MORPHOLOGICAL ASPECTS OF CONGENITAL AND ACQUIRED HEART DEFECTS

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Abstract

Heart defects remain one of the most significant causes of cardiovascular pathology and population disability, occupying a leading position in the structure of congenital and acquired heart diseases. Despite significant advances in clinical cardiology and cardiac surgery, the morphological aspects of the formation and progression of heart defects continue to be of high scientific interest. The development of heart defects is based on disturbances of embryogenesis, remodeling of the myocardium and valvular apparatus, as well as changes in the microcirculatory bed and extracellular matrix. Morphological remodeling includes hypertrophy and dilation of cardiac chambers, fibrosis, disorganization of cardiomyocytes, and alterations in the structure of the endocardium and valves. The aim of this review is to analyze current literature data on the morphological features of congenital and acquired heart defects, with an emphasis on histological and ultrastructural changes of the myocardium and valvular apparatus. The presented data emphasize the significance of morphological studies for understanding the pathogenesis of heart defects and for substantiating new diagnostic and therapeutic approaches.

Key words: heart defects, morphology, myocardium, valvular apparatus, remodeling, fibrosis.

Introduction

Heart defects represent a heterogeneous group of congenital and acquired diseases characterized by structural and functional disorders of the cardiovascular system. According to the World Health Organization, congenital heart defects occupy a leading

place among the causes of infant mortality, whereas acquired heart defects remain a significant problem in the adult population, especially under conditions of population aging and increasing prevalence of inflammatory and degenerative diseases of the valvular apparatus. Despite substantial progress in diagnosis and surgical correction, heart defects continue to be associated with a high risk of heart failure and reduced quality of life.

Modern studies increasingly focus on the morphological basis of heart defects, since structural changes in the myocardium, endocardium, and valvular apparatus determine the clinical course and prognosis of the disease. Morphological analysis allows a deeper understanding of the mechanisms of cardiac remodeling, identification of early signs of decompensation, and substantiation of new diagnostic and therapeutic approaches. In this regard, the study of morphological features of congenital and acquired heart defects remains a relevant direction of fundamental and clinical medicine.

Embryological Basis of Congenital Heart Defect Formation

The formation of congenital heart defects is associated with disturbances of cardiogenesis at early stages of embryonic development. The heart develops from a mesodermal primordium and undergoes a series of sequential stages, including the formation of the cardiac tube, its looping, chamber septation, and development of the valvular apparatus. Any adverse influences during critical periods of embryogenesis may lead to persistent structural anomalies.

Morphologically, congenital heart defects are characterized by defects of the interatrial and interventricular septa, disturbances in valve formation, and anomalies of great vessel outflow. At the histological level, immaturity of cardiomyocytes, impaired orientation of muscle fibers, reduced capillary network density, and alterations of the extracellular matrix are detected. Ultrastructural studies demonstrate disorganization of sarcomeres, mitochondrial dysfunction, and signs of energy deficiency in cardiomyocytes.

Of particular importance in the pathogenesis of congenital heart defects are disturbances in the migration of neural crest cells involved in the formation of cardiac outflow tracts and the aortopulmonary septum. These changes are accompanied by pronounced morphological defects that subsequently determine the severity of clinical manifestations of the disease.

Morphological Changes of the Myocardium in Acquired Heart Defects

Acquired heart defects are formed predominantly as a result of inflammatory, degenerative, or ischemic processes and are accompanied by progressive myocardial remodeling. The valvular apparatus is most frequently affected, leading to chronic volume or pressure overload and initiating compensatory and decompensatory morphological changes.

Macroscopically, the heart in acquired heart defects is characterized by hypertrophy and dilation of cardiac chambers, thickening or deformation of valves, and alterations in ventricular geometry. At the histological level, cardiomyocyte hypertrophy, interstitial and perivascular fibrosis, focal degeneration of muscle fibers, and signs of chronic hypoxia are observed. A significant role is played by remodeling of the microcirculatory bed, manifested by reduced capillary density and impaired myocardial perfusion.

Ultrastructural changes include mitochondrial damage, dilation of the sarcoplasmic reticulum, fragmentation of myofibrils, and disruption of intercellular contacts. These processes contribute to a decrease in myocardial contractile function and progression of heart failure. Morphological changes are stage-dependent, reflecting the transition from compensatory hypertrophy to decompensation and myocardial dysfunction.

Histological and Ultrastructural Features of the Myocardium

Histological studies of the myocardium in heart defects reveal a complex of structural changes reflecting chronic cardiac overload. A characteristic feature is cardiomyocyte hypertrophy accompanied by enlargement of nuclei, polyploidy, and increased synthetic activity of cells. At the same time, disorganization of muscle fibers,

expansion of intercellular spaces, and accumulation of extracellular matrix components are observed.

Interstitial fibrosis is one of the key morphological features of both congenital and acquired heart defects. It leads to decreased myocardial elasticity, impaired electrical conductivity, and deterioration of contractile function. Ultrastructural studies demonstrate mitochondrial damage, disruption of sarcomere structure, decreased density of myofibrils, and destruction of intercalated discs. These changes indicate the development of energy deficiency and progressive myocardial dysfunction.

Changes in the microcirculatory bed are characterized by rarefaction of the capillary network, thickening of basement membranes, and endothelial dysfunction. Taken together, these morphological alterations form the structural basis of heart failure in heart defects.

Conclusion

Thus, congenital and acquired heart defects are accompanied by pronounced morphological changes of the myocardium, valvular apparatus, and endocardium. These changes include disturbances of embryonic development, remodeling of cardiac chambers, fibrosis, disorganization of cardiomyocytes, and structural alterations of valves. Morphological studies allow a deeper understanding of the pathogenesis of heart defects, identification of stages of compensation and decompensation, and substantiation of new diagnostic and therapeutic approaches. A review of current literature data confirms the important role of morphology in the comprehensive assessment of heart defects and emphasizes the need for further fundamental and clinico-morphological research in this field.

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