When Transcatheter Aortic Valve Replacement Is Not Enough
A Step Toward Understanding When Concomitant Mitral Regurgitation Needs Treatment*

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Transcatheter aortic valve replacement (TAVR) has revolutionized the treatment of severe aortic stenosis (AS), providing a less morbid method of restoring valve function than conventional open heart surgery. As experience accumulates and technology advances, TAVR will progress from a treatment reserved only for patients with AS who have prohibitive or high surgical risk toward a treatment for younger, healthier patients who have longer life expectancy (1,2). In these patients, excellent long-term prognosis and functional status rival low procedural morbidity as important considerations when selecting treatment strategies. Consequently, issues of TAVR prosthesis durability and the treatment of concomitant cardiac disease—such as coronary artery disease, ventricular dysfunction, and mitral regurgitation (MR)—are of growing importance.

Significant MR is among the most common cardiac diseases associated with severe AS, affecting about 25% of patients with severe AS (3). There are several potential pathophysiological connections between severe AS and mitral valve disease. Severe AS can cause or exacerbate MR by causing increased left ventricular diastolic pressure and/or ventricular dilation, stretching the mitral annulus, and reducing leaflet coaptation (4). Aortic valve fibrosis and calcification can extend into the mitral valve annulus and leaflets, preventing proper valve function (5). Additionally, concomitant coronary artery disease can indirectly affect mitral valve function by way of ventricular and papillary muscle ischemia and infarction. With 1 or more of these factors potentially present, reduction in MR after aortic valve replacement can hypothetically range from dramatic to none at all.

In a study reported in this issue of JACC: Cardiovascular Interventions, Cortes et al. (6) examined a multicenter population of 1,110 patients undergoing TAVR who were followed clinically and with echocardiography for 6 months. Some of Cortes et al.’s (6) findings have been demonstrated previously by others, such as the frequent presence of significant MR (in this study, defined as ≥3+) in the setting of severe AS (~16%) and the increase in mortality associated with concomitant MR after TAVR (7,8). However, this latter finding is suspect because of inadequate statistical adjustment for the many prognostically important comorbidities associated with concomitant MR in patients with severe AS (9). Also shown by others is Cortes et al.’s (6) finding of some reduction in the severity of MR in about 60% of patients after TAVR, demonstrating the direct pathophysiological role severe AS can play in mitral valve dysfunction (10). Unfortunately, this improvement did not translate into reduced mortality. Whether this is due to the relatively short 6-month follow-up period, beyond which mortality differences could grow and functional MR could further diminish, or due to the excess mortality risk...
attributable to the aforementioned comorbidities, is not clear.

This study’s most unique and important contribution to our knowledge is not its clinical findings, but rather its demonstration of novel imaging predictors of MR persistence after TAVR. A blinded imaging core laboratory was used to evaluate the pre- and post-TAVR echocardiograms and TAVR-planning cardiac computed tomographic angiograms in the 177 patients with significant MR. Among all the echocardiographic and computed tomographic measurements studied, computed tomographic mitral annular calcification (more than one-third of the annulus calcified), mitral leaflet calcification (calcium deposits on both leaflets), and mitral annular diameter were found to be strong independent pre-TAVR predictors of persistent significant MR. Using receiver-operator characteristic analysis, Cortés et al. (6) showed that a mitral valve annular diameter of >35.5 mm had a moderate ability to predict persistent MR (area under the curve, 0.77; sensitivity, 70%; specificity, 77%). However, this study, like others, shows that conventional echocardiographic designation of organic versus functional MR etiology is not an independent predictor of MR improvement after TAVR (11,12). Independent confirmation of these imaging criteria, and perhaps even the development of a “scoring system” based on imaging and clinical features predicting MR improvement, would be the ideal next steps.

An interesting, more speculative finding in this study was that only 13% of patients with persistent significant MR after TAVR would qualify for percutaneous mitral valve therapy in the form of MitraClip and perhaps even the development of a study was that only 13% of patients with persistent MR after TAVR. The importance of comprehensively addressing all functionally and prognostically important concomitant cardiac disease in patients with severe AS will grow, in order to optimize long-term prognosis. This study takes us one small, but important, step forward in this area, providing us with clues as to who may need therapy for concomitant MR.

REFERENCES


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