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# ISCHEMIC MYOCARDIUM: PATHOPHYSIOLOGY, DIAGNOSIS AND MANAGEMENT

**Murtazayeva Xadicha Nuriddinovna**
Teacher of Termez branch Tashkent medical academy
**Choriyev Firdavs Farxodovich**
Student of Termez branch of Tashkent Medical Academy

**Abstract:** Ischemic myocardium results from insufficient coronary blood flow, leading to myocardial hypoxia and metabolic disturbances. The primary cause is atherosclerotic coronary artery disease (CAD), which contributes to chronic coronary syndromes (CCS) and acute coronary syndromes (ACS), including unstable angina, NSTEMI, and STEMI. This review explores the pathophysiological mechanisms, clinical presentation, diagnostic modalities, and management strategies for ischemic myocardium. It also discusses emerging therapies, including PCSK9 inhibitors, anti-inflammatory treatments, and regenerative medicine approaches. A multidisciplinary approach integrating medical therapy, lifestyle interventions, and revascularization techniques remains essential in reducing cardiovascular morbidity and mortality.

**Introduction:** Cardiovascular disease remains the leading cause of death worldwide, with ischemic myocardium playing a central role in the pathogenesis of ischemic heart disease (IHD). The primary mechanism involves an imbalance between myocardial oxygen supply and demand due to coronary artery obstruction, endothelial dysfunction, and microvascular disease. Understanding the underlying pathophysiology, early detection, and evidence-based management strategies is essential to improving patient outcomes.

## Pathophysiology of Ischemic Myocardium

Ischemic myocardium results from reduced perfusion, leading to metabolic derangements and myocardial injury. Key pathophysiological events include:

### Atherosclerosis and Coronary Obstruction

**Endothelial Dysfunction:** Early-stage atherosclerosis is characterized by reduced nitric oxide (NO) production and increased expression of pro-inflammatory cytokines, adhesion molecules (ICAM-1, VCAM-1), and oxidized LDL.

**Plaque Formation and Progression:** Macrophage infiltration and smooth muscle cell proliferation lead to fibrous cap formation, increasing the risk of rupture.

Plaque Rupture and Thrombosis: Exposure of thrombogenic plaque components triggers platelet aggregation and thrombus formation, precipitating ACS.

### Ischemic Cascade and Cellular Injury

Early Ischemia: Reduced oxygen supply shifts myocardial metabolism to anaerobic glycolysis, leading to ATP depletion, lactic acid accumulation, and intracellular acidosis.

**Myocyte Dysfunction:** Impaired Na+/K+ ATPase activity results in intracellular sodium and calcium overload, disrupting myocardial contractility and increasing the risk of arrhythmias.

**Irreversible Myocardial Necrosis:** Prolonged ischemia (>20-30 minutes) leads to cardiomyocyte death, inflammation, and fibrosis, contributing to heart failure (HF).

**Microvascular Dysfunction:** Patients with ischemia with non-obstructive coronary arteries (INOCA) exhibit microvascular dysfunction due to endothelial and smooth muscle abnormalities, impairing vasodilation and coronary flow reserve.

**Clinical Presentation of Chronic Coronary Syndrome (CCS)**

• Stable angina pectoris (predictable exertional chest pain, relieved by rest/nitroglycerin).

• Symptoms correlate with coronary stenosis (>70% luminal narrowing).

• May present as silent ischemia in diabetic and elderly patients.

**Diagnostic Approach**

Electrocardiography (ECG) **(ECG) remains the** first-line diagnostic tool **for detecting myocardial ischemia and infarction. It provides critical information about ischemic severity, location, and progression. Changes in ECG patterns correlate with the** degree of myocardial ischemia and infarction**, guiding treatment decisions.** Pathophysiology of ECG Changes in Ischemia
**Myocardial ischemia alters** transmembrane ion gradients**, particularly potassium (K+) and calcium (Ca2+), affecting** depolarization and repolarization**.**

• ST-segment depression (NSTEMI/unstable angina) vs. elevation (STEMI). • T-wave inversions in ischemic territories.

Biomarkers of Myocardial Injury. Serum troponins and CK-MB (creatine kinase-MB)are readily detectable and reliable cardiac-specific biomarkers of subclinical myocardial injury. This study explores the roles of cTnI (cardiac troponin I) and CK-MB in hypertrophic cardiomyopathy (HCM)

• Cardiac Troponins (I/T): High specificity for myocardial necrosis. •
Creatine Kinase-MB (CK-MB): Useful for detecting reinfarction.

**Invasive Coronary Angiography.** Cardiac catheterisation is an invasive diagnostic procedure that provides important information about the structure and function of the heart. It usually involves taking X-rays of the heart's arteries (coronary arteries) using a technique called coronary angiography or arteriography.

• Gold standard for high-risk patients to evaluate coronary stenosis.

Among patients who are candidates for treatment with thrombolytic agents, careful management of blood pressure is critical before and during the administration of alteplase (recombinant tissue plasminogen activator [rtPA]), and for the ensuing 24 hours. Excessively high blood pressures are associated with intracerebral hemorrhage after thrombolytic administration.

Thrombolytic therapy is not given to patients who have a systolic blood pressure above 185 mmHg or a diastolic blood pressure above 110 mmHg despite non-aggressive blood pressure-lowering attempts. While there is no established definition of "non-aggressive" blood pressure reduction, a common approach is to use a maximum of two to three attempts with parenteral medications, with options including labetalol, enalaprilat or nicardipine. Uncontrolled blood pressure is an uncommon reason for ineligibility of IV alteplase for AIS.

**Conclusion:** Ischemic myocardium remains a leading contributor to cardiovascular morbidity and mortality. Early diagnosis, evidence-based pharmacotherapy, and timely revascularization are crucial in improving survival. Future research should focus on novel lipid-lowering strategies, anti-inflammatory interventions, and regenerative therapies to enhance myocardial recovery and long-term outcomes.

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