Original Article

Transcatheter Closure of Ventricular Septal Defects in Malta - Initial Experience

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Introduction

Ventricular septal defects (VSD) consist of deficiencies of the wall separating the two ventricles. VSDs are the commonest congenital cardiac defects.¹ Small VSDs rarely require intervention, however, larger defects cause ventricular volume overload with or without heart failure or pulmonary hypertension and may, therefore, require closure.² Traditionally, closure has been an open heart surgical procedure.³ In recent years, various devices have been developed to close a wide variety of cardiac defects including atrial septal defects and patent arterial ducts through transcatheter interventional techniques.⁴ Recently, AGA Medical Corporation have

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introduced a range of Amplatzer VSD occluders which include specific devices for perimembranous (PM), muscular and post myocardial infarction (MI) VSDs. $^{5.6}$

Atrial septal defect and patent foramen ovale closure has been carried out at St. Luke's Hospital in Malta for the past three years.⁷ This year, for the first time, we have closed three VSDs in three individuals; two children with large perimembranous defects and an elderly gentleman with a large post-MI VSD. This paper will discuss this technique and initial results.

Patients

Case 1

MB (female) was referred to out-patients due to failure to thrive at 1 month of age. She had clinical evidence of a VSD, confirmed by echocardiography which showed a large PM VSD with left heart volume overload. She was commenced on caloric supplementation and diuretics to control heart failure. She thrived and diuretics were eventually successfully weaned off, despite evidence of a significant defect. At 4 years of age, she was admitted to hospital with high grade pyrexia, abdominal pain and vomiting. She underwent an appendicectomy and during her stay was found to have Staphylococcus aureus septicemia. Despite adequate prophylaxis and a normal echocardiogram during her admission, she re-presented two weeks later with tricuspid valve endocarditis for which she was treated with a standard six week course of antibiotics. However, the tricuspid valve was partially eroded by a large vegetation and remained significantly incompetent. The VSD was closed with an Amplatzer 14mm PM VSD occluder in July 2004 at five years of age. Angiogram confirmed good position of the occlusion device with a small residual left to right shunt. She will continue on antibiotic prophylaxis for life due to severe tricuspid regurgitation.

Case 2

BE (male) was noted to have a murmur at routine Well Baby Clinic follow-up. He had clinical evidence of a VSD, and a perimembranous, subaortic VSD was diagnosed by echocardiography. He was never in heart failure. However, he developed cardiomegaly with a dilated left ventricle and mild aortic regurgitation and the VSD was closed with an Amplatzer 12mm PM VSD occluder in July 2004 at 13 years of age Angiogram confirmed good position of the occlusion device with a small residual left to right shunt. No aortic regurgitation was noted after the procedure on angiogram or on echocardiogram.

Case 3

JA (male aged 76 years) has ischaemic heart disease, congestive heart failure, chronic renal failure and diabetes mellitus on insulin. He sustained an anterior myocardial infarction in August 2002 as a result of which, he developed an apical VSD that was demonstrated to be about 2 cm in diameter. He was not deemed suitable for surgical closure of the defect due to comorbidities and was moderately controlled with medical treatment, albeit with severely limited exercise tolerance. His glycaemic control was stabilized and he was started on an infusion of N-acetyl cysteine from the morning of the procedure and for 24 hours following the procedure as a renal sparing measure after contrast angiography. His VSD was closed uneventfully in July 2004 using a 24mm Amplatzer post-MI VSD device. At catheterization, a calcific aortic valve with mild stenosis was found. A small residual shunt from a separate hole remained. He felt better immediately following the procedure.

All cases were done under general anaesthesia using fluoroscopy and transoesophageal echocardiography guidance. They were discharged on aspirin for six months to prevent overexuberant clotting of the devices with potential thrombus formation on the left ventricular side of the device and risk of transient ischaemic attack, stroke or other embolic events. Patients should continue on antibiotic prophylaxis if there are residual defects such as shunting across a VSD or residual tricuspid regurgitation as in the first case or aortic stenosis as in the third case.

Technique A. Perimembranous defects

Deployment of these devices is under general anaesthesia using fluoroscopy, angiography and transoesophageal echocardiography control. The approach is from the femoral vein and due to catheter diameters, patient weight should be above six kilograms. The Amplatzer perimembranous VSD occluder is a self-expandable, double disc device with discs linked together by a connecting waist (Figure 1). The device is made from Nitinol wire mesh (Nickel Titanium National Ordinance Ltd), a memory alloy that returns a device to its original shape after being collapsed into a sheath for delivery within the heart. In order to increase its efficacy, the device is filled with three layers of Dacron fabric. Due to the close proximity of perimembranous defects to the aortic valve, this device is eccentric in shape with the top rim measuring 0.5 mm and the lower rim 5 mm in order to avoid impinging on the aortic valve. The device comes with a special TorqVue delivery system designed specifically to deploy the eccentric device in the correct orientation.5 The latter is confirmed by a radio-opaque top marker that is located at the lower end of the device.

B. Post-infarction defects

The Amplatzer post-MI VSD occluder is not eccentric and is an extension of the Amplatzer muscular VSD occluder.

The device is circular and consists of left (4 mm diameter) and right (3 mm diameter) discs joined together by a central waist with is 10 mm long and with a diameter that ranges from 16 to 24 mm. Patients with acute VSD following MI are adult patients with a thicker interventricular septum and the device therefore has a longer waist of one centimetre.

These devices are also implanted by a transcatheter technique through a venous or arterial or combined approach. Because of the path and orientation that the catheter takes as it goes through the heart, defects in the anterior outlet portion of the septum are approached from the femoral vein, whereas inferior or apical defects are easier to close from a jugular approach.

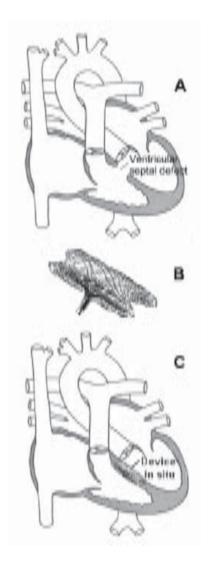


Figure 1:

A - Perimembranous ventricular septal defect. Note the very close proximity to the aortic valve. B - Eccentric perimembranous Amplatzer VSD occluder.

C - Device in situ closing off VSD.

Discussion

Device closure of VSD was first undertaken in 1985 using a Rashkind double umbrella device that was initially designed for closure of patent ductus arteriosus or atrial septal defect.⁸ The Amplatzer device has only been available since 2000.⁹

Although both types of defects can be closed from the right ventricular side, it is easier and safer to cross from the left ventricular side and create an arteriovenous circuit with a guide wire. The device is delivered left ventricular side first, followed by the waist and followed by the right ventricular disc. The position of the device is confirmed by transoesophageal echocardiography and by angiography prior to release.

VSD closure relieves ventricular volume overload or frank heart failure, reduces pulmonary hypertension and risk of bacterial endocarditis, and may reduce the chances of the eventual development of aortic incompetence. The advantages of this technique include avoidance of use of heart-lung bypass machine and blood products, and since it is done on a beating heart, it does not produce any myocardial ischaemia. Moreover, this technique permits the avoidance of intensive care, and entails a short hospital stay (24 hours) with no post-operative pain, surgical scars and other open-heart surgery complications. This technique also gives an alternative for patients who cannot undergo open heart surgery due to associated co-morbid conditions.

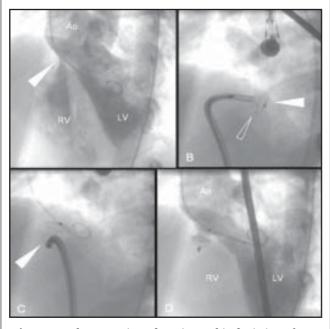


Figure 2: Fluoroscopic and angiographic depiction of steps in VSD closure. A: Left ventriculogram showing left-to-right shunting across a perimembranous VSD (arrow). B: Delivery catheter from femoral vein to right ventricle across VSD to left ventricle. The left ventricular disk has been deployed in the left ventricle (solid arrow). The outlined arrow depicts the radio-opaque marker that delineates the margin of the eccentric device that must point inferiorly. C: Right ventricular disk deployed in the right ventricle (solid arrow). D: Repeat left ventriculogram showing almost no flow across the VSD.

The risks of the procedure itself are low and some of these are similar to those encountered during routine cardiac catheterisation. These include allergy to contrast, air or thromboembolism with potential for cerebral ischaemia, bleeding around introducer sheaths, disturbance to the heart rhythm during the procedure, infection, injury to the femoral artery, vein or nerve, and perforation of the heart. All of these problems are very rare and similar ones may occur during open heart surgery. Risks specific to this procedure are interference with the function of the aortic valve, dislodgement of the device and incomplete closure of the defect.⁵

Small residual shunts are often present immediately after device deployment but usually close completely. If the shunt appears large, an attempt may be made to retrieve the device by a catheter technique, and a different device could then be deployed, if considered appropriate. If the device cannot be retrieved easily from the heart, cardiac surgery may be needed for its removal and to close the VSD simultaneously. Haemolysis after incomplete VSD closure is self-limitin g. Structural failure of the device has not been encountered so far. The risk of heart block is slightly higher than with surgical closure but is still considered a low risk and may occur some weeks after device implantation, therefore electrocardiographic follow-up is essential.

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