

A Review about Hepatitis B Virus: Transmission, Pathogenesis, Diagnosis, and Prevention

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Abstract
Hepatitis Liver inflammation can result from several causes, including viruses such as hepatitis B (HBV). It remains a serious global health concern: infection may become chronic andraise the risk of cirrhosis andliver cancer. HBV spreads through infected blood, sexual contact, or transmission from mother to child during childbirth. Vaccination, early diagnosis, andbasic precautions can help limit its spread. The World Health Organization aims to eliminate HBV as a health threat by 2030. The study stressed vaccination, safety measures, andeducation, particularly for healthcare workers, andoffered recommendations for additional research andprevention efforts.

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1. Introduction

Hepatitis is Hepatitis means inflammation of the liver, marked by the presence of inflammatory cells in liver tissue. It may result from infectious or non-infectious causes, including viruses, bacteria, toxins, drugs, andalcohol use. When a virus causes the inflammation, the condition is called viral hepatitis. Most cases come from one of five unrelated hepatotropic viruses: hepatitis A, hepatitis B, hepatitis C, hepatitis D, or hepatitis E. Other viruses can inflame the liver as well, including herpes simplex virus, cytomegalovirus (CMV), Epstein-Barr virus, yellow fever virus, coxsackie viruses, andadenovirus, among others [1]. Hepatitis B is a potentially life-threatening liver infection caused by hepatitis B virus (HBV), an enveloped deoxygenated ribonucleic acid (DNA) virus. Hepatitis B remains a major global health problem. It can persist as a chronic infection, raising the risk of cirrhosis andliver cancer, while its clinical course may be silent or mild, severe, or, rarely, fulminant hepatitis [2]. Hepatitis B virus (HBV) infection is the world's most common chronic viral infection. About 2 billion people are estimated to have been infected, andmore than 350 million carry the virus chronically. The Global Burden of Disease study ranked HBV infection among the world's leading health priorities andidentified it as the tenth leading cause of death, with 786 000 deaths each year. Those figures prompted WHO to add viral hepatitis to its major public health priorities [3]. The World Health Organization has pledged to eliminate HBV as a health threat by 2030. At present, however, no country is on track to meet that target [4]. Nurses who contract Hepatitis B after entering the hospital face serious health risks. Their infection also creates a risk that the virus could spread to patients or other healthcare workers. The central concern is occupational exposure, which makes effective prevention necessary to protect nurses andlimit transmission within healthcare facilities. This study aims to assess how much exposure nurses face in their work environments andidentify the factors that increase their likelihood of infection.

1.1. Background

The World Health Organization estimates that about 240 million people worldwide are infected with hepatitis B virus (HBV), andthat the infection causes roughly 780 thousanddeaths each year [5]. Among those patients, 50% may develop chronic liver disease, while 10% may develop liver fibrosis, which can lead to liver cancer [6].
In 1967, Krugman andhis colleagues established that hepatitis existed in at least two forms. One, later called serum hepatitis, spread through parenteral transmission. Independently, they identified an antigen known as the SH antigen in the blood of

patients with transfusion-associated hepatitis during the disease's incubation period. Further research showed that the Au andSH antigens were identical; both represented the hepatitis B surface antigen (HBsAg.) [7]

In 1970, Dane andhis colleagues identified the viral particle through electron microscopy; it became known as the Dane particle. By the early 1980s, researchers had sequenced the viral genome andwere testing improved versions of the first vaccine. The American Food andDrug Administration approved that vaccine, which was called an anti-cancer vaccine because it helped prevent primary liver cancer caused by HBV [8]

Lurman (1885) wrote that researchers began distinguishing among the different forms of acute hepatitis decades after World War 11, drawing on several clinical andepidemiological studies.

Chronic hepatitis B follows four recognized phases, although they do not always occur in sequence or appear in every patient, as shown in Figure (2-1). During the first phase—immune tolerance—HBeAg ("e" antigen) remains reactive, serum HBV deoxyribonucleic acid (HBV-DNA) levels are high, ALT is normal or only slightly elevated, andhistological findings are usually absent.

Signs of fibrosis andinflammation can appear. During the second phase, immune clearance, the body tries to remove the virus; if the infection persists, the chances of cirrhosis andhepatocellular carcinoma (HCC) rise [7]

Liver inflammation andelevated ALT occur alongside a modest drop in HBV-DNA levels, eventually leading to HbeAg loss andseroconversion into anti-

HBe. The next stage is the inactive-carrier phase. Disease activity remits, the prognosis generally improves, andhistology usually shows improvement as well. ALT returns to normal, while HBV-DNA falls below 10 thousandcopies/mL or 2,000 UI/mL, or becomes undetectable, as shown in Figure (2-2). Reactivation can occur later, either spontaneously or after immunosuppression. When it does, liver damage may progress, accompanied by renewed rises in ALT andHBV-DNA. Recognizing these phases can help predict liver damage, although applying them in practice remains difficult because the infection changes over time [5]

Once researchers realized that HBsAg could trigger production of anti-HBs—andthat this antibody protected against HBV infection—they began developing prototype hepatitis B vaccines. Production andlicensing expanded across several countries during the 1980s. By 1990, a worldwide campaign was under way to stop HBV transmission andreduce HBV-related chronic liver disease andPHC through routine vaccination of all infants [8].

1.2. Virus Structure

Double-stranded relaxed-circular DNA genome covalently bound to the viral polymerase. The outer envelope contains embedded proteins which are involved in viral binding. In general, Virion is spherical, but polymorphic forms, including filamentous forms, are also present. The DNA genome, comprising long andshort segments that converge to form a closed loop, is partly double-stranded andnon-segmented. The longer strandconsists of long nucleotides 3020-3320 while the shorter strandis long nucleotides 700-2800 The infectious portion of the virus is the viral core, while the outer coat carries the virus' main antigenic determinants, HBs Ag [9], as shown in figure (1)

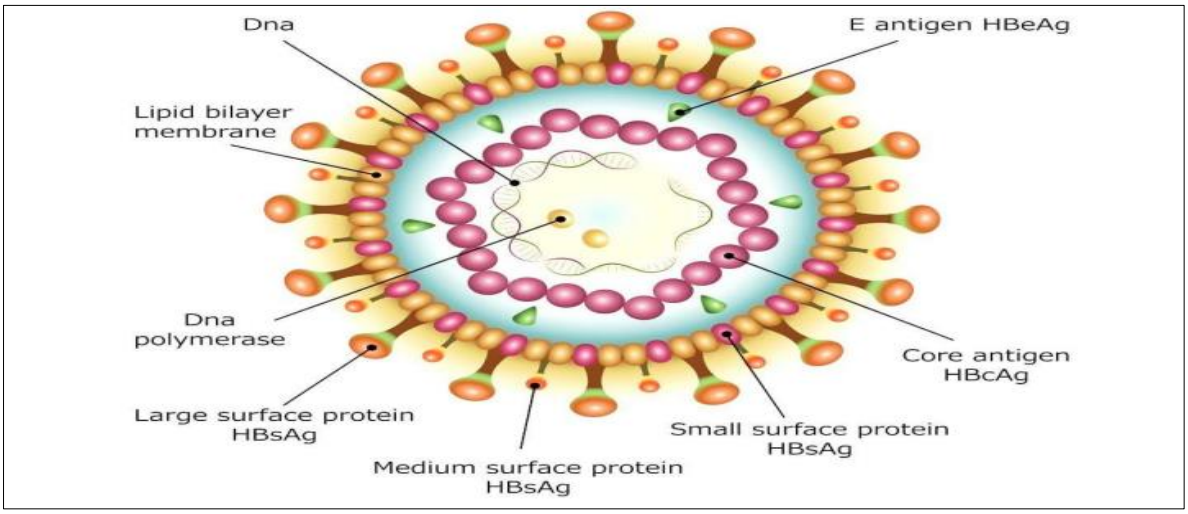


Fig 1: A simplified drawing of the HBV particle andsurface antigen

1.3. Epidemiology

The prevalence of chronic HBV infection varies greatly in different part of the world. The prevalence of chronic HBV infection worldwide could be categorized as high, intermediate andlow endemicity [10]. The age at the time of infection is associated with the endemicity of HBV infection **[11]** as see in table 1.

Table 1: Characteristics of endemic patterns of hepatitis B virus infection. Source: adapted from

Characteristic	Endemicity of infection		
	Low (%)	Intermediate (%)	High (%)
Chronic infection prevalence	0.5-2	2-7	≥8
Past infection prevalence	5-7	10-60	70-95
Perinatal infection	Rare	Uncommon	Common
	<10	10-60	>20
Early childhood infection	Rare	Common	Very common
	<10	10-60	>60
Adolescent/adult infection	Very common	Common	Uncommon
	70-90	20-50	10-20

1.4. Hepatitis B Virus Transmission

HBV is spread through contact with infected body fluids andthe only natural host is human. Blood is the most important vehicle for transmission, but other body fluids have also been implicated, including semen andsaliva. Currently, three modes of HBV transmission have been recognized: perinatal, sexual andparenteral/percutaneous transmission. There is no reliable evidence that airborne infections occur andfeces are not a source of infection. HBV is not transmitted by contaminated food or water, insects or other vectors [12].

1.5. Signs andSymptoms

The pathogenesis andclinical manifestations of hepatitis B are due to the interaction of the virus andthe host immune system, which leads to liver injury and, potentially, cirrhosis andhepatocellular carcinoma. Patients can have either an acute symptomatic disease or an asymptomatic disease. Icteric hepatitis is associated with a prodromal period, during which a serum sickness–like syndrome can occur. The symptomatology is more constitutional andincludes the following [13]: Anorexia ,Nausea ,Vomiting ,Low-grade fever ,Myalgia ,Fatigability ,Disordered gustatory acuity andsmell sensations (aversion to food andcigarettes) ,Right upper quadrant andepigastric pain (intermittent, mild to moderate) ,Patients with fulminant andsub fulminant hepatitis may present with the following [7]: Hepatic encephalopathy ,Somnolence ,Disturbances in sleep pattern ,Mental confusion ,Coma ,Ascites ,Gastrointestinal bleeding ,Coagulopathy .Patients with chronic hepatitis B infection can be immune tolerant or have an inactive chronic infection without any evidence of active disease, andthey are also asymptomatic. Patients with chronic active hepatitis, especially during the replicative state, may have symptoms similar to those of acute hepatitis [12].Inadequate adherence to standard precautions, such as handhygiene, personal protective equipment (PPE) use, andsafe injection practices, can increase the risk of HBV transmission.

1.6. Laboratory Diagnosis andManagement

Diagnosis is based on a combination of clinical andlaboratory findings. Because many persons infected with Hepatitis B have no symptoms or present with symptoms similar to many other diseases, definitive diagnosis is dependent on laboratory tests. Additionally, due to the fact that there are several types of viral Hepatitis, a blood test is needed to confirm which type of Hepatitis is present. Initial assessment of HBV infection begins with patient history, physical examination, evaluation of liver disease activity, andinterpretation of different hepatitis markers and/or their combinations such as HBsAg, HB core antigen (HBcAg), HBeAg, HB surface antibody (anti-HBs/HBsAb), HB core antibody (anti-HBc), antiHBc IgM, HB e antibody (anti-HBe), andfocus on the detection of antigens andantibodies [14].

The Hepatitis B Foundation (HBF) recommends screening all adults for HB with the triple serological marker panel that involves HBsAg, anti-HBs, andanti-HBc total. To classify the phases of the infection in HBV infected patients, the followings should be performed [15]:

a. the assays for HBsAg, HBeAg/anti-HBe, HBV DNA.
b. liver blood tests including aspartate aminotransferase (AST), alanine transaminase (ALT).
c. transient elastography (Fibroscan) as a noninvasive test or needle liver biopsy as an invasive method for the presence of cirrhosis.

All patients with HIV infection should be screened for HBV infection. The optimal screening strategy has not yet been defined but is usually based on the determination of HBsAg andanti-HBs. In the case that both markers are negative, an active hepatitis B vaccine should be administered. Vaccination is safe in HIV infected patients, but a higher proportion of these patients may not develop protective immunity (30% primary non-response to HBV vaccine in HIV infected individuals versus 2.5% in healthy individuals) or are more likely to lose their immunity over time. Testing for HBsAg andanti-HBs, however, will not identify patients with occult HBV infection who are typically serologically positive for anti-HBc only. In this situation anti-HBc andserum HBV DNA should be determined [16].

1.7. Treatment

Acute hepatitis B infection does not usually required treatment because most adults clear the infection spontaneously [17]. Early antiviral treatment may only be required in less than 1% of patients whose infection takes aggressive course (fulminant hepatitis) or who are immune compromise.

Chronically infected individuals with persistently elevated serum alanine aminotransferase andHBV- DNA levels are candidates for therapy. As of 2008, there are five antiviral medications andtwo immune-modulators licensed for the treatment of hepatitis B in the USA. This includes lamivudine (Epivir), adefovirs (Heps era), tenofovir (viread), telbivudine (Tyzeka), interferon alpha-2a andPegylated interferon alpha-2a (pegasys). Although none of these available drugs can clear the infection, they can stop the virus from replicating andminimizes liver damage such as cirrhosis andcancer [18].

Infant born to mothers known to carry hepatitis B can be treated with antibodies to HBV called hepatitis B immune globulin (HBIg) andwhen given within twelve hours of birth in combination with HBV vaccine, the risk of infection acquisition is reduced to 95% andthis allows the mother to safety breastfed her child [19].

1.8. Prevention andControl

HBV infection can be prevented by avoiding transmission from infected people andby inducing immunity in unexposed people. Screening of blood donors for HBsAg andimplementation of universal precautions resulted in a substantial reduction in transmission in health-care settings. The addition of HBV DNA testing to screening processes further decreases the incidence of transfusion-associated disease, but implementation is hampered by incremental cost.10 Counselling infected people to prevent [3].

Booster immunisations may also be required to prevent infection in late childhood andin young adults." Current recommendations by the World Health Organisation andthe Centers for Disease Control are for the universal immunisation of infants in all geographical areas, irrespective of endemicity. It is unfortunate that, despite the availability of safe andincreasingly cheaper vaccines there IS still widespread apathy andeven reluctance to implement these recommendations for non-economic factors. for example, despite the availability of safe andeffective vaccines for over a decade, the incidence of hepatitis B infection in the USA has increased by one-third with a postulated 300 000 new infections annually [20].

In developing countries, particularly in Africa, economic factors andpopulation numbers probably preclude the implementation of effective mass immunisation against HBV at current HBV vaccine prices. It is hoped that the anticipated redistribution of medical resources andthe greater emphasis on primary health care [20].

2. Conclusion

There are many factors that could cause andincrease for the infection of hepatitis B virus . Recommendation ,Encourage individuals to receive the Hepatitis B vaccine to prevent infection, especially those working in healthcare centers andhospitals ,Adhere to health precautions such as wearing masks andgloves, avoiding direct contact with infected individuals, andnot using contaminated or unsterilized tools. Also Raise awareness among the population about the importance of vaccination andprevention of Hepatitis B virus infection. Implement regular screening programs for Hepatitis B to detect andtreat infections early.

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