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EDITOR'S PAGE


Right Ventricle

Innocent Bystander or Wolf in Sheep's Clothes?

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With the realization of the frequency of the poor prognosis and profound undertreatment of tricuspid regurgitation (TR), there has been an explosion of interest in TR, its imaging, and the associated structural interventions. The TRILUMINATE Study With Abbott Transcatheter Clip Repair System in Patients With Moderate or Greater TR trial[1] has generated considerable discussion regarding the population selection and the outcomes, which were positive for the composite endpoint but with no significant benefit in survival or heart failure hospitalization. We eagerly expect the imaging substudy of the trial to detect the impact of the treatment arm on right ventricular (RV) function. A crucial question regards how RV function is quantified because the RV is a difficult structure to analyze, and its assessment is affected by a series of limitations that have to be taken into consideration.

RIGHT HEART REMODELLING

TR should be quantified, but not as single measurements and always in conjunction with right heart size and function. This involves not only RV assessment but, most importantly, right atrial (RA) volume, which has been demonstrated to be predictive of outcomes.[2] Patients with severe TR and severe RA enlargement are at higher risk for decompensation from right-sided heart failure than those whose RA has not yet remodeled.[3] RA dilatation is associated with inferior vena cava dilatation, reduction of its respiratory collapsibility, and hepatic vein flow reversal, harbingers of right-sided heart failure.

The assessment of RV remodeling involves qualitative and quantitative descriptors.[4,5] RV dilatation can be judged visually by echocardiography or by measuring RV diameters or volumes by 3-dimensional echocardiography, although this imaging method is short on spatial and temporal resolution. Recently, multimodality imaging has allowed us to quantitatively assess RV volumes using computed tomography or magnetic resonance. In addition to RV election fraction, these methods allow the quantification of RV strain. We are entering a phase of relatively large cohorts with increasing follow-up, which provides early insights into the value of these measurements on short- and long-term outcomes. Thus, it is essential that these detailed measurements be collected in routine practice and integrated into the clinical management of patients with TR.[6]

However, the RV is also profoundly affected by the type of overload that affects the circulation efficiency of patients with TR. When the RV is not only dilated but elongated and globular, then the suspicion of pressure overload and pulmonary hypertension may be raised.[7] When the right heart is disproportionally dilated compared to the left heart, precapillary pulmonary hypertension may be suspected, and right heart catheterization is the gold standard investigation to identify the pulmonary hypertension and pulmonary vascular resistance. When the RV shape is well preserved with its conical appearance and generally good function, it is generally affected by pure volume loading. This qualitative assessment of the type of RV remodeling is clinically important because the globular remodeling associated with pulmonary hypertension and RV dysfunction yields increased distance between papillary muscles and tricuspid annulus with leaflet tethering, resulting in their tenting above the tricuspid annulus.[7] These changes are consequential because annuloplasty tends to cinch the annulus,

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increasing the distance to the papillary muscle and resulting in acute exacerbation of tenting, sometimes with severely incomplete coaptation and severe residual TR. Thus, interpreting the type of RV overload and remodeling has a direct influence on the treatment of TR. In a number of patients, the combination of pressure overload (or RV dysfunction) and volume overload is possible, especially at late stages of pulmonary hypertension, left-to-right shunts, or severe TR.

VENTRICULAR VS ATRIAL TRICUSPID REGURGITATION

Recently, functional TR (FTR) has been subsegmented into ventricular (V-FTR) and atrial (A-FTR) forms that are based on the observed type of remodeling. V-FTR is associated with a dilated, deformed RV and, quite often, impaired systolic function. The exact assessment of RV systolic function is thus crucial to recognizing V-FTR using a multiparametric approach, with indices such as the fractional area change, the RV free wall tissue Doppler imaging S-wave, the isovolumic relaxation time, and the RV outflow tract acceleration time.[8] A parameter that is being measured widely, the tricuspid annular plane systolic exertion, is volume dependent, which explains why in certain clinical trials, tricuspid annular plane systolic exertion appears pseudo-normal in patients with severe TR.

A-FTR is often but not exclusively observed in patients with chronic atrial fibrillation. The tricuspid annulus dilates because of atrial dilatation, yielding FTR and subsequent further RA dilatation in those patients with severe TR.[9] The possibility that insufficient valvular tissue contributes to the increasing severity of FTR is difficult to prove because we generally do not have imaging of the leaflet before FTR occurred. Generally, the A-FTR mechanism is type I with normal leaflet movement, but with advanced RV remodeling, there may be a combination of type III and restricted leaflet movement.

Thus, in assessing the right heart, the volume dependency of echocardiographic indices should be taken into consideration and, together with a 3-dimensional evaluation of volumes and ejection fraction as well as RV-to-pulmonary artery coupling and RV free wall strain, a multiparametric approach will indicate the degree of RV dysfunction. A promising imaging approach is stress echocardiography for the assessment of RV contractile reserve, but this requires rigorous studies. A careful assessment of the RV is important in the prediction of the worsening of RV function postsurgery or after structural intervention, sometimes abruptly after the interventions, and the criteria for futility (in general and based on RV function) have to be refined.

FORGOTTEN NOT MORE?

Today, a great percentage of our patients are those who had a previous left-sided heart surgery, and they develop right-sided heart failure, often with severe TR. Those patients who develop moderate or more severe FTR may benefit from referral to a tertiary center, and this management pathway has started to increase with the increased realization of the importance of TR.[10] However, TR management remains challenging. Careful, detailed patient selection and very strict inclusion criteria for prospective structural trials are important for successful outcomes but limit those offered therapy. Thus, more work is necessary to address the profound undertreatment of TR. Whether, in the context of TR, the right heart is an innocent bystander of TR or a wolf in sheep's clothes will require more attention in the years to come and warrants advanced imaging techniques to provide patients with TR with optimal outcomes.

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