Percutaneous coronary intervention for delayed coronary obstruction due to endothelialization of self-expandable transcatheater heart valve: a case report

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Background
Coronary obstruction is one of the serious complications associated with transcatheter aortic valve implantation (TAVI). Delayed coronary obstruction (DCO) is a rare manifestation of coronary obstruction.

Case summary
A 91-year-old woman was diagnosed with severe aortic stenosis. She underwent TAVI with a self-expandable valve, without any complications. After 8 months, she was readmitted to our hospital for effort angina. Transthoracic echocardiogram and myocardial scintigraphy suggested left coronary artery ischaemia. Computed tomography revealed that the transcatheter heart valve (THV) frame was covered with a low-density mass that occluded the left coronary sinus (LCS). Transoesophageal echocardiogram showed a Doppler signal flowing from the non-coronary sinus to the LCS through the roundabout route between the aortic wall and the THV. Percutaneous coronary intervention was performed for the roundabout route. Although intravascular ultrasound after the implantation of one drug-eluting stent showed the underexpansion of the stent, another stent deployment improved the expansion. After the procedure, her symptom improved.

Discussion
Reportedly, the mechanism of DCO occurring months or years after TAVI is thought to be thrombus formation or THV endothelialization. In our case, the low-density mass was considered to be endothelium which developed along the THV frame. Low sinotubular junction height and higher THV position could be the underlying mechanisms of DCO. Percutaneous coronary intervention is a possible treatment option for DCO caused by THV endothelialization.

Keywords
Transcatheter aortic valve implantation • Delayed coronary obstruction • Transcatheter heart valve endothelialization • Percutaneous coronary intervention • Case report

Learning points
• Transcatheter heart valve (THV) endothelialization is a rare cause of delayed coronary obstruction (DCO) after transcatheter aortic valve implantation.
• Low sinotubular junction height and higher THV position can be risk factors for DCO.
• Percutaneous coronary intervention is a possible treatment option for this condition.
Introduction

Coronary obstruction is one of the serious complications associated with transcatheter aortic valve implantation (TAVI).\(^1,2\) While coronary obstruction is mainly considered as an acute complication, delayed coronary obstruction (DCO) has been reported to occur several months or even years after TAVI.\(^3\)

Herein, we report a case of DCO after self-expandable transcatheter heart valve (THV) implantation that was successfully treated with percutaneous coronary intervention (PCI) via the roundabout route from the non-coronary sinus (NCS).

Timeline

| Time            | Event                                                                 |
|-----------------|----------------------------------------------------------------------|
| 8 months prior  | The patient underwent transcatheter aortic valve implantation with a self-expandable valve, without any complications. |
| Re-admission to the hospital | The patient presented with effort angina. Transthoracic echocardiogram and myocardial scintigraphy suggested the left coronary artery ischaemia. Computed tomography revealed that the transcatheter heart valve frame was covered with a low-density mass that occluded the left coronary sinus (LCS). Transoesophageal echocardiogram showed a Doppler signal flowing from the non-coronary sinus to the LCS through the roundabout route. |
| Day 10          | Percutaneous coronary intervention was performed for the roundabout route. |
| Day 14          | The patient was discharged without any symptoms.                      |
| 4 months later   | The patient remained asymptomatic.                                     |

Case presentation

A 91-year-old woman with a history of paroxysmal atrial fibrillation, on edoxaban 30 mg/day, was admitted to our hospital with dyspnoea upon exertion. Electrocardiography showed a normal sinus rhythm and left ventricular hypertrophy (LVH). Transthoracic echocardiography (TTE) revealed left ventricular ejection fraction of 65%, LVH, mild mitral regurgitation, estimated right ventricular systolic pressure of 35 mmHg, severe aortic stenosis, an aortic valve area of 0.46 cm\(^2\), a peak velocity of 5.53 m/s, and mean pressure gradient of 70.0 mmHg. Due to her old age and frailty (clinical frailty scale of 4), she was considered ineligible for surgical aortic valve replacement. A pre procedural computed tomography (CT) revealed the aortic annulus area to be 351.0 mm\(^2\). The sinotubular junction (STJ) diameter was 22.3 × 21.0 mm. The height of STJ was 14.3 mm. The diameter of the sinus of Valsalva was 29.3 mm × 28.1 mm × 28.3 mm. The heights of the right and left coronary arteries were 10.1 and 10.0 mm, respectively. The patient had undergone transfemoral TAVI with a 26-mm EvolutR (Medtronic, Minneapolis, MN, USA). We selected EvolutR due its distinct characteristics such as bulky leaflet calcification and small STJ. While the THV was implanted at a depth of 0 mm, the postoperative aortography showed normal coronary flow and mild paravalvular leak without any complication (Video S1). Postoperative TTE revealed the effective orifice area (EOA) to be 1.62 cm\(^2\). Edoxaban therapy was continued after TAVI. The postoperative course was uneventful, and the patient remained asymptomatic at the 6-month follow-up.

However, 8 months after the procedure, she was readmitted to our hospital because she complained of effort angina. Electrocardiography showed new ST-segment depression in V5–6 leads. The TTE showed hypokinesis in the anterior segment and EOA of 1.56 cm\(^2\). Laboratory test results were within normal value, except for high-sensitivity troponin I levels at 429.2 pg/mL (<26.2 pg/mL). Adenosine stress myocardial perfusion scintigraphy performed to identify ischaemic area showed myocardial ischaemia in the anterior region. Computed tomography revealed that the THV frame was covered with a low-density mass between the leaflet and STJ, which occluded the left coronary sinus (LCS) (Figure 1). The transoesophageal echocardiogram (TOE) showed no colour Doppler signal flowing from the aorta to the LCS, but a Doppler signal flowing from the NCS to the LCS was detected through a gap between aortic wall and the THV (Figure 2A). The roundabout route was also confirmed using the corresponding CT image (Figure 2B).

Selective aortography from each THV cusp revealed the roundabout flow from the NCS into the LCS (Figures 2C–E and 3). While
we could not insert a guidewire directly from the aorta into the LCS, we did advance a coronary guidewire through the roundabout route from the NCS into the LCS (Videos 1 and 2). Using a dual-lumen microcatheter, we subsequently advanced another coronary guidewire into the distal left anterior descending artery (Video 3).

Following balloon angioplasty, a 4.00 mm \( \times \) 15 mm zotarolimus-eluting stent (ZES) was deployed into the roundabout route (Figure 4A–D). The intravascular ultrasound (IVUS) showed underexpansion of the stent, despite post-dilatation with a 4.0-mm non-compliant balloon (Supplementary material online, Figure S2A). We implanted another 4.00 mm \( \times \) 15 mm ZES into the first stent, which did improve stent expansion as shown in the IVUS (Supplementary material online, Figure S2B). Final coronary angiography showed revealed a roundabout route and improved flow to the left coronary artery (Figure 4E and F).

After the procedure, the patient’s symptom improved. Transthoracic echocardiography showed normalized left ventricular wall motion. Stress myocardial scintigraphy revealed improved myocardial ischaemia. Computed tomography showed optimal stent position: the proximal edge of the stent was placed just between the frame of EvolutR (Figure 5). Aspirin and clopidogrel therapy had been initiated before PCI, and aspirin therapy was discontinued at the time of discharge. At the 4-month follow-up, she remained asymptomatic.

**Discussion**

The DCO is a rare manifestation of coronary obstruction, and the incidence was reported to be 0.22%. The main underlying mechanism of acute coronary obstruction and DCO within 7 days after TAVI is reported to be the displacement of a native valve leaflet over the coronary ostium. However, the mechanisms of DCO that occurs months or years after TAVI is thought to be attributable to thrombus formation or THV endothelialization.

Some reports of late surgical complications of self-expandable THV revealed that the stent frame was infiltrated with endothelium and embedded in the aortic wall, especially at the STJ. THV thrombosis can be detected using CT by a hypo-attenuated leaflet thickening. Anticoagulation therapy can improve thrombosis. While the main manifestation of THV thrombus is restricted leaflet motion, it
Figure 2 Transoesophageal echocardiogram (A) revealed the roundabout flow from the non-coronary sinus to the left coronary sinus (arrow). Selective aortography from guide catheter (C–E) revealed the roundabout flow. The corresponding computed tomography shows the roundabout route (arrow) and where the guide catheter was placed in each images (B). LA, left atrium; LCS, left coronary sinus; NCS, non-coronary sinus; RA, right atrium; RCS, right coronary sinus.

Figure 3 Diagrams of the aortic root on the day after transcatheter aortic valve implantation (A) and 8 months after transcatheter aortic valve implantation (B). Left coronary artery was supplied with blood flow from the left cusp (i) on the day after transcatheter aortic valve implantation. However, the flow was obstructed by the transcatheter heart valve coverage, and roundabout route from the non-coronary sinus (ii) was the only blood source for left coronary artery 8 months after transcatheter aortic valve implantation, which resulted in myocardial ischaemia.

Video 2 Contrast injection after wiring through the roundabout route.
possibly causes DCO by obstructing the coronary flow or resulting in embolization.

In our case, CT played the most important role in ruling out native coronary artery disease, as well as other sources of chest pain, and identifying the DCO. In the CT and TOE, we observed THV coverage suggesting, the presence of THV endothelialization or thrombus. We assumed that the coverage was likely to be endothelium for two reasons: first, the coverage developed while the patient was under anticoagulation therapy. Second, the coverage was not detected at the bottom of the THV cusp where thrombus is usually observed. Instead, it developed along with the THV frame, suggesting the infiltration of the endothelium. Even if the coverage was thrombus, the same intervention was applicable since anticoagulation therapy had not improved DCO.

Because of low STJ height and the high implantation of EvolutR, the gap between the leaflet and STJ was only 3.0 mm. A minimal gap between the leaflet and STJ could be the underlying cause for endothelialization of the gap followed by complete occlusion of the blood flow path from the aorta into the LCS.

In a previous case report of DCO due to THV endothelialization, the patient was treated surgically.4 In our case, we first determined whether PCI was a possible treatment option for DCO due to THV endothelialization by implanting stents in the roundabout route and making a new orifice in the NCS.

![Video 3](https://academic.oup.com/ehjcr/advance-article/doi/10.1093/ehjcr/ytaa288/5903169)

**Video 3** The second wire insertion into the left coronary artery.

![Figure 4](https://academic.oup.com/ehjcr/advance-article/doi/10.1093/ehjcr/ytaa288/5903169)

**Figure 4** Diagrams (A–C) and fluoroscopic images of percutaneous coronary intervention (D–F). (A) Since we could not penetrate the coverage with a coronary guide wire (arrow), we advanced the wire through the roundabout route from the non-coronary sinus into the left coronary sinus. However, we could not advance the wire into the left coronary artery. (B) Using a dual-lumen microcatheter, we inserted the second coronary guide wire into the left coronary artery. (C) We deployed a zotarolimus-eluting stent into the roundabout route. (D) Fluoroscopic image just before stent implantation. (E and F) Final coronary angiography (right caudal view and left cranial view, respectively).
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 image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.
References
1. Ribeiro HB, Webb JG, Makkar RR, Cohen MG, Kapadia SR, Kodali S et al. Predictive factors, management, and clinical outcomes of coronary obstruction following transcatheter aortic valve implantation: insights from a large multicenter registry. J Am Coll Cardiol 2013;62:1552–1562.
2. Ribeiro HB, Nombela-Franco L, Urena M, Mok M, Pasian S, Doyle D et al. Coronary obstruction following transcatheter aortic valve implantation: a systematic review. JACC Cardiovasc Interv 2013;6:452–461.
3. Jabbour RJ, Tanaka A, Finkelstein A, Mack M, Tamburino C, Van Mieghem N et al. Delayed coronary obstruction after transcatheter aortic valve replacement. J Am Coll Cardiol 2018;71:1513–1524.
4. Ninomiya Y, Hamasaki S, Nomoto Y, Kawabata T, Fukumoto D, Yoshimura A et al. A case of acute coronary syndrome caused by delayed coronary ischemia after transcatheter aortic valve implantation. J Cardiol Cases 2018;17:107–110.
5. Mangi AA, Ramchandani M, Reardon M. Surgical removal and replacement of chronically implanted transcatheter aortic prostheses: How I teach it. Ann Thorac Surg 2018;105:12–14.
6. Jose J, Sulimov DS, El-Mawardy M, Sato T, Allali A, Holy EW et al. Clinical bioprosthetic heart valve thrombosis after transcatheter aortic valve replacement: incidence, characteristics, and treatment outcomes. JACC Cardiovasc Interv 2017;10:686–697.
7. Chakravarti T, Sandergaard L, Friedman J, De Backer O, Berman D, Kofod KF et al. Subclinical leaflet thrombosis in surgical and transcatheter bioprosthetic aortic valves: an observational study. Lancet 2017;389:2383–2392.
8. Yanagisawa R, Hayashida K, Yamada Y, Tanaka M, Yashima F, Inohara T et al. Incidence, predictors, and mid-term outcomes of possible leaflet thrombosis after TAVR. JACC Cardiovasc Imaging 2017;10:1–11.