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INTEGRATED DIAGNOSTIC APPROACHES TO VIRAL HEPATITIS B IN CONTEMPORARY CLINICAL PRACTICE

Berdiyarova Sh. Sh.
Assistant of the department of clinical laboratory diagnostics and with a course in clinical laboratory diagnostics PGF
Azamkulova P.A.
Cadet of the department of clinical laboratory diagnostics and with a course in clinical laboratory diagnostics PGF
Shukurova S.X.
Student of the Samarkand state medical university

Abstract. *Viral hepatitis B remains one of the most pressing problems of modern clinical medicine due to its high prevalence, risk of chronicity, and the development of severe complications, including liver cirrhosis and hepatocellular carcinoma. This article reviews current approaches to the comprehensive diagnosis of viral hepatitis B, encompassing clinical, serological, molecular biological, and instrumental methods of investigation. Special attention is given to the identification of HBV infection markers (HBsAg, HBeAg, anti-HBc, anti-HBs), quantitative determination of hepatitis B virus DNA using polymerase chain reaction (PCR), as well as assessment of liver functional status based on biochemical parameters. The significance of a comprehensive diagnostic approach is demonstrated for early detection of the disease, determination of the stage and activity of the infectious process, monitoring the effectiveness of antiviral therapy, and predicting disease outcomes. The application of modern diagnostic technologies contributes to improved diagnostic accuracy and enhanced quality of medical care for patients with viral hepatitis B.*

Keywords: *viral hepatitis B, comprehensive diagnosis, serological markers, HBsAg, HBeAg, anti-HBc, HBV DNA, PCR diagnostics, biochemical parameters, liver function, clinical practice.*

Introduction. Viral hepatitis B is one of the most significant medical and social problems of modern healthcare, which is обусловлено its wide prevalence, high level of population infection, and severe medical and biological consequences. According to the World Health Organization, hundreds of millions of people worldwide are carriers of the hepatitis B virus, while a substantial proportion of infected individuals are unaware of their disease, which contributes to further spread of the infection and late detection of pathology. The chronic course of hepatitis B often leads to progressive liver fibrosis, cirrhosis, and the development of hepatocellular carcinoma, significantly increasing disability and mortality rates among patients of working age [3,15,16].

The relevance of the problem is also determined by the clinical polymorphism and often asymptomatic course of viral hepatitis B in the early stages of the disease. The absence of pronounced clinical manifestations complicates timely diagnosis and leads to detection of the disease already at the stage of complications. Under these conditions, comprehensive diagnostics based on the combined use of clinical, serological, molecular biological, and biochemical methods becomes particularly important, allowing objective assessment of viral replication activity and the degree of liver damage. Modern clinical practice places high demands on the accuracy and informativeness of diagnostic methods, since laboratory test results form the basis for treatment strategy selection and monitoring the effectiveness of antiviral therapy. Determination of serological markers of HBV infection, quantitative assessment of viral DNA by polymerase chain reaction, and analysis of biochemical indicators of liver function allow not only confirmation of the diagnosis, but also determination of the disease phase, prediction of the risk of chronicity, and development of complications [3,12,15].

Comprehensive diagnosis of viral hepatitis B (VHB) in modern clinical practice is based on a stepwise and complementary assessment of clinical data, laboratory, and instrumental diagnostic methods. This approach makes it possible not only to confirm the presence of HBV infection, but also to determine the stage of the disease, the activity of viral replication, the degree of liver damage, and the prognosis of the pathological process. Clinical assessment includes analysis of patient complaints, medical history, and risk factors for infection. In the acute phase of viral hepatitis B, symptoms of intoxication, weakness, loss of appetite, dyspeptic disorders, jaundice of the skin and sclera may be observed; however, in a significant proportion of patients the disease proceeds subclinically or asymptomatically. In chronic hepatitis B, clinical manifestations are usually mild, which emphasizes the leading role of laboratory diagnostics [5,10,13,17].

Serological methods form the basis of laboratory diagnosis of HBV infection. Detection of HBsAg is used as the main screening marker indicating the presence of the virus in the body. Identification of HBeAg reflects active viral replication and high infectivity of the patient, whereas antibodies to HBeAg (anti-HBe) indicate a decrease in replicative activity. Antibodies to the viral core antigen (anti-HBc IgM and IgG) allow differentiation between acute and chronic forms of the disease and assessment of the phase of the infectious process. Molecular biological methods, primarily polymerase chain reaction (PCR), play a key role in assessing viral load. Quantitative determination of hepatitis B virus DNA enables objective evaluation of HBV replication activity, serves as an important criterion for initiating antiviral therapy, and is used to monitor its effectiveness. A decrease or disappearance of viral DNA during treatment is regarded as a favorable prognostic sign [6,14,16].

Biochemical studies are aimed at assessing liver functional status and the degree of cytolytic syndrome. Measurement of aminotransferase activity (ALT, AST), bilirubin levels, alkaline phosphatase, gamma-glutamyltransferase, and protein metabolism indicators allows detection of inflammatory changes in the liver and assessment of hepatocyte damage severity. Persistent elevation of ALT levels

combined with high viral load indicates active chronic hepatitis B. Instrumental methods complement laboratory diagnostics and are used to evaluate structural changes in the liver. Ultrasound examination, elastography, and other non-invasive methods make it possible to identify signs of liver fibrosis and cirrhosis and to perform dynamic patient follow-up. In certain cases, liver biopsy is used and remains the “gold standard” for assessing the degree of necroinflammatory changes and fibrosis [1,4,6,15].

The causative agent of viral hepatitis B is the hepatitis B virus (HBV), belonging to the Hepadnaviridae family. The virus is a DNA-containing pathogen with a complex structure, including an outer envelope with surface antigen (HBsAg) and an inner nucleocapsid core containing the core antigen (HBcAg), viral DNA, and DNA polymerase enzyme. High environmental stability of HBV and its pronounced infectivity determine the significant epidemiological hazard of this disease. The main source of infection is an infected person—patients with acute or chronic viral hepatitis B, as well as asymptomatic carriers. Transmission of HBV occurs predominantly via the parenteral route through contact with infected blood and biological fluids. The most significant transmission factors include blood transfusions and blood products, use of non-sterile medical instruments, injection drug use, and invasive medical and cosmetic procedures [9,11,13].

The sexual route of transmission plays a substantial role in the spread of viral hepatitis B, especially in individuals with multiple sexual partners and absence of barrier contraception. Vertical transmission from an infected mother to a child during childbirth is also of great epidemiological importance, as it often results in chronic infection in newborns. Factors contributing to HBV infection include occupational exposure among healthcare workers, low sanitary culture, and insufficient vaccination coverage against hepatitis B. Thus, the etiology of viral hepatitis B is closely related to the biological characteristics of the pathogen and its transmission routes, determining the need for comprehensive preventive and diagnostic measures in modern clinical practice [7,15].

Clinical manifestations of viral hepatitis B (VHB) are characterized by significant polymorphism and depend on the disease course, immune status, age, viral load, and presence of concomitant liver diseases. The disease may present in subclinical or manifest forms, including acute, chronic infection and viral carriage. The incubation period averages 45–180 days. During this period, clinical symptoms are usually absent, while laboratory markers such as HBsAg and viral DNA may already be detected in serum [7,14,15,19].

The prodromal (pre-icteric) period is characterized by nonspecific symptoms of general intoxication, including weakness, fatigue, loss of appetite, nausea, vomiting, discomfort in the right hypochondrium, arthralgia, myalgia, headache, low-grade fever, and dyspeptic disorders. Skin manifestations such as rash and pruritus may occur due to immunopathological reactions. The icteric period is accompanied by progressive jaundice of the skin, sclera, and mucous membranes, dark urine, acholic or hypocholic stools, hepatomegaly, and sometimes splenomegaly [9,14,15,17].

A distinctive feature of viral hepatitis B is the possibility of severe disease with development of cholestatic syndrome, manifested by intense pruritus, prolonged jaundice, and elevated alkaline phosphatase and gamma-glutamyltransferase levels. In rare cases, a fulminant course may develop, characterized by rapid onset of acute liver failure, encephalopathy, and coagulopathy, posing a direct threat to life. Chronic viral hepatitis B is defined by viral persistence for more than six months and may remain asymptomatic for a long time. As the disease progresses, fibrosis and cirrhosis may develop, accompanied by portal hypertension, ascites, esophageal varices, and impaired synthetic liver function [1,7,15,16].

Extrahepatic manifestations of viral hepatitis B are immune-mediated and include joint involvement, renal damage (glomerulonephritis), skin lesions (polyarteritis nodosa), as well as vascular and hematological disorders, which may dominate the clinical picture and complicate diagnosis. Laboratory diagnostics of viral hepatitis B occupies a key position in disease detection, staging of the infectious process, assessment of viral replication activity, and evaluation of liver function. A

comprehensive laboratory approach allows confirmation of diagnosis, prognosis of disease course, and monitoring of therapy effectiveness. The main laboratory methods include serological, molecular biological, and biochemical investigations [6,7,10,19].

The principal serological markers include HBsAg, anti-HBs, HBcAg, anti-HBc IgM and IgG, HBeAg, and anti-HBe. Detection of HBsAg is the main screening criterion for HBV infection. Anti-HBc IgM indicates acute infection, whereas anti-HBc IgG suggests chronic infection or past exposure. Molecular biological methods, particularly PCR, enable detection and quantification of HBV DNA. Viral load determination is essential for evaluating replication activity, infectivity, and selection of antiviral therapy, as well as for monitoring treatment response [3,8,11,18,20].

Biochemical blood tests assess hepatocellular damage and liver function. ALT and AST elevations reflect cytolytic syndrome. Cholestatic markers include alkaline phosphatase, gamma-glutamyltransferase, and bilirubin levels. Synthetic liver function is evaluated by albumin levels, prothrombin index, and international normalized ratio (INR). Complete blood count may reveal leukopenia, thrombocytopenia, and anemia, particularly in chronic disease and cirrhosis. Coagulation abnormalities reflect the severity of liver failure and hemorrhagic risk. Additional tests include fibrosis markers (hyaluronic acid, procollagen type III) and non-invasive indices (APRI, FIB-4), which allow indirect assessment of fibrosis without liver biopsy [3,6,14,19].

Functional diagnostics in viral hepatitis B is aimed at comprehensive assessment of liver structure and function, degree of damage, inflammatory activity, and complications. Ultrasound examination reveals hepatomegaly, altered echostructure, increased echogenicity, and signs of fibrosis or portal hypertension. Liver elastography (including transient elastography, FibroScan) is a modern non-invasive method for quantitative assessment of fibrosis and is widely used for dynamic monitoring and evaluation of antiviral therapy effectiveness. Doppler ultrasound assesses hepatic and portal blood flow and allows early detection of portal hypertension [3,11,15,21].

In some cases, imaging techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) are used to clarify structural changes, detect

nodular lesions, fibrosis, cirrhotic transformation, and complications including hepatocellular carcinoma. Functional liver reserve may also be assessed using dynamic tests, such as the 13C-methacetin breath test. Despite advances in non-invasive methods, liver biopsy remains a highly informative invasive method for morphological assessment in complex diagnostic cases and when non-invasive results are inconclusive [3,11,15].

Conclusion: Viral hepatitis B remains one of the most significant problems in modern clinical medicine due to its wide prevalence, variability of clinical forms, and high risk of chronicity. The diversity of clinical manifestations, including latent and mildly symptomatic forms, substantially complicates timely diagnosis and necessitates a comprehensive diagnostic approach. Comprehensive diagnosis of viral hepatitis B, based on the integration of clinical assessment with laboratory and functional diagnostic methods, enables reliable determination of the stage of the infectious process, the activity of viral replication, and the extent of liver damage. Serological and molecular biological markers play a key role in diagnostic verification and monitoring of therapeutic efficacy, while biochemical and functional studies provide an objective assessment of the structural and functional status of the liver.

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