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CASE REPORT

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Successful early percutaneous closure of acute ventricular septal rupture complicating acute myocardial infarction with Amplatzer ventricular septal occluder

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Abstract

Acute ventricular septal rupture — ventricular septal defect (VSD) following acute myocardial infarction remains a critical condition. We present an 80 year-old patient with an acute VSD following an acute inferior myocardial infarction. Percutaneous VSD closure with an Amplatzer ventricular septal occluder (AVSO) was performed successfully, two days after initial admission. In-hospital follow-up was uneventful.

We speculate that percutaneous VSD closure with an AVSO can be an alternative to surgical correction for these patients. Despite the impressive result, this innovative approach needs further investigation and refinements before it can be recommended as the treatment of choice for acute ischemic VSD. (Cardiol J 2007; 14: 411–414)

Key words: myocardial infarction, acute ventricular septal defect, Amplatzer ventricular septal occluder

Introduction

Despite substantial improvements in surgical and interventional cardiology techniques for the treatment of acute myocardial infarction (AMI), acute ventricular septal rupture — ventricular septal defect (VSD) remains a challenging clinical condition associated with high mortality and morbidity. There is acute ischemic insult and loss of the structural integrity of the interventricular septum that is, as is very well documented, essential for partitioning and providing mechanical support for both ventricles in the setting of AMI [1]. The interventricular septum also actively participates in the intracardiac conduction system [2, 3]. The consequences of an acute VSD include right ventricular failure, atrioventricular block, biventricular failure and, ultimately, cardiogenic shock. Two types of acute VSD are presently recognised, namely a simple direct perforation or a complex multifocal communication with a serpiginous course [4].

The Amplatzer ventricular septal occluder (AVSO), a self-expandable, self-centring wire-mesh double disc with a connecting central stent part (AGA Medical Corporation, Golden Valley, Minnesota, USA), has been shown to be effective in the treatment of congenital VSD, muscular VSD and some membranous VSD from 4 to 20 mm [5–7]. The device is now available in sizes of 6–24 mm that are delivered through 6 to 9 French sheaths [5–7]. Despite its success in congenital VSD, AVSO implantation faces a challenge in acute ischemic VSD in view of the location of the rupture, the underlying tissue quality (ischemic and/or necrotic) and, quite commonly, an already failing right ventricle.

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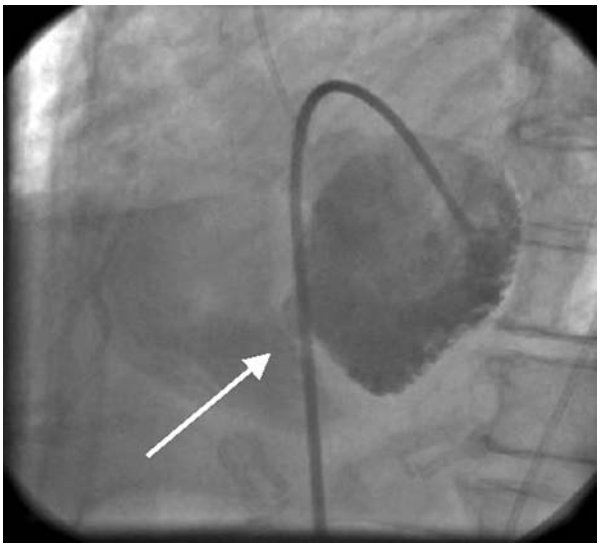

Figure 1. Left ventriculogram with ventricular septal defect (arrow).

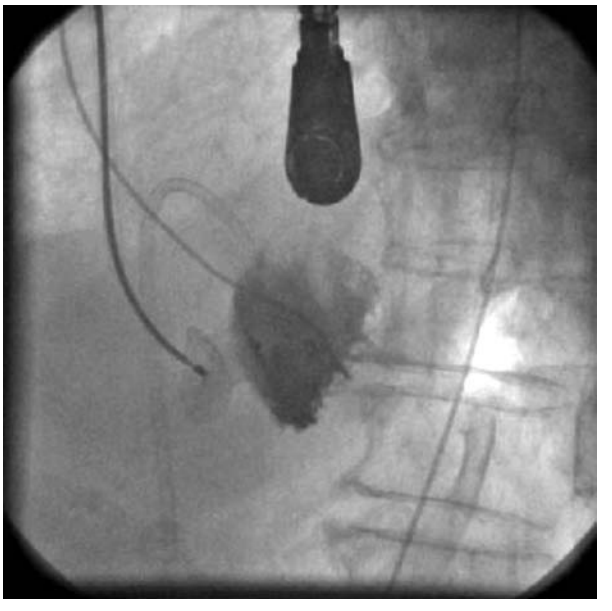

Figure 2. Amplatzer ventricular septal occluder in place. No para-device leak. Transesophageal echocardiography probe above.

Case description

We present an 80 year-old patient with a history of hypertension but without previous cardiac problems. He was admitted for an acute inferior wall myocardial infarction 6 hours after the onset of chest pain. Initial troponine T serum level was noted at 0.35 μg/l (N < 0.03 μg/l). On physical examination a loud holosystolic murmur was heard at the lower left sternum border. Following this finding, initial echocardiography was performed and a muscular VSD with a gradient of 55 mm Hg was described. Ejection fraction (EF) was calculated at 60% with akinetic inferior wall and right ventricle dilatation of up to 49 mm. The day after this the patient was referred to our institution for coronary angiography and VSD treatment. The coronary angiogram demonstrated a normal left main artery, non-significant atherosclerotic changes in the left anterior descending and very distal left circumflex 80% stenosis. The dominant right coronary artery was normal. The left ventriculography presented a VSD in the inferior septum with an estimated EF of 55% (Fig. 1). Cardiac output was calculated at 3.19 l/min with a cardiac index of 1.92 l/min/m^2. Qp/Qs was estimated at 1.67. The VSD diameter was estimated to reach 13–14 mm with a definite left-to-right shunt (Qp/Qs = 1.8) and right ventricular dilatation.

After discussion with the patient and our cardiac surgeons and in consideration of the respective interventional and surgical risks of VSD closure it was decided to proceed with percutaneous closure. Before the procedure the patient received 80 mg of Gentamycine IV and 2 g of Cefazoline IV for endocarditis prophylaxis as well as 8000 U of UFH. Then, general anesthesia was initiated with Fentanyl IV and 8 F right femoral vein and 5 F right femoral artery introducer sheaths were inserted. A 5 F Pigtail 155° catheter was advanced into the left ventricle. The VSD was measured before the procedure with transesophageal echocardiography (TEE) in the LAX view (VSD of 13–14 mm of diameter) for AVSO device size selection. An Extra Stiff 260 cm guidewire was inserted via the right femoral artery across the aortic valve, the VSD and the pulmonic valve, landing in the pulmonary artery. This wire was exteriorised from the right intimal jugular vein using a Microvena snare wire.

A 20 mm AVSO was inserted via the right jugular vein and advanced in the VSD and deployed under TEE guidance. Control left ventriculography showed a successful deployment with only minimal non-significant residual shunting (Fig. 2). This result was also confirmed by TEE (Fig. 3). Subsequently left and right ventricular pressures dropped from 110/0–20 to 99/0–20 mm Hg and from 50 mm Hg/19 mm Hg to 34 mm Hg/13 mm Hg respectively. Cardiac output and index rose to 7.9 l/min and 4.62 l/min/m^2 respectively. Post-procedural and hospital follow-up was uneventful. Six days later the patient was discharged. Endocardial prophylaxis was maintained for one year. Clopidogrel was prescribed for 6 months and acetylsalicylic acid indefinitely.

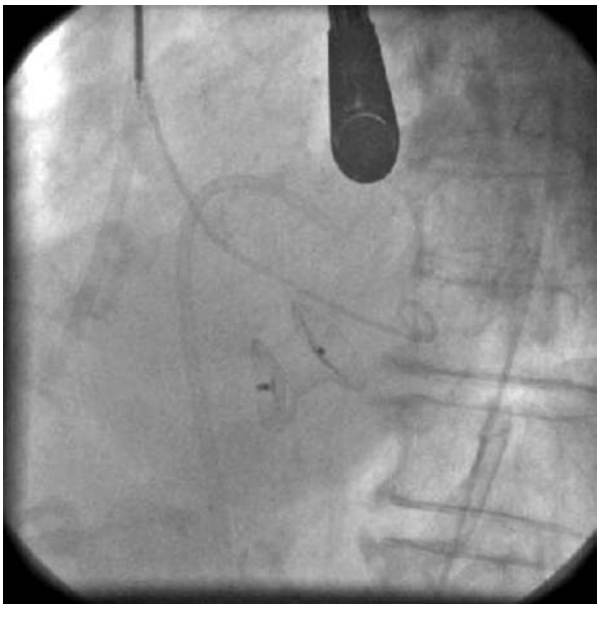

Figure 3. Final result. Amplatzer ventricular septal occluder in place. Transesophageal echocardiography probe above.

Discussion

Patients with post-AMI VSD may benefit from a relatively quick and simple closure which may allow hemodynamic stabilisation and survival. This procedure can be performed as the definitive therapy, as in the patient discussed here, or as a bridge to future surgical correction in optimal conditions. There are few reports on the efficacy of acute VSD treatment. Thielle et al. [8] describe a series of 23 patients with acute VSD who were surgically treated with prior insertion of an intra-aortic balloon pump (IABP). The mean age of the patients was 72 ± 10 years (ranging from 46 to 89) and the interval from the onset of AMI to the detection of VSD was 4 ± 4 days (ranging from 1 to 14). At the time of enrolment 12 patients were in cardiogenic shock (52%). The mean duration of IABP support was 10.8 ± 9.5 days (ranging from 1 to 25) and 20 patients (87%) underwent surgical closure of the VSD [8]. In the work of Thielle et al. [5] early surgical closure because of cardiogenic shock resulted in an ominously high mortality rate of 83%. The mortality rate in a hemodynamically stable group with IABP with delayed surgery was at 29%. Overall, perioperative mortality was 45%. Crenshaw et al. [9], in the largest study of VSD, described the outcomes of patients with VSD complicating AMI in the GUSTO 1 trial. Mortality with this complication remains extremely high despite improvements in medical therapy. Patients who underwent surgical repair had a lower mortality at 30 days and one year then the patients who were treated medically: 47% *vs.* 94% at 30 days ($p < 0.001$) and 53% *vs*. 97% at 1 year ($p < 0.001$). Mortality rates for all patients with VSDs were similar at 30 days and one year (74% and 78%). These data suggest that if the patient survives the initial treatment, the long-term prognosis is relatively good [9]. Similarly, Landzberg and Lock [10] reported that, despite improvements in the surgical management of ventricular septal rupture after AMI, perioperative risk may be excessive for some patients. They suggested that optimal management of patients with post-MI ventricular septal rupture requires surgical-medical collaboration and treatment selection based on individual patients. In a registry in the USA, of 18 patients with transcatheter closure of post-infarction VSD with an Amplatzer muscular VSD occluder, the 28 day-mortality was 28%. Five patients underwent the closure in the acute phase within 6 days of infarct; the remaining patients underwent closure at any time from 14 to 95 days after diagnosis [11].

Conclusions

In the current American College of Cardiology/American Heart Association guidelines urgent surgery is recommended for acute ventricular septal defect regardless of hemodynamic status [12]. Despite an inherent high mortality risk, surgical ventricular septal defect correction remains a life-saving procedure that cannot be deferred [13, 14]. Acute ventricular septal defect closure with the Amplatzer ventricular septal occluder may, however, represent a viable alternative for these patients. Nevertheless, long-term safety studies are required before this technique becomes widely used in routine clinical practice. The obvious heterogeneity of location and anatomy of ischemic ventricular septal defect calls for refinements in the technique and occlusive devices.

Despite the very encouraging result of this innovative approach, further data, namely larger series and long-term follow-up, need to be collected before we may change our current treatment options.

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