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Mechanical Complications of Acute Myocardial Infarction
A Review

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IMPORTANCE Mechanical complications of acute myocardial infarction include left ventricular free-wall rupture, ventricular septal rupture, papillary muscle rupture, pseudoaneurysm, and true aneurysm. With the introduction of early reperfusion therapies, these complications now occur in fewer than 0.1% of patients following an acute myocardial infarction. However, mortality rates have not decreased in parallel, and mechanical complications remain an important determinant of outcomes after myocardial infarction. Early diagnosis and management are crucial to improving outcomes and require an understanding of the clinical findings that should raise suspicion of mechanical complications and the evolving surgical and percutaneous treatment options.

OBSERVATIONS Mechanical complications most commonly occur within the first week after myocardial infarction. Cardiogenic shock or acute pulmonary edema are frequent presentations. Echocardiography is usually the first test used to identify the type, location, and hemodynamic consequences of the mechanical complication. Hemodynamic stabilization often requires a combination of medical therapy and mechanical circulatory support. Surgery is the definitive treatment, but the optimal timing remains unclear. Percutaneous therapies are emerging as an alternative treatment option for patients at prohibitive surgical risk.

CONCLUSIONS AND RELEVANCE Mechanical complications present with acute and dramatic hemodynamic deterioration requiring rapid stabilization. Heart team involvement is required to determine appropriate management strategies for patients with mechanical complications after acute myocardial infarction.

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Video

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Reperfusion therapies have led to a substantial reduction in the frequency of mechanical complications of acute myocardial infarction (MI).[1,2] Recent studies estimate that following ST-elevation MI, 0.27% to 0.91% of patients develop mechanical complications; papillary muscle rupture (PMR), ventricular free-wall rupture (FWR), and ventricular septal rupture (VSR) are estimated to occur in 0.05% to 0.26% of patients, 0.01% to 0.52% of patients, and 0.17% to 0.21% of patients, respectively.[3,4] However, unfortunately, there has been no significant decrease in associated mortality rates over the past 2 decades, and patients with mechanical complications are more than 4-fold more likely to experience in-hospital mortality than those without mechanical complications.[1,3] Mechanical complications are therefore infrequent but remain an important determinant of outcomes after MI. The purpose of this review is to highlight key clinical and diagnostic findings that may assist in the early diagnosis of mechanical complications and present an update on current management strategies.

FWR

Clinical Features

Three morphologies of FWR were originally described as follows: type 1 rupture is an abrupt tear usually within the first 24 hours of MI, type 2 rupture is a slower tear with localized myocardial erosion, and type 3 rupture is a thin-walled aneurysm perforation, which usually occurs more than 7 days after MI.[5] FWR usually occurs within 7 days after MI,[6] with a mean time to diagnosis of 2.6 days in a 2018 series.[7] Early autopsy studies demonstrated that an abrupt large tear would likely lead to sudden cardiac tamponade, cardiogenic shock, and cardiac arrest, whereas a smaller, more gradual tear may be limited by thrombus formation or a compliant pericardium but with hemodynamic instability and pericardial effusion.[8] These 2 types are also described in the surgical literature as the blowout and oozing types, respectively.[7,9]

In the SHOCK trial registry,[10] there was no significant sex difference in the incidence of FWR, but patients with FWR were less likely to have diabetes or a history of prior MI. It is postulated that the absence of certain cardiovascular risk factors denotes individuals who are less likely to have coronary artery disease and thus less likely to have developed collateral circulations that protect the myocardium in the setting of acute vessel occlusion.

Patients with FWR may present with chest pain, restlessness, hemodynamic compromise, or cardiogenic shock (**Table**). In a 2018 series,[7] more than 80% of patients presented with cardiac tamponade. FWR occurring late in the first week or beyond may also be associated with a history of straining, such as with coughing or vomiting.[11] Examination findings may be significant for a raised jugular venous

Table. Summary of the Main Features of Free-Wall Rupture, Ventricular Septal Rupture, and Papillary Muscle Rupture

Feature	Free-wall rupture	Ventricular septal rupture	Papillary muscle rupture
Timing	Within 7 d post-MI	Within 7 d post-MI	Within 7 d post-MI
Presentation	Chest pain; cardiogenic shock; cardiac arrest	Chest pain; heart failure; cardiogenic shock	Acute pulmonary edema; cardiogenic shock
Echocardiography	Pericardial effusion/tamponade; pericardial clots	Shunt flow across ventricular septum; simple apical defect or extensive irregular inferobasal defect	Ruptured papillary muscle; prolapse/flail leaflets; severe mitral regurgitation; hyperdynamic left ventricle
Nonsurgical management	Pericardiocentesis; IABP/ECMO	Diuretics/inotropes; IABP/ECMO	Diuretics/inotropes/vasodilators; IABP/ECMO
Recommended treatment	Urgent surgery	Urgent surgery	Urgent surgery
Percutaneous option	No	Yes	Yes, case reports

Abbreviations: ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; MI, myocardial infarction.

pulse, quiet heart sounds, or pulsus paradoxus suggestive of cardiac tamponade; acute pulmonary edema is less common.[6,10]

Diagnosis

A transthoracic echocardiogram (TTE) may show pericardial effusion, tamponade physiology, or epicardial clots or exudative material in the pericardial space (**Figure 1**C) (**Video 1**).[6] If the patient is stable, cardiac computed tomography or magnetic resonance imaging can also help confirm the presence and site of FWR.[9] In a 2018 report,[7] FWR most commonly affected the lateral wall (43%) and inferior wall (29%) and less commonly affected the anterior wall (17%) (Figure 1D) (Video 1). On coronary angiography, left anterior descending (LAD) or left circumflex (LCX) artery territory infarction is more commonly seen in patients with FWR than patients with post-MI cardiogenic shock from other causes.[10] A left ventriculogram may not show evidence of contrast shunting if there is no ongoing leak (Figure 1A and B) (Video 1).[6]

Treatment

The initial treatment of FWR is the same as for acute cardiac tamponade. Persistent hypotension may benefit from pericardiocentesis as a temporizing measure, but if the pericardial space contains predominantly thrombus, then drainage is unlikely to be helpful.[9] The presence of a hemopericardium is highly supportive of the diagnosis of FWR. Mechanical support with an intra-aortic balloon pump (IABP) or extracorporeal membrane oxygenation (ECMO) may also be required to maintain hemodynamic stability in the interim.[7,9] If patients are deemed unsuitable for surgery, then medical management may be all that is offered. However, in-hospital mortality rates for medically treated patients are extremely high at up to 90% compared with about 50% for those undergoing surgery.[12]

Surgery is the definitive therapy for FWR and aims to close the tear and prevent a recurrent rupture or formation of pseudoaneurysm while maintaining ventricular geometry.[9] Sutured and sutureless techniques using adhesives or surgical glues can be used (Figure 1E and F) (Video 1). A linear closure of the rupture line with sutures can be performed when sufficient nonischemic myocardium is present but is difficult in the presence of a large necrotic area. Direct suture of a patch covering the rupture and infarcted myocardium is another option and can also be done using surgical glue to adhere the patch to healthy myocardium but only in the absence of active bleeding. Infarct excision with direct suture or patch closure is less often used. Coronary artery bypass grafting (CABG) at the time of surgery has been associated with improved survival.[9] Coronary angiography to assess revascularization options can be considered before surgery if not already performed but depends on the degree of hemodynamic instability and urgency of operation. In-hospital survival in the SHOCK trial registry was 40%[10] but was recently reported at almost 66%[7]; hypertension, cardiac arrest at presentation, inotropes, preoperative ECMO, and postoperative IABP or ECMO were associated with higher in-hospital mortality.

VSR

Clinical Features

The 3 morphological types of rupture described for FWR are also applicable to VSR.[2] With no or delayed reperfusion, the development of VSR is described as having a bimodal distribution, with a high risk within the first 24 hours and then at 3 to 5 days.[2] However, in the SHOCK trial registry[13] and the GUSTO-I trial,[14] the median time to VSR was shorter at 16 hours and 1 day, respectively. VSR rarely occurs after 2 weeks post-MI.

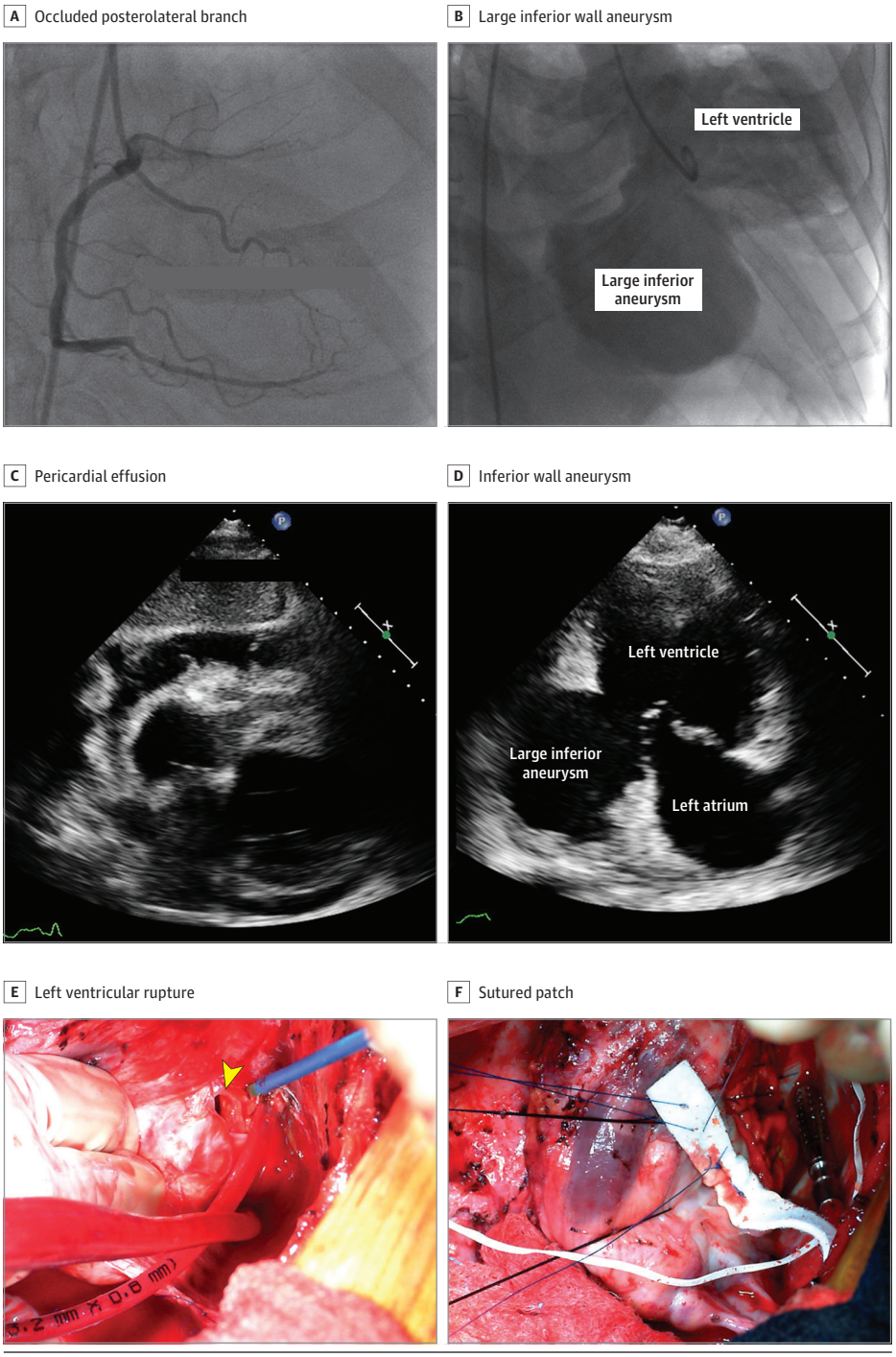
Female sex; older age; chronic kidney disease; and the absence of hypertension, diabetes, smoking, or a history of MI increase the risk of VSR.[13-17] Similar to FWR, these findings may suggest an important contribution of a lack of collateral circulation. VSR occurs less frequently with revascularization using percutaneous coronary intervention than thrombolytic therapy, less frequently in patients undergoing primary vs delayed percutaneous coronary intervention, and more frequently in those with anterior MI.[12,16]

VSR leads initially to a left to right shunt with right ventricular volume overload and consequent left atrial and ventricular volume overload. The ongoing direction and degree of the shunt will depend on the left and right ventricular function and pulmonary and systemic vascular resistance. Patients with VSR may present with chest pain, dyspnea, and evidence of cardiogenic shock (Table). Acute pulmonary edema is less common.[2] On examination, the typical murmur is a harsh pansystolic murmur at the left sternal border, and there may also be a palpable thrill. However, the murmur may not be heard in cardiogenic shock.

Diagnosis

A TTE is often the first test of choice and sufficient to identify the presence, region, and size of the rupture (**Figure 2**A) (**Video 2**). The direction of shunt flow, evidence of chamber enlargement with

Figure 1. Acute Rupture of a Left Ventricular Aneurysm



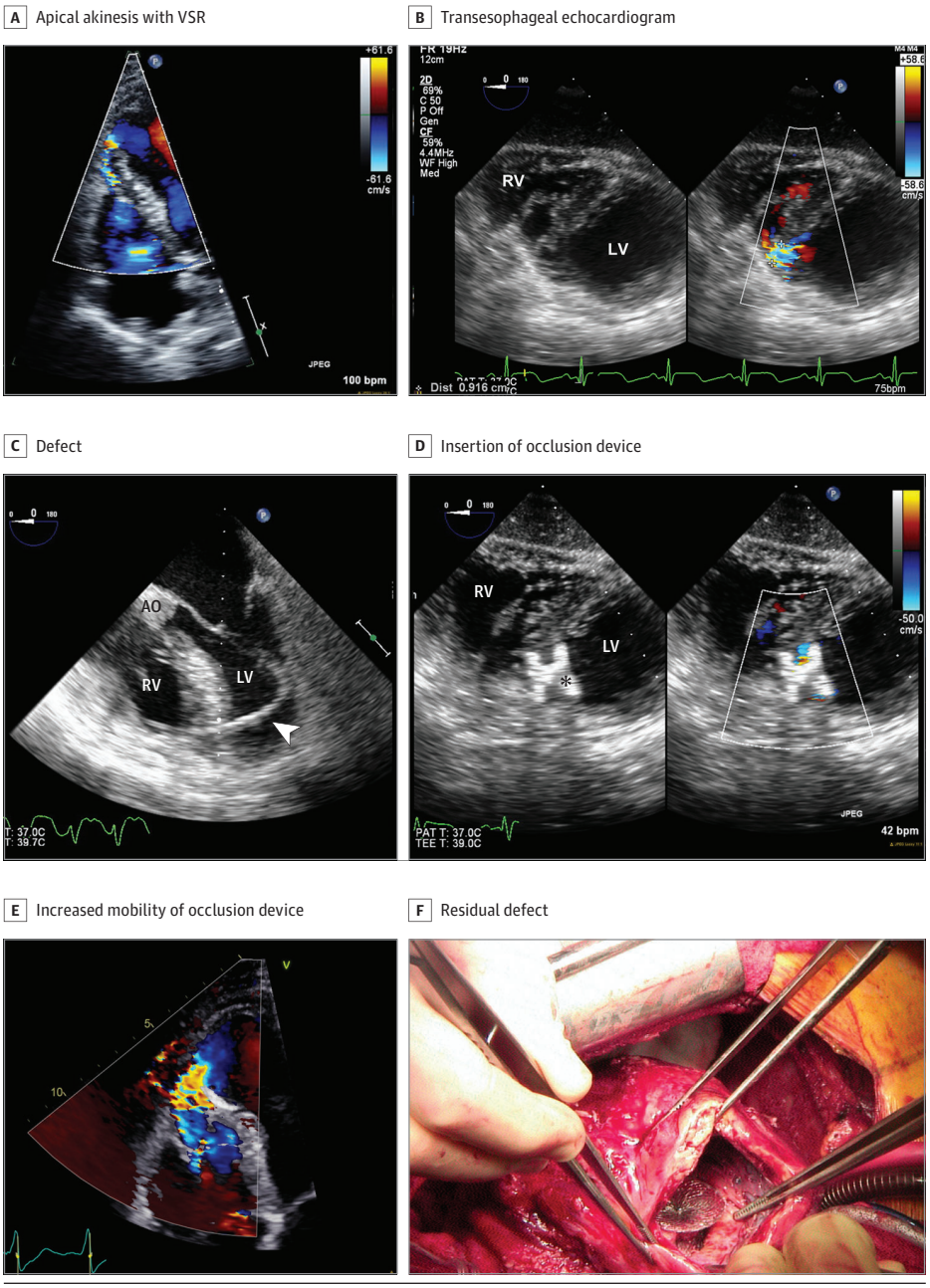
A man in his 50s presented with 3 weeks of fatigue and shortness of breath. A, Coronary angiography revealed an occluded posterolateral branch of the right coronary artery. B, Left ventriculography revealed a large inferior wall aneurysm. C, Prior to surgical assessment, he experienced a cardiac arrest, and bedside transthoracic echocardiography confirmed a large pericardial effusion in the subcostal view with tamponade physiology. D, Apical 2-chamber echocardiography revealed an inferior wall aneurysm. He received extracorporeal membrane oxygenation and was transferred for emergent surgery. Intraoperatively, perforation of a 4 × 8 × 4-cm aneurysm was identified. E, A left ventricular rupture (yellow arrowhead) was identified. F, A sutured patch was used to close the rupture.

dysfunction, and evidence of pulmonary hypertension can also be evaluated. In the event of suboptimal images on TTE, a transesophageal echocardiogram (TEE) can be performed (Figure 2B) (Video 2). If a VSR is suspected at the time of coronary angiography, a left ventriculogram can demonstrate left to right shunting of contrast. The LAD, dominant right coronary artery (RCA), or dominant LCX can all supply the ventricular septum. An anterior MI is more common in patients who develop a VSR than those who do not[15] and tends to produce simple apical defects. By contrast, inferior or lateral MIs tend to cause basal defects, which can be more irregular and extensive.[2,16] These can be missed on routine transthoracic imaging and may need additional imaging with TEE for diagnosis.

Treatment

Medical therapy, such as inotropes and diuretics, is a temporizing measure until more definitive treatment. An IABP or ECMO may also

Figure 2. Apical Ventricular Septal Rupture (VSR)



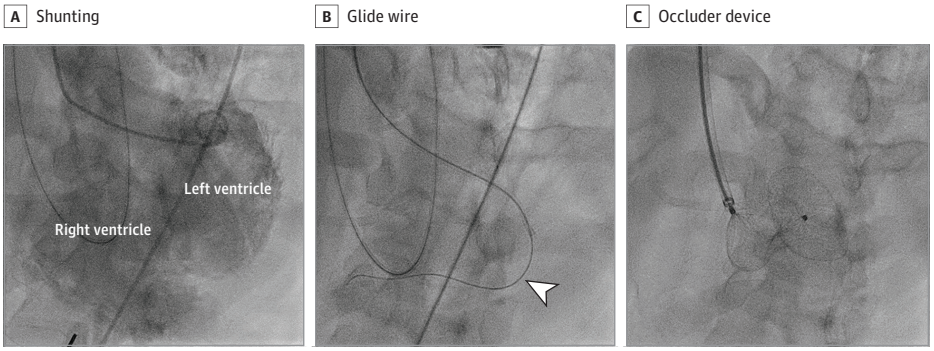
A man in his 50s presented with 2 days of persistent chest pain and an anterior ST-elevation myocardial infarction. Coronary angiography revealed left anterior descending occlusion, which was stented. A, Transthoracic echocardiography demonstrated apical akinesis with a VSR in the apex and left to right flow. B, A transesophageal echocardiogram measured the defect at 9 mm. C and D, After heart team discussion, the patient proceeded to percutaneous closure. The defect was cross from left to right via the aorta (AO) with a wire (arrowhead), facilitating insertion of a 16-mm occlusion device (asterisk) with trivial residual shunt. E, Six days later, he developed worsening heart failure, and transthoracic echocardiography showed increased mobility of the occlusion device with a large defect and significant left to right shunt. He proceeded to surgical patch closure of the VSR. F, Intraoperatively, the residual defect can be seen around the occluder device. LA indicates left atrium; LV, left ventricle; RV, right ventricle.

be required for unstable patients. In the GUSTO-I trial,[14] medical therapy alone was associated with 94% 30-day mortality and was double that of patients receiving surgery. Surgical management of VSR is the definitive treatment, but the optimal timing is unclear.

Findings from the Society of Thoracic Surgeons database showed that overall in-hospital or 30-day mortality for VSR repair was 43% but with a significant difference between mortality rates for repair performed 7 days or less and more than 7 days from MI (54% vs 18%, respectively).[18] Risk factors for operative mortality were increasing age, female sex, preoperative use of IABP, redo cardiac surgery, and emergent operation. The appropriate time to operate can be difficult to determine, as tissue friability is a concern for early repair, but delayed surgery also risks extension of the VSR and subsequent associated mortality. There are 2 established surgical techniques. The first involves making an incision into the infarcted myocardium and trimming this area before the defect is sutured closed; if the defect is large, a prosthetic patch is required.[19] The second is the infarct exclusion technique in which a patch larger than the infarcted area is sewn over the defect and infarcted myocardium directly onto healthy myocardium.[20] Modifications to these techniques have been made using more than 1 patch and additionally reinforcing the right ventricular septal wall.[21]

In contemporary practice, percutaneous techniques now offer a less invasive option for patients deemed at high operative risk. The

Figure 3. Closure of an Inferior Ventricular Septal Rupture on Fluoroscopy



A man in his 60s with delayed presentation of an inferior ST-elevation myocardial infarction underwent percutaneous closure of an inferior ventricular septal rupture. A, Left ventriculogram shows contrast shunting from left to right. B, The defect was crossed from left to right with a glide wire (arrowhead). C, The defect was large, and a 38-mm occluder device was required.

procedure is performed under general anesthesia with TEE and fluoroscopic guidance.[22,23] The TEE measurements or balloon sizing can be used to determine the appropriate device size. The Amplatzer devices (Abbott Laboratories) are most commonly used. Using both arterial and venous access, the defect is often crossed left to right, allowing the placement of a long sheath through which the occluder device can be placed at the VSR (Figure 2C to F and **Figure 3**) (Video 2). Based on recent Medicare data, between 2006 and 2014, the proportion of patients undergoing surgical repair decreased while the proportion undergoing percutaneous repair increased.[24] The procedural success rate for percutaneous device implantation has been reported at 89% across 13 studies.[25] A total of 46% of these patients had the procedure performed within 2 weeks of MI, and the 30-day mortality rate was 32%. Reported complications of percutaneous VSR closure include cardiac tamponade, persistent shunting, arrhythmias, bleeding, device embolization, hemolysis, and tricuspid leaflet chordal rupture.[22,25]

A 2018 systematic review[26] comparing medical, percutaneous, and surgical management for VSR showed significantly higher 30-day mortality for medical management compared with surgical or percutaneous treatments (92% vs 61% and 33%, respectively). There was no significant difference in mortality for surgical or percutaneous therapies performed within 14 days (56% vs 54%, respectively), but after 14 days, mortality was significantly higher for patients undergoing surgical vs percutaneous therapy (41% vs 16%). Given the complexities regarding the size and shape of the defect, hemodynamic stability, and other patient factors, heart team–based discussions should decide the optimal timing and method of closure.

PMR

Clinical Features

PMR accounts for more than half of acute severe mitral regurgitation that occurs after MI.[27,28] The remainder can be attributed to papillary muscle dysfunction from leaflet prolapse due to reduced tethering from an infarcted but intact papillary muscle, apical displacement, or ventricular dysfunction. As with other mechanical complications, PMR usually occurs within the first 7 days,[17] and the median time to presentation was 13 hours in the SHOCK trial registry.[29]

Patients with PMR are likely to be older and have hypertension and less likely to have diabetes or a prior MI.[27,30] Single-vessel occlusion is common,[27,30] and occlusion of the RCA is more common than the LCX, while LAD occlusion is less likely.[29-31] The anterolateral papillary muscle receives dual blood supply from the LAD and LCX, thereby protecting it from single-vessel occlusion, while the posteromedial papillary muscle is supplied only by the dominant RCA or LCX.[32,33] Therefore, the posteromedial papillary muscle is more often involved.

Acute pulmonary edema is the most common presentation with PMR (Table).[28] There may also be a history of delayed presentation or exertion or prolonged angina before the onset of symptoms. Examination findings of a classic holosystolic murmur may be absent in the setting of acute severe mitral regurgitation and high left atrial pressures.

Diagnosis

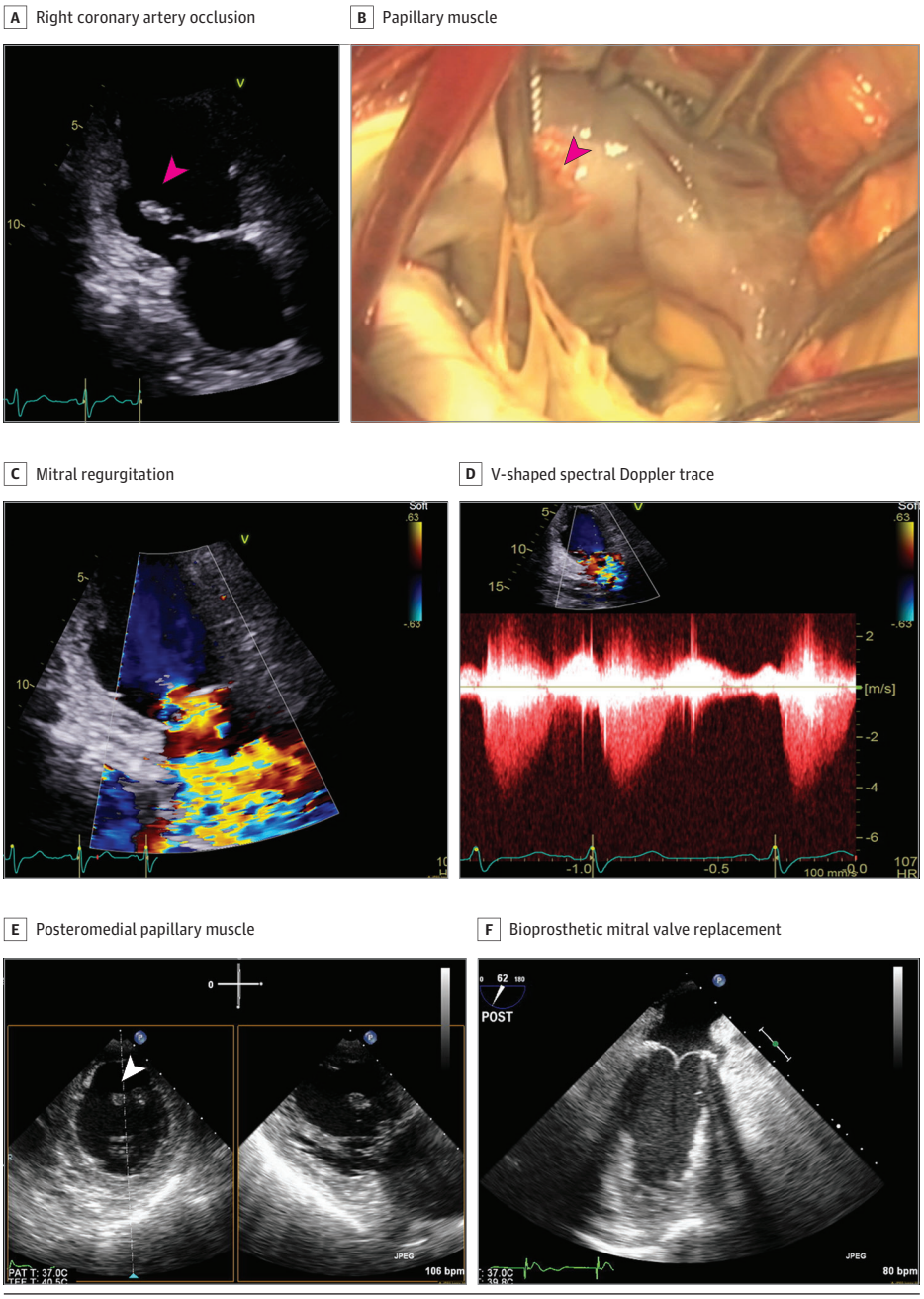
Chest radiography will confirm acute pulmonary edema. Findings of PMR may be seen on TTE, but TEE may be required. A rapidly moving ruptured papillary muscle prolapsing into the left atrium, prolapsing or flail leaflets, a severe regurgitant jet, or spectral Doppler tracing showing a V-shaped mitral regurgitation signal (rather than the typical rounded pattern) may provide a clue to the presence of PMR (**Figure 4**) (**Video 3**).[34] Because of the acuity of this condition, other imaging modalities are often not required.

Treatment

Acute management involves treatment of acute pulmonary edema, including noninvasive ventilation or intubation as required. Vasodilatation and afterload reduction can help to reduce mitral regurgitation and increase forward flow through the left ventricle (LV). Mechanical circulatory support (MCS) may be needed. However, mortality rates with medical management alone approach 50%,[31] and early surgery is generally recommended.

Mitral valve replacement is most commonly performed, but repair may be possible in some cases (Video 3).[35,36] The ruptured head of the papillary muscle may be sutured to an adjacent viable papillary muscle, but this may be unsuccessful if it is necrotic and friable. An isolated flail segment can be resected. Mitral valve repair is more difficult when severe mitral regurgitation is caused by papillary muscle dysfunction rather than rupture, as competency of the mitral valve achieved during surgery may be temporary in the setting

Figure 4. Papillary Muscle Rupture After Acute Myocardial Infarction



A woman in her 60s presented with acute onset chest pain. A and B, Coronary angiography showed right coronary artery occlusion with severe mitral regurgitation on echocardiography, and the patient underwent angioplasty, intra-aortic balloon pump insertion, and transfer to a surgical center. The ruptured papillary muscle (pink arrowhead) is seen on transthoracic echocardiogram and intraoperatively. C and D, Transthoracic echocardiography of the ruptured papillary muscle showed severe mitral regurgitation and a V-shaped spectral Doppler trace. She was taken to the operating theater on the evening of arrival. E and F, The intraoperative transesophageal echocardiogram shows the ruptured posteromedial papillary muscle and the bioprosthetic mitral valve replacement.

of ongoing ventricular remodeling. Nonetheless, previous studies have found no significant difference in mortality when comparing mitral valve repair and replacement.[31,37] Concomitant CABG has been associated with improved survival in many but not all studies.[28,37,38] Therefore, coronary angiography should be attempted if interim stabilization can be achieved. Perioperative mortality is reported to be between 8.7% and 24%.[28,37] For patients who survive beyond 30 days, 5-year mortality rates are similar to patients with MI but without PMR.[37]

Transcatheter mitral valve repair has been reported as an alternative treatment option in patients with severe mitral regurgitation after MI due to papillary muscle dysfunction (not rupture) who continue to experience hemodynamic instability despite medical therapy or MCS. The technique appears feasible in small case series with a reduction in mitral regurgitation, improved hemodynamics, and functional status maintained at up to 1 year.[39,40] However, more experience is required before routine consideration of transcatheter mitral valve repair.

True Aneurysms and Pseudoaneurysms

A pseudoaneurysm results from a rupture of the ventricular wall that remains contained by pericardium and fibrous tissue.[41] Myocardial

tissue is absent from a pseudoaneurysm. By contrast, a true aneurysm is a noncontractile outpouching of the ventricle and contains all the components of the myocardial wall.

Clinical Features

Patients with pseudoaneurysms can present with chest pain, dyspnea, cardiac tamponade, or even incidentally.[42] The potential for subacute presentation was illustrated in one series in which the median time from MI to diagnosis was 50 days, with approximately one-third presenting within 2 weeks after MI.[43] By contrast, true aneurysms usually form within the first 2 weeks after MI as a result of myocardial necrosis. Contemporary revascularization techniques limit the development of myocardial necrosis and have therefore decreased the incidence of true aneurysms; incidence was previously reported at up to 35% before thrombolysis[44] compared with 11% with thrombolysis.[45]

True aneurysms can present with angina, heart failure, ventricular tachycardia, or LV thrombus. Physical examination may reveal a prominent diffuse apical impulse or a third or fourth heart sound.[46] Q waves on the electrocardiogram correspond to the location of the aneurysm, and persistent ST segment elevation may also be seen. Chest radiography results may be unremarkable, show a bulge at the left heart border, or outline the aneurysm if calcification has occurred. Pseudoaneurysms most commonly present with heart failure but can also present with angina or be asymptomatic.

Diagnosis

As for all mechanical complications, TTE is often the first imaging modality used. A pseudoaneurysm is typically described as having a narrow neck while a true aneurysm has a wide neck (Figure 1). Additionally, a pseudoaneurysm more often occurs on the posterior or lateral wall, while a true aneurysm is more likely to occur on the anterior wall or apex.[42,47]

Differentiating between a pseudoaneurysm and true aneurysm can sometimes be difficult. In this setting, further assessment with cardiac computed tomography or magnetic resonance imaging may help to both differentiate the 2 pathologies and clarify the anatomy.

Treatment

Pseudoaneurysms have a high risk of expansion and rupture, and therefore, surgery is recommended. Direct closure of the defect with sutures or patch closure over an area of viable myocardium have both been described, while the pseudoaneurysm itself can be left unresected.[43] In-hospital mortality with surgery by either of these techniques has been reported at 20%. Percutaneous closure of pseudoaneurysms has also been attempted in patients at prohibitive surgical risk. In one series of 7 patients,[48] a septal occlusion device was successfully used to occlude the pseudoaneurysm with associated improvements in functional class. One patient experienced device embolization into the pseudoaneurysm. Experience with percutaneous therapies is otherwise limited.

More specific to true aneurysms is the use of prophylactic anticoagulation to prevent systemic embolization related to LV thrombus. The appropriate management remains unclear. Other medical management is that for heart failure. In the Coronary Artery Surgery Study (CASS) registry,[49] outcomes for medically treated patients were comparable with that of patients undergoing surgery: 1-year survival of 90% and 4-year survival of 71% with no significant difference compared with surgically treated patients with the same degree of LV dysfunction. Currently, concurrent aneurysm resection during CABG is recommended in the presence of a large aneurysm if there is a risk of rupture or large thrombus or if the aneurysm is contributing to recurrent arrhythmias.[50] Surgical techniques fall into 2 main types.[51] The direct suture technique is performed through a median sternotomy. The aneurysm is incised and resected with closure performed using a double row of sutures. The more recent technique is the patch ventriculoplasty; after the aneurysm is resected, circular sutures bring together the healthy and damaged myocardium to restore LV shape and then a patch can be directly sutured over this area. Thirty-day all-cause mortality was 5% for patients undergoing ventricular reconstruction in the Surgical Treatment for Ischemic Heart Failure (STICH) trial[52] and not significantly different to those who had CABG alone. However, the results remain controversial owing to what was felt to be an inadequate reduction in LV volumes with surgery.

MCS

Approximately 6% of patients with acute MI experience cardiogenic shock.[17] Patients with mechanical complications are particularly at risk of deteriorating to refractory cardiogenic shock, and in the SHOCK trial registry,[53] patients with mechanical complications accounted for 12% of those presenting with cardiogenic shock. MCS for these patients include IABP, percutaneous MCS, and ECMO. Unfortunately, despite the use of MCS, refractory cardiogenic shock continues to have a high mortality of 40% to 50%.[54] A meta-analysis evaluating ECMO use in cardiogenic shock found only 43% survival to discharge.[55] Reasons for the lack of perceived efficacy of MCS in managing acute MI–related cardiogenic shock have included delays in patient identification, delays in institution of therapy, and a lack of standardized approaches to decision-making and escalation of care.[56] Recent efforts to address these challenges have resulted in multidisciplinary shock teams akin to the heart team, consisting of interventional cardiologists, advanced heart failure cardiologists, cardiac surgeons, and critical care physicians. Shock teams are activated by predetermined criteria, and subsequent intervention and management is governed by a standardized protocol. The implementation of the shock team has been associated with significant improvements in survival[57,58] and is endorsed by a recent scientific statement from the American Heart Association[59] as a model of care to centralize management of patients with cardiogenic shock.

There are less data on the use of MCS for the management of cardiogenic shock associated with mechanical complications. Patients with mechanical complications are almost 7-fold more likely to use MCS than those without mechanical complications, and between 2003 and 2015, the rate of MCS use grew from 0% to 0.5% in 2003 to 7.6% to 8.4% in 2015.[3] Use of MCS, such as IABP or ECMO, for mechanical complications has been associated with increased mortality,[3,7,18] but it remains unclear whether this is largely a selection bias for sicker patients, related to complications of MCS, or related to delays in definitive therapy. Given that surgery remains definitive for mechanical complications, MCS remains a temporizing

measure. Management decisions for mechanical complications are complex with regards to timing and route of intervention and vary from patient to patient and, thus, particularly benefit from the multidisciplinary expertise of the shock team or heart team if hemodynamically stable.

Guideline Recommendations

Current guidelines from the American College of Cardiology Foundation/American Heart Association and the European Society of Cardiology recommend early surgical intervention for hemodynamically unstable patients.[60,61] Mortality rates associated with emergent surgery remain high and are reported at between 20% and 87% depending on the type of mechanical complication.[60,61] In view of more limited experience with percutaneous therapies for mechanical complications, the appropriate course of treatment and timing of intervention should be discussed with the heart team or shock team.[50]

Conclusions

Early revascularization is now the standard of care for patients presenting with acute MI. As a result, the incidence of mechanical complications is now less than 0.1%. However, when they do occur, the presentations are dramatic with acute hemodynamic instability and require urgent recognition. All patients require stabilization with interim medical management, but this alone results in extremely high mortality. In the absence of other factors that preclude any intervention, a decision is required regarding surgical or percutaneous treatment and the timing of this intervention. Surgery remains the definitive mode of treatment, but unsurprisingly, given the acuity and instability of patients undergoing early operations, mortality rates remain elevated. This has led to interest in acute percutaneous therapies, and our experience with these continues to grow. A multidisciplinary approach is required, and the heart team is aptly placed to guide the care of patients after MI with mechanical complications.

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Study concept and design: Gong, Maganti.
Acquisition, analysis, or interpretation of data: Vaitenas, Malaisrie.
Drafting of the manuscript: Gong, Maganti.
Critical revision of the manuscript for important intellectual content: All authors.
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