World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-31, February 2024
ISSN: 2749-3644

DISEASES OF THE HEART AND VASCULAR SYSTEM IN NEWBORNS

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Article history:

Received: December 20th 2023
Accepted: January 14th 2024
Published: February 21st 2024

Abstract:

The article presents the modern classification and clinical and morphological characteristics of primary and secondary cardiomyopathies in newborns and young children. The etiology and mechanisms of development of the most common cardiomyopathies are analyzed. Aspects of such rare cardiomyopathies as non-compact spongy myocardium and histiocytoid cardiomyopathy are covered. The issues of etiopathogenesis, clinical picture, pathomorphology and outcomes of posthypoxic cardiomyopathy, which occupies a leading place in the structure of causes of perinatal morbidity and mortality, are presented in detail.

Keywords: newborns, cardiomyopathy, morphology, posthypoxic cardiomyopathy

INTRODUCTION

Currently, in the structure of cardiovascular diseases in children, the proportion of myocardial lesions of non-inflammatory origin has increased, the development mechanism of which is closely related to metabolic disorders and a decrease in the contractile function of the heart.

MATERIALS AND METHODS

Cardiomyopathies remain one of the least studied cardiac diseases, being the object of research in actively developing modern cardiology. The increased interest in the problem of myocardial diseases is explained by the need for further study of the etiology and pathogenesis, the diversity and nonspecificity of their clinical manifestations, and significant diagnostic and therapeutic difficulties.

Hypoxic damage to the myocardium in fetuses and newborns is one of the pressing problems of neonatology. According to Russian literature, changes in the myocardium of hypoxic-ischemic origin (posthypoxic cardiomyopathy) are described in about 70% of children who have suffered antenatal hypoxia [1]. To this day, there is no unambiguous terminological interpretation of this lesion. Probably, both terminology and classification should reflect the pathogenetic essence of the changes occurring in the myocardium and take into account the dynamic assessment of these changes. Among the proposed options, the following should be highlighted: posthypoxic cardiomyopathy, transient myocardial ischemia, cardiopathy of the newborn period, functional cardiopathy of newborns, cardiac asphyxia syndrome, myocardial dysfunction, syndrome of maladaptation of the cardiovascular system [2].

RESULTS AND DISCUSSION

Hypoxic cardiopathy is a natural complication of a wide range of perinatal diseases accompanied by impaired oxygenation of the rhythmogenic, conductive and contractile structures of the heart. The leading pathogenetic factors are disturbances of the uteroplacental and umbilical cord blood circulation and respiratory distress syndrome. Hypoxic damage to the cardiovascular system of newborns is characterized by a complex of clinical symptoms designated "disadaptation syndrome of the cardiovascular system of newborns" with varying degrees of impairment of the conduction and contractility of the myocardium [3]. As a result of the direct impact of hypoxia on the body of the fetus and newborn, a cascade of metabolic and microcirculatory pathological reactions develops, leading to disturbances in the energy supply of the myocardium, as a result of which myocardial ischemia and associated disorders of central hemodynamics develop [4].

A drop in the partial pressure of oxygen in tissues induces the activation of a number of signaling systems. In particular, during hypoxia, activation of apoptosis or cell proliferation can occur in myocardial tissue, which is the most active consumer of oxygen. Myocardial ischemia and associated disorders of central hemodynamics and contractile function of the heart develop as a result of disorders of the energy supply of the myocardium under the influence of hypoxia [2].

There are a number of anatomical and physiological features that facilitate cardiac activity under hypoxic conditions. Thus, the cardiac muscle is represented by a symplast, consisting of thin, poorly separated myofibrils. There is no transverse striation. Cardiomyocytes have many nuclei, few mitochondria,

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and reduced activity of mitochondrial enzymes involved in fatty acid metabolism, which leads to L-carnitine deficiency [2]. In myofibrils, the β-isomer of myosin predominates with low ATPase activity and insufficient function of calcium channels. Connective tissue has few elastic fibers [3]. A feature of the biochemical processes in cardiomyocytes is the predominance of the carbohydrate component. Cardiomyocytes can also use metabolites (ketone bodies, amino acids, lactate), due to which the stable activity of the heart muscle is maintained, in contrast to skeletal muscles. Coronary circulation is characterized by a scattered type of vessels and a large number of anastomoses [4]. The nervous regulation of the heart's activity is dominated by the influence of the sympathetic nervous system. The above greatly facilitates the activity of the cardiovascular system of a healthy fetus and newborn, but, unfortunately, in general, hemodynamics are characterized by instability, and any deviation in homeostasis can provoke significant hemodynamic disturbances.

The connection between hypoxic myocardial damage and various disturbances of heart rhythm and conduction is evidenced by data from morphological and ultrastructural studies. Thus, in the conduction system of the heart, signs of apoptosis and dystrophy are found with a certain relationship between the severity of morphological changes and clinically detected rhythm and conduction disorders. The final morphological result of hypoxic damage to the heart can be focal dystrophy, which has two possible outcomes: either complete resolution and restoration of functions, or the formation of focal cardiosclerosis [2]. The persistence of autonomic dysfunction can lead to the formation of persistent vegetative-visceral disorders, one of the manifestations of which is functional cardiopathy [5].

Metabolic features in cardiomyocytes explain the increased sensitivity of the heart to hypoxia. Cardiomyocytes are among the cells with a predominant aerobic metabolism. Under conditions of ischemic cell damage during chronic circulatory failure, there is a disruption in the energy supply of the cell, at the level of both ATP production and energy transport and utilization. Impaired ATP synthesis is associated with suppression of oxidative processes caused by oxygen deficiency and secondary damage to the structure and enzymes of mitochondria. In this case, the activity of enzymes involved in ATP synthesis is suppressed. The activity of the main aerobic energy-producing processes decreases: the oxidative formation of acetyl-CoA from fatty acids, pyruvate and amino acids, the metabolism of acetyl groups in the tricarboxylic acid cycle, and the transport of electrons to oxygen associated with phosphorylation. In this case, the cell responds by mobilizing energy from intracellular reserves and reducing energy consumption. Mobilization of intracellular energy reserves is carried out through the use of energy-rich substances: primarily creatine phosphate, mobilization of glycogen, glucose, triglycerides, and activation of glycolysis. Activation of glycolysis can be considered as one of the compensation mechanisms.

Morphologically, the myocardium has a spongy appearance and is represented by a large number of trabeculae and intertrabecular depressions, which sometimes reach the subepicardial zone. They are sent out by the endothelium, which passes to the inner surface of the ventricles. You can see the connections between the sinusoids. There is no capillary network in trabeculae [3]. The pathological structure of the non-compact myocardium of the left ventricle causes its dysfunction, leading to progressive heart failure, the severity of which depends on the number of "non-compact" segments and the degree of "non-compactness". The causes of myocardial systolic dysfunction have not been sufficiently studied, but chronic ischemia caused by impaired coronary microcirculation is considered one of the mechanisms of its occurrence [2].

Excessive formation of connective tissue fibers, histologically resembling fibroelastosis, is often observed in the endocardium. Specific histological markers for non-compact myocardium of the left ventricle have not been described [2].

CONCLUSION

Macroscopically, cardiomegaly is noted, caused by a combination of myocardial hypertrophy with dilatation of the left ventricular cavity. Under the endocardium of the left ventricle, papillary muscles, in the atria, tricuspid valve leaflets, many round yellow-white nodules are visible. Histologically, the nodules represent a proliferation of large polygonal foamy and granular cells. They are often located inside the conduction system, and there is an assumption that this is a tumor of Purkinje cells.

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