Survivor of a Double Mechanical Complication After Myocardial Infarction: Papillary Muscle Rupture and Contained Free-wall Rupture

To the Editor,

We present the case of a 75-year-old man, referred to our hospital in a state of cardiogenic shock, with a diagnosis of free mitral regurgitation following myocardial infarction. The patient attended the first hospital with a 3-week history of chest pain. In the emergency department, he was diagnosed with evolving inferoposterior myocardial infarction, with Q waves and new-onset inverted T waves on electrocardiogram and mildly elevated markers of myocardial damage. He was admitted to the intensive care unit (ICU) for treatment and underwent coronary angiography, which showed a 60% lesion at the right coronary artery (RCA) ostium and chronic distal disease in the presence of right dominance; therefore, it was decided to treat medically. During his stay in the ICU, he remained asymptomatic, so was discharged to the ward. After 2 days on the ward, the patient deteriorated, with hemodynamic compromise and acute pulmonary edema. Transthoracic echocardiography was performed, which showed free mitral regurgitation secondary to posteroomedial papillary muscle (PM) rupture, with no other significant findings. In the absence of a cardiac surgery team in the hospital, it was decided to perform emergency simple angioplasty of the RCA, and subsequently the patient was sent to a center with cardiac surgery facilities. On arrival, the patient required vasoactive support and intra-aortic balloon counterpulsation; therefore, emergency intervention was performed. The estimated surgical risk was 30.15% (logistic EuroSCORE). The inferior wall was examined via midline sternotomy and with conventional extracorporeal circulation, and a large infarcted area with friable tissue was observed (Figure 1A). Left atrial atriotomy was performed, which allowed the posteroomedial PM rupture to be visualized (Figure 1B). The anterior leaflet of the mitral valve was resected, along with part of the muscle anchored to the primary chordae (Figure 1C). The valve was replaced with a 27-mm Carpentier Magna Ease (Edwards) biological mitral prosthesis and the inferior wall was revascularized with a saphenous vein graft to the posterolateral branch of the RCA. The surgery was uneventful, but the patient remained in the ICU for 2 weeks due to a tracheobronchitis that required prolonged intubation and antibiotics. After this resolved, the patient progressed well and was discharged home at 1 month after surgery. On postoperative transthoracic echocardiography, a 2.8 × 1 cm cavity was noted in the midregion of the inferior wall, with a 1.5-cm inlet and a gap in the ventricular wall with flow into the interior (Figures 2A and 2B), compatible with a pseudoaneurysm/contained free-wall rupture (FWR) following infarction. This finding was confirmed with computed tomography, which showed the close relationship to the artery that had caused the infarction.

Figure 1. Intraoperative images. A: Arrested and emptied heart positioned for revascularization of the posterolateral artery. The infarcted area of the inferior wall can be seen (dashed circle). B: After left atriotomy was performed, the complete rupture of the posteroomedial PM could be seen; the forceps are holding the head of the PM (arrow). C: Surgical specimen of the anterior leaflet of the mitral valve with the chordae tendineae anchored to the ruptured head of the PM (arrow). F, feet; H, head; L, left; LA, left atrium; PM, papillary muscle; R, right.
Reintervention was ruled out due to the high surgical risk at that time. At 2-months’ follow-up, the patient remained asymptomatic and was in New York Heart Association functional class I; however, he is seen in clinic regularly for echocardiography, to closely monitor his progress and to determine whether surgical reintervention may be necessary.

The mechanical complications of myocardial infarction, such as FWR, interventricular communication, and PM rupture, have become less frequent since the introduction of primary angioplasty. The incidence of PM rupture after infarction has decreased from 1.5% to 2% in the 1980s to less than 0.5% since the 1990s. In most cases, these complications occur in the first week, and the risk of mortality without surgery is extremely high.1–4 Preservation of subvalvular apparatus and revascularization of the affected territory can help improve outcomes, and the life expectancy for survivors at 1 month postsurgery is similar to that of patients who have undergone mitral valve intervention. The posteromедial PM is most frequently affected because it is supplied by a single arterial source (RCA).5 However, not all mechanical complications require intervention. Sometimes, FWR can have a good outcome with reperfusion and medical treatment, thus offering an alternative without the surgical risk.4 Mitral repair is an option with similar outcomes to mitral replacement, although replacement is preferable for most patients with hemodynamic instability.5

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Leadless Pacemaker Implantation in a Patient With a Severe Thoracic Deformity

Implante de un marcapasos sin cables en un paciente con deformidad torácica grave

To the Editor,

We present the case of a 76-year-old man who was urgently hospitalized for syncope. The electrocardiogram documented atrial fibrillation with high-grade atroventricular block. The patient’s clinical history included hypertensive heart disease with moderate ventricular dysfunction and permanent atrial fibrillation, as well as a chest deformity due to kyphoscoliosis.

During hospitalization, the absence of drugs or analytical abnormalities that could explain the clinical picture was confirmed, and tests showed left ventricular dilatation and dysfunction (ejection fraction = 40%) with normal coronary arteries. Continuous monitoring provided evidence of atrial fibrillation with preserved atroventricular conduction, but with symptomatic paroxysmal episodes of high-grade block that required isoproterenol administration; a definitive single-chamber pacemaker was indicated. During the implantation procedure, marked venous tortuosity (Figure 1A), related to anterior displacement of the heart due to the barrel chest deformity (Figure 1B), made it impossible to advance the electrode to the superior vena cava from either a right or left approach. The alternative, epicardial implantation, was ruled out because of the patient’s frail clinical status and the drawbacks of a procedure requiring general anesthesia. We decided to implant a leadless pacemaker (Micra, Medtronic Ibérica SA; Madrid, Spain) using a right femoral approach. Computed tomography angiographic study of the venous system showed distortion of the abdominal organs, a polycystic liver, and a large diaphragmatic hiatal hernia, whereas the femoral and abdominal venous system was confirmed to be accessible (Figure 1C). The implantation procedure progressed without incident from the access to the right atrium, with some difficulty releasing the device because of the position of the heart (right atrial dilatation, ventricular dilatation, and anterior elongation of the inferior vena cava-right atrial junction). The device was placed in the apical area of the right ventricle with no complications and with good parameters (sensing = 11 mV, impedance = 600 Ω, threshold 0.75 V to 0.24 ms). It was programmed in VVI mode at 50 bpm (Figure 2). The patient was discharged at 24 hours following the procedure without incident. The device parameters were confirmed to be stable over follow-up.

Conventional transvenous pacemaker implantation is currently the treatment of choice for symptomatic bradyarrhythmia. The procedure requires a central venous access, usually a thoracic vein (subclavian, axillary, or cephalic), as this technique provides high success rates and good long-term outcomes. However, these access routes are sometimes unavailable because of

![Figure 1](image_url)

Figure 1: A: Anteroposterior venography of the left subclavian vein showing a tortuous course up to the superior vena cava. B: Chest CT transverse slice depicts barrel chest deformity causing a large separation of the heart from the descending aorta; the transverse:anteroposterior chest diameter ratio was 1.3 (normal:2.5); a diaphragmatic hiatal hernia ($) is visible. C: Thoracoabdominal CT angiography sagittal image showing vascular distortion; in the enlarged image of the area of interest (asterisk), siphon configuration of the left subclavian vein (arrowheads). Ao, aorta; CT, computed tomography; IV, inferior vena cava; SVC, superior vena cava.