# Outcomes of surgical tricuspid valve intervention after heart transplant

**Macit Bitargil[1#], Mohammad Alomari[1#], Osama Haddad[2], Si M. Pham[1], Richard C. Daly[3], Kevin P. Landolfo[1], Pankaj Garg[1], Magdy M. El-Sayed Ahmed[1], Parag C. Patel[4], Basar Sareyyupoglu[1]**

[1]Department of Cardiothoracic Surgery, Mayo Clinic, Jacksonville, FL, USA; [2]Department of Cardiothoracic Surgery, University of Miami, Miami, FL, USA; [3]Department of Cardiovascular Surgery, Mayo Clinic, Rochester, MN, USA; [4]Division of Advanced Heart Failure and Transplantation, Department of Transplantation, Mayo Clinic, Jacksonville, FL, USA

[#]These authors contributed equally to this work as co-first authors.

*Correspondence to:* Basar Sareyyupoglu, MD. Department of Cardiothoracic Surgery, Mayo Clinic, 4500 San Pablo Road, Jacksonville, FL, USA. Email: Sareyyupoglu.Basar@mayo.edu.

**Background:** Tricuspid regurgitation (TR) is common following heart transplant (HTx). Data on the outcomes of tricuspid valve (TV) surgery after HTx is limited. This study evaluates the short- and long-term outcomes of TV surgery after HTx and identifies predictors of mortality.

**Methods:** A retrospective analysis of all HTx recipients undergoing TV surgery between 2000–2019 within the Mayo Clinic Health System was performed. Demographic, echocardiographic, hemodynamic, and postoperative outcomes were collected. Survival was estimated using Kaplan-Meier analysis, and univariable Cox regression analysis was used to identify mortality-associated risk factors.

**Results:** Forty-one patients (mean age 53.9±14.4 years) underwent TV surgery after HTx. Biopsy-related leaflet injury (22 patients) and annular dilation (16 patients) were the most common mechanisms of TR. Nine patients underwent TV surgery during the index hospitalization of HTx, while 32 patients were operated on during follow-up. After TV surgery, 5- and 10- year survival was 60.5% and 23.5%, respectively. There were 23 deaths (5 during the index hospitalization for TV surgery, while 18 died during follow-up). Preoperative predictors of mortality were elevated right atrial pressure (RAP), right ventricular end-diastolic pressure (RVEDP), and mean pulmonary artery pressure (mPAP), and international normalized ratio (INR), along with hypoalbuminemia ($P<0.05$). Postoperative predictors were right ventricular (RV) dysfunction, elevated RAP, RVEDP, mPAP, reduced pulmonary artery pulsatility index (PAPi), hyperbilirubinemia, and an elevated INR (all $P<0.05$).

**Conclusions:** TV surgery after HTx is associated with significant early and late mortality. In HTx recipients, TV surgery should be performed before the onset of RV dysfunction and hepatic dysfunction.

**Keywords:** Heart transplant (HTx); tricuspid regurgitation (TR); tricuspid valve surgery (TV surgery); right ventricular dysfunction (RV dysfunction)

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## Introduction

Tricuspid regurgitation (TR) is the most common valvular complication after orthotopic heart transplant (HTx), with a prevalence ranging from 19% to 84% (1,2). TR results in progressive right ventricular (RV) dilation and right heart failure (RHF) culminating in hepatic and renal dysfunction (3). Severe TR after HTx is associated with increased morbidity and mortality (4,5). In a study including 542 HTx recipients, early TR was associated with increased 30-day, 1-year, and 5-year mortality ($P<0.01$) and persistence of moderate to severe TR during follow-up was associated with approximately 50% mortality at 1-year (6).

The incidence of TR early after HTx remains substantial

despite advances in allograft preservation and the bicaval technique of HTx. TR may develop early after transplant or during follow-up and the mechanism can be functional or anatomic. Early TR is usually functional and results from right atrial and RV remodeling and tricuspid annular dilatation or distortion. Causes of functional TR are RV dysfunction due to pulmonary hypertension (PH), primary graft dysfunction (PGD), > Grade 2 allograft rejection, and leaflet interference from permanent pacemaker leads (6). Less common factors are size mismatch with a small donor heart in a large recipient's pericardial cavity, allograft anastomosis technique, and pre-existing TR in the donor heart (4,7-9). The biatrial technique compared to the bicaval technique leaves a large portion of the recipient's left and right atria, which can result in the distortion of the tricuspid annular geometry, increasing the likelihood of TR. Late TR can be functional or anatomic. Functional TR is related to repeated episodes of acute allograft rejection, chronic allograft vasculopathy, and implantation technique. Anatomic TR is caused by flail leaflets due to direct injury to the leaflet or subvalvular apparatus during repeated endomyocardial biopsies (EMBs), impingement of the tricuspid valve (TV) leaflet by an endocardial RV pacemaker lead, and rarely infective endocarditis (4,7,10).

Severe TR after HTx is associated with adverse clinical outcomes, including reduced exercise tolerance, RHF, renal dysfunction, and mortality (11,12). Studies have shown that concomitant prophylactic tricuspid annuloplasty of the donor heart at the time of HTx reduces the incidence of TR without increasing the surgical complexity (5,6,8,13,14). However, due to a significant reduction in the incidence of TR requiring intervention at 1-year post-HTx (6,15,16) and the lack of evidence for short- or long-term benefits of prophylactic tricuspid valve repair (TVr) (17,18); this practice remains contentious. Tricuspid annuloplasty also carries a risk of conduction system injury and the need for a permanent pacemaker.

The majority of patients with TR improve with medical therapy and approximately 6% may require TV intervention (7,19). Surgical treatment of the TV is generally reserved for patients with refractory symptoms despite optimal medical therapy as the isolated TV surgery in non-HTx patients is associated with significant perioperative and long-term mortality (20-22). However, there is a dearth of data related to the outcomes of post-HTx TV surgery. The present study aims to evaluate the outcome of post-HTx TV surgery, focusing on survival, complications, and predictors of mortality.

## Methods

This retrospective study included HTx recipients who underwent TV surgery at three Mayo Clinic hospitals (Rochester, Florida, and Arizona) between January 2000 and December 2019. Institutional review board approval (No. 215714) was obtained and the requirement for patient consent was waived due to the retrospective design of the study. Electronic medical records were reviewed for the demographic characteristics, operative technique, postoperative complications, hemodynamic data, and long-term survival. Preoperative and postoperative transthoracic echocardiography (TTE) and/or transesophageal echocardiography (TEE) were assessed to characterize the mechanism and severity of TR, leaflet and subvalvular morphology, RV size and function, left ventricular function, and other valvular pathology. For preoperative assessment of the heart, including the TV, we relied on TEE due to better image quality. In the postoperative period and during follow-up, we collected TTE parameters as TEE was only performed in a few patients. Tricuspid annular plane systolic excursion (TAPSE) <16 mm and fractional area change (FAC) <35% were considered indicators of RV dysfunction. For RV enlargement, the criteria used were RV basal diameter >4.2 cm and a right ventricle-to-left ventricle area ratio >0.6 (23). RV dysfunction was considered to be persistent if the signs of RV dysfunction were present beyond 4 to 6 months (24). TV surgery was offered according to guideline-driven indications for refractory symptomatic TR [New York Heart Association (NYHA) III/IV], progressive RV dilation/dysfunction, or hemodynamic compromise despite medical optimization. For postoperative hemodynamic parameters, we collected Swan-Ganz data at 72 hours after surgery. We collected the biochemical parameters at the end of one week or at the time of death, whichever was earlier, since it takes longer for biochemical parameters to normalize after cardiopulmonary bypass (CPB) (25).

Based on the timing of surgery, patients were divided into the early TV surgery group (surgery during index HTx hospitalization or within 6 months of HTx) and the Late TV surgery group (>6 months after HTx). The primary outcome was mortality. Secondary outcomes were postoperative change in right-heart hemodynamics and RV function, complications, and freedom from reintervention during follow-up.

### Statistical analysis

Continuous variables were reported as median and

 

interquartile range (IQR) or mean ± standard deviation, and categorical variables as frequency (percentage). Pre- and post-intervention parameters were compared using paired signed-rank or McNemar's tests. Survival was estimated using the Kaplan-Meier method. Univariable Cox regression was used to identify predictors of mortality, with P<0.05 considered statistically significant.

## Results

During the study period, 1,347 patients underwent HTx in three Mayo Clinic centers. A total of 264 (19.6%) developed moderate-to-severe TR; 223 patients remained asymptomatic on medical management, while 41 patients (3% of all HTx recipients; 15.5% of the TR cohort) required TV surgery for symptomatic TR despite optimal medical treatment, including diuretics. Nine (22%) patients underwent TV surgery early after HTx (early TV surgery group), and 32 (78%) patients required TV surgery during follow-up (late TV surgery group).

The mean age of the patients was 53.9±14.4 years, and 68.3% (n=28) were male. Thirty-seven (90.2%) patients were NYHA Class III/IV despite being on maximum tolerated oral or intravenous diuretics. The mechanism of TR was biopsy-related leaflet injury in 53.7% (n=22) patients, annular dilation in 39% (n=16) patients, pacemaker lead impingement in 4.9% (n=2) patients, and inflammatory etiology in 2.4% (n=1) patients. The cardiac implantation technique used was biatrial in 63.4% (n=26) patients and bicaval in 36.6% (n=15) patients. TR was severe in 85.4% (n=35) patients, and 39% (n=16) patients had moderate-to-severe RV dysfunction. Right heart catheterization showed elevated right heart pressures [right atrial pressure (RAP) 14.2±5.2 mmHg, right ventricular end-diastolic pressure (RVEDP) 13.1±5.5 mmHg and mean pulmonary artery pressure (mPAP) 23.2±6.1 mmHg], low pulmonary artery pulsatility index (PAPi) 1.4±0.9 and increased pulmonary vascular resistance (PVR) 2.7±1.4 Wood units. Total bilirubin was elevated (5.3±4.7 mg/dL) while albumin was preserved (3.9±0.45 g/dL). The mean interval from HTx to TV surgery was 7.1±5.5 years. Preoperative temporary mechanical circulatory support (TMCS) was required in 7.3% (three patients). All 9 patients (22%) in the Early TR group had developed PGD after HTx and had severe symptomatic TR despite optimal medical management and use of TMCS in three patients. Among the three patients on TMCS, two patients improved after TV surgery. The third patient had pre-existing severe PH and continued to have severe RV dysfunction despite tricuspid valve replacement (TVR); this patient subsequently developed thrombus over the TV bioprosthesis and died of sepsis.

For TV surgery, redo sternotomy was performed in 39 patients (95.1%), and the heart was arrested in 20 patients (48.8%). TVr was performed in 15 patients (36.6%), while 26 patients (63.4%) underwent TVR (when repair was deemed nondurable or challenging) (*Table 1*).

Postoperative complications occurred in 16 patients (39%). Acute kidney injury (AKI) requiring dialysis was the most common complication, developing in 14 patients (35%), and six patients (14.6%) were discharged on dialysis. Permanent pacemaker implantation was required in three patients (7.3%), while deep sternal wound infection and stroke occurred in one patient each (2.5%). After TV surgery, three patients (7.3%) required TMCS for PGD, and three patients (7.3%) required reintervention (including two redo-HTx). Moderate to severe TR persisted in three of 41 patients (7.3%), while the incidence of moderate to severe RV dysfunction reduced to 8 in 16 patients (50%). Freedom from reintervention at 5-years was 22 out of 25 surviving patients (88%). Right heart filling pressures and bilirubin levels improved significantly after TV surgery (all P<0.05, *Tables 2*,*3*).

There were 23 deaths. Five deaths were in-hospital (one in the early TR group, four in the late TR group), and 18 occurred during follow-up (one in the early TR group, 17 in the late TR group) (*Table 4*). Mean survival after TV surgery was 6.9±1 years, with actuarial survival of 60.5% (25 patients) at 5 years, and 23.5% (10 patients) at 10 years. Arrested heart TV surgery was associated with improved survival [median 10 *vs.* 4.4 years; P=0.006; hazard ratio (HR): 0.32, 95% confidence interval (CI): 0.13–0.78]. Technique of HTx (biatrial *vs.* bicaval) and type of TV surgery (TVR *vs.* TVr) (*Figures 1*,*2*) had no significant impact on survival.

Preoperative predictors of mortality were elevated RAP (HR: 1.12; 95% CI: 1.01–1.24; P=0.025), elevated RVEDP (HR: 1.12; 95% CI: 1.01–1.24; P=0.031), elevated mPAP (HR: 1.08; 95% CI: 1.00–1.16; P=0.044), low serum albumin (HR: 0.20; 95% CI: 0.05–0.80; P=0.024), and elevated international normalized ratio (INR) (HR: 2.09; 95% CI: 1.53–2.86; P<0.001). Arrested heart TV surgery was protective (HR: 0.32; 95% CI: 0.13–0.78; P=0.012). Postoperatively, persistent moderate-to-severe RV dysfunction, elevated RAP, elevated PA pressures, elevated RVEDP, low PAPi, elevated bilirubin, and elevated INR were associated with increased mortality (all P<0.05, *Figure 3*, *Table 5*).

**Table 1** Demographics, preoperative, and intraoperative characteristics in patients operated for tricuspid valve intervention after heart transplant

| Variable | Values |
|---|---|
| Age in years | 53.9±14.4 |
| Gender (male) | 28 [68.3] |
| NYHA class III/IV | 37 [90.2] |
| Preoperative dialysis | 5 [12.2] |
| Mechanism of TV regurgitation | |
| Ruptured chord/flail leaflet | 22 [53.7] |
| Annular dilatation with leaflet tethering | 16 [39] |
| Inflammatory | 1 [2.4] |
| Permanent pacemaker lead impingement | 2 [4.9] |
| Technique of HTx | |
| Biatrial | 26 [63.4] |
| Bicaval | 15 [36.6] |
| Early cardiac allograft rejection | 3 [7.3] |
| Late cardiac allograft rejection | 18 [45] |
| Number of EMBs | 19.8±16.5 |
| Number of EMBs in late TV group | 26.4±14 |
| Echocardiography | |
| TV regurgitation | |
| Moderate | 6 [14.6] |
| Severe | 35 [85.4] |
| RV function | |
| Normal/mild dysfunction | 25 [61] |
| Moderate/severe dysfunction | 12 [29.2] |
| Severe dysfunction | 4 [9.8] |
| LV function | |
| Normal | 32 [78] |
| Mild dysfunction | 9 [22] |
| Preoperative right heart catheterization parameters | |
| Right atrial pressure (mmHg) | 14.2±5.2 |
| RV end diastolic pressure (mmHg) | 13.1±5.5 |
| PA systolic pressure (mmHg) | 33.4±7.5 |
| PA diastolic pressure (mmHg) | 16.6±5.6 |
| PA mean pressure (mmHg) | 23.2±6.1 |
| PA pulsatility index (mmHg) | 1.4±0.9 |
| Pulmonary vascular resistance (Woods units) | 2.7±1.4 |
| Preoperative biochemical parameters | |
| INR | 1.2±0.2 |
| Creatinine (mg/dL) | 2.1±1.2 |
| Albumin (g/dL) | 3.9±0.45 |
| Bilirubin (mg/dL) | 5.3±4.7 |
| Interval between HTx and TV intervention (years) | 7.1±5.5 |
| TV intervention during the index HTx admission | 9 [22] |
| Preoperative TMCS | 3 [7.5] |
| RVAD + IABP | 1 [2.5] |
| RVAD | 1 [2.5] |
| ECMO + IABP | 1 [2.5] |
| Prior surgery before HTx | 12 [29.3] |
| LVAD | 5 [12.2] |
| HTx | 1 [2.5] |
| Congenital anomaly repair | 6 [14.6] |
| Surgical approach | |
| Sternotomy | 39 [95.1] |
| Thoracotomy | 2 [4.9 ] |
| Aortic cross clamp used | 20 [48.8] |
| Aortic cross clamp time (min) | 56.4±32.1 |
| CPB time (min) | 83.5±41.6 |
| Type of valve intervention | |
| Repair | 15 [36.6] |
| Replacement | 26 [63.4] |

Data are presented as mean ± standard deviation or n [%]. CPB, cardiopulmonary bypass; ECMO, extracorporeal membrane oxygenation; EMB, endomyocardial biopsies; HTx, heart transplant; IABP, intra-aortic balloon pump; INR, international normalized ratio; LV, left ventricle; LVAD, left ventricular assist device; MCS, mechanical circulatory support; NYHA, New York Heart Association; PA, pulmonary artery; RV, right ventricle; RVAD, right ventricular assist device; TV, tricuspid valve.

**Table 2** Post-tricuspid valve intervention complications

| Variables | N (%) |
|---|---|
| Complications | |
| Deep sternal wound infection | 1 (2.5) |
| Permanent pacemaker | 3 (7.3) |
| In-hospital dialysis | 14 (35.0) |
| Discharge on dialysis | 6 (14.6) |
| Stroke | 1 (2.5) |
| Need of TMCS | 3 (7.3) |
| Need of reintervention | 3 (7.3) |
| Tricuspid valve repair | 1 (2.5) |
| Re-transplant | 2 (4.9) |
| In-hospital mortality | 5 (12.2) |
| Tricuspid valve replacement group mortality | 3 (11.5) |
| Tricuspid valve repair group mortality | 2 (13.3) |
| Cause of in-hospital mortality | |
| Sepsis | 1 (2.5) |
| Stroke | 1 (2.5) |
| Sudden cardiac arrest | 1 (2.5) |
| Tricuspid valve thrombosis | 1 (2.5) |
| Severe right ventricular dysfunction | 1 (2.5) |

TMCS, temporary mechanical circulatory support.

## Discussion

Key findings of our study are: (I) early TR after HTx was related to PGD while late TR was related to biopsy related TV injury or annular dilatation; (II) severe TR was present in 85% of patients while moderate to severe RV dysfunction was present in 39% patients; (III) despite the intent to repair the TV, 63.4% of patients underwent TVR; (IV) 56% patients died after TV surgery. Predictors of mortality were the presence of moderate-to-severe RV dysfunction (elevated RAP, RVEDP, and mPAP), and liver dysfunction (low serum albumin and elevated INR) prior to surgery and persistent moderate-to-severe RV dysfunction (elevated RAP and PA pressure, low PAPi), and liver dysfunction (elevated bilirubin and INR) after TV surgery.

In patients with HTx, PGD is an important risk factor for early TR. PGD results in RV dilatation and an increase in RV length along the superior-inferior axis, leading to TV tethering, reduced coaptation, and TR (10). There is a direct correlation between the significant TR in the first post-HTx echocardiogram and the need for TMCS. TMCS is usually required for a brief period, until the improvement in RV function and resolution of PGD; however, patients who continue to have significant TR despite improvement in the RV function usually need TV surgery (26). In our series, nine patients with PGD and significant TR required TV surgery and three of these nine patients also required TMCS. One patient who required TMCS and underwent TVR with a bioprosthesis subsequently died due to bioprosthetic valve thrombosis and sepsis.

EMB remains an integral part of the HTx management and multiple biopsies are performed over the years, especially during the first year. Studies have demonstrated a direct correlation between the number of EMBs and the development of TR (27-29). Nguyen *et al*. (28) in their study reported a negligible incidence of TR when EMB number was ≤18 and as high as 60% once the EMB numbers were >31. Further, in 47% of patients with new onset TR after HTx, chordal tissue was demonstrated in their EMB specimen (27). In our cohort, the mean number of biopsies in the late TR group was 26.4±14 and there was evidence of ruptured chordae and flail leaflets in 54% of patients. Measures to reduce the TV injury are limiting the number of EMBs, use of long bioptome sheath (30), and use of non-invasive genetic expression profiling for the detection of cardiac allograft rejection (31,32). Recently, cell free deoxyribonucleic acid (cfDNA) levels in the recipients' blood have been increasingly used for predicting acute cardiac allograft rejection (33). If these tests become standard of care in the future, it may reduce the need for cardiac allograft biopsies. At our institution, we have started to use cfDNA in patients who did not have rejection early after transplant to reduce the need for biopsy. Among bicaval, modified bicaval, and biatrial techniques of allograft implantation (4,7,34); biatrial technique is associated with the highest incidence of early and late TR, possibly due to the large redundant RA and anatomic distortion of the TV annulus (4,35). In the present series, 63.4% of patients had undergone HTx by biatrial technique. However, whether the biatrial technique was responsible for TR or was a contributory factor cannot be predicted from our study due to the small number of patients.

There is limited data available on the timing and outcome of isolated TV surgery in the HTx recipients. Asymptomatic patients are usually managed medically and followed up with echocardiography. Development of symptoms and/or

 

**Table 3** Comparison of preoperative and postoperative echocardiographic, hemodynamic, and biochemical parameters in patients operated for tricuspid valve intervention after orthotopic heart transplant

| Variables | Preoperative | Postoperative | P value |
|---|---|---|---|
| Echocardiography | | | |
| Tricuspid regurgitation (moderate/severe) | 41 [100] | 3 [7.3] | 0.001 |
| RV dysfunction (moderate/severe) | 16 [40] | 8 [20] | 0.12 |
| LV dysfunction (moderate/severe) | 0 | 3 [7.3] | 0.48 |
| Right heart catheterization parameters | | | |
| RA pressure (mmHg) | 14.2±5.2 | 12.0±5.2 | 0.017 |
| RV end diastolic pressure (mmHg) | 13.1±5.5 | 9.7±5.7 | 0.047 |
| PA systolic pressure (mmHg) | 33.4±7.5 | 33.2±8.7 | 0.48 |
| PA diastolic pressure (mmHg) | 16.6±5.6 | 15.5±5.1 | 0.24 |
| PA mean pressure (mmHg) | 23.2±6.1 | 23.5±6.1 | 0.55 |
| PA pulsatility index (mmHg) | 1.4±0.9 | 1.8±0.9 | 0.18 |
| PVR (Woods units) | 2.7±1.4 | 3.9±2.8 | 0.97 |
| Biochemical parameters | | | |
| INR | 1.2±0.2 | 1.3±0.4 | 0.38 |
| Creatinine (mg/dL) | 2.1±1.2 | 2.0±1.0 | 0.57 |
| Albumin (g/dL) | 3.9±0.45 | 3.9±0.59 | 0.76 |
| Bilirubin (mg/dL) | 5.3±4.7 | 0.7±1.1 | 0.0004 |

Data are presented as mean ± standard deviation or n [%]. INR, international normalized ratio; LV, left ventricle; PA, pulmonary artery; PVR, pulmonary vascular resistance; RA, right atrium; RV, right ventricle.

RHF and evidence of anatomic issue, e.g., chordal rupture, flail leaflet and TV annular distortion usually prompt TV surgery. Isolated TV surgery for symptomatic severe TR in non-HTx patients is associated with increased morbidity, 8–10% in-hospital mortality, and 25–30% 5-year mortality (36,37). Further, in patients undergoing TV surgery after HTx, the incidence of cardiogenic shock, AKI, and AKI requiring hemodialysis is significantly high (34). Presence of moderate to severe PH, severe RV dysfunction, and severe TR are ominous signs with increased perioperative mortality and dismal long-term survival (7). Some patients may need re-HTx due to persistent RV dysfunction after TV surgery. In our cohort, 12.2% had in-hospital mortality and 43.9% died during follow-up. Further, two patients underwent re-HTx, and an additional patient was listed but died before re-HTx. In our study, predictors of death were markers of advanced RHF and hepatic dysfunction. These findings underscore that once long-standing TR progresses to clinically significant RV failure and congestive hepatopathy, hepatic reserve continues to deteriorate despite correction of TR. Presently, most patients are referred for TV intervention only after progression to advanced RV failure and hepatic compromise. Our findings reinforce that timing is critical: meaningful survival is far more likely when intervention occurs before irreversible RV and hepatic injury develops. Hence, patients with HTx should be considered for the TV surgery before the onset of RV dysfunction or hepatic dysfunction. Studies have reported a 5-year survival of 72.5% after HTx (38) and 70–75% after isolated TV surgery in non-HTx patients (36,37). In the present study, 5-year survival after TV surgery was 60.5%. Our results show that patients undergoing TV surgery after HTx remain at a higher risk of perioperative and long-term mortality. Hence, patients with TR after HTx should be intervened on sooner rather than later, and the timing of surgery should be decided on a case-by-case basis.

Choice of surgical intervention also remains a matter of debate. Filsoufi *et al.* (19) recommended TVR for anatomic

 

**Table 4** Comparison of long-term outcome in various groups

| Outcome | Values | P value |
|---|---|---|
| Survival, years, mean ± standard deviation | 6.9±1 | |
| Long-term survival after TV operation, % | | |
| 5 years | 60.5 | |
| 10 years | 23.5 | |
| 20 years | 17.6 | |
| Median survival with aortic cross-clamp use, years | | 0.006 |
| Yes | 10 | |
| No | 4.4 | |
| Overall mortality, n [%] | 23 (56.1) | |
| In-hospital mortality | 5 (12.2) | |
| Long-term mortality | 18 (43.9) | |
| Mortality based on aortic cross-clamp use, n [%] | | 0.008 |
| Yes | 7 [35] | |
| No | 16 [76] | |
| Mortality based on type of TV operation, n [%] | | 0.58 |
| Replacement | 16 [61.54] | |
| Repair | 7 [46.7] | |
| Mortality based on technique of transplant, n [%] | | 0.09 |
| Biatrial | 15 [68.2] | |
| Bicaval | 8 [42] | |

TV, tricuspid valve.

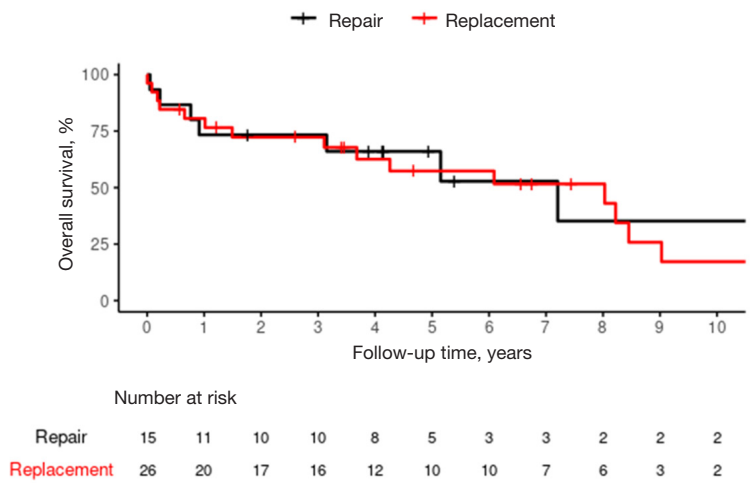


**Figure 1** Kaplan-Meier curve of survival after tricuspid valve operations.

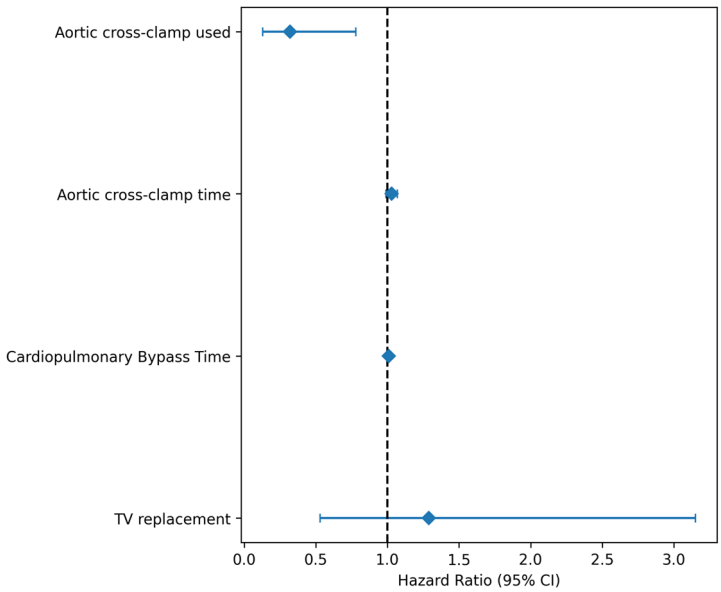


**Figure 2** Forest plot of operative variables associated with outcome. CI, confidence interval; TV, tricuspid valve.

causes, while TVr for functional etiologies. In patients undergoing TVR, a bioprosthetic valve is preferred as it allows continued access to the RV for biopsy in addition to a low risk of thrombosis, acceptable durability in the low-pressure system, and does not require long-term anticoagulation (7,39,40). Mohammed *et al*. (34) in a review of 366 patients with TV surgery after HTx reported the use of a mechanical valve in 16% of patients. In-hospital mortality was 17% in the mechanical valve group compared to 9% in TVr group and none in the bioprosthetic valve group; although, the difference was not statistically significant. Further, the incidence of AKI requiring hemodialysis was significantly higher in the mechanical valve group compared to the bioprosthetic valve and TVr groups (33% *vs.* 17% *vs.* 3%, respectively; P=0.024). In our study, no patient underwent TVR with a mechanical valve, and in-hospital mortality between TVR and TVr was not statistically significant.

With the availability of transcatheter tricuspid valve interventions (TTVI) in recent years, suitable high-risk patients with post-HTx severe TR can now be managed with a less invasive and vital alternative (Class IIa, Level A recommendation in recent guidelines). A recent study has reported effective symptom reduction and improved survival with transcatheter tricuspid edge-to-edge repair (T-TEER) and transcatheter tricuspid valve replacement (TTVR) compared to medical therapy. T-TEER devices [the Pascal Precision system (Edwards Lifesciences) and TriClip (Abbott)] have shown high fidelity in reducing symptomatic severe TR to moderate or less, with specific evidence in post-HTx patients (41). When the TV anatomy is unsuitable for repair (large coaptation gap, severe annular dilatation), orthotopic TTVR devices [EVOQUE (Edwards Lifesciences), a U.S. Food and Drug Administration (FDA)-approved system and in clinical trials or compassionate use system, e.g., the Intrepid (Medtronic), Lux-Valve Plus (Jenscare), Topaz (TRiCares), and NaviGate (NaviGate Cardiac Structures)] are an alternative for surgical TVR in high-risk patients. As per the 2022 European Society of Cardiology (ESC) and European Association for Cardio-Thoracic Surgery (EACTS) guidelines, TTVI is a Class IIb recommendation in patients with suitable anatomy but deemed inoperable surgically, and can expect a meaningful improvement in quality of life or survival (42). American societies currently do not mention TTVI options in their guidelines. However, a number of promising TTVI strategies are being tested. Given the complexity of the disease and the high-risk nature of this population, personalized evaluation by a multi-disciplinary team, including a heart failure cardiologist, cardiac surgeon, interventional cardiologist, cardiac anesthetist, and intensivist, is crucial for the successful outcome.

TV surgery can be performed with arrested heart or beating heart. In our study, based on surgeon's preference,

 

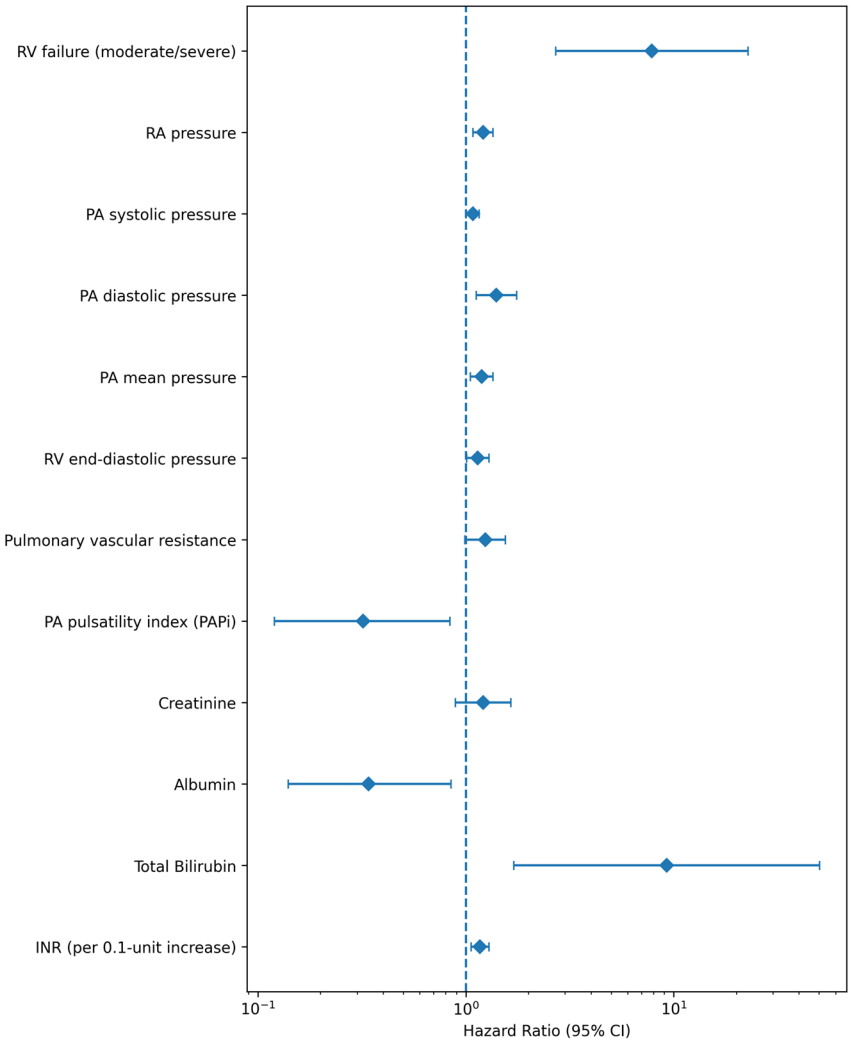


**Figure 3** Forest plot of postoperative variables associated with outcome (log scale). CI, confidence interval; INR, international normalized ratio; PA, pulmonary artery; RA, right atrial; RV, right ventricle.

TV surgery was performed either on a beating heart or an arrested heart. Our results indicate that the long-term and mean survival is significantly better with the arrested heart. Possible reasons for the better long-term survival with arrested heart may be related to more complete annular and subvalvular correction, although we could not test this hypothesis in our study due to limited sample size. Our findings are consistent with Baraki *et al*., who in a cohort of 92 patients operated for TV reported numerically better but statistically non-significant 1-, 5-, and 10-year survival with the arrested-heart approach compared to the beating-heart (39). On the other hand, Russo *et al*. in a multicenter study of 406 patients operated for isolated TV, reported better survival in the beating heart group compared to the arrested heart group after 6 years, although the difference was not statistically significant (40). A key limitation of both studies is the lack of focus on HTx recipients. Since, in HTx recipients, factors such as difficult pericardial dissection, presence of severe RV dysfunction, and renal failure not only increase the perioperative risk but also influence the surgeon's preference to avoid the aortic cross-clamp. We recommend larger studies dedicated specifically to post-HTx TV surgery to resolve these questions.

Our study has several limitations. Due to the retrospective nature of the study, confounding and data selection bias cannot be ruled out. Data related to chronic

**Table 5** Univariable Cox regression model predicting overall mortality after tricuspid surgery

| Variables | Hazard ratio (95% CI) | P value |
|---|---|---|
| Preoperative variables | | |
| Age | 1.01 (0.98–1.04) | 0.523 |
| Female | 0.61 (0.23–1.65) | 0.332 |
| Severe TR | 1.05 (0.31–3.56) | 0.937 |
| LV dysfunction none/mild | 1.15 (0.42–3.1) | 0.786 |
| RV dysfunction moderate/severe | 1.11 (0.47–2.63) | 0.81 |
| RA pressure | 1.12 (1.01–1.24) | 0.025 |
| RV end-diastolic pressure | 1.12 (1.01–1.24) | 0.031 |
| PA mean pressure | 1.08 (1–1.16) | 0.044 |
| Creatinine | 1.38 (0.96–1.98) | 0.084 |
| Albumin | 0.2 (0.05–0.8) | 0.024 |
| Total bilirubin | 0.97 (0.87–1.09) | 0.633 |
| INR (per 0.1-unit increase) | 2.09 (1.53–2.86) | <0.001 |
| Preoperative dialysis | 2.14 (0.61–7.48) | 0.235 |
| Late cardiac allograft rejection | 2.11 (0.84–5.3) | 0.114 |
| Number of biopsies | 1.01 (0.97–1.04) | 0.731 |
| Repair during same HTx admission | 0.47 (0.11–2.08) | 0.321 |
| Time from HTx to TV surgery (years) | 1.03 (0.96–1.12) | 0.41 |
| Operative variables | | |
| Aortic cross-clamp used | 0.32 (0.13–0.78) | 0.012 |
| Aortic cross-clamp time | 1.03 (0.99–1.07) | 0.158 |
| Cardiopulmonary bypass time | 1.01 (1–1.02) | 0.142 |
| TV replacement | 1.29 (0.53–3.15) | 0.576 |
| Postoperative variables | | |
| RV failure (moderate/severe) | 7.85 (2.71–22.79) | <0.001 |
| RA pressure | 1.21 (1.08–1.35) | 0.001 |
| PA systolic pressure | 1.08 (1–1.16) | 0.038 |
| PA diastolic pressure | 1.4 (1.12–1.76) | 0.003 |
| PA mean pressure | 1.19 (1.05–1.35) | 0.007 |
| RV end-diastolic pressure | 1.14 (1.01–1.29) | 0.030 |
| Pulmonary vascular resistance | 1.24 (0.99–1.55) | 0.058 |
| PA pulsatility index | 0.32 (0.12–0.84) | 0.021 |
| Creatinine | 1.21 (0.89–1.65) | 0.234 |
| Albumin | 0.34 (0.14–0.85) | 0.021 |
| Total bilirubin | 9.25 (1.7–50.22) | 0.01 |
| INR (per 0.1-unit increase) | 1.17 (1.06–1.29) | 0.002 |

CI, confidence interval; HTx, heart transplant; INR, international normalized ratio; PA, pulmonary artery; PVR, pulmonary vascular resistance; RA, right atrium; RV, right ventricle; TR, tricuspid regurgitation; TV, tricuspid valve.

allograft vasculopathy, donor-specific antibodies, and immunosuppression regimens were not available for all the patients, hence not included. For the diagnosis of RV dysfunction, we relied on TAPSE and FAC since these were available for all the patients. We did not include RV strain as it was available only in a few patients. The small sample size and the limited number of events limited the use of a multivariable model and the statistical power to identify risk factors associated with survival.

## Conclusions

TR after HTx remains a significant problem, although only a small proportion of patients require surgical intervention. Morbidity and mortality after TV surgery in HTx recipients remain high. Presence of moderate to severe RV dysfunction and liver dysfunction at the time of surgery and persistence of moderate to severe RV dysfunction and liver dysfunction after TV surgery are associated with increased perioperative and long-term mortality. Patients with HTx should be considered for the TV surgery before the onset of RV dysfunction or hepatic dysfunction. Both repair and replacement can provide durable benefits when performed early in the clinical course. The survival advantage observed with TV surgery on arrested heart warrants confirmation in larger HTx-specific cohorts. Moving forward, severe TR after HTx should be approached proactively rather than as a salvage operation, incorporating earlier referral, careful ventricular/hepatic assessment, and consideration of re-HTx in appropriately selected patients.

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