Hybrid closure of postinfarction ventricular septal rupture enlargement after transcatheter closure with Amplatzer occluder

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Abstract

Ventricular septal rupture (VSR) is nowadays a rare complication of myocardial infarction (MI), but with a mortality rate still very high. Urgent surgical correction is recommended, although in specific cases percutaneous closure of a post-infarct VSR is a therapeutic option or a bridge to surgical correction. We report a case of an 80-year-old woman, with a subacute anterior MI with an antero-septal VSR. Rapid clinical deterioration in a high-surgical-risk patient led us to attempt percutaneous VSR closure at day 8 post MI. A 16-mm Amplatzer post-infarction (PI) muscular VSD closed the defect with intra-cardiac echocardiography guidance, that allowed conscious sedation. Clinical and haemodynamic improvement was immediate. Unfortunately, a small orifice distal to the device persisted, which enlarged to 8 mm over the following days, with a Qp/Qs shunt of 1.9. At day 17 post MI, the VSR was surgically closed by suturing the Amplatzer device to the septum. A residual shunt was evident, but with no progression, being the patient discharged in NYHA class I. Percutaneous closure of a post-MI VSR as a bridge to surgery is a therapeutic option in patients with high surgical risk, allowing haemodynamic stabilization and thus gaining time for a further surgical intervention if needed, improving these patients grim prognosis. Intra-cardiac echocardiography for monitoring the percutaneous procedure instead of a transoesophageal approach, as well as the surgical technique, make this case unique.

Keywords

Amplatzer, myocardial infarction, ventricular septal rupture

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Case study

Ventricular septal rupture (VSR) is a rare complication of myocardial infarction (MI). Currently, the incidence of cardiac rupture in MI in the myocardial reperfusion era is of 0.45% and VSR of 0.26%.¹ However, mortality still remains above 90%, being of 100% in patients who progress to cardiogenic shock.¹ Urgent surgical correction is therefore recommended.² In specific cases, percutaneous closure of a post-infarct VSR is a therapeutic option^{3,4} or a bridge to surgical correction.⁵

We report a case of an 80-year-old woman, with a subacute anterior MI not submitted to thrombolysis, referred to our centre for angioplasty. On admission she was haemodynamically stable, with no heart murmur. Cardiac catheterization showed occlusion of the middle segment of the left

anterior descending coronary artery. Angioplasty was performed, with good final angiographic result. Maximum troponin I was of 449 ng/ml.

At day 4 post MI, a holosystolic murmur grade 3/6 was audible and heart failure symptoms developed. Transthoracic echocardiography revealed an antero-apical VSR (Figure 1A) with a maximum gradient LV/RV of 72 mmHg, moderated pulmonary hypertension, with Qp/Qs shunt of 1.8 and left ventricle ejection fraction of 38%.

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Figure 1. VSR complicating an anterior myocardial infarction. (A) Colour flow Doppler on transthoracic echocardiography revealing left-to-right shunt through anterior-apical. (B) Intracardiac echocardiography showing left ventricle and anterior-apical ventricular septal rupture (VSR, arrow). (C) Left ventriculography showing left-to-right shunt through VSR. (D) Left ventriculography post-Amplatzer implantation occluding VSR. (E) Amplatzer device in intra-operative view on the ventricular septum. (F) Amplatzer device in intra-operative view partially covering the VSR.

Clinical condition evolved into cardiogenic shock, needing inotropic support and intra-aortic balloon pump.

The rapid clinical deterioration in a high-risk surgical patient (EuroSCORE of 86%), led us to decided to attempt percutaneous VSR closure at day 8 post MI.

Under conscious sedation, right femoral artery and right jugular vein were cannulated. Non-fractionated heparin and cefazolin were administrated. A 10-mm VSR was visualized by intra-cardiac echocardiography (Acunav 8 Fr probe, right femoral vein) (Figure 1B) and left ventricle angiography (Figure 1C). The VSR was crossed using an arterial approach, and wire was crossed over the VSR forming an arterial-venous loop. 16-mm Amplatzer PI muscular VSD was delivered using an 8-Fr sheath, under fluoroscopic and intra-cardiac echocardiography guidance. Left ventricle angiography showed only minimal shunting through the device (Figure 1D).

Clinical and haemodynamic improvement was immediate, allowing removal of intra-aortic balloon pump in less than 12 hours and inotropic support reduction.

Unfortunately, a minor shunt persisted due to a small orifice distal to the device, which enlarged to 8 mm over the

following days, with a Qp/Qs shunt of 1.9. At day 17 post MI, VSR was surgically closed. Intra-operatively it was evident that the device was in place (Figure 1E), but without covering the entire defect (Figure 1F). The VSR was repaired by suturing the septum to the Amplatzer device, without the need of a prosthetic patch.

Recovery from surgery was uneventful. However, a residual shunt was evident on transthoracic echocardiography with a minor Qp/Qs shunt of 1.1 and without causing heart failure. Patient was discharged at day 23 post surgery.

The first reported attempts of percutaneous closure in post-infarct VSR are from the 1980s. Since then, there has been an improvement in device technology and many isolated cases of procedural success have been reported.^{6,7} The transcatheter closure approach in post-infarct VSR are typically left to high-surgical-risk patients, in cardiogenic shock, VSR surgery without success, or VSR with a complex location to allow surgical correction,^{4,6} with the patients selected on a case-by-case basis. However, a recent review of all series of post-infarct VSR percutaneously closed published until April 2010 helped to define which patients benefit the most from device closure.⁵ Patients

with simple VSR and VSR under 15 mm in diameter in subacute to chronic phase, when the septum becomes fibrotic and scar develops, appear to benefit the most from this technique, since in this group the percutaneous closure allowed total correction. In patients who are not expected to survive surgery, to achieve haemodynamic stabilization, percutaneous intervention is also accepted as a bridge to surgery.⁵

A persistent residual shunt of variable severity after the device closure as been reported.⁴ In the majority of cases, the flow is through the Amplatzer device, with no apparent worsening in long-term follow up. Surgical correction is also associated with residual shunts being as high as 37%, with up to 11% of patients requiring a second surgical procedure.⁶ However, friable tissue surrounding the infarct area might sometimes confer bad support to the Amplatzer device and increase the size of the VSR, as in our case, and even lead to Amplatzer dislodgement or embolization.^{6,7}

The increase of the VSR post-Amplatzer closure imposed the need for correction. In this particular case, surgery was the option, with no patch used, being the Amplatzer device sutured to the necrotic septum already in a subacute phase, giving a better support and with a good final result.

The majority of transcatheter closures in post-infarct VSR are performed under anaesthesia. Intra-cardiac echocardiography guidance, instead of transoesophageal approach, allowed in this case the use of conscious sedation and better haemodynamic stability during the procedure.

Percutaneous closure of a post-infarct VSR as a bridge to surgery is a therapeutic option in patients with high surgical risk, allowing haemodynamic stabilization and thus a

gain in time before further surgical intervention, improving these patients' grim prognosis.

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