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Acute Coronary Syndrome – A Clinical Update and Review

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Abstract

Background: Acute coronary syndrome continues to pose a major global mortality burden. Many studies have sought to classify and define its major etiology and pathophysiology. This review discusses the definition, classification, etiologies, diagnostic criteria, and differential diagnostic methods, as well as the newest updates in management strategies for each ACS subtype.
Main: This review separates acute coronary syndrome into subgroups based on etiology and pathogenesis, including ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), unstable angina, and newer subtypes such as type 2 MI and myocardial infarction with non-obstructive coronary arteries (MINOCA). Management strategies differ for each group: timely reperfusion is emphasized for STEMI, early invasive therapy for high-risk NSTEMI, and evidence-based pharmacotherapy, including dual antiplatelet therapy, high-intensity statins, and renin–angiotensin system inhibitors is indicated for all type of ACS. Secondary prevention through cardiac rehabilitation and lifestyle modification remains essential.
Findings: Evidence highlights the importance of early recognition, especially in patients with atypical presentations such as the elderly, women, and individuals with diabetes. Guideline-directed therapies and revascularization have shown to reduce mortality even though complications including arrhythmias, cardiogenic shock, and heart failure persist. Emerging therapies—such as PCSK9 inhibitors, anti-inflammatory agents, and digital health tools—offer promise in reducing residual risk.
Conclusion: Acute coronary syndrome continues to account for a significant proportion of morbidity and mortality worldwide despite advances in diagnosis and treatment. Recognition of subtypes such as type 2 MI and MINOCA has refined management, while early diagnosis, reperfusion, and secondary prevention remain central to improving outcomes. Ongoing challenges highlight the need for precision medicine, novel therapies, and integrated long-term strategies.

Keywords: Acute Coronary Syndrome, ST-elevation myocardial infarction, Non-ST-elevation myocardial infarction, Myocardial infarction with non-obstructive coronary arteries.

Introduction

Acute coronary syndrome (ACS) remains a major contributor to global disease prevalence, accounting for 9.5% among 5,071,185 individuals aged ≥60 years and 3.8% among 2,982,671 individuals aged <60 years[1]. Over the years, diagnostic modalities and management strategies for ACS have evolved substantially, driven by the continuous emergence of high-quality scientific evidence. This article aims to provide an updated synthesis of current knowledge regarding the etiology, pathogenesis, diagnostic approaches, and therapeutic strategies for ACS, with the objective of enhancing clinical recognition and optimizing patient outcomes. Specifically, this review addresses the definition, classification, etiologies, diagnostic and differential diagnostic methodologies, as well as evidence-based treatment plans for each ACS subtype.

Definition and Classification

The term acute coronary syndrome (ACS) refers to patients with suspicion or confirmation of myocardial ischemia[2]. ACS is divided into 3 main types: ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI) and unstable angina (UA). Myocaridal infarction (MI) is defined as an event of myocardial ischemia with the accompanying evidence of myocardial injury[2]. Confirmed evidence composed of the rise and fall of cardiac biomarkers (troponin) with at least one value >99th percent upper reference limit[2]. Unstable angina is defined when patients lack the evidence of changes in ECG pattern or biomarkers indicative of myocardial ischemia[2].

The joint task force has refined the definition of MI by creating a clinical classification based on

Table 1: Types of Myocardial Infarction (MI) according to the Fourth Universal Definition of MI.
CABG: coronary artery bypass graft; CAD: coronary artery disease; cTn: cardiac troponin; PCI: percutaneous coronary intervention; URL: upper reference limit.

Type	Definition	Key Criteria / Mechanism
1	Myocardial infarction (MI) resulting from acute atherothrombotic coronary artery disease	Usually triggered by atherosclerotic plaque disruption (rupture or erosion).
2	MI due to imbalance between myocardial oxygen supply and demand	Potential mechanisms include coronary dissection, vasospasm, coronary embolism, microvascular dysfunction, or increased oxygen demand, with or without pre-existing CAD.
3	MI with typical clinical features of myocardial ischemia/infarction resulting in unexpected death before biomarker confirmation	Examples include new ischemic ECG changes or ventricular fibrillation, with death occurring before cardiac troponin sampling or detection.
4a	MI related to percutaneous coronary intervention (PCI)	cTn >5× 99th percentile URL (if baseline normal) or >20% increase to a value >5× URL (if baseline elevated/stable/falling) *and* supporting evidence of new ischemia from ECG, imaging, or PCI-related complications (e.g., coronary dissection, major artery/side branch occlusion or thrombus, collateral flow interruption, slow/no-reflow, distal embolization).
4b	PCI-associated MI due to stent or scaffold thrombosis	Confirmed via angiography or autopsy, using diagnostic principles similar to Type 1 MI.
5	MI associated with coronary artery bypass graft (CABG) surgery	cTn >10× 99th percentile URL (if baseline normal) or >20% increase to >10× URL (if baseline elevated/stable/falling) *and* at least one of: new pathological Q waves, angiographic evidence of graft or native vessel occlusion, or imaging evidence of new loss of viable myocardium/regional wall motion abnormality consistent with ischemia.

the presumed underlying cause of the myocardial ischemia[2] (Table 1)

Etiology and Pathogenesis

Most myocardial infarctions arise when an unstable atherosclerotic plaque in a coronary artery ruptures or erodes, triggering the formation of a thrombus and, in some cases, microemboli. These events markedly impair coronary blood flow, resulting in myocardial ischemia and subsequent tissue damage. Less frequent causes of myocardial ischemia include coronary artery spasm, embolism, and arterial dissection[3].

Risk factors substantially impact the predisposition to myocardial infarction and are systematically classified into modifiable and non-modifiable categories. Modifiable factors, such as tobacco use, hypertension, dyslipidemia, diabetes mellitus, central obesity, psychosocial stressors, sedentary behavior, suboptimal dietary patterns, and regular alcohol intake, account for the vast majority of MI risk globally, exceeding 90%[4]. Conversely, non-modifiable risk determinants include advancing age (defined as >45 years in men and >55 years in women), male gender, a positive family history of premature coronary artery disease, and inherited genetic susceptibilities[5]. Emerging risk factors encompass psychosocial stress, exposure to air pollution, and inflammatory disorders, including autoimmune diseases and chronic infections[6, 7]. In younger populations, myocardial infarction may more commonly result from other causes such as hypercoagulable conditions (e.g., pregnancy, factor V Leiden mutation) or substance use, including cannabis, cocaine, and androgenic anabolic steroids[8].

Table 2: Categories of risk factors for myocardial infarction (MI) and their impact.
ASCVD: atherosclerotic cardiovascular disease; BMI: body mass index; FH: familial hypercholesterolemia; LDL: low-density lipoprotein.

Category	Examples	Evidence and Impact
Modifiable	Smoking, hypertension, dyslipidemia (e.g., high LDL), diabetes, obesity (BMI >30), sedentary lifestyle, poor diet (low fruits/vegetables)	Account for ∼90% of risk; smoking doubles MI risk[9], hypertension increases it by 2-3 fold[10].
Non-modifiable	Age, male sex, family history (first-degree relative with MI <50 years), genetics (e.g., familial hypercholesterolemia)	Risk rises exponentially with age[11]; FH increases ASCVD risk 4,1 times higher, accelerates onset in both genders[12].
Emerging / Other	Psychosocial stress, air pollution, autoimmune diseases, infections	Stress and pollution contribute to plaque instability[13–15]; infections may trigger acute events [7].

Atherosclerosis is the fundamental pathological process in the majority of myocardial infarction cases. It initiates with endothelial dysfunction caused by risk factors such as hypertension and dyslipidemia, which promotes lipid deposition, inflammation, and the development of fibrous plaques [16–18]. Stable plaques, marked by thick fibrous caps, generally lead to stable angina, with symptoms precipitated

by physical exertion. In contrast, unstable plaques possess a lipid-rich core and thin fibrous caps, making them vulnerable to rupture and thereby elevating the risk of acute coronary syndrome (ACS). Within these plaques, inflammatory cells like macrophages release matrix metalloproteinases that degrade the extracellular matrix, weakening the structural integrity of the fibrous cap [19, 20]. Minor mechanical or physiological stress can cause cap rupture, exposing the thrombogenic lipid core to the bloodstream [21]. This exposure triggers platelet activation and the coagulation cascade, resulting in thrombus formation and subsequent coronary artery occlusion, which characterizes type 1 myocardial infarction [22].

Partial occlusion of a coronary artery reduces myocardial blood flow, resulting in an imbalance between oxygen supply and demand that predominantly affects the subendocardial region of the heart muscle. Clinically, this condition is often manifested as unstable angina or non-ST-elevation myocardial infarction (NSTEMI) [3]. In contrast, complete coronary artery occlusion causes a more severe impairment of blood flow, leading to extensive myocardial cell death in the absence of timely reperfusion. This full-thickness damage, or transmural infarction, commonly presents as ST-elevation myocardial infarction (STEMI) [3].

Ischemia lasting beyond 20-30 minutes initiates a cascade of cellular damage: ATP depletion, mitochondrial dysfunction, sarcolemmal disruption, and cellular edema. Necrosis progresses in a wavefront from the subendocardium to the epicardium, with STEMI involving full-thickness damage and NSTEMI primarily subendocardial. Reperfusion may worsen damage via oxidative stress and inflammation [18]. Type 2 myocardial infarction arises from an imbalance between myocardial oxygen supply and demand, and can occur in individuals with or without preexisting coronary artery disease. A reduction in oxygen supply may result from coronary artery occlusion due to mechanisms such as vasospasm or dissection, or from systemic conditions leading to decreased perfusion, including hypotension, bradycardia, or anemia. Conversely, an increase in myocardial oxygen demand, commonly seen in sustained tachyarrhythmias, can also precipitate this type of infarction. Unlike type 1 myocardial infarction, type 2 MI does not involve an acute atherothrombotic event as the primary mechanism [2].

Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) is a heterogeneous condition characterized by myocardial infarction without significant coronary artery obstruction [23]. Its pathogenesis includes atherosclerotic plaque disruption with transient thrombosis, coronary artery spasm, spontaneous coronary artery dissection (SCAD), microvascular dysfunction, and thromboembolism. These mechanisms lead to myocardial ischemia and injury despite angiographically normal or near-normal vessels [24]. Nonischemic myocardial injury, such as in sepsis, involves necrosis of myocardial tissue without ischemia [25]. The pathophysiology includes inflammatory cytokines (e.g., TNF-α), catecholamine toxicity, and direct myocyte damage from toxic agents [25].

Clinical Presentations

The clinical presentation of myocardial infarction (MI) varies widely, ranging from classic chest pain to subtle or absent symptoms, complicating timely diagnosis. Accurate recognition of both typical and atypical presentations is critical for initiating prompt treatment and improving outcomes. Diagnosis relies on a combination of patient history, physical examination, electrocardiogram (ECG) changes, and cardiac biomarkers, such as troponins.

The hallmark of MI is acute retrosternal chest pain, typically described as a dull, squeezing pressure or tightness, lasting more than 20 minutes [3]. This pain commonly radiates to the left chest, arm, shoulder, neck, jaw, or epigastrium, and is often precipitated by exertion or emotional stress [26]. Symptom relief after nitrate administration is not a diagnostic criterion for cardiac ischemia, as it lacks specificity [27]. The peak time of MI occurrence is often in the morning, likely due to circadian increases in sympathetic tone and coagulability [28, 29]. Commonly associated symptoms encompass exertional dyspnea, pallor, nausea, vomiting, and diaphoresis, alongside anxiety that is frequently described as a sensation of impending doom. Patients may also present with dizziness, lightheadedness, or syncope [30].

Physical examination findings vary depending on MI severity and complications. Tachycardia, often due to sympathetic discharge, or arrhythmias, such as ventricular tachycardia, are common. Signs of congestive heart failure (CHF), including orthopnea, pulmonary edema (rales on auscultation), or jugular venous distension, may indicate left ventricular dysfunction. Cardiogenic shock, characterized by hypotension, tachycardia, and cold extremities, suggests extensive myocardial damage. A new heart murmur, such as an S4 gallop or mitral regurgitation murmur from papillary muscle dysfunction, may be detected on auscultation. Less commonly, a pericardial friction rub or signs of right ventricular failure (e.g., elevated jugular venous pressure) are present. [26, 31]

Atypical Presentations [38, 39]

Atypical presentations are more common in elderly patients, diabetic individuals, and women, often delaying diagnosis and treatment [40]. Up to 30% of MI cases, particularly in these groups, present with minimal or no chest pain, termed “silent MI.” Silent MI is more frequent in patients with diabetes due to polyneuropathy, which impairs pain perception. Atypical chest pain, described as stabbing or sharp, may occur instead of the classic squeezing sensation. Autonomic symptoms, such as nausea, diaphoresis, or epigastric pain, are more prevalent in inferior wall infarction. Women may present with fatigue, dyspnea, or abdominal discomfort rather than chest

Table 3: Common clinical symptoms and findings in myocardial infarction (MI).
CHF: congestive heart failure; MI: myocardial infarction.

Symptom / Finding	Description	Prevalence / Notes
Chest pain	Dull, squeezing, retrosternal pain radiating to arm, neck, or jaw	Present in ∼90% of cases [32]; less common in women, elderly [33, 34]
Dyspnea	Shortness of breath, often exertional	Common, especially in the presence of heart failure.
Nausea / Vomiting	Often accompanied by diaphoresis	More frequent in inferior MI.
Diaphoresis	Profuse sweating	Indicates autonomic activation.
Tachycardia / Arrhythmias	Increased heart rate or irregular rhythm	Observed in approximately 50% of cases [35].
CHF signs	Orthopnea, rales, jugular venous distension	Indicate left ventricular dysfunction.
Cardiogenic shock	Hypotension, cold extremities	Occurs in ∼7.5–10% of MI cases [36, 37].

pain. Elderly patients often report vague symptoms like malaise, confusion, or weakness.

Specific findings may point to distinct MI subtypes. Right ventricular infarction, often associated with inferior MI, presents with a clinical triad of hypotension, elevated jugular venous pressure, and clear lung fields. Bradycardia, due to vagal stimulation or atrioventricular block, is more common in inferior MI. In myocardial infarction with non-obstructive coronary arteries (MINOCA), symptoms may be atypical, with dyspnea or fatigue predominating over chest pain. Recognizing these variations is crucial, as atypical presentations are associated with higher mortality due to delayed intervention.

Diagnosis

The diagnosis of acute MI applies when there is acute myocardial injury accompanied by clinical evidence of acute myocardial ischemia, demonstrated by a rise and/or fall in cardiac troponin (cTn) levels with at least one measurement exceeding the 99th percentile upper reference limit, together with at least one of the following:

- Symptoms indicative of myocardial ischemia
- New ischemic changes on ECG
- Appearance of pathological Q waves
- Imaging findings showing new loss of viable myocardium or new regional wall motion abnormality consistent with ischemia
- Coronary thrombus confirmed by angiography or autopsy (applicable to types other than type 2 and type 3 MI) [41]

Furthermore, post-mortem identification of acute atherothrombosis in the artery supplying the infarcted myocardium fulfills the diagnostic criteria for type 1 MI. In contrast, evidence of a mismatch between myocardial oxygen supply and demand that is not related to acute atherothrombosis corresponds to type 2 MI. Cardiac death occurring in a patient with symptoms consistent with myocardial ischemia and presumed new ischemic ECG changes, before cardiac troponin (cTn) levels become available or abnormal, is classified as type 3 MI.

In certain patient populations—such as older adults, individuals with diabetes, and women—symptoms may present atypically, and ECG findings may be nonspecific. In such cases, when ischemic heart disease is suspected and a characteristic troponin pattern is observed, further evaluation with imaging studies may be warranted.

ECG changes

As outlined in the 2018 Universal Definition of Myocardial Infarction by the European Society of Cardiology, American College of Cardiology Foundation, American Heart Association, and World Heart Federation, the criteria below represent the standard ECG findings for the two primary categories of ECG manifestations of acute myocardial ischemia [2]:

Table 4: Electrocardiographic (ECG) diagnostic criteria for myocardial infarction (MI).
ECG: electrocardiogram; J-point: junction point between the end of the QRS complex and the start of the ST segment; MI: myocardial infarction; STEMI: ST-elevation myocardial infarction; NSTEMI: non-ST-elevation myocardial infarction; UA: unstable angina.

Condition	ECG Criteria
STEMI	New ST-segment elevation at the J-point in at least two contiguous leads. Cut-off values: ≥1 mm in all leads except V2–V3. In V2–V3: ≥2 mm in men ≥40 years, ≥2.5 mm in men <40 years, and ≥1.5 mm in women of any age. Measurements based on standard calibration (1 mV = 10 mm) (see Figure 1).
NSTEMI or UA	New horizontal or downsloping ST-segment depression of ≥0.5 mm in at least two contiguous leads and/or T-wave inversion >1 mm in two contiguous leads with a prominent R wave or R/S ratio >1.

The J point

The J point on an ECG refers to the junction where the QRS complex transitions into the ST segment, signifying the approximate end of ventricular depolarization and the onset of repolarization as recorded on the surface ECG. This transition typically occurs over an interval of about 10 milliseconds [42]

In many healthy young men, the J point lies slightly above the baseline. Its position can shift in conditions such as early repolarization, epicardial or endocardial ischemia or injury, pericarditis, right or left bundle branch block (RBBB/LBBB), right or left ventricular hypertrophy (RVH/LVH), or as an effect of digitalis [42]

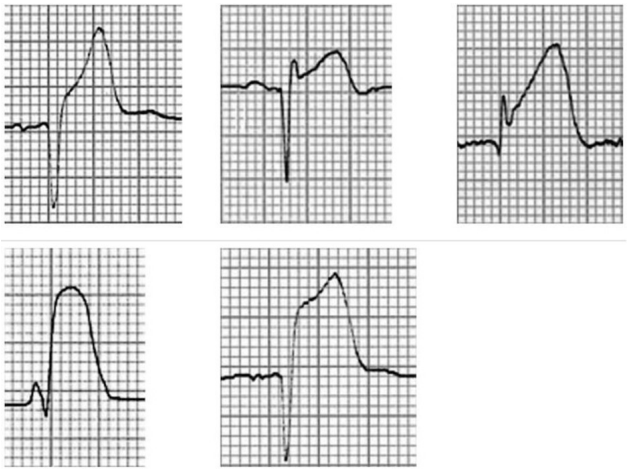

Figure 1: Differentiate between different ST segment changes morphology of acute MI [43]

In most ECGs, the J point can be readily identified as the boundary between the QRS complex and the beginning of the ST segment. However, with more advanced electrophysiological investigations and detailed analysis of the cellular and ionic events corresponding to each ECG phase, the distinction between these components can appear less well-defined (Figure 2) [42]

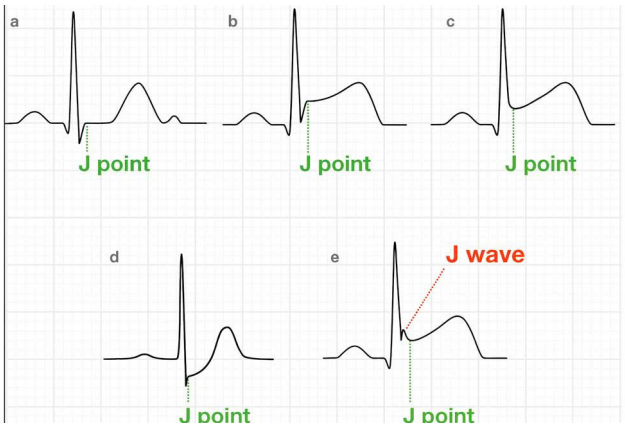

Figure 2: Distinct J point in a) normal; b) c) J point elevation; d) J point depression; e) with J wave (Osborn wave).

Note: On an ECG, the letter J is used to describe two distinct and unrelated phenomena. The J point refers to the moment marking the transition from the QRS complex to the ST segment, present on all ECGs. In contrast, the J wave is a far less common, prolonged, slow deflection of uncertain origin, originally described in association with hypothermia [42]

Pathological Q wave

A Q wave is defined as any negative deflection that appears before an R wave on the ECG. It reflects the normal left-to-right depolarization of the interventricular septum [44]. Pathological Q waves are typically defined by the following characteristics [44]:

- Width greater than 40 milliseconds (1 mm on the ECG)
- Depth exceeding 2 mm
- Depth measuring at least 25% of the amplitude of the corresponding QRS complex
- Present in leads V1 to V3

Differential Diagnosis

Accurate diagnosis of myocardial infarction (MI) necessitates a vigilant approach to identify not only ischemic heart disease but also other potentially life-threatening conditions that may present with similar clinical features. The differential diagnosis can be categorized by presenting features such as chest pain, elevated troponin, and ST-segment elevation on the electrocardiogram (ECG).

Potentially fatal diagnoses such as aortic dissection, aortic aneurysm, pulmonary embolism, tension pneumothorax, myocarditis, and esophageal ruptures or perforated ulcers can present with symptoms and signs overlapping with MI. Less critical but frequent causes include musculoskeletal pain, gastroesophageal reflux disease, esophageal spasm, pleurisy, pneumonia, and anxiety, which can closely mimic MI symptoms [45]. Key clinical distinctions lie in the character, location, and triggers of pain, associated symptoms such as dyspnea or diaphoresis, and reproducibility on palpation or positional changes. Integration of risk factors and clinical context enhances diagnostic accuracy.

Elevated troponin levels, while sensitive indicators of myocardial injury, are not specific to MI and may also be observed in myocarditis, pulmonary embolism, heart failure, severe systemic illness, and coronary involvement by aortic dissection. Similarly, ST-segment elevations on ECG, though hallmark signs of ST-elevation myocardial infarction (STEMI), can occur in pericarditis, myocarditis, aortic dissection affecting coronary arteries, pulmonary embolism, and other non-ischemic cardiac conditions [46].

Distinguishing these entities requires an integrated approach including detailed clinical evaluation, serial ECG analysis, biomarker trends, and appropriate imaging studies. Recognizing and promptly diagnosing these life-threatening mimics of MI is vital, as they necessitate specific therapeutic strategies that differ fundamentally from the treatment of primary coronary artery occlusion, underscoring the importance of a comprehensive and nuanced diagnostic strategy in patients with acute chest pain syndromes [47].

Management

Acute Management

Current expert consensus supports administering antiplatelet therapy as early as possible, ideally at the first point of medical contact, to improve survival outcomes in acute coronary syndromes [3]. Aspirin should be given as a chewable loading dose of 162–325 mg followed by 81 mg daily [3, 48]. A P2Y12 inhibitor should be loaded before PCI in eligible patients; ticagrelor and prasugrel are preferred over clopidogrel due to greater reduction in ischemic events [3, 49, 50].

Supplemental oxygen is indicated only when arterial saturation is < 90%, as showed no benefit in normoxemic patients [3, 51, 52]. Nitrates may be used sublingually or intravenously for ongoing ischemic pain if hypotension and right ventricular infarction are excluded [3, 53]. Morphine should be reserved for refractory symptoms because of observational evidence suggesting delayed P2Y12 absorption [3, 54].

In STEMI, reperfusion therapy remains the primary intervention. PCI is preferred when first medical contact–to-device time is $\leq$ 90 minutes for PCI-capable centers or $\leq$ 120 minutes when transfer is required [3, 55]. If PCI cannot be performed within these limits, fibrinolysis should be initiated within 30 minutes of hospital arrival [3, 56, 57].

For NSTE-ACS, early invasive management within 24 hours is advised for high-risk patients (elevated troponin, dynamic ECG changes, GRACE score > 140) because it reduces recurrent ischemia and adverse events [3, 58, 59].

Revascularization

Angiographic findings should guide the revascularization approach. In STEMI, complete revascularization of non-culprit lesions before discharge is associated with fewer major cardiovascular events [60]. In patients with complex multivessel disease, left main involvement, or anatomy unsuitable for PCI, CABG is appropriate after multidisciplinary review [3, 61]. For NSTE-ACS, PCI timing should be risk-based, with immediate intervention (< 2 hours) reserved for those with hemodynamic instability, refractory angina, or malignant arrhythmias [3, 59, 62].

Post-MI and Secondary Prevention

After discharge, therapy should target recurrence prevention and functional recovery. All patients should receive high-intensity statins, with addition of ezetimibe or PCSK9 inhibitors when LDL-C remains $\geq$ 70 mg/dL despite maximal therapy [63, 64]. Beta-blockers are indicated for LVEF $\leq$ 40% or arrhythmias, but long-term use in lower-risk patients is no longer routinely recommended [3]. ACE inhibitors or ARBs are appropriate for those with LVEF $\leq$ 40%, diabetes, hypertension, or CKD; ARNI may be considered selectively [3, 65, 66]. Structured cardiac rehabilitation, smoking cessation, dietary optimization, and regular exercise remain key components of care, with program participation linked to a 26–47% mortality reduction [3, 67].

Table 5: Key Acute MI Interventions[3] (2025 ACC/ AHA/ ACEP/ NAEMSP/ SCAI Guideline).
ACS: acute coronary syndrome; FMC: first medical contact; GRACE: Global Registry of Acute Coronary Events; IV: intravenous; NSTE-ACS: non-ST-elevation acute coronary syndrome; PCI: percutaneous coronary intervention; RV: right ventricle; SpO_2: peripheral oxygen saturation.

Timepoint / Condition	Intervention	Class / Level of Evidence
First medical contact	Chewable aspirin 162–325 mg loading, then 81 mg daily	I / A
First medical contact	Oral $P2Y_{12}$ inhibitor loading (ticagrelor or prasugrel preferred over clopidogrel)	I / A
SpO_2 < 90%	Supplemental oxygen	I / B-R
Persistent ischemic pain without hypotension or RV infarct	Sublingual or IV nitrates	I / B-NR
STEMI, PCI-capable, FMC-to-device $\leq$ 90 min ($\leq$ 120 min if transfer)	Primary PCI	I / A
STEMI with anticipated PCI delay > 120 min	Fibrinolysis within 30 min of hospital arrival	I / A
High-risk NSTE-ACS (elevated troponin, dynamic ECG changes, GRACE > 140)	Early invasive strategy within 24 h	I / A
Hemodynamic instability, refractory angina, or malignant arrhythmias (NSTE-ACS)	Immediate invasive strategy (< 2 h)	I / C-LD

Complication

Early complications after myocardial infarction are key determinants of in-hospital survival. Ventricular arrhythmias are the leading cause of sudden cardiac death within the first 48 hours, but routine prophylactic antiarrhythmic therapy is not advised due to evidence of increased mortality [3, 68]. Bradyarrhythmias and high-grade atrioventricular block occur more frequently in inferior infarctions and may require temporary pacing until conduction recovers [3]. Cardiogenic shock develops in roughly 5–10% of patients and is associated with mortality rates exceeding 40%; in this setting, limiting PCI to the culprit lesion has been shown to improve short-term outcomes compared with immediate multivessel intervention [69]. Mechanical complications (papillary muscle rupture, ventricular septal defect, free wall rupture) are most common 3 to 7 days after infarction, coinciding with peak inflammatory infiltration and myocardial weakening [3, 70].

Late complications reflect the longer-term structural and functional consequences of infarction. Heart failure frequently arises from adverse ventricular remodeling but can be mitigated by timely initiation of ACE inhibitors and beta-blockers [3, 65, 71]. Left ventricular aneurysms may form and predispose to mural thrombus and systemic embolization, for which anticoagulation may be indicated in selected cases [3]. Pericarditis occurring weeks after infarction, often referred to as Dressler's syndrome, is thought to

represent an autoimmune inflammatory process and is treated with NSAIDs or colchicine [3, 72].

Prognosis

Patient outcomes after myocardial infarction depend on the timeliness of reperfusion, the completeness of revascularization, and adherence to proven secondary prevention measures [3, 73]. Among available prognostic variables, left ventricular ejection fraction and Killip class remain the important predictors of both short- and long-term survival [3]. Reduced LV ejection fraction below 40% is associated with markedly higher one-year post-discharge mortality [3, 74]. Higher Killip class at presentation correlates with steep rises in in-hospital mortality [3].

Long-term prognosis is also improved by participation in structured cardiac rehabilitation programs, which provide supervised exercise, patient education, and comprehensive risk factor management. Engagement in such programs is linked to substantial reductions in all-cause mortality and recurrent cardiovascular events [67, 75]. These benefits reinforce the need to integrate lifestyle modification and rehabilitation into post-MI care alongside optimal medical therapy and complete revascularization.

Conclusion

Acute coronary syndrome remains a leading cause of morbidity and mortality worldwide, with a complex interplay of traditional, non-modifiable, and emerging risk factors driving its burden. Advances in our understanding of its pathophysiology, particularly the role of atherosclerotic plaque instability, inflammation, and oxygen supply demand mismatch, have refined both diagnostic and therapeutic strategies. Contemporary definitions and classifications now allow clinicians to more accurately identify subtypes of myocardial infarction, including type 2 MI and MINOCA, which require tailored management approaches.

Early recognition remains critical, as atypical presentations, particularly in elderly patients, women, and those with diabetes, contribute to delays in diagnosis and lower the quality of outcomes. Modern diagnostic algorithms, integrating high-sensitivity troponins, ECG interpretation, and advanced imaging, have improved accuracy but demand careful differentiation from non-ischemic causes of myocardial injury.

Management of ACS continues to emphasize timely reperfusion in STEMI, early invasive strategies in high-risk NSTE-ACS, and evidence-based pharmacotherapy, including dual antiplatelet therapy, high-intensity statins, and renin–angiotensin system inhibition. Structured cardiac rehabilitation and aggressive modification of lifestyle and risk factors remain essential pillars of long-term care.

Despite significant progress, challenges persist, including high residual risk, complications such as heart failure and arrhythmias, and disparities in access to reperfusion and secondary prevention programs. Future directions lie in precision medicine, novel biomarkers, anti-inflammatory therapies, and broader implementation of digital health technologies to enhance prevention, adherence, and early detection.

Ultimately, optimizing ACS outcomes requires an integrated, multidisciplinary approach that combines rapid acute care with comprehensive long-term strategies—shifting focus from not only saving lives in the acCitationute setting but also improving quality of life and reducing recurrence in the years that follow.

List of Abbreviations

CABG: coronary artery bypass graft;
CAD: coronary artery disease;
cTn: cardiac troponin;
PCI: percutaneous coronary intervention;
URL: upper reference limit;
ASCVD: atherosclerotic cardiovascular disease;
BMI: body mass index;
FH: familial hypercholesterolemia;
LDL: low-density lipoprotein;
CHF: congestive heart failure;
MI: myocardial infarction;
ECG: electrocardiogram;
J-point: junction point between the end of the QRS complex and the start of the ST segment;
MI: myocardial infarction;
STEMI: ST-elevation myocardial infarction;
NSTEMI: non-ST-elevation myocardial infarction;
UA: unstable angina;
ACS: acute coronary syndrome;
FMC: first medical contact;
GRACE: Global Registry of Acute Coronary Events;
IV: intravenous;
NSTE-ACS: non-ST-elevation acute coronary syndrome;
PCI: percutaneous coronary intervention;
RV: right ventricle;
SpO_2: peripheral oxygen saturation.

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Competing interests

None of the authors have conflicts of interest to declare.

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