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MODERN PATHOGENETIC MECHANISMS OF PERINATAL LESIONS OF THE CENTRAL NERVOUS SYSTEM IN NEWBORNS

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Abstract:

The review article presents modern data of foreign authors on the pathogenetic aspects of the formation of perinatal lesions of the central nervous system in newborns. The author systematized medical, social and metabolic factors that are of significant importance in the development of pathology.

Keywords: Newborns, Perinatal Damage Of The Central Nervous System, Encephalopathy

According to the literature, among the children recognized as disabled for the first time, about half are patients with cerebral palsy (cerebral palsy), the formation of which in most children has a perinatal hypoxic-ischemic origin [1,2].

The medical and social significance of this problem is given by the fact that children who have undergone perinatal hypoxia further develop a wide range of neuropsychiatric (including in 15-40% of severe children) and numerous somatic disorders, ultimately leading to disability, social maladaptation and a reduction in overall life expectancy [3-6]. The active introduction of advanced medical technologies into healthcare practice has significantly expanded the possibilities of studying the origins of many diseases and pathological conditions in newborns and has allowed to clarify the etiology, pathogenetic mechanisms, clinical and morphological structure, as well as the localization of cerebral disorders typical for different gestational ages [7-10].

Despite the fact that pregnancy and childbirth are the most natural and physiological events in human life, these fundamental processes are extremely vulnerable and subject to numerous endogenous and exogenous influences [4,8]. There are three main groups of causes of the development of chronic intrauterine hypoxia, which turns into hypoxia of the newborn: extragenital diseases of the mother (smoking, neurocirculatory dystonia, thyroid diseases, obesity, diabetes mellitus, bronchial asthma, anemia, cardiac arrhythmias), disorders of uteroplacental and fetoplacental circulation (history of abortions, gestosis, candidiasis colpitis, sexually transmitted infections, isthmic-cervical insufficiency) and diseases of the fetus itself [5,11]. The pathogenetic bases of acute and chronic variants of hypoxia are hemodynamic disorders leading to ischemia and hemorrhagic lesions of organs, disruption of their function and severe metabolic disorders [5,10].

It is believed that more than 60% of diseases of the central nervous system of childhood are directly related to or mediated by perinatal fetal hypoxia, and more than half of all cases of encephalopathy are caused not by acute hypoxia in childbirth, but by prolonged chronic hypoxia of the fetus and newborn [1-3]. Hypoxia provokes high activity of lipid peroxidation processes against the background of low antioxidant protection, which leads to the failure of cell membranes [4]. Currently, the concept of pathogenesis of perinatal hypoxic-ischemic injuries has been developed and confirmed by many studies, implying that the chain of pathological events is based on a generalized violation of lipid metabolism with the development of membrane-destructive processes. Evidence has been obtained that the severity of hypoxic ischemic lesions of the central nervous system in a newborn is directly correlated with changes in lipid metabolism that persist in the early postnatal period and are expressed in changes in the composition of blood plasma lipids and erythrocytes [7,11,13].

Gestationally immature children are most predisposed to the damaging effects of chronic intrauterine hypoxia and acute intranatal asphyxia due to morpho-functional immaturity of the brain, impaired cerebrovascular autoregulation, decreased activity of antioxidant systems, features of metabolic processes, energy deficiency and low level of plastic processes [7-9]. So, if the frequency of perinatal lesions of the central nervous system, depending on the nature of the course of the ante- and intranatal periods, in full-term children ranges from 15 to 60%, then in premature infants this indicator increases to 65-85%. Lesions of the central nervous system of various genesis in premature infants, due to predisposing circumstances, can lead to the formation of persistent neurological disorders with subsequent chronization of the process, disability, social maladaptation and a decrease in the quality of life in general. In the structure of childhood disability, 70-80% of neurological pathology has a perinatal genesis [9-12]. It should be noted that intrauterine hypoxia is a universal damaging factor affecting not only the

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nervous system, but also having a multi-organ effect, negatively affecting the function of the bronchopulmonary, cardiovascular, urinary, endocrine, immune systems, gastrointestinal tract, metabolic and reparative processes [3-6].

Given the unity and interconnection of all body systems, pathological processes in any of them lead to a violation of the functional activity of the other, thereby the pathology of the central nervous system will be accompanied by a violation in other vital organ systems, and the latter aggravates the defeat of the neurological sphere, forming a vicious circle. The main etiological factor in the development of intrauterine hypoxia is the pathological course of pregnancy, including toxicosis, gestosis, acute and chronic infectious processes in the mother, intestinal dysbiosis, various somatic pathology, burdened obstetric and gynecological history, etc. All these factors lead to increased permeability of the uteroplacental barrier for toxic products, microorganisms, antibodies, chronic disruption of placental-fetal blood flow with the development of circulatory ischemia of the brain and other internal organs, suppressing the growth of the capillary bed, inhibiting plastic processes and negatively affecting subsequent postnatal adaptation [1-3, 9-11].

Hypoxic damage to the vascular endothelium with the development of edematous destructive processes in it with a decrease in the lumen of the capillary bed also contributes to an increase in resistance to cerebral blood flow. The cerebral vasoconstrictor effect is promoted by electrolyte changes against the background of hypoxia in the form of a decrease in extracellular calcium, accompanied by an increase in vascular tone [10].

An imbalance in the processes of lipid peroxidation and antioxidant activity also affects the level of the erythrocyte membrane, reducing the content of reduced glutathione and thereby leading to a decrease in elasticity and increased rigidity of erythrocyte membranes. As a result, the degree of deformability of erythrocytes changes, which increases their aggregation ability and leads to the development of thrombosis not only in the central nervous system, but also in the vascular bed of other organ systems, predisposing against the background of general hypocoagulation to the development of disseminated intravascular coagulation syndrome [10-12].

The development of intraventricular hemorrhages is associated with metabolic changes in the form of activation of lipid peroxidation against the background of a decrease in antioxidant activity, activation of anaerobic glycolysis with lactic acidosis, increased content of malondialdehyde, hyperglycemia. But in premature infants, hyperglycemia is more often replaced by hypoglycemia due to depletion of liver glycogen reserves due to prolonged antenatal and acute intranatal hypoxia, as well as due to the development of adrenal insufficiency and a decrease in the stimulating effect of glucocorticoids on gluconeogenesis due to a decrease in their formation. Consequently, the energy supply of cerebral reparative and neurophysiological processes is sharply reduced due to the deficiency of the main energy substrate of the nervous system – glucose [9].

However, in premature infants suffering from chronic intrauterine hypoxia, hypocoagulation is more common with a predisposition to the development of hemorrhagic lesions of the central nervous system, represented more often by subependimal and intraventricular hemorrhages, the source of which is the hermenative matrix of the lateral ventricles. Intraventricular hemorrhages, the genesis of which is hypoxia, are one of the main causes of deaths and account for about 70% in premature infants [12,13].

CONCLUSION

Pathogenetic aspects of the formation of perinatal hypoxic lesions of the central nervous system are as follows. First of all, this is the development of cerebral hypoperfusion due to the increase in hypoxia and hypercapnia in conditions of fetoplacental insufficiency. These metabolic changes lead to a pronounced vasoconstrictor effect in the cerebral vascular bed and further disruption of autoregulation of cerebral blood flow (defective initially in a premature baby).

The important role of rheological properties of blood in the development of cerebral hypoxia and hemorrhagic brain damage has been established. The infectious process, chronic somatic pathology in the mother, acute intranatal asphyxia can lead to hypercoagulation of the hemostasis system with the development of thrombosis in the microcirculatory bed, leading to the formation of foci of ischemia with subsequent necrotizing of brain tissue, and the development of pseudocysts at the site of necrosis with replacement by connective tissue in the future.

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