Delayed subtotal coronary obstruction after transapical aortic valve implantation

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Abstract
We report on an obstruction of the left main coronary artery which occurred after completion of an uneventful transapical aortic valve implantation (TAVI) procedure. This delayed subtotal coronary obstruction was detected by routine final transthoracic echocardiographic examination. Emergency implantation of a coronary stent eliminated myocardial ischemia. This case demonstrated that coronary artery obstruction can occur not only during the TAVI procedure but also some time later. This finding reinforces the idea that these patients must also be carefully evaluated in the early post-procedure period.

Keywords: Aortic stenosis; Transapical aortic valve implantation; Intraoperative transthoracic echocardiography; Ischemia; Emergency percutaneous intervention

1. Introduction
Transapical aortic valve implantation (TAVI) is a novel approach for high-risk patients with severe aortic valve stenosis. The early results are excellent [1, 2], but the procedure poses a risk of possibly dangerous and life-threatening complications that may occur at any moment during the procedure [1, 2]. One of these is obstruction of coronary ostia with a reported incidence of 0.4–4.1% [1–4]. We present a first case of delayed obstruction of the left main coronary artery that occurred after TAVI procedure had been completed.

2. Case report
A 68-year-old female with a logistic EuroSCORE of 8.8% and an STS score of 6.1% underwent TAVI for severe aortic stenosis. The patient was referred and accepted for TAVI because of severe adipositas with a body mass index of 58 kg/m². Coronary artery disease was excluded. Intraoperative transthoracic echocardiography (TEE) confirmed severe aortic stenosis (aortic valve area 0.5 cm², \(dP_{\text{max}}\) 75 mmHg, \(dP_{\text{mean}}\) 46 mmHg) with left ventricular concentric hypertrophy and an ejection fraction of 65%. There were no regional wall motion abnormalities (Fig. 1a). The standard intraoperative TEE measurements showed the following: left ventricular outflow tract, 18 mm; aortic valve annulus, 21.6 mm; aorta at the level of sinuses of Valsalva, 26.4 mm; sinotubular junction, 23 mm; ascending aorta, 26 mm. We chose a 26 mm Edwards SAPIEN prosthesis (Edwards SAPIEN THV, Edwards Lifesciences, Irvine, CA, USA) according to the valve manufacturer’s instructions. TAVI was performed in a standard manner with some modifications [5]. Balloon dilation of the native aortic valve and consequent deployment of the prosthetic valve were uneventful. The heart recovered immediately thereafter without any inotropic support and echocardiographic (ECHO) showed sinus rhythm with a rate of 60–70 beats/min with isolectric ST segment. TEE showed excellent function of the new valve, and the procedure was finished by removal of the 26-Fr sheath and the guide wires and suture of the left ventricle (LV) apex puncture. Final angiographic check by aortography (20 ml of contrast medium, 20 ml/s) revealed no coronary obstruction, excellent filling of the left anterior descending (LAD), ramus circumflex (RCX) and right coronary artery (RCA) and good valve position without valvular or paravalvular leak (Video 1a, Fig. 2a). Furthermore, all echocardiographic findings had been completely normal during the procedure. Hemodynamic state was continuously stable with mean arterial blood pressure (MAP) between 75 and 80 mmHg. Twenty minutes after valve deployment, a routine final TEE was performed according to our institutional standard protocol. The valve findings were unchanged, showing normal aortic prosthetic function with a calculated \(dP_{\text{max}}\) of 6.0 mmHg and \(dP_{\text{mean}}\) of 3.3 mmHg, an effective orifice area of 1.8 cm² and no valvular or paravalvular regurgitation. During TEE examination, MAP decreased from 75 to 68 mmHg and, unexpectedly, TEE revealed new regional wall motion abnormalities with anterior and anteroseptal hypokinesia (Fig. 1b). At that time five-channel ECG showed new ST segment depression. Two-dimensional echocardiographic
Fig. 1. M-mode echocardiogram recorded through the short axis of LV at midpapillary level shows anterior and inferior wall motion. (a) Preprocedural normokinesia, (b) ischemic period with anterior hypokinesia (arrow), (c) hyperkinesia after epinephrine, (d) normokinesia following emergency percutaneous coronary intervention.

Video 1. (a) Initial angiography after TAVI shows no coronary obstruction and excellent filling of the LAD, RCX and RCA. (b) Selective coronary angiography showing left coronary obstruction and slow filling of the LAD and RCX. TAVI, transapical aortic valve implantation.
Fig. 2. (a) Initial angiographic check after TAVI shows no coronary obstruction and excellent filling of the LAD, RCX and RCA. The distance between LCA and aortic valve annulus is relatively short but the LCA ostium is free. The position of the stented valve and its relationship to the LCA is as in classical findings after conventional aortic valve replacement. There was no aortic regurgitation. The image corresponds to Video 1a. (b) Selective coronary angiography showing left coronary obstruction and slow filling of the LAD and RCX. The image corresponds to Video 1b. (c) Stent inflation in the proximal left main trunk. The image corresponds to Video 2a. (d) Angiography after stent implantation shows a patent coronary artery without any stenosis. The image corresponds to Video 2b.

TAVI, transapical aortic valve implantation.

dure but in rare cases – like in the reported case – also after the procedure. Therefore, we suggest a final TEE examination after finishing the procedure and before removing the probe.

Coronary obstructions are mostly caused by compression of an altered native aortic valve leaflet that contains a huge amount of calcified tissue or by the new valve prosthesis itself. To achieve the best valve position without coronary artery obstruction, we use angiographic visualization of the aortic root while the prosthetic valve is slowly and gradually deployed [5].

The cause of LCA obstruction in our particular case remains unclear. It can be speculated that the possible causes of obstruction were hematoma or simply leaflet distortion and apposition on the LCA ostium.

We conclude that coronary artery obstruction can occur following completion of the TAVI procedure and that any hemodynamic instability demonstrated by the patient should prompt an immediate search for impaired blood flow into a coronary artery. This finding reinforces the idea that TAVI patients must be carefully evaluated also in the early postprocedure period.

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References


