

CASE REPORT

Acute myocardial infarction shortly after valve-in-valve transcatheter aortic valve implantation successfully managed with challenging percutaneous coronary intervention

Kapak içi kapak transkateter aort kapak implantasyonu (TAVİ) sonrasında ortaya çıkan akut miyokart enfarktüsü ve bunun perkütan koroner girişimle tedavisi

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Summary– In recent years, transcatheter aortic valve implantation (TAVI) has been considered a novel option for the management of surgically high-risk patients requiring aortic valve replacement. Presently described is a case of acute coronary syndrome (ACS) managed with a challenging primary percutaneous coronary intervention (PCI) shortly after a valve-in-valve TAVI intervention. This case highlights 2 important issues: PCI may be an option for the management of coronary heart disease in patients after TAVI even in the setting of demanding features associated with coronary osteal engagement, and secondly, TAVI may serve as a potential risk factor for future coronary ischemic syndromes, largely due to its potential adverse effects on coronary flow dynamics, etc. However, the latter notion is quite speculative, and should be tested in further studies.

Özet– Son yıllarda, transkateter aort kapak değişimi (TAVİ) aort kapak değişimi gerektiren ve cerrahi açıdan yüksek risk taşıyan hastalar için yeni bir tedavi seçeneği olarak ortaya çıkmıştır. Sunulan olgu, yakın zamanda uygulanan bir kapak-içi kapak TAVİ işleminden kısa bir süre sonra gözlenen bir akut koroner sendrom (AKS) olgusu ve bunun işlemsel olarak oldukça sıkıntılı özellikler barındıran bir primer perkütan koroner girişim (PKG) ile başarılı olarak tedavisini anlatmaktadır. Bu olgu iki önemli noktaya işaret etmektedir: Birincisi TAVİ sonrası PKG'ler, koroner osteal kateterizasyonun çok zor olabileceği durumlarda bile oldukça uygun bir seçenek olabilir. İkincisi TAVİ, koroner akım dinamikleri dinamikleri üzerindeki olası kötü etkileri nedeniyle, geç dönem koroner iskemik sendromları için potansiyel bir risk faktörü olabilir. Ancak ikinci nokta oldukça spekülattir ve daha ileri çalışmalarla desteklenmesi gerekmektedir.

In the last decade, interest in transcatheter aortic valve implantation (TAVI) as a promising option, particularly for high-risk, inoperable patients requiring aortic valve replacement, has risen substantially.^[1]

Herein, a case of acute coronary syndrome (ACS) in a patient with a recent history of valve-in-valve TAVI (about 1 month

before ACS presentation) is described. Fortunately, the ACS was successfully managed with percutaneous coronary intervention (PCI), despite a variety of potentially challenging features associated with the procedure in this setting.

CASE REPORT

A 75-year-old man with a history of valve-in-valve TAVI about 1 month earlier presented with angina pectoris and electrocardiogram findings (Fig. 1) suggestive of an acute anteroseptal ST-elevation myocardial infarction (STEMI), along with an unremarkable physical examination on admission. Emergent coro-

Abbreviations:

ACS	Acute coronary syndrome
CAD	Coronary artery disease
CAG	Coronary angiogram
DES	Drug eluting stent
LAD	Left anterior descending
LVEF	Left ventricular ejection fraction
PCI	Percutaneous coronary intervention
RCA	Right coronary artery
STEMI	ST-elevation myocardial infarction
TAVI	Transcatheter aortic valve implantation

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Figure 1. Admission electrocardiogram consistent with an acute anteroseptal ST-elevation myocardial infarction (leads V1-4 demonstrating ST segment elevation).

nary angiogram (CAG) and primary PCI was planned. The patient had a history of coronary interventions beginning a few years prior, and TAVI about 1 month before presentation.

The initial coronary intervention was left anterior descending (LAD) artery stenting with a drug eluting stent (DES) (about 3½ years earlier), and was followed by a repeat CAG (about 1 year earlier) that demonstrated a fully patent LAD stent, a non-critical plaque in the circumflex artery, and a chronically occluded right coronary artery (RCA). RCA occlusion was managed with a successful intervention (about 8 months earlier) at another medical center.

More recently (a few months after the elective PCI mentioned above), the patient also underwent a successful TAVI (about 1 month before the ACS admission). He had been experiencing a gradually worsening dyspnea on his previous admissions, and was

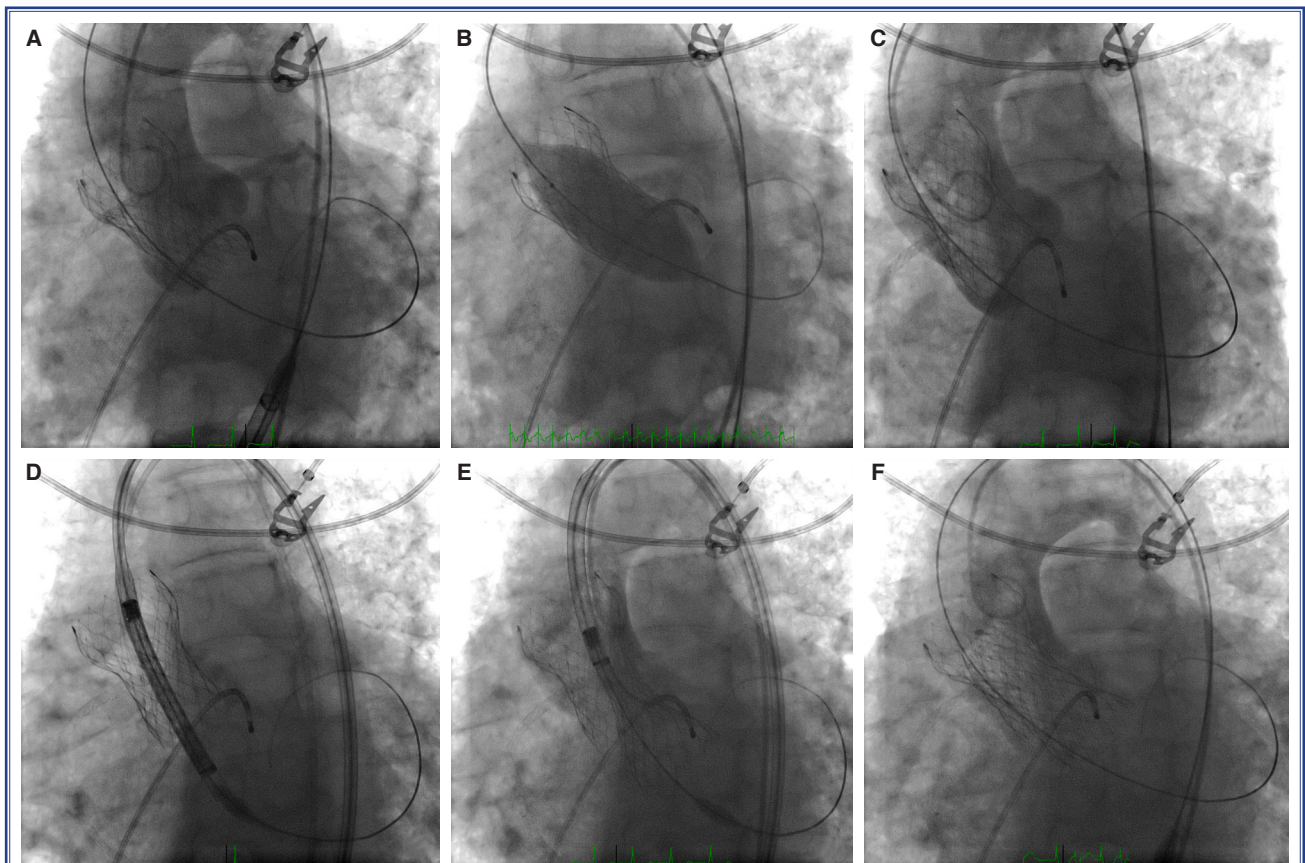


Figure 2. Valve-in-valve transcatheter aortic valve implantation about 1 month before the latest presentation with an anteroseptal ST-elevation myocardial infarction. **(A)** Initial 29-mm CoreValve Evolute-R (Medtronic, Inc., Minneapolis, MN, USA) implantation with incomplete expansion, **(B)** post-dilatation with a 25-mm balloon, **(C)** slight distal subluxation of the initial valve missing the aortic annulus, **(D)** a second 29-mm CoreValve Evolute R just prior to expansion, **(E)** gradual expansion of the second valve, **(F)** final image of the 2 valves (valve-in-valve).

found to have a severe aortic valvular pathology on transthoracic echocardiogram demonstrating heavily calcified aortic cusps with an aortic valve area of 0.9 cm² and an accompanying moderate-severe valvular regurgitation. Moreover, the initial left ventricular ejection fraction (LVEF) value of 55% then declined to 43% on follow-up. Therefore, the patient was to undergo an urgent aortic valvular intervention due to the emerging symptomatology and gradual deterioration in LVEF value. Based on our institution's multidisciplinary consensus, the patient was deemed to have prohibitively high-risk features for surgical aortic valve replacement due to multi-organ failure (pulmonary, renal, cardiac) and moderate frailty. Therefore, the multidisciplinary team considered TAVI a possibly safer option. TAVI was performed with a variety of emerging procedural challenges: a second valve was deemed necessary in the same session (valve-in-valve) due to a distal sublaxation of the initially deployed valve, possibly during balloon post-dilatation (both valves were self-expandable, 29-mm CoreValve Evolut-R bioprostheses; Medtronic, Inc., Minneapolis, MN, USA) (Fig. 2). Unfortunately, the patient also needed a peripheric surgical operation at

the end of the intervention due to a suture problem in the femoral access. Moreover, he had a prolonged hospitalization course due to an attack of pneumonia, but was eventually discharged in good health under dual antiplatelet therapy (acetylsalicylic acid and clopidogrel) that he had already been receiving for at least 5 months preceding the TAVI.

His acute STEMI was successfully managed with primary PCI in the setting of recent TAVI despite a variety of procedural challenges. After several attempts, we were able to intubate the left main coronary ostium through the double-layered valve struts with a 6-F Judkins diagnostic catheter. On engaging the left coronary artery, coronary opacification displayed a critical thrombotic stenosis of the LAD (proximal-mid part), just on the distal segment of the previous stent (reminiscent of a very late stent thrombosis). Once we re-cannulated the LAD with a 6-F Judkins guiding catheter after several maneuvers, and crossed a floppy wire through the lesion, a DES of 3.0x18 mm was successfully deployed with an overlapping segment on the previous stent (Fig. 3). As the culprit artery had already been found, we did not try to cannulate the RCA to avoid further loading the patient

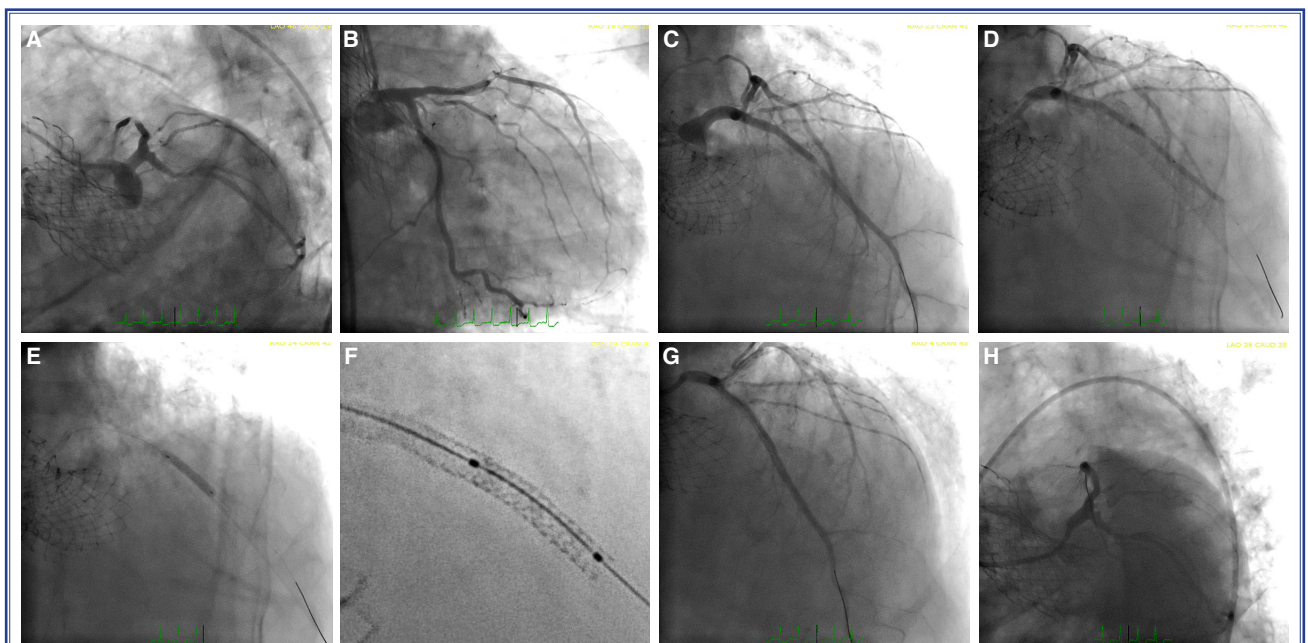


Figure 3. Coronary angiogram (A, B, C) and percutaneous intervention (D, E, F, G, H) of the critical in-stent thrombotic lesion in the left anterior descending artery about 1 month after valve-in valve transcatheter aortic valve implantation. A drug-eluting stent 3.0x18 mm in size was implanted with an overlapping segment on the previous proximal stent. (A) Left anterior oblique caudal view, (B) right anterior oblique caudal view, (C) right anterior oblique cranial view, (D) drug-eluting stent (DES) just before inflation, (E) inflated DES overlapping on the distal segment of the previous stent, (F) two overlapping stents (clear-stent mode), (G) post-implantation right anterior oblique cranial view, (H) post-implantation left anterior oblique caudal view.

with contrast medium, particularly during repetitive attempts at ostial engagement. However, despite a successful PCI, the left ventricular ejection fraction (LVEF) on echocardiogram dropped significantly from an initial value of 55% (pre-TAVI) to a value of about 30% (post-PCI), possibly as a result of delayed admission, reperfusion injury, etc. Unfortunately, the patient succumbed on the 17th day of hospitalization due to severe chronic respiratory problems and heart failure. Of note, the patient had an event-free period of at least 3½ years (from the initial LAD stenting to the latest ACS admission). Importantly, the patient had been receiving dual antiplatelet therapy (acetylsalicylic acid 100 mg 1x1 and clopidogrel 75 mg 1x1) for at least the 6 months preceding his ACS admission (possibly initiated for his last elective coronary intervention at another center). Therefore, this was a STEMI that evolved under dual antiplatelet therapy about 1 month after TAVI.

DISCUSSION

The present case highlights 2 important issues: firstly; post-TAVI coronary interventions may be feasible even in the setting of problematic cases for coronary ostial engagement. Despite the rapidly growing number of novel valve choices with more favorable features,^[1] coronary intervention after TAVI has generally been regarded as a significant challenge in clinical practice, particularly in the setting of supracoronary valve position (self-expandable valves),^[2] valve frames superimposed on the coronary ostia (mostly self expandable valves) and implantation that is too high, resulting in lower skirt impingement on the coronary ostia (self-expandable valves).^[3] Furthermore, valve-in-valve TAVI might be necessary in certain settings, and might further complicate a future PCI.^[3] Regardless of the valve type, interventionalists generally prefer to manage coronary stenoses in the pre-TAVI setting, possibly in an effort to obviate future PCI failures. With this fear of failure in mind, lesions with a borderline severity (with no objective evidence of ischemia) may unnecessarily be managed in a hasty manner in the pre-TAVI setting, suggesting potential overdiagnosis and overtreatment of these lesions. Conversely, clinicians may be reluctant to perform PCI after TAVI, even in an ACS scenario, and may be inclined to pursue a merely conservative strategy,

including dose adjustment of anti-ischemics, thrombolytic therapy, etc. This may be considered undertreatment, and may even become harmful in certain settings (for instance, bleeding due to thrombolytic therapy for ACS instead of PCI in dual antiplatelet recipients after TAVI). We already had all of the above-mentioned procedural challenges regarding post-TAVI PCI (2 self expandable valves with a supracoronary valve-in-valve placement with high implantation of the initial valve) that might potentially hamper cannulation of the left coronary artery. However, we pursued an invasive strategy and were able to perform primary PCI with a good angiographic result. Similarly, in a prospective series of patients undergoing post-TAVI PCI, a very high rate of procedural success was reported regardless of the valve type.^[3] However, there was no PCI performed in the setting of ACS or valve-in-valve TAVI in that study.^[3] Post-TAVI PCI may be considered a possible option in clinical practice even in the setting of uniformly agreed-upon procedural challenges. This may suggest the necessity of an ischemia-guided strategy rather than one guided by TAVI for percutaneous management of coronary artery disease (CAD), both in the pre- and post-TAVI settings.

Secondly, and speculatively, the present case raises the question of whether TAVI, per se, may elicit a proclivity for future coronary ischemic events, even in the absence of late mechanical complications (including late device dislodgement resulting in ostial occlusion, etc.^[4]). The present case had an event-free period of at least 3½ years, and suffered an ACS (even under dual antiplatelet therapy) without an evident ACS trigger or a device-related mechanical complication within a month after TAVI, which might potentially be associated with the coronary ischemic event through a variety of poorly understood mechanisms (alteration in coronary flow dynamics, etc.). However, this notion is quite speculative, and needs to be tested through further studies.

In conclusion, coronary interventions in the post-TAVI setting may be an entirely feasible option, even in apparently challenging cases. Accordingly, percutaneous intervention, when necessary, (particularly in the setting of ACS) should be the primary management strategy in these patients, just as in other CAD patients without TAVI. Clinicians should adopt an ischemia-guided strategy, rather than a TAVI-guided

strategy, for the management of CAD in both the pre- and post-TAVI setting. Interestingly, a subtle association between TAVI and late coronary ischemic events might also exist in certain settings. However, this issue remains speculative at present, and warrants further investigation.

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