



Atrial functional tricuspid regurgitation: An underappreciated cause of secondary tricuspid regurgitation

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Secondary tricuspid regurgitation (TR) caused by right ventricular enlargement in the setting of left heart disease/pulmonary hypertension has been well described. In contrast, that associated with right atrial enlargement—atrial functional TR (AF-TR)—remains largely underappreciated. AF-TR most often occurs in the setting of lone atrial fibrillation, although it is also seen in its absence (idiopathic AF-TR). Several recent studies have found that the prevalence, hemodynamic significance, and prognosis of AF-TR are not inconsequential, suggesting increased physician awareness of this novel clinical entity is warranted. This article discusses the pathogenesis, echocardiographic findings, and treatment of this underappreciated cause of secondary TR.

KEYWORDS

atrial functional tricuspid regurgitation, lone atrial fibrillation, secondary tricuspid regurgitation, tricuspid annulus, tricuspid valve repair

1 | INTRODUCTION

Interest in secondary tricuspid regurgitation (TR) caused by right ventricular (RV) enlargement (ventricular functional TR) in the setting left heart disease/pulmonary hypertension has increased in the past few years.¹ In contrast, secondary TR caused by right atrial enlargement, so-called atrial functional TR (AF-TR) remains largely underappreciated. AF-TR most often occurs in the setting of lone atrial fibrillation, although it is also seen in its absence (idiopathic AF-TR), notably in the elderly.^{2,3}

Atrial functional tricuspid regurgitation was first noted in 2002 by Zhou, et al⁴ who observed significant (moderate or severe) TR in approximately 1/3 of a small cohort with lone atrial fibrillation. In a more recent echocardiographic study of 300 subjects,⁵ Abe, et al found that the prevalence of significant AF-TR was 17% in those with lone atrial fibrillation of 1–10 years duration and 25% in those with lone atrial fibrillation for >10 years. The selective nature of such studies notwithstanding, it nonetheless appears that the burden of significant AF-TR is not insignificant, underscoring the need for increased physician awareness of this novel clinical entity.

2 | PATHOGENESIS

Atrial functional tricuspid regurgitation is thought to result from the increase in annular area that accompanies right atrial enlargement.² Notably, dilatation of the annulus is confined to its more pliable, fat-rich portion found along the RV free wall (Figure 1).^{6–8} Regurgitation develops as the free margins of the tricuspid valve leaflets are moved apart by the expanding annulus. In vitro modeling suggests that as much as a 40% increase in annular area can be tolerated before coaptation reserve is exhausted and regurgitation develops.⁹ In contrast to AF-TR, annular dilatation is less extensive and does not appear to play a significant role in ventricular functional TR. In this instance, TR is primarily the result of leaflet tethering caused by papillary muscle displacement that accompanies RV remodeling² (Figures 2 and 3).

In the past few years, the phenomenon of adaptive leaflet growth (valvulogenesis) has been shown to be an important disease modifier that decreases valvular regurgitation by increasing leaflet coverage area relative to closing area. Adaptive leaflet growth has been demonstrated in ventricular functional TR,¹⁰ ischemic mitral regurgitation,¹¹ and AF-mitral regurgitation.¹² Additional research is needed to determine to what extent it influences TR severity in AF-TR.

3 | ECHOCARDIOGRAPHIC FINDINGS

Patients with AF-TR due to lone AF typically have bi-atrial enlargement, however, combined significant MR and TR occurs in only 5%

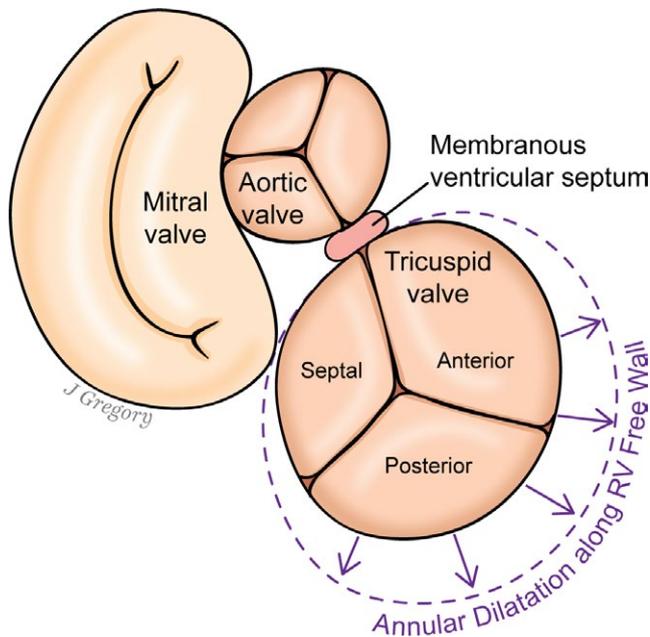


FIGURE 1 Anatomic relations of the tricuspid annulus. Note that annular enlargement is confined to the portion of the annulus attached to the right ventricular free wall

of patients.⁵ Leaflet effacement caused by annular enlargement decreases the height of the zone of coaptation but leaves the leaflets confined to the annular plane such that tenting height is not significantly increased (Figures 2 and 3).² Less well appreciated is the fact that RV enlargement may occur in AF-TR. This, unlike the more diffuse RV enlargement (increased ventricular length and sphericity) seen with ventricular functional TR, is largely confined to the base of the ventricle (Figure 2).² However, since this region is devoid of papillary muscles, leaflet tethering, in contrast to ventricular functional TR, is characteristically absent (Figures 2 and 3).² Guidelines for assessing TR severity echocardiographically emphasize the need for concordance of multiple parameters (vena contracta width, effective regurgitant orifice area, jet size, adequacy of leaflet coaptation, spectral Doppler shape and density, RV size and function and hepatic venous flow).¹³ Reliance on any single parameter can lead to incorrect conclusions regarding TR severity. For example, jet size often underestimates the amount of TR when there is poor leaflet coaptation; in such instances downstream (right atrial) flow tends to become laminar, resulting in less prominent TR jets.¹³ It should also be noted that the presence hepatic vein systolic flow reversal, an otherwise specific marker of severe TR, is not useful in the setting of atrial fibrillation, where it instead reflects the absence of atrial activity.¹³ Last, it is important to appreciate that the regurgitant orifice in AF-TR can assume an elliptical shape which may result in underestimation of the effective regurgitant orifice area because a hemispheric proximal convergence zone can no longer be assumed.¹⁴

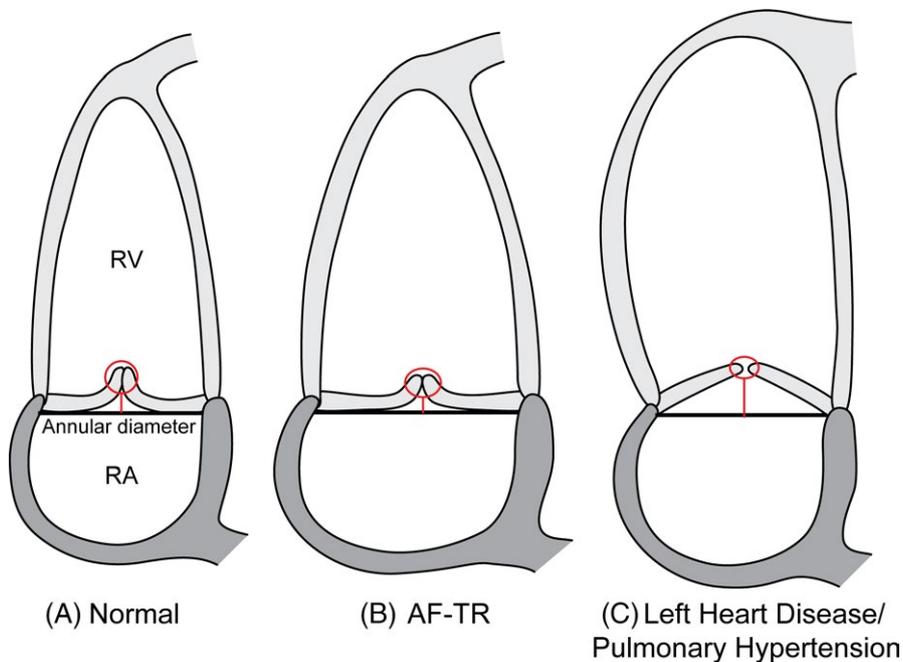


FIGURE 2 Patterns of right ventricular (RV) remodeling in secondary tricuspid regurgitation (TR). The encircled regions represent the zone of coaptation. Tenting height is indicated by vertical red lines. A, Normal tricuspid annulus, right atrium (RA) and RV. Note the tall coaptation zone. B, RV enlargement in AF-TR is largely confined to the base. Annular enlargement causes the leaflets to move apart reducing the height of the zone of coaptation. Tenting height does not increase. C, RV enlargement due to left heart disease/pulmonary hypertension increases chamber length (apex-forming RV) and sphericity. Annular enlargement is less prominent than in AF-TR, however, tenting height is increased due to leaflet tethering. Complete leaflet effacement has eliminated the coaptation zone

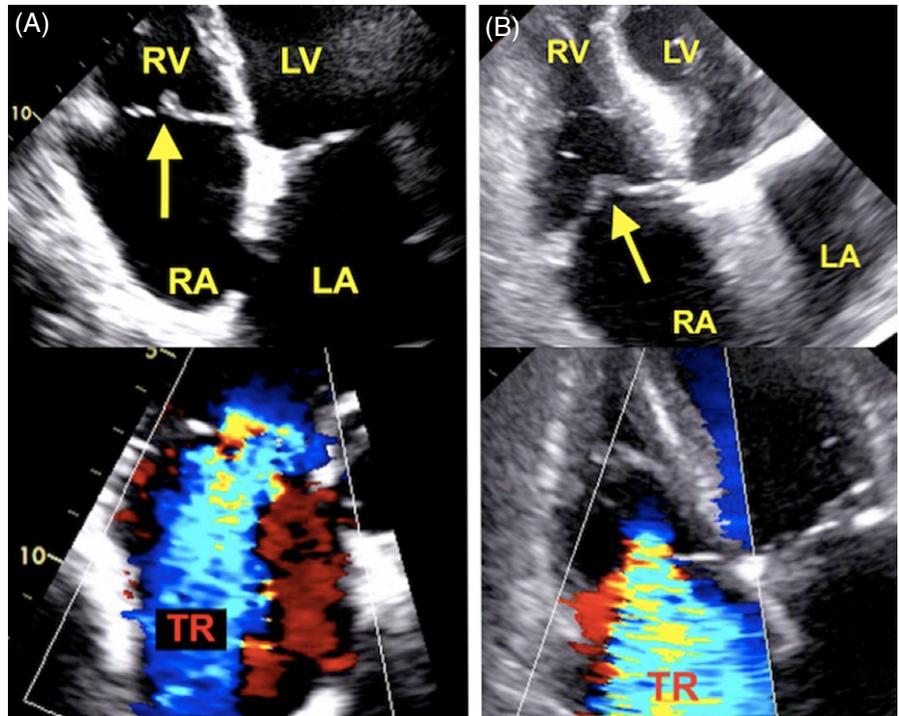


FIGURE 3 Echocardiographic images of secondary tricuspid regurgitation (TR). A, Severe atrial functional (AF)-TR. Note that the tricuspid valve leaflets remain within the annular plane (arrow). B, Severe secondary TR due to left heart disease/pulmonary hypertension. Note the presence of marked leaflet tenting (arrow)

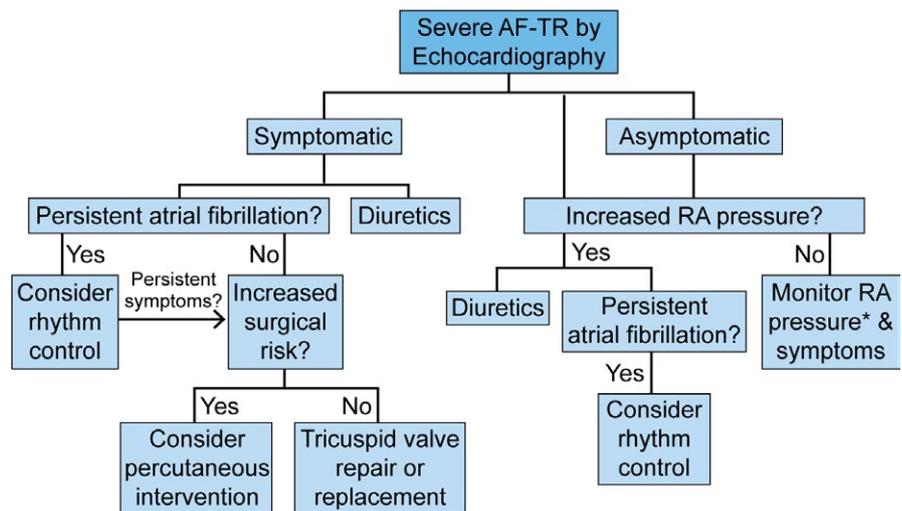


FIGURE 4 Suggested algorithm for medical and surgical management of atrial functional tricuspid regurgitation. See text. RA = right atrial

*Monitor RA pressure by physical examination, echocardiography, or hemodynamic catheterization

4 | TREATMENT

Current guidelines¹⁵ do not specifically address therapy of AF-TR, however, a treatment algorithm has recently been proposed by Nishimura, et al¹⁶ (Figure 4). Medical therapy, consisting of maintenance of sinus rhythm and reducing elevated right atrial pressure with diuretics may promote reverse atrial remodeling, reduce annular size and decrease TR severity.¹⁶ It is suggested that surgical intervention be reserved for patients with symptomatic severe AF-TR. While surgery has been shown to improve symptoms in these patients, there are few data on long-term outcomes.¹⁷ Unoperated patients have, however, been shown to have a poor prognosis. One study revealed 5- and 10-year mortality rates of

35% and 65%, respectively.¹⁷ The role of novel percutaneous tricuspid valve interventions in patients with AF-TR remains to be determined.¹⁸

5 | CONCLUSION

Atrial functional tricuspid regurgitation is a common cause of secondary TR that is easily recognized echocardiographically. The foregoing discussion underscores the need for increased physician awareness of the clinical significance this disorder. Additional studies are needed to determine the optimal timing and outcomes of medical, surgical, and percutaneous interventions.

DISCLOSURES

No specific grant from funding agencies in the public, commercial, or not-for-profit sectors was received for this work. There are no conflicts of interest to report.

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How to cite this article: Silbiger JJ. Atrial functional tricuspid regurgitation: An underappreciated cause of secondary tricuspid regurgitation. *Echocardiography*. 2019;00:1–4. <https://doi.org/10.1111/echo.14327>