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Research Article

Unforeseen Threats: Mechanical Complications in the Aftermath of Acute Coronary Syndromes

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Abstract

Introduction: Acute coronary syndromes (ACS) are cardiovascular emergencies resulting from the rupture or erosion of atherosclerotic plaque in the coronary arteries. Despite therapeutic advances that have reduced mortality, serious mechanical complications, such as myocardial rupture, acute valvular insufficiency and ventricular septal defect, can still occur, increasing the risk of mortality. The present work aims to describe mechanical complications in ACS in our setting, examine their frequencies, pathophysiological mechanisms and therapeutic approaches through our series and literature review.

Materials and methods: This retrospective study included 20 patients with mechanical complications of myocardial infarction, mainly diagnosed at the thoracic pain unit of the Mohammed VI University Hospital, Marrakech, and hospitalized in cardiology over a two-year period. Selected patients presented with myocardial rupture, acute mitral insufficiency, interventricular septal rupture, right ventricular free wall rupture or left ventricular aneurysm. Data on incidence, diagnosis and clinical outcome were analyzed.

Results: During the study period, 20 patients, with a mean age of 69 and a female predominance, presented with post-infarction mechanical complications without a history of ischemic heart disease. The main complications included ventricular septal defects (50%), intramyocardial dissections (15%), acute mitral insufficiency (15%) and ventricular wall rupture. Echocardiography enabled rapid diagnosis. Half the patients were stable on admission, while three quarters progressed to cardiogenic shock, often fatal.

Discussion and conclusion: Mechanical complications of ACS are rare but serious events that complicate patients' prognosis. Early detection by echocardiography or cardiac MRI, followed by surgical management, remains crucial to improving survival. Advances in early revascularization, notably coronary angioplasty, have helped to reduce the incidence of these complications, but optimal treatment of advanced cases relies on an experienced multidisciplinary team.

Keywords: acute coronary syndrome; mechanical complications; heart murmur; st-segment elevation; echocardiography; cardiogenic shock

Introduction

Acute coronary syndromes (ACS) represent a group of cardiovascular emergencies linked to the rupture or erosion of atherosclerotic plaque in the coronary arteries. Although current treatments significantly reduce mortality, serious mechanical complications can still occur, particularly in the absence of prompt intervention. These complications include myocardial wall rupture, acute valvular insufficiency and ventricular septal defect, each contributing to a high risk of mortality.

The present work aims to describe mechanical complications in ACS in our setting, examine their frequencies, pathophysiological mechanisms and therapeutic approaches through our series and literature review.

Materials and method:

This was a retrospective study of 20 patients with a mechanical complication of myocardial infarction diagnosed mainly in the thoracic

pain unit and subsequently hospitalized in the cardiology and vascular diseases department at the Mohammed VI University Hospital, Marrakech, over the past two years.

Inclusion criteria included clinical studies of patients with myocardial infarction complicated by myocardial rupture, acute mitral insufficiency, interventricular septal rupture, LV free wall rupture and LV aneurysm. Data on incidence, diagnostic methods and clinical outcomes were extracted and analyzed

Results:

During the study period, 20 patients developed post-infarction mechanical complications. The mean age of the patients was 69 years, with an age distribution ranging from 60 to 85 years, and a predominance of women. No history of ischemic heart disease was found in this cohort. All cases presented to the emergency department with acute coronary syndrome, characterized by ST-segment elevation beyond the therapeutic window for thrombolysis.

Of the complications observed, 50% corresponded to post-infarction ventricular septal defects with left-to-right shunting, 15% of cases were intramyocardial dissections, while the same percentage involved acute mitral insufficiency linked to papillary muscle dysfunction. Ten percent of patients presented with right ventricular free wall rupture, and the remaining cases were left intra-ventricular aneurysms. Anterior involvement on the electrocardiogram was the most frequent, and echocardiography was decisive in establishing the diagnosis of mechanical complications on admission.

Half the patients were admitted in stable clinical condition, while the remainder had left ventricular failure graded between Killip III and IV. Three quarters of patients progressed to cardiogenic shock, with a fatal outcome in hospital.

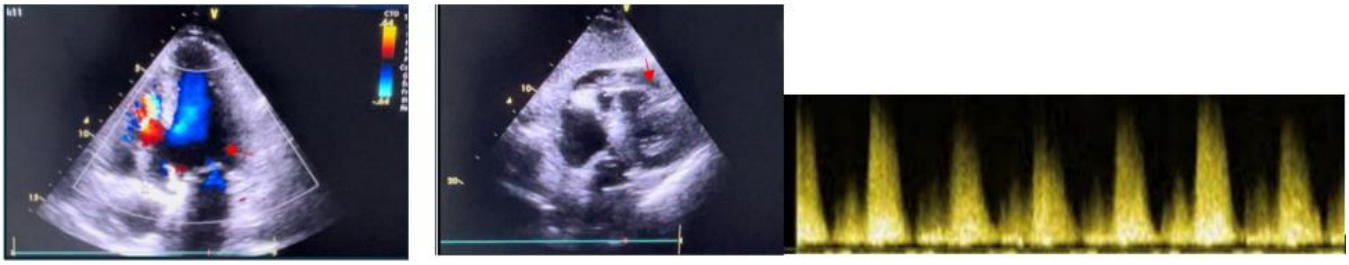

Figure 1: This is a 76-year-old patient admitted with inferobasal STEMI. The echocardiography showed segmental hypokinetic heart disease with preserved systolic function, a ruptured image in the inferobasal segment of the interventricular septum, and a breached right ventricle opposite its free wall, with systolic dysfunction.

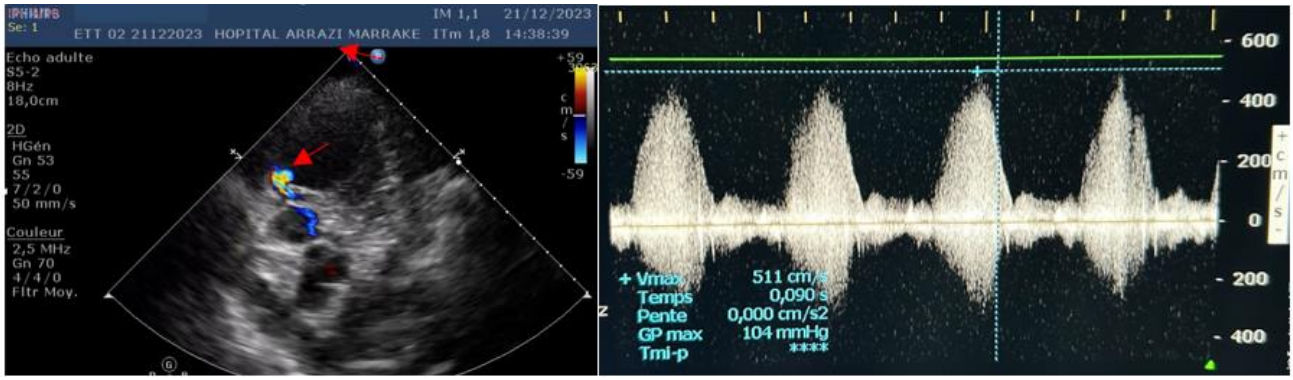

Figure 2: This 66-year-old patient was admitted with STEMI anteriorly. Echocardiography showed segmental hypokinetic heart disease with severe LV dysfunction (30-35%), the site of an apical aneurysm, associated with a 7 mm apical muscular IVC image responsible for a left-right shunt.

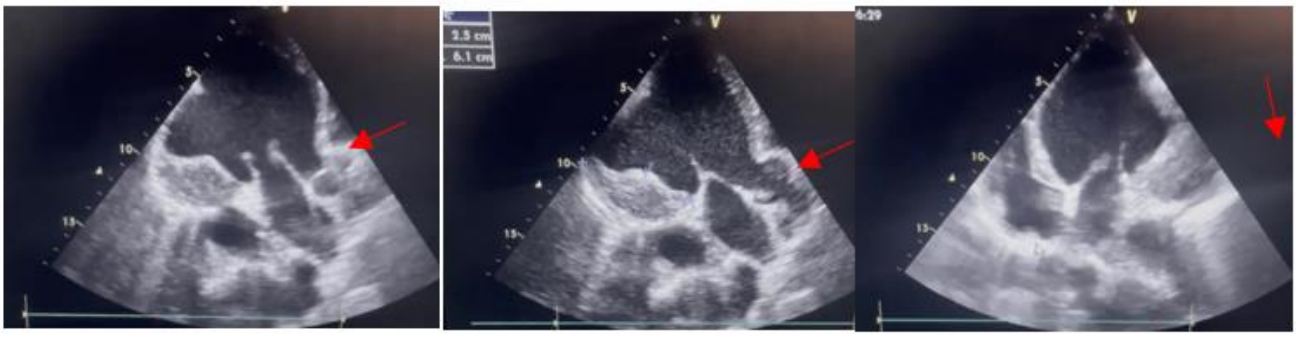

Figure 3: This 70-year-old patient was admitted with extensive anterior STEMI. Echocardiography showed segmental hypokinetic heart disease with severe LV dysfunction (20-25%) and an aneurysmal anterolateral wall containing a thrombus measuring 6.1 x 2.5 mm.

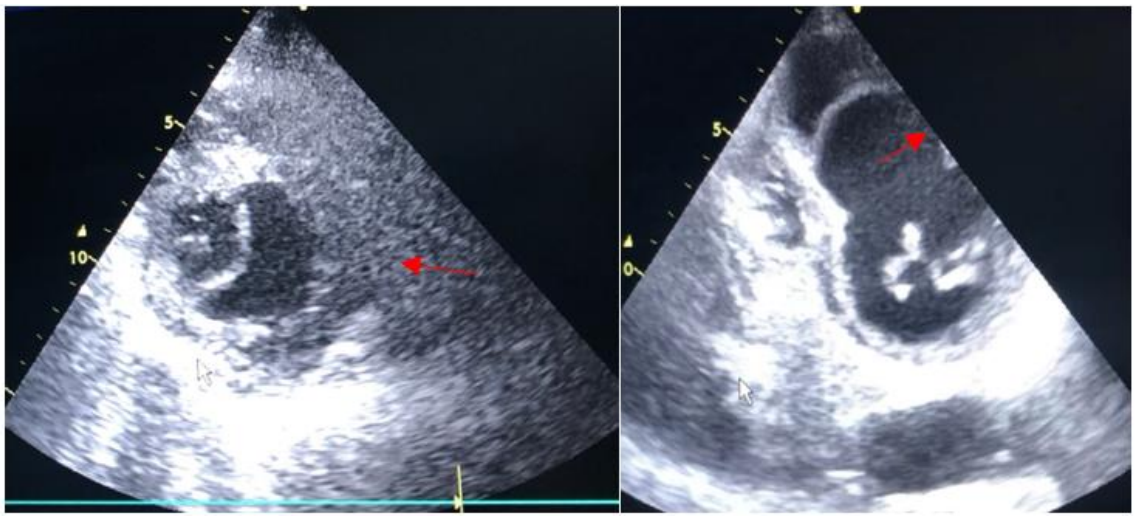

Figure 4: This 84-year-old patient was admitted with STEMI anteriorly. Echocardiography showed segmental hypokinetic heart disease with severe LV dysfunction (30%), a thickened, pulsatile LV cavity with dyskinetic motion, surrounded by a thin endomyocardial membrane in association with a haematoma secondary to intra-myocardial rupture.

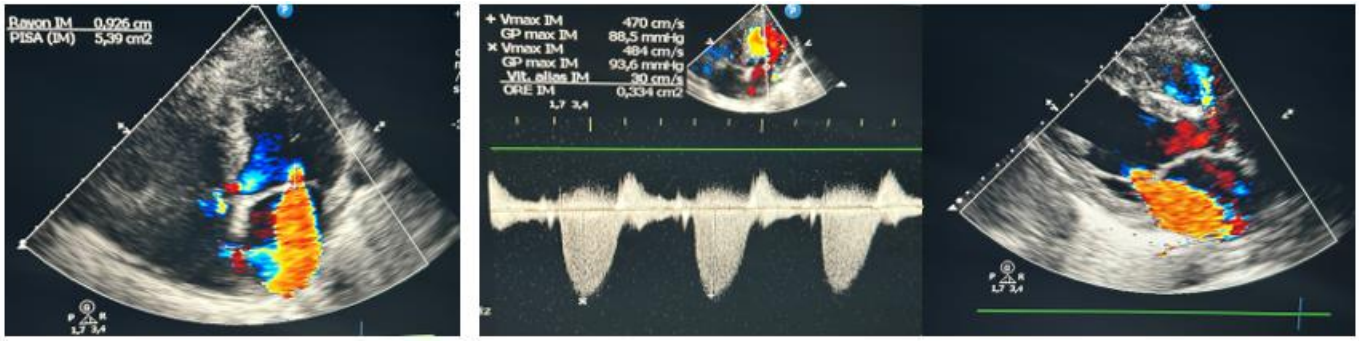

Figure 5: This 77-year-old patient was admitted with post-MI angina in inferior position, extended to the right shunts at H24, complicated by cardiogenic PAO. Echocardiography showed acute mitral insufficiency, on segmental hypokinetic cardiopathy with severe 30-35% LV dysfunction.

Discussion :

Reperfusion therapies have significantly reduced the frequency of mechanical complications following acute myocardial infarction. Studies estimate that after a myocardial infarction with ST-segment elevation, between 0.27% and 0.91% of patients develop mechanical complications. Papillary muscle rupture, ventricular wall rupture, and septal rupture occur in 0.05% to 0.26%, 0.01% to 0.52%, and 0.17% to 0.21% of cases, respectively [1,2]

Unfortunately, mortality rates associated with these complications have not significantly decreased over the past two decades, and patients with such mechanical complications are four times more likely to die in the hospital than those without complications. Although rare, mechanical complications remain a critical determinant of post-infarction prognosis [1,2,3]. While the proportion of patients with ST-segment elevation myocardial infarction has decreased over time, contemporary patients facing mechanical complications are often older, predominantly female, with a history of heart failure and chronic kidney disease, and frequently experiencing their first myocardial infarction [4,5,6]. Furthermore, socioeconomic disparities have a substantial impact on post-MI outcomes [7].

These complications include; Rupture of the left ventricular free wall, Acute severe MR, papillary muscle displacement, papillary muscle rupture, Ventricular septal rupture, Pseudo aneurysm formation, RV infarction, Dynamic LV outflow obstruction, LV thrombus.

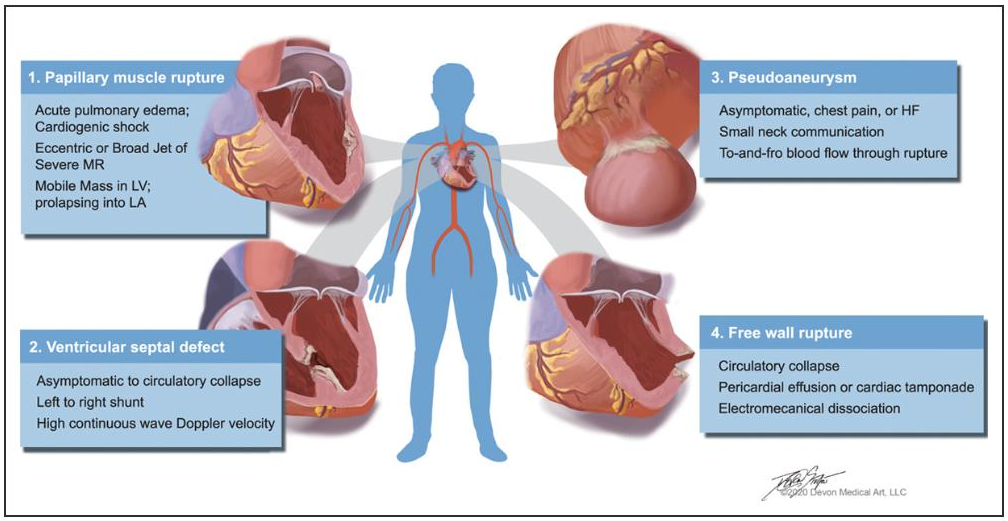

Figure 6: Characteristics of mechanical complications of acute myocardial infarction (7).

HF indicates heart failure; LA, left atrium; LV, left ventricle; and MR, mitral regurgitation. ©2020 Devon Medical Art LLC.

Aspects to consider	Ventricular septal rupture	Papillary muscle rupture	Free-wall rupture
Onset	Onset: 3–7 days from AMI	Onset: 3–7 days from AMI	Onset: 3–7 days from AMI
Clinical scenario	New acute chest pain episode, cardiogenic shock, pulmonary congestion, right heart failure signs and symptoms.	New acute chest pain episode, cardiogenic shock, acute pulmonary oedema.	New acute chest pain episode, cardiogenic shock, cardiac arrest, signs of cardiac tamponade.
Diagnosis/ workup	New cardiac murmur, echocardiographic evidence of ventricular septal defect, left-to-right ventricular shunt.	New cardiac murmur, echocardiographic evidence of mitral regurgitation with complete or partial rupture of PM (prolapse or flail of MV leaflet). Pulmonary hypertension. Hyperdynamic left ventricle.	Signs of cardiac tamponade, echocardiographic evidence of pericardial effusion, clots, contained rupture.
Management strategy	Haemodynamic stabilization (vasodilator, diuretics, IABP, inotropes). If possible, delayed surgical repair (beyond 7 days from diagnosis) with non-invasive or invasive systems like veno-arterial ECMO or other temporary percutaneous circulatory assist devices. Prompt surgery if refractory shock persistent or unresponsive right ventricular dysfunction develops.	Haemodynamic stabilization (vasodilator, diuretics, IABP, inotropes). If possible, delayed surgical repair (beyond 7 days from diagnosis) with non-invasive or invasive systems like veno-arterial ECMO or other temporary percutaneous circulatory assist devices. Prompt surgery if refractory shock persistent or unresponsive right ventricular dysfunction develops.	Immediate surgery.
Surgical treatment	Interventricular patch application. Prophylactic MCS in cases of left, right, or biventricular compromise and dysfunction. Heart transplant can be considered in patients with unsuccessful surgical repair and VSR recurrence with criteria for such a therapy and likely unsuccessful re-operation for VSR re-closure.	MV replacement (with rest of the native valve preserved besides resection of the involved PM and leaflet). In selected candidates (partial PM rupture), MV repair and PM reconstruction has been described.	Immediate surgery. Sutureless technique suggested if no large rupture present. Peri-operative support for reduced LVEDP

Table 1: Summary of Major Mechanical Complications of Acute Myocardial Infarction (8)

1.Post-myocardial infarction ventricular septal defect:

Post-infarction ventricular septal rupture (VSR) is defined as a rupture of the interventricular septum at the boundary between healthy and infarcted tissues [9,10]. The underlying mechanism involves the formation of an intramural hematoma within the infarcted area, leading to tissue dissection followed by rupture. The rupture typically occurs within 5–7 days after myocardial infarction (MI) but can sometimes occur within the first 24 hours due to a direct tear of the interventricular septum. The hemodynamic consequences include the presence of a left-to-right (L-R) shunt, resulting in volume overload of the left ventricle and pressure and volume overload of the right ventricle, which leads to biventricular failure with a predominance of right-sided failure [9,10].

The incidence of VSR represents 0.2% of all STEMI cases, compared to 1–2% before the advent of the percutaneous revascularization era. Risk factors for developing post-MI VSR include advanced age, female sex, hypertension, absence of prior infarction, and delayed presentation to the emergency department [11].

The patients' history typically reveals two-phase retrosternal chest pain (initial infarction followed by interventricular septal rupture), accompanied by dyspnea. The clinical presentation includes a loud holosystolic murmur at the left parasternal region with a characteristic "wheel-like" radiation, as well as signs of right-sided heart failure, such as jugular venous distension, a hyperdynamic apical impulse, and low-output state indicators (10). The electrocardiogram typically shows signs of acute or chronic ischemia, including ST-segment elevation, ST-segment depression, T-wave inversions, or pathological Q waves.

Initial management focuses on stabilizing hemodynamics through the use of vasodilators, diuretics, dobutamine, or norepinephrine as needed. An intra-aortic balloon pump (IABP) is recommended to reduce the left-to-right shunt, improve coronary perfusion, and limit oxygen consumption in cases of post-infarction ventricular septal rupture. For persistent

instability, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) may be considered (12). The timing of surgical intervention is critical: early repair is limited by tissue fragility, while delays increase the risk of complications and mortality. Surgical techniques include excision and suturing of the infarcted myocardium with or without a prosthetic patch, or the infarction exclusion technique, which involves attaching a patch to healthy myocardium. Modifications such as the use of multiple patches and reinforcement of the right septal wall are sometimes necessary to enhance postoperative stability [13,14].

Acute Mitral Regurgitation Secondary to Papillary Muscle Rupture

The incidence of severe acute mitral regurgitation (MR) caused by papillary muscle rupture (PMR) has decreased in the reperfusion era, ranging between 0.05% and 0.26%. However, in-hospital mortality rates remain high, ranging from 10% to 40%.

The mitral valve is supported by two papillary muscles: the anterolateral muscle, supplied by the left anterior descending artery (LAD) and the diagonal or marginal branch of the circumflex coronary artery; and the posteromedial muscle, which receives blood supply from the circumflex coronary artery or the right coronary artery, depending on dominance. Thus, rupture of the anterolateral papillary muscle is extremely rare, whereas rupture of the posteromedial papillary muscle is typically associated with inferior or lateral STEMIs. The rupture may be complete or partial, influencing the severity of clinical symptoms [15].

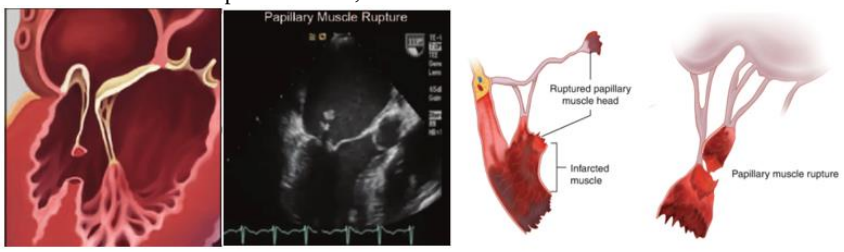

Figure 7: Ischaemic mitral valve disease. Mitral regurgitation caused by myocardial ischaemia following rupture of the papillary muscle results in excessive valve movement [15].

Risk factors for papillary muscle rupture (PMR) include advanced age, female sex, a history of heart failure, chronic kidney disease, and delayed presentation during a first acute myocardial infarction [16].

PMR typically occurs within days following an MI, with approximately half of the patients presenting with pulmonary edema that can rapidly progress to cardiogenic shock (see Table 1). Due to the rapid equalization of left atrial and ventricular pressures, a cardiac murmur may be absent. Modern cardiac intensive care units use bedside echocardiography or point-of-care ultrasound to manage acute cardiovascular diseases, facilitating the diagnosis of acute mitral regurgitation due to PMR. However, in cases of partial PMR, transthoracic echocardiography may not suffice for diagnosis, whereas transesophageal echocardiography has high diagnostic sensitivity. The left ventricular ejection fraction is often normal or slightly reduced, and coronary angiography typically reveals single- or double-vessel coronary artery disease with complete occlusion of the infarct-related artery [17].

Management of acute post-infarction mitral regurgitation includes hemodynamic stabilization with vasodilators and mechanical circulatory support (MCS) if necessary, although early surgery remains crucial due to high mortality (50%) with medical treatment alone. Mitral valve replacement is the preferred method, although repair may be considered in specific cases. Postoperative outcomes are similar between the two approaches, with perioperative mortality ranging from 8.7% to 24% and 5-year survival comparable to post-MI patients without mechanical complications. Transcatheter valve repair, although promising in isolated cases, requires further validation before widespread adoption [18,19].

LV Pseudoaneurysm and LV Aneurysms

Left ventricular (LV) pseudoaneurysms develop when cardiac rupture is contained by pericardial adhesions. Although they can occur following cardiovascular surgery, blunt or penetrating chest trauma, or infectious endocarditis, they are most commonly associated with anterior myocardial infarction.

Unlike true aneurysms, pseudoaneurysms more frequently affect the inferior or lateral wall, likely due to the pericardial adhesions that form in these regions during recovery from an infarction. While acute anterior wall rupture typically results in massive hemopericardium, catastrophic tamponade, and immediate death, other pseudoaneurysms may remain undiagnosed for several months [20, 21].

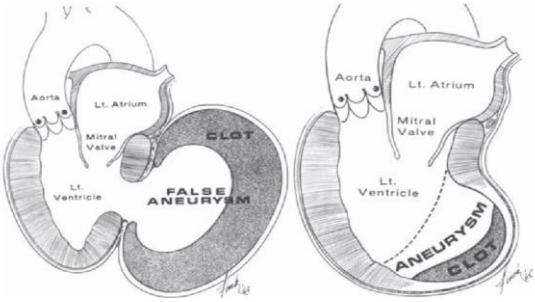

Figure 8: difference between pseudoaneurysm and aneurysm of the left ventricle (22)

Narrow base	Wide base
Walls composed of pericardium and thrombus	Walls composed of myocardium
High risk of rupture	Low risk of rupture

Patients with pseudoaneurysms may exhibit a variety of signs and symptoms, none of which are pathognomonic (Table 1). While earlier case series suggested that approximately half of the patients might be asymptomatic at the time of diagnosis, more recent studies indicate that the majority present with congestive heart failure, chest pain, or dyspnea. Some may also develop symptomatic arrhythmias, signs of systemic embolization, or even sudden cardiac death. Most patients are male and display electrocardiographic abnormalities (e.g., ST-segment changes) and radiographic findings (e.g., mass-like protuberance on X-ray or cardiomegaly) [23].

The diagnosis requires a high degree of suspicion and often necessitates the use of multiple complementary imaging tools, including coronary angiography, ventriculography, two-dimensional transthoracic echocardiography, transesophageal echocardiography, cardiac computed tomography, and magnetic resonance imaging. Pseudoaneurysms are typically characterized by a narrow neck and the absence of normal structural elements found in an intact cardiac wall [24]

Although left ventricular (LV) aneurysm is commonly a delayed complication of myocardial infarction, it is associated with an increased risk of angina pectoris, partly due to elevated LV end-diastolic pressure, thrombus formation, worsening heart failure, and the occurrence of severe ventricular arrhythmias. The aneurysm consists of a thin, scarred, or fibrotic myocardial wall, most often affecting the anterior or apical walls of the LV. The primary pathogenesis is total thrombotic occlusion of the left anterior descending artery, although involvement of the inferior or basal walls linked to right coronary artery occlusion can also be observed. In most cases, management is conservative [25].

1.Free Wall Rupture

The rupture of the free wall is the most commonly reported mechanical complication following acute myocardial infarction (AMI), although its true incidence remains unknown due to its frequent presentation as out-of-hospital sudden cardiac death and the lack of systematic autopsies. The incidence has declined thanks to rapid reperfusion therapies for ST-elevation myocardial infarction (STEMI); however, initial fibrinolysis trials demonstrated an increased risk of rupture with delayed reperfusion due to intramyocardial hemorrhage and dissections [26].

This complication should be suspected in any patient presenting with hemodynamic instability or collapse after an AMI, particularly in cases of delayed or ineffective reperfusion. Clinical signs include jugular vein distension, paradoxical pulse, muffled heart sounds, and occasionally electromechanical dissociation. Although rapidly fatal, rupture can sometimes be confirmed via bedside echocardiography, necessitating emergency surgical intervention. However, in-hospital mortality rates exceed 35%.

In cases of circulatory collapse, ECMO support can provide temporary stabilization to allow for surgical repair, although tamponade may sometimes limit ECMO blood flow. While surgical techniques for managing free wall rupture continue to evolve, primary repair using a patch to cover the defect and, when feasible, sutureless repair employing a patch with glue or a collagen sponge patch may be utilized in a small subset of patients as a complementary therapeutic option [27].

5. Management of mechanical complications of acute coronary syndrome.

Management of patients with mechanical complications of myocardial infarction requires a multidisciplinary approach in a cardiac intensive care unit (CICU). Multi-organ involvement is common and necessitates the collaboration of various specialists, including physicians, nurses, cardiologists, and surgeons [28].

Shared decision-making, led by an intensivist, enhances clinical outcomes and improves patient and family satisfaction. Severe complications often require urgent surgical intervention and prompt assessment by a specialized team [29].

Guidelines from the American College of Cardiology Foundation/American Heart Association and the European Society of Cardiology recommend rapid surgical intervention for patients with hemodynamic instability. However, mortality associated with these emergency procedures remains high, ranging from 20% to 87%, depending on the nature of the mechanical complications. Given the limited experience with percutaneous therapies for these complications, the selection and timing of the intervention should be discussed in consultation with the cardiology or intensive care team [30,31]

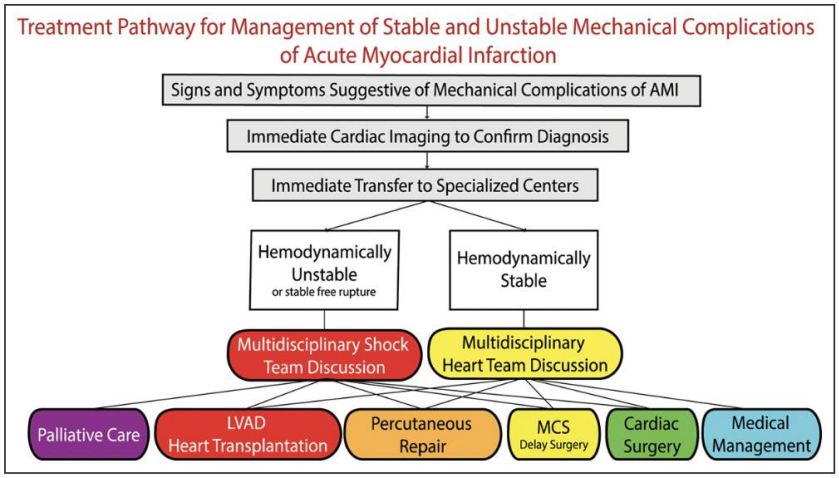

Figure 9: Treatment pathway for management of stable and unstable mechanical complications of acute myocardial infarction [30, 31,32]. AMI indicates acute myocardial infarction; LVAD, left ventricular assist device; and MCS, mechanical circulatory support.

Management of mechanical complications of acute myocardial infarction involves distinct pathways for stable and unstable patients. For unstable cases, early consultation with a multidisciplinary Shock Team is recommended to guide immediate medical management and assess eligibility for surgical or interventional treatment prior to interhospital transfer. In situations where interhospital transfer poses significant risks, alternative on-site therapies and transfer strategies should be explored for non-surgical candidates. These decisions must be made in close collaboration with the Shock Team, considering the expertise available at the current facility and the logistical characteristics of the regional healthcare system (32).

Despite advances in revascularization and the organization of care for STEMI, the incidence of mechanical complications remains stable, likely due to the increasing prevalence of cardiovascular risk factors and the aging population.

Conclusion :

Mechanical complications of ACS are rare but serious events that complicate patients' prognosis. Early detection by echocardiography or cardiac MRI, followed by surgical management, remains crucial to improving survival. Advances in early revascularization, notably coronary angioplasty, have helped to reduce the incidence of these complications, but optimal treatment of advanced cases relies on an experienced multidisciplinary team.

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