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CASE REPORT

INTERMEDIATE

CLINICAL CASE

# Rare Cause of Late Left Aortic Sinus Obstruction Following Transcatheter Aortic Valve Replacement

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## ABSTRACT

A 74-year-old woman with a history aortic stenosis with prior transcatheter aortic valve replacement presented with non-ST-segment elevation myocardial infarction secondary to a delayed left coronary sinus obstruction. With physiology and intravascular ultrasound guidance, the patient was treated with stents through the valve struts and to the left main. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2023;14:101828)


## HISTORY AND PRESENTATION

A 74-year-old woman presented with progressive dyspnea with acute exacerbation over the preceding 3 days as well as a single episode of chest pain 2 days before her admission. The patient was hemodynamically stable, with a blood pressure of 106/91 mm Hg and a heart rate of 88 beats/min. She was afebrile, and oxygen saturation was 100% on room air. On physical examination, the patient seemed comfortable and was well perfused. The jugular veins were not distended, lung and heart sounds were normal without gallops or murmurs, and no peripheral edema was noted. Complete blood count showed a hemoglobin of 7.0 g/dL (baseline was 9.0 g/dL) and normal white blood cell count. The creatinine was 0.49 mg/dL, and blood culture results were negative. The electrocardiogram showed a normal sinus rhythm with a left bundle branch block and left-axis deviation, which was unchanged. Her first high-sensitivity troponin I level was 16,358 ng/L. The chest x-ray was unremarkable, demonstrating previous transcatheter aortic valve replacement (TAVR).

### LEARNING OBJECTIVES

- To be able to identify the pathophysiology leading to non-ST-segment elevation myocardial infarction in the post-TAVR patient using multimodality imaging.
- To be able to offer an appropriate treatment to patients presenting with delayed coronary obstruction after TAVR.

## PAST MEDICAL HISTORY

She underwent transaxillary TAVR for aortic stenosis with a 26-mm Evolut PRO+ valve 10 months before presentation, for which she was on prasugrel alone after 1 month of dual antiplatelet therapy. The pre-TAVR computed tomography angiography did not

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**ABBREVIATIONS AND ACRONYMS**

**DES** = drug-eluting stent(s)

**IVUS** = intravascular ultrasound

**LAD** = left anterior descending artery

**LM** = left main

**MSA** = minimal surface area

**PCI** = percutaneous coronary intervention

**RFR** = resting full-cycle ratio

**TAVR** = transcatheter aortic valve replacement

**TTE** = transthoracic echocardiogram

demonstrate any high-risk features for coronary obstruction. The coronary artery ostium height was 15 mm for the left and 16.4 mm for the right, and the sinus of Valsalva diameter was greater than 26 mm. The pre-TAVR coronary angiography showed a right coronary artery chronic total occlusion, mild disease in the left anterior descending (LAD) and circumflex arteries, and a normal left main (LM). Post-TAVR aortography showed good perfusion of the left coronary system. The last transthoracic echocardiogram (TTE) 9 months before the current presentation showed a left ventricular ejection fraction of 55% to 60% with a normally functioning aortic valve prosthesis. Her past medical history was also notable for anemia secondary to Heyde syndrome with normal recent endoscopy findings.

## DIFFERENTIAL DIAGNOSIS

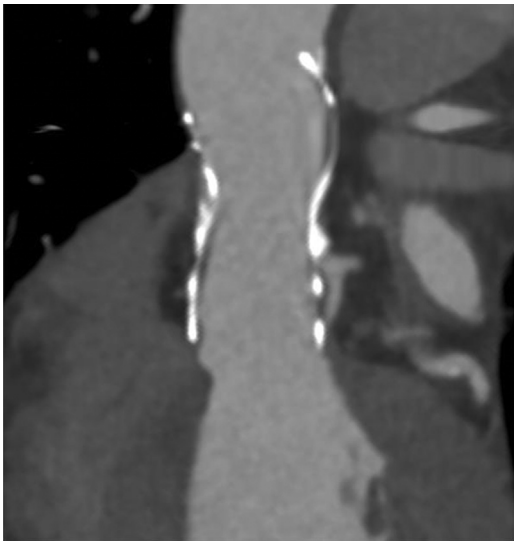
At the time of presentation, the differential diagnosis included type 1 non-ST-segment elevation myocardial infarction, bioprosthetic valve degeneration or thrombosis, and type 2 myocardial infarction in the context of anemia.

## INVESTIGATIONS

A TTE showed a mildly reduced left ventricular ejection fraction at 45% to 50% with regional wall motion abnormality in the LAD territory and a normally functioning TAVR. No thrombus or vegetation was visualized on TTE. With the hemoglobin stabilizing at 9.0 g/dL, the patient underwent a coronary angiography, from the radial approach, that showed a stable coronary anatomy, but there was poor filling of the left coronary sinus because of restricted patency of the TAVR struts. Supplemental Figure 1 and Video 1 show the diagnostic angiogram. Selective canulation of the LM was not possible, and the catheter engagement was limited behind the valvular stent, resulting in severe pressure dampening. At this point, flow obstruction to the left coronary sinus was suspected as the cause of the patient's presentation, and the mechanism was thought to be chronic thrombosis caused by poor flow and/or tissue growth from the native valve leaflets entrapped between the sinuses and the valvular frame struts with reduction of flow in this area.

Computed tomography angiography confirmed a normal LM, but a small shadow was present between the valve struts and the LM, possibly representing soft tissue growth, which was not attached to the native valve leaflets, compatible with thrombosis (**Figure 1**). There was also an organized thrombus in the right and noncoronary sinuses trapped behind the TAVR frame, which supported the hypothesis of this to be the mechanism of the left coronary sinus underfilling as well.

**FIGURE 1 Computed Tomography Scan With Shadowing Behind the Transcatheter Aortic Valve Replacement Struts**

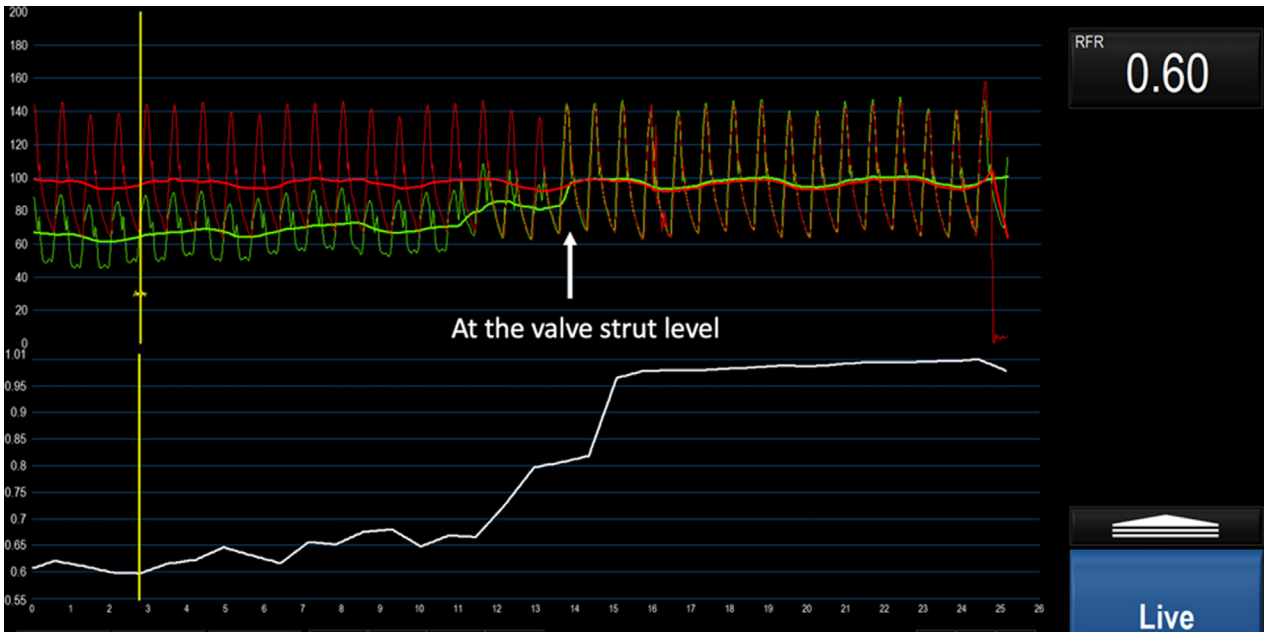


## MANAGEMENT

After a heart team discussion and consideration of coronary artery bypass graft, it was decided to perform physiology and intravascular ultrasound (IVUS)-guided percutaneous coronary intervention (PCI).

Resting full-cycle ratio (RFR) was strongly positive at 0.60 in the LAD. Pullback demonstrated a jump exclusively at the level of the TAVR struts (**Figure 2**). On IVUS, the LM was normal. On the outer side of the TAVR struts, a tissue growth was seen as a pannus creating a severe stenosis of the TAVR cells with a slit-like opening (**Figure 3**). It was decided to treat with PCI from the distal LM to the aorta across the left sinus and the TAVR struts. A first drug-eluting stent (DES), the Resolute Onyx 5.0 × 18 mm, was implanted from the distal LM to the aorta, trying to hang the least possible length of the stent in the aorta while preserving its radial force. Because the TAVR frame is nitinol, recoil was inevitable despite good stent expansion during deployment (Supplemental Figure 2). It was decided to implant a second DES Resolute Onyx 5.0 × 12 mm within the proximal

**FIGURE 2** **Resting Full-Cycle Ratio Pullback**



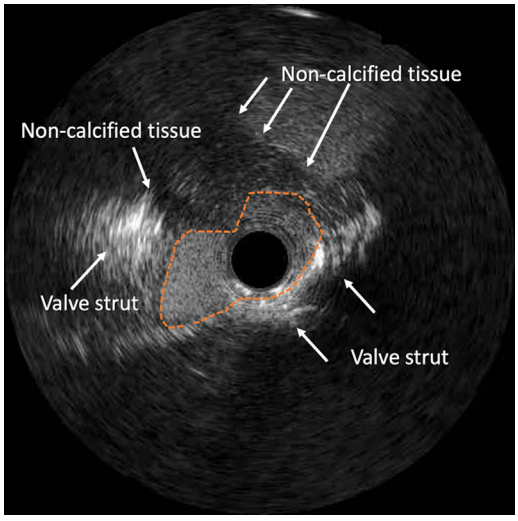

portion of the first stent to increase the radial strength. An IVUS was done at this time showing a minimal surface area (MSA) of 8 $mm^2$ (Supplemental Figure 3). Given the large territory at risk and the potential challenges of repeated PCI in the future, it was decided to aim for a larger MSA. To achieve better radial force, we deployed a peripheral bare metal stent, the Herculink 6.0 × 15 mm, at high pressure. Post-PCI angiogram showed a good result (Supplemental Figure 4, Video 2). The final IVUS was satisfactory, with an MSA of 12.7 $mm^2$ (Supplemental Figure 5). Post-PCI RFR was 0.93. Follow-up angiography at 3 months showed a patent stent and left sinus (Supplemental Figure 6).

## DISCUSSION

Myocardial infarction after TAVR occurs in about 2% of patients at 1 year, most of them associated with concomitant coronary artery disease.[1] The prosthetic valve can lead to coronary flow obstruction by different mechanisms that differ depending on the timing of presentation. After the index procedure, the incidence of delayed coronary obstruction is estimated to be very rare, at 0.22%,[2] and can occur early within 7 days postprocedure, usually caused by continuing expansion of the self-expandable valve platform, dissection, or hematoma. Late coronary flow obstruction can be seen more than 7 days after the procedure and can be attributable to a valve thrombosis, endocarditis, fibrosis, and endothelization of the valve stent. Risk factors for late coronary obstruction include valve-in-valve procedure; inappropriate antithrombotic regimen; and, possibly, self-expandable valves.[2] In this case, the only risk factor was the self-expandable valve.

The best treatment of delayed coronary obstruction is yet to be elucidated and is probably dependent on the underlying mechanism, but it has been previously treated with surgical removal and surgical aortic valve replacement.[3] More recently, PCI was used in a patient where blood to the left coronary sinus was flowing through a channel from the noncoronary sinus. In that case, the coronary stent was placed outside the valve stent.[4] We decided to treat this patient with PCI presuming that the mechanism was endothelization or thrombus formation, both of which we could not diagnose with absolute certainty. Although the higher tissue characterization obtained by optical coherence tomography would have been helpful to achieve a more definitive diagnosis, we were concerned about the inability to clear blood to gather a good optical coherence tomography image because the lesion was not in a coronary artery. Treatment of the obstruction to the LM flow was mandatory because of the high-risk presentation and drop in the left ventricular ejection fraction.

**FIGURE 3 Intravascular Ultrasound Imaging of the Stenosis at the Level of the Transcatheter Aortic Valve Replacement Struts**




## FOLLOW-UP

The patient remained asymptomatic after the procedure. We performed a surveillance coronary angiography 3 months after the PCI that showed no further recoil of the stent (Supplemental Figure 6) and a stable angiographic result. An aortogram done at the same time did not show any aortic regurgitation.

## CONCLUSIONS

Our case highlights a few novel approaches: first, we documented the clear ischemic nature of the problem, with RFR, with precise identification of the location of the obstruction of flow by pullback and step up. IVUS confirmed and guided therapy by identifying the presence of stenosis outside of the LM ostium into the open cell of the TAVR nitinol frame. The combination of a DES and a peripheral renal bare metal stent was required to overcome the important coronary stent recoil caused by the nitinol valve constriction through a single open cell strut. Finally, we confirmed the feasibility of performing PCI through a valve single open cell stent strut.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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**KEY WORDS** coronary artery disease, delayed coronary obstruction, percutaneous coronary intervention, transcatheter aortic valve replacement

**APPENDIX** For supplemental figures and videos, please see the online version of this paper.