SCIENTIFIC LETTER

SUCCESSFUL SURGICAL MANAGEMENT OF THREE MECHANICAL COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION: VENTRICULAR SEPTAL DEFECT, SEVERE MITRAL REGURGITATION AND VENTRICULAR ANEURYSM

ABSTRACT

Mechanical complications after acute myocardial infarction have a very high mortality rate on their own (40-50%), and the sum of these increases the risk of mortality after surgical repair. In this case report, we present a patient with a postinfarction ventricular septal defect of posterior location and early presentation with hemodynamic stability. Subsequently, the patient presented with severe acute mitral regurgitation plus posterior ventricular aneurysm. A surgical procedure with successful resolution is performed. Early diagnosis, patient selection, and adequate multidisciplinary management optimize successful outcomes. This communication aims to expose a clinical case with these characteristics since, in the literature, there are very few cases of living patients with these three complications who survived the acute event and even more after surgical repair.

Keywords: *post-infarction complications, ventricular septal defect, mitral regurgitation, ventricular aneurysm.*

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INTRODUCTION

Although the incidence of mechanical complications in patients after acute myocardial infarction (AMI) is low (0.21%), the associated mortality rate separately is very high (40-50%)[1]. However, given the sum of these and their presentation with subsequent localization and, in addition, the time to surgical resolution, especially in elderly patients, the therapeutic options are usually complex and require the experience of a multidisciplinary team.

Over the past 30 years, improvements in timely reperfusion techniques within regionalized care systems and advances in optimal medical therapies have reduced AMI mortality rates. However, nowadays, patients present with more comorbidities. The clinical characteristics of these mechanical complications differ and depend on the rupture site (the free wall of either the ventricle, the interventricular septum, or the papillary muscles). Ventricular septal rupture (75%) is the most frequent type of mechanical complication after AMI; the remaining patients develop mitral regurgitation (19%) due to papillary muscle rupture or free wall rupture (6%)[2]. The optimal timing of corrective surgery for mechanical complications is still controversial in the medical community. The main factor determining survival after the surgical procedure is the timing of the procedure, so most centers defer corrective surgery whenever possible[3].

CASE PRESENTATION

A 71-year-old male patient, hypertensive, with a history of chronic kidney disease KDIGO III and positive smoking, presented to the emergency department with precordial pain 9/10 on the visual analog scale (VAS); an electrocardiogram was performed showing positive ST-segment elevation in DII, DIII, and AVF with initial Q wave, as well as third-degree atrioventricular block. A temporary transvenous pacemaker was placed. Ten days later, the patient presented hemodynamic instability and bacteremia from a urinary tract infection (with the presence of pyuria). Antibiotic treatment was started. Once hemodynamically stable and no longer receiving vasopressor drugs, the patient presented a pericardial effusion of 18 mm without cavity collapse detected by transthoracic echocardiography (TTE). It was decided to admit him to the post-surgical therapy unit for close monitoring. A positive blood culture for Acinetobacter baumannii was received three days later, so the antibiotic was rotated. The patient evolved with clinical improvement, and two weeks after the initial event, he presented hemodynamic instability and cardiorespiratory arrest that required orotracheal intubation and amine management. A Levine grade III-IV mitral murmur radiating to the axilla was detected and blood cultures were requested, which showed Pseudomonas aeruginosa. Conservative treatment was decided, and the patient progressed to anuria. With hemodialysis support, the administration of vasopressor drugs was suspended, diuresis improved, and cultures were negative. Forty days after the initial event, a transesophageal echocardiogram (TEE) was performed, which showed an apical and posterior ventricular septal defect, 5 mm long, with left to right shunt with maximum trans ventricular septal defect gradient of 64 mmHg, Vmax of 4 m/s, ratio between pulmonary output and systemic output (Qp/Qs) of 3.3; basal and mid-inferior inferolateral akinesia, presence of pseudoaneurysm involving the basal and mid-segment, with a neck of 19 mm and a depth of 21 mm, and a left ventricular ejection fraction (LVEF) at rest of 48%. The mitral valve showed moderate insufficiency secondary to symmetric tenting of its leaflets, with a 5 mm jet vena contracta and a pulmonary artery systolic pressure of 40 mmHg.

The patient was referred to the hemodynamics service with evidence of a single lesion in the circumflex artery and posterobasal VSD (*Figure 1*), and it was decided to perform surgical correction of the three post-infarction complications.

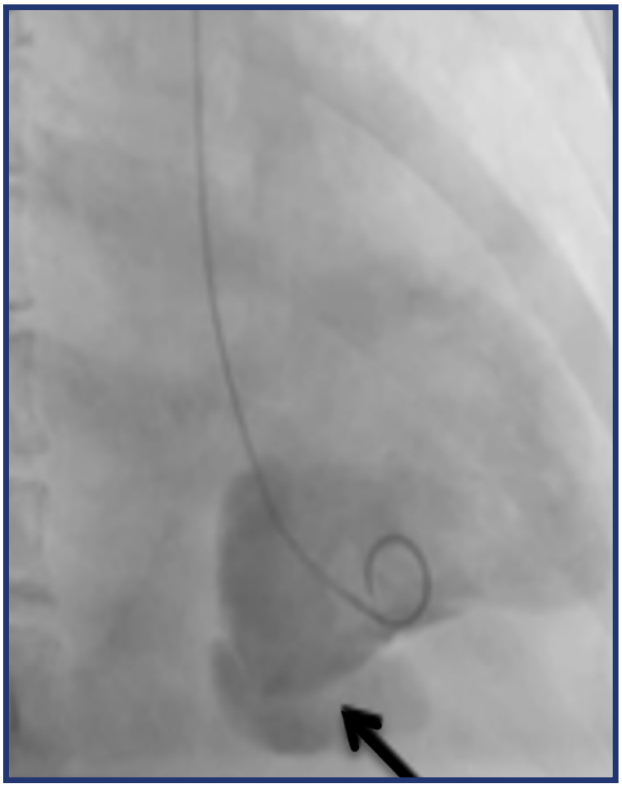

FIGURE 1. Catheterization with ventriculogram. There is evidence of a high posterior ventricular septal defect.

Due to the history of hemodynamic instability and the presence of an echocardiographic image compatible with contained ventricular rupture, dissection and peripheral cannulation of the femoral artery and vein were performed before sternotomy. Once the sternotomy was performed, lax adhesions were observed, with an area of hematoma on the posterolateral and inferior aspect of the left ventricle (*Figure 2*, yellow arrow).

Once free of adhesions, the superior vena cava was cannulated, aortic clamping was performed, and the cardioplegic solution was administered, in addition to maintaining the patient in moderate hypothermia (32-34 °C) for myocardial protection. Left ventriculotomy was performed through the area of the pseudoaneurysm, which extended very close to the posterior atrioventricular groove. Once the ventricular cavity was exposed, the ventricular septal defect was visualized approximately 1.5 cm x 1.5 cm, 5 mm from the posteromedial commissure (*Figure 2*, green arrow).

A patch was placed from Dacron™ with a 2-0 Ethibond suture with pledget and reinforced with a 3-0 Prolene continuous surge (*Figure 3*).

An endoaneurysmorrhaphy was performed with a 3-0 Prolene pericardial patch (*Figure 4*), and the closure was reinforced with the sandwich technique.

An incision was made in Waterston's groove for the left atrial approach. Visual examination of the mitral valve confirmed the presence of an area of mitral regurgitation between P3 and A3 (*Figure 5*).

According to the pre-surgical TTE, the U-stitch plasty was performed with 5-0 Prolene, and a hydraulic test was performed, which showed a competent mitral valve. It was decided not to revascularize due to the pathological process and repair zone in the circumflex artery territory. The patient was weaned from extracorporeal circulation, and decannulation was performed with minimal doses of inotropic drugs.

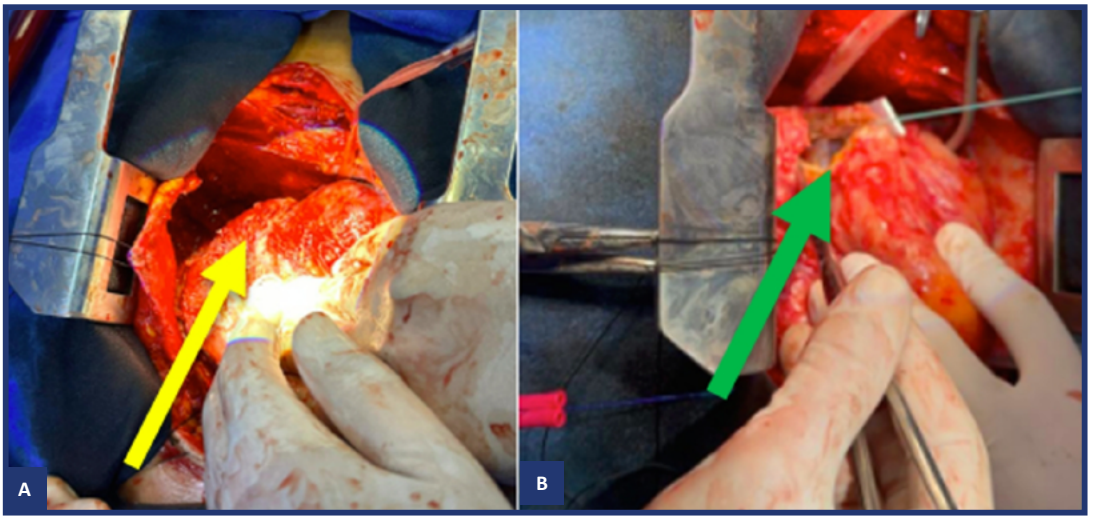

FIGURE 2. A. Collapsed aneurismatic zone (left ventricle is empty) (yellow arrow). **B.** Approach of the ventricular septal defect through the aneurysm (green arrow).

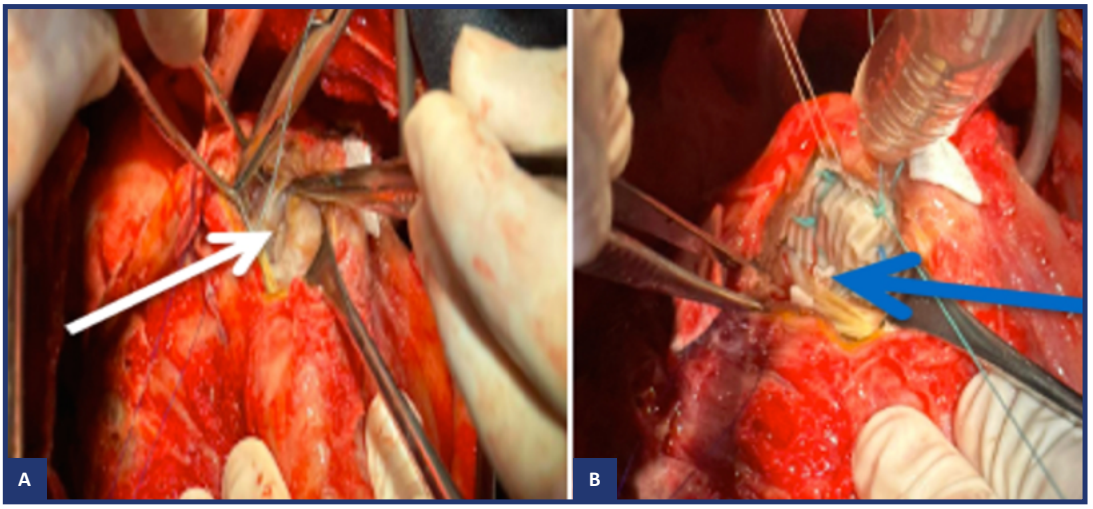

FIGURE 3. A. Presence of a 1.5 cm x 1.5 cm ventricular septal defect (VSD) (white arrow). **B.** Closure of the VSD with a patch of Dacron™ (blue arrow).

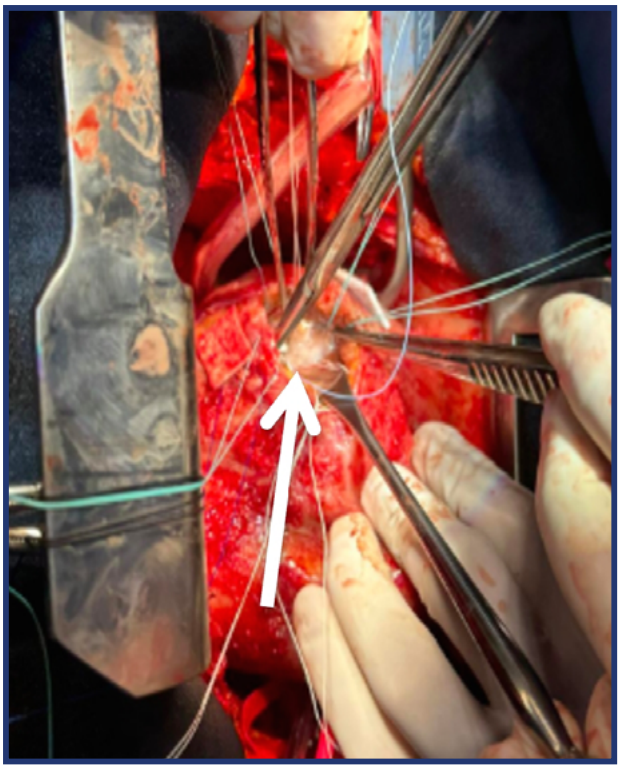

FIGURE 4. Pericardial patch placement for endoaneurysmography.

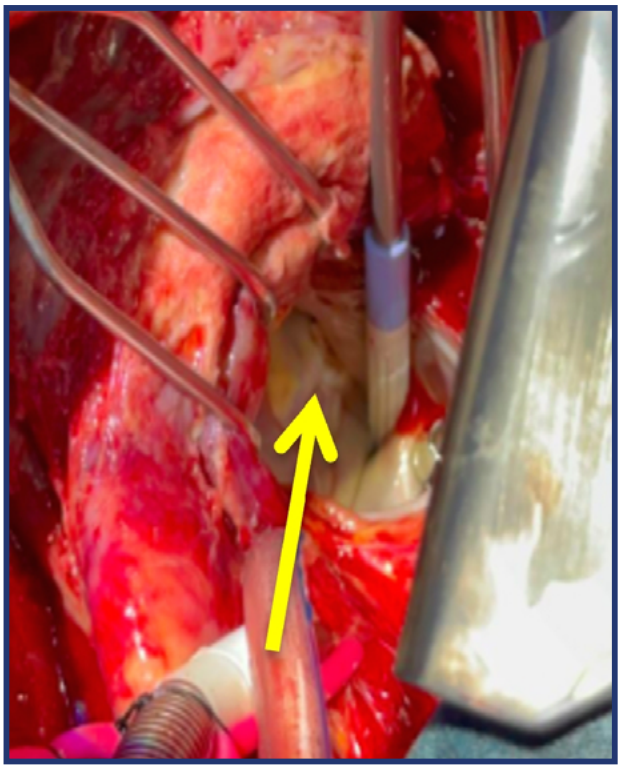

FIGURE 5. Alfieri mitral valve plasty at A3 and P3.

In the postoperative evolution, minimal doses of inotropic drugs and vasopressors were used, and the patient presented adequate diuresis and normal acid-base balance during the first hours of the postoperative period (POP). He was weaned from mechanical ventilation at 48 hours without complications.

Eight days postoperatively, the patient was transferred to the general ward, already extubated and with hemodynamic status. An echocardiogram showed an LVEF of 43%, no residual interventricular shunt, and no evidence of residual aneurysm and mild mitral insufficiency (*Figure 6*).

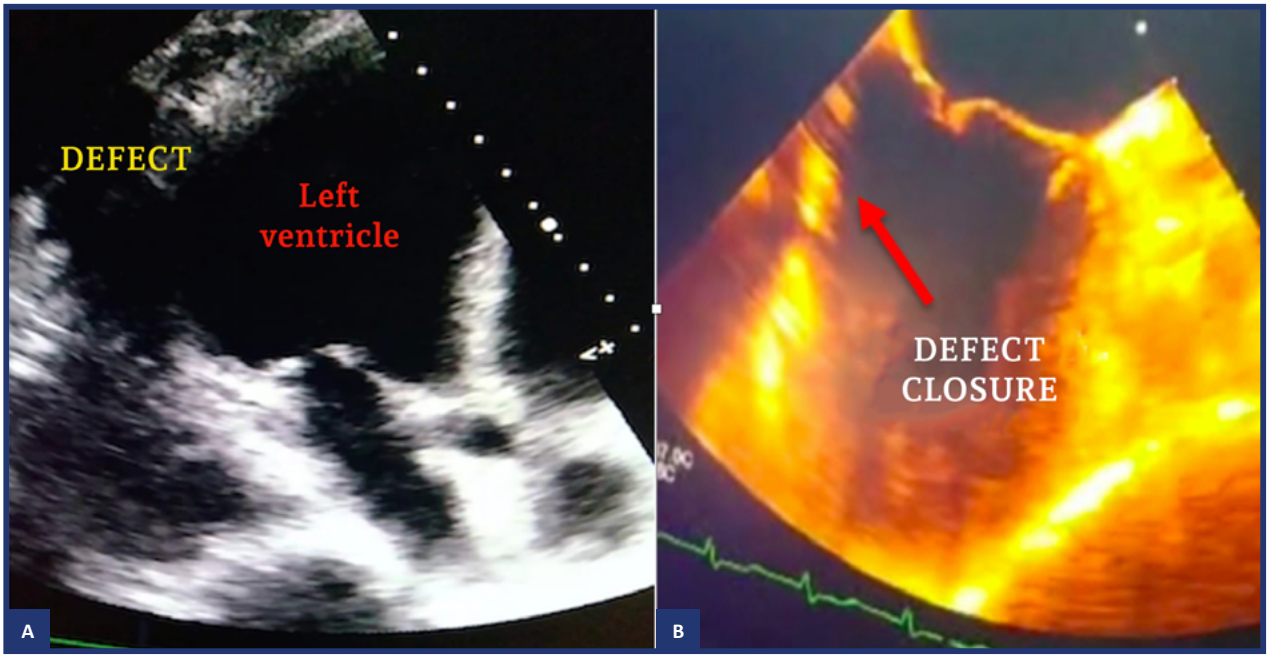

FIGURE 6. Transesophageal echocardiogram before **(A)** and after surgical repair **(B)**.

DISCUSSION

The optimal timing of definitive surgical repair is still difficult to establish. Although the 2013 guidelines of the American College of Cardiology and the American Heart Association[4] recommended emergency surgical repair of postinfarction ventricular septal defect (PIVSD), whatever the hemodynamic status, the timing of surgery in the setting of ventricular septal rupture is still a matter of controversy and should be considered in each patient on an individual basis.

Early corrective surgery should be considered in hemodynamically stable patients with favorable cardiac function and anatomy because sudden and unpredictable hemodynamic compromise is often observed.

Deferred surgery may be considered in stable patients when the surgical anatomy is complex, and there is concern about tissue fragility and the ability to perform a definitive repair.

The perceived benefit of delayed surgery has a mechanistic basis: after infarction, metalloproteinase activity and tissue degradation peak at day 7, whereas new collagen deposition begins between days 2 and 4, and collagen completely replaces necrotic myocytes by 28 days; therefore, a delay may facilitate successful repair by allowing friable tissue to organize, strengthen, and differentiate well from surrounding healthy tissue.

In this scenario, close follow-up in the intensive care unit may allow tissue healing and promote the chances of definitive repair[5] or use some form of ventricular support to manage hemodynamic instability if appropriate.

The 2017 European Society of Cardiology guidelines promote late elective repair in patients initially responding to aggressive conservative management6. Another of the most challenging problems for cardiac surgeons to solve is determining how best to approach a patient with ischemic mitral insufficiency (IMI).

The problem in IMI is not the valve but a ventricle disease due to the dilatation. For this reason, the evolution of IMI is very dynamic (mild to severe insufficiency); this is mainly related to preload and afterload conditions but often also to ongoing ischemia[7]. The surgeon's expertise is establishing the surgical treatment, which may be valve replacement or plasty. In the case presented here, it was decided to perform only mitral valve repair because it was complex (defined by the presence of two other mechanical complications).

In their publication, Kron et al.[7], a team composed mainly of surgeons who analyzed and synthesized the data and established consistent recommendations for surgeons, including how and when to repair or replace the valve, have accepted that surgical treatment is daunting. In addition, they discussed in detail the role of percutaneous therapy.

Myocardial revascularization in this type of patient is still controversial, mainly because of morbidity and mortality, which increase the longer the surgical time and cardiopulmonary bypass. On the other hand, in patients with recent infarction, inflammatory tissue in the infarct zone and, as in the case presented here, adhesions limit the exposure of the coronary vessel to be revascularized. If the hybrid approach is achieved, the result will be better.

Finally, aneurysms, most commonly affecting the LV, occur after myocardial infarction secondary to transmural myocardial necrosis; this tends to be a late complication where the affected segments of myocardial tissue are gradually replaced by akinetic fibrotic tissue, which is scar tissue that can dilate[8]. In our case, we performed endoaneurysmorrhaphy with a pericardial patch, and, as seen on TTE, there was no macroscopic difference in ventricular volume despite infarction and repair.

Declarations

The authors declare no conflict of interest.

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