



Case report

Extremely Late Post-Infarction Ventricular Septal Rupture: A Case Report of Successful Surgical Repair

Diego Davila-Flores^{1,a}, Adriana Gutiérrez-Yllu^{2,b}, Brenda Baltodano-García^{3,b}, Wendy Junes-Gonzales^{1,c}, Renee Montesinos-Segura^{1,a}

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Authors' affiliation

¹ Instituto Nacional Cardiovascular, INCOR-EsSalud, Lima, Perú.

² Facultad de Medicina, Universidad Peruana de Ciencias Aplicadas, Lima, Perú.

³ Facultad de Medicina, Universidad de San Martín de Porres, Lima, Perú.

^a Cardiologist.

^b Medical Doctor.

^c Cardiologist subspecialized in cardiovascular imaging.

Correspondence

Diego Davila-Flores
Av. Coronel Zegarra 417, Jesús María, Lima, Perú.

Email

diegodavilafmh2408@gmail.com

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ABSTRACT

Mechanical complications of ST-elevation myocardial infarction have become rare in high-income settings due to timely reperfusion. However, they remain more prevalent and carry a high mortality in regions lacking access to early reperfusion. We present a 62-year-old man with inferior STEMI managed conservatively without reperfusion. At four months, he developed progressive dyspnea. One year later, echocardiography revealed a large basal ventricular septal rupture and severe eccentric mitral regurgitation, all absent at discharge. The patient's transfer to a cardiovascular surgery center was delayed by two years, a consequence of the COVID-19 pandemic. Imaging confirmed an inferobasal left ventricular pseudoaneurysm, basal ventricular septal rupture, severe mitral regurgitation, and a left ventricular ejection fraction of 45%. Right heart catheterization showed a significant left-to-right shunt; coronary angiography revealed multivessel coronary artery disease. He underwent successful surgical repair and remained asymptomatic at the three-year follow-up.

Keywords: ST Elevation Myocardial Infarction; Pseudoaneurysm; Ventricular Septal Rupture; Mitral Regurgitation (Source: MeSH-NLM).

RESUMEN

Ruptura del septum ventricular postinfarto extremadamente tardía: reporte de caso de reparación quirúrgica exitosa

Las complicaciones mecánicas del infarto agudo de miocardio con elevación del ST son infrecuentes en contextos con reperusión oportuna, pero persisten con alta mortalidad en regiones con acceso limitado. Presentamos el caso de un varón de 62 años con infarto inferior con elevación del ST, manejado sin reperusión, quien desarrolló disnea progresiva a los cuatro meses. Un año después, la ecocardiografía evidenció comunicación interventricular basal y regurgitación mitral excéntrica severa, ausentes al alta. La referencia a cirugía cardiovascular se retrasó dos años debido a la pandemia por COVID-19. La imagen multimodal confirmó pseudoaneurisma inferobasal del ventrículo izquierdo, comunicación interventricular basal, regurgitación mitral severa y fracción de eyección del 45%. El cateterismo cardíaco derecho mostró un cortocircuito izquierda-derecha significativo y la angiografía coronaria mostró enfermedad coronaria multivaso. El paciente fue sometido a una reparación quirúrgica exitosa y permaneció asintomático a los tres años de seguimiento.

Palabras clave: Infarto del Miocardio con Elevación del Segmento ST; Pseudoaneurisma; Ruptura Septal Ventricular; Insuficiencia de la Válvula Mitral (Fuente: DeCS-BIREME)

Introduction

In the reperfusion era, the overall incidence of mechanical complications (MC) following acute myocardial infarction has decreased with timely revascularization, estimated at 0.9%.^(1,2) However, mortality remains high—reaching up to 42.4%—despite advances in surgical techniques and critical care.^(1,2) In health systems with limited access to early revascularization and structured follow-up, these complications are more prevalent, associated with higher mortality rates, and frequently diagnosed late due to the absence of standardized clinical and echocardiographic surveillance.^(1,2) We report the case of a patient who developed progressive heart failure symptoms for over one year following a non-revascularized inferior myocardial infarction and was subsequently diagnosed with ventricular septal rupture (VSR), a left ventricular pseudoaneurysm (LVPA), and severe functional mitral regurgitation (MR). Due to delays related to the COVID-19 pandemic, corrective surgery was performed two years after the initial diagnosis.

Case report

A 62-year-old male patient, with a notable history of hypertension and dyslipidemia, presented with an inferior ST-elevation myocardial infarction (STEMI). Management was conservative due to delayed presentation, without reperfusion therapy. He was discharged one week later on aspirin, clopidogrel, and bisoprolol. Transthoracic echocardiography (TTE) showed a preserved left

ventricular ejection fraction (LVEF) of 55% and no evidence of mechanical complications.

Four months later, the patient developed progressive dyspnea, NYHA class II, worsening to class III over the next year. No medical evaluation was performed during this period due to limited access to healthcare services in a remote rural area.

Because of the persistence of symptoms, the patient eventually sought a cardiology evaluation at a regional hospital. On examination, his blood pressure was 112/62 mmHg, heart rate 75 beats per minute, respiratory rate 22 breaths per minute, and oxygen saturation 99% on room air. Cardiac auscultation revealed a grade 3/6 holosystolic murmur along the left lower sternal border, and pulmonary examination revealed bilateral basal crackles. The electrocardiogram showed sinus rhythm with Q waves and negative T waves in leads II, III, and a VF.

TTE revealed a large posterobasal muscular VSR measuring 18 mm, a Qp/Qs ratio of 2.5, severe eccentric functional MR due to posterior leaflet tethering, and a mildly reduced LVEF of 45%.

Medical therapy was initiated with furosemide, enalapril, bisoprolol, spironolactone, and aspirin. A referral for definitive management was initiated to a national cardiovascular surgery center. Nonetheless, a two-year delay in completing the referral was primarily attributable to healthcare system backlogs stemming from the COVID-19 pandemic. Throughout this period, the patient managed his condition at home, presenting with stable NYHA class II dyspnea.

At the referral center, repeat TTE demonstrated a dilated left ventricle with an LVEF of 45%. Key findings included a significant posterobasal muscular VSR (measuring 15 × 11 × 18 mm) causing a systolic-diastolic left-to-right shunt, an inferobasal LVPA (**Figure 1A-C**), and severe eccentric functional MR attributed to posterior

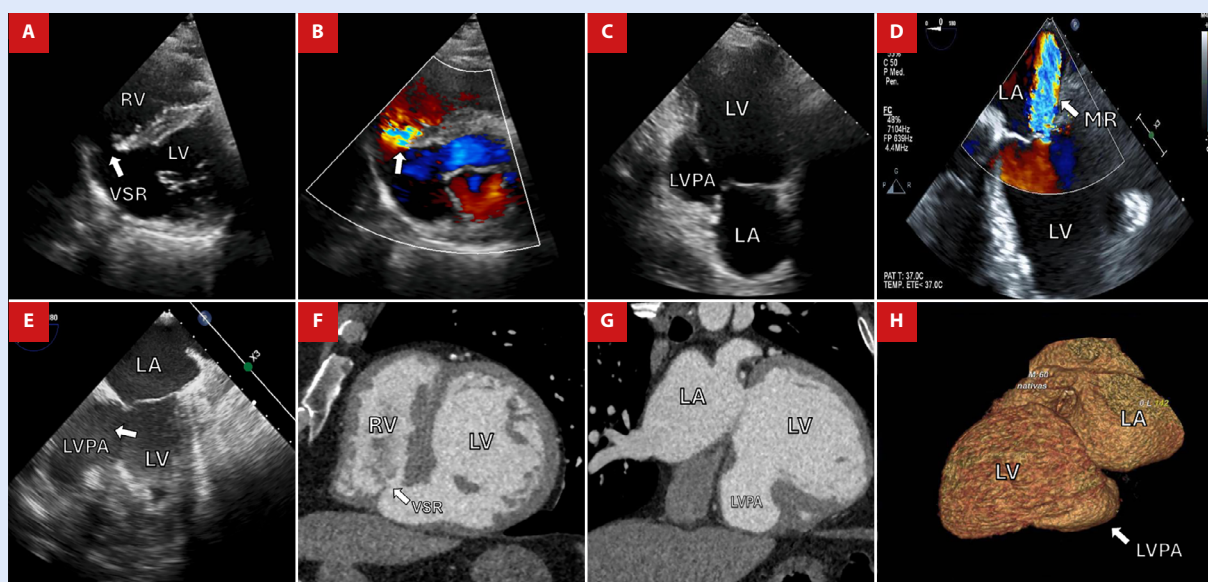


Figure 1. (A-C) TTE showing a basal VSR with left-to-right shunting through the defect (arrow) and an inferior basal LVPA. (D, E) the TEE demonstrates posterior leaflet tethering (arrow), resulting in severe functional mitral regurgitation and an inferior LVPA. (F, G) CCT showing a basal inferoseptal VSR. And confirming the LVPA with a narrow neck. (H) Postprocessing 3D CCT reconstruction showing the inferior basal LVPA (arrow) in relation to the LV and LA.

TTE: transthoracic echocardiography. VSR: ventricular septal rupture. CCT: cardiac computed tomography. TEE: transesophageal echocardiography. LVPA: left ventricular pseudoaneurysm. 3D-CCT: three-dimensional cardiac computed tomography. LV: left ventricle. LA: left atrium. RV: right ventricle. MR: mitral regurgitation.

leaflet tethering. The right ventricle was dilated and showed segmental dyskinesia involving the mid and posterior free wall, with reduced global function, evidenced by a fractional area change (FAC) of 30%.

Transesophageal echocardiography (TEE) confirmed severe MR with preserved subvalvular apparatus and revealed an image suggestive of a posterobasal LVPA with a 23 mm neck (**Fig. 1D, 1E**). Cardiac computed tomography (CCT) confirmed an inferobasal LVPA with a 28 × 48 mm neck, a depth of 32 mm, and a maximal sac diameter of 66 × 38 mm, as the associated VSR (**Figure 1F-H**). Coronary angiography showed severe three-vessel disease, including total occlusion of the mid-segment of the right coronary artery, severe proximal stenosis of the left anterior descending (LAD) artery, and severe distal stenosis of the circumflex artery (**Fig. 2A-2D**). Right heart catheterization revealed post-capillary pulmonary hypertension and a significant left-to-right shunt, with a Qp/Qs ratio of 8.17.

Based on the multimodal imaging findings and the severity of the structural complications, the multidisciplinary heart team recommended surgical repair. A median sternotomy was performed for on-pump surgical repair with aortic, superior vena cava, and left femoral venous cannulation. Myocardial protection was achieved using cardioplegia with histidine-tryptophan-ketoglutarate solution (Custodiol). A 27-mm mechanical mitral valve was implanted, followed by inferior left ventriculotomy to access the VSR and LVPA. The VSR was closed using a bovine pericardial patch via the exclusion technique. The pseudoaneurysm was resected and repaired, and ventriculorrhaphy was completed using the sandwich method. Additionally, coronary artery bypass grafting was performed using the left internal mammary artery to the LAD and a saphenous vein graft to the posterior descending artery. Total aortic cross-clamp time was 3 hours and 30 minutes, and total cardiopulmonary bypass time was 4 hours and 20 minutes.

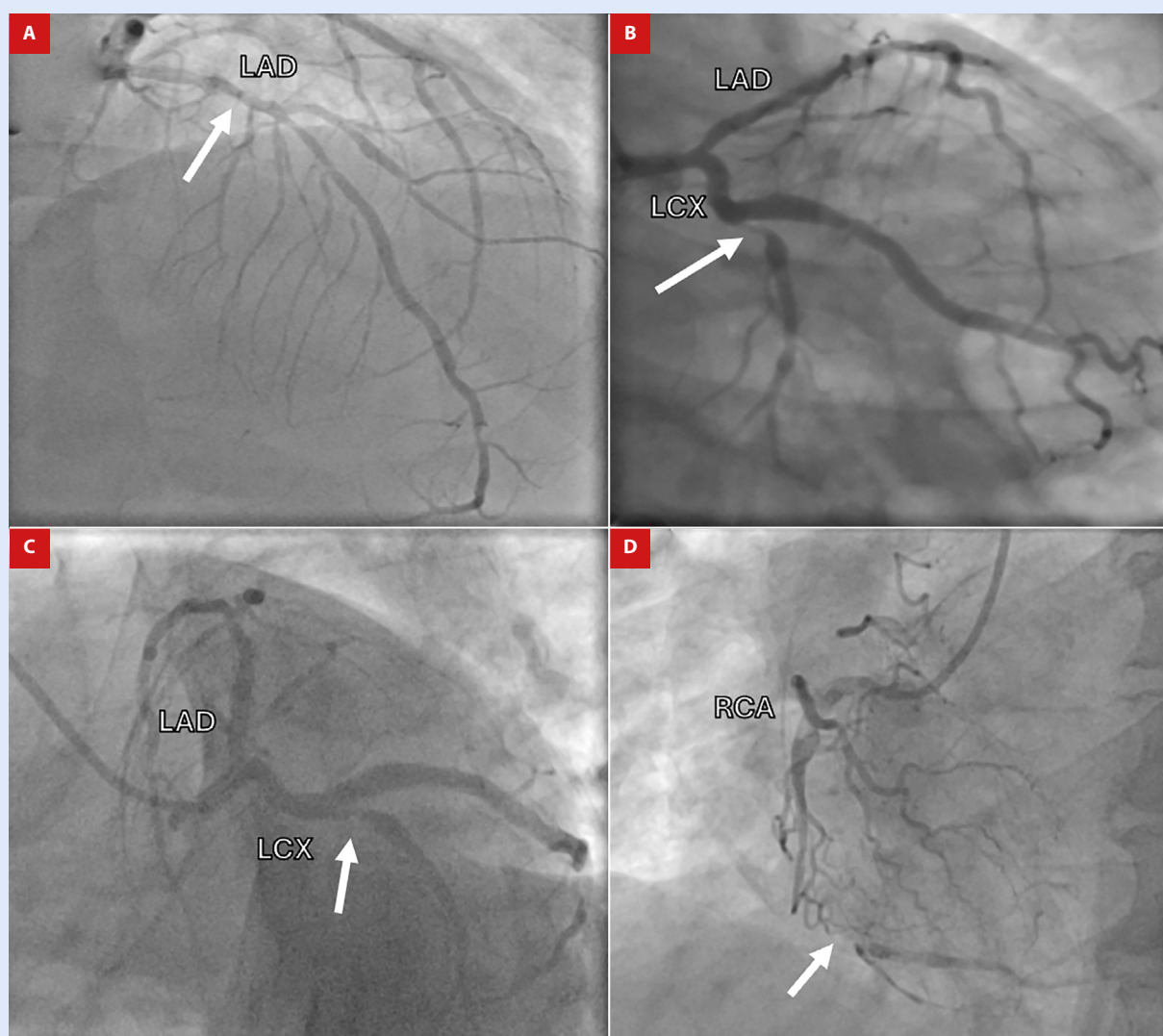


Figure 2. Coronary angiography. (A) Severe stenosis in the proximal left anterior descending artery (arrow). (B, C) Severe stenosis in the distal circumflex artery (arrows). (D) Chronic total occlusion of the mid right coronary artery with Rentrop grade 3, CC1 collateral circulation (arrow).

LAD: left anterior descending artery. LCX: left circumflex artery. RCA: right coronary artery. CC: collateral connection.

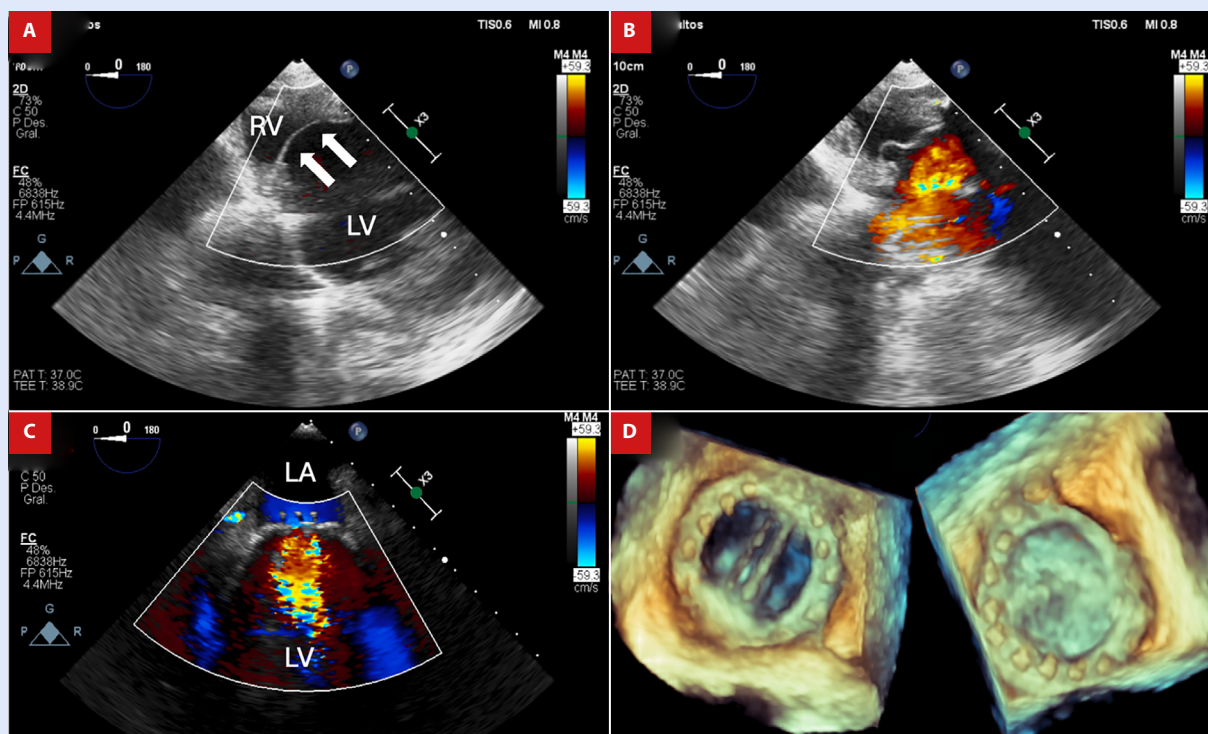


Figure 3. Postoperative transesophageal echocardiography. (A) Transgastric view highlighting the pericardial patch (arrows) used for the closure of the ventricular septal defect. (B) Color Doppler confirmation of the absence of residual interventricular shunting. (C) Mid-esophageal view illustrating normofunctional prosthetic flow and the absence of periprosthetic leaks. (D) 3D reconstruction evidencing the normofunctional bileaflet mechanical mitral valve.

LV: left ventricle. RV: right ventricle. LA: left atrium.

Postoperative TEE demonstrated preserved function of the prosthetic mitral valve without paravalvular leak, no residual interventricular shunt, an LVEF of 55%, and a right ventricular FAC of 35% (Figure 3). The postoperative course was uneventful, and the patient was discharged on day 10, asymptomatic, on warfarin, bisoprolol, furosemide, enalapril, and aspirin. At the three-year follow-up, he remained asymptomatic, with preserved prosthetic valve function, no residual shunt, and an LVEF of 55% on TTE.

Discussion

MC such as free wall rupture, VSR, and papillary muscle rupture are now rare in high-income countries due to timely revascularization.⁽²⁾ Nevertheless, retrospective studies still report high in-hospital mortality.⁽²⁻⁴⁾ In settings with limited access to reperfusion, these complications remain more frequent, as was notably observed during the COVID-19 pandemic.^(4,5)

The patient had an inferior STEMI managed conservatively due to delayed presentation. No MCs were evident at discharge. While such complications typically arise within the first week post-infarction, delayed presentations are possible in the absence of reperfusion.⁽²⁾

Despite symptom onset by month four, geographic and systemic barriers delayed the diagnosis of VSR, LVPA, and severe MR for nearly a year. The lack of reperfusion likely promoted transmural necrosis and adverse remodeling, favoring these late complications. Gradual progression with partial hemodynamic compensation may explain the subacute presentation.^(2,6)

VSR occurs in 0.3% of STEMI cases in the reperfusion era, typically in a bimodal pattern: within 24 hours or between days 3–5.^(2,7) Furthermore, while VSR is more frequently associated with anterior infarctions, inferior VSRs are anatomically more complex and often involve right ventricular dysfunction.^(2,7) Presentation beyond several weeks after myocardial infarction has rarely been reported⁽⁸⁾, and a three-year delay, as observed in this case, is exceptionally uncommon in contemporary literature.

LVPA is a rare but severe complication arising from a contained myocardial rupture limited by adherent pericardium or scar tissue.⁽⁶⁾ It often develops weeks after infarction, affecting the inferior wall in 20% to 25%.^(2,6) Unlike true aneurysms, LVPAs lack a complete myocardial wall and carry a high risk of rupture (30% to 45%), especially in acute phases.^(2,6) In this case, chronic evolution without overt hemodynamic collapse likely promoted LVPA stabilization, thereby allowing delayed surgical repair.

In contrast to structural rupture, ischemic MR results from adverse post-infarction remodeling rather than tissue discontinuity. In the absence of papillary muscle rupture, ischemic

MR stems from geometric distortion of the LV, leading to papillary muscle displacement, leaflet tethering, and annular dilation.^(2,9,10) Inferior infarctions are more frequently associated with this form of MR, which evolves gradually and leads to volume overload, high left atrial pressures, and congestion.^(2,9)

Multimodal imaging is essential for diagnosing and planning the treatment of post-STEMI MCs.^(6,7,9,10) TTE is the initial modality of choice due to its availability and its ability to detect VSR, pericardial effusion, and MR.^(6,7,9,10) The sensitivity of this approach is, however, limited when dealing with posterior defects.^(7,10) TEE provides better resolution of posterior septal defects, leaflet tethering, and pseudoaneurysm morphology.^(6,7,9,10) In cases of LVPA, TEE and CCT allow detailed anatomical assessment—particularly for evaluating neck width, chamber dimensions, and distinguishing pseudoaneurysms from true aneurysms.⁽⁶⁾

Structured clinical and echocardiographic surveillance is essential after STEMI to detect evolving complications.^(6,7,9,10) In this case, the absence of structured follow-up likely delayed recognition, allowing complications to progress subclinically. Standardized surveillance protocols could help mitigate these delays, especially in underserved settings.

The simultaneous occurrence of VSR, LVPA, and functional MR is infrequent and associated with poor outcomes, as patients often deteriorate due to volume overload, pulmonary hypertension, and biventricular dysfunction.^(6,7,9,10) Remarkably, this patient remained stable for nearly three years, suggesting an unusual degree of hemodynamic adaptation. This heterogeneity shows that, in selected compensated cases, delayed surgery may still be effective.

Early VSR repair (<7 days) carries high operative mortality (43%) due to myocardial fragility, while delayed repair after scar formation improves surgical outcomes.⁽⁷⁾ In chronic LVPA, progressive enlargement, symptoms, or rupture risk often justify surgical intervention over conservative management.⁽⁶⁾ The benefit of concomitant CABG in MC remains debated. However, current evidence supports its use in patients with multivessel coronary artery disease and LV dysfunction.^(2,11) The decision to perform CABG was warranted due to the widespread coronary disease and aimed to diminish the risk of subsequent ischemic events.

This case also reflects the collateral effects of the COVID-19 pandemic, including reduced STEMI admissions, delayed presentation, and limited access to interventional and surgical care⁽⁵⁾. The patient's prolonged delay in referral and treatment mirrors these systemic disruptions. Because serial imaging and structured follow-up were unavailable, the timing of complication onset remains uncertain. This case reinforces the need for routine surveillance, particularly in non-revascularized or underserved patients, to detect subclinical progression and enable timely intervention. Even in delayed settings, favorable outcomes are possible with delayed surgical repair in selected cases.

Author's contributions

DDF, AGY, BBG, WJG, RMS: conceptualization, investigation, writing-original draft, writing-review and editing.

Ethical considerations

Informed consent was obtained from the patient, and the study was approved by the institutional ethics committee.

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