CLINICAL COURSE AND OUTCOMES OF CONGENITAL CHRONIC VIRAL HEPATITIS B AND C IN CHILDREN

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КЛИНИЧЕСКОЕ ТЕЧЕНИЕ И ИСХОДЫ ВРОЖДЁННОГО ХРОНИЧЕСКОГО ВИРУСНОГО ГЕПАТИТА В И С У ДЕТЕЙ

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Annotation. Viral hepatitis is a serious medical and social problem. Socially significant diseases are one of the main threats to the health of the entire population and especially adolescents and young people. They are difficult to treat, pose a risk of infecting other people and can be fatal. To prevent the spread of socially significant diseases, it is necessary to search for methods of early diagnosis, means and forms of primary prevention as the most effective methods of combating the spread of these pathologies. Viral hepatitis is one of the most pressing problems of our time [3, 6, 7].

Key words: viral hepatitis B and C, clinical course, outcome, diagnostics.

Аннотация. Вирусные гепатиты представляют собо▫й серьезную медицинскую и соци▫альн▫ую проблему. Социально значимые забо▫лева▫ния – это одна из осно▫вных угроз здоровью всего насе▫лени▫я и прежде всего подр▫остк▫ов и молодёжи. Они труд▫но поддаются лечению, представляют опас▫ност▫ь заражения других людей и могу▫т приводить к летальному исхо▫ду. Для предотвращения распространения соци▫альн▫о значимых заболеваний, необходим поис▫к мпособов ранней диагностики, сред▫ств и форм первичной проф▫илак▫тики как наиболее эффективных мето▫дов борьбы с распространением данн▫ых патологий. Вирусные гепатиты явля▫ются одной из самых акту▫альн▫ых проблем современности [3, 6, 7].

Ключевые слова: вирусные гепатиты В и С, клиническое течение. исход, диагностика.

Introduction. The problems of early diagnosis, clinical course, choice of treatment method and outcomes of perinatal viral hepatitis B and C in children remain complex and poorly understood [2, 18]. Perinatal infection of newborns with hepatitis B virus (HBV) or C virus (HCV) can occur in utero, during passage through the birth canal or after birth during care [1, 8]. Introduction. The problems of early diagnosis, clinical course, choice of treatment method and outcomes of perinatal viral hepatitis B and C in children remain complex and poorly understood [2, 18]. Perinatal infection of newborns with hepatitis B virus (HBV) or C virus (HCV) can occur in utero, during passage through the birth canal or after birth during care [1, 8].

The aim of the study: to analyze the results of observation of children with CHB and CHC, born to mothers infected with hepatitis B and/or C viruses, and to study the characteristics of the clinical course and outcomes.

Materials and methods of the study. We observed 164 children with chronic hepatitis B (CHB) and chronic hepatitis C (CHC) aged 1–18 years, of whom 78 (48%) had perinatal infection (CHB – 46; CHC – 32). Chronic hepatitis B and C were characterized by subclinical signs, with minimal fibrosis, in the immune-active phase of HBV (69%) and high HCV viremia (56%) [2, 4, 9]. The following was performed: collection of anamnesis; clinical, immunological and virological studies with determination of HBV and HCV markers by ELISA; determination of DNA HBV RNA HDV, RNA H1C by PCR Real Time, HC genotype. Ultrasound of internal organs; determination of the degree of fibrosis by elastography (FibroScan) or FibroTest; puncture biopsy of the liver. Patients with an active viral process and the stipulated criteria received antiviral therapy [1, 16, 19].

All patients were observed in the clinic for 5 years. The basis for establishing the perinatal route of infection of the child was the presence of confirmed CHB (and in

some cases, HBsAg carriage) or CHC in mothers before pregnancy with the child [2, 10, 12].

Results and discussion. Perinatal infection was detected in 48% (CHB - 46 and CHC - 32). The average age of children was 10.5 ± 0.71 years with a predominance of males. The average age of children at the time of primary detection of chronic hepatitis was 4.5 ± 0.71 years. All mothers of the observed children had confirmed CHB (or chronic carriage of HBsAg) and CHC before pregnancy with this child. 26% of children with CHB and 71% with CHC at birth were vaccinated against HBV according to the 0-1-6 schedule. Primary chronic viral hepatitis B in 39% and C in 66% of children was diagnosed at the age of 1-5 years, in one third of patients - at the age of 6-10 years. Only in 11% of children with CHB and 3% with CHC was the infection diagnosed in the first year of life. CHB in 12 patients was combined with the delta virus (superinfection). Clinical signs of primary chronic hepatitis B and C were asymptomatic, with palpable minor hepatomegaly up to 2-3 cm below the costal margin of average density in one third of patients, and pronounced hepatomegaly up to 4 cm was noted in 53% with CHB. CHB in 70% and CHC in 50% were accompanied by chronic gastroduodenitis, acalculous and calculous cholecystitis. The activity of the viral process in 57% of children with CHB and in 50% with CHC was accompanied by hyperenzymemia ($p < 0.05$). High viral activity was characteristic of 69% of children with CHB with positive HBeAg and prevalence of the immunoactive phase ($p < 0.05$). The immunotolerant phase of the chronic viral B process with DNA HBV within the range of > 109—< 1012 uI/ml was found in 11%, minimal viremia and DNA HBV < 2000 uI/ml — in 20% and absence of replication — in 15%. CHB with delta virus was characterized by low viral load of HBV, absence of HBeAg, high viremia of HDV > 106 - <109 uI/ml and pronounced cytolysis.

In 35% of children with CHB who did not receive antiviral therapy, a decrease in viremia was observed, while in 24% it increased. Spontaneous seroconversion of HBeAg/antiHBe was detected in 1 (2%) observed child aged 10 years.

In CHB F0, the degree of fibrosis was detected in one third, while F1 - in 65%, F2 - 13% and F3 - in 13% of children. In 2 cases of CHB with delta agent, the degree of fibrosis during observation increased to F2 (1) and F3 (1).

For CHC, high replication of HSC > 600,000 ul/ml, minimal hyperenzymemia (50%) and prevalence of genotype 1b (88%) were characteristic. Genotype 3a of HSC was detected in 3 children, and genotype 2 - in 1 child. In all children with CHC and their infected mothers, the genotypes of the virus coincided. In 23% of children with CHC, liver fibrosis was absent (F0), minimal fibrosis F1 was detected in 45%, and F2 - in 12%.

All children with CHB and CHC in the active phase received symptomatic treatment and hepatoprotectors. In 23 children with CHB, monotherapy with alpha-2b

interferon or pegylated alpha-2b interferon in combination with lamivudine was performed, with remission achieved in 83% of cases. 24 children with CHC received combination therapy with pegylated alpha-2b interferon and ribavirin for 24-48 weeks, depending on the genotype of the virus, with remission occurring in 63%.

Conclusion. Our results allow us to conclude that 48% of children with CHB and CHC were infected perinatally. The diagnosis of primary chronic viral hepatitis B (39%) and C (66%) in perinatally infected children was confirmed at the age of 1-5 years. Chronic hepatitis B and C were characterized by subclinical signs, with minimal fibrosis, in the immunoactive phase of HBV (69%) and high HCV viremia (56%). Etiopathogenetic therapy contributed to the development of clinical and virological remission in 83% of children with CHB and in 63% with CHC.

Children born to HBV-infected mothers with HBeAg and viremia require vaccination against HBV according to the accelerated schedule 0-1-2-12 in the first 12 hours with the introduction of specific immunoglobulin. For early detection of possible primary chronic hepatitis, children born to HCV-infected women require continuous monitoring during the first 2 years of life. Given that maternal antibodies to HCV can persist for up to 1.5 years, it is advisable to examine HCV RNA to exclude infection when clinical signs appear.

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