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Diagnosis and Management of Papillary Muscle Rupture Complicating Acute Myocardial Infarction: A Case Report and Review of the Literature

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Abstract

Introduction: The incidence of mechanical complications related to myocardial infarction has decreased over the last decades, and revascularization certainly plays a major role in this change. However, mortality still remains elevated. This is a case of acute papillary muscle rupture secondary to myocardial infarction leading to cardiogenic shock.

Case Presentation: A 71-year-old woman presented to an outside hospital complaining of chest pain and shortness of breath. An electrocardiogram was obtained and revealed depression of the ST segments from leads V1 to V4. Troponin I was elevated at 3.0 ng/mL. She was transferred to our facility for a higher level of care. She was found in cardiogenic shock at arrival. A bedside echocardiogram was ordered, which demonstrated papillary muscle rupture with severe mitral regurgitation. A coronary angiogram followed, which diagnosed severe three-vessel disease. After the insertion of an intra-aortic balloon pump, she was transferred emergently to the surgical suite for mitral valve replacement and revascularization. The operation was uneventful. She was discharged to a rehabilitation center after approximately 1 month of hospital stay.

Conclusions: Mortality from papillary muscle rupture remains elevated. Survival largely depends on the early surgical repair or the replacement of the mitral valve.

Keywords: Papillary Muscle Rupture, Acute Mitral Regurgitation, Echocardiography, Cardiogenic Shock, Acute Myocardial Infarction

1. Introduction

The incidence of mechanical complications related to myocardial infarction has decreased over the last decades, and revascularization certainly plays a major role in this change. However, the mortality rate still remains elevated despite developments in medical and surgical sciences. This is a case of acute papillary muscle rupture secondary to myocardial infarction leading to cardiogenic shock.

2. Case Presentation

A 71-year-old woman with known hypertension and hypothyroidism arrived at an outside hospital with a 4-hour history of chest pain. She had a similar pain about 4 days earlier, which subsided after a 2-hour period. Her pain was described as a pressure sensation localized in the middle of the chest and associated with diaphoresis and dyspnea. She was diagnosed with an acute coronary syndrome. Her electrocardiogram revealed ST-segment depression from leads V1 to V4 (Figure 1), and laboratory testing showed a troponin I elevation of 3.0 ng/mL. Transfer by ambulance to our institution was arranged for a higher level of care. At arrival, she still complained of chest pain. She had a heart rate of 110 beats per minute, blood pressure of 80/40 mmHg, respiratory rate of 30 breaths per minute, temperature of 96.0 °C, and oxygen saturation of 88%. Diffuse crackles were auscultated throughout her lung fields. S1 was diminished, and a cardiac murmur could not be auscultated. Her extremities were cool to touch.

2.1. Diagnosis

An echocardiogram was ordered since the patient's clinical presentation was indicative of cardiogenic shock. The cardiac chambers did not appear enlarged, and left ventricular ejection fraction was measured at 55%. The inferolateral and anterolateral walls were not thickening properly during systole (Videos 1 and 2). Right ventricular systolic function seemed normal, although image acquisition was suboptimal. The mitral valve leaflets lacked adequate coaptation with a gap during systole. A bell-shaped structure with chordal attachments to the mitral valve leaflets was moving from the left ventricle to the left atrium throughout the cardiac cycle (Videos 1 and 3). With color Doppler, a large

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jet occupying an extensive area of the left atrium was evident (Figure 2). Continuous-wave Doppler of the mitral valve revealed a dense and early-peaking triangular signal during systole (Figure 3). These findings denoted complete papillary muscle rupture leading to severe mitral regurgitation. A portable chest X-ray demonstrated diffuse patchy infiltrates with cephalization of the pulmonary vessels (Figure 4). She was transferred emergently to the cath lab for a coronary angiogram. The left anterior descending artery (LAD) had an 80% proximal stenosis, the right coronary artery (RCA) had a 90% proximal stenosis and the left circumflex artery was completely occluded (Figure 5) (Videos 4 to 7). The RCA appeared to be nondominant.

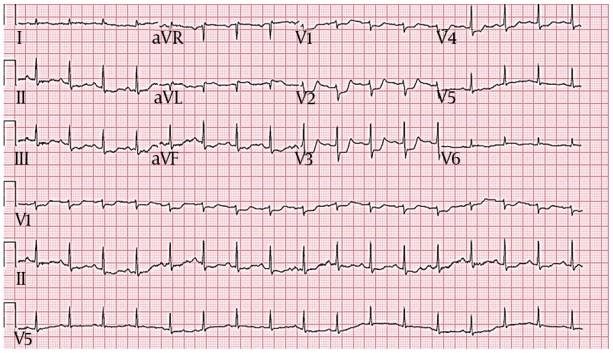
2.2. Initial Management

Oxygen levels worsened, so an orotracheal tube was inserted. Hypotension developed after an intravenous infusion of midazolam and fentanyl and improved with an intravenous drip of norepinephrine. While in the catheterization lab, an intra-aortic balloon pump (IABP) was placed for hemodynamic support and left ventricular afterload reduction. Her vital signs were temporarily stable. The cardiothoracic surgeon opted for surgery.

2.3. Definitive Treatment

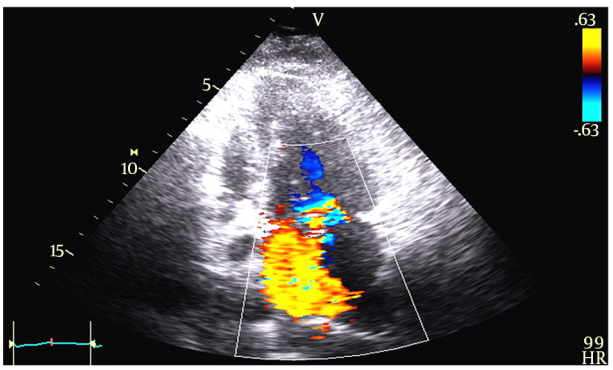
The cardiothoracic surgeon decided to intervene via mitral valve replacement and coronary artery bypass grafting (CABG). A saphenous vein graft was harvested from the left thigh and left upper leg. Cardiopulmonary bypass had to be initiated precipitously due to sudden hemodynamic deterioration. The saphenous vein was then sutured end to side to the RCA. The left atrium was accessed through the interatrial groove with adequate exposure of the mitral valve. Necrosis of the head of the posteromedial papillary muscle was confirmed, and extensive debridement of the necrotic tissue was performed. The anterior leaflet was excised to facilitate the placement of a 29 Hancock II porcine mitral bioprosthesis. After closing the interatrial groove, the left internal mammary artery was sutured end to side to the LAD. Normal sinus rhythm resumed after weaning off cardiopulmonary bypass. The patient was transferred to the cardiac care unit with intravenous infusions of norepinephrine, epinephrine, and vasopressin. After 1 week, the inotropes and vasopressors were weaned off and the IABP was removed. Eventually, support from mechanical ventilation was minimal and the orotracheal tube was removed. With ongoing physical and occupational therapy, the patient was discharged to a skilled nursing facility after a month of hospital stay. The prolonged requirement of vasopressor agents unfortunately led to the necrosis of her toes. She was followed up in the plastic surgery clinic, where amputation was recommended.

Figure 1. Initial 12-Lead Electrocardiogram



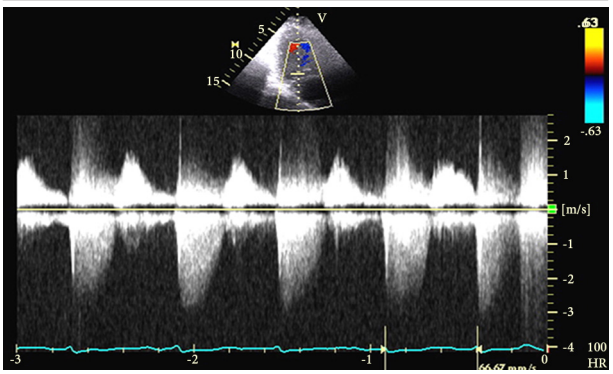
A prominent depression of the ST segments are visible from V1 through V4, raising the concern for the injury pattern of the posterior wall; a less prominent ST-segment depression at the inferior leads is observed, with ST-segment elevation in aVL.

Figure 2. Transthoracic Echocardiogram in the Four-Chamber View and Color Doppler of the Mitral Valve



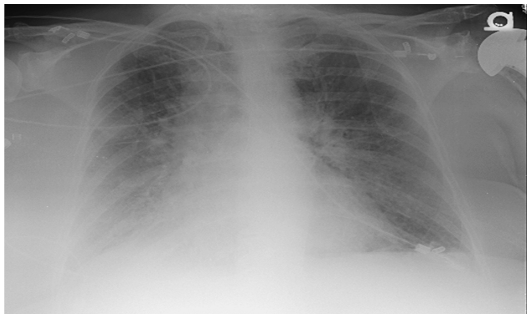
A large color jet is directed toward the medial wall of the left atrium. This phenomenon is named the Coanda effect and suggests severe mitral regurgitation. The color jet can occupy a variable amount of left atrial area when observed from different windows, necessitating complementary data to assess severity.

Figure 3. Continuous-Wave Doppler of the Mitral Valve



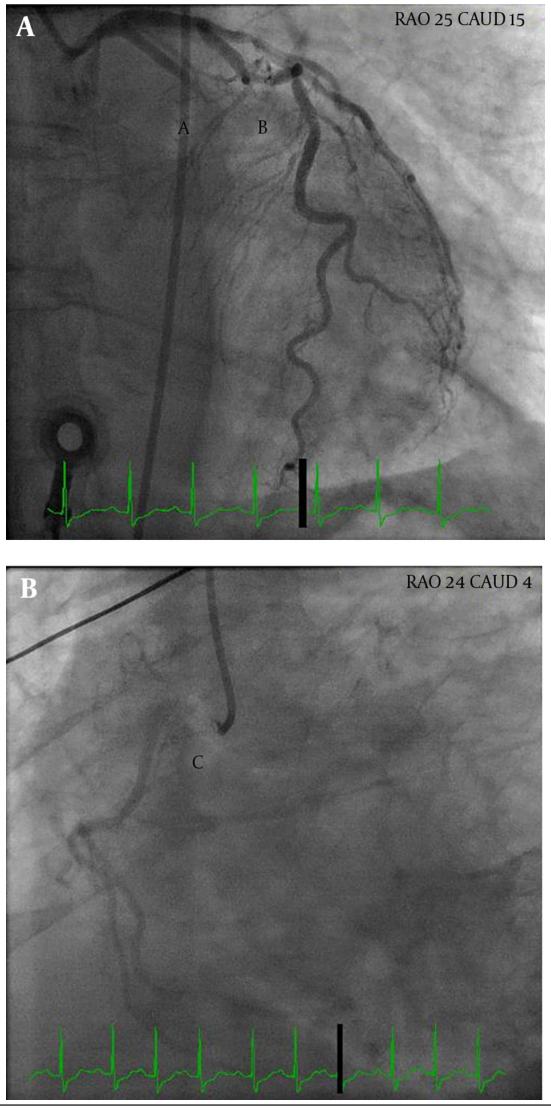
There is a very dense signal during systole with an early-peaking triangular shape, consistent with severe mitral regurgitation. This early peaking represents fast equalization of pressures between the left ventricle and the atrium.

Figure 4. Anteroposterior View of a Chest X-Ray



There are interstitial and alveolar opacities in a butterfly pattern, consistent with cardiogenic pulmonary edema.

Figure 5. Coronary Angiogram



A, The left circumflex artery appears totally occluded; B, the left anterior descending artery has an 80% proximal stenosis; C, the right coronary artery is not filled adequately with contrast, its proximal portion has a 90% stenosis.

3. Discussion

Ischemic heart disease can lead to mitral regurgitation through a variety of mechanisms including dilatation of the mitral annulus, dilatation of the left ventricular cavity, displacement and rupture of the papillary muscles, and rupture of the chordae tendineae.1 Chronic damage to the mitral valve system provides the heart with time to compensate for the secondary volume overload, and patients may remain asymptomatic for a long period of time. With the progression of the disease, left ventricular systolic function begins to decline leading to the insidious development of symptoms.

Acute mitral regurgitation (AMR) often results from the rupture of the chordae tendineae or the papillary muscles (1). One must be aware of the variety of causes as treatment can differ, and early recognition can confer life-saving interventions. These include ischemia, infective endocarditis, traumas, (2) procedures, (3, 4) degeneration of the mitral valve apparatus, (5) and malfunction of a prosthetic valve (1). Symptoms can develop in a matter of hours and include those secondary to cardiogenic shock (altered mental status, oliguria, and chest pain) and an increase in pulmonary venous pressures (shortness of breath and hypoxemia) (6). Effective stroke volume is decreased since now a fraction of the stroke volume is directed back to the left atrium. Unless the left atrium is enlarged as a consequence of a preexisting condition (chronic mitral regurgitation or diastolic dysfunction), its compliance is normal or even reduced. The left atrium is then unable to contain the additional volume of blood returning from the left ventricle (7). More pressure is transmitted to the pulmonary veins, precipitating congestion and pulmonary edema (8). This same pressure can acutely reduce right ventricular systolic function and contribute to cardiogenic shock (1). The findings of physical examination can be remarkably different from those in chronic mitral regurgitation. The typical holosystolic murmur may not be auscultated because of the early equalization of pressures between the left ventricle and the left atrium. Often, a low-pitched decrescendo murmur is auscultated at the beginning of systole. Sometimes there is not an audible murmur (9).

3.1. Diagnosis

An echocardiogram in general reveals a normal-sized left ventricle and atrium. The end-systolic diameter decreases with a lesser resistance for left ventricular output now that the blood is forwarded to the aorta and the left atrium. The left ventricle appears with a hyperdynamic contractile function and normal or above-normal ejection fractions. This does not portend a better outcome when compared to subjects who suffer from cardiogenic shock and left ventricular systolic dysfunction as demonstrated in the SHOCK Trial (10). A flail leaflet can develop with loss of continuity in any component of the subvalvular apparatus. Specifically in papillary muscle rupture, a bullet-

shaped mass may be seen floating indiscriminately from the left ventricle to the left atrium (11, 12). In up to 35% of the patients and despite complete rupture, the head of the papillary muscle is not floating into the left atrium since it becomes trapped within the chordal structures (13). The anterolateral papillary muscle complex is harder to visualize than is the posteromedial, and disruption is easier to identify in long-axis views than in short-axis views.

Color and flow Doppler can further establish the severity of mitral regurgitation (14). A large area of color occupying left atrial space during systole is visualized frequently, although the size may vary according to the echocardiographic equipment and settings used and the acoustic window from which the mitral valve is observed. Another problem with color Doppler is its tendency to underestimate the severity of mitral regurgitation when compared to contrast ventriculography (15). In order to make a more accurate measurement, color Doppler needs to be complemented with objective measurements. A prolapsed or flail leaflet or a flail papillary muscle strongly suggests severe mitral regurgitation. Continuous-wave Doppler of the mitral valve illustrates a dense signal with an early-peaking triangular configuration. This, in other words, means that there is a large regurgitant volume and that the pressures of the left ventricle and left atrium equalize early in systole. Quantitative measurements such as effective regurgitant orifice area, regurgitant volume, and regurgitant fraction vary with preload and afterload and should not be used to estimate severity in AMR (7).

The direction of the regurgitant jet and not the prolapsed leaflet determines what papillary muscle is involved. Each papillary muscle has multiple chordal attachments to both anterior and posterior leaflets (16). Color Doppler can also identify the other mechanical complications of acute myocardial infarction (AMI) such as ventricular septal and free-wall rupture. Distinguishing them is essential since their clinical presentation can be similar to AMR and they sometimes occur simultaneously.

A case series with 4 patients described echocardiographic differences between partial and complete papillary muscle rupture. In complete rupture we can find a cut-off sign, a mobile mass attached to the chordae and the mitral leaflets, and a flail or prolapsed mitral leaflet (17). In partial rupture, the muscle can be seen attached to its trunk with incomplete loss of continuity and some chordal attachments extending to the mitral leaflets (18). A flail or prolapse leaflet may also be present. Not uncommonly, these findings are less obvious or even missed with transthoracic echocardiography. Prompt diagnosis provides a window of opportunity to intervene before complete rupture develops with subsequent hemodynamic deterioration and death (19). A transesophageal echocardiogram (TEE) has an increased sensitivity compared to transthoracic imaging, although it may fail to identify partial rupture if the leaflet is not flail or prolapsing. In a case report, partial rupture was only seen after using live three-dimensional (3D) TEE (20).

TEE is recommended in AMI with hemodynamic instability when a transthoracic echocardiogram is suboptimal (21). Sometimes transthoracic imaging reveals severe mitral regurgitation without being able to exclude papillary muscle rupture (22). This situation is commonly encountered in patients with obesity and pulmonary emphysema. Tachycardia and early pressure equalization can reduce the period of time during which the regurgitant jet is observed, while a narrow eccentric jet may be misinterpreted as mild regurgitation. Since the TEE probe is in more proximity to the left-sided heart structures, a higher frequency can be used with better imaging resolution. The subvalvular apparatus should be visualized from both mid-esophageal and transgastric views (23).

The combination of TEE with 3D imaging allows better definition of the anatomy of the mitral apparatus and may aid in the surgeon's choice of repair or replacement (24, 25). In the past, 3D images were obtained by the reconstruction of 2D pictures, where resolution could be troubled by gating and respiratory variation. Today, the availability of matrix array transducers allows the acquisition of real-time 3D images (26, 27). The live images are obtained as small pyramidal data sets. A larger volume can be studied either by zooming (with lower resolution although still real time) or full-volume acquisition, which consists of merging multiple smaller pyramidal data sets (improved resolution albeit subjected to gating artifacts, specifically stitching artifacts). The quantification of the severity of mitral regurgitation is also possible. 3D-guided color Doppler allows measurement of the vena contracta with accuracy similar to cardiac magnetic resonance imaging. A more precise localization of the regurgitant jet improves correlation with the anatomical defect. More research with this imaging modality is necessary before it can become standardized in this group of patients.

Right-heart catheterization helps identify the etiology of shock, perform invasive hemodynamic monitoring, and diagnose even which of the mechanical complications of AMI a patient has. In severe AMR, pulmonary capillary wedge pressure is elevated and often has a prominent v wave in its tracing. This is commonly associated with a noncompliant left atrium, which allows the transmission of the pressure into the pulmonary vasculature from the left ventricle during systole. Postoperative state, ischemia, and rheumatic heart disease also increase left atrial stiffness (28). The v wave is less notorious and sometimes absent when the left atrium is enlarged and compliant, or when left ventricular systolic function is reduced. The typical scenario is that of a patient with chronic mitral regurgitation or sometimes AMR exacerbating chronic mitral regurgitation (29). The clinical presentation is of paramount importance when investigating the origin of a prominent v wave. A prominent v wave can also exist in patients with ventricular septal rupture. The latter can be distinguished by identifying an increase in oxygen saturation from the right atrium to the pul-

monary artery. This can occasionally be seen in papillary muscle rupture when the oxygenated blood from the left atrium contaminates the sample obtained in the pulmonary artery (30). A pattern simulating constrictive physiology can occur when acute volume overload increases the diastolic pressure in the right ventricle and reaches values similar to those in the left ventricle.

Coronary angiography should be performed in accordance with the current guidelines (31, 32). Revascularization either by surgery or by percutaneous coronary intervention increases the patient's chance of surviving cardiogenic shock. We will discuss below that many studies have supported surgical revascularization during the repair or the replacement of the mitral valve. The role that ischemia can play in precipitating left ventricular systolic dysfunction and hemodynamic instability, beyond the effects already generated by AMR, is variable. And this may explain why in certain studies revascularization did not result in a morbidity or mortality benefit. The posteromedial papillary muscle will more often suffer rupture than will its anterolateral counterpart due to the single-vessel blood supply from the posterior descending artery versus dual blood supply from the diagonal and obtuse marginal branches, respectively (33).

The severity of mitral regurgitation can also be assessed by injecting contrast inside the left ventricle (contrast ventriculography) through a pigtail catheter and then visualizing the amount of contrast returning into the left atrium during systole. It consists of a semiquantitative assessment, with the lowest grade of severity being +1 (contrast never fills the left atrium and it is cleared after each cardiac cycle) and the highest being +4 (there is contrast filling of the pulmonary veins) (28). The severity can be underestimated due to the under filling of the left ventricle when the pigtail catheter is placed in the left ventricular outflow tract. When the left atrium is markedly enlarged, the recommendation is to inject a larger volume of contrast than the standard. The catheter should not be positioned in the mitral apparatus lest it cause inadequate closure of the mitral valve. The amount of contrast given can increase the risk of contrast-induced nephropathy in a patient who already is hypotensive. Unless strictly necessary, contrast ventriculography should not be performed in a patient with papillary muscle rupture.

3.2. Initial Management

AMR secondary to papillary muscle rupture constitutes a life-threatening condition. It can manifest from day 1 to day 14 after an AMI, with peak incidence at 2 to 7 days (34). Older studies reported a mortality rate of about 80% with medical treatment (35-37). Initial optimization with vasodilators and/or an IABP should be interpreted as a window of opportunity to proceed with surgery. If medical therapy is continued alone, cardiogenic shock will most probably develop with an increased operative mortality (10). In a series of 54 patients, 91% presented to the hospital already with features of cardiogenic shock and pulmonary edema (35). There have been exceptional reports where surgery has been delayed for months (36). The use of new technologies such as the Impella device and the MitraClip may allow medical therapy to become a more reasonable option in the future (38, 39). If the repair or replacement of the mitral valve can be accomplished quickly, the patient has better odds of surviving, (36-40) and long term outcomes match those of an AMI without mechanical complications (35, 36). The mechanisms leading to post infarction AMR include complete or partial rupture of papillary muscles, acute dilatation of the mitral annulus, rupture of chordae tendineae, and displacement of the papillary muscle due functional impairment from ischemia (40). Necrosis without rupture appears to have no correlation with the presence or severity of mitral regurgitation. This was demonstrated in a study of 118 patients admitted with ST-segment elevation myocardial infarction (STEMI) who underwent cardiac magnetic resonance imaging at 9 ± 4 days and 8 ± 1 months after the event (41). Correlation was found instead with increased left ventricular volumes. Necrosis was detected by late gadolinium enhancement.

The posteromedial papillary muscle is more often compromised since its only source of blood supply is the posterior descending coronary artery (1). Russo et al. (35) recognized that the incidence of rupture for the former could be as high as 90%. Variants to this anatomical distribution have been reported (42, 43). The latter is potentially related to the large variety of anatomical locations and configurations of the papillary muscles. In one study of 116 human cadaveric hearts, the investigators found that only 3.4% had 2 papillary muscles as classically described (44).

The widespread use of reperfusion therapy has resulted in a decreased incidence rate of all mechanical complications in myocardial infarction. Older studies described the incidence of papillary muscle rupture to be 1 -3 % (35, 45). An updated review from 2010 found it to be 0.25% (46).

Management consists of early medical and surgical treatment (36). The aims of medical therapy are reducing afterload, increasing effective stroke volume, and lessening the amount of regurgitation. This can be achieved with intravenous vasodilators such as nitroprusside and nicardipine (21) and with mechanical support through an IABP. An IABP is inserted in the intra-arterial space through the femoral artery and positioned in the descending thoracic aorta distal to the origin of the left subclavian artery. Deflation of the balloon occurs during systole, generating negative pressure and driving more blood from the left ventricle to the aorta. During diastole, the balloon inflates and positive pressure increases the blood flow toward the coronary arteries. One study suggested a mortality benefit if the IABP is used during cardiogenic shock in this group of patients (47). The IABP-SHOCK and IABP-SHOCK II trials did not demonstrate a mortality advantage with the IABP in cardiogenic shock, although individuals with a mechanical complication

were excluded (48, 49). Despite conflicting evidence, the 2013 guidelines of the American Heart Association (AHA) for STEMI provide a IIa recommendation with level of evidence B for the use of the IABP in cardiogenic shock. An Impella left ventricular assist device contributes with a higher blood flow and a more significant preload reduction when compared to an IABP (3). Although its use in AMR has been reported scarcely, it is another option for mechanical support in patients with shock.

3.3. Definitive Treatment

Surgical management is key in patient survival since the heart adapts poorly to the hemodynamic changes resulting from acute regurgitation (7, 10) Older studies reported a mortality rate of up to 50% with surgical management (36, 37, 50) More contemporary studies suggest that this rate may be lower. Lorusso and colleagues5 in a retrospective multicenter study that included 279 patients from 1986 to 2007 found a 30-day postoperative mortality of 22.5%. Mortality from AMI was significantly higher when compared to infective endocarditis and degenerative disease. This study also compared mitral valve repair and mitral valve replacement and reported no difference in short and long-term outcomes. Patients who had simultaneous surgical revascularization demonstrated an improved long-term survival. Tavakoli (51) and Chevalier (52) reported a 22% and 24% postoperative mortality in patients undergoing emergent surgery of the mitral valve, respectively. Another study with 54 patients followed up from 1980 to 2000, where 91% presented with cardiogenic shock, found a 10-year survival rate of 35% and an operative mortality of 18.9% (35). Interestingly, the mortality rate decreased to 8.7% after 1990, although CABG was also performed in this group. Another study 51 reported a 10-year survival rate of 49.5% in a population of 54 patients. Postoperative mortality was not included. Chen et al. (53) studied 33 patients over a 22-year period and reported an in-hospital mortality of 21%, with 5- and 10-year survival rates of 65 ± 8.6 years and 32 ± 9.7 years, respectively. When ischemic AMR does not result from the disruption of the subvalvular apparatus, medical therapy including revascularization can suffice for patient recovery (7). Surgery is still recommended if the goals of treatment are not achieved. Tcheng et al. (45) followed up 50 patients with AMI and moderate-to-severe mitral regurgitation over the course of 4 years. Mitral regurgitation improved in only 3 patients who were revascularized with balloon angioplasty. Unfortunately, this research group failed to provide detail about the exact mechanism of valvular dysfunction (organic vs. functional).

The period of time between the onset of AMR and surgery must be as abbreviated as possible (40, 52). One study with 11 patients found that intervention within the first 24 to 48 hours was associated with increased survival (37). There is a general agreement that emergent surgery should be pursued when AMR is a consequence of papillary muscle rupture (52). Chevalier (52) did not find a mortality difference when comparing patients who had surgery before day 6 of admission with those who had surgery afterward. This information suggests that delay in surgery does not provide an additional benefit which sometimes is of concern for the surgeon since during the initial phase of the disease, the extent of the necrotic tissue can impair the adequacy of surgery. Multiorgan dysfunction usually ensues; and if surgery is implemented early enough, damage can be limited.

It is the surgeon's discretion how to return the mitral valve to its normal function. In chronic mitral regurgitation, mitral valve repair is superior to replacement (35, 54). A retrospective analysis of 58,370 patients in the Society of Thoracic Surgeons Adult Cardiac Database who had isolated mitral valve surgery found that reoperation and valve-related complications were less common in the mitral valve repair group (55). Although for our patient's scenario there is not enough evidence to advocate one of these methods in most of the studies published thus far the overwhelming majority of the patients had their mitral valve replaced (56, 57). Lorusso and et al. (5) did not find any difference in short- or long-term outcomes between the replacement and the repair of the mitral valve. Interestingly, the mitral valve was replaced in 70% of the patients, while repair was performed in the rest. Cohn (58) documented in his research an increase in the 5-year survival rate, in favor of mitral valve replacement. His assessment was that significant dilatation of the mitral annulus, scarring of the papillary muscles, and remodeling of the left ventricle reduced the probability of a good operative outcome if repair was to be done. Other authors have described annular dilatation and involvement of the posterior leaflet as favorable features for valve repair (7). Tavakoli (51) in his review of 93 patients did not demonstrate any difference between repair and replacement. Of note, only 19 of these patients underwent surgery emergently, and all the cases of papillary muscle rupture were managed with the replacement of the valve as repair was unsuccessful in the latter due to the significant amount of necrotic tissue. Bouma and colleagues55 followed up 50 patients and reported an 80% rate of valve replacement and 20% of repair. One valvular repair failed and was switched to replacement. The methods described for repair included annuloplasty, reimplantation of the papillary muscle, and quadrangular resection of the P2 segment. Mitral valve repair did not outperform replacement unless the subvalvular apparatus was not preserved during the operation. Perhaps preservation further protects left ventricular contractile function (59). Schroeter et al. (60) retrospectively analyzed 28 patients between 2002 and 2010 and reported that 25 patients had their mitral valve replaced and 3 had repair.

Studies support concurrent revascularization with mitral valve surgery. In one of the largest trials to date, CABG performed simultaneously reduced the death rate from 42% to 8.7% when compared with non-revascularized patients although statistical significance was not achieved (34). Lorusso (5) described a 15-year survival of approximately 60% with CABG versus 23% in the group who only

had mitral valve surgery. In the study by Tavakoli, (51) 56% of the patients were alive at 10 years and all of them had surgical revascularization. Chevalier (52) actually observed an impact in perioperative mortality, although the population studied was different from that of many of the previous reports as patients were taken up for surgery within the first month, and many within the first 24 hours. Mortality was reduced from 34% to 9% in the group who had CABG and mitral valve surgery combined. This finding has been reproduced in other studies (61) and is probably related to the contribution of coronary artery disease to myocardial ischemia and the patients' poor hemodynamics. Other authors have not found a survival advantage with concomitant CABG.55 Schroeter et al. (60) found a nonstatistically significant impact of CABG on survival, although there was a difference of 24% when compared to those who did not have CABG in favor of those who did. Moreover, 32% of the patients had percutaneous coronary intervention. An analysis comparing patients with any type of revascularization with those who did not was able to show a statistically significant difference. Some limitations of these studies include a small sample size and the absence of any mention of revascularization on further follow-up. A complex assessment should be made to determine whether ischemia has a significant contribution to cardiogenic shock and whether revascularization is necessary emergently or can be safely delayed. We know from the SHOCK Trial that a 13% absolute risk reduction in mortality at 1 year can be reached with early revascularization in patients with cardiogenic shock secondary to AMI (62).

As was mentioned above, mortality is almost certain if the mitral valve is not repaired or replaced. Thus, discriminating individuals who would not benefit from surgery is extremely difficult. Refractory heart failure, infective endocarditis, and prosthetic valve dysfunction elevate the risk of death in the perioperative setting (1). Bouma et al. (54) found that a EuroSCORE of more than 40%, EuroSCORE II more than 25%, preoperative use of inotropes, and valve replacement without preservation of the subvalvular apparatus were associated with a higher long-term mortality. Chen et al. (53) also identified the preoperative use of inotropes in addition to renal failure and mechanical ventilation as predictors of mortality. For those patients who do not qualify for surgery based on the criteria of the cardiovascular team, the MitraClip may be an alternative. Its use in this scenario has been described in case reports (38, 39).

Indicators of poor prognosis in the postoperative setting have been studied. These can be of help when deciding how much more aggressive our management should be and whether certain interventions are worth implementing. Hemodialysis and extracorporeal membrane oxygenation are linked to poor survival (61).

3.4. Conclusions

Papillary muscle rupture is a life-threatening mechanical complication of AMI. Physical examination has poor sensitivity and specificity in its diagnosis. Echocardiography with color and flow Doppler is the gold standard and has the ability to rule out other mechanical complications such as ventricular septal rupture and free-wall rupture. Quantitative measurements such as regurgitant fraction and volume and regurgitant orifice area tend to underestimate the severity in contrast to the situation in chronic mitral regurgitation. TEE is recommended when transthoracic imaging provides poor acoustic windows. High image resolution can be obtained with real-time 3D TEE and sometimes aid the surgeon in deciding how to approach the valvular lesion.

Emergency surgery with the repair or replacement of the mitral valve is in general the only treatment that can save the patient's life since medical treatment is associated with an 80% mortality rate and when combined with revascularization survival can be as high as 80%.

3.5. Videos

Video 1. Parasternal long-axis view in a transthoracic echocardiogram. There is incomplete coaptation of the mitral valve leaflets with the prolapse of the posterior leaflet during systole. Akinesis of the inferolateral wall is also evident.

Video 2. Four-chamber view in a transthoracic echocardiogram. Akinesis of the anterolateral wall with compensatory hyperkinesis of the septum is evident. Prolapse of the posterior leaflet is somewhat visible.

Video 3. Two-chamber apical view in a transthoracic echocardiogram. A dumbbell structure evident during diastole inside the left ventricle appears attached to the mitral valve, representing a ruptured papillary muscle.

Video 4. An LAO 30° view of the left coronary system. A cut-off sign is present at the proximal portion of the left circumflex coronary artery.

Video 5. An RAO 20° view with a caudal 20° view of the left coronary system. Again, a cut-off sign is seen at the proximal portion of the left circumflex coronary artery. The proximal left anterior descending artery has what appears to be a moderate stenosis, although a better assessment of its severity is done in the LAO 45° view with a cranial 25° view.

Video 6. An LAO 45° view with a cranial 25° view of the left coronary system. The proximal stenosis of the left anterior descending artery is measured here at 80%.

Video 7. An RAO 30° view of the right coronary artery. Engagement is poor due to severe proximal stenosis. The right coronary artery appears to be nondominant and with a high bifurcation.

Footnote

Authors' Contribution: Keith Suarez contributed in writing the abstract and the case presentation; Kipp Slicker and Victor Torres contributed in writing the discussion and the references.

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