CHANGES IN MYOCARDIAL STRUCTURE AND FUNCTION AFTER CORONARY ARTERY BYPASS SURGERY

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Abstract: *Cardiovascular disease remains the leading cause of death, and coronary bypass grafting is one of the most effective methods of surgical revascularization of the myocardium in cases of multivascular coronary artery disease. However, the restoration of coronary blood flow is not always accompanied by a complete structural and functional restoration of the heart muscle. The article examines the clinical and morphological changes in the myocardium in the early and long-term postoperative periods after coronary bypass grafting. Particular attention was paid to the phenomenon of "blinded myocardium," ischemic-reperfusion damage, cardiomyocyte apoptosis, and the development of fibrotic changes. It has been shown that, despite improved coronary perfusion and clinical reduction of ischemia symptoms, some patients retain microcirculation disorders and signs of chronic myocardial remodeling. The importance of morphological and ultrastructural changes in the heart muscle in assessing the effectiveness of coronary bypass grafting and predicting postoperative outcomes, as well as the prospects of surgical revascularization methods on a working heart to reduce the severity of myocardial damage, is emphasized.*

Keywords: *coronary heart disease; coronary artery bypass grafting; myocardium; ischemic-reperfusion lesion; stunned myocardium; fibrosis; microcirculation.*

Ischemic heart disease (IHD) remains the leading cause of death among cardiovascular diseases worldwide [1,2]. Coronary artery bypass grafting (CABG) is an effective method of surgical myocardial revascularisation in patients with

multivessel coronary artery disease and improves the prognosis and quality of life of patients [3].

At the same time, restoration of blood flow through the epicardial arteries is not always accompanied by complete structural and functional recovery of the myocardium. This is due to the presence of chronic ischemia, reperfusion injury, systemic inflammatory response, and initial morphological changes in the heart muscle [4,5].

Coronary artery bypass grafting (CABG) is one of the most effective methods of surgical myocardial revascularisation in patients with ischaemic heart disease. Despite clinical improvement, the postoperative period is accompanied by a complex of functional and morphological changes in the myocardium caused by ischaemia-reperfusion, systemic inflammatory response, and the preoperative condition of the heart muscle. In the early postoperative period after CABG, patients often develop transient left ventricular dysfunction, known as "stunned myocardium" syndrome [6]. This condition is characterised by a temporary decrease in myocardial contractility in the absence of irreversible cardiomyocyte necrosis.

Clinically, this manifests itself in a decrease in ejection fraction, cardiac arrhythmias, and the need for inotropic support [7]. In the long term, with successful revascularisation, most patients experience a reduction in angina symptoms, improved cardiac pump function, and increased exercise tolerance [3,8].

Morphological and ultrastructural studies of the myocardium after CABG reveal signs of reversible damage to cardiomyocytes, including cell swelling, cytoplasmic vacuolisation, myofibril disorganisation, and mitochondrial swelling [4,9].

Reperfusion injury is accompanied by apoptosis activation, which is confirmed by increased caspase expression and an increase in the number of TUNEL-positive cardiomyocytes [5]. Prolonged and severe ischaemia may lead to the formation of foci of coagulation necrosis, especially in peri-infarct areas [10]. Chronic myocardial ischemia leads to the development of interstitial and perivascular

fibrosis, which persists even after surgical revascularisation [11]. According to morphometric studies, the degree of fibrosis directly correlates with the duration of IHD and the severity of heart failure [11,12].

After CABG, the progression of fibrotic changes may slow down due to improved coronary perfusion, but scar tissue that has already formed, especially in areas of previous myocardial infarction, is irreversible [12].

Despite the restoration of blood flow through the main coronary arteries, microcirculation dysfunction persists in some patients. Morphologically, this manifests itself as endothelial dysfunction, thickening of the capillary basement membrane, stasis of blood cells, and microthrombosis [13]. Microcirculation disorders limit oxygen delivery to cardiomyocytes and may reduce the functional effectiveness of coronary bypass grafting [13,14]. The use of cardiopulmonary bypass during CABG is accompanied by the development of a systemic inflammatory response, complement activation, and the release of pro-inflammatory cytokines such as interleukin-6 and tumour necrosis factor-α [15]. These processes contribute to the exacerbation of ischaemic-reperfusion injury to the myocardium.

In recent years, methods of coronary artery bypass grafting on a beating heart have been actively studied, allowing to reduce the severity of inflammatory reactions and morphological damage to the myocardium in high-risk patients [8,15].

Conclusion. Clinical and morphological changes in the myocardium after coronary artery bypass grafting are a multifactorial process involving reversible and irreversible damage to cardiomyocytes, interstitial fibrosis, and microcirculation disorders. The degree of restoration of myocardial structure and function is determined by the initial condition of the heart muscle, the duration of ischaemia, and the characteristics of the surgical technique. In-depth study of these changes is an important area for improving the results of surgical treatment of ischaemic heart disease.

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