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SPECIAL ARTICLE
Ischaemic heart disease

Ventricular free-wall rupture, ventricular pseudoaneurysm, and papillary muscle rupture complicating acute myocardial infarction

A clinical consensus statement of the ESC Working Group on Cardiovascular Surgery, the Association for Acute CardioVascular Care of the ESC, the European Association of Percutaneous Cardiovascular Interventions of the ESC, the ESC Working Group on Pulmonary Circulation and Right Ventricular Function, the European Association of Preventive Cardiology of the ESC, the Heart Failure Association of the ESC, the European Association of Cardiovascular Imaging of the ESC, and the European Association for Cardio-Thoracic Surgery

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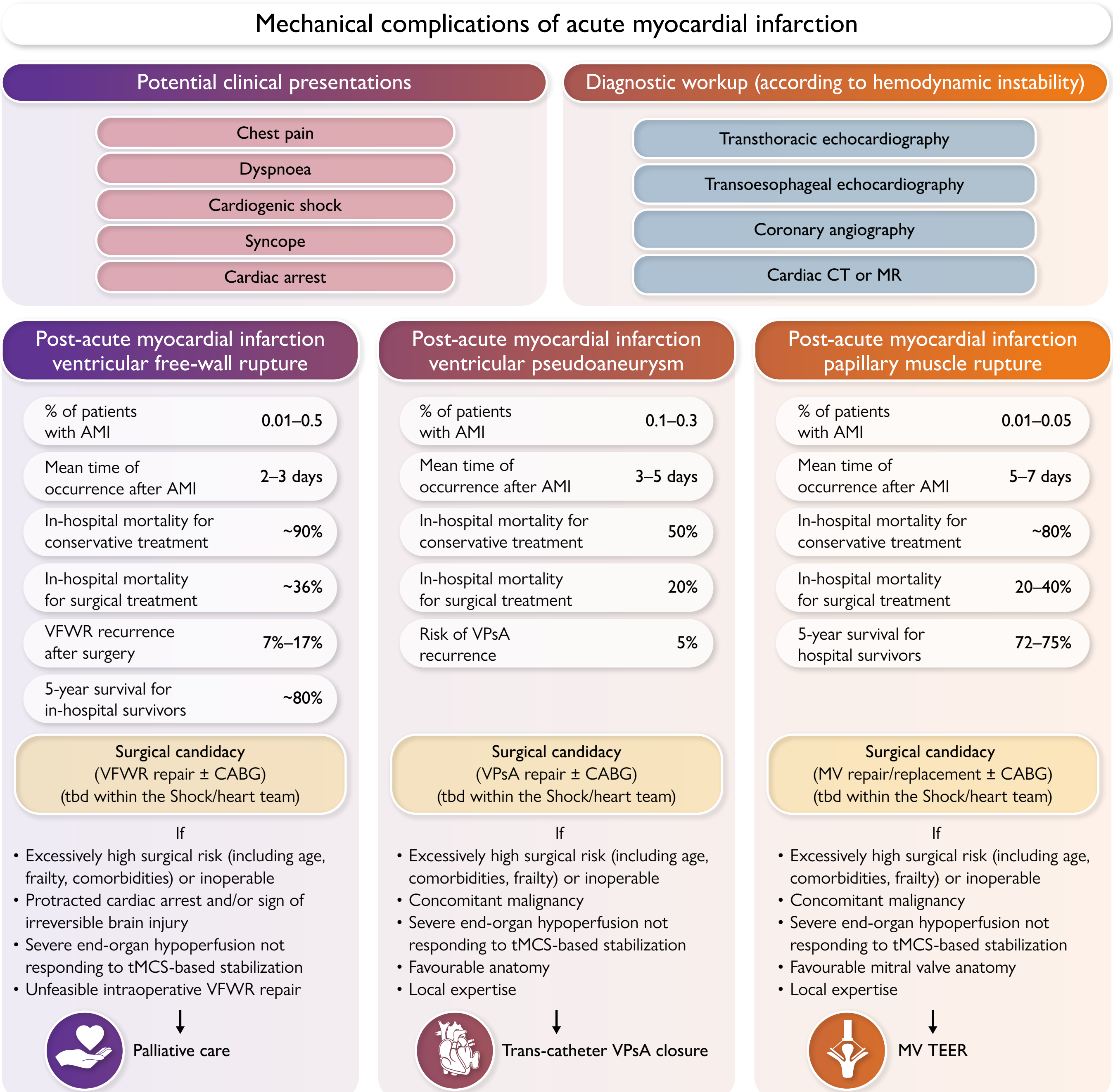
Abstract

The prevalence of mechanical complications following acute myocardial infarction has steadily declined in recent years owing to advances in prompt coronary revascularization, and they now occur in <1% of acute myocardial infarction cases. Nevertheless, significant haemodynamic impairment may already be present at hospital admission, requiring immediate diagnostic evaluation and urgent intervention. Until recently, surgical repair was the only treatment option, with non-negligible in-hospital mortality rates, particularly among patients with acute cardio-circulatory failure. Advances in transcatheter percutaneous procedures have now introduced alternative treatment strategies, especially for high-risk or inoperable patients. Recurrence of post-acute myocardial infarction mechanical complications, even shortly after the repair of the underlying lesion, has a critical impact on patient outcome and underscores the need for careful monitoring during hospitalization as well as after discharge. The role of concomitant coronary revascularization remains controversial, with variable effects on both early and late outcomes, and warrants further investigation. Temporary mechanical circulatory support has shown encouraging results, either for pre-procedural haemodynamic stabilization ('bridge-to-procedure') or for prophylactic, extended peri-procedural support to facilitate myocardial recovery ('bridge-to-recovery'). Optimal management should be guided by a multidisciplinary Heart Team approach (including Shock Team involvement where appropriate) with integration of palliative care into the decision-making process.

Graphical Abstract



AMI, acute myocardial infarction; **CABG**, coronary artery bypass grafting; **CT**, computed tomography; **tMCS**, temporary mechanical circulatory support; **MRI**, magnetic resonance imaginig; **MV**, mitral valve; **TBD**, to be discussed; **TEER**, trans-catheter edge-to-edge repair; **VFWR**, ventricular free-wall rupture; **VPSA**, ventricular pseudoaneurysm

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Keywords Acute myocardial infarction • papillary muscle rupture • ventricular free-wall rupture • ventricular pseudo-aneurysm • cardiogenic shock • temporary mechanical circulatory support • multidisciplinary heart team • coronary artery disease

Introduction

Over the past three decades, advances in the diagnosis and management of acute myocardial infarction (AMI) have significantly reduced post-infarction heart failure (HF), cardiogenic shock, and mechanical complications. Thanks to timely percutaneous coronary intervention (PCI), mechanical complications now affect <1% of ST-elevation (STEMI) and non-ST-elevation (NSTEMI) myocardial infarction cases.[1,2] Nevertheless, morbidity and mortality remain high, with in-hospital mortality ranging from 30% to 40%.[1,3] The management of ventricular free-wall rupture (VFWR), ventricular pseudoaneurysm (VPsA), and papillary muscle rupture (PMR) has evolved with the introduction of transcatheter techniques. Temporary mechanical circulatory support (tMCS) now enables haemodynamic stabilization, bridge-to-procedure and peri-procedural protection against cardiogenic shock, and low cardiac output syndrome (LCOS). Despite their clinical significance, post-AMI mechanical complications are underrepresented in major scientific documents. The current position paper provides an up-to-date summary of epidemiology, diagnosis, and treatment of VFWR VPsA and PMR and identifies research gaps to improve outcomes (*Graphical Abstract*). The ventricular septal rupture (VSR) has been addressed in a dedicated expert consensus document recently published.[4]

Methods and definition

A comprehensive literature review was conducted by four authors (R.L., M.M., D.R., G.M.) using PubMed, Embase, and the Cochrane Central Register of Controlled Trials (1 January 1950–31 May 2024). All studies addressing post-AMI mechanical complications were considered, including case reports, case series, registries, reviews, meta-analyses, and editorials. Keywords included 'post-infarction mechanical complications', 'cardiac rupture', 'heart rupture', 'myocardial rupture', 'ventricular free-wall rupture', 'ventricular pseudoaneurysm', 'papillary muscle rupture', 'acute mitral regurgitation', 'myocardial infarction', and 'acute myocardial infarction'.

Ventricular free-wall rupture is defined as a discontinuity in the ventricular free wall (either right or left) corresponding to the necrotic area following AMI. It can be characterized as complete rupture of the ventricular chamber, causing direct communication with the pericardial cavity (blowout VFWR type), or as subacute myocardial wall tearing generating slow blood extraversion into the pericardial cavity through the infarcted wall (oozing VFWR type).[5–7]

Ventricular pseudoaneurysm is a contained ventricular rupture, either by epicardial clots or pericardial adhesions, forming a thin-walled accessory cavity lacking endocardial and myocardial layers and connected to the ventricle through a narrow neck. Differentiating VPsA and true left ventricular (LV) aneurysm is essential for pathophysiologic, therapeutic, and prognostic purposes.[5,6,8]

Papillary muscle rupture involves partial or complete rupture of one mitral valve papillary muscle (PM) following AMI, causing acute mitral regurgitation (MR).[6,9,10]

Consensus for all the different parts of this document was achieved through multiple rounds of co-author reviews, with unanimous acceptance and based on extensive and systematic reviews of the available literature.

Ventricular free-wall rupture

Epidemiology

The incidence of VFWR has declined due to the improvement in primary revascularization strategies.[11] A recent analysis, assessing more than 9 million AMI hospitalizations, revealed that VFWR occurred in .01% of STEMI patients,[1] while other trials found an incidence between .2% and .5%.[2,11] However, the true incidence of VFWR is likely underestimated due to out-of-hospital sudden cardiac death.[12] Older reports demonstrate that VFWR is commonly diagnosed at 5–7 days after AMI,[13] while recent data show that it typically occurs within 24–48 h. Pathophysiologically, it is likely related to reperfusion injury and intramyocardial haemorrhage as well as myocardial dissection.[7,14,15]

Risk factors

Major risk factors for VFWR include late presentation [i.e. Q wave at electrocardiogram (ECG)] and large infarct size (i.e. creatine kinase-MB peak > 150 IU/L), associated with an almost 10-fold increased risk.[16] Lack of previous AMI and involvement of the left anterior descending or circumflex artery are considered to be major predictors of VFWR, probably due to the lack of compensatory collateral circulation that might develop in case of chronic coronary artery disease. Patients with VFWR are less likely to have diabetes, peripheral artery disease, and prior episodes of myocardial ischaemia.[15–17] Finally, advanced age, female sex, and history of hypertension are independent prognostic factors of VFWR.[16]

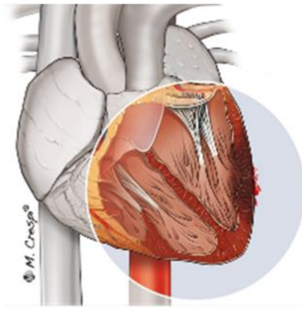
Classification

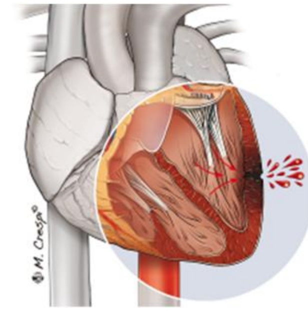
Different classifications of VFWR have been proposed, mostly based on surgical data or autopsy. Morphologically,[18] VFWR is classified as follows: *Type I rupture*, abrupt, slit-like myocardial tear, occurring within 24 h after AMI; *Type II rupture*, slowly progressive myocardial erosion (>24 h after AMI onset); and *Type III rupture*, thinned myocardial perforation (>7 days after AMI). Haddadin *et al.*[19] divided VFWR into two groups based on observing the rupture during surgery. The *blowout type*, defined as a macroscopic defect of the epicardium, with LV cavity-pericardial communication or the *oozing type*, represents a smaller tear or epicardial extravasation related to an intramyocardial haematoma. Two clinical classifications have been recently proposed: the first includes three clinical phenotypes—*cardiac arrest type*, *unstable type*, and *stable type*.[20] The second classification distinguishes *acute* and *subacute* VFWR.[5]

Pathophysiology and clinical presentation

Clinical presentation may include dyspnoea, chest pain, cardiogenic shock, or cardiac arrest.[14,21,22] Indeed, the clinical manifestations of VFWR are dependent on the rate and extent of pericardial bleeding as well as consequent pericardial tamponade.[23] The *blowout* pattern typically presents acutely with cardiogenic shock or cardiac arrest secondary to tamponade, and it often precludes treatment. The *oozing* pattern may present at a later stage with intermittent bleeding, chest pain, or acute HF.[14,21–24] Prodromal manifestations of VFWR with low

Table 1 Characteristics of ventricular free-wall rupture according to the type of rupture





Type of rupture	Oozing	Blowout
Pathological aspect	Myocardial erosion	Myocardial tear
Clinical course	Subacute (progressive bleeding)	Acute (massive bleeding)
Manifestations	Mildly symptomatic or haemodynamic instability	Cardiogenic shock or cardiac arrest
Diagnosis	Emergency echocardiography or CT (pericardial effusion; tamponade)	Emergency echocardiography (tamponade; flow across the defect in free wall)
Preferred repair	Sutureless technique	Sutured technique

Figures reproduced with permission from Lorusso *et al.*[5]
CT, computed tomography

specificity include persistent chest pain, intractable vomiting, restlessness, ECG signs as persistent ST-segment elevation, and positive T-wave deflection that remains for 72 h after the onset of chest pain.[23] *Table 1* highlights the main characteristics of VFWR according to the type of rupture.

Diagnosis and diagnostic workup

Ventricular free-wall rupture should be suspected in any patient with haemodynamic instability after AMI, particularly in the setting of delayed reperfusion. Since patients may rapidly deteriorate, the diagnostic workup of VFWR requires rapid identification and treatment. The diagnosis of VFWR is best established by emergency transthoracic echocardiography (TTE)[13] identifying pericardial effusion or tamponade (*Figure 1A*).[25] When the diagnosis of VFWR is not definitive, transoesophageal echocardiography (TEE) can identify even small pericardial collections. In stable patients, cardiac computed tomography (CT) (*Figure 1B*) and/or cardiac magnetic resonance (CMR) can provide essential information to confirm the diagnosis and delineate anatomy when uncertain.[26] Cardiac CT additionally offers a comprehensive view of the thoracic cavity, providing a broader differential diagnosis vs other life-threatening conditions, such as aortic dissection or pulmonary embolism. Contrast-enhanced CMR offers superior tissue characterization, delineating the extent of myocardial damage, pericardial effusion, and any associated haemorrhage.[27] Despite these advantages, the use of CMR is often constrained by its availability, duration of the exam, and contraindications in unstable conditions, which is not uncommon in patients with post-AMI VFWR.[28]

In patients with unknown coronary status, the opportunity to perform coronary angiography before surgery to assess revascularization options depends on the degree of haemodynamic instability. Additionally, contrast ventriculography showing extravasation of the contrast to the pericardium is outdated, considering the other diagnostic modalities available.[29]

Management

Medical therapy and other management modalities

Medical therapy aims at patient's stabilization during the acute phase, as a bridge to surgery. The initial management of VFWR requires invasive blood pressure monitoring, frequent bedside echocardiographic assessment, and central venous line for proper drug administration. To reduce tension on damaged myocardium and avoid expansion of the rupture, strict blood pressure control and balanced use of inotropic drugs to avoid hypoperfusion are paramount. In the presence of haemodynamic instability, bedside pericardiocentesis under direct echocardiographic guidance may be considered. The primary goal is immediate stabilization as a bridge to emergency surgery. Of note, pericardiocentesis may increase intrapericardial bleeding by dislodging the covering thrombus and is unlikely to be helpful in cases where the pericardial space is occupied by fully organized clots.[21]

Temporary mechanical circulatory supports, mainly veno-arterial extracorporeal life support (V-A ECLS), have also been advocated for haemodynamic stabilization and maintenance of organ perfusion, particularly in cases of cardiac arrest or profound haemodynamic instability. Veno-arterial extracorporeal life supportmay provide time to carry out preparatory surgical steps before the repair but should not prolong the initiation of emergency surgery unless facing the above-mentioned extreme clinical scenarios. In contrast, micro-axial flow pump (mAFP) should be considered contraindicated due to the risk of interfering with the ongoing myocardial rupture.[14,21]

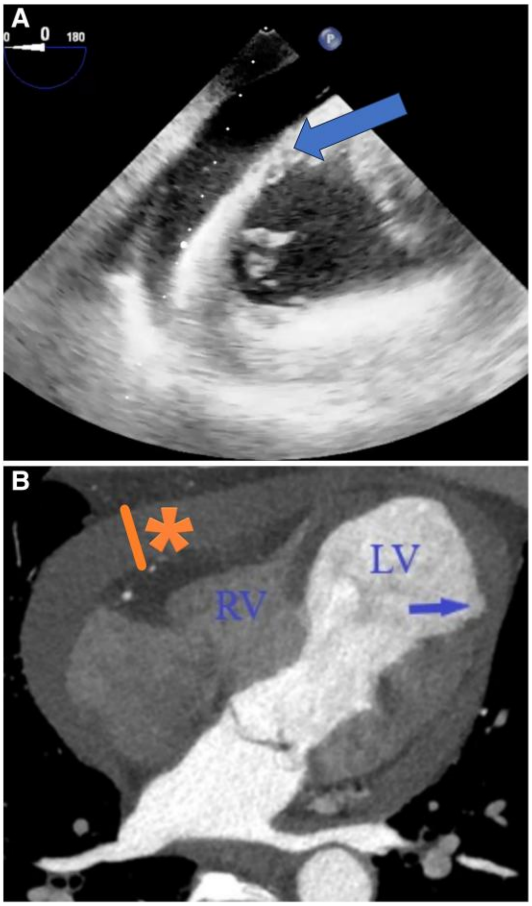


Figure 1 (*A*) TTE demonstrating left VFWR (arrow) with pericardial effusion. By courtesy of Alvaro Perazzo (Department of Cardiovascular Surgery of the Heart Institute at the Clinics Hospital of the Medical School of University of São Paulo, Sao Paulo, Brazil). (*B*) Cardiac computed tomography showing left VFWR (arrow) with contrast medium tearing from the left ventricle (LV), causing significant pericardial effusion (upper line with asterisk), which compresses the right ventricle (RV). TTE, transthoracic echocardiography; VFWR, ventricular free-wall rupture

Indications and timing for intervention

The *hyperacute form* of VFWR usually presents as a sudden catastrophic event with electromechanical dissociation and death following within minutes. Treatment attempts are usually futile in this context, although anecdotal successful reports of emergent pericardiocentesis and surgery have been published.[30] In the *subacute form*, patients with VFWR usually present with a narrow therapeutic window, generally minutes or few hours, for intervention.[21] Thus, the diagnosis of VFWR represents an indication for emergency surgical evaluation and potential intervention.

Concomitant coronary revascularization of non-necrotic areas should be considered after weighing the risks of prolonging surgery vs the benefit of coronary artery bypass grafting (CABG). Revascularization of non–infarct-related coronary arteries to avoid further deterioration in left ventricular ejection fraction (LVEF) and to improve long-term outcomes has been suggested in patients with multivessel coronary artery disease.[31,32] However, further investigations are warranted to define the optimal strategy of concomitant coronary revascularization.[32]

Surgical techniques

Principles guiding surgical VFWR repair include relieving cardiac tamponade, closing/covering the tear, and stopping any bleeding (*Figure 2*). Careful judgement of myocardial tissue and avoiding heart geometry distortion are necessary to prevent tension, repeat-rupture, or VPsA formation.

The choice of surgical repair mainly depends on the rupture type, its location, the quality of surrounding tissue, and the presence of concomitant disease.[21] Two different surgical approaches can be applied to treat VFWR depending on whether or not sutures are used: *sutured* (ST) or *sutureless* (STL) techniques (*Figure 3*).[33] Initially, only ST techniques were used, including infartectomy and linear closure with Teflon stripes (either directly or with prosthetic patch) or covering the defect with either a patch or with biological glue. The availability of effective adhesive materials (e.g. collagen sponge patches) and surgical glues has facilitated the wide adoption of STL techniques during the last two decades. Thanks to this development, the need for stitching on friable infarcted myocardium—especially in *oozing-type* VFWR—has been reduced.[21]

Resection of the necrotic area—known as infartectomy—has become less favoured and is now reserved for *blowout ruptures* with large myocardial tear or concomitant lesions, such as VSR.[34,35] Epicardial patch covering is suitable for non-actively bleeding VFWR.[36,37] Nevertheless, concerns remain regarding the permanent efficacy of STL repair. Indeed, the risk of acute re-rupture or VPsA formation generated by incomplete myocardial tear containment and recurrence of bleeding should be considered. Recently, hybrid approaches and the use of the triple-patch technique have been proposed to treat complex cases of VFWR.[38] To date, the optimal surgical technique to treat VFWR is still debated. In a recent review, STL and ST techniques showed comparable in-hospital mortality.[33] However, higher post-operative bleeding was detected in the ST repair, while recurrent rupture occurred more frequently in the STL.[33] Thus, while ST techniques have been the most traditional approach, STL repair offers potential advantages in expeditiousness, simplicity, and preservation of LV geometry. Nonetheless, further research is essential to refine these techniques and provide consistent information and confirmation on the proposed management pathway (*Figure 4*) as well as on the actual outcomes (*Figure 5*).

Post-procedural care

Post-AMI VFWR repair is associated with a high rate of recurrence, ranging from 7% to 17% within hours or a few days from the procedure, with the highest peak during patient awakening and extubation.[5,7,33] This is believed to be related to the development of hypertensive peaks, with consequent deleterious effect on the weak infarcted and recently repaired myocardium, especially in case of VFWR treated with STL technique.[33,39] As such, strict blood pressure control and rigorous pharmacological management are essential in minimizing the risk of re-rupture, bleeding, or VPsA formation.[21] Although speculative, other strategies focused on maintaining adequate LV unloading may be appropriate to reduce LV tension and enhance myocardial recovery.

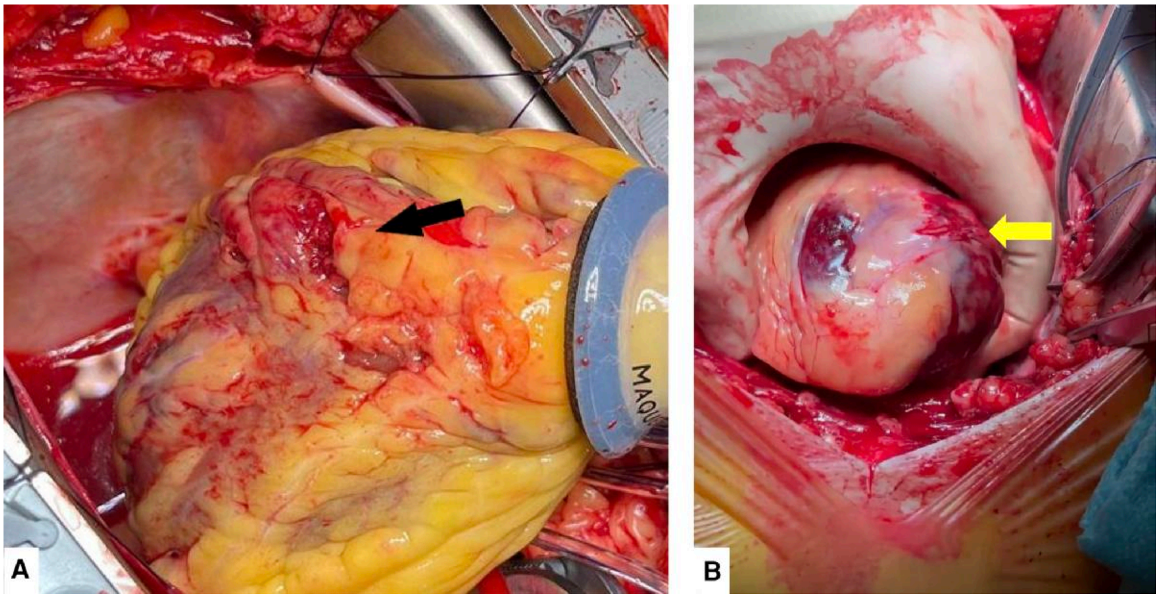


Figure 2 Intra-operative picture of (*A*) posterior blowout-type VFWR (arrow) and (*B*) oozing-type VFWR (arrow). VFWR, ventricular free-wall rupture

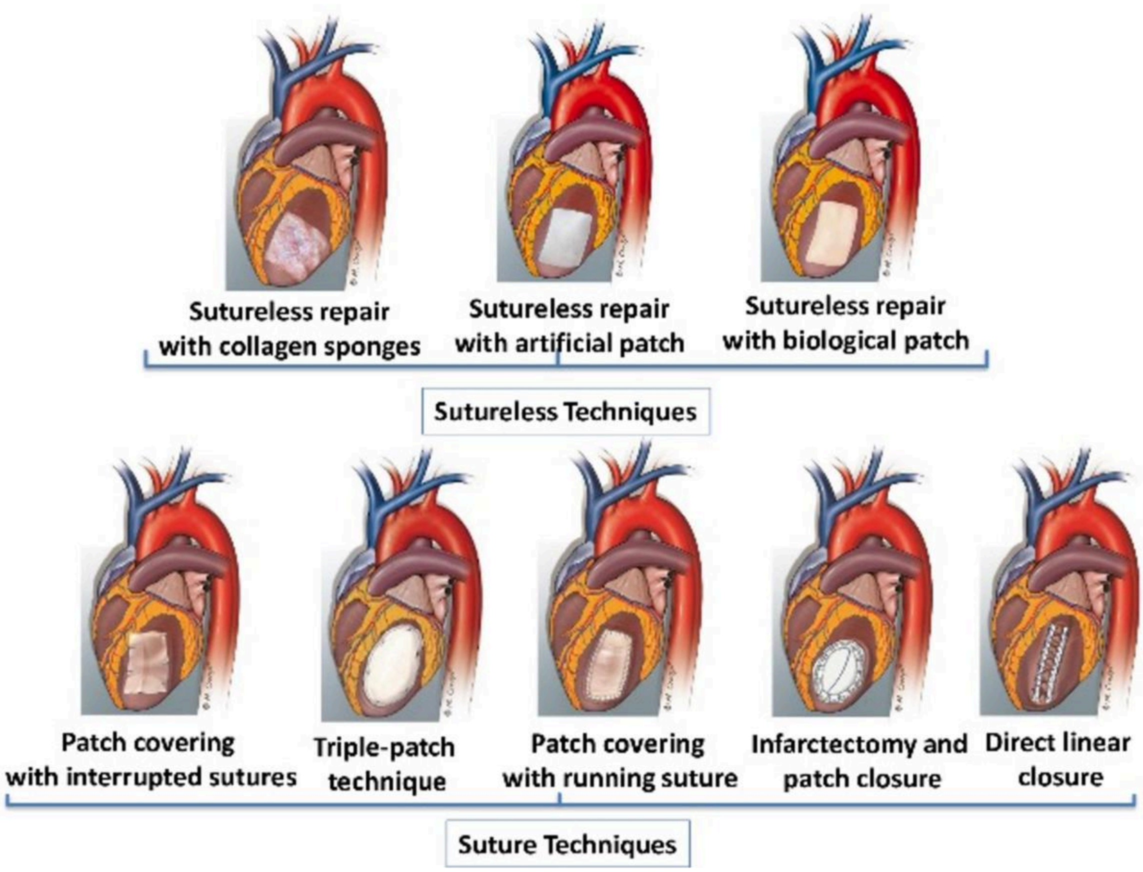


Figure 3 Techniques for surgical repair of VFWR, including the most frequently described sutured and sutureless techniques. Modified with permission from Matteucci *et al.*[21] VFWR: ventricular free-wall rupture

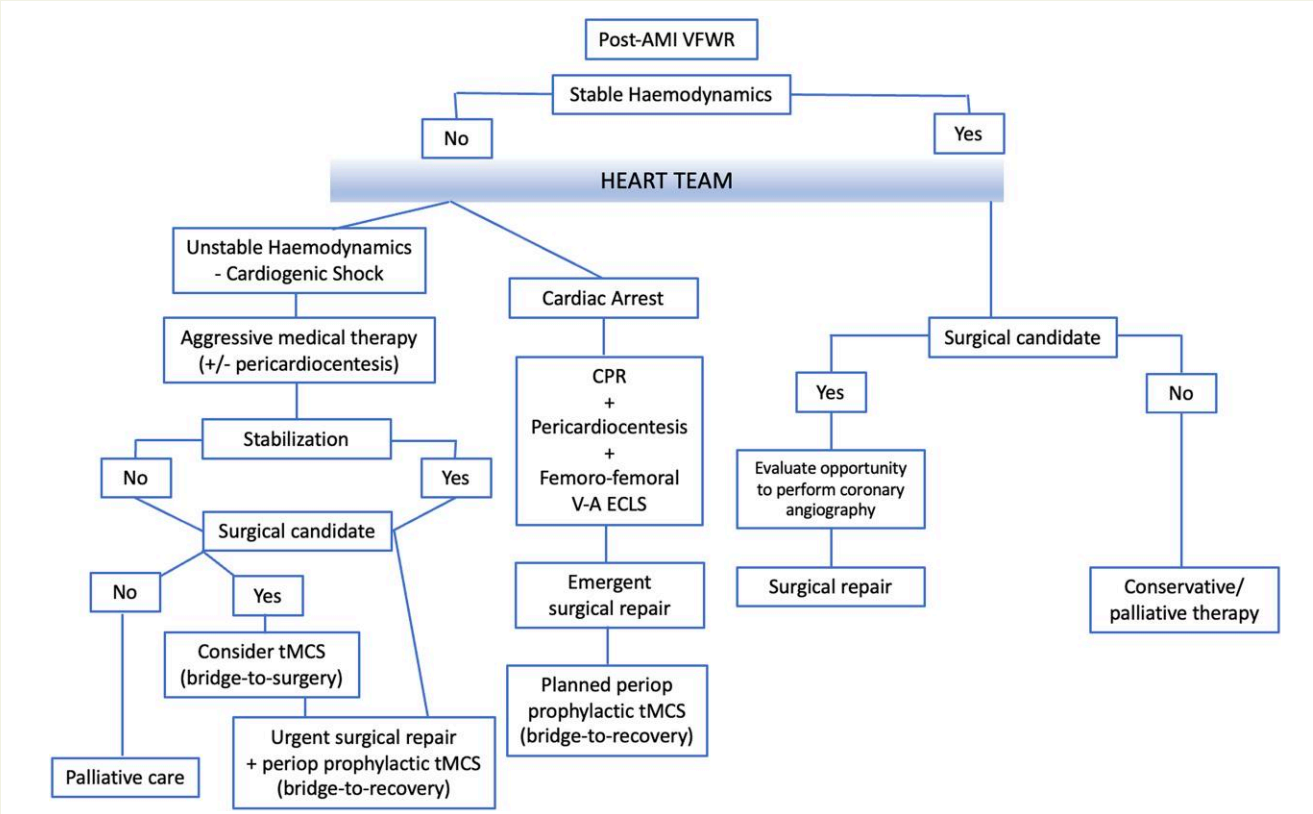


Figure 4 Flowchart for patient management in the presence of post-AMI VFWR. AMI, acute myocardial infarction; CPR, cardiopulmonary resuscitation; tMCS, temporary mechanical circulatory support; V-A ECLS, veno-arterial extracorporeal life support; VFWR, ventricular free-wall rupture

Indeed, the post-operative course after surgical VFWR repair is also characterized by a high incidence of LCOS.[7] The pre-emptive use of tMCS with concomitant LV unloading might therefore reduce VFWR recurrence as well as post-operative LCOS.

Non-surgical treatment

Although surgery remains the standard of care for VFWR, surgical risk may be prohibitive in some patients. In those individuals, available options include non-validated percutaneous approaches, such as intrapericardial fibrin glue (or thrombin) injection therapy[40] and palliative medical therapy.

Mortality rates for conservatively treated individuals exceed 90%.[41] Medical management for inoperable patients with VFWR who have recovered from the acute phase includes maintenance of fluid infusion and inotropic support.[21] The use of an intra-aortic balloon pump (IABP) may also be appropriate if not contraindicated, even in the absence of further surgical treatment.[42–46]

Once the patient is stabilized, post-intensive care follows with strict blood pressure control and initiation of the HF therapy.[21] Unlike in usual contemporary critical care, prolonged bed rest may be advised to prevent hypertensive crisis, a precipitating cause of re-rupture. Subjects who survive commonly have small tears that might close spontaneously by epicardial fibrin deposits, but potential progression to VPsA should be ruled out and monitored. Palliative care consultation can provide value-added service to the multidisciplinary team by helping determine patient-centred goals of care and assist in end-of-life discussions.

Early and late outcomes

Despite surgical repair being the gold standard, it remains a challenging operation associated with high early mortality (*Figure 5*).[47,48] In the recently published multicentre CAUTION study, the operative mortality was 36.4%, and the most frequent major adverse event after surgery was LCOS.[7] Predictors of adverse outcomes included female sex, poor pre-operative ventricular function, cardiac arrest at presentation, and V-A ECLS support.[7,14,48]

Information regarding follow-up for post-AMI VFWR is limited. According to the available data, outcomes for patients who survive index hospitalization appear more favourable than in the acute phase.[14] The CAUTION study confirmed a favourable life expectancy for hospital survivors, with 5- and 10-year survival of 79.6% and 68.8%, respectively.[3] Older age and post-operative LCOS were independent predictors of overall long-term mortality. *Table 2* summarizes the main features of VFWR as discussed above.

Ventricular pseudoaneurysm

Epidemiology

Ventricular pseudoaneurysm represents a rare, and often neglected, post-AMI mechanical complication, with more limited epidemiological data than other mechanical complication types of AMI.[8,49] Moreover, VPsA may pass unrecognized at the time of onset and be later misdiagnosed as a true LV aneurysm.[50] However,

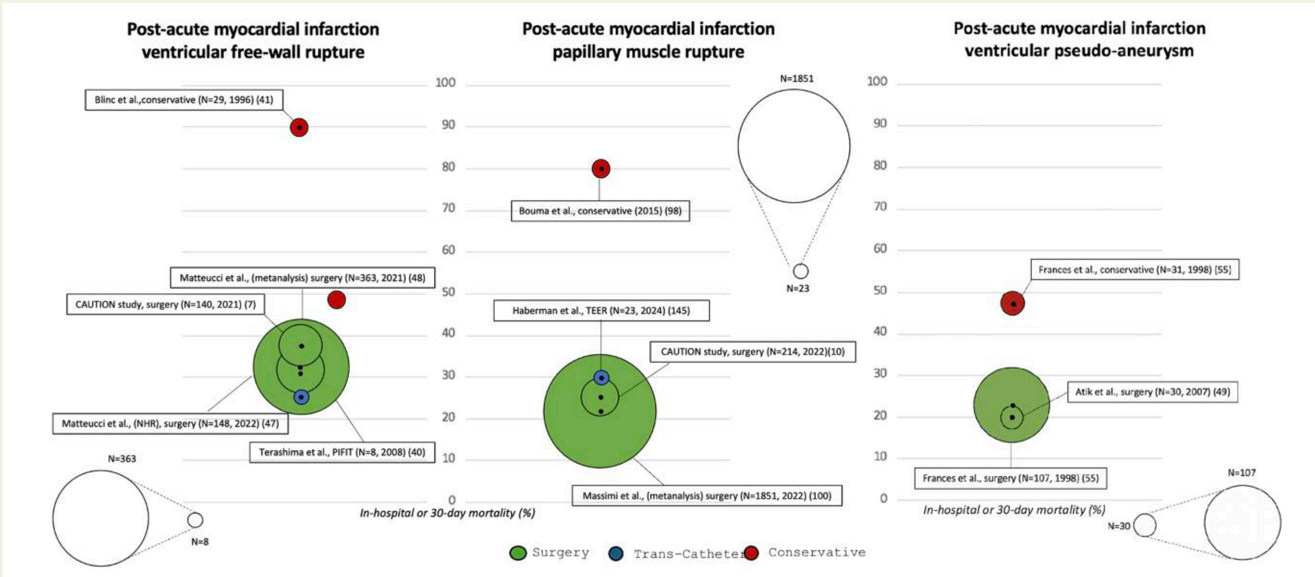


Figure 5 Treatment strategies and associated 30-day or in-hospital mortality of largest selected registries, reports, and meta-analyses for post-AMI VFWR, VPsA, and PMR. AMI, acute myocardial infarction; PMR, papillary muscle rupture; VFWR, ventricular free-wall rupture; VPsA, ventricular pseudoaneurysm

Table 2 Main features of ventricular free-wall rupture

Incidence	0.01%–0.5% of AMI cases
Definition	Whole-thickness discontinuity in integrity of myocardial free wall *Blowout* or *oozing type*
Typical clinical signs	Chest pain, dyspnoea, cardiogenic shock, cardiac arrest with cardiac tamponade
Diagnostic tools	TTE (gold standard), TEE (superior sensitivity), CMR, and cardiac CT (only in haemodynamically stable patients) Coronary angiography (if not previously performed only in haemodynamically stable patients)
Treatment options	*Surgical:* gold standard, often emergent, sutured vs sutureless techniques *Conservative:* palliation (inoperable patients), blood pressure control (non-validated percutaneous treatment options available)
Mechanical circulatory supports	*Pre-operative:* rescue or cardio-circulatory support before emergent surgery *Peri-operative:* myocardial recovery and prophylactic decrease of LV afterload to prevent re-rupture *Device choice:* IABP (if haemodynamically stable), V-A ECLS (in haemodynamic instability or cardiac arrest, LCOS or prophylactic in severe biventricular dysfunction), micro-axial flow pump (peri-operative prophylaxis)
Co-medications	Strict blood pressure control and inotropic support, as needed
Mortality	*Surgery:* 30%–35% (in-hospital), 20.4% (5-year, for hospital survivors), 31.2% (10-year, for hospital survivors) *Conservative:* 90% (in-hospital)
Most frequent complications	LCOS, re-rupture, bleeding, ventricular arrhythmias
Recurrence rate	7%–17%

AMI, acute myocardial infarction; CMR, cardiac magnetic resonance; CT, computed tomography; IABP, intra-aortic balloon pump; LCOS, low cardiac output syndrome; LV, left ventricular; TEE, transoesophageal echocardiography; TTE, transthoracic echocardiography; V-A ECLS, veno-arterial extracorporeal life support.

still representing a special subtype of cardiac rupture, the epidemiology of VPsA mirrors that of VFWR.[1,5,6] Similarly, starting from about .5% of AMI events,[51] the incidence of VPsA has decreased in the reperfusion era (.1%–.3%).[1,52] Nevertheless, given the high potential of spontaneous rupture with sudden death, its real incidence is unknown and might be underestimated.[53]

Table 3 Multi-modality identification of ventricular pseudoaneurysm

	Advantages	Disadvantages	Characteristics
TTE	• Widely available, particularly in emergency conditions • Feasible at bedside or in ICU • Doppler haemodynamics	• Limited echocardiographic window	• Narrow neck to maximal diameter (<0.5) • Turbulent flow by pulsed wave/colour Doppler
TEE	• Higher spatial resolution than TTE • Better visualization than TTE	• More invasive than TTE	• Narrow neck to maximal diameter (<0.5) • Turbulent flow by PW/colour Doppler • Contrast inflow in pouch
CMR	• Not limited by windows • Scar and thrombus visualization (LGE)	• Time-consuming • Not feasible in unstable patients	• Abrupt loss of myocardial thickness in scarred myocardium (cutoff sign) • Pericardial enhancement
CT	• 3D capabilities • Quick image acquisition • High spatial resolution • Late enhancement possible	• No haemodynamic data • Contrast media	• Abrupt loss of myocardial thickness in scarred myocardium (cutoff sign)

Modified with permission from Alexander and Ashwath.[63]
3D, three-dimensional; CMR, cardiac magnetic resonance; CT, computed tomography; ICU, intensive care unit; LGE, late gadolinium enhancement; TEE, transoesophageal echocardiography; TTE, transthoracic echocardiography.

Risk factors and localization

Clinical presentation frequently lacks specificity, with no pathognomonic signs and symptoms, thus making diagnosis challenging.[52,54] Representing a peculiar subgroup of VFWR, the risk factors for VPsA occurrence overlap. However, one of the largest series reporting characteristics of VPsA showed that older age, male sex, hypertension, non-reperfused AMIs, and inferior and lateral AMI were predisposing factors.[55] The posterior wall is the most prevalent site of VPsA (43%), followed by lateral (28%) and apical locations (24%).[5,55] This localization is relatively specific to this mechanical complication, especially if compared with true ventricular aneurysms, which more often develop from anterior AMI and rarely involve the posterolateral surface of the left ventricle.[56,57] This is probably related to the fact that anterior LV ruptures are rarely contained by the surrounding tissue and evolve more commonly into a complete VFWR.[58] Additionally, an extensive posterior infarction can also associate with PMR or VSR.[6,9,10,58–60]

Pathophysiology and clinical presentation

The VPsA wall has no endocardium or myocardium and generally contains organized thrombi. Although pericardial adhesions might contain rupture expansion, VPsA remains prone to rupture with an estimated risk of conversion to VFWR of ∼30%–45%.[5,8,61,62]

Ventricular pseudoaneurysm can be diagnosed at index hospitalization for an AMI, often presenting as acute HF. However, up to 50% of cases may be found incidentally as subacute or chronic VPsA during follow-up in asymptomatic patients or in subjects presenting with symptoms of congestive HF weeks to months, or even years, after AMI.[8,61] When diagnosed late, the VPsA wall can be fibrotic or partially calcific.[8] The usual clinical presentation of post-AMI VPsA includes chest pain (up to one-third of cases) and dyspnoea, as well as ventricular arrhythmias, syncope, and thromboembolic events.[55,61] A systolic murmur can be found in more than half of the cases either due to a turbulent flow towards and from the VPsA neck or due to concomitant MR.[55]

Diagnosis and diagnostic workup

Differential diagnosis of VPsA requires adequate imaging, especially to differentiate from true LV aneurysm. Non-specific ECG and chest X-ray abnormalities are almost always present. The diagnosis, therefore, relies on echocardiography, often supplemented with CT or CMR (*Table 3*).[5,8]

Historically, ventricular angiography was the standard for diagnosing VPsA, revealing delayed contrast distribution in the non-contracting VPsA with a distinct contour and absence of surrounding coronary arteries. However, this technique is no longer advised due to VPsA rupture and LV-based thrombi embolization risks. Nevertheless, these patients generally undergo coronary angiography at the time of AMI. In case VPsA is diagnosed in patients with previous silent AMI, if they remain haemodynamically stable, coronary angiography is advised to identify potentially stenotic coronary arteries and treat them accordingly.

As for other post-AMI mechanical complications, emergency TTE is the first-line diagnostic tool. Typical echocardiographic features of VPsA are an outpouching of the left ventricle, most often in the inferior or posterolateral wall,[57,64] with sharp discontinuity in the endocardial image at the site of communication and a narrow orifice, with the neck-to-fundus ratio of the VPsA typically measuring less than .5, as compared with true LV aneurysms (neck-to-fundus ratio of .9–1).[51] Additionally, pulsed[65] or colour Doppler imaging[66] may reveal forward and reverse flow into the VPsA. Imaging may be facilitated by contrast administration[67] or TEE.[55] Furthermore, three-dimensional (3D) echocardiography may be helpful in better understanding the anatomical extent and defining treatment strategies.[68] In haemodynamically stable patients, which is often the case of VPsA, cine-CMR is a key diagnostic technique, offering better visualization in patients with poor echocardiographic windows

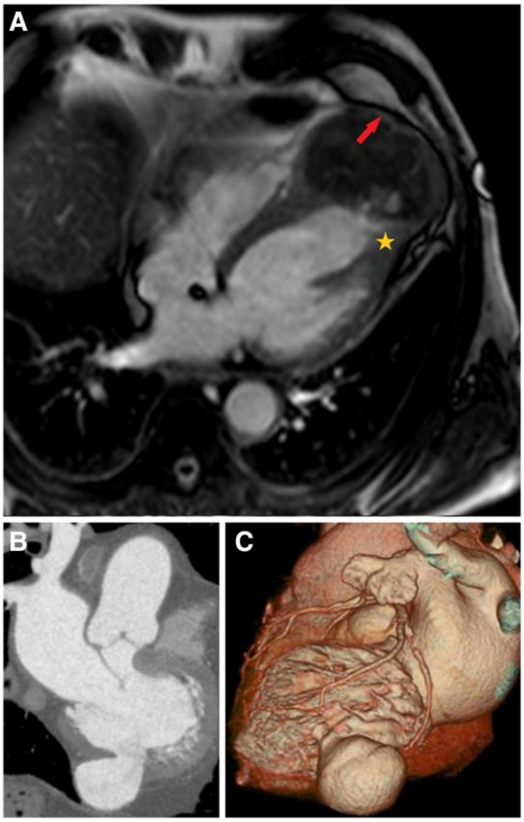


Figure 6 VPsA appearance on CMR (*A*) with fibrotic epicardial tissue lining (arrow) and transition zone from vital to fibrotic myocardium (star). Contrast-gated cardiac CT assessment of VPsA (*B*) with 3D anatomic reconstruction, showing the bulging on left ventricular wall caused by VPsA (*C*). CMR, cardiac magnetic resonance; CT, computed tomography; VPsA, ventricular pseudoaneurysm; 3D, three-dimensional

and allowing to visualize the neck of the aneurysm and flow in the pouch (*Figure 6A*). More importantly, late gadolinium enhancement allows for precise evaluation of scar tissue, place of rupture, and thrombus identification. Abrupt loss of myocardial thickness at the neck (i.e. 'cutoff sign')[69] and enhancement of the overlying pericardium represent typical VPsA features.[70] These aspects were reported to be highly sensitive and specific, also for differential diagnosis with true LV aneurysms, which generally show the integrity of the LV wall layers.[5] Cardiac CT can also precisely characterize VPsA with excellent spatial resolution and allows 3D anatomical reconstructions, presenting the advantage of faster image acquisition and being particularly useful for interventional planning, especially when transcatheter repair is indicated (*Figure 6B* and *C*).[5]

Indications and timing for treatment

Because of the rarity of VPsA, its wide clinical presentation, and the limited evidence available, the natural history and the optimal treatment are not well established.[6,21,55] The timing of surgery represents a balance between the risks of surgery and sudden rupture (*Figure 7*). Representing a subgroup of VFWR, albeit contained, all patients with a diagnosis of acute VPsA, defined as VPsA occurring within the first 2 weeks from the index AMI event, should undergo urgent or emergent surgery, which remains the gold standard, whenever operable.[5] Emergent surgery for acute VPsA is required in case of high-risk features or impending rupture (*Figure 7*). In stable patients with low-risk profile, delayed surgery up to 7–14 days could be attempted, to allow myocardial scar development and improved tissue resistance for repair. The indication and timing of treatment for VPsA are essential to prevent catastrophic complications, such as rupture and thromboembolism. Several factors must be taken into consideration by the Heart Team, including age, imaging findings (e.g. the size of the VPsA), evolution (e.g. VPsA expansion), presence of signs and symptoms, and the patient's overall clinical status (*Figure 7*). Nonetheless, due to its unpredictable clinical evolution, a low threshold for early surgery is generally advised after VPsA diagnosis.[5,6] In all operable patients showing symptoms or diagnosed with a large (≥3 cm) VPsA within 2–3 months after AMI (i.e. subacute), early surgical repair is advisable (i.e. within the following 48 h).[71] In chronic cases found incidentally, the indication is more controversial, especially for asymptomatic patients with small (<3 cm) VPsA and no evidence of expansion or calcific evolution of the false cavity (*Figure 7*).[8,61,72] In these cases, both conservative management as long as the patient remains asymptomatic[55,57,73–75] and surgery due to the potential risk of rupture at follow-up, albeit very low, have been described.[49,76] Considering the potentially fatal outcome in case of rupture, surgical repair should be indicated for every patient diagnosed with VPsA. Surgical repair can be usually performed with satisfactory results by experienced surgeons.[76] However, high-risk features for spontaneous rupture might be accounted for such a decision. The presence of at least one high-risk feature may advise prompt treatment (*Figure 7*). Although generally preferred, early surgery is associated with increased operative mortality.[73,77] Another frequent concern following VPsA repair is residual/recurrent communication through the VPsA neck,[78] even in case VPsA results from previous VFWR repair.[5,76] Moreover, ominous signs, including the appearance of pericardial fluid as well as expansion of the VPsA, should expedite the decision for surgery.[79]

Management

Surgical techniques

The first successful surgical repair of a VPsA was described in 1957.[80] Case reports and small series remain the primary source of evidence, showing generally encouraging results.[5,8,49,81] Surgical repair of VPsA aims to exclude the communication between the left ventricle and the false cavity (*Figure 8*).[5,8,82] Peripheral cardio-pulmonary bypass may be required in cases of tamponade, overt rupture, or significant rupture risk during pericardiotomy, especially in case of a reintervention or in case of conversion for patients who are supported with peripheral V-A ECLS. Ventricular pseudoaneurysm rupture may occur during dissection of pericardial adhesions, making VPsA localization difficult.[5,83] Cardioplegic arrest is commonly employed early to minimize VPsA rupture or embolic risk from thrombus mobilization. Nonetheless, induced ventricular fibrillation and on-pump beating heart have been reported.[84] Once the false cavity is opened, cautious thrombus removal is followed by

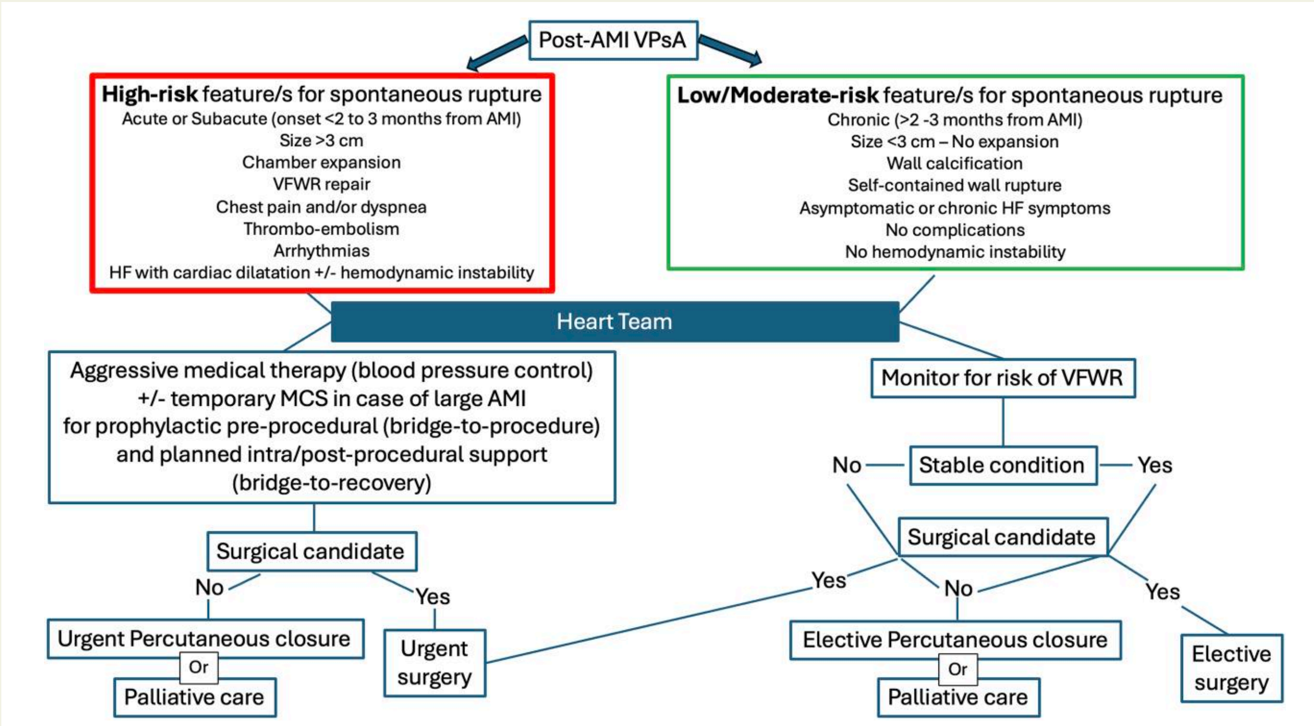


Figure 7 Flowchart for patient management in the presence of post-AMI VPsA, including the risk profile for spontaneous rupture to guide indication for urgent surgical repair. AMI, acute myocardial infarction; HF, heart failure; MCS, mechanical circulatory support; VFWR, ventricular free-wall rupture; VPsA, ventricular pseudoaneurysm

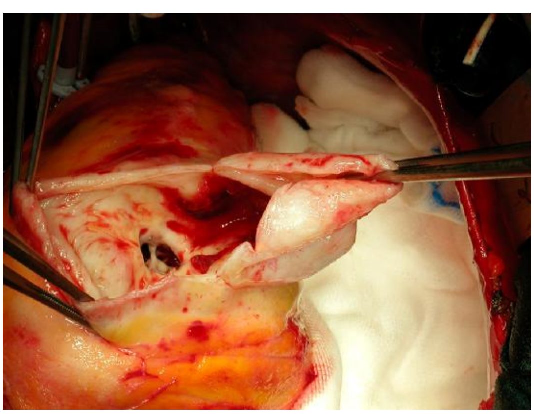


Figure 8 Intra-operative view of an opened left ventricular pseudoaneurysm with the neck shown at the centre of the opened sac

closure of the VPsA neck, usually with a felt-supported direct suture, especially in cases of scar-edged tissue, or with a prosthetic or biological patch.[75,85] For VPsA, patch repair around the internal aspect of the orifice often represents the most effective approach. The variety of surgical techniques accommodates the reported neck sizes ranging from .1 to 9 cm in diameter (median 2 cm).[55,86]

Peri-operative use of tMCS to reduce intraventricular pressure after VPsA repair and support the impaired LV function may be used, particularly in patients with previous extensive AMI and reduced cardiac function. In-hospital mortality associated with surgical repair of VPsA is quite variable according to different reports and ranges from 0% to 30%, likely influenced by local expertise, centre volume, clinical conditions at surgery, and LV function.[49,55,57,73,75,76] Concomitant CABG or mitral valve surgery is frequently reported, as needed,[73] although the role of CABG in the setting of cardiac rupture repair remains a controversial topic.[32]

Conservative management

The conservative management of VPsA includes maintenance of fluid infusion and inotropic support, as needed, prolonged bed rest, and strict blood pressure control to prevent sudden ventricular rupture.[76] However, high mortality has been reported in patients treated conservatively (48%–64%).[55,57,76] Indeed, the almost universal indication to VPsA repair is related to the high risk of rupture in the first 3 months, which may reach up to 50% in the following 2 years, even in asymptomatic patients.[61,74] However, since a high risk of stroke from VPsA thromboembolism has been reported (up to 32.5% at 4 years), life-long anticoagulation is advised for all patients.[75]

Transcatheter treatment

Percutaneous closure of VPsA may be appropriate in patients who are at extremely high surgical risk or deemed inoperable.[5,8,87] Most of the data available in literature arise from case reports or limited case series.[5,88,89] Different approaches can be proposed to close the communication between VPsA

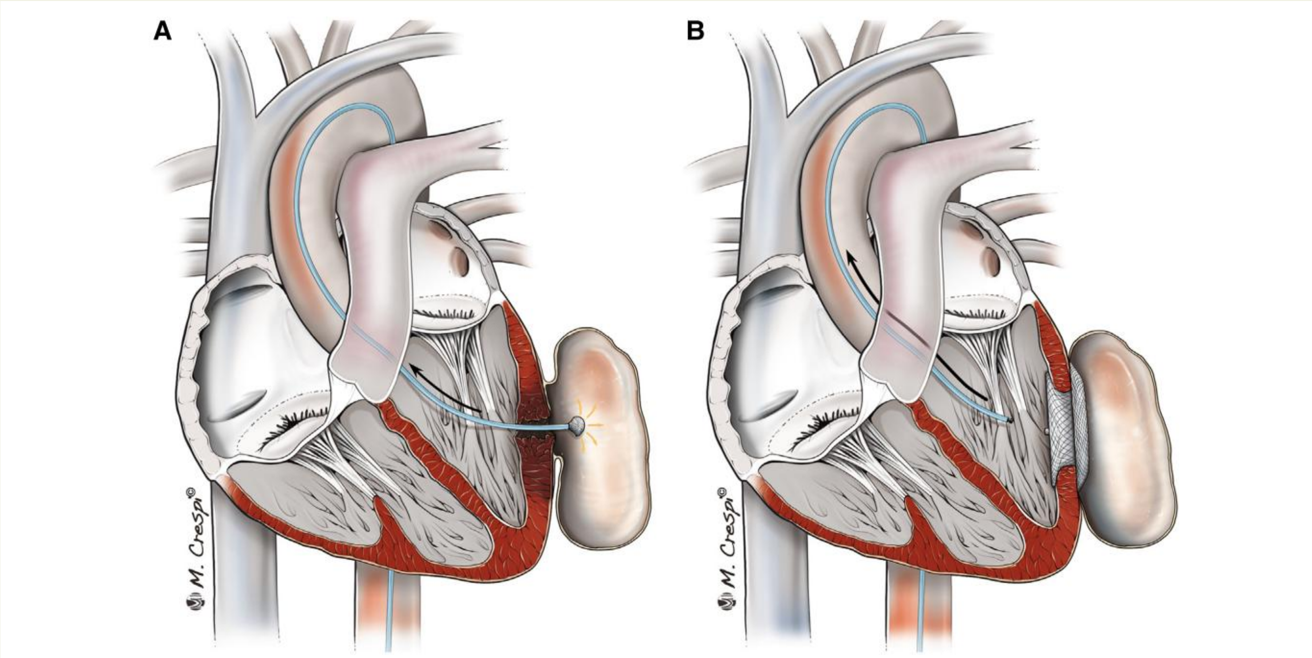


Figure 9 Transcatheter closure of VPsA with an Amplatzer device with retrograde trans-aortic approach. Modified with permission from Lorusso *et al*.[5] VPsA, ventricular pseudoaneurysm

and the true LV cavity. Currently, coil embolization and neck closure with Amplatzer occluders (*Figure 9*) are the most common techniques.[88] Coils are relatively easy to deliver and have little radial force and limited impact on the weakened LV wall but can result in incomplete closure and may be dislodged in case of large-neck VPsA. Various occluder devices (e.g. ventricular septal defect, atrial septal defect, and duct occluders) can be used, albeit sharing a common design.[90] Amplatzer-like occluders are technically more complex to implant compared with coils and require robust and well-defined neck borders (to ensure proper anchoring and prevent dislodgement) as they can damage recently infarcted, friable LV wall. Furthermore, occluders could conflict with nearby anatomical ventricular structures. Regarding device deployment, it is important to obtain a good delivery axis for the device, crucial for achieving stability. Access routes include antegrade trans-septal, retrograde trans-aortic, trans-apical, and direct thoracotomy approaches.[5,89] Hence, the optimal choice for transcatheter VPsA closure (device and route) depends on (i) VPsA dimensions (i.e. neck diameter, length and shape, and aneurysmal cavity dimensions); (ii) VPsA location and presence of adjacent structures; (iii) VPsA neck borders (i.e. well defined or not); (iv) risk of thrombus embolization; (v) accessible transcatheter access routes; and (vi) availability of adequate intra-procedural imaging for safe and effective VPsA closure. A careful pre-intervention multi-modality evaluation (including CT scan with possible 3D model reconstruction) and Heart Team discussion are mandatory to propose an individualized and appropriate strategy, especially considering that most often such an approach is offered to high-risk patients with multiple risk factors and comorbidities.[91,92]

Post-procedural care

Post-procedural care for patients who have undergone VPsA repair is key. It aims at preventing early complications in the first days after the intervention, mainly suture dehiscence with rupture recurrence. Therefore, guideline-directed HF therapy post-AMI and proper blood pressure control may help to prevent such events, when introduced in a timely manner.[44,93] Peri-procedural care should also focus on the prevention of LCOS, which is a significant risk, especially in patients operated in acute settings.[48]

Recurrence of VPsA is seen in ∼5% of treated patients, and this risk is higher in the first days after repair and in patients operated shortly after AMI, due to more fragile myocardial tissue.[55,94] The supplementary adoption of tMCS, such as IABP or trans-aortic mAFP, in addition to optimized medical therapy, might sustain myocardial recovery while decreasing afterload and LV end-diastolic pressure as well as wall stress to prevent suture dehiscence and rupture recurrence.[44] The detection of a recurrent VPsA can be challenging, as patients are often asymptomatic and haemodynamically stable or present with non-specific symptoms, including signs of congestive HF, breathlessness, chest pain, ventricular arrhythmias, thromboembolic complications, and pleuritic chest pain. Regular re-evaluation by echocardiography is required particularly during early follow-up and even in asymptomatic patients. Importantly, (recurrent) VPsA may not have a typical echocardiographic appearance. The presence of any of the above symptoms or signs must prompt comprehensive imaging (e.g. echocardiography, ECG-gated CT angiography, CMR, and LV angiography), paramount for the detection of recurrence.

Early/late outcome and follow-up management

The mortality rate associated with conservative management remains notably higher, estimated at ∼50% (*Figure 5*), whereas

Table 4 Main features of ventricular pseudoaneurysm

Incidence	0.5% of AMI cases
Definition	Free-wall rupture contained by epicardial tissue or pericardial adhesions, not involving the whole thickness *Acute*, *subacute*, or *chronic*
Typical clinical signs	Asymptomatic (50%), dyspnoea, chest pain, syncope, acute or chronic heart failure (depending on acuity of presentation), ventricular arrhythmias, thromboembolism
Diagnostic tools	TTE (first line), TEE (superior sensitivity), CMR (gold standard in stable patients), and cardiac CT Coronary angiography (if not previously performed only in haemodynamically stable patients and in chronic VPsA)
Treatment options	*Surgical:* gold standard, elective to emergent, linear suture or patch closure *Transcatheter:* high-risk or inoperable patients, coil embolization or Amplatzer-like device occlusion *Conservative:* in small, chronic, asymptomatic, calcific, low-risk VPsA, guideline-directed medical therapy for heart failure
Mechanical circulatory supports	*Pre-operative:* rescue or cardio-circulatory support before emergent surgery (acute VPsA or impending rupture) *Peri-operative:* myocardial recovery and prophylactic decrease of LV afterload to prevent re-rupture *Device choice*: IABP (in acute or subacute VPsA), V-A ECLS (in haemodynamic instability, LCOS or prophylactic in severe biventricular dysfunction), micro-axial flow pump (peri-operative prophylaxis)
Co-medications	Strict blood pressure control, inotropes as needed, anticoagulation
Mortality	*Surgery*: 20% (in-hospital) *Conservative:* 50% (in-hospital)
Most frequent complications	LCOS, wall rupture, ventricular arrhythmias, thromboembolism
Recurrence rate	5% (higher in acute VPsA)

AMI, acute myocardial infarction; CMR, cardiac magnetic resonance; CT, computed tomography; IABP, intra-aortic balloon pump; LCOS, low cardiac output syndrome; LV, left ventricular; TEE, transoesophageal echocardiography; TTE, transthoracic echocardiography; V-A ECLS, veno-arterial extracorporeal life support; VPsA, ventricular pseudoaneurysm.

post-operative in-hospital mortality may achieve 23%,[55] although recent advancements in surgical methods have improved patient outcome.[8,55] The risk of rupture decreases progressively over time, particularly for smaller VPsA (<3 cm).[8] Indeed, some investigators have shown promising survival rates in carefully selected patients who did not undergo surgery.[57,74] It is important to acknowledge, however, that the true natural progression of untreated VPsA remains uncertain, since an undetermined proportion of VPsA patients may experience sudden cardiac death due to VPsA rupture before reaching the diagnosis. Patient follow-up should be tailored based on the initial management. Strict clinical and echocardiographic monitoring is mandatory for patients managed conservatively, along with tailored rehabilitation. Suggestive symptoms warrant thorough evaluation to rule out the possibility of VPsA recurrence. Yet, since patients with VPsA or recurrent VPsA may remain asymptomatic, regular echocardiographic re-evaluation is mandatory in all patients. As per the initial diagnosis, ECG-gated angio-CT scan, TEE, and CMR represent the gold standard imaging tests for accurate monitoring, even in the presence of a negative TTE. Evaluation of mitral function is also important, because the mitral apparatus can be compromised by the VPsA location or after its repair. Finally, adherence to guideline-directed medical therapy is a measure for primary and secondary prevention.

Table 4 summarizes the main features of VPsA as discussed above.

Papillary muscle rupture

Epidemiology

Post-AMI PMR complicates .01%–.05% of AMI patients in the reperfusion era with no differences in STEMI and NSTEMI cases between 2003 and 2015 (.04% vs .05% in STEMI and .009% vs .019% in NSTEMI).[1,10] Historically, the proportion of men who had post-AMI PMR was significantly higher compared with women (over 60%), with a higher incidence in the seventh decade of life.[10,95,96] A recent sex-specific analysis of the US National Inpatient Sample database found no significant difference between sex in the occurrence of post-AMI PMR.[97] Post-AMI PMR usually develops (~80%) within 5–7 days after AMI, especially as an evolution of inferior and lateral AMI, but a delayed rupture several weeks or months after AMI has also been described.[98,99]

Risk factors and classification

More information on risk factors for PMR are derived from older patient cohorts compromising their contemporary validity.[11] Female sex, advanced age, diabetes, HF, and chronic kidney disease are risk factors for post-AMI PMR,[11,100] but it is not clear whether this holds true in the era of immediate reperfusion. PMR may frequently complicate smaller infarcts.[100,101] In patients with AMI and cardiogenic shock, LV dysfunction is more common in patients with PMR than in those without.[101]

Table 5 Post-acute myocardial infarction papillary muscle rupture main features and surgical insights

PMR: main features		
Incidence	0.01%–0.05% of AMI cases	
Definition	Rupture of papillary muscle leading to acute mitral regurgitation *Partial PMR:* A rupture involving one of the heads of the PM. MR is related to the extent of leaflet prolapse and varies according to the territory supplied by the ischaemic head. When partial PMR concerns to the main head of the PM, it is sometimes reported as an incomplete PMR.[102,103] *Complete PMR:* A rupture involving one of the heads of the PM. MR is related to the extent of leaflet prolapse and varies according to the territory supplied by the ischaemic head.[102,103]	
Clinical signs	New-onset systolic murmur, pulmonary oedema, hypotension, and/or cardiogenic shock	
Diagnostic tools	*TTE* (gold standard), *TEE* (superior sensitivity), *Cardiac CT* (in subacute settings of unexplained heart failure) *Coronary angiography* (if not previously performed only in haemodynamically stable patients).	
Treatment options	*Surgical:* gold standard, urgent, mitral valve replacement (most frequent, especially in complete PMR) vs repair *Transcatheter:* high-risk or inoperable patients or as bridge-to-surgery, transcatheter edge-to-edge repair *Conservative:* palliation (inoperable patients), supportive therapy	
Mechanical circulatory supports	*Pre-operative:* decrease afterload and MR severity, reduce lung congestion, and achieve haemodynamic stability *Peri-operative:* protected myocardial recovery *Device choice:* IABP (any pre-operative patient and low-risk post-operative patients), V-A ECLS with unloading device (achieve haemodynamic stabilization and recovery as bridge-to-surgery or post-operative), micro-axial flow pump (post-operative recovery)	
Medications	Diuretics, vasopressors, and inotropes, as needed	
Mortality	*Surgery:* 20%–40% (in-hospital), 24.8% (5-year, for hospital survivors), 46.4% (10-year, for hospital survivors) *Transcatheter:* 30%–40% (in-hospital) *Conservative:* 80% (in-hospital)	
Most frequent complications	LCOS, atrial and ventricular arrhythmias, respiratory failure, MR recurrence (after repair)	
Recurrence rate	10%–15% (after repair)	
PMR: surgical insights		
	MVR	***MVr***
Choice	≈80%	≈20%
Type of PMR	All types of PMR	Partial PMR in selected patients
Surgical technique	Replacement with mechanical or biological mitral valve prosthesis, with (when feasible) chordal sparing	Leaflet plication or resection; PM reimplantation; chordal transfer or replacement (neochordae); ‘edge-to-edge’ Annuloplasty ring always mandatory
Advantage	Reduced duration of surgery; no risk of re-PMR	No prosthesis related complications, better myocardial remodelling

AMI, acute myocardial infarction; CMR, cardiac magnetic resonance; CT, computed tomography; IABP, intra-aortic balloon pump; LCOS, low cardiac output syndrome; MR, mitral regurgitation; MVR, mitral valve replacement; MVr, mitral valve repair; PM, papillary muscle; PMR, papillary muscle rupture; TEE, transoesophageal echocardiography; TTE, transthoracic echocardiography; V-A ECLS, veno-arterial extracorporeal life support.

Papillary muscle rupture may be complete or partial, with different haemodynamic impact (*Table 5*).[102] Partial rupture is more common than complete, but this may reflect a survival bias in AMI registries.

Pathophysiology and clinical presentation

The natural history of post-AMI PMR is extremely poor, and studies in the presurgical era showed, under medical treatment alone, an in-hospital mortality of up to 80%.[101] The classic presentation of PMR occurs in the setting of inferior AMI with the appearance of a new systolic murmur, pulmonary oedema, and hypotension that may quickly progress to cardiogenic shock. A harsh and new apical systolic murmur is the typical sign of PMR but may be absent because of the rapid equalization of left atrial (LA) and LV pressures in massive MR. The ECG reveals evidence of recent infarction (Q waves) and/or ongoing ischaemia (abnormal ST-T and/or T-wave changes). However, ECG

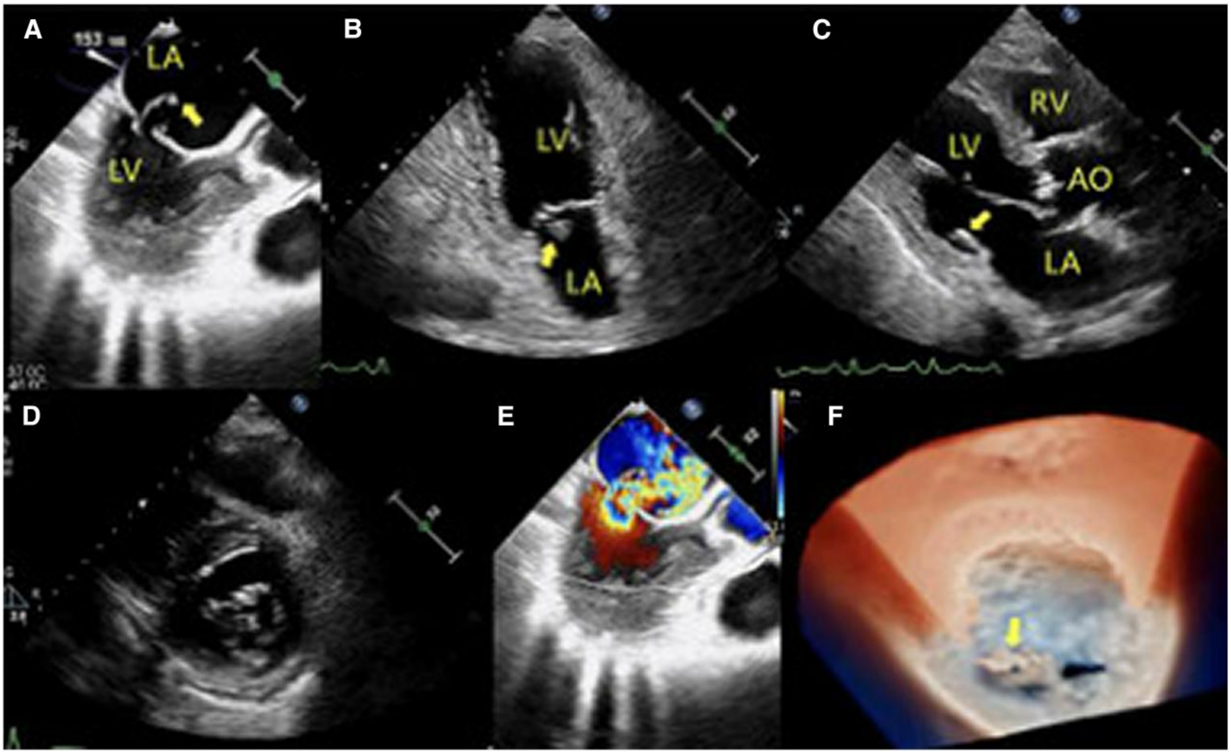


Figure 10 TTE and TEE evaluation of post-AMI PMR in the acute setting. Echocardiographic findings: mitral valve posterior leaflet flail with systolic prolapse into the LA (*A*); mobile mass attached to the chordae tendineae (arrow, *A*,*B*, *C*, and *F*), usually eccentric mitral regurgitation (indicating the leaflet involved, (*C* and *D*), with colour Doppler (*E*) and 3D reconstruction (*F*). AMI, acute myocardial infarction; AO, aorta; LA, left atrium; LV, left ventricle; PMR, papillary muscle rupture; RV, right ventricle; TEE, transoesophageal echocardiography; TTE, transthoracic echocardiography; 3D, three-dimensional

changes are often limited and not in relation with patient's haemodynamic instability. This finding is consistent with the pathologic identification of small areas of infarction affecting the area of PMs (posteromedial or anterolateral segments). Normally, the anterolateral PM has a dual blood supply from the diagonal branches of the left anterior descending and obtuse marginal branches of the circumflex coronary artery. The posteromedial PM is usually supplied by either posterolateral branches of the right coronary artery (in case of a right dominant system) or of the circumflex artery (in case of a left dominant system).[99] Therefore, posteromedial PMR is much more common (77% of PMR cases according to a recent meta-analysis[99]). Clinical presentation depends on the presence of partial or complete PMR. Complete PMR usually results in acute flailing of both the anterior and the posterior mitral leaflets. Most reports describe worse outcomes with complete PMR, probably because of its higher association with haemodynamic instability, pulmonary oedema, and more rapid development of cardiogenic shock.[99]

Diagnosis and diagnostic workup

Early PMR diagnosis is challenging but critical, considering its low incidence and high mortality.[11,104] Therefore, especially in patients with risk factors for PMR, the appearance of suggestive symptoms and signs during the first week after AMI should raise a high degree of suspicion for PMR.[96] Immediate TTE is the first-line imaging modality and can be very useful in excluding the presence of MR before the acute event and in distinguishing functional MR from PMR in the acute setting. Transthoracic echocardiography may identify a flail mitral valve leaflet or a mobile mass attached to the mitral chordae tendineae (*Figure 10A–F*). Severe, usually eccentric, MR (indicating the leaflet involved), with colour Doppler, and a cutoff sign with continuous wave Doppler may be observed. Additionally, the mechanisms of regurgitation can be confirmed in detail,[105] simultaneously evaluating the haemodynamic consequences of both AMI and PMR.[106] Transthoracic echocardiography may be challenging in some patients, owing to technical difficulties in critically ill patients, and TEE[99] may be required for final diagnosis.[107] Sometimes, the PM head prolapsing in the left atrium may be missing at TTE and TEE. The echocardiographic presentation depends on the anatomical variants of the PM and the extent of the rupture.[108] Indeed, more specific PMR features have been proposed, like any erratic motion of the PM in the left ventricle that could be properly observed in the trans-gastric view and/or an extended prolapse of the mitral leaflets into the left atrium (*Figure 10*).[108] Given the severity of PMR and the diagnostic accuracy provided by echocardiography, the role of CMR is very limited in this setting.[109,110] Cardiac CT also has a limited role in the acute setting but can be useful in patients with an unexplained new HF and a murmur and when echocardiography is inconclusive.[111] Coronary angiography should be promptly performed, if not already available from AMI treatment, according to the clinical condition, as evaluation of coronary disease is helpful in decision-making regarding approach to revascularization.[99]

Management

Principles of medical therapy

Treatment strategies are tailored to the patient's haemodynamic status, with fluid challenge reserved for the few cases of hypovolemia, particularly in the context of underlying right ventricular infarction.[112] Management of LCOS in the cardiac intensive care unit generally necessitates inotropic and/or vasopressor agents (almost 40% of patients with LVEF < 30%) alongside invasive mechanical ventilation.[99] Although the selection of pharmacological agents is predominantly empirical due to the lack of randomized clinical trials specifically addressing post-AMI mechanical complications, norepinephrine is commonly preferred as the vasopressor, while dobutamine is frequently utilized as the adrenergic inotrope. Levosimendan may be appropriate, typically combined with norepinephrine, albeit without a bolus dose. Notably, observational studies have suggested a correlation between preoperative inotropic support and poorer long-term survival in PMR patients.[98] Inotropes are generally advised for a short period as a bridge to more advanced therapies, such as tMCS and MR treatment, considering their potential to augment myocardial oxygen consumption and predispose to arrhythmias.[91,113] Nitrate administration may be indicated to reduce systemic vascular afterload in stable patients with elevated systemic vascular resistances. Anti-thrombotic therapy should be based on indications to single antiplatelet therapy with acetylsalicylic acid, dual antiplatelet therapy (e.g. previous primary PCI), or anticoagulation, with appropriate bridging to surgery according to the current guidelines.[114] Importantly, guideline-directed medical therapy for chronic HF should be avoided or withheld during the acute phase.

Treatment solutions

Surgery remains the mainstay treatment for post-AMI PMR. However, the ideal timing of surgery for these patients remains unclear. In fact, a significant portion of patients are considered too risky for immediate surgery, especially in the presence of decreased cardiac output, hepato-renal failure, severe pulmonary congestion, and metabolic acidosis.[115] In these high-risk surgical patients, alternative therapies, including tMCS and percutaneous mitral valve repair (MVr), are attractive options. However, few centres have developed sufficient expertise to manage haemodynamically unstable patients with acute severe post-AMI MR, particularly in the presence of PMR.[116] Indeed, most studies with AMI complicated by severe MR have excluded patients with PMR from the percutaneous MVr treatment.[117] In parallel, despite the remarkably high in-hospital mortality, data suggest that the application of tMCS is underutilized in patients with cardiogenic shock related to post-AMI mechanical complications, either before or immediately after the operation, despite their potentially favourable role.[3,118] Intra-aortic balloon pump still represents the first line of cardio-circulatory assistance in patients with acute post-AMI MR by improving LV forward flow, reducing myocardial workload, increasing coronary perfusion, and reducing LV end-diastolic pressure. Micro-axial flow pump represents a useful tool for intra-operative or early post-operative support with dedicated LV assistance if no respiratory support is needed. Veno-arterial extracorporeal life support provides circulatory and/or respiratory support, immediate haemo-metabolic improvement, and time for complete workup in these critically ill patients and can be used as a bridge to surgery. Furthermore, these patients are at a particularly high risk for post-cardiotomy shock and may remain dependent upon tMCS in the first postoperative days. In a large series of 3 244 799 admissions with AMI, PMR patients supported mechanically included IABP in 71.2% of cases, micro-axial flow pump in 23.3%, and V-A ECLS in 5.5%.[9] The use of tMCS was associated with reduced mortality and should be encouraged as bridge-to-surgery in unstable patients to preserve or recover organ function before definitive intervention and bridge-to-recovery after the therapeutic procedure.[112,119]

Indications and timing for intervention

Due to its potentially dramatical clinical presentation, post-AMI PMR diagnosis automatically signals the indication intervention, on top of medical support and tMCS, when appropriate. These patients must be immediately assessed by a multidisciplinary team as part of the decision-making process.[6] In general, partial PMR or sole rupture of a limited number of chordae tendineae are associated with a less dramatic clinical presentation and less MR volume. Patients with complete PMR will require immediate surgical treatment. In contrast to other post-AMI mechanical complications, a watchful waiting strategy does not add any significant advantage and is related to higher mortality due to excessive lung and secondary organ complications. Nevertheless, patients with emergency indications experience high mortality.[120] Schroeter *et al.*[121] reported on 28 patients who were diagnosed with post-AMI PMR and acute MR. The condition occurred within 48 h of AMI in 54% of the patients, and all underwent immediate surgical correction. Mortality rate at 30 days was as high as 39% in this series.[121] Low cardiac output syndrome, requiring V-A ECLS, and secondary renal failure were independent predictors of mortality. In a report by Kettner *et al.*,[122] cardiogenic shock and refraining from surgery were the only independent predictors of 30-day mortality. The authors focused on the timepoint of intervention by evaluating patients presenting with cardiogenic shock receiving immediate treatment and stable patients (operation postponed for a median of 9 days). In patients presenting with cardiogenic shock, a shorter time from onset of AMI to surgery was associated with better survival.[122]

Surgical techniques

The first cases of surgically treated PMR are credited to Adicoff *et al.*[123] and Austen *et al.*[124] For decades, a substantial proportion of the knowledge came from post-mortem studies.[103] Sixty years later, PMR is still a severe and highly morbid complication of AMI. The CAUTION study has confirmed that surgical patients present with unstable haemodynamics and cardiogenic shock in over 50% of the cases, especially in with complete PMR (60% of patients), and 80% of the patients require mitral valve replacement (MVR) (*Table 5*).

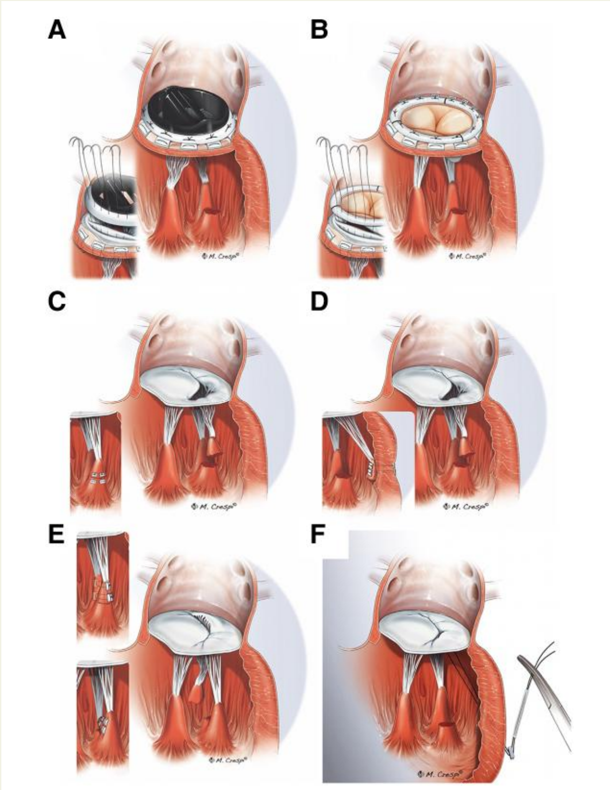


Figure 11 Mitral valve replacement due to papillary muscle rupture with biological (*A*) and mechanical (*B*) prostheses. Mitral valve repair with subvalvular techniques in papillary muscle rupture: reimplantation of the papillary muscle onto the corresponding papillary muscle site (*C*), reimplantation of the papillary muscle on the left ventricular free wall (*D*), reimplantation of the papillary muscle on the corresponding 'healthy' papillary muscle (*E*), and replacement of the corresponding chordae with PTFE sutures (*F*). An annuloplasty ring is always added to stabilize the repair. Modified with permission from Estevez-Loureiro *et al.*[9] PTFE, polytetrafluoroethylene

Mitral valve replacement

Mitral valve replacement is a well-tested, reliable option in post-AMI PMR, most frequently performed since early surgical reports (*Figure 11A and B*).[96] The main advantages of MVR are predictability and proven durability regardless of the surgical access and the type of valve prosthesis. It represents 70%–90% of practice with operative mortality in the range of 20%–30%.[3,10,126] The technique of chordal-sparing MVR is advocated.[6,125] The type of replacement prosthesis (i.e. biological vs mechanical) is still controversial.[120] Age may retain an influence on choosing the prosthesis regardless of aetiology.[127,128] The need for tMCS may also have impact on the decision due to the increased risk of valve thrombosis especially when a mechanical prosthesis is used, though large experience is still lacking.[6,104]

Mitral valve repair

Mitral valve repair is less frequently attempted in post-AMI PMR (10%–20%) as it entails substantial patient selection according to anatomy and pre-operative condition as well as tissue quality, which are usually poor.[10] Several techniques have been used, including leaflet plication or resection (*Figure 11C–F*).[123,124] Subvalvular repair techniques, including PM reimplantation at different levels, chordal transfer, or replacement accompanied by ring annuloplasty, have been attempted. Most of these experiences are limited series[129–131] or isolated reports with short, if any, follow-up.[132] The adoption of artificial chordae requires them to be secured on a PM not affected by the infarct-related scar. This may be challenging in some cases, but it can be safely used in patients with small AMI affecting only one PM head.[133]

Finally, the role of concomitant CABG in PMR patients remains controversial with regard to long-term outcomes, although it should be considered in patients with significant coronary disease.[121] Due to the scarcity of data, comparisons between MVR and MVr are unlikely to be reliable and meaningful.[134]

Transcatheter treatment

Although the surgical approach to post-AMI PMR remains the first-line treatment, it is acknowledged that patients included in surgical series are highly selected, and many PMR patients are not operated.[101,104] Therefore, the development of less invasive approaches to tackle MR in this setting, including transcatheter techniques, is paramount. Transcatheter edge-to-edge repair (TEER) has proven safe and effective for reducing MR in patients with MR at excessive risk or unsuitable for open-heart surgery, although more conclusive evidences for long-term outcomes are still warranted.[135–137] Indeed, in-hospital complications were common, with a mortality rate as high as 30%, indicating the need for additional investigations to elucidate actual potentials of such a procedure in the setting of post-AMI PMR.[9] No reports are available for transcatheter MVR in the setting of PMR.[138] Initially described as case reports, PMR can be treated percutaneously with acute MR reduction and haemodynamic improvement.[139–141] These reports refer to patients considered at unacceptable surgical risk, where TEER was offered as a 'last resort' option, also according to current guidelines.[6,142,143] The largest series of PMR treated by TEER included 23 patients turned down for surgery due to excessive surgical risk, or tMCS without improvement.[144,145] Acute procedural success was obtained in 87% of patients accompanied by rapid haemodynamic improvement. Interestingly, five patients who could be discharged received successful surgical MVR during follow-up, highlighting the potentially significant role of bridging to a better clinical condition, to undergo delayed surgery in a more stable situation. A practical approach, including the percutaneous treatment, is shown in *Figure 12*.

There are several potential advantages of a percutaneous approach to this condition. First, TEER is expected to induce an almost immediate haemodynamic improvement by relieving the amount of MR with, in turn, a decrease in left chambers and pulmonary artery pressures and increase in cardiac output, leading to a faster recovery.[144,145] Second, it can avoid the deleterious effects of the cardio-pulmonary bypass pump and post-surgical potential complications, which is paramount in high-risk or inoperable patients suffering from acute lung and myocardial damage.[146] Third, TEER does not preclude delayed cardiac surgery in case the device fails, or recurrent MR appears.[144] In fact, the role of TEER as a bridge to a lesser risk surgery is appealing (*Figure 12*).[147]

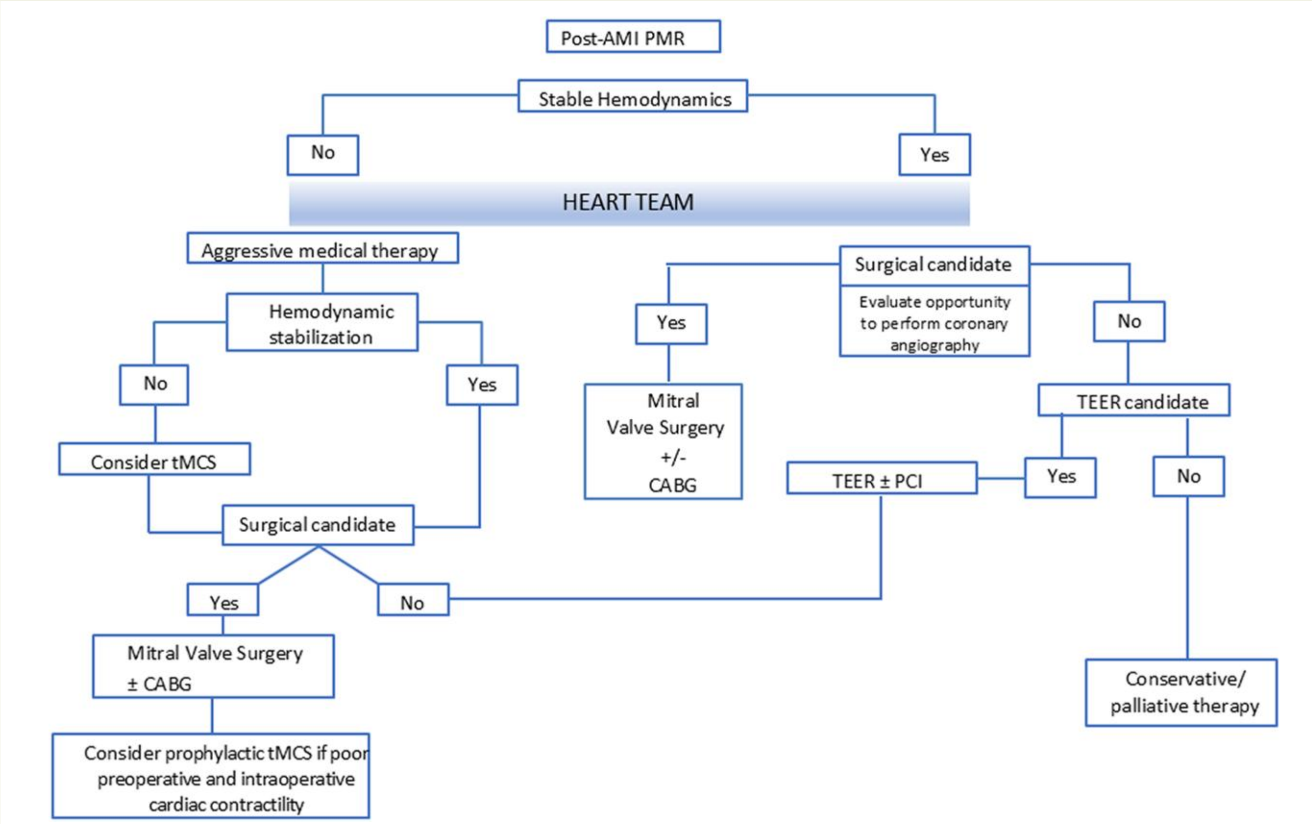


Figure 12 Flowchart for patient management in the presence of post-AMI PMR. AMI, acute myocardial infarction; CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention; PMR, papillary muscle rupture; TEER, transcatheter edge-to-edge repair; tMCS, temporary mechanical circulatory support

Pre- and post-procedural care

Some patient- and procedure-related feature assessments are advised for the planning of post-operative care, such as the type of procedure; length of time on cardio-pulmonary bypass, if used; intra-procedural complications; and patients' comorbidities.[104] However, a Heart Team can be required in certain scenarios due to the lack of evidence-based therapeutic strategies and complex decision-making. The maintenance and restoration of an adequate haemodynamic status must be the primary objective, using drugs or tMCS.[148] Transthoracic echocardiography and/or TEE must be performed post-procedurally to assess the effects of the mitral-related procedure and biventricular function.[145,148–150] Continuous ECG monitoring for early diagnosis and prompt treatment of potential ventricular and atrial arrhythmias must be provided in all cases. In haemodynamically stable patients, ventricular rate control can be obtained using beta-blockers, non-dihydropyridine calcium channel blockers, or amiodarone.[149] Conversely, in haemodynamically unstable patients, immediate cardioversion must be performed.

Monitoring for secondary coagulopathies, including early suspicion of heparin-induced thrombocytopenia, is also advised.

Early/late outcome

The mortality of post-AMI PMR is up to 80% after the first week when managed conservatively.[98,99] Despite the lack of supporting data from randomized trials, national registries, multicentre studies, and meta-analyses have shown that in-hospital mortality with early surgical treatment ranges between 20% and 40% (*Figure 5*), with LCOS, prolonged ventilation due to respiratory disease, and acute renal failure as the most frequent post-operative complications.[9,10,95,96,99] According to the largest cohort report from the USA, the main predictors of adverse outcomes associated with mortality included Hispanic race and comorbidities such as older age, hypertension, smoking, coagulopathy, and chronic kidney dysfunction. Other independent predictors of in-hospital mortality were cardiac arrest, cardiogenic shock, cardio-pulmonary bypass pump duration, and reduced LVEF.[3,10,95,96] Operative mortality increased in complete PMR, MVR, and emergent salvage status.[96,99,151] Mitral valve replacement is usually reserved for subjects with complete PMR or partial/incomplete PMR and compromised haemodynamic stability at surgery to reduce cardio-pulmonary bypass times and related risks.[99] Mitral valve repair in post-AMI PMR has a reduced risk of in-hospital mortality, but its feasibility is limited to selected patients.[96,99]

To date, the impact of concomitant CABG in PMR is debated. Several studies have demonstrated improved in-hospital mortality in patients who underwent concomitant CABG,[10] while a recent meta-analysis showed no difference.[99] The scarcity of literature data, particularly the long-term survival in concomitant CABG, cannot clearly define its specific impact on patient outcome.

Information regarding long-term results of PMR is also limited but confirms a favourable life expectancy for surgical survivors, with 5- and 10-year survival between 72%–75% and 56%–65%, respectively.[3,98] Older age, pre-operative inotropes, post-operative LCOS, and MVR without preservation of subvalvular apparatus were independent predictors of overall mortality.[3,98] Concerning TEER, the first series reported a 30% in-hospital mortality, which is still high but promising, considering that patients operated were not surgical candidates.[9,144]

Table 5 summarizes the main features of PMR as discussed above.

Special considerations

Some specific aspects in the management of post-AMI mechanical complications are common to all the types of complications, although bearing their respective peculiarities, and deserve dedicated discussion.

Role of the Heart Team and the Shock Team

Depending on the haemodynamic presentation of each patient, the Heart Team and the Shock Team are pivotal beyond procedural choice, although many cases are surgical emergencies.[152] Therefore, a Heart Team involvement is not always applicable.[6] In case of severe haemodynamic compromise, a Shock Team evaluation should be quickly involved.[6,153] The multidisciplinary team, involving clinical cardiologists, interventional cardiologists, cardiac surgeons, intensivists, experts in cardiovascular imaging, and physicians with palliative care expertise, should identify a personalized treatment pathway for each patient. Clinical evaluation of the single cases should be thoroughly carried out examining operative candidacy according to the risk profile, comorbidities, age, fragility, haemodynamic stability, severity of the complication, etc. Although surgery represents the gold standard for operable patients, the Heart Team should explore alternative individual treatment pathways, including transcatheter solutions, although based on the still currently limited experience, in case of high-risk surgery or inoperable patients. Heart Team evaluation should also encompass the overall management of these patients, including bridging strategies while waiting for intervention and palliative care for patients deemed not eligible for any type of procedure, as well as personalized management of the potential peri-operative sequelae for patients undergoing surgery.[154]

Indications for coronary angiography

Coronary angiography is a milestone in the diagnosis and management of AMI, and therefore it should be always performed unless severe, refractory haemodynamic instability requires expeditious surgery.[32,42] First, it is noteworthy that, in most cases, coronary angiography is already available since performed soon after AMI diagnosis, allowing primary PCI on the culprit lesion at least 24–48 h before the occurrence of post-AMI mechanical complications. For the remaining patients with undiagnosed AMI facing mechanical complications, the possibility to perform coronary angiography before surgical repair remains of critical importance to plan any concomitant revascularization.[32,42,44] The most controversial aspect is related to the advantage of revascularizing the infarct-related artery.[32]

Management of anti-thrombotic therapy

Considering the suggested potential impact of antiplatelet therapies and early reperfusion (including primary PCI) on the evolution of intramyocardial haematoma and possible rupture expansion, it is important to understand whether these patients might require personalized peri-operative protocols for antiplatelet therapy management in case of mechanical complication occurring early after primary PCI balancing the potential risk of rupture expansion and excessive intra-operative bleeding with that of stent thrombosis.[14,15,32,155] However, it is noteworthy that such impact of antiplatelet drugs on cardiac rupture has only been suggested by some authors, but it has not been clearly demonstrated with dedicated studies.[15,32,155] Therefore, until stronger evidence becomes available, the peri-operative anti-thrombotic management should be performed according to the current guidelines, based on the patients' baseline condition, presence of coronary stents, and other indications to anticoagulation.[153,154,156,157] Differently, for patients requiring emergent surgery, no specific modulation of these drugs is feasible, and thus the higher risk of bleeding should be managed accordingly.

Role of temporary mechanical circulatory support in post-acute myocardial infarction mechanical complications

Current European Society of Cardiology (ESC)/European Association for Cardio-Thoracic Surgery Guidelines on myocardial revascularization in the presence of post-AMI mechanical complications[42] and 2021 ESC guidelines for acute and chronic HF management in refractory cardiogenic shock,[93] and the 2023 ESC guidelines for Acute Coronary Syndrome advise the use of tMCS,[44] although conclusive and consistent evidence are still lacking in this respect, requiring dedicated investigations.

Pre-operative

Pre-operative tMCS might serve as multi-purpose management: it may provide expeditious cardio-pulmonary support in case of cardiac arrest and transfer to the operating room if indicated, and allow improvement of critical haemodynamic–metabolic conditions and shock. It may also 'protect' the patient during the transition to delayed intervention by reducing the risk of further deterioration for more favourable anatomical and functional conditions (bridge-to-surgery) and favour the decision-making about operative candidacy (bridge-to-decision). Based on the various patterns and related clinical scenarios, different timing, type, and extent of cardio-circulatory respiratory support may be warranted.[43] *Table 6* provides a comprehensive scenario of type and modality of tMCS in the presence of different clinical conditions of post-AMI mechanical complications.

Peri-procedural

Temporary MCS might also be required or maintained after surgery to provide prolonged peri-operative support, reducing intraventricular pressure and cardiac work, limiting the frequent development of LCOS, and reducing the risk of recurrences.[21] In VFWR, peri-operative tMCS, mainly IABP or

Table 6 Suggested role of temporary mechanical circulatory support in patients with post-acute myocardial infarction VFWR, VPsA, and PMR based on consensus

<table>
<tr><th></th><th>VFWR</th><th>VPsA</th><th>PMR</th></tr>
<tr><td>Intra-aortic balloon pump</td><td colspan="3">• It should be advised to stabilize and improve haemodynamic and metabolic conditions before surgery, reducing afterload, especially in patients developing haemodynamic instability or cardiogenic shock (bridge-to-surgery).[44]
• It can be added to V-A ECLS to provide adequate LV unloading.
• IABP maintenance for 48–72 h after surgery is advised to reduce LV afterload and rupture recurrence risk, especially in VFWR and VPsA patients.</td></tr>
<tr><td>Veno-arterial extracorporeal life support</td><td>• It should be advised before surgery in patients with refractory cardiogenic shock or in cardiac arrest as a rescue to allow for transfer to the operating room.[44]
• It may be advised in haemodynamic instability as bridge-to-delayed repair to allow for patient recovery and myocardial scar maturation.
• It may be advised after surgery in case of severe biventricular dysfunction to allow for myocardial recovery and prevent LCOS.</td><td>• It may be advised in case of severe myocardial dysfunction and cardiogenic shock (bridge-to-surgery).
• It may be advised in haemodynamic instability as bridge-to-delayed repair to allow for patient recovery and myocardial scar maturation.
• It may be advised after surgery in case of severe biventricular dysfunction to allow for myocardial recovery and prevent LCOS.</td><td>• It should be advised before surgery in patients with cardiogenic shock, if IABP support is not sufficient (bridge-to-surgery).[112]
• It may be advised in case of severe cardio-respiratory compromise or cardiac arrest as a rescue (bridge-to-surgery).
• It may be advised after surgery in case of severe biventricular dysfunction to allow for myocardial recovery and prevent LCOS.</td></tr>
<tr><td>Concomitant left ventricular unloading</td><td colspan="2">• It may be advised during peri-operative support (in case of peripheral V-A ECLS) to reduce rupture recurrence risk related to increased LV afterload.
• Unloading can be achieved via trans-septal or ventricular apex vent or adding an unloading device (e.g. IABP or micro-axial flow pump).</td><td>• In case of peripheral V-A ECLS, it is advised, to prevent worsening pulmonary congestion related to LV afterload increase in the presence of severe MR.[112]
• Unloading can be achieved via trans-septal or ventricular apex vent or adding an unloading device (e.g. IABP or micro-axial flow pump).</td></tr>
<tr><td>Trans-aortic micro-axial flow pump (e.g. Impella CP or 5.5)</td><td colspan="2">• It is generally not advised before surgery due to risk of rupture worsening.
• It may be advised after surgery to reduce risk of rupture recurrence and enhance myocardial recovery.</td><td>• Pre-operative adoption may be advised in patients with cardiogenic shock and acute pulmonary oedema without need for respiratory support, to allow LV unloading and circulatory support.
• It can be added to V-A ECLS to provide adequate LV unloading.
• Peri-operative adoption may be appropriate, alone or in combination with V-A ECLS, in patients with severe myocardial dysfunction (bridge-to-recovery).</td></tr>
</table>

IABP, intra-aortic balloon pump; LCOS, low cardiac output syndrome; LV, left ventricular; MR, mitral regurgitation; PMR, papillary muscle rupture; V-A ECLS, veno-arterial extracorporeal life support; VFWR, ventricular free-wall rupture; VPsA, ventricular pseudoaneurysm.

mAFP, might be advisable until just after patient extubation or for 24–48 h to achieve such goals. Escalating to V-A ECLS might be appropriate in patients with more severe haemodynamic impairment, with the advice of using a combination of LV unloading to decrease afterload and risk of VFWR recurrence (7%–17%) linked with high mortality.[7,33,39] Similar concepts apply for VPsA and PMR. Critical care management focused on maintaining adequate LV unloading is a cornerstone and should not be underestimated, potentially guiding also device selection.

Device selection

According to current ESC guidelines for acute coronary syndrome treatment, IABP should be considered in case of post-AMI mechanical complications,[44] particularly in the presence of haemodynamic instability or cardiogenic shock (*Table 6*). Obviously, the role of IABP is limited in the presence of severe cardio-circulatory compromise, requiring a more powerful tMCS. A preventive/prophylactic strategy (i.e. reducing MR extent in PMR and stabilizing haemodynamic condition in all mechanical complications) and reducing LV end-diastolic pressure (paramount in case of VFWR and VPsA) might represent useful adjuncts to actual effective circulatory assistance. The use of mAFP is receiving more attention and an increasing use, particularly in VSR, but also in PMR, which was a previous contraindication for the risk of device malfunction due to floating PM potentially impinging the suction part of the device. Also based on the recent positive trial of mAFP in post-AMI CS, such an approach might promote more frequent application of this device, but more evidence is warranted regarding patient selection and actual impact on outcome.

Post-discharge management

The limited evidence available suggests that post-discharge survival of post-AMI mechanical complications is expectedly satisfactory, although very little is known about the quality of life and recurrent cardiac events at follow-up.[3] However, since these conditions are characterized by frequent post-operative complications, adequate post-discharge rehabilitation is of paramount importance, including physical training to recover sufficient exercise tolerance. From the clinical point of view, this process should then focus on the specific sequelae that occurred after surgery. Once overcome the first months from the surgery, regular cardiological follow-up with clinical assessment should be in line with the other patients following uncomplicated AMI. Similarly, specific therapies should be established according to the current guidelines for patients affected by ischaemic cardiomyopathy. The cardiological follow-up should be better addressed by HF specialists, evaluating the trajectory of cardiac recovery, to potentially anticipate further workup in case of worsening for possible consideration of non-conventional therapies, such as heart transplant.

Sex-related difference in clinical presentation and outcome

Although female sex has been historically identified as a risk factor for the development of post-AMI mechanical complications and, in some reports, also as a predictor of worse in-hospital outcome, very few studies have directly addressed sex differences in the presentation, management, and outcome of such adverse events. Hence, although a higher rate of late-presenting AMI and other different baseline characteristics could be suggested to explain such results, as well as a recently reported lower rate of concomitant CABG at time of surgical repair of these conditions, any potential association would be merely speculative and just hypothesis-generating.[97] A higher surgical risk related to biological factors (like increased myocardial tissue fragility or myocardial ischaemia susceptibility, based on a less developed coronary collateral circulation) might be also involved in the pathophysiology and related differences in management outcome. Additional investigations are therefore still warranted to disclose the underlying mechanisms for such a disparity in outcome compared with male patients.

Palliative care

Despite several advancement in treatment modalities along the years (surgical techniques, cardiogenic shock management, the advent of new tMCS devices and application, and percutaneous and less invasive procedures), procedural futility must be taken into consideration in some patient categories, based also on the high morbidity and mortality rates in specific circumstances. Advanced age (particularly with age >80 years), the occurrence of cardiac arrest along the disease presentation with undefined cerebral injury, severe comorbidities, profound cardiogenic shock status (SCAI Stage E), and presence of above-mentioned factors in association of other cardiac diseases (previous chronic HF and the need for additional combined procedures) may represent unsurmountable barriers for a potential favourable outcome. Therefore, abstaining from further proceeding in the treatment pathway and concomitantly warranting palliative care consideration and application might be considered in these circumstances. The presence of palliative care specialists in the Shock Team, although still not common, due to the increasingly ageing population and the likelihood to more frequent chance to face such clinical decision-making, might therefore account for a valuable as well as critical role in this field of acute cardiac diseases.

Gaps in knowledge

The low incidence, severity of clinical presentation, and high related mortality make systematic enrolment of post-AMI complications in clinical trials challenging and subject to multiple potential biases, even with the aim of identifying more effective pathways of care. Furthermore, the variability in managing patients is influenced by several factors, disease-specific and patient-specific, limited experiences by multidisciplinary teams, and availability of percutaneous devices. Therefore, multiple gaps remain in the care of such entities, requiring dedicated studies, possibly randomized controlled trials, to improve the steadily unsatisfactory outcomes, despite the last decade's technological advancements. Hence, while recognizing the peculiarity of each type of mechanical complication that deserves specifically designed trials, the remaining gaps in evidence about the treatment of such conditions include the following:

- The identification of predictors for the occurrence of such post-AMI complications
- Sex differences in different types of mechanical complications with therapeutic and survival implications
- The role and timing of pre-operative as well as the prophylactic perioperative role of tMCS devices
- Most appropriate timing of intervention according to presentation (e.g. acute vs subacute or chronic VPsA)
- The impact of concomitant CABG on short- and long-term survival, especially concerning reperfusion of the infarct-related artery
- The impact and indication of different surgical corrective interventions (e.g. STL vs ST repair for VFWR or MVr vs MVR for PMR)
- Percutaneous vs surgical repair for specific groups of patients, to identify the best treatment according to the patient's risk

stratification, especially concerning TEER for PMR and device occlusion for VPsA
- The role and choice of peri-operative tMCS, also prophylactically, to prevent peri-operative LCOS due to uni- or biventricular dysfunction

Conclusions

Despite a substantial reduction in prevalence, post-AMI VFWR, VPsA, and PMR still represent clinical challenges, with persisting high in-hospital mortality rates. Although conservative and medical therapy has clearly shown a dismal prognosis, surgical treatment still shows a relatively high rate of unfavourable outcomes, mainly due to peri-operative recurrence-related or LCOS events. The advent of percutaneous techniques, particularly in the setting of PMR, appears promising, and additional multicentre and larger series are warranted to confirm such positive results. Overall, early patient triage and diagnosis, multidisciplinary decision-making, and potentially a more extensive use of tMCS in the pre- as well as peri-procedural phase reducing or preventing LCOS-related events might improve patient management by reducing those untoward complications which most frequently negatively influence patient outcomes.

Supplementary data

Supplementary data are not available at *European Heart Journal* online.

Declarations

Disclosure of Interest

R.L. received research grants from Medtronic and LivaNova and consultancy fees from LivaNova, Medtronic, and Abiomed and is a member of the Medical Advisory Board of Eurosets, Xenios, HemoCue, and ChinaBridge Medical, Chair of the ESC Cardiovascular Surgery Working Group, and Editor-in-Chief of MultiMedia Manual of Cardio-Thoracic Surgery (EACTS publishing group). S.P. is the ESC Vice-President and Chair of the ESC Advocacy Committee. M.C. received consulting fees from Terumo Aortic and Medica and is a shareholder of TEVAR Ltd. and AtriCure Medical. R.E.-L. received consulting fees from Abbot Vascular, Edwards Lifesciences, Boston Scientific, Venus Medtech, Jenscare, and Valgen and honoraria for presentations from Abbot Vascular, Valgen, and Venus Medtech. G.P. received grants from GE Healthcare, Heartflow, and Bracco and honoraria for presentation from GE HealthCare, Heartflow, and Bracco and is a member of the Medical Advisory Board for GE HealthCare, Heartflow, Alexion, and Novo Nordisk. C.H. received research grants from the Danish Heart Foundation, the Lundbeck Foundation, and the Novo Nordisk Foundation and is the ESC Vice-President. O.C. received support for meeting participation from Servier and Boehringer Ingelheim. M.A. received honoraria for presentation from Abbott Structural Heart and Edwards Lifesciences. F.G. is an Abbott full-time employee (since 7 April 2025). G.G. received research grants from AstraZeneca, Boehringer Ingelheim, Galenica, Pfizer, Menarini, Roche Diagnostics, and WinMedica; honoraria for presentations from Pfizer, Roche Diagnostics, Bayer, MSD, AstraZeneca, Boehringer Ingelheim, Janssen, Novartis, Menarini, Genesis Pharma, Sanofi, ELPEN Pharmaceuticals, Amgen, Gilead Sciences, VIANEX, Servier, Ferrer, and Gossamer Bio; and support for meeting participation from Galenica, Sanofi, Menarini, Pfizer, Bayer, Amgen, and Vianex and is a member of the Medical Advisory Board from Boehringer Ingelheim, Janssen, MSD, AstraZeneca, GSK, Sanofi, Amgen, Roche Diagnostics, Bayer, and Novartis. N.A. received research grant from Abbott Vascular; consulting fees from Abbott Vascular, Boston Scientific, Shockwave Medical, and GE Healthcare; and honoraria for presentation from Abbott Vascular and Boston Scientific. A.S. received research grants from Sanofi and Daiichi Sankyo; consulting fees from 4TEEN4, Amgen, Becton-Dickinson, and Sanofi; honoraria for presentation from Amgen, Becton-Dickinson, and Daiichi Sankyo; and support for congress participation from Amgen and Daiichi Sankyo. S.E.P. received research grants from EU, BARTS Charity, Medical Research Council, NIH, and British Heart Foundation; royalties from OUP for EACVI CMR Textbook; and consulting fees from Circle Cardiovascular Imaging and is the Chair of the DSMB of the PROTEUS Trial. K.H. is the Past-President of ESC ACVC and the Chair of ESC International Affairs. D.P. received research grants from Artivion, Corcym, and Terumo Aortic, consulting fees from Terumo Aortic, and support for meeting attendance from Terumo Aortic. F.A.K. received research grants from Bayer, BMS, BSCI, AstraZeneca, AngioDynamics, MSD, Leo Pharma, Actelion, VarmX, The Netherlands Organisation for Health Research and Development, The Dutch Heart Foundation, and the Horizon Europe programme and is the Chair of Dutch Thrombosis Network, Dutch Society of Vascular Medicine, and the ESC WG on PCRVF. F.S. received honoraria for presentations from Abbot, Medtronic, and Edwards Lifesciences and is a member of the Medical Advisory Board for Abbott, Edwards Lifesciences, Medtronic, Sanofi, Amarin, Amgen, Philips, and Shockwave. A.G.-A. received honoraria for presentation from ALNYLAM and support for meeting participation from Alnylam, Pfizer, and Novo Nordisk. B.G. received consulting fees from BSM and Novartis and is the Editor-in-Chief of *EHJ Cardiovascular Imaging*. S.A. received honoraria for Chairmanship from AstraZeneca, Roche Diagnostics, and GlaxoSmithKline and is a member of the Medical Advisory Board for GlaxoSmithKline (related to RSV vaccine in Greece). N.B. received research grants from Edwards Lifesciences and Corcym, consulting fees from AtriCure, and honoraria for presentations from Edwards Lifesciences and Medtronic. The remaining co-authors have nothing to declare.

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