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Management strategies and possible risk factors for ventricular septal defects after transcatheter aortic valve replacement: Case series from a single center and review of literature[☆]

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SUMMARY

Development of membranous ventricular septal defects (VSD) is a rare complication of transcatheter aortic valve replacements (TAVR), and is recognized using intraoperative and postoperative imaging. We present two cases of this rare but serious complication; one was successfully managed conservatively and the other with valve-in-valve therapy. Management strategies for post-TAVR VSDs varies, but should be individualized to the clinical scenario. We performed a literature search and sought to identify various risk factors which may predispose patients to the development of VSD after TAVR.

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Abbreviations: VSD, ventricular septal defect; TAVR, transcatheter aortic valve replacement; MSCT, multi-slice computer tomography; CAD, coronary artery disease; CKD, chronic kidney disease; CVA, cerebrovascular accident; STS, Society of Thoracic Surgeons; LVOT, left ventricular outflow tract; VT, ventricular tachycardia; TEE, transesophageal echocardiogram; THV, transcatheter heart valve.

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1. Introduction

Transcatheter aortic valve replacement (TAVR) has emerged as an alternative to surgery in patients with severe aortic stenosis, who are at intermediate, high or prohibitive risk for a traditional surgical aortic valve replacement [1,2]. Development of a membranous ventricular septal defect (VSD) after TAVR carries a high mortality and the optimal management strategy remains unclear. Reported treatments have included percutaneous closure, surgical repair and conservative

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management. We present two cases of membranous ventricular septal defects at our institution and a literature review of currently reported cases of post-TAVR VSD from January 2002 until June 2016. Furthermore, we seek to identify potential risk factors which can predispose patients to this rare but potentially fatal complication.

2. Case 1

The patient was an 87-year-old male with severe aortic stenosis and New York Heart Association (NYHA) Class III symptoms that was referred for TAVR. His other medical history included coronary artery disease (CAD), chronic kidney disease (CKD), cerebrovascular accident (CVA) and paroxysmal atrial fibrillation on anticoagulation. Echocardiography revealed normal left ventricular systolic function and severe aortic stenosis with peak and mean aortic valve gradients of 90 mmHg and 58 mmHg, respectively. The calculated aortic valve area was 0.93 cm². Based on multi-slice computer tomography (MSCT), the average annulus diameter was 26 mm with an average area of 530 mm² and mild left ventricular outflow tract (LVOT) calcification. Both iliofemoral arteries were suitable for percutaneous TAVR access (Fig. 1A and B). His calculated Society of Thoracic Surgeons (STS) risk score was 6.1%. Despite his intermediate risk score, he was felt to be high risk for a surgical aortic valve replacement due to advanced age and frailty.

The procedure was performed via transfemoral access. The aortic valve was predilated with a Z MED II 22 mm × 5 cm × 100 cm valvuloplasty balloon. A 29 mm Edwards Sapien XT valve (Edwards Lifesciences, Irving CA)

was deployed during rapid ventricular pacing (Fig. 1C). The procedure was complicated by the development of a left bundle branch block, without requiring a permanent pacemaker. Post procedural echocardiography revealed a well seated valve with a mean gradient of 5 mmHg, a peak gradient of 8 mmHg and a mild paravalvular leak. His post-operative course was unremarkable and he was discharged home after 3 days.

One week after his discharge, he had a brief episode of chest pain and pre-syncope and was found to be in monomorphic ventricular tachycardia (VT) at a rate of 250 beats per minute without hemodynamic compromise. He received an amiodarone bolus in the field and converted to atrial fibrillation prior to transfer to our institution. Transthoracic echocardiography revealed a new ventricular septal defect of 4 mm, with normal left and right ventricular function (Fig. 1D, Supplemental Video 1). The estimated pulmonary artery pressure was within normal range. Amiodarone was continued and no further episodes of VT were observed. An implantable cardioverter defibrillator was recommended by the electrophysiology team, but the patient preferred conservative management with amiodarone. He was discharged home with a defibrillator vest while he continued his amiodarone load.

At 30 days and 6-month follow-up visits, he was doing well and reported NYHA Class II symptoms. He had no further hospitalizations for heart failure and no documented arrhythmias. Thirty-day follow-up transthoracic echocardiogram noted the VSD to be stable in size with a left to right shunt. The right ventricle remained normal in size and function with normal pulmonary artery pressures. As a result, it was decided not to proceed with percutaneous closure. Patient is now 2 years out of his initial procedure and doing well, with minimal residual VSD.

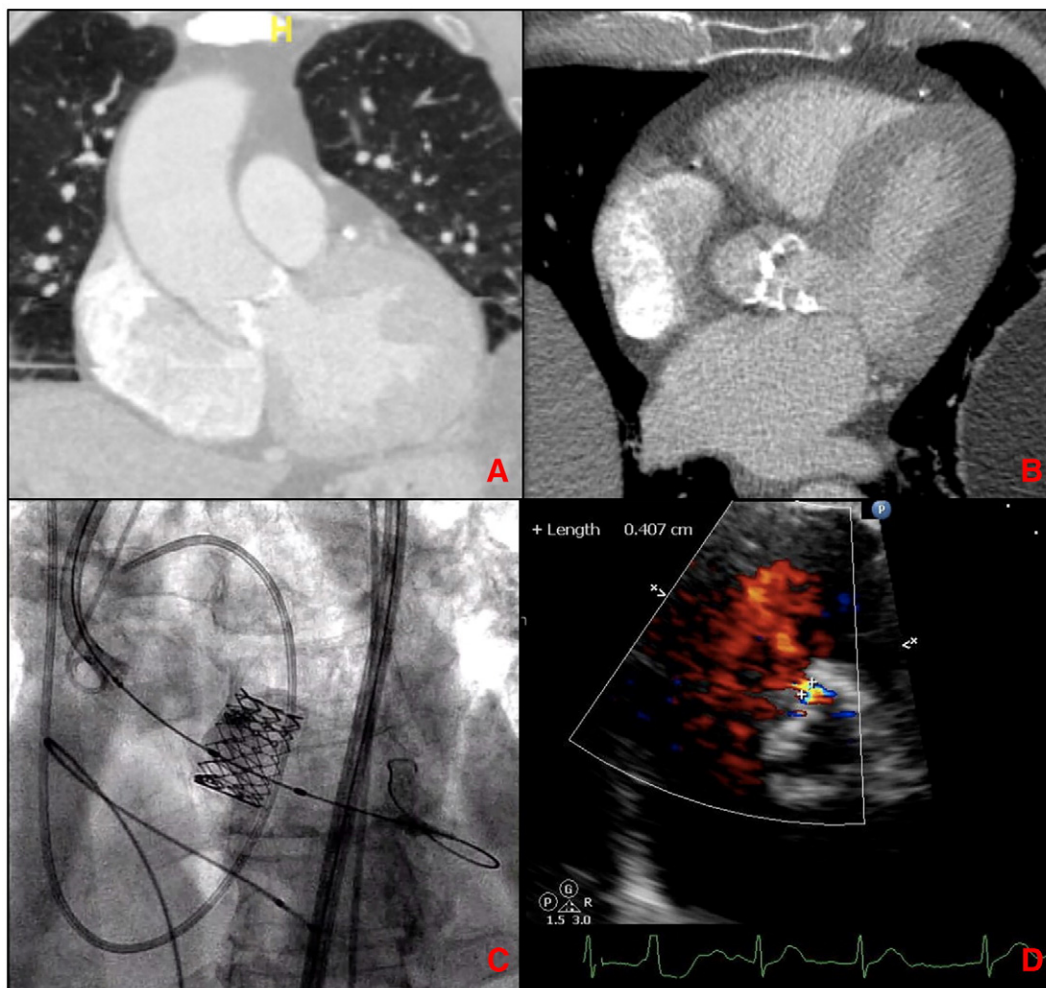


Fig. 1. (A, B) CT scan showing minimal LVOT calcification. (C) Transfemoral deployment of 29 mm Edwards Sapien XT valve. (D) Transthoracic echocardiogram with parasternal short-axis view at the base showing 4 mm membranous ventricular septal defect with left to right shunt. (Abbreviations: LVOT = left ventricular outflow tract).

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