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Mechanical Complications after Myocardial Infarction: A Comprehensive Review

Alexis D. Aparicio-Ortiz[1], María Natalia Alonso-Jimenez[1], Adrian Espejel-Guzman[1], Aldo Cabello-Ganem[1], Javier Serrano-Roman[1], Santiago Luna-Alcala[1], Nilda Espinola-Zavaleta[1,2*]

[1]Department of Nuclear Cardiology, National Institute of Cardiology Ignacio Chavez, Mexico City, Mexico
[2]Department of Echocardiography, ABC Medical Center, Mexico City, Mexico
Email: *niesza2001@hotmail.com

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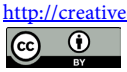

Abstract

Mechanical complications of myocardial infarction are potentially fatal events that can occur after an acute myocardial infarction. While the introduction of primary percutaneous reperfusion and fibrinolysis has reduced the incidence of these complications to less than 1%. These complications pose significant hemodynamic consequences and necessitate prompt diagnosis. Echocardiography, cardiac magnetic resonance imaging, and computed tomography are valuable tools for establishing an accurate and expedited diagnosis. Consequently, it is imperative to conduct further scientific research to enhance hemodynamic stabilization techniques such as intra-aortic balloon counterpulsation and extracorporeal membrane oxygenation, in addition to exploring new surgical procedures that can reduce mortality resulting from mechanical complications. This article aims to provide a comprehensive review of mechanical complications following myocardial infarction and their correlation with multi-imaging, facilitating a better understanding of these complications.

Keywords

Infarction, Mechanical Complications, Echocardiography, Magnetic Resonance

1. Introduction

Acute myocardial infarction (AMI) can develop several short and long-term complications classified as inflammatory, arrhythmic, embolic, or mechanical [1]; the latter includes Papillary Muscle Rupture (PMR), Ventricular Septal Rupture (VSR), Left Ventricle Free Wall Rupture (LVFWR), Left Ventricular

Aneurysm (LVA), and Left Ventricular Pseudoaneurysm (LVP) [2] [3].

Mechanical complications (MC) due to ischemic heart disease are rare in developed countries, where fibrinolysis and Percutaneous Coronary Intervention (PCI) have played an essential role in decreasing its incidence by less than 0.27%. Nevertheless, patients with mechanical complications still have higher mortality rates, up to 4 times more than those without MC [3] [4]. Furthermore, since a state of emergency and restrictions were declared in several countries due to the COVID-19 pandemic and because of the fear of getting infected, there was a significant rise (fourfold higher) in the incidence of late presentations of AMI and a reduced amount of PCI, both considered as established risk factors for MC [5] [6] [7].

The objective of this revision is to highlight the value of noninvasive cardiovascular imaging such as transthoracic echocardiography (TTE), cardiac magnetic resonance (CMR), and computed tomography (CT) as tools for an accurate diagnosis and prompt therapy election.

2. Papillary Muscle Rupture

PMR incidence is about 1% - 5%, and it is considered a lethal complication that causes cardiogenic shock in 85% of the patients with AMI [8]. The posterior descending artery, a branch of the right coronary artery or the left circumflex artery, usually supplies the posteromedial Papillary Muscle (PM) while the left anterior descending artery and the left circumflex artery typically supply the anterolateral papillary muscle (ALP). The obstruction of any of these arteries provokes ischemic necrosis leading to papillary dysfunction or rupture, causing mitral regurgitation followed by decompensated heart failure [9] (HF), which may result in pulmonary edema and cardiogenic shock within 1 - 7 days posterior of the AMI [3].

Risk factors for developing PMR include elderly, hypertension, female gender, complete occlusion of the coronary artery, history of HF, inadequate collateral circulation of the affected muscle, lack of revascularization, delayed presentation, chronic kidney disease and first myocardial infarction [7] [10] [11] [12].

2.1. Clinical Features

The PMR symptoms are related to several factors including the extension of the AMI, pulmonary edema, hypoxia, the severity of the mitral regurgitation, and occurrence of cardiogenic shock. Suspicion of PMR should be raised in patients presenting shortness of breath and chest pain [9]. In cases of total PMR, severe mitral regurgitation occurs, increasing the mortality to the point that patients die before any surgical procedure [13]. At the physical examination, there is a mid, late, or holosystolic murmur, and as PMR worsens, it tends to disappear due to the equalization of the LA and LV pressures [7] [13].

2.2. Diagnosis

The gold standard for the diagnosis of PMR is echocardiography, where transe-

sophageal echocardiography (TEE) has the highest sensitivity (92% - 100%), while transthoracic echocardiography (TTE) has a lower sensitivity (65% - 85%) [9]. This technique allows evaluating different aspects of the pathology, such as 1) Rupture of the MP or chordae tendineae; 2) The anatomy and function of the mitral leaflets and the subvalvular apparatus; 3) The function of the left ventricle (LV) (**Figure 1** and **Figure 2**). PMR can cause a holosystolic, mid, or late murmur that tends to disappear as left atrial (LA) and LV pressures equalize [3] [14].

The echocardiogram shows PMR as free-moving echo densities that prolapse into the LA (**Figure 1**). Depending on the site and size of the rupture, different images involving one or more segments of a flail mitral valve are seen in approximately one-third of the patients with PMR. The medial commissure is the most affected segment of the mitral valve, followed by A3 and A2, P2 - P3 [9] [13]. Also, a backward flow is shown in color Doppler due to the mitral regurgitation (**Figure 2**); the duration of the backward flow reduces as the severity of

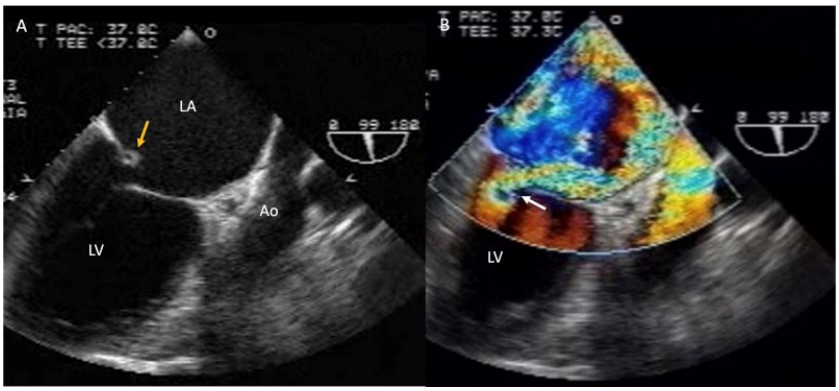

Figure 1. Papillary muscle rupture (A) 99˚ transesophageal echocardiogram with a rupture of the posteromedial papillary muscle (orange arrow). (B) Color Doppler shows torrential mitral regurgitation (white arrow). Abbreviations: LA: left atrium, LV: left ventricle, Ao: Aorta.

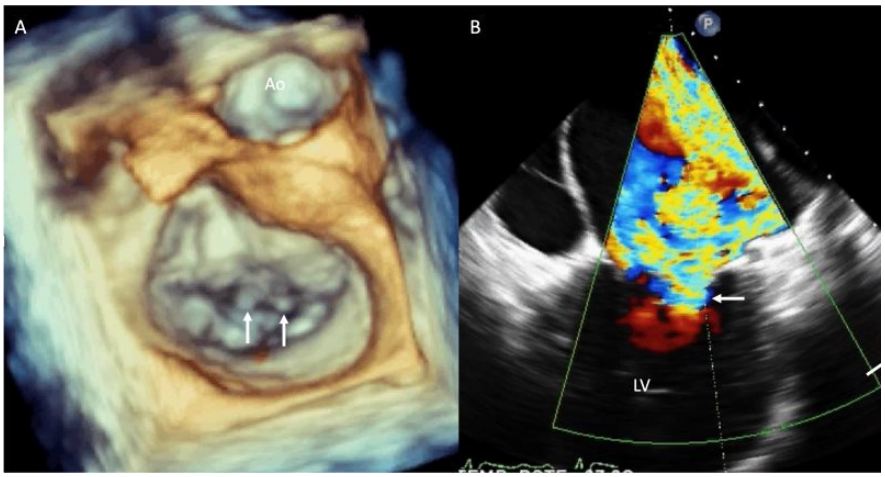

Figure 2. 2D and 3D transesophageal echocardiogram. (A) Surgical view of the mitral valve with ruptured chordae tendineae pointed by the white arrows, (B) Color Doppler showing severe mitral regurgitation (white arrow). Abbreviations as before.

the mitral regurgitation worsens because of the equalization of the LA and LV pressure. In the LV assessment, ejection fraction usually stays normal or decreases a little bit [9] [14].

CMR is not usually used to evaluate PMR due to the urgent nature of the condition, requiring immediate assessment typically provided by echocardiography. However, CMR provides high resolution, a wide field of view and high soft tissue contrast that can be enhanced with gadolinium, CMR shows an increased movement of the mitral valve and an abnormal movement of the LV wall, related to the infarction and mitral regurgitation [15].

Chest radiography may be helpful in cases with pulmonary edema and angiography can be used to determine the extension of the coronary artery involvement [16].

2.3. Treatment

The initial treatment involves hemodynamic and airway support and management for afterload reduction to decrease mitral regurgitation, pulmonary edema, arrhythmias, and PCI. As mortality rises to 50% in PMR, early surgery is required; as mitral valve repair, which is more complex, the most common procedure is mitral valve replacement [3]. No evidence demonstrates a superiority between mitral valve replacement and mitral valve repair; on the other hand, it is essential to know the severity of the necrosis because the greater extension of the necrosis, the lesser the survival rate [14].

Mitral valve replacement is usually practiced in patients with complete PMR; consequently, it has higher mortality rates [17]. Mitral valve techniques include: 1) Reimplantation of the ruptured PM to adjacent PM; 2) Reimplantation of the ruptured PM in the LV wall; 3) Neochordal; 4) Chordal translocation; 5) Resection of prolapse leaflet segment; 6) Annuloplasty ring or bands. Also, new off-pump transapical mitral valve repairs, such as NeoChord DS1000 device, Tendyne, and Intrepid, allow minimum invasive valve repair, but further research is needed to determine their feasibility in PMR [9].

3. Left Ventricular Free Wall Rupture

The incidence of left ventricular free wall rupture (LVFWR) is about 0.56% - 2% of AMI. Half of the patients debut with sudden death. The most common sites of the rupture are inferolateral (43%), lateral (28%), and apical (24%) [17] [18].

There are several classifications for LVFWR. Old classifications of LVFWR are anatomical-based and are of little help in providing an appropriate treatment. Becker and colleagues found on early phase AMI (<24 hours), type I rupture that is characterized by an abrupt, slit-like myocardial tear; type II rupture, where myocardial degradation is visible and denotes a gradually worsening rip; the type III rupture that occurs during the late phase of AMI (>7 days) with a pronounced weakening of the myocardium and perforation in the center region of the aneurysm [19].

On the other hand, according to autopsy or surgery, Purcaro *et al.* divided FWR into six pathological categories [20]. Haddadin *et al.* classified the free wall rupture (FWR) as either oozing or blow-out types based on the appearance of the rupture site during surgery [21]. Gong and colleagues proposed a new classification based on the clinical presentation of the patient, which may be readily identified during FWR diagnosis and serve as a basis for their treatment. The newly proposed classification is as follows: Cardiac Arrest Type, which is characterized by an abrupt rupture with massive bleeding into the pericardial cavity, followed by a loss of consciousness and cardiac arrest; 90.8% of this type of patients had electromechanical dissociation and their period from start to circulatory collapse was exceedingly brief. The unstable type is characterized by syncope, brief arrhythmia, temporary gatism, transient electromechanical dissociation, abrupt angina pectoris with moderate to massive pericardial effusion but an additional requirement of pericardiocentesis showing bloody fluid or no/mild pericardial effusion 24 hours before FWR has to be accomplished, the average time between onset and death on the unstable type was 4.5 hours, considered the "golden time" for surgery. Stable type LVFWR was classified when patients had stable hemodynamics, hypotension, and moderate to massive pericardial effusion with a pericardiocentesis showing bloody fluid; they might deteriorate, which means they may need careful monitoring of pericardial effusion and vital signs [22].

Uchida *et al.* also proposed a new classification based on surgical findings, highlighting that there is no objective in distinguishing between blowout and oozing type LVFWR, and pointing out that classification is based on the subjective judgment of the surgeon, and that the definition of blowout type an oozing type is unclear, proposing a new classification that considers cases where at the time of the pericardiotomy, the bleeding from the LV had already ceased on its own. They classified these cases of spontaneous hemostasis as "sealed type" and created the blowout type that results from combining blowout and oozing types with continuous bleeding [23].

We strongly believe that having a lot of classifications worsens the understanding of the pathology and that an in-depth analysis should be done to evaluate the effectiveness of each classification focusing on early diagnosis and treatment, which could help improve overall outcomes in patients with this condition. In this article, we use the old classification, which classifies LVFWR as "blowout type" (abrupt rupture with active bleeding and macroscopically large tear in the infarcted region) and "oozing type" (incomplete rupture with epicardial extravasation) or slow bleeding which may be temporarily occluded by a clot or pericardial adhesion) [24].

Risk factors for LVFWR are no history of AMI, female gender, elderly, arterial hypertension, severe one-vessel coronary disease, long angina episode, delayed hospital attention, ST-segment elevation, progressive hypotension, Peak MB-creatine kinase above 150 international units and electromechanical dissociation.

In addition, mortality is up to 75% - 90% in LVFWR; surprisingly, previous myocardial infarction and multivessel disease are protective factors [18] [25].

3.1. Diagnosis

Echocardiography is the angular stone in LVFWR diagnosis; its sensitivity and specificity are over 90% [25]; it demonstrates cardiac tamponade, hemopericardium, and reduction of myocardial wall thickness (**Figure 3**).

Once the diagnosis is confirmed, emergency surgery is needed [1]. Unfortunately given the high mortality of LVFWR, alternative imaging methods are ruled out, however, CMR provides a detailed visualization of the heart, allowing a clear differentiation between pericardium, myocardium, thrombus and fat, showing LVFWR content, compression of the right atrial wall, pericardial effusion, cardiac tamponade and the site of infarction; additionally, contrast-enhanced CMR may be useful for better planning the surgical approach [17] [26] [27]. CT (**Figure 3**) and contrast-enhanced CT are valuable alternatives when CMR is not available, especially to differentiate from conditions such as aortic dissection. Coronary angiography may be essential in determining whether a coronary graft is needed [27] [28].

3.2. Treatment

When the diagnosis is confirmed, it may be necessary to provide hemodynamic and airway support. The intra-aortic balloon pump is effective for hemodynamic support; if a drainable clot tamponade is present, pericardiocentesis can be performed; otherwise, options include pericardium decompression through subxiphoid drainage or sternotomy. Various methods to repair LVFWR exist, but their mortality rates are up to 32%. Suture and sutureless techniques have been developed, aiming to close the tear, stop the bleeding, repair, and reduce the

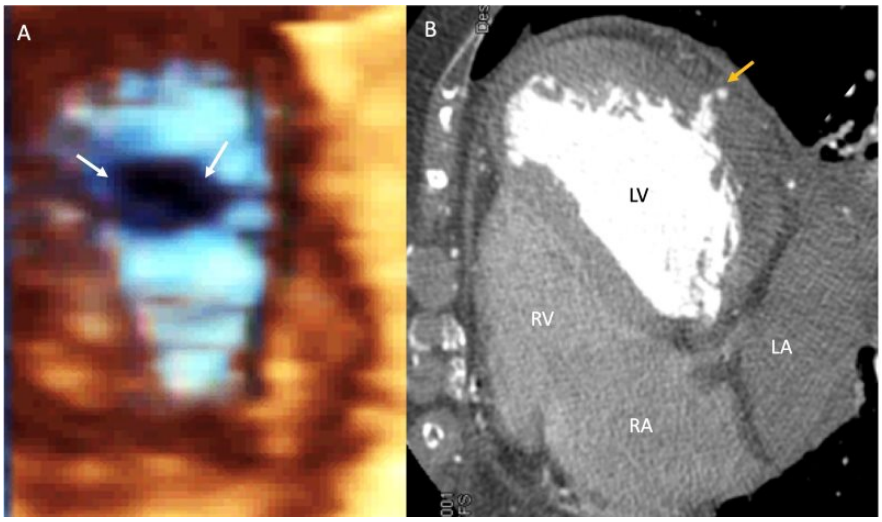

Figure 3. 2D and 3D transthoracic echocardiogram. (A) 3D echocardiogram with an internal view of free wall rupture (white arrows); (B) CT 4-chamber view showing free wall rupture with mild pericardial effusion (orange arrow). Abbreviations as before.

heart distortion; the former is the most practiced, achieved with suture of the edges with prolene buttressed by Teflon and the addition of Dacron, Teflon, or biological patches (bovine or pericardium). Infarctectomy has fallen into disuse due to its higher mortality, and to the development of new techniques, it is reserved for a massive blow-out type. A covering patch graft may be considered in cases without bleeding LVFWR, consisting of suturing a patch graft with prolene surrounding the infarcted area; glue injection beneath the patch is needed to strengthen the union to the myocardium and prevent blood leakage. Suture-less repair on the oozing type LVFWR without active bleeding reduces the mortality by 40% compared to the suture techniques. It consists of gluing a prosthetic patch around the infarcted area with fibrin-based glues or gelatin hydrogels that leave a stable fibrin matrix after the degradation of the fibrinogen. Collagen sponge patches like Tachochomb and Tachosil may be used by pressing them over the dry surface around the infarcted area over the healthy myocardium providing a sealing and hemostasis effect. Percutaneous intra-pericardial fibrin-glue injection therapy (PIFIT) is a new technique that induces hemostasis, achieving a sealant effect. Conservative treatment is not recommended since survival is about 10 %. Extracorporeal membrane oxygenation may be helpful but not recommended since anticoagulation is needed [17] [23] [24] [25] [29].

4. Ventricular Septal Rupture

Ventricular septal rupture (VSR) is a rip of the interventricular septum caused by necrosis ischemia due to an AMI [3]; it causes immediate left-to-right shunting, increases pulmonary flow, and biventricular failure [13]. Percutaneous coronary intervention has led to a decline in VSR incidence; between 0.17% and 0.31% of the patients die in the first week, and 62% - 82% within two months [16] [30]. VSR is classified into three types. Type I rupture shows an abrupt, slit-like tear associated with acute infarcts. Type II rupture demonstrates the erosion of the infarcted myocardium and correlates clinically with a sub-acute presentation. Type III rupture exhibits concomitant aneurysm formation associated with older infarcts [30], which occur 24 hours or 3 or 5 days after de AMI [2] [3]. Female sex, advanced age, and delayed reperfusion are risk factors for developing VSR [7].

4.1. Clinical Features

All patients with a hemodynamic compromise during AMI should be rapidly examined for systolic murmur over the precordium and palpable thrill. Other physical exam findings result from augmented right-sided flow and may include a loud pulmonic component of the second heart sound, left or right S3 gallop, or tricuspid regurgitation [30].

4.2. Diagnosis

Usually, the diagnosis is made by TTE since it can identify the region and size of the rupture; coronary angiography may incidentally demonstrate the interven-

tricular shunt as the contrast goes through it [3]. The AMI causing the VSR can be in the left anterior descending coronary artery (apical VSR), dominant right coronary artery (basal VRS), or the dominant left circumflex artery [31].

Echocardiography can evidence the direction of the shunt, chamber enlargement, biventricular dysfunction, and pulmonary hypertension. The color Doppler can also help us to assess the defect's anatomical size (**Figure 4**) [3] [30].

CMR imaging shows left-to-right shunt, thinning, and akinesia of the inferoseptal and inferior wall from the basal level to the midventricular level, consistent with VSR, and provides detailed information on tissue integrity, site, size, the location of the infarcted area and the lesion of the interventricular septum, important data for surgical repair (**Figure 4**) [32] [33].

CT can be used to better define the anatomy of the lesion showing the interventricular septum with a discontinuous area in addition to regional akinesia, as a marked reduction in myocardial thickening during systole which is of concern for surgical procedure; it also can be used to rule out the differential diagnosis [34] [35].

Angiography and cardiac catheterization confirm VSR and lesions of the coronary arteries. In addition, right heart or pulmonary artery catheterization can show an "oximetric jump", characterized by an increase in oxygen saturation between the right atrium and the pulmonary artery, forming a "ventricular shunt" [36].

4.3. Treatment

VSR is a surgical emergency; the procedure consists of closure of the VSR and placement of a coronary artery bypass graft to stabilize the patient [31]. Additionally, hemodynamic support is necessary; the reduction of afterload is carried out

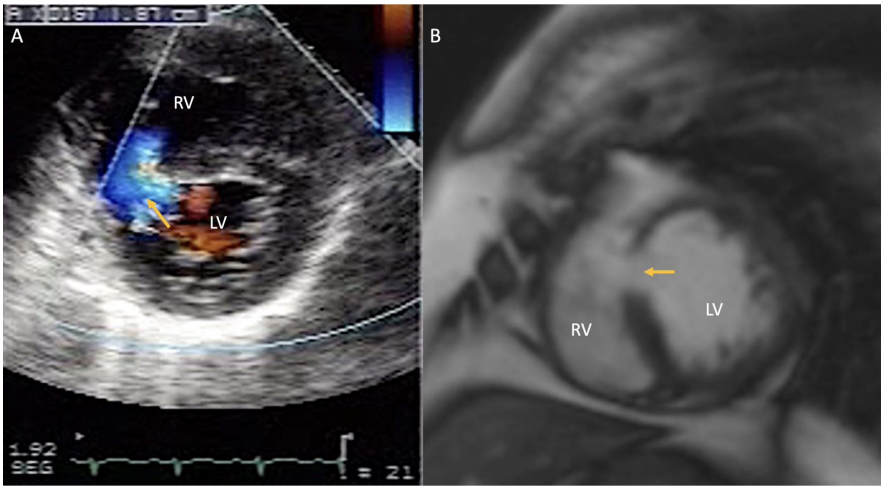

Figure 4. Transthoracic echocardiogram and cardiac magnetic resonance (A) Parasternal short axis at the level of both ventricles with color Doppler, showing rupture of the interventricular septum in its middle portion (orange arrow). (B) CMR at the level of both ventricles with thinning and rupture of the interventricular septum in its middle portion (orange arrow). Abbreviations as before.

with the administration of vasodilators, inotropics, intra-aortic balloon counterpulsation and the Impella device in cases with low cardiac output [31] [37].

Two surgical techniques are described; David infarct exclusion, that is superior to Daggett direct septal closure for early and late survival. In the former, the infarcted septum and free wall myocardium are excluded with a patch of bovine pericardium; the latter consists of infarctectomy with direct closure of the VSR with or without the use of patches [38]. The percutaneous closure of a VSR is only a temporary fix because residual VSR develops in 10% - 25% of cases [39].

The mortality of surgical intervention within 24 hours of AMI is over 60%. In contrast, untreated VSR has a 40% to 80% mortality. Surgical intervention within seven days of this complication has a mortality of 54.1% [40].

5. Left Ventricular Aneurysm

Left ventricular aneurysm (LVA) is a protrusion secondary to a thinning of a dyskinetic wall segment of the ventricle after AMI that affects the ventricular wall along diastole, it is a late mechanical complication that affects mainly the anteroseptal wall (64.7%) followed by the apex (21.3%) and posterior (8.6%) and lateral walls (5.3%) [32] [41] [42]. Since the development of percutaneous coronary interventions, LVA has decreased from 38% to 15 or 8% [43]. The risk factors are elderly (>65 years old), female gender, occlusion of the descending anterior coronary artery, high NT-pro-BNP levels, and the impossibility of achieving accurate reperfusion of the infarcted coronary artery [41] [44].

The clinical presentation of the aneurysm depends on its size; small aneurysms tend to be asymptomatic and large aneurysms present with fatigue, dyspnea, chest pain, syncope, palpitations, lower edema, ischemia in the lower limbs and hypotension [41]. On physical examination, apical pulses are prominent diffuse or a third or fourth heart sound may be detected [3].

5.1. Diagnosis

Since LVAs are asymptomatic, they are usually incidental findings. Echocardiography is the first tool to diagnose LVA; it establishes a differential diagnosis between true LVA and left ventricular pseudoaneurysm (LVP). TTE has a specificity and sensitivity that exceeds 90% (**Figure 5**) [43]. Color Doppler shows abnormal flow within the aneurysm and oscillatory movement (fro-motion) [38]. The electrocardiogram (ECG) may show persistent ST-segment elevation (more than three weeks), Q waves throughout the aneurysm territory, and reduced T waves amplitude [3] [43].

CMR is very useful since it allows better differentiation between fat, thrombus, myocardium and pericardium; it is the best option to evaluate biventricular function and morphological structures, such as the wide neck and its transition to the thin wall of the aneurysm, and the ratio between the maximum diameter and the maximum internal diameter (0.9 - 1.0), the presence of calcifications or fatty replacement of the myocardium, adherent thrombus at the site of the aneurysm and dyskinesia of the infarcted myocardium (**Figure 5**) [32] [42].

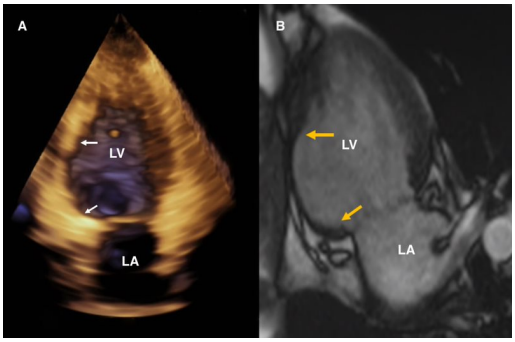

Figure 5. Transthoracic 3D echocardiogram and cardiac magnetic resonance. (A) 3D echocardiogram in apical 2-chamber view showing an aneurysm in the basal portion of the inferior wall of the left ventricle (white arrows). (B) CMR image with an inferobasal left ventricular aneurysm (orange arrows). Abbreviations as before.

CT allows visualization of the wide neck and the transition from the normal myocardium to the thinned myocardium and akinesia or dyskinesia during systole.

Contrast-enhanced CT shows an anomalous opacified cavity posterolateral to the dilated LV that connects to the LV lumen through a wide neck. The maximum diameter of the aneurysmal cavity is approximately twice as large as its neck; it also allows us to characterize the site and extent of the anterior and posterior LV aneurysms and the calcification or fatty replacement of the infarcted myocardium [45] [46].

5.2. Treatment

Medical treatment involves antihypertensive and anticoagulation therapy to avoid thrombus formation; surgery should be reserved for patients with refractory heart failure, systemic embolization, recurrent embolism despite anticoagulant therapy and arrhythmias [32] [43]. Patients considered for surgery should undergo right or left heart catheterization with coronary arteriography and left ventriculography. Aneurysmectomy, aneurysmorrhaphy, and ventricular restoration are the techniques for LV aneurysm repair [41]. Its goal is to decrease the LV size to reduce oxygen requirements, eliminate refractory arrhythmias, and enhance the diastolic function of the LV. Despite the information presented, there is bias through new data about the best therapy; some studies will show the superiority of surgical against conservative treatment and vice versa, but there is no clear evidence of benefit between each other [7] [13] [41].

6. Left Ventricle Pseudoaneurysm

Left ventricular pseudoaneurysm (LVP) occurs when LV tears form an outpouching contained by fibrous tissue of the pericardium or hematoma [3] [32] [42] [47]. It usually presents after two weeks of the AMI and can be asymptomatic or onset with angina and heart failure (HF). The most common site where LVP occurs is the inferior, inferolateral wall over the left circumflex artery area and the anterior wall or apex [3] [32]; it has an incidence of 4% with a risk of

rupture and death of 30% - 45% [48]. It is mainly characterized by sudden chest pain, dyspnea, cardiac arrest, congestive heart failure (CHF), syncope, tamponade, and embolism [49]. Sometimes patients have nonspecific symptoms such as cough, altered mental status, and dizziness. On physical examination, systolic-diastolic or holosystolic murmurs related to mitral regurgitation may be found [50] [51].

6.1. Diagnosis

Echocardiography is the main modality for diagnosis TEE having an effectiveness higher (75%) compared to TTE (26%) [51]. The ratio between the maximum diameter neck and the maximum inner diameter of the LVP cavity is 0.25 - 0.5 (**Figure 6**). A turbulent flow within the LVP is observed with color Doppler simultaneously, and spectral Doppler shows bidirectional intraventricular flow, one flow in proto-diastole from the LVP to the ventricle and the other in proto-to-systole from the ventricle to the pseudoaneurysm [47].

CMR has a sensitivity of 100% and a specificity of 83% for diagnosing LVP [51]. In addition, it demonstrates an akinetic focal convexity or outpouching of the LV wall through a narrow neck that can provoke hemopericardium and tamponade; it also establishes the difference between the pericardium and the thrombus, scar, or myocardium and shows the loss of epicardial fat, also by seeing the destruction of the myocardial-pericardial interface and the delayed enhancement of the pericardium overlaying it (**Figure 6**) [32] [42] [47]. CT allows better visualization of the anatomy to differentiate it from a true aneurysm. The contrast-enhanced CT shows a hyperdense cavity with distal dilatation greater than the proximal neck, compatible with LVP [46] [51]. Angiography is considered the best available method for diagnosis. It helps distinguish false aneurysms and it is necessary before surgery to assess the severity and location of coronary lesions [50] (**Table 1**).

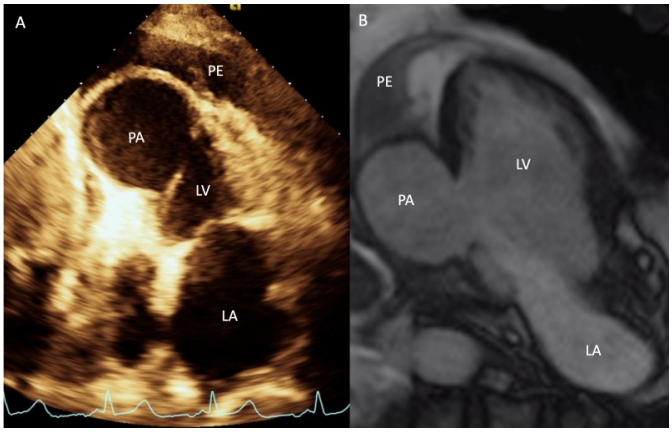

Figure 6. 2D transthoracic echocardiogram and cardiac magnetic resonance. (A) Apical 2-chamber view of the left chambers showing a pseudoaneurysm in the middle portion of the left ventricular inferior wall and pericardial effusion. (B) CMR image in the apical 2-chamber view with the presence of a pseudoaneurysm in the inferior wall of the left ventricle and pericardial effusion. Abbreviations as before, PE = pericardial effusion.

Table 1. Diagnostic imaging modalities for mechanical complications.

Mechanical complications	Echocardiogram	Cardiac Magnetic Resonance	Computed Tomography	Others
Papillary Muscle Rupture	• The Gold Standard • TEE has the highest sensitivity 92% - 100% • TTE sensitivity 65% - 85% • Free moving echo densities that prolapse into the LA • Mitral regurgitation • Normal or mildly reduced LVEF	• Increased mitral valve motion • Abnormal movement of the LV related to the infarction • Brings valuable information for the surgical procedure	• Only used when echocardiogram and magnetic cardiac resonance are not available	**Chest X-ray** • Pulmonary edema **Angiography** • Helps to determine the extension of the infarction
Left Ventricular Free Wall Rupture	• Sensitivity and specificity over 90% • Demonstrate cardiac tamponade, hemopericardium, reduction of the myocardial wall thickness	• Ruled out due to the severity of the complication	• Helps to confirm the diagnosis • Provides an entire visualization of the heart • Helps to differentiate between pericardium, myocardium and fat • Shows the contained LVFWR, pericardial effusion, cardiac tamponade, site of the infarction **Contrast-enhanced CMR** • Useful for planning a surgical approach	
Ventricular Septal Rupture	• Can identify the severity and size of the rupture • Demonstrates the presence of a shunt, chamber enlargement, biventricular disfunction and pulmonary hypertension	• Shows left to right shunt, thinning of the septum • Provides detailed information about tissue integrity, site, size and location of the infarcted area and location of the lesion	• Shows lesion in the interventricular septum, regional akinesia, reduction in the myocardial thickening	**Angiography** • Demonstrates the interventricular shunt as the contrast goes through it
Left Ventricular Aneurysm	• It is the first tool to make the diagnosis • Doppler color shows abnormal flow within the Aneurysm and oscillatory movement (fro-motion)	• Allows better differentiation between fat, thrombus, myocardium and pericardium • It is preferable for evaluating biventricular function and morphologic structures, such as the wide neck and its transition to the thin wall of the aneurysm.	Focal thinning of LV wall with aneurysmal dilation showing the wide neck, calcification and fatty replacement of the infarcted myocardium	

Continued

		• The ratio between the maximum and maximum inner diameters (0.9 - 1.0). • Possible calcifications or fatty replacement of the myocardium. • Adherent thrombus at the aneurysm site. • Dyskinesia of the infarcted myocardium.	**Contrast CT** shows an anomalous opacified cavity posterolateral to the dilated LV that connects with the LV lumen through a wide neck	
Left Ventricular Pseudoaneurysm	• Low accuracy for the diagnosis • Turbulent flow within the LVP is observed with color Doppler. • Spectral Doppler shows bidirectional intraventricular flow.	• Sensitivity 100% • Specificity 83% • Demonstrates akinetic wall, outpouching of the left ventricle, hemopericardium, tamponade, destruction of the myocardium, delayed enhancement of the pericardium overlaying it • Allows better differentiation between tissues	• Anatomic assessment of the LV **Contrast-enhanced CT** • Hyperdense cavity with distal dilatation greater than the proximal neck	**Angiography** • Helps to distinguish between false aneurysms • Brings information for the surgical procedure • Allows to assess the severity and location of the coronary lesions

6.2. Treatment

Little is known about the medical treatment of LVP [7]. The objective is to reduce the aneurysm growth, afterload, and the risk of thromboembolism, surgical repair of LVP follows the same principle as that of an LVA [2] [51]. Due to the significant risk of rupture (30% - 45%) and abrupt death by tamponade, urgent surgical treatment of LVP is recommended whenever possible, but further investigation is needed [3] [13] [43].

7. Discussion

In the last decades, the diagnostic techniques and the treatment of MC have been continuously improving. However, the mortality rate remains high despite efforts to reduce it. In addition, MC are characterized by hemodynamic instability in most patients, mainly those with VSR or PMR, so treatment with extracorporeal membrane oxygenation and intra-aortic balloon pump counterpulsation must be perfected [37] [52]. Also, new advances in this field must be studied in greater depth to be considered a standard in diagnosing and treating MC.

8. Conclusions

Post-infarction mechanical complications present significant challenges in clinical practice, both in terms of diagnosis and treatment. Advanced imaging tech-

niques, such as echocardiography, CMR imaging, and CT, have evolved to enable a more timely, accurate, and comprehensive assessment of these complications, focusing on their etiology, anatomy, and location. While echocardiography remains the initial modality of choice due to its ability to detail the lesion's etiology, anatomy, location, and size, the use of complementary imaging techniques like CMR and CT enhances diagnostic precision, providing essential anatomical details for surgical planning and repair.

In the realm of treatment, there have been considerable advances. Techniques such as the use of neochordae, annuloplasty rings or bands, and minimally invasive procedures for PMR, as well as sutureless methods, prosthetic patches, and biological glues for LVFWR, have significantly improved outcomes and reduced the invasive treatments. These innovations not only offer enhanced treatment options but also have the potential to redefine clinical approaches in the management of MC.

Despite these advances, several critical areas require further investigation. The debate over the superiority between mitral valve replacement and repair in PMR cases remains unresolved, highlighting the need for more research and clinical studies. A standardized and comprehensive classification system for LVFWR is crucial to enhance diagnosis and treatment efficacy. The effectiveness of surgical versus conservative approaches for LVA, as well as the outcomes and developments in surgical treatments for LVP, also necessitate additional research due to the scarcity of current information.

To summarize, the understanding and treatment of post-infarction mechanical complications has progressed significantly, driven by advances in imaging and treatment techniques. However, it is imperative to continue research to fill gaps in existing knowledge, refine clinical decision making, and optimize therapeutic outcomes. Continued collaboration and research are key to developing more effective diagnostic tools and personalized treatment strategies, which ultimately improve the prognosis and quality of life of these patients.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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