

## HEPATITIS B: ETIOLOGY, EPIDEMIOLOGY, CLINICAL FEATURES, AND TREATMENT

*Turdiyev Tuymurod O'tkir o'g'li*

*Email: turdiyevtuumurod92@gmail.com*

*https://orcid.org/0009-0003-5191-7927*

*Bukhara State Medical Institute*

*named after Abu Ali Ibn Sino, Uzbekistan,*

*Bukhara city. A.Navoiy street 1*

*Tel: +998(65) 223-00-50 e-mail: mail.bsmi.uz*

Hepatitis B, caused by the Hepatitis B virus (HBV), is a contagious disease that leads to inflammation of the liver cells, often accompanied by jaundice and metabolic disorders. It frequently progresses to chronic hepatitis and can eventually cause liver cirrhosis. The virus, discovered in 1967, belongs to the Hepadnaviridae family. Its genome consists of two strands of DNA. The internal membrane is connected to an external membrane, which makes it highly resistant to external environments. It can survive for up to three months at room temperature. In the refrigerator, it can remain viable for six months, and when frozen, it can persist for up to 15-20 years.

### **Etiology and Virus Structure**

#### **Structure of Hepatitis B Virus**

Hepatitis B virus (HBV) belongs to the Hepadnaviridae family. The viral particle (virion) consists of an external lipid envelope and a protein core, forming an icosahedral nucleocapsid. The diameter of these virions ranges from 30 to 42 nm. The nucleocapsid contains viral DNA with reverse transcriptase activity and DNA polymerase. The external envelope incorporates embedded proteins that play a role in viral attachment and entry into susceptible cells. Hepatitis B virus is one of the smallest enveloped viruses in animals. The 42 nm virions, known as "Dane particles," are capable of infecting hepatocytes. In addition to the Dane particles, serum from infected individuals may contain non-infectious, nucleocapsid-free, filamentous and spherical structures. These particles are composed of lipids and proteins, which form part of the virion surface, and are referred to as surface antigens (HBs antigens). These surface antigens are produced in excess during the virus life cycle.

### **Epidemiology**

The spread of HBV is extensive. The infectious source includes individuals with various clinical forms of the disease and chronic carriers. The mechanisms of transmission are as follows:

1. Parenteral transmission.
2. Transmission through blood.

### Modes of Transmission

**Artificial Transmission:** Transmission can occur through manipulations that cause damage to the skin or mucous membranes, such as surgeries, injections, endoscopic examinations, surgical procedures, needle-stick injuries, manipulations, pedicure procedures, and tattoos. **Natural Transmission:** HBV can also be transmitted through broken skin layers. The transmission factors include:

1. Blood.
2. Plasma.
3. Erythrocytes.
4. Fibrinogen.
5. Prothrombin.

Additionally, transmission can occur through intimate contact, including shared household items, toothbrushes, razors, etc. The likelihood of contracting HBV infection is significantly higher than that of HIV infection—up to 100 times more likely. HBV predominantly affects individuals under the age of 12 and those over the age of 30. Immunity after infection is typically long-lasting.

### Clinical Presentation

The Hepatitis B virus (HBV) primarily affects the liver parenchyma, specifically hepatocytes.

1. **Jaundice (Icteric) form.**
2. **Non-jaundice (Anicteric) form.**
3. **Asymptomatic form:**
  - a) Subclinical.
  - b) Viral infection without clinical symptoms.

The severity of the disease can be classified as mild, moderate, or severe. The incubation period ranges from 40 to 180 days. The pre-jaundice phase typically lasts 1-2 weeks but may occasionally extend longer. The following clinical syndromes may present:

- **Arthralgic syndrome:** Often characteristic.
- **Dyspeptic syndrome:** Gastrointestinal symptoms such as nausea and loss of appetite.
- **Astenovegetative syndrome:** Fatigue, lethargy, dizziness, headaches, insomnia.
- **Fungal form:** Rare.
- **Mixed form:** A combination of various symptoms.
- **Occasionally asymptomatic:** A hidden or silent phase.

The disease often begins gradually. After 2-5 days following meals, symptoms like weakness, lethargy, loss of appetite, nausea, vomiting, skin discoloration, and rashes may occur. There may also be constipation or diarrhea. Patients may experience a feeling of heaviness and pain in the right hypochondrium.

- **Arthralgic syndrome:** Pain and discomfort in bones and joints.
- **Astenovegetative syndrome:** Generalized weakness, lethargy, dizziness, headaches, and insomnia.

At the end of the pre-jaundice phase, urine may turn a brownish color, while feces will first show pale coloration in the sclera and then later in the skin. The jaundice phase can last from 3 to 5 weeks or even longer, with symptoms of intoxication intensifying. Jaundice and other signs of liver dysfunction may also worsen. During this phase, sleep disturbances, euphoria, apathy, and irritability are common. Bradycardia, hypotension, and hepatomegaly (liver enlargement) or splenomegaly (enlarged spleen) are characteristic findings.

Blood tests typically show:

- Leukopenia (low white blood cell count).
- Lymphocytosis (increased lymphocytes).
- Increased ESR (erythrocyte sedimentation rate).

The chronic phase can last up to 12 months or more.

### Complications

1. **Acute hepatic encephalopathy** (liver coma).
2. **Ascitic syndrome** (fluid accumulation in the abdomen).

### Diagnosis

1. **Special tests** are used to detect specific markers in the serum.
2. **Biochemical tests** are performed to assess liver function.
3. **Detection of HBs antigens** is crucial in diagnosing HBV infection.

### Treatment

Hospitalization is mandatory for treatment. The approach is complex and includes the following:

1. **Daily regimen:** Structured daily activity and rest.
2. **Dietary regimen:** A No. 5 diet (low-fat, easily digestible foods) to support liver function.
3. **Parenteral detoxification:** To remove toxins and support the liver.
4. **Symptom-based treatment:** Medications are given according to symptoms. In severe cases, glucocorticosteroids such as **prednisone** (40-60 mg daily) may be used. **Panangin** and **qushqonmas** are prescribed to correct electrolyte imbalances, particularly in hypokalemia. **Antispasmodics** such as **No-Spa** and **euphylline** may be administered. If the lower intestine is involved in absorption, antibiotics like **neomycin** may be used. In cases of cholestasis (bile flow impairment), **ursodeoxycholic acid** (e.g., **Urosan**, **Ursofalk**) is prescribed.

### Prevention

**Immunization** is the most effective preventive measure. The development of the commercial HBV vaccine in 1982 allowed for effective control of this infection. In

Kazakhstan, the national vaccination schedule for HBV was implemented in 1992.

Vaccination against HBV is recommended for the following groups:

1. **Newborns:** Vaccination is given within 24 hours after birth.
2. **Healthcare workers** who handle blood and bodily fluids.
3. **Blood recipients** (e.g., hemodialysis patients, hematology patients, kidney transplant recipients).

4. **Medical students and healthcare staff** in tertiary medical centers.

HBV vaccination is performed with a **recombinant DNA vaccine**, which contains HBV antigens. This vaccine is produced in several countries, including **North Korea, South Korea, China, Belgium, Cuba, France, Japan, Switzerland,** and the **United States**. The vaccine is administered in three intramuscular injections:

1. **First dose:** Given at birth or in the first 24 hours.
2. **Second dose:** Given 2 months after the first dose (alongside DTP vaccination).

3. **Third dose:** Administered 4 months after the second dose (alongside DTP vaccination).

For **older children and adults**, the vaccination schedule includes:

1. **First vaccination.**
2. **Second dose:** 2 months after the first.
3. **Third dose:** 6 months after the first dose.

HBV vaccines can be combined with other vaccines like **BCG, DTP, measles,** and **mumps** in a single combination vaccine, but they should not be mixed with other vaccines in a single syringe.

### **References**

1. "Hepatitis Viruses," Baron's Medical Microbiology, 4th edition, Baron S., University of Texas Medical Branch, 1996. .
2. "Molecular Virology of Hepatitis B Virus," Seminars in Liver Disease, 2004.
3. Desk Encyclopedia of General Virology, Boston: Academic Press, 2009,
4. "The Biology of Hepadnaviruses," Journal of General Virology, 1986.