RISK OF CARDIOVASCULAR DISEASES IN INDIVIDUALS WITH LOW BIRTH WEIGHT

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Annotation. The article examines the main clinical aspects, analyzes perinatal risk factors for cardiovascular pathology, and explores the impact of low birth weight (due to prematurity or intrauterine growth restriction) on the risk of subsequent cardiovascular diseases. A relationship has been identified between typical perinatal disorders in preterm infants (such as hypoxic maladaptation of the cardiovascular system, persistent fetal circulation, and others) and subsequent structural and rhythmic abnormalities of the heart. The influence of perinatal factors has a global impact on all future risks of cardiovascular diseases, including the development of myocardial infarction and strokes in adulthood.

Keywords: Preterm newborns, cardiovascular system, low birth weight.

Preterm birth is the result of the disruption of the "fetus-mother" system, caused by external or internal factors. The most significant factors contributing to pathological processes in preterm infants include the necessity of obtaining oxygen through pulmonary gas exchange and hypoxic-ischemic damage or immaturity of the nervous system, which subsequently leads to the development of chronic bronchopulmonary and neurological pathologies, as well as delays in cognitive and neuro-sensory development [1]. An important aspect of the development of complex pathology is also the functional and structural immaturity of the cardiovascular system, leading to varying degrees of hemodynamic disturbances during intensive care and neonatal management, as well as during subsequent follow-up observations.

The adaptation of the cardiovascular system in preterm infants has certain distinctive features. In particular, the heart of such an infant operates with

minimal reserve capacity, meaning that any adverse impact on the myocardium can lead to functional decompensation [2].

Low birth weight is associated with a reduced number of cardiomyocytes, which can be explained by low levels of tissue growth factors in the fetus due to nutritional deficiencies and impaired uteroplacental blood flow. Additionally, there is a direct correlation between the concentration of growth factors in the blood and tissues and the gestational age. Increased myocardial workload against the background of low reserve capacity, combined with comorbid conditions, disrupts the autonomic regulation of the heart and coronary vessels and impairs energy metabolism in the cardiac muscle [3].

These factors contribute to cardiovascular maladaptation in newborns, leading to various hemodynamic disturbances that can significantly affect the prognosis for life and health in infants with very low and extremely low birth weight [4].

Hypoxia plays a key role in cardiovascular damage in preterm newborns, with the incidence of myocardial ischemia increasing in proportion to the severity of respiratory pathology in neonates [5]. According to various studies, the prevalence of post-hypoxic cardiovascular maladaptation in children ranges from 40% to 70%, making it one of the leading conditions in neonatal pathology [6]. Moreover, preterm infants tend to experience prolonged disturbances within this spectrum

The frequency of transient myocardial ischemia is influenced by gestational age. According to I.V. Vinogradova, this condition occurs in 58% of infants with extremely low birth weight and in 46.1% of infants with low birth weight. In addition to hypoxic maladaptation, contributing factors include hypercatecholaminemia, carnitine deficiency, and a dispersed type of coronary vessel arrangement [7].

Conclusion. Preterm birth and low birth weight increase the risk of cardiovascular maladaptation in newborns, primarily due to hypoxia-related

myocardial ischemia and structural immaturity. Impaired autonomic regulation and energy metabolism contribute to long-term complications. The high prevalence of post-hypoxic cardiovascular dysfunction underscores the need for early monitoring and targeted interventions to improve outcomes in preterm infants.

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