Unusual Cause of Left Ventricular Outflow Tract Obstruction Following Transcatheter Mitral Valve-in-Ring Replacement

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INTRODUCTION

Left ventricular outflow tract (LVOT) obstruction after transcatheter mitral valve replacement (TMVR) is a potentially catastrophic complication with a reported incidence of 12.5% during valve-in-valve procedures.[1] This obstruction is typically caused by either the valve assembly itself or, less commonly, by anterior displacement of the anterior mitral valve leaflet (AMVL).[2] In this case report we present a case of LVOT obstruction uniquely caused by anterior displacement of the anterolateral papillary muscle into the LVOT, causing a fixed outlet obstruction. We detail the successful management of this complication and explain the unique role transesophageal echocardiography (TEE) played in both diagnosis and guiding treatment.

This patient's Health Insurance Portability and Accountability Act authorization form was obtained and completed.

CASE PRESENTATION

A 71-year-old woman who had previously undergone mitral valve (MV) repair with a 28-mm Carpentier-Edwards Physio II annuloplasty ring (Edwards Lifesciences, Irvine, CA) presented with symptomatic severe mitral stenosis (MS; Figure 1, Video 1). At the time of her MV repair, she also underwent concomitant aortic valve replacement and tricuspid valve repair. The patient's other comorbidities included diabetes mellitus, chronic obstructive pulmonary disease with intermittent use of home oxygen, and atrial fibrillation. Because of the multiple comorbidities she was deemed prohibitively high risk for open heart surgery to treat her MS and was subsequently referred to the structural heart team for possible percutaneous TMVR with a tissue heart valve (THV) in the mitral position. After thorough preoperative medical optimization, the patient was evaluated to determine suitability for the procedure with high-resolution computed tomography (Figure 2). This imaging modality is used to identify the optimal landing zone and virtually assess the dimensions of the neo-LVOT after THV deployment. In this patient the predicted neo-LVOT was measured at 118 mm^2, and although the patient was thus deemed to be at risk for LVOT obstruction, the decision was taken to proceed with the procedure, taking care to ensure the THV was deployed as atrially as possible in order to reduce the risk for LVOT obstruction.[3]

After induction of general anesthesia, TEE was performed, which confirmed the diagnosis of severe LVOT obstruction. Under transesophageal echocardiographic and fluoroscopic guidance, a transseptal puncture was performed in the inferoposterior portion of the intra-atrial septum. Under real-time three-dimensional transesophageal echocardiographic and fluoroscopic guidance, an Edwards SAPIEN

VIDEO HIGHLIGHTS

Video 1: Midesophageal four-chamber view focused on the MV showing **(A)** thickened MV leaflets with restricted leaflet motion and **(B)** flow acceleration proximal to the MV orifice with the formation of a proximal isovelocity surface area shell signifying mitral stenosis. **(C)** 3-dimensional en-face view of the MV showing an annuloplasty ring along with thickened MV leaflets with limited excursion.

Video 2: Three-dimensional TEE of the MV viewed from the left atrial perspective in the "en face" or "surgeon's view" showing **(A)** deployment of the SAPIEN valve in the mitral position and **(B)** deployed SAPIEN valve in the mitral position showing all three leaflets opening well.

Video 3: Midesophageal view of the deployed SAPIEN valve in the mitral position showing **(A)** well-anchored valve with normal leaflet motion and **(B)** no valvular or paravalvular leak when interrogated with color flow Doppler.

Video 4: **(A)** Midesophageal and **(B)** transgastric long-axis views showing turbulent flow in the neo-LVOT due to anterior displacement of the anterolateral papillary muscle causing a fixed outlet obstruction.

Video 5: Midesophageal long-axis view showing guidewire and balloon placed across the neo-LVOT and dilation of the balloon during rapid ventricular pacing.

Video 6: Transthoracic echocardiographic images taken after ASA. **(A)** Parasternal long-axis view showing the anteriorly displaced anterolateral papillary muscle in continuity with the strut of the SAPIEN valve that was previously deployed in the mitral position. The size of the neo-LVOT has increased in size because of septal necrosis from the ASA. **(B)** Color flow Doppler across the LVOT in the parasternal long-axis view showing turbulent flow in the LVOT during systole.

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Keywords: Mitral stenosis, Structural heart disease, Transcatheter mitral valve replacement, Left ventricular outflow tract obstruction, Echocardiography, Alcohol septal ablation, Balloon dilation

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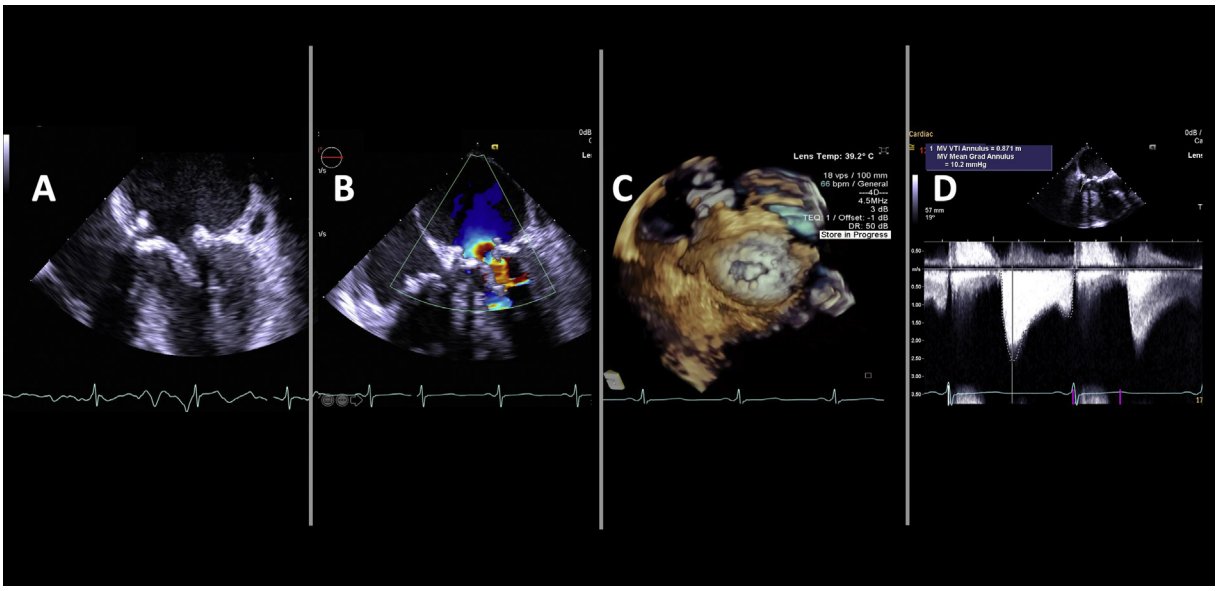

Figure 1 Midesophageal four-chamber view focused on the MV showing **(A)** previous annuloplasty ring with thick leaflets and restricted motion, **(B)** formation of a proximal isovelocity surface area shell signifying flow acceleration proximal to the orifice of the MV, and **(C)** transmitral velocities through the MV showing a high mean gradient consistent with severe MV stenosis.

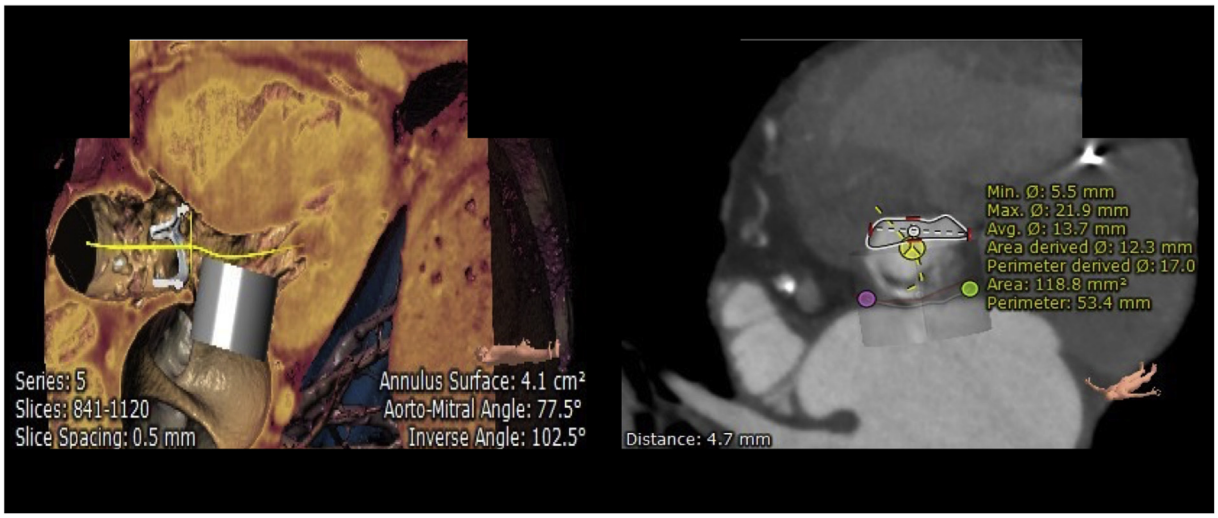

Figure 2 Predicting neo-LVOT obstruction on postcontrast cardiac computed tomography. A simulated 29-mm valve is inserted in the mitral position and allows the operator to evaluate the neo-LVOT for potential obstruction before the procedure while also optimizing the landing zone for the THV.

3 THV (Edwards Lifesciences) was then deployed in the mitral position, and successful deployment was confirmed on TEE (Video 2).

Postdeployment imaging showed a THV that was well seated in the mitral position, with no valvular or paravalvular leaks and a normal transmitral mean gradient (Figure 3, Video 3). However, on further imaging, TEE also revealed the presence of turbulent flow in the outflow tract, as seen on color flow Doppler, and a high LVOT peak gradient of 136 mm Hg when measured using continuous-wave Doppler in the deep transgastric window (Figure 4). TEE demonstrated the cause of this obstruction to be the anterolateral papillary muscle that was displaced into the LVOT, resulting in a fixed outflow obstruction (Figure 5, Video 4). This papillary muscle displacement is thought to have occurred because of increased tension on the chordae tendineae from displacement of AMVL while deploying the THV.

After optimization of the patient's hemodynamic parameters through preload, afterload, and heart rate augmentation, her LVOT gradient improved from 136 mm Hg to a still elevated 45 mm Hg, with stable hemodynamics (Figure 6). After a multidisciplinary team discussion, the decision was made to refrain from surgical intervention because of this patient's multiple comorbidities and high surgical risk, and she was subsequently extubated in the operating room. The tentative plan was to medically manage the patient as best as possible and to provide minimally invasive interventions only if she were to become symptomatic.

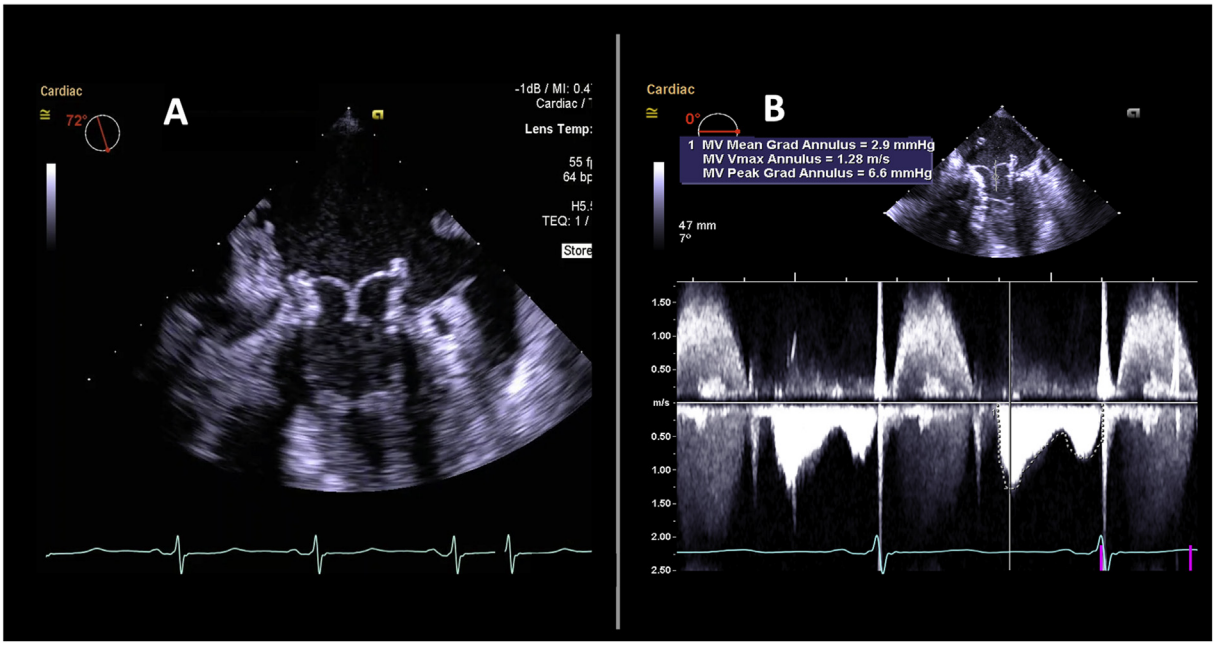

Figure 3 Midesophageal four-chamber view focused on the MV showing **(A)** SAPIEN valve deployed in the mitral position and **(B)** transmitral flow through the newly deployed SAPIEN valve showing normal transmitral gradients.

In the initial postoperative period, the patient made good clinical progress, but symptoms soon developed with even mild exertion because of her LVOT obstruction, despite maximal medical therapy. To attempt to reduce the gradient in the LVOT and alleviate some of her symptoms, the decision was made to perform septal reduction with alcohol septal ablation (ASA) of the septal perforators and also to perform TEE-guided percutaneous balloon dilation of the neo-LVOT that was created between the displaced papillary muscle and the wall of the LVOT.

Four weeks after her initial procedure, the patient was brought back to the operating room for these interventions. Following induction of general anesthesia, TEE was performed, which confirmed turbulent flow in the LVOT with an outflow tract gradient measured at 62 mm Hg (Figure 7). Using transesophageal echocardiographic

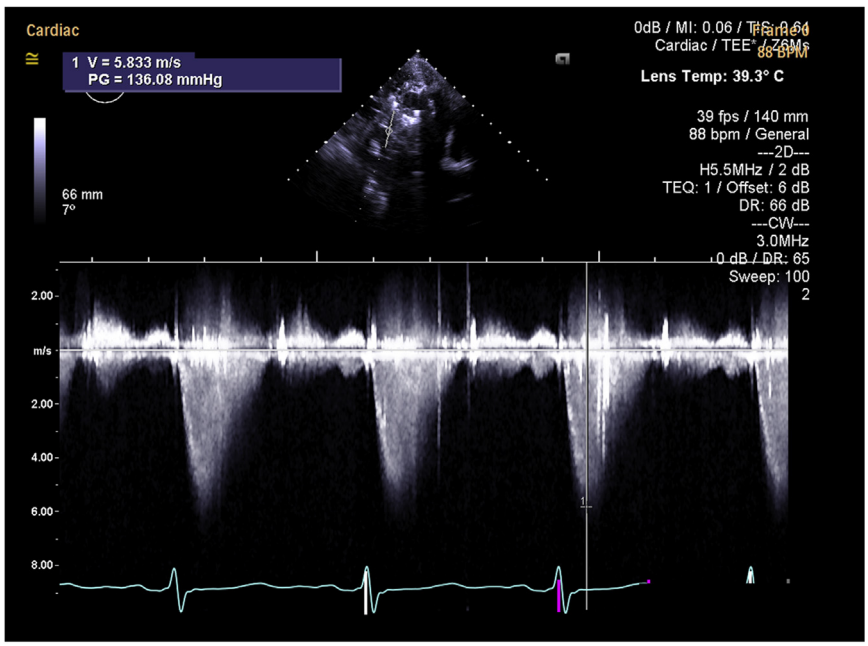

Figure 4 Continuous-wave Doppler through the LVOT in the deep transgastric window showing high peak velocities signifying outlet obstruction.

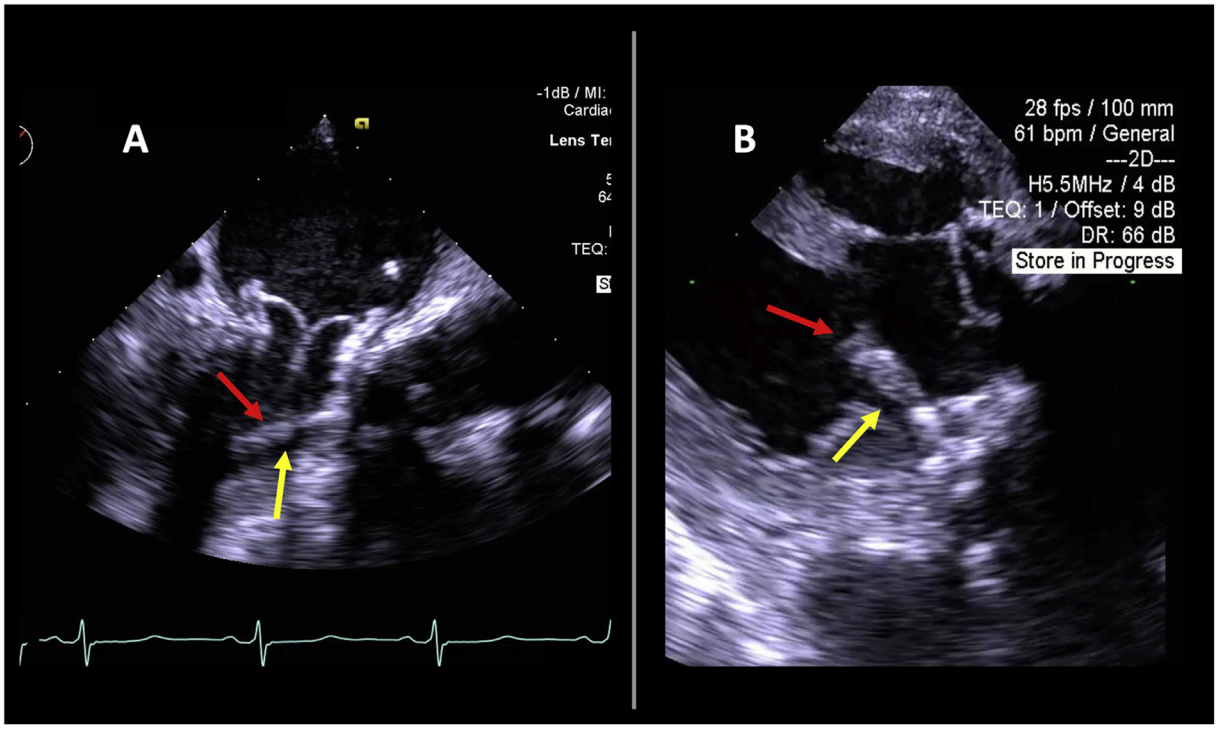

Figure 5 **(A)** Midesophageal and **(B)** transgastric long-axis views showing anterior displacement of the anterolateral papillary muscle (*red arrow*) and the newly created narrowed neo-LVOT (*yellow arrow*).

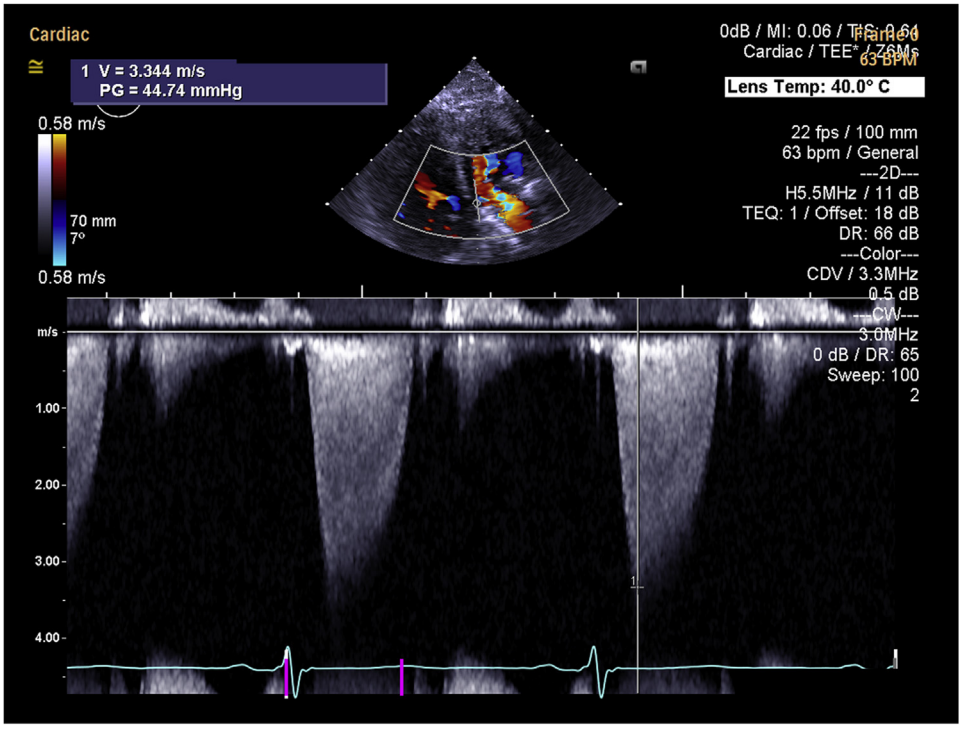

Figure 6 Continuous-wave Doppler through the LVOT in the deep transgastric window showing a reduction in the peak velocity after preload, afterload, and heart rate augmentation.

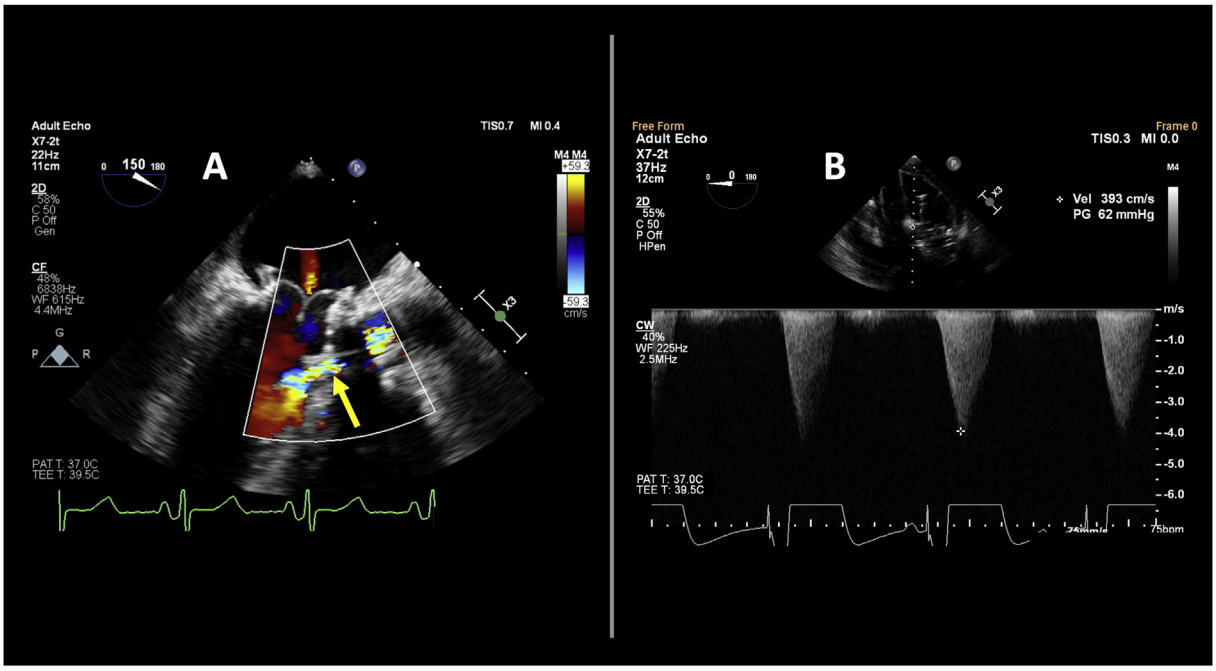

Figure 7 **(A)** Midesophageal long-axis view with color flow Doppler showing turbulent flow in the neo-LVOT (*yellow arrow*) and **(B)** continuous-wave Doppler through the LVOT in the deep transgastric window showing high peak velocities.

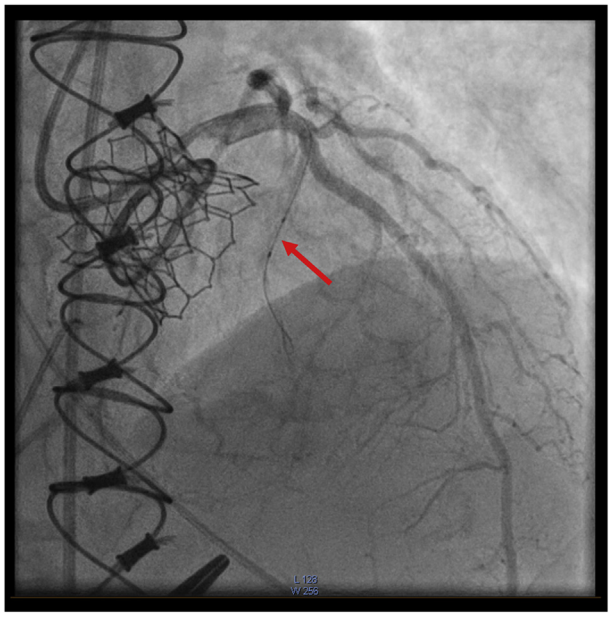

Figure 8 Fluoroscopic image of guidewire in first septal perforator to allow delivery of ethanol into the first septal artery.

guidance with intravenous echocardiographic contrast and fluoroscopic guidance, septal 1 was chosen for ablation of the basal septum. After confirming complete isolation of septal 1 from the left anterior descending coronary artery with angiography, 2 ml ethanol was slowly injected into the septal 1 artery (Figure 8). After septal ablation was performed, we proceeded to balloon dilation of the neo-LVOT. The aortic valve was crossed with a guidewire, and then a Charger balloon (Boston Scientific, Marlborough, MA) was delivered across the neo-LVOT. Short inflations were performed at 10 atm during rapid pacing at 180 beats/min (Video 5). Initial results after dilation of the neo-LVOT showed a reduction of the outflow gradient down to 49 mm Hg (Figure 9). The patient was extubated after these procedures and made a good postoperative recovery with significant symptom alleviation. She was subsequently discharged to a rehabilitation facility, and follow-up transthoracic echocardiography after the effect of ASA had taken place demonstrated further decrease of the LVOT gradient down to a peak gradient of 34 mm Hg, with an absence of symptoms on mild exertion (Figure 10, Video 6).

DISCUSSION

Percutaneous TMVR with a THV, such as the Edwards SAPIEN 3, is growing in popularity as a viable option for treatment of patients with severe symptomatic MS who are otherwise deemed prohibitively high risk for traditional surgical intervention.[4] TMVR may be performed in patients with MS because of severe mitral annular calcification (valve–in–mitral annular calcification) or stenotic prosthetic valve (valve-in-valve) or in patients with annuloplasty rings and stenotic native valvular leaflets (valve-in-ring), as was the situation in the case presented here. The literature to date suggests that patients who undergo valve-in-valve procedures seem to have a higher success rate and better outcomes, but good success rates and outcomes have also been shown in patients who undergo valve-in-ring and also valve–in–mitral annular calcification procedures.[5-7] Although TMVR is a good alternative to surgery in certain patient subsets, current-generation THVs are not designed for mitral position implantation, because of intrinsic geometry that makes them suboptimal, leading to the risk for LVOT obstruction, as a result of either the valvular apparatus itself or, less commonly, displacement of the AMVL.[2] LVOT obstruction is a potentially catastrophic complication with prevention primarily through appropriate patient selection,

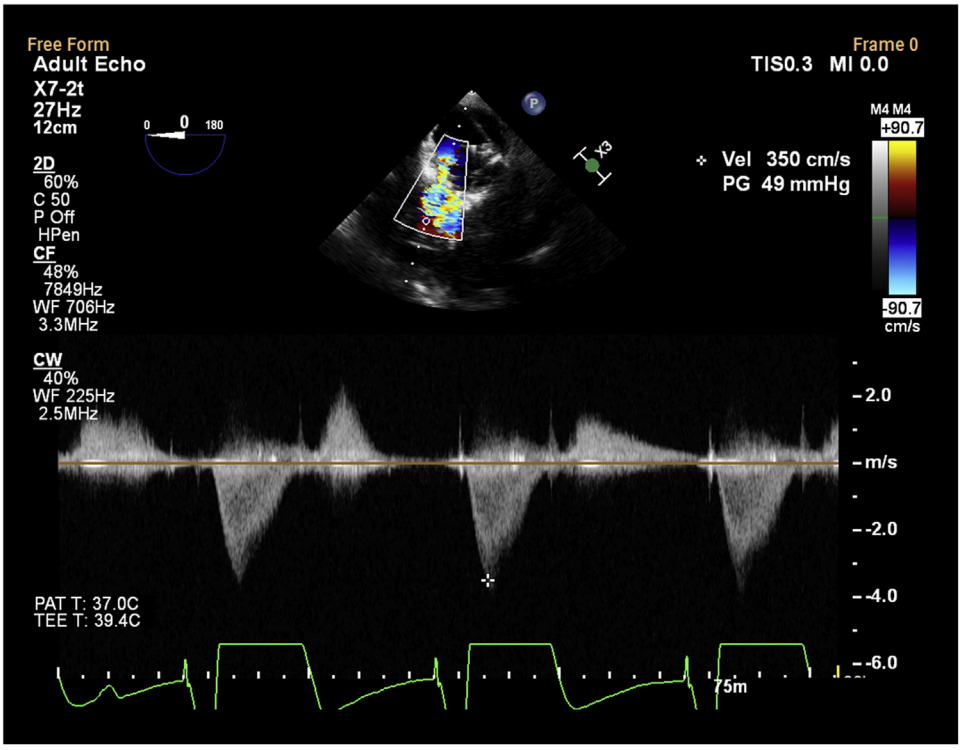

Figure 9 Continuous-wave Doppler through the LVOT in the deep transgastric window showing a reduction in the peak velocity after balloon dilation of the neo-LVOT and ASA.

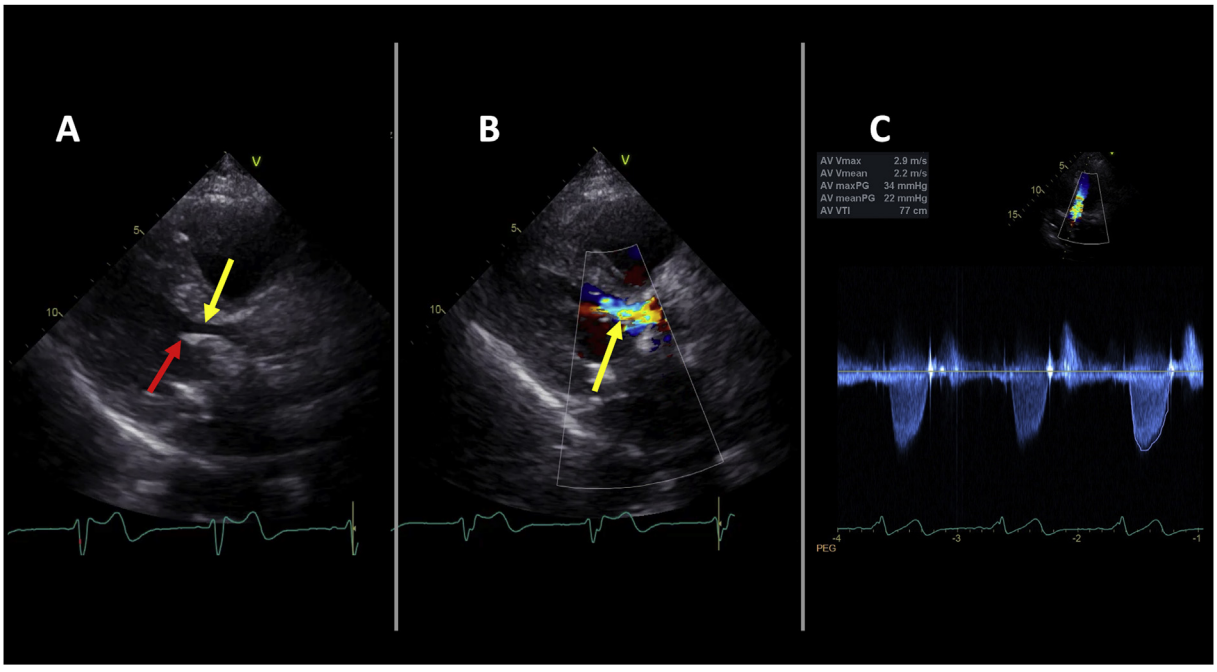

Figure 10 Transthoracic echocardiographic images taken after ASA. **(A)** Parasternal long-axis view showing the anteriorly displaced anterolateral papillary muscle in continuity with the strut of the SAPIEN valve that was previously deployed in the mitral position. The size of the neo-LVOT has increased in size because of septal necrosis from the ASA. **(B)** Color flow Doppler across the LVOT in the parasternal long-axis view showing turbulent flow in the LVOT during systole. **(C)** Continuous-wave Doppler across the LVOT and aortic valve showing elevated gradients but significant improvement from previous.

preoperative planning, and certain prophylactic interventions.[8] All patients should undergo extensive preoperative planning to reduce the chance of LVOT obstruction. Currently, the gold standard is high-resolution cardiac computed tomography with virtual implantation of a THV to optimize the appropriate landing zone. Patients found to be at risk for LVOT obstruction as defined by a predicted neo-LVOT area $<$ 189.4 mm^2 may be deemed unsuitable for TMVR or may undergo preprocedural interventions such as ASA or the electrosurgical LAMPOON (laceration of the anterior mitral leaflet to prevent outflow obstruction) technique, which have both been shown to reduce the risk for LVOT obstruction.[9-11] The use of ASA as either a bailout or a preemptive strategy to reduce the risk for LVOT obstruction has being previously described, with good success in both instances.[9,10] Although ASA is an option to reduce the risk for LVOT obstruction, there are also limitations associated with this procedure. Such limitations include the need for an adequate septal perforator artery, risk for an iatrogenic ventricular septal defect, conduction abnormalities requiring pacemaker insertion, and the need to allow time for myocardial remodeling to occur after ASA and before the proposed TMVR procedure. The LAMPOON technique is another intervention that can be used at the time of TMVR to reduce the risk for LVOT obstruction.[8,11] This procedure is performed percutaneously before THV deployment and uses electrocautery to split the AMVL. After laceration of the AMVL, the THV is then deployed and the AMVL is splayed and subsequently parts away from the LVOT. Blood is then able to flow through the open cells of the THV frame in the LVOT, which may have otherwise been prevented from doing so by the AMVL. Although this technique has been shown to be effective in preventing LVOT obstruction due to displacement of the AMVL, it is not effective in overcoming obstruction from the THV skirt.

CONCLUSION

Despite optimal planning and patient optimization, unforeseen complications can occasionally occur, as in this case with a high-risk patient developing the rare complication of anterior displacement of the anterolateral papillary muscle, resulting in LVOT obstruction. Although the displaced papillary muscle resulted in the majority of the LVOT obstruction seen in this patient, it is very likely that the AMVL and THV assembly also contributed to some degree. Although it was not performed at the time of the initial procedure, this patient may have benefited from the LAMPOON technique at the time of TMVR. However, although the degree of obstruction may not have been as severe, there still likely would have been some degree of LVOT obstruction from the displaced papillary muscle requiring intervention. This patient presented us with a dilemma on how to best treat this complication while mitigating further surgical risk. Our strategy to use a combination of two minimally invasive interventions, ASA and balloon dilation of the neo-LVOT, to avoid open surgery was a novel approach that was ultimately successful. The balloon dilation we performed likely temporarily reduced the LVOT gradient and alleviated symptoms, and this effect was subsequently maintained as the septal necrosis occurred after ASA. As structural heart teams take on more complex patients for interventions such as TMVR, we are likely to experience unforeseen complications such as we encountered in this case, and we will need to tailor therapeutic interventions that are patient specific to treat those complications.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2021.02.002.

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