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CASE REPORT
Other

A case report of ventricular septal defect complicating transcatheter aortic valve implant for aortic regurgitation: novel complication and technical considerations

Jack Hartnett [1], Lisa Brandon [1], Deirdre Waterhouse[2], Ross T. Murphy[1,2], Kevin P. Walsh[3], Mark S. Spence[4], and Andrew O. Maree [1,2]*

[1]Department of Cardiology, St James's Hospital, James's Street, Dublin 8 D08 W9RT, Ireland; [2]Department of Cardiology, Blackrock Clinic, Rock Road, Blackrock, Co Dublin A94 E4X7, Ireland; [3]Department of Cardiology, Our Lady's Children's Hospital Crumlin, Dublin 12 D12 N512, Ireland; and [4]Department of Cardiology, Royal Victoria Hospital, Belfast Trust, 274 Grosvenor Road, Belfast BT12 6BA, UK

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Background Transcatheter aortic valve implantation (TAVI) has proven efficacy in the treatment of aortic stenosis (AS). Understandably, there is increasing enthusiasm for its use to treat aortic regurgitation (AR). However, there are significant anatomical differences between AS and AR which make TAVI for AR more complex.

Case summary We present the case of technically challenging TAVI for severe AR, which was complicated by a traumatic ventricular septal defect (VSD) that required percutaneous closure. To our knowledge, this is the first published case of VSD post-TAVI for AR.

Discussion This unanticipated complication highlights anatomical differences between TAVI use in AS and AR. Lack of aortic valve calcification and excessive annular compliance made stable deployment of a self-expanding valve extremely challenging. Despite device oversizing, repeated embolization of the prosthesis into the left ventricular outflow tract traumatized the interventricular septum.

Keywords Case report • Aortic regurgitation • Transcatheter aortic valve implantation • Ventricular septal defect

Learning points

- There are significant anatomical differences between a regurgitant and stenosed aortic valve. These include less valvular calcium and increased annular compliance.
- Transcatheter aortic valve implantation in aortic regurgitation is less stable and valve embolization may result in trauma to the interventricular septum.

Introduction

Transcatheter aortic valve implantation (TAVI) has revolutionized the management of severe aortic stenosis (AS) and is an established percutaneous alternative to surgical aortic valve replacement (SAVR).[1]

Given TAVI's expanding role in the treatment of AS there has understandably been increased interest in its use to treat aortic

* Corresponding author. Tel: +353 (0)14103523, Email: amaree@tcd.ie
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regurgitation (AR); a condition with traditionally few therapeutic strategies outside of heart failure (HF) management and SAVR.[2] Indeed, recent data suggest TAVI in AR may have comparable mortality to SAVR but with a favourable complication profile.[3] The evidence-based however is lacking and other studies indicate higher complication rates.[4,5] This is generally attributed to difficulty anchoring the prosthesis in a non-calcified aortic valve. Here, we provide the first description of a traumatic ventricular septal defect (VSD) that occurred during implantation of a self-expanding valve prosthesis. Ventricular septal injury was the result of device instability and repeated embolization.

Timeline

Month 0	Referral for transcatheter aortic valve implantation (TAVI) with dyspnoea, orthopnoea, and exertional chest pain from severe symptomatic aortic regurgitation (AR)
Month 3	TAVI for AR with immediate post-procedure ischaemic stroke
Month 4	Presentation with worsening heart failure (HF) and transoesopheageal echocardiogram confirming serpiginous ventricular septal defect (VSD)
Month 5	Percutaneous VSD closure using Amplatz™ device
Month 7	Resolved HF symptoms on review in clinic

Case presentation

An 86-year-old lady was referred for consideration for TAVI to treat severe symptomatic AR. Symptoms included New York Heart Association (NYHA) III dyspnoea, orthopnoea, and exertional chest pain that were refractory to medical therapy. On cardiovascular examination, an early diastolic murmur was present. No leg swelling was noted. Past medical history included prior pulmonary emboli, Barrett's oesophagus, diverticular disease, and T7 vertebral fracture.

Routine laboratory investigations were within normal range. Transthoracic echocardiography confirmed a severe central jet of aortic incompetence through a trileaflet, non-calcified, non-stenosed aortic valve. Further investigations included pulmonary function testing, right heart catheterization, and coronary angiography that excluded key differentials such as pulmonary hypertension and obstructive coronary artery disease. As such, the patient's progressive symptoms were attributed to severe AR. The case was discussed at a multidisciplinary Heart Team meeting and in light of the patient's age and comorbidities she was not considered a candidate for surgery and off-label TAVI was recommended. In hospital mortality from SAVR was estimated at 2.38% using the validated EuroSCORE II.

Application for off-label use of the Evolut™ R (Medtronic, Minneapolis, MN, USA) was approved and a 34 mm Evolut™ R bioprosthesis recommended given the aortic annulus perimeter of 79.9 mm, aortic annulus diameter of 25.4 mm, and sino-tubular junction diameter 33.3 mm (minimum)—33.5 mm (maximum). Planning multidetector computed tomography imaging highlighted two key procedural considerations (i) the lack of aortic annular calcification (Figure 1) (ii) the horizontal angulation of the aorta (69°).

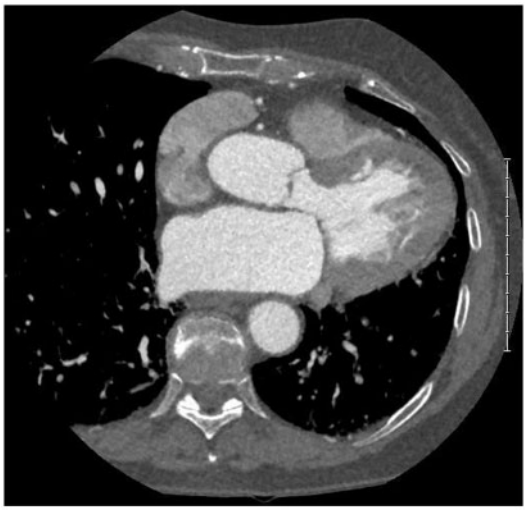


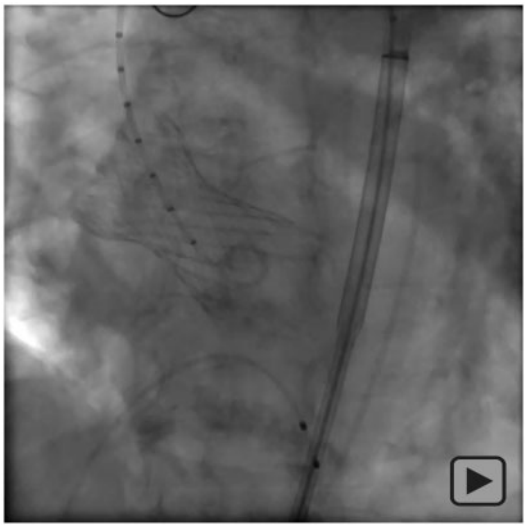
Figure 1 Axial image at level of aortic annulus from computed tomography-coronary angiogram (CT-CA). Note the absence of calcification at the aortic valve complex.

The procedure was initially uncomplicated. Under conscious sedation a right femoral approach was employed and the aortic valve crossed with a pigtail catheter and a small safari wire positioned in the left ventricle. Via the left common femoral vein a 5F balloon tipped temporary pacing wire was inserted into the right ventricular apex. An activated clotting time of 310 was established and the valve was inserted and deployment attempted with concomitant right ventricular rapid pacing at 160 b.p.m. to temporarily reduce cardiac output and enhance stability during difficult deployment.

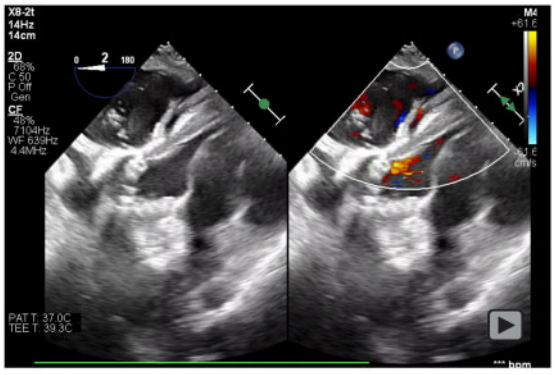
Despite multiple attempts, upon partial expansion of the 34 mm Evolut™ R, the valve repeatedly prolapsed superiorly into the aorta or inferiorly into the ventricle. Deployment of the smaller 29 mm Evolut™ PRO was then attempted without success. With progressive attempts at valve repositioning the AR jet increased, pulse pressure widened, and the patient became unstable. The procedure was converted to general anaesthesia and a surgical theatre readied in case the valve could not be implanted successfully. The safari wire was exchanged for a more stiff Lunderquist wire to provide additional stability during the technically challenging procedure. Finally, the 34 mm Evolut™ R valve was deployed successfully (*Video 1*) with a total of three bioprosthesis deployment attempts required; in keeping with Evolut™ R technical guidelines. The valve position was slightly supra-annular with initial 2+ paravalvular AR.

The patient was transferred to the intensive care unit post-procedure where she subsequently developed new onset expressive aphasia and right-sided hemiparesis. Imaging confirmed an acute ischaemic stroke.

After initial improvement in stroke symptoms the patient developed progressive HF. A transoesophageal echocardiogram performed 6 days post-TAVI identified a serpiginous muscular VSD



Video 1 Aortogram post final transcatheter aortic valve implantation deployment. Note the supravalvular position of the prosthetic valve and the 2+ paravalvular leak into the left ventricle. The lack of aortic valve calcification is notable.

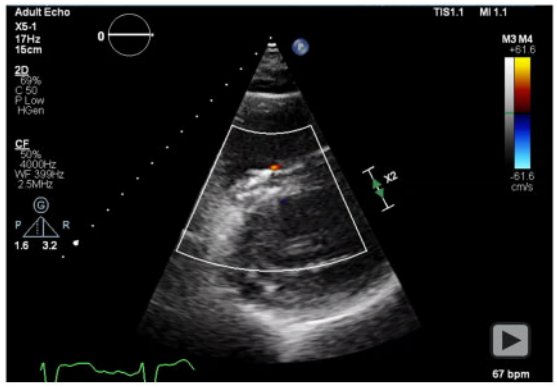


Video 2 Transoesophageal echocardiogram image in modified transgastric short-axis view demonstrating ventricular septal defect with serpiginous cleavage of interventricular septum.

inferior to the prosthetic valve with associated left-to-right shunting (*Video 2*).

The progressive clinical picture and echocardiogram findings clearly demonstrated that the VSD was symptomatic and unlikely to improve without intervention. A multidisciplinary decision was made to close the VSD percutaneously using an Amplatz™ device.

Under general anaesthesia and transoesophageal echocardiogram guidance a right femoral approach was used. A left ventriculogram demonstrated the VSD which measured 0.9 mm in maximal diameter (Supplementary material online, *Video S1*). The interventricular septum diameter was estimated at 1.3 cm. A 12 mm Amplatz™ muscular VSD device was selected. The defect was crossed using a 5F JR4 catheter and a 0.035 terumo angled guidewire. The catheter was advanced into



Video 3 Transthoracic echocardiogram image of interventricular septum demonstrating Amplatz™ device *in situ* with no flow through ventricular septal defect at 4-month follow-up post-closure.

the right ventricular outflow tract and the wire exchanged for an Amplatz™ super stiff wire. The VSD device was inserted from the right ventricular side and the discs deployed on either side of the septum. Although well positioned there was some persistent flow through the VSD seen on intraoperative left ventriculogram (Supplementary material online, *Video S2*). However, the patient improved steadily with resolution of HF symptoms and minimal flow evident through VSD on transthoracic echocardiogram 4 months post-closure (*Video 3*).

Discussion

There is an unmet need for percutaneous treatment of AR. In the past, those considered unsuitable for surgery would have been confined to medical HF therapies. However, TAVI is becoming increasingly popular in this cohort. Indeed, recent editorials have proclaimed the benefit of TAVI in AR based on recent encouraging small non-randomized studies.[6] A national analysis in the United States found no difference in in-hospital mortality between SAVR and TAVI. Furthermore, TAVI was associated with reduced risk of post-operative complications that included acute kidney injury (AKI), cardiogenic shock, and respiratory sequalae.[3]

However, when compared with TAVI for AS results for AR are associated with increased in-hospital mortality.[4,7,8] Two comparative studies noted a lower device success rate for AR (76.9–81.8% vs. 91.3–96.0%) and more complications, particularly AKI and paravalvular leak.[7,8] In another retrospective analysis, heart block requiring permanent pacemaker insertion occurred in 18.2% and need for second valve implantation in 16.6%.[9] In contrast, lower rates (4.5% and 1.7%, respectively) were reported in the original PARTNER AS trial.[1]

Worse outcomes cannot be completely explained by lesser experience with TAVI for AR. The more likely reason is anatomical differences: (i) aortic annulus calcification, (ii) aortic angulation and dimensions, and (iii) aortic root rigidity.

A calcified aortic annulus is key to successful TAVI. On initial valve crossing calcification acts as a radio-opaque landmark that facilitates prosthesis positioning. In its absence valve positioning is more difficult.[10] Valvular calcium also acts as an anchor for the deployed

prosthesis and reduces embolization and paravalvular leak. While the aortic valve is usually heavily calcified in AS, calcification is often absent in AR. Yoon *et al.*[9] demonstrated that no/low aortic valve calcification was associated with less device success (70.6% vs. 87.2%, $P = 0.03$) vs. a heavily calcified leaflet. In our case, aortic calcification was absent on imaging (Figure 1). The elastic native valve in AR can expand during prosthetic deployment thus valve oversizing is advised. However, in the absence of guidelines review of the literature suggests oversizing by 15–20%.[10] Our patient had an annulus diameter of 25.4 mm and a 34 mm Evolut™ R bioprosthesis (recommended for annulus diameter 26–30 mm) was used.

Risk of valve dislodgement and embolization is higher when a prosthesis is expanded into a dilated aorta.[11] This is also relevant to AR, which is also associated with more aortic dilation and friability than AS. Our patient's mean ascending aorta diameter was greater than that reported in AS studies (35.7 mm vs. 32.0 mm in one large AS study[11]). High degrees of aortic angulation also reduce device efficacy and increase paravalvular leak.[12] Angulation in our case with 69° and is often referred to as a 'horizontal aorta'.

Our patient's unfavourable anatomy led to valve dislodgement inferiorly into the ventricle. Repetitive trauma to the interventricular septum by the partially expanded valve resulted in a VSD. To our knowledge, this is the first case of a VSD post-TAVI for AR. Ventricular septal defect post-TAVI is rare with less than 30 cases reported in the literature.[13–15] All prior cases were for AS and the majority used balloon expandable stents. Interestingly, proposed risk factors include aortic annulus dilation and valve oversizing.[14] In a systematic review of 20 cases, percutaneous closure was the most common intervention for VSD repair (6 cases).[14]

New AR-specific TAVI prostheses with alternative anchoring mechanisms may be required along with evidence-based cut-off criteria for key anatomical variables such as aortic angulation, dilation and degrees of calcification. Finally, large randomized controlled trials are necessary to determine which subgroups of AR patients would most benefit from TAVI over SAVR or medical management.

Conclusions

Ventricular septal defect is an uncommon but recognized complication of TAVI. We describe here the first case of VSD occurring during TAVI to treat AR that resulted from repetitive trauma to the interventricular septum from inferior embolization of the partially expanded valve prosthesis.

Lead author biography

Jack Hartnett is a graduate of the School of Medicine, Trinity College Dublin. Currently working as a junior doctor in St James's Hospital Dublin, Jack plans to specialize in Cardiology.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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