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The clinical and pathogenetical analysis of the lethal case of viral hepatitis c in child

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During the last years the problem of hepatitis C (HC) for medical science and practical public health has grown. According to official statistics in Ukraine, the infected adult population with HCV on an average is 2%, however in reality the level of morbidity of HC is 5-6 times higher [1].

According to the WHO forecast, in the next 10-20 years HC will become the main problem of public health, as is expected the result of this considerable number of patients with liver cirrhosis can increase to 60% and the death rate by 2-3 times [3]. Consequently, the study of clinical and pathogenetical features of HC in children with a severe clinical course and lethal outcome for determine the direct cause of death is an actual problem in medicine.

Aim: study of clinical and pathogenetical features of the lethal case of HC in the child.

Materials and methods. The clinical and pathomorphological analysis of the lethal case of HC in the child was conducted. The HC diagnosis was confirmed by the presence of anti-HCV.

Results and their discussion. Patient S., 3,5 months old, was hospitalized in the Vinnitsia's children's regional clinical infectious hospital with complaints of yellow coloring of skin, sclera and mucus membranes, darkening of urine, acholic feces, repeated vomiting, fever up to 39^0C, languor, violation of sleep. From anamnesis the disease is known, that during the last week the child was treated in the regional hospital with pneumonia. However a few days ago the mother noticed that the child had yellow coloring of skin and sclera, darkening of urine, hypocholia of feces, hemorrhagic syndrome. The well being of the child progressively got worse, therefore the child was transferred the Vinnitsia's regional hospital.

This child was born from 1st normal course pregnancy. The new-born period was complicated by ABO hemolytic anemia, for this blood transfusions were performed three times and other parenteral manipulations. Objectively: the child's state at hospitalization was very severe, expressed jaundice, hemorrhagical rash on the skin, hepatosplenomegaly.

The deceased child had considerable pathological changes in the hemogram: normochromic anemia, considerable thrombocytopenia - till $35 \cdot 10^9$/l. In the biochemical blood test hyperbilirubinemiya (106-137 mcmol/l) was marked, both due to the level of conjugated (94-73 mcmol/l) and unconjugated (12-64 mcmol/l) bilirubin. It is necessary to notice the decrease of the level of conjugated and the increase of unconjugated bilirubin before death, which testifies of the development of total hepatic insufficiency and violation of uptake and conjugation of unconjugated bilirubin. The syndrome of cytolysis was confirmed by the rise of ALT (2,8) and AST (2,0) activity, thymol test was 2,6; hypoglycemia (1,5 mmol/l) which was related to the violation of glycogenesis in the liver and exhausting of glycogen reserve. As a result of the violation of proteinsynthesis function of the liver, the indexes of coagulation were substantially violated: prothrombin index - 56%, fibrinogen - 0,77 g/l; time of plasma recalcification - 135sec. On the basis of clinical and laboratory data the clinical diagnosis was made: HC, severe course; acute hepatic insufficiency, hepatic coma; III phase of DIC syndrome.

In spite of the conducted intensive treatment, on the 3rd day of hospitalization, the child died. Progressive hepatic insufficiency with appearances of the expressed hemorrhagic syndrome, edema of cerebrum was the direct cause of death.

The obtained results coincide with data [2], in accordance with which, the main cause of death in acute viral hepatitis is progressive hepatic insufficiency, which is quickly complicated by a hepatic comma that leads to death in 2-3 days. All signs of hemorrhagic syndrome can be of great significance in thanatogenesis in a hepatic comma. We can not consider hepatic insufficiency as the cause of death in viral hepatitis in the case of absence of jaundice and hemorrhagic syndrome [2].

Consequently, the conducted research outlined the clinical course of the malignant stage of HC in this child demonstrating expressed signs of cholemia and acholia with growth of hepatic-cellular insufficiency which caused the development of hepatic comma and the death.

Conclusions

1. HC in children is a serious medical and social problem and requires further study with the purpose of prophylaxis and improvement of medical tactics for prevention of the development of dangerous complications.

2. The cause of death in HC is progressive hepatic-cellular insufficiency, of which the main dangerous complication is the development of hemorrhagic syndrome.

Literature

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