Progression of Functional Tricuspid Regurgitation With Aortic Valve Stenosis After Intervention

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Functional tricuspid regurgitation (FTR) associated with aortic valve disease has recently become a focus of research. Because late significant FTR during follow-up is associated with a poor long-term mortality and high incidence of right-sided heart failure, the management of FTR with aortic valve disease is as important as that of FTR with mitral valve disease. However, strategies for management of FTR associated with aortic valve disease have not yet been determined.

The mechanism of FTR with aortic stenosis (AS) involves the following negative cascade: (1) left ventricular (LV) filling pressure increase is caused by aortic valve flow obstruction, causing concentric myocardial hypertrophy and declining compliance (diffuse fibrosis/extracellular matrix expansion of the LV myocardium), and increasing the LV end-diastolic pressure (LVEDP); (2) LVEDP increase also raises the left atrial (LA) pressure and pulmonary capillary wedge pressure; (3) pulmonary vascular compliance declines and adds to the increasing resistance against the right ventricle (RV) and as a result, the RV end-diastolic pressure increases and dilatation of the RV occurs (RV pressure overload and dilatation); (4) dilatation of the RV causes tricuspid annular dilatation and leads to RV failure, resulting in FTR progression; and (5) FTR progression also causes volume overload in the RV, and TR further exacerbates right-sided heart failure (Figure). Hence, an aortic valve intervention (aortic valve replacement

Figure. Mechanism of functional tricuspid regurgitation progression in patients with aortic valve stenosis. EDP, end-diastolic pressure; LA, left atrium; LV, left ventricle; PA, pulmonary artery; RV, right ventricle. (Reproduced from references 2,7,8 with permission.)
AVR or transcatheter aortic valve implantation) leads to LV reverse remodeling, which results in a reduction of LV mass, and reverse remodeling can interfere with the negative cascade of FTR progression. Previous studies report a positive effect of AVR for FTR improvement compared with conservative treatment. However, similar to FTR progression after mitral valve surgery, some FTR progression has been noted during follow-up after aortic valve intervention. It might be caused by an irreversible change in the pulmonary vascular bed, LV remodeling (the degree of diffuse interstitial myocardial fibrosis remains essentially unchanged), RV remodeling, and/or LA dysfunction (which also influences the occurrence of atrial fibrillation). Because of irreversible or inadequate reverse remodeling, the negative cascade of FTR will not be interrupted, and FTR will develop. Therefore, the timing of the intervention or indication of operation is very important in FTR management.

In previous issue of the Journal, Yajima et al report that the tricuspid annulus diameter index is an independent predictor of late significant FTR progression. They show that a tricuspid annulus diameter index >21 mm/m² is a sign of irreversible or inadequate reverse remodeling, and this parameter is very important as a cutoff value for the operative indication of FTR. Considering other parameters, the preoperative pulmonary artery (PA) pressure and/or LA diameter may be also adopted as a criterion for post-operative FTR progression. Pulmonary hypertension reflects long-standing stenosis, leading to LV diastolic dysfunction. LA diameter is also a parameter of LVEDP in AS, and correlates with PA pressure and the occurrence of atrial fibrillation, which are associated with right-sided heart failure.

In the study by Yajima et al, the postoperative aortic valve area, aorta-LV peak pressure gradient, and prosthesis-patient mismatch were not associated with late significant FTR progression, and there was no case of prosthetic valve failure in patients with late significant FTR progression. However, the degree of LV reverse remodeling during follow-up was not examined. Hence, the effects of reverse remodeling on FTR progression during follow-up were not clarified in their report, and other factors affecting FTR progression might exist. As a factor considered to exacerbate FTR, postoperative change might be a cause of late significant FTR progression. Paradoxical motion of the interventricular septum restricts the tricuspid septal leaflet motion and contributes to FTR progression. Igarashi et al reported that LV diastolic dysfunction and LV myocardial hypertrophy are the risk factors for FTR progression after AVR. Pericardial adhesion after operation might influence the diastolic dysfunction and add to the pressure overload. Regarding myocardial hypertrophy, FTR might develop in patients with hypertensive heart disease if the patient does not have valve disease. We expect to continue various investigations of FTR associated with aortic valve disease and accumulate data regarding this condition in the future.

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