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**CELL NECROSIS AND REPERFUSION INJURY ARE THE MECHANISMS OF
MYOCARDIAL INFARCTION**

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Abstract: This article analyzes the pathological physiological mechanisms of cell necrosis and reperfusion injury in myocardial cells during myocardial infarction (MI). It examines changes at the cellular level, oxidative stress, intracellular calcium accumulation, inflammatory processes, and apoptosis. Additionally, it discusses new injury mechanisms occurring during reperfusion, including microvascular dysfunction and free radical formation. The article is based on clinical and laboratory findings and provides a scientific basis for the prevention and treatment strategies of MI.

Key words: Myocardial infarction, cell necrosis, reperfusion injury, oxidative stress, apoptosis, microvascular dysfunction, inflammatory mediators

Introduction: Myocardial infarction (MI) is a clinical disease characterized by necrosis of heart muscle cells due to oxygen deprivation and is the most common cause of cardiovascular disease in the world. The development of MI is usually associated with myocardial ischemia, which occurs as a result of coronary artery occlusion or spasm. During ischemia, energy resources in cells decrease, adenosine triphosphate (ATP) deficiency develops, and as a result, cell membranes and organelles are damaged. At the end of this process, cell necrosis begins.

Research Objective: The aim of this study is to identify the pathological physiological mechanisms of necrosis and reperfusion injury in heart muscle cells during myocardial infarction (MI), analyze their changes at the cellular level, and create a scientific basis for developing strategies for the prevention and treatment of MI. The study aims to study intracellular metabolic and biophysical changes, the effects of inflammatory mediators, and oxidative stress during MI.

Materials and methods: The study used clinical and experimental data to study cell necrosis and reperfusion injury during myocardial infarction. In experimental studies, changes at the cellular level were observed by creating ischemia-reperfusion models on heart muscle cells in mice. At the same time, ATP levels, calcium accumulation, and oxidative stress levels in cells were measured. Cells and tissues were examined by ultrastructural analysis and microscopy. The degree of apoptosis and necrosis was determined by immunohistochemical methods. In clinical studies, electrocardiogram (ECG) results, troponin, and CK-MB levels of patients with MI were analyzed. The data obtained were analyzed using statistical methods, and their mean values and standard deviations were calculated. Through these methods, changes at the cellular level, oxidative stress and inflammatory processes occurring during reperfusion, and additional injury mechanisms were studied in detail.

Results and discussion: According to the results of the study, during myocardial infarction, the level of ATP in cells decreases, intracellular calcium accumulation increases, and cell membranes are damaged, which accelerates the necrosis process. During reperfusion, free radicals are formed, oxidative stress increases, and the apoptosis process is activated. At the same time, microvascular dysfunction occurs, which leads to additional damage to the heart muscle. In clinical studies, troponin and CK-MB levels have been shown to reflect cell damage.

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The results show that MI and reperfusion injury negatively affect cardiac function through mutually reinforcing mechanisms. Therefore, proper planning of the reperfusion strategy and therapies that provide cell protection are important in the treatment of MI.

Conclusion: During myocardial infarction, necrosis and reperfusion injury in cells occur through pathological physiological processes. Intracellular calcium accumulation, oxidative stress, and inflammatory mediators play an important role in these processes. During reperfusion, additional damage occurs through cell apoptosis and microvascular dysfunction. Appropriate use of cytoprotective therapies and reperfusion strategies is essential for the prevention and treatment of MI.

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