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VARIANTS OF CLINICAL COURSE AND METHODS OF TREATMENT OF VIRAL HEPATITIS DELTA

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Abstract
Hepatitis D is an inflammation of the liver caused by the hepatitis D virus (HDV), which requires hepatitis B virus (HBV) for its replication. Hepatitis D infection cannot occur in the absence of HBV. HDV–HBV co-infection is considered the most severe form of chronic viral hepatitis due to more rapid progression towards hepatocellular carcinoma and liver-related death. Vaccination against hepatitis B can prevent HDV infection. HDV has recently been classified as carcinogenic to humans (class I) by the IARC mongraphprogramme, just like hepatitis B and C.

Keywords: Hepatitis D virus, RNA test, intoxication, splenomegaly, jaundice, arthralgia.

Introduction

Clinical manifestations of viral hepatitis Delta Hepatitis D occurs in both acute and chronic forms, caused by the hepatitis D virus (HDV), which requires the HBV virus for replication. Infection with the hepatitis D virus occurs only in the presence of the hepatitis B virus. This virus is more likely than other causative agents of viral hepatitis to cause fulminant hepatitis and cirrhosis of the liver. Every patient with a positive hepatitis B surface antigen (HBsAg) should be tested for hepatitis D virus. The first diagnostic step is often a hepatitis D antibody test, followed by an HDV RNA test. Hepatitis D is considered the most severe type of viral hepatitis, as it accelerates the progression of liver cirrhosis. In addition, the infection has also been associated with a significantly higher risk of developing HCC. Persistent HDV replication leads to cirrhosis at annual rates of 4% and HCC at annual rates of 2.8%. The severity of the disease depends on the host and viral factors. HDV infection can clinically occur in two variants: Co-infection with HBV and HDV. Co-infection involves simultaneous infection with both HDV and HBV, which leads to the development of acute hepatitis D and B. This type of infection is usually acute, self-limited and clinically indistinguishable from classical acute hepatitis B. A short prodromal period is noted: high fever; often migrating pain in large joints; increasing intoxication in the icteric period; often pain syndrome (pain in the projection of the liver or epigastrium); occurrence 2-3 weeks after the onset of the disease or clinical and laboratory exacerbation. The course is relatively benign, however, it can cause severe hepatitis with a high risk of developing fulminant hepatitis. Acute hepatitis B with Delta agent. The incubation period is no different from that of acute hepatitis without a D-agent, and lasts from 6 weeks to 6 months. The pre-icteric period is shorter and has a more acute course with early manifestations of intoxication. Hepatitis of mixed etiology is characterized by high temperature and pain in the liver in half of the patients. More often than with acute hepatitis without a D-agent, patients are bothered by migrating arthralgia of large joints. In the icteric period, subfebrile temperature persists or develops, which lasts

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for 7-12 days; intoxication continues to increase, pain in the right hypochondrium intensifies. Urticarial rashes and splenomegaly appear more often than with acute hepatitis without a D-agent. A significant feature of coinfection is the two-phase course of the disease with clinical and enzymatic exacerbation in more than half of the patients: usually on the 15-32 day from the onset of jaundice, there is an increase in general weakness, dizziness, pain in the right hypochondrium, an increase in the liver size and an increase in the level of aminotransferases. An increase in enzymes can occur without clinical manifestations. Biochemical parameters of blood serum reflect a pronounced cytolysis syndrome. Hyperbilirubinemia is persistent, persists longer, the maximum values of aminotransferase activity are higher than similar indicators in acute hepatitis B without a D-agent. Repeated exacerbation of the process is characterized by an increase in enzyme activity, mainly due to an increase in AST, while the de Ritis coefficient becomes more than 1.0. An increase in the thymol test is noted, which is not typical of acute hepatitis B without a D-agent. With an increase in the severity of the disease, the prothrombin index, sublimate test and the content of beta-lipoproteins decrease. Acute hepatitis B with D-agent occurs mainly in a moderate form, about 1/3 of patients suffer a mild form. In case of coinfection, there is a high risk of developing severe and fulminant forms of the disease. In case of liver failure with hepatic encephalopathy, the diagnosis is formulated as follows: acute hepatitis B with delta agent (coinfection) with hepatic coma. Thus, coinfection is characterized by high activity, two peaks of ALT and AST increase, a severe course than hepatitis B monoinfection, a rare frequency of storage (5-10%). HDV superinfection is infection with the hepatitis D virus of a person infected with the hepatitis B virus. Short incubation and pre-icteric periods (3-5 days) with high fever, severe intoxication, repeated vomiting, pain syndrome, arthralgia are noted. Characterized by severe jaundice, development of edematous-ascitic syndrome, severe hepatosplenomegaly, repeated clinical and laboratory exacerbations. In this variant, the development of a malignant (fulminant) form of the disease with a fatal outcome is possible. Unlike coinfection, spontaneous elimination of HDV occurs only in a small number of patients. As a rule, acute hepatitis with a short incubation period leads to chronic hepatitis D in 80% of cases. Wu et al. described three phases of HDV superinfection: acute phase, active HDV replication and suppression of HBV with high ALT levels;chronic phase, HDV decline and HBV reactivation with moderate ALT levels; and late phase, development of cirrhosis and hepatocellular carcinoma caused by viral replication or remission resulting from marked reduction of both viruses. Thus, HBV replication, although inhibited by HDV, appears to play an important role in maintaining HDV pathogenicity. Superinfection may cause acute failure of pre-existing chronic HBV liver disease or severe chronic active hepatitis rapidly progressing to cirrhosis. HDV suppresses HBV replication in most people infected with hepatitis D virus. This is evidenced by the absence of HBeAg in most patients (77%) and the presence of HDV RNA in 85% of cases. Acute Delta (super) infection. The incubation period is often shorter than in acute Delta (super) infection without D agent - from 1 to 2 months. The pre-icteric period is also much shorter and lasts 3-5 days. The disease begins acutely with the appearance of asthenovegetative and dyspeptic symptoms, intense pain in the right hypochondrium, repeated vomiting, and arthralgia in 30% of cases. Almost half of the patients have a fever, often above 38°C. In the pre-icteric period, various manifestations of edematous-ascitic syndrome can be observed. The icteric period is characterized by pronounced intoxication, increasing general weakness, nausea, and aversion to food. A feeling of heaviness in the right hypochondrium is disturbing. Swelling of the shins increases,

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sometimes joint swelling appears, and edematous-ascitic syndrome is determined. Fever for 3-5 days is characteristic of this variant of acute delta infection. Hepatosplenomegaly is observed in almost all patients. The spleen size increases significantly, in some cases more than the liver size, which is not typical of acute hepatitis B without a D-agent. Acute delta infection is characterized by early signs of chronic hepatitis. The protein-synthetic function of the liver is clearly impaired, as evidenced by a decrease in the sublimate test, the content of serum albumins with a significant increase in the gamma-globulin fraction of the protein spectrum in the early stages of the icteric period. The thymol test increases significantly. The activity of AST and ALT remains high longer than in acute hepatitis B without a D-agent. An essential feature of acute delta infection is the multi-wave nature of the disease with repeated clinical and enzymatic exacerbations, accompanied by fever for 1-2 days, edematous-ascitic syndrome and jaundice. In case of liver failure with hepatic encephalopathy, the diagnosis is formulated as follows: acute delta superinfection of a hepatitis B virus carrier with hepatic coma. The recovery period is longer than in acute delta superinfection without a D agent. Hospitalization and discharge of patients is carried out according to the same clinical indications as in acute delta superinfection without a D agent. Chronic infection causes accelerated progression of fibrosis and early decompensation in the presence of cirrhosis. Approximately 60-70% of patients with chronic hepatitis D develop cirrhosis, which usually develops within 5 to 10 years, but can occur 2 years after the initial infection. Compared with patients with HBV monoinfection, patients with HDV have a decreased platelet count, greater variations in endoscopy, and smaller liver sizes. Data analyzed in the Hepatitis Delta International Network (HDIN) registry showed that HDV is associated with impaired liver function, as evidenced by low platelet counts.

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