MYOCARDIAL INFARCTION AND EARLY PREVENTION METHODS

Qovulova Nurizar Mo'min qizi
4-th grade student of Medical Faculty of Jizzakh State of Pedagogical University

ABSTRACT

Myocardial infarction (MI), or heart attack, remains one of the leading causes of death globally. It is primarily caused by a sudden reduction in coronary blood flow due to atherosclerotic plaque rupture and subsequent thrombosis. The condition leads to irreversible myocardial necrosis if not managed promptly. Major risk factors include hypertension, dyslipidemia, diabetes mellitus, smoking, obesity, sedentary lifestyle, and a family history of cardiovascular disease. Advances in preventive cardiology have highlighted the importance of early identification of at-risk individuals through regular screening, biomarker assessment, and non-invasive imaging techniques. Primary prevention focuses on lifestyle modifications, pharmacological management of risk factors, and public education, while secondary prevention targets individuals with established cardiovascular disease through strict control of contributing conditions and use of antiplatelet, statin, and antihypertensive therapies. This article aims to provide a comprehensive overview of myocardial infarction and the evidence-based strategies used for its early prevention, emphasizing the importance of a multidisciplinary and proactive approach in reducing incidence and improving outcomes.

Keywords: Myocardial infarction, ischemic heart disease, prevention, atherosclerosis, risk factors, early detection, cardiovascular health.

Introduction

Myocardial infarction (MI), commonly referred to as a heart attack, is a critical manifestation of ischemic heart disease and remains one of the leading causes of death and disability worldwide. According to the World Health Organization (WHO), cardiovascular diseases are responsible for approximately 17.9 million deaths annually, with myocardial infarction accounting for a significant proportion of these cases. The condition is characterized by the interruption of blood supply to a portion of the heart muscle, most commonly due to the rupture of an atherosclerotic plaque in the coronary arteries, followed by thrombus formation and coronary artery occlusion. The clinical consequences of MI vary from mild chest discomfort to sudden cardiac death, depending on the extent and location of myocardial injury. Early diagnosis and intervention are critical to limit infarct size and improve patient outcomes. Despite advancements in acute management strategies such as thrombolytic therapy,

percutaneous coronary intervention (PCI), and cardiac rehabilitation, primary and secondary prevention remain the most effective long-term strategies for reducing the burden of MI. Prevention involves identifying and modifying risk factors such as hypertension, hyperlipidemia, diabetes mellitus, smoking, sedentary lifestyle, and poor dietary habits. Modern preventive cardiology integrates lifestyle changes, pharmacological interventions, and public health initiatives aimed at reducing the incidence of first and recurrent myocardial infarctions. This article aims to review the pathophysiology of myocardial infarction and highlight evidence-based early prevention strategies, including risk assessment, lifestyle modification, and medical therapies, to reduce morbidity and mortality associated with this condition.

Myocardial infarction (MI), colloquially known as "heart attack," is caused by decreased or complete cessation of blood flow to a portion of the myocardium. Myocardial infarction may be"silent," and go undetected, or it could be a catastrophic event leading to hemodynamic deterioration and sudden death. Most myocardial infarctions are due to underlying coronary artery disease, the leading cause of death in the United States. With coronary artery occlusion, the myocardium is deprived of oxygen. Prolonged deprivation of oxygen supply to the myocardium can lead to myocardial cell death and necrosis. Patients can present with chest discomfort or pressure that can radiate to the neck, jaw, shoulder, or arm. In addition to the history and physical exam, myocardial ischemia may be associated with ECG changes and elevated biochemical markers such as cardiac troponins. This activity describes the pathophysiology, evaluation, and management of myocardial infarction and highlights the role of the interprofessional team in improving care for affected patients.

Classification of Myocardial Infarction

According to the Fourth Universal Definition of Myocardial Infarction (2018) and confirmed by key cardiology books, myocardial infarction is classified into five major types, each with specific pathophysiological features and clinical relevance.

- Type 1 MI – Spontaneous Myocardial Infarction

Definition: MI related to ischemia due to a primary coronary event such as plaque rupture, ulceration, fissuring, or dissection leading to intraluminal thrombus formation.
Cause: Atherosclerotic plaque rupture with thrombosis.
Clinical scenario: Acute coronary syndrome (ACS).
Troponin: Significantly elevated with rising/falling pattern.

- Type 2 MI – Secondary to Ischemia

Definition: MI secondary to ischemia from increased oxygen demand or decreased supply, without acute atherothrombosis.
Examples: Coronary spasm, anemia, hypotension, arrhythmias.

Mechanism: Imbalance between myocardial oxygen supply and demand.
Troponin: Elevated but usually without dynamic ECG changes.

Type 3 MI – Sudden Cardiac Death

Definition: Cardiac death with symptoms suggestive of myocardial ischemia, accompanied by presumed new ECG changes or ventricular fibrillation before blood samples can be obtained or before cardiac biomarkers appear.
Diagnosis: Post-mortem or clinical suspicion.
ECG: Often shows ventricular arrhythmias or ST elevation before death.

Type 4 MI – Related to Percutaneous Coronary Intervention (PCI

Type 4a: MI associated with PCI; defined by troponin elevation >5× upper reference limit (URL) plus evidence of ischemia (e.g., chest pain, ECG changes).
Type 4b: MI due to stent thrombosis.
Type 4c: MI due to in-stent restenosis without thrombosis.

Type 5 MI – Related to Coronary Artery Bypass Grafting (CABG)

Definition: MI occurring during or after CABG surgery, with:
Troponin elevation >10× URL
New Q waves, angiographic evidence, or imaging confirmation of infarction.

Additional Subclassifications:

ST-Elevation Myocardial Infarction (STEMI)
Transmural MI with persistent ST elevation.
Urgent reperfusion is required.
Non-ST-Elevation MI (NSTEMI)
Subendocardial MI without persistent ST elevation.
Summary Table:
Type Name Key Feature Common Cause
1 Spontaneous MI Atherothrombosis Plaque rupture
2 Supply/Demand MI Oxygen imbalance Anemia, tachycardia
3 Sudden death MI Cardiac death < biomarkers Prehospital fatal events
4a PCI-related MI Post-PCI elevation Iatrogenic
4b Stent thrombosis Thrombotic occlusion In-stent clot
4c In-stent restenosis Restenosis > thrombosis Neointimal hyperplasia
5 CABG-related MI Post-op infarct Surgical complications

Global Burden

MI remains a leading cause of death worldwide, particularly in low- and middle-income countries.
According to the World Health Organization (2023):

Cardiovascular diseases (CVDs) account for ~17.9 million deaths per year, of which 85% are due to MI and stroke.

MI incidence is higher in industrialized regions but increasing rapidly in developing countries due to urbanization, sedentary lifestyle, and unhealthy diet.

Incidence & Prevalence

United States (CDC, 2023):

Approx. 805,000 MIs/year.

STEMI: ~25-30%

NSTEMI: ~70-75%

About 1 in 5 MIs are silent (unrecognized).

Europe (ESC Data, 2022):

Annual incidence: ~200–300 per 100,000 population.

Decreasing mortality due to improved treatments, though aging populations are at higher risk.

Asia:

Rising trend in MI, especially among younger individuals in India, China, and Central Asia.

South Asians are at higher risk due to genetic predisposition and early metabolic syndrome.

Age & Gender Distribution

Age: MI incidence increases with age, typically affecting:

Men ≥ 45 years

Women ≥ 55 years or postmenopausal

Gender:

Men have higher incidence at a younger age.

Women have worse outcomes post-MI due to late presentation and atypical symptoms.

Risk Factors (Updated)

Traditional Risk Factors	Emerging Risk Factors
Smoking	Chronic inflammation
Hypertension	Lipoprotein(a)
Hyperlipidemia	Air pollution exposure
Diabetes mellitus	Psychosocial stress
Obesity	Gut microbiota changes
Sedentary lifestyle	Sleep disorders

Etiology of Myocardial Infarction (MI)

Myocardial infarction occurs due to interruption of coronary blood flow, leading to ischemia and necrosis of the heart muscle. The etiology of MI can be classified into atherothrombotic (primary) and non-atherothrombotic (secondary) causes.

1. Atherothrombotic Causes (Most Common)

A. Atherosclerotic Plaque Rupture or Erosion

Pathogenesis:

Chronic atherosclerosis → plaque formation.

Plaque rupture or erosion → platelet aggregation and thrombus → coronary occlusion.

Main cause of Type 1 MI.

B. Plaque Hemorrhage

Intraplaque hemorrhage increases plaque volume → luminal narrowing → rupture.

2. Coronary Artery Vasospasm

Transient or sustained spasm causes ischemia even without significant atherosclerosis.

Associated with Prinzmetal's (variant) angina, drug use (e.g., cocaine, amphetamines), or endothelial dysfunction.

3. Coronary Embolism

Sources:

Left atrial thrombus (e.g., atrial fibrillation).

Vegetations (infective endocarditis).

Prosthetic valves or paradoxical emboli.

Leads to acute coronary occlusion.

4. Coronary Artery Dissection

Spontaneous Coronary Artery Dissection (SCAD):

Seen in young women, often postpartum.

Associated with connective tissue disorders.

Causes intramural hematoma and vessel obstruction.

5. Supply-Demand Mismatch (Type 2 MI)

No acute plaque rupture; due to imbalance between oxygen supply and demand.

Causes:

Severe anemia

Hypotension/shock

Tachyarrhythmia or bradyarrhythmia

Hypoxemia (e.g., pulmonary embolism, COPD)

Severe hypertension (↑ afterload)

6. Other Rare Causes

A. Myocardial Bridging

Intramyocardial course of a coronary artery segment → dynamic compression during systole.

B. Vasculitis

Diseases like Kawasaki disease, Takayasu arteritis, or polyarteritis nodosa can affect coronary arteries.

C. Radiation-Induced Coronary Disease

Seen in patients with prior chest radiation (e.g., Hodgkin's lymphoma survivors).

D. Infiltrative Diseases

Amyloidosis or sarcoidosis causing microvascular dysfunction.

Summary Table: Etiological Categories

Category	Specific Cause	Common Type
Atherosclerotic	Plaque rupture, erosion, hemorrhage	Type 1 MI
Vasospastic	Cocaine, Prinzmetal angina	Type 1/2 MI
Embolic	Atrial fibrillation, endocarditis	Type 1 MI
Mechanical/Dissection	SCAD, trauma, catheter-induced	Type 1 MI
Supply-demand mismatch	Anemia, hypoxia, hypotension, tachycardia	Type 2 MI
Inflammatory/Autoimmune	Vasculitis, lupus, Kawasaki disease	Type 1/2 MI
Structural/Anatomic	Myocardial bridging, congenital anomalies	Type 2 MI

Myocardial Pathophysiology of Myocardial Infarction

infarction (MI) is the necrosis of myocardial tissue due to prolonged ischemia, most commonly caused by acute thrombotic occlusion of a coronary artery following plaque rupture or erosion. The pathophysiological process unfolds in several well-defined stages and involves vascular, cellular, and molecular mechanisms.

1. Atherosclerosis and Plaque Development

The initial stage involves the development of atherosclerotic plaques within the coronary arteries, beginning with endothelial dysfunction triggered by risk factors such as hypertension, hyperlipidemia, smoking, and diabetes mellitus. Low-density lipoprotein (LDL) cholesterol penetrates the endothelial lining and becomes oxidized, initiating an inflammatory response. Macrophages engulf oxidized LDL to become foam cells, forming the fatty streak, which evolves into a fibrous plaque. Over time, the plaque enlarges and becomes vulnerable due to a lipid-rich core and a thin fibrous cap, prone to rupture.

2. Plaque Rupture and Thrombus Formation

In most cases of acute MI, the plaque rupture exposes thrombogenic material, such as collagen and tissue factor, to the circulating blood. This triggers:

Platelet adhesion and aggregation

Activation of the coagulation cascade

Formation of a fibrin-rich thrombus

This thrombus can partially or completely occlude the coronary artery. In ST-segment elevation myocardial infarction (STEMI), the thrombus usually causes a complete occlusion, while NSTEMI results from a subtotal obstruction.

3. Myocardial Ischemia and Necrosis

Once the coronary blood flow drops below the level required for myocardial oxygen demand:

Ischemia occurs within seconds
ATP depletion begins within 1–2 minutes
Irreversible cell injury begins within 20–30 minutes
Transmural necrosis can develop if blood flow is not restored within 6 hours
Necrosis typically starts in the subendocardium, the innermost and most vulnerable layer, and progresses outward to the epicardium. The infarcted area is gradually replaced by granulation tissue and later by fibrous scar tissue.
4. Inflammatory and Repair Response
Post-infarction, necrotic myocardium triggers a sterile inflammatory response:
Neutrophils infiltrate within the first 24 hours
Macrophages remove dead cells over days
Fibroblasts and endothelial cells proliferate and deposit collagen
A fibrous scar replaces lost myocardium within 6–8 weeks
However, the fibrous scar is non-contractile and may contribute to ventricular remodeling, dilation, and eventual heart failure if the infarct is extensive.
5. Electrical and Mechanical Complications
Myocardial infarction can lead to a range of complications:
Arrhythmias: due to disruption of conduction pathways
Heart failure: due to impaired systolic function
Papillary muscle rupture, ventricular septal defect, or free wall rupture
Pericarditis or Dressler's syndrome
Left ventricular aneurysm
These complications are directly related to the size and location of the infarct and the timeliness of reperfusion therapy.
Summary of Pathophysiological Events
Stage Key Features
Endothelial dysfunction LDL infiltration, oxidative stress
Plaque formation Foam cell accumulation, fibrous cap formation
Plaque rupture Exposure of thrombogenic core, thrombus formation
Myocardial ischemia Oxygen imbalance, ATP depletion
Cell death and necrosis Subendocardial to transmural progression
Inflammation and repair Leukocyte infiltration, scar formation
Functional and structural impact Loss of contractility, arrhythmias, heart failure

Clinical Manifestations of Myocardial Infarction

Myocardial infarction (MI) presents with a wide spectrum of clinical features. Classic symptoms are well recognized, but atypical or silent presentations are also important, particularly in certain populations like the elderly, diabetics, and women.

1. Chest Pain (Angina Pectoris)

▪ Characteristics:

Intense, crushing, or squeezing pain (retrosternal)

Described as "pressure," "tightness," or "heaviness"

May radiate to:

Left arm

Neck

Jaw

Back

Lasts >20 minutes and is not relieved by rest or nitroglycerin

▪ Associated Features:

Diaphoresis (sweating)

Sense of impending doom

Often occurs at rest or early in the morning due to circadian increases in sympathetic activity

2. Dyspnea (Shortness of Breath)

Reflects left ventricular dysfunction and pulmonary congestion

May be the only symptom in the elderly or diabetics

In posterior infarctions, may occur without chest pain

3. Autonomic Symptoms

Nausea and vomiting – especially in inferior wall MI

Diaphoresis

Cold, clammy skin due to sympathetic overactivity

Hypotension or syncope – due to reduced cardiac output or arrhythmia

4. Palpitations and Arrhythmias

Patients may feel irregular or fast heartbeat

Underlying cause: ischemia-induced arrhythmias such as:

Ventricular tachycardia (VT)

Ventricular fibrillation (VF)

Atrial fibrillation (AF)

Heart blocks

5. Fatigue and Weakness

Frequently reported in older adults and women

Reflects decreased cardiac output and systemic hypoperfusion

6. Silent Myocardial Infarction
Common in:
Elderly
Diabetics (due to autonomic neuropathy)
Chronic kidney disease patients
Detected only via ECG changes or cardiac biomarkers
7. Physical Examination Findings
Sign Possible Cause
Tachycardia Pain, anxiety, hypoperfusion
Hypotension LV failure, RV infarct
S4 gallop Decreased LV compliance
Rales or crackles Pulmonary edema
Jugular venous distension RV infarction or heart failure
New systolic murmur Papillary muscle rupture or VSD
Cyanosis or pallor Peripheral vasoconstriction, low output
8. ECG Changes and Laboratory Markers (Support Diagnosis)
Though not symptoms, ECG and biomarkers are key in confirming clinical suspicions:
ST-segment elevation or depression
T-wave inversion
Pathologic Q waves
Elevated troponin I or T
Summary Table of Common and Atypical Presentations
Clinical Feature Frequency / Notes
Chest pain Most common (85–90% of STEMI
Dyspnea Common in elderly or LV failure
Diaphoresis Often accompanies pain
Nausea/vomiting More common in inferior MI
Syncope or presyncope May indicate arrhythmia or low perfusion
Silent infarction Common in diabetics, elderly
Confusion Seen in elderly; may be sole manifestation

Diagnosis of Myocardial Infarction (MI)

The diagnosis of MI relies on clinical evaluation, electrocardiography (ECG), cardiac biomarkers, and imaging studies. According to the Fourth Universal Definition of MI, at least one cardiac biomarker (preferably troponin) must be elevated with evidence of myocardial ischemia.

1. Clinical History and Physical Examination
Classic symptoms: Chest pain > 20 minutes, pressure-like, radiating

Associated signs: Diaphoresis, dyspnea, nausea, palpitations
Risk factor assessment: HTN, diabetes, smoking, family history
2. Electrocardiography (ECG)
ECG is essential and should be done within 10 minutes of arrival.
Typical findings:

Type of MI	ECG Findings
STEMI	ST-segment elevation ≥1 mm in ≥2 leads
NSTEMI	ST depression, T-wave inversion
Posterior MI	ST depression in V1–V3, tall R waves
Q-wave MI	Pathologic Q waves (late finding)

Reference:
Marriott's Practical Electrocardiography, 13th Ed. ISBN: 9781496397454
3. Cardiac Biomarkers
Troponins I and T:
Most sensitive and specific
Rise within 3–6 hours, peak at 12–24 hours, remain elevated up to 10–14 days
CK-MB:
Less specific than troponin
Peaks earlier; useful for detecting reinfarction

Marker	Rise (hrs)	Peak (hrs)	Return to Normal
Troponin	3–6	12–24	7–14 days
CK-MB	4–6	24	48–72 hrs

4. Imaging Studies
A. Echocardiography
Assesses regional wall motion abnormalities
Identifies mechanical complications (e.g., papillary muscle rupture)
B. Cardiac MRI
Gold standard for assessing myocardial viability, infarct size
Detects late gadolinium enhancement (LGE)
C. Coronary Angiography
Performed in all STEMI and high-risk NSTEMI
Identifies culprit lesion, guides PCI (percutaneous coronary intervention)
5. Additional Laboratory Tests
CBC: Leukocytosis may be present
Lipid panel: Ideally within 24 hours
Serum electrolytes: Potassium, magnesium (important for arrhythmia risk)
Renal function tests: For medication safety

BNP/NT-proBNP: If heart failure suspected

Diagnostic Criteria (Universal Definition)

MI is diagnosed when troponin is elevated plus one of the following:

1. Symptoms of ischemia
2. New ischemic ECG changes
3. Development of pathologic Q waves
4. Imaging evidence of new loss of myocardium or wall motion abnormality
5. Identification of thrombus by angiography or autopsy

Treatment of Myocardial Infarction (MI)

Treatment depends on the type of MI (STEMI vs. NSTEMI), time since symptom onset, hemodynamic stability, and comorbid conditions. The goals are to restore perfusion, reduce myocardial damage, and prevent complications.

1. Immediate Emergency Management (First 10 Minut)

Drug Purpose

Morphine For chest pain unrelieved by nitrates

Oxygen If $SpO_2 < 90\%$

Nitrates Sublingual nitroglycerin (vasodilation, pain relief)

Aspirin Chewed, 160–325 mg (antiplatelet effect)

2. Reperfusion Therapy

A. STEMI: Primary Percutaneous Coronary Intervention (PCI)

Preferred within 90–120 minutes of first medical contact

Involves angioplasty and stent placement

Superior to thrombolytics in reducing mortality

B. Thrombolytic Therapy (If PCI not available within 120 minutes)

Administer within 12 hours of symptom onset

Agents:

Tenecteplase (TNK-tPA)

Alteplase

Streptokinase (less preferred)

Contraindications: bleeding, stroke history, trauma, hypertension

3. Antithrombotic Therapy

Drug Class Examples Notes

Antiplatelets Aspirin + P2Y12 inhibitor (clopidogrel, ticagrelor) Dual antiplatelet therapy (DAPT)

Anticoagulants Enoxaparin, unfractionated heparin Given during acute phase, especially before PCI

4. Beta-Blockers
Reduce myocardial oxygen demand and mortality
IV or oral (e.g., metoprolol)
Avoid in acute heart failure, bradycardia, or hypotension
5. ACE Inhibitors / ARBs
Start within 24 hours
Prevent adverse remodeling and reduce mortality
Examples: Ramipril, Lisinopril, Losartan
6. Statins
High-intensity statin (e.g., atorvastatin 80 mg/day)
Initiated early to stabilize plaques and reduce inflammation
7. Aldosterone Antagonists
In patients with heart failure or diabetes with LVEF < 40%
Example: Eplerenone, Spironolactone
8. Pain Management
Morphine sulfate IV if pain persists after nitrates
Monitor for hypotension and bradycardia
9. Ongoing Monitoring & Support
Telemetry monitoring for arrhythmias
Serial ECGs and cardiac enzymes
Echocardiography to assess function and complications
Lifestyle Modifications and Secondary Prevention

Intervention Details
Smoking cessation Counseling, pharmacologic aids
Diet Low saturated fat, high-fiber, Mediterranean diet
Physical activity Cardiac rehabilitation, supervised exercise
Weight control Target BMI <25
Glycemic control HbA1c <7% for diabetics
BP control Target <130/80 mmHg
Long-Term Pharmacotherapy (Post-MI)
Aspirin indefinitely
P2Y12 inhibitor for 12 months
Statin long-term
Beta-blocker (especially if reduced EF)
ACEi/ARB and aldosterone antagonist if indicated

Advanced Prevention Methods of Myocardial Infarction

Prevention of myocardial infarction is categorized into:

Primary prevention – in patients with risk factors but no prior MI.

Secondary prevention – in patients who already had MI or coronary artery disease (CAD).

1. Risk Stratification & Personalized Screening

A. Coronary Artery Calcium (CAC) Scoring

Non-invasive CT scan to detect subclinical atherosclerosis

Improves risk prediction in intermediate-risk patients

B. Genetic Risk Profiling

Polygenic risk scores (PRS) to assess inherited predisposition

2. Pharmacologic Primary Prevention in High-Risk Individuals

A. PCSK9 Inhibitors

For high LDL-C not controlled by statins

Examples: Alirocumab, Evolocumab

B. Aspirin (Selective Use)

In high cardiovascular risk + low bleeding risk only

Avoid routine use in low-risk individuals

C. Anti-inflammatory Therapies

Canakinumab (IL-1β inhibitor) – shown to reduce recurrent MI in CANTOS trial

Colchicine – emerging anti-inflammatory in secondary prevention

3. Advanced Lifestyle Intervention Models

A. Multidisciplinary Cardiac Rehabilitation

Supervised exercise, diet education, psychosocial therapy

Reduces mortality and recurrence

Personalized and technology-aided versions exist (e.g., apps, wearable ECG monitors)

B. Precision Nutrition Programs

Based on genetic tests, gut microbiota profiling, and metabolic markers

4. Implantable and Wearable Monitoring Devices

Implantable loop recorders for arrhythmia detection

Smartwatches and patches for continuous ECG, BP, and heart rate monitoring

AI-enhanced ECG interpretation for early ischemia detection

5. Comorbidity Management

MI prevention is multidisciplinary and involves tight control of systemic diseases:

Condition Strategy

Hypertension Intensive BP control (<130/80 mmHg), ambulatory monitoring

Diabetes Use of SGLT2 inhibitors and GLP-1 receptor agonists (shown to reduce MI)

CKD Limit nephrotoxic drugs, manage electrolytes

Obstructive sleep apnea CPAP improves cardiovascular outcomes

6. Vaccination for MI Prevention

Influenza and COVID-19 Vaccination

Prevent viral-induced plaque rupture and inflammation

Recommended annually for CAD patients

7. System-Based and Population-Level Prevention

National screening programs for lipids, BP, glucose

Taxation and regulation of tobacco, trans fats

Digital health platforms for mass education and behavior change

8. Psychosocial and Behavioral Risk Modification

Chronic stress, depression, and social isolation increase MI risk

Interventions:

Cognitive Behavioral Therapy (CBT)

Mindfulness-based stress reduction

Group support programs

Conclusion

Myocardial infarction remains one of the most critical and life-threatening manifestations of cardiovascular disease globally. Despite remarkable progress in our understanding of its pathophysiology, clinical features, and risk stratification, MI continues to be a leading cause of morbidity and mortality. Early diagnosis—through clinical assessment, ECG, cardiac biomarkers, and imaging—is essential for timely intervention and improved outcomes.

Therapeutic approaches have evolved to include not only immediate reperfusion strategies, such as percutaneous coronary intervention (PCI) and thrombolysis, but also comprehensive pharmacological management and long-term secondary prevention. Medications such as dual antiplatelet therapy, beta-blockers, statins, ACE inhibitors, and newer agents like PCSK9 inhibitors and anti-inflammatory drugs play a pivotal role in reducing recurrent events and improving survival.

Furthermore, the concept of prevention has become increasingly personalized and multidisciplinary. Primary prevention now involves advanced risk assessment tools such as coronary calcium scoring and genetic profiling, while secondary prevention integrates cardiac rehabilitation, psychosocial support, and the use of wearable technologies for ongoing monitoring.

In conclusion, the effective management of myocardial infarction hinges on a combination of early recognition, evidence-based treatment, and sustained preventive efforts that address not only the biological but also the behavioral and societal dimensions of cardiovascular health.

Continued research and innovation, alongside public health initiatives, are essential to further reduce the global burden of this disease.

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