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CLINICAL AND MORPHOLOGICAL CHANGES IN THE MYOCARDIUM OF THE HEART AFTER CORONARY ARTERY BYPASS GRAFTING

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Abstract: Ischemic heart disease (IHD) remains a leading cause of death globally, and coronary artery bypass grafting (CABG) is a primary surgical intervention for patients with multivessel coronary artery disease. However, while CABG improves clinical outcomes, the restoration of blood flow does not always lead to complete recovery of myocardial structure and function. This study investigates the clinical and morphological changes in the myocardium following CABG, highlighting the effects of ischemia-reperfusion injury, systemic inflammation, and the preoperative myocardial condition. The methodology involved a review of ultrastructural and morphological changes observed in myocardial tissue post-surgery. Findings suggest that myocardial damage, including reversible cardiomyocyte swelling and irreversible fibrosis, persists despite improved coronary perfusion. Inflammatory responses and microcirculation dysfunction further complicate recovery. These findings underscore the need for a deeper understanding of myocardial pathophysiology to improve the long-term outcomes of CABG. The implications of these results point to the importance of optimizing surgical techniques and developing therapies to mitigate reperfusion injury and improve microcirculation in CABG patients.

Keywords: *Ischemic heart disease, coronary artery bypass grafting, myocardial damage, ischemia-reperfusion, fibrosis, microcirculation, systemic inflammation*

Introduction

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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