Current Cardiology Reports (2024) 26:393–404
https://doi.org/10.1007/s11886-024-02042-5

ECHOCARDIOGRAPHY (JM GARDIN AND AH WALLER, SECTION EDITORS)

Echocardiography in the Recognition and Management of Mechanical Complications of Acute Myocardial Infarction

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Accepted: 11 March 2024 / Published online: 25 March 2024

Abstract

Purpose of Review Although rare, the development of mechanical complications following an acute myocardial infarction is associated with a high morbidity and mortality. Here, we review the clinical features, diagnostic strategy, and treatment options for each of the mechanical complications, with a focus on the role of echocardiography.

Recent Findings The growth of percutaneous structural interventions worldwide has given rise to new non-surgical options for management of mechanical complications. As such, select patients may benefit from a novel use of these established treatment methods.

Summary A thorough understanding of the two-dimensional, three-dimensional, color Doppler, and spectral Doppler findings for each mechanical complication is essential in recognizing major causes of hemodynamic decompensation after an acute myocardial infarction. Thereafter, echocardiography can aid in the selection and maintenance of mechanical circulatory support and potentially facilitate the use of a percutaneous intervention.

Keywords Papillary muscle rupture · Ventricular septal rupture · Free wall rupture · Pseudoaneurysm · Cardiogenic shock · Mechanical complications

Introduction

Over recent decades, substantial progress has been made in the treatment of acute myocardial infarction (AMI) due to advances in coronary reperfusion and medical therapy. The advent of fibrinolytic therapy marked the onset of a reperfusion-focused era for ST-segment elevation myocardial infarction (STEMI), leading to a noteworthy 40% reduction in the overall mortality rate [1]. The use of primary percutaneous coronary intervention (PCI) as the preferred treatment approach ushered in a further decline in both short and long-term AMI mortality [2, 3]. Moreover, widespread adoption and prompt utilization of primary PCI for AMI have also resulted in decreased rates of mechanical complications, affecting 0.27% of patients with STEMI and 0.06% of patients with non-STEMI. Unfortunately, in-hospital mortality in this population still remains extremely high—42.4% and 18.0%, respectively [4•].

The recognition of mechanical complications is complex and necessitates a high clinical suspicion. The portability, availability, and diagnostic yield of echocardiography make it the cornerstone of diagnosis (Table 1). However, inexperience or poor image quality can result in delays in establishing the correct diagnosis and subsequent treatment. This comprehensive review aims to provide a detailed overview of the use of echocardiography in the recognition and management of mechanical complications of AMI, specifically focusing on papillary muscle rupture (PMR), ventricular septal rupture (VSR), free wall rupture (FWR), and pseudoaneurysms.

Papillary Muscle Rupture

Clinical Features

While the occurrence of acute severe mitral regurgitation (MR) due to PMR has declined in the reperfusion era

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Table 1 Characteristic echocardiographic findings of mechanical complications after acute myocardial infarction

	2D/3D	Color Doppler	PW/CW Doppler
Papillary muscle rupture	Mobile/torn papillary muscle Mitral valve leaflet flail or prolapse	Large flow convergence Jets may appear *very* eccentric *Do not rely exclusively* on quantitative metrics (e.g., EROA) Vena contracta ≥ 7 mm Color Doppler splay	Densely echogenic regurgitant profile Triangular-shape profile Peak velocity < 5 m/s Pulmonary vein systolic flow reversal
Ventricular septal rupture	Defect/discontinuity of the interventricular septum • *Anterior MI*—usually simple, apical • *Inferior MI*—usually complex, posterobasal	LV to RV color flow Turbulent flow during systole in the RV *should raise suspicion for VSR*	Systolic (or continuous) flow from the LV to the RV
Free wall rupture	Defect/discontinuity of the free wall of the LV (or RV) *New* pericardial effusion, particularly with spontaneous echo contrast or echodensities Extravasated UEA (LV or RV) or agitated saline (RV *only*) in the pericardial space	Color flow through the rupture site into the pericardium	
Pseudoaneurysm	*Typically* narrow neck Associated complex pericardial effusion (particularly with spontaneous echo contrast or echodensities) Appearance of disrupted myocardium	"To-and-fro" flow, if the orifice is narrow	"To and fro" flow, if the orifice is narrow
All mechanical complications	WMA in the infarct-related territory Normal LA/LV size		

2D two-dimensional, *3D* three-dimensional, *CW* continuous wave, *EROA* effective regurgitant orifice area, *LA* left atrium, LV left ventricle, *MI* myocardial infarction, *PW* pulsed wave, *RV* right ventricle, *UEA* ultrasound enhancing agent, *VSR* ventricular septal rupture, *WMA* wall motion abnormalities

(ranging from 0.05 to 0.26%), in-hospital mortality remains notably high (up to 40%) [5, 6]. Anatomically, chordae tendinae prevent the mitral valve from everting into the left atrium during systole. These chordae are attached to two papillary muscles: the anterolateral papillary muscle, which receives dual arterial supply from both the left anterior descending artery (LAD) and the left circumflex coronary artery (LCx), and the posteromedial papillary muscle, which receives a single blood supply either from the LCx or the right coronary artery (RCA), contingent upon left or right dominance [7]. As a result, posteromedial PMR is the dominant phenotype, typically in conjunction with inferior or lateral STEMI [7].

PMR usually occurs 2–7 days after AMI. Risk factors include older age, hypertension, and single-vessel occlusion, while diabetes and prior MI are less common [8, 9]. Patients most typically present with the acute onset of pulmonary edema combined with features of cardiogenic shock, including jugular venous distension (JVD), cool extremities, and hypotension with a narrow pulse pressure. While "classic" auscultatory findings of a holosystolic murmur (heard loudest at the apex) are characteristic for *chronic* severe MR, this can be elusive in the acute setting (both due to the clinical environment and the rapid hemodynamic equalization of left atrial and left ventricular pressures). Indeed, in up to 30% of patients with acute severe MR and AMI, no audible murmur is noted [10].

Echocardiography

Bedside echocardiography is critical for confirming the presence of PMR, while excluding other etiologies of acute MR and cardiogenic shock. While transthoracic echocardiography (TTE) represents the first-line imaging modality for diagnosis, if clinical suspicion is high and TTE imaging is suboptimal, transesophageal echocardiography (TEE) should be pursued.

Two-dimensional (2D) and three-dimensional (3D) echocardiography will demonstrate a complete or partial thickness tear of the papillary muscle. Complete thickness tears will show a flail component of mitral valve leaflet attached to chordae tendinae and a mobile segment of papillary muscle

(Fig. 1, Supplemental Video 1). Conversely, partial thickness tears may demonstrate flail or prolapsed areas of the mitral valve with incomplete transection of the entire head of the muscle itself. Of note, unless the patient has pre-existing heart failure or a prior history of arrhythmia, left atrial (LA) and left ventricular (LV) sizes will typically be normal.

Even with difficult TTE imaging, color Doppler will typically clue the clinician into acute valvular regurgitation as the etiology of clinical decompensation. Large areas of flow convergence with a prominent vena contracta are often seen; however, careful attention must be paid to avoid underestimating the extent of MR in eccentric jets (Fig. 1, Supplemental Video 2). In these cases, color Doppler splay may be a clue to the presence of significant MR [11]. It is worth noting that quantitative measures for valvular regurgitation (e.g., effective regurgitant orifice area, regurgitant volume) should not be exclusively relied upon in the context of significantly abnormal hemodynamics and cardiogenic shock [12].

Pulsed and continuous-wave Doppler can further corroborate the diagnosis of acute severe MR. Systolic flow reversal in the pulmonary veins has high specificity for severe MR [13]. Continuous-wave Doppler profiles of the MR jet will typically be densely echogenic, have a low peak velocity (< 5 m/s), and demonstrate an early peaking, asymmetric, triangular shape, representing rapid equalization of the LA and LV pressures in a noncompliant LA (Fig. 1C).

Treatment

While initial (and immediate) medical management of pulmonary edema and cardiogenic shock is essential, definitive therapy for PMR is surgical. However, as previously noted, post-operative mortality may be as high as 40% [14]. As such, many patients are initiated on mechanical circulatory support (MCS) for stabilization (see "Temporary Mechanical Circulatory Support" section).

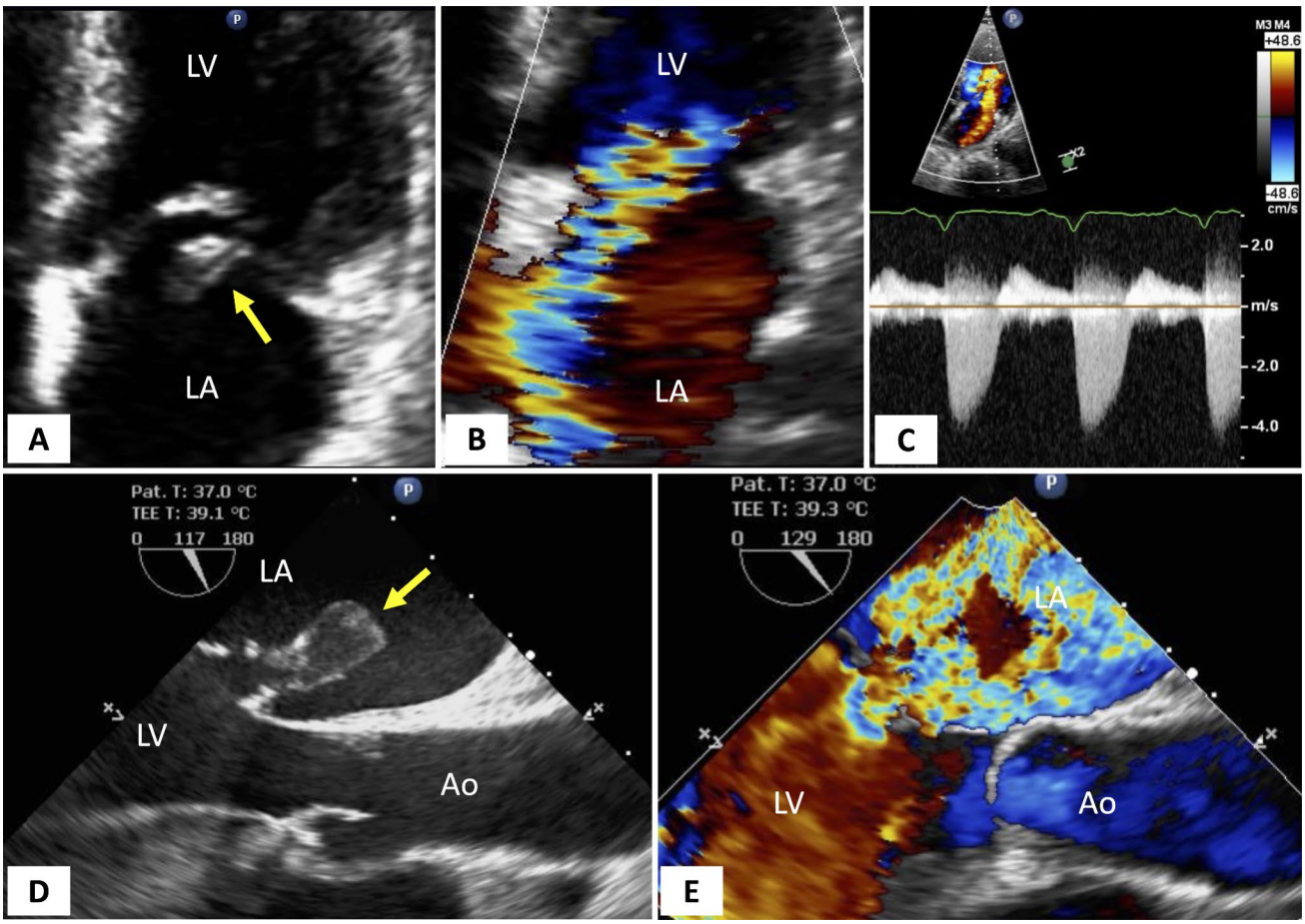

Fig. 1 **A**–**C** Transthoracic echocardiography after a delayed presentation of an acute myocardial infarction demonstrating a flail posterior mitral valve leaflet with an associated ruptured papillary muscle (*yellow arrow*). There is severe, eccentrically directed mitral regurgitation. Continuous-wave Doppler demonstrates a densely echogenic profile, with a low peak velocity (< 5 m/s) and an early peaking, asymmetric, triangular shape. **D**, **E** Transesophageal echocardiography demonstrating flail segments of the anterior and posterior mitral valve leaflets with associated chordae tendinae and a ruptured papillary muscle (*yellow arrow*). There is severe, eccentrically directed mitral regurgitation. *Ao*, aorta; *LA*, left atrium; *LV*, left ventricle

In patients who may not be suitable for surgery, transcatheter edge-to-edge repair may be a viable option, although experience is limited to case series and case reports [14]. Unsurprisingly, these cases are often technically challenging, with large flail widths and flail gaps, necessitating multiple clips. Patients are also at risk for further decompensation from an acute decline in LV function due to increased afterload when MR is successfully reduced.

Ventricular Septal Rupture

Clinical Features

Like PMR, the development of VSR is quite uncommon after AMI (approximately 0.3% of patients) [4•]. Pathophysiologically, absent (or delayed) reperfusion results in infarction of the interventricular septum and, ultimately, rupture. Studies have demonstrated that this patient population has lower rates of well-developed collaterals in the infarct territory and higher rates of single vessel disease (versus triple vessel disease) when compared to patients without VSR [15]. These angiographic patterns support the protective role of collateral support in the infarct territory. As expected, VSR is less commonly observed following revascularization through PCI compared to thrombolytic therapy and lower in patients undergoing primary early PCI as opposed to delayed intervention [16].

When reperfusion is absent or delayed, the emergence of VSR follows a bimodal distribution. The risk is notably elevated within the initial 24 h and then again between 3 and 5 days. Conversely, it is rarely observed beyond two weeks following a myocardial infarction (MI) [17]. Advanced age, anterior infarct location, female sex, lack of smoking history, hypertension, and no previous myocardial infarction are risk factors for VSR [18–21]. The lack of many traditional cardiovascular risk factors also supports the protective role of collateralized myocardium.

Clinical symptoms may vary from an incidentally discovered murmur to pulmonary edema to frank circulatory collapse, primarily depending on the size of the VSR and the degree of left-to-right shunting. During physical examination, the distinctive murmur is characterized by its harsh, pansystolic quality and is typically audible at the left lower sternal border. It may be accompanied by a precordial thrill.

Echocardiography

VSR tends to relegate to specific areas of the interventricular septum based on the location of AMI (Fig. 2, Supplemental Video 3). Specifically, anterior MI tends to be associated with defects that are simple (i.e., occurring at the same level on both sides of the septum) and apically positioned. Conversely, inferior MI is associated with complex posterobasal defects that take serpiginous routes through a hemorrhagic and necrotic septum [16, 22]. As such, conducting a meticulous 2D and color Doppler echocardiographic assessment of these specific regions becomes paramount. This may require off-axis or non-traditional imaging in order to visualize the defect. In cases where image quality may be compromised, the use of ultrasound enhancing agents (UEA) or agitated saline may aid 2D echocardiographic assessment.

Color Doppler imaging typically demonstrates a shunt from the LV to the right ventricle (RV) through the VSR (Fig. 2, Supplemental Video 4). However, should TTE imaging be challenging, the presence of turbulent systolic flow in the RV should lead to a high index of suspicion for a VSR, particularly in patients with hemodynamic instability and deteriorating clinical status. As such, careful color Doppler interrogation of the *right ventricle* (including on parasternal long axis and RV-focused apical 4 chamber views) should be undertaken after AMI.

Continuous-wave (CW) spectral Doppler assessment will demonstrate predominantly systolic flow from the LV into the RV. However, continuous (or near-continuous) shunting may be seen due to elevated LV diastolic pressure in the setting of AMI. Peak flow velocity across the site of rupture measured by CW Doppler interrogation corresponds to the pressure gradient between the LV and RV and can be employed to estimate RV systolic pressure (RVSP). In the absence of LV outflow tract obstruction or aortic valve stenosis, the systolic blood pressure (SBP) would equal the LV systolic pressure, while the RVSP is calculated by subtracting the pressure gradient between the LV and RV from the SBP.

Treatment

Given the high mortality rate associated with uncorrected defects (approaching 80% at 30 days), conservative medical therapy alone is limited to patients with hemodynamically insignificant defects or those with prohibitive surgical risk [16].

Surgical intervention stands as the definitive approach for addressing VSR, although the optimal timing remains uncertain. Findings from the Society of Thoracic Surgeons database showed that overall in-hospital or 30-day mortality for VSR repair was 43% but with a significant difference between mortality rates for repair performed before versus after 7 days from AMI (54% vs. 18%, respectively) [23]. The appropriate time to operate can be difficult to determine, as tissue friability is a concern for early repair, but delayed surgery also risks extension of the VSR and subsequent associated mortality. The improved outcome with delayed surgery may be related to one of two factors, including (1) evolution of the infarct with collagen deposition, resulting in

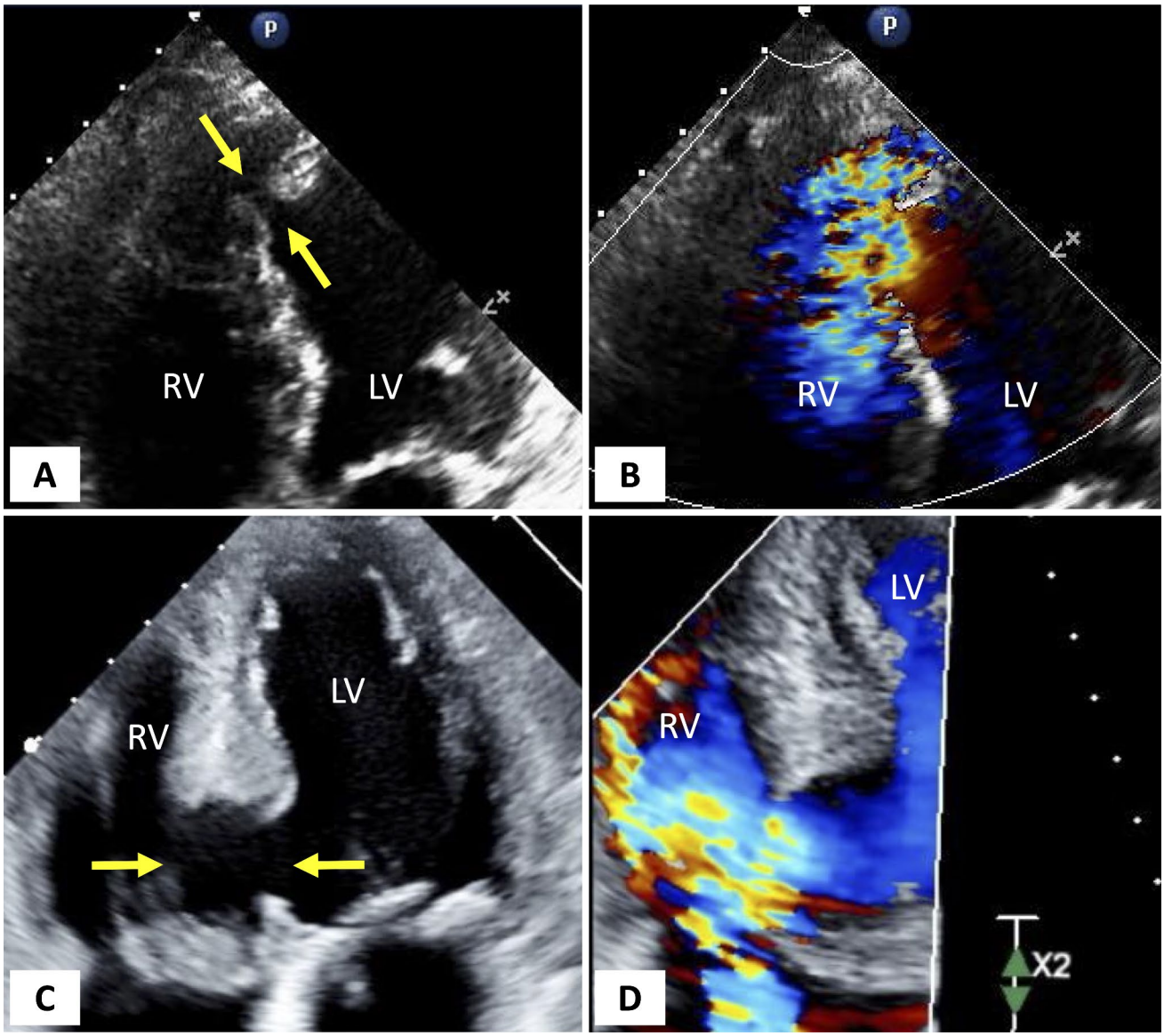

Fig. 2 **A**, **B** Transthoracic echocardiography demonstrating ventricular septal rupture with a simple defect of the apical septum (*yellow arrows*) after a delayed presentation of an LAD ST-segment elevation myocardial infarction. Note the turbulent flow in the RV, a result of high velocity left-to-right shunting. **C**, **D** Transthoracic echocardiography demonstrating ventricular septal rupture with a very large defect of the basal inferoseptum (*yellow arrows*) after a delayed presentation of an RCA ST-segment elevation myocardial infarction. Again, note the turbulent flow in the RV as a result of left-to-right shunting. *LAD*, left anterior descending artery; *LV*, left ventricle; *RCA*, right coronary artery; *RV*, right ventricle

greater stability of the cardiac tissue allowing a more effective repair, or (2) survival bias (i.e., higher-risk patients with marked hemodynamic instability are operated on more emergently). Like those with PMR, MCS has been utilized as a bridge for stabilization and is increasingly used to delay surgery to improve rates of technical success (see "Temporary Mechanical Circulatory Support" section).

For patients deemed unsuitable for surgical VSD repair due to excessive risk, TEE-guided percutaneous closure has emerged as a treatment option (Fig. 3). Despite a procedural success rate of approximately 89% in specialized centers (varying between 80 and 100%), the associated in-hospital/30-day mortality is roughly 32% [24]. In hopes of mitigating the aforementioned, some experts recommend delayed percutaneous intervention to allow for tissue fibrosis and stability.

Patient selection and device sizing is highly dependent on the morphology and location of the VSR due to technical feasibility. Anatomical studies have demonstrated that only 22% of VSR defects are exclusively confined to the septum

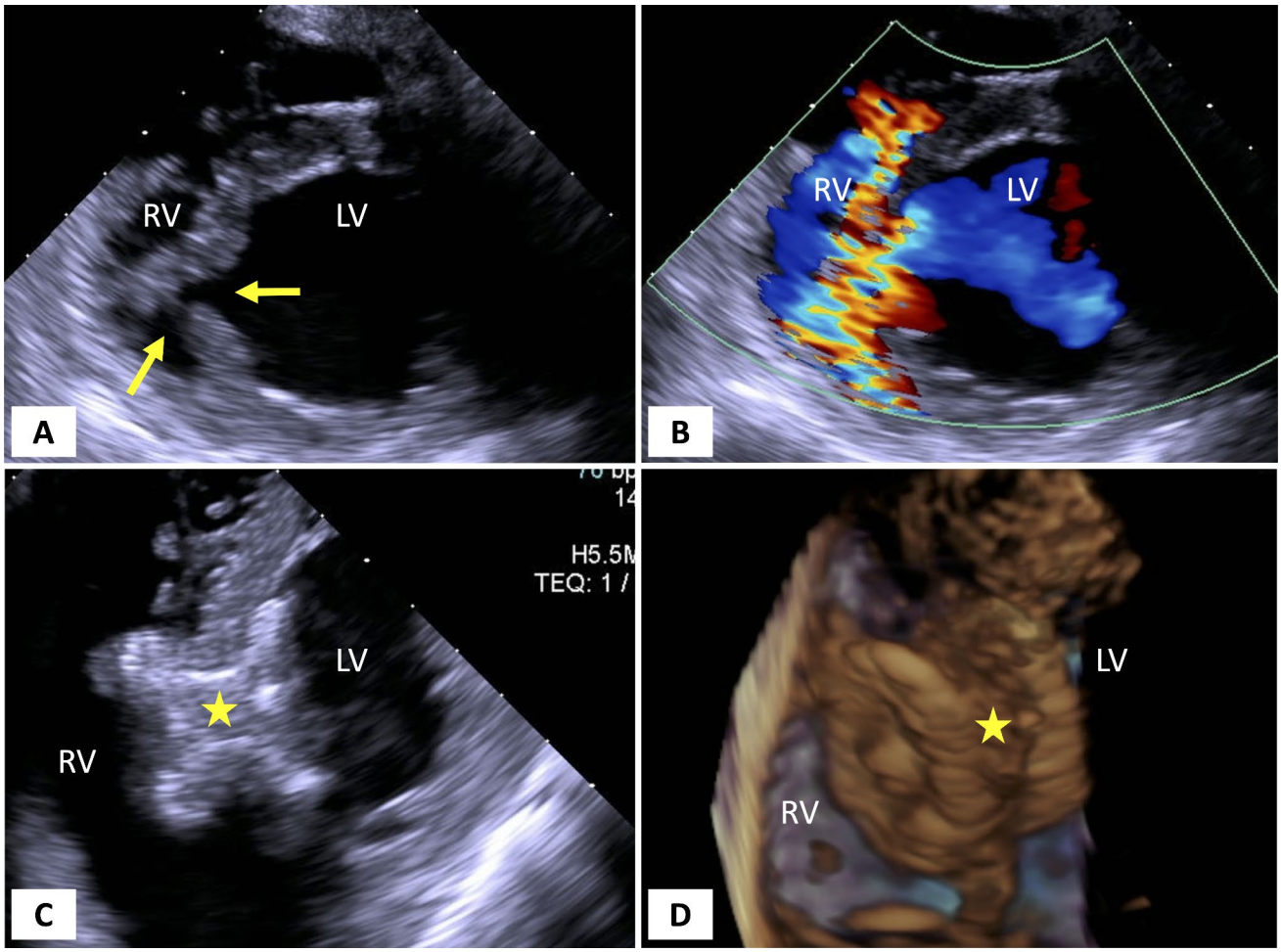

Fig. 3 **A**, **B** Transesophageal echocardiography demonstrating a simple ventricular septal rupture (*yellow arrows*) with left-to-right flow by color Doppler. **C**, **D** Percutaneous closure with a 24-mm Amplatzer post-infarct muscular VSD occluder device (*yellow star*) as seen by 2D and 3D transesophageal echocardiography. *LV*, left ventricle; *RV*, right ventricle; *VSD*, ventricular septal defect

and only 50% of defects can be filled by the stem of the largest available Amplatzer Muscular VSD occluder device (St. Jude Medical; St. Paul, MN) [24]. Percutaneous VSR closure is also associated with complications in up to 41% of cases, including device embolization, arrhythmias, hemolysis, and incomplete VSD occlusion necessitating subsequent surgical intervention [25]. Finally, TEE guidance for VSR closure is complex and requires specialized structural knowledge for echocardiographically guided sizing and positioning.

Free Wall Rupture

Clinical Features

While historically described as the most common mechanical complication after AMI, the actual incidence of FWR remains elusive due to instances that manifest as out-of-hospital sudden cardiac death combined with the lack of routine autopsies. FWR predominantly occurs within the first 7 days following AMI, most commonly 3–5 days after initial symptoms [26].

In a study that evaluated predictors of FWR in reperfused AMI, multiple characteristics were identified, including age > 70, female gender, anterior location, and treatment more than 2 h after symptom onset [27]. Treatment with PCI was also associated with a lower risk of FWR compared to thrombolysis [27]. Interestingly, in the SHOCK trial registry, patients with FWR were less likely to have a history of MI, raising the possibility that collateral circulation may offer myocardial protection [28].

Clinically, most patients will experience a sudden hemodynamic decompensation or cardiac arrest (the classically associated rhythm being pulseless electrical activity). In some cases, a friable infarct zone is associated with an oozing bloody pericardial effusion, which may manifest

as a more gradual or delayed presentation. Given that FWR is inexorably linked to hemopericardium, signs and symptoms of pericardial irritation or tamponade should prompt an immediate evaluation (e.g., sudden onset chest pain, muffled heart sounds, new jugular venous distension, and diffuse ST-segment elevation on electrocardiogram).

Echocardiography

The diagnosis of FWR is most readily established by echocardiography. Characteristically, the presence of a new complex pericardial effusion with spontaneous echo contrast or echodensities suggestive of thrombus is diagnostic (Fig. 4, Supplemental Video 5). However, the presence of

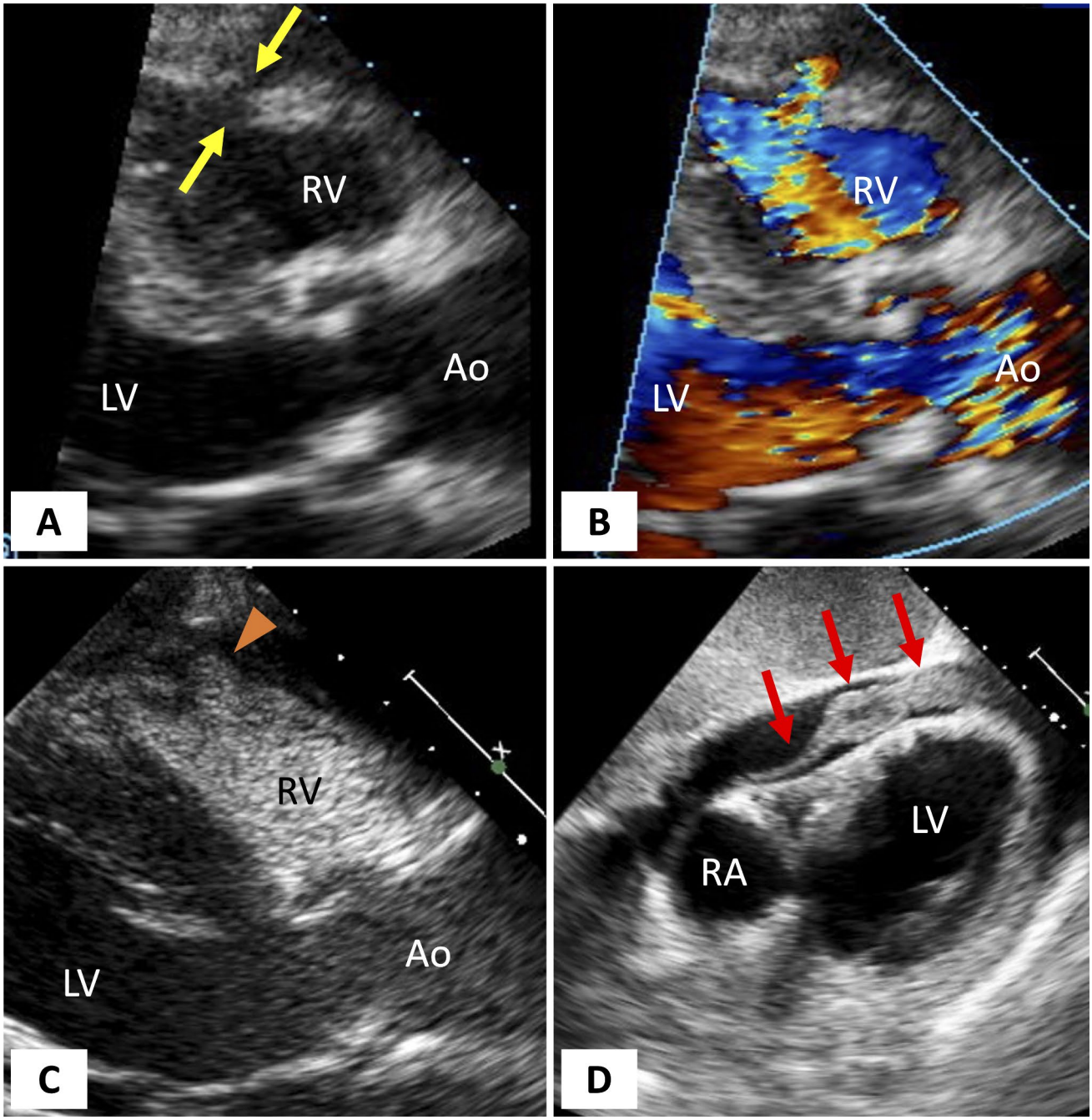

Fig. 4 **A**, **B** Transthoracic echocardiogram showing right ventricular free wall rupture (*yellow arrows*) with color Doppler demonstrating flow through the defect. **C** With the injection of agitated saline, microbubbles can be seen passing through the right ventricular wall and into the pericardial space (*orange arrowhead*). **D** Echodense material in the pericardial space (*red arrows*), consistent with hemopericardium due to free wall rupture. *Ao*, aorta; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle

any pericardial effusion on TTE accompanied by sudden hemodynamic compromise following an AMI should raise suspicion for the diagnosis. Echocardiographic features of tamponade may also be present and include right atrial collapse, diastolic collapse of the right ventricle, a dilated and non-collapsible inferior vena cava (IVC), and marked respiratory variation in mitral and tricuspid valve inflow velocities.

Color Doppler can be a useful adjunct in attempting to localize the rupture site (usually located at the juncture of normal and infarcted myocardium) and typically demonstrates blood flow through the site of rupture and into the pericardial space (Fig. 4, Supplemental Video 6). However, these are often difficult to locate or may be sealed over (with thrombus) by the time of assessment. In cases where diagnostic uncertainty arises, UEA may be employed [29••]. Agitated saline contrast can also be used, but should be reserved for the primary assessment of RV rupture (Fig. 4). The detection of extravasated microbubble contrast within the pericardial space subsequently confirms the diagnosis of FWR. FWR may culminate in the development of a pseudoaneurysm due to containment facilitated by epicardial clots or pericardial adhesions.

Treatment

While emergency surgical repair is the definitive treatment of choice of FWR, in-hospital mortality is substantial (35–66%) [29••]. For patients who suffer a cardiac arrest or for those who need temporary (i.e., peri-operative) stabilization, emergent MCS with venoarterial extracorporeal membrane oxygenation (VA-ECMO) should be considered (see "Temporary Mechanical Circulatory Support" section). Medical therapy, including the use of pericardiocentesis for hemopericardium, is associated with abysmal outcomes, with in-hospital mortality reaching up to 90% [30, 31].

Left Ventricular Pseudoaneurysm and Aneurysm

Clinical Features

A pseudoaneurysm represents a FWR that is contained by the pericardium and fibrous tissue [7]. It typically involves the inferior or lateral walls and is hypothesized to result from the formation of dependent pericardial adhesions in patients convalescing in a recumbent position after AMI [22]. For patients who do not immediately decompensate from FWR, pseudoaneurysms can elude diagnosis for extended periods, stretching into months or longer. Evidence suggests that the risk of pseudoaneurysm rupture is elevated during the initial 3 months following an infarction, subsequently transitioning into a chronic and stable state, exhibiting a propensity to remain intact over the course of several years [32]. As such, patient presentations vary widely, from incidental discovery to symptoms of chronic heart failure.

LV aneurysms, on the other hand, are thinned, dyskinetic areas involving all three myocardial layers [7]. Compared to pseudoaneurysms, more than 80% of LV aneurysms are situated anterolaterally in proximity to the apex and are typically associated with complete occlusion of the LAD in the absence of collateral blood supply [7]. Roughly half of individuals with moderate or large aneurysms experience heart failure symptoms, either with or without concurrent angina. Around 15% present with symptomatic ventricular arrhythmias, which may prove refractory and be life-threatening [7]. Mural thrombi are discovered in nearly half of individuals diagnosed with chronic LV aneurysms, with systemic embolic events more likely to manifest shortly following AMI.

Echocardiography

Differentiating between LV pseudoaneurysms and true aneurysms can be challenging. On 2D echocardiography, pseudoaneurysms will typically have a "narrow" orifice/neck (i.e., less than half of the maximal diameter of the outpouching) (Fig. 5, Supplemental Video 7) [22]. Unfortunately, the maximal size of the defect itself cannot delineate the two, as pseudoaneurysm diameters have been shown to vary widely (from 1.5 to 20 cm) [33]. Signs of contained free wall rupture may also be present, including discontinuities in the myocardial wall or an adjacent pericardial effusion containing echodensities or loculations (although the latter may easily be confused with an unrelated complex pericardial effusion). 3D TEE may provide enhanced visualization of the anatomy of the pseudoaneurysm and provide insights into the possibility of percutaneous closure [22].

While color and spectral Doppler will show flow in and out of both pseudoaneurysms and aneurysms, the former may demonstrate turbulence due to the (typically) narrower orifice, typically referred to as "to-and-fro" flow (Fig. 5C, Supplemental Video 8). In certain instances, an echo-free space adjacent to the left ventricle may be present, but the abnormal communication might not be distinctly visible. In such cases, employing color Doppler and UEA may be helpful.

Of note, while many of the aforementioned features may strongly suggest a pseudoaneurysm, no individual echocardiographic criteria exist with sufficient specificity to definitively differentiate it from a true LV aneurysm. As such, cardiac-gated computed tomography (CT) imaging and cardiac magnetic resonance imaging (MRI) are valuable adjuncts in diagnosis.

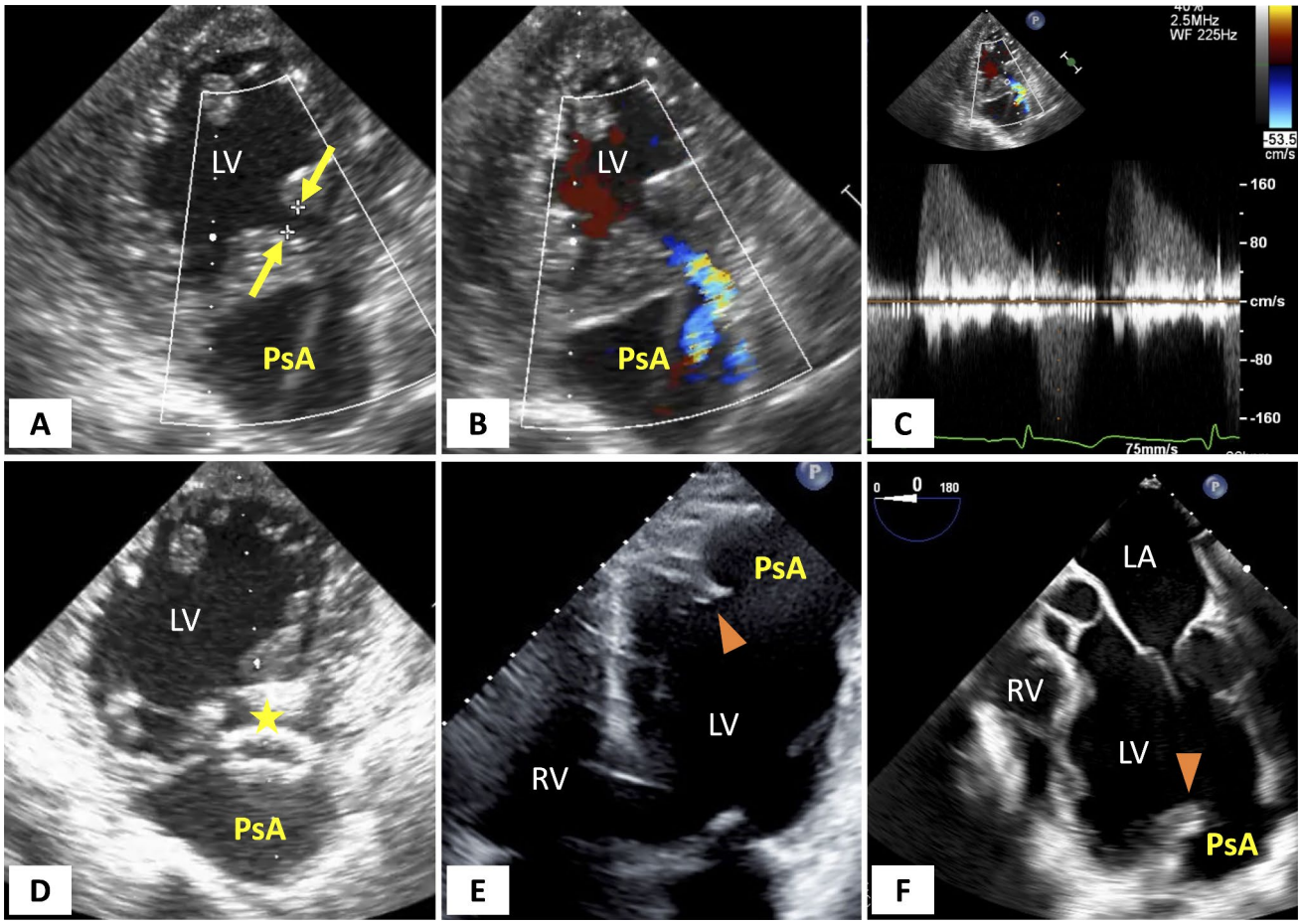

Fig. 5 **A**–**C** Transesophageal echocardiogram demonstrating pseudoaneurysm with a narrow neck (*yellow arrows*). Turbulent flow is seen entering the pseudoaneurysm from the left ventricle by color Doppler and demonstrates "to-and-fro" flow by continuous-wave Doppler assessment. **D** Well-positioned percutaneous closure device (*yellow star*) across the neck of the pseudoaneurysm. **E**, **F** Transthoracic and transesophageal echocardiogram demonstrating a wide-neck pseudoaneurysm. Note the presence of disrupted myocardium (*orange arrowhead*). *LA*, left atrium; *LV*, left ventricle; *PsA*, pseudoaneurysm; *RV*, right ventricle. **A**–**D** Courtesy of Carlos E. Ruiz, MD, PhD

Treatment

LV pseudoaneurysms demand immediate surgical intervention due to their heightened susceptibility to progressive expansion and rupture. Nevertheless, surgical intervention has an in-hospital mortality rate of about 20% [34]. In situations where surgical risks are prohibitive, percutaneous closure appears to be a viable alternative (Fig. 5) [35]. 2D and 3D TEE are essential modalities for assessing the size of the defect and its associated rims, which can then in turn inform the appropriate closure device size. Unsurprisingly, care must be taken when instrumenting the pseudoaneurysm so as not to disrupt the pericardium stabilizing the contained rupture.

LV aneurysms, on the other hand, generally only require medical therapy for heart failure and treatment of thrombus (if present). However, surgical excision may be performed with the aim of ameliorating clinical symptoms, primarily addressing heart failure symptoms, embolic phenomenon from left ventricular thrombus, and potentially life-threatening tachyarrhythmias [36, 37].

Temporary Mechanical Circulatory Support

Temporary MCS devices offer crucial support to patients with mechanical complications and cardiogenic shock. Specifically, they play a critical role as a bridge to a definitive surgical (or percutaneous) correction [38]. While a number of MCS devices are available, the specific choice is influenced by a variety of factors including the mechanical complication encountered, other cardiac abnormalities, the degree of hemodynamic instability, the severity of hypoxemia, the presence or absence of significant

peripheral arterial disease, institutional availability, and clinician familiarity/comfort. Intra-aortic balloon pump (IABP), Impella (Abiomed, Danvers, Massachusetts), TandemHeart (Cardiac Assist, Inc.; Pittsburgh, PA), VA-ECMO, and left atrial VA-ECMO (LAVA-ECMO) may all be considered for stabilization of patients with mechanical complications after AMI [29••, 39].

Echocardiography plays a crucial role in the choice of MCS, confirmation of appropriate positioning, and monitoring for the development of complications. As such, during the initial assessment for mechanical complications following AMI, a complete echocardiogram should be performed (if possible) for the identification of other cardiac abnormalities that may preclude the use of specific types of mechanical circulatory support. This includes the severity of aortic regurgitation (AR), aortic valve stenosis, the presence of a mechanical aortic valve, intracardiac thrombi (i.e., LV or LA appendage thrombus), and intracardiac shunts. Notably, the existence of substantial AR acts as a contraindication for the utilization of most types of temporary MCS, although there has been reported success when direct LA unloading is used (i.e., TandemHeart and LAVA-ECMO) [40, 41].

Conclusion

While rare, mechanical complications after AMI are a significant source of morbidity and mortality. Not only does echocardiography play an essential role in diagnosis, but it is a critical component of the selection, initiation, and maintenance of MCS for stabilization prior to definitive surgical correction. In select patients, echocardiographically guided percutaneous correction can be considered as an alternative to surgery.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s11886-024-02042-5.

Author Contributions All authors contributed text and figures to the manuscript, in addition to providing a comprehensive review after completion of the manuscript.

Data Availability No datasets were generated or analyzed during the current study.

Compliance with Ethical Standards

Conflict of Interest The authors report that they have no relationships relevant to the contents of this paper to disclose.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- •• Of major importance

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