Clinical Spotlight

Percutaneous Closure of an Aortic Prosthetic Paravalvar Leak: An Australian First

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Percutaneous intervention is becoming an increasingly recognised modality for the management of prosthetic paravalvar leaks (PVLs) with particular utility in severely symptomatic non-surgical candidates. To date, application of this intervention has predominantly involved closure of mitral valve PVLs. Consequently, current literature on its application to aortic PVLs is limited. This article describes what we believe to be the first percutaneous closure of an aortic prosthetic PVL in Australia.

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Introduction

Significant paravalvar regurgitation around prosthetic valves is well-recognised following heart valve surgery with a reported incidence of 1–5% [1,5]. Most commonly, paravalvar leaks result from rupture of one or more sutures securing the prosthesis to the valve annulus, and typically occur during the first post-operative year [2]. Clinical manifestations are a consequence of reduced net cardiac output and/or transfusion-dependent haemolytic anaemia. Re-operation is currently the gold standard treatment, however, morbidity and mortality increases with each re-operation [3,4].

Transcatheter percutaneous closure of prosthetic paravalvar regurgitation may be offered to patients whose re-operation risks are prohibitively high. Mostly, this technology has been applied to mitral paravalvar leaks via a transvenous, transeptal, antegrade approach. In this report, we describe what we believe to be a novel percutaneous solution for an aortic paravalvar leak in Australia.

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Case

In early 2009, an 85 year-old male underwent porcine aortic valve replacement surgery (Carpentier-Edwards) with two coronary artery bypass grafts. Immediate post-operative transthoracic echocardiography (TTE) showed normal aortic valve function with no apparent regurgitation. Ten days later, a repeat TTE showed the development of mild (1/4) paravalvar aortic regurgitation.

The aortic regurgitation progressed, and by the seventh post-operative month, severe clinical heart failure had manifested. Echocardiography showed the aortic valve replacement to be well seated with severe anterior paravalvar regurgitation originating from the right coronary cusp position (see Fig. 2A) There was no evidence of intracardiac infection. On the basis of the recurrent heart failure admissions, progressive left ventricular enlargement and clinical heart failure, intervention was felt to be indicated. Due to high perceived surgical risk, he was declined open re-operation. On compassionate grounds, percutaneous options were discussed and subsequently accepted by the patient.

Percutaneous closure was performed with a retrograde femoral approach under general anaesthesia with 2D/3D TOE and fluoroscopic guidance. Ascending aortography confirmed severe (Grade 4/4) aortic regurgitation originating from under the right coronary artery. Coronary angiography showed the grafts to be patent. The paravalvar defect was located, defined and crossed using 3D zoomed *en face* imaging of the valve annulus. Initially the defect was crossed with a 0.035-in. angled slippery wire and a multipurpose catheter. This was exchanged for a 0.035-in., 'exchange length', atrial septal defect (ASD),

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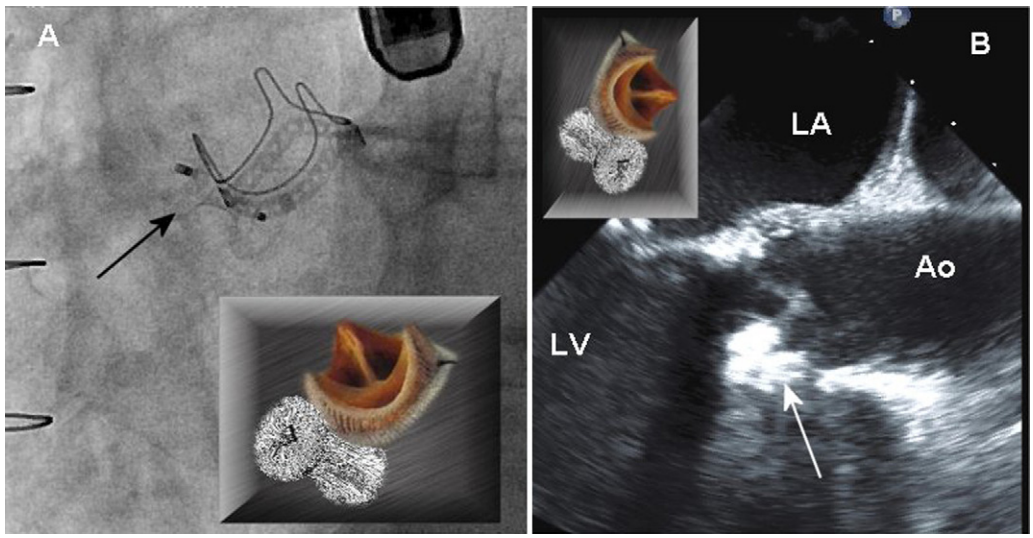

Figure 1. *Panel A: Angiographic imaging of the paravalvar defect closure device (black arrow) adjacent to the aortic prosthesis. The orientation of the devices is demonstrated in the inset panel. Panel B: Omniplane transoesophageal echocardiographic imaging of the paravalvar defect closure device (white arrow) adjacent to the aortic prosthesis (Ao: aorta; LA: left atrium; LV: left ventricle).*

stiff Amplatzer (AGA Medical Corporation, Golden Valley, MN) wire. A size 6 French 180° Torque View sheath was placed across the defect into the left ventricular outflow tract. Inflation of a 6 mm sizing balloon significantly reduced the amount of aortic regurgitation, and an 8 mm Amplatzer muscular ventricular septal defect (VSD) occluder was selected for deployment. This device was chosen because of its "double flange" configuration, with one flange sited either side of the valve sewing ring (see Fig. 1).

The device seated easily in the paravalvar tract (see Fig. 1). Despite this, significant aortic regurgitation persisted on TOE, arising from around the device owing to the elliptical nature of the defect. A brief attempt was made to reposition the device, however, given its stable placement and the perceived risk of dislodgement, it was deployed. Repeat aortography and TOE showed the aortic regurgitation to be reduced (Grade 2/4). Leaflet mobility of the prosthetic valve and the right coronary ostium remained unimpinged.

Recovery was initially complicated by severe postural hypotension, which rapidly improved with alterations to medical management. By day four the regurgitation was trivial – mild (Grade 1/4), likely due to seating of the device and oedema of the surrounding tissues. Repeat TTE one month post-procedure showed the Amplatzer device to be well seated and the left ventricle to have returned to normal size and function. There was trivial regurgitation (see Fig. 2B), with the device presumably fibrosed in place to minimise the leak.

At late follow-up 18 months post paravalvar leak closure, the patient was well and active. There was no haemolysis and aspirin was the only anti-platelet agent. Ventricular function remained normal. There was no evidence of left ventricular outflow tract obstruction (LVOT V_{max} 1.2 m/s), and the peak transaortic gradient was 36 mmHg, which is considered satisfactory for this valve. There was trivial residual aortic regurgitation.

Discussion

In the early period following valve replacement surgery, the occurrence of paravalvar leaks has been reported as high as 47.6% [5]. Most of these are benign with clinically significant regurgitation present in only 1–5% of replaced valves; 60% of which arise within the first year of replacement [1,6]. Mechanical prostheses are more prone than biological valves to develop clinically significant paravalvar leaks with mitral valves affected twice as frequently as aortic valves [7,8].

Paravalvar leaks are most commonly caused by suture dehiscence between the sewing ring and the valve annulus [5]. This process may be precipitated by annular calcification, inherent tissue friability, infection, or be secondary to surgical technique [9].

Symptomatically, paravalvar leaks can manifest as diminished functional capacity, heart failure, pulmonary hypertension, arrhythmias or transfusion-dependent haemolytic aenemia. Typically, small (<3 mm) or multiple leaks around the valve are associated with haemolysis, whereas larger leaks (>6 mm) tend to result in a heart failure predominant picture [1,10].

Re-operation with replacement or re-suturing of the prosthesis is currently the gold standard treatment for symptomatic patients not responding to medical therapy [10]. Re-operation is high-risk with mortality reported as 12.6%, 14.9% and 37% following first, second and third reoperations respectively [3]. Age, preoperative NYHA functional class, urgency of the surgery, presence of active infective endocarditis and concomitant valve surgery or valve dysfunction are all independent risk factors for in-hospital mortality [3,4]. Given frequently poor outcomes