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MICROBIOLOGICAL MAIN CHRACTERISTICS OF VIRAL HEPATITIS

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Article history:		Abstract:
Received:	July 6th 2022	The goal of this is to determine the etiological structure of hepatitis B, the level and, in part, the clinic of combined (mixed) forms of hepatitis B in children
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INTRODUCTION

Beginning in the second half of the 20th century , viral hepatitis (HB) has become the most common infections , second only to acute respiratory viral infections and, in certain periods, acute intestinal infections. The tension of the epidemiological situation persists at the beginning of the XXI century. The number of registered cases of viral hepatitis is 50-60 times less than the number of flu and acute respir- atory infections, but their average duration is 3-5 times longer and the severity of the course is much more pronounced, not to mention the tendency of some forms of viral hepatitis to a chronic course, the development of cirrhosis and even liver can- cer [1, 2, 3, 5].

Uzbekistan, according to WHO, belongs to the territories endemic for viral hepatitis. In the dynamics of the incidence of viral hepatitis for 2000-2019, there have been years of rise and fall in the incidence. During the observed period, relatively high incidence rates of viral hepatitis were observed in 2000, when incidence rates were 882.0 per 100 thousand of the population, a dynamic decrease in the inci- dence rate was observed in subsequent years, however, in 2005 and 2007 there was an abrupt increase in the incidence of viral hepatitis. At the same time, the inci- dence of viral hepatitis «B. was characterized by a dynamic decrease, from 155.9 in 2000 to 29.5 in 2001 per 100 thousand of the population. In 2001, the first among the Central Asian states in the Republic of Uzbekistan was introduced into the practice of public health services the immunization of newborns against viral hepa- titis •B., as a result of which the incidence of viral hepatitis B, as a whole, de- creased in comparison with 2000 in 2009 almost 60 times, accounting for 2.6 against 155.9 per 100 thousand people [4, 5, 6].

There are a number of unresolved issues, in particular, specific diagnostic methods (ELISA) are not everywhere introduced, therefore, mainly diagnoses of hepatitis A and hepatitis B are established on the basis of clinical, biochemical and epidemiological data, without taking into account other etiological forms of hepatitis B, mixed (mixed) hepatitis is not detected.

GOAL

Determine the etiological structure of hepatitis B, the level and, in part, the clinic of combined (mixed) forms of hepatitis B in children.

RESEARCH MATERIAL AND METHODS

Under observation were 1,400 children with acute viral hepatitis from 0 to 14 years old who were admitted to hepatitis wards during the year. A continuous serological examination was conducted to identify the etiology of hepatitis and the level of mixed forms among them. Known hepatitis virus markers were determined in blood serum in an enzyme-linked immunosorbent assay with test systems of ROSH JSC (Russia-Switzerland) and the NPO Diagnostic Systems (Nizhny Novgorod, Russia).

THE RESULTS OF THE RESEARCH AND THEIR DISCUSSION

A planned serological examination of 400 children admitted to the hepatitis de- partments during the year revealed the etiological structure of hepatitis B in chil- dren. At the same time, the specific gravity of GA was 37.3 %, HS - 8.01 %, HS- 9.5 %, GE – 1.0 % and TTV – 1.6 %, combined forms (HB-mixed) were revealed in 40.0 %. Of these, 10.3 % accounted for a combination of HS with HS, 7.0 % – HA with HS, 5.8 % - HS with HD. 3.5 %– HS with TTV, 3.0 %– HS with GE. 2.0 %- GW with TTV and 1.5 - GW with GE. In 7.5 % cases, the detection of concomitant in- fections with the identification of markers of 2, 3 and even 4 types of hepatitis B virus.

In accordance with the objectives of the work, 32 cases verified by the detection of serological markers of both infections (HBsAg. Anti-HBc, IgM. HBEAG antenna- HCVc test systems of the second generation) were monitored to study the clinical picture of mixed B + C infections. Control groups with mono-infections comprised 40 children patients with OV and 56 – with OVHS.

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In the epidemiological history of HBV patients, 9.3 % of children had a transfu- sion of blood and its components, 18.1 % had I information about various parenter- al I manipulations (injections, blood sampling, dental procedures, circumcisions, etc.). Moreover, in 18.2 % of children with hepatitis B there was information about contact with a patient with HB virus infection (acute chronic hepatitis B, HBsAg carriage) in the family. At the same time, in 53.4 % of patients, there is no infor- mation on parenteral manipulations and contact with patients with HB. In the group of children with mixed hepatitis B+ HS infection, the epidemiolog- ical history had similar data with the group of children with hepatitis B (transfusion of blood and its preparations - 78.14 various parenteral manipulations – 12.5 %, family contact - 6.2 %, and not there was information in 3.1 %) of children). With regard to the severity and course of the disease, with mixed B'C infection compared with patients with HBV, moderate to severe forms were somewhat more frequent (P < 0.05). The difference in severity compared with the group of patients with HCV showed a significant increase (P< 0.001) severe forms of the disease. Thus, with a mixed course of HBV and HCV, it was possibleto identify a num- ber of features in the clinical course, the severity of the disease, the severity of individual clinical symptoms, which must be taken into account in practical work. Interesting results of a comparative analysis of virology cal indicators in the dy- namics of manifest forms of mixed hepatitis B+ C.

During the height of the disease, the onset of HBcAg / anti-HBc seroconversion was established significantly earlier, and hence the cessation of HBV active replication in hepatitis B + C compared 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 hepatitis C virus, which to some ex- tent inhibits the hepatitis B virus. In addition, statistically significant differences in the registration of serum HCV markers were observed. First of all, a rarer indication for hepatitis B + C compared with hepatitis C. anti HCVcorclgM (13.2; 37.9 % and 24.2 %; p < 0.05), and HCV RNA (15.7 and 24 %; p « 0.05) indicating active HCV replication. In turn, they suggested the presence of a depressing effect on the part of HBV. The detection of an average of half of patients with both hepatitis C (51.2 %) and hepatitis B C (56.1 %) antibodies to the 4th non-structural protein indicated the chronic nature of HCV infection, Considering the fact that during the period of hep- atitis B + C high, HBV DNA was detected in blood serum in 70 % of patients, while HCV RNA in only 16 % (p < 0.05), we can confidently talk about the dominant role of HBV in development clinical manifestation with combined HBV / HCV liver damage. The extremely rare simultaneous indication of the genomes of both viruses indicated the possibility of their mutual suppression. Control studies of serum HBV markers during the convalescence period showed that HBEAB / anti HBe serocon- version occurred in all patients with mixed hepatitis B + C, while in some patients with monohepatitis B HBCAC continued to be detected (3.8 %). Moreover, HBsAg (88.4 and 95.2 %; p < 0.05) and HBV DNA (23.9 and 52.4 %; p< 0.05) were record- ed much more often also in the comparison group. The ego testified that elimination from the patient's body took place at a faster, faster pace with the simultaneous presence of HCV in patients with mixed hepatitis during the convalescence period, which was generally comparable with that of monohepa titis C. In both groups, total antibodies continued to be detected, as well as with a similar HCV RNA frequency (17.4 and 21.2 %; p < 0.05). Apparently, this sit- uation was determined by the further persistence of HCV. Thus, a comparative analysis of the results of serological and molecular biologi- cal studies suggests that during the infection process of the combined HBV / HCV etiology there was a mutual suppression of the activity of hepatotropic viruses. At the same time, the hepatitis B virus dominated in the beginning, which was the reason for the manifest clinical picture of the disease. Subsequently, HBV was eliminated from the patient's body, more rapidly in the presence of hepatitis C.

The subsequent course of HCV infection itself continued in accordance with its inherent patterns. Among the combined HA + HS, the combination of OVGA + OVGV was detected in 39 (18.5 %) OVGA in the presence of HBSA.- carriage in 70 (33.2 %) and in OVGA in the presence of HBVA in 102 (48.3 %). An analysis of the severity of the disease revealed that with GA in HBsAg carriers. there was no difference in severity compared with the group of patients with OVHA (PO.G5). as for the combined course of OVGA with OvGv and OVG A with HVGV. then an increase in medium-heavy (re- spectively 46.0 and 42.0 % versus 37.2 %) and severe forms (respectively 13.0 and 9.0 % versus 2.7 %) is revealed here

In rebuild; In the combined period of OVGA + OVGV and OVGA combined with HBsAgc carriage, a significant slowdown in recovery was noted with a more frequent formation of a protracted course of the disease (10.0 and 8.0 % versus 4.5 %; P < 0.001). With a long-term (2-year-old chic) dispensary, in no one group of patients did the chronization of the

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process occur. Of 294 patients undergoing OVGV. 51 re- vealed an acute delta virus infection. In 16 of them (5.4), co-infection was noted, in 45 (15.4 %) superfection in carriers of HBsAg. This separation using modern methods of laboratory verification of the diagnosis allowed us to identify a number of clinical features and differences in IOP in co-infection and superinfection. In the first case, the percentage of severe forms increased (19.0 %). Fulnnant hepatitis oc- curred in 25.0 % of cases, with three deaths (19.0 %). Chronic hepatitis was ob- served in 12.0 % of patients, versus 9.1 % with HBV. In the second case, fulminant hepatitis occurred in 13.0 % of patients with three fatal outcomes (7.0 %), and the formation of chronic hepatitis occurred in 76.0 % of cases. Regarding the nature of the relationship between HBVand HDV, it is determined not only by the use of HBSAg to form the outer shell of HDV, but also by other, not completely prevale nt interactions, so HDV inhibits HBV replication, which leads to a decrease in the ex- pression of HBsAg and HBsAg and inhibition of the activity of DNA polymer rase dur ing acute infection. One possible announcement of this fact is data'on the stimula- tion of HDV intracellular synthesis of interferon, which inhibits the replication of HBV. Thus, a deltaviral infection, both with co-infection and superinfection, can cause otia glaucous and prolonged course of hepatitis B.

CONCLUSIONS

Thus, the use of the highly sensitive IFD method allows us to clearly conduct the etiological diagnosis of hepatitis B in children and, thereby, to deter mine the true ratio of hepatitis B in children, the presence of combined forms of the disease. This allows us to predict the incidence of various forms of hepatitis B in the region and contributes to rational measures to reduce the incidence of hepatitis B.

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