Clinical and Pathogenetic Aspects of Combined Hepatitis "B" And "C"

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ABSTRACT

In order to study the issues of pathogenesis, clinic and diagnosis of combined liver lesions with hepatitis B and C viruses, 38 patients with various forms of mixed hepatitis B + C were comprehensively examined. During the infectious process of combined HBV/HCV etiology, indirect signs of mutual inhibition of two hepatotropic viruses were observed. The severity of clinical, laboratory and histological changes in the liver in latent forms is mainly due to HCV infection; it is characterized by the rapidity of HBeAg/HBeAb seroconversion and the disappearance of HBV DNA from the patient's blood against the background of ongoing HCV persistence. Isolation of various variants of the course of mixed hepatitis is uninformative in relation to the outcome and prognosis of the disease. At the same time, a thorough assessment of the activity of viral replication of both pathogens determines the choice of rational therapy.

Keywords: Viral hepatitis, B+C mixed hepatitis, clinical and laboratory characteristics.

One of the characteristic features of hepatitis B and C viruses is the possibility of combined liver damage with the development of mixed hepatitis. Associated diseases occur when two hepatotropic viruses are simultaneously introduced into the human body or when one infection is superimposed on another. This is facilitated, first of all, by the common epidemiological characteristics of HBV and HCV infections [1,2].

The number of registered cases of combined hepatitis B + C in the general structure of parenteral viral hepatitis increases from year to year. Significant improvement in the etiological interpretation of viral hepatitis also affected the frequency of detection of combined HBV/HCV lesions.

Purpose of the study. The study of some issues of pathogenesis, clinic and diagnosis of combined liver lesions with hepatitis B and C viruses.

Materials and methods. 138 patients were under observation, of which 121 had mixed hepatitis B and C in a manifest (more often icteric) form, in 17 - in a latent one. The comparison groups consisted of 179 patients with monohepatitis B (manifest forms in 38, latent forms in 141) and 309 patients with monohepatitis C (manifest forms in 140, latent forms in 169). All patients were males aged 16 to 37 years (mean age 21.3±0.2 years).

Results. With latent forms of both mixed hepatitis and monohepatitis, complaints and objective manifestations of the disease were completely absent, with the exception of a

minimal increase in the liver in 1/3 of patients. Elevated levels of alonyl transaminase (Alt) were observed in 71% of patients (up to 4-5 norms on average), biochemical signs of dysproteinemia were detected in 88%. Noteworthy is the predominant registration of HCV RNA - 57% ($p<0.05$), as well as the absence of cases of simultaneous detection of DNA and RNA.

Manifest hepatitis B + C is clinically manifested by a short preicteric period (4-5 days) mainly of the dyspeptic type, a moderate increase in intoxication with the manifestation of jaundice, more often proceeded in mild (40.5%) and moderate (45.4%) forms. At the same time, an intermediate in severity position of combined hepatitis between monohepatitis B and C was noted. In particular, mild and moderate forms in hepatitis B were recorded in 17, 1 and 56.2%, respectively, and in hepatitis C - 72.7% and 27, 3%. Interesting results of a comparative analysis of virological indicators of indicators in the dynamics of manifest forms of mixed hepatitis B + C. During the peak of the disease, a significantly earlier onset of HBeAg/anti-HBe seroconversion was established, and hence the cessation of active HCV replication in hepatitis B + C in comparison with monohepatitis B (61.2 and 46.7%; p ... 0.05) . A possible reason for this could be the simultaneous presence in the patient's body of the hepatitis C virus, which to some extent inhibits the hepatitis B virus. In addition, there were statistically significant differences in the registration of serum HCV markers. First of all, a rarer indication in hepatitis B + C compared with hepatitis C is anti-HCVcoreIgM (13.2; 37.9% and 24.2%; $p<0.05$), and HCV RNA (15.7 and 24%; $p<0.05$) indicates active HCV replication. In turn, the presence of a depressing effect from HBV was also assumed. Detection of antibodies to the 4th non-structural protein, on average, in half of the patients with both hepatitis C (51.2%) and hepatitis B + C (56.1%) indicated the chronic nature of HCV infection. Taking into account the fact that during the height of hepatitis B+C, HBV DNA was detected in the blood serum of 70% of patients, while HCV RNA was found only in 16% ($p<0.05$), we can confidently speak about manifestations in combined HBV/ HCV - liver damage. The extremely rare simultaneous indication of the genomes of both viruses indicated the possibility of mutual suppression.

Control studies of serum HBV markers conducted during the convalescence period showed that HBeAg/anti-HBe seroconversion occurred in all patients with mixed hepatitis B HBeA .. continued to be detected (3.8%). Moreover, HBVAg (88.4 and 95.2%; $p<0.05$) and HBV DNA (23.9 and 52.4%; $p<0.05$) were registered significantly more frequently also in the comparison group. This indicated that the elimination of HBV from the patient's body occurred at a faster pace, while the presence of HCV in patients with mixed hepatitis during the convalescence period was generally comparable to that in monohepatitis C. In both groups, total antibodies continued to be detected, as well as with a similar frequency of RNA HCV (17.4 and 21.2%; $p<0.05$). Apparently, this situation was determined by the further persistence of HCV.

Thus, a comparative analysis of the results of serological and molecular - biological studies suggested that during the infectious process of combined HBV / HCV - etiology, there was a mutual suppression of the activity of hepatotropic viruses. At the same time, at the beginning, the hepatitis B virus dominated, which to a greater extent was due to the manifest clinical picture of the disease. Subsequently, the elimination of HBV from the patient's body occurred, and at a faster pace in the presence of the hepatitis C virus. The subsequent course of the HCV infection itself continued in accordance with its inherent patterns. Of all the immunological studies we conducted, the results of the study of some cytokines should be noted. Thus, in all compared groups of patients, hyperproduction of pro-inflammatory cytokines was observed: interleukin (IL) - 1β (hepatitis B + C - 4.6 ± 0.7 pg / ml, hepatitis B - 4.4 ± 0.6 pg-ml, hepatitis C - 2.2±0.8 pg/ml). In addition, control immunological studies during the convalescence period showed that clinical recovery, primarily in hepatitis B + C and C, was not accompanied by the normalization of

these immunopathological changes. A possible reason for this could be the continued persistence of HCV infection.

Conclusions. It is likely that the simultaneous introduction of two hepatotropic viruses into the body causes an earlier manifestation, often with an exacerbation (reactivation) of chronic hepatitis. The obtained ultrasound data compared with the results of virological studies made it possible to think that in 34% of patients clinically manifest acute HBV / HCV - coinfection is most likely, and in 66% - HBV superinfection against the background of chronic hepatitis C. To determine exactly which virus infection occurred earlier, and which later, especially in the presence of fibrosis in the liver, is extremely difficult, and given the similarity and outcome of the disease, such a definition is of little use. The literature data, as well as our results, indicate that acute hepatitis B + C, acute hepatitis B against the background of chronic HCV infection, and even acute hepatitis C against the background of chronic HBV infection ends, as a rule, with HBV seroconversion and the formation of chronic hepatitis. C. It seems practically important to study the replication activity of both hepatotropic viruses during the course of the disease in order to prescribe antiviral and pathogenetic therapy, the choice of which is primarily determined by the dominant role of one or another pathogen.

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