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CASE REPORT

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Unusual cause of myocardial infarction following transcatheter aortic valve replacement

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Key Clinical Message

Left coronary artery embolism from aortic valve leaflet tissue mass is a rare but potentially life-threatening complication following transcatheter aortic valve replacement. It is important for interventional cardiologists to be aware of this rare complication for rapid identification and prompt treatment which is the key to a successful outcome.

Abstract

An 81-year-old female presented for elective transcatheter aortic valve replacement (TAVR) for severe low-flow low-gradient aortic stenosis. Immediately post-procedure, she developed unexplained, persistent hypotension. There was no bleeding. There was no aortic injury. Activated clotting time was in therapeutic range. Coronary angiography revealed hazy filling defects in left anterior descending and left circumflex. Intravascular ultrasound showed heterogeneous, hypoechoic mass with mild calcification consistent with embolized valve leaflet tissue. This was treated with emergent percutaneous coronary intervention with excellent results. Left coronary artery embolism from aortic valve leaflet tissue is a rare, but potentially life-threatening complication following TAVR. Prompt recognition is key to a successful outcome.

KEYWORDS

coronary thrombosis, left coronary artery embolism, myocardial infarction (MI), transcatheter aortic valve replacement (TAVR)

1 | INTRODUCTION

We present an unusual case of myocardial infarction following transcatheter aortic valve replacement (TAVR).

2 | CASE DESCRIPTION

An 81-year-old female presented for planned TAVR for symptomatic severe aortic stenosis. Past medical history included chronic systolic congestive heart failure (CHF) (left ventricular ejection fraction [LVEF] 35%–40%), left bundle branch block (LBBB), hypertension, chronic kidney disease Stage III, chronic obstructive pulmonary disease, and remote history of pulmonary embolism on lifelong anticoagulation with warfarin.

2.1 | Preoperative imaging

2.1.1 | Echocardiogram

Transthoracic echocardiogram (TTE) showed left ventricular ejection fraction (EF) 35–40% with abnormal septal

motion due to baseline LBBB. Low dose dobutamine stress echocardiogram showed aortic valve area (AVA) 0.8 cm^2 and mean gradient (MG) 24 mmHg at rest and AVA 0.6 cm^2 and MG 40 mmHg at peak dobutamine infusion (20 mcg/kg/min); hence, a diagnosis of low-flow low-gradient severe aortic stenosis was made.

2.1.2 | Cardiac catheterization

Cardiac catheterization showed right dominant circulation with normal coronary arteries without any significant coronary artery disease (Figure 1, Video S5).

2.1.3 | Computed tomography (CT) chest abdomen and pelvis

Gated CT chest abdomen and pelvis showed adequate transfemoral access for TAVR and anatomic suitability for TAVR with 23 mm balloon expandable valve with adequate sinus of Valsalva diameters (24.7 × 28.6 × 28.5 mm for right, left, and non-coronary sinus, respectively), adequate coronary heights, mild leaflet calcification and overall low risk of coronary obstruction (Figure S1).

2.2 | Pre-procedure planning

Based on the imaging studies, transfemoral TAVR with balloon expandable valve [23 mm Sapien 3 Ultra (Edwards Lifesciences)] was planned under monitored anesthesia care. Warfarin was held 5 days prior to the planned procedure with international normalized ratio (INR) value drifting down from 2.4 six days prior to the procedure to 1.3 on the day of the procedure.

2.3 | Procedure description

Bilateral femoral arterial and right femoral venous access were obtained under ultrasound guidance. Temporary pacing wire was inserted via right femoral vein access. A 5 Fr pigtail catheter was advanced to the right coronary sinus via right femoral arterial access. Preclosure of the left femoral artery was performed using standard technique following which a 14 Fr valve delivery was inserted. Intraprocedural anticoagulation was accomplished with intravenous heparin and activated clotting time (ACT) greater than 250 s was confirmed prior to valve insertion. Following standard technique, the 23 mm Sapien 3 Ultra (Edwards Lifesciences) valve was positioned at the level of the aortic annulus and deployed under rapid ventricular pacing (Video S1). TTE following valve deployment showed EF 35%–40% and well-functioning TAVR valve with no paravalvular leak. The valve delivery system and the 14 Fr sheath were removed from the left femoral access site and adequate hemostasis was achieved.

2.4 | Unusual complication

Soon after, the patient started developing significant unexplained hypotension requiring vasopressor support with frequent vasopressor boluses. Contralateral angiogram of left femoral access site performed via right femoral access showed no evidence of bleeding from the large bore access site. Repeat TTE confirmed no pericardial effusion and left ventricular function and wall motion appeared unchanged. Three-lead electrocardiogram (EKG) on the monitor did not demonstrate any changes other than the baseline LBBB. Aortogram performed via pigtail catheter positioned in the ascending aorta did not reveal any aortic or LVOT injury or aortic dissection; however, there was possibility of decreased

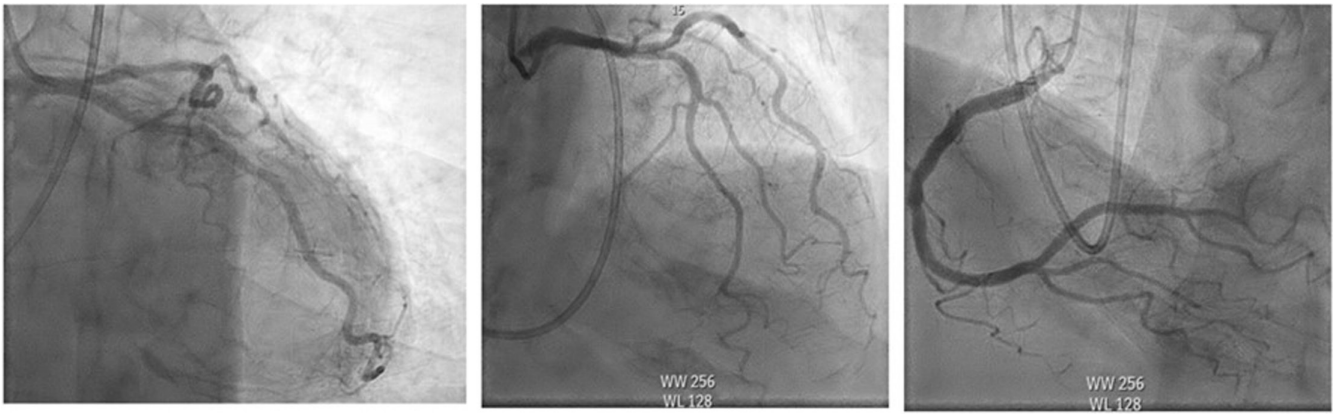

FIGURE 1 Preoperative coronary angiogram showing normal coronary arteries.

flow in the left circumflex (LCX) on nonselective imaging (Video S2). Given ongoing hemodynamic instability and no other apparent explanation for the same, we decided to perform coronary angiography. ACT greater than 250 s was reconfirmed at this time.

Right coronary angiography did not reveal any notable changes from baseline. Left coronary angiography showed 100% thrombotic occlusion in proximal LCX with a visible filling defect as well as large, hazy filling defect in proximal left anterior descending (LAD) causing 80%–90% hazy stenosis with thrombolysis in myocardial infarction (TIMI)-2 flow in distal LAD (Figure 2A, Video S6).

2.5 | Complication management

Left main (LM) was engaged using a 6 Fr Judkins Left (JL) 4 guide and both LAD and LCX were wired using two 0.014″ workhorse wires. Percutaneous transluminal coronary angioplasty (PTCA) of proximal LCX followed by proximal LAD was performed using a 3.0 mm non-compliant (NC) balloon. An Export Advance™ (Medtronic) coronary aspiration catheter was introduced in the LCX; however, no thrombus was retrieved. Subsequently, percutaneous coronary intervention (PCI) of ostial to proximal LCX was performed using a 3.5 × 32 mm drug-eluting stent (DES). Repeat angiogram showed restoration of TIMI-3 flow in the LCX; however, there was shift of embolized material to ostial LAD (Figure 2B). At this time, we decided to proceed with intravascular ultrasound (IVUS) imaging. IVUS of LCX showed well-apposed and expanded stent without any edge dissection (Video S3). IVUS of LAD showed heterogeneous hypoechoic mass with mild calcification in ostial LAD (Figure 3, Video S7). Subsequently, DES PCI of distal LM to proximal LAD was performed using a 3.5 × 20 mm DES after removing the LCX wire. Subsequently, the LCX was recrossed, and final kissing balloon inflation was performed using two 3.5 mm NC balloons in LAD and LCX, each inflated at nominal pressure (Figure 2C). Finally, proximal optimization technique (POT) was performed using 4.0 × 8 mm NC balloon in distal LM inflated at nominal pressure (Figure 2D). Final IVUS showed excellent stent expansion and apposition in LAD and LM (Video S8). Final angiogram showed widely patent stents with TIMI-3 flow in LCX and TIMI-2 flow in LAD (Figure 4, Video S8). An intra-aortic balloon pump (IABP) was inserted via right femoral access.

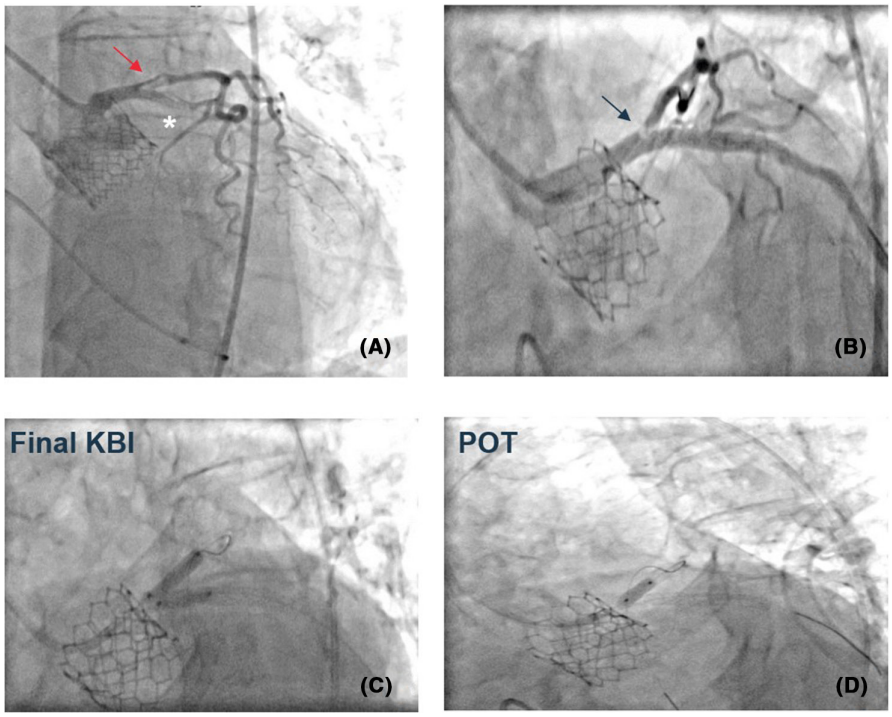

FIGURE 2 Post-procedure coronary angiogram and interventional management. (A) Post-procedure left coronary angiogram showing hazy filling defect with 100% proximal left circumflex occlusion (white asterisk) as well as hazy filling defect in proximal to ostial left anterior descending (LAD) resulting in 90% stenosis (red arrow). (B) After left circumflex stenting, there was shift of embolized mass to ostial LAD (black arrow). (C) After distal left main to LAD stenting, the left circumflex was recrossed and final kissing balloon inflation (KBI) was performed using two 3.5 mm non-compliant balloons in LAD and left circumflex. (D) Proximal optimization technique (POT) with 4.0 × 8 mm non-compliant balloon in distal left main.

2.6 | Postoperative course

The patient continued to improve clinically and did not require any further vasopressor support. IABP was weaned and removed the next day. The patient was discharged home on the fourth postoperative day. Warfarin was resumed at discharge. At 1 month follow-up, she reported improvement in her symptoms with TTE showing EF 40% and normal TAVR valve function.

3 | DISCUSSION

We report an unusual cause of myocardial infarction following TAVR that was thromboembolic in nature and occurred immediately after valve deployment. Potential origins of this thromboembolic mass in LAD and LCX in our patient in order of increasing likelihood include as follows: (1) undiagnosed left atrial appendage (LAA) or left ventricular (LV) thrombus that dislodged and embolized into the LM; (2) thrombus formation on left ventricular wire or TAVR valve hardware that embolized into the LM after valve deployment; and (3) valve leaflet tissue mass that embolized into the LM. Our patient did not have a history of atrial fibrillation or flutter making LAA thrombus unlikely. Preoperative echocardiogram was performed with contrast echocardiography and did not reveal any evidence of LV thrombus. Given patient's history of coagulation disorder, it is possible that there may have been thrombus formation on wire or valve hardware despite intraprocedural heparin; however, ACT values greater than 250 s were confirmed on multiple occasions. Therefore, the most likely etiology of this complication was embolized valve leaflet tissue mass into the LM. IVUS images obtained in the LAD revealed that the embolized mass was heterogeneous with hypoechoic areas as well as evidence of mild calcification, making it more likely to be leaflet tissue mass rather than thrombus.

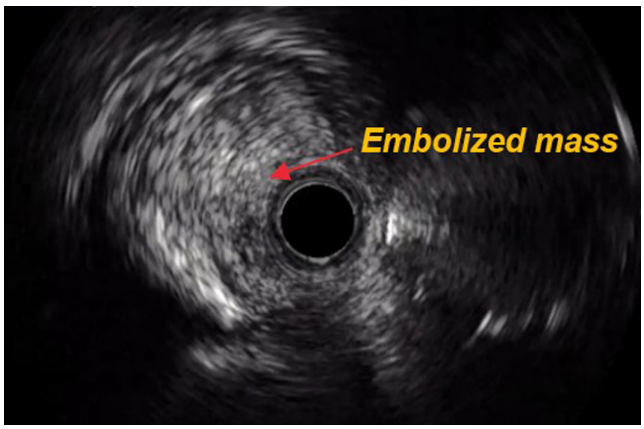

FIGURE 3 Still image of intravascular ultrasound (IVUS) of left anterior descending (LAD) showing heterogeneous, hypoechoic mass in ostial LAD with mild calcification, consistent with embolized valve leaflet tissue.

There are prior reports of unexpected LM embolism from dislocated valve leaflet tissue.[1–3] In these studies, IVUS showed a heterogeneous, hypoechoic mass,[1,2] and calcified leaflet material,[3] similar to the IVUS findings in our case. Optical coherence tomography (OCT) was performed in the study by Katagiri et al[2] which showed a mixture of calcification and lipid that was likely part of the aortic valve. In the first two reports,[1,2] there was a mobile mass attached to aortic cusp after balloon aortic valvuloplasty which subsequently embolized to the LM after TAVR deployment. This was identified and treated promptly while the patients were still on the table. In the third report, the patient developed hypotension and ST-elevation myocardial infarction 16 h following balloon-expandable valve (BEV) implantation, with urgent coronary angiography showing hazy high-grade stenosis at distal LM bifurcation that was confirmed on IVUS to be irregular and mobile calcified leaflet tissue.[3] This was treated successfully with simultaneous kissing stent technique. In all three cases, PCI was successful with the aid of intra-coronary imaging and the patients had an excellent clinical and hemodynamic recovery. In all three cases as

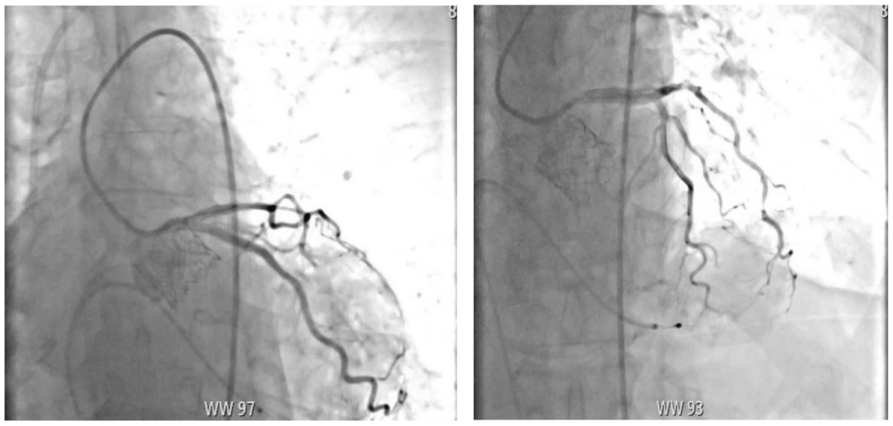

FIGURE 4 Final left coronary angiogram after left main, left anterior descending, and left circumflex stenting in caudal (left image) and cranial (right image) views.

well as in our case, BEV was used for TAVR, hence making this rare complication likely to occur with forced valve expansion with BEV or aggressive predilatation (e.g., balloon aortic valvuloplasty) and unlikely to occur with the use of a self-expanding valve (SEV) without predilatation.

Our case was further complicated by the presence of baseline cardiomyopathy and LBBB making EKG changes or wall motion abnormalities indiscernible. Fortunately, we considered performing coronary angiography due to persistent, recurrent hypotension with no apparent etiology; and this prompted immediate recognition as well as quick and timely treatment of left coronary artery embolism from aortic leaflet tissue. Our report highlights the importance of early recognition of this unusual complication as well as the importance of maintaining coronary interventional skills for structural interventional cardiologists who may encounter this rare but potentially fatal complication.

4 | CONCLUSION

Left coronary artery embolism from aortic valve leaflet tissue is a rare, but potentially life-threatening complication following TAVR. This can occur despite therapeutic anticoagulation. Prompt recognition is key to a successful outcome.

AUTHOR CONTRIBUTIONS

Neeraj Shah: Conceptualization; data curation; investigation; resources; writing – original draft; writing – review and editing. **L. Wiley Nifong:** Supervision; validation; writing – review and editing.

FUNDING INFORMATION

None.

CONFLICT OF INTEREST STATEMENT

The authors have no conflict of interest to declare.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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