

See Article page 91.



Commentary: Coronary revascularization following aortic valve replacement: More than just a trivial event?

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Degenerative aortic stenosis and coronary artery disease (CAD) share similar pathophysiological processes.¹ A high proportion of patients with severe aortic stenosis have some degree of CAD, with a prevalence of CAD in transcatheter aortic valve replacement (TAVR) recipients of approximately 50%.² In randomized trials comparing TAVR and surgical aortic valve replacement (SAVR), coronary revascularization at the time of the replacement operation was performed in approximately 12% of patients (either by percutaneous coronary intervention [PCI] or coronary artery bypass graft [CABG]), and this proportion reached 25% in real-world TAVR registries.² Three recent studies investigated the incidence, clinical characteristics, and outcomes of acute coronary syndrome (ACS) following TAVR³⁻⁵; however, most aspects of coronary events following SAVR have not been completely elucidated.

In this issue of the *Journal*, Çelik and colleagues⁶ report the risk of late coronary events among 420 patients who underwent isolated SAVR with a mean follow-up of 17 years.

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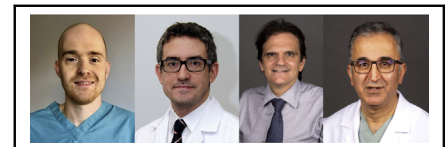
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CENTRAL MESSAGE

Coronary revascularization was infrequent following surgical aortic valve replacement. Further studies in low-risk patients undergoing TAVR are needed to better assess the late need and outcomes of coronary revascularization.

The authors evaluated the incidence of coronary revascularization after isolated SAVR and the risk factors associated with late coronary revascularization post-SAVR. The study provides insight into the late incidence of coronary revascularization following SAVR; a rate of revascularization of 6.2 per 1000 patient-years was reported, along with an independent link between previous coronary interventions and the need for subsequent revascularization post-SAVR. The authors address a compelling clinical question, as the risk of late coronary disease may influence the selection of TAVR or SAVR for patients with severe aortic stenosis, and this is highly relevant for low-risk and younger patients. Nonetheless, some aspects of the authors' work merit further discussion.

First and foremost, patients were excluded if they had not been followed at the Erasmus Medical Center. These patients represented approximately 75% of all patients undergoing isolated SAVR, and thus the study's validity is seriously compromised by the likely profound selection bias. Only 24 patients underwent late coronary revascularization and were included in longitudinal analyses, and it is difficult to draw meaningful conclusions from these limited data. In addition, patients with concomitant CABG were not included in this study. This is relevant, given that approximately 30% of patients with aortic stenosis undergoing SAVR have concomitant CABG⁷ and 25% of TAVR recipients undergo PCI before TAVR.² Thus, the authors' findings might not systematically apply to the whole spectrum of patients undergoing AVR.

One of the stated objectives of the study was to provide relevant information regarding low-risk patients undergoing SAVR, which could then be applied to the low-risk TAVR population. Two recent studies including intermediate- to high-risk patients reported a rate of readmission for ACS after TAVR of 5% after a median follow-up of 1 year and 10% after 2 years.^{3,5} In contrast, Çelik and colleagues report notably lower rates of coronary revascularization during follow-up, with 0.5% at 30 days, 2.2% at 1 year, 4.1% at 10 years and 6.9% at 20 years.

Several issues may have led to the authors' underestimation of the true coronary event burden following SAVR. Their data suggest that patients with previous revascularization are at highest risk of coronary revascularization following SAVR. However, patients with concomitant CABG were not included in the analysis. Also, approximately one-half of the study population underwent SAVR before 2002, and 67% received a mechanical prosthesis, a practice that is no longer representative of the current management of surgical aortic stenosis. These patients were prescribed a lifelong vitamin K antagonist, which may have modulated the risk of a late coronary event. Finally, some of the observed deaths were probably related to a cardiac cause. Although considered in the statistical analyses, the competing risk of death may have led to underestimation of the coronary event burden, given that preceding mortality obviously decreases the risk of a coronary event requiring revascularization to zero.

Çelik and colleagues found that coronary revascularization during follow-up was more frequent in patients with previous revascularization before SAVR. This is not surprising, given that revascularization procedures are the most frequent cause of rehospitalization after PCI in patients with ACS.⁸ Of note, this result is consistent with findings of 2 previous studies that identified previous CAD as an independent predictor of ACS following TAVR.³⁻⁵ Owing to the advent of primary PCI, mortality following ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction is now similar.⁹ In contrast, STEMI following TAVR is associated with an increased risk of death compared with other types of ACS.^{4,5} It would be of value to compare the risks of death according to coronary event type among patients who underwent coronary revascularization after SAVR. Unfortunately, the small numbers of events did not allow Çelik and colleagues to investigate

the prognostic factors predicting coronary events following SAVR, opening a new venue for future investigations.

In conclusion, this article by Çelik and colleagues examines the highly relevant issue of the late risk of coronary events following SAVR. In contrast, available data in TAVR cohorts come from intermediate- to high-risk populations. We caution readers to resist the urge to extrapolate the authors' data to the expected rate of coronary events in low-risk patients evaluated for TAVR. They likely underestimated the risk of late coronary revascularization, and their study design does not (and cannot) account for the alternative pathophysiological mechanisms linked to the transcatheter heart valve (eg, impaired coronary flow, leaflet thrombosis or late valve migration) leading to an ACS post-TAVR.² Future studies focusing on low-risk patients undergoing TAVR are needed to better assess the incidence, clinical features, and outcomes of coronary revascularization following surgical AVR and TAVR.

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