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Delayed closure of postinfarction ventricular septum defect in a patient with single vessel disease

Case Report

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Abstract: A 65-year-old patient with a systolic murmur that developed five days after acute anteroseptal myocardial infarction was referred to our Institution. He had previously been treated with fibrinolytic therapy. The patient was in a stable hemodynamic condition when admitted, with sustained diuresis. Blood gas analysis revealed normal parameters, whereas a chest X-ray showed signs of pulmonary congestion. Transthoracic echocardiography revealed a 1.5x1.2 cm post-infarction ventricular septum defect (VSD) in the apical part of the septum. Because the patient's hemodynamic conditions were stable, we decided to postpone the operative treatment to allow scarring of the infarcted area to make VSD repair feasible, thereby increasing the chance for success. Operative treatment was performed three weeks after admission. We performed closure of the VSD with a bovine pericardial patch. The patient was discharged in good condition and remained well three months after the surgery.

Keywords: *Ventricular septum defect • Acute myocardial infarction • Surgery*

1. Background

One of the most devastating mechanical complications of acute myocardial infarction (AMI) is ventricular wall rupture. Most of the ruptures occur in the free wall of the left ventricle, and these are usually fatal. However, 15%–20% of ruptures occur in the interventricular septum; these are also often fatal unless surgically treated [1-4]. The incidence of postinfarction ventricular septum defects (VSD) has been significantly reduced with adoption of modern acute revascularization strategies (thrombolytic therapy and percutaneous coronary interventions); currently, it is 0.2% (4_7183.pdf 1).

Medical treatment alone yields extremely poor results: 94% of patients die within 30 days (4_7183.pdf 1), although surgical treatment of interventricular septum postinfarction rupture is also associated with high operative mortality when performed during AMI. Operative procedures to correct acute septal rupture have consisted of infarctectomy and reconstruction of the septum and ventricular walls with one or more Dacron fabric patches. David *et al*. [5]. have introduced a new operative procedure that excludes the left ventricular cavity from the infarcted myocardium with a pericardial patch sutured to the endocardium of the left ventricle, thereby avoiding infarctectomy entirely.

2. Case report

A 65-year-old male patient was referred to our Institution 5 days after an onset of anteroseptal AMI. The patient

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had previously been treated with fibrinolytic therapy at a small community hospital prior to his referral. Upon admission, auscultatory systolic and diastolic murmur was detected with evident radiographic signs of pulmonary congestion. Biomarkers for myocardial necrosis were in the normal range (total creatine kinase, 87 U/L; creatine kinase, MB 17 U/L).

Electrocardiography demonstrated atrial fibrillation with a ventricular rate of 120/min. Echocardiography revealed the presence of postinfarction VSD of 1.5 x 1.2 cm at the apical position with left-to-right shunt (Figure 1). The gradient was 80 mmHg; the Qp:Qs ratio was 2.5:1. Ejection fraction of the left ventricle was assessed at 40% with apparent signs of mild mitral insufficiency (MR+2) and a small anteroseptal postinfarction aneurysm. The cardiac index was 2.4 l/m^2.

The patient was in a stable hemodynamic condition with normal systemic pressure. Microcatheterization of the right heart demonstrated elevated mean-pulmonary pressure of 50 mmHg. Coronary angiography revealed single vessel disease with occlusion of the left anterior descending (LAD) artery in the region of the distal third (Figure 2), whereas the proximal part of the LAD artery showed no signs of atherosclerotic lesions (TIMI flow II).

Dual antiplatelet therapy (aspirin and clopidogrel) along with unfractionated heparin was initiated upon admission.

The patient was committed for the operation 3 weeks later because his hemodynamic condition deteriorated, mainly through progressive hypotension. Dual antiplatelet therapy (aspirin and clopidogrel), started upon admission, was discontinued 3 days prior to surgery and the patient was transferred to unfractionated heparin. A decision was made to insert an intra-aortic balloon pump (IABP) preemptively. Transventricular approach to VSD was performed through the postinfarction aneurysm of the left ventricle. A 1.2 x 1.5 cm postinfarction VSD in the lower third of the interventricular septum was detected (Figure 3). The decision was made to perform closure of the VSD with a bovine pericardial patch using Prolene 3/0 sutures reinforced with 3/0 pledgeted sutures. The operation was carried out without an infarct excision, but instead with securing of the bovine pericardium patch to the endocardium. Aneurysmectomy and linear closure was performed after completion of the VSD closure.

Following the operation, the patient started to regain normal hemodynamic conditions. The patient was extubated during the next postoperative day and discharged from the ICU on the fourth postoperative day in good condition, and not requiring inotropoic support. He was discharged from the hospital on the 21st postoperative day. Three months after the operation the patient was in sinus rhythm; control echocardiography revealed no

Figure 1. Four-chamber view on transthoracic echo – color Doppler showing left-to-right shunt in the apical segment of interventricular septum.

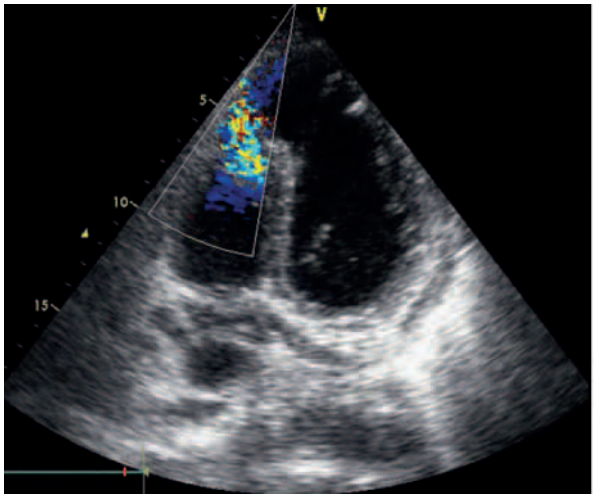


Figure 2. Coronarography showing occlusion in the distal part of left anterior descending artery.

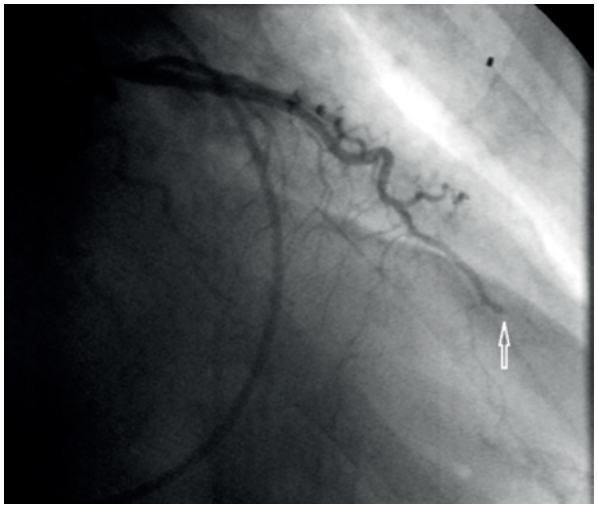
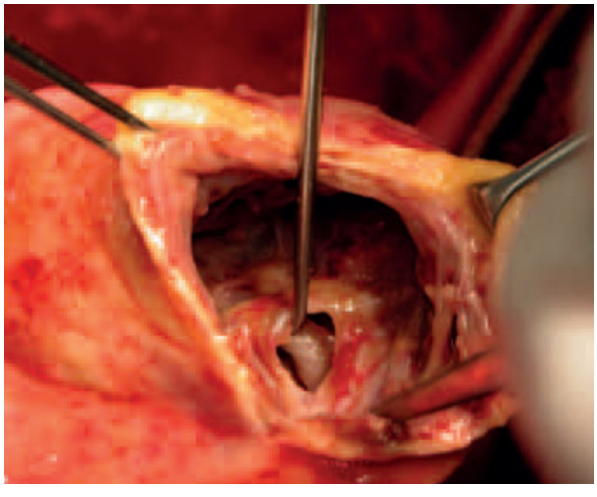


Figure 3. Intraoperative finding of postinfarction ventricular septum defect in the apical region.



signs of the residual interventricular shunt, with good ejection fraction of the left ventricle (around 50%), and only a mild mitral regurgitation (MR +1).

3. Discussion

Postinfarction VSD is a complication seen in approximately 1%–2% of patients with AMI and accounts for about 5% of early deaths following AMI [1,2]. The average time from AMI onset to wall rupture has been reported as between 2 and 4 days, but it may be as short as a few hours or as long as 2 weeks. Angiographic evaluation of patients with postinfarction ventricular rupture indicate that septal rupture is usually associated with complete occlusion rather than severe stenosis of coronary artery, as we observed in our case [6]. These patients have slightly less extensive coronary artery disease (CAD) as well as less developed septal collaterals than do other patients with CAD. Postinfarction VSDs are most commonly located in the anteroapical septum as a result of a full-thickness anterior infarction (approximately 60% of cases).

As in our case, the first signs of postinfarction VSD development are newly diagnosed systolic murmur and hemodynamic deterioration. This systolic murmur should be differentiated from acute postinfarction mitral regurgitation. Advancement and omnipresence of transthoracic and transesophageal echocardiography, especially Doppler color flow mapping, have revolutionized the diagnosis of presence and enabled determination of the exact site of septal rupture. This method, according to Smylie *et al*. [7], has 100% specificity and 100% sensitivity when using Doppler color flow mapping to detect or differentiate diagnosis of postinfarction VSD.

The first surgical repair of postinfarction VSD was performed by Cooley *et al*. [8]. The practice of waiting several weeks after ventricular septal rupture before proceeding with surgery is applicable only to minority of patients in whom the hemodynamic disturbance is less severe and is better tolerated. The delay to operate on those patients promotes the better outcome because the necrotic tissue gets replaced by fibrous tissue, enabling the surgeon to perform a more secure defect closure. Unfortunately, this is not possible in the majority of patients because of the developing cardiogenic shock. Cardiogenic shock was identified as the single most important determinant of operative mortality [9].

The experience of Madsen *et al* suggests that patients in cardiogenic shock represent a true surgical emergency and require immediate operative repair [10]. Death incidences among these patients are typically a result of multisystem failure as a consequence of organ hypoperfusion, therefore, delay in operative repair for patients in cardiogenic shock represents a failed therapeutic strategy. Few patients who are completely stable, having no clinical deterioration and requiring no hemodynamic support, can undergo operative repair when convenient during their hospitalization. The larger group of patients that make up the middle between the two extremes should be operated on early (usually within 12 to 24 hours) after appropriate preoperative evaluation. According to the literature, the group of patients in stable condition constitutes 5% or less of the total population of patients with postinfarction septal rupture [4].

The goals of preoperative management are: 1) to reduce the systemic vascular resistance and the left to right shunt; 2) to maintain cardiac output and arterial pressure to ensure peripheral organ perfusion; and 3) to maintain or improve coronary artery blood flow, which is best accomplished with IABP. It must be stressed that pharmacological therapy and IABP are intended primarily to support patients in preparation for surgery and should not in any way delay urgent operations of critically ill patients.

David *et al*. have published a review of this novel technique, with the majority of patients operated in the acute phase with nearly half being in the state of cardiogenic shock [5]. The concept presented is that of preservation of left ventricular function that provides the groundwork for evolution in the surgical approach to postinfarction VSD. This technique for a ventricular septal rupture repair is a modification of Dor's technique of ventricular endoaneurysmorrhaphy [11].

In recent years, an alternative approach to postinfarction VSD treatment has been suggested. Primary transcatheter closure with Amplatzer devices provides new potentials and options for patients with this deleterious complication (reference 4-7813). There is no randomized study to compare the outcome of the two different approaches, but transcatheter closure may be reserved for patients with suitable anatomy (usually small VSDs) and completed necrosis or for those for whom surgery is not an option because of high risk. It is important to stress the completeness of the myocardial necrosis, as an attempt to pass the device through the VSD may increase the size of the rupture and tear residual fragile tissue (reference Percutaneous.pdf). New hybrid techniques are also currently evolving where small VSDs may be closed on the beating heart with the VSD occluder through a lower ministernotomy incision (reference Percutaneous.pdf 20)

Controversy exists in literature concerning the advantages and disadvantages of concurrent coronary artery grafting in patients undergoing emergent repair of postinfarction ventricular septal rupture. Some who

have argued that revascularization offers no survival benefit, subject their patients to preoperative left heart catheterization, which is a time consuming and potentially dangerous diagnostic procedure [12]. That approach is also based on the findings that multivessel disease is much less prevalent in those with an apical septal rupture as a result of anterior infarction. However, recent revascularization guidelines from both the American College of Cardiology Foundation/American Heart Association and the European Society of Cardiology do recommend coronary artery bypass grafting in patients undergoing emergency surgical repair of AMI mechanical complications such as VSD.

In conclusion, in rare cases of postinfarction VSD with no signs of developing cardiogenic shock, it is possible to postpone operative closure. The postponement enables the infarcted myocardium to scar, thereby increasing the chance of a successful surgical repair.

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