

Exposure and acute viral hepatitis

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Acute hepatitis Epidemiology

In Australia, 300–500 cases of hepatitis A virus (HAV) infection and around 250 cases of newly acquired hepatitis B virus (HBV) infection are reported annually.¹ An estimated 9,700 new cases of hepatitis C virus (HCV) infection occurred in 2005, but only 354 cases of newly acquired HCV were reported because most cases are subclinical and go unnoticed.^{1,2} Acute hepatitis secondary to excessive alcohol consumption is also common. Various forms of chronic liver disease may present clinically as an acute hepatitis. These include autoimmune hepatitis and Wilson's disease, as well as chronic HBV, which may present as a hepatitis flare. Drug-induced hepatitis also should be considered in all cases of sudden liver enzyme elevation.

Outcomes of acute hepatitis

Less than 1% of all cases of viral hepatitis with jaundice develop acute liver failure. Infection with HAV causes acute hepatitis but is not associated with the development of chronic infection. In contrast, infection with HCV and HBV can result in acute and chronic infection (Table 5.1). Infants and children with HBV infection are more likely to develop chronic HBV infection than adults. Early studies of HCV infection suggested that a significant proportion (85%) of people acutely infected develop chronic viraemia, but later studies suggested that rates of chronic infection may be as low as 55%.³ A recent meta-analysis of the natural history of acute hepatitis C suggested that spontaneous clearance rates were around 25%.⁴ Chronic hepatitis secondary to HCV and HBV infection may progress to cirrhosis, liver failure and hepatocellular carcinoma (HCC). Some patients with chronic HCV infection may develop glomerulonephritis, mixed cryoglobulinaemia, or a syndrome of non-deforming arthritis similar in distribution to rheumatoid arthritis. Chronic HBV may also be associated with extrahepatic manifestations.

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 probably under-recognised.
- 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 viral hepatitis. 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.
- Preventive interventions should be offered to persons with clinical acute hepatitis, those recognised to be in at risk populations and those who have been exposed to hepatotropic viruses.

Symptoms and signs of acute hepatitis

The symptoms and signs of acute viral hepatitis are not specific for a particular aetiological agent and are the same for acute hepatitis and chronic viral hepatitis (Chapter 7). They include nausea, vomiting, anorexia, lethargy, jaundice and tender hepatomegaly. Patients who present with a prolonged prodromal illness, including arthralgia and rash, may have immune complex disease associated with HBV infection. Rarely, acute liver failure supervenes. Signs and symptoms of acute liver failure include intractable vomiting, encephalopathy, asterixis (liver flap) and fetor hepaticus.

Incubation periods

The average time from exposure to the development of symptoms varies for the three major hepatotropic 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**Hepatitis A virus**

- Approximately 0.1% of patients with HAV develop acute liver failure. Less than 40% of patients with acute liver failure die or receive a liver transplant.
- Chronic hepatitis does not occur following HAV infection.
- Lifelong immunity occurs after infection.

Hepatitis B virus

- Less than 1% of clinical cases develop acute liver failure. 80–90% of patients with acute liver failure die or receive a liver transplant.
- Less than 5% of adults with acute HBV infection develop chronic hepatitis.
- 90% of infants infected at birth develop chronic hepatitis.
- Those who go on to develop chronic infection are at risk of cirrhosis, hepatocellular carcinoma and liver failure.
- Those with chronic infection have persistent HBsAg and are infectious to others.
- Those who clear infection have lifelong immunity, maintain anti-HBc, and may or may not preserve anti-HBs.

Hepatitis C virus

- Acute liver failure is rare, but may occur in persons with HBV co-infection.
- Approximately 75% of adults with acute HCV infection develop chronic HCV.
- Those who go on to develop chronic infection are at risk of cirrhosis, hepatocellular carcinoma and liver failure.
- 5% of infants born to HCV-infected women develop HCV infection.
- If infection resolves and the virus is cleared, the person is NOT immune and can be re-infected. After resolution of infection, antibodies persist for a variable amount of time (20 years in some cases).

Diagnostic approach

The diagnosis of acute hepatitis relies predominantly on serological testing, although other features are important to consider.

History should include consideration of:

- Symptoms consistent with acute hepatitis
- A review of any symptoms that may suggest an alternative diagnosis (e.g. infectious mononucleosis)
- Epidemiological clues (Table 5.1 and Chapter 2)
- A history of alcohol and drug use (including illicit drugs, over-the-counter medications and complementary therapies)
- Travel history
- Vaccination history
- Family history of liver disease

An awareness of current epidemiological information is useful (such as a current outbreak of HAV).

Examination should specifically include evaluation for fever, icterus, rash, arthritis, tender hepatomegaly, splenomegaly, injection sites, tattoos, piercings and signs

of hepatic encephalopathy (asterixis, fetor hepaticus and altered mental state). A general 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 with the aspartate aminotransferase (AST)/ALT ratio less than one.
- In alcoholic hepatitis, the ALT is generally 2–10 times the upper limit of normal with the AST/ALT ratio greater than 1.5; bilirubin is usually elevated.
- In drug-induced hepatitis, a mixed profile may be seen with raised hepatic (AST and ALT) and cholestatic (alkaline phosphatase and GGT) markers.

- 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 counselling 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 and Tables 5.2–5.4.

If the diagnosis is unclear, the initial serological investigations may be repeated after 1–2 weeks. Serological investigation of Epstein-Barr virus infection and investigation of 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

In the context of acute HAV infection, anti-HAV IgM is invariably present. False negative results are rare.

Acute HBV infection is best detected by testing for HBsAg and anti-HBc IgM. Anti-HBc IgG and anti-HBs appear later in the course of the illness. HBV DNA 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 (there have been virtually no cases of HDV in Australia for 10–20 years).

In acute HCV infection, HCV antibody may be present at the onset of hepatitis or may develop in the following weeks. If it is not present, and HCV is suspected on epidemiological grounds, HCV RNA polymerase chain reaction (PCR) should be performed to detect viraemia directly. HCV antibodies are usually present within three months of 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 and all patients with an ALT greater than 1000 IU/L, or progressive rise in bilirubin (greater than 60 mmol/L) and INR greater than 1.3. The most ominous signs are falling ALT and rising bilirubin and INR as this indicates severe liver injury with significant loss of hepatocytes. These patients may exhibit signs of encephalopathy.

Most drugs should be avoided during acute hepatitis. Analgesics are generally not required and aspirin,

TABLE 5.2 Clues to diagnosis – epidemiological and exposure risks

• Knowledge of current epidemiology, e.g. HAV cluster
• Contact with a case of acute or chronic hepatitis
• Travel to endemic area without vaccination or passive prophylaxis – HAV, HBV, yellow fever
• Travel to endemic areas – HAV, HBV, HEV, dengue fever, leptospirosis etc.
• Unprotected penetrative sex – HBV
• Unprotected oro-anal sex – HAV
• Occupation, e.g. sewerage workers, childcare workers – HAV
• Occupation, e.g. health care workers – HAV, HBV, HCV
• Injecting drug use – HAV, HBV, HCV
• Alcohol consumption
• Family history – HBV, Wilson's disease, alpha1-antitrypsin deficiency
• Country of birth – HAV, HBV
• Tattoos and/or body piercings – HBV, HCV
• Blood transfusion and medical/dental procedures – HBV, HCV
• Needle-stick injury or other significant occupational exposure – HBV, HCV
• History of imprisonment – HCV

narcotics and sedatives should be avoided. Small amounts 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.

Specific therapy

There is little role for specific agents in the management of acute viral hepatitis A and B. However, in prolonged cholestasis after HAV infection, corticosteroids may reduce serum bilirubin and relieve itch. In the case of acute HBV, infection will resolve spontaneously in the majority of adults and antiviral therapy is not usually indicated. The role of nucleoside analogues in the treatment of acute HBV is not established. However, recent guidelines do suggest consideration of the use of a nucleoside analogue (lamivudine, telbivudine or entecavir) for cases of fulminant or protracted severe

TABLE 5.3 Serodiagnosis of HAV and HCVs

Interpretation	anti-HAV IgM	anti-HAV total	HBsAg	anti-HBs	anti-HBc		HBeAg	anti-HBe	HBV DNA	anti-HCV	HCV PCR
					IgM	total					
Acute hepatitis A	+	-	-	-	-	-	-	-	-	-	-
Past hepatitis A	-	+	-	-	-	-	-	-	-	-	-
Acute hepatitis C	-	-	-	-	-	-	-	-	-	+ or -	+
Chronic hepatitis C (symptomatic or asymptomatic)	-	-	-	-	-	-	-	-	-	+	+
Resolved hepatitis C	-	-	-	-	-	-	-	-	-	+ or -	-

Note: co-infection or superinfection may make interpretation more complicated.

TABLE 5.4 Serological, virological and biochemical profiles of HBV⁵

	HBsAg	Anti-HBs	Anti-HBc (total)	Anti-HBc IgM	HBeAg	Anti-HBe	HBV DNA (IU/ml)	ALT
Acute HBV	+	-	+	+	+	+/-	High	↑
Chronic HBeAg positive								
Immunotolerant Phase	+	-	+	-	+	-	High	N
Immunoclearance Phase	+	-	+	-	+	+/-	High	↑
Chronic HBeAg Negative								
'Inactive Carrier state'	+	-	+	-	-	+	<20 000	N
'Precore mutant'	+	-	+	-	-	+	>20 000	↑
'Occult'	-	-	+	-	-	+/-	Very low	N
Reactivation HBV	+	-	+	+/-			High	↑
Vaccinated	-	+	-	-	-	-	-	N
Resolved HBV	-	+	+	-	-	+/-	-	N

* By kind permission of Dr Mark Danta
+ = positive, - = negative, N = normal, ↑ = elevated.

acute hepatitis B.⁶ Patients with undiagnosed chronic HBV may develop severe spontaneous flares of hepatitis which appear clinically as an acute hepatitis. In this situation, resolution may be enhanced with nucleoside analogue therapy (Chapters 1 and 11).

In the case of acute hepatitis C there is evidence that treatment with pegylated (PEG)-interferon-based therapy alone in the acute phase of infection results in greater rates of viral clearance than treatment in the chronic stage of hepatitis C infection.^{7,8} However, since some people will spontaneously clear HCV without treatment, the ideal time to commence PEG-interferon therapy remains to be determined. A strategy of waiting 12 weeks to establish whether spontaneous clearance will occur and commencing PEG-interferon therapy if not has been proposed.⁹ This subject is still the focus of ongoing clinical trials and referral to a specialist for further advice should be considered for all patients diagnosed with acute hepatitis C. Section 100 of the PBS does not fund PEG-interferon 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.

Acute liver failure is the most serious complication of viral hepatitis, occurring in less than 1% of HAV and HBV cases. It remains unclear whether acute HCV can result in acute liver failure. In viral hepatitis, acute liver failure results from massive, immune-mediated hepatocyte necrosis. Risk factors for the development of acute liver failure in viral hepatitis 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.

Altered mental status (hepatic encephalopathy) and coagulopathy in the setting of acute hepatitis defines acute liver failure. Typically, non-specific symptoms such as malaise, nausea, intractable vomiting and sleep disturbance develop in the previously healthy person, followed by jaundice, the rapid onset of altered mental

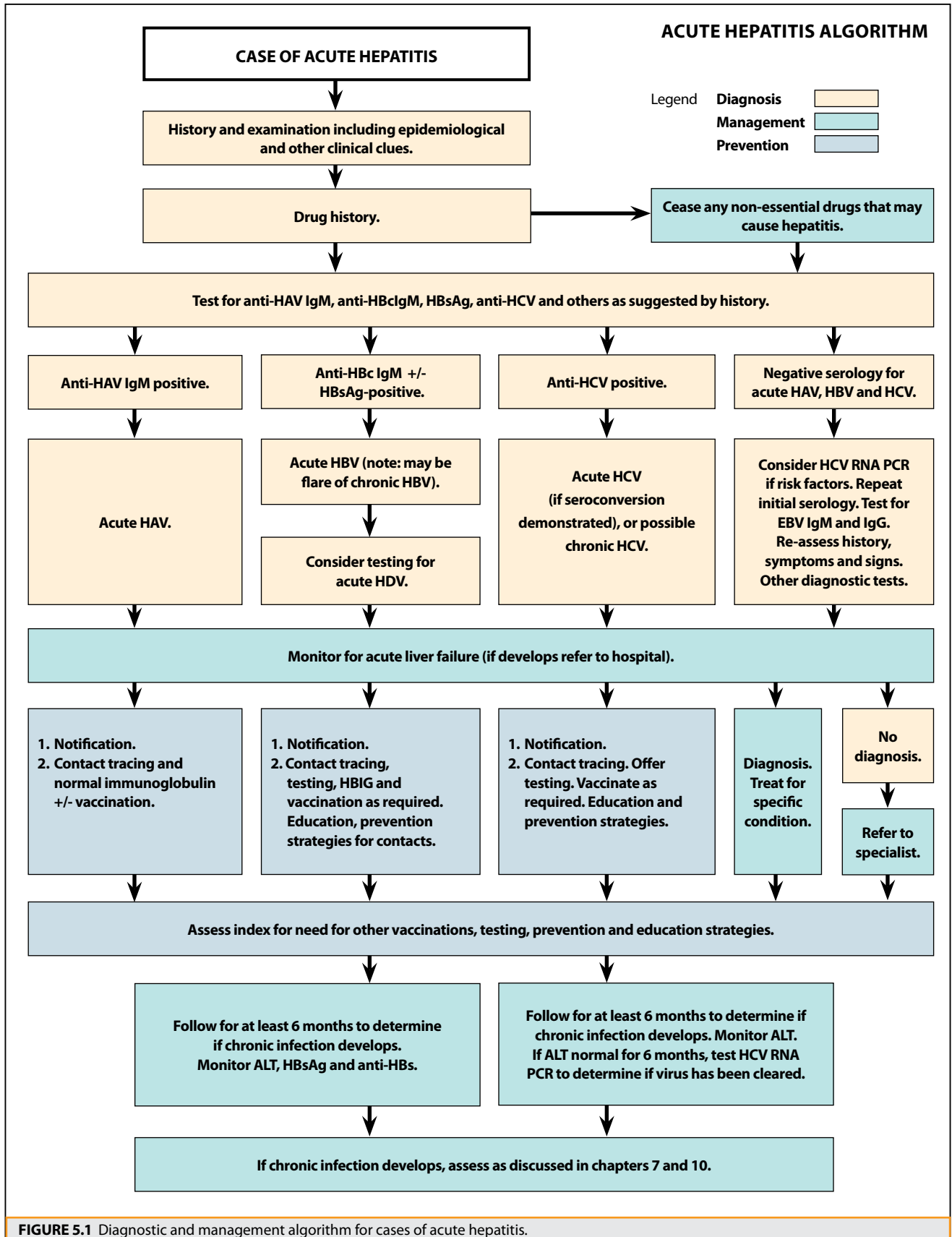


FIGURE 5.1 Diagnostic and management algorithm for cases of acute hepatitis.

status and coma. Thus, the patient goes from being healthy to moribund within 2–10 days. Supportive laboratory findings include high serum ALT, low blood glucose levels and worsening coagulopathy.

The management of acute liver failure begins with the recognition that patients with coagulopathy or encephalopathy may die. Due to the potential for rapid deterioration in their clinical status and the need for close monitoring, patients with acute liver failure are best cared for 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

To determine whether chronic infection has been established, the recommended follow-up time for acute hepatitis is at least six months. Repeatedly normal ALT results and a negative HCV RNA PCR at six months indicate viral clearance. Table 5.4 and Figure 5.1 provide 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 preventative measures to be implemented. Discussion with the patient regarding how to proceed with contact tracing may be appropriate. The clinician may ask the patient to consider recent blood-to-blood or sexual contacts as well as recent blood donations. With regard to HAV, household and occupational contact tracing may be relevant. It is recommended that primary care practitioners keep up to date with the relevant State or Territory guidelines.

Public health notification

Cases of 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 implementing harm reduction and preventive measures, such as vaccination, should also be taken.

Specialist and hospital referral

Referral to hospital is appropriate in cases where the primary care clinician assesses an individual to have severe hepatitis or possible acute liver failure. Specialist referral is recommended:

- Where the primary care clinician is unable to make a definitive diagnosis
- Where multiple diagnoses appear to co-exist
- For consideration of antiviral therapy in acute hepatitis
- Where other, treatable conditions have been diagnosed

CASE STUDY 1

Hepatitis B diagnosis: managing the anxious patient

Anxious patient with acute viral hepatitis

Peter is a 19-year-old man of European background who presents to a general practice clinic. He has recently been told by another service that he has hepatitis B after an episode of jaundice. Peter has no idea whether he has acute or chronic infection and believes that it is 'for life'. He is distressed and expresses fear about sharing food with his family, kissing and hugging. Peter believes that he will never be able to have sex again because he is contagious.

Peter is confused about the differences between acute and chronic infection, and he has an exaggerated sense of how easily HBV can be transmitted. People with the infection are often extremely fearful of infecting loved ones and need accurate information from health professionals to enable them to continue in their usual activities and maintain closeness with family and friends.

The clinician contacts the other service, establishes how the diagnosis was made and uses the serology and other investigations to determine that Peter has acute hepatitis B infection. Peter is queried regarding symptoms such as intractable vomiting, disturbed sleep and altered mental state, and examined for physical signs including asterixis (hepatic flap) and fetor hepaticus, to ensure that there is no evidence of liver failure. Peter agrees to have further liver function tests and INR as recommended by the clinician.

Although serology shows Peter is negative for HCV and HIV antibodies as well as HAV IgM, the clinician assesses Peter for risk factors for viral hepatitis and discusses the ways in which other blood-borne viruses and sexually transmitted infections can be prevented.

The clinician explains how HBV is transmitted and, importantly, also discusses ways in which it is not transmitted (Chapters 1 and 2). The clinician states that over 90% of adults clear acute HBV infection (Table 5.1) but even if Peter does develop ongoing or chronic infection, he can still kiss, hug, share food and even have sex without transmitting HBV. The clinician explains that an effective vaccine is available for his loved ones ('Post-exposure prophylaxis' and 'Immunisation' in this chapter), although Peter will need to use condoms for sexual intercourse until any sexual partners are effectively vaccinated.

The clinician tells Peter that he requires follow-up for at least six months to ascertain clearance or persistence of HBV infection. Peter is invited to return the following week to discuss his test results and review other issues discussed during the consultation.

Because of the fear and uncertainty associated with viral hepatitis, it is especially important that health professionals give accurate information about transmission and prognosis at the time of diagnosis, and explore the availability of treatment options if chronic infection with HBV develops.

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 childcare 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 13). 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 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 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 exposure to 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 viral infections in the source. If the source is available and willing, screening 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.¹⁰

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 hepatitis B immunoglobulin (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

HAV

Post-exposure prophylaxis is recommended for the close contacts of people with HAV. This includes household and sexual contacts who have had contact with the index case two weeks before, or up to one week after, the onset of jaundice. Normal human immunoglobulin (NHIG) is recommended and should be given within 14 days of the exposure. The standard dose is 2.0 mL (1.0 mL for persons 25–50 kg; 0.5 mL for persons under 25 kg in weight). It is given as a single intra-muscular injection. If the patient is a food handler, all other food handlers at his or her place of work should receive normal human immunoglobulin. 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, NHIG should be offered to children and susceptible staff in the relevant age groups or classes at the facility. HAV vaccine can be commenced simultaneously with normal human immunoglobulin and should be considered for those at ongoing risk of HAV infection.¹¹

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 exposure) should be given HBIG as soon as possible within 12 hours of birth; within 72 hours of percutaneous/ocular/mm exposures and 14 days of sexual contact.¹¹ (The dose of HBIG is 400 IU for adults and 100 IU for children.) Concomitantly, HBV vaccination should be injected at a separate 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 or chronic infection. Serological follow-up after exposure to HBV and HCV should occur at one, three and six months as both infections can have prolonged incubation periods.

The HCV RNA PCR assay is currently funded 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 four weeks, although some may have transient viraemia that clears before this time. A single negative HCV RNA result does not exclude infection with HCV 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 15).

The anxieties and concerns of the patients regarding transmission to sexual partners and family can be addressed by a discussion of 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

Newborn babies of HBV-infected mothers should receive HBIG and be started on a course of HBV vaccination at birth. This strategy effectively prevents transmission of HBV infection. There are no effective strategies to prevent perinatal transmission of HCV, although avoidance of invasive foetal monitoring may be important. Potential benefits of caesarean section have not been proven and there is no place for routine caesarean sections in HCV-infected mothers. Breastfeeding is regarded as safe unless blood is present in the milk.

Immunisation¹¹

HAV vaccination is recommended for some populations at high risk (Table 5.5). Screening for immunity prior to immunisation is recommended for persons born before 1950, for those who spent their childhood in endemic countries (China, South East Asia and Pacific countries) and for those who report previous 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, two months, four months and six or 12 months) and, in school-based programs, to adolescents between 10 and

TABLE 5.5 Persons for whom hepatitis A vaccine is recommended¹¹

(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])

• Travellers to endemic areas
• Visitors to rural and remote Aboriginal communities
• Childcare and pre-school personnel
• The intellectually disabled and their carers
• Health care workers who provide care for substantial populations of Indigenous children
• Sewage and waste disposal workers
• Men who have sex with men
• Injecting drug users
• Persons with chronic liver disease
• Persons with chronic HCV infection
• Sex workers
• People with HBV

TABLE 5.6 Persons for whom hepatitis B vaccination is recommended¹¹

• Infants and young children	• Persons with HCV infection
• Young people aged 10 to 13 who have never received a primary course of HBV vaccine	• Persons with clotting disorders who require multiple blood product administration
• Liver transplant recipients	• Health care workers with direct patient or human tissue contact
• Household contacts of people with acute HBV or HBV carriers	• Prisoners and staff of long-term correctional facilities
• Sexual contacts of people with acute HBV or HBV carriers (these people should also be offered hepatitis B immunoglobulin)	• Residents and staff of facilities for persons with intellectual disabilities
• Men who have sex with men	• Embalmers
• Injecting drug users	• Haemodialysis patients
• Individuals adopting HBsAg+ children	• At-risk emergency services personnel, police and waste disposal workers
• People with HIV infection or impaired immunity	• People with chronic liver disease
• Tattooists and body piercers	• Sex workers

13 years of age who have not previously been vaccinated. HBV vaccination is also recommended for populations at high risk (Table 5.6). Vaccination is safe for people with HIV, although protection is likely to be weak or transient compared with the highly effective, protective immunity produced among immunocompetent individuals. Serological confirmation of post-vaccination immunity is not required after routine child and adolescent vaccination but is recommended for some high risk individuals (see current edition of the Australian Immunisation Handbook).¹¹ Booster doses are not recommended in immunocompetent people but may be required for those with impaired immunity, who should have regular monitoring of their anti-HBs levels at six to 12 month intervals.

The recombinant HBV vaccine entails an initial dose followed by two further doses at one and six months. The vaccination schedule may vary according to likelihood of compliance. The rapid schedule (0, 7 and 21 days) may be more appropriate in highly mobile populations.¹¹ Access to free HBV vaccination is available through sexual health clinics, some councils and other selected clinics.

A combined vaccine for HAV and HBV is available and should be considered for individuals at risk of both infections and for people with chronic HCV. Such persons may include health care workers and students, long-term visitors to endemic countries, men who have sex with men, injecting drug users, prisoners and prison workers. There is no vaccine for HCV.

Education and harm minimisation

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

Concurrent assessment for drug treatment programs may be considered for those who inject drugs. Referral to injecting drug user groups (such as the Australian IV League or local equivalent) for education and support may also be considered (Chapter 15).

Travellers require accurate advice and appropriate vaccination or passive immunisation prior to 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 infected individuals. 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 minimisation strategies should be fully explored to reduce ongoing transmission.

References

- 1 National Centre for HIV Epidemiology and Clinical Research (NCHECR). HIV/AIDS, viral hepatitis and sexually transmitted infections in Australia. Annual Surveillance Report 2006. Sydney: NCHECR, 2006.
- 2 Ministerial Advisory Committee on AIDS, Sexual Health and Hepatitis: Hepatitis C Sub-Committee. Hepatitis C virus projections working group: estimates and projections of the hepatitis C virus epidemic in Australia 2006. Sydney: University of New South Wales, 2006.
- 3 Alter HJ, Seef LB. Recovery, persistence and sequelae in hepatitis C infection: a perspective on long-term outcome. *Sem Liver Disease* 2000;20:17–35.
- 4 Micallef JM, Kaldor JM, Dore GJ. Spontaneous viral clearance following acute hepatitis C infection: a systematic review of longitudinal studies. *J Viral Hepatitis* 2006;13(1):34–41.
- 5 Matthews G, Robotin M (eds). *B Positive: all you wanted to know about hepatitis B - a guide for primary care providers*. Sydney: Australasian Society for HIV Medicine and Cancer Council of NSW, (at press).
- 6 Lok AS, McMahon BJ. Chronic hepatitis B. *Hepatology* 2007;45(2):507–39.
- 7 Wiegand J, Buggisch P, Boecher W, Zeuzem S, Gelbmann CM, Berg T, et al. for the German HEP-NET Acute HCV Study Group. Early monotherapy with pegylated interferon alpha-2b for acute hepatitis C infection: the HEP-NET acute-HCV-II study. *Hepatology* 2006;43(2):250–6.
- 8 Zekry A, Patel K, McHutchison JG. Treatment of acute hepatitis C infection: more pieces of the puzzle? *J Hepatology* 2005;42(3):293–6.
- 9 Kamal SM, Fouly AE, Kamel RR, Hockenjos B, Al Tawil A, Khalifa KE, et al. Peginterferon alfa-2b therapy in acute hepatitis C: impact of onset of therapy on sustained virologic response. *Gastroenterology* 2006;131(3):979. Published erratum on *Gastroenterology* 2006;130(3):632–8.
- 10 Dore GJ, Kaldor JM, McCaughan GW. Systematic review of the role of the polymerase chain reaction in defining infectiousness among people infected with hepatitis C virus. *Br Med J* 1997;315:333–7.
- 11 National Health and Medical Research Council. *The Australian Immunisation Handbook*. 9th edn. Canberra: Department of Health and Aged Care; 2008.