ESC European Society of Cardiology

European Heart Journal - Case Reports (2018) **2**, 1–7
doi:10.1093/ehjcr/yty112

CASE REPORT

Acute left main stem coronary occlusion following transcatheter aortic valve replacement in a patient without recognized coronary obstruction risk factors: a case report

Roberto Spina, Omar Khalique, Isaac George, and Tamim Nazif*

Structural Heart and Valve Center, New York Presbyterian Hospital/Columbia University Medical Center, 177 Fort Washington Ave, New York, NY 10032, USA

Received 4 April 2018; accepted 4 September 2018; online publish-ahead-of-print 23 October 2018

Background Acute coronary obstruction following transcatheter aortic valve replacement (TAVR) is an uncommon but life-threatening event.

Case summary A 78-year-old man developed acute left main obstruction following transfemoral TAVR with a balloon-expandable valve. Cardiac arrest ensued, requiring emergent peripheral cardiopulmonary bypass. Percutaneous coronary intervention (PCI) to the left main coronary artery was performed with one drug-eluting stent. Intravascular ultrasound (IVUS) demonstrated focal underexpansion of the stent in its proximal segment which was not responsive to high-pressure non-compliant balloon dilatation, suggesting stent compression from either valve strut or calcific native leaflet. Therefore, to increase radial strength of the scaffolding at the site of compression, we deployed a second stent within the first stent, and further expanded that segment with high-pressure balloon inflations. Final IVUS demonstrated better expansion of the focally compressed segment. Following PCI, left ventricular function normalized completely. The patient was discharged from hospital on Day 3 post-procedure. At 12 weeks follow-up, his dyspnoea had improved significantly, and follow-up transthoracic echocardiography demonstrated normal left ventricular systolic function and normal aortic valve function.

Discussion Established risk factors for coronary ostial occlusion include a short distance between the aortic annulus and the coronary ostia (<10 mm) and a narrow aortic root (<28 mm at the sinuses of Valsalva). These two factors increase the likelihood that the native valve leaflets are displaced over and obstruct the coronary ostia when the aortic bioprosthesis is deployed. Perplexingly, our patient did not present with any of the recognized risk factors for acute coronary occlusion, suggesting other factors might be at play. We suggest that a leaflet length to coronary sinus height ratio greater than 1 might be an additional useful predictor of coronary occlusion during TAVR. In addition, we suggest that if residual focal stent compression from either valve strut or calcific leaflet exists after stent deployment and the latter is resistant to balloon dilatation, deploying a second concentric layer of stent might improve the radial strength of the scaffolding and improve overall stent expansion.

Keywords TAVR • TAVI • Aortic stenosis • Coronary obstruction • Acute coronary occlusion • Case report

* Corresponding author. Tel: +1 212 342 0444, Fax: +1 646 426 0204, Email: tmn31@columbia.edu. This case report was reviewed by Eugenio Caradonna, Marco De Carlo, Nikolaos Bonaros, Cemil Izgi, Christian Fielder Camm, Mark Philip Cassar, and Peregrine Green.

Learning points

- Acute coronary obstruction following transcatheter aortic valve replacement (TAVR) is an uncommon but life-threatening event with a high mortality rate. Established risk factors for coronary ostial occlusion include a short distance between the aortic annulus and the coronary ostia (<10 mm) and a narrow aortic root (<28 mm at the sinuses of Valsalva).
- Although coronary protection may mitigate the risk of adverse events in patients with low-lying coronary ostia or small sinuses of Valsalva, this complication may occur in the absence of well-established high-risk features, suggesting other factors might be at play. A leaflet length to coronary sinus height ratio greater than 1 might be an additional useful predictor of coronary occlusion during TAVR.
- If residual focal stent compression from either valve strut or calcific aortic leaflet persists after stent deployment and the latter is resistant to balloon dilatation, deploying a second concentric layer of stent might improve the radial strength of the scaffolding and improve overall stent expansion.

Introduction

Transcatheter aortic valve replacement (TAVR) has emerged in the last decade as the accepted treatment modality for severe symptomatic aortic valve stenosis in patients who are inoperable,[1,2] and as equivalent to surgery for patients whose risk of mortality is moderate or high from surgical aortic valve replacement.[3–5] Albeit being a less invasive therapeutic modality compared to surgery, the TAVR procedure is associated with potentially serious complications, including death, aortic rupture or dissection, pericardial tamponade, acute coronary obstruction, stroke, and flow-limiting peripheral arterial dissection.[1–5] Acute coronary obstruction following TAVR is an uncommon but life-threatening event with a high morbidity and mortality rate.

Timeline

Time	Events
Initial presentation [elective transcatheter aortic valve replacement (TAVR)]	Admitted for transfemoral TAVR under general anaesthesia with transoesophageal guidance in the hybrid operating room.
Procedure (day 0)	Balloon aortic valvuloplasty. Deployment of Edwards Sapien 29 mm aortic bioprosthesis under rapid ventricular pacing. Blood pressure fails to rise above 50/20 mmHg post-valve deployment. On transoesophageal echocardiography, the anterior wall of the left ventricle appeared severely hypokinetic. On aortography, the left coronary system failed to opacify, suggesting acute left main stem coronary obstruction. Cardiac arrest ensued, and peripheral cardiopulmonary bypass was initiated emergently. Percutaneous coronary intervention to the ostium of the left main coronary stem was performed with two concentric layers of drug-eluting stents deployed with intravascular ultrasound guidance and expanded at high pressure with non-compliant balloons. At the completion of the procedure, left ventricular function had normalized, and haemodynamic parameters had stabilized.
Day 1	Extubated, transferred to the cardiology ward from the intensive care unit
Day 3	Recovers uneventfully, discharged home
Day 7	Re-admitted to hospital with light-headedness. Bradycardia with second degree heart block. Insertion of permanent pacemaker. Discharged home the following day.
One month post-procedure: follow-up in cardiologist office	The patient feels well. Repeat transthoracic echocardiogram demonstrates normal left ventricular systolic function.

Patient information/diagnostic assessment/follow-up and outcomes

A 78-year-old male with a background of hypertension, cerebrovascular disease with two transient ischaemic attacks in 2015, and severe symptomatic aortic stenosis was evaluated by a multidisciplinary team in an outpatient setting. He had developed progressively worse dyspnoea on exertion in the 6 months prior to evaluation.

On physical examination, the pulse rate was 65 b.p.m., and the blood pressure 135/76 mmHg. The jugular venous pressure was not raised. An ejection-systolic murmur, 3/6 in intensity and radiating to the carotids, was auscultated in the aortic area. The lungs were resonant to percussion, and clear to auscultation. The abdomen was soft and non-tender to palpation. There was no peripheral oedema. Transthoracic echocardiography demonstrated normal left and right ventricular size and systolic function, severe aortic stenosis (peak

velocity= 4.1 m/s; mean transvalvular gradient= 37 mmHg; estimated valve area= 0.90 cm^2) and no other significant valvular abnormality. A 12-lead electrocardiogram demonstrated sinus rhythm with no conduction abnormalities.

He was deemed at low risk for surgical aortic valve replacement, with a Society of Thoracic (STS) score of 3%. He agreed to participate in a clinical trial comparing transcatheter to surgical aortic valve replacement in patients at low risk of mortality from aortic surgery, and was randomized to the TAVR arm. A recommendation for transfemoral TAVR with a balloon-expandable bioprosthesis was made, consistent with institutional preference. Contrast-enhanced computed tomography (CT)—derived annular area and perimeter measurements were 580 mm^2 and 87 mm, respectively, mandating a 29 mm Edwards Sapien 3 aortic bioprosthesis (*Figure 1A*). Careful analysis of the CT reconstructions revealed adequate coronary ostia heights and adequate sinuses of Valsalva dimensions (*Figure 1B*, *D*, and *E*). Computed tomography revealed bulky leaflet calcification and moderate left ventricular outflow tract calcium on the non-coronary cusp (*Figure 1C*). The latter finding prompted the use transoesophageal echocardiography (TOE) during valve deployment. The procedure was performed under general anaesthesia in the hybrid operating room. Balloon aortic valvuloplasty was performed with an Edwards Sapien 25 mm balloon. The 29 mm ES3 bioprosthesis was deployed under rapid pacing in a satisfactory position (*Figure 2A* and *B*, Supplementary material online, *Video S1*). Immediately following deployment, the systolic blood pressure failed to rise above 50 mmHg. Transoesophageal echocardiography ruled out pericardial effusion, annular rupture or aortic dissection, and initially at least, the left main stem coronary flow appeared intact. Within less than a minute, however, the left ventricular anterior wall appeared significantly hypokinetic, and on root aortography, the left coronary system failed to opacify. Ventricular tachycardia and fibrillation ensued, requiring cardiopulmonary resuscitation and defibrillation. Peripheral cardiopulmonary bypass was established emergently via the left common femoral artery and vein, respectively. On the first selective contrast injection with a Judkins left 4.0 guide catheter, the left main appeared partially obstructed by a native leaflet (*Figure 2C*, the 'white line sign',[6] Supplementary material online, *Video S2*). A 7 Fr extra back-up 3.50 coronary guide catheter with a guide extension catheter (Guideliner) was used to engage the left main stem. A Hi-Torque balance middle weight coronary guidewire was manoeuvred into the distal left anterior descending artery. Following balloon angioplasty, a 3.50 mm × 15 mm everolimus-eluting stent was deployed into the left main and expanded at high pressure with a non-compliant balloon. Intravascular ultrasound (IVUS) demonstrated stent malapposition and underexpansion (*Figure 3A* and *B*). We postulated that aggressive post-dilatation was not sufficient to achieve adequate stent apposition and expansion because of the resistance offered by either the valve stent struts or the calcific native valve cusp. A second 4.0 mm × 15 mm overlapping everolimus-eluting stent was then deployed at high-pressure proximal into the first stent with the aim of achieving improved radial strength of the stent scaffolding. Following repeat high-pressure balloon dilatation, repeat IVUS showed improved and adequate stent expansion (*Figure 3C* and *D*). Following angioplasty and stent placement, coronary blood flow in the left coronary system was normal [Thrombolysis in Myocardial Infarction (TIMI) 3 flow

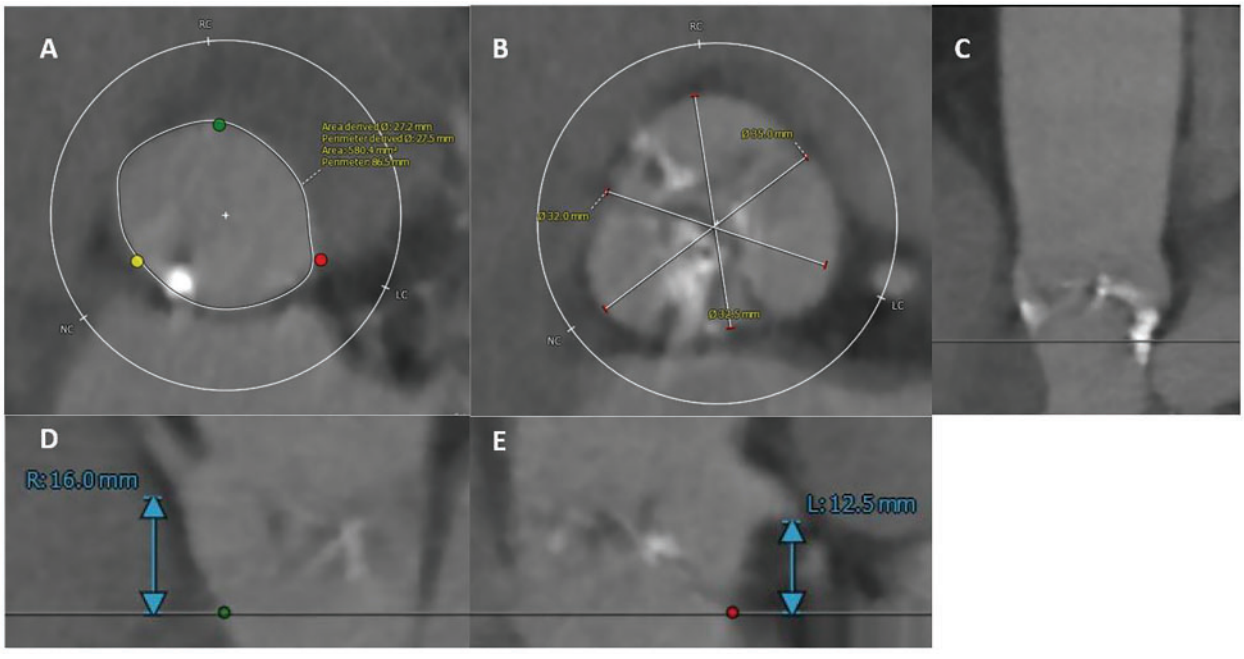

Figure 1 Multidetector computed tomography-derived aortic annulus reconstructions. (*A*) Annular area: 580 mm^2 and annular perimeter: 86.5 mm. (*B*) Sinuses of Valsalva measurements: 32 mm × 32.5 mm × 33 mm. (*C*) Stretched view demonstrating significant left ventricular outflow tract calcification. (*D*) Left coronary artery ostium height: 12.5 mm. (*E*) Right coronary artery ostium height: 16 mm.

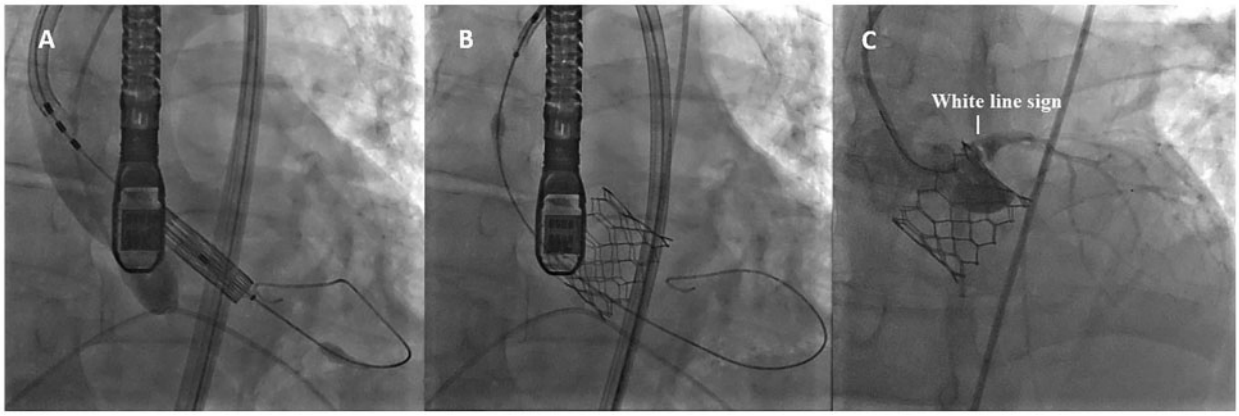

Figure 2 (*A*) Angiographic still depicting the Edwards Sapien 3 29 mm aortic bioprosthesis position prior to deployment. (*B*) Deployed valve. (*C*) On the first selective contrast injection with a JL 4.0 guide catheter, the left main appeared partially obstructed by a native leaflet (the "white line sign").[5]

(*Figure 4*), Supplementary material online, *Video S3*), and left ventricular function had normalized completely. Aspirin and clopidogrel were administered via a nasogastric tube. With haemodynamic stabilization, peripheral cardiopulmonary bypass was discontinued in the operating room. The patient was transferred to the intensive care unit with stable vital signs and without inotropic or vasopressor support and he was extubated the following day. He was discharged from hospital on Day 3 post-procedure. He developed a bradyarrhythmia requiring a permanent pacemaker 3 days following discharge. At 4 weeks follow-up, his exertional dyspnoea had improved significantly and he had resumed his usual activities. Follow-up transthoracic echocardiography demonstrated normal aortic valve function and normal left ventricular systolic function.

Discussion

Acute coronary obstruction following TAVR is an uncommon event, with a reported prevalence of less than 1% in contemporary practice.[7–9] The left coronary artery is the most commonly involved (87%). Established risk factors for coronary ostial occlusion include a short distance between the aortic annulus and the coronary ostia (<10 mm) and a narrow aortic root (<28 mm at the sinuses of Valsalva).[7–9] These two factors increase the likelihood that the native valve leaflets are displaced over and obstruct the coronary ostia when the aortic bioprosthesis is deployed. Other cited predictors of coronary occlusion include bulky calcification of the native leaflets, and signs of coronary compromise during balloon aortic valvuloplasty, such as ST-segment elevation in the electrocardiogram, and extrinsic compression of or reduced flow in the coronary artery on TOE (when used). Leaflet avulsion and migration into the coronary ostium, aortic root dissection or haematoma extending near or into the coronary ostia, and dislodgement of calcified material, are less common causes of coronary obstruction.[10] Complete coronary obstruction usually presents with severe persistent hypotension, ST segment elevation on the electrocardiogram, ventricular arrhythmias, and cardiac arrest. Partial obstruction may lead to either less dramatic or delayed haemodynamic compromise. Emergent percutaneous coronary intervention is the preferred treatment option. In a large (>6500 TAVR procedures) multicentre registry study of acute coronary occlusion during TAVR, PCI was attempted in 33/44 patients (75%), and it was successful in 81.8% of cases.[7] Urgent coronary artery bypass surgery or mechanical haemodynamic support were needed in 14% and 36% of the patients, respectively, underscoring the importance of performing these procedures in highly experienced centres with cardiac surgery backup. Mortality after acute coronary obstruction is high. In a recent systematic review of 96 cases published between 2012 and 2016, coronary obstruction following TAVR in native aortic valve stenosis was associated with a 30 days mortality rate of 35.3%.[9]

Perplexingly, our patient did not present with any of the recognized risk factors for acute coronary occlusion following TAVR. In particular, the vertical distance between the aortic annulus and the coronary ostia and the dimensions of the aortic annulus were significantly greater than the minimum thresholds identified in the literature. The sinuses of Valsalva measured 32 mm × 32.5 mm × 33 mm (*Figure 1B*), whereas the height of the left main coronary stem was 12.5 mm (*Figure 1E*). In this context it is worth noting that in the large multicentre registry mentioned above, 20% of cases of acute left main artery occlusion occurred in patients with an LCA (left coronary artery) height greater than 12 mm, and 35% of cases occurred in patients with sinus of Valsalva diameter greater than 30 mm. Although the non-coronary leaflet exhibited moderate calcification, the left leaflet did not appear significantly calcified (*Figure 1*). Some authors have suggested that curved leaflet length in relation to sinus dimension is a better predictor of coronary artery occlusion compared with annular diameter. Okuyama *et al.*[11] report a case of acute left main occlusion in a patient with a left coronary height of 17.1 mm. The authors estimated the ratio between the leaflet length and the curved coronary sinus height at 1.06, and postulated that given this finding, the relatively non-calcified and mobile left leaflet was long enough to cover the coronary ostium with valve expansion. They

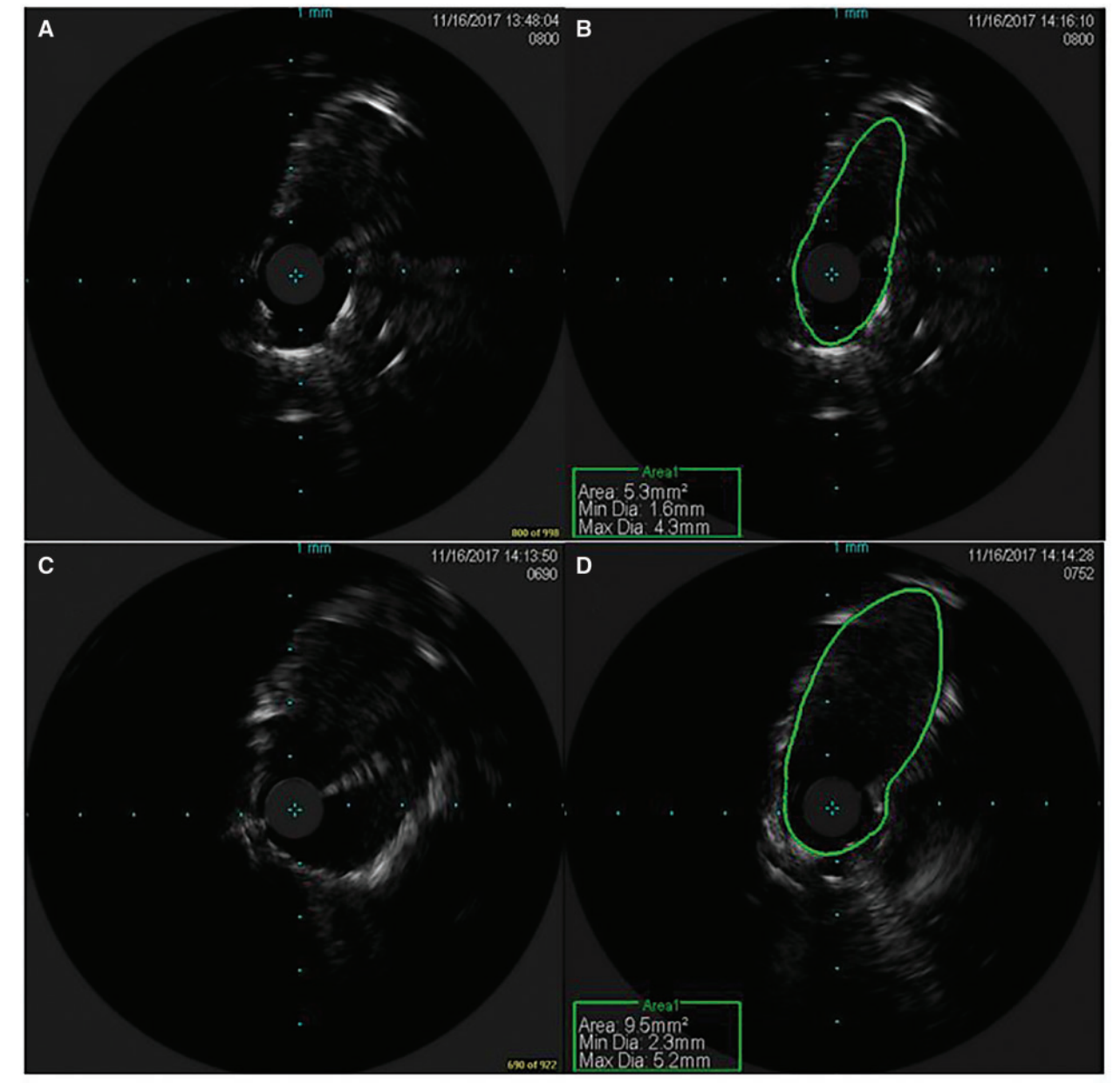

Figure 3 Following balloon angioplasty, a 3.50 mm × 15 mm everolimus-eluting stent was deployed into the left main coronary artery. (*A*) Intravascular ultrasound demonstrates stent malapposition and underexpansion at the site of contact with the valve frame struts. (*B*) The calculated minimum lumen area is 5.3 mm^2. (*C*) Following a second stent deployment and high-pressure balloon angioplasty with non-compliant balloon, intravascular ultrasound demonstrates stent better stent expansion at the site of contact with the valve frame struts. (*D*) The calculated minimum lumen area has increased to 9.5 mm^2.

suggest that a ratio >1 between leaflet length and coronary sinus height could be a new and useful predictor of coronary occlusion. In our patient, the left coronary leaflet (LCL) measured 16.1 mm from hinge point to the tip of the leaflet, and the curved coronary sinus (CCS) height was 13.2 mm (*Figure 5*), giving a ratio LCL/CCS of 1.219—significantly greater than 1.

The management of this case raises a technical point concerning focal stent compression from the valve frame struts or the displaced calcified native leaflet. The first stent, a 3.50 mm × 15 mm everolimus-eluting stent, was deployed into the left main and expanded at high pressure with a non-compliant balloon. Despite a satisfactory angiographic appearance, on IVUS it appeared underexpanded in its proximal segment. Repeated post-dilatation with non-compliant balloons inflated at high pressure did not improve stent expansion significantly. We postulated that the cause of the residual compression was the resistance exerted by the calcified native

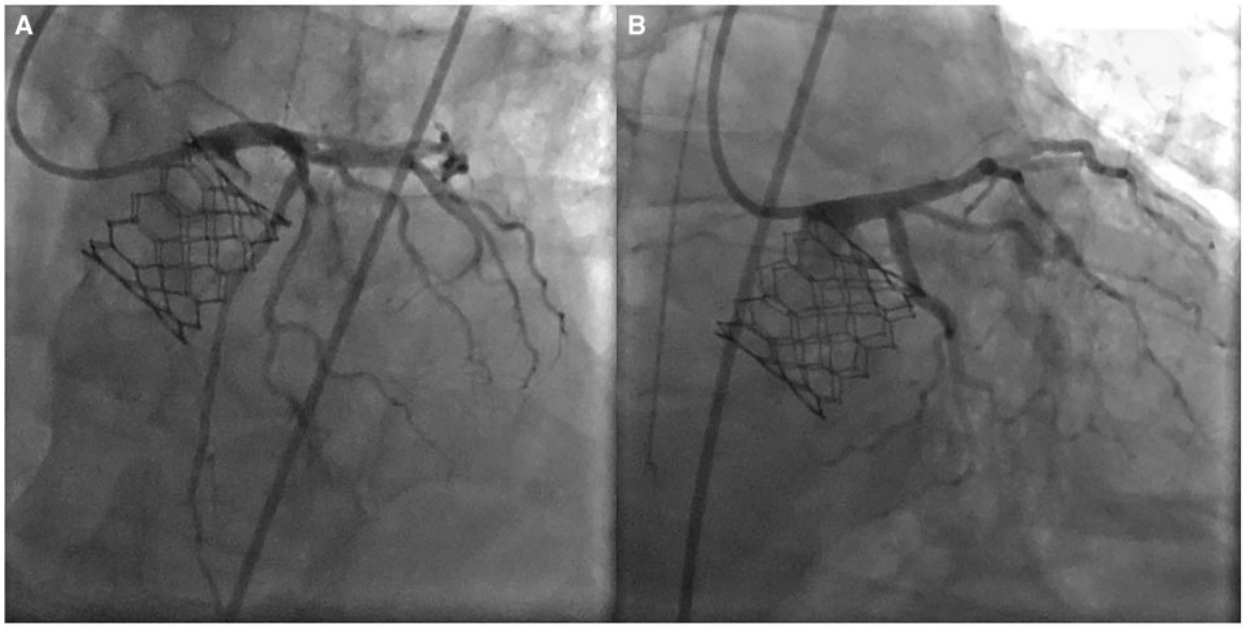

Figure 4 Final angiographic appearance after a second stent was deployed at the ostium. (*A*) Left anterior oblique, cranial view. (*B*) Straight caudal view.

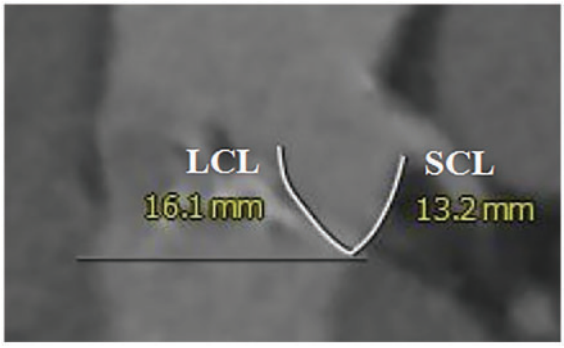

Figure 5 Multidetector computed tomography-derived aortic annulus reconstructions. The left coronary leaflet length is 16.1 mm from hinge point to the tip of the leaflet, and the sinus curved length is 13.2 mm, giving a left coronary leaflet/sinus curved length ratio of 1.219, significantly greater than 1. LCL, left coronary leaflet; SCL, sinus curved length.

valve leaflet or the struts of the valve stent. We, therefore, decided to deploy a second stent, a 4.0 mm × 15 mm overlapping everolimus-eluting stent, at high pressure, with the aim of achieving improved radial strength of the stent scaffolding against the valve struts. Repeat IVUS showed improved and adequate stent expansion. In the systematic review by Ribeiro *et al*, 3 patients (13%) needed a second stent due to significant compression of the first implanted stent unresponsive to balloon post-dilation,[7] and other authors have suggested implanting a second stent when the first one is under-expanded due to extrinsic compression.[12,13]

Conclusion

Acute coronary obstruction is a life-threatening complication of TAVR with a high-mortality rate. Although coronary protection may mitigate the risk of adverse events in patients with low-lying coronary ostia or small sinuses of Valsalva, this complication may occur in the absence of well-established high-risk features, suggesting other factors might be at play. A leaflet length to coronary sinus height ratio greater than 1, as described in this case and one previous report, might be an additional useful predictor of coronary occlusion during TAVR. In addition, we suggest that if residual focal stent compression from either valve strut or calcific aortic leaflet persists after stent deployment and the latter is resistant to balloon dilatation, deploying a second concentric layer of stent might improve the radial strength of the scaffolding and improve overall stent expansion.

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 author/s 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.

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