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Tricuspid Regurgitation in Tuberculous Constrictive Pericarditis Underwent Pericardiectomy

ABSTRACT

Background: The presence of constrictive pericarditis (CP) in conjunction with tricuspid regurgitation (TR) and the worsening of TR following pericardiectomy are associated with a reduction in patient survival. The purpose of this study was to investigate the prevalence of tuberculous CP in conjunction with TR, the incidence of worsening regurgitation following pericardiectomy, and the analysis of associated factors.

Methods: Seventy-five consecutive patients who underwent pericardiectomy for tuberculous CP at the institution between January 2021 and December 2023 were retrospectively analyzed. Their clinical, imaging, and hemodynamic characteristics were analyzed.

Results: Among the 75 patients with tuberculous CP, 29 patients (38.7%) had mild or greater TR pre-operatively and 27 patients (36%) had worsening TR after pericardiectomy. In patients with worsening TR, the pericardial thickness of the right ventricular (RV) lateral wall was significantly thickened preoperatively, and there was a reduction in the tricuspid annular plane systolic excursion (TAPSE), right ventricle S′ tissue Doppler velocity (S′), and right ventricle fractional area change (FAC) postoperatively. The preoperative inferior vena cava diameter and the postoperative right atrial and RV basal diameters were significantly larger in patients with worsening TR compared with patients with non-worsening TR, whereas the TAPSE, S′, and FAC were significantly lower before and after the surgery ($P < .05$). The FAC [OR = 0.354; 95% CI (0.165-0.761), $P = .008$] and pericardial thickness of the RV lateral wall [OR = 1.887; 95% CI (1.206-2.953), $P = .005$] were independently associated with worsening TR.

Conclusion: Patients with tuberculous CP often have coexisting TR, and pericardiectomy can lead to worsening TR. The pericardial thickness of the RV lateral wall and FAC are independently associated with worsening TR following pericardiectomy.

Keywords: Constrictive pericarditis, pericardiectomy, tricuspid regurgitation, tuberculous

INTRODUCTION

Constrictive pericarditis (CP) results in impaired ventricular diastolic filling due to thickening and fibrosis of the pericardium, which ultimately leads to right heart failure. Tuberculosis infection is the main cause of CP in developing countries, accounting for approximately 38%-83% of all CP, and currently, pericardiectomy is the only effective treatment for CP.[1,2] The the coexistence of CP and tricuspid regurgitation (TR) renders the diagnosis of the disease more challenging and increases the risk of postoperative mortality in affected patients. In particular, those exhibiting moderate to severe TR face a mortality rate that is more than 2-fold higher than that observed in individuals without or with trace regurgitation. Additionally, pericardiectomy causes worsening of TR, leading to a recurrence of symptoms and a reduction in long-term survival.[3-5] Therefore, the assessment of TR before and after surgical intervention is of great clinical value. Currently, there are no studies to assess the incidence and factors affecting tuberculous CP coexisting with TR and the worsening of TR after surgery. In this study, the incidence of tuberculous CP coexisting with TR, the incidence of worsening TR after pericardiectomy, and analyzed the related factors were retrospectively analyzed, with the

ORIGINAL INVESTIGATION

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aim of providing some help for further clinical diagnosis and treatment options.

METHODS

Study Population

The study was a retrospective single-center observational study and enrolled a total of 86 patients with tuberculous CP who underwent successful pericardiectomy from January 2021 to December 2023 in our hospital. All patients had a history of anti-tuberculosis treatment before surgery or pathologically confirmed tuberculous pericarditis postoperatively, with pre- and postoperative echocardiography available. Eleven patients were excluded from analysis as they underwent other concomitant cardiac operations at the time of pericardiectomy, of whom 5 underwent coronary artery bypass grafting, 3 underwent valvuloplasty, 1 underwent valve replacement, and 2 underwent a secondary pericardiectomy. Seventy-five patients were finally included. Clinical data including demographic information, past history, and laboratory tests were collected. The study was conducted in accordance with the Declaration of Helsinki and was approved by our institutional ethics committee. Because this was a retrospective study, written informed consent for this study was waived.

Surgical Technique

Pericardiectomy was performed via median sternotomy in all cases, without cardiopulmonary bypass. The primary surgical goal was total pericardiectomy, which was defined as the excision of the anterior pericardium up to the phrenic nerves and the diaphragmatic surface. The visceral and parietal pericardium were removed whenever technically possible. Any excision less than total was defined as partial.

Assessment of Cardiac Imaging

Preoperative computed tomography (CT) was performed to measure the pericardial thickness of the right ventricular (RV) lateral wall, as well as to observe whether the thickened pericardium was combined with calcification or pericardial effusion. Pericardial thickness >3 mm was considered to be pericardial thickening.[6]

All patients underwent transthoracic echocardiography 1-3 days preoperatively and 7-10 days postoperatively, using the Philips EPIQ 7C (Philips Medical Systems, Andover) or ACUSON SC2000 (Siemens Medical Solutions USA, Mountain View, CA) equipment. Valve regurgitation grading was based on multiparametric criteria of echocardiography guidelines, taking into account vena contracta width (VCW) and color flow jet area (quantified), along with visual assessment of tricuspid valve morphology, color, and continuous wave Doppler properties.[7] Tricuspid regurgitation was classified as no regurgitation, trace regurgitation (regurgitant bundle confined to the orifice and VCW <2 mm), mild regurgitation (2 mm ≤ VCW <3 mm and jet area <5 cm^2), moderate regurgitation (3 ≤ VCW <7 mm and 5 ≤ jet area <10 cm^2), and severe regurgitation (VCW ≥7 mm and jet area ≥10 cm^2), where no regurgitation and trace regurgitation were considered to be at the same grade. Worsening TR was defined as a postoperative increase in regurgitation of at least one grade compared with preoperative TR. Routine parameters of cardiac structure and function were recorded, of which indicators of RV systolic function included right ventricle fractional area change (FAC), the tricuspid annular plane systolic excursion (TAPSE), and right ventricle S′ tissue Doppler velocity (S′).

Statistical Analysis

Statistical analysis was performed using SPSS Statistics version 26.0 (SPSS, Inc., Chicago, IL, USA). The Shapiro–Wilk test was used to test for normality. Normally distributed data are presented as mean ± SD, and non-normally distributed data are presented as median [M (Q25, Q75)]. Continuous numeric data were compared using the independent samples *t*-test and paired *t*-test for normally distributed data, or Mann–Whitney U test and Wilcoxon test for non-normally distributed data. Categorical variables are expressed as frequency (percentage) and compared using the X^2 test or Fisher's exact test. Multifactorial analysis was conducted by binary logistic regression analysis.

RESULTS

Evolution of Tricuspid Regurgitation

Seventy-five patients who underwent pericardiectomy for tuberculous CP in the study were retrospectively analyzed. In the majority of patients (61.3%), preoperative TR was none or trace, with only 6.7% exhibiting moderate regurgitation. The incidence of postoperative moderate/severe TR was found to be significantly higher in 18.7% of patients, as shown in Table 1. In this study, 27 patients (36%) exhibited worsening TR, while 48 patients (64%) demonstrated non-worsening TR.

HIGHLIGHTS

- This research reveals that tuberculous constrictive pericarditis coexisted with mild or greater tricuspid regurgitation (TR), accounting for 38.7% preoperatively.
- Pericardiectomy can lead to worsening TR, with an incidence of 36%.
- The research highlights the independent factors associated with the worsening of TR following surgery, including the right ventricle fractional area change and the pericardial thickness of the right ventricular lateral wall.

Table 1. Cohort Tricuspid Regurgitation Characteristics—Frequency (Percentage)

	Pre-Operative n = 75	Post-Operative n = 75	*P*
TR overall			.035
No/Trace	46 (61.3%) #	33 (44%) ▲	
Mild	24 (32%) #	28 (37.3%) #	
Moderate/Severe	5 (6.7%) #	14 (18.7%) ▲	

Values are presented as n (%).
Different symbols (#, ▲) indicate differences between groups.
TR, tricuspid regurgitation.

Comparison of General Clinical Data

Patients with worsening TR had higher C-reactive protein (CRP) and longer duration of the disease compared to patients with non-worsening TR ($P < .05$), while their differences in demographics, past history, and laboratory tests were not statistically significant ($P > .05$), as shown in Table 2.

Comparison of Computed Tomography Parameters

Patients with worsening TR had thicker pericardial thickness in the RV lateral wall and more patients with pericardial thickening and calcification compared to those without worsening TR ($P < .05$), as shown in Table 3.

Comparison of Echocardiographic Parameters

In patients with worsening TR, RV basal diameter, VCW, and colored flow jet area of TR were significantly increased, whereas TAPSE, S′, and FAC were decreased after pericardiectomy ($P < .001$). In patients without worsening TR, the postoperative left and right atrial diameters and VCW were reduced ($P < .05$), whereas the pre- and postoperative TAPSE, S′, and FAC differences were not significant ($P > .05$).

Preoperative inferior vena cava diameters were increased in patients with worsening TR compared with those without worsening TR. In addition, right atrial and RV basal diameters were significantly increased postoperatively. In contrast, S′, TAPSE, and FAC decreased significantly both before and after surgery ($P < .05$). Postoperative mitral valve E-wave velocity, lateral mitral annular early diastolic velocity e′, and E-wave/mean e′ were significantly increased in worsened and non-worsened patients compared with the preoperative period ($P < .001$), as shown in Table 4. Figure 1 shows an example of worsened TR after pericardiectomy.

Multiple logistic regression analysis showed that FAC [OR = 0.354; 95% CI (0.165-0.761), $P = .008$] and RV lateral wall pericardial thickness [OR = 1.887; 95% CI (1.206-2.953), $P = .005$] were independently associated with worsening TR (Table 5).

DISCUSSION

The results of the present study show the following:① the coexistence of mild or greater TR and tuberculous CP is present in 38.7% of patients preoperatively. ② Pericardiectomy can lead to worsening TR in 36% of patients.③ Patients with worsening TR have enlarged right ventricles and reduced RV function postoperatively. ④ The pericardial thickness of the RV lateral wall and FAC are independently associated with worsening TR following pericardiectomy.

Table 2. Baseline Patient Characteristics

	Worsened TR	Non-Worsened TR	*P*
Number of patients	27	48	
Demographics			
Age (year)	52 (24, 63)	51 (31, 58)	.675
Male (%)	19 (70.4%)	33 (68.8%)	.884
BMI (kg/m^2)	22.43 ± 3.31	23.14 ± 3.36	.383
Duration of disease (month)	6 (3, 12)	3 (2, 7)	.033
NYHA			.923
I	1 (3.7%)	3 (6.3%)	
II	5 (18.5%)	11 (22.9%)	
III	18 (66.7%)	29 (60.4%)	
IV	3 (11.1%)	5 (10.4%)	
Past history			
Hypertension	2 (7.4%)	5 (10.4%)	.667
Diabetes	4 (14.8%)	4 (8.3%%)	.383
Atrial fibrillation	3 (11.1%)	1 (2.1%)	.095
Laboratory tests			
WBC (10^9/L)	5.53 (4.86, 7.72)	5.04 (3.98, 6.30)	.113
CRP (mg/L)	19.17 (9.72, 28)	6.34 (2.01, 23.50)	.018
ALT (U/L)	14.80 (9.50, 22.90)	16.50 (12.60, 28.85)	.122
AST (U/L)	23.10 (18.0, 32.5)	26.55 (18.85, 33.95)	.497
GGT (U/L)	70.80 (38.80, 150.3)	82.60 (49.25, 122.0)	.707
TBIL (mol/L)	20.22 (12.88, 27.30)	14.17 (8.86, 21.61)	.059
DBIL (mol/L)	7.62 (4.10, 11.61)	4.34 (2.42, 8.53)	.069
IDBL (mol/L)	11.75 (7.30, 15.92)	8.75 (6.58, 13.31)	.099

Values are represented as mean (SD) or n (%) or median [M (Q25, Q75)].
ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; CRP, c-reactive protein; DBIL, direct bilirubin; GGT, gamma-glutamyl transpeptidase; IDBL, indirect bilirubin; NYHA, New York Heart Association; TBIL, total bilirubin; WBC, white blood cell count.

Tricuspid regurgitation can result from primary, secondary, or multiple causes and is strongly associated with high morbidity and mortality and was previously an underestimated clinical problem.[3] Tricuspid regurgitation is a common and important comorbidity of CP, and studies have shown that moderate/severe TR is an independent predictor of late postoperative mortality in CP (OR = 2.9, 95% CI: 1.5-5.6).[8] However, previous studies on the prevalence of TR coexisted with CP were scarce

Table 3. CT Imaging Characteristics

	Worsened TR	Non-Worsened TR	*P*
Right ventricular lateral wall pericardial thickness (mm)	10.96 (9.51, 14.27)	6.71 (4.62, 7.93)	<.001
Thickened pericardium combined with calcification/effusion			.001
Simple thickening	13 (48.1%) #	33 (68.8%) #	
Thickening combined with calcification	9 (33.3%) #	1 (2.1%) ▲	
Thickening combined with effusion	5 (18.5%) #	14 (29.2%) #	

Values are represented as n (%) or median [M (Q25, Q75)].
Different symbols (#, ▲) indicate differences between groups.

Table 4. Echocardiographic Parameters

	Worsened TR		Non-worsened TR					
	Preoperative	Postoperative	Preoperative	Postoperative	*P1*	*P2*	*P3*	*P4*
LAD, mm	40.03 ± 7.00	38.11 ± 6.21	39.00 ± 3.99	36.68 ± 6.71	.416	.369	.082	**.003**
LVEDD, mm	40.55 ± 3.99	41.85 ± 2.69	42.29 ± 3.79	42.37 ± 3.69	.066	.86	.075	.887
LVESD, mm	27.77 ± 3.17	28.48 ± 3.11	28.54 ± 2.90	28.62 ± 2.68	.295	.835	.187	.827
LVEF, %	57.66 ± 6.00	59.14 ± 3.71	58.75 ± 6.28	59.75 ± 8.39	.469	.725	.162	.49
RAD, mm	38.77 ± 6.25	40.25 ± 5.57	40.56 ± 3.43	37.72 ± 5.02	.114	.048	.287	<.001
RVBD, mm	32 (28, 35)	37 (35, 39)	34 (30, 36)	35 (33, 36)	.052	.001	<.001	.009
IVCD, mm	24 (22, 26)		22 (19, 24)		.002			
E, cm/s	65 (54, 81)	82 (64, 91)	73 (57.75, 86.75)	80.5 (66.25, 95.70)	.229	.934	.005	.004
A, cm/s	56.96 ± 19.70	61.86 ± 17.69	63.47 ± 18.35	65.24 ± 22.88	.381	.544	.765	.413
Medial e', cm/s	11 (7, 16)	9 (6, 12)	10 (8, 14)	9 (6, 10)	.63	.333	.024	<.001
Lateral e', cm/s	9 (8, 10)	10 (9, 12)	9 (8, 10)	10 (8, 11)	.669	.093	.001	.007
E/medial e'	5 (4.2, 9.16)	8.2 (6.61, 11.83)	6.97 (5.52, 9.75)	9.11 (7.31, 11.16)	.142	.377	.001	.001
E/lateral e'	7.13 (5.44, 10)	7.63 (5.92, 9.77)	8.37 (6.50, 10.16)	9.30 (7.16, 9.56)	.223	.096	.381	.234
E/average e'	6.09 (5.48, 10.08)	7.92 (6.45, 9.80)	7.87 (6.16, 9.33)	9.06 (7.60, 10.22)	.125	.074	.006	.003
S', cm/s	9 (8, 10)	8 (6, 9)	11 (10, 12)	11 (10, 11)	<.001	<.001	.001	.277
TAPSE, mm	13 (12, 14)	11 (10, 13)	14 (13, 15)	13 (12, 15)	<.001	<.001	.003	.051
RVFAC, %	37 (35, 38)	35 (33, 37)	40 (38, 43)	40 (38, 43)	<.001	<.001	.016	.092

Values are represented as mean (SD) or n (%) or median [M (Q25, Q75)].
P1 represents the comparison of preoperative parameters between the worsened TR group and non-worsened TR group;
P2 represents the comparison of postoperative parameters between the worsened TR group and non-worsened TR group;
P3 represents the comparison of pre-and postoperative parameters in the worsened TR group;
P4 represents the comparison of pre-and postoperative parameters in the non-worsened TR group;
A, mitral valve A-wave velocity; E, mitral valve E-wave velocity; IVCD, inferior vena cava diameter; LAD, left atrial diameter; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; Lateral e', lateral mitral annular early diastolic velocity e'; Medial e', medial mitral annular early diastolic velocity e'; RAD, right atrial diameter; RVBD, right ventricular basal diameter; RVFAC, right ventricle fractional area change; S', right ventricle S' tissue Doppler velocity; TAPSE, tricuspid annular plane systolic excursion.

and highly variable. In this study, the prevalence of preoperative TR coexisted with tuberculous CP was shown to be 38.7% (of which 32% were mild and only 6.7% were moderate), and 3 patients who underwent valve repair for severe regurgitation were excluded from this study. This is similar to the results of Calderon–Rojas, which included 518 patients and showed mild TR in 37% (191 patients) and moderate/severe in 10% (51 patients), but the study included mainly cardiac surgeries related to CP and did not include tuberculous CP.[4] A study by Góngora et al[8] on combined TR in 261 patients who underwent pericardiectomy showed that moderate/severe TR accounted for 20% of the cases (54 patients), but there were 9 pacemaker implantations (3%) and 15 cases of radiation pericarditis (6%) among these 54 patients. The prevalence of coexisting TR in CP of different etiologies varies, which may be strongly related to the comorbid underlying disease of the study population, such as a history of pacemaker implantation, atrial fibrillation, pulmonary disease, etc. There is noconsensus on the mechanism of coexisting TR in CP, preferring to believe that TR is secondary to the structural and functional remodeling of the patient's right atrium and right ventricle during the pathological processes of CP.[5]

Cardiac surgery can lead to worsening TR, with an incidence of approximately 17%-50%, and is associated with a reduction in long-term patient survival after surgery.[9,10] However,

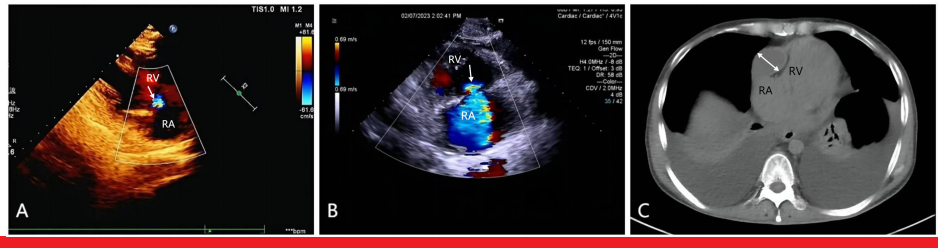

Figure 1. A case of worsened TR after pericardiectomy. Transthoracic echocardiography of the right ventricular inflow tract showed trace TR (A) preoperatively and severe TR (B) postoperatively. CT showed significant thickening of the pericardium on the right ventricular lateral wall and compression of the right ventricular cavity (C) preoperatively (as indicated by the white arrow).

Table 5. Binary Logistic Regression Analysis

Parameters	OR	95% CI	*P*
Pericardial thickness of right ventricular lateral wall	1.887	1.206-2.953	.005
Right ventricle fractional area change	0.354	0.165-0.761	.008
Right ventricle S′ tissue Doppler velocity	0.667	0.287-1.554	.348
Tricuspid annular plane systolic excursion	0.74	0.348-1.572	.433
Inferior vena cava diameter	1.286	0.973-1.699	.077

P values < .05 are statistically signifcant.

worsening of TR due to pericardiectomy is mostly reported in case reports, and scarce clinical studies have been performed.[11] It was found that the incidence of worsening TR after pericardiectomy was 36%, which is different from the 51% reported by Tabucanon et al,[5] who included echocardiographic data within 1 year postoperatively, whereas the study included data from 7 to 10 days after the operation and is a short-term observation.[9] There are 2 main hypotheses regarding the mechanism of worsening TR after pericardiectomy: one suggests that it is due to dilatation of the annulus, and the other suggests that it is due to RV enlargement as a result of surgical relief of pericardial constraint on the ventricle.[11,12] The present study showed that patients with worsening TR had an increased postoperative right ventricle and reduced RV systolic function, which is consistent with the results of previous studies.[5,12] Choudhry et al[13] demonstrated that reduced RV systolic function was an independent predictor of heart failure and death after pericardiectomy in patients with CP. The deterioration of RV systolic function in the process of pericardial constriction can be explained in 3 aspects. Firstly, the subepicardial myocardium undergoes ischemic necrosis, atrophy, and fibrosis due to a reduction in coronary blood flow, which is caused by the inflammatory infiltration of the subepicardial myocardium and the mechanical compression of the subepicardial coronary artery by the diseased pericardium.[14,15] Furthermore, the adhesion of the epicardium to the pericardium increases the difficulty of surgical dissection, which in turn results in additional myocardial damage and a reduction in ventricular systolic function.[16] In addition, the thin RV wall and chronic RV systolic dysfunction may result in an imbalance between the supply and demand of myocardial oxygen following a sudden increase in filling after pericardiectomy. This may lead to acute RV dilation and dysfunction, which could subsequently lead to worsening TR. The 2 patients in this study with a grade 2 or greater increase in postoperative TR were found to have significant preoperative pericardial thickening, whereas postoperative RV dilatation was evident, with markedly reduced systolic function and symptoms of low cardiac output.

This study showed that FAC was an independent factor contributing to postoperative worsening TR, suggesting that the overall contractile function of the RV is more closely related to the worsening of TR. Although the RV myocardium is predominantly subepicardial longitudinal myocardium, which plays a major role in contractile function, the degree of damage to the RV myocardium of the corresponding segments is different due to the different sites and degrees of pericardial thickening in patients with CP. Tricuspid annular plane systolic excursion and S′ can only reflect the longitudinal contractile function in the localized area (proximal to the annulus) rather than the overall contractile function.[17] Therefore, in tuberculous CP, the overall contractile function of the RV should be emphasized before surgery to prevent worsening of TR.

In this study, it was also found that the RV lateral wall pericardium was thicker and more often combined with calcification in patients with worsening TR, and the thickness of the RV lateral wall pericardium was an influencing factor in the worsening of TR. The pericardial thickness was negatively correlated with the longitudinal strain of the RV wall in the study by Kusunose et al.[18] The degree of compression of the cardiac chambers is directly proportional to the thickness of the pericardium. Additionally, the adherence of the pericardium to the RV wall is enhanced in cases of thickening or calcification, resulting in greater local epicardial myocardial damage and increased difficulty in separating it during surgery. This, in turn, contributes to a further reduction in RV systolic function.

This study found that preoperative CRP was higher in patients with worsening TR. It is a sensitive indicator for assessing the state of infection and inflammation and is associated with the prognosis of the disease. Fernandes et al showed an association between high CRP levels and death in patients after pericardiectomy.[19] This detail should not be overlooked when evaluating patients preoperatively.

Study Limitations

It should be noted that this study is subject to a number of limitations. Firstly, this was a single-center surgical experience, which may have introduced a degree of selection bias. Secondly, the tricuspid annulus was not subjected to analysis in order to ascertain the role of annular dilatation in the worsening of TR. Thirdly, the perioperative data collected in this study lack clarity regarding the mid- and long-term follow-up and survival outcomes of patients with worsened TR after tuberculous CP, which represents a gap in the literature that requires further investigation.

CONCLUSION

Tricuspid regurgitation often complicates patients with tuberculous CP, and pericardiectomy can lead to worsening TR. The thickness of the pericardium of the RV lateral wall and FAC are independently associated with worsening TR following pericardiectomy. It is imperative that clinicians monitor TR in patients with tuberculous CP, both pre- and

post-operatively, in order to facilitate the development and adjustment of treatment programs.

It is confirmed that artificial intelligence (AI)–assisted technologies (such as Large Language Models [LLMs], chatbots, or image creators) were not used in the production of the submitted work.

Ethics Committee Approval: The study was approved by the Institutional Ethics Committee (Date: October 20, 2019; number: S-183).

Informed Consent: This was a retrospective study, written informed consent for this study was waived.

Peer-review: Externally and internally peer-reviewed.

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