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Percutaneous paravalvular leak repair for severe aortic regurgitation after transcatheter aortic valve implantation (TAVI)

Dear Editor,

Paravalvular leaks (PVLs) are not uncommon after surgical valve replacement. The need for re-operation for clinically significant leaks is between 1 and 3%.[1] Percutaneous PVL closure has become increasingly performed and is an alternative to surgery.[1] PVLs occur more frequently after transcatheter aortic valve implantation (TAVI), as the transcatheter heart valve (THV) is anchored by radial force alone.[2] The experience of percutaneous repair of significant paravalvular aortic regurgitation (AR) after TAVI is limited.[3-9] We report a case of heart failure due to severe PVL after TAVI that has been treated with percutaneous repair with a good 4-year clinical outcome.

A 72-year-old man presented with exertional dyspnoea (New York Heart Association [NYHA] Class III) over 6 months. Echocardiography demonstrated a severely impaired left ventricular ejection fraction (LVEF) of 30% and severe paravalvular AR around a bioprosthetic valve (Fig. 1A). Transoesophageal echocardiogram (TEE) confirmed severe paravalvular AR with a pressure half-time of 213ms, holodiastolic reversal in the descending thoracic aorta, and PVL jet occupying 35% of the prosthesis circumference. The PVL defect width was approximately 6mm.

The man underwent TAVI 4 years ago with a 31mm self-expanding CoreValve (Medtronic plc, Dublin, Ireland) bioprosthesis for severe aortic stenosis. LVEF was 25%. Pre-TAVI computerised tomography showed that the aortic annulus perimeter and diameter were 86.5mm and 27.5mm, respectively. The 31mm CoreValve was selected at that time as the largest balloon-expandable valve, the 26mm Sapien XT (Edwards Lifesciences Corp, Irvine, US) available in Singapore at that time, was too small. CoreValve position and expansion were satisfactory, but there was residual moderate paravalvular AR despite post-dilatation with a 25mm Z-Med balloon (BVM Medical Ltd, Hinckley, UK). Nevertheless, the man experienced symptomatic improvement (NYHA Class II) with increase in LVEF at 1 month to 45%. His other medical conditions included right lobectomy for lung carcinoma, chronic obstructive pulmonary disease, as well as coronary and iliac artery angioplasty and stenting.

Due to the high surgical risk, percutaneous PVL closure was performed. The procedure was performed under general anaesthesia and TEE guidance. Attempts to cross the paravalvular defect retrograde via right and left femoral artery using various catheters and wires were unsuccessful (Fig. 2A). Via the right radial artery,

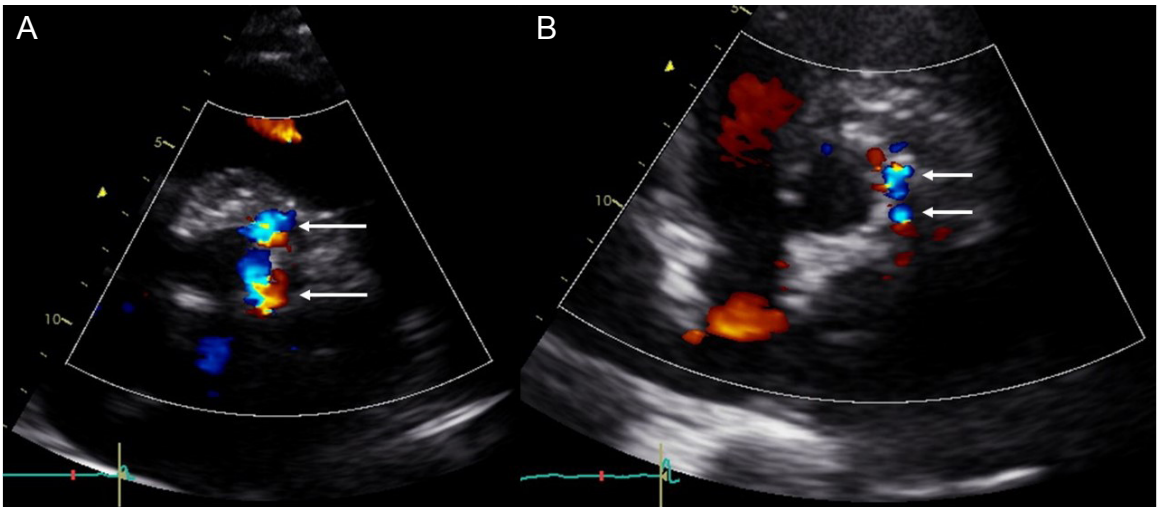

Fig. 1. (A) Echocardiographic image of the CoreValve THV in parasternal short axis view at baseline. The arrows denote 2 jets of PVLs along the THV circumference from 1 to 4 o'clock positions. (B) Echocardiographic image of the CoreValve THV in parasternal short axis view showing a reduction in PVL (arrows denote PVL from 2 to 3 o'clock positions) post-procedure.
PVL: paravalvular leak; THV: transcatheter heart valve

an angled hydrophilic guide wire and a 5-French sized multipurpose A1 catheter finally crossed the PVL into the left ventricular cavity (Fig. 2B). Both TEE and fluoroscopy confirmed that the wire and catheter were within the paravalvular defect. Attempts to exchange the 5-French guide catheter for a 6-French sized catheter over an extra-stiff wire were unsuccessful as the 6-French catheter was too large to cross the defect. Through a 5-French Judkins right 4 curve guide catheter, an 8mm Amplatzer Vascular Plug (AVP) 2 was advanced but extreme resistance was felt midway and the AVP2 could not be advanced further. The AVP2 was then removed and a smaller profile 8mm AVP4 was successfully deployed across the PVL (Fig. 2C). The diastolic pressure rose from 40mmHg at baseline to 50mmHg, and the left ventricular end diastolic pressure decreased from 23mmHg to 19mmHg. Echocardiography showed a reduction from severe to moderate paravalvular AR (Fig. 1B). We decided not to deploy a second plug to further reduce the AR due to the technical difficulties and risk of dislodging the first plug.

The patient was discharged the next day. He reported symptomatic improvement (NYHA class II), and echocardiography revealed that LVEF had returned to baseline (45%) at 3 months. He remained in Class NYHA II at 4 years after percutaneous PVL closure, without evidence of haemolysis. Echocardiography revealed unchanged LVEF (45%), moderate paravalvular AR (pressure half-time 465ms, PVL jet occupying 15% of the prosthesis circumference) and a mean aortic valve gradient of 8mmHg.

TAVI has become an established treatment for patients with severe aortic stenosis,[10] and clinical outcomes have improved with newer devices. Although PVLs after TAVI have become less frequent,[11,12] they still occur and may occasionally be clinically significant. It has been demonstrated that mild or greater PVL after TAVI can increase long-term mortality.[2] Successful percutaneous symptomatic PVL repair after TAVI has been shown to reduce hospitalisation related to heart failure and mortality as compared to conservative treatment.[4]

Most PVLs after TAVI remain stable or improve over time, though it may worsen in approximately 20% of patients.[2] PVLs occur due to an undersized THV relative to the aortic annulus, heavy calcification preventing sealing of the THV against the annulus, or a suboptimal THV implant position.[2] If the THV position was ideal, the simpler manoeuvre would be to perform a balloon post-dilatation to ensure full THV expansion and sealing. This was not performed in our patient at the second intervention as the THV appeared well expanded on fluoroscopy, and to avoid leaflet damage that may worsen the AR and reduce THV durability.

The success rate of percutaneous repair for symptomatic PVL after TAVI ranges from 60–89%. Complications, though infrequent, can include cardiac tamponade, stroke or THV dislodgement.[3-6] It is

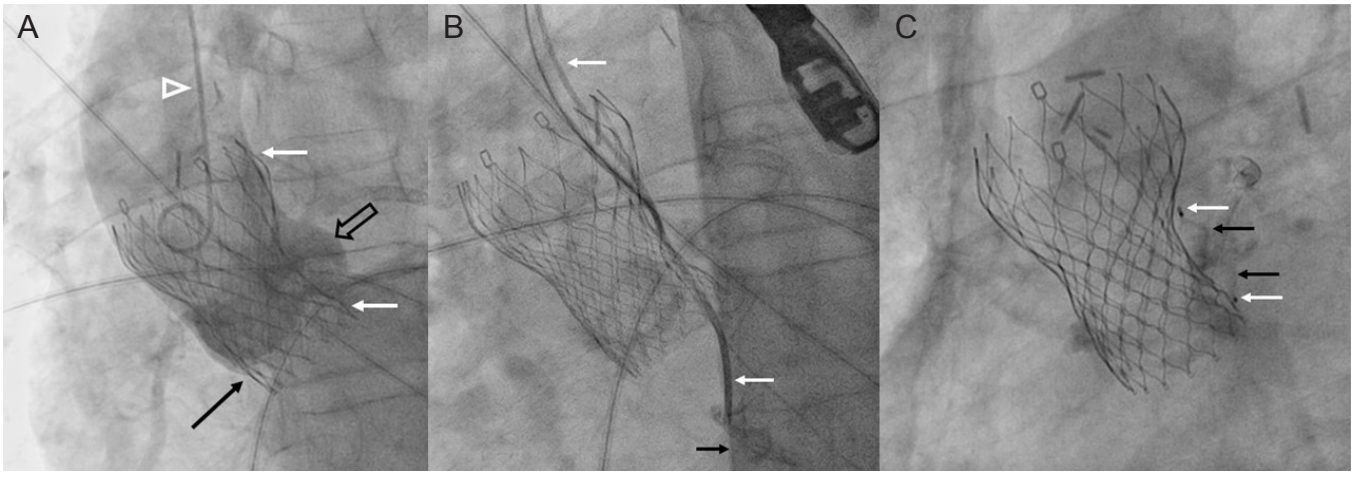

Fig. 2. (A) Fluoroscopic image of the CoreValve THV during an aortogram. White arrows denote the length of the CoreValve frame, black arrow denotes the plane of the native aortic annulus, black open arrow denotes the limited space in the sinus of Valsalva, and white arrowhead denotes the pigtail catheter. (B) Fluoroscopic image showing multipurpose catheter (white arrows) crossing the CoreValve frame and paravalvular defect. Contrast injection (black arrow) confirms that the catheter is in the left ventricle. (C) Fluoroscopic image showing the AVP2 plug deployed within the paravalvular defect (external to the CoreValve frame). The white arrows denote the radiopaque tips and black arrows denote the body of the plug.
AVP2: Amplatzer Vascular Plug 2; THV: transcatheter heart valve

technically challenging with a lower success rate as compared to PVL closure of surgical bioprostheses.[6] This is due to the retained native calcific aortic leaflets (compressed between the THV frame and aortic wall) and aortic annular calcium that is not removed (unlike surgical aortic valve replacement). The self-expanding THVs tall stent frame pose additional challenges and the supra-annular location of its leaflets limits the space for catheter manipulation. Procedural failures have been due to the inability to cross the defect with a wire or catheter.[3,4]

The devices used most frequently in PVL closure are the AVPs, which were developed for use in arterial and venous embolisation. The availability of the low profile AVP4 plug has increased procedural success as it can be delivered through small 4-French diagnostic catheters, which are more likely to cross irregularly shaped calcific defects.[7,9] This has led to procedural success in our case.

To the best of our knowledge, we report Asia's first percutaneous closure of severe PVL after TAVI with a 4-year outcome. It illustrates that sustained clinical benefit can be derived from partial reduction of paravalvular AR post-TAVI. Percutaneous repair for severe paravalvular AR after TAVI is feasible despite technical challenges. This resulted in symptomatic improvement and a good outcome in a high-risk patient.

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Paul TL Chiam, [1,2]*MRCP*, Choon Pin Lim, [1]*MRCP*, Yean Teng Lim, [3,4]*MRCP*, Cumaraswamy Sivathasan, [5]*FRCS*

[1] The Heart and Vascular Centre, Mount Elizabeth Hospital, Singapore
[2] Department of Physiology, Yong Loo Lin School of Medicine, National University of Singapore, Singapore
[3] Cardiology Associates Pte Ltd, Mount Elizabeth Hospital, Singapore
[4] Department of Medicine, Yong Loo Lin School of Medicine, National University of Singapore, Singapore
[5] The Heart, Lung and Vascular Centre, Mount Elizabeth Hospital, Singapore

Correspondence: Assoc Prof Paul TL Chiam, The Heart and Vascular Centre, #08-06 Mount Elizabeth Medical Centre, 3 Mount Elizabeth, Singapore 228510.
Email: paulchiam@heartvascularcentre.com