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Parenteral Acute Viral Hepatitis B and C: Modern Diagnosis, Prevention, and Treatment

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Abstract: Questions of diagnostics, feature of clinic, treatment and preventation of active virus hepatites with parenteral mechanism of transmission of an infection (hepatites B, C,) which make up a significant number of all acute virus hepatites are considered. A wide circulation, features of modern diagnostics and frequency of progress of chronic forms defi ne a problem of diagnostics and treatment of a virus hepatites,as one of important for domestic healthcare.Data about the importance and features serological markers of viruses of a hepatites B, C, , are presented and their clinical interpretation is analyzed. Modern drug therapy is in detail resulted including preparations of interferon. The need of specific preventive actions, post-exposure preventive maintenance and passive immunization is considered.

Key points: Acute hepatites B, C; ALT (Alanine aminotransferase), AST (Aspartate aminotransferase), ELISA, RIBA , Genome Variability.

Acute Viral Hepatitis (AVH) is an infectious disease characterized by acute necrosis and inflammation of the liver caused by hepatitis viruses A, B, C, and others. Hepatitis A and E are classified as enteral infections with a fecal-oral transmission mechanism, while hepatitis B and C belong to the group of parenteral hepatitis.

Regardless of the virus type, the clinical manifestation is primarily centered around liver damage, and the disease typically follows a cyclical course. Liver damage can also occur due to other viruses during generalized infections, such as cytomegalovirus, Epstein-Barr virus, adenovirus, echovirus, and TT virus [1].

The clinical presentation encompasses a wide range of syndromes, from subclinical cases to rapidly progressing and fatal outcomes. In most cases, acute viral hepatitis is self-limiting and uncomplicated. However, depending on the viral agent, the frequency of extrahepatic manifestations and the risk of developing chronic liver disease vary significantly. Occasionally, the disease may remain subclinical.

SEROLOGICAL MARKERS IN VIRAL HEPATITIS

(Table 1).

Pathogen	Marker	Diagnostic Significance
HBV Virus	**HBsAg**	Positive in most cases of acute or chronic hepatitis.
	HBcAg	Typically undetectable in serum.
	HBeAg	Transiently positive during viral replication.
	anti-HBc (IgM, IgG)	Positive in cases of acute and chronic infection and in carriers of HBV infection markers; non-protective; anti-HBc IgM indicates viral replication.
	anti-HBe	Transiently positive during convalescence, in some chronic infections,

		and in carriers; non-protective, indicates low infectivity.
	Pre-S1 Ag	Marker of infectivity and high risk of vertical transmission of HBV.
	anti-HBs	Becomes positive in the late convalescence phase in most acute cases.
	anti-Pre-S2	Marker of recovery from HBV infection and high vaccination efficacy.
HCV Virus	**anti-HCV IgM**	Marker of active HCV replication.
	anti-HCV IgG	Non-protective; seropositive individuals should be considered infected.

...leading to chronic liver disease with the development of cirrhosis and even hepatocellular carcinoma.

After varying incubation periods, viral replication in liver cells reaches its peak, resulting in the presence of viral components in bodily fluids and/or excreta, hepatocyte necrosis combined with an inflammatory response. This corresponds to the clinical symptoms of liver damage and changes in laboratory parameters. The immune response plays a significant but not fully understood role in the pathogenesis of the disease.

Acute viral hepatitis with parenteral transmission ranks among the leading infectious diseases in Russia, due to its high incidence, severe clinical course, and the considerable frequency of chronic forms. Addressing this serious public health issue is closely tied to improving the diagnosis, prevention, and treatment of acute viral hepatitis.

Diagnosis of Acute Viral Hepatitis (AVH)

At the initial stages, in most cases, the diagnosis is made through clinical and biochemical studies. Significant information for diagnosing hepatitis B and C includes data about:

- Blood transfusions and its components,
- Parenteral procedures,
- Chronic hemodialysis,
- Multiple injections,
- Prolonged hospitalization of the patient.
- Intravenous drug use,
- Possible sexual transmission.

Clinical Criteria

Special attention must be given to:

- The cyclic nature of disease manifestations,
- The duration of individual symptoms.
- It is important to track how the symptoms of the pre-icteric period change as jaundice develops.

Functional Criteria

Early diagnosis in the pre-icteric stage and detection of anicteric and subclinical forms can be helped by examining the activity of:

- ALT (Alanine aminotransferase),
- AST (Aspartate aminotransferase),
- Aldolase.

A significant increase in ALT is characteristic. Bilirubin appears in the urine, and its level in the blood serum usually rises as clinical symptoms appear.

Reliable diagnostic criteria include the results from the study of serological markers of various etiological variants of acute hepatitis (Table 1).

Liver biopsy is not necessary for diagnosing AVH. It is indicated in cases of atypical disease progression (e.g., prolonged form, persistence of symptoms, or biochemical test deviations lasting more than 4 weeks).

A liver biopsy is performed when there is a mild elevation in aminotransferase levels along with persistent HBsAg for more than 16 weeks, as this symptom complex suggests the possible transition to chronic hepatitis.

Differential Diagnosis

Anicteric and subclinical forms of acute hepatitis need to be distinguished from:

- Gastritis,
- Enterocolitis,
- Enteroviral infection.

The key diagnostic factor is clinical and laboratory signs of liver damage.

The icteric form of AVH can sometimes be very difficult to distinguish from acute toxic and allergic hepatitis caused by medications (e.g., MAO inhibitors, antituberculous and sulfonamide drugs).

For diagnosis, the information about the drugs the patient has taken and their possible hepatotoxic effects is critical.

In clinical practice, it is extremely important to differentiate the cholestatic form of AVH from subhepatic jaundice.
Clinical and biochemical indicators may sometimes be insufficient, and a reliable diagnosis may be possible only after duodenoscopy with retrograde pancreatocholangiography and percutaneous cholangiography.

There can be significant difficulties in distinguishing AVH from acute alcoholic hepatitis. Substantial assistance comes from knowledge of the extrahepatic signs of alcohol intoxication. In doubtful cases, liver biopsy is used, revealing:

- Centrilobular necrosis of hepatocytes,
- Mallory bodies (alcoholic hyaline) in their cytoplasm,
- Portal tract infiltrates, predominantly composed of polymorphonuclear leukocytes.

Clinical Features

The incubation period of acute hepatitis B varies from 4 weeks to 6 months, with an average duration of 50 days. The pre-icteric period is characterized by gradual onset, the absence of high fever, but possible low-grade fever, arthralgia (mainly at night), and malaise in 20-30% of patients. Symptoms commonly include fatigue, weakness, easy fatigue, reduced appetite, nausea, vomiting, and a feeling of heaviness or dull pain in the right hypochondrium. In the pre-icteric period, there is an increase in the activity of serum transaminases, and HBV-specific markers can be detected. In some patients, prodromal symptoms may be absent, and dark urine and jaundice of the sclera are the first signs of the disease.

The icteric period progresses with pronounced and persistent clinical symptoms: increasing weakness, nausea, anorexia, and itching of the skin (found in 20% of patients). Jaundice peaks during the 2nd to 3rd week. The urine remains dark, and the stool is pale. The liver becomes enlarged, somewhat firm, and tender to palpation. It should be noted that the icteric form occurs in a smaller proportion of patients, approximately 30% have subclinical or anicteric forms.

Extrahepatic manifestations are often seen, including urticaria and other rashes, arthritis, and, less commonly, glomerulonephritis and vasculitis. HBV infection is associated with over one-third of cases of nodular periarteritis.

Laboratory testing reveals a rise in the activity of transaminases from 1000 to 2000 IU/L, with ALT typically being higher than AST. An increase in transaminases does not correlate with the prognosis. The best predictor of prognosis is the prothrombin time. The reduction of ALT activity typically occurs within 1 to 4 months, at which time bilirubin levels normalize.

Diagnosis of Hepatitis B

The first serological markers of viremia may appear as early as 2 weeks after infection, especially in cases of massive parenteral exposure. HBsAg begins to be detectable in the serum between 2 weeks and 2 months before the clinical manifestations of the disease. Anti-HBc antibodies are detected approximately simultaneously with clinical symptoms and the elevation of serum transaminases. Initially, anti-HBcIgM is present in high titers and persists in the serum for several months to 1 year. Later, anti-HBcIgG antibodies become dominant. Anti-HBcIgG can persist for several years after acute hepatitis and be found in all chronic carriers. These antibodies do not provide protective immunity but rather serve as a marker of past HBV infection.

Markers of Active Replication

- HBeAg, anti-HBcIgM, DNA polymerase, and HBV DNA can typically be detected in the serum before the rise in transaminase activity.
- The duration of HBsAg positivity varies widely, from several days to 2-3 months. Persistence for more than a few months may suggest a chronic process. It is characteristic that HBsAg disappears before the appearance of anti-HBs antibodies. These antibodies are present in 80-90% of patients, especially during convalescence, and indicate relative or absolute immunity. Their detection suggests an adequate immune response to the infection.

Interpretation of Serological Test Results in Acute Hepatitis B

Several important considerations must be taken into account when interpreting the results:

1. In a certain number of cases of acute viral hepatitis B, HBsAg may not be detectable in the serum, usually due to its low concentration. Therefore, the absence of HBsAg does not rule out the diagnosis of acute viral hepatitis B. In this case, anti-HBc IgM is a more sensitive marker and may be the only serological indicator of HBV infection.
2. Negative anti-HBc IgM results likely exclude the diagnosis of acute hepatitis B. On the other hand, a positive anti-HBc IgG result in the absence of HBsAg may simply reflect a past HBV infection. Anti-HBc IgG is also commonly found in carriers.
3. Patients with HBsAg-negative but anti-HBc-positive markers can be differentiated based on anti-HBs levels. Positive anti-HBs results at the onset of the disease are inconsistent with a diagnosis of acute hepatitis B.
4. The detection of anti-HBc IgM indicates either a recently resolved acute hepatitis B or chronic hepatitis B during the phase of active viral replication. In patients with a history of acute hepatitis and clinical signs of active liver disease, absence of anti-HBc IgM may suggest a superinfection with HDV or another viral or non-viral cause of liver disease.
5. A positive test for HBeAg or HBV DNA provides definitive evidence of ongoing HBV replication.

Hepatitis C

In 1989, Houghton and colleagues identified the HCV virus, which replaced the term "non-A, non-B hepatitis with a parenteral transmission mechanism." HCV is an RNA-containing flavivirus, ranging from 30-75 nm in diameter, and covered by a lipid envelope. On its 5' terminal end, there is a segment containing 329-341 nucleotides, which is 92% homologous across different HCV types.

This segment is responsible for genome translation and is highly conserved, which makes it useful for detecting HCV RNA via Polymerase Chain Reaction (PCR).

The viral genome also contains a core region, two segments encoding envelope glycoproteins (E1 and E2), and four non-structural (NS) regions, which code for enzymes crucial in virus replication. The core protein forms the nucleocapsid and participates in replication. The glycoproteins E1 and E2 play roles in the virus's entry into cells. Within the E2 gene, there is a hypervariable region. Changes in this region and the corresponding antigenic determinants of E2 are pivotal for the virus's ability to evade the primary immune response to infection.

A distinctive feature of HCV is its ability to persist long-term in the body, leading to a high chronicity rate, with 50-80% of cases progressing to chronic infection. There are at least 6 genotypes of HCV, classified based on the analysis of the 5' terminal segment of the NS5 non-structural region. The distribution of HCV genotypes varies geographically.

Recombinant Proteins Used in ELISA and RIBA Test Systems *(According to Weiland O., 1994)*

(Table 2).

Method	Generation	Antigen	Region
HCV ELISA	1st Generation	C100	-
	2nd Generation	C100	-
		C22	-
		C33	-
	3rd Generation	C100	-
		C22	-
		C33	-
		N35	-
		NS4	-
RIBA	3rd Generation	C100-3	Core
		C33	NS3
		C22-3	NS4
		NS5	NS5

This table outlines the recombinant antigens used in various generations of ELISA and RIBA assays for detecting Hepatitis C Virus (HCV). Each generation shows improvements in sensitivity and specificity by incorporating additional antigens from different regions of the HCV genome.

- C100: Early recombinant antigen targeting a region within NS4.
- C22: Corresponds to the Core region of HCV.
- C33: Represents the NS3 region.
- N35 and NS4/NS5: Target non-structural proteins (NS), which are crucial for identifying chronic infections.

The distribution of **hepatitis C virus (HCV)** genotypes is uneven. In particular, in European countries, genotype 1b accounts for 50–91% of cases (Germany — 59%, Belgium — 65%, Hungary — 84%, Italy (Sicily) — 91%), while genotype 1a accounts for no more than 40%. In the United States, the prevalence of genotypes 1a and 1b is 37% and 30%, respectively.

In Central Asia and Central Africa, genotype 4 is common, whereas genotypes 2 and 3 are predominant in Northern and Central Europe. In Southeast Asia and the Far East, genotypes 1, 2, and 6 are the most prevalent【4,5】.

In Russia, genotype 1b is dominant, accounting for:

- 64.7% in various regions of Northern Eurasia,

- 80–83% in the Far East,
- 50–56% in the Central Black Earth and Volga-Vyatka regions.

Genotype 1a is most frequently detected in the Central, Northwestern, and Volga-Vyatka regions, ranging from 11.2% to 21.9%. Genotypes 3a, 2a, and 2b are considered rare in Russia【6】.

Genetic Heterogeneity of HCV

The genetic heterogeneity of HCV leads to differences in the course and outcome of the disease, as well as the effectiveness of therapy. Chronic HCV infection develops after acute hepatitis in:

- 92% of cases with genotype 1b,
- 33–**50% of cases with other genotypes**.

Moreover, significant differences exist within the same genotype, primarily in the **E2/NS1 region** (also known as the hypervariable region). These genetic variations can rapidly emerge during acute or chronic infections and are important because they may lead to **false-negative results** in tests for HCV antibodies or viral RNA.

This occurs because primers used in diagnostic tests may fail to recognize the nucleotide sequences of such altered viral variants.

Genome Variability

Genome variability also plays a crucial role in the fact that previous HCV infection does not provide immunity against reinfection. Therefore, multiple infections with different HCV variants are possible.

These characteristics of the virus hinder the development of a vaccine against HCV and can impact the effectiveness of **antiviral and immunomodulatory therapy** in chronic HCV infection.

Diagnosis. Detection of Antibodies to HCV.

The enzyme immunosorbent assay (ELISA) has gained the most widespread use due to its reliability and sensitivity. The first-generation test system (ELISA-1) detected antibodies to the C-100 antigen. Subsequently, new HCV RNA clones were obtained, producing other viral antigens for detecting the corresponding antibodies (Table 2), leading to the development of second- and third-generation ELISA. ELISA-3 is widely used for donor screening, offering significantly higher sensitivity and specificity compared to previous-generation test systems.

An important feature of ELISA-3 is its ability to detect antibodies to the NS5 region, as up to 5% of virus carriers have only this type of antibody in their blood, which leads to false-negative results when using earlier ELISA generations. The use of ELISA-3 provides almost a 100% guarantee of detecting anti-HCV carriers during donor screening and in diagnosing viral liver diseases.

However, antibodies may not be detected in two cases:

1. The appearance of anti-HCV in the blood can occur up to 6 months after infection (on average, after 12 weeks), meaning there is a "serological window" during a certain period of infection.
2. Antibodies may not be detected in patients receiving immunosuppressive therapy (e.g., after organ transplantation).

Despite the high specificity of ELISA-3 (99.7%), false-positive results are also possible. To address this, confirmatory tests have been proposed, such as recombinant immunoblot assay (RIBA) and the less commonly used synthetic peptide analysis (Inno-Lia).

In RIBA, HCV antigens are applied separately to nitrocellulose strips and incubated with the patient's serum. If the corresponding antibodies are present, they are visualized. The third-generation test system (RIBA-3), widely used in Europe, contains synthetic peptides of the core region, NS4, and recombinant NS3 and NS5 (see Table 2).

RIBA results are considered positive if antibodies to more than one HCV region are detected. In most RIBA-positive individuals, the virus is in a replication state, as confirmed by detecting HCV in 75–80% of cases.

The absence of RNA in the presence of anti-HCV antibodies may be due to:

- Elimination of the virus after infection,
- A viremia level below the sensitivity threshold of the polymerase chain reaction (PCR), or
- A false-positive antibody detection result.

There is a noted correlation between the absence of HCV RNA in anti-HCV-positive patients and the absence of inflammatory changes in liver biopsy specimens, which may indicate virus elimination. However, HCV RNA may not be detected in patients after antiviral therapy; in such cases, its presence at sub-threshold levels or virus persistence in tissues, making it undetectable, cannot be ruled out.

Thus, individuals who test positive for anti-HCV, even in the absence of signs of viremia, should be considered potentially infectious regarding HCV transmission.

Detection of Viral RNA

Determining the presence of HCV RNA in serum is necessary for:

1. Confirming HCV infection in anti-HCV-positive individuals or in cases of suspected infection when antibodies to the virus are absent;
2. Early diagnosis of acute hepatitis;
3. Monitoring perinatal transmission of the virus;
4. Determining indications for antiviral therapy;
5. Monitoring the effectiveness of antiviral therapy.

The most widely used method is PCR, which involves synthesizing multiple copies of DNA based on viral RNA using reverse transcriptase, followed by electrophoresis in a polyacrylamide gel. PCR can be used to detect RNA both in serum and in liver biopsies. Recently, quantitative RNA determination has gained popularity, utilizing techniques such as serial dilution analysis and branched DNA amplification. It is important to note that the latter method, while providing the most accurate assessment of viremia, is less sensitive (about 70% of PCR sensitivity) and therefore requires parallel PCR testing. Less commonly used methods include ligase chain reaction and isothermal amplification of nucleic acids.

Genotyping can assist in determining the epidemiology of hepatitis C and in developing a personalized approach to patient care. It plays a decisive role in recommendations regarding the choice of antiviral drugs and the duration of treatment. Genotyping is carried out based on sequence analysis using sequencing and reverse hybridization. Viral load may vary, but the genotype remains unchanged during the infection. Superinfection with a different genotype is rarely detected. For reliable genotyping, 5'URT (5' untranslated region) is insufficient by itself, and sequencing of NS5B is the gold standard.

Clinical Features

The incubation period after infection lasts from 5 to 7 weeks, after which there is an increase in transaminase activity and other clinical manifestations. Acute hepatitis C is characterized by a high proportion of anicteric forms (more than 80%), which most often proceed asymptomatically. Clinical symptoms and laboratory findings of hepatitis C are indistinguishable from other forms of acute hepatitis. In general, acute hepatitis C is much milder than other acute viral hepatitis.

Diagnosis

Specific markers confirming the presence of acute hepatitis C are antibodies to the hepatitis C virus (anti-HCV), which are detected in immunoassay using modern test systems, starting from the 2nd to 3rd week of the disease. Radioimmunoblotting (RIBA) analysis is used to identify false-positive samples, ensuring an accurate result in more than 95% of cases.

The only absolutely reliable test is the detection of hepatitis C RNA using polymerase chain reaction. The HCV RNA test becomes positive no earlier than 2 weeks after infection. Testing for HCV RNA may be helpful in unclear cases, although a positive RIBA test correlates with the presence of HCV RNA. PCR testing may be indicated to assess viral replication activity in individuals with liver damage caused by multiple etiological factors.

Treatment

Patients with acute viral hepatitis (AVH) in our country are hospitalized in infectious disease departments and hospitals. During the peak of the illness, bed rest is prescribed. Hospitalization duration ranges from 2-4 weeks to 6 weeks or even several months, depending on the severity of the disease. Most patients in the USA and Europe are not hospitalized and are monitored at home. Rest is recommended, but strict bed rest is not mandatory if the patient does not experience significant weakness. In the case of home treatment, appropriate medical supervision is required. Examinations are conducted 2-3 times during the first week of the disease, and then at longer intervals. Biochemical tests are performed twice a week during the first two weeks of illness. If anorexia and/or vomiting occur or biochemical parameters worsen, immediate hospitalization is required.

Adherence to bed rest is particularly important when there are severe clinical symptoms, prolonged hyperbilirubinemia lasting more than 2-3 weeks, a decrease in prothrombin index, and in weakened patients or those over 40 years old. Alcohol and all medications, especially narcotics, analgesics, and tranquilizers, should be excluded. Sedative medications should also not be prescribed due to impaired elimination by liver cells. The use of oral contraceptives is prohibited. Special dietary restrictions are not necessary for patients with mild to moderate illness; for most patients, a diet with reduced fat and high carbohydrate content is recommended. Diet No. 5, along with sufficient fluid intake (up to 1.5-2 liters per day), is advised. Mineral waters such as Borjomi, Essentuki No. 4, 17, and Mirgorodskaya can be used.

In severe cases, during the acute phase of the illness, anorexia and nausea may be so pronounced that oral food intake is minimized. In such cases, water-salt balance monitoring is essential; frequent intake of small amounts of liquid is desirable. For pronounced nausea, medications that normalize gastrointestinal motility (e.g., cerucal, motilium, cisepride) are recommended. Parenteral infusion therapy with detoxification purposes is indicated. Intravenous drip administration of Ringer's solution and 5% glucose solution, up to 2 liters per day, is recommended. pH and electrolyte levels of the blood are monitored for necessary corrections: 5% ascorbic acid is used for severe alkalosis, and 3% sodium bicarbonate solution (50-100 ml) for severe acidosis.

Medications

Pharmacological treatment should be minimal. The administration of vitamins B1, B2, B6, B12 through injections is not indicated unless there is a specific deficiency. Ascorbic acid and riboxin can be used as non-specific immune stimulators.

Hepatitis B

In mild, moderate, and severe forms of acute hepatitis B, antiviral therapy is not indicated. There are few reports on the effectiveness of lamivudine in a dose of 100 mg/day for 1 to 6 months in cases of severe prolonged acute hepatitis B. The criteria for prolonged disease include the presence of HBeAg for more than 30 days. In severe cases with the risk of liver coma development, nucleoside analogs are recommended in standard doses until the disappearance of HBsAg, and in case of liver transplantation, to reduce the risk of transplant infection.

Hepatitis C

Given the serious prognosis and the lack of criteria for predicting chronicity, in addition to basic therapy, the prescription of interferon drugs is reasonable. Treatment initiated after 3 months of disease can lead to a sustained virological response in more than 80% of patients with acute hepatitis C. These results are observed with both short and pegylated interferons. The course duration ranges from 12 to 24 weeks. The recommended doses of interferon are from 3-5 million IU daily for 4 weeks, followed by 3-5 million IU every other day for 20 weeks. Another regimen is possible: 10 million IU daily for 4-6 weeks (until transaminase levels normalize), followed by 3 million IU every other day for up to 12 weeks. Pegylated interferons are prescribed in standard doses.

Patients with fulminant forms of AVH showing signs of hepatic encephalopathy are transferred to intensive care units, where central venous pressure, pH, blood sugar, and electrolytes are monitored, and intracranial pressure is monitored if necessary.

PROPHYLAXIS

Unlike hepatitis A and E, hepatitis B and C are not usually transmitted through the fecal-oral route, although secretions from infected individuals should be considered potentially infectious. Virus transmission can occur through needle punctures with contaminated needles (or equivalent contact with infected materials) or through intimate, particularly sexual, contact, especially during the period of HBsAg positivity. The prevention of serum hepatitis relies on mechanical cleaning and the effective sterilization of instruments. Each procedure (vaccination, diagnostic testing) should use reliably sterilized needles and syringes. An important task is ensuring that healthcare institutions have single-use syringes available. Proper monitoring of blood donors is also essential. A highly effective measure in preventing post-transfusion hepatitis is medically justified limitation of blood and blood product transfusions.

IMMUNIZATION AGAINST VIRAL HEPATITIS

Hepatitis B

Although the pathogen is not primarily transmitted via the fecal-oral route, the same hygiene measures as for hepatitis A should be followed. Transmission typically requires close contact with an infected person or the parenteral introduction of infected material. Strict isolation is generally not necessary.

Specific prophylaxis is achieved through the use of recombinant HBV vaccines. Effective and safe vaccines have been developed using recombinant products. These vaccines are made using HBsAg synthesized by microorganisms. The vaccine is administered three times: the second dose is given after 1 month, and the third dose after 6 months. This regimen leads to the production of anti-HBs in 90% or more of healthy recipients. The vaccine is administered intramuscularly into the deltoid muscle, with a dose of 10–20 mcg for adults and 2.5–10 mcg for children. Higher doses are used for patients on hemodialysis or those who are immunosuppressed. The recombinant vaccine is safe for pregnant women. Most individuals who receive the three-dose vaccination become immune to hepatitis B infection. Exceptions are most commonly seen in immunosuppressed individuals. The duration of immunity varies but lasts for at least 5 years, after which a single booster dose is recommended.

Active immunization is carried out for high-risk groups. These include healthcare workers (surgeons, dentists, dialysis unit staff); patients undergoing hemodialysis; patients receiving multiple transfusions (e.g., those with hemophilia); individuals with a history of drug use; individuals who have heterosexual or family contacts with HBsAg carriers; and homosexual men. For emergency prevention in unvaccinated healthcare workers (in cases of cuts or needle-stick injuries), hyperimmune specific immunoglobulin with a high titer of anti-HBsAg antibodies is used alongside active immunization with the hepatitis B vaccine.

In Russia, WHO recommendations have been adopted, and hepatitis B vaccination is now included in the immunization schedule. Priority is given to vaccinating newborns from mothers who are carriers of the hepatitis B virus. The vaccination is administered at birth, on day 1, followed by additional doses at 1, 2, and 12 months after the first dose. For hyper-endemic areas, this vaccination scheme is recommended for all newborns due to the high risk of infection in the first months of life. For other regions, vaccination is recommended to start at 4–5 months of age.

POST-CONTACT PROPHYLAXIS AND PASSIVE IMMUNIZATION

Hepatitis B

Non-immune individuals can be infected with HBV in the following situations:

- Accidental inoculation with infected HBV material, such as a needle stick injury from a HBsAg-positive patient or transfusion of HBsAg-positive blood.
- Exposure of eyes or damaged skin to HBsAg-positive material.
- Ingestion of HBsAg-positive material.
- Sexual contact with a person with acute hepatitis B (immunization should be done within 14 days after sexual contact).
- Newborns of HBsAg-positive mothers, especially those who had acute hepatitis B in the last trimester of pregnancy or in the first 2 months after birth, or those who were positive for both HBsAg and HBeAg during delivery.

For these individuals, early passive immunization with hepatitis B immunoglobulin (serum immunoglobulin with a high anti-HBs titer, 0.06 ml per kg of body weight) is recommended, in combination with active HBV vaccination. To determine the exact indications for immunization, urgent testing of the serum from both the "donor" and the "recipient" for hepatitis markers is advisable.

Passive immunization with hepatitis B immunoglobulin is effective if administered within 14 days after sexual contact. In all cases where the infected person has been vaccinated, additional passive immunization is not required if the individual has a sufficient anti-HBs titer (10 million IU per ml).

If the vaccination was started but not completed, or if anti-HBs titers are low, a single dose of hepatitis B immunoglobulin should be administered, followed by the completion of the vaccination series.

Hepatitis C

General preventive measures for hepatitis C are similar to those for hepatitis B, although there is no evidence of transmission through family or sexual contacts. The main focus is on the prevention of post-transfusion hepatitis through donor screening.

The usefulness of administering immunoglobulin before a transfusion is unclear, as there is no evidence of protective antibodies against HCV. As a result, passive immunization is not currently recommended for hepatitis C.

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