

# Obstruction of 2 Coronary Arteries

from Different Causes Immediately after  
Transapical Transcatheter Aortic Valve Replacement

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*Coronary obstruction, a rare complication of transcatheter aortic valve replacement, can be fatal. Few data exist on this phenomenon, and, to date, authors have reported only single coronary lesions. We present a case in which 2 coronary arteries obstructed immediately after transapical transcatheter aortic valve replacement.*

*The patient was an 81-year-old woman with symptomatic severe aortic stenosis who underwent transapical transcatheter aortic valve replacement. Immediately after an Edwards SAPIEN XT valve was deployed, she experienced sudden cardiogenic shock resulting from obstruction of the left main coronary artery ostium and the distal left anterior descending coronary artery. The left main obstruction was caused by direct compression from a large calcified mass and the valve frame. The left anterior descending coronary artery obstruction was caused by ambient myocardial tightening and external compression around the apical sutures. Revascularization was achieved through coronary stent placement and suture removal, respectively. Our patient's case highlights the risk for coronary obstructions after transapical transcatheter aortic valve replacement, and we discuss how they can be managed. (Tex Heart Inst J 2020;47(1):30-4)*

**Key words:** Aged, 80 and over; aortic valve stenosis/complications; coronary occlusion/diagnosis/therapy; heart valve prosthesis implantation/adverse effects; percutaneous coronary intervention; suture techniques/adverse effects; transcatheter aortic valve replacement/adverse effects/methods; treatment outcome

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**T**ranscatheter aortic valve replacement (TAVR) has become an alternative to conventional surgery in high-risk patients who have symptomatic severe aortic stenosis.<sup>1,2</sup> The incidence of coronary obstruction, a catastrophic complication of TAVR, is estimated to be 0.3% to 0.7%.<sup>3</sup> The prognosis is poor, few data are available, and reported cases have been of single lesions only. The most frequently reported potential mechanism is compression of the left main coronary artery (LMCA) ostium caused by a displaced calcified nodule or native aortic leaflet.<sup>3,4</sup> We report a case of 2 coronary obstructions after transapical TAVR. We discuss their different causes and how we managed them.

## Case Report

In April 2016, an 81-year-old woman was referred to our institution for treatment of symptomatic severe aortic stenosis (New York Heart Association functional class III). She had a pacemaker because of complete atrioventricular block. Her baseline electrocardiogram (ECG) showed left bundle branch block associated with right ventricular pacing (Fig. 1). A baseline transthoracic echocardiogram showed severe degenerative aortic stenosis and a severely calcified aortic valve (valve area, 0.77 cm<sup>2</sup>; peak and mean gradients, 92 and 56 mmHg, respectively; and left ventricular [LV] ejection fraction, 0.65). Multidetector computed tomograms (MDCT) showed an aortic annular diameter of 21.6 to 25.6 mm and an annular area of 469 mm<sup>2</sup>. The left sinus of Valsalva was large (diameter, 31.8 mm). The ostial height of the left coronary artery was low, at 10.3 mm from the annulus ring, and that of the right coronary was 12.8 mm. The right and noncoronary cusps were moderately calcified; the left coronary cusp (LCC) was only mildly so (Fig. 2A). There was no substantial stenosis in the left or right coronary artery. A bulky, calcified nodule was seen in the abdominal aorta, in addition to severe arterial degeneration known as “shaggy aorta” (Fig. 2B).

The patient's estimated operative death risk in terms of the logistic EuroSCORE was 12.4%, and her Society of Thoracic Surgeons score was 9.3%; therefore, we decided to perform TAVR. The calcified nodule and shaggy aorta made a transfemoral approach unsuitable because of the risk of distal embolization and acute aortic dissection. With the aid of MDCT and transthoracic echocardiography, we decided on a transapical

approach through a left anterolateral minithoracotomy in the 5th intercostal space (Fig. 2C).

The patient was placed under general anesthesia, and transesophageal echocardiography was used for guidance. After identifying the left anterior descending coronary artery (LAD) on echocardiography and by sight, we approached the LV apex directly, then placed double triangular, apical, pledgeted sutures in the apical cannulation site, which was lateral to the LAD. Using a large suture needle (½ circle, 36 mm long), we made a puncture lateral to the apex and placed a 24F Ascendra™ valve replacement system with introducer sheath (Edwards Lifesciences Corporation) in the LV. After using a 20-mm balloon for valvuloplasty, we successfully implanted a 26-mm Edwards SAPIEN XT® prosthesis (Edwards Lifesciences) during rapid ventricular pacing. However, immediately after valve deployment, the patient became persistently hypotensive (blood pres-

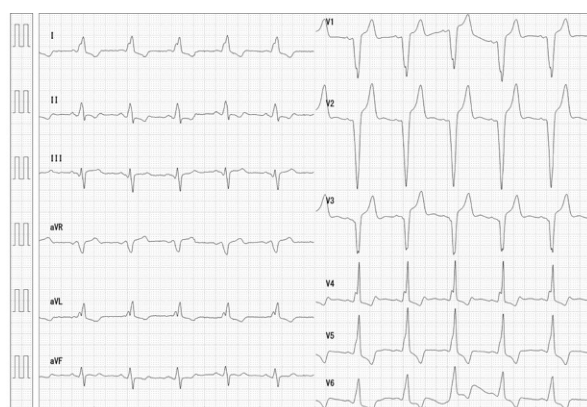
sure, 78/40 mmHg). Infused inotropic drugs raised her blood pressure to 104/64 mmHg, averting the need for cardiopulmonary bypass. Aortic angiograms showed a filling defect in the LMCA ostium and slow LAD filling. There was no change from baseline in the patient's ECG, nor additional ischemic change. However, LV apical wall motion was severely depressed.

Strongly suspecting an obstruction at the LMCA ostium, we acquired an emergency coronary angiogram. It confirmed subtotal obstruction of the LMCA ostium—and, unexpectedly, showed complete obstruction of the distal LAD (Fig. 3A). An intravascular ultrasonogram showed a hyperechoic mass compressing the LMCA ostium. The mass had a minimum luminal area of 2.87 mm<sup>2</sup> and a minimum luminal diameter of 1.2 × 3 mm (Fig. 3B). To treat this obstruction, we implanted a 3.5 × 18-mm Nobori® biolimus-eluting stent (Terumo Medical) at a pressure of 14 atm and postdilated it with a 4.5-mm noncompliant balloon catheter at the same pressure, achieving a good outcome (Fig. 3C).

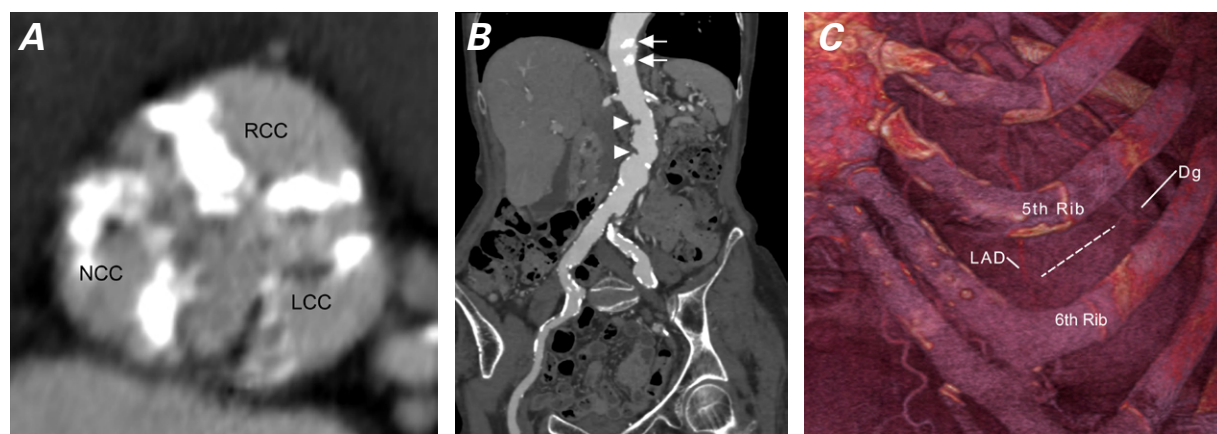
Treating the distal LAD obstruction was more challenging. Our attempts at intracoronary vasodilator infusion and coronary guidewire-crossing were unsuccessful. When we saw that one apical suture had been placed close to the LAD (Fig. 3C), we suspected that external compression around it was causing obstruction. We removed the suture, and the LAD obstruction promptly resolved (Fig. 3D). Although the first purse-string suture was not sufficient to control bleeding from the puncture hole in the ventricle, adding purse-string sutures more lateral to the first restored hemostasis.

Thereafter, the patient's blood pressure and LV wall motion rapidly improved. Remediating both obstructions took approximately 50 min.

Postprocedural MDCT showed the displaced calcified mass and valve frame over the LMCA ostium,



**Fig. 1** Baseline electrocardiogram shows right ventricular pacing with left bundle branch block.



**Fig. 2** Preprocedural multidetector computed tomograms show **A)** a left coronary cusp (LCC) that is less calcified than the noncoronary cusp (NCC) and the right coronary cusp (RCC), and **B)** a bulky, calcified nodule (arrows) in the abdominal aorta, and a shaggy aorta (arrowheads). **C)** Intraoperative multidetector computed tomogram (3-dimensional reconstruction) shows the site of left anterolateral minithoracotomy in the 5th intercostal space (dotted line).

Dg = major diagonal branch; LAD = left anterior descending coronary artery

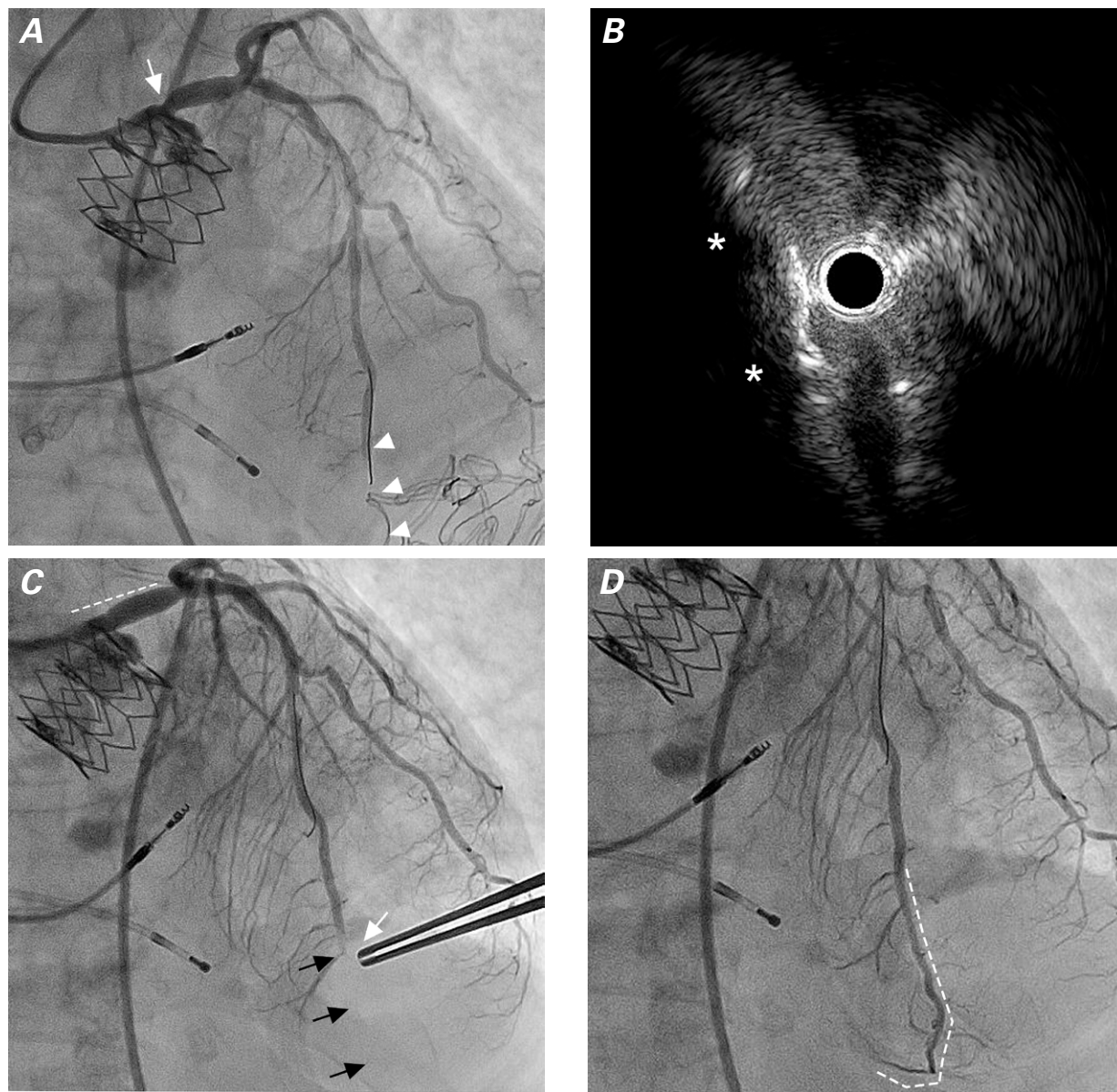
without aortic dissection or hematoma (Fig. 4A). Notably, no remaining stenosis or periprocedural injury was seen at the previously obstructed portion of the LAD (Fig. 4A), and the reconstructed apical suture was adjacent to but did not impinge upon the distal LAD (Fig. 4B).

An echocardiogram showed a well-seated prosthesis (mean transvalvular gradient, 10 mmHg), mild paravalvular leak, and no LV asynergy. The patient's cardiac enzyme levels did not increase substantially; she needed only 0.04 µg/kg/min of noradrenaline. She remained hemodynamically stable, and the inotropic drug was

stopped the next day. She spent 4 days in intensive care, had an uneventful clinical course, and was discharged from the hospital after 8 days in total. At her 12-month follow-up examination, she was in stable condition and in New York Heart Association functional class I.

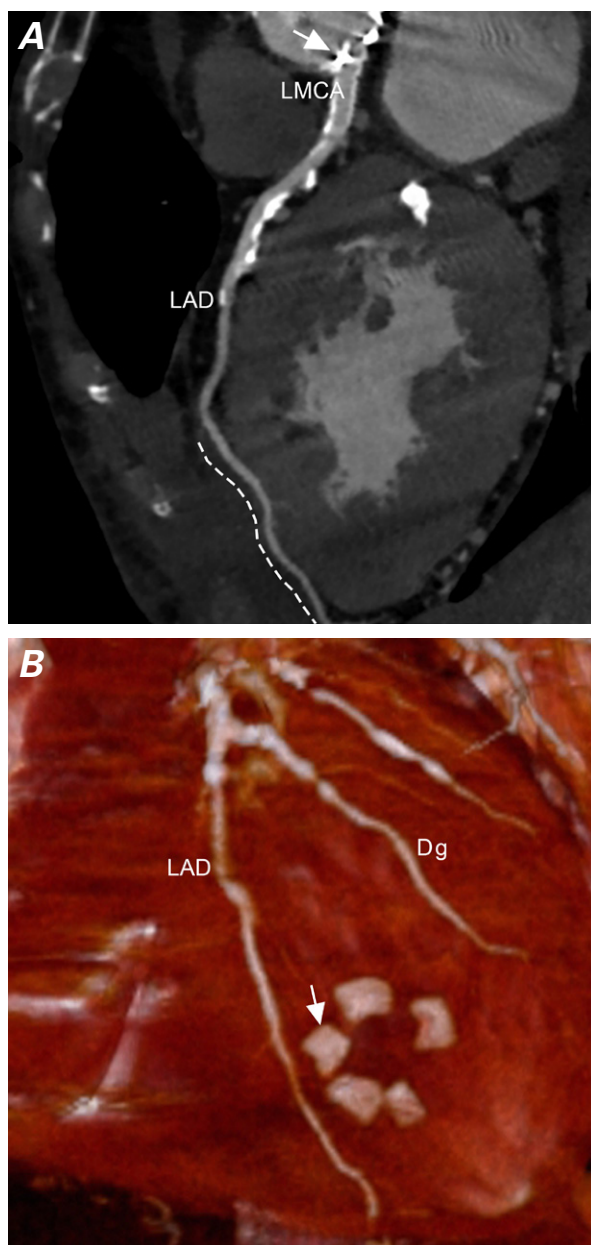
## Discussion

We have seen no other reports about 2 coronary artery obstructions from different causes after transapical TAVR. The LMCA ostial obstruction, a result of direct compression from a calcified mass and the newly im-



**Fig. 3** **A)** Coronary angiogram after valve implantation shows obstruction of the left main coronary artery (LMCA) ostium (arrow) and complete obstruction of the distal left anterior descending artery (LAD) (arrowheads). **B)** Intravascular ultrasonogram shows the hyper-echoic mass (asterisks) compressing the LMCA ostium (minimum luminal area, 2.87 mm<sup>2</sup>; minimum luminal diameter, 1.2 × 3 mm). **C)** Coronary angiogram shows good stent expansion in the LMCA (dotted line) and a completely obstructed distal LAD (black arrows). White arrow and forceps indicate the apical suture nearest the LAD. **D)** Final coronary angiogram shows resolution of LAD obstruction (dotted line).





**Fig. 4** Postprocedural multidetector computed tomograms show **A**) the displaced calcified mass and the valve frame at the ostium of the left main coronary artery (LMCA) (arrow), no substantial stenosis or avulsion in the previously obstructed distal left anterior descending coronary artery (LAD) (dotted line), and **B**) the reconstructed apical suture near the distal LAD (arrow).

Dg = major diagonal branch

planted valve frame, was resolved by coronary stenting. The second lesion, an LAD obstruction from ambient myocardial tightening and external compression, was resolved by removing the pertinent apical suture.

Coronary obstruction during TAVR can be fatal. Potential mechanisms include displacement of calcified nodules or native aortic leaflets, aortic root hematoma, high implantation of a valvular prosthesis, coronary

spasm, and embolization of calcific debris.<sup>5-7</sup> During TAVR, obstruction of the ostial LMCA has been the most frequent and catastrophic coronary complication. Predisposing factors include bulky leaflet calcifications, a low-lying coronary ostium (height, <12 mm), and shallow sinuses of Valsalva (diameter, <30 mm).<sup>8</sup> We considered protecting our patient's left coronary artery by using a coronary guidewire and balloon catheter before we deployed the valve. However, her left sinus of Valsalva was relatively large, and her LCC was only mildly calcified, so we did not take these steps. The low height (10.3 mm) of her left coronary artery was presumably the main cause of the LMCA obstruction.

We found only 2 reports of indirect LAD obstruction during transapical TAVR.<sup>9,10</sup> In acute distal LAD obstruction during transapical TAVR, possible mechanisms are external compression, periprocedural injury, coronary spasm, and distal coronary embolization. In our patient, our attempts at intracoronary vasodilator infusion and coronary guidewire-crossing were not successful in restoring blood flow, and postprocedural MDCT had ruled out direct LAD injury from the apical sutures or distal coronary embolization (Fig. 4). During the procedure, however, one suture had been placed adjacent to the patient's LAD, a large suture needle (½ circle, 36 mm long) had been used, and the needle had been obliquely inserted into the myocardium, all of which could have caused excessive ambient myocardial tightening and external compression, thus leading to indirect LAD obstruction.

In addition, from a technical perspective, the apical suture and sheath should be placed to prevent impingement upon the LAD. When the transapical approach is used in TAVR, inserting the sheath at the apicolateral area of the LV has been recommended because a more lateral or true apical approach can cause complications.<sup>9</sup> In our patient, a preprocedural 3-dimensional CT reconstruction image of the left chest wall (Fig. 2C) showed that the left anterolateral minithoracotomy surgical area was enclosed by the LAD, a major diagonal branch, and ribs. In hindsight, the operative field was substantially limited, precluding proper placement of the sheath and apical sutures.

The best treatment of indirect LAD obstruction caused by apical sutures is unclear. In the 2 previous cases,<sup>10,11</sup> the operators used coronary stenting and apical suture removal, with subsequent infusion of a selective vasodilator. If external compression is suspected as the main cause, removing the suture may be a simple and reasonable treatment by itself.

Our patient had a permanent pacemaker because of complete atrioventricular block. Her baseline ECG showed left bundle branch block in association with a right ventricular pacing pattern. Because there were no significant ischemic changes on the surface ECG, diagnosing the coronary obstructions was difficult. Only

the emergency coronary angiogram revealed subtotal obstruction of the LMCA ostium—and, unexpectedly, complete obstruction of the distal LAD.

Having encountered these simultaneous coronary obstructions during transapical TAVR, we recognized the importance of promptly identifying their causes and appropriately managing them.

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