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Prevention and control of viral hepatitis

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Aims and intended learning outcomes

The aim of this article is to provide an essential overview of viral hepatitis with an emphasis on primary prevention in the community and preventing exposure and infection in healthcare settings. After reading the article you should be able to:

- Define acute and chronic viral hepatitis.
- Describe the characteristics of hepatitis-specific viruses.
- Discuss the transmission patterns and clinical outcomes associated with different hepatotropic viruses.
- Give accurate primary prevention advice to clients.
- Identify potential strategies for preventing exposure and infection in healthcare environments.

Introduction

Every year, millions of people throughout the world become infected with viruses that cause acute and chronic hepatitis; the associated burden of ill health has become a major public health concern in all countries. Equally serious is the potential risk to nurses, midwives and other healthcare providers of becoming exposed and infected during direct care activities. An understanding of the sometimes confusing world of the hepatitis viruses is an important first step to developing sound infection prevention and control strategies.

Physiology of the liver

The liver, which is located in the upper right portion of the abdomen just behind the lower portion

of the ribs (which protect it from injury), is the largest and most metabolically complex organ in the body. It carries out hundreds of different functions designed to maintain a favourable internal environment in the body (metabolic homeostasis) and helps protect against infections (Box 1).

The liver receives oxygen-rich blood from the heart via the hepatic artery and nutrient-rich blood from the capillaries in the intestinal wall, which drain into the liver via the hepatic portal vein. The blood then flows through a latticework of tiny vascular channels inside the liver where the terminal portal venules and hepatic arterioles enter small sac-like units known as acini. In each hepatic acinus an exchange of nutrients and macromolecules takes place with nearby hepatocytes – the functional cells of the liver. This exchange enables the metabolic and other processes that maintain the body's internal milieu. Following passage through the liver, the blood returns to the heart via the hepatic vein. At any given moment the liver is processing approximately 13 per cent (or one pint) of the body's total blood supply.

Liver tissue is mainly composed of hepatocytes (also known as parenchymal cells), but other non-parenchymal cells in the liver are equally essential for efficient hepatic functioning. Special endothelial cells line the terminal portal venules and hepatic arterioles that enter the hepatic acini. Unlike the endothelium lining blood vessels elsewhere, the endothelium in these vessels lacks a basement membrane and contains numerous pores (fenestrae) that permit the exchange of nutrients and macromolecules between the blood and hepatocytes. Additionally,

In brief

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Summary

Viral hepatitis is a common disease caused by various hepatotropic viruses. An awareness of the characteristics and transmission patterns of these viruses will help nurses to understand the diverse clinical outcomes of infection and develop effective patient education and infection prevention and control strategies.

Key words

- Hepatitis
- Infection control
- Virus diseases

These key words are based on subject headings from the British Nursing Index. This article has been subject to double-blind review.

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Box 1. Principal functions of the liver

- **Nutrient metabolism:** processes and converts substances in digested food into proteins, fats and carbohydrates, and controls their levels in the blood.
- **Protein synthesis:** manufactures essential protein compounds, such as albumin, clotting factors, complement factors, transferrin.
- **Storage and release:** stores sugar as glycogen and releases it into the bloodstream as glucose when needed for energy; also stores other nutrients, vitamins (A, D, B₁₂, K and folate), iron and copper, and releases them when required.
- **Cholesterol synthesis:** manufactures about half of the body's cholesterol which is needed as a component of cell membranes and to make bile and certain hormones, such as oestrogen, testosterone and adrenal hormones.
- **Bile formation:** manufactures and excretes bile salts (via the gallbladder where it is stored and concentrated) when needed for the digestions of fats.
- **Detoxification:** neutralises or degrades and excretes harmful substances absorbed from the intestines and blood, such as toxins (including alcohol), drugs, hormones and micro-organisms.
- **Protection against infection:** liver macrophages (Kupffer's cells) destroy bacteria and viruses found in the blood as it flows through the liver.

Box 2. Acute and chronic hepatitis

- **Acute hepatitis** is a sudden diffuse liver inflammation generally caused by infection with hepatotropic viruses and usually lasting only a few weeks or months.
- **Chronic hepatitis** is long-lasting liver inflammation (more than six months, but usually for years), which may result in cirrhosis and chronic liver failure.

the liver is the largest reticuloendothelial organ in the body and resident macrophages in the liver known as Kupffer's cells attack and destroy (phagocytose) bacteria and viruses and clear antigen-antibody immune complexes, damaged red blood corpuscles and various endotoxins that are in the blood being filtered by the liver. These cells also produce specialist immune system messenger proteins known as cytokines that help direct immune responses.

TIME OUT 1

Identify clinical problems that patients may experience when disease interferes with the normal functions of the liver and relate these to patients you have cared for previously.



TIME OUT 2

Reflecting on patient problems during the phases of acute viral hepatitis, identify likely nursing assessments and interventions.



Box 3. Hepatotropic viruses

- Hepatitis A virus (HAV)
- Hepatitis B virus (HBV)
- Hepatitis C virus (HCV)
- Hepatitis D virus (HDV) (Delta)
- Hepatitis E virus (HEV)

Hepatitis

Hepatitis refers to an acute or chronic inflammation of the liver (Box 2), usually caused by hepatitis-specific viruses, alcohol and drugs. Less commonly, hepatitis can be caused by other viruses, including the Epstein-Barr, cytomegalovirus, herpes simplex viruses, and some parasitic infections, such as malaria, amoebiasis and schistosomiasis. This article concentrates on the five hepatitis-specific viruses that are the major cause of hepatitis throughout the world. Because these viruses specifically target and infect the hepatocytes, they are known as hepatotropic viruses (Box 3).

Clinical features of acute viral hepatitis

Following infection, most people developing acute viral hepatitis have similar clinical features (Box 4),

although immunosuppressed people, such as those infected with human immunodeficiency virus (HIV), may have a more severe illness.

During the incubation period (also called the prodromal or pre-icteric phase) individuals commonly feel progressively unwell and most eventually develop jaundice (icteric phase). Despite worsening jaundice during this phase, symptoms usually begin to regress and the patient starts to feel better. As the jaundice fades and symptoms resolve, the patient begins to make a recovery (recovery phase).

These three phases represent the natural history of acute viral hepatitis, although there is significant variation in the length of each phase, the severity of illness experienced by the patient and the long-term outcomes in relation to viral carriage and chronic liver disease. The degree of difference in natural history is influenced by the individual patient's immune competence and health reserves, and the characteristics of the specific, infecting hepatotropic virus. For example, the incubation period for acute disease caused by hepatitis A or hepatitis E viruses is significantly shorter than acute hepatitis caused by hepatitis B or hepatitis C viruses. A carrier state and the potential for chronic liver disease is only associated in some people following infection with hepatitis B and hepatitis C viruses.

Characteristics of hepatotropic viruses

The hepatotropic viruses are a mixture of viral species from different virus families, showing similarities and differences to each other – each with a well-known natural history and its own menu of potential health outcomes (Box 5). It is important to understand these different viral characteristics, which influence the patient's medical and nursing management, quality of life and long-term prognosis.

Hepatitis A and hepatitis E viruses are enteric pathogens transmitted by the faecal-oral route. They will be discussed first, followed by hepatitis B, D and C viruses, which are bloodborne viruses, transmitted parenterally, sexually and from mother-to-child (vertical transmission).

Hepatitis A

Hepatitis A virus (HAV) is an enterovirus that infects cells in the gut from where it spreads to the liver via the blood. Following infection, HAV is found

principally in the faeces; transmission generally occurs from person to person by a faecal-oral route. HAV can also be transmitted as a result of faecal contamination of food or water (and faecally contaminated water used to wash salads and other raw and unpeeled vegetables and fruits). Shellfish may also be contaminated from filter-feeding in water that has been contaminated by human faeces. All of this most commonly occurs where sanitation and hygiene are poor. When these standards improve, as has happened in the developed world, the incidence of infection and the prevalence of natural antibody to this virus fall. Standards of personal and food hygiene in the UK are high and the incidence of HAV infection has progressively fallen during the past century. Because of this, most people today have not been previously exposed to HAV and do not have natural antibodies to this virus. Consequently, the majority of the UK population is now susceptible to HAV infection (Crowcroft *et al* 2001). HAV can also be transmitted sexually, for example, following some types of sexual foreplay and/or oral-anal sexual activity. Finally, during the incubation period, there is a transient viraemia when HAV can be found briefly in the serum and, occasionally, HAV transmission can occur following exposure to infectious blood or other body fluids.

Hepatitis A has a short incubation period. Following exposure, an infected person excretes the virus in his or her faeces and urine for two to three weeks before the onset of symptoms, and for one to two weeks thereafter (Burns 2002). The disease is generally mild and complete recovery usually occurs within four to eight weeks. Progression to chronic hepatitis is rarely, if ever, seen. The severity of the illness ranges from asymptomatic (subclinical) infection (common), through to clinical hepatitis (with or without jaundice) to (rarely) fulminant hepatic failure. In areas of high prevalence, most children have antibodies to HAV by the age of three years, and these early childhood infections are generally asymptomatic or very mild. In later life, HAV infection tends to cause clinical disease, with 70-80 per cent of adults developing jaundice, preceded typically by malaise, anorexia, nausea and fever (Collier and Oxford 2000). Patients aged over 49 and those with existing liver disease have a higher risk of morbidity and mortality (Crowcroft *et al* 2001).

Following primary infection, IgM antibodies to HAV appear and are present for two to six months before disappearing and being replaced by IgG HAV antibodies. The presence of specific HAV IgM antibodies is indicative of primary infection, whereas the presence of HAV IgG antibodies indicates previous exposure and immunity from further attacks (Finlayson *et al* 1999).

HAV infection is not associated with a carrier state.

Box 4. Clinical features of acute viral hepatitis

- Pre-icteric phase: 'prodromal' or incubation phase occurs two to three weeks before the onset of jaundice, with the development and a progressive worsening of anorexia, malaise, nausea and vomiting, fever, distaste for cigarette smoking (if a smoker). Sometimes urticarial, pruritic hives, maculopapular lesions and/or fleeting, irregular patches of erythema; arthralgias (joint pains) occasionally occur, especially in HBV infection. Myalgias (muscle pains), chills and right upper quadrant abdominal pain may occur.
- Icteric phase (jaundice): lasts one to three weeks. Dark urine and jaundice: eyes show scleral icterus, and faeces may be clay-coloured. Temperature usually normal, as are vital signs, however, there may be a bradycardia (slow heart rate) if the patient has severe hyperbilirubinaemia. Serum bilirubin may increase to 20 times normal; hepatic transaminases levels (AST, ALT) may increase to 100 times normal and an increase in alkaline phosphatase (one to three times normal) may also be seen. Systemic symptoms begin to regress and the patient feels better, despite worsening jaundice.
- Recovery phase: jaundice gradually recedes during a four-to-eight-week recovery phase.

Faecal excretion of the virus declines rapidly once clinical symptoms appear and usually ceases within two weeks following the onset of clinical hepatitis. HAV infection is prevalent throughout the world and is maintained in the human population through faecal-oral contamination routes.

Hepatitis E

Hepatitis E virus (HEV) is a calicivirus and, like HAV, is transmitted enterically (faecal-oral route). It is a major cause of epidemic, water-borne hepatitis in many parts of the world, but not in the UK. The incubation period and clinical features are much the same as in HAV infection, except the illness may be more severe, especially in pregnant women, in whom high mortality rates may be seen (Finlayson *et al* 1999). This virus is endemic in tropical and subtropical developing countries, where infection is highly associated with water contaminated by human faeces. Visitors and tourists to these regions may become infected and develop acute viral hepatitis when they return home. HEV infection is not associated with a carrier state or progression to chronic liver disease. Hepatitis E is diagnosed by finding HEV antibody (anti-HEV) in the blood.

TIME OUT 3

Before reading on, consider the advice you would give patients for preventing exposure to HAV and HEV.



Preventing hepatitis A and hepatitis E Good personal hygiene helps to avoid exposure. Food handlers in particular must consistently use effective hand hygiene measures, especially after using the toilet. Travellers to regions where there is likely to be unreliable sanitation should ensure that they drink

**Box 5. Characteristics of hepatotropic viruses**

Hepatotropic virus	Hepatitis A virus (HAV)	Hepatitis B virus (HBV)	Hepatitis C virus (HCV)	Hepatitis D virus (HDV)	Hepatitis E virus (HEV)
Virus group	Enterovirus	Hepadnavirus	Flavivirus	Incomplete	Calicivirus
Nucleic acid in genome	ssRNA ⁺	dsDNA	ssRNA ⁺	Incomplete RNA [#]	ssRNA ⁺
Serologic diagnosis	IgM anti-HAV	HBsAg [▼]	Anti-HCV	Anti-HDV	Anti-HEV
Incubation period (weeks)	2-4	6-23	2-26	6-9	3-8
Transmission					
* Faeces	Yes	No	No	No	Yes
* Blood	Uncommon	Yes	Yes	Yes	Uncommon
* Saliva	Uncommon	Yes	Yes	Questionable	Uncommon
* Sexual	Possible	Yes	Uncommon [◊]	Yes	Possible
* Mother-to-child	No	Yes	Uncommon [◊]	Yes	No
Epidemics	Yes	No	No	No	Yes
Chronic infection	No	Yes (5-10%)	Yes (>50%)	Yes	No
Liver cancer	No	Yes	Yes	Yes	No
Prevention					
Passive immunisation	Normal immunoglobulin	Hepatitis B immunoglobulin	No	Prevented by preventing HBV infection	No No
Active immunisation [^]	Various formaldehyde inactivated vaccines	Various vaccines containing inactivated HBsAg prepared from yeast cells by recombinant DNA technique	No	Prevented by preventing HBV infection	No

[#] HDV requires presence of HBV for replication

[▼] HBsAg = Hepatitis B surface antigen

* All body fluids are potentially infectious, though some, for example, urine, are less infectious

[◊] HCV: sexual and mother-to-child transmission may be more efficient in patients co-infected with HIV

[^] Combined hepatitis A and hepatitis B vaccines are available

bottled or boiled water and avoid ice cubes in drinks unless they are confident they have been made from safe water. They should also refrain from eating raw shellfish and peel all fruit and vegetables. Salad ingredients may have been washed in contaminated water so they should be avoided in areas where the water supply is unsafe.

Passive immunisation, using human normal immunoglobulin (HNIG), can be used for temporary and immediate protection against Hepatitis A. Active immunisation is available by the use of one of several inactivated HAV vaccines (Mehta 2002).

Individuals should be tested for HAV IgG prior to either form of immunisation if time allows and if they are aged over 50 (especially if they have lived overseas) or have a history of jaundice (DoH 1996a). The reason for this is that if they are positive for HAV IgG, they may not require immunisation (depending on the titre of antibody in their blood), as a protective level of HAV IgG demonstrates previous infection and immunity to disease following new HAV infections. Unfortunately, neither passive nor active immunisation products are available to prevent Hepatitis E.

Hepatitis B

Hepatitis B virus (HBV) is a hepadnavirus and has distinct, intimately related antigen-antibody (Ag-Ab) systems:

- **HBV surface antigen (HBsAg).** This is associated with the viral surface coat and its presence in serum usually provides the first evidence of acute HBV infection. Excess viral coat protein (HBsAg, or 'Australia antigen') is produced and these extra pieces of HBsAg outnumber the intact hepatitis B virion (sometimes called the 'Dane particle'). HBsAg characteristically appears during the incubation period, usually one to six weeks before clinical or biochemical illness develops, and disappears during convalescence. The corresponding antibody (anti-HBs) appears only weeks or months later following clinical recovery, and usually persists for life. In up to 10 per cent of patients, HBsAg persists after acute infection and anti-HBs does not develop. These patients become asymptomatic carriers of the virus and some will develop chronic hepatitis (Collier and Oxford 2000).

- **HBV core antigen (HBcAg).** This is associated with the viral inner core. Antibody to the core (anti-HBc) appears at the onset of clinical illness, with gradually diminishing titres thereafter, usually for years or life.

- **HBV 'e' antigen (HBeAg).** This appears to be a peptide derived from the viral core (not to be confused with hepatitis E virus). It is found only in patients with detectable HBsAg in their blood. Its presence reflects more active viral replication and is generally associated with greater infectivity of blood (and other body fluids, such as semen) and a greater likelihood of progression to chronic liver disease. In contrast, presence of the corresponding antibody (anti-HBe) signals relatively lower infectivity and a better outcome.

HBV infection is diagnosed by identifying HBsAg in blood: its presence indicates acute primary HBV infection. The corresponding antibody, anti-HBs, appears after recovery and persists for life. Its presence indicates previous infection and immunity from disease following new HBV infections.

During primary infection, an impressive level (titre) of virus is found in the blood, for example, as high as 500(g/ml of viral antigen and 10 trillion extra particles of surface antigen (HBsAg) in each millilitre of blood (Dienstag *et al* 1991). Following infection, HBV can be found in most body fluids, for example, in blood, saliva, seminal fluid, cerebrospinal fluid, breast milk and urine (Burns 2002).

HBV transmission HBV is transmitted parenterally, typically by contaminated blood or blood products in many countries where screening of human blood

for transfusion is unreliable or inconsistent. In the UK, all donor blood is screened for HBV infection and transmission rarely, if ever, occurs from blood transfusions or blood products (UKBTS 2002). Injecting drug users frequently become infected with HBV (and other parenterally transmitted viruses, such as HIV and hepatitis C virus) following exposure resulting from sharing blood-contaminated injecting equipment. Occupational exposure to HBV following needlestick injuries is a recognised risk in healthcare settings.

Non-parenteral spread occurs, including sexual transmission between heterosexual and homosexual partners.

Mother-to-child (vertical) transmission also occurs, during pregnancy or birth, or following birth from breast milk. Breastfeeding is the most frequent mode of transmission in some developing countries, especially in the Far East, where it is an important cause of HBV perpetuation in the population. In the UK, all pregnant women are offered and recommended HBV testing during antenatal care (NHS Executive 1998) and, therefore, it is uncommon for HBV to be transmitted vertically in this country.

The incubation period for hepatitis B ranges from six to 23 weeks following exposure to the virus. Approximately 2-10 per cent of those infected as adults will become chronic carriers of HBV (DoH 1996a). Those carriers in which e-antigen (HBeAg) can be detected in their serum are the most infectious to others, as opposed to carriers where antibody to this antigen (anti-HBe) is detected in serum and who are generally of low infectivity. Chronic viral carriage is more frequent in those infected as children and rises to 90 per cent in those infected perinatally (DoH 1996a). Worldwide, 20-25 per cent of chronic carriers develop progressive liver disease, often leading to cirrhosis and hepatocellular carcinoma (DoH 1996a).

Hepatitis B is endemic in most countries and, globally, remains one of the most serious, persistent viral infections. In South and East Asia, up to 20 per cent of the population are carriers, and in Africa and Central and South America, up to 10 per cent of the population may be carriers (Roure 1995, Van Damme *et al* 1995). Although the incidence of overt cases of Hepatitis B in the UK is low and shows a decreasing trend (DoH 1996a), there are estimated to be one million cases of acute hepatitis B and 90,000 new cases of chronic hepatitis B in Europe every year (Brook 2001, Roure 1995).

Hepatitis D

The hepatitis D virus (HDV), also called the 'delta agent', is a unique, defective virus that can replicate only in the presence of HBV, never alone. It

**Box 6. Liver function tests**

- Full (complete) blood count (FBC or CBC)
- Coagulation tests
- Prothrombin time
- Biochemical tests
- Urea and electrolytes
- Liver function tests (plasma)
- Aspartate aminotransferase (AST)
- Alanine aminotransferase (ALT)
- Alkaline phosphatase
- Gamma-glutamyl transferase (GGTP)
- Proteins (total and albumin)

occurs as either a co-infection with acute HBV infection, or as a superinfection in established chronic hepatitis B.

Injecting drug users are at a relatively high risk of acquiring HDV infection in Europe and North America. In other parts of the world, HDV is more commonly acquired following sexual exposure. Mother-to-child transmission also occurs.

Clinically, HDV infection is typically manifested by unusually severe (fulminant) acute viral hepatitis; acute exacerbation in chronic HBV carriers (superinfection); or a relatively aggressive course of chronic hepatitis B. HDV is diagnosed by detecting antibodies to the virus (anti-HDV) in the blood.

Non-A, non-B hepatitis

Following the introduction of testing blood donors for HBV infection in the 1970s, and HAV infection in the 1980s, and excluding those found positive, it was hoped that post-transfusion viral hepatitis would virtually disappear. However, this did not happen and, as people continued to develop hepatitis following blood transfusions or from the use of blood components, it was clear that there must be other, unknown hepatotropic bloodborne viruses. Viral hepatitis not caused by HAV or HBV infection was referred to as 'non-A, non-B hepatitis'. The responsible viruses were transmitted parenterally and sexually (and sometimes vertically), similar to HBV transmission, or enterically, infecting people by a faecal-oral route of transmission, exactly like HAV infection. In 1988, the 'non-A, non-B' virus responsible for the vast majority of cases of post-transfusion hepatitis was identified and termed hepatitis C virus. A few years later, in 1990, scientists identified the hepatotropic virus being transmitted enterically, principally in faecally contaminated water in many countries in the developing world. This virus was named the hepatitis E virus (discussed earlier).

Hepatitis C

Hepatitis C virus (HCV) belongs to the flavivirus family. It is endemic worldwide with high prevalence rates in South and East Asia and Eastern Europe (Brook 2001). There are at least six closely related strains (genotypes) of HCV, referred to as 1-6, and more than seven subtypes, referred to as: a, b and c. The prevalence of different genotypes varies in different geographical locations throughout the world. In the UK, Europe and North America, genotype 1, 2 and 3 are the most common (Morgan-Capner and Simmonds 2002).

HCV transmission HCV is predominantly transmitted by blood and blood products. Prior to the introduction of HCV screening of all donor blood

in 1990, most infections in Europe and North America occurred following blood transfusions or the use of blood products. This mode of transmission has now been virtually eliminated. Today, the greatest risk of acquiring HCV infection in the industrially developed world is from exposure to HCV-contaminated blood as a result of injecting drug use. Sexual and mother-to-child HCV transmission also occurs and HIV co-infection may increase this risk (Thomas *et al* 1998). There is no evidence that HCV is transmitted by breast milk and, consequently, breastfeeding is considered safe (DoH 2002a). Healthcare workers are at an increased risk of HCV infection following occupational exposure to blood or blood-contaminated body fluids, especially if the source patient was viraemic, that is, their blood was positive for HCV RNA (CDC 1998, Ramsay 1999).

Clinical outcomes The natural history and clinical course of hepatitis C can be influenced by virus characteristics, such as the specific genotype, viral load, co-infection with other viruses, including HIV, geographical location, alcohol use, drug use (including some antiretroviral and antituberculosis drugs), and other unexplained factors (Morgan-Capner and Simmonds 2002).

Acute hepatitis C Following primary HCV infection, most patients will remain asymptomatic with only one third presenting with malaise, weakness, anorexia and sometimes jaundice (CDC 1998). However, all patients will sustain some degree of liver cell injury during this period, as demonstrated by blood tests for liver function (Box 6). Antibodies to HCV (anti-HCV) will appear in almost all patients within 90 days, but only about 15-25 per cent will be able to clear the virus and make a complete recovery from acute hepatitis C (DoH 2002a). They will remain anti-HCV positive but they will become HCV RNA negative. Most (75-85 per cent) HCV-infected people will develop chronic infection with persistent (sometimes intermittent) viraemia and it is this propensity for chronic infection that is a unique hallmark of hepatitis C (DoH 2002a).

Chronic hepatitis C Most people with chronic HCV infection are asymptomatic for the first two decades following primary infection. Some may develop non-specific symptoms during this period, such as malaise and fatigue. Somewhere between 60 and 70 per cent of chronically infected people will have abnormal liver function tests (Box 6), especially persistent or fluctuating ALT (alanine aminotransferase) elevations which indicate active liver disease. The remaining 30-40 per cent of chronically infected people will have normal ALT levels and will not progress to active liver disease (CDC 1998). The course of chronic liver disease is variable and over a 20-30 year period, 10-20 per cent of people will develop cirrhosis (DoH

2002a, CDC 1998). This condition, leading to end-stage liver disease, may develop rapidly in some patients, especially if there is concomitant alcohol use (DoH 2002a). Between 1 and 5 per cent of people with chronic liver disease, usually those with cirrhosis, will develop hepatocellular carcinoma. A variety of other extrahepatic conditions may also be caused by chronic hepatitis C, including arthritis and glomerulonephritis (CDC 1998, DoH 2002a).

TIME OUT 4

Before reading on, consider the advice you would give patients on preventing exposure to HBV and HCV.



Preventing HBV, HDV and HCV infection Hepatitis B vaccination is recommended for all people at risk of exposure, including those with hepatitis C (CDC 1998, DoH 1996a). Additionally, hepatitis A and hepatitis B vaccination is strongly recommended for people with chronic hepatitis C. This is because the risk for fulminant hepatitis associated with hepatitis A appears to be increased in HCV co-infected people and hepatitis B is a co-factor for progression of hepatitis C disease (DoH 1996a). Antenatal screening for hepatitis B is important in preventing neonatal HBV infection (NHS Executive 1998) and screening all donor blood, plasma, tissues, organs and semen for HBV and HCV infection will prevent infections in people without behavioural risks for these infections. Safer sexual practices and harm-minimisation techniques for injecting drug users (Box 7) will help those whose behaviour or lifestyle place them at risk to avoid infection (CDC 1998, 1999).

Treatment of chronic hepatitis B and C

The treatment of chronic hepatitis B and hepatitis C is a rapidly changing area of clinical practice. Current drugs licensed for use in chronic viral hepatitis include interferon alfa (and peginterferon alfa 2b), ribavirin and lamivudine (Mehta 2002).

Chronic hepatitis B Interferon alfa is given by injection for three to four months, after which treatment is assessed. If there is no improvement at this time, interferon alfa is usually discontinued. The anti-retroviral drug lamivudine can be used for those patients for whom interferon alfa either cannot be used or is not effective. Both of these agents are associated with significant side effects and nurses need to ensure they consult the latest edition of the British National Formulary when administering these drugs (Mehta 2002). Interferon alfa is usually given by subcutaneous injection but some preparations can be given by intramuscular or intravenous injection.

Box 7. Prevention messages for people with high-risk behaviours

People who use or inject recreational drugs should be advised:

- To stop injecting recreational drugs.
- To enter and complete substance-abuse treatment, including relapse-prevention programmes.
- If continuing to inject drugs:
 - to never re-use or share syringes, needles, water or drug preparations equipment; if injection equipment has been used by other people, to first clean the equipment with bleach and water.
 - to use only sterile syringes and needles obtained from a reliable source, e.g. syringe and needle exchange programme or pharmacy (chemist).
 - to use a new sterile syringe and needle to prepare and inject drugs.
 - if possible, to use sterile (boiled) water to prepare drugs; otherwise to use clean water from a reliable source (such as fresh tap water).
 - to use a new or disinfected container ('cooker') and a new filter ('cotton') to prepare drugs.
 - to clean the injection site before injection with a new alcohol swab; and to safely dispose of syringe and needle after one use.
 - to get vaccinated against hepatitis A and hepatitis B.

People who use intranasal recreational drugs ('snort') should be advised:

- To be aware that this practice has been associated with HCV transmission.
- To not share equipment, for example, straws, with other users.

People considering tattooing or body piercing should:

- Be informed of potential risks of acquiring bloodborne infections which could be transmitted if equipment is not sterile or if effective infection prevention precautions are not followed, such as washing hands, using latex gloves, and cleaning and disinfecting instruments.

To reduce the risks for acquiring bloodborne infections, all clients should:

- Be advised not to share dental appliances, razors or other personal care articles.

People who are at risk for sexually transmitted diseases should be advised:

- That safer sexual practices should be encouraged.
- To use latex or polyurethane condoms correctly and every time for penetrative sexual intercourse to protect themselves and their partners from diseases spread through sexual activity.
- That if condoms are not used, to use safer, non-penetrative sexual practices.
- To get vaccinated against hepatitis A and hepatitis B.

Chronic hepatitis C Guidelines from the UK National Institute for Clinical Excellence recommend that patients with severe to moderate chronic hepatitis C should be treated with a combination of interferon alpha and ribavirin for six months (NICE 2000). Extension of treatment by a further six months is recommended only in those patients infected with HCV of genotype 1 and who have responded to treatment during the first six months (as judged by clearing of circulating HCV RNA) (Mehta 2002, NICE 2000).

Nursing assessment and intervention

The nursing history and ongoing nursing observations will elicit the typical signs and symptoms of

Infection control



Box 8. Fulminant hepatitis with encephalopathy

Onset: sudden rapid clinical deterioration with the onset of hepatic encephalopathy. Patient becomes lethargic and sleepy with personality and behavioural changes. Coma may develop within hours. An early sign is asterixis, the irregular flapping of forcibly dorsiflexed, outstretched hands. Bleeding is common, resulting from liver failure and DIC (disseminated intravascular coagulation). An increasing prothrombin time is a grave prognostic indicator.

Prognosis: meticulous nursing care and competent medical management of each specific complaint is required. Survival in adults is rare, although when it does occur, survivors often make a good recovery with minimal liver damage.

(Beers and Berkow 1999)

acute viral hepatitis (Box 4). Nursing interventions are focused on actual and potential patient problems, including activity intolerance, altered nutrition and potential fluid volume deficit. There is no substantial evidence to suggest that either dietary or activity restrictions have any benefit for patients with acute hepatitis, but alcohol intake is usually restricted to minimise liver damage. The patient's appetite usually returns to normal during the icteric phase of acute hepatitis. The use of drugs that are toxic to the liver, such as paracetamol (acetaminophen), some antiretroviral and anti-tuberculosis drugs, should be avoided.

Ongoing evaluation of care and continuing nursing observations will detect signs of complications, especially fulminant hepatitis with encephalopathy (Box 8). This is a rare condition, often associated with HBV or HCV infection. Although survival from this complication is rare, it does occur, usually because of highly skilled nursing care. Patient education initiatives might centre on reducing risk to other bloodborne viruses, such as safer sexual behaviour and harm-minimisation techniques used for injecting drugs (Box 7). Patients should be advised to avoid alcoholic drinks during acute and chronic phases of hepatitis.

TIME OUT 5

Before reading on, reflect on the care you would plan for a patient with either acute or chronic viral hepatitis and consider the precautions you would take to avoid exposure to these viruses.



Preventing occupational exposure to hepatitis viruses

Exposure to body substances, blood and other body fluids during patient care activities puts nurses and midwives at potential risk of infection with hepatotropic viruses. The estimated likelihood of becoming infected with bloodborne viruses following a single percutaneous exposure to blood or blood body fluids from a patient known to be infected has been calculated (Box 9) (CDC 1998, PHLS 1999). Percutaneous exposure (where the skin is cut or

penetrated by a needle or other sharp object) to blood from a patient with active HBV disease, and who is also 'e' antigen positive and has a high HBV DNA viral load, is associated with a high risk of transmission and subsequent infection in susceptible people. At the upper extreme of probability, one out of every three exposures may result in transmission. Percutaneous exposure to HCV is associated with a higher risk of infection than HIV exposure but significantly less than HBV exposure, with approximately one out of every 30 HCV exposures resulting in infection (PHLS 1999).

Mucocutaneous exposures, that is, where the eye(s), inside of the nose or mouth or an area of non-intact skin is exposed, usually by splashing or spilling incidents, have been implicated in viral transmission in healthcare settings, but the risk of this happening is less well-defined: it is certainly less than the risk following percutaneous exposure. There is no evidence to indicate that contact with blood or body fluids with intact skin presents a risk of viral transmission. However, most nurses, midwives and other healthcare personnel may not have intact skin on their hands, especially in the area of their fingernail beds.

Standard principles for preventing exposure and infection

Accepting that all blood and body fluids from all individuals are always potentially infectious, and then consistently adhering to current evidence-based guidelines for preventing healthcare-associated infections (HAI), offers the best protection against exposure and subsequent infection (Garner and HICPAC 1996, Loveday *et al* 2001, Pratt *et al* 2001). Preventing needlestick and sharp injuries and other parenteral exposures, along with the judicious use of gloves for procedures where contact with blood, body fluids or mucous membranes is anticipated, is at the very core of universal infection prevention precautions. As these recommendations have evolved, they are now referred to in the UK as *Standard Principles for Preventing HAI* (Loveday *et al* 2001, Pratt *et al* 2001) and some of the salient recommendations focused on preventing exposure to bloodborne pathogens (BBP), including recommendations from the Health and Safety Commission (1999) are listed in Box 10.

Box 9. Probability of infection with percutaneous exposure to bloodborne pathogens

Virus	Risk of infection	Approximate probability
Hepatitis B virus (HbsAg-positive)	HbeAg-negative: 5% HbeAg-positive: 19-33%	(One in three)
Hepatitis C virus	1.8-3.3%	(One in 30)
Human immunodeficiency virus	0.31%	(One in 300)

TIME OUT 6

Identify any obstacles you may encounter in your own practice in consistently using standard infection prevention principles in all clinical situations with all patients all the time.



Management of occupational exposures to BBP

Immediately following any exposure incident, the site of the exposure should be washed liberally with soap and water but without scrubbing. There is no evidence that antiseptic/disinfectant skin preparations are more effective than soap and water in this situation; they are probably best avoided as their effect on local defences is unknown.

Free bleeding of the puncture wounds is 'gently' encouraged. Exposed mucous membranes, including conjunctivae, should be irrigated copiously with water, before and after removing any contact lenses (DoH 1998).

In addition to immediate first aid, medical advice should be immediately sought so that the need for any additional preventive measures can be assessed and serological testing of the source patient and baseline serology of the nurse considered. In general, source patients can only be tested with their informed consent.

Medical advice is usually accessed through the occupational health services or the A&E department. Every exposure incident needs to be reported to management, documented and investigated thoroughly.

Exposure incidents are psychologically traumatic for staff and counselling support needs to be available. This is frequently accessed from occupational health. Active and/or passive immunisation or post-exposure chemoprophylaxis (PEP) helps reduce the risk of infection and is available following exposure to HBV and HIV but not for HCV.

Hepatitis B All nurses, midwives and other health-care staff (including students) who work in NHS clinical areas where they may have direct contact with blood and other body fluids, who perform exposure-prone procedures (EPP) (Box 11) or who are at risk of injury from blood-stained sharp instruments, are required to be immunised against HBV infection (DoH 1996b). The vaccine is normally given intramuscularly in the deltoid region, but not in the buttock as this may reduce vaccine efficacy (DoH 1996a).

Antibody levels (titres) should be checked following vaccination and a booster is usually given every five years if antibody titres fall below 100miu/ml. Specific hepatitis B immunoglobulin (HBIG) may be used for passive protection in unvaccinated people or those who did not respond to vaccination. Currently, there are no specific anti-hepatitis B antiviral drugs that are recommended following exposure (PEP) to abort infection.

Hepatitis C Unfortunately, there is no vaccine or PEP available currently to prevent infection following exposure to HCV.

Box 10. Standard principles for preventing exposure to BBP**Hand hygiene**

- Hands must be decontaminated immediately before each and every episode of direct patient contact.
- Hands must be washed if they are visibly or potentially contaminated with dirt or organic material.
- Alcohol-based handrub may be used to decontaminate hands between caring for different patients and different caring activities for the same patient.
- Effective technique ensures thorough hand decontamination and protects skin integrity.

Gloves

- Gloves must be worn for invasive procedures, contact with sterile sites, and non-intact skin, mucous membranes, and all activities that have been assessed as carrying a risk of exposure to blood, body fluids, secretions and excretions, sharp or contaminated instruments.
- Gloves must only be worn once, for one aspect of care and one patient.
- Dispose of gloves as clinical waste and decontaminate your hands following their removal.

Aprons and gowns

- Disposable plastic aprons should be worn when there is a risk that clothing or uniform may become exposed to blood, body fluids, secretions and excretions, with the exception of sweat.
- Full body fluid-repellent gowns should be worn where there is a risk of extensive splashing of blood, body fluids, secretions and excretions, with the exception of sweat, on to the skin.

Face masks and eye protection

- Face masks and eye protection should be worn where there is a risk of blood, body fluids, secretions and excretions splashing into the face and eyes.

Sharps and needles

- Do not pass sharps from hand to hand and keep handling to a minimum.
- Do not bend or break needles, recap or disassemble needles and syringes by hand prior to disposal.
- Used sharps must be discarded at the point of use into a sharps' container (conforming to UN3291 and BS7320 Standards).

Box 11. Exposure-prone procedures (EPPs)

- EPPs are those where there is a risk that injury to the healthcare professional may result in the exposure of the patient's open tissues to the blood of the healthcare professional. These include procedures where the healthcare professional's gloved hands may be in contact with sharp instruments, needle tips and sharp tissues (spicules of bone or teeth) inside a patient's open body cavity, wound or confined anatomical space where the hands or fingertips may not be completely visible at all times.

- General nursing procedures do not include EPP but the duties of operating theatre nurses, nurses practising in A&E departments and haemodialysis units should be considered individually. In midwifery, simple vaginal delivery and the use of scissors to make an episiotomy cut are not exposure-prone. Infiltration of local anaesthetic prior to episiotomy, suturing of an episiotomy and attaching sharp scalp electrodes to a baby's head are considered exposure-prone.

(DoH 1998, 2002b)

Conclusion

In reviewing the information in this article and completing the Time Out activities, you should have acquired adequate information and insights to care



Further information

The internet sites below have links to related sites and host a variety of useful resources.

British Liver Trust and the Liver Nurses Forum: www.britishlivertrust.org.uk

The British Liver Trust publishes up-to-date information for patients and services, and encourages and funds liver disease research. It is also the internet home for the Liver Nurses Forum, which enables the exchange of information and promotes nurse-led liver disease research.

UK National Hepatitis C Resource Centre: www.hep-ccentre.com

This Department of Health-supported site provides information for people living with hepatitis C, healthcare professionals, and the general public. It also provides a peer perspective on personal experiences of HCV-positive individuals regarding day-to-day living, treatment, alternative therapies and support.

The Centers for Disease Control and Prevention:

<http://www.cdc.gov/ncidod/diseases/hepatitis/index.htm>

The CDC provides user-friendly, accurate, up-to-date information on acute and chronic viral hepatitis.

American Liver Foundation: www.liverfoundation.org

This is dedicated to the prevention, treatment and cure of hepatitis and other liver disease through education, research and advocacy. This is a huge website and has useful links to other related sites.

UK Public Health Laboratory Service:

http://www.phls.co.uk/topics_az/hepatitis_a/guidelines.htm

A comprehensive review of the evidence on control measures for preventing hepatitis A virus infection has been published by the UK Public Health Laboratory Service.

effectively and safely for patients with viral hepatitis. When assessing, planning and evaluating nursing care, you will now have a better understanding of how changes in liver function resulting from infection with hepatotropic viruses are linked to patient problems. Being well informed on how these viruses are transmitted will help you effectively engage in patient education encounters focused on primary prevention. A better understanding of how the potential for viral exposure and transmission can occur during healthcare interventions, and universally incorporating standard principles for preventing exposure and infection into your everyday routine clinical practice, will allow you to care more safely for patients with infections ■

TIME OUT 7

Now that you have completed the article, you might like to think about writing a practice profile. Guidelines to help you are on page 55.



REFERENCES

- Beers M, Berkow R (1999) Hepatic and biliary disorders. In *The Merck Manual of Diagnosis and Therapy*. 17th edition. Whitehouse Station NJ, Merck Research Laboratory.
- Brook M (2001) European guideline for the management of hepatitis B and C virus infections. *International Journal of STD & AIDS*. 12, Suppl 3, 48-57.
- Burns S (2002) Picornaviruses. In Greenwood D *et al* (Eds) *Medical Microbiology*. 16th edition. Edinburgh, Churchill Livingstone.
- Centers for Disease Control and Prevention (1999) USPHS/IDSA guidelines for the prevention of opportunistic infections in persons infected with human immunodeficiency virus: US Public Health Service (USPHS) and Infectious Diseases Society of America (IDSA). *Morbidity and Mortality Weekly Report*. 48, RR-10, 1-66.
- Centers for Disease Control and Prevention (1998) Recommendations for prevention and control of hepatitis C virus (HCV) infection and HCV-related chronic disease. *Morbidity and Mortality Weekly Report*. 47, RR-19, 1-39.
- Collier L, Oxford J (2000). *Human Virology*. Second edition. Oxford, Oxford University Press.
- Crowcroft N *et al* (2001) Guidelines for the control of hepatitis A virus infection. *Communicable Disease and Public Health*. 4, 3, 213-227.
- Department of Health (2002a) *Hepatitis C Strategy for England*. London, Stationery Office.
- Department of Health (2002b) *HIV Infected Health Care Workers: A Consultation Paper on Management and Patient Notification*. London, Stationery Office.
- Department of Health (1998) *Guidance for Clinical Health Care Workers: Protection Against Infection with Blood-borne Viruses. Recommendations of the Expert Advisory Group on AIDS and the Advisory Group on Hepatitis*. London, Stationery Office.
- Department of Health (1996a) *Immunisation against Infectious Disease*. London, Stationery Office.
- Department of Health (1996b) *Protecting Health Care Workers and Patients from Hepatitis B: Recommendations of the Advisory Group on Hepatitis*. HSG(93)40 1993 and Addendum issued under cover of EL(96)77. London, Stationery Office.
- Dienstag J *et al* (1991) Acute hepatitis. In Wilson J *et al* (Eds) *Harrison's Principles of Internal Medicine*. 12th edition. International Edition. London, McGraw-Hill.
- Finlayson N *et al* (1999) Diseases of the liver and biliary system. In Haslett C *et al* (Eds) *Davidson's Principles and Practice of Medicine*. 18th edition. Edinburgh, Churchill Livingstone.
- Edinburgh, Churchill Livingstone.
- Garner J, Hospital Infection Control Practices Advisory Committee (1996) Guideline for isolation precautions in hospitals. *Infection Control and Hospital Epidemiology*. 17, 53-80.
- Health and Safety Commission (1999) *Control of Substances Hazardous to Health Regulations 1999: Approved Codes of Practice*. London, HSE Books.
- Loveday H *et al* (2001) The epic project: developing national evidence-based guidelines for preventing healthcare associated infections. Standard principles for preventing hospital-acquired infections. *Nursing Times*. 97, 13, 36-37.
- Mehta D (Ed) (2002) *British National Formulary No. 44*. London, British Medical Association and the Royal Pharmaceutical Society of Great Britain.
- Morgan-Capner P, Simmonds P (2002) *Togavirus and hepacivirus*. In Greenwood D *et al* (Eds) *Medical Microbiology*. 16th edition. Edinburgh, Churchill Livingstone.
- National Institute for Clinical Excellence (2000) *Guidance on the Use of Ribavirin and Interferon Alpha for Hepatitis C*. www.nice.org.uk/article.asp?a=11676. (Last accessed February 12 2003.)
- NHS Executive (1998) *Screening of Pregnant Women for Hepatitis B and Immunisation of Babies at Risk*. Health Service Circular HSC 1998/127. London, Department of Health.
- Pratt R *et al* (2001) The epic project: developing national evidence-based guidelines for preventing healthcare-associated infections. Phase 1: Guidelines for preventing hospital-acquired infections. *Journal of Hospital Infection*. 47, Suppl, S1-S82.
- Public Health Laboratory Service AIDS & STD Centre (1999) *Occupational Transmission of HIV*. London, PHLS.
- Ramsay M (1999) Guidance on the investigation and management of occupational exposure to hepatitis C. *Communicable Disease and Public Health*. 2, 4, 258-262.
- Roure C (1995) Overview of epidemiology and disease burden of hepatitis B in the European region. *Vaccine*. 13, Suppl 1, S18-S21.
- Thomas S *et al* (1998) A review of hepatitis C virus vertical transmission to infants born to mothers with and without HCV viraemia or HIV. *Internal Journal of Epidemiology*. 27, 1, 108-117.
- UK Blood Transfusion Services (2002). *Guidelines for the Blood Transfusion Service in the United Kingdom*. Sixth edition. London, Stationery Office.
- Van Damme P *et al* (1995) Hepatitis B prevention in Europe: a preliminary economic evaluation. *Vaccine*. 13, Suppl 1, S54-S57.