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EDITORIAL COMMENT

Coronary Artery Disease and Revascularization in T-TEER

Signal or Bystander?

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Tricuspid regurgitation (TR) has emerged as an important and often under-recognized contributor to heart failure, impaired quality of life, and excess mortality.[1] Historically neglected, TR is frequently diagnosed late in its course, when right ventricular dysfunction, systemic congestion, and multiorgan impairment are already advanced. Surgical correction carries substantial risk in this population, leading to the rapid development and adoption of transcatheter tricuspid interventions, particularly tricuspid transcatheter edge-to-edge repair (T-TEER), as a less invasive therapeutic option. Coronary artery disease (CAD) commonly coexists in patients undergoing structural heart interventions, reflecting shared risk factors and advanced age. Although coronary revascularization may alleviate symptoms and, in selected cases, improve outcomes, randomized data have challenged the routine use of percutaneous coronary intervention (PCI) prior to left-sided structural procedures, favoring a more selective, indication-driven approach.[2,3] In contrast, the role of CAD in patients undergoing tricuspid interventions remains poorly defined. The pathophysiology of functional TR is distinct, often driven by right ventricular remodeling, atrial fibrillation, and pulmonary hypertension rather than ischemic myocardial injury.[4] Consequently, the extent to which CAD contributes to disease progression or modifies outcomes after T-TEER is uncertain. Moreover, it remains unclear whether coronary revascularization confers prognostic benefit in this setting or simply reflects underlying comorbidity. In this issue of *JACC: Cardiovascular Interventions*, Al Zaidi et al[5] provide an important and timely analysis addressing the role of CAD and revascularization in patients undergoing T-TEER. In this single-center cohort of 361 consecutive patients undergoing T-TEER, the investigators performed systematic pre-procedural coronary angiography with quantitative assessment of CAD complexity using the SYNTAX (Synergy Between PCI With Taxus and Cardiac Surgery) score. Obstructive CAD, defined as lesions with >70% angiographic stenosis or >50% in case of left main lesions, was present in 34% of patients, with relatively low anatomical complexity (median SYNTAX score 6). Among patients with CAD, approximately one-third underwent PCI, resulting in a reduction in residual SYNTAX score. At 12 months, overall mortality was 16.7%, consistent with contemporary T-TEER populations.[6] Notably, the presence of obstructive CAD did not affect survival, with similar mortality rates in patients with and without CAD (17.5% vs 16.3%). Furthermore, neither baseline nor residual SYNTAX score was associated with mortality in the overall cohort. The principal finding of the study emerges from interaction analyses incorporating the TRI-SCORE, a validated composite risk score for T-TEER patients.[7] Among patients with low or intermediate procedural risk (TRI-SCORE <6), incomplete revascularization, defined as a residual SYNTAX score ≥4, was associated with significantly higher mortality (20.8% vs 6.4%). In contrast, in high-risk patients (TRI-SCORE ≥ 6), outcomes were poor irrespective of CAD burden or completeness of revascularization. These findings suggest that the prognostic impact of CAD in T-TEER is not uniform but instead depends on the

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underlying disease stage, with residual CAD influencing outcomes only in patients with sufficient physiological reserve.

This study represents a meaningful step forward in understanding the interplay between CAD and tricuspid interventions, addressing a major evidence gap in structural heart disease. Several aspects merit discussion. First, the investigators move beyond the traditional binary classification of CAD by incorporating quantitative measures of anatomical complexity (SYNTAX score) and residual disease. This approach aligns with contemporary revascularization paradigms and allows a more nuanced assessment of coronary disease burden. Second, the integration of CAD metrics with the TRI-SCORE provides a clinically relevant framework that acknowledges the dominant role of systemic disease severity in T-TEER outcomes. By demonstrating a significant interaction between residual CAD and global risk, the study introduces a stage-dependent model that may better reflect real-world patient heterogeneity. However, several limitations warrant careful consideration. Foremost, the observational and single-center design introduces the potential for selection bias, particularly in the decision to perform PCI. Although the investigators report that revascularization was driven primarily by anatomical complexity rather than clinical risk, unmeasured confounding cannot be excluded. Patients selected for PCI may differ in ways not fully captured by baseline variables or risk scores. Second, the overall CAD burden in this cohort was modest. The median SYNTAX score of 6 indicates predominantly low-complexity disease, which may limit the ability to detect a prognostic signal and restricts generalizability to patients with more advanced or diffuse CAD. Third, the study relied on angiographic assessment without incorporating physiological measures such as fractional flow reserve or instantaneous wave-free ratio. In contemporary practice, the functional significance of coronary lesions has become central to revascularization decisions. Whether a physiology-guided approach would yield different associations between residual disease and outcomes remains unknown. Fourth, the relatively limited sample size and event rates, particularly within subgroup analyses, raise concerns regarding statistical power and the robustness of interaction effects. The observed association between residual SYNTAX score and mortality in low- and intermediate-risk patients, although compelling, should be viewed as hypothesis generating rather than definitive. Finally, causality cannot be inferred. Residual CAD may act as a surrogate marker for more diffuse cardiovascular disease, rather than being a direct mediator of adverse outcomes. Whether more complete revascularization would improve survival in lower risk T-TEER patients remains an open question.

The results of this study must be interpreted within the broader context of evolving evidence on coronary revascularization in structural heart interventions. In transcatheter aortic valve replacement (TAVR), the role of PCI has been the subject of randomized investigation. The ACTIVATION (Percutaneous Coronary Intervention Prior to Transcatheter Aortic Valve Implantation) trial[3] demonstrated no benefit of routine PCI prior to TAVR and reported increased bleeding complications, challenging the historical practice of systematic revascularization. More recently, the NOTION-3 (Revascularization in Patients Undergoing Transcatheter Aortic Valve Implantation) trial[2] suggested that PCI in patients with significant CAD may reduce a composite endpoint of death, myocardial infarction, and urgent revascularization, although the benefit appeared driven primarily by reductions in myocardial infarction and repeat revascularization rather than mortality. Together, these trials support a selective, patient-centered approach to revascularization, rather than routine treatment of all angiographic lesions. In mitral transcatheter edge-to-edge repair, the evidence is less definitive but follows a similar path. CAD is highly prevalent in this population, particularly among patients with secondary mitral regurgitation, for which ischemic cardiomyopathy is often a key driver. Revascularization may influence left ventricular remodeling and, in some cases, reduce the severity of mitral regurgitation itself. However, no randomized trials have established that PCI improves outcomes specifically in patients undergoing mitral transcatheter edge-to-edge repair. Importantly, even in patients with ischemic left ventricular dysfunction, patient age represents an important modifier, which inversely correlates with the ability of PCI to achieve improvements in cardiovascular mortality.[8] Current practice therefore emphasizes treatment of prognostically significant or symptomatic lesions rather than complete anatomical revascularization.[9] Against this background, the present study highlights important distinctions in the tricuspid disease phenotype. Compared with TAVR and mitral transcatheter edge-to-edge repair populations, patients undergoing T-TEER appear to have a lower prevalence and complexity of CAD. This likely reflects fundamental differences in pathophysiology. In this setting, CAD may play a less central role in disease progression. The stage-dependent findings of Al Zaidi et al[5] further

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reinforce this concept. In advanced disease stages—characterized by right ventricular dysfunction, renal and hepatic impairment, and high TRI-SCORE—outcomes are driven predominantly by comorbidities. In such patients, revascularization is unlikely to modify the clinical trajectory. This paradigm parallels broader trends in cardiovascular medicine, in which the benefit of intervention is increasingly understood to depend on the stage of disease progression. Just as earlier intervention in valvular heart disease yields better outcomes, the potential impact of coronary revascularization may be greatest before the onset of irreversible right-sided failure.

Al Zaidi et al[5] provide a thoughtful and hypothesis-generating contribution that advances our understanding of CAD in T-TEER. Their findings challenge the notion of CAD as a uniform prognostic factor and instead support a stage-dependent model, in which residual coronary disease influences outcomes primarily in patients with lower overall risk. From a clinical perspective, the data argue against routine coronary revascularization prior to T-TEER and instead favor a selective, individualized strategy, integrating coronary anatomy with global risk assessment. Future studies, ideally prospective and incorporating physiological lesion assessment, are needed to determine whether targeted revascularization in selected patients can meaningfully improve outcomes. Until then, the present work serves as a valuable reminder that in structural heart disease, as in many areas of medicine, context matters as much as anatomy.

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