Mechanical Complications of Acute Myocardial Infarction

Ramesh S. Kutty, MBBS, MRCS\textsuperscript{a}, Nicola Jones, MA, BM BCh, MRCP, FRCA, FFICM\textsuperscript{b}, Narain Moorjani, MB ChB, MRCS, MD, FRCS (C-Th)\textsuperscript{a}.*

KEYWORDS

- Acute myocardial infarction
- Ventricular septal rupture
- Papillary muscle rupture
- Left ventricular free wall rupture
- Infarct exclusion technique

KEY POINTS

- The major mechanical complications after acute myocardial infarction (AMI) include rupture of the left ventricular free wall, papillary muscle rupture, and ventricular septal rupture.
- Primary percutaneous coronary intervention has significantly reduced major mechanical complications since its introduction as a treatment strategy in AMI.
- Echocardiography with color-flow Doppler is the investigation of choice in the diagnosis and differentiation of the conditions.
- Preoperative optimization, with an intra-aortic balloon pump and vasodilators, may help to reduce the afterload on the compromised ventricle following AMI and may improve cardiac output in the short term, but should not delay expedient surgical intervention.
- Surgical intervention remains the mainstay of treatment in patients with mechanical complications of AMI, with dismal outcomes for patients treated medically.

INTRODUCTION

Acute myocardial infarction (AMI) can result in ischemic, mechanical, arrhythmic, embolic, or inflammatory complications. The development of mechanical complications following AMI is associated with a significantly reduced short-term and long-term survival (Fig. 1).\textsuperscript{1} Since the introduction of primary percutaneous coronary intervention (PCI) as the principal reperfusion strategy following acute ST-elevation myocardial infarction (STEMI), the incidence of mechanical complications has reduced significantly to less than 1\%, including rupture of the left ventricular free wall (0.52\%), papillary muscle (0.26\%), and ventricular septum (0.17\%).\textsuperscript{1}

VENTRICULAR SEPTAL RUPTURE

Introduction

Ventricular septal rupture (VSR) represents a defect in the interventricular septum caused by ischaemic necrosis following AMI. Before the introduction of thrombolysis and primary PCI, VSR occurred in 1\% to 2\% of patients following AMI and usually presented between 3 and 5 days after AMI.\textsuperscript{2,3} Since the introduction of early reperfusion therapy, however, the incidence
of VSR has decreased to 0.17% and is usually diagnosed within the first 24 hours following presentation of AMI.\textsuperscript{4}

**Pathophysiology**

VSR occurs following a transmural infarct, and may be subclassified into simple and complex VSR.\textsuperscript{5} A simple VSR represents a single defect with openings in both ventricles at approximately the same level, whereas a complex VSR represents a meshwork of serpiginous channels with hemorrhage and disruption of myocardial tissue, which are more commonly found following posteroinferior AMI.

Before the introduction of early reperfusion, risk factors for VSR included hypertension, age, female sex, and the absence of previous history of ischemic heart disease (angina or myocardial infarction [MI]).\textsuperscript{6–8} The presence of angina or MI may have led to development of collateral coronary vessels as well as myocardial preconditioning, both of which reduce the risk of a transmural infarct and subsequent VSR development.\textsuperscript{8} In patients undergoing thrombolysis, advanced age, female sex, and the absence of smoking have been described as risk factors for the development of VSR, whereas the absence of prior angina or infarction has not been associated with an increased risk.\textsuperscript{5,9}

AMI complicated with VSR can progress into left ventricular (LV) or right ventricular (RV) failure, cardiogenic shock, and potentially irreversible end-organ malperfusion.\textsuperscript{10} The immediate effect of VSR is shunting of blood from the left to the right ventricle. The magnitude of this shunt is determined by the left- and right-sided pressures, as well as the size of the defect, which in turn determines the extent of hemodynamic compromise. Whereas LV dysfunction is related to the extent of the AMI, RV dysfunction is related to the volume overload produced by the intraventricular shunt as well as the initial infarct.

**Diagnosis**

Patients characteristically present for the first time following AMI, with no previous history of angina and with increasing dyspnea, often deteriorating rapidly into cardiogenic shock.\textsuperscript{11} A harsh pansystolic murmur, which is heard loudest at the left sternal edge, is present in 90% of patients, with a parasternal thrill palpable in 50% of patients.\textsuperscript{12} Mitral regurgitation (MR) secondary to papillary muscle rupture is an important differential diagnosis but has a murmur loudest at the apex.

Electrocardiography confirms primarily an anterior or inferior STEMI, with an associated atrioventricular conduction block noted in approximately 40% of patients.\textsuperscript{13} Chest radiography is generally nonspecific and may demonstrate cardiomegaly, pulmonary congestion, or pleural effusions.

Echocardiography with color-flow Doppler is the gold standard for diagnosis, monitoring, and planning treatment, distinguishing between papillary muscle rupture and VSR, as well as assessing

![Graph showing survival after mechanical complication](image-url)
LV and RV function. The sensitivity and specificity of color Doppler echocardiography has been reported in the literature to be as high as 100%. Echocardiography can identify the rupture, characterize the site and size of the defect, and estimate the degree of left-to-right shunt by assessing flow across the pulmonary and aortic valves.

Although right heart catheterization has now been superseded by echocardiography as a diagnostic tool, it can help to provide a diagnosis if the echocardiographic data are unclear. A demonstrated “step-up” between samples taken from the right atrium and pulmonary artery confirms the presence of a ventricular shunt. The ratio of flows between the pulmonary and systemic circulations provides an estimate of the size of the shunt. The excess pulmonary blood flow represents the amount of blood passing through the defect. Oxygen saturation samples are taken simultaneously to estimate the pulmonary to systemic flow ratio from the right atrium (SvO₂), pulmonary artery (SpaO₂), and systemic artery (Sao₂), which represents aortic saturations. Oxygen saturations from the pulmonary veins (SpvO₂) are not usually measured, but are assumed in a patient with healthy lungs to be fully oxygenated at 100%. The shunt ratio can then be calculated by the following formula:

\[ Q_p/Q_s = (Sao_2 - SvO_2)/(SpvO_2 - SpaO_2) \]

where \( Q_p \) is pulmonary blood flow and \( Q_s \) is systemic blood flow.

A \( Q_p/Q_s \) ratio of greater than 2 suggests a large shunt, which is usually poorly tolerated by patients. Standard pulmonary artery catheter reference values need to be interpreted with caution in patients with a shunt, and other continuous real-time hemodynamic monitoring tools, such as the LiDCO (London, UK) or PICCO (Pulsion AG, Munich, Germany) systems, may be more useful.

**Preoperative Resuscitation and Optimization**

Patients presenting with VSR are often in extremis, secondary to acute pulmonary congestion. Management of these patients may require intubation for a definitive airway or noninvasive ventilatory support. The mainstay of medical treatment is to achieve afterload reduction, using pharmacologic and nonpharmacologic methods.

Pharmacologic agents include angiotensin-converting enzyme inhibitors, intravenous nitrates, or hydralazine. Phosphodiesterase-3 inhibitors, such as milrinone and enoximone, are also used as inodilators, as they increase myocardial contractility as well as producing vasodilation. The calcium sensitizer, levosimendan, has also been used to beneficial effect to counteract the ventricular stunning associated with reperfusion injury following AMI. Levosimendan improves ventricular function as it acts as a positive inotrope by increasing the sensitivity of myofilaments to calcium by binding to troponin C, thereby increasing contractility without increasing myocardial oxygen consumption. It also has vasodilatory effects, thereby reducing preload and afterload as well as increasing coronary blood flow. Patients with significant RV dysfunction may also benefit from pulmonary vasodilators, such as inhaled nitric oxide.

Nonpharmacologic measures include the use of an intra-aortic balloon pump (IABP), the Impella Recover device (Abiomed, Aachen, Germany), extracorporeal membrane oxygenation (ECMO), a ventricular assist device (VAD), a total artificial heart to unload the failing myocardium, and hemofiltration to treat the volume overload and manage fluid status. Insertion of an IABP decreases LV afterload, thereby reducing the volume of the shunt, and improves coronary blood flow. The resultant increased cardiac output produces improved systemic pressures and end-organ perfusion. The quest for optimization, however, should not delay definitive surgical treatment.

**Operative Technique**

Standard surgical repair of a postinfarction VSR involves using the infarct exclusion technique (Fig. 2). This procedure comprises a left ventriculotomy through the infarcted anterior or inferior wall, 2 to 3 cm parallel to the left anterior descending artery or posterior descending artery, respectively. A glutaraldehyde-fixed bovine pericardial patch is then sutured to healthy endocardium deep in the left ventricle, to exclude the infarct and VSR from the high-pressure area of the left ventricle. This patch is then brought out through the ventriculotomy and incorporated in the closure. The ventriculotomy is closed in 2 layers, buttressed by Teflon strips. Using this technique, LV geometry and volume can be restored. Surgical repair of a posterior VSR is technically more difficult because of access to the inferobasal LV wall, especially involving necrotic and friable myocardium immediately after an AMI. Involvement of the posteromedial papillary muscle, either in the ischemic area of the infarct or by the suture line of the repair, increases the risk of papillary muscle dysfunction with resultant compromise of the mitral valve apparatus. In these patients, concomitant mitral valve repair or replacement might be required. Once surgical repair of the
VSR is complete, coronary artery bypass grafting (CABG) is completed.

**Percutaneous closure**
The role of percutaneous devices to primarily close defects following VSR is limited at present to selected patients with simple defects that are less than 15 mm in diameter and after approximately 3 weeks following AMI. Percutaneous closure has also been used in the acute setting, with variable results. VSR without a suitable rim or a basal VSR in the vicinity of the mitral apparatus or the aortic valve represent a contraindication to percutaneous closure. In the majority of cases, rapid hemodynamic stabilization occurs by reducing the left-to-right shunt, and may provide a bridge to definitive surgery. Percutaneous closure devices are also used for patients with residual defects after surgical repair.

**Postoperative management**
The principal goal of postoperative care in these patients is optimizing cardiac output and reversing any end-organ dysfunction that may have occurred following the AMI or in the perioperative period. This care involves continuation of preoperative cardiac support, including the use of an IABP, positive inotropic agents, antiarrhythmic agents, and vasodilatory agents, to reduce the preload and afterload on the impaired myocardium. In particular, optimization of RV function may be required with phosphodiesterase-3 inhibitors, nitric oxide, or an RV assist device. In addition, renal replacement therapy with hemodialysis, nasogastric feeding, and prolonged ventilator support is often required in these patients.

**Controversial Issues**

**Should surgery be performed on all patients with a VSR?**
Although current guidelines recommend immediate surgical treatment for patients with VSR following AMI to avoid end-organ failure and improve survival, certain patients are likely to have a poor outcome despite optimal medical or surgical management. Risk factors predisposing patients to increased operative mortality include cardiogenic shock, age, RV failure, and renal impairment. Some patients with multiple comorbid risk factors may be best suited to a trial of maximal nonoperative management, and undergo operative intervention if they can be hemodynamically stabilized.

**Concomitant coronary revascularization or not?**
There has previously been controversy in the surgical literature over the benefits of concomitant CABG at the time of surgical VSR repair. Initial studies demonstrated that concomitant CABG
did not confer a long-term survival advantage and that performing a left heart catheterization is time consuming, which may contribute to perioperative morbidity and mortality in these already hemodynamically compromised patients.\(^3^9\) In most series, however, more than 60% of patients with septal rupture have significant involvement of at least 1 vessel in the noninfarcted area.\(^4^0\) More recently, incomplete myocardial revascularization has been demonstrated to be a significant predictor of late mortality following surgical repair of postinfarction VSR.\(^4^1\) In view of this, concomitant CABG to all stenotic coronary arteries, including those supplying the noninfarcted area, is recommended for patients undergoing surgical repair of VSR.\(^4^2\) The improvement of the collateral flow to the myocardium is thought to contribute to enhanced ventricular recovery.

**Is there a role for delayed surgery?**

The strategy of operating following stabilization for around 6 weeks, often with IABP counterpulsation, is an attractive option, as it allows an infarcted septum to “mature” with fibrotic healing.\(^4^3\) The sickest patients fail to survive this trial of delayed surgery, through a process of “unnatural selection.”\(^4^4\) As outcomes of immediate and delayed surgery have been shown to be similar, more patients who otherwise may not survive to 6 weeks could potentially be saved by early surgery.\(^3^8\) The use of nonpharmacologic cardiac support in acute VSR management is predominantly limited to IABP support. However, in some patients who are at high risk of perioperative death but in whom surgery cannot be safely deferred, the use of mechanical circulatory support, such as using an ECMO circuit or Impella Recover device, may provide hemodynamic stability and the potential to correct multorgan dysfunction, until a definitive procedure is performed.\(^2^6–^2^8\)

**Outcomes**

Patients treated medically have a universally dismal prognosis, with 24% mortality at 24 hours, 46% mortality at 1 week, and 82% mortality within 2 months.\(^4^5\) In a review of 64 patients presenting with VSR, medically treated patients had a 30-day mortality of 100% (Fig. 3).\(^1^0\)

Recent data from the Society of Thoracic Surgeons registry of 2876 patients presenting with postinfarction VSR demonstrated an overall operative mortality of 42.9%.\(^4^6\) Patients who underwent surgical repair within 7 days from MI (54.1%) had a higher operative mortality compared with those in whom the surgical repair was performed more than 7 days following the MI (18.4%). Multivariable analysis identified several factors associated with increased risk of operative death, including preoperative dialysis, age, female sex, cardiogenic shock, preoperative IABP, mitral insufficiency, and redo cardiac operation.\(^4^5\) The Swedish national registry data of 189 patients undergoing surgical repair of postinfarction VSR reported a 30-day mortality rate of 41% and 5 year survival of 38% (Fig. 4).\(^4^7\) For patients that survived the first 30 days (n = 112), the 5-year cumulative survival was 67%. The investigators observed that posterior septal rupture was independently associated with an increased risk of operative death.

**Summary of Ventricular Septal Rupture**

VSR requires the expeditious stabilization of a hemodynamically compromised patient followed by
prompt surgical management of the rupture. Despite the high mortality associated with surgical repair of VSR, good long-term outcomes in operative survivors make it a worthwhile endeavor, as it is potentially the only option for survival following this mechanical complication of AMI.

**ACUTE MITRAL REGURGITATION**

*Introduction*

Acute MR is a catastrophic complication of an AMI that, if recognized, is amenable to emergent surgical intervention. Mild to moderate chronic MR is found in 15% to 45% of patients after AMI. In this population this degree of MR is well tolerated, and is usually transient and asymptomatic. Acute MR secondary to papillary muscle rupture, however, is a life-threatening complication with a poor prognosis. Papillary muscle rupture occurs in 0.25% of patients following AMI and represents up to 7% of patients in cardiogenic shock following AMI. Although acute MR secondary to papillary muscle rupture is usually diagnosed between 2 to 7 days after

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**Fig. 4.** Cumulative survival following operative repair of postinfarction ventricular septal rupture in (A) all surgical patients and (B) surgical patients who survived 30 days. *(From Jeppsson A, Liden H, Johnsson P, et al. Surgical repair of post infarction ventricular septal defects: a national experience. Eur J Cardiothorac Surg 2005;27:217–8; with permission.)*
AMI, the median time to papillary muscle rupture is approximately 13 hours. Papillary muscle rupture following AMI may be partial (occurring at either one of the muscle heads) or complete.

Pathophysiology

Following AMI, papillary muscle dysfunction or rupture, in combination with changes in LV shape and regional wall function, results in acute MR. Even slight modifications of LV geometry caused by regional wall-motion abnormality may contribute to the increased frequency of MR after AMI. In the acute setting, pulmonary edema and cardiogenic shock may ensue, as there is not enough time for the left ventricle to dilate or compensate. Acute MR secondary to papillary muscle dysfunction or rupture occurs most commonly following an inferior MI, owing to the single blood supply to the posteromedial papillary muscle from the posterior descending coronary artery. The anterolateral papillary muscle, however, has a dual blood supply (left anterior descending and circumflex coronary arteries) and, therefore, is more likely to be protected following an AMI in a single-vessel territory.

Diagnosis

When acute MR accompanies complete papillary muscle rupture, patients may present with immediate pulmonary edema, hypotension, and, in some cases, cardiogenic shock. A new pansystolic murmur is heard loudest at the cardiac apex, with a diastolic component, and radiates to the axilla. VSR secondary to AMI is an important differential diagnosis, and can be distinguished by a harsh pansystolic murmur heard loudest at the left sternal edge.

Electrocardiography usually confirms an inferior or posterior MI. Chest radiography demonstrates pulmonary edema, which occasionally is localized to the right upper lobe, if the flow is directed at the right superior pulmonary vein.

Echocardiography with color-flow Doppler is the standard for diagnosis, monitoring, and planning of surgical treatment, distinguishing between papillary muscle rupture and VSR, as well as assessing LV and RV function. It usually identifies either a flail chord or papillary muscle, with resultant leaflet prolapse and MR. Echocardiography usually overestimates the LV function, as the ventricle is hyperdynamic in the presence of MR. Transesophageal echocardiography is particularly useful in identifying the anatomy of the mitral valve and demonstrating the pathology of the MR (Fig. 5).

Right heart catheterization has limited use in the diagnosis of acute MR, but can be useful in differentiating it from VSR (see earlier discussion). The pulmonary capillary wedge pressure (PCWP) trace may show giant V waves but these are generally

![Fig. 5](image)

**Fig. 5.** Transesophageal echocardiogram images demonstrating prolapse of the anterior mitral valve leaflet, secondary to flail papillary muscle (arrow), and corresponding color-flow Doppler images illustrating a resultant posteriorly directed jet of severe mitral regurgitation. Ao, aorta; LA, left atrium; LV, left ventricle.
nonspecific, as they may also be seen in VSR or severe LV failure.57

Treatment

Prompt diagnosis with immediate initiation of aggressive medical therapy is vital until emergent surgical intervention can be performed. Urgent cardiac catheterization needs to be performed to identify coronary anatomy, as concomitant revascularization during mitral valve surgery is associated with improved short-term and long-term outcomes (Fig. 6).58,59

Medical therapy aims to reduce the afterload, with a resultant decreased regurgitant fraction and increased forward stroke volume and cardiac output.60 This goal can be achieved pharmacologically with vasodilators and inodilators, such as nitrites, sodium nitroprusside, diuretics, and phosphodiesterase-3 inhibitors.61,62 Intra-aortic balloon counterpulsation during acute MR decreases afterload, resulting in less MR and more forward flow from the left ventricle into the aorta.25 Similarly to patients with VSR, these patients may also benefit from mechanical cardiac support, such as an Impella Recover device,26 ECMO circuit,27 or VAD.28 In addition, positive-pressure ventilation is used with great effect in such patients with acute pulmonary edema and pulmonary congestion.63 As medical therapy is associated with a very poor survival, emergent surgery remains the cornerstone of treatment.58 During the operation, careful assessment of the mitral valve and the subvalvular apparatus will allow for decision making regarding repair or replacement. If there is evidence of papillary muscle necrosis or there are concerns about subtle, ongoing progression of ischemic injury, mitral valve replacement provides a definitive treatment of the failing mitral valve apparatus.64 Intra-aortic balloon counterpulsation should continue for at least 24 hours following surgery, in addition to the supportive postoperative care that will be required for these patients with multiorgan dysfunction (see the section on postoperative management of VSR).65

Outcomes

Acute postinfarction MR is associated with an inhospital mortality of between 70% and 80% with medical treatment.49 In the largest series of patients (N = 126) who underwent surgical intervention for papillary muscle rupture, operative mortality was 26.9% with a 15-year survival of 39%.58 Although there was no difference in early mortality between patients undergoing concomitant CABG and those who were not revascularized at the time of emergent mitral surgery (CABG 27.3% vs no CABG 26.4%; P>.9), long-term survival was significantly improved in patients undergoing concomitant revascularization at 15 years (CABG 64% vs no CABG 23%; P<.001). In a more recent study, concomitant CABG has also been shown to improve operative mortality (odds ratio 0.18; 95% confidence interval 0.04–0.83; P = .011).66 However, no significant difference in 5-year survival has been demonstrated between mitral valve repair or mitral valve replacement in this patient cohort (62.1% vs 66.7%; P = .48).66
Of note, the 5-year survival rate of the operative survivors following surgical repair of postinfarction papillary muscle rupture (79.4%) is similar to that of matched controls with an uncomplicated MI.66

Summary of Acute Mitral Regurgitation

Patients presenting with the catastrophic mechanical complication of acute MR secondary to papillary muscle rupture following MI benefit from combined mitral valve surgery and myocardial revascularization, with satisfactory early and late outcomes despite the increased operative mortality. Mitral valve replacement rather than repair may be indicated in patients following acute papillary muscle rupture, or in cases with severe restriction of the mitral valve caused by papillary muscle dysfunction after AMI.

RUPTURE OF LEFT VENTRICULAR FREE WALL

Introduction

Rupture of the LV free wall occurs in 0.5% of patients following AMI and is associated with 20% mortality.1 A high index of suspicion and early diagnosis are vital to the survival of these high-risk patients. Approximately 50% of patients with free-wall rupture are diagnosed within 5 days of the AMI, with 90% diagnosed within 2 weeks.5 Risk factors for the development of free-wall rupture following AMI include age, female gender, hypertension, first MI, and poor coronary artery collateralization.6

Pathophysiology

Early free-wall rupture, seen within the first 24 hours, represents a small-tear, full-thickness rupture, which is temporarily sealed by clot and fibrinous pericardial adhesions. The use of thrombolysis may result in the necrotic tissue developing into a hemorrhagic infarct, with a sudden “blow-out” rupture, which is associated with 35% to 60% mortality.67 Late free-wall rupture develops 1 to 3 days following the AMI and is due to erosion at the border zone between infarction and normal myocardium.68 Since the advent of thrombolytic agents and primary PCI, there has been a reduction in the incidence of late rupture.69 Cardiac rupture has been also been classified according to pathology, with type I representing an abrupt myocardial tear in the absence of myocardial thinning; type II representing erosion of the infarcted myocardium followed by dehiscence and subsequent covering by thrombus; and type III representing myocardial thinning and perforation at the center of the ventricular aneurysm.70 Pseudoaneurysm of the left ventricle represents free-wall rupture that is contained by pericardial adhesions. The culprit coronary lesion associated with LV rupture has been reported as the left anterior descending artery in 42% of patients, the circumflex coronary artery in 40% of patients, and the right coronary artery in 18% of patients.71

Diagnosis

Sudden onset of pain following coughing or straining may be suggestive of myocardial rupture following AMI. A proportion of patients who develop free-wall rupture will present with a subacute course of pain in keeping with pericarditis, nausea, and hypotension.72 Acute rupture, however, often presents as electromechanical dissociation and sudden death (type I) or hemorrhage, tamponade, hypotension, and state of low cardiac output (types II and III).73 The nature of acute rupture does not always lend itself to diagnostic investigation. If time allows, echocardiography is the modality of choice, with a sensitivity and specificity of 93% to 98%.74 Echocardiography will demonstrate a pericardial collection with signs of cardiac tamponade, including collapse of the right atrium and ventricle in diastole, a dilated inferior vena cava, and marked respiratory variation in mitral and tricuspid valve inflow. Similarly, if a pulmonary artery catheter is in situ, it will demonstrate the hemodynamic abnormality of cardiac tamponade with equalization of right atrial, RV diastolic, and pulmonary capillary wedge pressures.75 Computed tomography or magnetic resonance imaging may also be useful in determining the extent of the free-wall rupture or pseudoaneurysm (Fig. 7).

Management

LV wall rupture requires emergent salvage surgery. The use of emergency pericardiocentesis is controversial.76 Although emergency pericardiocentesis may provide hemodynamic short-term improvement by relieving the tamponade, it can cause a dangerous increase in blood pressure with increased tension on damaged myocardium, with the potential for extension of a small tear to a rupture. Traditionally surgical repair is performed by direct suture of the myocardium over the infarct zone with reinforcement by Teflon felt strip,77 or repair using the infarct exclusion technique.78 More recently, a bovine pericardial patch has been used over the repair, reinforced with surgical glue beneath the patch, with or without epicardial suture on the patch borders (patch and glue technique).78 Angiographically guided complete revascularization should be attempted following repair of the rupture. If angiography is not performed,
complete revascularization to the noninfarcted areas is recommended.\textsuperscript{79,80}

**Outcomes**

Successful surgical management of patients with LV rupture has been reported in small series (N = 25), with an operative mortality of 12\% to 30\%.\textsuperscript{78,81} The long-term outcomes of these patients is primarily related to cardiac function following the AMI rather than recurrent rupture, with a 7-year survival of 68\%.\textsuperscript{78}

**Summary of Rupture of LV Free Wall**

Rupture of the LV free wall represents a significant mechanical complication of AMI, associated with a high mortality. Patients with contained rupture of the left ventricle benefit from expeditious diagnosis and emergent surgical intervention. If possible, avoiding the use of cardiopulmonary bypass by using a minimally invasive operative approach to repair the free-wall rupture (patch and glue technique) may be beneficial in patients with an already hemodynamically compromised myocardium.

**SUMMARY**

Mechanical complications after AMI, including VSR, papillary muscle rupture, and rupture of the LV free wall, are surgical emergencies that require urgent diagnosis and rapid intervention for optimal outcomes. Diagnosis in the emergent setting can be difficult in patients with dyspnea and shock. A high index of suspicion and appropriate investigation with echocardiography is vital for rapid diagnosis followed by emergent surgical treatment, if possible. Preoperative optimization with IABP and vasodilators may help to reduce the afterload on the compromised ventricle following AMI and improve cardiac output in the short term, but should not delay expedient surgical intervention. Despite high operative mortality, the lack of an effective medical alternative makes surgical repair the mainstay of current management for these patients.

**REFERENCES**


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