

Transcatheter Aortic Valve Replacement to Treat Left Ventricular Outflow Tract Obstruction and Significant Paravalvular Leak Following Transcatheter Mitral Valve Replacement



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INTRODUCTION

Mitral regurgitation (MR) is a leading cause of valvular heart disease,¹ but up to half of patients with moderate to severe MR are not referred for conventional mitral valve surgery, because of advancing age or multiple comorbidities.² Transcatheter mitral valve replacement (TMVR) with the Tendyne device (Abbott Vascular, Santa Clara, CA) is an alternative therapy for a subset of patients with significant MR at prohibitive operative risk because it does not require cardiopulmonary bypass or sternotomy.³⁻⁵ Potential complications of TMVR devices include fixation of the native anterior mitral valve leaflet (AMVL) in the left ventricular outflow tract (LVOT) in systole, which may result in systolic anterior motion (SAM) of the AMVL and potential LVOT obstruction and paravalvular leak (PVL).⁶ We report a case of profound intravascular hemolysis causing acute kidney injury because of PVL and LVOT obstruction following Tendyne TMVR. Although PVL closure was attempted, definitive treatment was achieved with transcatheter aortic valve replacement (TAVR), which corrected both PVL and LVOT obstruction, with complete resolution of hemolysis and improvement of renal function.

CASE PRESENTATION

A 76-year-old man presented with exertional breathlessness. He had previously undergone coronary artery bypass grafting in 2004 and had a patent left internal mammary arterial graft to the left anterior descending coronary artery, a patent radial graft to the circumflex artery, and an occluded saphenous vein graft to the right coronary artery. He had hypertension and previous transitional cell carcinoma of the bladder (treated with intravesical mitomycin under annual surveillance). On examination, he weighed 75 kg, his body mass index was 25 kg/m², he was in New York Heart Association functional class II/III and sinus rhythm (heart rate 58 beats/min), and his blood pressure was 166/56 mm Hg; venous pressure was not elevated, heart sounds were dual, and he had a pansystolic murmur, loudest in the mitral region, associ-

ated with soft aortic diastolic murmur. He had no peripheral edema, and his lung fields were clear. Twelve-lead electrocardiography confirmed sinus rhythm with first-degree atrioventricular block (PR interval 286 msec), incomplete left bundle branch block (QRS duration 109 msec), and normal cardiac axis.

Transthoracic echocardiography (TTE) documented a dilated left ventricle (end-diastolic dimension 6.7 cm) with a left ventricular (LV) ejection fraction of 65%, apical septal infarct, severe MR, mild aortic stenosis, and significant aortic regurgitation (AR). Real-time three-dimensional (3D) transesophageal echocardiographic assessment of the mitral valve confirmed severe MR due to a restricted posterior mitral valve leaflet and chordal rupture of the lateral aspect of A2 and the medial aspect of A3 (Videos 1 and 2). Multislice computed tomographic imaging reported mildly calcified aortic valve leaflets (calcium score 1,370 Agatston units), aortic annular perimeter of 95 mm, an infrarenal abdominal aortic aneurysm (measuring 35 × 35 × 69 mm), and focal plaques of calcified atheromatous disease throughout the thoracoabdominal aorta but no significant luminal aortic stenosis (Figure 1).

The case was discussed at a structural heart multidisciplinary meeting. The patient was deemed high risk for conventional mitral and aortic valve replacement because of patent retrosternal arterial grafts and a European System for Cardiac Operative Risk Evaluation II score of 9.26%. His dominant valvular lesion was felt to be MR, and potential transcatheter options were discussed: the MitraClip device was not available at the time of assessment, and he was anatomically unsuitable for any other Conformité Européenne–marked device available. Postprocessing simulation of a Tendyne device within the mitral annulus on the basis of multislice computed tomographic images predicted a neo-LVOT > 250 mm² (Figure 2), and the patient was accepted for TMVR with a Tendyne device.

The patient gave informed consent for TMVR with a Tendyne device. The procedure was performed through a small left anterior thoracotomy under general anesthesia (full procedural details were reported previously⁷). The LV apex was exposed and noted to be extremely friable, with bleeding from epicardial veins. A Tendyne device was implanted under continuous real-time 3D transesophageal echocardiographic guidance (Video 3), with the valve positioned in a good location on both transesophageal echocardiography (TEE; Video 4) and fluoroscopy (Video 5). Mild PVL was noted (Figure 3), as was SAM from the mobile native AMVL, which resulted in a dynamic periprocedural LVOT gradient of 58 mm Hg on TEE. However, at a tether tension of 203 mm Hg, LV pressure was 95/37 mm Hg and aortic pressure was 108/34 mm Hg, resulting in a pull-back gradient on fluoroscopy of 13 mm Hg (Figure 4). Thus, we elected not to retrieve the Tendyne valve at that time.

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VIDEO HIGHLIGHTS

Video 1: Long axis two-dimensional TEE of the mitral valve showing chordal rupture of the AMVL and restriction of the posterior mitral valve leaflet.

Video 2: Real-time 3D TEE, en face view of the mitral valve from the left atrium, confirming chordal rupture of the lateral aspect of A2 and the medial aspect of A3 and restricted posterior mitral valve leaflet.

Video 3: Continuous real-time X-plane imaging on TEE showing deployment of the Tendyne in the mitral valve annulus.

Video 4: Real-time 3D TEE, en face view, of the Tendyne device from the left atrium, confirming correct clocking of the outer frame of the Tendyne device and central orifice with three mobile neo-mitral valve leaflets.

Video 5: Correct Tendyne positioning on fluoroscopy.

Video 6: A well-seated Tendyne device but SAM of the mitral valve into the LVOT on TTE.

Video 7: Real-time 3D TEE, en face view, of the Tendyne device from the left atrium showing two jets of PVL (one anteromedially and another anterolaterally).

Video 8: Long-axis two-dimensional TEE of the Tendyne device showing a mobile AMVL with SAM into the LVOT

Video 9: Real-time 3D TEE used to direct placement of an Agilis Steerable introducer to the anterolateral PVL.

Video 10: Placement of an Agilis Steerable introducer to the anterolateral PVL on fluoroscopy.

Video 11: Amplatzer Vascular Plug deployed in the anterior-lateral aspect of the Tendyne device on fluoroscopy.

Video 12: Anterior “lifting” of the Tendyne device on continuous real-time X-plane imaging after placement of the Amplatzer plug.

Video 13: Real-time 3D TEE en face view of the Tendyne device showing widening of the gap between the Tendyne device and the mitral annulus after placement of the Amplatzer plug.

Video 14: Long-axis two-dimensional TEE of the Tendyne device showing worsening of the PVL and distortion of the central mitral valve orifice shown by increased transvalvular color-flow turbulence.

Video 15: Long-axis two-dimensional TEE showing low deployment of a TAVR device to “pin back” the AMVL and prevent SAM of the AMVL.

Video 16: Position of the TAVR device confirmed against the anterior aspect of the Tendyne device on fluoroscopy.

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inine of 93 $\mu\text{mol/L}$ (normal range, 60–120 $\mu\text{mol/L}$), estimated glomerular filtration rate of 68 mL/min/1.73 m^2 (normal range, 60 mL/min/1.73 m^2), bilirubin of 17 $\mu\text{mol/L}$ (normal range, 0–20 $\mu\text{mol/L}$), liver alanine transaminase of 19 IU/L (normal range, 8–40 IU/L), and albumin of 42 g/L (normal range, 35–50 g/L). No PVL was noted on pre-discharge TTE, and the peak LVOT gradient was reported as 25 mm Hg.

The patient presented again 2 weeks after discharge feeling nonspecifically unwell. On examination, he was anemic and jaundiced. He was on no known hepatotoxic medications. TTE documented a well-seated Tendyne device with trivial PVL, a mobile AMVL resulting in SAM (Video 6), an LVOT gradient of 26 mm Hg, and moderate to severe AR. Laboratory investigations reported anemia and deranged liver function but normal renal function: hemoglobin 86 g/L, urea 7.0 mmol/L, creatinine 76 $\mu\text{mol/L}$, estimated glomerular filtration rate 86 mL/min/1.73 m^2 , bilirubin 55 $\mu\text{mol/L}$, alanine transaminase 119 IU/L, and albumin 32 g/L. Further investigations reported a haptoglobin level of <0.3 g/L (normal range, 0.6–2.9 g/L) and a grossly elevated lactate dehydrogenase level of 7,635 IU (normal range, 266–500 IU/L). Three-dimensional TEE reported a well-positioned Tendyne device that had not altered in position or rotation since the final periprocedural images, and there was no qualitative change in the Tendyne tether tension. However, the Tendyne appeared to “seesaw” across a bar of calcification in the center of the anterior annulus, and two PVL jets were easily identified either side of this calcification, one anteromedially and another anterolaterally (Video 7). Although the mobile AMVL and SAM were visualized (Video 8), the peak LVOT gradient on TEE was only 15 mm Hg. Cardiac computed tomography was performed (Figure 5), which confirmed appropriate positioning of the Tendyne device. The patient remained hemodynamically stable and was initially treated medically with packed red cells and β -blockade.⁸

However, 2 days later, the patient developed very rapid acute kidney injury, with urea and creatinine rising from normal to 29 mmol/L and 463 $\mu\text{mol/L}$, respectively, accompanied by a significant reduction in estimated glomerular filtration rate. Autoantibody, hemolytic uremic syndrome, and thrombotic thrombocytopenic purpura renal screens were all negative, and renal ultrasound demonstrated normal kidney echo texture and size, no hydronephrosis, and a collapsed urinary bladder containing a catheter. Random urinary albumin was grossly elevated at 524 mg/L (normal range, 0–19 mg/L), as was the urine albumin/creatinine ratio at 476 mg/mol (normal range, 0–3.0 mg/mmol). The etiology of the patient’s acute kidney injury was assumed secondary to intravascular hemolysis (plasma i.e., free hemoglobin level 2.9 g/L [normal range, <0.3 g/L]), and the patient was transferred to intensive care unit for renal support with hemofiltration.

The case was again discussed among the structural heart team. Initial consideration was given to retensioning the Tendyne device, but this option was rejected, in part because the LV apex had been very friable with LV bleeding at the time of Tendyne surgery and in part because the Tendyne was felt to be in a good position within the mitral annulus, and further tensioning of the tether might make the Tendyne device more vertical and worsen the PVL and LVOT gradient. Percutaneous PVL closure with device(s) was suggested as an alternative therapeutic option, but there was concern that this might cause lifting of the Tendyne device and subsequent worsening of the PVL. Following Tendyne TMVR, the native mitral valve leaflet is

The patient was hemodynamically stable off all inotropes periprocedurally and postoperatively and remained well on the ward. He was discharged on day 8 with hemoglobin of 146 g/L (normal range, 134–166 g/L), urea of 8.0 mmol/L (normal range, 2.5–7.8 mmol/L), creat-

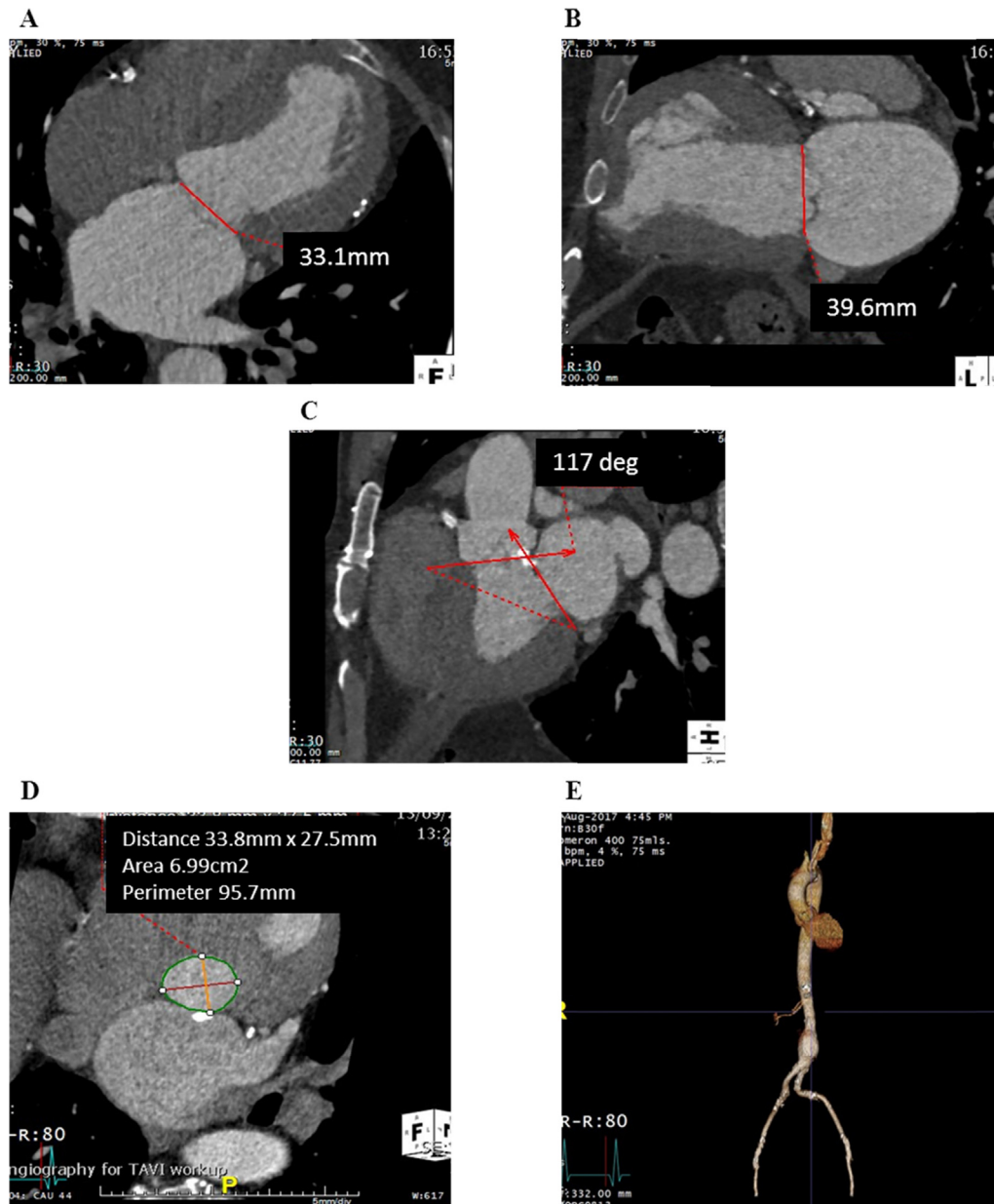


Figure 1 Preprocedural multislice computed tomographic assessment. The mitral annulus measured 33.1 mm in anteroposterior diameter (**A**) and 39.6 mm in intercommissural diameter (**B**), with an aortomitral angle of 117° (**C**). The aortic annular circumference was 95.7 mm (**D**), and an infrarenal aneurysm was noted (**E**).

fixed in an “open” position (i.e., the “SAM” position) and thus can obstruct the LVOT. In addition, because the PVL jets were mostly anterior, SAM itself could have been accentuating the PVL. Alternatively, the AR jet, directed straight onto the anterior aspect of the Tendyne device prosthesis, might have been accentuating SAM. Potential treatment with LVOT placement of a TAVR, which might have the dual benefit of relieving LVOT obstruction caused by SAM while relieving the AR while alleviating the SAM was considered. However, there were potential issues with TAVR, including a large aortic annulus (95 mm), relatively noncalcified aortic valve leaflets, and problematic femoral access. Alcohol

septal ablation, offered to patients with septal hypertrophy who develop SAM and LVOT obstruction after TMVR, was discussed. Finally, consideration was given performing a LAMPOON (laceration of the anterior mitral leaflet to prevent LVOT obstruction) procedure, whereby the AMVL could be cauterized using a trans-septal and retrograde aortic approach,⁹ potentially reducing the buildup of high LV pressure behind the AMVL contributing to the PVL.

The postprocedural real-time 3D transesophageal echocardiographic and multislice computed tomographic images were re-reviewed. Seating depth of the Tendyne valve was deemed

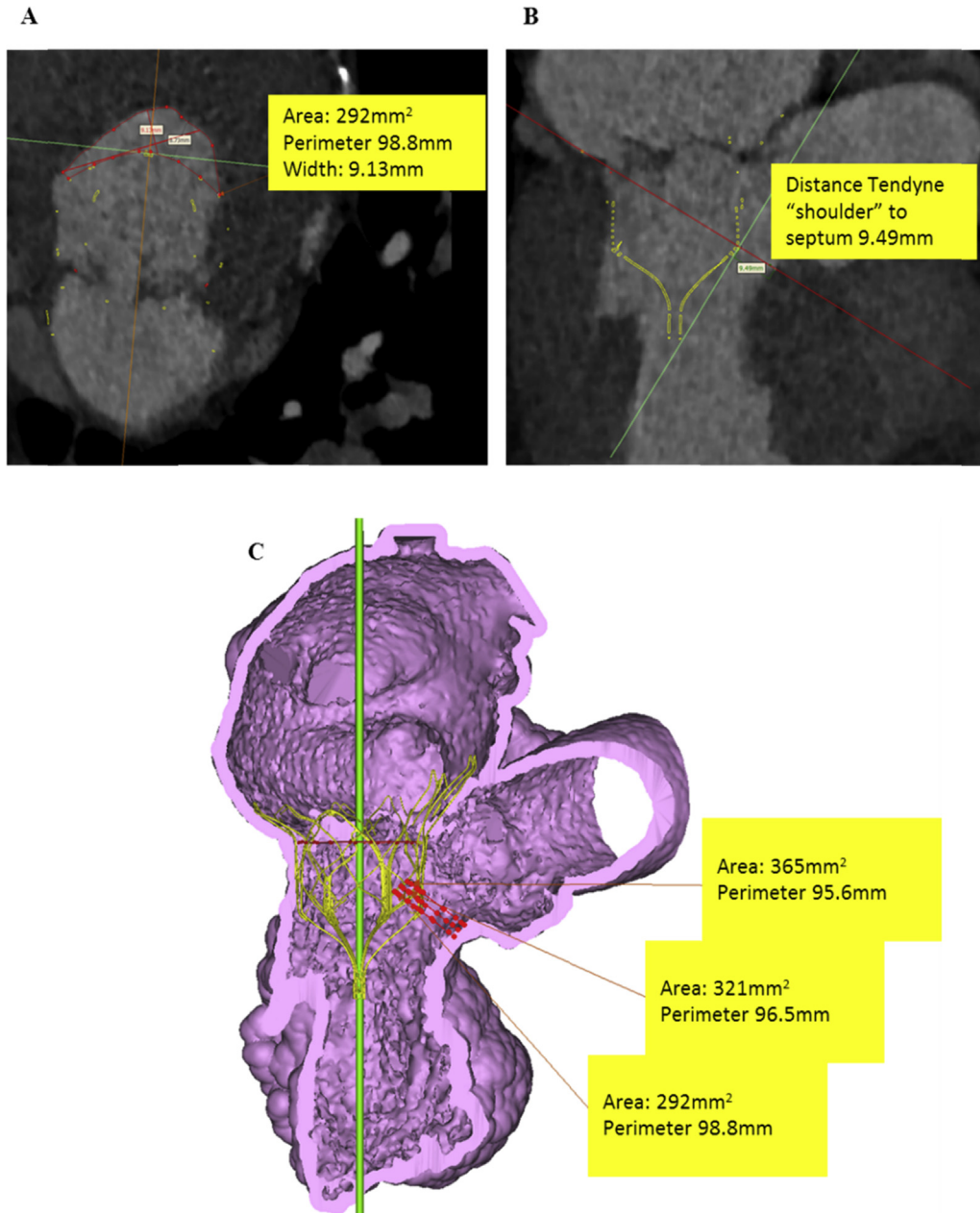


Figure 2 Assessment of predicted LVOT dimensions with simulated valve superimposed on multislice computed tomographic images. Neo-LVOT area was 292 mm² (**A**), with the smallest clearance from the shoulder of the device to the septal wall of 9.49 mm (**B**). On volume-rendered computed tomography, the smallest predicted LVOT area was 292 mm² (**C**). At end-systole, a neo-LVOT area of 250 mm² and clearance distance from the shoulder of device to the septum of 5 mm suggested a low possibility of LVOT obstruction after Tendyne implantation. Septal bump, aortomitral angle, length and shape of outflow, and anterior mitral leaflet characteristics and dynamics are other important factors.

appropriate, with the level of the mitral annulus at the point of transition between the atrial cuff and sealing body of the valve circumferentially (**Figure 4**). However, a very focal area of calcification at A2 (midanterior aspect of the AMVL), 1.2 cm in length, and noted on preprocedural 3D TEE (**Video 2**), was found to protrude 4 mm into the bioprosthetic Tendyne valve between the A2 (anterior) and A3

(anteromedial) region of the anterior mitral annulus (**Figure 6**), causing a 4-mm gap between the anterior mitral annulus and Tendyne valve.

In the first instance, we elected to attempt to address the PVL with closure devices under general anesthesia, which we believed was the least invasive therapy. A transseptal puncture was performed using a

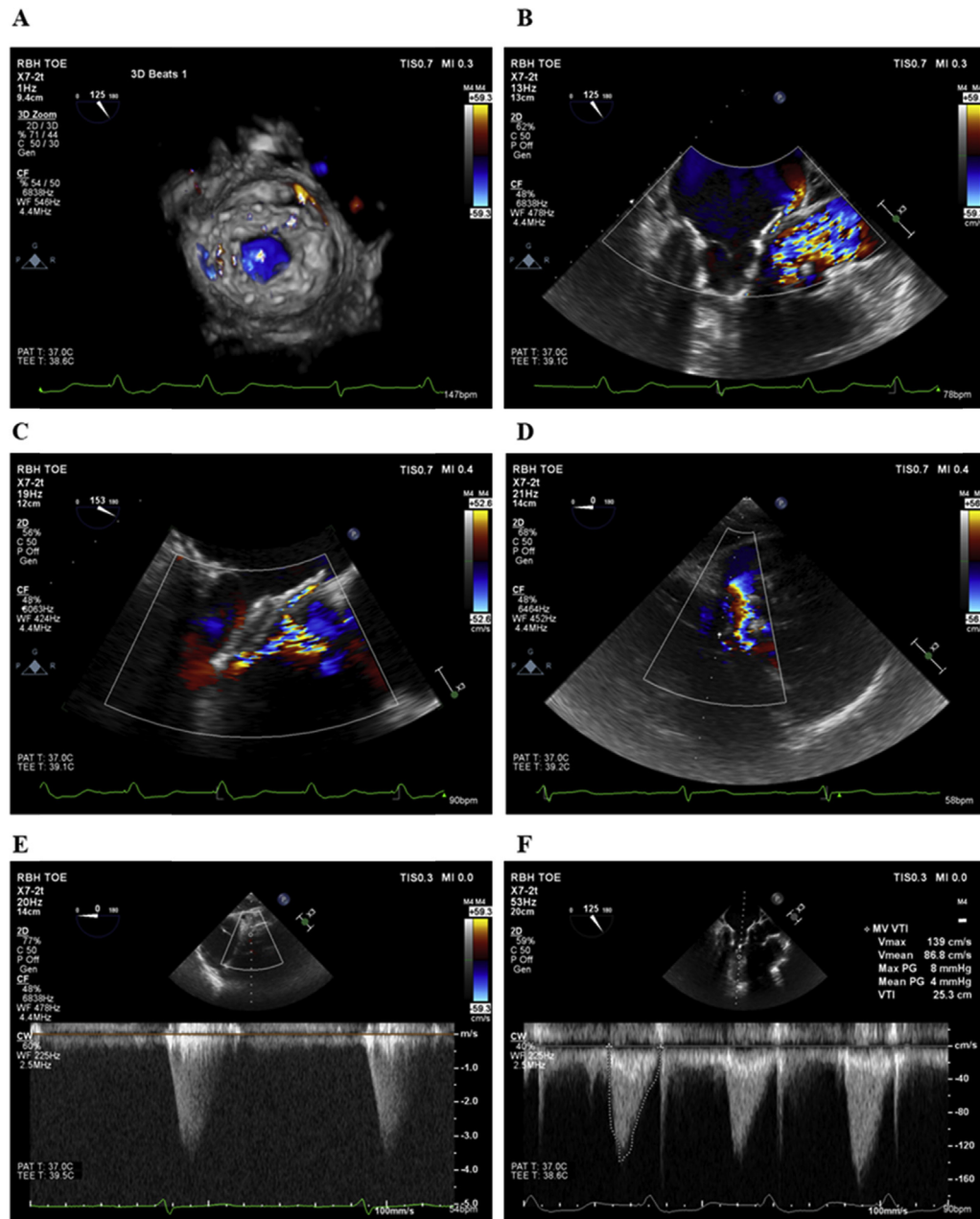


Figure 3 Periprocedural TEE. Mild anterolateral PVL (A, B) with flow acceleration in LVOT (C, D), peak LVOT gradient of 58 mm Hg (E), and mean transvalvular mitral gradient of 4 mm Hg (F).

BRK needle (St. Jude Medical, St. Paul, MN) under continuous real-time 3D TEE, and transesophageal echocardiographic measurements were made to size the Amplatzer device of choice for both PVL sites (Figure 7).

Real-time 3D TEE was then used to direct placement of an Agilis Steerable introducer (St. Jude Medical) to the anterolateral PVL (Videos 9 and 10). A 12-mm Amplatzer Vascular Plug II device (St. Jude Medical) was deployed in the correct position (Video 11). However, the Amplatzer device resulted in “lifting” of the Tendyne de-

vice (Videos 12 and 13), worsening of the PVL, and distortion of the central mitral valve orifice (Video 14) and was removed with a 15-mm goose-necked snare. Instead, a 34-mm Evolut R TAVR device (Medtronic, Minneapolis, MN) was positioned deliberately low in the LVOT under rapid LV pacing (Videos 15 and 16). The patient had three periarrest episodes with ventricular tachycardia at points when the underdeployed TAVR was obstructing cardiac output, but eventual deployment of the TAVR device in the correct position resulted in instantaneous removal of SAM and resolution of PVL.

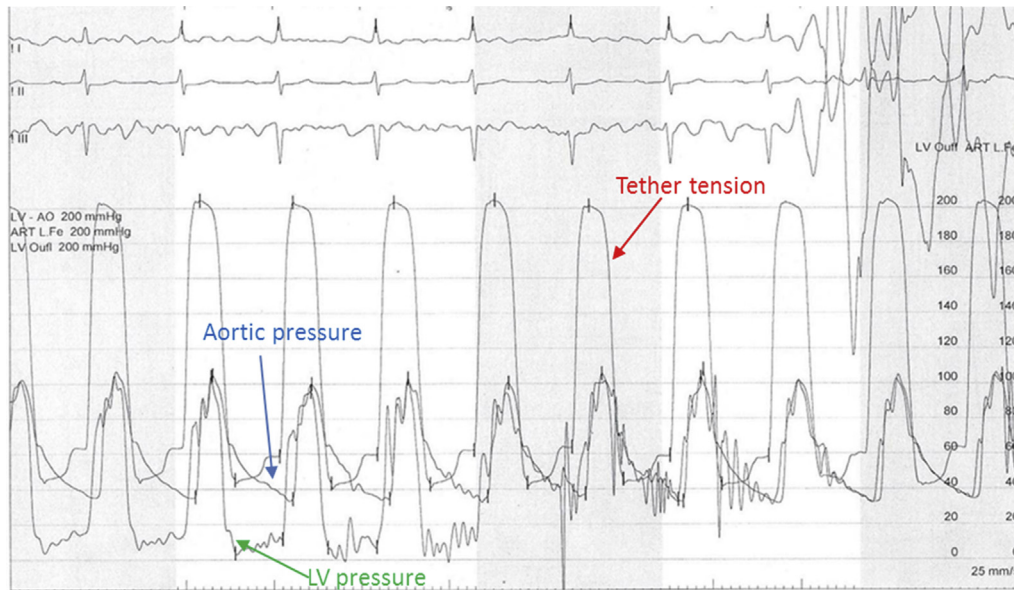


Figure 4 Hemodynamics at end of procedure. At a tether tension of 200 mm Hg, there was no significant pull-back gradient from the LVOT to the aorta. Tether tension is the pressure exerted on the apical pin when pulling the Tendyne device down toward the LV apex by the Tendyne tether device before final apical pin fixation.

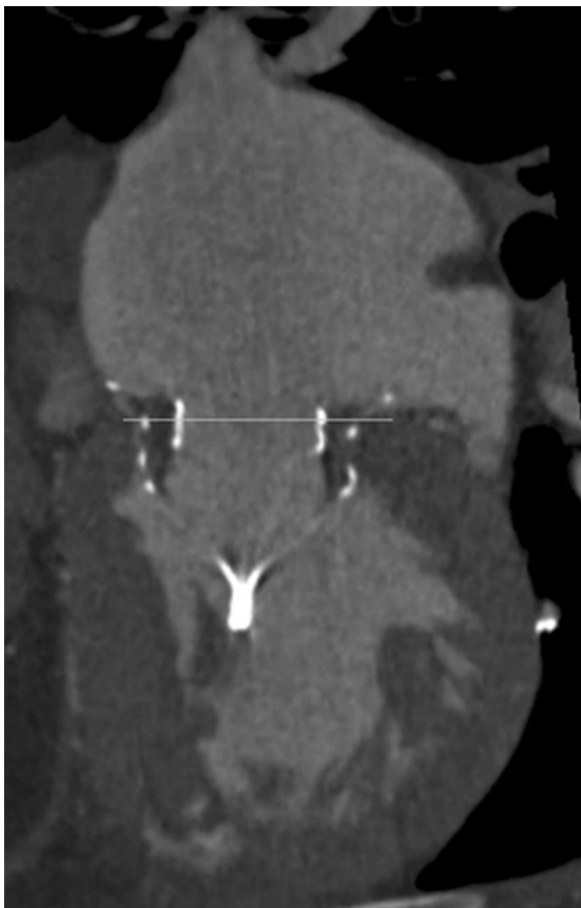


Figure 5 Postprocedural multislice computed tomography documenting well-seated intra-annular position of Tendyne device (medial and lateral aspects of the device positioned at the level of the mitral annulus with no rocking or movement of the device).

Correction of PVL removed the intracardiac hemolysis driving acute renal failure, such that plasma hemoglobin fell dramatically (from 2.9 to 0.3 g/L) within the next 24 hours. The patient required less frequent and finally no hemofiltration; on discharge, the urea had decreased to 15 mmol/L and creatinine had fallen to 266 μ mol/L. A biventricular pacemaker was required for complete heart block after transcatheter aortic valve implantation. On discharge (4 weeks after re-presentation), TTE reported a well-seated Tendyne with no PVL, a well-seated TAVR with peak gradient of 28 mm Hg, and mild to moderate PVL, LV ejection fraction of 48%, and normal right ventricular size and function with right ventricular systolic pressure of 27 mm Hg (Figure 8). The patient's acute kidney injury continued to improve, and 8 weeks after TAVR, urea was 12 mmol/L, creatinine 172 μ mol/L, and glomerular filtration rate 33 mL/min/1.73 m².

DISCUSSION

Acute kidney injury may occur with hemoglobin breakdown products, because hemoglobin proteins are toxic to tubular epithelial cells, causing acute tubular necrosis.¹⁰ Biochemical diagnosis of hemolysis is based on normocytic anemia, raised plasma hemoglobin, raised lactate dehydrogenase, elevated unconjugated bilirubin, reduced haptoglobin (a protein that binds to free hemoglobin released in active hemolysis), raised reticulocyte count (>2%), and red blood cell fragments and polychromasia on a blood film examination.

PVL is an uncommon but potentially serious complication following valve replacement surgery, resulting from incomplete apposition of the prosthetic sewing ring to the native annulus, leading to isolated or multiple blood flow jets through the communication between the two heart chambers related to the valve. The prevalence of mitral PVL following conventional mitral valve surgery is 7% to 17%¹¹ but may be as high as 20% after TMVR.⁶ Shear stress placed on red blood cells passing through

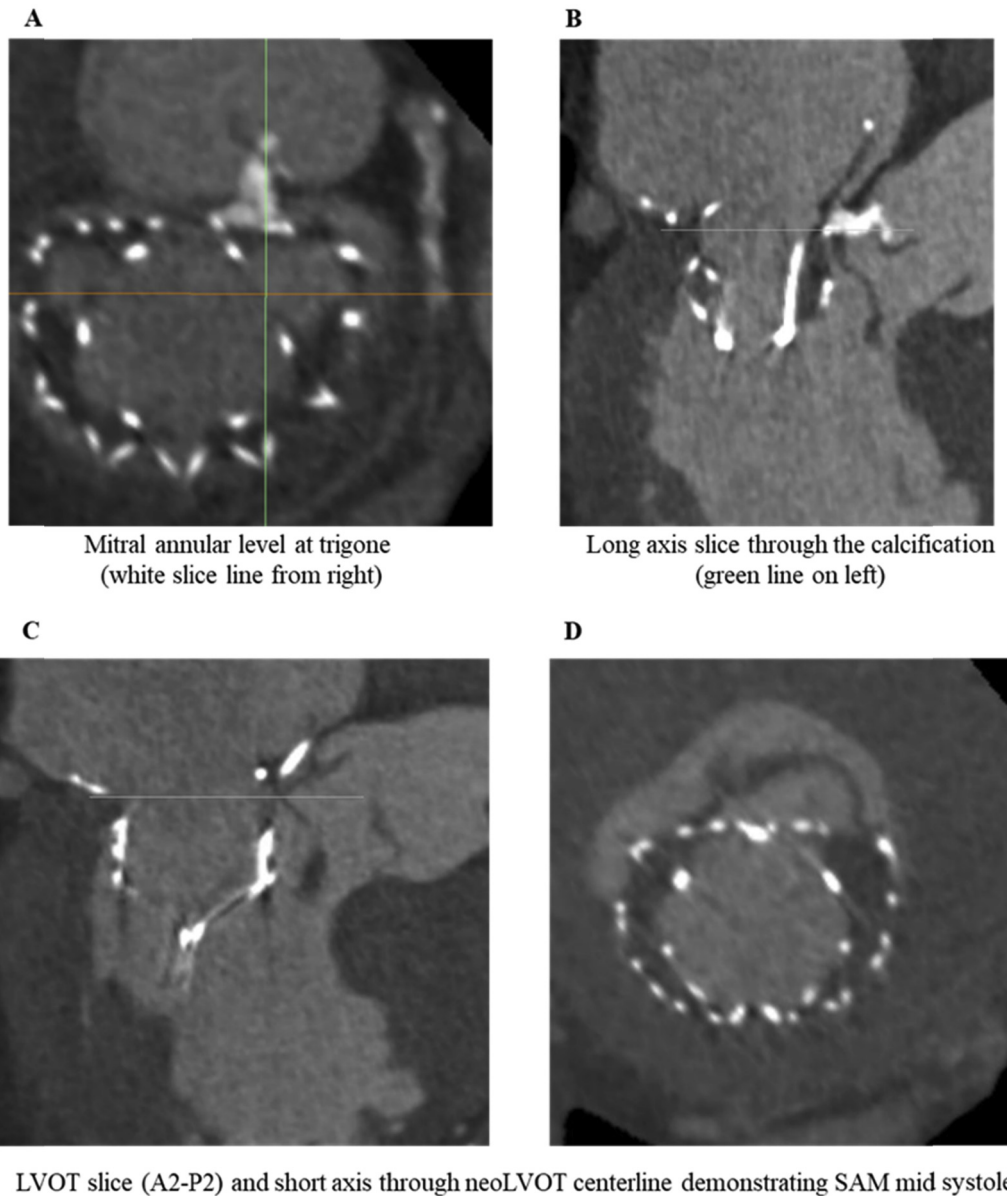


Figure 6 Postprocedural multislice computed tomography. Defined area of calcification noted in A2/A3 region of mitral valve, approximating to the D-shaped portion of the Tendyne device (**A**). SAM (**B–D**) with contrast connection in conduit between left ventricle and left atrium.

the abnormal channel and associated high-velocity jets across a nonendothelialized surface cause intravascular hemolytic anemia, which may be clinically subacute⁶ or result in profound hemolysis.¹² Resolution of severe hemolysis has been reported after successful PVL closure.^{13,14}

In the present case, we attempted PVL closure¹⁵ even though we thought this might prove technically difficult because of the unique Tendyne device shape (we believed that this was a lower risk procedure than TAVR). Although challenging, we successfully placed a guidewire and catheter across the defect with deployment of the closure device with the aid of continuous real-time 3D TEE.

However, placement of the Amplatzer device resulted in lifting of the Tendyne, with worsening of the PVL. This may be because specific closure devices for PVL following Tendyne TMVR have yet to be developed. We went on to deploy a TAVR device low in the LVOT, which successfully reduced the high-velocity jet across the PVL by relieving LVOT obstruction caused by SAM of the AMVL. Our group had previous experience of treating SAM causing LVOT obstruction in a patient who had undergone Tendyne TMVR by inserting a cardiopulmonary stent into the LVOT,⁶ but that patient had no aortic valve disease, and we have not identified a case in the literature in which LVOT obstruction

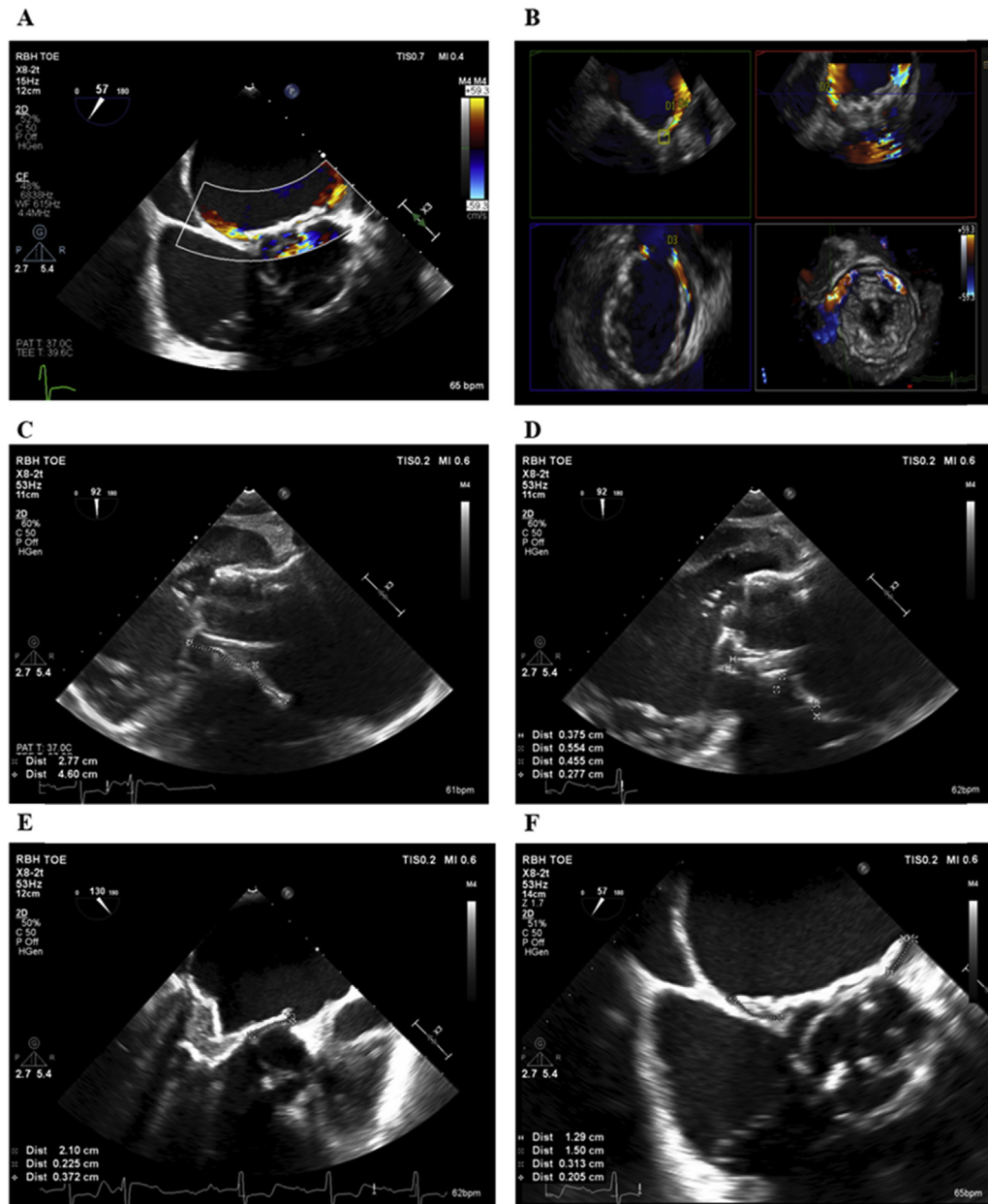


Figure 7 Real-time 3D TEE to locate and size the PVLs. Two jets were identified, one anterolateral and one anteromedial (**A**, **B**). The anterolateral defect measured 2.77 mm in length (**C**), with the tunnel width between device and annulus/left atrial wall device 2.77 to 5.54 mm (**D**). The anteromedial defect measured 2.1 cm in length (**E**), with a tunnel width of 0.31 mm (**F**).

after any TMVR device was treated with a TAVR device. Removal of SAM resolved the PVL and reversed the patient’s hemolysis within 24 hours, while relieving the patient of his AR.

CONCLUSIONS

We present a complex case of acute kidney injury after TMVR, caused by massive intravascular hemolysis, driven by the presence of two

small but significant PVLs, and accentuated by SAM of the residual AMVL. The heart team required cardiologists, cardiac surgeons, and cardiac imagers experienced in TMVR therapy, transseptal puncture, PVL closure, and TAVR therapy. Successful placement of a transcatheter aortic valve device removed SAM of the AMVL and obliterated the high-velocity jets across the PVLs, which in turn resolved the drive for severe intravascular hemolysis and resulted in recovery of our patient’s renal function.

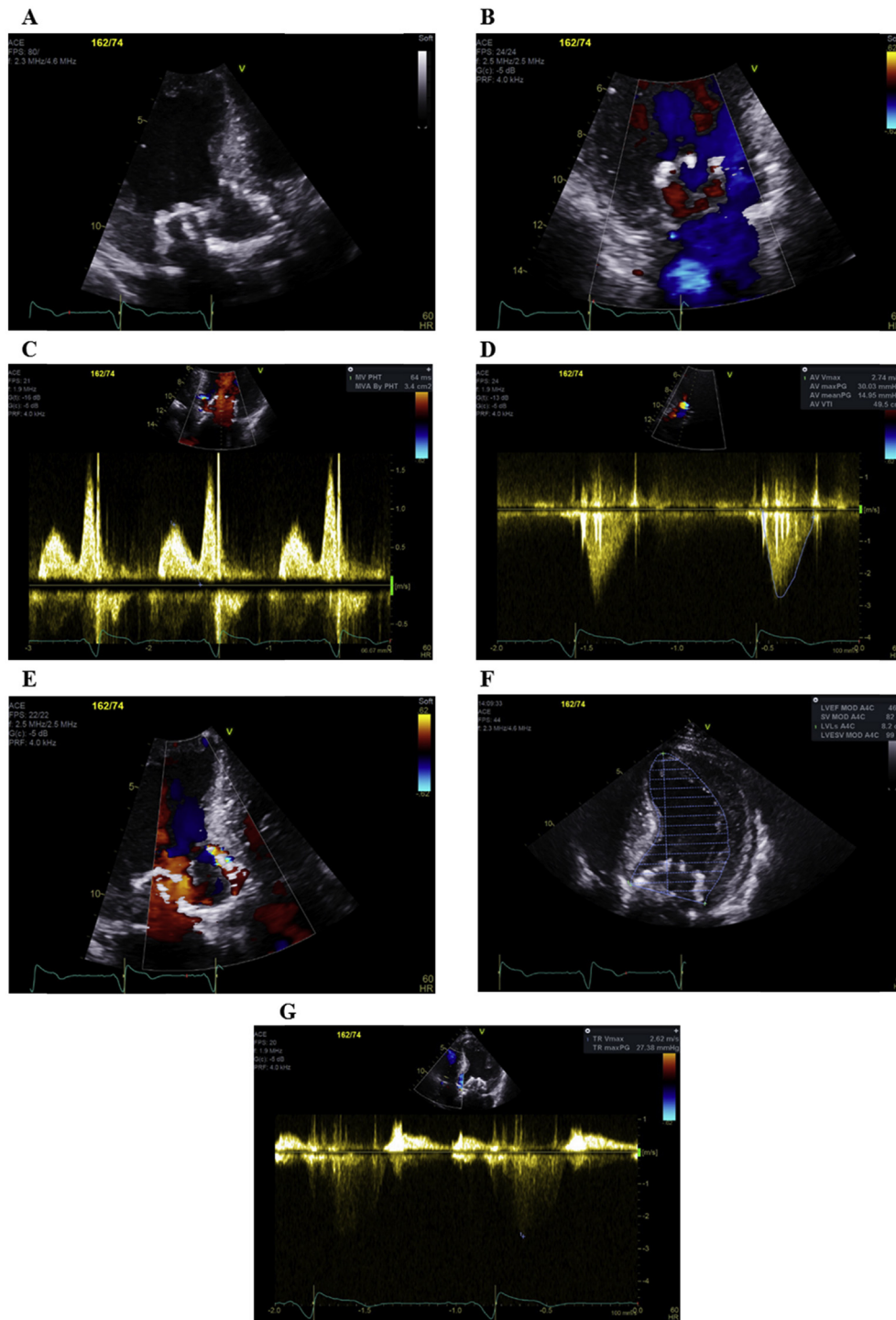


Figure 8 On TTE, the Tendyne TMVR was well seated (**A**) with no PVL (**B**) or mitral stenosis (**C**), the TAVR device was well-seated (**A**) with a peak gradient of 30 mm Hg (**D**) and mild to moderate PVL (**E**), LV ejection fraction of 48% (**F**), and right ventricular systolic pressure of 27 mm Hg (**G**).

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SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.case.2019.02.005>.

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