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CASE REPORT

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Subacute left ventricular outflow tract obstruction after transapical closure of a mitral paravalvular leak in the region of the aortomitral curtain

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We report a rare case of progressive left ventricular outflow tract (LVOT) obstruction after percutaneous device closure of a mechanical prosthetic mitral valve (MV) paravalvular leak (PVL) in the region of aortomitral curtain in a patient who also had small mechanical aortic valve prosthesis with patient–prosthesis mismatch.

KEYWORDS
angiography, aortic, cardiac catheterization, mitral, paravalvular leak, rheumatic heart disease

1 | INTRODUCTION

Transcatheter closure of PVL has become relatively common and is generally successful, particularly with newer generation occlusion devices.[1] Complications are uncommon and often relate to the access approach. Several device-related complications have been recognized, including embolization, impingement on mechanical valve leaflets, endocarditis, new onset or worsening of hemolysis, and erosion of the device through a bioprosthetic valve leaflet.[2,3]

2 | CASE REPORT

A 62-year-old, 45-kg woman with history of multivalvular rheumatic heart disease and prosthetic valves in the mitral (MV), aortic (AOV), and tricuspid (TV) positions, presented with ascites, shortness of breath at rest, and lower extremity edema. She initially underwent mitral valve replacement (MVR) at the age of 29 years, and 11 years later she developed severe prosthetic mitral stenosis and native aortic stenosis, for which she underwent replacement of the MV prosthesis with a 29 mm Omniscience titling disk valve and AOV replacement with a 19 mm Omniscience tilting disk valve.

There was a 30 mm Hg peak gradient (peak velocity 2.7 m/s) across the mechanical AOV from the outset. Two years later, she underwent TV replacement with a 29 mm Carpentier-Edwards porcine valve for severe functional tricuspid regurgitation, after which her heart failure symptoms improved. She was relatively asymptomatic for the next 19 years, but then presented in NYHA class IV with symptoms of pulmonary hypertension and right heart failure. She complained of progressive shortness of breath, significantly reduced exercise tolerance from 5 blocks to less than half a block in the last year, severe abdominal distension, and lower extremity swelling.

Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) demonstrated severe bioprosthetic TV stenosis (Fig. 1A, C, and E with corresponding movie clips), severe PVL around the mechanical MV prosthesis (Fig. 2A and C with corresponding movie clips), thrombus in the left atrial appendage, normal left ventricular (LV) function, an estimated maximum instantaneous LV outflow tract (LVOT) of ~30–35 mm Hg (2.9–3.1 m/s), and an estimated RV pressure of at least 65 mm Hg. The decision was made to treat the bioprosthetic TV stenosis alone as a first intervention and proceed with the mitral PVL closure as a second intervention, if her status did not improve.

2.1 | First intervention

Using a femoral venous approach, the TV bioprosthesis was dilated with a 20 mm × 4 cm Atlas balloon (C.R. Bard, Inc., Murray Hill, NJ, USA) at high pressure, after which a Melody valve (Medtronic,

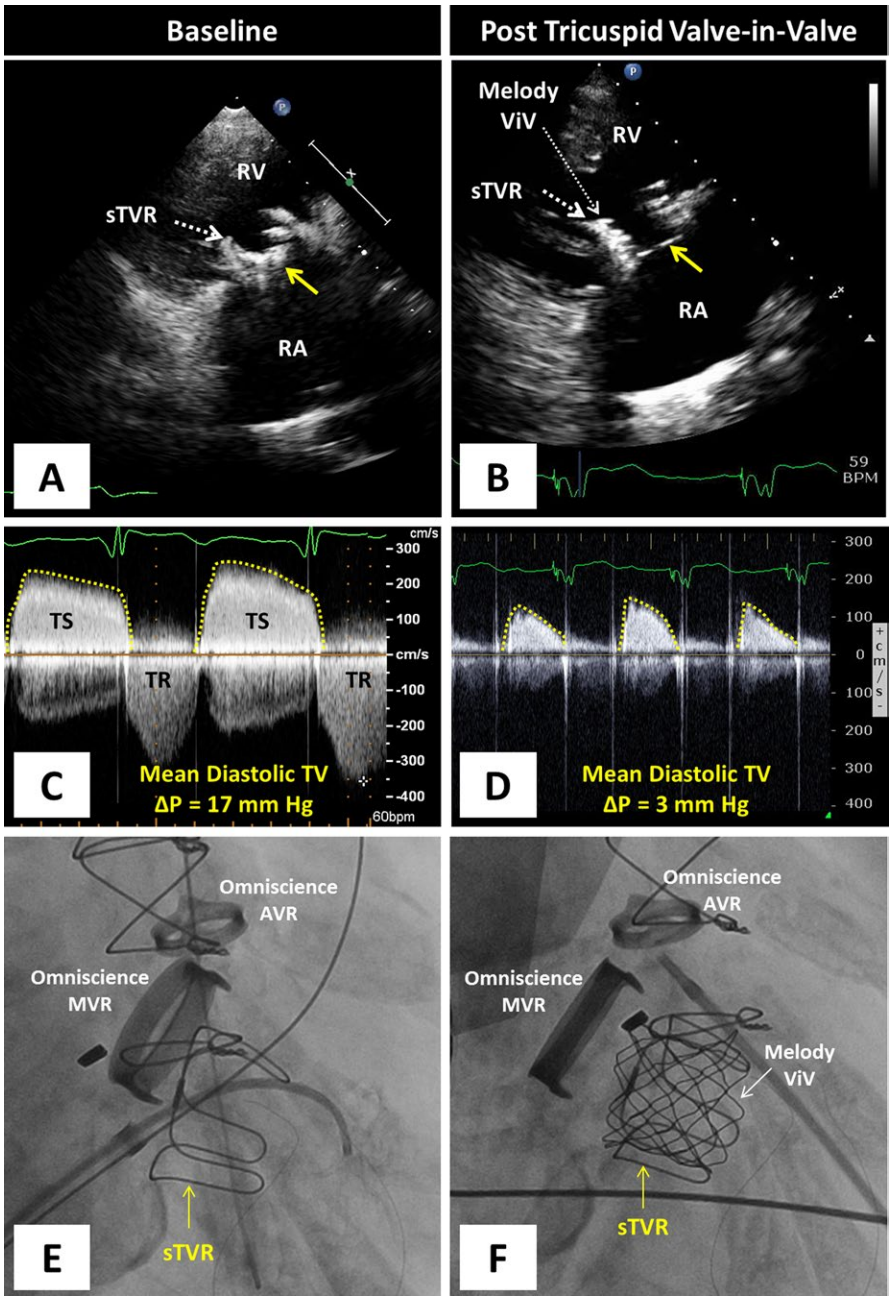

FIGURE 1 Tricuspid Melody valve-in-valve procedure. **A**, **C**, and **E.** demonstrate severe stenosis of a surgical tricuspid bioprosthesis at baseline, while **B**, **D**, **F.** show status posttricuspid Melody valve-in-valve procedure. **A**, **B.** Right ventricular inflow view on transthoracic echocardiogram. **A.** demonstrates severe thickened leaflets (yellow arrow) of a surgically implanted tricuspid bioprosthesis. In (**B**), tricuspid stenosis is no longer present following percutaneous implantation of a Melody valve inside the surgical bioprosthesis. **C, D.** Continuous-wave spectral Doppler across the tricuspid valve. **A.** demonstrates a markedly elevated mean diastolic gradient of 17 mm Hg at baseline. **B.** demonstrates resolution of tricuspid stenosis and normalization of the mean diastolic gradient at 3 mm Hg postpercutaneous tricuspid Melody valve-in-valve implantation. **E, F.** Fluoroscopy of tricuspid valve before (**E**) and after (**F**) percutaneous implantation of a Melody valve inside a surgical tricuspid bioprosthesis. AVR = aortic valve replacement; MVR = mitral valve replacement; RA = right atrium; RV = right ventricle; sTVR = surgically implanted tricuspid valve bioprosthesis; TV = tricuspid valve; ViV = valve-in-valve

Minneapolis, MN, USA) was implanted on a 22 mm delivery system (Fig. 1B, D, and F with corresponding movie clips). The mean trans-TV gradient was reduced from 10 to 2 mm Hg, the mean right atrial pressure went from 34 to 24 mm Hg, RV end-diastolic pressure decreased from 28 to 26 mm Hg, but RV pressures increased from 82 to 98 mm Hg.

2.2 | Second intervention

Despite a minimal trans-TV gradient and no TR, her symptoms improved only minimally, so PVL closure was planned. Computerized tomography scan and transesophageal echocardiography (TEE) were performed to assess the anatomy of the PVL and the location of the LV apex relative to surrounding structures, in anticipation of transapical percutaneous access.

Under direct 2D/3D TEE (Fig. 2B and D with corresponding movie clips) and fluoroscopic guidance (Fig. 3 with corresponding movie clips), access was obtained percutaneously through the left lateral chest wall into the LV apex with a 22G needle, which was exchanged for a 6Fr sheath using a micro puncture set. The PVL appeared by TEE as an aneurysmal defect in the anterolateral region of the mitral valve and was ultimately localized and crossed using a glide catheter and a 0.035" Terumo wire (Terumo Medical Corporation, Somerset, NJ, USA). An Amplatzer super stiff wire (St. Jude Medical, Inc., St. Paul, MN, USA) was positioned in the left atrium and followed by a 6 Fr main pulmonary artery guide catheter. The defect was closed with a 10 mm Amplatzer vascular plug II device, and position was confirmed by TEE and fluoroscopy before release. TEE demonstrated an excellent result with trivial residual

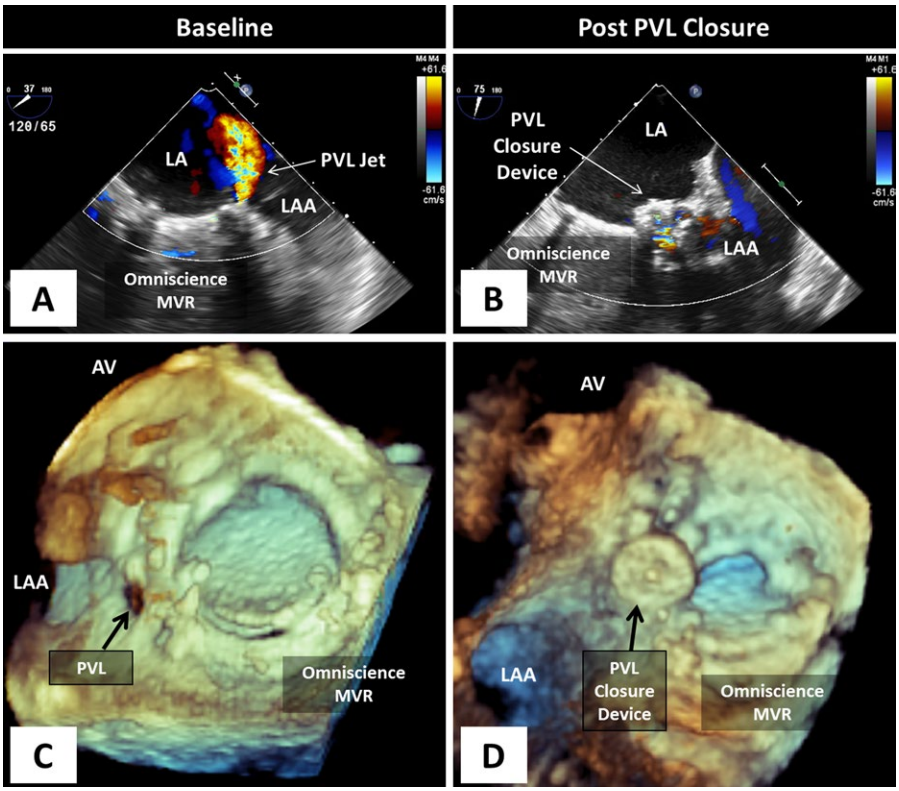

FIGURE 2 2DTEE and 3DTEE of Mitral Omniscience Mechanical Prosthesis. **A, B.** 2DTEE color Doppler images demonstrates severe paravalvular mitral regurgitation at baseline (**A**). After percutaneous device closure, paravalvular mitral regurgitation is no longer present (**B**). **C, D.** 3DTEE demonstrates an Omniscience mechanical mitral prosthesis in the standard surgical view from the left atrial perspective. At baseline (**C**), there is a paravalvular leak (PVL) adjacent to the left atrial appendage (LAA). **D.** PVL closure device

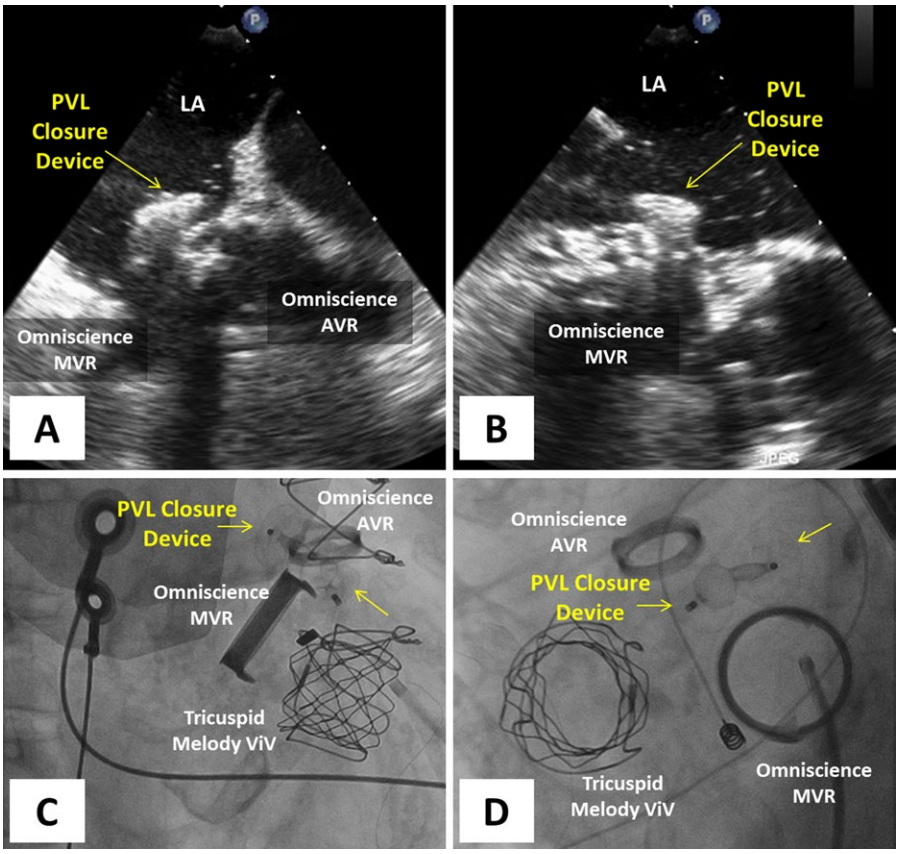

FIGURE 3 3DTEE and fluoroscopic biplane imaging of PVL closure device. Percutaneously implanted PVL closure device is visualized on biplane 3DTEE (**A, B**) and fluoroscopy (**C, D**). AVR = aortic valve replacement; LA = left atrium; MVR = mitral valve replacement; PVL = paravalvular leak; ViV = valve-in-valve

PVL and no change in the LVOT gradient. The LV apical puncture site was closed using a 6/4 mm Amplatzer duct occluder with no complications.

She had modest improvement in her heart failure symptoms and was discharged successfully home on anticongestive and anticoagulation medications 5 weeks later. A predischarge echocardiogram confirmed

no change in the LVOT gradient. During the next 3 months, her clinical status gradually improved, but 4 months after PVL closure she presented again with exacerbation of symptoms and was readmitted.

Transthoracic echocardiography and TEE showed progressively increasing gradients across the LVOT, with a peak velocity of 3.0 m/s at readmission up to 4.1 m/s and 5.7 m/s 16 and 17 weeks later (Fig. 4). There was no evidence of endocarditis, thrombus, recurrent PVL, or AOV prosthesis dysfunction. Initially, she was noted to have low baseline hematocrit with no other laboratory findings suggestive of hemolysis. Subsequently, she developed clinical features of septic shock with multiorgan failure. After her clinical condition continued to worsen, the family decided not to escalate care and the patient expired.

3 | DISCUSSION

The incidence of clinically significant mitral valve PVL is reported at 1%–5% following surgical valve replacement.[4] Most of the reported complications in patients undergoing transcatheter PVL have been periprocedural, including bleeding, immediate embolization of the device, interference with mechanical valve leaflets, or coronary artery obstruction. However, subacute and chronic complications can also occur, such as hemolysis leading to significant anemia and renal dysfunction, reconfiguration of the device and recurrence of regurgitation, endocarditis, and device embolization.

In this case, we observed a previously unreported adverse event, namely progressive LVOT obstruction after MV PVL closure in the context of AOV prosthesis with patient–prosthesis mismatch. This case highlights several important considerations:

- If a device that is used to close either a MV or AOV prosthetic PVL in the region of aortomitral continuity protrudes into the LV, it may contribute to LVOT obstruction. Accordingly, the LVOT gradient should be assessed with Doppler echocardiography before and after device placement, and if the available vascular access allows, with directly measured pressure measurements as well.
- Second, in the presence of both mitral and aortic valve prostheses, it can be challenging to visualize a PVL closure device in fine detail

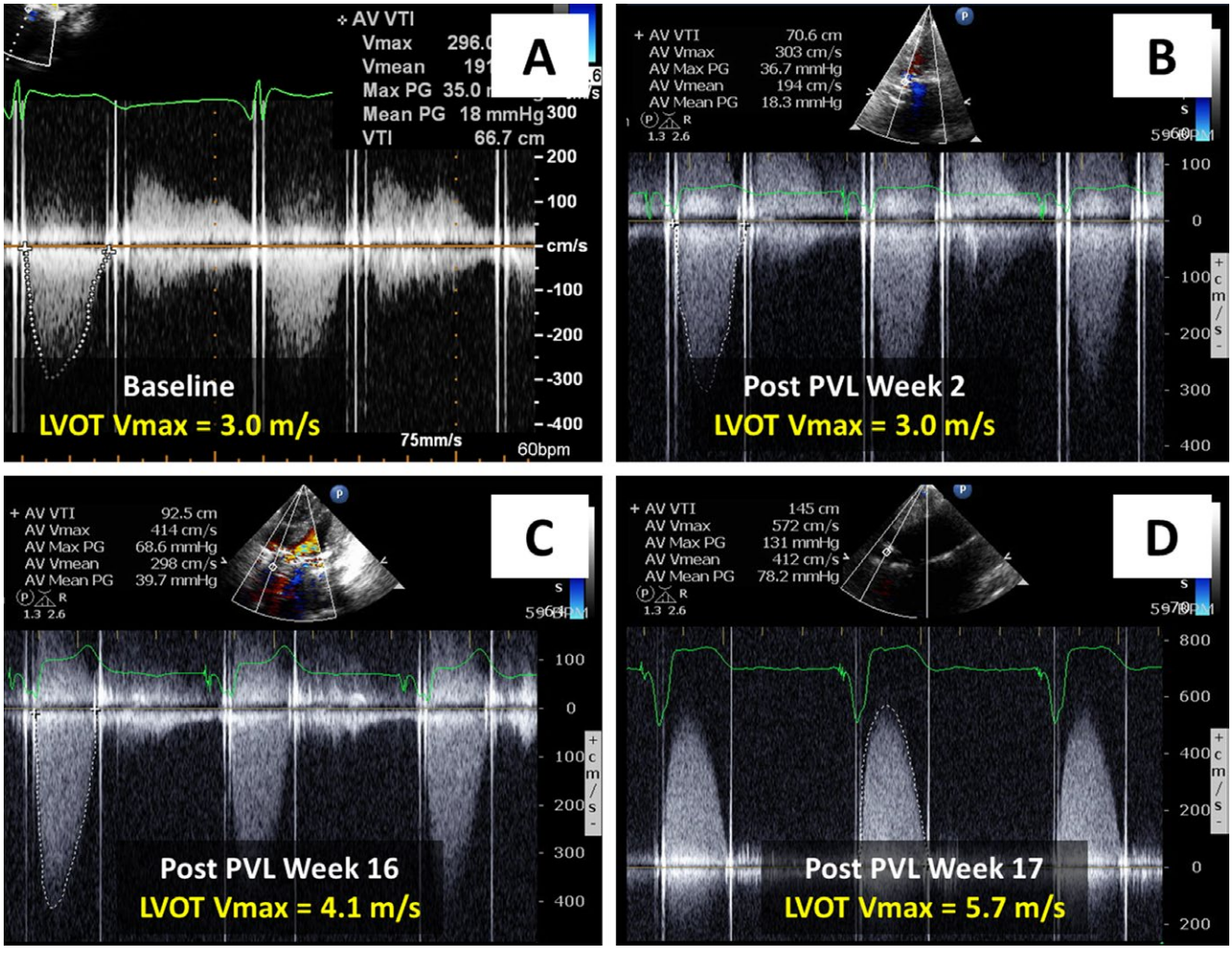

FIGURE 4 Progression of LVOT Obstruction. **A–D**. Spectral Doppler imaging demonstrates a progressive increase in the peak velocity (Vmax) across the LVOT from 3.0 m/s at baseline and at 2 weeks post procedure (**A, B**) to 4.1 m/s and 5.7 m/s (**C, D**) postpercutaneous device closure of the mitral mechanical prosthesis PVL

with echocardiography, potentially limiting the ability to evaluate the device in detail, and/or to identify vegetations or other thrombus.

- The importance of understanding the anatomy of aortomitral curtain is crucial in planning and assessing treatment of PVL in this region, particularly in patients with AOV and MV prosthetic valves. In particularly, in the setting of a small AOV prosthetic or impingement of the MV prosthetic on the LVOT, baseline LVOT obstruction may potentially be exacerbated by the presence of a PVL closure device protruding into this region.[5]

The mitral valve is located posterior and to the left of aortic valve, the ends of aorto-mitral fibrous continuity are thickened to form the left and right trigones. The inter-leaflet area between the left and noncoronary cusp is part of aortomitral curtain, and placement of aortic valve prosthesis too low in the LVOT may cause impingement of mitral valve and alters the function.[5] In our patient, the progressive in LVOT velocities was likely a combination of endothelialization and self-reorientation of the device along with flow-related changes from progressive anemia and sepsis. However, the magnitude of this increased gradient across LVOT is unlikely to be explained by anemia and or sepsis alone.

Although this is likely to be an uncommon complication that occurs in very specific circumstances, it is important to be aware that it is possible and to consider LVOT obstruction during early and subsequent follow-up in patients undergoing AOV or MV PVL closure. Even in the absence of early gradient changes, progressive obstruction may develop in the setting of improved cardiac output or potentially in other circumstances.

Surgery was obviously high risk in this patient, and transcatheter management was determined to provide the best hope of improvement. Alternative approaches to closure that may have avoided LVOT obstruction could have included utilization of a different device, transseptal closure with the device drawn firmly back against the sewing ring, or other techniques that would have minimized the portion of the device on the LV side of the PVL.

4 | CONCLUSION

Devices in region of the aortomitral curtain can potentially contribute to obstruction, particularly in patients with patient–prosthesis mismatch of aortic valve prosthesis. In any event, impact of MV and AOV devices on the LVOT should be assessed if they are in the region of the aortomitral curtain.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

Movie clip S1. Right ventricular inflow on transthoracic echocardiogram demonstrates severe leaflet thickening of a surgically implanted tricuspid bioprosthesis at baseline. This video corresponds to Fig. 1A.

Movie clip S2. Right ventricular inflow on transthoracic echocardiogram demonstrates resolution of severe tricuspid stenosis after percutaneous implantation of a Melody valve inside the surgical tricuspid bioprosthesis. This video corresponds to Fig. 1B.

Movie clip S3. Three prosthetic valves are demonstrated on fluoroscopy at baseline (mechanical Omniscience MVR and AVR) as well as a surgical tricuspid bioprosthesis (sTVR). This video corresponds to Fig. 1E.

Movie clip S4. Following the valve-in-valve procedure (ViV), a Melody valve is visualized inside the surgical tricuspid bioprosthesis (sTVR). This video corresponds to Fig. 1F.

Movie clip S5. 2D color Doppler TEE imaging demonstrates a large paravalvular leak (PVL) of the patient's mitral mechanical Omniscience prosthetic valve. This video corresponds to Fig. 2A.

Movie clip S6. Post percutaneous PVL closure, 2D color Doppler TEE imaging demonstrates complete resolution of paravalvular mitral regurgitation. This video corresponds to Fig. 2B.

Movie clip S7. At baseline, 3D TEE demonstrates an Omniscience mitral mechanical prosthesis with a paravalvular leak (PVL) adjacent to the left atrial appendage (LAA). This video corresponds to Fig. 2C.

Movie clip S8. Post percutaneous PVL closure, 3D TEE imaging from the left atrial perspective demonstrates PLV closure device obliterating prior PVL. This video corresponds to Fig. 2D.

Movie clip S9. PVL closure device is visualized by biplane 3D TEE imaging. This video corresponds to Fig. 3A and B.

Movie clip S10. PVL closure device is visualized on fluoroscopy in the right anterior oblique (RAO) projection at RAO 37°, CRANIAL 1°. This video corresponds to Fig. 3C.

Movie clip S11. PVL closure device is visualized on fluoroscopy in the left anterior oblique (LAO) projection at LAO 44°, CAUDAL 21°. This video corresponds to Fig. 3D.

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