THE PUTATIVE MECHANISM OF RECURRENT VALVE REGURGITATION AFTER VALVE REPAIR IN ISCHEMIC MITRAL VALVE REGURGITATION

To the Editor:

We read with interest the articles written by Kron and colleagues,[1-3] who conducted the first prospective randomized clinical trial to compare mitral valve (MV) repair and MV replacement in patients with ischemic mitral regurgitation (IMR). In that study, mitral regurgitation (MR) recurred more frequently in the MV repair group (32.6% at 1 year and 58.8% at 2 years).

The authors attempted to address the complex issue of predicting MR recurrence after MV repair in patients with IMR. If we can discriminate between a subgroup of patients who would develop progressive MR recurrence after MV repair and those who would not, based on the preoperative clinical data, then we can maximize the treatment efficiency of MV repair by identifying patients who will benefit from MV repair preoperatively. Meanwhile, we can minimize the heart infarction caused by recurrent MR through the choice of MV replacement for patients who will experience severe MR recurrence.

The authors failed to prove the predictive values of preoperative echocardiography parameters of MV geometric tethering and ventricle remodeling, however.[4-6] Notably, the presence of a basal aneurysm/dyskinesis (BAD) remained the sole independent predictor of recurrent IMR after multivariate analysis.[1] This finding seems to be confounding and unreasonable, but the authors failed to explain the underlying mechanism.

Fundamentally, the mechanism of IMR relates to a mismatch between the ventricular and MV spatial geometry.[1] It is associated to remodeling and distortion of the ischemic left ventricle (LV) after myocardial infarction (MI).[7] LV myocardial ischemia leads to myocardial infarction, ventricle wall thinning, contraction decrease, and subsequent displacement of papillary muscle. Myocardial thinning also causes LV remodeling, leading to the enlargement of the MV annulus, affecting leaflet closure. Meanwhile, changes in anchoring position caused by papillary muscle displacement may tether the leaflets and affect valve closure, resulting in MR. Hsuan and associates[8] have identified MV tenting and interpapillary muscle distance as independent predictors of significant functional IMR in patients. Jensen and colleagues[9] reported that the distance between anterior and posterior papillary muscle showed the strongest correlations with regurgitant volume in a porcine model of IMR. The position of the anterior papillary muscle is superior, mural, and to the left, whereas that of the posterior papillary is inferior, septal, and to the right in the long axis of the heart. An infarction of myocardium between those 2 papillary muscles may lead to ventricle wall thinness and a change in distance that causes IMR. Chinits and colleagues[10] found that occlusion of the circumflex coronary artery caused a posterolateral MI between the anterolateral papillary muscle (APM) and posteromedial papillary muscle (PPM), and their multivariate analysis identified infarct size as a predictor of IMR grade.

This trial has not made the evaluation of the ravascularization strategy, as well as the stenosis status of coronary arteries, especially the artery dominating the inferior LV wall. Assessing the effect of revascularization alone during severe MR progression is difficult; however, analyzing the degree of coronary artery stenosis may facilitate our understanding of IMR. In disagreement with Kron and colleagues, we suggest that BAD is not an indicator of severe LV ischemic remodeling. The IMR patients with BAD represent a subgroup with different outcomes following MV repair. Taking anatomic factors into consideration, BAD is caused mainly by inferior MI after the corresponding coronary artery occlusion, usually the posterior descending artery from the dominant artery.

Because the PPM is often involved in posterior MIs, IMR is frequently detected in patients with congestive heart failure resulting from posterior LV dilatation.[11] An inferior MI might not cause BAD in all cases, but patients with BAD are likely to have a history of inferior MI or myocardial ischemia. Similar to inferior MI, BAD may be related to a severe form of LV ischemic remodeling. In this study, the majority of patients (roughly 80%) had a history of MI; however, the authors do not provide detailed data regarding MI position and coronary artery disease. Based on current limited data, we doubt that, if the ratio of inferior MI in the patients developing MR recurrence is higher than that in the patients not developing MR recurrence, just as the authors report, the ratio of BAD in the patients developing MR recurrence is higher than that in the patients not developing MR recurrence.

It may be that the underlying inferior MI affects the clinical outcome of MR repair, rather than the BAD. The underlying mechanisms of functional IMR caused by anterior MI and posterior MI are demonstrably different.[12,13] Anterior MI causes predominantly global LV dilatation, whereas inferior LV wall infarction causes less. Furthermore, PPM displacement and functional IMR grade are significantly

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higher in patients with inferior MI, indicating that inferior MI produces more severe geometric changes in the MV apparatus.[12] Agricola and associates[13] reported that the MV tethering caused by anterior wall MI is symmetric, whereas inferior wall infarction causes asymmetric tethering. For patients with previous inferior MI, remodeling can occur between these 2 papillary muscles or between the PPM and the posterior septum.[14]

In their study, Kron and colleagues did not further group the patients according to different MI positions; however, the technique of MV repair does not differ based on MI position. A mitral annuloplasty ring was applied to downsize the annulus diameter. The perioperative outcomes were desirable, with all patients demonstrating mild or no MR, confirmed by intraoperative transesophageal echocardiography.

Over time, patients with previous inferior MI or corresponding coronary artery disease may be at greater risk for recurrence of MR than patients with anterior MI. Regardless, concomitant coronary artery bypass surgery procedure was performed in 76% of the patients.[1] We do not have data on the revascularization of involved coronary arteries and postoperative catheterization to examine coronary artery bypass patency over time. Revascularization may retard the LV remodeling process due to MI or ischemia, but cannot totally prevent or reverse it, especially when local outpouching of the LV occurs. Furthermore, the PPM has 2-vessel perfusion in 37% of patients, compared with 71% for the APM,[15] making the PPM more vulnerable to ischemia. The PPM abnormality caused by an adjacent MI may more serious than an APM abnormality, and then the improvement of PPM dysfunction after revascularization would be less significant than that of APM dysfunction.

As LV remodeling and papillary muscle dysfunction progress, the risk of recurrence of MR will differ between patients with anterior MI and those with posterior MI. Continued LV dilation in patients with anterior MI is related to lower rate of recurrence, owing to a restriction of MV annulus dilatation by the artificial annuloplasty ring. In contrast, the progressive mechanistic abnormalities of PPM cause newly emerging leaflet tethering that cannot be eliminated by annulus downsizing, and thus those patients are at greater risk for early recurrence.

The inference above may help to understand why BAD can predict MR recurrence more accurately than the widely reported measurements of MV tethering and LV remodeling. This novel finding needs to be validated in further studies with larger numbers of subjects and the underlying mechanism requires detailed subgroup analysis.

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