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Review

Mechanical, inflammatory, and embolic complications of myocardial infarction: An emergency medicine review

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ABSTRACT

Introduction: Despite the declining incidence of coronary heart disease (CHD) in the United States, acute myocardial infarction (AMI) remains an important clinical entity, with many patients requiring emergency department (ED) management for mechanical, inflammatory, and embolic complications.
Objective: This narrative review provides an evidence-based summary of the current data for the emergency medicine evaluation and management of post myocardial infarction mechanical, inflammatory, and embolic complications.
Discussion: While 30-day mortality rate after AMI has decreased in the past two decades, it remains significantly elevated at 7.8%, owing to a wide variety of subacute complications evolving over weeks. Mechanical complications such as ventricular free wall rupture, ventricular septal rupture, mitral valve regurgitation, and formation of left ventricular aneurysms carry significant morbidity. Additional complications include ischemic stroke, heart failure, renal failure, and cardiac dysrhythmias. This review provides several guiding principles for management of these complications. Understanding these complications and an approach to the management of various complications is essential to optimizing patient care.
Conclusions: Mechanical, inflammatory, and embolic complications of AMI can result in significant morbidity and mortality. Physicians must rapidly diagnose these conditions while evaluating for other diseases. In addition to understanding the natural progression of disease and performing a focused physical examination, an electrocardiogram and bedside echocardiogram provide quick, noninvasive determinations of the underlying pathophysiology. Management varies by presentation and etiology, but close consultation with cardiology and cardiac surgery is recommended.

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1. Introduction

Atherosclerotic cardiovascular disease is the leading cause of death around the world [1]. Despite the declining incidence of coronary heart disease (CHD) in the United States over the last few decades many, but not all, observational studies have found no reduction in the incidence of acute myocardial infarction (AMI), accounting for 208 cases per 100,000 person-years, with roughly 22% representing ST-segment elevation myocardial infarction (STEMI) [2]. While 30-day mortality rate after AMI has decreased in the past two decades, it remains significantly elevated at 7.8% [2].

Complications of AMI include a wide variety of mechanical, embolic, ischemic, dysrhythmic, and inflammatory processes evolving over weeks. These complications are associated with significantly increased morbidity and mortality [3,4]. Following the widespread adoption of interventional percutaneous coronary intervention (PCI) as the preferred treatment for acute STEMI, the incidence of these complications has been reduced significantly to under 1% [3,4]. This includes ventricular free wall rupture (VFWR) with an incidence of 0.52%, papillary muscle rupture with an incidence of 0.26%, and ventricular septal rupture (VSR) with a 0.17% incidence [3,4]. Acute mitral valve regurgitation (MR) after AMI is most commonly secondary to left ventricular dilatation or papillary muscle rupture (PMR) and is associated with a mortality rate of up to 24% at 30 days [5]. Dysrhythmias, most commonly atrial fibrillation, ventricular fibrillation, and ventricular tachycardia, affect nearly 21% of this population [6]. Ischemic complications can affect the kidneys resulting in renal failure, the brain in the form of embolic strokes secondary to dysrhythmias, and the heart with the formation of left ventricular (LV) aneurysm [7-9]. Ischemic complications may also include stent thrombosis and secondary heart failure with subsequent exacerbations [10,11].

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2. Methods

Authors searched PubMed and Google Scholar for articles using the keywords "myocardial infarction" OR "acute coronary syndrome" OR "acute coronary syndromes" AND "complications", AND "emergency" for production of this narrative review. Authors included case reports and series, retrospective and prospective studies, systematic reviews and meta-analyses, clinical guidelines, and other narrative reviews. The literature search was restricted to studies published in English. Initial literature search revealed over 600 articles. Authors reviewed all relevant articles and decided which studies to include for the review by consensus, with focus on emergency medicine-relevant articles, including guidelines. A total of 177 resources were selected for inclusion in this review. As this is a narrative review, authors did not pool individual study data.

3. Discussion

3.1. Ventricular free wall rupture

3.1.1. Etiology

VFWR occurs in 0.5–2.7% of patients following AMI and contributes to 20–30% of AMI-related deaths [12-16]. Following the advent of PCI and thrombolytic agents, the incidence of late VFWR occurring at least 1 day after MI has diminished, although this may be under-reported due to decreased autopsy rates during the same time [17]. Patients with VFWR typically present within the first week after an AMI [12,13,18]. Subacute or late-occurring VFWRs present 1–3 days after an AMI and account for up to one third of VFWRs [19,20]. Altogether, 50% present within the first 5 days after MI, with 90% presenting within the first 2 weeks [20,21]. However, rupture has been reported to occur up to 1 month post-AMI [22,23]. Left VFWR accounted for 1.7% of patients in cardiogenic shock from the SHOCK registry [18]. Early VFWR occurring within the first 24 h is due to full-thickness rupture associated with a small myocardial tear [4]. This myocardial tear may temporarily become covered by clot or late-developing pericardial adhesions [4]. In contrast, subacute or late-occurring VFWRs present 1–3 days after an AMI due to a slow erosion of the border zone between healthy and infarcted myocardium, progressing to a full-thickness rupture [4].

As with ventricular septal rupture (VSR), VFWRs may be further classified as simple or complex, and can involve the anterior or lateral and posterior LV wall [13,18,20]. Simple VFWRs are direct through-and-through defects, while complex VFWRs are characterized by serpiginous dissection tracts extending from the primary tear of the rupture [24]. The lateral and posterior wall AMIs are theorized to be more prone to free wall rupture but are less prevalent due to the overall higher proportion of anterior wall AMIs [25]. Inferior infarctions are present in the majority of subacute VFWRs [19,26]. The infarcted coronary distribution leading to VFWRs has been reported as the circumflex coronary artery (LCX) in 40% of cases, left anterior descending artery (LAD) in 42% of cases, and the right coronary artery (RCA) in 18% of cases [27].

3.1.2. Risk factors

Risk factors for VFWRs include female gender, age >55 (most commonly 65–70), hypertension, large infarct size, single vessel disease (usually a completely occluded vessel), transmural infarction, pseudoaneurysm formation, and delayed or incomplete revascularization [12,28-34]. VFWRs are more likely to occur following a first AMI in patients without prior history of angina, suggesting that these patients have limited development of a collateral coronary blood supply [35-38].

3.1.3. Clinical presentation and diagnosis

The emergency physician must have a high index of suspicion, as timely diagnosis and management are vital to the survival of this high-risk population. Clinical presentation varies, and patients may present with prolonged episodes of angina lasting for 6 h or more [13,30,39]. Some patients have sentinel anginal episodes lasting >30–60 min in the preceding days [30,39]. In these instances, continued ischemia may trigger VFWR as the initial infarction extends transmurally [40]. Persistent arterial hypertension does not appear to affect the development of VFWRs; however, physical exertion—such as persistent coughing, retching, or excessive straining during defecation—is thought to play a role [4,39]. These patients may also present with syncope, dyspnea, or hypotension after a silent AMI complicated by VFWR and subsequent pericardial tamponade [39]. Hemodynamic stability depends on the amount of accumulated pericardial bleeding, rate of bleeding, and presence of a clot sealing any pericardial leaks [41]. Patients with rapid accumulation of pericardial blood may suffer from sudden electromechanical dissociation and death [39]. VFWR patients may present in extremis, characterized by any combination of tamponade, hypotension with associated hemorrhage, or stigmata of low cardiac output [39,42-44]. Physical examination findings may be consistent with cardiac tamponade, including jugular venous distension, pulsus paradoxus, hypotension, and diminished heart sounds [20,45].

The electrocardiogram (ECG) has a limited specificity for diagnosing VFWR, but may reveal low voltages throughout, or the presence of electrical alternans [46]. The cardiac rhythms found in VFWR most commonly include junctional rhythms, asystole, pulseless electrical activity, sinus bradycardia, complete heart block, or idioventricular rhythms [40]. Other possible ECG findings include persistent ST-segment elevation (>0.3 mV) and pseudonormalization of pre-existing negative precordial T waves up to 3 days post-AMI [40]. Within the literature, ST-segment elevation in leads other than the infarction-related leads is a sensitive marker for an impending free wall rupture [47]. It is important to note, however, that the aforementioned findings vary with the location of infarction. The presence of an intraventricular conduction delay may be less prevalent in patients with VFWR, although the subsequent development of a new conduction delay may portend increased risk for rupture [28,36,40].

Echocardiography will demonstrate a pericardial effusion with signs of cardiac tamponade, including 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 in-flow velocities as a surrogate for pulsus paradoxus [4,48]. Presence of increased echogenicity within the pericardium suggests presence of a hematoma developing into a fibrin clot [25,49]. Chest radiography is poorly diagnostic of free wall rupture and associated pericardial effusion but may be a useful adjunct to assess for other potential etiologies for the patient's presentation [50]. Computed tomography angiography, while not routinely necessary for the diagnosis, may be useful for surgical planning, determining the extent of rupture, and excluding aortic dissection in stable patients [51-53].

3.1.4. Management

Management of VFWR centers on hemodynamic resuscitation and emergent surgical intervention [4,25,54]. Early consultation with the cardiothoracic surgical team and rapid mobilization of the operating room (OR) is essential [4,20]. Intravascular resuscitation with intravenous crystalloids or blood products and hemodynamic support with vasopressors and/or inotropes should be initiated in hemodynamically unstable patients, but this should not delay definitive treatment [20].

While pericardiocentesis (PC) provides rapid improvement in the patient's hemodynamics by relieving the underlying tamponade physiology, emergent PC in these patients remains controversial [55]. By relieving the tamponade, there is a reflex increase in blood pressure and subsequent tension on the LV myocardium, which may convert a small tear to a full-thickness VFWR [4]. As PC can only act in a

temporizing fashion, it should be reserved for those patients in extremis refractory to supportive therapy en route to the OR [20,55].

3.1.5. Prognosis

Prognosis for all VFWR remains poor, with a mortality rate between 75 and 94% [56]. However, patients with subacute VFWRs are less likely to experience rapid hemodynamic deterioration [20]. In a prospective study of 1247 consecutive patients with AMI that included 33 patients with subacute VFWR, 76% (25 of 33) survived their initial surgical repair, with 48.5% (16) of them becoming long-term survivors [32]. This rate of successful surgical management has been demonstrated in several small series as well [57,58].

3.2. Ventricular septal rupture (VSR)

3.2.1. Etiology

VSR, a defect in the intraventricular septum, is typically caused by ischemic necrosis and causes up to 4.6% of cardiogenic shock after an AMI [20,59]. Before the current era of thrombolysis and PCI, VSR occurred in approximately 1–2% of patients with AMI; however, the current incidence is closer to 0.17% [36,60,61]. VSR, much like VFWR, represents a transmural infarction of the myocardium and may be subdivided into simple or complex presentations [4,21]. Simple VSRs consists of an abrupt, slit-like "through-and-through" intraventricular defect occurring at the same level of both ventricles. A complex VSR is characterized by disruption of the intraventricular septum followed by hemorrhage formation and development of a meshwork of channels connecting the ventricles [4]. Location of the VSR may provide an indication as to VSR subtype, with complex forms associated with posteroinferior infarctions and simple forms more commonly seen following anterior infarctions [20].

3.2.2. Risk factors

Risk factors for the development of VSR and subsequent mortality mirror those of VFWR, including hypertension, elevated body mass index, anterior wall AMI, increased age, female gender, first AMI, single vessel occlusion, and absence of smoking history [17,62-65]. Historically, VSR is most commonly associated with ischemia of the anterior or anterolateral distributions supplied by the LAD, occurring in 60% of cases [60,66]. Comparatively, occlusion of the dominant RCA or LCX accounts for VSR formation in up to 40% of cases [20].

3.2.3. Clinical presentation and diagnosis

As shown in the SHOCK trial and further validated by APEX-MI and GUSTO-I, VSR timecourse is variable, but the large majority of patients will present within the first week following an AMI, with a median time to presentation of 16 h [21,67-69]. Patients characteristically present for the first time following an AMI and are clinically stable before experiencing the sudden onset of recurrent angina followed by shortness of breath secondary to the creation of a left-to-right shunt (LRS) through the VSR [4,20]. This LRS typically leads to acute pulmonary edema with right sided heart failure, and patients decompensate rapidly into cardiogenic shock characterized by oliguria (81% of patients) and cold, poorly perfused extremities (83%) [4,20,68]. Physical examination reveals a palpable parasternal thrill in approximately one half of presentations, with a new, harsh pan-systolic murmur radiating to the apex and base best heard at the left lower sternal border occurring in up to 90% of cases [70-73]. In comparison, mitral regurgitation, which may present similarly or occur concomitantly, has a mid-frequency pansystolic murmur best heard at the apex [73]. Other physical examination findings are secondary to increased right-sided flow and subsequent right-sided failure, including jugular venous distention, a left or right S3 gallop, tricuspid regurgitation with cannon A waves, and possibly a loud pulmonic component of the second heart sound [73,74].

STEMI is present in the majority of patients with VSR, and anterior STEMI (60% of patients) is more common than inferior STEMI (40%) [20,68]. There is an associated atrioventricular or infranodal conduction abnormality in 40% of cases [4,20,75]. Chest radiography lacks specificity and sensitivity for diagnosing VSR but may show cardiomegaly, pulmonary congestion, or a pleural effusion [20].

When the emergency clinician suspects VSR, point-of-care transthoracic (TTE) or transesophageal echocardiography (TEE) with color-flow Doppler is the imaging modality of choice for early diagnosis and therapy guidance [76,77]. Echocardiography is able to determine the size and site of VSR, assess the magnitude of the LRS as well as biventricular function, and evaluate for any concurrent papillary muscle rupture or MR [4,20]. Both the sensitivity and specificity of TTE with color-flow Doppler reach 100% within the literature [78,79]. Characteristic findings on TTE include right ventricular dilation, pulmonary hypertension, perforation of the ventricular septum, and demonstration of left-to-right flow across the septum on color-flow Doppler [74]. Magnitude of the LRS can be assessed by comparing flow across the aortic and pulmonic valves [4,80,81]. Unless there is a large territory of infarction or pre-existing dysfunction, TTE reveals a hyperdynamic left ventricle [74]. While TTE remains the most accessible bedside technique, TEE should be considered in those patients in which the TTE is undiagnostic and there remains a high index of suspicion [74].

3.2.4. Management

Due to the significant morbidity and mortality associated with untreated VSR, consultation with interventional cardiology and cardiac surgery is essential, as early closure via surgery or percutaneous intervention improves outcomes, even in the hemodynamically stable patient [20]. Patients presenting with VSR commonly present in extremis secondary to cardiogenic shock and fulminant pulmonary edema, necessitating the use of non-invasive ventilation (NIV) or definitive airway management [4]. However, these patients are at high risk for peri-intubation mortality and should be hemodynamically optimized prior to intubation if possible.

Treatment depends on patient blood pressure and perfusion. Temporizing management for hypertensive patients with VSR includes reducing LRS and increasing LV stroke volume via afterload reduction. Afterload reduction is initially achieved by pharmacologic means with intravenous nitrates, which have the advantage of being rapidly titratable [4,74]. Phosphodiesterase-3 inhibitors, such as milrinone, may be used as an inodilator, providing afterload reduction while simultaneously increasing myocardial contractility [82-84]. However, milrinone is slow in onset with a long half-life and may not be readily available in the emergency department. Dobutamine is another viable option for inodilation [85]. If available, pulmonary vasodilators, such as inhaled nitric oxide, may benefit patients with significant right ventricular dysfunction [86]. However, afterload reduction should be avoided in patients presenting with hypotension [20]. In patients unable to tolerate afterload reduction, inotropic support may be necessary [4]. Hypotensive patients may require vasopressors, such as norepinephrine, to improve peripheral perfusion and ensure adequate coronary blood flow to maintain adequate cardiac output for end-organ perfusion [20,74,82].

Non-pharmacologic measures for afterload reduction include the use of an intra-aortic balloon pump (IABP) which also augments coronary blood flow, Impella placement, and veno-arterial extracorporeal membrane oxygenation (ECMO), which also augments cardiac output [87-91]. IABP decreases afterload, thereby reducing the degree of LRS, and increases cardiac output, systemic pressures, and end-organ perfusion. Hemodynamic optimization should not delay definitive treatment via percutaneous closure device or primary surgical treatment [4,20,74].

3.2.5. Prognosis

Thirty day mortality for patients with VSR approaches 87% in those with VSR and concomitant cardiogenic shock, and VSR is always fatal in patients who do not receive operative repair [68,92]. Though there is a high mortality associated with surgical repair of VSR (~43%), there

are favorable long-term outcomes in operative survivors with a 5-year cumulative survival of 57% [90,93]. Patients should be referred to surgery or percutaneous closure as soon as possible.

3.3. Mitral valve regurgitation (MR)

3.3.1. Etiology

Acute MR remains a prominent complication of AMI, accounting for 8.3% of cardiogenic shock presentations per the SHOCK registry [59,94]. While mild to moderate MR is found in up to 45% of patients following an AMI, it is generally well tolerated, transient, and asymptomatic [5]. However, acute MR with subsequent cardiogenic shock following papillary muscle rupture or LV dilation is a life-threatening diagnosis [95,96]. The underlying pathophysiology of acute MR following an AMI may be categorized as mechanical or functional in nature. Functional MR occurs secondary to LV dilation with concomitant mitral valve annular dilation, or poor leaflet valve coaptation due to papillary muscle dysfunction with resultant segmental wall motion abnormality at the muscle insertion site [97-99]. This functional MR is seen in even slight modification of LV geometry in the presence of regional wall motion abnormalities [100].

Conversely, structural MR may occur due to chordae rupture, papillary muscle dysfunction, or PMR leading to a flail mitral valve leaflet [4,20]. Structural MR is closely tied to coronary anatomy, as the posteromedial papillary muscle has a singular arterial supply via the posterior descending artery which arises from the RCA or the LCX in right and left dominant systems, respectively [20,101,102]. The anterolateral papillary muscle is 6–12 times less likely to suffer rupture, as it receives duel blood supply from the LAD and LCX [20,70].

3.3.2. Risk factors

Patients presenting with cardiogenic shock secondary to acute MR are more likely to have an RCA lesion with right dominant circulation, as well as preexisting coronary artery disease and diabetes [96]. Additional risk factors for development of acute MR include female gender, increased age, and presence of cerebrovascular disease [96].

3.3.3. Clinical presentation and diagnosis

MR most commonly occurs within 2–7 days post-AMI, with a median time to onset of 13 h [4,94]. Clinical presentation varies according to degree of MR, with fulminant MR following PMR characterized by acute pulmonary edema, hypotension, and cardiogenic shock [4]. Physical examination is non-specific and does not identify 50% of patients with moderately severe to severe MR [96]. However, the presence of a soft pansystolic murmur heard loudest at the cardiac apex, with a diastolic component and radiation to the left axilla, may be suggestive of MR [4,20,73]. However, the characteristic murmur may be absent in patients with impaired LV systolic function or elevated left atrial pressure [103].

ECG may show tachycardia, or confirm the presence of a posterior or inferior AMI, while chest radiography may reveal pulmonary edema with right upper lobe predominance suggestive of increased flow in the right superior pulmonary vein [4,104]. Bedside echocardiography with color-flow Doppler is the imaging modality of choice for initial diagnosis of MR, clinical monitoring, and surgical planning [4]. TTE may reveal the size and direction of the regurgitant jet via color-Doppler, while it is possible to visualize flail leaflets and evidence of malcoaptation [103]. Likewise, TTE may help distinguish between VSR and PMR with associated MR, while evaluating biventricular function [4,105]. The presence of MR with normal LV size is highly suggestive of acute MR [103]. While TTE is useful, and often the initial imaging modality, it has a sensitivity between 65 and 85% for diagnosing PMR [106]. Therefore, TEE may be necessary if there remains a high index of suspicion PMR not diagnosed by TTE [20].

3.3.4. Management

Prompt diagnosis and emergent surgical correction of MR are vital to improve survival [4,107]. Immediate stabilization involves management of pulmonary edema, necessitating the use of non-invasive ventilation (NIV) or definitive airway management via endotracheal intubation [4]. The goals of temporizing medical management is to increase forward flow from the left ventricle and decrease the amount of MR. Nitroprusside or nitroglycerin can be used to provide vasodilation and afterload reduction as tolerated by the blood pressure. Inodilators such as dobutamine, are helpful by improving both contractility and vasodilation to improve forward flow. Vasopressors, such as norepinephrine, may be needed to maintain blood pressure despite the adverse effect of increasing afterload. If patients are hypertensive with pulmonary edema, vasodilator therapy with nitrates is recommended [20,108]. Maintaining ventricular function is vital by avoiding cardiodepressant drugs including beta-blockers, as well as augmenting cardiac output via catecholamine inotropes such as dobutamine [4,103]. Diuretics, as well as hemofiltration, may be used in patients with acute pulmonary edema and pulmonary congestion [4]. Similarly to patients with VSR, mechanical circulatory support in the form of IABP, Impella placement, or ECMO may prove useful [89,109-113]. While the decision for primary surgical repair is largely based on the severity of MR and underlying etiology (functional vs. structural), it is important to involve the surgical specialists as well as interventional cardiology [4,20].

3.3.5. Prognosis

Prognosis for acute MR following AMI remains poor, with a mortality rate of 75% at 24 h and 95% at 2 weeks if left untreated [114]. Operative mortality approaches 39%, with an in-hospital mortality of 55% [59,94].

3.4. Post-myocardial infarction pericarditis (PMIP)

3.4.1. Etiology

PMIP, also known as postmyocardial infarction syndrome or Dressler's Syndrome (DS), is a form of pericarditis presenting with or without pericardial effusion after an AMI [115]. However, PMIP represents a small part of the post-cardiac injury syndrome (PCIS), which expands to include post-traumatic pericarditis secondary to iatrogenic causes such as PCI and postpericardiotomy syndrome following cardiac surgery [116]. While the exact pathophysiology of PMIP remains unknown, it is presumed to be triggered by initial insult to pericardial mesothelial cells as well as minor bleeding into the pericardial space, releasing cardiac antigens, stimulating an inflammatory and autoimmune response in predisposed patients [115,116]. This inflammation causes a spectrum of clinical presentation ranging from simple, uncomplicated pericarditis to more complicated disease including cardiac tamponade, pleural effusions, and/or pleuropericarditis [115,116].

Two time-related forms of PMIP are recognized. Early PMIP occurs within the first 2–4 days after an AMI, and late PMIP typically presents after the first week following an AMI [117]. Historically, the incidence of early PMIP was thought to occur in 10–20% of post-AMI patients [117], although this has decreased to an incidence of roughly 6% in the post-thrombolytic era [118,119]. Conversely, late PMIP, formerly known as DS, had an incidence of 3–4% in the past [120], but contemporary studies show an incidence of <1% [117,119].

3.4.2. Risk factors

The risk factors for the development of PMIP include delayed reperfusion, higher cardiac biomarkers, larger infarction size, younger age, anterior location of ischemia, inferior infarcts accompanied by right ventricular involvement, and more frequently reduced left ventricular ejection fraction [121-124].

3.4.3. Clinical presentation/diagnosis

Patients who develop PMIP typically present with signs and symptoms similar to those seen in patients presenting with acute pericarditis and/or pericardial effusion [115,116,124]. Most patients present with sharp, pleuritic chest pain that is centrally located (>80%), 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 (>80%), the presence of a pericardial effusion (>80%), new pleural effusion with or without pulmonary infiltrates (>60%), and evidence of a pericardial or pleural rub (30–60%) [116,125]. ECG changes are present in 60–80% of cases of pericarditis in different series [115]. Elevated troponin levels may reveal myocardial involvement and the presence of myopericarditis [126].

Typical ECG changes seen in pericarditis, including diffuse ST segment elevations in association with PR segment depressions, are often masked by post-AMI changes [116,127]. However, ST segment elevation with persistently upright T waves or T waves that become upright after having been inverted suggest PMIP [128,129]. Chest radiography is typically normal in patients with PMIP but may show new-onset pleural effusion or pulmonary infiltrates and an enlarged cardiac silhouette, present in pericardial effusions >200 mL [115,116,127]. TTE should be performed in patients with PMIP, specifically to evaluate for pericardial effusion, as well as evidence of tamponade [130,131]. Patients with a PMIP and effusions >10 mm are at significantly increased risk of subacute VFWR [130,131].

There are no formalized criteria for diagnosing PMIP [115,116]. However, the diagnosis requires the presence of a prior injury to the pericardium, myocardium, or pleura and evidence of pleuropericardial inflammation (Table 1) [116].

3.4.4. Management

While there is little literature to guide treatment for PMIP, management principles may be extrapolated from studies evaluating acute pericarditis. For patients presenting with early PMIP, which is usually self-limited, the recommended approach is to avoid using NSAIDs for the initial 7–10 days post-AMI, with the exception of once daily aspirin for secondary prevention [132]. For those with severe symptoms requiring analgesia, acetaminophen remains the preferred agent, with high-dose aspirin second line [132].

Treatment for late occurring PMIP is mostly supportive, with medical management including non-steroidal anti-inflammatory drugs (NSAIDs), colchicine, and corticosteroids [115-117]. Aspirin should be preferentially used in patients with PMIP due to the patient's requirement for antiplatelet therapy, as well as the fact that the anti-inflammatory actions of other NSAIDs may interfere with myocardial healing and scar formation [115,133-136]. Indomethacin should be avoided, as it decreases coronary blood flow [136]. High-dose aspirin (800 mg orally every 6–8 h for 7–10 days followed by gradual tapering of the dose by 800 mg per week for 3 weeks) has been shown to be efficacious in multiple studies [127,137,138]. Ibuprofen, which has been shown to increase coronary flow, may be prescribed at a dose of 600 to 800 mg every 6–8 h, with tapering of the total daily dose by 400 to 800 mg every week for a treatment period of 3–4 weeks [124]. Gastric protection, commonly with a proton pump inhibitor, should be provided in all patients treated with an NSAID [115].

Colchicine (0.5 mg BID for patients >70 kg and 0.5 mg once daily for patients <70 kg for 4–6 weeks) has been shown to be effective in relieving pain in patients with acute pericarditis and preventing recurrences, and it may play a role in PMIP prevention [138-140]. Treatment with colchicine is recommended by multiple guidelines [115,141,142]. However, colchicine should be avoided in those patients at risk for bone marrow suppression, as well as those with liver disease, gastrointestinal motility disorders, and renal insufficiency [115]. Low-dose corticosteroid therapy (i.e. prednisone 0.2–0.5 mg/kg/day for four weeks followed by a taper) has been shown to be effective in patients in whom aspirin/NSAIDs are contraindicated, and those who have failed to respond to more conservative therapy [115,116,143]. If steroids are used, dosages must be tapered very slowly to avoid recurrences of PMIP [138]. In the case of PMIP complicated by cardiac tamponade, PC is indicated [115,117,124].

Table 1
Diagnostic features of post-myocardial infarction pericarditis.

Evidence of prior pericardial, myocardial, or pleural injury, plus
1. Pericarditis ± pericardial or pleural effusion
2. Evidence of inflammation without alternative etiology

3.4.5. Prognosis

While the prognosis of PMIP is good for the majority of patients, there remains a recurrence rate of 10–15% [116]. Inpatient admission is not necessary for all patients with PMIP; however, patients with high-risk features should be hospitalized [137,144]. These include patients with leukocytosis, fever (temperature > 38 °C), immunosuppressed state, subacute course (symptoms developing over days to weeks), concurrent oral anticoagulant use, elevated troponin levels, large effusion or tamponade, and aspirin or NSAID failure [115,144].

3.5. Left ventricular aneurysm (LVA)

3.5.1. Etiology

AMI may be complicated by the formation of a LVA, broadly defined as a large area of abnormal left ventricular akinesia or dyskinesia that causes a reduction in LV ejection fraction [145-147]. LVAs characteristically are a well-delineated, thin, scarred, fibrotic wall containing necrotic myocardium secondary to a healing transmural AMI [148]. This is in contrast to a LV pseudoaneurysm, which represents a VFWR contained by the surrounding pericardium [39]. While the mechanism of LVA formation is not completely elucidated, there appears to be a component of transmural infarction with poor collateral blood supply or incomplete coronary reperfusion, followed by a period of increased wall stress in the month following an AMI [145]. This increased wall stress may arise from preserved contractility of adjacent myocardium, ventricular dilation, decreased wall thickness, or a combination thereof [145,149].

Approximately 85% of LVAs are anterolateral in nature, corresponding to the territory perfused by the LAD artery, with 5–10% located posteriorly in the distribution of the RCA, and the remaining LVAs arising from the lateral myocardium supplied by the obtuse marginal arteries [147,150]. Historically LVA was thought to develop in up to 35% of patients following an AMI [151], but current literature reports 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 LAD occlusion, hypertension, and incomplete reperfusion [149,154].

3.5.3. Clinical presentation/diagnosis

Clinical presentation varies and is often nonspecific. Small and moderate-sized aneurysms are often asymptomatic, while large LVAs present with persistent dyspnea and evidence of heart failure [145]. The paradoxical movement of the LVA during systole results in decreased cardiac output related to both systolic and diastolic dysfunction [145]. A portion of the LV volume is retained in the poorly-contractile LVA instead of flowing through the aortic valve while the fibrotic wall stiffens, impairing diastolic relaxation [155-157]. This increased myocardial tension and wall stiffening cause increased oxygen demand, which may lead to further myocardial ischemia. Myocardial ischemia and increased stretch lead to enhanced automaticity and increased

ventricular activity [158]. Symptoms related to ventricular dysrhythmias occur in up to 44%% of patients, with the clinical presentation ranging from palpitations to syncope and sudden cardiac death [159-161]. Some degree of functional mitral valve dysfunction is present due to the distorted LV geometry and dyskinetic movement in these patients [145]. Similarly, an LVA may enlarge over time and rupture, although this is a rare event owing to the dense fibrotic wall [162]. Over half of patients with LVAs have a mural thrombus on autopsy or surgery, due to the stasis of flow in the aneurysmal cavity as well as the procoagulant nature of the fibrotic tissue within the LVA [163,164]. Systemic embolization occurs in 13.7% of these patients [165]. Palpation of the chest may demonstrate an apical systolic thrust or the presence of a double impulse. A third or fourth heart sound may be present as well. There may be an apical pansystolic murmur in the case of concomitant mitral regurgitation.

ECG usually shows persistent ST segment elevations; however, this finding does not necessarily imply the presence of an LVA [166]. The frequency of ST elevations ranges from 84 to 100% in patients with known LVA [167,168]. While the presence of ST elevations is highly sensitive for LVA, specificity is limited, as post-AMI ST elevations may be due to ventricular hypertrophy, left axis deviation, scarring, or left bundle branch block [167].

While the presence of an LVA may be suspected from chest radiography, it is poorly sensitive for evaluation of a suspected LVA [169]. Echocardiography is the imaging modality of choice and will typically show a dyskinetic LV wall with diastolic deformity [170]. Mural thrombus and LV wall calcification may be suggestive of LVA. TTE can also evaluate associated mitral valve dysfunction and differentiate a true aneurysm from pseudoaneurysm by detecting any discontinuity in the myocardium, indicative of myocardial perforation and pseudoaneurysm formation [171]. In cases in which echocardiography is not diagnostic, cardiac CTA may confirm the diagnosis and may be helpful to distinguish between a true and pseudoaneurysm [172].

3.5.4. Management

Management of an LVA focuses on medical therapy for associated complications and consideration of an aneurysmectomy. Asymptomatic small to moderately sized LVAs may be managed medically in consultation with cardiology and cardiac surgery; however, the role for medical management for symptomatic patients or those with asymptomatic large aneurysms is uncertain [145]. Additional surgical indications include intractable ventricular dysrhythmias, severe angina, heart failure unresponsive to medical management, and systemic embolization in patients who cannot take oral anticoagulants [132,145]. Therapy for symptomatic patients includes afterload reduction for those with LV enlargement, anti-ischemic medications for angina, and parenteral anticoagulation if there is thrombus present within the aneurysm or LV. 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 reported efficacy with off-label use of direct oral anticoagulants in this population, further studies are needed before adopting this practice [174]. Cardiology and cardiac surgery consultations may help guide therapeutic management and consideration for surgical repair in these patients.

3.5.5. Prognosis

The prognosis and natural history for patients with LVA in the contemporary era is improved, with an anticipated 5 year survival rate of up to 90% for medically treated small to moderate sized LVAs [175]. The 30-day mortality following surgical LVA repair ranges from 2 to 8%, with 5 year survival ranging from 73 to 90% [145].

3.6. Additional complications

There remains a variety of subacute complications, including acute ischemic stroke, acute renal failure, congestive heart failure, and cardiac dysrhythmias. While the presentation, evaluation, and management of these conditions in patients following AMI are similar to the general population, there are important epidemiological differences. For instance, the rate of ischemic stroke is increased in the subacute post-AMI period, with 12.2 ischemic strokes occurring per 1000 AMIs at 30 days [176]. This represents roughly a 44-fold increase compared to the general population [177]. While the incidence of acute heart failure following AMI has diminished from approximately 16 per 100 person years in 1998 to 14 per 100 person-years in 2010, survival remains poor, with a 1 year mortality rate of 45.5% [178]. Additionally, these patients are at high risk for sudden death secondary to sustained ventricular dysrhythmias, which account for roughly 50% of all deaths in the post-AMI period [179]. Atrial fibrillation is the most common sustained cardiac dysrhythmia, with an incidence of 6–21% [6]. Acute kidney injury (AKI) is a well-described complication of AMI, affecting up to 19.4% of patients, including 7.1% with mild, 7.1% with moderate, and 5.2% with severe AKI [180]. In patients following PCI, stent thrombosis is uncommon, occurring in 1.4% of patients within 30 days of placement, but carries a mortality rate of 20–45% [181,182].

4. Conclusions

Despite improvements in reperfusion therapy and medical management, the 30-day mortality rate after AMI remains significantly elevated at 7.8%, secondary to a variety of both acute and subacute complications [2]. These complications include a wide variety of mechanical, embolic, ischemic, dysrhythmic, and inflammatory processes evolving over weeks after an AMI. Patients may present to the acute care setting with VFWR, VSR, acute mitral valve dysfunction, LVA, and PMIP [3,4]. Dysrhythmias, most commonly atrial fibrillation, as well as ischemic events such as renal failure or stroke affect these patients. In addition to understanding the natural progression of disease and performing a focused physical examination, an ECG and bedside echocardiogram provide quick, noninvasive determinations of the underlying pathophysiology. Management varies by presentation and etiology, but close consultation with cardiology as well as cardiac surgery, when indicated, is recommended.

Conflicts of interest

None.

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