Editorial

Tricuspid regurgitation and right ventricular dysfunction: preoperative non-procedural interventions to improve procedural outcomes

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Tricuspid regurgitation (TR), once dismissed as a secondary and relatively benign finding, is now recognized as a risk marker of morbidity and mortality (1).

The pathophysiology of TR is characterized by a vicious cycle of volume overload, right ventricular (RV) dilation, and progressive dysfunction. Chronic regurgitation exposes the RV to volume stress, causing remodeling and impaired contractility. As RV function deteriorates, forward flow decreases and systemic venous pressures rise, leading to hepatic congestion, renal impairment, and intestinal edema. These systemic consequences have been associated with poor outcomes, reduced tolerance to interventions, and hinder recovery (1-3).

With the growing use of transcatheter tricuspid valve interventions and renewed surgical interest, in recent years, the focus has shifted toward optimizing outcomes in a patient population that is often elderly, frail, and burdened by comorbidities.

The etiologic phenotype of TR, the presence and degree of RV dysfunction and its ability to recover, the degree of systemic congestion, and the existence and reversibility of organ dysfunction all strongly influence outcomes and procedural success (2-4). Early detection is therefore crucial. Yet, most patients with TR are identified late, when right heart failure (HF) is advanced.

Transthoracic and transesophageal echocardiography are fundamental to assess TR severity and tricuspid valve anatomy, to identify the mechanism underlying TR, and to evaluate RV and right atrial dimensions and function. A multiparametric evaluation of RV function should encompass conventional indices like tricuspid plane systolic excursion, RV fractional area change, tissue Doppler S', as well as more accurate and reliable parameters such as RV free wall longitudinal strain (RVFWLS) and three-dimensional RV ejection fraction (RVEF) (5,6). Conventional parameters may be overestimated in patients with TR mainly due to their load-dependency, while RVFWLS and RVEF have improved the accuracy of echocardiography in assessing RV function and prognostic stratification of patients. RV dysfunction is defined as the presence of an RVEF <50% and/or a RVFWLS <23% (5,6). Although current echocardiographic guidelines for the echocardiographic assessment of the right heart in adults (5) do not propose a grading system for the severity of RV dysfunction, the Tricuspid Valve Academic Research Consortium (4) has suggested possible cutoffs for the identification of moderate and severe RV dysfunction (15–19% and 15% respectively for RVFWLS and 35–45% and <35% respectively for RVEF). Cardiac magnetic resonance remains the most reproducible method for assessing chamber volumes, RVEF, as well as myocardial structural changes, and may have utility in patients with TR and poor acoustic window or in uncertain cases (6). In selected patients, low-dose dobutamine stress echocardiography

may provide insights into RV contractile reserve, although there is no evidence in patients with severe TR undergoing surgical or transcatheter intervention.

Since echocardiography can misestimate pulmonary pressure in the setting of severe TR and right HF, right heart catheterization has a key role to confirm or exclude pulmonary hypertension, to differentiate pre- from post-capillary phenotypes, and to define TR's hemodynamic impact (2,7). All these assessments should ideally be performed when patients are euvolemic, on stable diuretic therapy, with optimized pulmonary pressure and normal systemic blood pressure (2,4).

Identifying the etiological phenotype of TR is of the utmost importance to determine which patients may benefit from specific pre-procedural treatment to improve TR and which are less likely to benefit (2-4). Restoration of sinus rhythm, correction of severe left-sided valvular heart disease, and specific drug and resynchronization therapy in patients with HF and reduced ejection fraction (HFrEF) have been associated with reductions in TR severity and/or improvement in RV function (2,7-11). To date, no specific drug has demonstrated a positive effect on symptoms, quality of life, clinical course and prognosis in patients with TR; however, a small randomized controlled trial showed that sodium-glucose cotransporter 2 inhibitors, in addition to other HF drugs, were found to be more effective in improving RV function in patients with HFrEF as compared to other drugs alone (8). Moreover, sacubitril/valsartan was found to improve RV performance and pulmonary hypertension in patients with HFrEF independently of left ventricular reverse remodeling (9). Pulmonary vasodilators can reduce pulmonary hypertension and consequently improve TR (10). Although diuretic therapy did not improve outcomes or delay TR treatment when indicated, volume status optimization is fundamental for achieving an optimal procedural result (7). Loop diuretics remain first-line therapy. At advanced stages of disease, diuretic resistance can occur due to renal congestion and malabsorption, and sequential nephron blockade with thiazide-like agents or acetazolamide could be added. Mineralocorticoid receptor antagonists modulate the expression/activity of sodium and potassium channels in the distal nephrons, avoiding hypokalemia and exhibit pleiotropic effects. In cases of refractory congestion, ultrafiltration offers a controlled, isotonic removal of fluid, reducing RV preload without worsening electrolyte imbalance. Inotropic agents and vasopressors can be considered in case of peripheral hypoperfusion and or as a bridge to tricuspid intervention (7).

Regardless of the treatment strategy, it has been shown that patients with atrial secondary TR with isolated atrial fibrillation or HF and preserved ejection fraction have a more favorable outcome when diagnosed early. Conversely, those with ventricular secondary TR, especially in the presence of pre-capillary pulmonary hypertension, exhibited a poor prognosis and TR intervention in these patients could be futile (2,3,7,12). In conclusion, TR represents a complex interplay of hemodynamic and systemic derangements. Comprehensive pre-procedural optimization, encompassing volume and afterload management, organ support, arrhythmia control, and structured assessment of RV dimension and function and pulmonary pressures, is essential to enhance procedural success and patient recovery. Early recognition, multidisciplinary collaboration, careful patient selection, and timely intervention are essential to improve outcomes. Bridging current evidence gaps through targeted research will refine protocols and advance the care of patients with TR.

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Footnote

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