



Acute Coronary Syndrome in Modern Cardiology: Early Diagnosis, Emergency Management, and Outcome Optimization

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ABSTRACT:

Acute Coronary Syndrome (ACS) represents a spectrum of acute cardiovascular emergencies including ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina. It is primarily caused by sudden reduction or complete blockage of coronary blood flow due to atherosclerotic plaque rupture and thrombus formation. Despite significant advances in interventional cardiology and pharmacotherapy, ACS continues to be a leading cause of mortality globally. Early recognition of symptoms, rapid diagnostic evaluation using ECG and cardiac biomarkers, and timely reperfusion therapy are the most critical determinants of survival. This review provides an expanded discussion of pathophysiology, clinical features, diagnostic advancements, emergency treatment strategies, complications, and future perspectives in ACS management.

KEYWORDS: Acute coronary syndrome, myocardial infarction, STEMI, NSTEMI, troponin, ECG, reperfusion therapy, PCI, thrombolysis, emergency cardiology, atherosclerosis.

INTRODUCTION

Acute Coronary Syndrome is a medical emergency that requires immediate attention due to its rapid progression toward irreversible myocardial damage. It arises when coronary blood flow becomes insufficient to meet myocardial oxygen demand.

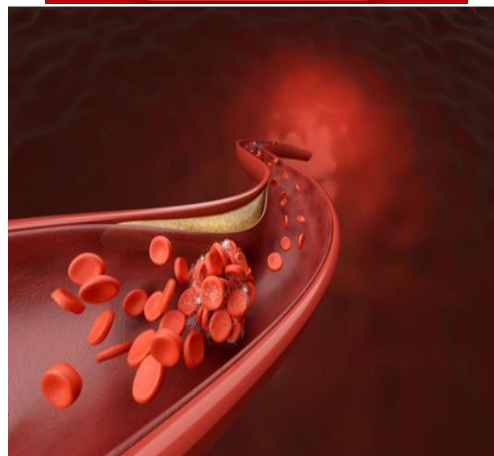
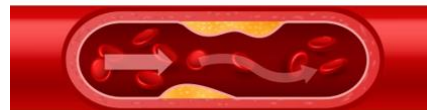
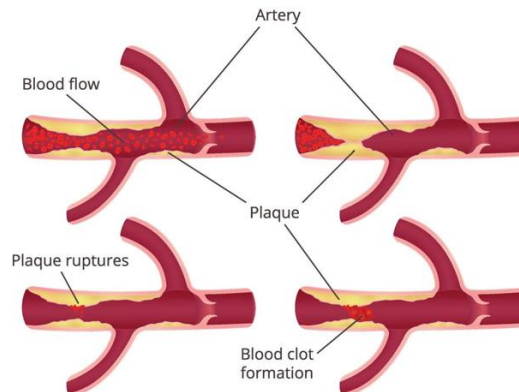
Globally, ACS accounts for millions of deaths annually, and its incidence is increasing due to lifestyle changes, urbanization, obesity, diabetes mellitus, and hypertension. The disease burden is especially high in low- and middle-income countries where delayed diagnosis and limited access to cardiac care facilities worsen outcomes.

ACS is clinically categorized into:

- ST-Elevation Myocardial Infarction [STEMI] – complete coronary occlusion
- Non-ST-Elevation Myocardial Infarction [NSTEMI] – partial occlusion
- Unstable Angina – ischemia without myocardial necrosis.

2. DETAILED PATHOPHYSIOLOGY OF ACS

ATHEROSCLEROSIS mechanisms of plaque formation



The development of ACS involves multiple pathological steps:

i. Atherosclerotic Plaque Formation

- Endothelial injury due to hypertension, smoking, or hyperlipidemia
- LDL cholesterol infiltration into arterial walls
- Formation of fatty streaks and fibrous plaques

ii. Plaque Instability and Rupture

- Thin fibrous cap becomes vulnerable
- Inflammatory cell infiltration weakens plaque structure
- Sudden rupture exposes thrombogenic core

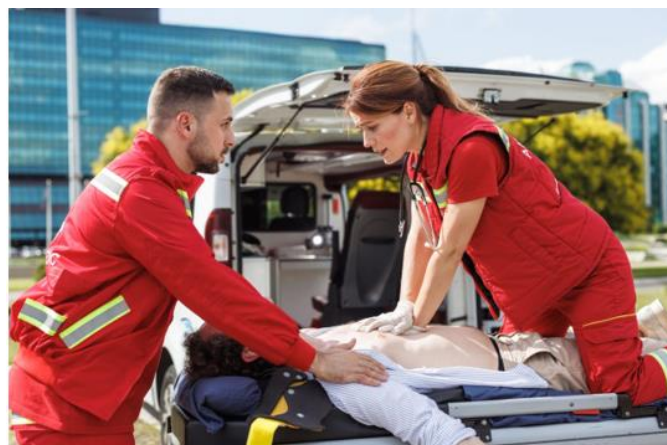
iii. Thrombus Formation

- Platelet activation and aggregation
- Activation of coagulation cascade
- Formation of occlusive or partially occlusive clot

vi. Myocardial Ischemia and Necrosis

- Oxygen deprivation to cardiac muscle
- Cellular ATP depletion
- Irreversible myocardial cell death if not treated quickly

3. CLINICAL FEATURES OF ACS (DETAILED)



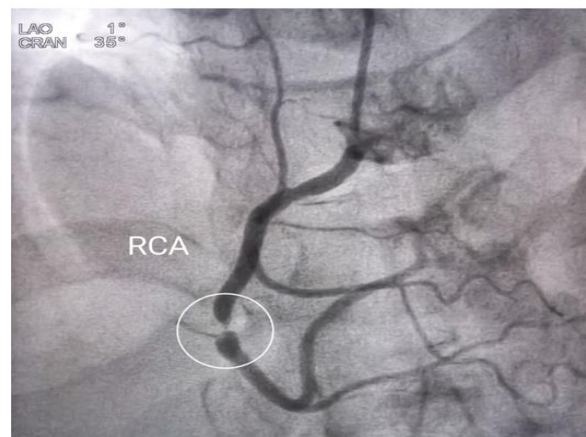
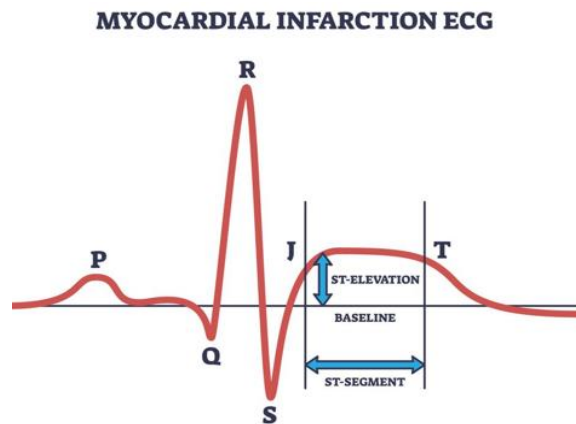
i. Typical Symptoms

- Severe retrosternal chest pain (crushing or pressure-like)
- Pain radiating to left arm, neck, jaw, or back
- Profuse sweating (diaphoresis)
- Shortness of breath (dyspnoea)
- Nausea, vomiting, and anxiety

ii. Atypical Presentations

- More common in diabetic patients, women, and elderly
- Silent ischemia without chest pain
- Fatigue, epigastric discomfort, or syncope

4. DIAGNOSTIC EVALUATION



i. Electrocardiogram (ECG)

- First-line diagnostic tool
- ST elevation indicates STEMI
- ST depression or T-wave inversion suggests ischemia

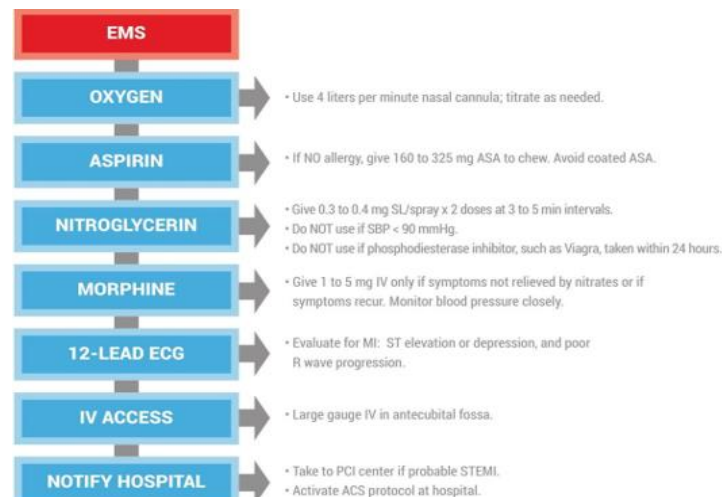
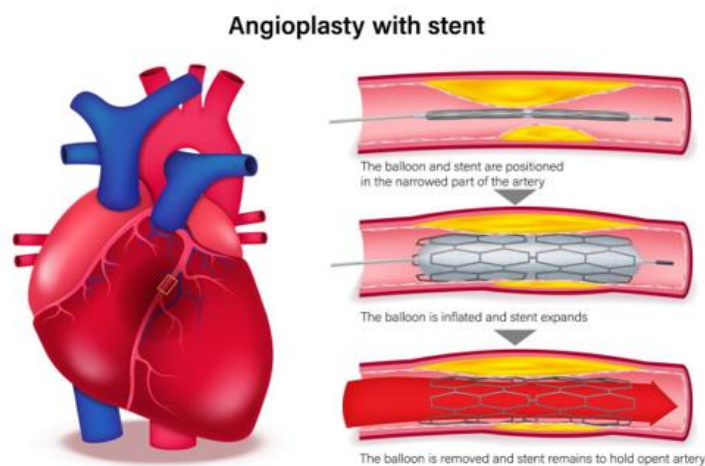
ii. Cardiac Biomarkers

- Troponin I and T are most sensitive markers
- Rise within 3–6 hours after myocardial injury
- Remain elevated for 7–14 days

iii. Imaging Techniques

- Echocardiography for wall motion abnormalities
- Coronary angiography for vessel visualization
- CT coronary angiography for non-invasive assessment

5. EMERGENCY MANAGEMENT OF ACS (EXPANDED)





i. Immediate Medical Therapy

- Aspirin: prevents platelet aggregation
- P2Y12 inhibitors (clopidogrel, ticagrelor)
- Nitro glycerine: reduces myocardial oxygen demand
- Beta-blockers: reduce heart rate and workload
- Anticoagulants: prevent clot extension

ii. Reperfusion Therapy

- Primary PCI (Percutaneous Coronary Intervention)
- Gold standard treatment
- Restores coronary blood flow
- Thrombolytic therapy
- Used when PCI is not available
- Must be given within 30–60 minutes

6. COMPLICATIONS OF ACS

- Life-threatening arrhythmias (ventricular fibrillation)
- Acute heart failure
- Cardiogenic shock
- Pericarditis (post-MI inflammation)
- Mechanical complications (papillary muscle rupture, septal defect)

7. PROGNOSIS AND RISK ASSESSMENT

- Time-to-treatment is the strongest predictor of survival
- Elevated troponin levels indicate poor prognosis
- Reduced left ventricular ejection fraction (LVEF) worsens outcomes
- Diabetes, renal disease, and hypertension increase mortality risk

8. FUTURE DIRECTIONS IN ACS MANAGEMENT

- AI-assisted ECG interpretation for early diagnosis
- Nanomedicine for targeted clot dissolution
- Gene therapy for vascular repair
- Wearable continuous cardiac monitoring devices
- Personalized antiplatelet therapy based on genetic testing

9. CONCLUSION

Acute Coronary Syndrome remains a major global health challenge requiring rapid diagnosis and immediate intervention. Advances in interventional cardiology, biomarker research, and emergency protocols have significantly improved survival rates. However, early recognition of symptoms and timely access to care remain critical. Future developments in precision medicine, artificial intelligence, and regenerative cardiology are expected to further transform ACS management.

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