Can Degenerative Changes Create Significant Tricuspid Valve Regurgitation?

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Dear Editor,

The predominant form of tricuspid regurgitation (TR) originates from annular dilation and right ventricular (RV) enlargement, mostly due to left heart diseases especially mitral valve stenosis rather than primary intrinsic valve involvement.

Also, pulmonary hypertension of any origin or pulmonary stenosis that increases RV systolic pressure to > 55 mm Hg (pressure overload) can cause secondary TR (1).

As the tricuspid valve (TV) annulus is a shared structure with the RV, secondary TR is typically associated with annular dilation. Hence, its elliptical and nonplanar shape converts to a much more circular pattern, mainly in the anterior and posterior portions neighboring RV free wall. However, because of fibrous skeleton, septal segment expansion is somewhat limited (2).

Apart from what was mentioned, some other various situations lead to organic valve deformation and primary TR. For example, in the congenital category, the Ebstein anomaly is a well-known TV involvement that is recognized as the apical displacement of the septal and, less commonly, posterior leaflets. It leads to inadequate coaptation and at least moderate TR in most patients. Congenitally corrected transposition of the great arteries with late pulmonary stenosis that increases RV systolic pressure to > 55 mm Hg (pressure overload) can cause secondary TR (1).

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A rheumatismal TV predominantly coexists with other valvular involvements. Isolated TR or a combination of TR and tricuspid stenosis (TS) happens owing to restricted mobility and short chordae tendinae.

The carcinoid syndrome affects the valve by the deposition of collagen, smooth muscle cells, and elastic tissue. TS/TR conjunction is a feature mostly noticed (4).

Myxomatous changes of the TV have coincidence with approximately 20% of mitral valve prolapses, and they may also be accompanied by atrial septal defects. When associated with syndromes like Marfan’s, the extent of degeneration increases.

Endocardial pace leads or implantable cardioverter-defibrillator leads are likely a preventable iatrogenic cause of TR. Mechanical interference and lead impingement as well as delayed RV activation and dysynchrony are the suggested mechanisms (5).

Penetrating or blunt trauma, infective endocarditis, radiation therapy, endomyocardial fibrosis, recurrent endomyocardial biopsies, and drug exposure to methysergide have been mentioned as other causes of TR (6).

We have faced several old patients (> 60 years) with thickened, calcified annuli and valves, obviously degenerative TV with significant TR without rheumatic or prolaptic changes, and important left-heart valve involvement (in transthoracic and even for a few of them in transesophageal echocardiography; 2D or 3D). However, in our expanded literature review, we did not clearly find this entity except for in an animal review study, which revealed that chronic degeneration manifested as thick and nodular lumpy valves evident in old dogs (manifesting in nearly 75% of them). The affected valves comprised the mitral valve (60%), both mitral valve and TV (30%), and the TV (merely 10%). The risk was elevated in older dogs, especially in breeds (7).

Accordingly, it seems that a new entity of degenerative change of the TV or “degenerative TR” is encountered, which cannot be classified in either of the etiologies that have been discussed until now (e.g., rheumatic and myxomatous-prolaptic) and needs further attention.

References


