Nazarova Nodira Sharipovna

Samarkand State Medical University

"Department of Dentistry of the Faculty of post-diploma education" t.f.d., associate professor

Shukurov Sherzod Shuxratovich

Assistant of the Department of Pediatric Dentistry of Samarkand State Medical University

Raxmonova Moxinur Baxronjon qizi

Clinical resident of the Department of Pediatric Dentistry of Samarkand State Medical University

PARODONT, WHICH OCCURS IN PATIENTS WITH CHRONIC VIRAL HEPATITIS DIFFERENTIAL APPROACH TO THE TREATMENT OF INFLAMMATORY DISEASES

ANNOTATION

As a rule, hepatitis is defined as chronic with a duration of the disease for more than 6 months, although these time frames are conditional. Hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most frequent causes of chronic hepatitis; 5-10% of cases of HBV infection (with or without co-infection with hepatitis D virus) and approximately 75% of cases of HCV infection go to chronic form. Hepatitis A and E viruses do not cause chronic hepatitis. Although the mechanism of the development of the chronic process is not completely clarified, liver damage is mainly determined by the body's immune response to infection.

Key words: approximately, idiopathic. In a high percentage, autoimmune hepatitis

Many cases are idiopathic. In a high percentage of cases of idiopathic chronic hepatitis, it is possible to detect pronounced signs of immune hepatocellular injury (autoimmune hepatitis), including the presence of serological immune markers; association with histocompatibility antigens haplotypes characteristic of autoimmune diseases (eg, HLA-B1, HLA-B8, HLA-DR3, HLA-DR4); prevalence of T-lymphocytes and plasma cells in histological preparations

of the affected areas of the liver; disorders of cellular immunity and immunoregulatory function in in vitro studies; association with other autoimmune diseases (eg, rheumatoid arthritis, autoimmune hemolytic anemia, proliferative glomerulonephritis), and a positive response to therapy with glucocorticoids or immunosuppressants. Sometimes chronic hepatitis has manifestations of both autoimmune hepatitis, and other chronic liver disorders (eg, primary biliary cirrhosis, chronic viral hepatitis). These states are called cross-over syndromes.

Many medications, including isoniazid, methyldopa, nitrofurans and sometimes paracetamol, can cause chronic hepatitis. The mechanism of hepatitis development depends on the drug and may include a modified immune response, the formation of cytotoxic intermediate metabolites or genetically caused metabolic disorders.

Other causes of chronic hepatitis include alcoholic hepatitis and non-alcoholic steatohepatitis. More rarely, the cause of chronic hepatitis is a deficiency of α 1 -antitrypsin or Wilson's disease.

Previously, chronic hepatitis was classified based on the histological pattern and chronic persistent hepatitis, chronic lobular and chronic active hepatitis were isolated. The latter classification takes into account the etiology, the intensity of inflammation and necrosis (severity), as well as the degree of fibrosis (stage), determined by histological examination. Inflammation and necrosis are potentially reversible; fibrosis is usually irreversible.

Clinical manifestations are different. Approximately one-third of cases develop after acute hepatitis, but more often gradually. In many patients, the disease is asymptomatic, especially with chronic HCV infection. Often there are signs such as malaise, anorexia and fast fatigue, sometimes with subfebrile temperature and undefined discomfort in the upper abdomen. Jaundice is usually absent. Often, especially with HCV infection, the first clinical manifestations are signs of chronic liver disease (eg, splenomegaly, vascular spiders or asterisks, palmar erythema, pain in the right side). In some patients with chronic hepatitis, cholestasis can develop. In the autoimmune process, especially in young women,

the manifestation of the disease can involve virtually any body system and include signs such as acne, amenorrhea, arthralgia, ulcerative colitis, pulmonary fibrosis, thyroiditis, nephritis and hemolytic anemia.

Chronic HCV infection is sometimes accompanied by lichen planus (Wilson's lichen), skin-mucous vasculitis, glomerulonephritis, late cutaneous porphyria and, possibly, non-Hodgkin's B-cell lymphoma. Approximately 1% of patients develop cryoglobulinemia with fatigue, myalgia, arthralgia, neuropathy, glomerulonephritis and skin rash (hives, purpura, or leukocytoclastic vasculitis); more characteristic is asymptomatic cryoglobulinemia.

Diagnosis should be expected in patients with similar symptoms, with occasional detection of an increase in the level of aminotransferases and if there is an indication in the history of acute hepatitis. Functional hepatic tests (if not previously studied) are investigated, which should include the determination of the level of ALT and ACT, alkaline phosphatase and bilirubin in serum. An increase in the level of aminotransferase is the most characteristic laboratory evidence. Although enzyme levels may vary, they are typically 100-500 IU / L. ALT is usually higher than ACT. Aminotransferase levels in chronic hepatitis can be normal if the course of the disease is stable, especially with HCV infection.

Alkaline phosphatase is usually normal or slightly elevated, but sometimes it can be noticeably high. Bilirubin, as a rule, is within the norm with a mild course and no progression of the disease. However, changes in these laboratory tests are not specific and may be a consequence of other diseases, such as alcoholic liver disease, recurrence of acute viral hepatitis and primary biliary cirrhosis.

If the results of laboratory tests confirm the clinical manifestations of hepatitis, serological tests for viruses are performed to exclude HBV and HCV. If these studies do not confirm a viral etiology, further research is needed. Initially, studies include the determination of autoantibodies, immunoglobulins, and the level of a1-antitrypsin. Children and adolescents undergo a screening examination for Wilson's disease with the determination of the level of ceruloplasmin. The detected increases in serum immunoglobulins suggest chronic autoimmune

hepatitis, but are not final. Autoimmune hepatitis is usually diagnosed by the presence of antinuclear antibodies (AHA) in titres more than 1:80 (in adults) or 1:20 (in children), anti-smooth muscle antibodies or antibodies to liver microsomes and kidney type 1 (anti-LKMI).

In contrast to acute hepatitis, a liver biopsy is necessary if there is a suspicion of chronic hepatitis. Certain cases of chronic hepatitis can be manifested only by insignificant hepatocellular necrosis and inflammatory cell infiltration, usually in the area of portal venules, with normal acinar architectonics and little fibrosis or no fibrosis at all. Such cases rarely appear clinically and, as a rule, do not transform into cirrhosis of the liver. In more severe cases, biopsy usually reveals periportal necrosis with mononuclear cell infiltration, accompanied by periportal fibrosis and proliferation of bile ducts of varying severity. Acinar architectonics can be deformed by zones of damage and fibrosis, sometimes obvious cirrhosis of the liver is combined with signs of ongoing hepatitis. A biopsy is also performed to assess the severity and stage of the disease.

In most cases, a specific cause of chronic hepatitis can not be established on the basis of a biopsy, although cases caused by HBV infection can be differentiated by the presence of hepatocytes such as "frosted glass" and special coloration of HBV components. Autoimmune hepatitis usually has more pronounced lymphocytic and plasma cell infiltration. Patients with histological but non-serological signs of chronic autoimmune hepatitis should be diagnosed with different variants of it; many of them can correspond to cross-over syndromes.

Serum albumin and albumin should be examined to assess the severity of the process; liver failure is characterized by a low level of albumin and prolonged PV. If symptoms or signs of cryoglobulinemia develop in chronic hepatitis, especially in chronic hepatitis C, cryoglobulin levels and rheumatoid factor should be investigated; high levels of rheumatoid factor and low levels of complement also suggest cryoglobulinemia.

Patients with chronic hepatitis B to exclude hepatocellular carcinoma should undergo an annual ultrasound and an analysis of the vagal a-fetoprotein, although

opinions on the profitability of such tactics diverge. Patients with chronic hepatitis C should undergo a screening test for HCC only in the case of liver cirrhosis.

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