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CLINICAL AND LABORATORY CORRELATION OF BILIRUBIN INDUCED NEUROLOGICAL DYSFUNCTION IN NEONATAL CHILDRENS

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Annotation. According to the WHO, complications of pathological jaundice in children are a leading cause of disability and mortality and continue despite universal newborn screening. Further research into the neurological and clinical aspects of bilirubin encephalopathy and jaundice is needed. This article reviews various clinical and pathological conditions associated with jaundice and bilirubin encephalopathy, as well as diagnostic tests used in patients with these conditions. The authors aim to convey to pediatric neurologists and neuropathologists the importance of early diagnosis and prevention of the disease. Acute bilirubin encephalopathy (ABE) is a cause of morbidity and mortality worldwide, particularly among infants and young children.

Key words: bilirubin encephalopathy, reflex, BIND, dystonia, correlation, autopsy, basal ganglia.

Abstract. According to WHO, complications of pathological jaundice in children are a leading cause of disability and mortality and continue despite universal newborn screening. More research is needed into the neurological and clinical aspects of bilirubin encephalopathy and jaundice. This article reviews the various clinicopathological conditions associated with jaundice and bilirubin encephalopathy, as well as the diagnostic tests used in patients with these conditions.

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The authors try to convey to pediatric neurologists and neuropathologists the importance of early diagnosis and prevention of the disease. Acute bilirubin encephalopathy (ABE) is a cause of morbidity and mortality worldwide, particularly among infants and young children.

Key words: bilirubin encephalopathy, reflex, BIND, dystonia, correlation, autopsy, basal ganglia.

Introduction. High levels of bilirubin are the cause of kernicterus (KBE). Too much bilirubin in the body can cause jaundice, or yellowing of the skin. [1] Unless the material is bound to albumin (a protein) in the blood, when bilirubin levels are too high in a newborn, it leaves the blood and accumulates in brain tissue. This can lead to hearing loss and brain damage. The yellow color caused by bilirubin is called "kernicterus." After autopsy, it is seen in certain areas of the brain. [2, 3] Research has shown a correlation between blood bilirubin levels and damage to the nervous system, particularly the brainstem, basal ganglia, and cerebellum. This can lead to the development of acute bilirubin encephalopathy (ABE). It plays a significant role in the development of lifelong disabilities such as cerebral palsy (CP) and hearing impairment in newborns. Furthermore, studies show that drug treatment is effective if EB is detected and treated early. [3] Without standardized tests, EB may be difficult to detect in the early stages, and treatment becomes more difficult if it is detected late. Therefore, it is important to develop standardized laboratory tests and accurate neurological examinations for better diagnosis and treatment of EB. Previous studies have shown that the bilirubin-to-albumin (B/A) ratio and total serum bilirubin (TSB) are early indicators of poor outcome and are closely associated with the development of PE. may be performed.[4] In addition to the biochemical tests mentioned above, we also perform clinical findings such as neurological and clinical tests, neurosonography, and magnetic resonance imaging (MRI). The BIND score has also been used to determine the prognosis of diseases in the early stages. These tests can detect any changes in the structure and function of the brain that may indicate the severity of the disease. In addition, they are able to identify any underlying conditions that may contribute to BE, such as infections, inflammation, or trauma.[5,6] Worldwide, morbidity and mortality from acute bilirubin encephalopathy (ABE) is particularly common in infants and children. Clinical manifestations. Transient bilirubin encephalopathy in infants may be reversible if bilirubin toxicity begins early. Acute bilirubin encephalopathy in neonates with moderate to severe hyperbilirubinemia can progress to kernicterus or reverse the process. Despite significant risk factors for the development of high-level hyperbilirubinemia in patients, timely and appropriate treatment with intensive phototherapy and blood transfusions can completely eliminate the toxic effects of bilirubin on the brain.Posttransfusion lethargy in infants and persistent damage to the auditory brainstem are clinical manifestations of this condition, called BIND. Bilirubin-induced neurologic dysfunction (BIND) Bilirubin toxicity to the brain, defined as BIND, causes lesser neurologic dysfunction than kernicterus, which is a serious neurologic dysfunction. This complication is characterized by subtle disturbances of vision (vasomotor dysfunction), use, neuromotor functions, speech, perception, and language, and new dystonia. Other hallmarks of this disease include reflex hyperexcitability and a number of neurologic features in neonates. Currently, bilirubin levels in neonates are not as severe as those associated with common jaundice.

Research methods. Each newborn was closely monitored with clinical, anamnestic, and laboratory examinations. Accurate diagnosis and examination of newborns are more difficult than other disease groups. A complete medical history is collected for each infant and young child with pathology based on the examination plan. Neurological status parameters,

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developmental risk factors, and obstetric history were studied. The neurological status of newborns was assessed using the Bayley scale. The detection and assessment of the clinical and neurological status of encephalopathy caused by bilirubin poisoning was studied using the BIND scale. BIND indicators are a tool for objectifying and facilitating the clinical diagnosis of BPE, as well as for monitoring neonatal neurological examination in young children with progressive hyperbilirubinemia and the clinical course of encephalopathy. The BIND scale classifies the clinical course of bilirubin encephalopathy in children into three levels (mild, moderate, and severe) based on the child's mental state, muscle tone, and crying. Clinical assessments of the control group were conducted regularly. A select number of children in the control group underwent monthly re-examinations, beginning with a clinical and neurological examination and continuing until age one.

Results of the study The degree and severity of bilirubin disorders are determined by the amount of bilirubin and its various components in the blood. Since bilirubin and its fractions assess the acute or chronic course of the disease and the state of neurological diseases, bilirubin and its fractions are checked and monitored at different times in the blood test of each subject. According to the anamnesis, the main complaints are: jaundice of the body in 100% of children, restlessness in 30 (60%), slow sucking in 21 (41.5%), jumping during sucking in 23 (46.7%), sleep disturbances in 35 (71%)... Jaundice begins on the first day in 24 (47.5%) newborns and the amount of OTC in the blood, estimated in zone 4-5 on the Kramer scale, is 367±80.34 μmol/L. Jaundice developed in 26 (52.5%) infants on days 2–3. Studies show that in the first week of the neonatal period, UBI and its fractions, CB, and unconjugated bilirubin increase to a maximum level, which causes the development of OBE. In newborns with HDI 30 (60%) anemia and jaundice, the UBI level was 448±68.34 μmol/L in the first three and seven days of life. As a result of intensive treatment in the second half of the neonatal period, the UBI level decreased to 268±35.24 μmol/L. 10 (20%) infants were born with enzymopathy. Polycythemia 12 (6) Liver diseases other than infection in the fetus WBC 358±55.34 μmol/L and UB 338±43.34 μmol/L In 4 (8%) of the studied material, the level of unconjugated bilirubin 22±7.2 is measured in μmol. /L ABE and nuclei. The results of laboratory tests show that hypohemoglobinemia and hypoproteinemia play an important role in the development of bilirubin encephalopathy. According to observations, hypohemoglobinemia of 72.45±8.43 g/L (52% of Hb) was detected in 10 participants, 94.45±14.43 g/L in 16 participants, and 102.45±11.43 g/L in 15 participants. The total protein content in the control group was 50.4±4.43 g/l, while in the main group it was 47.45±6.83 g/l. The weight of the observed children was 42.45±5.43 g/l and 45.25±3.43 g/l. The complete blood count showed that people had 3.45±0.49 10^12 g/l of erythrocytes. The color index of erythrocytes was: 0.77±0.04, ECT 8.8±5.43 mm/h. 2.7±0.73 10*12 g/l, ECT 22.45±2.43 g/l of erythrocytes was detected in 10 children (20%) in the comprehensive blood test. In 40 (80%) individuals, no clinically significant differences were found between the color index of the erythrocyte sediment and the total number of erythrocytes. However, it should be noted that the nucleation rate in 2-year-old children is 3-4 times higher. -3 degree anemia and varying degrees of hypotrophy. showed. The total protein content in the comparison group was 50.4±4.43 g/l, while in the respondents of the main group it was 47.45±6.83 g/l. In four children under observation, the indicators were 42.45±5.43 g/l and 45.25±3.43 g/l, respectively. The results of the general blood test show that the number of erythrocytes in each person is 3.45±0.49 x 10^12 g/l. Presence of erythrocytes, color 0.77±0.04 ECT 8.8±5.43 mm/h.After a comprehensive blood test, the red blood cell count in 10 (20%) newborns was 2.7±0.73 10*12 g/L, and the ECT was 22.45±2.43 g/L. In 40 cases (80%), no clinically significant differences

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were found in the total red blood cell count and the amount of sediment formed by the red blood cell color index. As a result of the study, 13 (52%) cases of SPC were identified in the comparison group. Of these, 6 (24%) had nuclear anemia and jaundice, polycythemia + sepsis, 3 (12%) liver disease + cephalohematoma, 3 (12%) enzymopathy + reactive hepatitis, 2 (8%) enzymopathy + sepsis (2 (8%)). From the first day of hyperbilirubinemia on the 15th-40th day, the child had persistent jaundice. The amount of OTC was 367±82.34 µmol/l (310-430 µmol/l), and in the second half of the neonatal period it was 275±60.34 µmol/l. According to the Cramer scale 4-5 zones in the first days of treatment, 2-3 zones at the end of the neonatal period. BIND scale 6 points 4 (30.6%) 4 points 3 (23%) 5 points 4 (30.76%) 3 points 2 (15.35%). The main complaints: sleep disturbance 6 (46.54%) tremor of the chin in the distal part of the hands 5 (38.45%) slow reaction to sound 4 (30.7%) restlessness 8 (61.57%). Weak absorption 3 (23%). Unconditioned reflex of newborns: the sucking reflex is weak, low Bauer-Moreau is observed. Ankle reflex hyporeflexia 2 (15.4%) nystagmus 5 (38.45%) dystonia 4 (30.7%) hypotonia 9 (69.7%) Ankle reflex hyperreflexia 8 (61.84%) anisoreflexia 3 (23%) convulsive syndrome was observed (2). %) children. Low response to sound exposure was revealed in 3 (23%) children, intestinal muscle rigidity - in 4 (30.7%) children. Conclusion. In newborns with hyperbilirubinemia, the development of bilirubin encephalopathy may occur due to a reactive state of the body, for example, sepsis, fermentopathy or intrauterine infection leading to neurological dysfunction. The most important neurological manifestation of neonatal bilirubin encephalopathy may be associated with changes in muscle tone, basal ganglia syndrome, and physiological reflexes of the newborn, particularly walking, support, and Robinson's syndrome. Hyperbilirubinemia results from elevated bilirubin levels, which are closely associated with abnormal muscle tone.According to the Kramer scale 4-5 zones in the first days of treatment, 2-3 zones at the end of the neonatal period. BIND scale 6 points 4 (30.6%) 4 points 3 (23%) 5 points 4 (30.76%) 3 points 2 (15.35%). Main complaints: sleep disturbance 6 (46.54%) chin tremor in the distal part of the hands 5 (38.45%) slow reaction to sound 4 (30.7%) restlessness 8 (61.57%). Weakness of suction 3 (23%). Unconditioned reflex of newborns: the sucking reflex is weak, low Bauer-Moro is observed. Ankle reflex hyporeflexia 2 (15.4%) nystagmus 5 (38.45%) dystonia 4 (30.7%) hypotonia 9 (69.7%) Ankle reflex hyperreflexia 8 (61.84%) anisoreflexia 3 (23%) convulsive syndrome was observed (2). %) children. Low response to sound exposure was revealed in 3 (23%) children, intestinal muscle rigidity - in 4 (30.7%) children. Conclusion. In newborns with hyperbilirubinemia, the development of bilirubin encephalopathy may occur due to a reactive state of the body, for example, sepsis, fermentopathy or intrauterine infection leading to neurological dysfunction. The most important neurological manifestation of neonatal bilirubin encephalopathy may be associated with changes in muscle tone, basal ganglia syndrome, and physiological reflexes of the newborn, particularly walking, support, and Robinson's syndrome. Hyperbilirubinemia results from elevated bilirubin levels, which are closely associated with abnormal muscle tone.According to the Kramer scale 4-5 zones in the first days of treatment, 2-3 zones at the end of the neonatal period. BIND scale 6 points 4 (30.6%) 4 points 3 (23%) 5 points 4 (30.76%) 3 points 2 (15.35%). Main complaints: sleep disturbance 6 (46.54%) chin tremor in the distal part of the hands 5 (38.45%) slow reaction to sound 4 (30.7%) restlessness 8 (61.57%). Weakness of suction 3 (23%). Unconditioned reflex of newborns: the sucking reflex is weak, low Bauer-Moro is observed. Ankle reflex hyporeflexia 2 (15.4%) nystagmus 5 (38.45%) dystonia 4 (30.7%) hypotonia 9 (69.7%) Ankle reflex hyperreflexia 8 (61.84%) anisoreflexia 3 (23%) convulsive syndrome was observed (2). %) children. Low response to sound exposure was revealed in 3 (23%) children, intestinal muscle rigidity - in 4 (30.7%) children. Conclusion. In newborns with hyperbilirubinemia, the development of

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