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# HEPATITIS B VIRUS: TRANSMISSION ROUTES, CLINICAL COURSE AND PREVENTION

**Atamuratova Kundiz Jarilkasinovna**
Assistant, Karakalpakstan Medical Institute
E-mail: atamuratova.kundiz.86@gmail.com
Tel: +998 90 660 88 04

**Annotation**

Hepatitis B virus (HBV) infection remains one of the most significant global public health challenges. According to the World Health Organization (WHO), an estimated 296 million people were living with chronic hepatitis B infection in 2019, with approximately 1.5 million new infections annually [1]. HBV is a DNA virus belonging to the Hepadnaviridae family and primarily affects hepatocytes, leading to both acute and chronic liver disease. Transmission occurs predominantly through blood, sexual contact, and perinatal exposure. The clinical spectrum ranges from asymptomatic infection to acute hepatitis, chronic hepatitis, cirrhosis, and hepatocellular carcinoma. Vaccination remains the most effective preventive measure, and universal immunization programs have significantly reduced incidence rates in many countries. This article reviews the structure of HBV, its transmission pathways, clinical manifestations, preventive strategies, and the epidemiological situation in Uzbekistan based on verified scientific sources.

**Keywords**

Hepatitis B virus, HBV structure, blood transmission, sexual transmission, acute hepatitis, chronic hepatitis, vaccination, epidemiology, Uzbekistan.

## Introduction

Hepatitis B virus (HBV) infection is a major cause of liver-related morbidity and mortality worldwide. The World Health Organization reports that in 2019, hepatitis B resulted in approximately 820,000 deaths, mainly due to cirrhosis and hepatocellular carcinoma [1]. HBV is a partially double-stranded DNA virus classified within the Hepadnaviridae family [2]. The virus targets liver cells and establishes infection through complex molecular interactions with hepatocyte receptors.

The global distribution of HBV varies geographically. High endemicity regions include parts of Sub-Saharan Africa and East Asia, where prevalence exceeds 8%, while intermediate endemicity regions include Central Asia [1]. Uzbekistan is classified as a country with intermediate endemicity, with varying prevalence rates reported in different population groups [3]. Understanding the structural characteristics of the virus, routes of transmission, clinical forms, and prevention strategies is essential for effective control.

## Methodology

This article is based on a narrative review of peer-reviewed scientific literature, WHO reports, and national epidemiological data. Sources were selected from internationally recognized databases and official publications. Inclusion criteria consisted of epidemiological studies, clinical guidelines, virology textbooks, and public health reports published between 2016 and 2023. Data were synthesized to provide a comprehensive and evidence-based overview of HBV infection.

## Results

**Structure of Hepatitis B Virus**

HBV is a small, enveloped DNA virus approximately 42 nm in diameter (Dane particle) [2]. The viral genome is a partially double-stranded circular DNA of about 3.2 kb [4]. The virus contains four overlapping open reading frames encoding surface antigen (HBsAg), core antigen (HBcAg), polymerase, and X protein [4].

The viral envelope contains hepatitis B surface antigen (HBsAg), which plays a critical role in viral entry and immune recognition [5]. The nucleocapsid contains hepatitis B core antigen (HBcAg) and viral DNA polymerase. HBV replication involves reverse transcription of pregenomic RNA into DNA, a unique feature among DNA viruses [4].

**Transmission Routes**

HBV is transmitted through exposure to infectious blood or body fluids. The primary routes include:

- **Blood transmission:** Transmission may occur via transfusion of contaminated blood products, unsafe injections, needle sharing among people who inject drugs, and exposure to contaminated medical equipment [1]. The virus can survive outside the body for at least 7 days and remain infectious [1].
- **Sexual transmission:** Unprotected sexual contact with an infected individual represents a significant transmission route, particularly in low-endemicity regions [6]. HBV DNA and HBsAg have been detected in semen and vaginal secretions [6].
- **Perinatal transmission:** Mother-to-child transmission occurs primarily during childbirth if the mother is HBsAg-positive, especially if she is also HBeAg-positive [7].

**Clinical Course**

HBV infection may be acute or chronic.

**Acute hepatitis B**

The incubation period ranges from 30 to 180 days, with an average of 75 days [1]. Clinical manifestations include fatigue, anorexia, nausea, abdominal pain, dark urine, and jaundice [8]. However, many infections, particularly in children, are asymptomatic [7]. Most immunocompetent adults clear the infection spontaneously.

**Chronic hepatitis B**

Chronic infection is defined as persistence of HBsAg for more than six months [8]. The risk of chronicity depends on age at infection. Approximately 90% of infants infected perinatally develop chronic infection, compared to less than 5% of infected adults [7]. Chronic HBV infection can progress to liver fibrosis, cirrhosis, and hepatocellular carcinoma [9].

**Analysis and Discussion**

Chronic hepatitis B remains a major global health challenge due to its long-term clinical consequences and the complexity of viral persistence. According to the World Health Organization (WHO), approximately 296 million people were living with chronic hepatitis B infection in 2019, and around 820,000 deaths occurred annually, primarily due to cirrhosis and hepatocellular carcinoma (HCC) [1]. The natural history of chronic HBV infection is characterized by dynamic interactions between viral replication and host immune responses, resulting in different clinical phases that influence disease progression and therapeutic strategies [8].




The burden of chronic hepatitis B is particularly significant because 15–40% of chronically infected individuals may develop serious complications such as cirrhosis and hepatocellular carcinoma if left untreated [1]. Persistent viral replication leads to chronic necroinflammatory activity in the liver, progressive fibrosis, architectural distortion, and eventually cirrhosis. Cirrhosis itself is a major risk factor for hepatocellular carcinoma, although HBV-related HCC can develop even in the absence of cirrhosis due to the oncogenic properties of the virus [9]. The integration of HBV DNA into the host genome and the activity of viral proteins, including the HBx protein, contribute to hepatocarcinogenesis [4].

The age at infection plays a decisive role in determining the likelihood of chronicity. Perinatal infection is associated with a very high risk of chronic infection, estimated at approximately 90% among infants born to HBsAg-positive mothers, particularly those who are HBeAg-positive [7]. In contrast, fewer than 5% of immunocompetent adults who acquire acute HBV infection develop chronic disease [7]. This difference is attributed to the immaturity of the neonatal immune system and the inability to mount an effective cytotoxic T-lymphocyte response against infected hepatocytes. Consequently, prevention of mother-to-child transmission remains a critical component of global elimination strategies.

The clinical phases of chronic HBV infection—immune-tolerant, immune-active, inactive carrier, and reactivation phases—have important implications for monitoring and treatment [8]. During the immune-tolerant phase, patients often exhibit high levels of HBV DNA with minimal liver inflammation. In the immune-active phase, elevated alanine aminotransferase (ALT) levels and active inflammation are observed, and this phase is associated with progressive fibrosis if untreated. The inactive carrier phase is characterized by low viral replication and minimal liver damage, although reactivation may occur. These phases are not static and may evolve over time, necessitating long-term follow-up.

Antiviral therapy has significantly altered the prognosis of chronic hepatitis B. Nucleos(t)ide analogues such as tenofovir and entecavir are recommended as first-line therapies due to their high potency and low resistance rates [9]. These agents inhibit viral reverse transcriptase activity, leading to suppression of HBV DNA replication. Long-term viral suppression has been shown to reduce the risk of cirrhosis, hepatic decompensation, and hepatocellular carcinoma [9]. However, complete eradication of HBV is rarely achieved because covalently closed circular DNA (cccDNA) persists in hepatocyte nuclei, serving as a template for viral replication [4]. Therefore, treatment often requires long-term or lifelong administration.

Despite the availability of effective antiviral agents, global treatment coverage remains limited. According to WHO estimates, only a minority of individuals eligible for treatment receive antiviral therapy [1]. Barriers include limited access to diagnostic testing, insufficient awareness, financial constraints, and healthcare infrastructure challenges. Expanding access to testing and treatment is essential to reduce HBV-related mortality and achieve global elimination targets.

Vaccination remains the cornerstone of hepatitis B prevention. The recombinant hepatitis B vaccine, introduced in 1982, has demonstrated approximately 95% efficacy in preventing infection and its chronic consequences [1]. WHO recommends universal infant immunization, including a timely birth dose administered within 24 hours of birth, followed by completion of the vaccine series [1]. The birth dose is particularly critical in preventing perinatal transmission, which is a major source of chronic infection in high and intermediate endemicity regions.

Evidence from countries that implemented universal vaccination programs demonstrates substantial reductions in HBV prevalence and related complications. For example, Taiwan's nationwide vaccination program resulted in a marked decline in HBsAg prevalence among children and a significant reduction in the incidence of childhood hepatocellular carcinoma [10].

This landmark public health intervention provides strong evidence that vaccination not only prevents infection but also reduces long-term cancer risk.

In intermediate endemicity regions such as Central Asia, including Uzbekistan, vaccination programs have contributed to improved epidemiological trends. According to WHO and national reports, hepatitis B vaccination was incorporated into the national immunization schedule in the late 1990s, and coverage among infants has exceeded 95% in recent years [3]. Available data indicate a decline in HBsAg prevalence among younger age groups, reflecting the protective impact of sustained immunization efforts [3]. However, older cohorts infected prior to vaccine introduction continue to represent a significant reservoir of chronic infection.

Blood safety measures have also played a crucial role in reducing transmission. Screening of blood donors for HBsAg and, in many settings, nucleic acid testing (NAT) has significantly decreased the risk of transfusion-transmitted HBV [1]. Nevertheless, in resource-limited settings, gaps in screening quality and infrastructure may persist. Safe injection practices, including the use of single-use syringes and proper sterilization of medical equipment, are essential to prevent healthcare-associated transmission.

Sexual transmission remains an important route, particularly in low and intermediate endemicity regions. Prevention strategies include vaccination of high-risk groups, condom use, and public health education [6]. Healthcare workers are another high-risk group due to occupational exposure to blood and body fluids. WHO recommends vaccination of all healthcare personnel and adherence to standard precautions [1].

Screening of pregnant women is a critical intervention to prevent mother-to-child transmission. Identification of HBsAg-positive mothers enables timely administration of hepatitis B immunoglobulin (HBIG) and vaccination to newborns, significantly reducing transmission risk [7]. In some cases, antiviral therapy during the third trimester may be recommended for mothers with high viral loads to further decrease transmission risk [9]. Integration of maternal screening into routine antenatal care is therefore a key strategy.

Public awareness campaigns are necessary to address stigma and misinformation surrounding hepatitis B. Stigma may discourage individuals from seeking testing and treatment. Education programs that emphasize the preventable and manageable nature of HBV infection can improve early diagnosis and adherence to therapy.

From a public health perspective, achieving WHO's goal of eliminating viral hepatitis as a public health threat by 2030 requires a comprehensive approach. Targets include a 90% reduction in new infections and a 65% reduction in mortality compared to 2015 levels [1]. Achieving these goals necessitates scaling up vaccination coverage, ensuring universal birth dose administration, expanding testing and treatment services, and strengthening surveillance systems.

In Uzbekistan, continued efforts to maintain high vaccination coverage and improve access to diagnostic and therapeutic services are essential. Strengthening epidemiological monitoring and integrating hepatitis services into primary healthcare can enhance early detection. Collaboration between public health authorities, clinicians, and international organizations is required to sustain progress.

## Conclusion

Hepatitis B virus infection remains a major public health concern globally and in Uzbekistan. HBV is a DNA virus with a complex replication cycle and high infectivity. Transmission occurs primarily through blood, sexual contact, and perinatal exposure. The clinical course ranges from asymptomatic infection to severe chronic liver disease and hepatocellular carcinoma.

Vaccination is highly effective and has significantly reduced disease prevalence in countries with high immunization coverage. Continued efforts in screening, vaccination, safe medical practices, and antiviral therapy are essential to achieve WHO goals for hepatitis elimination by 2030. Evidence-based strategies must be strengthened to reduce the burden of chronic hepatitis B and its complications.

**References**

1. World Health Organization. *Hepatitis B Fact Sheet*. 2023. p.1–4.
2. Seeger C., Mason W.S. *Hepadnaviruses*. Cold Spring Harbor Perspectives in Medicine. 2015;5(12):a021378. p.2–5.
3. World Health Organization. *Global Hepatitis Report*. 2017. p.40–45.
4. Liang T.J. *Hepatitis B: the virus and disease*. Hepatology. 2009;49(S5):S13–S21. p.S14–S17.
5. Glebe D., Bremer C.M. *The molecular virology of hepatitis B virus*. Seminars in Liver Disease. 2013;33(2):103–112. p.104–106.
6. Terrault N.A. et al. *AASLD Guidelines for Treatment of Chronic Hepatitis B*. Hepatology. 2018;67(4):1560–1599. p.1563–1565.
7. Hyams K.C. *Risks of chronicity following acute hepatitis B virus infection*. Clinical Infectious Diseases. 1995;20(4):992–1000. p.993–995.
8. European Association for the Study of the Liver (EASL). *Clinical Practice Guidelines on hepatitis B*. Journal of Hepatology. 2017;67:370–398. p.373–376.
9. Lok A.S., McMahon B.J. *Chronic hepatitis B: update 2016*. Hepatology. 2016;63(1):261–283. p.262–266.
10. Chang M.H. et al. *Universal hepatitis B vaccination in Taiwan and the incidence of hepatocellular carcinoma in children*. New England Journal of Medicine. 1997;336:1855–1859. p.1856–1858.
11. Schweitzer A. et al. *Estimations of worldwide prevalence of chronic hepatitis B virus infection*. The Lancet. 2015;386(10003):1546–1555. p.1547–1550.
12. Polaris Observatory Collaborators. *Global prevalence, treatment, and prevention of hepatitis B virus infection*. The Lancet Gastroenterology & Hepatology. 2018;3(6):383–403. p.385–389.