SCIENTIFIC LETTER

DEFERRED CLOSURE IN THE SUCCESSFUL TREATMENT OF POST-INFARCTION VENTRICULAR SEPTAL RUPTURE

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ABSTRACT

Ventricular septal rupture is a rare but highly lethal mechanical complication of acute myocardial infarction. The gold-standard treatment is surgical repair; with conservative treatment, 90% mortality is estimated in the following two months. We present the case of an 81-year-old patient with post-infarction SVR who underwent angioplasty and was later admitted to the intensive care unit to assess hemodynamics to prepare him for delayed closure. Surgical repair of the defect was performed 21 days later, and the patient was discharged to the cardiovascular intensive care unit with adequate postoperative evolution.

Keywords: *mechanical complications of acute myocardial infarction, acute myocardial infarction, ventricular septal rupture, surgical repair ventricular septal rupture.*

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INTRODUCTION

Ventricular septal rupture (VSR) is a mechanical complication of acute myocardial infarction (AMI). The gold standard of treatment is surgical intervention; however, it has a very high mortality rate. Early repair is recommended when hemodynamic instability is present; however, the time indicated to establish management has yet to be precisely defined. Due to the low incidence of this condition, most of the information comes from cohorts or national patient registries.

CASE PRESENTATION

We present the case of an 81-year-old male patient referred for acute coronary syndrome of the acute myocardial infarction type with positive ST-segment elevation; he has a history of systemic arterial hypertension of ten years of evolution and receives treatment with angiotensin two receptor antagonists (ARA 2). He has no other chronic degenerative diseases.

The current presentation began at 18:00 hours the day before admission with oppressive chest pain of intensity 10/10 on the visual analog scale, accompanied by a vagal response characterized by diaphoresis and nausea. He came to the general hospital at 19:00 hours with vital signs: blood pressure of 188/83 mmHg, heart rate of 96 beats per minute, respiratory rate of 22 breaths per minute, and oxygen saturation of 96%. The first electrocardiogram (ECG) showed positive ST-segment elevation of V1-V4; acetylsalicylic acid, clopidogrel, atorvastatin, and enoxaparin were administered. The patient was transferred to the coronary intensive care unit with a diagnosis of AMI; he was admitted 5 hours and 50 minutes after the onset of symptoms, hemodynamically stable, with anginal pain of intensity 5/10 and no other added symptoms. A new ECG was performed, which confirmed anterior infarction; auscultation showed a bar murmur predominantly in the apex. A transthoracic echocardiogram was requested, showing a rupture of the interventricular (IV) septum of 5 mm in the apical portion, left ventricular ejection fraction of 45%, and mobility alterations (septoapical, lateroapical, inferoapical and anteroapical akinesia; inferoseptal hypokinesia of the middle segment, anterior akinesia of the middle segment and anteroseptal akinesia of the middle segment). In the hemodynamics department, coronary angiography showed disease of two main vessels: anterior descending artery (AD) with 99% subocclusive lesion in the middle segment and posterior descending artery with focal distal segment lesion of 75%. The percutaneous coronary intervention of the LAD was performed with the placement of a 3 x 38 mm XIENCE Sierra™ stent with TIMI 3 end-flow (thrombolysis in myocardial infarction). In the second stage, we worked on the right coronary artery by placing a 3 x 38 mm Resolute Onyx™ stent in the distal segment with normal arterial flow (TIMI 3). He was admitted to the cardiovascular intensive care unit. A new ECG showed a 15 mm IV septum rupture with a left to right shunt and a pressure difference of 3:1. During his stay, a medical-surgical session was held, and it was agreed to close the defect with a pericardial patch three weeks after the AMI. On the 21st day after the IV septum rupture, hemodynamic deterioration, data suggestive of cardiogenic shock, and heart failure refractory to treatment, so vasopressor therapy was initiated. Hemodynamic studies showed evidence of decreased peripheral vascular resistance, so it is considered for emergency surgery.

The patient is admitted to the operating room; the approach is performed by median sternotomy, arterial and bicaval cannulation, the total cardiopulmonary shunt is started, and aortic clamping is performed. A right ventriculotomy is performed with a cut parallel to the IV septum, a 3 x 2 cm defect is identified (*Figure 1*), a bovine pericardium patch is placed (*Figure 2*) and closed with Prolene™ U-stitches 4-0 with pledgets (*Figure 3*). Then a ventriculorrhaphy is performed with a sandwich technique with Prolene™ 2-0 with pledgets (*Figure 4*). Trans-operative findings were global grade III cardiomegaly, a macroscopic trace of infarction in the inferior and anterior face, and an apical fenestrated ventricular septal defect of 3 x 2 cm. After surgery, he was admitted to the intensive care unit, intubated, and with aminergic and vasopressor support; he was decannulated 24 hours later, and vasopressor support was withdrawn 48 hours later. The patient was transferred to the general ward at 72 hours, clinically stable, without mechanical ventilatory support or vasopressor support, with borderline mean arterial pressure, afebrile, and no evidence of shock. A new control echocardiogram was performed, where the residual defect of the surgical closure was reported as hemodynamically not significant, with a left ventricular ejection fraction of 32% and ischemic cardiomyopathy with moderate myocardial damage. He had a good postoperative evolution and was discharged home without complications.

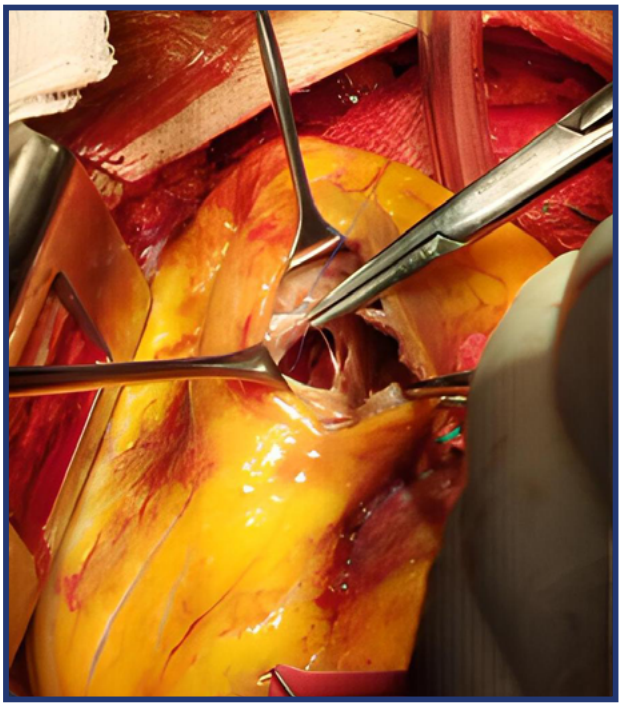

FIGURE 1. Interventricular septal defect.

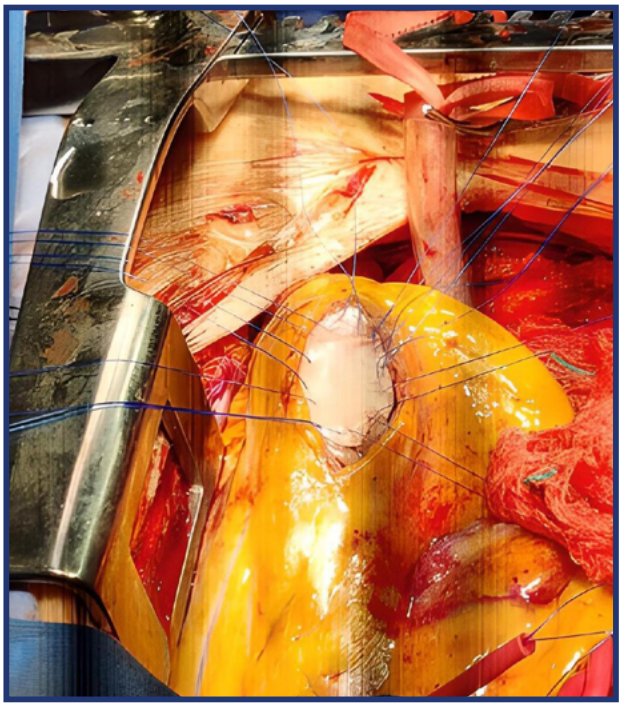

FIGURE 2. Placement of a bovine pericardium patch in a 3 x 2 cm defect.

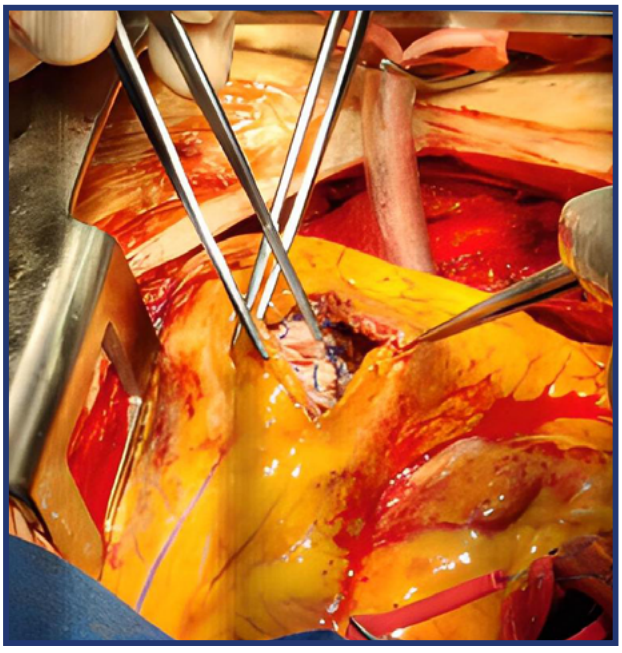

FIGURE 3. U-point closure with pledgets.

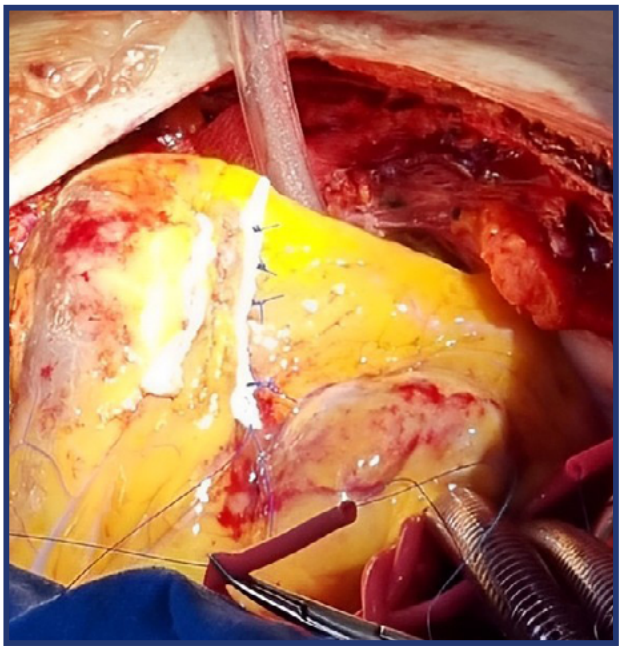

FIGURE 4. Ventriculorrhaphy with sandwich technique with pledgets.

DISCUSSION

SVR is the most common mechanical complication of AMI and carries a very high lethality; it occurs in approximately 0.21% of patients with ST-segment elevation infarctions and 0.04% of those with non-ST-segment elevation infarctions[1].

In this reperfusion era, risk factors for developing post-infarct SVR include older age, female sex, smoking, hypertension, right ventricular infarction, and extensive AMI[2]. The report Elbadawi et al. obtained from the National Inpatient Sample (NIS) database from 2003 to 2015 evaluated mechanical complications arising from ST-segment elevation and non-ST-segment elevation AMI. The sample included 13,767 patients who had mechanical complications; 10,344 (75%) developed SVR, and the remaining patients had acute mitral insufficiency due to papillary muscle rupture (19%) or free wall rupture (6%)[3]. ST-segment elevation infarcts are more likely to be associated with transmural infarction than non-elevation infarction. A transmural AMI is a prerequisite for SVR and is, therefore, more

common after ST-segment elevation infarction[1]. This mechanical complication usually occurs between day 2 and day 6, although it can occur at any time in the 2 weeks following acute infarction. The average time to onset was one day in the GUSTO-I study (Global Utilization of Tissue Plasminogen Activator and Streptokinase for Coronary Artery Occlusion), four days in the American Heart Association Study of Mechanical Complications of AMI, and 16 hours in the SHOCK study[4,5]. Depending on the presentation of the septal rupture (the size of the infarct, the degree of shunt present, and the associated right ventricular failure), patients may present with relative hemodynamic stability or frank cardiogenic shock. The most important determinant of the outcome of SVR management is the development of heart failure (left, right, or both) and cardiogenic shock. The severity of heart failure is related to the extent of myocardial necrosis and left-to-right shunt[1]. Today, surgical closure is the definitive treatment for post-infarct ventricular septal rupture. Management usually includes excision of all necrotic tissue, patching of the defect, and myocardial revascularization[6]. Without surgical treatment, 90% of patients will die within two months[4]. Arnaoutakis et al. published results obtained from the 1999-2000 cohort of the STS (Society of Thoracic Surgeons) National Database, where they studied 2,876 patients who underwent surgical repair for post-infarction SVR. They reported an overall 30-day in-hospital mortality rate of 42.9%, with a marked decrease with deferred closure (54.1% mortality with surgical repair in the first seven days versus 18.4% after this time). Risk factors that increased trans-operative mortality included age, female sex, shock, inferior infarction, use of intra-aortic balloon counterpulsation, dialysis, mitral insufficiency, reoperation, emergency surgery, and time to repair4. Other investigators found that mortality was higher (60%) in patients who underwent surgery within the first 24 hours[7,8].

The international multicenter retrospective multicenter retrospective cohort study CAUTION (Mechanical Complications of Acute Myocardial Infarction) included patients who received surgical treatment for mechanical complications arising from acute myocardial infarction. The study included 475 patients who underwent surgery for post-infarction SVR from 26 centers in different countries from January 2001 to December 2019. The main findings of this study were as follows:

- The early mortality rate was 40.4%.
- Advanced age, preoperative cardiac arrest, percutaneous revascularization, and postoperative requirement of intraaortic balloon counterpulsation or extracorporeal membrane oxygenation were independently associated with early mortality.
- Longer times between AMI and SVR and between SVR and surgery were associated with lower mortality.
- Recurrent SVR was not associated with increased mortality[9].

It is still difficult to determine the optimal time for definitive surgical repair. The American College of Cardiology and the American Heart Association guidelines recommend emergency surgical repair regardless of the patient's hemodynamic status, so the ideal timing is also controversial and should be individualized. Early repair should be considered in stable patients without organ failure and favorable anatomy. In stable patients with complex anatomy or friable tissues, delayed intervention should be considered[5].

The pathophysiological mechanism involves excessive transmural myocardial necrosis followed by rupture or extensive scarring of the affected tissue. The conventional mechanism of SVR involves coagulation necrosis of ischemic tissue with neutrophil infiltration, which causes thinning and weakness of the septal myocardium; this subacute process requires three to five days. On the other hand, rupture occurring within 24 hours of presentation is more likely to result from dissection of an intramural hematoma or hemorrhage in the ischemic myocardium[8].

In early repair, the intervention is performed around the infarcted area in friable myocardial tissue, which increases the possibility of expanding the size of the SVR. This would explain the high in-hospital mortality rate compared to delayed closure[6].

The intervention delay has a mechanistic rationale: after infarction, metalloproteinase activity and tissue degradation peak on day seven, whereas new collagen deposition begins between days two and four, and necrotic myocytes are entirely replaced by collagen by 28 days. Therefore, deferral could facilitate successful closure by allowing friable tissue to organize, strengthen, and differentiate well from surrounding healthy tissue activity, allowing peak tissue breakdown to occur on day 7. The connective tissue and scar formation around the defect promote better anchorage for the suture material and decrease the potential for patch dehiscence[5]. In surgical repair, myocardial revascularization should be performed first to prioritize myocardial protection. Techniques include Dagget's technique, primary closure of the defect, and David's technique, which consists of endocardial patch placement with infarct

exclusion and is currently the most widely used in the world[5]. For anterior SVR, the infarcted area of the anterolateral left ventricle should be incised parallel to the LAD, as the septal defect is usually located below the incision. A patch of pericardium or synthetic material with U-sutures with pledgets should be used in the non-infarcted area of the right ventricle to exclude the entire portion of the LV septum from the mitral annulus to the anterolateral LV wall. True apical SVRs can be repaired, and primary closure can be performed by amputating the apex. Posterior SVRs are approached via ventriculotomy on the infarcted posterior LV wall parallel to the posterior descending, with patch suturing to the LV side of the non-infarcted septum with patch closure, primary closure, or infarct exclusion, depending on how much of the LV free wall is involved[5]. If deferred closure is chosen, ventricular assist devices (VADs) are a valuable bridge to surgery; decreasing afterload and preload helps to increase coronary perfusion in the affected myocardium. Venoarterial extracorporeal membrane oxygenation has also demonstrated numerous benefits compared to VADs, as it prevents sternotomy, provides oxygenation support, and is easily reversible; it also allows hemodynamic stabilization, recovery or prevention of organ failure and washout of the dual antiplatelet effect, and is a decision-making strategy[4]. Presently, percutaneous closure devices allow less invasive management in this type of patient; in selected cases (defects <1.5cm, subacute stage, and patients who are not good candidates for surgery), they are viable. However, in a case series of 29 patients with this type of management, 41% experienced procedure-related complications, and the overall survival rate at 30 days was 35%, with higher mortality in patients with cardiogenic shock[6].

CONCLUSIONS

Post-infarct SVR is a severe condition with challenging medical and surgical treatment; despite multiple advances in infarct management and surgical repair techniques, the mortality rate has not changed significantly for decades. According to the databases and cohorts reviewed, a better prognosis has been observed with deferred closure; however, more prospective studies are required that include improved surgical techniques and preoperative management to enhance the current suboptimal early mortality rate. In our Institute, and based on literature reports and our center's experience, the management algorithm for this mechanical complication consists of admitting the patient to the coronary intensive care unit, optimization of pharmacological management, support with ventricular support, and performing surgical repair after 14 days following the event. In case of hemodynamic instability, intervention with mechanical circulatory support and surgical correction after seven days is considered since satisfactory results are obtained.

Declarations

The authors declare no conflict of interest.

Acknowledgments

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