REVIEW ARTICLE

MANAGEMENT OF TRICUSPID REGURGITATION IN PATIENTS WITH RHEUMATIC MITRAL DISEASE

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Abstract

Background: *Rheumatic heart disease (RHD) remains a major cause of cardiovascular morbidity and mortality in low and middle-income countries, with tricuspid regurgitation (TR) frequently complicating rheumatic mitral pathology. While mild TR often stabilizes after mitral correction, management of moderate and severe TR in this context remains controversial due to limited evidence and applicability of guidelines largely derived from degenerative etiologies. This review synthesizes current data on diagnostic strategies, surgical decision-making, and outcomes in rheumatic TR.*

Methods: *A comprehensive literature search was performed across PubMed, Google Scholar, and OMNI databases (2010–2024) using the keywords "rheumatic mitral disease," "tricuspid regurgitation," and "management." Eligible studies included clinical trials, case series, reviews, and meta-analyses focusing on TR associated with rheumatic mitral disease. Data was extracted and critically appraised for study design, population, and outcome relevance.*

Results: *Evidence for managing TR in rheumatic mitral disease primarily stems from retrospective cohorts and small prospective studies, with few randomized trials. Predictors of TR progression include annular dilation (>21 mm/m^2), right ventricular dysfunction, pulmonary hypertension, and atrial fibrillation. Mild TR generally regresses following mitral surgery and is managed conservatively. For moderate TR, concomitant repair may prevent late progression and improve hemodynamics, though risks of postoperative arrhythmia and pacemaker implantation persist. Severe TR warrants surgical correction, preferably rigid ring annuloplasty, while valve replacement is reserved for advanced calcific disease. Emerging transcatheter therapies show promise for high-risk patients but lack robust rheumatic-specific data.*

Conclusion: *Optimal management of TR in rheumatic mitral disease requires individualized, Heart Team–based decision-making due to limited high-quality evidence. Future research should prioritize multicenter trials comparing repair, replacement, and transcatheter approaches, integrating advanced imaging for risk stratification and addressing access disparities in resource-limited settings.*

INTRODUCTION

Rheumatic heart disease (RHD) is a leading cause of cardiovascular morbidity and mortality in low- and middle-income countries (LMICs), driven by untreated streptococcal infections. The 2021 Global Burden of Disease Study estimates 54.8 million RHD cases globally (95% uncertainty interval [UI], 43.3–67.6), contributing to 360,000 deaths and 10.67 million disability-adjusted life years annually, with the highest burden in sub-Saharan Africa, South Asia, and Oceania[1]. Acute rheumatic fever, triggered by Group A Streptococcus (GAS), initiates an immune-mediated inflammatory response targeting heart valves, predominantly the mitral valve (MV), resulting in thickening, scarring, and deformity[2].

RHD commonly affects the MV, resulting in isolated stenosis, regurgitation, or combined lesions (Table 1)[3]. It can also involve the tricuspid valve (TV), causing rheumatic

tricuspid valve disease, characterized by leaflet thickening, commissural fusion, chordal shortening, and calcification[3]. Tricuspid regurgitation (TR) in RHD is classified as primary or secondary. Primary TR is direct rheumatic damage to TV leaflets/subvalvular structures, whereas secondary, also called functional TR, is due to left-sided valvular disease and pulmonary hypertension causing right ventricular (RV) remodeling and annular dilatation[4]. This differs from isolated functional TR, where leaflets are structurally normal, or degenerative TR, driven by prolapse or flail leaflets, necessitating distinct management strategies due to differences in tissue integrity and progression [5].

Functional TR is present in over 96% of patients with rheumatic MV disease (mild 83.7%, moderate 8.5%, severe 4.3%), with worse outcomes: 3-year event-free survival drops from 91% (mild/absent TR) to 72% (moderate) and 62% (severe)[6]. TR severity is categorized as mild, moderate, or severe based on retrograde flow from the right ventricle to the right atrium during systole[7]. Mild TR is often asymptomatic and managed conservatively, while severe TR typically requires surgical intervention to prevent right heart failure. Moderate TR management remains controversial due to limited evidence, unlike degenerative cases with clearer guidelines. Therapeutic strategies must be tailored to the patient's clinical profile, pathology, and physician expertise.

Prior reviews often focused on functional or degenerative TR, with limited emphasis on rheumatic-specific aspects[8]. A 2010 review addressed rheumatic tricuspid valve disease (RTVD) epidemiology but lacked integration with mitral disease[4]. Recent analyses explored TR progression in RHD but omitted comprehensive treatment comparisons[6]. Key gaps include underrepresentation of RTVD, pitfalls in applying degenerative data, detailed surgical technique comparisons, and emerging transcatheter options in rheumatic contexts. This narrative review summarizes current practices for managing TR in rheumatic MV disease, emphasizing decision-making, surgical versus conservative approaches, and postoperative outcomes, addressing these gaps.

Search Methods

For this narrative review, an electronic search was conducted in PubMed, Google Scholar, and OMNI databases, prioritizing studies on RHD with a focus on rheumatic mitral and tricuspid pathology. Inclusion criteria: 1) publications from 2010–present; 2) case series, case reports, systematic reviews, randomized controlled trials, and clinical trials; 3) English language; 4) papers addressing TR management in rheumatic mitral disease. Exclusions: inaccessible full texts or studies primarily addressing non-rheumatic etiologies without rheumatic-specific insights.

MeSH keywords included: "rheumatic mitral disease," "tricuspid regurgitation," "rheumatic disease," "mitral disease," "surgery," "management." Titles and abstracts were screened for relevance, followed by full-text review. Disagreements were resolved by consensus. A secondary search reviewed reference lists of included papers. Studies were critically appraised for design (e.g., retrospective vs. prospective), sample size, and limitations, with quantitative data from meta-analyses presented where applicable.

Findings

The evidence base for managing TR in rheumatic mitral disease is derived from a heterogeneous mix of study designs, including predominantly retrospective cohort studies, a limited number of prospective trials, and meta-analyses, with a notable scarcity of large-scale randomized controlled trials. This body of literature often suffers from methodological limitations, such as small sample sizes (frequently under 200 participants), reliance on single-center data, and inclusion of studies dated over a decade old, which may not fully reflect advancements in surgical techniques, imaging modalities, or perioperative care[9]. For example, many retrospective analyses are prone to selection bias, confounding variables, and incomplete follow-up, potentially overestimating or underestimating treatment effects. Recent meta-analyses, such as that by Awad et al. in 2023, offer more robust insights by pooling data from multiple sources but are constrained by interstudy heterogeneity, as evidenced by varying definitions of TR severity and inconsistent reporting of long-term outcomes (odds ratio for TR progression 0.08, 95% CI 0.05–0.16)[10]. Distinctions between study types are essential for interpretation: retrospective designs provide real-world data but lack randomization, while prospective studies, though fewer, offer better control over variables but are often underpowered. Overall, these limitations underscore the need for high-quality, multicenter randomized trials to strengthen recommendations, particularly in rheumatic populations where evidence remains weaker compared to degenerative or functional TR etiologies.

Predictors of late TR progression in rheumatic mitral disease encompass a range of clinical, hemodynamic, and imaging parameters that inform risk stratification and therapeutic decision-making. Key factors include tricuspid annular dilation, persistent atrial fibrillation, right ventricular (RV) dysfunction and dilation, elevated pulmonary artery pressures, and preoperative TR severity, all of which contribute to progressive right heart remodeling if unaddressed[11,12]. Imaging thresholds play a pivotal role in guiding intervention timing: two-dimensional (2D) echocardiography typically recommends a tricuspid annulus (TA) diameter cutoff of >40 mm or indexed >21 mm/m^2 for considering repair, but three-dimensional (3D) echocardiography provides superior accuracy, suggesting thresholds of >42 mm or >23 mm/m^2 to account for the annulus's non-planar geometry, as 2D methods can underestimate dimensions by up to 20% [11,12]. Annular dilation exceeding 21 mm/m^2 is associated with an elevated risk of TR recurrence (hazard ratio 1.8,

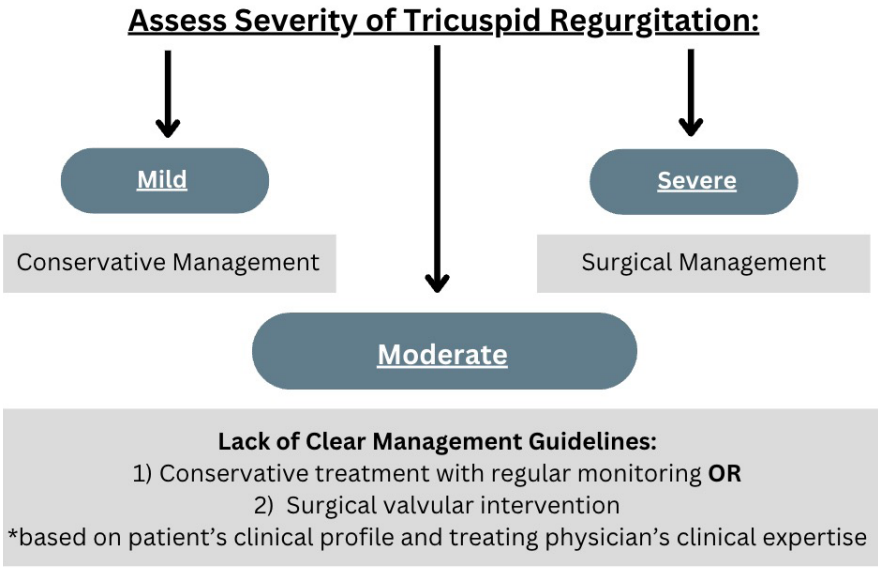

Figure 1 *Management of TR based on level of severity*

95% confidence interval [CI] 1.2–2.7), highlighting its prognostic value. Additional predictors from recent studies include postoperative RV remodeling failure and residual pulmonary hypertension, which can exacerbate TR progression even after successful mitral intervention[22]. These parameters not only aid in preoperative assessment but also emphasize the importance of multimodal imaging, including cardiac magnetic resonance for precise RV function evaluation, to optimize patient selection and outcomes in this challenging population.

Patients with Mild TR

In patients with rheumatic heart disease presenting with mild TR, the primary therapeutic emphasis is placed on correcting the underlying rheumatic mitral valve pathology, rather than pursuing direct intervention on the tricuspid valve itself (Figure 1)[13]. This approach is supported by evidence indicating that mild TR exerts only a minimal influence on overall cardiac function and long-term patient outcomes, often remaining stable or even regressing spontaneously following successful mitral valve surgery[9]. For instance, studies have demonstrated that isolated mitral intervention can lead to a reduction in pulmonary pressures and subsequent improvement in right ventricular loading conditions, thereby mitigating mild TR without additional procedures[18]. However, the decision to forego tricuspid valve surgery is also driven by the potential risks associated with concomitant interventions, including an increased likelihood of requiring permanent pacemaker implantation, iatrogenic right coronary artery lesion, and aortic valve distortion, bleeding complications from right atriotomy, and a higher incidence of postoperative atrial arrhythmias[13]. A comprehensive 2023 meta-analysis encompassing 1,456 patients from retrospective studies reinforced this conservative stance, revealing no significant mortality benefit from prophylactic tricuspid repair in mild TR cases (risk ratio [RR] 1.02, 95% CI 0.85–1.22), although the analysis was limited by non-randomized designs and potential confounding factors such as varying surgical expertise across centers[10].

Conservative management strategies for mild TR involve meticulous monitoring and supportive care to prevent progression. Regular clinical evaluations, incorporating serial echocardiography, are indispensable for tracking TR severity, right ventricular function, and associated hemodynamic changes. Antibiotic prophylaxis is recommended to avert recurrent streptococcal infections, which could precipitate further valvular deterioration and exacerbate rheumatic involvement[2]. Optimization of heart failure therapy, including fluid status control through diuretics and lifestyle modifications such as sodium-restricted diets and moderate physical activity, plays a crucial role in alleviating symptoms and preserving cardiac reserve[3]. Follow-up intervals with a cardiologist should be tailored to individual risk profiles, typically every 6–12 months with echocardiography,

Table 1 **Overview of rheumatic heart disease and associated pathologies**

Rheumatic Heart Disease	Characteristics
Pathophysiology	· Rheumatic mitral disease originates from inadequately treated Group A streptococcal infections.
Mechanism of Valve Damage	· Initiated by acute rheumatic fever. · Immune-mediated inflammation damages heart valves, primarily the mitral valve. · Results in thickening, fibrosis, and deformation of valve leaflets.
Mitral Valve Pathology	· Leads to: Isolated mitral stenosis Isolated mitral regurgitation Combined valvular lesions
Tricuspid Valve Pathology	· Frequently involved in rheumatic heart disease. · Dysfunction resembles mitral valve pathology. · Regurgitation may develop secondary to mitral valve-induced hemodynamic changes.
Cardiac Hemodynamics	· Combined valvular dysfunction increases right heart chamber workload. · Potential progression to right-sided heart failure.
Therapeutic Considerations	· Address primary mitral valve pathology. · Monitor and manage tricuspid regurgitation. · Aim to prevent progressive cardiac dysfunction and optimize patient outcomes.

escalating to 3–6 months in the presence of worsening symptoms or echocardiographic evidence of progression[3]. In cases complicated by atrial fibrillation, anticoagulation is warranted to mitigate thromboembolic risks, further underscoring a multidisciplinary approach that integrates medical therapy with vigilant surveillance to ensure timely escalation if TR advances to moderate or severe grades[14].

Patients with Moderate TR

The management of moderate functional TR in patients undergoing surgery for rheumatic mitral valve disease presents a multifaceted clinical challenge, characterized by ongoing debates regarding the merits of concomitant tricuspid valve repair versus observation (Central Illustration)[14]. Proponents of intervention argue that addressing moderate TR at the time of mitral surgery can yield substantial benefits, including a decreased incidence of postoperative low cardiac output syndrome, reduced need for late tricuspid re-interventions, and lower rates of rehospitalization for congestive heart failure[15]. This perspective is bolstered by evidence showing that concurrent repair enhances overall cardiac hemodynamics and patient quality of life, particularly through techniques like De Vega's suture annuloplasty, which has demonstrated durable results in moderate secondary TR within rheumatic contexts[17]. Furthermore, successful postoperative reduction in systolic pulmonary artery pressure (sPAP) and right ventricular systolic pressure (RVSP) has been linked to halted TR progression, even among patients with severe preoperative pulmonary hypertension, as mitral correction alleviates left-sided overload and its downstream effects on the right heart[18]. A prospective cohort study involving 120 patients illustrated these advantages, reporting an 85% freedom from severe TR at 5 years with repair compared to 70% without, although the study's small sample size and single-center nature temper its generalizability[15].

Conversely, outcomes vary when treatment decisions are based solely on indexed tricuspid annulus dimensions (e.g., <21 mm/m^2), with some cohorts experiencing inconsistent efficacy and suboptimal long-term functional results[19]. Notably, up to 16.3% of patients who undergo tricuspid valve repair may develop persistent moderate-to-severe tricuspid insufficiency and subsequent heart failure during follow-up, highlighting interpatient variability and the necessity for individualized strategies[18]. Preoperative evaluation of tricuspid annulus fractional shortening has emerged as a valuable prognostic tool, with values below 25% correlating to a 57.1% rate of adverse late events, in contrast to superior outcomes in those exceeding this threshold, thereby advocating for comprehensive preoperative risk assessment to guide surgical planning[19]. Medical management parallels that of mild TR, incorporating diuretics and beta-blockers for symptom alleviation and congestion control, alongside regular monitoring every 3–6 months to detect progression early14. Recent investigations emphasize the role of patient-specific factors, such as RV function and atrial fibrillation burden, in tipping the balance toward intervention, reinforcing the need for Heart Valve Team discussions to weigh surgical risks against potential long-term benefits in this intermediate-risk group[15,16].

Table 2 **Comparative summary of key studies on tr management in rheumatic mitral disease**

Study	Design	Patient Cohort (n)	Surgical Intervention	TR Outcomes	Limitations
Arafat et al., 2022 [3]	Retrospective cohort	480	Compare long-term survival and TR recurrence after concomitant TV repair in rheumatic vs. degenerative MV disease	Recurrence of TR was higher with MV replacement compared with repair (shr: 1.69 [1.03−2.78], P = .038) Degree of TR did not differ between groups before/after matching	Non-randomised, selection bias
Kim et al., 2023 [7]	Retrospective	541	Concomitant TV repair for mild-moderate TR	No significant differences between repair vs no repair.	Heterogeneity, non-randomised
Fawzy et al., 2020 [8]	Prospective cohort	974	TV repair for moderate TR	Concomitant TV repair reduced the need for later TV reintervention and CHF rehospitalization compared with mv surgery alone	non-randomised, single-centre
Naqshband et al., 2010 [9]	Prospective follow up	106	Tricuspid valve repair	100% Freedom from TR with de vega's repair at the end of 7 years vs existing TR with suture bicuspidization and no repair	Dated, small n
Kim et al., 2020 [11]	Retrospective	227	Tricuspid annuloplasty	Rate of freedom from development of moderate or severe TR at 5 years was 96.2%	Single center, small sampled
Colombo et al., 2011 [12]	Prospective analysis	50	Tricuspid annuloplasty	Effective in terms of clinical efficacy and of late functional result	Small sample size
Jeong et al., 2013 [13]	Retrospective	106	Reoperation post-MV	Can be performed at low risk with good clinical outcomes	Small n
Ariyoshi et al., 2013 [14]	Retrospective	99	MV surgery	93.6% Improvement rate in tricuspid annuloplasty	Small sample size
Zhong et al., 2021 [15]	Retrospective	170	Concomitant annuloplasty	Concomitant tap was associated with postoperative rv volume reduction ($p < 0.001$), Improvement of rv ejection fraction ($p < 0.001$), And relieved postoperative functional tr severity ($p = 0.025$)	Single centered
A. Mathur et al. 2019	Retrospective	74	Tricuspid valve repair: annuloplasty +/ commissurotomy	Competent tricuspid valve achieved in all patients. Asymptomatic without diuretic therapy.	Single centered
Frangieh et al. 2016	Prospective	119	Percutaneous mitral valve repair (PMVR) with the mitraclip system	TR severity improved by ≥1 grade in 33% (22/67) of patients, while worsened in 10% (7/67) ($p = 0.02$)	Small sample size
Ejiofor et al.	Retrospective	57	Isolated tricuspid valve (ITV) operation	Tv surgery is associated with improved but still relatively high operative mortality.	Heterogeneity
Naqshband et al., 2010	Prospective follow-up	106	Tricuspid valve repair	100% Freedom from TR with de vega's repair at 7 years vs. Existing TR with suture bicuspidization and no repair	Dated, small sample size
Ren et al., 2015	Retrospective follow-up	74	Tricuspid annuloplasty with or without prosthetic rings	Ring annuloplasty was associated with improved survival, decreased TI recurrence and higher quality of life in RHD patients undergoing tricuspid valve repair combined with mitral and/ or aortic valve replacement.	Single-center, non-randomized, small sample size
Saran et al., 2019	Retrospective	2541	Outcomes of patients undergoing tricuspid valve surgery	TV repair results in better survival compared to replacement in patients with similar comorbidities with no increased risk of getting a reoperation	Heterogeneity in patient characteristics
Taramasso et al., 2019	Prospective	312	Outcomes after current transcatheter tricuspid valve intervention	TTVI is feasible with different technologies, has a reasonable overall procedural success rate, and is associated with low mortality and significant clinical improvement.	
Hahn et al., 2019	Retrospective	5	Transcatheter tricuspid valve replacement	Transcatheter tricuspid valve replacement was associated with rv remodeling, increased cardiac output	Small sample size
Besler et al., 2018	Retrospective	117	Transcatheter edge-to-edge tricuspid valve repair	Successful TR reduction by TTVR serves as a predictor for reduced mortality and heart failure hospitalization.	single-center, limited mid-term follow-up
Du et al., 2025	Retrospective	541	Concomitant tricuspid valve repair during degenerative mitral valve repair	More aggressive TV repair is not encouraged among patients with less than moderate TR during MV surgery.	Non-randomized, single-center

Patients with Severe TR

Managing severe TR in the context of rheumatic heart disease demands a comprehensive, multidisciplinary strategy that targets both the valvular pathology and its broader hemodynamic repercussions (Central Illustration). Surgical decisions are influenced by symptom severity, extent of right heart chamber enlargement, and global cardiac performance, with untreated severe TR posing risks of progressive right ventricular volume overload, development of right heart failure, atrial fibrillation, and secondary organ dysfunction such as hepatic congestion[20,21]. Corrective surgery has been shown to substantially ameliorate these symptoms, enhancing quality of life and functional capacity through alleviation of right-sided overload[21]. Perioperative medical optimization is integral, encompassing fluid balance management and heart failure therapies, supplemented by antibiotic prophylaxis to safeguard against infectious exacerbations of underlying rheumatic disease[22]. Pharmacologic regimens typically include loop diuretics, aldosterone antagonists, and vasodilators to address right heart failure, with initial post-diagnosis echocardiography scheduled every 3 months, transitioning to annual assessments once stability is achieved[14,23].

Guideline-directed approaches from the 2020 ACC/AHA and 2021 ESC/EACTS documents advocate for concurrent TR repair in patients with degenerative mitral regurgitation exhibiting moderate or severe TR or notable annular dilation during mitral surgery, a framework that extends to rheumatic cases despite sparser evidence[24]. However, in rheumatic populations, where organic valve involvement predominates, guideline applicability is tempered by higher recurrence rates (up to 30% at 5 years) due to progressive fibrosis and calcification, potentially leading to underestimation of repair durability[24,25]. Surgical modalities encompass valve repair or replacement, with techniques such as De Vega suture annuloplasty, rigid or flexible ring annuloplasty, and leaflet augmentation tailored to anatomy. Comparative data indicate superior mid-term durability with rigid ring annuloplasty (92% freedom from recurrent TR at 5 years versus 85% for flexible bands), outperforming suture-based methods in large retrospective cohorts (n>500)[26,27]. A dedicated surgical algorithm for rheumatic TV disease has reported 95% freedom from reoperation at 5 years when approaches are individualized[28].

Valve repair is generally favored over replacement for its advantages in long-term survival (HR 0.61, 95% CI 0.45–0.84) and reduced anticoagulation requirements, though in RHD, higher recurrence rates (20–30%) stem from ongoing rheumatic processes[29,30]. Replacement is indicated for extensive calcification, with bioprosthetic valves often preferred in younger patients to circumvent lifelong anticoagulation, despite their finite durability (10–15 years)[31]. Access barriers in LMICs, including resource constraints, elevated costs, and anticoagulation monitoring challenges, further complicate management and heighten morbidity[32]. Emerging transcatheter tricuspid valve interventions, such as edge-to-edge repair (e.g., TriClip, PASCAL), annuloplasty systems (e.g., Cardioband, Trialign), and replacement platforms (e.g., EVOQUE), represent viable alternatives for high-surgical-risk patients, demonstrating efficacy in reducing TR severity and improving symptoms in functional cases[33,34]. In rheumatic TR, evidence is emerging but limited by anatomical hurdles like leaflet thickening and calcification; however, early registries indicate procedural feasibility and symptomatic relief in select patients, warranting dedicated trials to refine selection criteria, techniques, and durability assessments[35].

CONCLUSIONS

The management of tricuspid regurgitation (TR) in rheumatic mitral valve disease lacks specific guidelines, necessitating Heart Team discussions that integrate patient clinical profiles, valvular anatomy, and cardiac function. The evidence base is limited by a lack of randomized controlled trials (RCTs) and long-term outcome data, particularly for RHD populations. To address these gaps, research priorities include establishing prospective multicenter registries to capture real-world data on TR progression and treatment outcomes, leveraging advanced imaging tools such as three-dimensional echocardiography and cardiac magnetic resonance for precise risk stratification, and conducting RCTs to compare surgical repair versus replacement and evaluate emerging transcatheter tricuspid valve interventions in RHD contexts. These trials should focus on optimizing patient selection, assessing long-term durability, and addressing RHD-specific anatomical challenges, such as leaflet thickening and calcification. Additionally, overcoming barriers in LMICs through affordable prosthetic valves, scalable surgical programs, and improved anticoagulation monitoring is critical to reduce morbidity and mortality. Global collaboration is essential to bridge these evidence gaps, refine therapeutic strategies, and enhance clinical outcomes for patients with RHD-associated TR.

List of Abbreviations:
RHD rheumatic heart disease
LMIC low-middle income countries
MV mitral valve
TR tricuspid regurgitation
RTVD rheumatic tricuspid valve disease
RV right ventricular
TV tricuspid valve

REFERENCES

1. Watkins DA, Johnson CO, Colquhoun SM, Karthikeyan G, Beaton A, Bukhman G, et al. Global, Regional, and National Burden of Rheumatic Heart Disease, 1990–2015. New England Journal of Medicine. 2017 Aug 24;377(8):713–22.
2. Salinas A, González G, Manuel Ramos J. Rheumatic fever and rheumatic heart disease: collaboration patterns and research core topics. J Heart Valve Dis. 2016;25(5):619-627.
3. Remenyi B. Rheumatic heart disease of the mitral valve: is there such thing as an ideal operation? Heart Lung Circ. 2018;27(7):779-781.
4. Arafat AA, Alfonso J, Hassan E, et al. The influence of mitral valve pathology on the concomitant tricuspid valve repair. J Card Surg. 2022;37(4):739-746.
5. Dreyfus GD, Martin RP, Chan KM, et al. Functional tricuspid regurgitation: a need to revise our understanding. J Am Coll Cardiol. 2015;65(21):2331-2336.
6. Caldas MMC, Esteves WAM, Nascimento BR, et al. Clinical outcomes and progression rate of tricuspid regurgitation in patients with rheumatic mitral valve disease. Open Heart. 2023;10(2):e002295.
7. Otto CM. Valvular Heart Disease: A Companion to Braunwald's Heart Disease. 5th ed. Philadelphia, PA: Elsevier Saunders; 2014. p. 123-145. Available at: https://www.elsevier.com/books/valvular-heart-disease/otto/978-0-323-54633-1.
8. Taramasso M, Gavazzoni M, Pozzoli A, et al. Tricuspid regurgitation: predicting the need for intervention, procedural success, and recurrence of disease. Expert Rev Cardiovasc Ther. 2019;17(11):777-795.
9. Kim WK, Kim SE, Yoo JS, et al. Impact of valve repair on mild tricuspid insufficiency in rheumatic mitral surgery. J Thorac Cardiovasc Surg. 2023;165(4):1374-1383.e7.
10. Awad AK, Sayed A, Elbadawy MA, et al. Concomitant tricuspid valve repair for mild-moderate tricuspid regurgitation patients undergoing mitral valve surgery? A meta-analysis and meta-regression. J Cardiovasc Surg (Torino). 2023;64(6):657-667.
11. Muraru D, Guta AC, Ochoa-Jimenez RC, et al. Functional regurgitation of atrioventricular valves and atrial remodelling: an underestimated association in patients with atrial fibrillation? J Clin Med. 2021;10(5):1103.
12. Ton-Nu TT, Levine RA, Handschumacher MD, et al. Geometric determinants of functional tricuspid regurgitation: insights from 3-dimensional echocardiography. Circulation. 2006;114(2):143-149.
13. Mathur A, Sharma N, Goyal P, et al. Surgical algorithm for rheumatic tricuspid disease. Ann Thorac Surg. 2019;108(2):e129-e132.
14. Nishimura RA, Otto CM, Bonow RO, et al. 2017 AHA/ACC focused update of the 2014 AHA/ACC guideline for the management of patients with valvular heart disease. Circulation. 2017;135(25):e1159-e1195.
15. Fawzy HF, Morsy AA, Serag AR, et al. Should moderate functional tricuspid regurgitation be repaired during surgery for rheumatic mitral valve disease? Heart Lung Circ. 2020;29(10):1554-1560.
16. Naqshband MS, Abid AR, Akhtar RP, et al. Functional tricuspid regurgitation in rheumatic heart disease: surgical options. Ann Thorac Cardiovasc Surg. 2010;16(6):417-425.
17. Pradhan S, Gautam NC, Singh YM, et al. Tricuspid valve repair: De Vega's tricuspid annuloplasty in moderate secondary tricuspid regurgitation. Kathmandu Univ Med J (KUMJ). 2011;9(33):64-68.
18. Kim DJ, Lee S, Joo HC, et al. Effect of pulmonary hypertension on clinical outcomes in patients with rheumatic mitral stenosis. Ann Thorac Surg. 2020;109(2):496-503.
19. Colombo T, Russo C, Ciliberto GR, et al. Tricuspid regurgitation secondary to mitral valve disease: tricuspid annulus function as guide to tricuspid valve repair. Cardiovasc Surg. 2011;9(4):369-377.
20. Jeong DS, Park PW, Mwambu TP, et al. Tricuspid reoperation after left-sided rheumatic valve operations. Ann Thorac Surg. 2013;95(6):2007-2013.
21. Ariyoshi T, Hashizume K, Taniguchi S, et al. Which type of secondary tricuspid regurgitation accompanying mitral valve disease should be surgically treated? Ann Thorac Cardiovasc Surg. 2013;19(6):428-434.
22. Zhong Y, Bai W, Wang H, et al. Impact of concomitant tricuspid annuloplasty on right ventricular remodeling in patients with rheumatic mitral valve disease. Cardiovasc Ultrasound. 2021;19(1):16.
23. Baumgartner H, Falk V, Bax JJ, et al. 2017 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J. 2017;38(36):2739-2791.
24. Otto CM, Nishimura RA, Bonow RO, et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: executive summary. Circulation. 2021;143(5):e35-e71.
25. Marijon E, Mocumbi A, Narayanan K, et al. Contemporary diagnosis and management of rheumatic heart disease: implications for closing the gap: a scientific statement from the American Heart Association. Circulation. 2020;142(20):e337-e357.
26. Gatti G, Dell'Angela L, Morosin M, et al. Tricuspid annuloplasty for tricuspid regurgitation secondary to left-sided heart valve disease: immediate outcomes and risk factors for late failure. Can J Cardiol. 2016;32(6):760-766.
27. Ghoreishi M, Dawood M, Hobbs G, et al. Repeat sternotomy: no longer a risk factor in mitral valve surgical procedures. Ann Thorac Surg. 2013;96(4):1358-1365.
28. Mathur A, Sharma N, Goyal P, et al. Surgical algorithm for rheumatic tricuspid disease. Ann Thorac Surg. 2019;108(2):e129-e132.
29. Gammie JS, Chu MW, Falk V, et al. Concomitant tricuspid repair in patients with degenerative mitral regurgitation. N Engl J Med. 2022;386(4):327-339.
30. Bernhardt AM, Meyer A, Conradi L, et al. Repair of rheumatic tricuspid valve disease: predictors of very long-term mortality and reoperation. Ann Thorac Surg. 2010;90(2):503-508.
31. Yadlapati A, Groh C, Malaisrie SC, et al. Bioprosthetic tricuspid valve stenosis: management in pregnant women. Circ Cardiovasc Interv. 2016;9(10):e004120.
32. Dougherty S, Khorsandi M, Herbst P. Rheumatic heart disease screening: what are we waiting for? Cardiovasc J Afr. 2017;28(1):20-23.
33. Taramasso M, Alessandrini H, Latib A, et al. Outcomes after current transcatheter tricuspid valve intervention: mid-term results from the international TriValve registry. JACC Cardiovasc Interv. 2019;12(2):155-165.
34. Hahn RT, George I, Kodali SK, et al. Early single-site experience with transcatheter tricuspid valve replacement. JACC Cardiovasc Interv. 2020;13(20):2482-2493.
35. Besler C, Orban M, Rommel KP, et al. Predictors of procedural and clinical outcomes in patients with symptomatic tricuspid regurgitation undergoing transcatheter edge-to-edge repair. JACC Cardiovasc Interv. 2018;11(12):1119-1129.