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Valve-in-valve transcatheter mitral valve replacement procedure in prosthetic valve stenosis

Mridul Bansal^a, Aryan Mehta^a, David X. Zhao^a, Saraschandra Vallabhajosyula^{b,*}

^aSection of Cardiovascular Medicine, Department of Medicine, Wake Forest University School of Medicine, Winston-Salem, North Carolina, USA

^bDepartment of Implementation Science, Division of Public Health Sciences, Wake Forest University School of Medicine, Winston-Salem, North Carolina, USA

Abstract

A patient presented with acute respiratory failure and shock due to severe prosthetic mitral valve stenosis. A valve-in-valve transcatheter mitral valve replacement procedure was performed via the transeptal approach due to his high-risk presentation with good results.

Keywords

Interventional cardiology; Cardiovascular surgery; Mitral valve interventions; Structural heart disease; Transcatheter mitral valve replacement

1. Introduction

Bioprosthetic valves used for valve replacement surgery have a distinct advantage over mechanical valves, in that they generally don't require lifelong anticoagulation, but however, are more prone to structural valve degradation leading to valve failure.¹ Although the gold standard treatment for prosthetic valve stenosis remains redo valve replacement surgery, current American Heart Association (AHA)/American College of Cardiology (ACC) joint committee guidelines recommend the use of percutaneous interventions in the group of patients considered high risk for surgery.² We present such a case, in which the patient had symptomatic prosthetic mitral stenosis (MS). He had multiple comorbidities therefore needed an urgent novel valve-in-valve (ViV) transcatheter mitral valve replacement (TMVR) procedure.

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*Corresponding author. Section of Cardiovascular Medicine, Department of Medicine Wake Forest University School of Medicine, 306 Westwood Avenue, Suite 401, High Point, North Carolina, 27262, USA. svallabh@wakehealth.edu (S. Vallabhajosyula).

Author contributions

Study design, literature review, statistical analysis: MB, AM, SV.

Data management, data analysis, drafting manuscript: MB, AM, DXZ, SV.

Access to data: MB, AM, DXZ, SV.

Manuscript revision, intellectual revisions, mentorship: DXZ, SV.

Final approval: MB, AM, DXZ, SV.

Declaration of competing interest

None.

2. Case presentation

A male in his 60's presented with shortness of breath for two weeks and initial room air oxygen saturation of 80%. He had prior severe rheumatic mitral stenosis which was treated surgically 7 years ago with a 27 mm Carpentier-Edwards Perimount bioprosthetic mitral valve implant and concomitant ligation of left atrial appendage. His other vitals were unremarkable and on auscultation he had a grade 3/6 mid-diastolic murmur was in the mitral area. Investigations showed elevated levels of leukocytes ($14,000 \times 10^9/L$), serum lactate (4.8 mmol/l), creatinine (1.6 mg/dl), transaminases and B-type natriuretic peptide (2233 pg/ml). Chest imaging revealed right middle lobe consolidation with pleural effusion, cardiomegaly and no evidence of pulmonary embolism. The patient was evaluated for undifferentiated shock of either septic (pneumonia) or circulatory (heart failure) etiology. The patient was started on empiric broad-spectrum antibiotics. Transthoracic echocardiogram (TTE) at admission demonstrated severe right atrial and right ventricular dilatation and enlargement, severe tricuspid regurgitation, severely elevated right ventricular systolic pressure (106 mm Hg) severe prosthetic mitral valve stenosis (mean gradient 26 mm Hg; heart rate 71 bpm), and left atrial enlargement (Fig. 1A–D). For comparison, a TTE performed elsewhere three years ago, showed normal prosthetic mitral valve functioning with a mean mitral valve gradient of 6 mmHg.

However, after a few hours the patient developed worsening hypoxemic respiratory failure and needed emergent endotracheal intubation and mechanical ventilation. Despite three days of broad-spectrum antimicrobial treatment, there was minimal improvement in the clinical status. Over the course of 24–48 hours the cardiogenic shock became more apparent. Further examination with a transesophageal echocardiography (TEE) confirmed the prosthetic mitral stenosis to be severe (mean gradient 22 mmHg, heart rate 53bpm) with presence of structural damage to mitral valve (Fig. 2A–D). However, on hospital day six, he developed flash pulmonary edema and he was deemed a high-risk surgical candidate for redo valve replacement surgery due to frailty.

A decision was made to proceed with ViV TMVR. A transeptal puncture was performed using TEE and fluoroscopic guidance, A Bayliss sheath was used to cross the interatrial septum and exchanged for an Edwards-Sapien E-sheath. After balloon atrial septostomy, an Agilis catheter was used to cross the mitral valve. A 26 mm Sapien 3 (S3) valve (Edwards Lifesciences, Irvine, California) was deployed under rapid pacing. TEE showed significant left-to-right shunt through the iatrogenic atrial septal defect, which was subsequently closed with a 28–32 mm Amplatzer septal occluder device (Abbott Vascular, Santa Clara) (Fig. 3A–C). He was successfully extubated on post-operative day one. On hospital day 23 the patient was discharged in good clinical condition. At 1-month post-hospitalization follow-up, on TTE with the mitral prosthetic valve was well functioning with trace paravalvular leak, reduced right ventricular size, and improved function. The transmitral gradient was 6 mmHg (71 bpm).

3. Discussion

An acquired intrinsic bioprosthetic valve abnormality known as structural valve degeneration involves the deterioration of the leaflets or supporting structures resulting in thickening, calcification, tearing, or disruption of the prosthetic valve materials, culminating in hemodynamic dysfunction resulting in stenosis or regurgitation of the valve.¹ Historically, the treatment of choice for a prosthetic stenotic valve has been valve replacement surgery. However, repeat valve surgery, especially in an acute and inpatient setting, has been associated with an increased risk of complications, mortality, and resource utilization.³ Currently, the recommendations both European and American societies have included the use of transcatheter percutaneous intervention approaches in patients who are high-risk surgical candidates.^{2,4}

Data comparing the difference between ViV TMVR, and surgical MVR is limited. In one such study, it was noted that ViV TMVR is associated with lower mortality, periprocedural morbidity, and resource utilization compared with patients who had a redo SMVR (Surgical Mitral Valve Replacement).⁵ In the same registry, it was observed that 2.7 % of patients with surgical MVR required tricuspid valve intervention which is not currently feasible using transcatheter approaches. The prognostic role of concomitant tricuspid pathology on patients who underwent redo surgery mitral valve is well established. Thus, each approach should be individualized depending upon concomitant pathologies.

Even though the percutaneous approach is feasible, it may be associated with significant complications such as left ventricular perforation and left ventricular outlet tract (LVOT) obstruction. Preprocedural Computed Tomography imaging is recommended to better delineate the anatomy and help in the reduction of incidence of LVOT.⁶ Other challenges associated with TMVR include paravalvular leaks, prosthesis-patient mismatch, valve thrombosis, and limited durability of prostheses.⁷ Lastly, there is limited long term data on the efficacy and durability of ViV TMVR and further data are awaited. In conclusion, in this case, we report a successful urgent ViV TMVR. Appropriate planning, close clinical assessment and multidisciplinary input is important to help in the successful conduct of such procedures.

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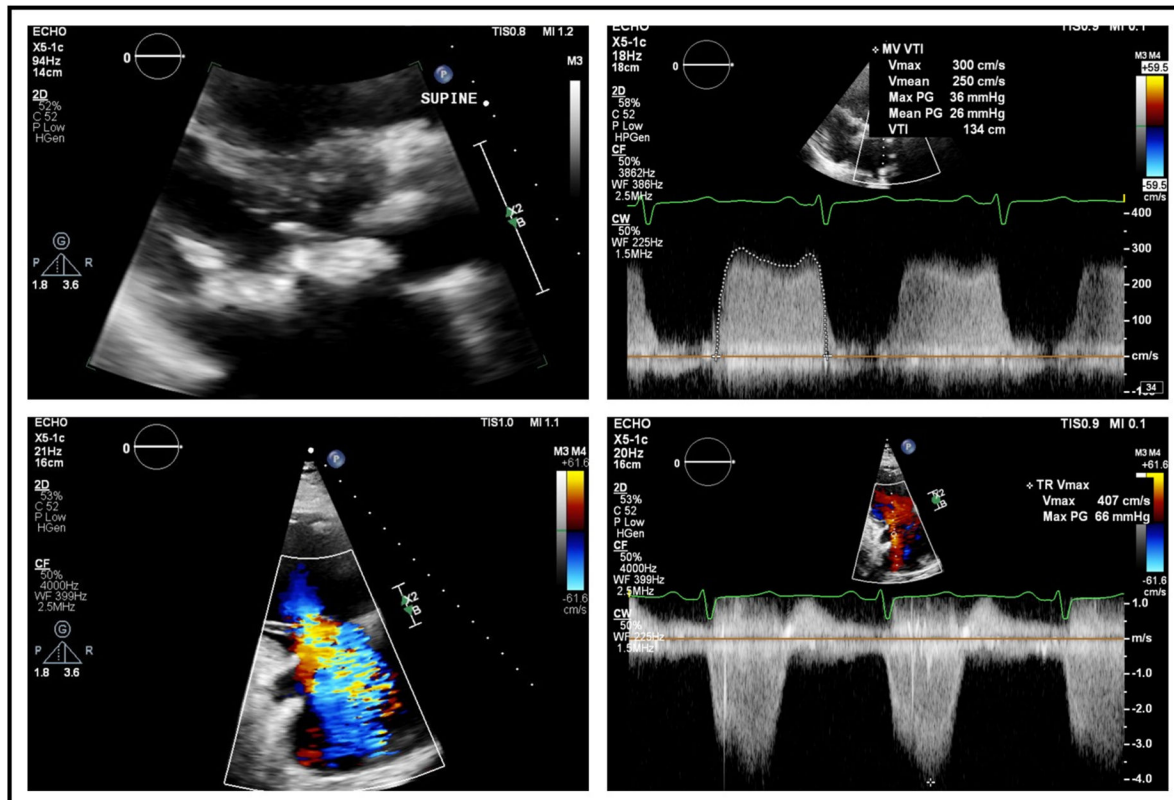


Fig. 1. Transthoracic echocardiogram at presentation demonstrating severe mitral stenosis in parasternal long axis (**1A**) with a 26 mm Hg gradient (**1B**), severe tricuspid regurgitation on right ventricle dedicated view with Doppler waveform (**1C and 1D**).

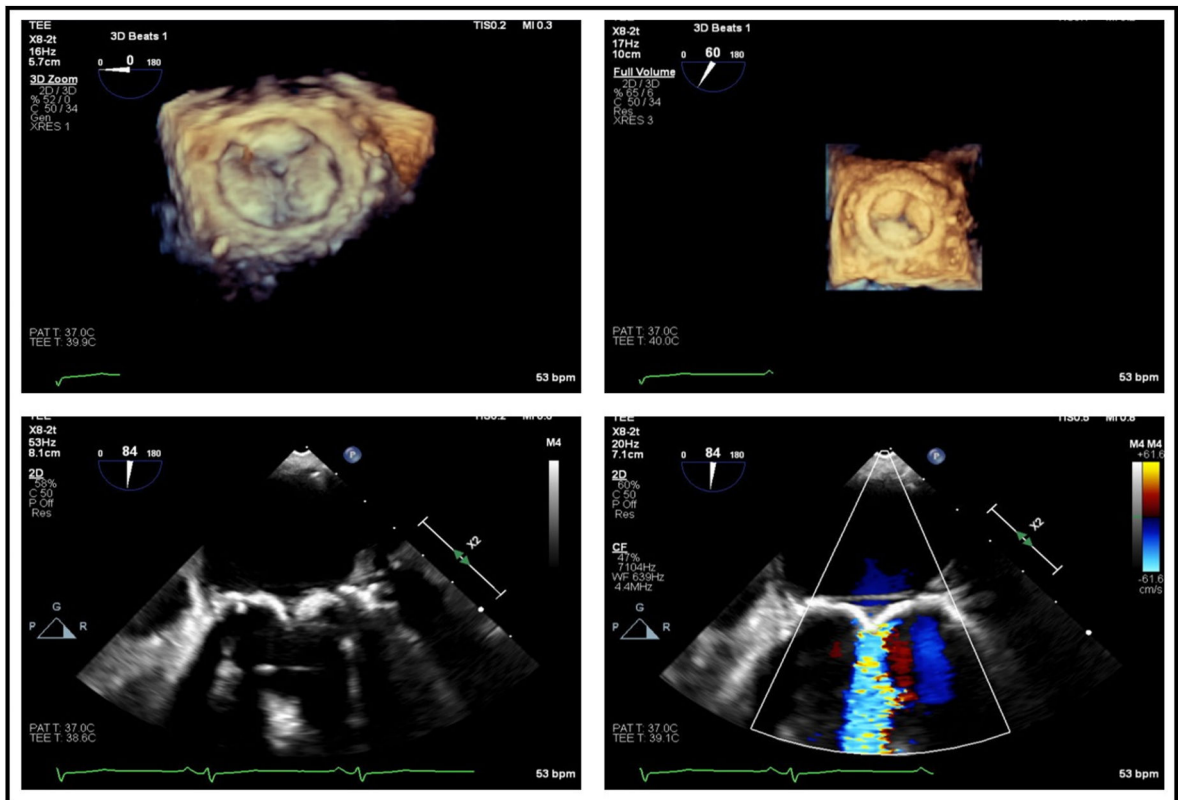


Fig. 2. Transesophageal echocardiogram re-demonstrating severe mitral stenosis in 3-dimensional imaging (2A-B), 2D mid-esophageal view (2C) with increased transmitral velocity on color Doppler imaging (2D).

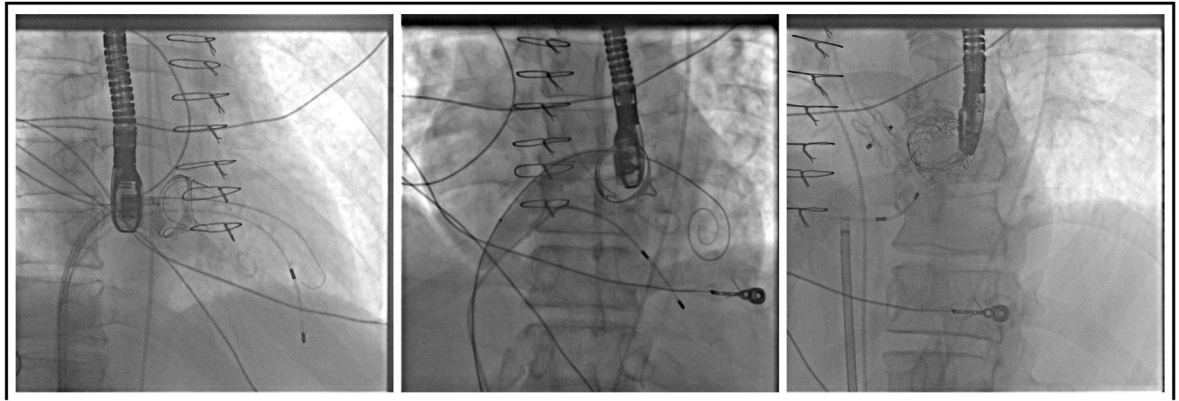


Fig. 3. Transcatheter mitral valve-in-valve replacement procedure demonstrating crossing of prosthetic mitral valve (**3A**), balloon mitral valvuloplasty (**3B**) and final transcatheter valve in-situ with subsequent iatrogenic atrial septal defect closure with Amplatz occluder (**3C**).