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COMBINATION OF TRANSITOR HYPERAMMONIEMIA WITH POST-HYPOXIC SYNDROME IN NEWBORN

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Abstract:

Reactions that reflect the process of adaptation to childbirth, new living conditions, are called borderline (transitional, transient, physiological) states of newborns. The day of these conditions, in contrast to the anatomical and physiological characteristics of the newborn, is characterized by the fact that they appear during childbirth or after birth and then disappear. They are called borderline not only because they occur at the border of two periods of life (intrauterine and extrauterine), but also because they are usually physiological for newborns, under certain conditions they can take on pathological features. [1, 3, 9, 10, 17] The literature describes more than 50 such transient reactions affecting the metabolism and functioning of all systems, including: transient hyperammonemia. [2, 4, 5, 14, 16]

Keywords: Borderline conditions, transient hyperammonemia, nitrogen balance, posthypoxic syndrome, premature newborns.

INTRODUCTION. Transient hyperammonemia is a borderline condition, first identified in the 70s in very premature babies with IUGR, but later verified in full-term newborns. Transient hyperammonemia - a rise, usually on the second or third day of life, of ammonia nitrogen concentration above 40 - 45 μmol / l. Some children with transient hyperammonemia did not show any clinically pronounced disorders, but others were found to have: signs of CNS depression of varying severity (lethargy - up to adynamia, sucking lethargy, muscle hypotension, decreased tendon reflexes, stupor, lethargy, coma), shortness of breath with alkalosis, respiratory disorders, often requiring mechanical ventilation, jaundice due to hemolysis (in ¾ of children with transient hyperammonemia, the level of carboxyhemoglobin in the blood is increased), and often intraventricular and other intracranial hemorrhages, convulsions, dehydration. [8, 12] The frequency of development of transient hyperammonemia in very preterm infants reaches 50%. Its main provoking factor is perinatal hypoxia. Transient hyperammonemia is found in approximately 1/4 - 1/3 of premature newborns who have undergone the latter (regardless of whether the child was born in asphyxia or not). Usually, a high level of ammonium in the blood lasts for several days, but in some children who have undergone severe perinatal hypoxia, combined with severe hyperbilirubinemia, posthypoxic pneumo- and encephalopathies, hyperammonemia can persist for several weeks [11, 15, 20, 21].

MATERIALS AND RESEARCH METHODS. Taking into account this information, we studied nitrogen metabolism, namely the level of urea and creatinine in the blood serum using the KONE-2000 apparatus in newborns with posthypoxic syndrome, depending on the complications.

There were 38 patients under observation, 23 of them were full-term, 15 were premature. By age from 1 to 5 days - 16 newborns, from 10 to 14 days 14 newborns, from 22 to 28 days 8 newborns. In the anamnesis, 5 mothers had a TORCH complex - toxoplasmosis, cytomegalovirus infection, herpes virus, 7 - ARVI with pyelonephritis, 7 - dirty amniotic fluid, 7 - a long anhydrous period, 5 - prolonged preeclampsia, 4 - prolonged labor, 3 - home birth, a combination of aggravated factors in 70% of mothers.

RESULTS OF RESEARCH AND DISCUSSION. Against the background of perinatal pathology of the nervous system, 8 patients had pneumonia, 18 - hypertension syndrome, 12 - depression syndrome, hyperbilirubinemia. In the dynamics of observation, these children showed areflexia, hypotension, signs of arousal syndrome were transformed into a syndrome of depression. Against the background of severe symptoms of hypoxic-ischemic encephalopathy, 8 children showed signs of cerebral hemorrhage on days 2-4. The depression syndrome was manifested by a decrease in motor activity, muscle hypotension, weakening of reflexes and the predominance of parasympathetic autonomic tone.

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Indicators of nitrogen metabolism in dynamics reflected the level of urea in full-term in the first days 6.5 ± 0.72, on days 5-7 of treatment 5.1 ± 0.01: P<0.05,
with indicators in healthy newborns from 2.5-4.5 mmol / l. In premature babies in the first days, 6.4 ± 0.6 to 6.5 ± 0.15 on days 5-7 of treatment. At the same time, the level of creatinine in full-term in the first days from 78.5 ± 19.0 and then on days 5-7 of therapy, up to 60.0 ± 0.01, in healthy people from 88 to 100 mmol / l. In preterm infants, these indicators varied by 74.5 ± 5.5 and 73.5 ± 4.5, respectively; P<0.05.

The catabolic orientation of metabolism during hypoxic complications in the full-term newborns observed by us was expressed in shifts in nitrogen metabolism, an increase in the level of urea upon admission with a slow tendency to decrease against the background of ongoing treatment in dynamics. An increase in the level of urea in premature infants without any significant changes in dynamics compared with full-term newborns with normal creatinine levels indicates an indirect relationship with hyperammonemia, which is characteristic of preterm infants during the period of adaptation. But the absence of a tendency to decrease in dynamics indicates the possibility of developing posthypoxic complications of the urinary system.

5 children had a syndrome of increased neuro-reflex excitability. Manifested by emotional anxiety against the background of sufficient physical activity, prolongation of the period of active wakefulness. The level of urea in the blood serum of these newborns ranged from 4.4 to 6.2 mmol/l, creatinine from 74.8 to 99.0 mmol/l. In newborns with intraventricular hemorrhage, the level of urea in the blood serum ranged from 5.9 - 7.5 mmol / l, the level of creatinine in 3 newborns from 78 - 100 mmol / l, in 5 from 102 - 151 mmol / l.

If we analyze the observed metabolic changes during perinatal hypoxia in newborns with intraventricular hemorrhage and without intraventricular hemorrhage, the level of urea and creatinine in newborns with intraventricular hemorrhage is higher than in children without intraventricular hemorrhage, which confirms the information that transient hyperammonemia, although it is considered a borderline condition , in the presence of aggravated factors with clinical and instrumental confirmation, it acquires a pathological orientation.

Two phases of the toxic effect of ammonia on the central nervous system can be assumed. The first early phase is associated with the toxic effect of ammonia on certain functions and structures of the brain, while causing limited changes. It is believed that this phase is due to a sharp decrease in the level of glotomat in the brain, which determines the early stage of CNS depression. The late phase of the toxic effect of ammonia leads to the depletion of the energy reserve of the brain and leads to deeper cerebral disorders (coma or comatose state). [12, 13, 22, 24]

CONCLUSIONS. Thus, physiological changes in the main functional systems of the body during childbirth and immediately after birth, their adaptive changes in the neonatal period (boundary states) and pathological processes, the pathogenetic basis of which may be a specific borderline state, are formed as a result of stressful influences and adverse environmental factors. into which the child enters after birth. The obtained results of the research can be the basis for formulating an idea of the mechanisms of the pathogenesis of hyperammonemia in transient physiological and pathological conditions in newborns.

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