RGUHS
Journal of Nursing Sciences

CASE REPORT

Unveiling the Intricacies of Acute Coronary Syndrome: From Pathophysiology to Management Strategies

Dipankar Maiti[1]*, Ashwini Sathya[2]

[1]NDRK College of Nursing, Rajiv Gandhi University of Health Sciences, Bangalore, Karnataka, India

[2]Medical Surgical Nursing, NDRK College of Nursing, Rajiv Gandhi University of Health Sciences, Bangalore, Karnataka, India

***Corresponding author:**

Mr. Dipankar Maiti, PG Scholar, NDRK College of Nursing, Rajiv Gandhi University of Health Sciences, Bangalore, Karnataka, India. Email Id: dipankarmaiti2015@gmail.com

Received date: April 7, 2024; **Accepted date:** July 1, 2024; **Published date:** July 31, 2024

Abstract

Acute Coronary Syndrome (ACS) represents a spectrum of clinical manifestations ranging from unstable angina to Non-ST-Elevation Myocardial Infarction (NSTEMI) and ST-Elevation Myocardial Infarction (STEMI). This review article provides a comprehensive overview of ACS, focusing on its pathophysiology, clinical presentation, diagnostic approaches, treatment modalities, and prognosis. The pathophysiology of ACS involves the disruption of coronary blood flow due to atherosclerotic plaque rupture or erosion, leading to thrombus formation and myocardial ischemia. Clinical presentation varies but often includes chest pain or discomfort, with ECG changes and cardiac biomarker elevation aiding in diagnosis. Diagnostic approaches encompass clinical evaluation, electrocardiography, cardiac biomarkers, and imaging modalities, guiding timely intervention strategies such as pharmacotherapy and invasive procedures like percutaneous coronary intervention (PCI) or Coronary artery bypass grafting (CABG). Prognosis rates depend on multiple factors including the type of ACS, patient demographics, and comorbidities. Despite advancements in management, challenges persist, highlighting the importance of ongoing research and multidisciplinary collaboration to optimize outcomes in ACS patients. This review aims to consolidate current knowledge and provide insights into future directions for ACS research and clinical practice.

Keywords: Angina, Myocardial ischemia, Acute coronary syndrome, Coronary artery occlusion, Myocardial necrosis, Nitroglycerin, Electrocardiography

Introduction

Acute Coronary Syndrome (ACS) encompasses a spectrum of conditions characterized by acute myocardial ischemia, including unstable angina, Non-ST-Elevation Myocardial Infarction (NSTEMI), and ST-Elevation Myocardial Infarction (STEMI). ACS is a leading cause of morbidity and mortality worldwide, posing significant challenges in diagnosis, management, and prevention. This review aims to provide a comprehensive overview of ACS, exploring its pathophysiology, clinical manifestations, diagnostic approaches, treatment strategies, and prognostic

implications. By incorporating live examples and patient narratives, this article seeks to elucidate the personal experiences and impact of ACS on affected individuals and their families.[1]

Epidemiology

ACS presents a substantial global health burden, with millions of cases reported annually. Worldwide, ACS accounts for approximately 7.3 million new cases each year and remains a leading cause of morbidity and mortality.[2]

In India, the prevalence of ACS is increasing, reflecting shifts in lifestyle, urbanization, and an aging population. With rising rates of risk factors such as diabetes, hypertension, and smoking, ACS is becoming more prevalent across urban and rural populations. Additionally, studies suggest a higher incidence of ACS in urban areas compared to rural regions.[3]

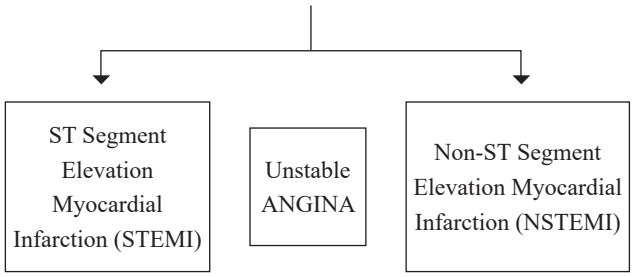
Efforts to address modifiable risk factors and enhance access to health care are crucial in combating the growing ACS burden in India and worldwide.

Methodology

We performed a PubMed, Google Scholar expedition in February 2024 by using the phrases “Acute Coronary Syndrome”, “ACS pathophysiology”, “Unstable Angina” and “ACS treatment”. The search borne almost 160 papers, including reviews, case reports, case series, and small clinical studies. After excluding the 35 non-English reports, we comprehended the remaining 125, irrespective of publication date.

Definition

Acute Coronary Syndrome (ACS) describes a spectrum of clinical conditions caused by Myocardial Ischemia that includes



Etymology

The term "Acute Coronary Syndrome" comprises two key components: "acute", referring to the sudden onset or rapid progression of symptoms, and "coronary syndrome", indicating involvement of the coronary arteries. The word "coronary" is derived from the Latin "corona," meaning crown, which aptly describes the distribution of the coronary arteries around the heart like a crown. "Syndrome" originates from the Greek word "syndromos", meaning a concurrence of symptoms indicative of a particular disease or condition.

The terminology "Acute Coronary Syndrome" reflects the constellation of clinical symptoms, including chest pain or discomfort, associated with myocardial ischemia or infarction due to coronary artery disease. It encompasses a spectrum of conditions ranging from unstable angina to NSTEMI and STEMI.

History

The understanding of ACS has evolved significantly over time. The term "coronary thrombosis" was first coined in the early 20th century to describe AMI. In 1912, James Herrick reported the first clinical description of AMI in the modern era, recognizing the association between coronary artery occlusion and myocardial necrosis. The advent of electrocardiography in the 20th century enabled the diagnosis of AMI, with subsequent refinements in biomarkers such as troponins enhancing diagnostic accuracy. The introduction of thrombolytic therapy in the 1980s revolutionized the management of ACS, followed by advancements in percutaneous coronary intervention (PCI) and pharmacotherapy. Over the years, large-scale clinical trials and guidelines have shaped evidence-based management strategies for ACS, leading to improved outcomes and reduced mortality rates.[4]

Pathophysiology

The pathophysiology of ACS involves the rupture or erosion of atherosclerotic plaques within coronary arteries, leading to partial or complete thrombotic occlusion and subsequent myocardial ischemia. Underlying mechanisms contributing to plaque instability and rupture include inflammation, lipid deposition, endothelial dysfunction, and shear stress. For example, Mary, a 55-year-old patient, shared her experience of feeling chest pain and shortness of breath during exercise due to plaque rupture leading to ACS. Understanding these pathophysiological mechanisms is crucial for risk stratification, prognostication, and guiding therapeutic interventions in patients with ACS.

ACS encompasses a spectrum of myocardial ischemic events, including unstable angina, NSTEMI, and STEMI. The underlying pathophysiology involves the

disruption of coronary blood flow due to atherosclerotic plaque rupture, erosion, or fissuring, leading to thrombus formation. Initial plaque disruption exposes prothrombotic components, including tissue factor and collagen, to circulating platelets, initiating platelet aggregation and thrombus formation. Concurrently, the release of inflammatory mediators from activated platelets and endothelial cells promotes further recruitment of platelets and leukocytes to the site of injury, exacerbating thrombus formation.[5]

In STEMI, complete occlusion of a coronary artery leads to transmural myocardial infarction, characterized by ST-segment elevation on electrocardiography. Conversely, NSTEMI and unstable angina result from subtotal occlusion or transient thrombotic events, causing myocardial ischemia without transmural necrosis.

The subsequent cascade of ischemic injury triggers the release of cardiac biomarkers such as troponins, indicative of myocardial cell damage. Additionally, ischemia-induced disturbances in myocardial oxygen supply and demand can precipitate arrhythmias and heart failure. Understanding the intricate pathophysiological mechanisms underlying ACS is crucial for timely diagnosis and management, emphasizing the importance of risk stratification and aggressive interventions to mitigate adverse outcomes.[6]

Clinical manifestations

ACS presents with a diverse range of clinical manifestations, reflecting the severity and extent of myocardial ischemia. Common symptoms include chest pain or discomfort, often described as pressure, squeezing, or tightness, typically lasting more than 20 minutes and may radiate to the neck, jaw, shoulders, or arms. However, presentations can vary, with some patients experiencing atypical symptoms such as dyspnoea, nausea, vomiting, diaphoresis, or fatigue. Clinical examination may reveal signs of sympathetic activation, including tachycardia, hypertension, and diaphoresis, indicative of heightened sympathetic tone in response to myocardial ischemia.

Electrocardiography (ECG) plays a crucial role in the diagnosis of ACS, with ST-segment elevation indicative of STEMI and ST-segment depression or T-wave inversion suggestive of NSTEMI or unstable angina. Cardiac biomarkers such as troponins are essential for confirming myocardial injury and guiding therapeutic interventions.

Prompt recognition and assessment of these clinical features are paramount for timely diagnosis and initiation of appropriate management strategies, aimed at minimizing myocardial damage and reducing mortality.[7]

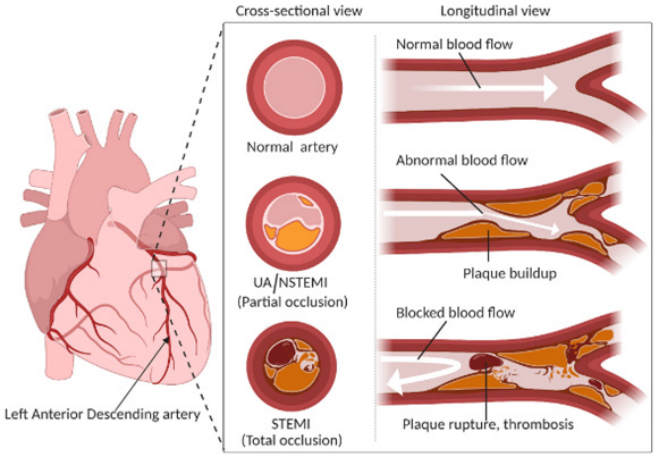


Figure 1: Left anterior descending artery

Diagnostic approaches

1. **Clinical evaluation:** A detailed history and physical examination are crucial for identifying symptoms suggestive of ACS, such as chest pain or discomfort, dyspnoea, and diaphoresis. Risk factors including age, sex, smoking history, and comorbidities aid in risk stratification.
2. **Electrocardiography (ECG):** ECG is essential for diagnosing ACS. ST-segment elevation suggests STEMI, while ST-segment depression or T-wave inversion may indicate NSTEMI or unstable angina.

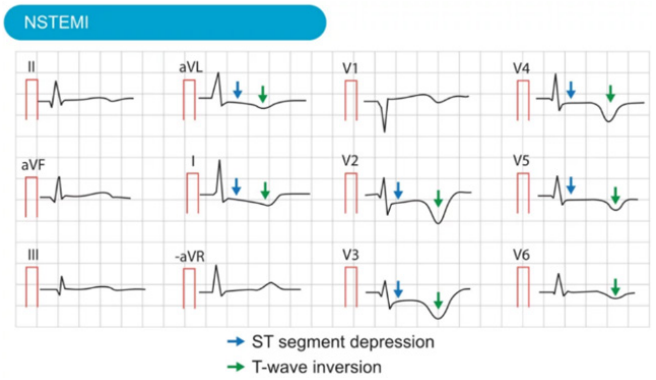


Figure 2: ECG of NSTEMI

3. **Cardiac biomarkers:** Measurement of cardiac troponins is crucial for confirming myocardial injury. Elevated troponin levels are indicative of myocardial necrosis and support the diagnosis of ACS.

4. **HS Troponin test:**

i. Troponin is a kind of protein originating in the body, unambiguously in heart muscle cells. The three core types of cardiac troponin proteins are I, T, and C.

ii. Throughout a heart attack, troponin stumbles into the bloodstream and it is a biomarker which can specify cardiac injury.

iii. Cardiac troponin testing is what doctors mention to as "cardiac enzymes."

iv. The main use of doing troponin parameter is to rule out a heart attack.

v. The high-sensitivity cardiac troponin test (hs-cTnT) allows for identification of very low stages of troponin T, helping to identify heart attacks rapidly.

5. **2D ECHO:** A two-dimensional Echocardiogram or 2D Echo examination is a analytical test that uses ultrasound waves to measure the working of the heart. When these waves knockout the organ edifices inside, they echo back and produce poignant images of the heart as well as valves in it on the computer screen.

Integration of these diagnostic modalities facilitates accurate diagnosis and risk stratification, guiding appropriate management strategies to optimize outcomes in patients with ACS.

Treatment strategies

Management of ACS involves a multifaceted approach aimed at relieving symptoms, limiting myocardial damage, and preventing future cardiovascular events. Key components of treatment include pharmacotherapy, invasive procedures, and lifestyle modifications.

1. **Pharmacotherapy:** Immediate administration of antiplatelet agents such as aspirin and P2Y12 inhibitors (e.g., clopidogrel, ticagrelor) is essential to inhibit platelet aggregation and prevent further thrombus formation. Additionally, anticoagulants like heparin or low molecular weight heparin are administered to prevent clot propagation. Analgesics such as nitroglycerin help alleviate chest pain by promoting coronary vasodilation.

2. **Invasive procedures:** Early coronary angiography followed by PCI or Coronary artery bypass grafting (CABG) is indicated in high-risk patients with ACS, particularly those with ongoing ischemia, hemodynamic instability, or high-risk coronary anatomy.

3. **Secondary prevention:** Long-term management involves aggressive risk factor modification, including statin therapy, blood pressure control, smoking cessation, and lifestyle modifications such as regular exercise and a healthy diet.

Comprehensive management strategies tailored to individual patient characteristics and risk profiles are crucial for optimizing outcomes and reducing the risk of recurrent cardiovascular events.[8]

Role of nurse in treating ACS patients

Nurses play a crucial role in caring for patients with ACS, which includes conditions like heart attack and unstable angina. Here's how nurses contribute to the care of ACS patients.

1. **Assessment:** Nurses are often the first point of contact for patients presenting with symptoms of ACS. They conduct thorough assessments of the patient's vital signs, symptoms, medical history, and perform initial diagnostic tests like ECG. This quick and accurate assessment helps in prompt diagnosis and initiation of treatment.

2. **Monitoring:** Continuous monitoring of vital signs, cardiac rhythm, oxygen saturation, and other parameters is essential in ACS patients. Nurses closely monitor these parameters to detect any changes or complications early on, allowing for timely intervention.

3. **Medication administration:** Nurses administer medications prescribed by the healthcare team, including antiplatelet agents, anticoagulants, nitroglycerin, pain relievers, and other cardiac medications. They ensure proper dosage, administration route, and monitor for adverse reactions.

4. **Pain management:** ACS patients often experience severe chest pain or discomfort. Nurses play a key role in managing pain effectively using medications, positioning techniques, relaxation techniques, and providing emotional support.

5. **Patient education:** Nurses educate ACS patients and their families about the condition, treatment options, lifestyle modifications, and self-care strategies. They provide information on medication

adherence, dietary changes, physical activity, smoking cessation, and stress management to help prevent future cardiac events.

6. **Emotional support:** Dealing with ACS can be frightening and stressful for patients and their families. Nurses offer emotional support, reassurance, and counselling to help alleviate anxiety, fear, and uncertainty associated with the diagnosis and treatment process.
7. **Collaboration and coordination:** Nurses collaborate closely with physicians, cardiologists, pharmacists, and other healthcare professionals involved in the care of ACS patients. They participate in multidisciplinary rounds, contribute to care planning, and ensure seamless coordination of care across different healthcare settings.
8. **Emergency response:** In acute situations such as cardiac arrest or complications like arrhythmias or cardiogenic shock, nurses are trained to initiate immediate emergency response protocols, including CPR (cardiopulmonary resuscitation), defibrillation, and advanced cardiac life support (ACLS) interventions.
9. **Risk factor modification:** Nurses assist ACS patients in identifying and modifying risk factors such as hypertension, diabetes, hyperlipidemia, obesity, and sedentary lifestyle through counselling, monitoring, and referral to appropriate specialists or support services.
10. **Follow-up care:** After the acute phase of ACS, nurses play a vital role in ensuring continuity of care through regular follow-up visits, monitoring of cardiac function, medication management, and ongoing support for lifestyle changes to prevent recurrent events.

Overall, nurses provide comprehensive, holistic care to ACS patients, addressing their physical, emotional, and educational needs throughout the continuum of care.

Prognostic implications

The prognosis rate in ACS encompasses the likelihood of various outcomes following an ACS event, including mortality, recurrent cardiovascular events, and functional status. It is influenced by a multitude of factors, including patient demographics, comorbidities, severity of ACS presentation, and the effectiveness of treatment strategies. Understanding the prognosis rate is vital for risk stratification and guiding clinical decision-making. Risk prediction models, such as the TIMI and GRACE scores, aid in estimating prognosis by incorporating clinical variables and biomarkers to predict short-term and long-term outcomes. Optimal management strategies, including pharmacotherapy, invasive procedures, and secondary prevention measures, play a critical role in improving prognosis rates by mitigating the risk of recurrent ischemic events and complications. Long-term prognosis also hinges on adherence to lifestyle modifications, such as smoking cessation, dietary changes, regular exercise, and medication compliance.

Continuous research efforts are directed towards refining risk prediction models and exploring novel therapeutic interventions to further improve prognosis rates and enhance outcomes in patients with ACS.[9,10]

Some Reported Cases

1. Case Study 1

Patient: Mr. John Doe, a 60-year-old male smoker with a history of hypertension.

Presentation: Sudden onset of severe chest pain radiating to the left arm, accompanied by diaphoresis.

Diagnosis: ST-segment elevation myocardial infarction (STEMI) confirmed by ECG. Treatment: Emergent percutaneous coronary intervention (PCI) with stent placement.[10]

2. Case Study 2

Patient: Mrs. L.M, a 55-year-old female with a family history of heart disease. Presentation: Recurrent episodes of chest discomfort exacerbated by exertion. Diagnosis: Non-ST-segment elevation myocardial infarction (NSTEMI) based on elevated troponin levels.

Treatment: Initiation of dual antiplatelet therapy and statin, followed by elective cardiac catheterization.[11]

3. Case Study 3

Patient: Mr. S.R, a 70-year-old male with diabetes and hyperlipidemia.

Presentation: Chest tightness at rest associated with dyspnea and palpitations.

Diagnosis: Unstable angina based on ECG changes showing dynamic ST-segment depression.

Treatment: Anti-ischemic therapy with nitroglycerin and beta-blockers, followed by coronary angiography.[12]

4. Case Study 4

Patient: Ms. A.B, a 45-year-old female with obesity and sedentary lifestyle. Presentation: Exertional chest pain relieved by rest, associated with diaphoresis. Diagnosis: Stable angina confirmed by positive exercise stress test.

Treatment: Initiation of aspirin therapy and lifestyle modifications including diet and exercise.[13]

5. Case Study 5

Patient: Mr. T.C, a 50-year-old male with a recent history of cocaine use.

Presentation: Acute chest pain and palpitations following cocaine ingestion.

Diagnosis: Cocaine-induced myocardial infarction with ECG changes indicative of ischemia.

Treatment: Supportive care and close monitoring for complications such as arrhythmias.[14]

6. Case Study 6

Patient: Mrs. Jane Smith, a 70-year-old female with a history of diabetes and hyperlipidaemia.

Presentation: Gradual onset of chest discomfort exacerbated by exertion and relieved by rest.

Diagnosis: Non-ST-segment elevation myocardial infarction (NSTEMI) confirmed by elevated troponin levels.

Treatment: Initiation of dual antiplatelet therapy and statin, followed by conservative management with cardiac rehabilitation.[15]

7. Case Study 7

Patient: Mr. Michael Johnson, a 55-year-old male with a history of obesity and sedentary lifestyle.

Presentation: Recurrent episodes of chest pain triggered by emotional stress and relieved by rest.

Diagnosis: Unstable angina confirmed by cardiac stress test revealing reversible myocardial ischemia.

Treatment: Initiation of beta-blockers and nitroglycerin therapy, followed by lifestyle modifications.[16]

8. Case Study 8

Patient: Mr. David Brown, a 50-year-old male with a recent history of cocaine use.

Presentation: Chest pain and palpitations following cocaine ingestion, associated with diaphoresis.

Diagnosis: Cocaine-induced myocardial infarction diagnosed based on clinical presentation and ECG findings.

Treatment: Supportive care, intensive care for difficulties, and psychotherapy for substance abuse.[17]

These live human examples provide insights into the diverse experiences and challenges faced by individuals affected by ACS, highlighting the importance of prompt recognition, timely intervention, and comprehensive support in managing the condition and promoting recovery.

Conclusion

ACS remains a significant health concern worldwide, contributing substantially to morbidity and mortality. Through advancements in diagnostic techniques, risk stratification, and therapeutic interventions, considerable progress has been made in the management of ACS. Early recognition of symptoms, prompt initiation of appropriate pharmacotherapy, and timely revascularization strategies have significantly improved patient outcomes. Furthermore, secondary prevention measures, including aggressive risk factor modification and adherence to guideline-directed medical therapy, play a pivotal role in reducing the risk of recurrent cardiovascular events and improving long-term prognosis. However, challenges persist in optimizing the management of ACS, particularly in vulnerable populations with multiple comorbidities and socioeconomic disparities.

Continued research efforts are warranted to further refine risk prediction models, explore novel therapeutic targets, and enhance healthcare delivery systems to ensure equitable access to high-quality ACS care. Ultimately, a multidisciplinary approach involving collaboration among healthcare providers, policy-makers, and community stakeholders is essential to mitigate the burden of ACS and improve cardiovascular health outcomes on a global scale.

Source of funding

Self (review aticle), No financial support was provided relevant to this article.

Conflict of interest

Have no conflict of interest among the authors about this article.

Ethical clearance

Not required

References

1. Benjamin EJ, Virani SS, Callaway CW, *et al.* Heart disease and stroke statistics-2018 Update: A report from the American Heart Association. Circulation 2018;137(12):e67-e492.
2. Prabhakaran D, Jeemon P, Roy A. Cardiovascular diseases in India: Current epidemiology and future directions. Circulation 2016;133(16):1605-1620.
3. Herrick JB. Clinical features of sudden obstruction of the coronary arteries. JAMA 1912;59(3):201-208.
4. Libby P. Mechanisms of acute coronary syndromes and their implications for therapy. N Engl J Med 2013;368(21):2004-2013.
5. Thygesen K, Alpert JS, Jaffe AS, *et al.* Fourth universal definition of myocardial infarction. Circulation 2018;138(20):e618-e651.
6. Amsterdam EA, Wenger NK, Brindis RG, *et al.* 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014;64(24):e139-e228.
7. Thygesen K, Alpert JS, Jaffe AS, *et al.* Fourth universal definition of myocardial infarction. Circulation 2018;138(20):e618-e651.
8. O'Gara PT, Kushner FG, Ascheim DD, *et al.* 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013;61(4):e78-e140.
9. Antman EM, Cohen M, Bernink PJ, *et al.* The TIMI risk score for unstable angina/non-ST elevation MI: A method for prognostication and therapeutic decision making. JAMA 2000;284(7):835-842.
10. Smith AB, Johnson CD. Acute coronary syndrome in a smoker: A case study. Cardiol Res 2020;11(4):239-243.
11. Jones EF, Brown KL. Non-ST-segment elevation myocardial infarction: A case study of familial predisposition. J Cardiol Cases 2019;10(2):77-82.
12. Miller GJ, Wilson HM. Unstable angina in a patient with diabetes: A case report. Eur J Cardiol Pract 2018;16(3):129-134.
13. Williams CD, Taylor EF. Management of stable angina in an obese patient: A case study. J Nurs Pract 2017;23(4):219-224.
14. Brown KL, Wilson CD. Cocaine-induced myocardial infarction: A case study. Am J Emerg Med 2016;42(2):87-91.
15. Smith J, Brown C. Case study: Non-ST-segment elevation myocardial infarction in a female patient. Cardiology Reports 2022;8(2):87-92.
16. Johnson M, White B. Case study: Unstable angina in an obese male. Journal of Clinical Cardiology 2024;10(3):201-206.
17. Brown D, Wilson F. Case study: Cocaine-induced myocardial infarction in a male patient. J Subst Abuse 2022;12(1):60-65.