

## DIAGNOSTICS OF BILIARY ATRESIA IN CHILDREN USING INSTRUMENTAL EXAMINATION METHODS

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**Introduction.** Biliary atresia is one of the most difficult problems of modern neonatology. Despite the fact that the first reports of infants with biliary atresia appeared in the 19th century, this pathology is still considered by all researchers as a complex and difficult to correct anomaly of the biliary tract, with an unfavorable prognosis. This pathology is the most common cause of cholestasis in newborns and the most common indication for liver transplantation in pediatrics. The disease is based on progressive obliteration of the extrahepatic bile ducts with gradual involvement of the intrahepatic biliary tract and the formation of biliary cirrhosis of the liver.

**Purpose of the study.** Study of clinical and laboratory manifestations of biliary atresia in children at different age periods.

**Material and methods of research.** A total of 70 children aged 10 days to 6.5 months with biliary atresia were examined. Anamnestic, clinical, laboratory and instrumental manifestations of the disease were analyzed. Most children were born full-term with anthropometric indices corresponding to the norm. Jaundice appeared on the 2nd-4th day of life, 52% of patients had a "light interval" in the course of jaundice. The earliest clinical sign of the disease was discolored stool, the appearance of which in most cases (94%) was preceded by the passage of meconium. Absence of hepatomegaly at birth with subsequent gradual increase in liver size by the end of the 1st month of life was characteristic of all children. Clinical and laboratory manifestations of cholestasis and transaminase indices increased in children after 1 month of life. By this age, hemorrhagic syndrome was manifested in 5 (7%) patients. The indicators of the liver synthetic function remained normal up to 3.5-4 months of life. According to the ultrasound examination data, the gallbladder was visualized as a "cord" (77%) or was not visualized (23%). Without surgical treatment, signs of biliary cirrhosis formation appeared by 5-6 months of life.

**The results obtained and their discussion.** Morphological studies of liver biopsy revealed a direct dependence of the degree of organ damage, including bile duct proliferation, signs of fibrosis and cholestasis, on the patient's age. We believe that the dynamics of other clinical and laboratory parameters also serve as a reflection of progressive liver damage with gradual formation of cirrhosis during the first 4-5 months of life. In most children under 2 weeks of age, transaminase activity was within normal limits, in other groups it was increased.

The degree of increase in enzyme activity was directly proportional to age, with the exception of children older than 5 months, who had lower values, which was also due to the formation of biliary cirrhosis. Indicators of liver synthetic function remained within normal limits in all groups, with the exception of children older than 5 months. According to the ultrasound examination, hepatomegaly was absent in all children under 2 weeks of age; in older children, sonographic signs of hepatomegaly

were directly proportional to the child's age. In 2 children aged  $4.0 \pm 0.5$  months and in most children over 5 months, signs of portal hypertension (ascites, esophageal varices, portal blood flow disorders, splenomegaly) were detected. Our results do not contradict the available literature data. All children, regardless of age, showed a significant increase in the content of total lipids in feces, mainly due to fatty acids and triglycerides. These changes reflect a violation of the processes of digestion and absorption of fats, associated with the absence of bile in the intestine. The severity of steatorrhea depended on the type of feeding and did not depend on the age of the patient.

**Conclusion.** Clinical and laboratory manifestations of cholestasis were minimally expressed up to 1 month of life and increased thereafter. After 1 month of life, a delayed moderate increase in the activity of transaminases was observed, gradually increasing in dynamics. Indices of the synthetic function of the liver in all patients remained normal up to 3.5–4 months of life. By the age of 1 month of life, the development of hemorrhagic syndrome (intracranial hemorrhage - in 2 children, bleeding from the mucous membrane of the gastrointestinal tract - in 3) was noted, caused by a deficiency of vitamin K-dependent blood clotting factors.