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Tricuspid Regurgitation Worsening After Pericardiectomy in Tuberculosis Constrictive Pericarditis: An Overlooked Prognostic Concern

LETTER TO THE EDITOR

To the Editor,

The article by Wang et al[1] entitled "Tricuspid Regurgitation in Tuberculous Constrictive Pericarditis Underwent Pericardiectomy," recently published in your esteemed journal, was read with great interest. The authors are sincerely thanked for this valuable contribution, which highlights an often underrecognized component of right-sided heart involvement in constrictive pericarditis (CP), especially in the setting of tuberculosis.

Wang et al[1] have provided important insights into the prevalence and predictors of tricuspid regurgitation (TR) following pericardiectomy in patients with tuberculous CP. Their findings underscore the need to appreciate TR not merely as a bystander lesion but as a dynamic and prognostically significant abnormality, even in modest grades.[2-4] The association between preoperative right ventricular (RV) dysfunction and postoperative TR progression aligns with evolving data emphasizing the importance of RV-TR interaction.[5]

Constrictive pericarditis due to tuberculosis remains a critical surgical challenge in endemic regions, and pericardiectomy, while effective in relieving pericardial constraint, may unmask latent RV dysfunction or geometrical distortion. Several studies have suggested that even with technically complete pericardial resection, residual fibrosis, adherence, or myocardial tethering may contribute to deteriorating tricuspid valve competence.[3]

Furthermore, the dynamic interplay between RV contractility, pericardial compliance, and annular dilation is gaining recognition. Right ventricular fractional area change has emerged as a particularly sensitive marker of intrinsic systolic dysfunction, superior to TAPSE or tissue Doppler indices in postoperative settings where volume loading and longitudinal motion may be compromised. This may explain why patients with preoperative RV systolic impairment tend to experience worsening TR despite successful decompression.[3]

Importantly, pericardial thickness—especially over the RV free wall—may serve as a surrogate for inflammatory burden and technical complexity during surgery. Imaging modalities such as cardiac magnetic resonance imaging and computed tomography have shown utility in preoperative stratification of such risks.[4,5]

In conclusion, Wang et al[1] deserve commendation for directing attention to this clinically important, yet often overlooked sequela of pericardiectomy in tuberculous CP. Their work supports the growing consensus that routine evaluation of RV function and pericardial morphology should inform surgical planning and postoperative surveillance strategies. Their call for further prospective studies is echoed to refine the understanding of TR evolution and its prognostic implications in this unique patient population.

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