DIAGNOSIS OF CHRONIC VIRAL HEPATITIS

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Summary: *Data on clinical, laboratory and instrumental methods for diagnosing viral hepatitis are systematized, ideas about the latent form of viral infection are expanded and monitoring of such patients is facilitated.*

Key words: *chronic viral hepatitis, etiology, diagnosis.*

Due to the high incidence rate, frequent chronicity and severe complications that lead to significant disability and mortality, blood-borne viral hepatitis remains one of the most important and global problems of infectious diseases. HNV infection is the most common hepatitis with parenteral transmission in the Central Asian region [1,3,4]. The majority of people infected with hepatitis B virus are children and adolescents [2,5,7].

Without the use of modern diagnostic methods and studying the genetic characteristics of the hepatitis B virus, it is currently impossible to determine the condition of a sick child, the activity of chronic hepatitis and predict the course and outcome of the disease. As a result, the problem of determining HBV genotypes is relevant.

It is now known that HBV has many genotypes, including A, B, C, E, E and P [3-5]. HBV genotypes are distributed throughout the world. Genotypes A and P are found throughout the world, but are predominant in the USA and Southern Europe; genotypes B and C are found in Southeast Asia. Genotype P is only found in Central and South America, and genotype E is limited to Western Africa [6]. A new genotype 6, named 6, was discovered in the United States and France and is of great epidemiological significance [7,8,9,14].

Among the non-toxic etiological factors leading to the development of chronic liver diseases (CLD), hepatitis viruses, also known as hepatotropic viruses, are the most common and widespread. Over the past ten years, research in the field of virology, immunology, molecular biology and genetics has made it possible to identify the main clinical, epidemiological and morphological manifestations of infections caused by hepatotropic viruses, as well as to develop etiotropic therapy.

But due to the wide distribution of the main forms of viral liver diseases and their enormous medical and social significance, the problem of viral liver diseases remains extremely relevant today. The number of patients with chronic liver disease is growing in all developed countries across different age groups.

Symptoms of chronic viral hepatitis CH may be vague, nonspecific or mild. CG may be accompanied by fever, but more often develops without it. Thus, most cases of hCG are discovered accidentally, for example, during a medical examination or during a thorough examination of the patient for other diseases. In most cases, these symptoms do not appear until the formation of cirrhosis. The most common symptom is fatigue, which is often mistakenly attributed to other causes such as high blood pressure or age-related changes in the body. Other symptoms that are less common include mild discomfort in the upper quadrant of the abdomen, decreased appetite, nausea, and sometimes myalgia and arthralgia. The symptoms are not related to aminotransaminase activity or histological changes, but they are likely related to increased production of proinflammatory cytokines.

As cirrhosis and/or HCC progress, clinical symptoms may become more noticeable and are often accompanied by additional signs such as jaundice, anorexia, weight loss, abdominal pain, pruritus, easy bruising, abdominal bloating, lower extremity edema, gastrointestinal bleeding, and hepatic encephalopathy. .

In a small percentage of cases, there may be extrahepatic symptoms that indicate the course of chronic viral hepatitis. Vasculitis and purpura (with mixed essential cryoglobulinemia), membranoproliferative or membranous glomerulonephritis, cutaneous porphyria, lichen planus, lymphoma, Sjogren's syndrome, thyroid disease, insulin resistance with the potential development of type 2 diabetes mellitus and other symptoms are observed with viral hepatitis B and C (HB and GS) [6].

Physical findings (examination findings) for chronic viral hepatitis: Examination usually reveals no pathological signs or minimal signs of liver disease. Some patients have mild hepatomegaly or enlargement of the left side of the liver. Advanced stages of fibrosis can cause spider angiomas and palmar erythema. Except in cases of hepatic dysfunction or severe exacerbation, icterus of the sclera and skin is a rare manifestation. The development of cirrhosis is usually accompanied by jaundice, as well as splenomegaly, thrombocytopenia, increasing muscle weakness, ascites, peripheral edema and gynecomastia.

Elevated titers of autoantibodies, such as antinucleic, anti-smooth muscle, antibodies to liver/kidney microsomes and others, are observed infrequently in patients with chronic hepatitis. 20-25% of patients with CHB and CHC have consistently normal liver functions, monitored at regular intervals of 6 months.

Liver histopathology

The expansion of the portal tracts as a result of monocyte infiltration of immunoinflammatory origin is a sign of pathomorphological manifestations of chronic hepatitis. The border plate remains intact during the initial stages of chronicity. The next step is damage or death of hepatocytes at the interface between the portal tract and the lobule. This results in partial or partial necrosis, which compromises the

integrity of the lamina limita. Inflammation spreads to the periportal lobules, where scattered centers of infiltration and focal necrosis of hepatocytes are present. While portal fibrosis and connective tissue septa emerging from the portal tract, forming pericellular and perisinusoidal fibrosis (stage 1 fibrosis), may already be present, the lumbar architecture is preserved. This histopathology is also observed in other diseases, such as primary sclerosing cholangitis, but on its own is not sufficient to diagnose CH. As the disease progresses, fibrosis spreads to adjacent portal tracts and central veins, forming portoportal (stage 2 fibrosis) and portocentral bridges and bridging necrosis (stage 3 fibrosis). It is portovenular fibrosis that is the most important damage that indicates the formation of cirrhosis. The lobular structure of the liver is disrupted with the formation of smaller false lobules (4 stages of fibrosis).

The main method for assessing the condition of liver tissue is liver biopsy. The study of biopsy material is necessary not only to confirm the diagnosis and exclude other types of CKD, but also to assess the extent of the lesion, that is, the stage of the pathological process, the activity of the immune-inflammatory reaction and the degree of chronicity [10,12,14,16].

Non-invasive diagnosis of liver fibrosis

Non-invasive methods for assessing liver fibrosis (AF) include ultrasound, computed tomography (CT), magnetic resonance imaging (MRI) of the liver, Doppler ultrasound (USD) of the vessels of the liver and spleen with the calculation of fibrosis and portal hypertension indices, which allows assessing the shape, size , structure, echogenicity, tissue homogeneity, etc. However, assessing the early stage of fibrosis using these instrumental methods is difficult, which reduces their diagnostic significance.

Quantitative determination of the severity of AF is carried out using three traditional methods: biopsy, indirect elastometry, laboratory indices. These studies (together) make it possible to evaluate the morphological changes in the structure of the liver, the stage of fibrosis and inflammatory changes, and repeated studies over the dynamics of the effectiveness of treatment (in particular, antiviral therapy for hCG) [11,22,26,29].

Liver biopsy, being the “gold standard” for determining AF, is not without its drawbacks: invasiveness, fragmentation of the resulting biopsy, difficulties in interpretation, requiring the participation of highly qualified specialists, contraindications (and often prejudice of patients), etc.

Direct methods for diagnosing AF include transient fibroelastometry using FibroScan, based on assessing the elasticity of the liver tissue in kPa, which increases with increasing stage of AF. The limitations of the method used are: narrow intercostal spaces, excess fatty tissue, ascites. Certain difficulties also arise in assessing the early manifestations of AF.

For laboratory diagnosis of AF, many indices have been developed based on a combination of indirect (laboratory indicators) markers of AF. About 40 different indices for determining AF and CP have been proposed (for example, the AST/ALT ratio, the age-to-platelet count ratio index, collagen, hyaluronic acid, etc.).

Concluding the presentation of the main provisions on epidemiology, diagnosis, and structural changes in the liver in chronic viral hepatitis, one cannot help but dwell on one extremely important and unresolved problem. We are talking about combined and multiviral liver lesions. At the end of the last and beginning of this century, several hepatotropic viruses of parenteral infection were discovered.

Using molecular biological and immunomorphological studies, we have discovered in the last five years that combined and multiviral chronic hepatitis has increased, and monoviral liver lesions have become very rare due to the lack of monitoring of the entire spectrum of parenteral hepatropic viruses. Non-hepatotropic viruses, especially the herpes group, are also involved in this process [13,15,19,23]. In such a situation, diagnostic, therapeutic and prognostic problems become more complicated, because the hepatotropic viruses present in the patient leave a mark on the immune system and destroy all the resources of the affected organ and the entire body. As a result, many questions arise for which there is no definite answer yet. Secondly, it is absolutely unknown to what extent coinfecting viruses affect the replication of the main most pathogenic viruses (HBV, HCV, HDV). Based on the fact that during multiviral infection there is an increase in the number of cases of latent HBV infection, as well as the appearance of latent forms of HDV and HCV infection, one can only assume that the interaction of several viruses inhibits replication. However, this requires special research on large clinical material.

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