AN EMERGENCY MEDICINE REVIEW OF THE MECHANICAL, INFLAMMATORY, AND EMBOLIC COMPLICATIONS OF MYOCARDIAL INFARCTION

Abstract

Introduction:

Despite the declining incidence of coronary heart disease (CHD) in the United States, acute myocardial infarction (AMI) remains a significant clinical issue. Many patients still require emergency department (ED) management due to mechanical, inflammatory, and embolic complications.

Objective:

This narrative review aims to provide a summary of the current evidence on the evaluation and management of post-myocardial infarction mechanical, inflammatory, and embolic complications in emergency medicine.

Discussion:

Although the 30-day mortality rate following AMI has decreased in recent years, it remains high at 7.8% due to various subacute complications that develop over weeks. Mechanical complications, such as ventricular free wall rupture, ventricular septal rupture, mitral valve regurgitation, and left ventricular aneurysm formation, pose significant morbidity risks. Other complications include ischemic stroke, heart failure, renal failure, and cardiac dysrhythmias. This review outlines several guiding principles for managing these complications, emphasizing the importance of understanding their nature and adopting appropriate management approaches to optimize patient care.

Conclusions:

Mechanical, inflammatory, and embolic complications resulting from AMI can lead to substantial morbidity and mortality. Physicians should promptly diagnose these conditions while considering other potential diseases. In addition to understanding the natural progression of the disease and conducting a focused physical examination, an electrocardiogram and bedside echocardiogram can provide quick, noninvasive assessments of the underlying pathophysiology. The management approach may vary based on the specific presentation and etiology, but close consultation with cardiology and cardiac surgery specialists is strongly recommended.

1. INTRODUCTION

Atherosclerotic cardiovascular disease is the leading global cause of mortality [1]. Although the incidence of coronary heart disease (CHD) has declined in the United States, studies have shown that the occurrence of acute myocardial infarction (AMI) has not decreased uniformly, with an estimated rate of 208 cases per 100,000 person-years, of which approximately 22% are ST-segment elevation myocardial infarction (STEMI) [2]. Despite improvements in the 30-day mortality rate following AMI over the past two decades, it remains significantly high at 7.8% [2]. AMI complications encompass a diverse range of mechanical, embolic, ischemic, dysrhythmic, and inflammatory processes that evolve over several weeks. These complications are associated with increased morbidity and mortality [3,4]. The widespread adoption of percutaneous coronary intervention (PCI) as the preferred treatment for acute STEMI has significantly reduced the incidence of these complications to less than 1% [3,4]. Notably, the incidence of ventricular free wall rupture (VFWR) is 0.52%, papillary muscle rupture (PMR) is 0.26%, and ventricular septal rupture (VSR) is 0.17% [3,4]. Acute mitral valve regurgitation (MR) following AMI is most commonly attributed to left ventricular dilation or papillary muscle rupture and is associated with a 30-day mortality rate of up to 24% [5]. Dysrhythmias, particularly atrial fibrillation, ventricular fibrillation, and ventricular tachycardia, affect nearly 21% of patients with AMI [6]. Ischemic complications can impact the kidneys, leading to renal failure, the brain through embolic strokes resulting from dysrhythmias, and the heart with the development of left ventricular (LV) aneurysms [7-9]. Ischemic complications may also involve stent thrombosis and secondary heart failure with subsequent exacerbations [10,11].

2. METHODS

For this narrative review, a comprehensive search was conducted on PubMed and Google Scholar using the keywords "myocardial infarction" OR "acute coronary syndrome" OR "acute coronary syndromes" AND "complications" AND "emergency." The inclusion criteria comprised case reports, case series, retrospective and prospective studies, systematic reviews and meta-analyses, clinical guidelines, and other narrative reviews. The search was limited to English-language publications. Initially, over 600 articles were identified, and through consensus among the authors, relevant articles were selected for inclusion in the review, with a focus on emergency medicine-related literature and guidelines. A total of 177 resources were ultimately included. As a narrative review, individual study data were not pooled.

3. RESULTS AND DISCUSSION

3.1 Ventricular free wall rupture

3.1.1 Etiology

Ventricular free wall rupture (VFWR) occurs in approximately 0.5-2.7% of patients following AMI and contributes to 20-30% of AMI-related deaths [12-16]. The incidence of late VFWR occurring at least 1 day after MI has decreased with the advent of percutaneous coronary intervention (PCI) and thrombolytic agents, although this may be underestimated due to decreased autopsy rates [17]. VFWR typically presents within the first week after an AMI, with subacute or late-occurring VFWRs accounting for up to one-third of cases [12,13,18,19,20]. The majority (90%) of VFWR cases present within the first 2 weeks, although rupture has been reported up to a 1-month post-AMI [20-23]. Left VFWR accounts for 1.7% of patients in cardiogenic shock [18]. Early VFWR, occurring within the first 24 hours, is characterized by full-thickness rupture associated with a small myocardial tear, which may be temporarily covered by a clot or late-developing pericardial adhesions. Subacute or late-occurring VFWRs,

on the other hand, present 1-3 days after an AMI due to a slow erosion of the border zone between healthy and infarcted myocardium, progressing to full-thickness rupture [4].

VFWRs, similar to ventricular septal rupture (VSR), can be classified as simple or complex, and their location may involve the anterior or lateral/posterior LV wall [13,18,20,24]. Simple VFWRs are direct through-and-through defects, while complex VFWRs exhibit serpiginous dissection tracts extending from the primary tear [24]. Although the lateral and posterior wall AMIs are more prone to free wall rupture, they are less prevalent due to a higher proportion of anterior wall AMIs [25]. Subacute VFWRs are predominantly associated with inferior infarctions [19,26]. The infarcted coronary distribution leading to VFWRs has been reported as follows: circumflex coronary artery (LCX) in 40% of cases, left anterior descending artery (LAD) in 42% of cases, and right coronary artery (RCA) in 18% of cases [27].

3.1.2 Risk factors

Risk factors for VFWRs include female gender, age above 55 (typically 65-70), hypertension, large infarct size, single vessel disease (often complete occlusion), transmural infarction, pseudoaneurysm formation, and delayed or incomplete revascularization [12,28-34]. Patients without a prior history of angina who experience their first AMI are more likely to develop VFWR, suggesting limited development of collateral coronary blood supply [35-38].

3.1.3. Clinical Presentation And Diagnosis

Timely diagnosis and management are crucial for the survival of patients with ventricular free wall rupture (VFWR), necessitating a high index of suspicion in the emergency setting. The clinical presentation of VFWR can vary. Some patients may present with prolonged episodes of angina lasting for 6 hours or more, while others may experience shorter anginal episodes lasting 30-60 minutes in the days leading up to the rupture. Continued ischemia can trigger VFWR as the initial infarction extends transmurally. Physical exertion, such as persistent coughing, retching, or excessive straining during defecation, has been implicated as a contributing factor. Patients may also present with syncope, dyspnea, or hypotension, indicating the presence of VFWR and subsequent pericardial tamponade. Hemodynamic stability depends on the amount and rate of pericardial bleeding, as well as the presence of a clot sealing any pericardial leaks. Rapid accumulation of pericardial blood can lead to sudden electromechanical dissociation and death. VFWR patients may present in critical condition, showing signs of tamponade, hypotension with associated hemorrhage, or evidence of low cardiac output. Physical examination findings may include jugular venous distension, pulsus paradoxus, hypotension, and diminished heart sounds.

The electrocardiogram (ECG) has limited specificity in diagnosing VFWR but may reveal low voltages throughout or the presence of electrical alternans. Common cardiac rhythms found in VFWR include junctional rhythms, asystole, pulseless electrical activity, sinus bradycardia, complete heart block, or idioventricular rhythms. Other ECG findings may include persistent ST-segment elevation (≥0.3 mV) and pseudo normalization of pre-existing negative precordial T waves up to 3 days post-AMI. ST-segment elevation in leads other than the infarction-related leads can serve as a sensitive marker for an impending free wall rupture. It is important to note that these findings can vary depending on the location of the infarction. VFWR patients may exhibit a reduced prevalence of intraventricular

conduction delay, although the development of new conduction delays may indicate an increased risk of rupture.

Echocardiography plays a vital role in the diagnosis of VFWR, demonstrating the presence of pericardial effusion and signs of cardiac tamponade. These signs include diastolic right ventricular collapse (high specificity), systolic right atrial collapse (earliest sign), a plethoric inferior vena cava with minimal respiratory variation (high sensitivity), and exaggerated respiratory cycle changes in mitral and tricuspid valve inflow velocities as a surrogate for pulsus paradoxus. Increased echogenicity within the pericardium suggests the presence of a developing hematoma or fibrin clot. Chest radiography has limited diagnostic value for VFWR and associated pericardial effusion but may help assess other potential causes of the patient's presentation. Computed tomography angiography, while not routinely necessary for diagnosis, can aid in surgical planning, determine the extent of rupture, and exclude aortic dissection in stable patients.

3.1.4 Management

The management of VFWR focuses on hemodynamic resuscitation and emergent surgical intervention. Early consultation with the cardiothoracic surgical team and prompt mobilization of the operating room is crucial. In hemodynamically unstable patients, intravascular resuscitation with intravenous crystalloids or blood products and hemodynamic support with vasopressors and/or inotropes should be initiated, without delaying definitive treatment. Although pericardiocentesis (PC) can provide rapid improvement in hemodynamics by relieving tamponade, its use in VFWR remains controversial. PC may lead to an increase in blood pressure and tension on the left ventricular myocardium, potentially converting a small tear into a full-thickness VFWR. Therefore, PC should be reserved for patients in extremis who are refractory to supportive therapy and require it as a temporizing measure en route to the operating room.

3.1.5 Prognosis

The prognosis for VFWR remains poor, with a mortality rate ranging from 75% to 94%. However, patients with subacute VFWRs are less likely to experience rapid hemodynamic deterioration. In prospective studies and small series, successful surgical repair has been associated with survival rates ranging from 48.5% to 76%. Long-term survival has been observed in a significant proportion of patients who undergo successful surgical management.

3.2 Ventricular Septal Rupture (VSR)

3.2.1 Causes

VSR, a condition characterized by a defect in the intraventricular septum, is primarily caused by ischemic necrosis following an acute myocardial infarction (AMI). It accounts for approximately 4.6% of cases of cardiogenic shock after an AMI. The incidence of VSR has decreased to around 0.17% with the advent of thrombolysis and percutaneous coronary intervention. VSR can be classified as simple or complex, depending on the nature of the septal defect. Simple VSRs are characterized by a single, slit-like defect in the intraventricular septum, while complex VSRs involve multiple channels connecting the ventricles, formed after the rupture of the septum. The location of the VSR can provide insight into the subtype, with complex forms associated with posteroinferior infarctions and simple forms more commonly seen following anterior infarctions.

3.2.2 Risk Factors

The risk factors for VSR and subsequent mortality are similar to those for VFWR. These include hypertension, elevated body mass index, anterior wall AMI, increased age, female gender, first AMI, single-vessel occlusion, and absence of smoking history. Historically, VSR is most commonly associated with ischemia of the anterior or anterolateral distributions supplied by the

left anterior descending artery (LAD), occurring in 60% of cases. Occlusion of the dominant right coronary artery (RCA) or left circumflex artery (LCX) accounts for VSR formation in up to 40% of cases.

3.2.3 Clinical Presentation and Diagnosis

Most patients with VSR present within the first week following an AMI, with a median time to presentation of 16 hours. The onset of VSR is characterized by recurrent angina followed by shortness of breath due to the creation of a left-to-right shunt through the septal defect. This leads to acute pulmonary edema and right-sided heart failure, resulting in rapid decompensation into cardiogenic shock. Physical examination findings may include a palpable parasternal thrill, a new harsh pan-systolic murmur radiating to the apex and base, and other signs of right-sided heart failure. STEMI is commonly observed in VSR patients, with anterior STEMI being more prevalent than inferior STEMI. Echocardiography, including transthoracic (TTE) or transesophageal (TEE) echocardiography with color-flow Doppler, is the preferred imaging modality for early diagnosis and guidance of therapy. TTE can reveal the size and location of the VSR, assess the magnitude of the left-to-right shunt, evaluate the biventricular function, and detect any associated complications. TEE is considered when TTE is inconclusive but there is a high suspicion of VSR.

3.2.4 Management

Consultation with interventional cardiology and cardiac surgery is crucial for the management of VSR. Early closure of the defect through surgical or percutaneous intervention improves outcomes, even in hemodynamically stable patients. Patients presenting with VSR often require non-invasive ventilation or definitive airway management due to cardiogenic shock and pulmonary edema. Hemodynamic optimization should be performed before intubation, if possible, as these patients are at high risk for peri-intubation mortality. Treatment strategies depend on the patient's blood pressure and perfusion. Hypertensive patients may benefit from afterload reduction through pharmacologic means, such as intravenous nitrates or phosphodiesterase-3 inhibitors. Inotropic support with medications like milrinone or dobutamine may be necessary. Hypotensive patients may require vasopressors like norepinephrine to improve perfusion. Non-pharmacologic measures for afterload reduction include intra-aortic balloon pump (IABP) placement, Impella placement, or veno-arterial extracorporeal membrane oxygenation (ECMO). Hemodynamic optimization should not delay definitive treatment through percutaneous closure or surgery.

3.2.5 Prognosis

The 30-day mortality rate for patients with VSR and concomitant cardiogenic shock is high, approaching 87%. Untreated VSR is fatal, highlighting the importance of operative repair. Surgical repair of VSR is associated with a mortality rate of approximately 43%, but long-term survival is favorable for operative survivors, with a 5-year cumulative survival rate of 57%. Early referral for surgery or percutaneous closure is essential.

3.3.2 Risk Factors

Acute mitral regurgitation (MR) secondary to cardiogenic shock following an acute myocardial infarction (AMI) is associated with several risk factors. These include right coronary artery (RCA) lesions with right dominant circulation, preexisting coronary artery disease, and diabetes. Other risk factors for the development of acute MR include female gender, increased age, and the presence of cerebrovascular disease.

3.3.3 Clinical Presentation and Diagnosis

Acute MR typically occurs within 2-7 days after an AMI, with a median time to onset of 13 hours. The clinical presentation varies depending on the degree of MR, with fulminant MR following papillary muscle rupture characterized by acute pulmonary edema, hypotension, and cardiogenic shock. Physical examination findings are often nonspecific, and a significant number of patients with moderately severe to severe MR may not have a detectable murmur. However, a soft pan systolic murmur heard loudest at the cardiac apex, with a diastolic component and radiation to the left axilla, may suggest MR. However, in patients with impaired left ventricular systolic function or elevated left atrial pressure, the characteristic murmur may be absent. Electrocardiography (ECG) may show tachycardia or confirm the presence of a posterior or inferior AMI. Chest radiography may reveal pulmonary edema with right upper lobe predominance, indicating increased flow in the right superior pulmonary vein. Echocardiography with color-flow Doppler is the preferred imaging modality for the initial diagnosis of MR. Transthoracic echocardiography (TTE) can provide valuable information about the size and direction of the regurgitant jet, visualize flail leaflets and evidence of mal coaptation, and evaluate biventricular function. In cases where TTE is inconclusive, transesophageal echocardiography (TEE) may be necessary, especially if there is a high suspicion of MR not being diagnosed by TTE.

3.3.4 Management

Prompt diagnosis and emergent surgical correction of MR are crucial for improving survival. Immediate stabilization involves managing pulmonary edema, which may require non-invasive ventilation (NIV) or endotracheal intubation. Medical management aims to increase forward flow from the left ventricle and reduce MR. Nitroprusside or nitroglycerin can be used to provide vasodilation and afterload reduction, while inotropic agents like dobutamine can improve contractility and vasodilation. Vasopressors such as norepinephrine may be needed to maintain blood pressure. Hypertensive patients with pulmonary edema may benefit from vasodilator therapy with nitrates. It is important to avoid cardio-depressant drugs like beta-blockers and to maintain ventricular function through the use of catecholamine inotropes like dobutamine. Diuretics and hemofiltration may be employed in patients with acute pulmonary edema and congestion. Mechanical circulatory support with devices like an intra-aortic balloon pump (IABP), Impella, or extracorporeal membrane oxygenation (ECMO) may be considered. The decision for surgical repair or percutaneous intervention depends on the severity of MR and underlying etiology and involves collaboration between surgical specialists and interventional cardiology.

3.3.5 Prognosis

The prognosis for acute MR following an AMI is poor if left untreated. The mortality rate at 24 hours is around 75%, and it increases to 95% at 2 weeks. Operative mortality is approximately 39%, with an in-hospital mortality of 55%.

3.4. Post-myocardial infarction pericarditis (PMIP)

3.4.1. Etiology

Post-myocardial infarction pericarditis (PMIP), also known as postmyocardial infarction syndrome or Dressler's Syndrome (DS), is a condition characterized by inflammation of the pericardium, with or without pericardial effusion, following an acute myocardial infarction (AMI) [115]. PMIP is a subset of the broader post-cardiac injury syndrome (PCIS), which encompasses pericarditis occurring after cardiac procedures like percutaneous coronary intervention (PCI) and postpericardiotomy syndrome following cardiac surgery [116]. The exact cause of PMIP is not fully understood, but it is believed to be triggered by damage to pericardial

mesothelial cells and minor bleeding into the pericardial space, leading to the release of cardiac antigens and subsequent inflammatory and autoimmune responses in susceptible individuals [115,116]. This inflammation can manifest as simple pericarditis or progress to more complex conditions such as cardiac tamponade, pleural effusions, and/or pleuropericarditis [115,116].

PMIP can be categorized into two time-related forms. Early PMIP occurs within the first 2-4 days after an AMI, while late PMIP typically presents after the first week following an AMI [117]. In the past, early PMIP was estimated to occur in 10-20% of post-AMI patients [117], but the incidence has decreased to around 6% in the era of thrombolytic therapy [118,119]. Late PMIP, previously known as DS, used to have an incidence of 3-4% [120], but contemporary studies indicate an incidence of less than 1% [117,119].

3.4.2. Risk factors

Several risk factors contribute to the development of PMIP. These include delayed reperfusion, higher levels of cardiac biomarkers, larger infarction size, younger age, anterior ischemia, inferior infarcts with right ventricular involvement, and reduced left ventricular ejection fraction [121-124].

3.4.3. Clinical presentation/diagnosis

Patients with PMIP typically exhibit signs and symptoms similar to those seen in acute pericarditis and/or pericardial effusion [115,116,124]. The common clinical presentation includes centrally located sharp, pleuritic chest pain (in approximately 80% of cases), low-grade fever (50-60%), and dyspnea (50-60%) [116,125]. Clinical evaluation may reveal leukocytosis, elevated inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein (in more than 80% of cases), pericardial effusion (in more than 80% of cases), new pleural effusion with or without pulmonary infiltrates (in approximately 60% of cases), and the presence of a pericardial or pleural rub (in 30-60% of cases) [116,125]. ECG changes are observed in 60-80% of pericarditis cases [115]. However, ST segment elevations associated with PR segment depressions, typical ECG changes in pericarditis, are often masked by post-AMI changes [116,127]. In PMIP, ST-segment elevation with persistently upright T waves or T waves that become upright after being inverted can suggest the condition [128,129]. Chest radiography is typically normal in PMIP but may show new-onset pleural effusion or pulmonary infiltrates and an enlarged cardiac silhouette, particularly in pericardial effusions larger than 200 mL [115,116,127]. Transthoracic echocardiography (TTE) should be performed in patients with PMIP to evaluate the presence of pericardial effusion and signs of tamponade [130,131]. Patients with PMIP and effusions greater than 10 mm are at a significantly higher risk of subacute ventricular free-wall rupture [130,131].

There are no standardized criteria for diagnosing PMIP [115,116]. However, the diagnosis requires the presence of a prior injury to the pericardium, myocardium, or pleura, along with evidence of pleuropericardial inflammation [116].

3.4.4. Management

Although there is limited literature specific to the management of PMIP, treatment principles can be extrapolated from studies on acute pericarditis. In cases of early PMIP, which typically resolves on its own, it is recommended to avoid using non-steroidal anti-inflammatory drugs (NSAIDs) during the initial 7-10 days after an AMI, with the exception of once-daily aspirin for secondary prevention [132]. For patients with severe symptoms requiring analgesia, acetaminophen is the preferred agent, with high-dose aspirin considered as a second-line option [132].

Management of late PMIP is mainly supportive, and medical treatment includes NSAIDs, colchicine, and corticosteroids [115-117]. Aspirin is preferred for patients with PMIP due to the need for antiplatelet therapy and the potential interference of other NSAIDs with myocardial healing and scar formation [115,133-136]. Indomethacin should be avoided as it can decrease coronary blood flow [136]. High-dose aspirin (800 mg orally every 6-8 hours for 7-10 days, followed by gradual tapering of the dose by 800 mg per week for 3 weeks) has demonstrated efficacy in multiple studies [127,137,138]. Ibuprofen, which has shown to increase coronary flow, can be prescribed at a dose of 600 to 800 mg every 6-8 hours, with a tapering of the total daily dose by 400 to 800 mg every week for a treatment period of 3-4 weeks [124]. It is important to provide gastric protection, commonly with a proton pump inhibitor, to all patients treated with an NSAID [115].

Colchicine (0.5 mg twice daily for patients weighing more than 70 kg and 0.5 mg once daily for patients weighing less than 70 kg) has been shown to effectively relieve pain in patients with acute pericarditis and prevent recurrences, and it may have a role in PMIP prevention [138-140]. Multiple guidelines recommend treatment with colchicine [115,141,142]. However, colchicine should be avoided in patients at risk of bone marrow suppression, as well as those with liver disease, gastrointestinal motility disorders, and renal insufficiency [115]. Low-dose corticosteroid therapy (e.g., prednisone 0.2-0.5 mg/kg/day for four weeks followed by a taper) has demonstrated effectiveness in patients who are contraindicated for aspirin/NSAIDs or who have not responded to conservative therapy [115,116,143]. If steroids are used, dosages must be tapered very slowly to prevent PMIP recurrence [138]. In cases of PMIP complicated by cardiac tamponade, pericardiocentesis is indicated [115,117,124].

3.4.5. Prognosis

The prognosis for most patients with PMIP is favorable, with a recurrence rate of approximately 10-15% [116]. Hospitalization is not necessary for all PMIP patients, but those with high-risk features should be admitted [137,144]. High-risk features include leukocytosis, fever (temperature above 38 °C), immunosuppressed state, subacute symptom development over days to weeks, concurrent use of oral anticoagulants, elevated troponin levels, large effusion or tamponade, and failure of aspirin or NSAID therapy [115,144].

3.5. Left ventricular aneurysm (LVA)

3.5.1. Etiology

A left ventricular aneurysm (LVA) can occur as a complication of an acute myocardial infarction (AMI) and is characterized by a large area of abnormal left ventricular akinesia or dyskinesia that leads to a reduction in left ventricular ejection fraction [145-147]. LVAs typically have a well-defined, thin, scarred, and fibrotic wall containing necrotic myocardium, resulting from the healing process following a transmural AMI [148]. It is important to differentiate LVAs from left ventricular pseudoaneurysms, which are characterized by a ventricular free wall rupture contained by the surrounding pericardium [39]. The exact mechanism of LVA formation is not fully understood, but it involves transmural infarction with poor collateral blood supply or incomplete coronary reperfusion, followed by increased wall stress during the month following an AMI [145]. This increased wall stress may result from preserved contractility of adjacent myocardium, ventricular dilation, decreased wall thickness, or a combination of these factors [145,149].

Approximately 85% of LVAs are located in the anterolateral region, corresponding to the territory supplied by the left anterior descending (LAD) artery, while 5-10% are posteriorly located in the distribution of the right coronary artery (RCA), and the remaining LVAs arise

from the lateral myocardium supplied by the obtuse marginal arteries [147,150]. Historically, LVA was believed to develop in up to 35% of patients following an AMI [151], but current literature suggests an incidence between 8 and 15%, likely due to contemporary reperfusion strategies [149,152,153].

3.5.2. Risk factors

Risk factors for LVA formation include female gender, absence of previous angina, single-vessel disease, total occlusion of the LAD artery, hypertension, and incomplete reperfusion [149,154].

3.5.3. Clinical presentation/diagnosis

The clinical presentation of LVA can vary and is often nonspecific. Small to moderate-sized aneurysms are often asymptomatic, while large LVAs present with persistent dyspnea and signs of heart failure [145]. The paradoxical movement of the LVA during systole leads to decreased cardiac output due to systolic and diastolic dysfunction [145]. A portion of the left ventricular volume is retained in the poorly contractile LVA instead of being ejected through the aortic valve, while the fibrotic wall stiffens, impairing diastolic relaxation [155-157]. This increased myocardial tension and wall stiffness result in increased oxygen demand, which can lead to further myocardial ischemia. Myocardial ischemia and increased stretch contribute to enhanced automaticity and increased ventricular activity [158]. Ventricular dysrhythmias can occur in up to 44% of patients, with clinical manifestations ranging from palpitations to syncope and sudden cardiac death [159-161]. Functional mitral valve dysfunction is commonly observed due to distorted left ventricular geometry and dyskinetic movement [145]. Although rare, an LVA can enlarge over time and rupture. Over half of the patients with LVAs have a mural thrombus on autopsy or surgery, resulting from flow stasis within the aneurysmal cavity and the procoagulant nature of the fibrotic tissue [163,164]. Systemic embolization occurs in 13.7% of these patients [165]. Physical examination may reveal an apical systolic thrust or a double impulse. A third or fourth heart sound may be present, and there may be an apical pan systolic murmur in the presence of concomitant mitral regurgitation. Electrocardiography (ECG) typically shows persistent ST segment elevations, although this finding does not necessarily indicate the presence of an LVA [166]. The frequency of ST elevations in patients with known LVA ranges from 84 to 100% [167,168]. However, the presence of ST elevations on ECG has limited specificity, as post-AMI ST elevations can also be caused by ventricular hypertrophy, left axis deviation, scarring, or left bundle branch block [167].

Chest radiography may raise suspicion of an LVA, but it is not highly sensitive for evaluating a suspected LVA [169]. Echocardiography is the preferred imaging modality and typically shows a dyskinetic left ventricular wall with diastolic deformity [170]. The presence of a mural thrombus and left ventricular wall calcification may suggest the presence of an LVA. Transthoracic echocardiography (TTE) can also assess associated mitral valve dysfunction and differentiate between a true aneurysm and a pseudoaneurysm by detecting any discontinuity in the myocardium, indicative of myocardial perforation and pseudoaneurysm formation [171]. If echocardiography is inconclusive, cardiac computed tomography angiography (CTA) can confirm the diagnosis and help differentiate between a true aneurysm and a pseudoaneurysm [172].

3.5.4. Management

The management of an LVA involves medical therapy for associated complications and consideration of aneurysmectomy. Asymptomatic small to moderately-sized LVAs can be managed medically in consultation with cardiology and cardiac surgery. However, the role of medical management in symptomatic patients or those with large asymptomatic aneurysms is

uncertain [145]. Surgical intervention may be indicated in cases of intractable ventricular dysrhythmias, severe angina, heart failure unresponsive to medical treatment, and systemic embolization in patients who cannot take oral anticoagulants [132,145]. Symptomatic patients may require afterload reduction for left ventricular enlargement, anti-ischemic medications for angina, and parenteral anticoagulation if a thrombus is present within the aneurysm or left ventricle. Preferred agents for parenteral anticoagulation include unfractionated heparin or low-molecular-weight heparin, which should be continued until effective anticoagulation with warfarin is achieved [173]. Although isolated case reports have shown efficacy with off-label use of direct oral anticoagulants in this population, further studies are needed before adopting this practice [174]. Collaboration with cardiology and cardiac surgery specialists can help guide therapeutic management and consider surgical repair in these patients.

3.5.5. Prognosis

The prognosis and natural history of LVAs have improved in the contemporary era, with an expected 5-year survival rate of up to 90% for medically managed small to moderately-sized LVAs [175]. The 30-day mortality following surgical LVA repair ranges from 2 to 8%, and 5-year survival rates range from 73 to 90% [145].

3.6. Additional complications

There are various subacute complications that can arise following an acute myocardial infarction (AMI), including acute ischemic stroke, acute renal failure, congestive heart failure, and cardiac dysrhythmias. While the presentation, evaluation, and management of these conditions are similar to the general population, there are notable differences in their occurrence in patients after an AMI.

The rate of ischemic stroke is significantly increased in the subacute post-AMI period, with approximately 12.2 ischemic strokes occurring per 1000 AMIs at 30 days. This represents a substantial 44-fold increase compared to the general population. Acute heart failure following AMI has shown a decrease in incidence over time but remains associated with poor survival, with a 1-year mortality rate of 45.5%.

Sudden death secondary to sustained ventricular dysrhythmias is a significant risk in the post-AMI period, accounting for approximately 50% of all deaths. Atrial fibrillation is the most common sustained cardiac dysrhythmia, with an incidence ranging from 6% to 21%. Acute kidney injury (AKI) is a well-known complication of AMI, affecting up to 19.4% of patients, with varying degrees of severity.

In patients who undergo percutaneous coronary intervention (PCI), stent thrombosis is a relatively uncommon but serious complication, occurring in 1.4% of patients within 30 days of stent placement and carrying a high mortality rate of 20-45%.

In conclusion, despite advancements in reperfusion therapy and medical management, the 30-day mortality rate after AMI remains significantly elevated due to a range of acute and subacute complications. These complications include mechanical, embolic, ischemic, dysrhythmic, and inflammatory processes that develop over several weeks following an AMI. Various presentations such as ventricular free wall rupture, ventricular septal rupture, acute mitral valve dysfunction, left ventricular aneurysm, and post-myocardial infarction pericarditis can occur. Effective management requires a thorough understanding of the underlying pathophysiology, and close consultation with cardiology and cardiac surgery, when necessary, is recommended.

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