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CASE REPORT

CLINICAL CASE

Strut Inversion During Valve-in-Valve Transcatheter Aortic Valve Replacement

An Unknown Complication?

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ABSTRACT

A 74-year-old man presented with failure of a bioprosthetic aortic valve implanted 7 years earlier, with a mean gradient of 44 mm Hg across the aortic valve. During valve-in-valve transcatheter aortic valve replacement, we came across an unusual complication of strut inversion at the lower end of the valve. (Level of Difficulty: Advanced.) (J Am Coll Cardiol Case Rep 2022;4:460-463) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

A 74-year-old man presented with exertional dyspnea on minimal activity and presyncope for the previous 6 months. He had undergone surgical aortic valve replacement (AVR) 7 years earlier and with an Epic Supra 19-mm bioprosthesis valve for severe calcific aortic stenosis (AS). During the intervening 6.5 years he had remained well and was able to perform work without limitation.

LEARNING OBJECTIVES

- To understand the importance of achieving appropriate cell strut geometry while performing ViV TAVI, despite achieving good hemodynamics.
- To understand the importance of proper valve crimping and loading, and predilation of the surgical prosthesis, to prevent valve inversion/indentation.

On examination, the pulse (80 beats/min) and blood pressure (100/60 mm Hg) were within normal limits. Cardiac examination revealed a heaving apex and a systolic thrill in the right second intercostal space, radiating to the carotids. A harsh, grade 5/6 ejection systolic murmur was heard in the same location.

MEDICAL HISTORY

After AVR, the patient continued with 6-monthly follow-up visits. The last echocardiogram performed 1 year earlier had shown a normally functioning aortic bioprosthesis with peak and mean gradients of 24 mm Hg and 14 mm Hg, respectively, with a normal left ventricular ejection fraction. After that the patient had been lost to follow-up until the current presentation. There was no other relevant history.

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ADVANCED

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

DIFFERENTIAL DIAGNOSES

Degeneration of the aortic bioprosthesis was strongly considered based on the initial assessment. However, associated coronary artery disease could not be entirely excluded clinically.

INVESTIGATIONS

An echocardiogram revealed thickening and calcification in the bioprosthetic aortic valve with transvalvular peak and mean gradients of 100 mm Hg and 44 mm Hg, respectively. There was significant left ventricular hypertrophy with a normal left ventricular ejection fraction. Coronary angiography revealed normal coronaries. Other baseline laboratory parameters were within normal limits. In view of the patient's recent-onset disabling symptoms secondary to severe aortic stenosis in the setting of a degenerated aortic bioprosthesis, a heart team discussion was undertaken, and it was decided to perform valve-invalve (ViV) transcatheter aortic valve implantation (TAVI) as the treatment modality. His Society of Thoracic Surgeons score of redo-AVR for periprocedural morbidity and mortality was 10%.

Detailed computed tomography angiography was performed and was analyzed for feasibility of TAVI (Table 1).

MANAGEMENT

The patient underwent TAVI according to the standard operating procedure of the center. Right femoral arterial access was obtained. A Confida wire was placed into the left ventricle through a pigtail catheter. Predilation of the native valve was not performed. A 23-mm Evolute R self-expanding valve was crimped and loaded onto the advancer. Accurate loading of the valve and its positioning were confirmed and were found appropriate on cinefluoroscopy before being advanced into the inline sheath.

The valve was positioned across the native aortic valve and 80% deployed. The valve started functioning with a peak transvalvular gradient of 24 mm Hg, without paravalvular leak on transesophageal echocardiography. However, on fluoroscopy, the diamond-shaped configuration of a few cells of the valve frame, predominantly in the lower part, appeared to be distorted (**Figure 1**, Video 1). As a result, it was difficult to make the prior and new valve coaxial. Despite waiting for 45 minutes, the configuration of the valve did not correct spontaneously. In the meantime, the valve was recaptured once and redeployed. However, the distorted configuration remained. A minimal leak across the aortic valve was seen on angiography, probably related to the Confida wire across the valve. Despite a good result with a peak transvalvular gradient of only 24 mm Hg, it was decided to recapture the valve and deploy a new valve, based on the unsightly appearance of the cell struts. The valve was

retrieved, and a new 23-mm Evolute R self-expanding valve was placed successfully at the desired location 3 to 4 mm below the radiopaque ring of the old aortic bioprosthesis. This time, the configuration of the valve frame appeared normal (Figure 2, Video 2). The valve acquired better configuration both on fluoros-copy and also on transoesophageal echocardiography (Videos 3 and 4). After confirming no paravalvular leak and a peak gradient of only 22 mm Hg, we decided to complete the procedure without any attempt to fracture the native ring of the aortic bioprosthesis or to postdilate the new valve. The access site was closed with a preplaced Perclose suture.

The first valve that was removed was examined because of abnormal configuration. It was found that 2 struts of the valve frame were inverted, leading to significant crimping of the lower end of the valve (Figure 3).

DISCUSSION

The therapeutic options for patients with degeneration of a bioprosthetic aortic valve are redo AVR or ViV TAVI. Recent evidence indicates that ViV TAVI is

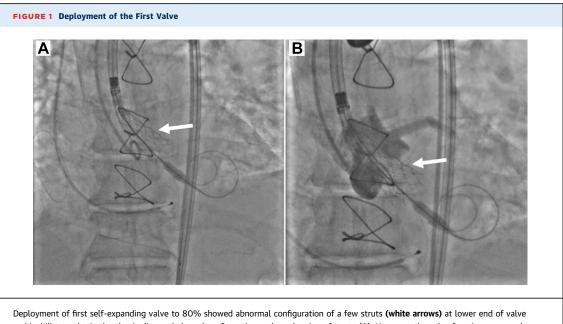
TABLE 1 Various Dimensions Angiography	of the Aortic Valve	e on Computed To	omography
Aortic annulus	Minimum	Maximum	Mean
Diameter, mm	14.2	17.2	15.7
Perimeter, mm	49.7		15.8
Area, mm ²	193.2		15.7
LVOT	Minimum	Maximum	Mean
Diameter, mm	14.8	20.3	17.6
Perimeter, mm	55.9		17.8
Area, mm ²	236		17.3
Aortic root angle, °		49	
Maximum ascending aorta, mm	32.3		
Sinotubular junction, mm	25.5 × 27.6		
Sinus of Valsalva			
Diameter, mm	28.8 LCC	28.2 RCC	29.5 NCC
Height, mm	17.5 LCC	21.2 RCC	18.1 NCC
Coronary ostia height	10.7 left	17.8 right	
Common iliac artery	7.3 \times 9.2 right	7.7×8.2 left	
External iliac artery	$5.1 \times$ 6.3 right	6.6×6.8 left	
Common femoral artery	6.0×6.3 right	7.1×7.9 left	
$\label{eq:LCC} \mbox{LCC} = \mbox{left coronary cusp; LVOT} = \mbox{left ventricular outflow tract; NCC} = \mbox{noncoronary cusp; } \mbox{RCC} = \mbox{right coronary cusp.}$			

ABBREVIATIONS AND ACRONYMS

AVR = aortic valve replacement

TAVI = transcatheter aortic valve implantation

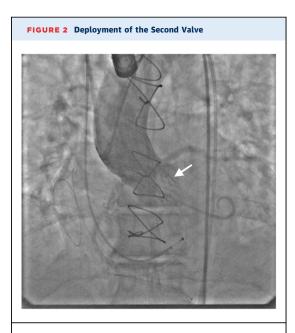
ViV = valve-in-valve



Deployment of first self-expanding valve to 80% showed abnormal configuration of a few struts (white arrows) at lower end of valve and inability to obtain the classic diamond-shaped configuration and overlapping of struts (A). However, the valve function was good on echocardiography, and there was minimal paravalvular leak on angiogram (B).

feasible and is a safer alternative to redo AVR.¹ A recent meta-analysis in a large population showed ViV TAVI to be associated with lower rates of 30-day mortality, stroke, permanent pacemaker implantation, major bleeding, and shorter hospital stay.² Both

self-expanding and balloon-expandable valves are used for ViV TAVI, and the common complications associated with them are valve embolization, coronary obstruction, high residual gradients, and valve thrombosis.³⁻⁵ Structural disorganization of the



After removal of the first valve, the second valve was deployed. The appropriate diamond configuration was seen **(white arrow)**, and it easily aligned with the prior aortic bioprosthesis. There was with no paravalvular leak, and well-flowing left and right coronary arteries were seen.



The first valve was removed and inspected for abnormality, and it showed 2 struts if the nitinol frame inverted, causing abnormal configuration of lower end of valve. nitinol frame of the valve causing indentations during valve deployment, as happened in the index case, is an unheard-of complication of ViV TAVI.

Despite the increasing popularity of ViV TAVI, it still is a relatively new treatment modality for failed surgical bioprosthetic valves. The index patient had a 19-mm bioprosthesis with an anticipated true internal diameter of 17 mm, the thickened leaflet and fibrosis around it being taken into consideration. To provide maximum benefit by a supra-annular valve position, a 23-mm selfexpanding valve was used. It was decided to perform valve fracture if the residual peak-to-peak gradient was >25 mm Hg. Even though good hemodynamics were obtained after 80% deployment of the self-expanding valve, the subtle abnormal configuration of valve struts prevented us from completely deploying it. Another reason was the inability to align the new valve with the old surgical valve. As a result, the first valve was removed, and a decision to deploy a fresh valve was made.

The possible explanations for the above complication are improper crimping and loading of the valve, a calcific spur at the valve not allowing the valve to open, or proceeding with ViV TAVI without predilation of the surgical bioprosthesis. Predilatation of the surgical valve before ViV TAVI was not performed because there was no major calcification at the valve level; thus, the complication of improper valve opening was not anticipated.

The fresh valve was successfully placed and a final peak transvalvular gradient of 22 mm Hg was achieved without valve fracturing. However, in retrospect, we maintain equal possibilities of improper crimping or inadequate predilation that could have led to this extremely uncommon complication during deployment of the self-expanding valve. Thus, it is extremely important to anticipate complications and to be vigilant while performing ViV TAVI. As more and more experience is gained, many unknown complications are anticipated to occur.

FOLLOW-UP

The patient was asymptomatic at the 9-month followup visit. A recent echocardiogram showed a mean gradient of 18 mm Hg.

CONCLUSIONS

Structural disorganization of the nitinol frame of the valve causing its indentation/inversion is an underrecognized complication during the performance of ViV TAVI. Improper crimping and loading or a calcific spur at the deployment site are possible explanations for this.

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KEY WORDS aortic bioprosthesis, strut inversion, transcatheter aortic valve implantation, valve-in-valve transcatheter aortic valve replacement

APPENDIX For supplemental videos, please see the online version of this article.