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Coronary artery disease in patients with severe aortic stenosis undergoing valve replacement: a rapidly evolving field

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We read with great interest the clinical consensus statement regarding the management of coronary artery disease (CAD) in patients undergoing transcatheter aortic valve implantation (TAVI).[1] We would like to congratulate the European Association of Percutaneous Cardiovascular Interventions, the ESC working group on Cardiovascular surgery, and all the authors for this very nicely written document. It entails a very pertinent clinical issue, and this document will provide many cardiologists with an excellent summary of the available evidence regarding this topic. Nevertheless, it is a rapidly evolving field, and very recently, some interesting papers were published giving us further insights into the interaction between CAD and aortic valve stenosis (AS). We want to mention and provide some context regarding these findings and will focus on two aspects: (i) impact of CAD on outcomes and the role of percutaneous coronary intervention (PCI) and (ii) coronary physiology in AS and its potential to guide treatment.

Firstly, the consensus document mentioned the lack of evidence for prognostic benefit of PCI on short-term outcomes after valvular replacement. Recently, a study showed that this lack of prognostic benefit even persisted after 5 years of follow-up.[2] This is an important finding since more lower risk patients are currently being considered for TAVI. Furthermore, a suggestion is made that proximal left anterior descending disease may be a more attractive candidate for PCI. These new data suggest that it is the complexity of CAD, and not only the location of individual lesions, that is important in determining the long-term prognosis after TAVI.[2] A potential beneficial effect of complete revascularization, that sometimes is advocated, was however not found.[2]

Secondly, it has been hypothesized that using coronary physiology to select lesions for treatment might improve the prognostic effect of PCI. The main benefit of this strategy may lay in PCI deferral of non-flow limiting lesions, avoiding unnecessary exposure to dual antiplatelet therapy. The increased bleeding risk related to PCI in this population has been shown in the ACTIVATION trial as mentioned in the document.[1] Nevertheless, the currently used indices and cut-offs have not been validated in a severe AS population due to many potential pathophysiological pitfalls (Table 1 of the consensus document).[1] Studies investigating changes in physiological indices before and immediately after TAVI have provided us with conflicting results. Nevertheless, recent studies have provided more important insights. One group showed that basal coronary flow significantly decreased 6 months after TAVI.[3] Another recent paper showed that relative hyperaemic myocardial perfusion was significantly lower in AS patients.[4] Based on these findings, one can hypothesize that the non-hyperaemic pressure indices (NHPIs) have a higher change of overestimating CAD importance while the fractional flow reserve (FFR) might underestimate the significant of coronary lesions pre-TAVI. Whether it might be correct to use a lower cut-off for the NHPIs and a potentially higher cut-off for FFR in the AS population is currently being studied. A multi-centre cohort study with non-invasive ischaemia assessment and 6 months follow-up after aortic valve replacement will report its results next year.[5]

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