A septal rupture of atypical location after myocardial infarction: Clinical case

Ruptura septal de localización atípica posterior a infarto del miocardio. Caso clínico

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A 46-year-old male patient with a personal history of hypertension, smoking and drinking, and a family-hereditary history of hypertension, started with symptoms of asthenia, adynamia, progressive dyspnea, edema of the lower limbs, and increased abdominal volume, which prompted him to visit his doctor, who decided to hospitalize him. The patient received medical treatment with captopril, furosemide, and spironolactone, with no symptoms improvement and for this reason he was referred to our institution. On arrival at the emergency department, the subject was stable. On physical examination, he was conscious and oriented, with skin adequate coloration and hydration, Grade II jugular engorgement, and symmetric carotid pulses of adequate amplitude. Auscultation revealed crackling rales in the bilateral subscapular region, hypoventilation in the right subscapular region, and ipsilateral effusion syndrome. The apex impulse was palpated at the sixth intercostal space, left clavicular midline, and left parasternal thrill was detected. Heart sounds were rhythmic, with S3 and Grade III/IV holosystolic murmur in the mesocardium, with eccentric irradiation, and without pericardial friction rub. Lower extremities were sound, symmetrical, with edema up to the bilateral tibial proximal third and immediate capillary filling. Pulses were symmetrical and of

adequate amplitude in all four extremities. Electrocardiogram showed normal sinus rhythm with HR of 83 bpm, dextrorotation, left atrial enlargement, and image of recent inferolateral infarction, with q wave, sub-epicardial lesion, and T-wave symmetric inversion in DII, III, aVF, V5, and V6 (Fig. 1). Chest X-ray showed Grade II cardiomegaly with venocapillary pulmonary hypertension (Fig. 2). Echocardiogram showed an apical septal rupture with a diameter of 17 mm (Fig. 3), Qp/Qs of 3.6 and gradient of 26 mmHg across the defect and pulmonary artery systolic pressure of 74 mmHg (Fig. 4), left atrial dilatation, and normal left ventricular systolic function, with ejection fraction of 58%. The patient was admitted to the hemodynamics department for placement of an intra-aortic balloon pump (IABP). Diagnostic coronary angiography showed no significant angiographic lesions on the trunk, circumflex and left anterior descending artery, with TIMI 3 distal flow. The right dominant coronary artery showed an ulcerated tubular lesion in the middle segment, with poorly defined borders, eccentric, with stenosis of 70% and chronic total occlusion of the posterior descending artery in the middle segment (TIMI 0 flow), with left anterior descending artery collateral circulation, Rentrop 3 (Fig. 5). Ventriculography revealed an apical ventricular septal defect (VSD), and

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Figure 1. Electrocardiogram showing normal sinus rhythm with HR of 83 bpm, dextrocardia, left atrial enlargement, and an image of recent inferolateral infarction, with q wave, sub-epicardial lesion, and T-wave symmetric inversion in DII, III, aVF, V5, and V6.

Figure 2. Chest X-ray revealing Grade II cardiomegaly, significant venocapillary pulmonary hypertension, pulmonary artery flotation catheter, and intra-aortic balloon pump.

left ventricular end-diastolic pressure was 35 mmHg. Dose-response-based treatment was started with loop diuretic, sodium nitroprusside, and levosimendan, with evident improvement. A Swan-Ganz catheter was placed, which demonstrated pulmonary capillary pressure of 34 mmHg.

After hemodynamic stabilization, intervention was carried out to close the VSD with a bovine pericardium patch and placement of a venous graft on the posterior descending artery.

During his evolution, the patient had a septic condition of pulmonary origin, due to the development of mechanical ventilation-related pneumonia. He developed hemodynamic deterioration despite the administration of high-dose vasopressors, as well as signs of persistent hypoperfusion, with cardiac output of 1.5 L/min, hypotension of 80/40 mmHg, and lactic acidosis. Transthoracic echocardiogram revealed the presence of cardiac tamponade with 2.1 cm pericardial leaves separation, right chamber collapse, and increased transmitral and transtricuspid flow variability. An emergency pericardial window was created with extraction of 140 mL of blood and fluid. The patient died that same day, with diagnoses of septic shock, cardiogenic shock, bacterial pneumonia, and acute myocardial infarction (AMI).

Despite recent advances in the diagnosis and treatment of ischemic heart disease, it remains the main cause of morbidity and mortality in the world. Even patients who survive an AMI are at risk of suffering mechanical complications that significantly decrease survival in the short and long term; more than 50% of deaths occur within the first 28 days after an AMI,

Figure 3. A: Two-dimensional transthoracic echocardiogram and color Doppler in apical four-chamber plane, showing septal rupture at the apical portion of the interventricular septum. **B:** Two-dimensional transesophageal echocardiogram showing the septal rupture at the apical portion of the interventricular septum. **C:** Two-dimensional transgastric echocardiogram and color Doppler, at the level of both ventricles, showing the septal rupture. **D:** Three-dimensional reconstruction of the septal rupture, which is oval in shape (white arrow). RV: right ventricle; LV: left ventricle.

Figure 4. Two-dimensional transthoracic echocardiogram with color Doppler showing apical septal rupture; the gradient across the ventricular septal defect (VSD) (27 mmHg) was calculated with continuous Doppler. Pulmonary artery systolic pressure was calculated with the following formula: systemic systolic pressure - trans-VSD gradient = 100 - 26 = 74 mmHg.

without the patient arriving at the hospital. The rest die within the first 24 h of hospitalization^{1,2}

With the advent of percutaneous coronary intervention (PCI), the incidence of AMI mechanical complications has significantly decreased in recent years³, and currently, it is < 1%. However, they continue to represent a clinical emergency and are a major cause of short-term mortality⁴.

AMI complications can be mechanical, electrical, inflammatory, ischemic, and embolic (Table 1)⁴ and their occurrence increases mortality, particularly in cases without reperfusion treatment or with late thrombolysis⁵. Cardiogenic shock is the most common mechanical complication and the main cause of death after AMI³.

The incidence of septal rupture before reperfusion⁶ was 1 - 3%, and with thrombolysis and PCI, it has dropped to 0.17 - 0.31%⁷. At present, this mechanical complication accounts for only 5% of all AMI-related deaths^{3,4,8}, but when it occurs, it has a mortality of up

Figure 5. Coronary angiography left anterior oblique projection at 45°. **A:** Dominant right coronary artery with ulcerated tubular lesion in the middle segment, with poorly defined borders, eccentric, with maximum stenosis site of 70% (white arrow) and chronic total occlusion of the posterior descending artery in the middle segment, TIMI 0 flow (blue arrow), with anterior descending artery collateral circulation, Rentrop 3. **B:** In right anterior oblique projection, the left coronary artery trunk is observed without significant angiographic lesions, the circumflex artery and the left anterior descending artery show no significant angiographic obstruction, with TIMI 3 distal flow. RCA: right coronary artery; LAD: left anterior descending.

Table 1. Acute myocardial infarction complications

Complications	Subtypes
Mechanical	Cardiogenic shock, free wall rupture, ventricular septum rupture, ventricular aneurysm, and acute mitral regurgitation
Electrical	Arrhythmias, AV blocks, and fascicular blocks
Inflammatory	Dressler syndrome
Ischemic	Post-infarction angina
Embolic	Mural thrombosis and systemic embolism

Modified from Moreno et al., 2017⁴.

to 46% within the 1st week and reaches 90% in the first 2 months in those patients who did not undergo surgical treatment^{9,10}. The patient underwent revascularization and VSD closure, but died within the first 2 months after the onset of the condition. The literature records that even with surgical repair, mortality in these patients is 40%^{7,11}.

Ventricular septal rupture clinical manifestations occur within the first 3-5 days after AMI, although some reports refer that they can occur even up to 30 days post-infarction¹². This case, which did not receive reperfusion treatment, developed septal rupture 4 days

after the ischemic episode. Among the risk factors that predispose to septal rupture, hypertension, old age, female gender, absence of angina, prolonged ischemia time, absence of collaterals, anterior AMI, and extensive AMI stand out^{1,13}. This patient was hypertensive, but the most significant risk factor for septal rupture was a prolonged ischemia time without reperfusion.

It should be noted that, in anterior infarctions, septal rupture is more common in the apical region, whereas in lateral or inferior infarctions, septal rupture appears in the posterobasal region^{6,14-16}. In contrast to these findings, this patient suffered a posteroinferior transmural AMI with septal rupture at the apical portion, a rare presentation of this complication. It should be remembered that most part of the ventricular septum (2/3) is irrigated by the left anterior descending artery, but one-third is irrigated by the right coronary artery. This might explain the unusual presentation of apical septal rupture in the presence of posterior descending artery obstruction.

In more than 70% of patients, the most common clinical presentation corresponds to hemodynamic collapse and holosystolic murmur¹⁷⁻¹⁹. Transthoracic echocardiogram is the diagnostic study of choice for AMI mechanical complications¹⁸, although sometimes transesophageal echocardiogram, especially the three-dimensional technique, is superior to accurately outline the full extent of the rupture¹⁶.

Although the ACC/AHA guidelines indicate that post-infarction ventricular septal rupture requires urgent intervention²⁰, some studies show that mortality from surgical treatment is higher the earlier the intervention is⁸. Patients who enter the operating room within the first 7 days have a mortality of 54.1%, in comparison with 18.4% for those undergoing surgery after 7 days. Mortality is higher than 60% if the procedure is practiced within the first 24 h⁶. Due to this, the use of IABP, inotropic drugs, vasodilators, and invasive methods is indispensable in patient pre-surgical stabilization¹. Surgical mortality in cases of cardiogenic shock ranges from 20-50%¹⁵.

Incidence of hemodynamically significant pericardial effusion has been described in 1-2% of all heart operations; the presence of kidney injury and prolonged time on cardiopulmonary bypass pump are independent risk factors for the development of this complication²¹; both alterations were present in this patient. Although the authors consider that cardiac tamponade was not the cause of death in this patient, it did contribute to hemodynamic deterioration and, consequently, to sustained tissue hypoperfusion.

Septal rupture percutaneous closure is an alternative that has shown favorable results in the short and long term^{1,3,18,20}; in this case, the borders of the defect were not suitable for fixing a percutaneous closure device.

Conclusion

This case is of special interest because it showed a transmural infarction of the posteroinferior left ventricular wall, which had a septal rupture in the apical portion of the interventricular septum, an atypical location of this complication, diagnosed by clinical data, physical examination, and echocardiography. Despite the performed interventions, the patient died from cardiogenic shock.

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Conflicts of interest

The authors have no conflicts of interest to declare.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this research.

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent. The authors have obtained informed consent from the patients and/or subjects referred to in the article. This document is in the possession of the corresponding author.

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