JOURNAL OF MULTIDISCIPLINARY SCIENCES AND INNOVATIONS
GERMAN INTERNATIONAL JOURNALS COMPANY
ISSN: 2751-4390
IMPACT FACTOR (RESEARCH BIB) : 9,08. Academic research index

ACUTE MYOCARDIAL INFARCTION: PATHOPHYSIOLOGY, CLINICAL FEATURES, AND CONTEMPORARY MANAGEMENT

Axmadulina Galiya Marsovna

Abstract: Acute myocardial infarction (AMI) arises from sudden occlusion of a coronary artery, most often following atherosclerotic plaque rupture and superimposed thrombus formation. Within minutes of vessel closure, downstream myocardium experiences ischemic necrosis that, if not promptly reperfused, leads to irreversible injury and loss of contractile function. Clinically, AMI manifests as prolonged chest discomfort – often crushing or constricting – accompanied by diaphoresis, dyspnea, and autonomic features. Electrocardiography differentiates ST-elevation from non-ST-elevation infarctions, while troponin assays confirm myocardial necrosis. Imaging modalities, including echocardiography, cardiac magnetic resonance, and coronary angiography, further delineate infarct extent, guide revascularization decisions, and assess left ventricular function. Early reperfusion via primary percutaneous coronary intervention or fibrinolysis, combined with antiplatelet, anticoagulant, beta-blocker, statin, and renin-angiotensin system inhibitor therapy, markedly reduces infarct size and improves survival. Post-AMI care focuses on prevention of recurrent events through lifestyle modification, pharmacotherapy, and structured cardiac rehabilitation. This review synthesizes current understanding of AMI pathophysiology, clinical presentation, diagnostic strategies, and evidence-based management to optimize patient outcomes.

Keywords: Acute myocardial infarction; Atherosclerotic plaque rupture; ST-elevation myocardial infarction; Percutaneous coronary intervention; Cardiac rehabilitation

Myocardial infarction occurs when an atherosclerotic plaque within a coronary artery ruptures or erodes, triggering the rapid formation of a thrombus that abruptly occludes blood flow. This sudden obstruction deprives downstream myocardium of oxygen and nutrients, leading to ischemic necrosis within minutes. Although collateral vessels can sometimes provide partial perfusion, they rarely compensate fully in the acute setting, and irreversible myocardial injury begins as early as twenty to thirty minutes after occlusion.

Clinically, patients with myocardial infarction often describe a prolonged, severe chest discomfort, distinguishing it from the transient pain of angina. The sensation may be described as crushing, constricting, or burning, typically located retrosternally but possibly radiating to the neck, jaw, left arm, or back. Accompanying symptoms frequently include profuse sweating, nausea, vomiting, dyspnea, and a sense of impending doom. In some individuals- particularly the elderly, women, and those with diabetes - symptoms may be atypical or muted, manifesting as unexplained fatigue, indigestion, or shortness of breath without significant chest pain.

Electrocardiographic changes are central to diagnosing MI. In ST-elevation myocardial infarction, persistent ST-segment elevation appears in leads corresponding to the infarcted territory, often accompanied by reciprocal ST depression in opposing leads. Pathological Q waves may develop within hours to days, reflecting transmural necrosis. In non-ST-elevation myocardial infarction, ST depression or T-wave inversion is seen without persistent ST elevation; biomarkers confirm necrosis. Cardiac troponins- now the gold standard- rise within three to six hours, peak at around twenty-four hours, and remain elevated for up to two weeks. Creatine

kinase-MB isoenzyme provides additional corroboration but lacks the sensitivity and specificity of troponin.

Beyond ECG and biomarkers, imaging plays an important role. Echocardiography reveals regional wall motion abnormalities, which correspond to areas of infarction and can help distinguish MI from other causes of chest pain. Cardiac magnetic resonance imaging offers detailed tissue characterization, identifying edema, necrosis, and scar formation. Coronary angiography confirms the location and extent of occlusion, guides revascularization, and assesses the need for stenting or bypass surgery.

Immediate management focuses on restoring perfusion to minimize infarct size and preserve left ventricular function. Reperfusion is achieved either pharmacologically- using fibrinolytic agents to dissolve the thrombus- or mechanically via primary percutaneous coronary intervention (PCI). When performed promptly, PCI offers superior outcomes, reducing mortality and the risk of complications. Adjunctive therapies include dual antiplatelet therapy (aspirin plus a $P2Y_{12}$ inhibitor), anticoagulation (e.g., heparin), and high-intensity statins to stabilize plaques and reduce inflammation. Beta-blockers decrease myocardial oxygen demand by slowing heart rate and reducing contractility, while ACE inhibitors or angiotensin receptor blockers mitigate remodeling and heart failure risk.

Complications span electrical, mechanical, and inflammatory domains. Ventricular arrhythmias- such as ventricular tachycardia and fibrillation- occur most commonly in the early hours and often cause sudden cardiac death. Heart block may result from ischemia of the conduction system, particularly in inferior infarctions. Mechanical complications, though less frequent in the era of rapid reperfusion, remain serious: papillary muscle rupture leads to acute severe mitral regurgitation and pulmonary edema; ventricular septal rupture causes a left-to-right shunt with hemodynamic collapse; free wall rupture precipitates tamponade. Myocardial inflammation can give rise to pericarditis, presenting days after the infarction.

Long-term care focuses on preventing recurrent events and managing heart failure. Cardiac rehabilitation- including supervised exercise, dietary counseling, and smoking cessation- improves functional capacity and quality of life. Optimizing medical therapy with ACE inhibitors, beta-blockers, statins, and antiplatelet agents reduces the risk of reinfarction and death. Patients undergo periodic assessment of left ventricular function, often with echocardiography, to guide decisions about implantable cardioverter-defibrillators in those with severely reduced ejection fraction.

Myocardial infarction represents the most severe manifestation of ischemic heart disease, marked by abrupt coronary occlusion, irreversible myocardial necrosis, and a spectrum of acute and chronic complications. Rapid recognition, prompt reperfusion, and comprehensive secondary prevention are essential to limit myocardial damage, avert life-threatening events, and improve long-term outcomes.

References:

1. A.Gadayev. Ichki kasalliklar. - Toshkent: “Turon zamin ziyo”, 2016. - 800 b.
2. Amsterdam E.A., Wenger N.K., Brindis R.G., et al. 2014 AHA/ACC Guideline for the Management of Patients With Non-ST-Elevation Acute Coronary Syndromes // *Journal of the American College of Cardiology*. 2014. Vol. 64, No 24. P. e139-e228.
3. Braunwald E., Zipes D.P., Libby P., Bonow R.O. *Heart Disease: A Textbook of Cardiovascular Medicine*. 10th ed. Philadelphia: Elsevier Saunders, 2013. 2496 p.