

Modified chimney / snorkel stenting of the left main coronary artery after transcatheter aortic valve implantation

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The predominant mechanism of the coronary flow impairment following transcatheter aortic valve implantation (TAVI) is mainly due to the displacement of the calcified native cusp over the coronary ostia¹ or rarely due to implanted bioprosthesis being positioned too high.

We present a 67-years-old woman after mitral valve replacement (MVR) and re-MVR (mechanical prosthesis) several years ago, treated with TAVI for critical aortic stenosis. Detailed preinterventional computed tomography angiography (CTA) measurements were done. The distance between the aortic annulus and the ostium of the left coronary artery (LCA) was 12 mm, and between the aortic annulus and the ostium of the right coronary artery, 18 mm; the maximal radius of the Valsalva sinuses was 31 × 31 mm; the left ventricle outflow tract diameter was 19 × 28 mm. No significant coronary lesions were visualized. Using femoral access, Evolut R 29 mm bioprosthesis (Medtronic Inc., Minneapolis, Minnesota, United States) was directly implanted and postdilated. The left main coronary artery (LMCA) remained unprotected during the procedure. Aortography performed immediately after TAVI showed both left and right coronaries with nonobstructed ostia. However, a too high position of the bioprosthesis with the left leaflet above the ostium of the LMCA could have been suspected.

Five months later, the patient was readmitted due to complaints of exertional dyspnea. Echocardiography showed diffused hypokinesia of the left ventricular (LV) anterolateral wall and reduction of LV ejection fraction from 60% to 30%. Troponin T levels were elevated at 131 ng/l

(upper normal limit, 14 ng/l). CTA and nonselective coronary angiography (CA) revealed coronary flow impairment to the LCA due to bioprosthesis being positioned too high with the ostium of the LMCA below the bioprosthesis leaflets (FIGURE 1A and 1B). Thus, percutaneous coronary intervention (PCI) was chosen as an optimal treatment strategy. Despite lack of selective LCA intubation with Amplatz-left guide, it was successfully wired. Next, an everolimus-eluting stent (3.5 × 15 mm) was positioned across the bioprosthesis leaflets and further across the bioprosthesis cell with landing zone in the distal LMCA. Then, it was directly deployed and postdilated with a 4-mm noncompliant balloon (FIGURE 1C and 1D). The final contrast injection showed appropriate LCA filling (FIGURE 1E). In-stent minimal lumen area was 7 mm² as measured by intravascular ultrasound (Supplementary material, Figure S1A). The proximal part of the implanted stent was located above the level of the bioprosthesis leaflets as confirmed by a postprocedural CTA (Supplementary material, Figure S1B and S1C). Echocardiography done 4 days later and 8 months after PCI revealed significantly improved LV function (ejection fraction, 50% and 60%, respectively).

High implantation of Evolut R bioprosthesis in our patient was intentional to avoid interference with previously implanted mitral prosthesis and unintentionally caused a too high bioprosthesis position (above the LCA ostium). This allowed mainly systolic LCA inflow and only residual diastolic LCA inflow (due to small paravalvular leak), which physiologically is predominant. That most probably caused ischemic deterioration of the LV function. Restoration of the diastolic LCA

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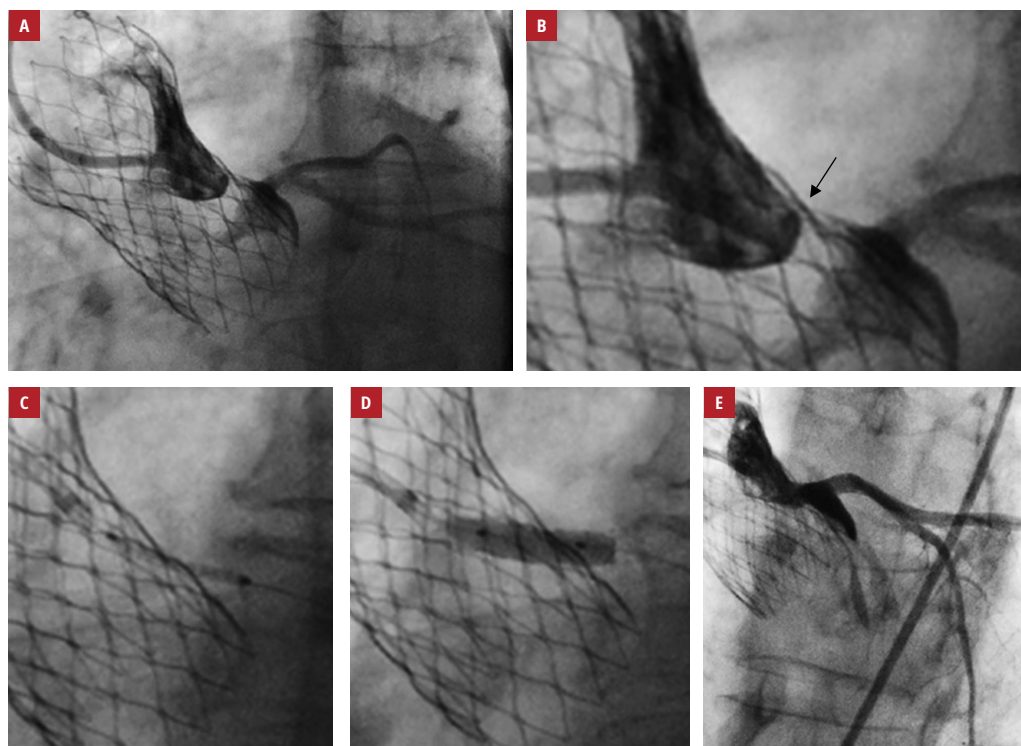


FIGURE 1 Chimney stenting: **A** – contrast injection showing the left leaflet of the bioprosthesis above the left coronary ostium; **B** – magnification of the image **A** with focus on the distance between the left leaflet (arrow) and the ostium of the left main coronary artery; **C** – positioning of the stent across the bioprosthesis cell; **D** – stent deployment; **E** – final angiographic result

inflow after LMCA chimney / snorkel stenting allowed significant improvement of LV function.

Evidence from previous trials does not support the routine use of periprocedural guidance by transesophageal echocardiography during TAVI. However, patients after MVR pose more challenge and transesophageal echocardiography–guidance during TAVI may be considered, especially in those with mechanical valves in the mitral position.

PCI after TAVI may be technically challenging (more difficult selective coronary intubation and poorer catheter support).² Subsequently, the success rate of PCI remains suboptimal.³

Several cases of intraprocedural chimney / snorkel stenting with prophylactic coronary wire protection during TAVI were reported before.⁴ At the end of such procedures, stent protrudes between the aortic wall and bioprosthesis-scaffolding. PCI in our patient differed in 2 ways: the stent crossed both the bioprosthesis leaflets and the bioprosthesis-scaffolding before entering LMCA (modified chimney / snorkel PCI).

Finally, optimal post-PCI intravascular ultrasound criteria⁵ suggest that the LMCA-minimal lumen area should be at least 8.2 mm². Despite postdilatation, this threshold was not achieved in our case. Further, interaction between the protruding stent and bioprosthesis leaflets may lead to damage of both structures over time.

In conclusion, aortic bioprosthesis positioned too high impairs blood flow into the LCA.

Modified chimney / snorkel stenting may be a reasonable treatment option. Control CA or CTA may be considered at follow-up.

SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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