees</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Commercial sex workers (Australia)</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Overall Australian seroprevalence</td>
<td>0.1</td>
</tr>
</tbody>
</table>

Adapted with permission from the national guidelines Post-exposure prophylaxis after non-occupational and occupational exposure to HIV14

Table 14.2: Transmission risk per act for sexual exposure and intravenous drug use

<table>
<thead>
<tr>
<th>Community group</th>
<th>HIV seroprevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Homosexual men (MSM – men who have sex with men)</td>
<td></td>
</tr>
<tr>
<td>• ACT</td>
<td>4.2</td>
</tr>
<tr>
<td>• Adelaide</td>
<td>5.4</td>
</tr>
<tr>
<td>• Brisbane</td>
<td>8.8</td>
</tr>
<tr>
<td>• Melbourne</td>
<td>8.1</td>
</tr>
<tr>
<td>• Perth</td>
<td>4.5</td>
</tr>
<tr>
<td>• Sydney</td>
<td>11.8</td>
</tr>
</tbody>
</table>

Actual seroprevalence may be higher than reported seroprevalence

Injecting drug users in Australia

• homosexual
• all others

Heterosexuals in Australia

• blood donors (% donations)
• STI clinic attendees

Commercial sex workers (Australia)

Overall Australian seroprevalence

0.1
HIV-negative participants and 9% of seroconverters.4 Since adherence to daily PrEP appears to be an essential condition for the effectiveness of daily PrEP at the population level,26 research continues on the development of the next generation of PrEP: alternative antiretroviral options, schedules of use and delivery mechanisms aiming to make PrEP easier to use and acceptable to a variety of user groups. Evidence from clinical trials on long-acting injectable antiretroviral drugs, medicated vaginal rings and rectal microbicides, among others, is expected to be available in the next 3-5 years.27

Other than adherence, issues which may affect the future implementation of PrEP include those that can influence its uptake, particularly the support from the anticipated target population groups and PrEP providers. Current evidence suggests very low levels of PrEP uptake, even in the US where PrEP is licensed and available. A lack of knowledge in communities about PrEP and varying degrees of community and provider support for PrEP have been reported as obstacles for PrEP implementation there.26

PrEP in Australia

There are no guidelines for PrEP prescription in Australia as yet. At the time of publication of this monograph, no antiretroviral drugs have been licensed by the Australian Therapeutic Goods Administration for preventive use. However, Australia maintains a high commitment to reducing rates of HIV infection and recognises that new technological developments should be considered for HIV prevention.24 During 2014-16, PrEP implementation models will be developed and tested in at least three demonstration projects across the country in New South Wales, Victoria and Queensland. All three projects are funded by state departments of Health committed to incorporate PrEP into a comprehensive package of HIV prevention interventions in keeping with the WHO recommendation to undertake implementation research.19 Their purpose is not only to give access to PrEP to homosexual men and heterosexual men and women who can benefit from it, but also to better understand the role of PrEP in response to the local HIV epidemic, to assess local barriers and facilitators to PrEP adherence among the users, and to exert the maximum effect from PrEP in local HIV prevention.19

Preliminary work has been undertaken in Australia to understand the needs of high-risk population groups for more effective HIV prevention strategies,31 awareness about and willingness to use daily PrEP among homosexual men21 and heterosexual men and women in serodiscordant relationships, as well as current levels of informal use21 and the patterns of use and associated factors.34 Similar to reports from other countries (e.g. the US),24 PrEP use in Australia remains low (about 2.5% of gay men in community samples) and is reported by gay men with highest risk practices for HIV, but interest in this prevention strategy is rapidly growing.35 At the time of this publication, there has been no evidence about the use of PrEP by heterosexual men or women in Australia.

Eligibility and risk assessment for PrEP

PrEP is a relatively new HIV prevention approach. It may represent a much-needed additional prevention method for some individuals at high risk for HIV infection. It is important to remember however that, to reduce the risk of getting HIV, this method is not intended to be used alone or to replace other prevention methods, but rather in combination with other methods, particularly condom use and safer sex practices. PrEP cannot replace behaviours that help avoid HIV exposure.

In addition, this prevention method will not suit everyone. It will be most effective if it is targeted to users who can benefit from it most. Based on the CDC recommendations, those eligible for PrEP are adults who are able to make an informed choice to use PrEP, who are confirmed to be HIV negative, and are at substantial and sustained risk for HIV infection.21 HIV-negative status should be confirmed no more than 7 days before initiation of PrEP, by using the standard-of-care testing procedures. Because the risk of HIV infection cannot be fully eliminated by PrEP, regular HIV testing is essential. At any time, PrEP should be discontinued immediately if a person has symptoms of seroconversion illness or is highly likely to seroconvert (e.g. a known exposure to HIV happened during the time when daily PrEP was not taken), because HIV resistance substitutions may develop in individuals with undetected HIV-1 infection who are taking Truvada alone. Truvada alone does not constitute a complete treatment regimen for HIV infection.

PrEP is meant to be used by people who are at high and ongoing risk of acquiring HIV. Table 14.3 summarises different practices and conditions which are known to be associated with increased HIV incidence among men who have sex with men in Australia (data from the Health in Men (HIM) study conducted in 2001-2007).25 Some recommendations on how to determine behavioural eligibility for PrEP are provided by the US clinical guidance for PrEP prescribers.21

Administration of PrEP for partners without HIV infection may offer an additional tool to reduce the risk of sexual transmission of HIV in periconception period. The US CDC guidelines recommend that
Clinicians discuss PrEP with heterosexually-active men and women whose partners are HIV positive (i.e. HIV-discordant couples) as one of several options to protect the partner without infection during conception. Clinicians should educate HIV-discordant couples who wish to conceive about the potential risks and benefits of available alternatives for safer conception and make referrals to a specialist if indicated. Irrespective of whether such a couple makes a choice in favour of PrEP, the partner with HIV infection should be on antiretroviral therapy before conception attempts (for the health benefit of that partner, and if the partner with infection is male to reduce the HIV viral load in semen).

The risk of HIV acquisition increases during pregnancy, as does the risk of HIV transmission to a child from a mother who acquired the infection during pregnancy or breastfeeding. Therefore, an HIV-negative woman at high risk of HIV infection may benefit from continuing PrEP use throughout her pregnancy and breastfeeding to protect herself and her infant. The US CDC guidelines recommended to discuss PrEP with women without infection at high risk of HIV infection who are pregnant or breastfeeding so that an informed decision can be made in full awareness of what is known and unknown about benefits and risks of continuing PrEP.

Clinicians need to obtain a thorough sexual and drug use history to determine PrEP eligibility and regularly discuss practices at high risk of HIV with their patients to assess continuing candidacy for PrEP. Patients should be followed regularly not only for HIV infection, but also for other STIs per STI testing guidelines (see Chapter 8), and renal and liver function due to the known side effects of Truvada (see prescribing information for details). Women taking PrEP should be regularly assessed for pregnancy. Clinicians should also offer extensive HIV risk-reduction counselling and encourage patients to continue condom use and other risk reduction approaches for safer sex. Referral to drug and alcohol counselling and mental health services may be indicated and should be considered.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Associated HIV incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients regardless of practices</td>
<td>0.78 per 100 PY 95% CI (0.59-1.02)</td>
</tr>
<tr>
<td>Regular sexual partner of a man with HIV infection with whom condoms were not</td>
<td>5.36 per 100 PY 95% CI (2.78-10.25)</td>
</tr>
<tr>
<td>consistently used in the last 3 months (HIV-positive partner is not on treatment and/or has detectable viral load)</td>
<td></td>
</tr>
<tr>
<td>At least one episode of receptive unprotected anal intercourse with any casual male partner with HIV infection or a male partner of unknown HIV status during the last 3 months</td>
<td>2.31 per 100PY 95% CI (1.48-3.63)</td>
</tr>
<tr>
<td>More than one episode of anal intercourse during the last 3 months when proper condom use was not achieved (e.g. condoms slipped off or broke)</td>
<td>1.30 per 100 PY 95% CI (0.95-1.77)</td>
</tr>
<tr>
<td>More than one episode of insertive unprotected anal intercourse where the serostatus of partner was not known or was HIV positive and not on treatment in the last 3 months</td>
<td>0.94 per 100 PY 95% CI (0.35-2.52)</td>
</tr>
<tr>
<td>In circumcised men</td>
<td>0.65 per 100 PY 95% CI (0.16-2.61)</td>
</tr>
<tr>
<td>In uncircumcised men</td>
<td>1.73 per 100 PY 95% CI (0.43-6.90)</td>
</tr>
<tr>
<td>Rectal gonorrhoea diagnosis</td>
<td>7.01 per 100 PY 95% CI (2.26-21.74)</td>
</tr>
<tr>
<td>Rectal chlamydia diagnosis</td>
<td>3.57 per 100 PY 95% CI (1.34-9.52)</td>
</tr>
<tr>
<td>Methamphetamine use</td>
<td>1.89 per 100 PY 95% CI (1.25-2.84)</td>
</tr>
</tbody>
</table>

PY: person-years; CI: confidence interval
* Data from the Health in Men (HIM) cohort study conducted in 2001-2007

**Table 14.3: Different practices and conditions associated with high HIV incidence among MSM**
Implementation of PrEP

At the time when this monograph is published, access to PrEP in Australia is not yet established and PrEP is only available through demonstration projects in New South Wales, Victoria and Queensland.

Information about PrEP


Truvada Risk Evaluation and Mitigation Strategy (REMS). Materials developed by Gilead Inc. and FDA. Available at: www.truvadaprepresources.com/truvadaprep-resources

Truvada prescription guide. Available at: www.accessdata.fda.gov/drugsatfda_docs/label/2013/021752s042lbl.pdf

AIDS Vaccine Advocacy Coalition (AVAC) website. Pre-Exposure Prophylaxis [Internet]. Available at: http://www.avac.org/prep

PrEP Watch: www.prepwatch.org/

Further information about PrEP and antiretroviral prescribing is available on the ASHM website at www.ashm.org.au/HIVguidelines

Other strategies to prevent HIV infection

HIV is a preventable infection and today there are more tools than ever available to avert it. The cornerstone of HIV prevention is risk reduction using approaches such as limiting of the number of sexual partners, never sharing needles, using condoms correctly and consistently, and testing regularly for HIV and STIs. In addition to these strategies, people at risk for HIV may be able to take advantage of biomedical prevention options. PEP and PrEP are just two elements of the antiretroviral-based and wider biomedical HIV prevention toolkit. The latter also includes a range of proven approaches (including biomedical prevention of mother-to-child transmission of HIV (discussed in Chapter 9), early treatment of HIV as prevention of onward transmission (antiretroviral treatment as prevention [TasP]) (Chapter 10), and medical male circumcision, as well as others under study (such as rectal and vaginal microbicides and HIV vaccines). These strategies have differing levels of efficacy, acceptance in the social and medical scientific communities, and differing levels of use. The highest impact in HIV prevention can be achieved by a combination of behavioural and biomedical strategies. Priority areas for action in HIV prevention in Australia are outlined in the national HIV prevention strategy.29

CASE STUDY 1

Non-occupational post-exposure prophylaxis (nPEP) presentation and issues of safer sex and disclosure

David is a middle-aged, married man who presents to his general practitioner, Dr Betheras, for non-occupational HIV post-exposure prophylaxis (nPEP) the morning after a condom break during receptive anal sex in a sex-on-premises venue.

Dr Betheras immediately organises referral to a general practitioner who can prescribe antiretroviral therapy (antiretroviral therapy prescribing practitioners’ contact details are listed in the ASHM Directory at: http://www.ashm.org.au/ashm-directory/). Before David leaves for his next appointment, Dr Betheras advises him that he will need to institute condom use when having sex with his wife and any other sexual partners until he has his final, week-12 test results. ‘How will I explain this to my wife?’ David asks. Dr Betheras explores his concerns about the risk episode and the fear, guilt and shame he is experiencing. She also discusses with him the issues involved in talking about the episode with his wife, if and when he decides to do so.

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Dr Betheras also discusses the case with her medical insurer and gets advice about the legal issues of duty of care and confidentiality regarding both David and his wife (Chapter 15). She continues to monitor the situation in conjunction with the general practitioner providing nPEP.
Case Study 2

nPEP or PrEP?

Jack is a young gay man who lives in Sydney and likes to enjoy life. His last HIV test was 5 months ago and the result was HIV negative. He has a few regular sexual partners, whom he knows, with whom he has unprotected sex. He also occasionally meets a new casual partner with whom he usually, but not always, has protected sex. A week ago Jack went to a party, where he met a group of new guys. He had sex without a condom with them thinking they were all HIV negative. For some reason, he feels concerned about getting HIV at that event. Jack comes to see Dr Betheras to get checked out and asks for PEP (one of his friends had been exposed to HIV previously and Jack knows that that friend successfully prevented infection by taking a course of pills).

Dr Betheras carefully collects all of the necessary information and clarifies that the last event of unprotected sex was a week ago. Dr Betheras knows that, unfortunately, it’s too late to start nPEP. She orders a 4th Generation Combo EIA test for HIV and the standard of care STI tests. Dr Betheras is aware of a new biomedical prevention strategy called PrEP and she decides that, if testing shows that Jack is HIV negative, she will discuss it with him.

Jack indeed was confirmed to be HIV-negative and has come for a follow-up visit. Dr Betheras and Jack discuss the issue of safer sex practices. She informs Jack about initiating nPEP within 72 hours and places of access to nPEP including after hours. She also tells Jack about PrEP. Dr Betheras advises Jack that he will still need to use condoms and safer sex practices and come to test for HIV and STI regularly.

Summary

HIV is a preventable infection. There are a number of effective behavioural and biomedical tools to prevent HIV and the prevention toolkit is rapidly expanding. PEP and PrEP are only two of a number of biomedical approaches which clinicians could recommend to their patients. PEP and PrEP have some similarities; both are based on the use of antiretroviral agents for prevention and should be used by only those individuals who are HIV negative. There are substantial differences in when, how and by whom these two strategies should be used. Clinicians should carefully evaluate the patients as to their eligibility for PEP or PrEP, provide detailed information about their correct use and support patients along the course of medication use. Either approach should not be used on its own or as replacement for other prevention methods. Patients would benefit most from a combination of biomedical and behavioural approaches to risk reduction.

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CHAPTER 15 LEGAL RESPONSIBILITIES IN RELATION TO HIV AND VIRAL HEPATITIS

2014 REVIEW

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Note: This chapter refers to a number of important Australian laws and policies relating to privacy, confidentiality and duty of care including a summary of leading legal cases. Although addressing some important questions, this information does not constitute legal advice. Practitioners who are uncertain about their statutory or common law obligations to patients or to the local health department, including privacy and reporting obligations, are strongly advised to contact their local health department, applicable privacy office or seek independent legal advice.
Introduction

While special issues arise concerning the treatment of people who may have the human immunodeficiency virus (HIV) or hepatitis infection, or are suspected of having the infection, for the most part those laws that pertain to the treatment of any patient also apply to these patients. This chapter will, where relevant, pass very briefly over those areas of law that deal with the treatment of patients generally and focus particularly on what is required of health-care practitioners regarding HIV, hepatitis B virus (HBV) and hepatitis C virus (HCV), diseases that are of greater social sensitivity.

Each state and territory’s body of law deals with this area differently. In most cases the differences are a matter of degree. Occasionally the differences are significant. Appendix 5 summarises these laws as they apply in each state and territory. It is useful in guiding health-care practitioners about their responsibilities to patients and in informing the content of counselling.

Provision of information to the patient

Normally given the misnomer of ‘informed consent’, the provision of information, and the exchange of information between a health-care provider and a patient, is a key element in any treatment or procedure. When this is done well it allows the health-care practitioner to discuss the risks and benefits of any treatment or procedure and the patient to consider these risks and benefits in the light of his or her circumstances. These discussions may be concluded in the course of a single consultation or may need to take place over the course of several consultations. The aim of such discussion is to enable the patient to consider the information that is provided in order to facilitate his or her decision-making. The result may be patient consent to the treatment or procedure, refusal or some other negotiated outcome. It is no longer possible to assert that the view of the health-care provider should take precedence.

In many jurisdictions testing for HIV is subject to additional laws. These laws were drafted or a number of reasons. During the early 1980s, many health-care practitioners in Australia were ill informed about HIV and responsible for poor treatment of those suspected of being HIV positive. A proliferation of horror stories from those people who had suffered at the hands of their practitioners in both surgery and hospital settings emerged. There was no treatment. Testing was often undertaken solely to prevent transmission to others. Testing was not necessarily in the interests of the patient, who would in all likelihood suffer from discriminatory behaviour. These circumstances indicated that legislative intervention was required. This testing regimen was called ‘voluntary counselling and testing’ (VCT).

Confidentiality

Health practitioners will be well aware of their duty to maintain the confidentiality of their patients. The reasons for this are clearly understood and relate, at the individual level, to the creation of a climate of trust between the health-care practitioner and the patient, and at the population level to the protection of public health. If people believe that their trust will be betrayed they will be less likely to seek attention, and this may have an impact on the general health of the community. This duty is now reinforced by Commonwealth privacy laws that apply to all medical practitioners. Some states (such as NSW and Vic.) impose concurrent duties under state acts.

There are also public health specific laws requiring that medical practitioners not disclose any information regarding a person who has tested positive to an infectious disease, and in particular, HIV.

In the ACT, NT, SA, Qld and Tas., disclosure of private information regarding infectious diseases is an offence. NSW legislation does not make disclosure a crime, but instead requires that medical practitioners and other people involved in the testing of HIV have appropriate systems in place for protecting the privacy of persons tested for HIV.

Notwithstanding such provisions, doctors, pathologists and hospitals are under a duty to notify government of instances of HBV, HCV and HIV. Ordinary notification of infectious disease is necessary for the purposes of
identifying risk factors, monitoring outbreaks, service planning, implementation and evaluation, and in some cases enabling contact tracing. Such information may also be used in research. Notification via the appropriate channels is authorised under the various privacy and public health acts; however a number of states require such notifications to be by way of a de-identified code.

Many patients are reluctant for the doctor to ring their home or workplace; some patients instruct their doctors not to ring under any circumstances, and other patients attend giving a false name and false contact details. In some situations, the doctor is unable to initiate passing the result to the patient but relevant public health authorities must, be informed of positive test results regarding notifiable diseases. At the same time, the clinician can report that the patient had not sought the result and could not be contacted. It would then be a matter for the public health authority to find the patient. Though extremely difficult, this may still be possible.

**Notification of third parties**

Health-care practitioners may become aware a patient has placed one or more people at risk of contracting HIV, HBV or HCV. In such instances the health-care practitioner may wish to encourage the patient to discuss the matter with those who may be at risk of infection. Alternatively, the health-care practitioner may advise that the patient bring his or her partner/s or contact/s in so they may be counselled. This situation can raise particular difficulties where, for example, a woman has been diagnosed with HIV infection and lives in a situation where she is exposed to violence from her partner.

There will be the occasional patient whom the health-care practitioner sincerely believes may have transmitted the infection to others and who refuses to cooperate and the practitioner will need to weigh the difficult moral issues in play. In such instances, the appropriate action to take is the notification of the appropriate health authorities, which will not breach any of a doctor’s duties of confidentiality under the law. In the very rare instance where the practitioner believes his or her patient is intentionally placing others at risk, the obligation to notify becomes compelling.

However the Courts have clearly held that doctors cannot directly, without express consent of their patient, provide information such as their patient’s HIV status to another party at risk of infection though a doctor may still owe a duty of care to such third parties. In example, the case of BT v Oei [1999] NSWSC 1082 examined whether a medical practitioner owes a duty of care to the spouse of his or her patient. Dr Oei had not ordered an HIV test for AT, his patient. BT argued that Dr Oei, by virtue of his specialist training and knowledge, should have known that given AT’s history and symptoms, AT was at risk of having HIV infection. This being so, it was reasonably foreseeable that AT, if HIV positive, would transmit the virus to sexual partners. After an extensive consideration of Australian and international law, the judge found that, had AT been appropriately counselled, he would have had a test for HIV which would have shown he had contracted HIV. Had AT been counselled properly, he would have understood the need to protect his partner from risk of infection. The couple would not then have engaged in unprotected sexual relations. The judge concluded that the doctor’s negligent failure to properly advise AT with respect to a possible diagnosis of HIV and the need for an antibody test materially contributed to BT’s acquiring the HIV infection.

Another relevant judgment, PD v Dr Nicholas Harvey & 1 Ors [2003] NSWSC 487, reinforces this point. A couple attended a general practitioner together for pre-marital counselling and sexually transmitted infection (STI) screening. The man was found to be HIV positive. When he was given the result, he was referred to a specialist HIV clinic. When the woman rang, having ascertained that she was HIV negative, she asked about the man’s result. She was told she could not be given the man’s result without his consent. He fraudulently told her his result was negative; they had unprotected intercourse, and she became HIV positive. She sued the doctors involved. The judge supported the doctors’ observance of their duty not to disclose the man’s result to the woman without his consent.

However, the judge found the pre-test counselling at the original joint consultation was negligently provided in that it did not meet the standard required under guidelines issued by the NSW Ministry of Health. In particular, the Court found that given the initial consultation was a joint one for the purposes of HIV testing for the couple, and in the light of a doctor’s conflicting requirements of patient confidentiality and duties under the Public Health Act 1991, he should have at least advised FH and PD of the need for each to consent to the supply of their results to the other. Had this happened, PD would have become aware of FH’s HIV status. The Court was also very critical of the doctor’s failure to follow up with FH after he did not attend at a specialist appointment for his HIV, and the manner in which the initial post-test counselling was provided to him.

The importance of appropriate record keeping and follow-up is further demonstrated by the recent case of C S v Anna Biedrzycka [2011] NSWSC 1213, where the Court found that a provider of administrative services to a medical centre was, in addition to the doctors, civilly liable to the partner of a patient of the Centre. The patient had had an indeterminate HIV test which was not properly advised to the patient, following which the patient’s partner also
Contact tracing

Contact tracing is the practice whereby a medical professional or the relevant governmental agency traces all the contacts of a person who has, or is suspected of having, an infectious disease. Faced with an outbreak of an infectious disease which spreads rapidly through person-to-person contact, public health officials can use contact tracing to identify people at risk of infection and places contributing to the spread of the disease.

The nature of HIV, HBV and HCV, along with society’s reaction to them, makes contact tracing a delicate exercise. Firstly, the stigma associated with HIV and the other blood-borne viruses means people who have the infection do not want every person they are in contact with to know of the infection. Secondly, the nature of transmission, requiring transfer of bodily fluids, makes it unnecessary to identify any of a person’s non-sexual contacts. Public health legislation in Australia is not cognisant of this difference in the nature of contact tracing between HIV, HBV and HCV and other infectious diseases, however. In every state or territory which has contact tracing provisions (ACT, NSW, NT, Qld, SA and Tas.), the powers of the relevant person to require and disclose information are identical no matter the type of condition.

Contact tracing powers vary between the various states and territories. In the ACT, NT and Qld authorised people can require that a person with HIV, HBV or HCV provide their name and address, the name and address of anyone they may have been in contact with, and information about how and in what circumstances the person acquired the infection. In Tas., the Director of Public Health can require only that a person with a notifiable disease provide the name and address of any person to whom he or she might have transmitted the disease. NSW limits the extent of contact tracing to advising a contact of the possibility that they may have been exposed to an STI or a blood-borne virus, and precautions to be taken to minimise the chance of infection or of passing it to others. Finally Vic. has the most circumscribed powers, only having power to require the name and address of contacts in the case of an infectious disease outbreak.

Additional powers exist in the ACT and Qld for an authorised officer or contact tracing officer to advise contacts they may have been in contact with an infectious disease even where the person with the disease has told the doctor he or she does not wish the contact to be told. These provisions are understandable in the context of an infectious disease like tuberculosis, which can spread rapidly and is difficult to contain, but are less appropriate in the context of HIV, HBV and HCV unless there is evidence to suggest the person with the infection is likely to endanger the health of others.

It is unclear from the legislation what role medical practitioners play in the collecting of contact tracing information. In each jurisdiction the powers of the relevant person to demand information appear unfettered (except in the case of Vic. which requires an outbreak) and therefore, as a matter of practicality it is likely that medical practitioners will be used as the major sources of such information. Many of the states have provisions which protect the medical practitioner from liability for any information given pursuant to an order under the relevant Act.

A list of relevant resources and professional guidelines relating to contact tracing can be found on the Australian Models of Care database available on the ASHM website (www.ashm.org.au).

Testing in health-care or custodial settings

Health-care and custodial settings are environments where the possibility of transmission of disease may be increased. In health-care settings, transmission may occur where proper infection control procedures are not observed. In custodial contexts, particularly for people detained in correctional facilities, blood-borne viruses can be spread between inmates through intravenous drug use, the use of unsterilised tattoo equipment or unprotected sex. There is some anecdotal evidence that the practice of drug testing prison inmates has resulted in a shift from cannabis use (which remains detectable for up to 6 months) to injecting drug use (which can be flushed out in under 48 hours). If this is true, the lack of clean needles in prison is likely to increase the prevalence of HIV, HBV and HCV and equally increase the risk to correctional workers.

If a person has, or may have contracted their infection in either of these settings, for the most part the standard laws regarding notification and testing will apply. Vic. does, however, have specific provisions aimed at incidents involving health-care workers that give rise to the risk of infection, with powers to order tests in these circumstances. This section covers situations where, for example, a health-care practitioner may be at risk of contracting an infection from a patient, for example through a needlestick injury.

Otherwise, under corrections legislation, prisoners must submit to medical testing when ordered to do so and must comply with the instructions of a medical practitioner.

Criminal law

There are also criminal offences associated with HIV, HBV and HCV with laws in every jurisdiction making it an offence to transmit the infection to another
person. The majority of these laws do not specify HIV, HBV or HCV, but instead refer to the infliction of harm, either recklessly or intentionally. In some jurisdictions, engaging in behaviour that endangers others is also a criminal offence. As with the other areas of legislation, the scope and requirements of these offences differ between jurisdictions.

Offences regarding the spread of HIV, HBV and HCV exist both in public health law and criminal law, though the offences under public health law generally penalise a failure to take reasonable precautions against the spread of infection, rather than transmission itself. Typically the offences contained in the public health law have much lighter penalties than do the criminal provisions and they also allow for defences that the criminal law does not.

The ACT, NSW, Tas. and Vic. have public health provisions which require a person with the infection to take reasonable precautions against the spread of disease. Qld and SA also penalise the transmission of disease. Penalties vary greatly between the jurisdictions. The least punitive requirements are the jurisdictions which only require that a person with an infectious disease take reasonable precautions not to pass it on. Stricter standards exist elsewhere; SA law, for example, prohibits the intentional or reckless causing of a serious risk to public health and prescribes up to a $1,000,000 penalty or a jail term of 10 years. In most jurisdictions it is a defence if the person who contracted the disease knew of, and voluntarily accepted, the risk.

The ACT and Tas. do not have any specific criminal provisions relating to the spread of HIV, HBV or HCV infection. The other states and the NT criminalise the spread of the infections in one of two ways. Either specific offences addressing HIV, HBV and HCV have been created or spreading a disease is incorporated within existing provisions which prohibit causing harm to another person.

SA, WA, NSW and the NT have adopted the latter approach and included ‘disease’ within the definition of ‘harm’ for the purposes of the criminal law. As a result, all of the provisions prohibiting harm also prohibit transmitting the disease to someone else. For example, the prohibition on intentionally causing serious harm also prohibits intentionally causing a serious disease; similarly the prohibition on negligently causing harm also prohibits negligently causing a disease. This approach makes use of an extensive existing body of law and is thus more likely to address sensitive issues of HIV, HBV and HCV infection more effectively.

Qld and Vic. have separate criminal offences addressing the intentional infection of another with a serious disease. While Qld does not expressly define ‘serious disease’, in Vic. ‘Very serious disease’ is specifically defined as HIV. This approach runs the risk of treating people accused of spreading HIV differently to someone accused of causing any other type of bodily harm. Although the consequences of infection with these diseases are long term, these are issues which can be considered when assessing the gravity of the harm caused.

Thus, although it has been regarded as politically populist to create HIV specific offences, doing so is arguably unnecessary and indeed runs counter to the position adopted by agencies such as the World Health Organization, the Joint United Nations Programme on HIV/AIDS and the Office of the High Commissioner on Human Rights.

**Anti-discrimination**

Anti-discrimination provisions exist in every Australian jurisdiction which make it illegal to discriminate against someone on the basis of their having HIV, HBV or HCV. In each jurisdiction, discrimination is prohibited either on the basis of disability or impairment and, whichever word is used, it includes organisms in the blood which cause, or are capable of causing, a disease. Appendix S summarises each of these provisions. (NSW differs from the other states and territories, as it is the only state that outlaws vilification on the grounds of HIV, of homosexuality and of being a transgender person. Vilification is defined as doing anything publicly that could encourage or incite hatred, contempt or severe ridicule). In addition, Commonwealth legislation also prohibits discrimination on the basis of disability and covers most service providers, public and private, in the country.

More pertinent questions for medical practitioners are, however, what constitutes discrimination on the basis of disability or impairment and what behaviour health-care practitioners must avoid when testing and treating people with HIV, HBV and HCV.

Discrimination on the basis of disability or impairment is, at its simplest, treating a person less favourably as a result of his or her (perceived) disability or impairment. Such treatment in a health-care setting could include refusing to see a patient or offering different or inappropriate treatment to the patient.

Most complaints about discrimination on the basis of HIV status are related to the perceived link between homosexuality and HIV status. Health-care workers must not treat someone as if they are HIV positive merely because they are homosexual and, similarly, they must not treat someone as homosexual merely because they are HIV positive. Treating homosexuality and HIV status as inextricably linked increases the stigma associated with each and makes both groups less likely to seek medical care.

Health complaints commissioners have, in the past, received complaints concerning doctors, dentists and other health-service workers placing persons...
with HIV infection last on their consultation lists. However, better training on effective infection control procedures appears to have been successful. Given the significant advances in treatment and the fact that most service providers and employers are required to employ standard precautions in infection control, organisations should be very slow to come to a decision that a person does pose a health risk. Discrimination legislation also places an onus to take an accommodating rather than exclusionary approach. Thus, rather than forcing a person to leave work or refusing to allow them to play sport, all possible accommodation of the disability or impairment should instead occur and any limitations on behaviour be only those necessary to protect the health of others.

Health-care workers with HIV, HCV or HBV infection

Health-care workers who perform exposure-prone procedures have a responsibility to know their infectious status with regard to HIV, HBV and HCV and are encouraged to undertake voluntary testing (Chapter 14). Health-care workers have an obligation to care for the safety of others in the workplace (including patients) under both common law and the Occupational Health and Safety and Welfare Act 1986.

Conclusion

It has often been noted that unlike many other infectious diseases, HIV, HBV and HCV are not easily transmitted to others. Hopefully a legal and social milieu, mindful of the impact of discriminatory and stigmatising behaviour, will facilitate an environment in which good health care is possible and the incidence of new infections is reduced.

References

Note that state specific guidelines also exist
CHAPTER 16 CONTACT AND REFERAL INFORMATION
The following list provides websites and phone numbers for a range of state, national and international organisations that will be useful to both clinicians and patients. For further details of HIV, sexual health and viral hepatitis specialists and services, please consult the ASHM Directory, available from the ASHM website at http://www.ashm.org.au/directory

<table>
<thead>
<tr>
<th>General contacts</th>
<th>Website</th>
<th>Phone number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australasian Society for HIV Medicine (ASHM)</td>
<td><a href="http://www.ashm.org.au">www.ashm.org.au</a></td>
<td>02 8204 0700</td>
</tr>
<tr>
<td>National Serology Reference Laboratory (NRL)</td>
<td><a href="http://www.nrl.gov.au">www.nrl.gov.au</a></td>
<td>03 9418 1111</td>
</tr>
</tbody>
</table>

**Professional bodies**

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<thead>
<tr>
<th>Professional bodies</th>
<th>Website</th>
<th>Phone number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australasian Sexual Health Alliance (ASHA)</td>
<td><a href="http://www.sexualhealthalliance.org.au/">http://www.sexualhealthalliance.org.au/</a></td>
<td></td>
</tr>
<tr>
<td>Australian College of Rural and Remote Medicine (ACRRM)</td>
<td><a href="http://www.acrrm.org.au">www.acrrm.org.au</a></td>
<td>07 3105 8200</td>
</tr>
<tr>
<td>Australasian Society for HIV Medicine (ASHM)</td>
<td><a href="http://www.ashm.org.au">www.ashm.org.au</a></td>
<td>02 8204 0700</td>
</tr>
<tr>
<td>Australasian Chapter of Sexual Health Medicine (ACHSHM)</td>
<td><a href="http://www.racp.edu.au">www.racp.edu.au</a></td>
<td>02 9256 9643</td>
</tr>
<tr>
<td>Gastroenterological Society of Australia (GESA)</td>
<td><a href="http://www.gesa.org.au">www.gesa.org.au</a></td>
<td>02 9256 5454</td>
</tr>
<tr>
<td>Royal Australian College of General Practitioners (RACGP)</td>
<td><a href="http://www.racgp.org.au">www.racgp.org.au</a></td>
<td>03 8699 0414</td>
</tr>
<tr>
<td>Royal Australian College of Physicians (RACP)</td>
<td><a href="http://www.racp.edu.au">www.racp.edu.au</a></td>
<td>02 9256 5444</td>
</tr>
<tr>
<td>Australian College of Nursing Australia</td>
<td><a href="http://www.acn.edu.au/">www.acn.edu.au/</a></td>
<td>02 6283 3400 or 1800 331 626</td>
</tr>
<tr>
<td>Royal College of Pathologists of Australasia (RCPA)</td>
<td><a href="http://www.rcpa.edu.au">www.rcpa.edu.au</a></td>
<td>02 8356 5858 1800 061 660</td>
</tr>
<tr>
<td>Australasian HIV and Sexual Health Nurses Association (ASHHNA)</td>
<td><a href="http://www.ashhna.org.au">www.ashhna.org.au</a></td>
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</tbody>
</table>

**INTERNET RESOURCES**

Below is a selection of useful and interesting websites for human immunodeficiency virus (HIV), hepatitis and sexually transmitted infections (STIs). This list is not intended to be exhaustive.

**HIV Australian**

- **Australasian Society for HIV Medicine** – www.ashm.org.au
- **Australian Research Centre in Sex, Health and Society** – www.latrobe.edu.au/arcshs/
- **Macfarlane Burnet Institute for Medical Research and Public Health** – www.burnet.edu.au
- **National Association of People Living with HIV Australia (NAPWHA)** – www.napwha.org.au
- **Kirby Institute** http://www.kirby.unsw.edu.au/ (formerly the National Centre in HIV Epidemiology and Clinical Research)
- **The Centre for Social Research in Health (CRSH)** https://crsh.arts.unsw.edu.au/
- **The National Drug and Alcohol Research Centre (NDARC)** – www.med.unsw.edu.au/ndarc/
- **Australian Federation of AIDS Organisations (AFAO)** – www.afao.org.au
- **AIDS Council of NSW (ACON)** – www.acon.org.au
- **HIV/AIDS Legal Centre** – www.halc.org.au
- **Queensland Association for Healthy Communities (QAHC)** – www.qahc.org.au
- **Tasmanian Council on AIDS, Hepatitis and Related Diseases (tasCAHRD)** – www.tascahrd.org.au
- **The AIDs Council of South Australia (ACSA)** – www.acsa.org.au
- **Northern Territory AIDS and Hepatitis Council (NTAHC)** – www.ntahc.org.au
- **The Western Australian AIDS Council (WAAC)** – www.waaid.org

**HIV: International**

- **AIDSmap** – www.aidsmap.com
- **AIDS.ORG** – www.aids.org
- **HIVdent** – www.hivdent.org
APPENDICES
### APPENDIX 1

**Patient information – natural history and transmission of HIV**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>What is HIV?</strong></td>
<td>Human immunodeficiency virus (HIV) is the virus that causes AIDS.</td>
</tr>
<tr>
<td><strong>What is AIDS?</strong></td>
<td>Acquired immune deficiency syndrome (AIDS).</td>
</tr>
<tr>
<td><strong>How does HIV affect people?</strong></td>
<td>When a person first contracts HIV, a flu-like illness called seroconversion illness may occur. In most cases, without treatment, HIV slowly causes damage to the immune system. The body becomes less able to fight infection and illness. As HIV infection advances, a person may develop AIDS. An AIDS diagnosis generally means that the immune system is severely weakened and that life-threatening illnesses may occur. These illnesses include opportunistic infections (e.g. pneumonia, recurrent respiratory tract infections) and some cancers such as Kaposi’s sarcoma (KS) and non-Hodgkin's lymphoma (NHL). Additionally, people with AIDS frequently have symptoms such as prolonged fevers, night sweats, swollen lymph nodes, chills, weakness and weight loss. Before treatments became available, it took an average of 8–12 years after initial HIV infection for AIDS to develop. Recently, more effective treatments have become available. As a result of these treatments, the life expectancy and quality of life of people with HIV now approaches and in some circumstances could equal that of people without HIV.</td>
</tr>
<tr>
<td><strong>How is HIV monitored?</strong></td>
<td>Regular check-ups and blood tests are conducted to monitor the progress of the disease, such as viral load and CD4 cell count. - viral load is the amount of virus in a sample, indicating viral activity - CD4 cell count indicates extent of damage to the immune system.</td>
</tr>
<tr>
<td><strong>How many people have HIV?</strong></td>
<td>In Australia in 2012, an estimated 25,708 people were living with diagnosed HIV infection giving a prevalence of 0.1%</td>
</tr>
<tr>
<td><strong>How is HIV transmitted?</strong></td>
<td>HIV is present in certain bodily fluids of people with the infection (i.e. blood, semen, vaginal fluids and breast milk). It may be passed on by sexual contact, exposure to infected body fluids or tissues, and from mother to child during pregnancy, delivery or breastfeeding. It is not passed on through normal household contact or by kissing.</td>
</tr>
<tr>
<td><strong>How is HIV transmission prevented?</strong></td>
<td>HIV transmission is significantly reduced by: - safer sex, which is any sexual activity that does not allow the transfer of one person’s body fluids (blood, semen, vaginal fluid) into another. This means reducing the number of sex partners and adopting safer sex practices, using condoms and water-based lubricants for any vaginal or anal intercourse and avoiding oral sex if there are cuts or sores on the genitals or in the mouth. If sex toys are shared, they should be covered by a condom (Chapter 3) - safe injecting using only sterile equipment (needles, syringes, swabs, spoons, filters, tourniquets and water) to inject each time or thoroughly cleaning equipment where this is not possible. Alternatively, drugs can be smoked, snorted or swallowed (Chapter 3 and Appendix 4) - testing for and treatment of STIs - interventions during pregnancy and labour, and avoidance of breastfeeding - standard precautions (Chapter 14). - infection can also be prevented by taking a course of post-exposure prophylaxis within 72 hours after being exposed to HIV, or by taking pre-exposure prophylaxis daily (Chapter 14) - HIV transmission from HIV-positive people can be substantially reduced by their early treatment which reduces the numbers of viruses in blood to undetectable levels. In general, HIV testing is an important part of HIV prevention as it enables early detection of HIV. As a result, steps can be made to link people with HIV to appropriate care which improves health outcomes, and prevents further transmission of HIV.</td>
</tr>
</tbody>
</table>
## Patient information – fact sheet for people living with chronic hepatitis B

### What is hepatitis B?

Hepatitis B is a liver infection caused by the hepatitis B virus. Your liver is very important for your wellbeing. When your liver is inflamed or damaged, it may not work properly and this can affect your health.

**Hepatitis B can be ‘acute’ or ‘chronic’**.

Most adults who acquire hepatitis B infection will get rid of the virus (clear it) within 6 months and develop life-long protection against it. This short-term illness is called acute hepatitis B. Once a person gets rid of the virus, he or she cannot get the hepatitis B infection again and cannot pass it on to others.

When the infection lasts for more than 6 months, the person has developed chronic hepatitis B. This happens to 90% of people who acquire the infection at birth or before 1 year of age. Most people in Australia with chronic hepatitis B got the hepatitis B infection at birth or in childhood, and not as adults.

Chronic hepatitis B can cause liver damage, liver scarring (cirrhosis) and liver cancer; however there are effective medicines that can greatly reduce damage and prevent cancer. There is also a lot you can do to help your liver. The most important thing is to have regular checks with your doctor regarding your hepatitis B, at least every 6 to 12 months.

### How do you get hepatitis B?

Hepatitis B is found in body fluids such as blood, semen and vaginal fluids of a person with hepatitis B infection. Hepatitis B is passed on (transmitted) when body fluids from a person with the infection enter another person’s bloodstream. Even amounts of fluid too small to be seen can transmit the virus.

In babies and young children transmission occurs:

- From a mother with hepatitis B to her baby around the time of birth if the baby is not vaccinated. This is the most common way the virus is spread worldwide.
- From a child with hepatitis B to another child who is not vaccinated against hepatitis B, through cuts and sores that are not covered.

In adults transmission occurs through:

- Vaginal, anal or oral sex without a condom.
- Sharing needles, syringes or any other equipment used to inject drugs.
- Tattooing or body piercing done with equipment that has not been sterilised properly.
- Sharing toothbrushes, razors, nail files or other personal items that may carry blood, including dry blood.
- Blood transfusions in some parts of the world, particularly in developing countries. In most developed countries including Australia, donated blood is checked for hepatitis B and other viruses so the risk of infection is extremely low.
- Medical and dental procedures, particularly in countries with less established health systems, may put people at risk, but very rarely cause infection in Australia and other countries with well-resourced health systems.
- Accidental injury with a needle or splashing of infected blood or body fluids, especially for healthcare workers.
- Contact sports.

Hepatitis B CANNOT be transmitted through:

- hugging.
- kissing.
- sharing food and eating utensils.
- insect bites.
- coughing.
- sharing bathroom and toilet facilities.
- swimming pools.
- breastfeeding, especially where the baby has been vaccinated against hepatitis B.
Regular checks with your doctor

Having chronic hepatitis B means you will need to think of your health a little differently. With many illnesses, you can tell if you are getting worse and need to see your doctor because you feel unwell. However, hepatitis B is different and you cannot rely on how you feel to know how the illness is affecting your liver. In fact, it is often the case that by the time you feel unwell, there is already liver damage.

Not every person with chronic hepatitis B will need treatment, but you will need to see your doctor every 6 to 12 months for regular checks, even if you feel well and have no symptoms. This is called monitoring.

Chronic hepatitis B is a complex disease that changes over time and it’s only through regular monitoring that you can know what chronic hepatitis B is doing to your liver and when to get treatment if you need it.

As well as blood tests, your doctor may order tests such as a Fibroscan®, a liver ultrasound or a liver scan. The tests allow your doctor to see if there have been any changes in the disease, if there is liver damage, scarring of the liver (cirrhosis) or cancer and decide if and when you may need treatment. If you need treatment, your doctor will refer you to a liver clinic or a liver specialist.

Seeing your doctor for regular monitoring is the most important thing you can do to look after yourself and your liver when you have chronic hepatitis B, as treatment at the right time can prevent scarring of the liver and cancer.

Treatment for hepatitis B

There are effective medications available that can control the virus. They can reduce the damage to your liver and the risk of liver cancer and also can help the liver repair itself.

The most common treatment consists of taking one pill a day. This treatment may last many years or continue for life.

There is another type of treatment that is offered to some patients. It consists of a weekly injection, for up to 12 months. This treatment can be very effective for some patients, but can have side effects.

Each treatment has different benefits and your doctor will discuss which one is best for you.

Reducing the risk of liver damage

There are a number of things you can do to reduce the risk of liver damage:

- drink less alcohol (for people with cirrhosis, it is recommended to drink no alcohol at all)
- eat a balanced healthy diet, avoiding too much fat
- there is some recent evidence that drinking coffee can be good for your liver
- maintain a healthy body weight
- stop smoking
- exercise regularly
- tell your doctor if you are taking any medicines or herbal remedies, including Chinese medicines. Some medications and herbs can be harmful to the liver
- protect yourself from other infections such as HIV and other hepatitis viruses, as they can severely affect your health and cause further liver damage:
  - get vaccinated for hepatitis A if you are not already protected against it
  - do not share equipment to inject drugs to avoid getting hepatitis C
  - practise safer sex (using condoms and lubricant) to avoid getting HIV
- Your doctor can refer you to services that can help you with these or may offer other support.

Protecting others from hepatitis B

You need to prevent passing hepatitis B on to others by taking the following precautions:

- make sure people you have close contact with are vaccinated against hepatitis B. Hepatitis B vaccine is often available free for household contacts and sexual partners of people living with hepatitis B
- practise safer sex: use condoms and lubricant during vaginal, anal and oral sex
- avoid blood-to-blood contact: do not share toothbrushes, razors or other personal items that may contain blood, including dry blood
- cover any open wounds and clean blood spills with bleach. Do not allow other people to touch your wounds or blood unless they are wearing gloves
- do not share needles, syringes or other equipment used to inject drugs
- do not donate blood, sperm, organs or body tissue
- if you are pregnant or planning to have a baby, talk to your doctor about the vaccinations your baby will need to be protected. You will be able to breastfeed
- if you are a health-care worker who performs invasive procedures (such as surgeons or dentists), you should seek expert medical advice, and expert occupational health and safety advice.
### Vaccination

The hepatitis B vaccine is very safe and provides immunity (protection against the virus) for almost all people who complete the course. The vaccine is usually given in two or three injections over 6 months, depending on the age of the person. In Australia, all babies are eligible for free vaccination. The first dose is given at birth, followed by three additional doses in the first year of life.

A baby born to a mother with hepatitis B will also receive hepatitis B immunoglobulin (HBIG) at birth. This adds to the protection provided by hepatitis B vaccine. Children of mothers who have hepatitis B should be tested to check whether they have become immune to hepatitis B or for hepatitis B infection at least 3 months after vaccination is completed.

Other people at high risk of contracting hepatitis B, such as household or sexual contacts of people living with hepatitis B, should also be tested 1 month after the final dose of vaccine, to show whether they have developed immunity or not.

### Do I need to tell others that I have hepatitis B?

While you don’t have to tell everyone that you have hepatitis B, you need to tell the people who live in your house and your sexual partner/s, so that they can be tested and vaccinated. If you need help telling them, talk to your doctor to get some advice.

There are also some situations in which you have to tell other people you have chronic hepatitis B. These include if:

- you are applying to join the Australian Defence Force
- your insurance company requires information about infections and illnesses
- you are a health-care worker who performs invasive procedures (such as surgeons or dentists)

You may want to tell your family so they can also be tested, especially if you come from a country where hepatitis B is common or you are Aboriginal or Torres Strait Island person.

Telling health-care workers, such as your dentist or other doctors, can help them give you the best medical care, but this is your choice. If you decide to tell them, they have a responsibility to protect your privacy and keep your information confidential, and they cannot discriminate against you.

You may find it helpful to talk to other people who can understand and support you, but should take your time to decide who you feel you can trust.

### Interpreters

Hepatitis B can be complex and difficult to understand. If you don’t speak or understand English well and you need help to communicate with your doctor, you can ask for an interpreter. An interpreter may help you to:

- understand everything you are being told
- ensure everything you say is understood
- ask questions and get answers
- give permission for tests or treatment.

Interpreters must protect your confidentiality.

Telephone interpreters can help you connect with services in your own language. Call TIS on 131 450 for the cost of a local call and ask to speak to someone in your language.

### Where can I find more information?

If you need more information talk to your GP or liver specialist. You can also check:

- Hepatitis Australia www.hepatitisaustralia.com
  For information in English and in other languages visit:
  - www.mhahs.org.au
  - http://www.hepatitisaustralia.com/community-resources/
- For personal stories of people who have chronic hepatitis B http://www.wdp.org.au/health-promotion/hepatitis-b

The Hepatitis Organisation in your state or territory can provide information about hepatitis B and what health and support services are available in your area.

- ACT www.hepatitisresourcecentre.com.au
- NSW www.hep.org.au
- NT www.ntahc.org.au
- Qld www.hepqld.asn.au
- SA www.hepatitissa.asn.au
- Tas www.tascahrd.org.au
- Vic www.hepvic.org.au
- WA www.hepatitiswa.com.au
Patient information – natural history and transmission of HCV

What is HCV?
Hepatitis C virus (HCV) is a blood-borne virus that can cause hepatitis. Hepatitis means inflammation of the liver.

What does the liver do?
The liver is the second largest organ and plays an important role in many vital functions of the body. Some of the liver’s many functions include:
- acting as a filter to remove alcohol and other toxic substances from the body
- processing and clearing drugs and medications
- manufacturing the many chemical substances needed by the body

How does HCV affect people?
When a person has HCV infection, his or her body produces tiny proteins called antibodies in an attempt to eliminate the virus. During the first 2–8 weeks, the person may feel slightly ill or off-colour but most people have no symptoms during this acute stage. Typical hepatitis symptoms (fatigue, yellowed skin or eyes) are uncommon with initial HCV infection.

Approximately 25% of people exposed to HCV will spontaneously clear the virus. The remaining 75% will progress to chronic HCV infection, which is a HCV infection that persists for more than 6 months. Most people eventually develop some signs of hepatitis C illness—usually after 10 years or so. The symptoms are generally mild for many people. However, for a minority of people, hepatitis C may be quite debilitating.

The exact nature and timing of short-term and long-term consequences of chronic HCV infection are not clear. The most commonly reported signs of hepatitis C illness are fatigue or pain in the upper right side of the abdomen. If chronic HCV remains untreated, approximately one person in five develops severe scarring of the liver or cirrhosis after 20 years of infection. About one in 20 people will develop liver failure or liver cancer after 30 years of infection. Liver disease can progress faster in some people due to lifestyle factors and other conditions, e.g. excessive alcohol consumption, obesity/insulin resistance and co-infection with hepatitis B virus (HBV) or human immunodeficiency virus (HIV). In a small proportion of people, the virus can cause problems in parts of the body other than the liver, such as the joints, skin and kidneys.

How many people have HCV?
It is estimated that:
- approximately 1–2 in every 100 Australians have HCV infection
- 80% of Australians with HCV infection acquired the virus through injecting drug use
- 5% of Australians living with HCV acquired the virus through transfusion of blood and blood products between the 1970s and 1980s
- 15% of Australians with HCV are from culturally and linguistically diverse backgrounds

How do people know what is happening to their liver?
A liver function test monitors the level of liver enzymes in the blood. If there is damage to liver cells, these levels may be raised. Liver function tests are often a poor indicator of illness outcome. It is recommended that a person with HCV is seen by a doctor every 6 to 12 months to see how the virus is affecting their liver. The doctor may refer them for a Fibroscan assessment, a non-invasive test that can measure liver stiffness and correlates with the level of fibrosis or scarring of the liver.

How is HCV transmitted?
HCV is spread through infected blood entering someone else’s bloodstream (e.g. by sharing and reusing contaminated syringes and other injecting equipment, non-sterile tattooing or piercing, and non-sterile medical procedures). Mother-to-child transmission is low and averages around 5%. Sexual transmission of HCV is regarded as rare, although anal penetration or the presence of blood may increase the risk of sexual transmission. The risk of sexual transmission may also be higher if the person has HIV and possibly other STI co-infection.

How is HCV transmission prevented?
The risk of HCV transmission is reduced by:
- safe injecting practices and avoid reusing items such as needles, tourniquets, spoons, swabs and snorting equipment (Chapter 3 and Appendix 4)
- avoiding sharing of household items such as razors or toothbrushes
- standard infection control precautions
- use of condoms if engaging in high risk sexual practices among men who have sex with men (MSM) or if HIV and HCV co-infection is present

Can HCV be treated?
Effective treatment for HCV is available, with up to 90% of people clearing the virus. The combination of drugs used will vary depending on the type of HCV (genotype) a person has. Treatment currently consists of pegylated interferon and ribavirin and, if a person has genotype 1, boceprevir or telaprevir is added. The length of treatment will also vary depending on the genotype, the presence of cirrhosis (scarring of the liver) and how the person responds to the treatment. Generally, current treatment ranges in length from 24 to 48 weeks. There are potential side effects associated with current HCV treatment, but close monitoring will help to minimise and manage these.
### APPENDIX 4

**Safer injecting and cleaning injecting equipment**

The sharing of injecting equipment is the single greatest risk factor for contracting hepatitis C virus (HCV) among those who inject drugs. There are options other than injecting drugs, such as smoking, snorting or swallowing drugs, which will significantly reduce the risk of contracting HCV, human immunodeficiency virus (HIV) and other blood-borne viruses. If snorting is the alternative mode of administration, the sharing of straws is not recommended due to a low risk of HCV transmission.

**Injecting with sterile equipment**

For people who do choose to inject drugs, transmission can be prevented through the exclusive use of sterile fits (needle and syringe), water and swabs (one to swab the spoon and one to swab the injecting site), clean filters, a clean and detachable tourniquet and clean hands. The injecting space should also be clean and all blood contact avoided. Sterile equipment is equipment that has undergone a process that destroys viruses, bacteria and germs. Sterile injecting equipment includes pre-packaged fits, water and swabs, that are marked as sterile. All other equipment needs to be cleaned with soap and water or with a swab.

There is no way of eliminating the risk of viral transmission from used syringes. If patients seek advice about re-using injecting equipment, the need for sterile equipment must be reiterated.

**Using cleaned injecting equipment**

People who decide to inject with a used fit must be advised that they risk acquiring HIV, hepatitis B virus (HBV) and HCV infection. In addition, they should be advised that:

- Using your own fit will be safer than a fit used by another person
- The more thoroughly a fit is cleaned, the less risk of infection
- Cleaning is important for people who already have an infection with a blood-borne virus because they can re-acquire infection with another strain of HCV, HBV or HIV. Re-infection with another strain of HCV or another hepatitis virus may place added strain on the liver.

**How to clean injecting equipment**

The following are directions on how to clean used injecting equipment. Clean workspace equipment and a safe area for fluid disposal (sink, bin, drain) are required. Hands should be washed before beginning.

1. **Equipment**
   - Three separate containers filled with:
     - Clean water from the cold tap, for rinsing the blood out of the fit.
     - Use water from the cold tap – preferably soapy water. This is best for rinsing out blood because water that is too hot or too cold can cause the blood to congeal and stick inside the fit, where it can shed microscopic particles into the mix.
     - Full strength bleach for soaking and bleaching the fit (5.25% sodium hypochlorite).
   - Clean water from the cold tap to rinse the bleach from the fit.

2. **Cleaning process**
   - **a. Rinsing:**
     - Rinse the fit in clean water from the cold tap from the first container.
     - Squirt the water out into the sink or safe fluid disposal area.
     - Repeat a number of times.
   - **b. Bleaching:**
     - Use full strength bleach (at least 5.25% sodium hypochlorite and check the use-by date).
     - Take the fit apart.
     - Soak it completely, covering it with bleach for at least 2 minutes.
     - **If you can't soak:**
       - Draw the bleach into the fit and shake it for at least 30 seconds (or while you count slowly to 30).
       - Squirt the bleach out into the sink or safe disposal area.
       - Repeat the process at least once, again counting slowly to 30 (as above).
   - **c. Flushing:**
     - Draw up fresh water from the third container.
     - Do NOT use water from the FIRST container as this has been contaminated with blood.
     - Squirt, flushing the water into the safe fluid disposal area or sink.
     - Repeat flushing process until all the bleach has been removed.
     - **FLUSH AT LEAST SIX TIMES.**

3. **When the fit has been cleaned follow the guidelines for safer injecting**
4. Some handy hints for being blood aware

- Stock up on sterile equipment so you won’t be caught short.
- Make sure the surface where you prepare your shot is clean.
- Wash your hands with warm soapy water before and after injecting. This will remove any traces of blood from your fingers, as well as any unhygienic dirt.
- No matter how well cleaned, never let your used equipment, or anyone else’s used equipment, come into contact with a group mix. Unless sterile fits are used to mix and divide up, then each member of the group must have their own water, spoon and filter (as well as their own fit).
- If someone is going to help you inject, make sure they wash their hands before and after.
- It is best to have your own tourniquet that you don’t share. Try not to get blood on your tourniquet. Detachable (medical) tourniquets will make this easier.
- Rinse your fit in clean water from the cold tap immediately after your shot, even if you are disposing of it. This will remove most of the blood that is present, and therefore reduce the chance of a virus staying alive in your fit. It will also prevent it from blocking, and help reduce the likelihood of a dirty shot if you have to use the fit again.
- If you are going to save your fit for personal re-use, keep track of it (mark it), and keep it safe and separate from others.
- Wash or swab your spoon after each shot, and wash your tourniquet with soapy water as soon as possible to remove blood spills.
- Always dispose of fits safely, in an approved disposal bin, sharps container or childproof, puncture-proof container. Whenever possible, return sharps containers and used fits to your local needle and syringe program.
- Do not reuse swabs, filters or opened sterile water: they become contaminated with bacteria and fungi when exposed to air. Dispose of them in the recommended sharps container you have used to dispose of your used fits, or place inside two plastic bags (double bagging). Return your sharps container to your local needle and syringe program.
- Also dispose of blood-contaminated materials as above. If you get blood on your clothes, throw them straight into the wash with a good measure of washing powder.

Appendix 4 adapted from:
### APPENDIX 5
Laws as they apply in each State and Territory

#### Commonwealth

<table>
<thead>
<tr>
<th>Subject</th>
<th>Section</th>
<th>Act</th>
<th>Notes / Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Privacy</td>
<td></td>
<td>Privacy Act 1988</td>
<td>Sets out the National Privacy Principles (NPP)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>The NPP govern how all health workers and hospitals must collect, handle, retain and disclose personal information.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>The general rule is that information should only be collected when necessary and the information should not be disclosed to any third party.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Privacy Amendment (Private Sector) Act 2000</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Extends the operation of the NPP to all private sector workers, including health workers.</td>
</tr>
</tbody>
</table>

#### Australian Capital Territory

<table>
<thead>
<tr>
<th>Subject</th>
<th>Section</th>
<th>Act</th>
<th>Notes / Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Notifiable condition</td>
<td>Schedule 1</td>
<td>Public Health Notifiable Condition Determination 2005</td>
<td>HV, AIDS and viral hepatitis are notifiable conditions.</td>
</tr>
<tr>
<td>Notification to patient</td>
<td>s 102</td>
<td>Public Health Act 1997</td>
<td>If a doctor or nurse believes a person has or may have a notifiable condition, he or she must give the patient information about transmission and prevention of transmission of that condition. Failure to do so may have implications under other legislation.</td>
</tr>
<tr>
<td>Notification to health department</td>
<td>s 102A</td>
<td>Public Health Act 1997</td>
<td>Doctor or nurse commits an offence if he or she believes a person has a notifiable condition and he or she fails to notify the Chief Health Officer (CHO). Doctor must also notify the CHO if a patient of the doctor dies of what the doctor believes is a notifiable condition.</td>
</tr>
<tr>
<td>Notification by pathologist</td>
<td>s 103</td>
<td>Public Health Act 1997</td>
<td>If a pathologist tests a person and the test indicates the person has or may have a notifiable condition, the pathologist, or the person in charge of the laboratory, must notify the CHO.</td>
</tr>
<tr>
<td>Notification by hospital</td>
<td>s 104</td>
<td>Public Health Act 1997</td>
<td>Hospital must notify CHO of any in-patient with a notifiable condition.</td>
</tr>
<tr>
<td>Contact tracing</td>
<td>s 106</td>
<td>Public Health Act 1997</td>
<td>Where an authorised officer believes on reasonable grounds a person has a notifiable condition, he or she may request that the person provide:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• The person’s name and address</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Information about how the person acquired the condition</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Information about the circumstances under which the person may have transmitted the condition</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• The name and address of any contact of the person</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>The person must not refuse this request without a reasonable excuse.</td>
</tr>
<tr>
<td>Informing contacts</td>
<td>s 108</td>
<td>Public Health Act 1997</td>
<td>If a person with a notifiable condition advise a responsible person that he or she refuses to tell a contact about the condition and refuses to give the responsible person permission to do so, the responsible person may contact the CHO who may then inform the contact. A responsible person is a doctor, nurse practitioner, counsellor or person responsible for the care, support and education of the person.</td>
</tr>
</tbody>
</table>
Privacy | s 110 | Public Health Act 1997 | A person shall not without good reason or consent of that person disclose any information regarding a person having a notifiable condition, unless for the purposes of the Act, or another law of Commonwealth, State or Territory, or authorised under a code of practice.

Privacy of doctors, pathologists | s 111 | Public Health Act 1997 | It is an offence for a person to disclose without reasonable excuse or consent any information regarding a person with a notifiable condition in which the doctor, pathologist or other health-care provider is identifiable.

### Sex work

Sexually transmitted infection (STI) | Dictionary | Prostitution Act 1992 | HIV is an STI. Hepatitis is not.

Operator responsibility | s 24 | Prostitution Act 1992 | Operator of a brothel must take reasonable steps to ensure that a sex worker does not provide commercial sexual service if the sex worker has an STI.

Sex worker responsibility | s 25 | Prostitution Act 1992 | A person shall not provide or receive commercial sexual services if he or she knows or can reasonably be expected to know he or she has an STI.

Medical testing | s 26 | Prostitution Act 1992 | It is an offence for an operator or owner of a brothel to fail to take reasonable steps to ensure that sex workers receive regular medical testing for STIs. It is also an offence for a sex worker to mislead a person about the results of a test.

Condoms | s 27 | Prostitution Act 1992 | Operator or owner of a brothel must take all reasonable steps to ensure that condoms are used. Penalties are provided for failure to comply.

### Offences

Unauthorised assertions | s 107 | Public Health Act 1997 | It is an offence to assert to a person who has been exposed to or may be a source of infection that a third person has a transmissible notifiable condition without the consent of the third person.

Transmission | r 21 | Public Health Regulation 2000 | A person with a transmissible notifiable condition must take reasonable precautions not to pass that condition on to another person. Penalty is 10 penalty units.

Non-discrimination | s 7 (j) | Discrimination Act 1991 | Must not discriminate on the basis of disability which includes ‘the presence in the body of organisms that cause or are capable of causing disease’ (s 5AA(e))

The ACT does not have a separate offence for transmitting a serious disease and the Crimes Act 1900 does not define harm to include inflicting a disease.

### New South Wales

<table>
<thead>
<tr>
<th>Subject</th>
<th>Section</th>
<th>Act</th>
<th>Notes / Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scheduled medical condition</td>
<td>Schedule 1</td>
<td>Public Health Act 1991</td>
<td>NSW legislation divides medical conditions into 5 categories. HIV and hepatitis B and C are Category 3 conditions. HIV and AIDS are Category 5 conditions. AIDS is also a Schedule 3 notifiable disease.</td>
</tr>
<tr>
<td>Notification</td>
<td>s 16</td>
<td>Public Health Act 1991</td>
<td>A positive test result for a Category 3 medical condition must be notified to the Director General in an approved form. Where the positive test result is for a medical condition that is also a Category 5 medical condition, such notification must not disclose the person’s name and address. The obligation falls on the person who certifies the result of the test, not the treating doctor.</td>
</tr>
</tbody>
</table>
**Confidentiality**

<table>
<thead>
<tr>
<th>Section</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>s 17</td>
<td>Public Health Act 1991</td>
<td>Notification to the Director General of a Category 5 medical condition must not include the person's name or address. A person who acquires information about Category 5 testing must take reasonable steps to prevent disclosure unless disclosure is with consent, in the course of administration of the Act, by court order or to a person involved in the care, treatment or counselling of the person affected.</td>
</tr>
</tbody>
</table>

**Advice**

<table>
<thead>
<tr>
<th>Regulation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>r 12</td>
<td>The Director General or an authorised medical practitioner may notify a person with a Category 2 or 3 condition of measures to be taken and activities to be avoided in order to minimise the danger of passing the condition to another person.</td>
</tr>
</tbody>
</table>

**Contact tracing**

<table>
<thead>
<tr>
<th>Regulation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>r 13</td>
<td>The Director General may notify a person whom they reasonably believe may have been in contact with a person suffering from a Category 2, 3 or 4 medical condition of measures to be taken, and activities to be avoided, in order to minimise the danger of the first person contracting the condition or passing it to a third person.</td>
</tr>
</tbody>
</table>

**Public Health Orders**

**Examination**

<table>
<thead>
<tr>
<th>Section</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>s 22</td>
<td>Public Health Act 1991</td>
<td>The Director General may make an order requiring that a person be tested for a Category 4 or Category 5 medical condition if the Director General believes on reasonable grounds that the person is suffering from a Category 4 or Category 5 medical condition.</td>
</tr>
</tbody>
</table>

**Behaviour**

<table>
<thead>
<tr>
<th>Section</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>s 23</td>
<td>Public Health Act 1991</td>
<td>The Chief Health Officer or a medical practitioner authorised by the Director General may make a public health order (PHO) if a person is believed on reasonable grounds to be suffering from a Category 4 or Category 5 medical condition and is behaving in a way that is endangering or is likely to endanger the public health. A PHO may require that the person refrain from specified conduct, undergo counselling or specified treatment, or if the PHO is based on a Category 5 medical condition, be detained at a specified place.</td>
</tr>
</tbody>
</table>

**Confirmation by Tribunal**

<table>
<thead>
<tr>
<th>Section</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>s 25</td>
<td>Public Health Act 1991</td>
<td>A PHO based on a Category 5 medical condition ceases to have effect if an application to the Administrative Decisions Tribunal is not served on the person to whom the PHO applies within 3 business days of the PHO, or if the Tribunal does not confirm the PHO, or vary the PHO and confirm it as varied.</td>
</tr>
</tbody>
</table>

**Offence to contravene**

<table>
<thead>
<tr>
<th>Section</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>s 28</td>
<td>Public Health Act 1991</td>
<td>It is an offence for a person to whom a PHO applies to contravene a PHO.</td>
</tr>
</tbody>
</table>

**Apprehension**

<table>
<thead>
<tr>
<th>Section</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>s 29</td>
<td>Public Health Act 1991</td>
<td>An authorised medical practitioner can give a certificate which is the grounds for apprehension by a police officer of a person who has contravened a PHO.</td>
</tr>
</tbody>
</table>

**Revocation**

<table>
<thead>
<tr>
<th>Section</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>s 31</td>
<td>Public Health Act 1991</td>
<td>If an authorised medical practitioner considers that the person is no longer a risk to public health, the practitioner must revoke the order immediately.</td>
</tr>
</tbody>
</table>

**Unlawful release**

<table>
<thead>
<tr>
<th>Section</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>s 34</td>
<td>Public Health Act 1991</td>
<td>It is an offence to release, or to attempt to release, a person detained under a PHO without lawful authority to do so.</td>
</tr>
</tbody>
</table>

**Transfer of human tissue**

**Prescribed contaminant**

<table>
<thead>
<tr>
<th>Regulation</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>r 14</td>
<td>Human Tissue Regulation 2005</td>
<td>HIV and viral hepatitis are prescribed contaminants.</td>
</tr>
</tbody>
</table>

**False statements**

<table>
<thead>
<tr>
<th>Section</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>s 20E</td>
<td>Human Tissue Act 1983</td>
<td>A donor must not sign a certificate that is false or misleading in a material particular.</td>
</tr>
</tbody>
</table>

**Restrictions on liability**

<table>
<thead>
<tr>
<th>Section</th>
<th>Act</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>s 20F</td>
<td>Human Tissue Act 1983</td>
<td>No action lies against the donor of blood unless the donor has signed a false certificate. No action lies against a supplier of blood products if the supplier was an exempt supplier, and the donor signed a certificate, and tests indicated that no prescribed contaminant was present in the blood.</td>
</tr>
</tbody>
</table>
### Correctional setting

**s 73** | **Crimes (Administration of Sentences) Act 1999** | Any prisoner can be forced to undergo any medical procedure deemed necessary by Justice Health for the preservation of the prisoner’s life or to prevent serious damage to the prisoner. This includes testing for HIV, HBV and HCV.

### Offences

<table>
<thead>
<tr>
<th>Offence</th>
<th>Section</th>
<th>Act</th>
<th>Notes / Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sexually transmissible medical condition</td>
<td>s 13</td>
<td>Public Health Act 1991</td>
<td>It is an offence for a person who knows they have a sexually transmissible medical condition to have intercourse with another person unless that person has been informed of the risk of contracting the disease and voluntarily accepts that risk. It is also an offence for an owner or occupier of premises who knowingly permits a person with a sexually transmissible medical condition to have intercourse with another person on their premises. Maximum penalty of 50 penalty units ($5500).</td>
</tr>
</tbody>
</table>

### Northern Territory

<table>
<thead>
<tr>
<th>Subject</th>
<th>Section</th>
<th>Act</th>
<th>Notes / Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Notifiable disease</td>
<td>s 6</td>
<td>Notifiable Diseases Act 1999</td>
<td>Minister may by notification in the Gazette declare a disease to be a notifiable disease.</td>
</tr>
</tbody>
</table>

### Notification

<table>
<thead>
<tr>
<th>Subject</th>
<th>Section</th>
<th>Act</th>
<th>Notes / Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical practitioner</td>
<td>s 8</td>
<td>Notifiable Diseases Act 1999</td>
<td>If a medical practitioner diagnoses that a person has an infection or considers that a person is a suspect person in relation to notifiable disease, the medical person must give specified information about the notifiable disease to a medical officer.</td>
</tr>
<tr>
<td>Pathology laboratory</td>
<td>s 16</td>
<td>Notifiable Diseases Act 1999</td>
<td>If a pathology laboratory diagnoses a person with a notifiable disease, the person in charge must give specified information about the notifiable disease to the Chief Health Officer (CHO).</td>
</tr>
<tr>
<td>Proprietor of hotel, hostel, boarding house</td>
<td>r 48</td>
<td>Public Health (Shops, Boarding Houses, Hostels and Hotels) Regulations</td>
<td>Proprietor who becomes aware that any person is suffering from or suspected to be suffering from an infectious disease on a premises must immediately notify the Medical Officer of Health of the circumstances, and must isolate the person.</td>
</tr>
<tr>
<td>Advice</td>
<td>s 10</td>
<td>Notifiable Diseases Act 1999</td>
<td>When a doctor diagnoses a notifiable disease, he or she must explain the nature of the disease and the measures necessary to prevent the spread of the disease. That advice may be provided to the parents of a person under 18 years of age.</td>
</tr>
<tr>
<td>Disclosure protected</td>
<td>s 30</td>
<td>Notifiable Diseases Act 1999</td>
<td>No action lies against a person including doctor or pathology laboratory for notifying the Minister or other person as required.</td>
</tr>
</tbody>
</table>
### Testing

<table>
<thead>
<tr>
<th>Person</th>
<th>s 7</th>
<th>Notifiable Diseases Act 1999</th>
<th>A person who has reasonable grounds to believe he or she may have an infection or suspected infection shall consult a medical practitioner at the first reasonable opportunity.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contact tracing</td>
<td>s 9</td>
<td>Notifiable Diseases Act 1999</td>
<td>A person who has an infection shall provide to a doctor or authorised person either the name and address of person he or she may have contracted the disease from or the name and address of all persons he or she has been in contact with during a specified period.</td>
</tr>
<tr>
<td>Behavioural order</td>
<td>s 11</td>
<td>Notifiable Diseases Act 1999</td>
<td>A medical officer may serve a person with infection with a notice in writing directing the person to carry out measures the officer believes necessary for the treatment or to prevent transmission of the disease.</td>
</tr>
<tr>
<td>Appeal</td>
<td>s 12</td>
<td>Notifiable Diseases Act 1999</td>
<td>A person can appeal to the Local Court against a notice given under s 11.</td>
</tr>
<tr>
<td>Enforcement</td>
<td>s 13</td>
<td>Notifiable Diseases Act 1999</td>
<td>The CHO may order compliance with an s 11 notice. CHO may also order orally or in writing that the person with the infection or suspected infection be detained in hospital, that premises be disinfected and bedding destroyed and take all steps necessary to give effect to the order made by the CHO.</td>
</tr>
<tr>
<td>Notice to attend</td>
<td>s 14</td>
<td>Notifiable Diseases Act 1999</td>
<td>CHO may by notice in the Gazette require a person or class of persons to attend at specified times for medical examination.</td>
</tr>
</tbody>
</table>

### Blood donation

| Liability of Red Cross | s 26B | Notifiable Diseases Act 1999 | In an action against the Red Cross for transmitting a notifiable disease through blood transfusion, it is a defence if the Red Cross complied with the specified requirements in taking, testing, processing and handling the blood. |

### Employment

| Barbers | r 18 | Public Health (Barbers' Shops) Regulations | A barber suffering from a contagious disease shall not attend to a customer. |
| Taxi | r 12 | Taxis Regulations | A taxi driver may refuse to pick up a person who is apparently suffering from an infectious disease. |
| Bus | r 45 | Motor Omnibus Regulations | A conductor of an omnibus shall not allow a person suffering from an infectious or contagious disease to be carried in the omnibus. |
| Correctional setting | s 75 | Prisons (Correctional Services) Act | If in the opinion of a visiting medical officer a prisoner is deemed a threat to him- or herself or others, the Director can order medical examination and treatment, including the provision of blood or bodily secretions. |

### Offences

| Bribe | s 35 | Notifiable Diseases Act 1999 | A medical practitioner or authorised person commits an offence if he or she accepts a reward on account of a failure to perform his or her duty. |
| Recklessly endangering life | s 174C | Criminal Code Act | Creates offence if reckless conduct gives rise to danger of death. |
| Recklessly endangering serious harm | s 174D | Criminal Code Act | Creates offence if reckless conduct gives rise to danger of serious harm. |
| Negligently causing serious harm | s 174E | Criminal Code Act | Creates offence if conduct negligently causes serious harm. |
### Queensland

<table>
<thead>
<tr>
<th>Subject</th>
<th>Section</th>
<th>Act</th>
<th>Notes / Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Notifiable condition</td>
<td>s 64(1)</td>
<td>Public Health Act 2005</td>
<td>Notifiable condition is defined in the regulations.</td>
</tr>
<tr>
<td>Schedule 1</td>
<td>Public Health Regulation 2005</td>
<td>HIV, AIDS and all hepatitis are notifiable conditions.</td>
<td></td>
</tr>
<tr>
<td>Notifiable diseases</td>
<td>Schedule 6</td>
<td>Stock Regulation 1988</td>
<td>The term 'notifiable disease' applies to animals and does not include HIV or AIDS.</td>
</tr>
<tr>
<td>Notifiable Conditions Register</td>
<td>s 67</td>
<td>Public Health Act 2005</td>
<td>Chief Executive (CE) must create and maintain a register of persons about whom notification has been received.</td>
</tr>
<tr>
<td>Notification by doctor</td>
<td>s 70</td>
<td>Public Health Act 2005</td>
<td>Doctor must notify CE when a person is diagnosed or provisionally diagnosed with a notifiable condition.</td>
</tr>
<tr>
<td>Anonymity</td>
<td>s 74</td>
<td>Public Health Act 2005</td>
<td>Notification may occur with an anonymity code.</td>
</tr>
<tr>
<td>Confidentiality</td>
<td>s 77</td>
<td>Public Health Act 2005</td>
<td>Confidential information must not be disclosed.</td>
</tr>
<tr>
<td></td>
<td>s 81</td>
<td>Public Health Act 2005</td>
<td>Confidential information can be disclosed where the disclosure is in the public interest (as judged by the CE and where the CE has, in writing, authorised the disclosure).</td>
</tr>
</tbody>
</table>

### Contact tracing

| Disclosure                    | s 80    | Public Health Act 2005                             | The prohibitions against disclosure of information in s 77 do not apply where the information is disclosed with authorisation by CE for the purpose of monitoring the patterns of notifiable conditions; identifying the sources of outbreaks; identifying persons who may transmit a notifiable condition to others; identifying persons who may have contracted, or may be at risk of contracting a notifiable condition to prevent or minimise transmission of the condition; or contact tracing by a contact tracing officer. |
| Functions                     | s 89    | Public Health Act 2005                             | A contact tracing officer has the following functions:                                            |
|                               |         |                                                    | a. identifying persons who may have contracted a notifiable condition                              |
|                               |         |                                                    | b. identifying persons who may transmit a notifiable condition to others                           |
|                               |         |                                                    | c. informing persons who may have contracted a notifiable condition so that they may seek medical examination and treatment |
|                               |         |                                                    | d. providing information to persons who may have contracted a notifiable condition to prevent or minimise transmission of the notifiable condition |
|                               |         |                                                    | e. obtaining information about the following to prevent or minimise transmission of a notifiable condition— |
|                               |         |                                                    | i. how a person has, or may have, been exposed to the notifiable condition                        |
|                               |         |                                                    | ii. how a person has, or may have, exposed other persons to the notifiable condition.            |
| Power to require information  | s 99    | Public Health Act 2005                             | Where a contact tracing officer reasonably believes that a person has a notifiable condition or has been in contact with someone who has a notifiable condition, the officer can, after explaining to the person that information is needed to attempt to prevent or minimise the spread of the condition, require that person to provide his or her name, address and the name and address of any person who may have transmitted the condition to the person or to whom the person may have transmitted the condition. |
| An offence not to provide information under s 99 | s 100 | Public Health Act 2005 | Person must comply with request under s 99 unless the person has a 'reasonable excuse'
|---|---|---|---
| Detention | s 113 | Public Health Act 2005 | CE may order that a person be detained if the CE believes 'the person's condition and likely behaviour constitutes an immediate risk to public health; and is satisfied the person has been counselled, or reasonable attempts have been made to counsel the person about the condition and its possible effect on the person's health and on public health.'
| Orders by magistrate Detention | s 116 | Public Health Act 2005 | Following a sworn application, CE may obtain order for detention from magistrate. Orders including a detention order may be made in the person's absence if magistrate believes the person represents an immediate risk to public health.
| Initial examination | s 118 | Public Health Act 2005 | If satisfied that a person has a controlled notifiable condition and that a medical examination is necessary, Magistrate may make an 'Initial Examination Order' requiring that a person submit to examination for a notifiable condition.
| Behavioural | s 125 | Public Health Act 2005 | If satisfied that a person has a controlled notifiable condition and the condition constitutes an immediate risk to public health, a Magistrate may make a 'Behavioural Order'.
| Detention | s 129 | Public Health Act 2005 | If satisfied that a person has a controlled notifiable condition and the condition constitutes an immediate risk to public health a Magistrate may issue a 'Detention Order' which requires that the person remain in detention for not more than 28 days.
| Warrant | s 136 | Public Health Act 2005 | Warrant for apprehension of a person may be issued to enforce Initial Examination Order or Detention Order.
| Correctional settings | s 21 | Corrective Services Act 2006 | A prisoner must submit to any medical examination or treatment deemed necessary by a doctor.
| Offences | | | |
| Recklessly spreading disease | s 143 | Public Health Act 2005 | Person must not recklessly spread a controlled notifiable condition.
| | | | It is a defence if the person placed at risk knew of the condition and voluntarily accepted the risk.
| | | | Maximum penalty of 400 penalty units ($30 000) or 2 years imprisonment.
| Grievous bodily harm | s 317 | Criminal Code 1899 | Offence to intentionally transmit a serious disease to another person.
| | | | Liable to life imprisonment.
| | | | A 'serious disease' is defined in s 1 as one that is likely to endanger life and would include HIV, AIDS and hepatitis.
| Risk minimisation | s 151 | Public Health Act 2005 | Every person involved in the provision of a declared health service must take reasonable precautions to minimise risk of infection to other people.
| | | | This includes dentists, nurses, employers with a first aid room and others.
| Employment | | | |
| Therapeutic goods | s 176 | Health Regulation 1996 | Person suffering from any contagious or infectious disease cannot be employed for the purpose of making therapeutic goods.
Sex work

s 77A

Prostitution Act 1999

It is an offence for a sex worker to provide or offer to provide sex services unless a prophylactic is used. A licensee or approved manager must take reasonable steps to ensure a prophylactic is used.

Sexually transmissible infections

Schedule 4

Prostitution Act 1999

HIV is an STI for the purposes of the Act.

s 90

Prostitution Act 1999

Sex workers with an STI are not allowed to work as a sex worker at a licensed brothel.

Non-discrimination

s 7(h)

Anti-Discrimination Act 1991

Must not discriminate on the ground of ‘impairment’ which includes ‘the presence in the body of organisms capable of causing illness or disease’ (Schedule Dictionary).

South Australia

<table>
<thead>
<tr>
<th>Subject</th>
<th>Section</th>
<th>Act</th>
<th>Notes / Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Notifiable disease</td>
<td>Schedule 1</td>
<td>Public and Environmental Health Act 1987</td>
<td>HIV, AIDS and viral hepatitis are notifiable diseases.</td>
</tr>
<tr>
<td>Controlled notifiable disease (CND)</td>
<td>Schedule 2</td>
<td>Public and Environmental Health Act 1987</td>
<td>HIV, AIDS and viral hepatitis are CNDs.</td>
</tr>
</tbody>
</table>
| Notification                  | s 30    | Public and Environmental Health Act 1987 | Where a doctor or person prescribed by regulation believes a person is suffering from a notifiable disease, he or she must inform the Department within 3 days.
|                              |         |                                          | If after receipt of the report the Department believes the person poses an immediate threat to public health it must notify the council for the Local Government area in which the person resides. |
| Order testing                 | s 31    | Public and Environmental Health Act 1987 | Where the South Australian Health Commission believes a person has a CND, it can require by written notice that the person be examined for that disease.
|                              |         |                                          | If the person refuses to be tested, a magistrate may issue a warrant requiring apprehension and examination. |
| Quarantine                    | s 32    | Public and Environmental Health Act 1987 | Where a medical practitioner has certified that a person has a CND and the Commission believes that person poses a risk to public health, a magistrate may issue a warrant for detention at a place of quarantine.
<p>|                              |         |                                          | The order is only to be for 3 days and after that time must be renewed by a magistrate up to a maximum of 6 months. The Supreme Court can extend an order beyond 6 months. |
| Behaviour                     | s 33    | Public and Environmental Health Act 1987 | The Commission may give appropriate directions to a person suffering from a CND to reside at a specified place, refrain from performing specified work, submit to an examination or other directions in order to limit the spread of that disease. |
| Report to Councils            | s 35    | Public and Environmental Health Act 1987 | The Department shall on a monthly basis provide reports to Local Councils as to the occurrence of notifiable diseases in its area and the threat, if any, they pose. |
| Actions to prevent spread of disease | s 36    | Public and Environmental Health Act 1987 | The Commission may take any action necessary to prevent the spread of a notifiable disease. |
| Sex work                      | s 13 s 21 s 25A | Summary Offences Act 1953 | It is an offence to consort with sex workers, occupy premises frequented by sex workers or engage in procurement for sex work. |
| Preventing transmission       | s 37    | Public and Environmental Health Act 1987 | A person with a CND will take all reasonable steps to prevent transmission of the disease to others. |</p>
<table>
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<tr>
<th>Subject</th>
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</thead>
<tbody>
<tr>
<td>Causing harm</td>
<td>s 23  s 24</td>
<td>Criminal Law Consolidation Act 1935</td>
<td>It is an offence to cause harm or serious harm to another person with intent to cause harm or serious harm. Physical harm includes infection with a disease (s 21).</td>
</tr>
<tr>
<td>Non-discrimination</td>
<td>Part 5</td>
<td>Equal Opportunity Act 1984</td>
<td>Part 5 prohibits discrimination on the basis of ‘mental or physical impairment’.</td>
</tr>
<tr>
<td>Infectious disease</td>
<td>Schedule 2</td>
<td>Health (Infectious Diseases) Regulations 2001</td>
<td>HIV, AIDS and viral hepatitis are infectious diseases.</td>
</tr>
<tr>
<td>Notifiable disease</td>
<td>Schedule 3</td>
<td>Health (Infectious Diseases) Regulations 2001</td>
<td>HIV and AIDS are notifiable diseases.</td>
</tr>
<tr>
<td>Order for test when spread by or to a care giver</td>
<td>s 120A</td>
<td>Health Act 1958</td>
<td>If the Secretary reasonably believes that an incident has occurred in which an infectious disease could have been transmitted while a care-giver or custodian is acting in that capacity and any of those people to whom the disease could have been transmitted have been counselled about the risk of transmission of the disease, the Secretary may make an Order requiring the person named in the Order to be tested for the disease.</td>
</tr>
<tr>
<td></td>
<td>s 120AB</td>
<td>Health Act 1958</td>
<td>These powers can also be exercised by a senior medical officer at a hospital.</td>
</tr>
<tr>
<td></td>
<td>s 120B</td>
<td>Health Act 1958</td>
<td>If the circumstances in s 120A apply and a specimen of the person's blood is available, that may be tested rather than making an order to have the person tested.</td>
</tr>
<tr>
<td></td>
<td>s 120D</td>
<td>Health Act 1958</td>
<td>When advising a person that he or she needs to be tested, the Secretary or authorised officer must not divulge the name of the person from whom the disease originated.</td>
</tr>
<tr>
<td>General orders</td>
<td>s 121</td>
<td>Health Act 1958</td>
<td>Secretary can order in writing a person to be tested if he or she reasonably believes a person has an infection or has been exposed to infection, the person is likely to transmit the disease and there is a serious risk to public health. If test is positive, Secretary can order counselling if appropriate. If counselling is unsuccessful, Secretary can impose restrictions on the person's behaviour or movements. If restriction is insufficient, Secretary can order detention.</td>
</tr>
<tr>
<td>Appeals</td>
<td>s 122</td>
<td>Health Act 1958</td>
<td>Person subject to an order under s 121 may appeal to the Supreme Court.</td>
</tr>
<tr>
<td>Emergency powers</td>
<td>s 123</td>
<td>Health Act 1958</td>
<td>Governor in Council can declare a state of public health emergency for the purpose of stopping, limiting or preventing the spread of an infectious disease.</td>
</tr>
<tr>
<td></td>
<td>s 124</td>
<td>Health Act 1958</td>
<td>If the Governor so declares, the Secretary can make orders requiring that persons of a specified class be prevented from leaving or entering a prescribed area, or that they be arrested without warrant and detained in the proclaimed area. Can also order any building or land to be seized, disinfected or destroyed if it is contributing to the spread of infection.</td>
</tr>
<tr>
<td>Testing for HIV</td>
<td>s 127</td>
<td>Health Act 1958</td>
<td>Doctor must not test for HIV unless he or she is satisfied that the person has received sufficient information on the medical and social consequences of a positive diagnosis. The person must not be advised of a positive test except in person by a doctor.</td>
</tr>
<tr>
<td>Privacy</td>
<td>s 128</td>
<td>Health Act 1958</td>
<td>Any person who discovers that another has been tested for or has tested positive for HIV must take all steps to develop and implement systems to protect the privacy of that person. A penalty exists for breach of this section.</td>
</tr>
<tr>
<td>Section</td>
<td>Act</td>
<td>Prescribed Categories</td>
<td>Penalty</td>
</tr>
<tr>
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</tr>
</tbody>
</table>
| s 130  | Health Act 1958 | i. homosexual male contact  
ii. coagulation factor recipient  
iii. injecting drug user  
iv. transfusion recipient  
v. heterosexual contact  
vi. occupational contact  
vii. screening recipient. | Pathology laboratories must keep records of the number of HIV tests, the number of people tested who fall into each prescribed category and any other information required by the regulations. |
| s 136  | Health Act 1958 | | A donor must not make a false statement regarding HIV/AIDS infection when donating blood or tissue. A penalty exists for breach of this section. |
| r 15  | Health (Infectious Diseases) Regulations 2001 | If the Secretary believes that an outbreak of infectious disease has occurred or may occur, the Secretary may:  
- enter any premises without a warrant and search for and seize any goods  
- in writing require any person who may have been in contact with a person with the infection to provide information about the contact  
- in the case of a premises where the disease may be spread:  
  - inspect the premises  
  - direct the proprietor to disinfect the premises and dispose of anything  
  - close a school  
  - give reasonable directions to a person to take any action that he or she considers necessary to prevent or limit the spread of the infectious disease. |
| s 29  | Corrections Act 1986 | Principal medical officer of a prison can direct a prisoner to submit to medical tests.  
In making decision medical officer is to consider the safety of everyone in the prison. |
| s 120  | Health Act 1958 | It is an offence to knowingly or recklessly transmit an infectious disease to another person.  
It is a defence if the person placed at risk knew of the condition and voluntarily accepted the risk. |
| r 28  | Health (Infectious Diseases) Regulations 2001 | A proprietor must ensure that condoms are used in a brothel. |
| r 29  | Health (Infectious Diseases) Regulations 2001 | Proprietor must not force a sex worker to provide service if he or she has refused on the basis that he or she suspects the client has an infectious disease or because the client has refused to wear a condom. |
| s 19  | Prostitution Control Act 1994 | The manager of a brothel must not allow a sex worker to provide services when the sex worker has an STI. |
| s 20  | Prostitution Control Act 1994 | A sex worker must not provide services if he or she has an STI. |
| s 6  | Equal Opportunity Act 1995 | Prohibits discrimination on the basis of ‘impairment’ which includes the presence in the body of organisms that may cause disease.
<table>
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<tr>
<th>Subject</th>
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<tbody>
<tr>
<td>Notifiable disease</td>
<td>Table 1</td>
<td>Notifiable Diseases Guidelines</td>
<td>HIV, AIDS and viral hepatitis are notifiable diseases.</td>
</tr>
<tr>
<td>Notification</td>
<td>s 48</td>
<td>Public Health Act 1997</td>
<td>The Director may require any person or class of person, agency or public authority to notify the Director of the presence of any notifiable disease in a sample of tissue, substance or secretion.</td>
</tr>
<tr>
<td>Information from doctor</td>
<td>s 50</td>
<td>Public Health Act 1997</td>
<td>If a doctor believes that a person he or she is attending has a notifiable disease, he or she must provide that person with any information about the transmission and prevention of the disease, and other matters as prescribed including any relevant counseling.</td>
</tr>
<tr>
<td>Order for examination</td>
<td>s 41</td>
<td>Public Health Act 1997</td>
<td>The Director may require that a person he or she believes to have a notifiable disease undergo a medical examination.</td>
</tr>
<tr>
<td>Directions</td>
<td>s 42</td>
<td>Public Health Act 1997</td>
<td>Director may make the following directions to someone who has or is suspected to have a notifiable disease:</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>• That the person be placed in isolation</td>
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<td>• That the person be placed in quarantine</td>
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<td>• That the person be placed under supervision</td>
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<tr>
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<td>• Further medical examination be conducted</td>
</tr>
<tr>
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<td>• That the person provide the name and address of any person she or he might have transmitted the disease to</td>
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<td></td>
<td>• Preventing the person from performing specified work</td>
</tr>
<tr>
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<td></td>
<td>• That the person do or not do anything as the Director determines</td>
</tr>
<tr>
<td>Warrant</td>
<td>s 43</td>
<td>Public Health Act 1997</td>
<td>Director may apply to a magistrate for a warrant to detain any person who has not complied with an order made under s 42.</td>
</tr>
<tr>
<td>Order by magistrate</td>
<td>s 46</td>
<td>Public Health Act 1997</td>
<td>A person arrested under a s 43 warrant must be brought in front of a magistrate as soon as practicable.</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>The magistrate may order the person to comply with the Director’s order and may also vary, add to or make any other order.</td>
</tr>
<tr>
<td>Appeal to Supreme Court</td>
<td>s 47</td>
<td>Public Health Act 1997</td>
<td>Any person subject to an order under s 45 may appeal that order to the Supreme Court.</td>
</tr>
<tr>
<td>Period of detention</td>
<td>s 44</td>
<td>Public Health Act 1997</td>
<td>Detention is not to exceed 48 hours (if for the purposes of medical examination) or 24 hours (detention for any other purpose). A magistrate may approve detention for a period of up to 6 months. Further detention must be with permission of the Supreme Court.</td>
</tr>
<tr>
<td>Report to Council and third parties</td>
<td>s 49</td>
<td>Public Health Act 1997</td>
<td>Director must provide a report to a council on the occurrence of any notifiable disease in its area. Director may also inform any other party who may be directly affected by any occurrence of a notifiable disease.</td>
</tr>
<tr>
<td>Transmission</td>
<td>s 51</td>
<td>Public Health Act 1997</td>
<td>A person who is aware of having a notifiable disease must take all reasonable measures and precautions not to transmit it to any other person and must not knowingly or recklessly place another person at risk unless that other person knew of, and voluntarily accepted the risk of contracting the disease. Maximum penalty: fine of 100 penalty units or imprisonment for up to 12 months.</td>
</tr>
<tr>
<td>Investigation</td>
<td>s 52</td>
<td>Public Health Act 1997</td>
<td>The Director may carry out any investigation or inquiry into any occurrence of any notifiable disease.</td>
</tr>
</tbody>
</table>
Preventing spread  s 53  Public Health Act 1997  The Director may require any person to take any action to stop the spread of any notifiable disease.

Correctional settings  s 30  Corrections Act 1997  Prison director may require a prisoner to undergo a test for HIV or other blood-borne disease, and in the event of refusal, undergo counseling as to the necessity for testing.

**Offences**

Intentionally or recklessly placing another at risk of becoming infected with HIV  s 20  HIV/AIDS Preventive Measures Act 1993  A person who is aware of being infected with HIV must take all reasonable measures to prevent the transmission of HIV and must inform in advance any sexual contact or person with whom needles are shared of that fact. Further a person infected with HIV must not knowingly or recklessly place another person at risk of infection unless that person was aware of the person's HIV-positive status and voluntarily accepted the risk of infection.

Where a medical practitioner responsible for the treatment of a person with HIV is aware that they are not compliant with these obligations, and has not requested contact tracing, the medical practitioner may, after approval from an approved specialist medical practitioner, inform the contacts of the person.

Non-discrimination  s 16  Anti-discrimination Act 1998  Must not discriminate on the basis of disability which includes ‘the presence in the body of organisms causing or capable of causing disease or illness’ (s 3).

**Victoria**

<table>
<thead>
<tr>
<th>Subject</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Notifiable condition</td>
<td>Schedule 4</td>
<td>Public Health and Wellbeing Regulations 2009</td>
<td>HIV and AIDS are group D notifiable conditions. Hepatitis A is a group A condition, while Hepatitis B – E are group B conditions.</td>
</tr>
<tr>
<td>Infectious disease</td>
<td>s3</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>Not specifically defined. Includes a human illness or condition due to a specific infectious agent.</td>
</tr>
<tr>
<td>Order for test when spread by or to a healthcare worker or police officer</td>
<td>s134</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>If the Chief Health Officer (CHO) believes that an incident has occurred in which: an specified infectious disease (including HIV and hepatitis) could have been transmitted to, or by a healthcare worker, police officer or other prescribed person; while acting in that capacity; and any of those people to whom the disease could have been transmitted have been counseled about the risk of transmission of the disease and the medical and social consequences of being infected, and counseling has offered to the person who may have transmitted the disease or does not have the capacity to consent to testing; the CHO may make an Order requiring the person named in the Order to be tested for the disease.</td>
</tr>
<tr>
<td></td>
<td>s137</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>The powers of the CHO under s134 can also be exercised by authorized senior medical officers at certain hospitals and multi-purpose services.</td>
</tr>
<tr>
<td></td>
<td>s135</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>If the circumstances in s134 apply and a specimen of the person's blood is available, that may be tested rather than making an order to have the person tested.</td>
</tr>
<tr>
<td></td>
<td>s140</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>When advising a person that he or she needs to be tested, the Secretary or authorised officer must not divulge the name of the person from whom the disease originated.</td>
</tr>
<tr>
<td>Topic</td>
<td>Section</td>
<td>Act</td>
<td>Description</td>
</tr>
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<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Examination and testing</td>
<td>s113</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>The CHO may make an examination and testing order if of the belief that: a person has an infectious disease or has been exposed and is likely to contract that disease; and is likely to transmit that disease; and the disease, the behavior of the person or both is likely to constitute a serious risk to public health. If a person fails to undergo the required examination, they can be detained for a period of not more than 72 hours to undergo the examination.</td>
</tr>
<tr>
<td>Notification</td>
<td>ss127 - 129</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>If a registered medical practitioners (s127) has a reasonable belief that a person has, or has died from a notifiable condition, they must notify the Secretary in accordance with the regulations. Pathology services (s128) must similarly notify the Secretary of any test results of persons resident in Victoria that indicate that a person has, or may have a notifiable condition. Health and Pathology Services (s129) must implement processes to ensure such notifications are made.</td>
</tr>
<tr>
<td>Pre Test Counseling</td>
<td>s131, r77</td>
<td>Public Health and Wellbeing Act 2008 and Public Health and Wellbeing Regulations 2009</td>
<td>A registered medical practitioner must not carry out or authorize an HIV test unless satisfied that the person has been given information about the medical and psychosocial consequences of the test and the meaning of possible results of the test.</td>
</tr>
<tr>
<td>Post Test Counseling</td>
<td>s132, r78</td>
<td>Public Health and Wellbeing Act 2008 and Public Health and Wellbeing Regulations 2009</td>
<td>Only a prescribed class of persons (the provider of test results) may provide a person with the results of a positive HIV test. In so doing, the provider of test results must counsel the person in person as to the medical and psychosocial consequences of the test, and ways to prevent transmission of HIV.</td>
</tr>
<tr>
<td>Closure of Court or Tribunal</td>
<td>s133, r79A</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>If evidence is proposed to be given in a court or tribunal regarding HIV or Hepatitis C, the court may close the session or prohibit publication for reason of the social or economic consequences on the person concerned.</td>
</tr>
<tr>
<td>Behavioral orders</td>
<td>s117</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>The CHO may make a public health order if of the belief that: a person has an infectious disease or has been exposed and is likely to contract that disease; and is likely to transmit that disease; and the disease, the behavior of the person or both is likely to constitute a serious risk to public health; and the person needs to take, or refrain from taking certain action to prevent that risk; and a reasonable attempt has been made to counsel the person; and it is necessary to make the order. If so satisfied, the CHO may make an order for a maximum period of 6 months that the person: participate in counseling; undergo psychiatric assessment; refrain from certain behaviour; reside at a particular, or notify change in residence; submit to supervision; receive specified prophylaxis or treatment; be detained.</td>
</tr>
<tr>
<td>Review of public health order</td>
<td>s121 - 122</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>A person subject to an order under s 117 may seek a review by either the CHO or the VCAT at any time.</td>
</tr>
<tr>
<td>Privacy</td>
<td>Schedule 1, Principle 2</td>
<td>Health Records Act 2001</td>
<td>Health Information must be treated in accordance with the principles outlined in the Health Records Act. Importantly, information can only be used for the purpose for which it was collected (the primary purpose). The person’s consent is needed before the information can be used for a secondary purpose, unless that secondary purpose is directly related to the primary purpose and the person would reasonably expect the information to be used in that way.</td>
</tr>
<tr>
<td>Blood and tissue donation</td>
<td>s155</td>
<td>Public Health and Wellbeing Act 2008</td>
<td>A donor must not make a false statement when donating blood or tissue. Maximum penalty of 120 penalty units or imprisonment of 1 year.</td>
</tr>
</tbody>
</table>
### Correctional settings

**Corrections Act 1986**
Principal medical officer of a prison can direct a prisoner to submit to medical tests.
In making decision medical officer is to consider the safety of everyone in the prison.

### Offences

**Public Health and Wellbeing Act 2008**
It is a principle of the Act that a person infected with an infectious disease must take all reasonable steps to ensure another person does not contract the disease. It is also a principle of the Act that persons who do not suffer from an infectious disease should take all reasonable precautions to prevent contracting an infectious disease.

### Sex Work

**Public Health and Wellbeing Act 2008**
Brothel proprietors must provide a free supply of condoms and lubricant, and must take reasonable steps to ensure that clients and sex workers use condoms in any encounter that involves penetration. (ss158, 159)
Brothel and escort agency proprietors must not require a sex worker to provide services to a client that the sex worker suspects is infected with an infectious disease, or who refuses to use a condom. (s160)

**Sex Work Act 1994**
Sex workers and clients must take all reasonable steps to minimize the risk of acquiring or transmitting an STI, including the use of a condom or appropriate barrier in the event of oral, vaginal or anal penetration.

**Sex Work Act 1994**
A provider of sex work services must not permit a sex worker to work during a period when the sex worker is infected with an STI. A provider of sex work services is presumed to have known that a sex worker was infected with an STI, unless the person believed on reasonable grounds that the sex worker had been undergoing tests on at least a quarterly basis for STIs, and was not infected with an STI.

### Non-discrimination

**Equal Opportunity Act 1995**
Prohibits discrimination on the basis of ‘impairment’ which includes ‘the presence in the body of organisms that may cause disease’.

### Criminal Offences

**Crimes Act 1958**
It is an offence to intentionally (16) or recklessly (17) cause serious injury. It is also an offence to engage in conduct that places or may place another person in danger of serious injury (23).
It is also an offence to intentionally cause another person to be infected with HIV (19A).
Courts have held serious injury to include HIV

### Western Australia

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<tbody>
<tr>
<td>Notification by doctor</td>
<td>s 276</td>
<td>Health Act 1911</td>
<td>Obligation of notification to the Executive Director, Public Health rests with clinician, nurse practitioner and responsible pathologist or pathology laboratory.</td>
</tr>
<tr>
<td></td>
<td>s 289</td>
<td>Health Act 1911</td>
<td>Doctor, nurse practitioner and responsible pathologist of pathology laboratory who notify of an infectious disease incur no civil liability and are taken not to have breached duty of confidentiality.</td>
</tr>
<tr>
<td>Notification by employer</td>
<td>r 2.5</td>
<td>Occupational Health and Safety Regulations 1996</td>
<td>Employer must notify the Commissioner if a person contracts HIV or viral hepatitis in the course of work which involves exposure to human blood products, body secretions, excretions or other material which may be a source of infection.</td>
</tr>
<tr>
<td>Employment</td>
<td>Section</td>
<td>Act</td>
<td>Description</td>
</tr>
<tr>
<td>------------------------------------------------</td>
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<td>-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Employment - food handling</td>
<td>s 246X</td>
<td>Health Act 1911</td>
<td>A person with an infectious disease commits an offence if he or she is engaged or employed in the handling and packaging of food. A medical officer can require a person engaged in food handling to submit to a test for an infectious disease.</td>
</tr>
<tr>
<td>Employment - apparel</td>
<td>s 279</td>
<td>Health Act 1911</td>
<td>An owner or occupier of a factory, workshop or place from which work is given commits an offence if he or she allows a person with an infectious disease to make wearing apparel on the premises unless he or she could not reasonably be aware that the person had an infectious disease.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Infectious diseases</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV/AIDS is a dangerous infectious disease</td>
<td>Schedule 1</td>
<td>Health (Dangerous Infectious Diseases) Notice 2000</td>
<td>HIV and AIDS are each declared to be dangerous infectious diseases.</td>
</tr>
<tr>
<td>Infectious diseases - removal to hospital</td>
<td>s 263</td>
<td>Health Act 1911</td>
<td>Medical Officer of Health may order any person suffering an infectious disease removed to hospital for treatment in any case where it is considered in the interest of public health to do so.</td>
</tr>
<tr>
<td>Infectious diseases - exposure</td>
<td>s 264</td>
<td>Health Act 1911</td>
<td>A person suffering an infectious disease commits an offence if he or she exposes him- or herself in a public place (or public vehicle) without precaution as to transmitting infection to others.</td>
</tr>
<tr>
<td>Non-discrimination</td>
<td>s 66A</td>
<td>Equal Opportunity Act 1984</td>
<td>Prohibits discrimination on the basis of ‘impairment’ which includes ‘any defect or disturbance in the normal structure or functioning of a person’s body’ (s 4).</td>
</tr>
<tr>
<td>Sexually transmitted disease</td>
<td>s 248</td>
<td>Health Act 1911</td>
<td>The Governor may declare any infectious disease to be a dangerous infectious disease for the purposes of the Act. Sexually transmitted disease is not an infectious disease for the purposes of the Act.</td>
</tr>
<tr>
<td>Giving blood</td>
<td>Schedule 1</td>
<td>Blood and Tissue (Transmissible Diseases) Regulations</td>
<td>Person giving blood must provide declaration that blood is free from HBV, HCV and HIV. All blood taken for transfusion is tested for HBV, HCV and HIV and if it tests positive, the person will be notified.</td>
</tr>
<tr>
<td>Sex work</td>
<td>s 8</td>
<td>Prostitution Act 2000</td>
<td>Condom must be used to prevent the transmission of bodily fluid from one person to another.</td>
</tr>
<tr>
<td>Correctional settings</td>
<td>s 95D</td>
<td>Prisons Act 1981</td>
<td>Medical officer can force a prisoner to undergo any medical treatment or testing deemed necessary.</td>
</tr>
<tr>
<td>Offences</td>
<td>s 294(8) or 297 as read with s 1(4)</td>
<td>Criminal Code</td>
<td>Sections 294 (8) and 297 make it an offence to do any act that is likely to result in a person having a serious disease or to cause grievous bodily harm to another. S.1(4) provides that any reference to causing or doing bodily harm to a person includes a reference to causing a person to have a disease which interferes with health or comfort or to have a serious disease.</td>
</tr>
<tr>
<td>Non-discrimination</td>
<td>s 66A</td>
<td>Equal Opportunity Act 1984</td>
<td>Prohibits discrimination on the basis of ‘impairment’ which includes ‘any defect or disturbance in the normal structure or functioning of a person’s body’ (s 4).</td>
</tr>
</tbody>
</table>
Guardianship and enduring powers of attorney

If there is a possibility that a patient may become incompetent, health-care practitioners should advise their patients to consider options that might include enduring powers of attorney or enduring powers of guardianship. The laws that apply to substituted decision making can be quite complex and vary between states. Patients should be referred to the relevant authorities in their jurisdiction for assistance.

<table>
<thead>
<tr>
<th>Name</th>
<th>State</th>
<th>Street address</th>
<th>Postal address</th>
<th>Phone numbers</th>
<th>Email address</th>
</tr>
</thead>
<tbody>
<tr>
<td>Public Advocate of the ACT</td>
<td>ACT</td>
<td>Level 3 12 Moore St, Canberra City 2601</td>
<td>PO Box 1001 Civic Square ACT 2608</td>
<td>Tel: (02) 6207 0707 Fax: (02) 6207 0688</td>
<td><a href="mailto:pa@act.gov.au">pa@act.gov.au</a></td>
</tr>
<tr>
<td>Guardianship and Management of Property Tribunal</td>
<td>ACT</td>
<td>Magistrates Court, 4 Knowles Place, Canberra ACT 2601</td>
<td>GPO Box 370 CANBERRA CITY ACT 2601</td>
<td>Ph: (02) 6217 4281 Fax: (02) 6217 4505</td>
<td><a href="mailto:tribunals@act.gov.au">tribunals@act.gov.au</a></td>
</tr>
<tr>
<td>Office of the Public Guardian</td>
<td>NT</td>
<td>2nd Floor, Casuarina Plaza Darwin</td>
<td></td>
<td>Ph: (08) 8922 7343 Ph: (08) 8922 7304</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Darwin Public Guardian Alice Springs Public Guardian</td>
<td></td>
<td>Ph: (08) 8922 7116 Ph: (08) 8951 6739</td>
<td></td>
</tr>
<tr>
<td>Guardianship Tribunal</td>
<td>NSW</td>
<td>Level 3 2a Rowntree Street Balmain NSW 2041</td>
<td>Guardianship Tribunal Locked Bag 9 Balmain NSW 2041</td>
<td>Toll free 1800 463 928 Main switch: (02) 9555 8500 Telephone typewriter: (02) 9552 8534 Fax: (02) 9555-9049</td>
<td><a href="mailto:gt@gt.nsw.gov.au">gt@gt.nsw.gov.au</a></td>
</tr>
<tr>
<td>Office of the Public Guardian</td>
<td>NSW</td>
<td>Sydney office: Level 15, 133 Castlereagh St Sydney NSW 2000</td>
<td>Sydney office: PO Box A231 Sydney South NSW 1235 DX 1335 Sydney</td>
<td>Ph: (02) 9265 3184 Fax: (02) 9283 2645</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Blacktown office: Level 2D, 15-17 Kildare Rd Blacktown NSW 2148</td>
<td>Blacktown office: PO Box 168 Blacktown NSW 2148 DX 8132 Blacktown</td>
<td>Ph: (02) 9671 9800 Fax: (02) 9671 9804</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gosford office: Suite 3, 40 Marn St Gosford NSW 2350</td>
<td>Gosford office: PO Box 487 Gosford NSW 2350 DX 7229 Gosford</td>
<td>Ph: (02) 4320 4888 Fax: (02) 4320 4818</td>
<td></td>
</tr>
<tr>
<td>The Office of the Adult Guardian</td>
<td>Qld</td>
<td>Level 3 Brisbane Magistrates Courts Complex 363 George Street Brisbane Qld 4000</td>
<td>PO Box 13554 George Street Brisbane Qld 4003</td>
<td>Ph: (07) 3234 0870 Outside Brisbane 1300 653 187 Fax: (07) 3239 6367</td>
<td><a href="mailto:adult.guardian@justice.qld.gov.au">adult.guardian@justice.qld.gov.au</a></td>
</tr>
<tr>
<td>Guardianship and Administration Tribunal</td>
<td>Qld</td>
<td>Level 9 259 Queen Street Brisbane Qld 4000</td>
<td>GPO Box 1639 Brisbane Qld 4001</td>
<td>Ph: (07) 3234 0666 Outside Brisbane: 1300 780 666 Fax: (07) 3221 9156</td>
<td><a href="mailto:guardianship@justice.qld.gov.au">guardianship@justice.qld.gov.au</a></td>
</tr>
<tr>
<td>Office of the Public Advocate</td>
<td>SA</td>
<td>Level 7, ABC Building 85 North East Road Collingwood 5082</td>
<td>Level 7, ABC Building 85 North East Road Collingwood SA 5081</td>
<td>Ph: (08) 8269 7575 Toll Free Number (for country South Australia only) 1800 066 969 Fax: (08) 8269 7490</td>
<td><a href="mailto:OPA@agd.sa.gov.au">OPA@agd.sa.gov.au</a></td>
</tr>
<tr>
<td>Guardianship Board</td>
<td>SA</td>
<td>Level 8, ABC Building 85 North East Road Collinswood 5082</td>
<td>Guardianship Board PO Box 138 Prospect SA 5082</td>
<td>Toll Free Number (for country South Australia Only) 1800 800 501 Ph: (08) 8368 5600 Fax: (08) 8368 5699</td>
<td><a href="mailto:guardianshipboard@agd.sa.gov.au">guardianshipboard@agd.sa.gov.au</a></td>
</tr>
<tr>
<td>---</td>
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</tr>
<tr>
<td>Office of the Public Guardian</td>
<td>Tas.</td>
<td>Level 3, 15 Murray Street, Hobart Tas 7000</td>
<td>GPO Box 825 Hobart Tas 7001</td>
<td>Ph: (03) 6233 7608 Fax: (03) 6233 4882</td>
<td><a href="mailto:public.guardian@info.tas.gov.au">public.guardian@info.tas.gov.au</a></td>
</tr>
<tr>
<td>Guardianship and Administration Board</td>
<td>Tas.</td>
<td>First Floor 54 Victoria Street Hobart 7000</td>
<td>The Registrar Guardianship and Administration Board GPO Box 1307 Hobart Tas 7001</td>
<td>Ph: (03) 6233 3085 Fax: (03) 6233 4509 After hours emergency service: (03) 6233 3085</td>
<td><a href="mailto:guardianship@justice.tas.gov.au">guardianship@justice.tas.gov.au</a></td>
</tr>
<tr>
<td>Victorian Civil and Administrative Tribunal</td>
<td>Vic.</td>
<td>55 King Street, Melbourne Vic 3000 DX 210576</td>
<td></td>
<td>Guardianship List: Tel: (03) 9628 9911 or 1800 133 055 Fax: (03) 9628 9932</td>
<td><a href="mailto:vcat@vcat.vic.gov.au">vcat@vcat.vic.gov.au</a></td>
</tr>
<tr>
<td>Office of the Public Advocate</td>
<td>Vic.</td>
<td>5th Floor 436 Lonsdale Street Melbourne Vic 3000</td>
<td></td>
<td>Tel 1300 309 337 Fax (03) 9603 9501 TTY (03) 9603 9529 ACE 136677 (03) 9603 9500</td>
<td><a href="mailto:publicadvocate@justice.vic.gov.au">publicadvocate@justice.vic.gov.au</a></td>
</tr>
<tr>
<td>Office of the Public Advocate</td>
<td>WA</td>
<td>Level 1 30 Terrace Road East Perth WA 6004</td>
<td></td>
<td>Tel: (08) 9278 7300 Country Freecall: 1800 807 437 Fax: (08) 9278 7333</td>
<td></td>
</tr>
<tr>
<td>State Administrative Tribunal.</td>
<td>WA</td>
<td>Level 4 12 St Georges Terrace Perth WA 6000</td>
<td></td>
<td>Tel: (08) 9219 3111 or 1300 306 017 (for regional STD callers) Fax: (08) 9325 5099</td>
<td></td>
</tr>
</tbody>
</table>
## Glossary

<p>| Ab | antibody |
| AChSHM | Australasian Chapter of Sexual Health Medicine of the RACP |
| ACRRM | Australian College of Rural and Remote Medicine |
| AFAO | Australian Federation of AIDS Organisations |
| Ag | antigen |
| AHC | Australian Hepatitis Council |
| AIDS | Acquired immune deficiency syndrome |
| AVL | Australian Injecting and Illicit Drug Users League |
| ALA | Australian Liver Association |
| ALT | alanine aminotransferase or alanine transaminase |
| ANA | antinuclear antibody |
| ANCAHRD | Australian National Council on AIDS, Hepatitis C and Related Diseases (obsolete; now replaced by MACASHH) |
| Anilingus | oro-anal sex |
| Anoscopy | inspection of anal canal and rectal lining through a (usually) disposable instrument called an anoscope (also called proctoscopy) |
| Anti-HAV IgM | antibody to HAV IgM - signifies recent exposure to HAV |
| Anti-HAV IgG | antibody to HAV IgG - signifies past exposure to HAV or successful vaccination |
| Anti-hBc IgM | antibody to hepatitis B core antigen - signifies recent exposure to HBV |
| Anti-hBc IgG | antibody to hepatitis B core antigen - signifies past exposure to HBV |
| Anti-hBe | antibody to hepatitis Be antigen |
| Anti-hBs | antibody to hepatitis B surface antigen - associated with non-replicative phase or successful vaccination |
| Anti-HCV | antibody for HCV - indicates infection with HCV has occurred |
| Anti-HDV IgG and IgM | antibody to the hepatitis D virus |
| APRI | AST-to-platelet ratio index |
| APTT | activated partial thromboplastin time |
| ART | antiretroviral therapy |
| ASHM | Australasian Society for HIV Medicine |
| ASMA | anti-smooth muscle antibody |
| AST | aspartate aminotransferase |
| AZT | azidothymidine, also called zidovudine |
| B-cell | a type of immune cell |
| Balanitis | inflammation of the glans penis |
| Balanoposthitis | inflammation of the glans penis and the prepuce (foreskin) |
| BBV | blood-borne virus |
| BCG | Bacille Calmette-Guerin (tuberculosis vaccine) |
| bd, bid | twice daily |
| b-DNA | branched deoxyribonucleic acid |
| Beats | public toilets, parks and other outdoor venues where MSM ‘beat a path’ looking for sexual partners |
| BV | bacterial vaginosis, a common complex syndrome resulting in a change in the vaginal ecosystem with raised vaginal pH; often asymptomatic but sometimes associated with an abnormal vaginal discharge |
| CAH | chronic active hepatitis |
| CALD | culturally and linguistically diverse |
| cART | combination antiretroviral therapy |
| C &amp; S | culture and sensitivity |
| CCR5 | chemokine co-receptor on the surface of cells which may be used in HIV-cell fusion |
| CD4 cell | a helper T-cell which carries the CD4 surface antigen. CD4 cells are the primary target of HIV and CD4 cell numbers decline during HIV disease |
| CD8 cell | a killer or cytotoxic T-cell which carries the CD8 surface antigen |
| Chancre | the painless ulcer of primary syphilis |
| Chancroid | a tropical STI caused by Haemophilus ducreyi, virtually never seen in Australia or New Zealand |
| CHAP smear | ano-rectal cytology for HPV in MSM |
| Chlamydia | a sexually transmitted infection caused by Chlamydia trachomatis |</p>
<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>CIN</td>
<td>cervical intraepithelial neoplasia</td>
</tr>
<tr>
<td>Circumcision</td>
<td>removal of the prepuce (foreskin)</td>
</tr>
<tr>
<td>Clue cells</td>
<td>vaginal epithelial cells with bacteria adhering to the surface and partially obscuring the borders, characteristic of bacterial vaginosis microscopically</td>
</tr>
<tr>
<td>CMV</td>
<td>cytomegalovirus</td>
</tr>
<tr>
<td>CNC</td>
<td>clinical nurse consultant</td>
</tr>
<tr>
<td>Condylomata acuminata</td>
<td>genital warts</td>
</tr>
<tr>
<td>Condylomata lata</td>
<td>moist warty growths occurring in perineum in secondary syphilis</td>
</tr>
<tr>
<td>Contact tracing</td>
<td>the following-up, diagnosis and (where possible) treatment of all sexual partners of a patient infected with an STI. Also called 'partner notification'</td>
</tr>
<tr>
<td>Crabs</td>
<td>see pubic lice</td>
</tr>
<tr>
<td>CRP</td>
<td>C-reactive protein</td>
</tr>
<tr>
<td>CT</td>
<td>computed tomography</td>
</tr>
<tr>
<td>Cunnilingus</td>
<td>oral sex - mouth to vulva</td>
</tr>
<tr>
<td>DAA</td>
<td>direct acting antiviral</td>
</tr>
<tr>
<td>DGI</td>
<td>disseminated gonococcal infection</td>
</tr>
<tr>
<td>DILI</td>
<td>drug-induced liver injury</td>
</tr>
<tr>
<td>Dipping</td>
<td>vaginal or anal sex without a condom for varying periods of time prior to ejaculation, i.e. the condom is only applied when the insertive partner is getting near ejaculation</td>
</tr>
<tr>
<td>DNA</td>
<td>deoxyribonucleic acid</td>
</tr>
<tr>
<td>cccDNA</td>
<td>covalently closed circular DNA</td>
</tr>
<tr>
<td>DoHHA</td>
<td>Commonwealth Department of Health and Ageing</td>
</tr>
<tr>
<td>Donovanosis</td>
<td>a rare STI of great chronicity causing considerable destruction of genital structures if untreated. Seen only in remote Indigenous communities in Australia</td>
</tr>
<tr>
<td>DRE</td>
<td>digital rectal examination</td>
</tr>
<tr>
<td>DRESS</td>
<td>drug rash with eosinophilia and systemic symptoms</td>
</tr>
<tr>
<td>EBV</td>
<td>Epstein-Barr virus</td>
</tr>
<tr>
<td>EIA</td>
<td>enzyme immunoassay; an immunoassay in which an enzyme, such as a peroxidase, is used as a marker to indicate the presence of specific antigens or antibodies (as in treponemal EIA, a specific serological test for syphilis)</td>
</tr>
<tr>
<td>ELISA</td>
<td>enzyme linked immunosorbent assay</td>
</tr>
<tr>
<td>Epididymo-orchitis</td>
<td>inflammation of epididymis primarily, spreading secondarily to testis</td>
</tr>
<tr>
<td>FBC</td>
<td>full blood count</td>
</tr>
<tr>
<td>FPU</td>
<td>first passed specimen of urine</td>
</tr>
<tr>
<td>FDA</td>
<td>US Food and Drug Administration</td>
</tr>
<tr>
<td>Fellatio</td>
<td>oral sex – mouth to penis</td>
</tr>
<tr>
<td>Fisting</td>
<td>sexual act where fist and forearm are inserted into vagina or ano-rectum</td>
</tr>
<tr>
<td>Fitz-Hugh-Curtis</td>
<td>transcoelomic spread of pelvic infection with Chlamydia trachomatis or Neisseria gonorrhoeae to the liver surface causing a perihepatitis</td>
</tr>
<tr>
<td>Fomites</td>
<td>materials (e.g. towels, sheets etc) which, once contaminated with a microbiological or virological agent, allow transmission of that infection to another individual</td>
</tr>
<tr>
<td>FTA-ABS</td>
<td>fluorescent treponemal antibody absorbed serology test, a specific serological test for syphilis</td>
</tr>
<tr>
<td>Genital herpes</td>
<td>infection of ano-genital region with sexually transmitted HSV-1 or HSV-2</td>
</tr>
<tr>
<td>Genital warts</td>
<td>exophytic clinical manifestation of sexually transmitted ano-genital HPV infection</td>
</tr>
<tr>
<td>GESA</td>
<td>Gastroenterological Society of Australia</td>
</tr>
<tr>
<td>GGT</td>
<td>gamma glutamyltransferase</td>
</tr>
<tr>
<td>GI</td>
<td>gastrointestinal</td>
</tr>
<tr>
<td>GIT</td>
<td>gastrointestinal tract</td>
</tr>
<tr>
<td>Gonorrhoea</td>
<td>a sexually transmitted infection caused by Neisseria gonorrhoeae</td>
</tr>
<tr>
<td>GP</td>
<td>general practitioner</td>
</tr>
<tr>
<td>gp120</td>
<td>glycoprotein on the surface of HIV which binds to the CD4 receptor</td>
</tr>
<tr>
<td>gp41</td>
<td>glycoprotein on the surface of HIV involved in fusion between HIV and the CD4 cell</td>
</tr>
<tr>
<td>GUD</td>
<td>(ano)-genital ulcerative disease</td>
</tr>
<tr>
<td>HAART</td>
<td>highly active antiretroviral therapy</td>
</tr>
<tr>
<td>HAND</td>
<td>HIV-associated neurocognitive disorder</td>
</tr>
<tr>
<td>HASTI</td>
<td>HIV/AIDS and Sexually Transmissible Infections Subcommittee of the Ministerial Advisory Committee on AIDS, Sexual Health and Hepatitis</td>
</tr>
<tr>
<td>HAV</td>
<td>hepatitis A virus</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Full Form</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>HAVAb</td>
<td>hepatitis A antibody test (IgM or IgG)</td>
</tr>
<tr>
<td>HBcAb</td>
<td>see anti-HBc</td>
</tr>
<tr>
<td>HBcAg</td>
<td>hepatitis B core antigen</td>
</tr>
<tr>
<td>HBeAb</td>
<td>see anti-HBe</td>
</tr>
<tr>
<td>HBeAg</td>
<td>HBV ‘e’ antigen - a marker of viral replication and infectivity</td>
</tr>
<tr>
<td>HBG</td>
<td>hepatitis B immunoglobulin</td>
</tr>
<tr>
<td>HBsAg</td>
<td>hepatitis B surface antigen - a marker of current infection which persists in individuals who become carriers</td>
</tr>
<tr>
<td>HBsAb</td>
<td>see anti-HBs</td>
</tr>
<tr>
<td>HBV</td>
<td>hepatitis B virus</td>
</tr>
<tr>
<td>HCC</td>
<td>hepatocellular carcinoma</td>
</tr>
<tr>
<td>hCG</td>
<td>human chorionic gonadotropin</td>
</tr>
<tr>
<td>HCV</td>
<td>hepatitis C virus</td>
</tr>
<tr>
<td>HDV</td>
<td>hepatitis D virus</td>
</tr>
<tr>
<td>HHV-8</td>
<td>human herpesvirus-8 - associated with Kaposi's sarcoma</td>
</tr>
<tr>
<td>HIV</td>
<td>human immunodeficiency virus</td>
</tr>
<tr>
<td>HPV</td>
<td>human papillomavirus</td>
</tr>
<tr>
<td>HSIL</td>
<td>high grade squamous intraepithelial lesion</td>
</tr>
<tr>
<td>HSV</td>
<td>herpes simplex virus</td>
</tr>
<tr>
<td>HVS</td>
<td>high vaginal swab</td>
</tr>
<tr>
<td>Hydrocoele</td>
<td>scrotal swelling caused by accumulation of fluid around the testis, between the serosal layers of the tunica vaginalis</td>
</tr>
<tr>
<td>IASHC</td>
<td>Indigenous Australians' Sexual Health Subcommittee of the Ministerial Advisory Committee on AIDS, Sexual Health and Hepatitis</td>
</tr>
<tr>
<td>IDU</td>
<td>injecting drug user</td>
</tr>
<tr>
<td>IFN</td>
<td>interferon</td>
</tr>
<tr>
<td>Ig</td>
<td>immunoglobulin</td>
</tr>
<tr>
<td>INR</td>
<td>international normalised ratio - a test of blood clotting</td>
</tr>
<tr>
<td>IRIS</td>
<td>immune reconstitution inflammatory syndrome</td>
</tr>
<tr>
<td>IV</td>
<td>intravenous</td>
</tr>
<tr>
<td>IVD</td>
<td>In vitro diagnostic medical devices</td>
</tr>
<tr>
<td>IU</td>
<td>international unit (measurement)</td>
</tr>
<tr>
<td>kPa</td>
<td>kilopascals</td>
</tr>
<tr>
<td>KS</td>
<td>Kaposi's sarcoma</td>
</tr>
<tr>
<td>Latency</td>
<td>the situation where an infection enters a quiescent asymptomatic phase and is only detectable by appropriate testing</td>
</tr>
<tr>
<td>LCR</td>
<td>ligase chain reaction - a NAAT</td>
</tr>
<tr>
<td>LFT</td>
<td>liver function test</td>
</tr>
<tr>
<td>LGV</td>
<td>lymphogranuloma venereum - a tropical STI caused by Chlamydia trachomatis serovars L1–L3, now becoming endemic among highly sexually active men who have sex with men</td>
</tr>
<tr>
<td>LKM</td>
<td>liver kidney microsomal</td>
</tr>
<tr>
<td>LSIL</td>
<td>low grade squamous intraepithelial lesion</td>
</tr>
<tr>
<td>MAC</td>
<td>Mycobacterium avium complex</td>
</tr>
<tr>
<td>MACASHH</td>
<td>Ministerial Advisory Committee on AIDS, Sexual Health and Hepatitis</td>
</tr>
<tr>
<td>MAI</td>
<td>Mycobacterium avium intracellulare</td>
</tr>
<tr>
<td>MBS</td>
<td>Medicare Benefits Schedule</td>
</tr>
<tr>
<td>µl</td>
<td>microlitre</td>
</tr>
<tr>
<td>ml</td>
<td>millilitre</td>
</tr>
<tr>
<td>mmol</td>
<td>millimole</td>
</tr>
<tr>
<td>MRI</td>
<td>magnetic resonance imaging</td>
</tr>
<tr>
<td>MSM</td>
<td>men who have sex with men - a rather unsatisfactory but widely used term to describe all men who ever have sex (of any type) with another man</td>
</tr>
<tr>
<td>MSSU</td>
<td>midstream specimen of urine</td>
</tr>
<tr>
<td>NAAT</td>
<td>nucleic acid amplification test</td>
</tr>
<tr>
<td>NAPWA</td>
<td>National Association of People Living with AIDS</td>
</tr>
<tr>
<td>NASH</td>
<td>non-alcoholic steatohepatitis</td>
</tr>
<tr>
<td>NATA</td>
<td>National Association of Testing Authorities Australia</td>
</tr>
<tr>
<td>NCHECR</td>
<td>National Centre in HIV Epidemiology and Clinical Research, former name of the Kirby Institute</td>
</tr>
<tr>
<td>Acronym</td>
<td>Definition</td>
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<tr>
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<tr>
<td>NGO</td>
<td>non-governmental organisation</td>
</tr>
<tr>
<td>NHIG</td>
<td>normal human immunoglobulin</td>
</tr>
<tr>
<td>NHMRC</td>
<td>National Health and Medical Research Council</td>
</tr>
<tr>
<td>NNRTI</td>
<td>non-nucleoside reverse transcriptase inhibitor</td>
</tr>
<tr>
<td>NPEP</td>
<td>non-occupational post-exposure prophylaxis</td>
</tr>
<tr>
<td>NRTI</td>
<td>nucleoside / nucleotide reverse transcriptase inhibitor</td>
</tr>
<tr>
<td>NSU</td>
<td>non-specific urethritis - urethritis where exhaustive laboratory testing fails to find a specific cause (a non gonococcal, non chlamydial, non herpetic, non trichomonal etc urethritis)</td>
</tr>
<tr>
<td>OCP</td>
<td>ova, cysts and parasites - looked for on microscopy of faecal specimens</td>
</tr>
<tr>
<td>OI</td>
<td>opportunistic infection</td>
</tr>
<tr>
<td>Oral sex</td>
<td>use of the mouth in sexual activity (i.e. anilingus, cunnilingus or fellatio)</td>
</tr>
<tr>
<td>p24</td>
<td>a core HIV protein, the primary protein detected by the HIV antigen test which is incorporated with the HIV antibody test in Australia</td>
</tr>
<tr>
<td>Partner notification</td>
<td>see contact tracing</td>
</tr>
<tr>
<td>Pathogenicity</td>
<td>the ability of a micro-organism to cause disease in its host</td>
</tr>
<tr>
<td>PBS</td>
<td>Pharmaceutical Benefits Scheme</td>
</tr>
<tr>
<td>PCB</td>
<td>postcoital bleeding</td>
</tr>
<tr>
<td>PCP</td>
<td>Pneumocystis pneumonia, also known as Pneumocystis jiroveci pneumonia</td>
</tr>
<tr>
<td>PCR</td>
<td>polymerase chain reaction, a NAAT</td>
</tr>
<tr>
<td>PCT</td>
<td>porphyria cutanea tarda</td>
</tr>
<tr>
<td>PEP</td>
<td>post-exposure prophylaxis</td>
</tr>
<tr>
<td>pg/ml</td>
<td>picogram per millilitre</td>
</tr>
<tr>
<td>PHI</td>
<td>primary HIV infection</td>
</tr>
<tr>
<td>PI</td>
<td>protease inhibitor</td>
</tr>
<tr>
<td>PID</td>
<td>pelvic inflammatory disease</td>
</tr>
<tr>
<td>Pili</td>
<td>hair like appendages found on the surface of some bacteria (especially Neisseria gonorrhoeae)</td>
</tr>
<tr>
<td>PML</td>
<td>progressive multifocal leukoencephalopathy</td>
</tr>
<tr>
<td>PMTCT</td>
<td>preventing mother-to-child transmission of HIV</td>
</tr>
<tr>
<td>POCT</td>
<td>point of care testing</td>
</tr>
<tr>
<td>PrEP</td>
<td>pre-exposure prophylaxis</td>
</tr>
<tr>
<td>Prepuce</td>
<td>foreskin</td>
</tr>
<tr>
<td>Proctitis</td>
<td>inflammation of rectal mucosa</td>
</tr>
<tr>
<td>Proctoscopy</td>
<td>inspection of the rectal mucosa through a (usually) disposable instrument called a proctoscope (also called anoscopy)</td>
</tr>
<tr>
<td>PT</td>
<td>Pubic lice</td>
</tr>
<tr>
<td>PWID</td>
<td>people who inject drugs</td>
</tr>
<tr>
<td>Qd</td>
<td>once daily</td>
</tr>
<tr>
<td>Qds, qid</td>
<td>four times daily</td>
</tr>
<tr>
<td>RACGP</td>
<td>Royal Australian College of General Practitioners</td>
</tr>
<tr>
<td>RACP</td>
<td>Royal Australasian College of Physicians</td>
</tr>
<tr>
<td>RF</td>
<td>rheumatoid factor</td>
</tr>
<tr>
<td>Rimming</td>
<td>anilingus, oro-anal sex</td>
</tr>
<tr>
<td>RNA</td>
<td>ribonucleic acid</td>
</tr>
<tr>
<td>RPR</td>
<td>rapid plasma reagin test - a non-specific quantitated serological test for syphilis</td>
</tr>
<tr>
<td>RT</td>
<td>reverse transcriptase</td>
</tr>
<tr>
<td>Salpingitis</td>
<td>inflammation of a Fallopian tube - a component of PID</td>
</tr>
<tr>
<td>SBP</td>
<td>spontaneous bacterial peritonitis</td>
</tr>
<tr>
<td>Scabies</td>
<td>skin infestation caused by Sarcoptes scabiei, often sexually transmitted in adults</td>
</tr>
<tr>
<td>Screening</td>
<td>testing for the presence of an asymptomatic condition in an apparently healthy individual</td>
</tr>
<tr>
<td>SDA</td>
<td>strand displacement amplification assay - a NAAT</td>
</tr>
<tr>
<td>Section 100</td>
<td>a section of the Pharmaceutical Benefits Scheme which provides access to highly specialised drugs</td>
</tr>
<tr>
<td>Seroconversion</td>
<td>process whereby a serological test for a given microbiological or virological agent changes from non-reactive to reactive, coinciding with recent infection</td>
</tr>
<tr>
<td>Serology</td>
<td>diagnostic identification of antibodies (usually), sometimes antigens, in serum</td>
</tr>
<tr>
<td>Serovar</td>
<td>group of closely related microorganisms distinguished by a characteristic set of antigens</td>
</tr>
<tr>
<td>Sexually transmitted infection (STI)</td>
<td>any infection which is mainly transmitted from one individual to another by sexual activity (e.g. all the usually accepted STIs such as syphilis, gonorrhoea and genital herpes)</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
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<tr>
<td>-------------------------------------------</td>
<td>------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Sexually transmissible infection (STI)</td>
<td>any infection which is capable of being transmitted from one individual to another by sexual</td>
</tr>
<tr>
<td></td>
<td>activity given suitable circumstances (e.g. enteric infection during oro-anal sexual contact).</td>
</tr>
<tr>
<td></td>
<td>These two terms are used interchangeably in this monograph</td>
</tr>
<tr>
<td>SIL</td>
<td>squamous intraepithelial lesion</td>
</tr>
<tr>
<td>SMA</td>
<td>smooth muscle antibody</td>
</tr>
<tr>
<td>SOPVs</td>
<td>sex on premises venues, such as saunas and sex clubs frequented by MSM</td>
</tr>
<tr>
<td>Spirochaete</td>
<td>family of motile, spiral shaped gram negative bacteria of which Treponema pallidum is the best</td>
</tr>
<tr>
<td></td>
<td>known</td>
</tr>
<tr>
<td>SSRI</td>
<td>selective serotonin re-uptake inhibitors</td>
</tr>
<tr>
<td>STD</td>
<td>sexually transmitted disease - a term now largely replaced by the more accurate term STI</td>
</tr>
<tr>
<td>SBP</td>
<td>spontaneous bacterial peritonitis</td>
</tr>
<tr>
<td>SVR</td>
<td>sustained virological response (negative HCV RNA and normal ALT six months after completion</td>
</tr>
<tr>
<td></td>
<td>of therapy for HCV)</td>
</tr>
<tr>
<td>Syndromic management</td>
<td>the approach of treating STI symptoms and signs based on the organisms most commonly</td>
</tr>
<tr>
<td></td>
<td>responsible for each syndrome (WHO definition)</td>
</tr>
<tr>
<td>Syphilis</td>
<td>a sexually transmitted infection caused by Treponema pallidum</td>
</tr>
<tr>
<td>TasP</td>
<td>antiretroviral treatment as prevention</td>
</tr>
<tr>
<td>T-cell</td>
<td>white blood cell or lymphocyte</td>
</tr>
<tr>
<td>Td, tds, tid</td>
<td>three times daily</td>
</tr>
<tr>
<td>TGA</td>
<td>Therapeutic Goods Administration</td>
</tr>
<tr>
<td>TMA</td>
<td>transcription mediated assay - a NAAT</td>
</tr>
<tr>
<td>TNF</td>
<td>tumour necrosis factor</td>
</tr>
<tr>
<td>TPHA</td>
<td>Treponema pallidum haemagglutination assay - a largely superseded specific serological test for</td>
</tr>
<tr>
<td></td>
<td>syphilis</td>
</tr>
<tr>
<td>TPPA</td>
<td>Treponema pallidum particle agglutination - a specific serological test for syphilis</td>
</tr>
<tr>
<td>Transmission</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- horizontal transmission of an infection from person-to-person in the community</td>
</tr>
<tr>
<td></td>
<td>- vertical transmission of an infection from mother-to-foetus or infant</td>
</tr>
<tr>
<td>Trichomoniasis</td>
<td>a sexually transmitted infection causing vaginitis and urethritis</td>
</tr>
<tr>
<td>U &amp; E</td>
<td>urea and electrolytes</td>
</tr>
<tr>
<td>VCT</td>
<td>voluntary counselling and testing</td>
</tr>
<tr>
<td>VVC</td>
<td>vulvo-vaginal candidiasis</td>
</tr>
<tr>
<td>VZV</td>
<td>varicella zoster virus</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
<tr>
<td>WSW</td>
<td>women who have sex with women - a rather unsatisfactory term used to describe all women</td>
</tr>
<tr>
<td></td>
<td>who ever have sex (of any type) with another woman</td>
</tr>
</tbody>
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ASHM Resources

ASHM produces a range of HIV and viral hepatitis related resources, available in electronic and hard copy format for health-care professionals. Resources may be downloaded directly from the ASHM website at www.ashm.org.au or ordered by contacting the ASHM office on +61 2 8204 0700 or email resources@ashm.org.au

Monographs
HIV Management in Australasia: a guide for clinical care
HIV, viral hepatitis and STIs: a guide for primary care (electronic only)
B Positive. All you wanted to know about hepatitis B: a guide for primary care (electronic only)

Handbooks
Co-infection HIV and Viral Hepatitis: a guide for clinical management (electronic only)
Djiyadi – Can we talk? A resource manual for sexual health workers who work with Aboriginal and Torres Strait Islander youth

DVDs and CDs
C Me, Hear Me. Hepatitis C in our words
B Seen, B Heard. Hepatitis B from our perspective
Clinical Science of HIV Medicine

Booklets
Aboriginal and Torres Strait Islander Health Workers and Blood-borne Viruses
Dental and Orofacial Health and Hepatitis C
Dentists and HIV
General Practitioners and Hepatitis C
General Practitioners and HIV (include online learning module)
Hepatitis B and Primary Care Providers
Nurses and Hepatitis C
Pharmacy and Hepatitis C
Antenatal Testing and Blood-borne Viruses
Correctional Officers and Blood-borne Viruses
Emergency Service Providers and Blood-borne Viruses
Ngarra 2013
Ngarra 2011
Police and Blood-borne Viruses

Clinical Fact Sheets
Decision Making in HBV
Decision Making in HCV
Decision Making in HIV
GP Companion Resource to the 12 questions to ask your doctor if you have been diagnosed with hepatitis B (electronic only)
GP Companion Resource to the 12 questions to ask your doctor if you have been diagnosed with hepatitis C
Hepatitis B Factsheet: For people newly diagnosed
  • This factsheet is available in hard copy in English, Khmer and Burmese
  • This factsheet is available online only in Arabic, Chinese, Dari, Greek, Indonesian, Italian, Korean, Thai, Turkish and Vietnamese
Hepatitis C in brief – patient fact sheet
  • Available online only in English, Arabic, Chinese, Greek, Indonesian, Italian, Khmer, Spanish and Vietnamese
Medicare Benefits Schedule: Items for use in diagnosing and managing hepatitis B infection
New Treatments in Hepatitis C
HIV Patient Fact Sheet
  • Available online only in Arabic, Chinese, Indonesian, Khmer, Maori, Spanish, Thai, Vietnamese

Other (online only)
Partner Notification (contact tracing) eLearning Modules https://lms.ashm.org.au/

To place an order for ASHM resources, please visit: www.ashm.org.au
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