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HEMORRHAGIC DISEASE OF THE NEWBORN

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Abstract. The article discusses the problems of hemorrhagic disease of newborns with late debut. Currently, hemorrhagic disease prevention is not carried out for all newborns in maternity hospitals. A clinical analysis of 9 cases of the disease was conducted. A dangerous feature of the late manifestation of hemorrhagic disease (after a month of life) is the development of massive intracranial hemorrhages against the background of increased bleeding.
Keywords: newborns, vitamin K, hemorrhagic disease, breastfeeding.

INTRODUCTION

In the practice of a pediatric resuscitator, non-traumatic intracranial hemorrhages in children in the first months of life are quite rare. It is not always possible to establish the true cause of the hemorrhage. At the same time, it is known that one of the causes of intracranial hemorrhages in children in the first months of life is late hemorrhagic disease of the newborn (LDN). It is impossible to predict the development of the late onset of the disease, so prevention of LDN at the stage of the maternity hospital is extremely important. The development of LDN in newborns, especially those who are breastfed, is preceded by a deficiency of vitamin K: gamma-carboxylation of vitamin K-dependent blood clotting factors is disrupted in the hepatocyte.

MATERIALS AND METHODS

The onset of the disease was almost identical in all children. 1–2 days before the onset of intracranial hemorrhage, hemorrhagic elements appeared on the skin or oral mucosa. Single deep ecchymoses with a diameter of 5 to 20 mm were detected by parents more often on the extremities, less often on the trunk. Small multiple hemorrhages on the oral mucosa were detected already during examination in the intensive care unit. One sick child had blood in the stool and prolonged bleeding from the injection site during repeated vaccination against hepatitis B. No pathology was detected during consultation with a surgeon.

RESULTS AND DISCUSSION

In all patients, intracranial hemorrhage manifested itself with sudden, painful, but short-term crying. In 8 children, persistent and persistent vomiting immediately developed, in two cases with an admixture of blood. Vomiting was not observed in only one child. At first, the children showed periodic restlessness, began to moan, refused to feed, then became apathetic and indifferent to their surroundings. Tonic convulsions were observed in 7 children. A sharp pallor of the skin increased [1]. Short-term subfebrile temperature was replaced by hypothermia. Almost all children were hospitalized very late: the time spent at home from the moment the hemorrhage occurred until hospitalization was from one day or more.

On admission to the intensive care unit, 8 children were in an extremely serious condition. All had decompensation of pulmonary ventilation, systemic circulatory disorders, focal and general cerebral neurological symptoms, and coagulation hemostasis disorders. In 6 cases, the admitted children had rare, arrhythmic breathing or no breathing at all. In 2 cases, respiratory disorders were only in the form of pronounced tachypnea (respiratory rate within 100/min). All patients had severe pallor of the

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skin and mucous membranes with a cyanotic tint, as well as bleeding from the injection sites. In 2 children, signs of mild pulmonary and gastric bleeding were observed, in the form of hemorrhagic discharge from the endotracheal tube and gastric tube. In 8 children, a slight icteric tint of the skin persisted upon admission [2]. The circulating blood volume deficit ranged from 30 to 39% of the norm (the normal circulating blood volume in children in the first months of life is 85 ml/kg; circulating blood volume fact = body weight/weight portion of circulating blood volume by hematocrit). Blood pressure fluctuated from 80/40 to 110/72 mm Hg. Tachycardia with muffled heart sounds was observed in 7 children. Disorders of central respiratory regulation were observed in the neurological status of 6 children. Pathological forms of respiration in the form of pronounced bradypnea or apnea developed in these children even before hospitalization and continued to progress until complete loss of automatic breathing. Trunk reflexes from the mucous membranes (cornea, pharynx, trachea) were not evoked. Sharp dryness and swelling of the mucous membranes of the oral cavity and sclera were noted. The oculocephalic reflex was absent. Fixed, bilateral, paralytic mydriasis and diffuse muscular atony were revealed. In all children, the large fontanelle was bulging, dense to the touch, with no pulsation. Severe hypothermia of the scalp was noted. Five children had attacks of hormetonia (periodic tonic tension of the muscles of the limbs and trunk, arising against the background of muscular atony spontaneously or under the influence of irritants, lasting no more than 10 s) [3].

In this case, the development of hormetonia is associated with damage to the brainstem at the level of the midbrain and pons due to transtentorial herniation, when the brainstem and cerebral hemispheres are functionally separated. As the coma deepened, the attacks of hormetonia ceased. Thus, all 6 patients were diagnosed with grade 3 (extreme) coma. All patients admitted to the intensive care unit in a state of extreme coma died. Two children were diagnosed with grade 1–2 coma upon admission to the intensive care unit. Tonic seizures, transient symptoms of damage to the III, VI, and VII pairs of cranial nerves, and tachypnea were noted. The large fontanelle was dense, bulging, but with preserved pulsation. The reaction of the pupils to light, brainstem reflexes from the mucous membranes (cornea, pharynx, trachea), and the oculocephalic reflex remained intact. One patient's neurological status showed signs of moderate intracranial hypertension.

In the peripheral blood tests, a significant decrease in the hemoglobin level was noted in most patients. In all patients, the blood clotting time (according to Morawitz) was sharply prolonged. The prothrombin index was determined only in one patient and was reduced. The platelet count was normal or increased, and tended to increase further during the day [4]. Normal fibrinogen levels were noted in 4 patients, in the rest fibrinogen was not determined. Direct bilirubin remained elevated in 8 children, liver enzymes were slightly elevated in 1.

Subarachnoid hemorrhages were detected in all children. In 8 cases, the hemorrhages were massive. Intracranial hemorrhages were diagnosed based on the anamnesis, clinical data and ultrasound examinations. Neuroimaging methods (CT, MRI of the brain) were impossible in the vast majority of patients due to the extremely serious condition of the patients. In 3 patients, after the bleeding subsided, when intracranial hypertension (irreversible cerebral edema) was not excessively pronounced, the hemorrhage was confirmed by lumbar puncture (after 24 hours). The cerebrospinal fluid most often flowed out under low or normal pressure, and was reddish-brown in color. Centrifugation revealed severe xanthochromia; microscopy revealed a large number of altered (hemolyzed) erythrocytes. Biochemical examination of cerebrospinal fluid revealed low glucose levels and high levels of protein and lactic acid (lactate).

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Therapy

8 children were on artificial ventilation. All of them underwent correction of blood volume, hemostasis and metabolism disorders, as well as anticonvulsant and neuroprotective therapy. Bleeding control began with the introduction of 10 mg of Vikasol through a tube, a single transfusion of fresh frozen plasma in a volume of 15 ml/kg. After the bleeding stopped (usually after 10-12 hours), further administration of sodium menadione (intramuscularly) was continued to create a depot of 5 mg/day for 2-3 days. Eight patients underwent transfusion of red blood cells due to the development of posthemorrhagic anemia. Subsequently, none of the patients had bleeding.

The basis of laboratory diagnostics of HDN is the determination of prothrombin time and index, reflecting the total level (three out of four) of vitamin K-dependent coagulation factors (II, VII, X). In HDN, the platelet content and thrombin time should be normal [5].

Blood breakdown products have pronounced toxicity (oxyhemoglobin, serotonin, bilirubin, etc.) and additionally cause severe cerebral ischemia, leading to cerebral infarction. And, finally, another feature of late HDN is the hypocoagulation trend of the results of available hemostasis assessment tests and their stabilization against the background of the introduction of vitamin K (Vikasol).

CONCLUSION

Based on the clinical observation, it can be concluded that late-onset HDN is prone to develop in full-term newborns, in whom a combination of factors such as the absence of prophylactic administration of sodium menadione, breastfeeding, and transient cholestasis has become possible. In newborns with expected breastfeeding, HDN prevention is especially relevant.

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