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Myocardial Infarction: Causes, Symptoms, and Emergency Response

Shazain Khan [a], Raheel Chaudhry [b], Arhum Mahmood [c], Mohd. Diya Masmoum [d], Shaf A. Altaf [e] and Muhammad Sohail S. Mirza [f*]

[a] Benedictine University, Illinois, USA.
[b] Baylor College of Medicine, Houston, USA.
[c] Henry Ford Health System, Detroit, USA.
[d] Alfaisal University, Riyadh, Saudi Arabia.
[e] King Edward Medical College, Lahore, Pakistan.
[f] Shandong University School of Medicine, Jinan, China.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Review Article

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ABSTRACT

Myocardial infarction (MI), often known as a heart attack, is a severe form of coronary artery disease that causes myocardial damage or necrosis as a result of persistent ischemia. Hypertension, atherosclerosis, diabetes, hyperlipidemia, smoking, lack of physical activity, and obesity are all significant risk factors. Recognizing symptoms such as chest pain, shortness of breath, heavy perspiration, and nausea is crucial for timely treatment. Advances in high-sensitivity troponin tests, coronary angiography, percutaneous coronary intervention (PCI), and

**Corresponding author: E-mail: drsohailmirza2024@gmail.com;*

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pharmaceutical therapies, such as antiplatelet medications, beta-blockers, and statins, have all improved patient outcomes. The reduction of MI morbidity and mortality is contingent on early detection, prompt intervention, and ongoing care. This comprehensive study underlines the need to identify MI symptoms early and call emergency medical services to improve patient outcomes. Emerging medicines and future tactics in MI therapy, including novel antiplatelet agents and precision medicine approaches, hold promise for enhancing patient care.

Keywords: Myocardial infarction; heart attack; profuse sweating; hyperlipidemia.

1. INTRODUCTION

The most dangerous type of coronary heart disease is myocardial infarction, which arises from either acute or chronic myocardial ischemia brought on by an imbalance between oxygen supply and demand. An increase in cardiac biomarkers and clinical evidence that correlates with changes in ECG indicates myocardial injury or necrosis. Imaging can show acute abnormalities in regional wall motion or new damage to viable myocardium [1]. Severe and ongoing chest pain is one of the clinical signs of myocardial infarction; it frequently coexists with dyspnea, nausea, and sweating. Angina, ischemic episodes, acute to severe arrhythmias, which can be life-threatening, and congestive heart failure are all possible outcomes of myocardial infarction. Therefore, depending on the kind and severity of the infarction, aggressive therapy should be started in the case of clinical doubt regarding myocardial infarction. Using supportive care and secondary prevention strategies is also crucial. The primary factors influencing the prognosis are the severity of the clinical symptoms, coexisting diseases, and the patient's reaction to the first round of treatment [2].

1.1 Definition and Explanation

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 may present with chest pain or pressure that spreads to the neck, jaw, shoulder, or arm. In addition to a history and physical exam, myocardial ischemia may be accompanied by ECG abnormalities and increased biochemical markers such as cardiac troponin. This exercise discusses the pathogenesis, examination, and therapy of myocardial infarction and emphasizes the role of the interprofessional team in enhancing care for afflicted individuals [3].

2. LITERATURE REVIEW

Epidemiology: Analyzing epidemiology Myocardial infarction is the primary cause of cardiovascular disease (CVD)-related death worldwide. In 2016, 17.9 million deaths worldwide, or 31% of all deaths, were related to CVD. It is estimated that 2,36,000,000 people will pass away from CVD by 2030. Over 75% of deaths from CVD happened in poorer nations. Eighty-two percent of these deaths from CVD happened in low- and middle-income nations [4]. The South Asian nations of Bangladesh, Sri Lanka, India, Pakistan, and Nepal have the highest rates of cardiovascular disease. adults over 75 are more likely than younger adults under 45 to get an age-specific myocardial infarction in developed countries. The opposite is true, too, in South Asian nations, where older adults over 60 are less likely than younger adults under 45 to experience an age-specific myocardial infarction [5].

The importance of MI is based on its influence on society and individual health. The World Health Organization (WHO) reports that, globally, MI and other cardiovascular diseases come first in causing death: they lead to 17.9 million annual fatalities. In the USA alone, every year, as many as 805000 people suffer from myocardial infarction, including more than 605 000 individuals who have experienced heart attacks for the first time. Meningococcal Inflammation is not only effective in making people sick and making people die, but it is also expensive in so far as society and medical care are concerned [5].

This is a review for people interested in learning about heart attacks. We will look at their signs, causes, and what to do when they occur. Myocardial infarction is the clinical term for a heart attack, which is a life-threatening condition that requires urgent attention. This article will help you understand more about this disorder by using information from different sources, including research articles and medical facts.

2.1 Causes of Myocardial Infarction

Atherosclerosis and plaque rupture: A condition leading to the accumulation of cellular debris, inflammatory cells, and cholesterol and fatty deposits in the walls of arteries called atherosclerosis. Atherosclerotic plaques are its outcome [6]. Plaques of this sort, being more prone to bursting than others due to instability, increase early rupture risks of coronary arteries, resulting in myocardial ischemia or infarction [7]. MI, alongside related cardiovascular ailments, emerges among the biggest causes of death according to the World Health Organization, which says they account for 17.9 million deaths every year on a global scale. Atherosclerosis-related events, including MI and stroke, cause most cardiovascular mortality, emphasizing the importance of atherosclerosis in cardiovascular morbidity and mortality.

Acute myocardial infarction (AMI) results from the sudden obstruction of a coronary artery, typically due to the rupture of an atherosclerotic plaque, which triggers the formation of a thrombus. Plaques that are particularly prone to rupture often have a large lipid core, a significant infiltration of inflammatory cells, and a thin fibrous cap. When these unstable plaques rupture, the resultant thrombus can obstruct blood flow, initiating an acute coronary event. This process involves blood coming into contact with the exposed plaque material, leading to platelet activation and aggregation, and ultimately, coronary thrombosis [8].

2.2 Risk Factors

2.2.1 Non-modifiable risk factors

Age: Those who are older have a larger chance of dying from an acute myocardial infarction [9]. It's still unknown what process causes the noticeable increase in mortality that comes with age [10]. People 65 years of age or older account for 80% of heart disease deaths [11].

Gender: Gender influences the risk of MI, with males experiencing heart attacks at a higher rate than premenopausal women. However, women's risk increases significantly at menopause, partly due to altered hormone levels and diminished cardioprotective effects of estrogen [12]. Women are more likely than men to get MI later in age if they experience atypical symptoms, which might delay identification and treatment [13].

Family history: A family history of premature coronary artery disease (CAD) or MI majorly predicts the likelihood of myocardial infarction (MI). In those individuals whose parent, sibling, or any other close relative had a heart attack before reaching 55 for males and 65 for women, the chances of developing CAD increase [14]. This means that bad lifestyle preferences like diet are one-factor affecting several family members and their offspring, although they might not be related by blood.

2.2.2 Modifiable risk factors

Hypertension: High blood pressure is one well-established modifiable risk factor for MI [15]. According to the Centers for Disease Control and Prevention (CDC), about 45% of Americans have hypertension, which raises their risk of MI. In addition to increasing the strain on the heart, hypertension causes atherosclerosis, which causes arterial stiffness, endothelial dysfunction, and a higher risk of MI [16,11].

Hyperlipidemia: Elevated cholesterol, especially low-density lipoprotein cholesterol (LDL-C), is linked to a higher risk of atherosclerosis and MI. Dyslipidemia raises a person's risk of MI because it encourages the development of atherosclerotic plaques in the coronary arteries [17]. It is usual practice to lower LDL-C levels and lower the risk of cardiovascular events with statin medication [11].

Diabetes: Diabetes mellitus of any form, including type 1, significantly increases the risk of MI. Chronic hyperglycemia accelerates atherosclerosis, oxidative stress, inflammation, and endothelial dysfunction, all of which contribute to the development of CAD and MI. According to the International Diabetes Federation, those with diabetes have a two to three times higher risk of MI than those without the condition [18].

Smoking: Smoking is one of the primary modifiable risk factors for MI because it damages

the heart. Tobacco smoke contains several toxic compounds that raise the risk of MI by encouraging inflammation, atherosclerosis, endothelial dysfunction, and platelet aggregation [19]. The World Health Organization reports that tobacco use kills over 8 million people worldwide and that smoking is the primary cause of MI [20].

Obesity: Obesity is a body mass index (BMI) of 30 kg/m2 or greater, associated with an increased risk of MI. Atherosclerosis and MI are risks that are increased by obesity [21]. In addition, it results in insulin resistance, dyslipidemia, metabolic issues, and systemic inflammation. According to the National Health and Nutrition Examination Survey, obesity has spread like wildfire and is having a catastrophic impact on cardiovascular health worldwide [22].

Sedentary lifestyle: Physical inactivity is a modifiable risk factor for MI, independent of other risk factors, including obesity and high blood pressure. In addition to other cardiovascular benefits, regular exercise enhances lipid profiles, blood pressure regulation, endothelial function, and weight management [23]. Sedentary behavior, on the other hand, has been connected to an increased risk of MI and other cardiovascular events. According to estimates from the World Health Organization, insufficient physical activity results in 3.2 million deaths annually throughout the globe [20].

Role of inflammation and endothelial dysfunction: Chronic inflammation plays a crucial role in the development of atherosclerosis, the primary cause of most myocardial infarctions (MIs). In response to endothelial injury, circulating monocytes migrate into artery walls to form foam cells, which release inflammatory mediators and hasten the formation and spread of plaque [24]. Moreover, inflammation causes plaques to become unstable, which raises the possibility that they may burst and that an acute coronary event, like MI, would follow. Intravascular imaging studies have demonstrated the presence of inflammatory cells in the responsible lesions, shedding light on inflammation in plaque susceptibility and rupture [25].

Endothelial dysfunction, a key feature of atherosclerosis associated with increased arterial permeability, a prothrombotic state, and diminished vasodilation, is common between MI and atherosclerosis [26]. Increased vascular tone and inadequate vasodilatory action from reduced nitric oxide (NO) bioavailability in injured endothelium raise the risk of hypertension and coronary artery spasm. Defective endothelium also promotes a pro-thrombotic phenotype, characterized by increased production of adhesion molecules, tissue factors, and platelet activators. In regions with plaque erosion or rupture, there is an increase in platelet aggregation and thrombus formation [27].

Numerous epidemiological studies have demonstrated the link between the risk of MI and inflammatory biomarkers, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and high-sensitivity C-reactive protein (hs-CRP). Elevated inflammatory marker levels, unaffected by conventional risk factors, can predict the onset of CAD and unfavorable cardiovascular events, such as MI [28].

2.3 Symptoms of Myocardial Infarction

2.3.1 Atypical symptoms

Shortness of breath: Dyspnea is one symptom of myocardial infarction that may appear even in the absence of the usual chest pain. Dyspnea or rapid, shallow breathing are common symptoms in patients [29]. A myocardial infarction (MI) may be indicated by dyspnea due to diminished cardiac output or pulmonary congestion from left ventricular dysfunction [30]. Respiratory conditions, including chronic obstructive lung disease and congestive heart failure, are frequently associated with dyspnea [31].

Research shows that shortness of breath (dyspnea) is typically the first indicator of a heart attack in old people, the elderly, and those who are affected by heart or lung complications simultaneously. The American Journal of Cardiology has reported that as many as 40% of individuals suffering from acute coronary syndrome show dyspnea as their primary symptom. Therefore, this justifies the need to consider dyspnea when evaluating possible MI patients [32].

Nausea and Vomiting: After a heart attack, nausea and vomiting can happen, especially among women and elderly persons. A person can feel nausea, vomit, or be sick to his/her stomach. These stomach-related signs may be linked to an activation of the autonomic nervous system or a referral of interior discomfort caused by cardiac ischemia [33].

Studies reveal that nausea and vomiting are more common among older women with MI. Women were more predisposed than men to develop atypical symptoms such as nausea, vomiting, or back pain following acute coronary episodes, based on the JAMA Internal Medicine publication. At the same time, research published in the American Journal of Medicine found that older people with MI complain more often of gastrointestinal problems [34].

Diaphoresis: Sweating too much is known as diaphoresis, and it may tell us someone is at risk of having a heart attack when the sympathetic nervous system becomes active because it does not get enough oxygenated blood [35]. Sometimes, even under the usual weather changes, normal people feel cold or hot and sweat more than usual. Excessive perspiration may indicate other symptoms of this disease, such as chest tightness or shortness of breath [36].

Studies show that diaphoresis is a very prevalent symptom in 20–30% of MI patients [37]. According to research published in the American Journal of Emergency Medicine, diaphoresis was more common in patients with ST-segment elevation myocardial infarction (STEMI) than in those without it. This finding raises the possibility that diaphoresis is an indication of more severe ischemia.

Gender differences in symptomatology: Though men and women react to acute cardiac events differently, chest discomfort is the most typical indication of myocardial infarction in both sexes. Compared to men, women are more likely to experience odd symptoms rather than merely typical chest discomfort. These symptoms include dyspnea, tiredness, nausea, and vomiting. Research indicates that women are less likely than males to recognize or think that their symptoms are due to heart ischemia; as a result, they delay seeking medical attention and receiving a diagnosis [32].

Women were less likely than men to report chest pain as their main symptom during acute coronary syndromes, with 43% of women reporting atypical symptoms compared to 31% of males, according to research published in Circulation: Cardiovascular Quality and Outcomes. Women also tended to present with symptoms other than chest pain, which made identification more difficult and prolonged the start of therapy [37].

2.4 Emergency Response to Myocardial Infarction

Recognizing symptoms and activating emergency medical services (EMS): For the best results and fastest care, it's imperative to identify the signs of MI and promptly activate EMS. Suspicion for MI should be raised in the event of chest pain or discomfort and related symptoms such as palpitations, nausea, vomiting, diaphoresis, and shortness of breath. Research has demonstrated that people with MI who delay seeking assistance and starting therapy usually have less favorable results [38]. The American Heart Association reports that 47% of people with acute myocardial infarctions wait more than two hours after symptoms begin to appear before seeking medical attention. This demonstrates how crucial it is to spread knowledge and create awareness of the necessity of quickly identifying and initiating emergency medical help [39].

Immediate interventions: The first care is to minimize symptoms as much as possible, keep the patient stable until more advanced treatment is available, and prevent further cardiac damage. These therapies are often started as soon as emergency medical services (EMS) arrive or continued by emergency hospital professionals.

Administration of aspirin: Unless there are restrictions, aspirin should be given to all suspected instances of MI immediately since it is a vital component of the therapy for acute MI. Aspirin inhibits cyclooxygenase-mediated platelet activation and aggregation, which lowers the risk of thrombus formation and myocardial ischemia [40]. As soon as it is feasible, individuals with a suspected MI should be given chewable aspirin (162–325 mg), according to the American College of Cardiology and the American Heart Association. After that, you should take maintenance medication consistently for the remainder of your life [39].

Use of nitroglycerin for chest pain relief: One typical therapy for MI-related chest discomfort symptoms is nitroglycerin. Since nitroglycerin mainly dilates venous capacitance arteries, which lowers oxygen demand and preload in the heart and lessens angina symptoms, it is categorized as a vasodilator. When treating individuals with suspected MI for chest discomfort, sublingual nitroglycerin is frequently the first medication utilized. Nitroglycerin can induce severe hypotension. Thus, persons who

already have hypotension or are using PDE inhibitors at the same time should be cautious [41].

Initiation of cardiopulmonary resuscitation (CPR) if necessary: Cardiopulmonary resuscitation (CPR) must start right away in instances of hemodynamic instability or cardiac arrest to preserve essential organ perfusion and circulation. Before performing advanced life support procedures, competent bystanders or medical professionals should respond right away to administer successful CPR, which consists of chest compressions and rescue breaths. For early defibrillation in patients with ventricular fibrillation or pulseless ventricular tachycardia, an automated external defibrillator (AED) may also be necessary [42].

2.5 Advances in Diagnosis and Treatment

High-sensitivity troponin assays for early diagnosis: Identifying myocardial damage at earlier stages of development is essential for preventing heart attacks (myocardial infarctions). This is important because once the muscle tissue has started to die, it is almost impossible to reverse the process. Myocardial infarctions are the leading cause of death among both men and women globally. In contrast to the regular/standard checks for the troponins, which have relatively broad detection levels, high-sensitivity assays use much narrower testing limits, thereby ensuring that we can even pick up a slight rise in troponin amounts sometime in the first few hours after symptoms have set in. [43].

Many studies have shown that high-sensitivity troponin tests are more effective than conventional diagnostic procedures when detecting patients who suffered a heart attack [43]. An example is research that appeared in JAMA Cardiology, which indicated that HS assays were more sensitive than their counterparts while having NPVs that allowed safe exclusion of MI. So, because of a faster diagnosis, the patient spent fewer days in the hospital. This means that using this tool helps improve treatment [44]. Detecting heart attacks early High-sensitivity troponin testing also improves clinical judgment and allows for better risk assessment in people brought to the hospital with chest pain, hence allowing for immediate commencement of drugs and their appropriate discharging aftercare in place.

Coronary angiography and percutaneous coronary intervention (PCI): Coronary angiography and percutaneous coronary intervention (PCI) are essential for therapeutic strategies that aim at treating acute myocardial infarction because they allow a conclusive diagnosis and revascularization of occluded coronary arteries. With coronary angiography, myocardial ischemic lesions can be detected, and the architecture of the coronary artery can be visualized. The percutaneous coronary intervention (PCI) is a minimally invasive procedure aimed at unblocking or dilating narrowed or obstructed coronary arteries to restore blood flow into ischemia areas.

Early coronary angiography and percutaneous coronary intervention (PCI) have been proven to be useful for everyone who has had a heart attack, and this is based on numerous research findings [45]. When primary PCI is carried out within a few hours of the beginning of symptoms, research published in the New England Journal of Medicine reveals that it is superior to fibrinolytic therapy in terms of lowering mortality, recurrent myocardial infarction, and the need for repeat revascularization. Furthermore, by causing quick and total reperfusion of the infarct-related artery, research like the TIMI (Thrombolysis in Myocardial Infarction) trials have shown how effective PCI is in improving outcomes for patients with ST-segment elevation myocardial infarction (STEMI) [46].

2.6 Pharmacological Interventions

Antiplatelet agents: Antiplatelet medications are essential to treating MI because they stop platelets from activating and aggregating, preventing thrombus formation and subsequent coronary artery blockage [47]. Most often, aspirin, a nonsteroidal anti-inflammatory drug, is used as an antiplatelet agent to treat MI. Thromboxane A2, a powerful platelet activator, is stopped permanently by inhibiting the cyclooxygenase-1 (COX-1) enzyme's activity. Additionally, to lower the risk of stent thrombosis and recurrent ischemic events, patients with acute coronary syndromes undergoing percutaneous coronary intervention (PCI) are advised to receive dual antiplatelet therapy (DAPT), which combines aspirin with a P2Y12 receptor inhibitor such as clopidogrel, ticagrelor, or prasugrel [48].

Antiplatelet treatment has been shown in several cutting-edge studies to be beneficial in lowering adverse cardiovascular events in individuals with MI. For instance, clopidogrel with aspirin decreased the incidence of cardiovascular

mortality, myocardial infarction, or stroke in patients with acute coronary syndromes when compared to aspirin alone, according to the Clopidogrel in Unstable Angina to Prevent Recurrent Events (CURE) study [49]. Ticagrelor and prasugrel were also more successful than clopidogrel at lowering the frequency of ischemic episodes in patients with acute coronary syndromes in the PLATO and TRITON-TIMI 38 trials [50].

Beta-blockers: The beta-blocker medication family is another commonly used to treat MI. It reduces symptoms, improves left ventricular function, and lowers the heart's oxygen demand. Beta-blockers decrease heart rate, myocardial contractility, and systemic blood pressure by blocking the action of beta-adrenergic receptors. Moreover, beta-blockers antiarrhythmic properties may protect MI patients against ventricular arrhythmias [51].

The use of beta-blockers in the treatment of MI is supported by significant studies like the COMMIT and ISIS-1 investigations, which showed that early beta-blocker medication in patients with acute MI lowered mortality and recurrent myocardial infarction. Despite current guidelines recommending its selective use in hemodynamically stable patients without contraindications such as heart failure or severe bronchospasm, beta-blocker use in treating MI has changed over time [52].

Statins: Statins, also known as 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors, are lipid-lowering medications that stabilize atherosclerotic plaques and lower low-density lipoprotein cholesterol levels [53]. This makes them essential for secondary prevention after MI. Their pleiotropic effects, which improve endothelial function, reduce inflammation, and prevent thrombosis, are the reasons why statins are good for the heart [54].

Two clinical trials that have shown the advantages of statin medication in lowering cardiovascular events and enhancing survival in patients with coronary artery disease, including those who have a history of MI, are the Scandinavian Simvastatin Survival Study (4S) and the PROVE IT-TIMI 22 study. Regardless of baseline cholesterol levels, guidelines from professional associations like the American College of Cardiology and the American Heart Association advise that all patients with acute coronary syndromes start high-intensity statin medication [55-58].

Emerging therapies and future directions: Recent scientific studies aim to find different ways to treat heart attack patients besides employing traditional medications that may increase their survival chances. Some of the future directions and emerging therapies in MI management include the development of novel antiplatelet agents with improved safety and efficacy profiles, the investigation of adjunctive therapies that target inflammation and stabilize plaque, and the exploration of precision medicine approaches based on genetic profiling and customized risk stratification. Exciting research areas include clinical studies evaluating the efficacy of anti-inflammatory drugs in preventing repeat cardiovascular events and the development of antiplatelet drugs that precisely target platelet activation pathways. By using individual risk prediction algorithms, improvements in precision medicine may refine treatment plans and enable personalized therapies that enhance the long-term prognosis of MI patients.

3. CONCLUSION

This thorough study demonstrates the importance of not wasting time getting treatment for myocardial infarction to improve patient outcomes. It is important to note that MI is one of the main causes of death worldwide; therefore, recognizing its symptoms promptly and calling emergency medical services (EMS) is paramount. The word electronic comes from the Greek word “Elektron,” meaning amber. Amber is a hard translucent fossil resin used for making jewelry. During the early history of electricity, amber was referred to as electrostatic. Precision medicine holds promise for bettering the treatment of MI by stabilizing plaque and controlling inflammation with cutting-edge methods. All things considered, this all-encompassing method emphasizes the significance of interdisciplinary efforts in strengthening treatment plans and boosting patient outcomes for MI patients.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during the writing or editing of manuscripts.

CONSENT AND ETHICAL APPROVAL

It is not applicablke.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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