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MOLECULAR AND FUNCTIONAL MECHANISMS OF CARDIAC PATHOPHYSIOLOGY: FROM CELLULAR INJURY TO ORGAN DYSFUNCTION

Rakhimov Khamidullo Odiljonovich
Assistant, Department of Pathological Physiology,

Andijan State Medical Institute

Abstract:

Background: Cardiac pathophysiology is driven by a complex interaction of molecular, structural, and systemic factors that impair myocardial performance.
Objective: To investigate the fundamental cellular and functional mechanisms contributing to cardiac dysfunction with a focus on ischemia-reperfusion injury, maladaptive remodeling, and electrophysiological disturbances.
Methods: A narrative review and analytical synthesis of 2012–2024 experimental and clinical studies from PubMed and Scopus databases. The study evaluated alterations in myocardial energy metabolism, calcium signaling, and extracellular matrix remodeling.
Results: Ischemia triggers ATP depletion and mitochondrial dysfunction, while reperfusion generates reactive oxygen species leading to oxidative damage. Persistent neurohormonal activation induces pathological hypertrophy and fibrosis, reducing ventricular compliance. Abnormal calcium cycling and gap junction remodeling create arrhythmogenic substrates.
Conclusion: Cardiac pathophysiology represents a dynamic continuum beginning at the molecular level and progressing to overt heart failure. Early detection of mitochondrial and calcium-handling abnormalities may provide novel therapeutic targets.

Keywords: cardiac pathophysiology, ischemia-reperfusion injury, ventricular remodeling, mitochondrial dysfunction, calcium signaling, heart failure.

Introduction

Cardiovascular diseases account for the majority of global deaths, necessitating a detailed understanding of the mechanisms leading to myocardial dysfunction. Pathophysiological changes in the heart arise from a combination of ischemic injury, metabolic disturbances, and maladaptive structural responses. These processes culminate in ventricular remodeling, arrhythmias, and heart failure.

At the molecular level, disruption of mitochondrial oxidative phosphorylation and calcium homeostasis are early events. At the tissue level, chronic pressure or volume overload induces hypertrophy and extracellular matrix remodeling. These mechanisms are interconnected and represent potential targets for preventive and therapeutic interventions.

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Materials and Methods

Data Sources

Literature was reviewed using PubMed and Scopus with the keywords: "cardiac pathophysiology," "ischemia-reperfusion injury," "ventricular remodeling," "calcium signaling," and "mitochondrial dysfunction."

Inclusion Criteria

Peer-reviewed studies between 2012–2024 focusing on cellular and systemic mechanisms of cardiac dysfunction.

Analysis

Data were categorized into ischemic injury mechanisms, hypertrophic remodeling, neurohormonal activation, and electrophysiological disturbances.

Results

Ischemia-Reperfusion Injury

Ischemia leads to ATP depletion, acidosis, and intracellular calcium overload. Reperfusion exacerbates injury through ROS generation, causing lipid peroxidation and mitochondrial permeability transition.

Pathological Ventricular Remodeling

Chronic neurohormonal stimulation (RAAS, sympathetic activation) induces fibroblast proliferation, collagen deposition, and loss of contractile reserve.

Calcium Handling and Arrhythmias

Abnormal function of SERCA2a and ryanodine receptors disrupts excitation-contraction coupling, predisposing to ventricular arrhythmias. Fibrotic tissue creates conduction block and reentry circuits.

Discussion

The study highlights the importance of early mitochondrial protection and calcium homeostasis regulation. Targeting oxidative stress pathways during reperfusion and modulating neurohormonal signaling are key strategies to prevent progression to heart failure. Current research emphasizes gene therapy aimed at restoring SERCA2a function and antifibrotic interventions as future directions.

Conclusion

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Cardiac pathophysiology begins with cellular energy and calcium disturbances, progressing through structural remodeling to organ-level dysfunction. A deeper understanding of these processes enables precision-based approaches to cardiovascular therapy.

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