ABC of Sexually Transmitted Diseases

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VIRAL HEPATITIS

Causes

Hepatitis A	4-6 weeks' incubation Faecal-oral, sexual (homosexual) transmission
Hepatitis B	12 weeks' incubation Parenteral or percutaneous, perinatal, sexual (homosexual and heterosexual) transmission Carrier state
Non-A non-B	(a) 5 weeks' incubation faecal-oral (epidemic) transmission (b) 8 weeks' incubation Parenteral or percutaneous transmission (? sexual) Carrier state

The three main viral causes of hepatitis are hepatitis A, hepatitis B, and non-A non-B virus(es). Cytomegalovirus and Epstein-Barr virus occasionally cause hepatitis, and the recently discovered delta agent (δ) may cause a superimposed hepatitis in a carrier of hepatitis B.

Hepatitis A is caused by a small RNA virus, which is excreted in the stools for up to two weeks before the onset of symptoms. With increasing standards of hygiene in the developed world an older section of the population becomes susceptible. Fewer than a fifth of young heterosexual adults in London are immune. As a sexually transmitted infection hepatitis A occurs mainly among homosexual men because of oral-anal contact. Recovery is the rule after hepatitis A.

Hepatitis B virus is a DNA virus. It is usually present in serum many weeks before the acute illness. In populations with a high carrier rate—for example, tropical Africa and South East Asia, where 8-20% of people carry the virus—the whole population is at risk of infection. Much infection occurs perinatally or during early childhood. In populations with a low carrier rate, such as the United Kingdom, where about 1 in 1000 of the population carry the virus, only certain groups are at high risk of infection. As a sexually transmitted disease hepatitis B in the United Kingdom occurs largely in homosexual men. Risk factors are multiple casual sexual partners, anal intercourse, and a high carrier rate (about 5% in those attending departments of genitourinary medicine).

The δ agent is a recently discovered defective RNA virus which needs hepatitis B virus to produce infection. It occurs largely as a superinfection in carriers of the B virus and may cause a transient infection, severe acute or fulminant hepatitis, or worsened chronic liver disease.

Non-A non-B virus(es) emerged as important causes of hepatitis once serological tests were available to identify A and B viruses, cytomegalovirus, and Epstein-Barr virus. There may be two agents transmitted via percutaneous routes, blood, and blood products. In developed countries the groups at risk are similar to those at risk of hepatitis B. A carrier state exists and is more common. Non-A non-B viruses account for 15-30% of cases of sporadic acute hepatitis. In 25-30% of patients there is no history of parenteral exposure and limited observations suggest occasional transmission by person to person and sexual contact. In homosexual men, however, the risks of hepatitis A and B are much higher. Chronicity after sporadic non-A non-B virus infection is unusual. A non-A non-B hepatitis spread by the oral-faecal route has caused epidemics in India and Asia with an incubation period similar to that of hepatitis A and no carrier state.

Hepatitis B groups at risk

- Endemic areas—whole population at early age
- Neonates of carrier mothers
- Homosexual men
- Prostitutes
- Intravenous drug abusers
- Sexual contact(s) of carriers
- Certain patients, laboratory and medical staff exposed to blood or blood products or in closed institutions

Diagnosis

Serological diagnosis of acute non-A non-B hepatitis

Exclude alcohol and drugs etc

HBsAg	negative
IgM anti-HBc	negative
IgM anti-HAV	negative
IgM anti-EBV	negative
IgM anti-CMV	negative

History and examination—It is important to inquire about sexual orientation and contacts and whether the partner has symptoms of hepatitis. Most cases of acute hepatitis diagnosed in departments of genitourinary medicine occur in homosexual men and are due to A or B viruses. A history of travel, intravenous drug abuse, tattoos, recent transfusion, or percutaneous exposure may also be relevant. Hepatotoxins such as alcohol and drugs should also be excluded as a cause of hepatitis. There are no major differences in the clinical features of acute hepatitis due to A, B, or non-A non-B viruses. More than half of acute infections are subclinical. The clinical illness begins with non-specific symptoms such as fever, headache, and fatigue, and jaundice follows. Symptoms and signs of other concurrent sexually transmitted diseases should be looked for.

Laboratory tests—Routine liver function tests are unhelpful in identifying the responsible virus; they may help to distinguish between hepatitis and cholestasis due to extrahepatic or intrahepatic lesions, but prolonged cholestasis does occur with viral hepatitis. Tests of synthetic function such as prothrombin time and serum albumin are useful in determining the severity of the hepatitis.

Specific serological tests—Hepatitis A, Epstein-Barr virus, and cytomegalovirus infections are best diagnosed by tests for specific serum IgM antibodies, which persist for a short time and indicate recent infection. IgG antibodies appear more slowly, persist for many years, and indicate immunity. Early in acute hepatitis B, hepatitis B surface (HBsAg) and hepatitis B e antigens (HBeAg) appear in the serum. In a minority HBsAg may disappear before the onset of clinical symptoms. Anti-HBc appears during the acute illness followed by anti-HBe. Neither of these antibodies provide immunity. Anti-HBs, which provides long term immunity, appears after HBsAg has disappeared, though there may be a long gap. In this serological gap anti-HBc will be the only evidence of hepatitis B infection and IgM anti-HBc evidence of recent infection. In a patient with acute hepatitis detection of HBsAg will usually indicate acute B virus infection but in a few cases may represent some other cause of hepatitis in a B virus carrier. There are no specific serological tests for non-A non-B viruses and the diagnosis is one of exclusion. A few major centres have assays for antibodies to δ antigen to evaluate superinfection in a B virus carrier.

In homosexual men it may be necessary to take specimens for microscopy and culture from exposed sites such as the throat, urethra, and rectum to exclude other sexually transmitted diseases. Finally, serological tests for syphilis must be performed.

Complications

Fulminant hepatitis is a rare complication occurring in less than 1 in every 100 cases of acute viral hepatitis. Some 5-10% of patients with acute hepatitis B develop a chronic infection (HBsAg persists in serum for more than 6 months). Early in the chronic carrier state the complete virion, and therefore HBsAg and HBeAg, are present in the serum. These carriers are of high infectivity (supercarriers). Liver biopsy is the most reliable way of assessing whether the chronic carrier has a normal liver, chronic persistent hepatitis, chronic active hepatitis, or cirrhosis.

Acute to chronic hepatitis B

Chronic persistent hepatitis is usually benign. Some patients do, however, progress to chronic active hepatitis and cirrhosis, particularly if HBeAg remains in their serum. Longstanding chronic hepatitis B infection and the development of cirrhosis are associated with a high risk of primary hepatocellular carcinoma.

Management

Indications for admission to hospital

Complications

Symptoms and signs of acute liver failure (earliest=neuropsychiatric)

Doubts about diagnosis

? extrahepatic cause for jaundice

Social factors

young adult living alone

Acute viral hepatitis is usually self limiting and management largely supportive. Most patients can be managed as outpatients, but acute liver failure and its complications demand urgent admission to intensive care.

In uncomplicated cases a low fat, high energy diet is more palatable and bed rest advisable during the early phase of the illness. Alcohol is excluded at least until liver function tests are normal.

In chronic hepatitis management is also largely supportive with antiviral therapy still experimental. Carriers with anti-HBe and normal biochemical and histological findings should be reassured. Alcohol even in moderation may hasten the progression of chronic liver disease.

Prevention

Prevention of hepatitis as a sexually transmitted disease

Contact tracing

Counselling of HBeAg positive carriers

Passive immunisation

Hepatitis A—normal pooled immunoglobulin

Hepatitis B—Hepatitis B immunoglobulin (HBIG)

Active immunisation

—vaccine

Because of the lack of specific treatment prevention has been the major aim in managing viral hepatitis, especially hepatitis B. Simple hygienic precautions, in the case of hepatitis A, and careful handling of blood, blood contaminated material, and instruments, in the case of hepatitis B, will significantly decrease the transmission from patients.

Regular sexual contacts of patients with acute hepatitis A or B should be traced and offered passive immunisation. Ideally all those who have had sexual contact with a patient with acute hepatitis B in the preceding three months should be traced. Unidentified HBeAg positive carriers may then be found as a source of infection. These patients should be counselled about their infectivity and reduction in number of casual partners, and their non-immune regular sexual contacts should be vaccinated.

Hepatitis B vaccine—Purified, inactivated, alum conjugated 22 nm HBsAg particle vaccines are now available for high risk groups. They are safe and protect over 90% of homosexual vaccinees for at least five years. The groups at risk are priority groups for vaccination. Savings can be made by pretesting for anti-HBs in those with high attack rates and pre-existing immunity such as homosexuals and not doing so in those with low attack rates such as certain medical staff.

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