

# A REVIEW OF MEDICAL EMERGENCIES RELATED TO THE EMBOLIC, INFLAMMATORY, AND MECHANICAL COMPLICATIONS IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

## Abstract

Although the incidence of coronary heart disease (CHD) has decreased in the United States, acute myocardial infarction (AMI) remains a major medical problem. Many patients still need emergency care in the emergency department (ED) due to complications due to mechanical, inflammatory, and embolic factors. This summary aims to provide a brief summary of the available evidence in clinical medicine regarding the evaluation and management of mechanical, inflammatory, and embolic complications after myocardial infarction. Although 30-day mortality after AMI has decreased in recent years, it remains high at 7.8% due to many subacute complications that occur within a few weeks. Mechanical problems such as ventricular free wall rupture, ventricular septal rupture, mitral regurgitation, and left ventricular aneurysm formation have a high risk of morbidity. The complications of MI can vary in severity and can affect different organs and systems of the body. Heart attacks can disrupt the normal electrical activity of the heart, leading to irregular heart rhythms, such as ventricular tachycardia or ventricular fibrillation. A heart attack can weaken the heart muscle, making it difficult for the heart to pump blood effectively. This can lead to heart failure, a condition where the heart is unable to meet the body's demand for blood and oxygen. In severe cases, a heart attack can cause cardiogenic shock, a condition characterized by a significant drop in blood pressure and inadequate blood flow to vital organs. This is a life-

threatening situation that requires immediate medical attention. Sometimes, a heart attack can cause a rupture in the wall separating the heart's chambers (ventricular septum), leading to the formation of a VSD. This condition allows blood to flow between the ventricles, decreasing the heart's ability to pump blood effectively.

Mechanical, inflammatory, and embolic complications associated with AMI can have a significant impact on morbidity and mortality. Physicians should check for existing conditions when considering other diagnoses.

In addition to monitoring disease progression and physical examination, electrocardiography and point-of-care echocardiography allow rapid and noninvasive assessment of the underlying pathophysiology. Management may vary depending on the particular presentation and etiology; however, relationships with cardiologists and cardiologists are recommended.

**Keywords:** emergency medicine, embolic complications, acute myocardial infarction, complicated myocardial infarction

## INTRODUCTION

Atherosclerotic cardiovascular disease is the leading cause of death worldwide [1] Although the incidence of coronary heart disease (CHD) has decreased in the United States In the United States, studies show that the incidence of myocardial infarction (AMI) is not decreasing, at approximately 208 per 100,000 person-years, of which it is approximately 22%. ST-segment elevation myocardial infarction (STEMI) [2]. Although there is an improvement in 30-day mortality after AMI, it remains as high as 7 days. 8% [2].

Ischemic complications can affect the kidneys, leading to renal failure, and embolic strokes from arrhythmias can affect the brain and heart with the development of left ventricular (LV)

aneurysms [7–9]. Ischemic complications may also be associated with stent thrombosis and secondary heart failure with increased morbidity [10,11]. In the 1980s the name of myocardial infarction was changed to include ECG evidence of MI. Q wave MI replaces the old definition of transmural MI and is used for MI involving all three layers of the myocardium, causing pathological Q waves in both leads on the ECG. Non-transmural MIs are referred to as non-Q-wave MIs on the assumption that the Q wave will not appear on the ECG unless the entire myocardial thickness is included. However, clinical trials in subsequent years have not confirmed that Q-wave MI is equivalent to transmural MI. Thus, in the 1990s, ST-segment elevation myocardial infarction (STEMI) and non-STEMI (NSTEMI) were considered the preferred terms.

## **METHODS**

Methods included include published literature, case reports, retrospective and prospective studies, systematic reviews and meta-analyses, clinical trials, and other reviews. Search is limited to English publications. Initially, more than 600 articles were identified and, with the agreement of the authors, important articles focusing on emergency medical information and guidelines were selected as medicine. Finally, 177 emergency treatment and referral programs were included. As for descriptive analysis, individual survey data were not collected.

## **LITERATURE REVIEW**

### **3.1 Ventricular free wall rupture**

#### **3.1.1 Etiology**

Left ventricular free-wall rupture is a critical mechanical complication that can arise from an acute myocardial infarction (heart attack). It primarily occurs in individuals with ST-elevation myocardial infarction (STEMI), although it can also be caused by infections or trauma. Timely

diagnosis relies on maintaining a high level of suspicion and employing transthoracic echocardiography to aid in the assessment. VFWR can be characterized as simple or complex and may involve the inner or posterior wall of the left ventricle (LV). Simple VFWRs provide direct access to the defect, while complex VFWRs exhibit convoluted anatomical bundles that connect with the primary tear.

Although posterior and posterior AMIs are more prone to white wall ruptures, they are less common in anterior AMIs because they are more common. Subacute VFWRs are often associated with inferior infarcts. The distribution of infarcted arteries causing VFWR was as follows: circumflex coronary artery (LCX) in 40% of patients, left anterior descending artery (LAD) in 42%, and right coronary artery (RCA) in 18%.

### **3.1.2 Risk Factors**

Risk factors for VFWR include coexistence, age over 55 years (usually 65-70 years), high blood pressure, large infarcts, single artery (usually total occlusion), transmural infarcts, pseudoaneurysm formation, delayed or incomplete revascularization. Patients without a history of angina are more likely to develop VFWR after their first AMI, suggesting a limited risk of coronary artery disease.

### **3.1.3 Diagnosis and Presentation**

Early detection of VFWR is important to patient survival. The clinical presentation may vary, with some patients experiencing prolonged or short-term angina in the days before rupture. Physical exertion and persistent ischemia can cause VFWR. Patients may also have sexual dysfunction, shortness of breath, or hypotension suggestive of VFWR and cardiac arrest. Electrocardiography (ECG) has limited specificity in diagnosing VFWR, but some findings such

as abnormal electrical activity or electrical changes may be seen. Echocardiography plays an important role in the diagnosis by showing signs of pericardial effusion and cardiac tamponade. Chest radiography and computed tomography angiography may provide additional information but are not usually required for diagnosis.

### **3.1.4 Prognosis**

The prognosis of VFWR remains poor, with a mortality rate ranging from 75% to 94%. Subacute VFWR is less likely to cause rapid hemodynamic deterioration. Effective surgical treatment was associated with a 48-year survival. Between 5% and 76% and most well-treated patients have long survival.

## **3.2 Ventricular septal rupture (VSR)**

### **3.2.1 Causes**

VSR can be classified as simple or complex. Simple VSRs are characterized by a single disorder in the interventricular system, while complex VSRs involve multiple pathways connecting the ventricles that develop after septal rupture. The location of the VSR can provide insight into subtypes such as the complex forms associated with posteroinferior infarcts and the simpler forms seen in posterior-anterior infarcts.

### **3.2.2 Diagnosis And Presentation**

Acute mitral regurgitation (MR) usually occurs 2-7 days after myocardial infarction (AMI) with an average duration of 13 hours. The clinical presentation varies according to the severity of MR. In the case of fulminant MR due to papillary muscle rupture, patients may develop pulmonary edema, hypotension, and cardiogenic shock. However, the physical examination may be nonspecific and some patients with mild to severe MR may have a nonspecific complaint.

However, a diastolic murmur towards the left axilla and a pan systolic murmur at the highest apex can cause MR.

Transthoracic echocardiography (TTE) can provide valuable information about the size and direction of the regurgitation jet, visualize the anterior surface and evidence of mal coaptation, and assess biventricular function.

In the case of incomplete TTE, transesophageal echocardiography (TEE) is required, especially if MR is suspicious despite incomplete TTE results.

### **3.2.8 Timely Management**

Early diagnosis and surgical treatment of MR are important to improve survival. Current management includes the management of pulmonary edema, which may require mechanical ventilation (NIV) or endotracheal intubation. Medical therapy aims to advance the anterior left ventricle and reduce MR.

Vasodilators such as sodium nitroprusside or nitroglycerin are used to reduce afterload, while inotropes such as dobutamine improve inotropy and vasodilation. Consider supplementing any device with devices such as an intra aortic balloon pump (IABP), Impella, or extracorporeal membrane oxygenation (ECMO). The decision for surgical treatment or percutaneous intervention depends on the severity and underlying etiology of MR and requires the cooperation of specialist physicians and interventional cardiologists.

### **3.3.5 Prognosis**

Chronic MR after AMI has a poor prognosis if left untreated. Mortality is approximately 75% in 24 hours and increases to 95% in 2 weeks. Surgical treatment has an operative mortality of approximately 39% and in-hospital mortality of 55%.

## **3.4. Pericarditis after myocardial infarction (PMIP)**

### **3. 4.1. Etiology**

It is caused due to the inflammation of the perimyocardium after myocardial infarction. This causes inflammation and an autoimmune response in exposed individuals. The inflammation may appear as simple pericarditis or progress to more serious conditions such as cardiac tamponade, pleural effusion, and/or pleuropericarditis.

PMIP can be divided into two types depending on time. Previously, early PMIP was estimated to occur in 10-20% of patients after AMI, but with the advent of thrombolytic therapy, this rate has dropped to about 6% [118,119].

The incidence of late PMIP, formerly known as DS, is 3-4%, but modern studies show the incidence to be less than 1%.

### **3.4.2. Risk Factors**

There are many risk factors that can contribute to the development of PMIP. These include delayed reperfusion, higher cardiovascular biomarker levels, smaller size, younger age, anterior ischemia, right ventricular and inferior infarction, and decreased left ventricular ejection fraction [121–124].

### **3.4.3. Clinical Diagnosis And Presentation**

Acute pericarditis, while often resolving on its own, can lead to notable complications. It is important to distinguish between acute idiopathic pericarditis and pericarditis resulting from cardiac injury, as they have distinct underlying mechanisms, complication profiles, and natural progressions. Measuring serum troponin I levels may help identify patients with elevated levels who paradoxically face a lower risk of treatment failure, recurrences, constrictive pericarditis, tamponade, or death.

Patients who exhibit a positive response to anti-inflammatory medications typically have a

favorable prognosis. Nevertheless, there is a possibility of experiencing complications such as cardiac tamponade, constrictive pericarditis, and recurrent episodes.

#### **3.4.4 Management**

Although specific information on PMIP management is limited, treatment guidelines can be determined from studies of acute pericarditis. For early PMIP, which usually resolves on its own, it is recommended not to use non-steroidal anti-inflammatory drugs (NSAIDs) other than once-daily aspirin for secondary prevention during the first 7-10 days after AMI.

Acetaminophen is the drug of choice for patients with severe symptoms requiring sedation, and high-dose aspirin is considered second-line [123].

Management of advanced PMIP usually includes supportive care and drug therapy with NSAIDs, colchicine and corticosteroids [115–117]. Because of the need for antiplatelet therapy and the potential interaction of other NSAIDs with myocardial damage and scar formation, aspirin is the first choice for PMIP patients [115]. Indomethacin should be avoided as it reduces blood flow [113]. High-dose aspirin (800 mg orally every 6-8 hours for 7-10 days, then tapering down to 800 mg weekly for 3 weeks) has been shown to be effective in most cases in research [127].

Ibuprofen has been shown to increase blood flow in the arteries and can be administered at a weekly dose of 400 to 800 mg for a period of 3-4 weeks, with doses of 600 to 800 mg every 6 to 8 hours [124]. It is important to provide gastrointestinal protection in all patients treated with NSAIDs, usually proton pump inhibitors [115].

Low-dose corticosteroid therapy (eg, prednisone 0.2-0.5 mg/kg/day for 4 weeks followed by tapering) has been shown to be effective in patients who discontinue aspirin/NSAIDs or who do not respond to treatment.

If steroids are used, the dose should be reduced to prevent the recurrence of PMIP. In the case of PMIP complicated by cardiac tamponade, pericardiocentesis is required [115,117].

### **3.6. Other Complications**

Many subacute complications can occur after acute myocardial infarction (AMI). These complications include ischemic stroke, kidney failure, heart failure, and cardiac arrhythmias. Some individuals may experience recurring chest pain or angina even after the heart attack has occurred. This can be caused by partial blockages in the coronary arteries or persistent narrowing of the blood vessels supplying the heart. Following a heart attack, inflammation of the pericardium (the sac surrounding the heart) can occur, causing chest pain, fever, and other symptoms. Pericarditis can sometimes lead to complications such as pericardial effusion (accumulation of fluid in the pericardium) or constrictive pericarditis (thickening and stiffening of the pericardium). Sometimes, a heart attack can cause a rupture in the wall separating the heart's chambers (ventricular septum), leading to the formation of a VSD. This condition allows blood to flow between the ventricles, decreasing the heart's ability to pump blood effectively.

## **CONCLUSION**

These complications include all embolic, ischemic, arrhythmic, and inflammatory processes that occur in the weeks after AMI. Ventricular white wall rupture, ventricular septum rupture, acute mitral valve insufficiency, left ventricular aneurysm, pericarditis after myocardial infarction, etc. may appear in the form. Effective management requires a good understanding of the underlying pathophysiology and collaboration with cardiology and cardiac surgery specialists is recommended when needed.

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