The forgotten valve: lessons to be learned in tricuspid regurgitation

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This editorial refers to ‘Geometric changes after tricuspid annuloplasty and predictors of residual tricuspid regurgitation: a real-time three-dimensional echocardiography study’†, by S.-Y. Min et al., on page 2871

Only limited information is currently available about the prognostic significance of tricuspid regurgitation (TR). The few existing studies, however, suggest a strong impact of TR on clinical outcome. Significant TR is associated with poor prognosis in patients with mitral stenosis after percutaneous balloon valvuloplasty and with a reduction in exercise capacity after mitral valve surgery. A significant increase in mortality among patients with moderate and severe TR has been reported, which was independent of left ventricular ejection fraction or pulmonary artery pressure. In 60 patients with flail tricuspid leaflet due to trauma, significant increases in atrial fibrillation, heart failure, and death were observed. TR was also an independent predictor of increased mortality in 1400 patients with left ventricular systolic dysfunction.

TR is only rarely caused by primary abnormalities of the tricuspid leaflets. In most instances it is ‘functional’ in nature and is the consequence of geometric alterations caused by right ventricular dilatation, distortion of the subvalvular apparatus, tricuspid annular dilatation, or a combination of these factors.

Significant functional TR most commonly occurs in combination with left-sided heart disease, which often dominates the clinical picture. The development of TR leads to a vicious cycle propagating further right ventricular dilatation and dysfunction, more tricuspid annular dilatation, leaflet tethering, and, consequently, worsening of TR.

Although incomplete leaflet closure is the immediate cause of functional TR, the leaflets themselves are usually normal. What leads to such incomplete closure is the currently poorly understood geometrical alteration of the tricuspid apparatus that is caused by interaction between the altered tricuspid annulus size and shape, right ventricular remodelling, and displacement of papillary muscles (Figure 1) which lead to leaflet tethering. Min and co-workers have analysed the tricuspid valve apparatus using real-time three-dimensional echocardiography to predict residual TR after surgical annuloplasty. Tenting volume and antero-posterior tricuspid annulus diameter before surgery were the independent pre-operative predictors of short-term residual TR. In addition, the leaflet tenting angle between the tricuspid annulus line and the septal leaflet was a predictor for operative success. It is noteworthy that annuloplasty led to reduction of annulus size at the expense of an aggravation of leaflet tenting by inward displacement of the annulus. This was accompanied by a reduction of the septal-lateral diameter of the right ventricle, while antero-posterior dimensions remain unchanged.

Min’s results are an important step forward as we gain knowledge about new tools for the prediction of immediate surgical success. Nevertheless, two major questions remain unanswered.

First, what do these measurements tell us about the long-term success of tricuspid surgery? Follow-up data in this context are sparse. One small study on only 39 patients showed that in addition to tricuspid valve tethering, left ventricular as well as right ventricular function and pressure influence repair durability. Recent data from the Cleveland clinic on >2000 patients report a high recurrence rate of significant TR years after surgery, irrespective of the mode of repair. By 3 months after surgery, 34% of patients had moderate or severe TR, which increased to 45% of patients at 5 years. Risk factors of recurrent TR include higher grade of pre-operative TR, female gender, mitral valve replacement, and left ventricular dysfunction. Echocardiographic measurements were, however, not included in this analysis.

Secondly, current ESC and AHA/ACC guidelines on tricuspid valve surgery are based on small retrospective studies as well as on expert consensus, and prospective randomized trials on the benefit of tricuspid valve surgery on outcome are lacking.

In functional mitral regurgitation there is growing evidence for adaptive and compensatory mechanisms taking place in the leaflets. Based on the observation that leaflet area is substantially increased in patients with functional mitral regurgitation, Dal-Bianco et al. recently used a sheep model to test the hypothesis...
that it is through cell activation that mitral valve leaflet surface area increases over time with the mechanical stretch created by papillary muscle displacement. The mechanical stress-induced increases in leaflet area and in matrix thickness were associated with endothelial–mesenchymal transdifferentiation, supporting the concept of an actively adapting mitral valve tissue. The existence of similar adaptive mechanisms for the tricuspid valve is plausible, but remains to be shown.

While there appear to be similarities between the mechanisms leading to functional mitral and tricuspid regurgitation there are bound to be differences that, at present, are only incompletely understood. It is likely that the mitral and tricuspid annulus behave differently from each other when subjected to either pressure or volume overload, just as there are differences between the left and right ventricular myocardium. Also, the relative contributions of annular dilatation and leaflet tethering toward the development of valvular incompetence may differ between the mitral and the tricuspid valve.

Clinical implications

Pre-operative leaflet tethering and the size of the tricuspid annulus seem to be important parameters for the persistence of functional TR after surgery. Other parameters include right and left ventricular size, shape, function, and pulmonary artery pressures. The interaction of these parameters is strong and needs to be considered carefully to identify those patients who should undergo tricuspid valve repair.

Three-dimensional echocardiography of the tricuspid valve apparatus seems to provide important additional information for the decision as to whether a leaking tricuspid valve can be repaired successfully. However, in addition to short-term results, long-term prospective studies are needed to confirm the predictive power of such measurements.

Conflict of interest: none declared.

References

A 48-year-old man with unrepaired Tetralogy of Fallot (ToF) was admitted to our institution with dyspnoea on exertion and several episodes of cyanosis.

The diagnosis of ToF had been established in early infancy, but surgical repair had been repeatedly refused.

Retrospectively, ECG-gated 64-slice computed tomographic angiography (CTA) showed multiple abnormalities of cardiovascular morphology and function, and ruled out coronary artery disease and pulmonary embolism in a single non-invasive examination.

The following findings of ToF were demonstrated on CTA: an overriding aorta (Ao), a large outlet ventricular septal defect (arrow), a dilated and hypertrophic right ventricle (RV) (Panel A), with reduced left ventricular function (ejection fraction: 30%) and interventricular septum flattening (Panel B).

Right ventricular outflow tract obstruction was minimal (overlapping double outlet right ventricle anatomy). The pulmonary artery (Panel C) was aneurysmal with a diameter of 47 mm. The pulmonary valve was bicuspid (Panel D). There were no filling defects in the pulmonary arteries.

The aortic root and ascending AO were not dilated, with a non-stenotic tricuspid aortic valve.

The coronary arteries were anomalous but not stenotic. The right coronary artery (RCA) and left anterior descending artery arose from the tubular ascending AO with a normal course subsequently. The left circumflex artery originated from the RCA and took a retro-aortic course, terminating in the atrio-ventricular groove (Panel E).

Owing to advanced heart failure and pulmonary hypertension, surgical correction was not feasible, and the patient was referred for heart–lung transplantation.