PATHOPHYSIOLOGY OF MYOCARDIAL INFARCTION

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Abstract: Myocardial infarction (MI), commonly known as a heart attack, occurs due to the sudden occlusion of a coronary artery, leading to ischemia and necrosis of the myocardial tissue. It is a major cause of morbidity and mortality worldwide. This article discusses the underlying mechanisms, including atherosclerosis, plaque rupture, thrombosis, and inflammatory responses, that contribute to MI pathophysiology.

Keywords: Myocardial Infarction, Pathophysiology, Ischemia, Atherosclerosis, Coronary Thrombosis, Cardiac Necrosis

1. Introduction

Myocardial infarction is a life-threatening cardiovascular event characterized by inadequate blood supply to the heart muscle. It is primarily caused by atherosclerotic plaque rupture and thrombus formation in the coronary arteries. Understanding the pathophysiological processes involved in MI is crucial for early diagnosis, prevention, and treatment.

2. Pathophysiological Mechanisms of Myocardial Infarction

2.1 Atherosclerosis and Plaque Formation

Atherosclerosis is a chronic inflammatory process involving lipid accumulation, endothelial dysfunction, and fibrous cap formation in the coronary arteries. The gradual narrowing of arteries leads to reduced myocardial perfusion.

2.2 Plaque Rupture and Thrombosis

The rupture of an unstable atherosclerotic plaque exposes prothrombotic substances, leading to platelet aggregation and thrombus formation. This results in sudden occlusion of the affected coronary artery, causing acute ischemia.

2.3 Ischemia and Myocardial Necrosis

Prolonged ischemia deprives cardiac myocytes of oxygen, leading to energy depletion, calcium overload, and the activation of apoptotic and necrotic pathways. This results in irreversible myocardial cell death.

2.4 Inflammatory Response and Tissue Remodeling

Following myocardial injury, an inflammatory cascade involving neutrophils, macrophages, and cytokines is activated. This process contributes to tissue repair but may also lead to adverse remodeling and heart failure.

3. Clinical Manifestations and Complications

Common symptoms of MI include chest pain, dyspnea, diaphoresis, and nausea. Potential complications include arrhythmias, cardiogenic shock, heart failure, and ventricular rupture.

4. Diagnosis and Treatment Strategies

4.1 Diagnosis

- **Electrocardiography (ECG):** Detects ST-segment changes characteristic of MI.
- **Biochemical Markers:** Elevated troponin and creatine kinase-MB indicate myocardial injury.
- **Imaging Techniques:** Coronary angiography and echocardiography help assess infarct severity.

4.2 Treatment Approaches

- **Reperfusion Therapy:** Percutaneous coronary intervention (PCI) and thrombolytic therapy restore blood flow.
- **Antiplatelet and Anticoagulant Therapy:** Aspirin and P2Y12 inhibitors prevent further thrombosis.
- **Beta-Blockers and ACE Inhibitors:** Reduce myocardial oxygen demand and prevent remodeling.
- **Lifestyle Modifications:** Smoking cessation, dietary changes, and regular exercise lower recurrence risk.

5. Conclusion

Myocardial infarction is a complex pathological event involving ischemia, thrombosis, and inflammatory responses. Early recognition and timely intervention are critical for improving patient outcomes and reducing cardiovascular mortality.

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