

Ventricular Septal Defect and Complete Atrioventricular Block Following Transcatheter Aortic Valve Implantation: A Case Report

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Abstract

Transcatheter aortic valve implantation (TAVI) is an established alternative to surgical aortic valve replacement for severe aortic stenosis (AS) in high-risk patients, yet rare, potentially life-threatening complications still occur. We describe a 75-year-old man with severe symptomatic AS who underwent TAVI using a balloon-expandable Myval prosthesis; the procedure was complicated by complete atrioventricular block requiring permanent pacemaker implantation. At 1-month follow-up, he presented with dyspnea. Transthoracic echocardiography demonstrated a newly developed perimembranous ventricular septal defect (VSD) adjacent to the prosthetic valve, resulting in a significant left-to-right shunt. This case underscores the need for meticulous pre-procedural assessment of the distribution of calcification, careful procedural planning and technique, and post-procedural vigilance for both conduction disturbances and rare structural complications, such as VSD, following TAVI.

Keywords: TAVI, aortic stenosis, ventricular septal defect, atrioventricular block, complication

INTRODUCTION

Aortic stenosis (AS) is the leading valvular disorder in older adults, affecting roughly 2-4% of people over 75 years of age.^[1] Surgical aortic valve replacement has traditionally been regarded as the standard therapy. Nevertheless, many candidates are unsuitable because of advanced age, frailty, or significant comorbid conditions. For this cohort, transcatheter aortic valve implantation (TAVI) has emerged as a less invasive and highly effective alternative.

Although TAVI has transformed care for severe AS, procedure-related complications still occur, including vascular trauma, paravalvular regurgitation, coronary obstruction, conduction system disturbances, and, on rare occasions, a ventricular septal defect (VSD). Post-TAVI VSD is a rare complication;

large contemporary series report incidences well below 0.5% (e.g., ~0.37%),^[2] whereas higher rates (up to ~1.5%) have been described in smaller cohorts with systematic echocardiographic screening that mainly detects small, restrictive defects.^[3] We present a rare case of combined complete atrioventricular (AV) block and perimembranous VSD following the implantation of a balloon-expandable valve.

CASE REPORT

A 75-year-old man with New York Heart Association class IV symptoms—dyspnea and angina at rest—was referred for severe AS. His medical history included hypertension, diabetes mellitus, and asthma. Baseline transthoracic echocardiography (TTE) showed preserved left ventricular systolic function (ejection fraction of 55%) and severe calcific AS (Figure 1), with an aortic

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valve area of 0.8 cm^2 , a mean gradient of 45 mmHg , and a peak gradient of 80 mmHg . Coronary angiography demonstrated normal epicardial arteries. With a Society of Thoracic Surgeons score of 5.42%, the Heart Team recommended TAVI.

Multislice cardiac computed tomography (CT) revealed extensive calcification predominantly involving the left coronary cusp. Multislice cardiac CT demonstrated severe asymmetric aortic valve calcification predominantly involving the left coronary cusp, extending toward the left ventricular outflow tract (LVOT) and membranous septum. Calcification severity was reported as an Agatston leaflet calcium score of 3200 or as severe qualitative calcification. CT sizing showed an annular area of 651 mm^2 , a perimeter of 90.4 mm , and coronary heights of 12 mm (left main coronary artery) and 14 mm (right coronary artery), supporting selection of a 29-mm balloon-expandable Myval™ valve. The Myval™ transcatheter heart valve is a balloon-expandable, tri-leaflet bovine pericardial valve mounted on a nickel-cobalt frame with a hexagonal-cell design and a sealing cuff; it is available in conventional, intermediate, and extra-large sizes, facilitating more granular annular sizing. The procedure was performed transfemorally under fluoroscopic guidance using a pre-defined coplanar projection. After pre-dilation with a 25-mm balloon, significant resistance was observed during balloon expansion under rapid pacing; subsequently, a 29-mm balloon-expandable Myval™ (Meril Life Sciences Pvt. Ltd., India) prosthesis was deployed. The final valve position was 2.8 mm ventricular to the annular plane (implantation depth measured on the final angiographic projection), consistent with a neutral/orthotopic implantation strategy. Hemodynamics improved immediately, and the mean transvalvular gradient fell to 10 mmHg . Because of heavy calcification and concern for annular rupture, post-dilation was not undertaken. No resistance or recoil was documented



Figure 1: Calcification seen on the aortic valve

during balloon expansion. No new conduction disturbance was observed during balloon pre-dilation; complete AV block developed immediately after valve deployment and required temporary pacing. Because conduction did not improve, a dual-chamber permanent pacemaker was implanted before discharge (on the second day after TAVI). Pre-discharge TTE with color Doppler interrogation of the interventricular septum (perimembranous region) showed no evidence of a VSD or an interventricular shunt, and no new murmur was noted during the index hospitalization.

At one-month follow-up, he reported worsening dyspnea. Compared with pre-discharge findings (no murmur and no shunt on TTE), a new pansystolic murmur was detected on examination. TTE confirmed normal prosthetic valve function (mean gradient of 10 mmHg), but demonstrated an 8-mm perimembranous VSD adjacent to the prosthetic valve edge (Figure 2A), with color Doppler evidence of a significant left-to-right shunt (Figure 2B) and elevated estimated pulmonary artery (PA) pressures ($62/38 \text{ mmHg}$) (Figure 2C). Estimated right ventricular (RV) systolic pressure was 38 mmHg , with mildly abnormal RV size and systolic function (tricuspid annular plane systolic excursion 15 mm , fractional area change 34%). Right-heart catheterization was planned to quantify shunt severity ($Qp/Qs: 1.9$) and to obtain comprehensive right-sided hemodynamics (right atrium, RV, PA, pulmonary capillary wedge pressure) prior to evaluation for percutaneous versus surgical closure of the VSD.

Written informed consent was obtained from the patient.

DISCUSSION

TAVI may be complicated by a spectrum of adverse events, including vascular and valvular complications, as well as conduction and structural problems. VSD is particularly uncommon but clinically important, with an estimated frequency of 0.37% after TAVI.^[2]

Mechanistically, iatrogenic VSD following TAVI is likely multifactorial. Severe asymmetric calcification can predispose the membranous septum to injury during balloon pre-dilation or valve deployment. Excess mechanical stress may shift calcified nodules into the interventricular septum, provoking both conduction block and mechanical disruption. Balloon pre-/post-dilatation in the presence of bulky, asymmetric annular/LVOT calcium may increase focal mechanical stress and can displace/compress calcific nodules toward adjacent structures, including the membranous septum, providing biological plausibility for septal disruption and VSD.^[3] In line with this, a review of reported post-TAVR VSD cases found that VSDs were more frequently associated with balloon-expandable valves and with the use of pre- or post-dilatation.^[4]

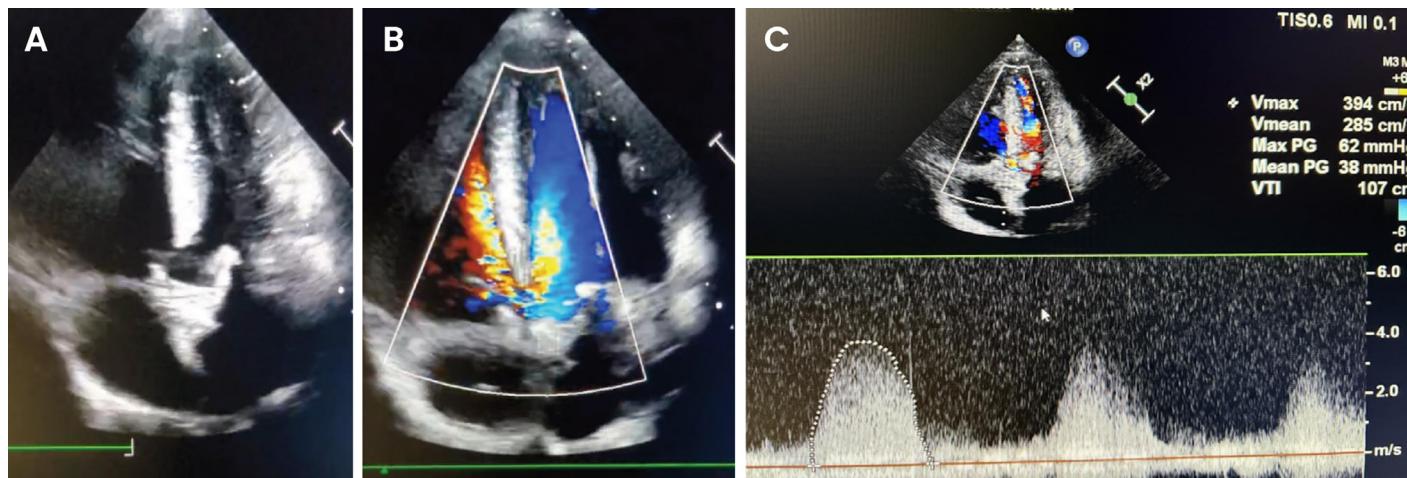


Figure 2: A) 8 mm VSD developing after TAVI, B) VSD shunt from left ventricular to right ventricular, C) 62/38 mmHg gradient formed on the VSD shunt

VSD: Ventricular septal defect, TAVI: Transcatheter aortic valve implantation

Conduction abnormalities are among the most frequent complications of TAVI due to the close anatomical relationship among the aortic annulus, the membranous septum, and the AV node. Situated at the apex of Koch's triangle, adjacent to the membranous septum and subaortic LVOT, the conduction system is vulnerable to compression or trauma during prosthesis expansion, which explains the propensity for AV block in these cases. In the randomized LANDMARK conduction substudy, 30-day new permanent pacemaker implantation rates were similar between Myval and contemporary valves (15% vs. 17%), with complete AV block being the most common indication, supporting the conclusion that clinically relevant conduction injury remains a platform-spanning issue influenced by anatomy, calcification, and deployment mechanics.^[5]

Valve design and deployment mechanics may influence the risk of septal injury. Balloon-expandable valves are deployed by acute balloon inflation, which can generate high instantaneous radial stress within the annulus/LVOT and may displace bulky, asymmetric calcification toward the membranous septum—particularly in the presence of moderate-to-severe LVOT calcification—thereby plausibly increasing the risk of adjacent septal trauma.^[6] Conversely, self-expanding valves expand more gradually and are often repositionable; however, meta-analyses comparing contemporary platforms have reported higher rates of conduction disturbances and permanent pacemaker implantation with some self-expanding systems than with balloon-expandable devices.^[7]

In our patient, severe asymmetric calcification coupled with balloon pre-dilation and prosthesis expansion likely displaced calcium toward the membranous septum, producing immediate

AV block and later culminating in a VSD. The absence of a shunt on pre-discharge echocardiography suggests a delayed or progressive septal injury, which became clinically evident at one month. Clinically, the patient developed dyspnea and a new murmur, and echocardiography confirmed a significant left-to-right shunt. Management of post-TAVI VSD depends on size, hemodynamic burden, and symptoms; small, stable defects can be observed, whereas larger or symptomatic defects may require percutaneous or surgical closure.

CONCLUSION

This case illustrates two rare but consequential post-TAVI complications—complete AV block necessitating permanent pacing and a perimembranous VSD with left-to-right shunting—most likely driven by severe asymmetric calcification and balloon manipulation. Thorough pre-procedural imaging, careful valve sizing, and judicious use of balloon dilatation are essential to reduce these risks, and clinicians should maintain vigilance for new murmurs or unexplained dyspnea during follow-up.

Ethics

Informed Consent: Written informed consent was obtained from the patient.

Footnotes

Authorship Contributions

Surgical and Medical Practices: S.E.Ö., Concept: S.E.Ö., K.K., Design: S.E.Ö., E.A., Data Collection or Processing: S.E.Ö., Literature Search: S.E.Ö., Writing: S.E.Ö., K.K.

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