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ORIGINAL ARTICLE

Long-term Survival after Isolated Tricuspid Valve Replacement

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Background Isolated replacement of the tricuspid valve is rare, and the decision to operate is difficult. This study reviews the in-hospital mortality and long-term survival after tricuspid valve replacement in the absence of concomitant left sided valve surgery. It identifies predictors of poor outcome.

Methods All patients who underwent tricuspid valve replacement between January 1995 and December 2011 were retrospectively reviewed. Patients having concomitant mitral or aortic valve surgery were excluded. Logistic regression was used to identify predictors of early and late death.

Results Twenty-nine cases were identified. There were six in-hospital deaths (20.6%), and eight late deaths. Ascites was associated with in-hospital death (hazard ratio 16.96; p=0.0052). Higher dose of Frusemide was associated with late mortality (hazard ratio 1.157 per 20 mg increase; p=0.0155). Frusemide dose and ascites were both significantly associated with death overall (p<0.01). Survival analysis estimated a 50% probability of surviving to 12.45 years.

Conclusions Isolated tricuspid valve replacement has a high peri-operative risk. Long-term survival in this study was consistent with other reports. Ascites and higher doses of Frusemide were associated with poor outcomes.

Keywords Heart valve diseases • Tricuspid valve replacement • Tricuspid valve insufficiency • Tricuspid valve stenosis • Rheumatic heart disease • Heart failure

Introduction

Tricuspid valve replacement (TVR) is an uncommon operation. Most reports contain a small number of patients with a high mortality [1–12]. The decision to operate on the tricuspid valve is difficult when it is the only valve requiring treatment, especially when surgery may lead to replacement rather than repair.

Most studies do not separate patients who have had tricuspid valve replacement alone from those who have also had left sided valve surgery during the same procedure, and often combine repair and replacement patients. Many include data back to the 1960s, with results which may not be applicable to current practice.

The aims of this study were to define the in-hospital and long term mortality of patients at our institution who underwent TVR without concomitant surgery to the left sided valves since 1995, and to identify factors associated with mortality.

Materials and Methods

A retrospective chart review was undertaken of all patients undergoing TVR without concomitant mitral or aortic valve surgery between January 1995 and December 2011 at the Green Lane Cardiothoracic Unit. Patient demographics, preoperative clinical status, operative details, postoperative

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morbidity and late outcomes were analysed. Late survival was verified from the National Registry of Births Deaths and Marriages.

Statistical analysis

Univariate Cox proportional hazards regression was used to determine predictors of in-hospital death and late death. Statistical analyses were performed with SAS software version 9.3 (SAS Institute, Cary, NC). All p-values resulted from two sided tests and a p-value of <0.05 was considered statistically significant.

Table 1 Patient demographics and pre-operative variables.

	Number of TVR = 29	Percent (%)
Age (mean)	46 (range 11-80)	
Gender (female)	21	72.4
Original lesion		
Rheumatic	9	31.0
Congenital	10	34.5
Endocarditis	5	17.2
Carcinoid	3	10.3
Other	2	6.9
NYHA class		
I/II	16	55.2
III/IV	7	24.1
Unknown	6	20.6
Bilirubin (pre-operative level)		
Raised (>25umol/L)	10	34.5
Normal	14	48.3
Unknown	5	17.2
Ascites (on pre-operative examination)		
Present	2	6.90
Absent	25	86.2
Unknown	2	6.90
Hepatomegaly (on pre-operative examination)		
Present	13	44.8
Absent	11	37.9
Unknown	5	17.2
Right ventricular function (on pre-operative echocardiogram)		
Normal	15	51.72
Mildly impaired	6	20.7
Moderately impaired	2	6.9
Severely impaired	2	6.9
unknown	4	13.8
Number of previous heart surgeries		
0	9	31.0
1	15	51.7
2+	5	17.2
Previous TV repair	5	17.2
Previous TV replacement	7	24.1

NYHA= New York Heart Association; TV=tricuspid valve.

Results

The cardiothoracic surgical database identified 30 patients. Two of these patients had a systemic right ventricle and were excluded. One patient with carcinoid disease underwent two TVRs during the study period so that the total number of replacements was 29 in 28 patients.

Demographics

Patient demographics are presented in Table 1. Mean age was 46 years (range 11-80). Twenty-one cases (72.4%) were female. The original native valve lesion was rheumatic in nine, congenital in 10, endocarditis in five, carcinoid in two patients (one of these was operated on twice during the study period), one following right ventricular infarction, and one idiopathic. This is distinct from the indication for surgery as listed in Table 2. Twenty (61%) had previously undergone open heart surgery. Five (17%) had undergone two or more previous cardiac surgeries. Prior procedures included Tricuspid valve repair or replacement, atrial septal defect closure, Pulmonary valve procedures, Mitral valve replacement, Aortic valve replacement, and coronary artery bypass grafting.

Surgical procedure

A midline sternotomy was performed in all patients. Standard aortic and bicaval cannulation was performed in 25 cases. Femoral cannulation was used in four. Ten cases were done with the heart beating. In the other 19 cases, antegrade

Table 2 Indication for Surgery.

	Number of TVR =29	Percent (%)
Severe TR		
- native valve	7	24.1
- failed repair	5	17.2
- prosthetic degeneration	2	6.9
- native valve +coronary artery disease	1	3.4
- native valve + free PR	2	6.9
Mixed TR/TS		
- native valve	2	6.9
- prosthetic degeneration	4	13.8
- native valve + PS (prior to hepatic surgery)	1	3.4
Infection	4	13.8
Recurrent thrombosis of mechanical TVR	1	3.4

TR=tricuspid regurgitation; PR=pulmonary regurgitation; TS=tricuspid stenosis; PS=pulmonary stenosis.

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Table 3 Univariate survival regression – in-hospital deaths.

Variable	Hazard Ratio	95% CI	P-value
BMI (per 5 kg/m^2 increase)	1.15	0.63 - 2.101	0.649
BSA (m^2)	1.107	0.051 - 24.026	0.9481
Total bilirubin	1.032	0.989 - 1.076	0.1437
Frusemide (per 20 mg increase)	1.121	0.96 - 1.308	0.1482
Ascites	16.963	2.328 - 123.613	0.0052
Hepatomegaly	2.628	0.273 - 25.272	0.4028
Previous heart surgery	0.993	0.311 - 3.172	0.9912
No concomitant procedure	1.793	0.328 - 9.798	0.5003
Valve implanted (mechanical vs tissue)	1.986	0.364 - 10.853	0.4282
Underlying cause (congenital vs other)	0.357	0.042 - 3.057	0.3473
NYHA Class (I/II vs III/IV)	1.53	0.255 - 9.179	0.642
Second Diuretic	0.302	0.05 - 1.811	0.1903

BMI=body mass index; BSA=body surface area; NYHA=New York Heart Association.

or combined antegrade and retrograde cold blood cardioplegia was used.

In our institution, repair is performed whenever feasible. The reasons for replacing the tricuspid valve in this series were: previous prosthetic tricuspid valve replacement (7), thickened and retracted leaflets with cordal shortening and fusion (7), failed repair (5), endocarditis with valve destruction (4), gross myxomatous enlargement of the leaflets with ruptured cords (2), Ebstein valve not amenable to repair (2), restrictive central orifice (1), and extreme annular dilatation (1).

Sixteen patients had TVR alone. Concomitant procedures were performed in 13 patients: pulmonary valve procedures (4), MAZE procedures (4), closure of patent foramen ovale/atrial septal defect or ventricular septal defect (3), coronary bypass grafting (2), epicardial lead placements (2), and bidirectional cavopulmonary shunt (1).

Choice of valve prosthesis was based on surgeon and patient preference. Twenty-three patients (79%) received a bioprosthesis; 14 Mosaic (Medtronic, Minneapolis, MN), five Perimount (Edwards Lifesciences, Irvine, CA), and four Intact (Medtronic, Minneapolis, MN). Six patients (21%) received a mechanical valve; five St Jude (St Jude Medical, St Paul, MN), and one ATS (ATS Medical, Inc, Plymouth, MN).

Early mortality and morbidity

There were six in-hospital deaths (20.6%). The cause of death was low cardiac output/right ventricular failure in four and sepsis in two. The only preoperative variable found to be statistically significantly associated with in-hospital mortality was the presence of ascites (hazard ratio 16.96; p=0.0052) (Table 3).

Early complications were as follows: nine patients (31%) required pacemakers, five patients (17%) had acute renal failure requiring renal replacement, six patients (21%) developed low cardiac output, and three patients (10%) were re-explored for bleeding. There was one deep sternal wound infection, one tracheostomy for prolonged ventilation and no strokes.

Long term outcome

There were eight late deaths (34.7% of those who survived to hospital discharge). At the time of review, 15 patients were still alive at a mean of 13.9 years since surgery. Survival analysis estimated a 50% probability of surviving to 12.45 years (Fig. 1).

Factors found to be associated with late mortality were higher preoperative dose of Frusemide (hazard ratio 1.157 per 20 mg increase in Frusemide dose), and higher body surface area (BSA) (hazard ratio 19.267 per m^2) (Table 4).

Frusemide dose and ascites were both statistically significantly associated with death overall. The hazard ratio for Frusemide was 1.143 per 20 mg increase in pre-operative Frusemide dose (p=0.0055). The hazard ratio for the presence of pre-operative ascites was 16.96 (p=0.0052) (Table 5).

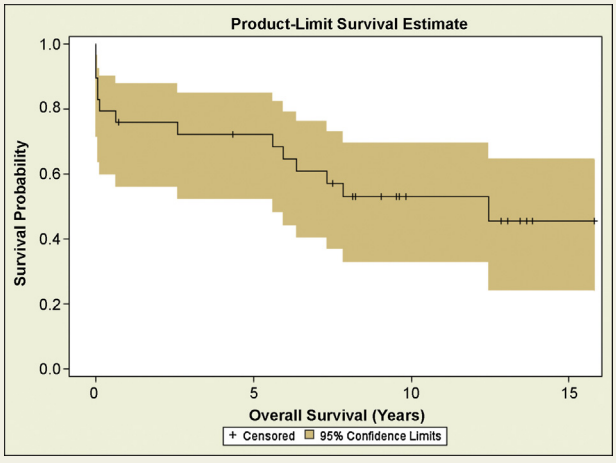

Figure 1 Survival Probability Curve.

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Table 4 Univariate survival regression – late deaths.

Variable	Hazard Ratio	95% CI	P-value
BMI (per 5 kg/m^2 increase)	1.45	0.915 - 2.296	0.1134
BSA (m^2)	19.267	1.095 - 339.098	0.0432
Total bilirubin	1.022	0.981 - 1.065	0.3006
Frusemide (per 20 mg increase)	1.157	1.028 - 1.301	0.0155
Hepatomegaly	3.031	0.61 - 15.047	0.175
Previous heart surgery	0.954	0.334 - 2.729	0.9305
No concomitant procedure	0.47	0.112 - 1.972	0.302
Valve implanted (mechanical vs tissue)	1.406	0.283 - 6.977	0.6767
Underlying cause (congenital vs other)	0.481	0.096 - 2.405	0.3725
NYHA Class (I/II vs III/IV)	6.072	0.992 - 37.173	0.0511
Second Diuretic	0.927	0.179 - 4.795	0.9283

BMI=body mass index; BSA=body surface area; NYHA=New York Heart Association.

Table 5 Univariate survival regression – all deaths.

Variable	Hazard Ratio	95% CI	P-value
BMI (per 5 kg/m^2 increase)	1.325	0.92 - 1.909	0.1303
BSA (m^2)	5.683	0.68 - 47.50	0.1087
Total bilirubin	1.027	0.99 - 1.06	0.076
Frusemide (per 20 mg increase)	1.143	1.04 - 1.256	0.0055
Ascites	16.963	2.33 - 123.61	0.0052
Hepatomegaly	2.893	0.78 - 10.71	0.1116
Previous heart surgery	0.972	0.446 - 2.118	0.9423
No concomitant procedure	0.831	0.29 - 2.37	0.7292
Valve implanted (mechanical vs tissue)	1.644	0.515 - 5.248	0.4011
Underlying cause (congenital vs other)	0.429	0.119 - 1.548	0.1962
NYHA Class (I/II vs III/IV)	3.023	0.857 - 10.662	0.0853
Second diuretic	0.564	0.179 - 1.784	0.3301

BMI=body mass index; BSA=body surface area; NYHA=New York Heart Association.

Discussion

In the majority of cases, patients are accepted for tricuspid valve surgery as part of an operation addressing left sided valve disease [2]. The decision to operate for symptoms due directly to the tricuspid valve is more difficult, particularly when the valve requires replacement.

Tricuspid replacement has long been known to carry substantial risk, with reported operative mortality as high as 33% [1–12]. The in-hospital mortality in our series was 21%. This is similar to Mangoni et al. who reported an in-hospital mortality of 20% in a series of 15 patients who underwent isolated TVR between 1984 and 1996 [3]. A meta-analysis of studies published between 1994 and 2003 of TVR (including both isolated TVR and those done concomitantly with left sided valve procedures) documented a mortality of 19.2% in 1258 patients from 11 series [13]. The consistently high perioperative mortality rates have led authors to suggest that tricuspid valve disease requiring replacement may be a marker for end-stage valvular heart disease [3,6].

Our findings suggest that the degree of heart failure, as manifest by the dose of diuretic required and the presence of ascites, should be carefully considered when assessing a patient's risk. This is supported by previous studies as reported by Viganò et al., who found that chronic right heart failure symptoms were associated with hospital mortality [9]. Mangoni et al. identified anasarca as the sole predictor of short-term mortality, and suggested anasarca was a marker for patients with the most advanced heart failure [3]. Filsoufi et al. observed that heart failure was the predominant cause of early and late deaths, emphasising the importance of timely referral before the development of end-stage cardiac impairment [6].

It is apparent that right ventricular (RV) function plays a central role in both preoperative clinical status and postoperative outcome. Assessment of RV function, however, is

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problematic. In this study there was limited echocardiography information available retrospectively about the RV and pulmonary pressures and no analysis was able to be made. This may be in part due to the inherent limitations of this imaging modality in measuring RV function. Echocardiography provides some indication of RV size and systolic function but is mostly qualitative and subjective in nature, owing to the complex geometry of the right ventricle. Magnetic resonance imaging (MRI) offers a more quantitative measure of RV volumes and ejection fraction and is emerging as the clinical reference technique due to its unlimited imaging planes, superior image resolution, and three-dimensional volumetric rendering [14]. Nevertheless there remains intra- and inter-observer variability in MRI assessment of RV function [15] and this imaging modality is not universally available.

More precise estimates of RV function may help identify those patients in whom TVR will not be tolerated. Iscan et al. suggested that when chronic pressure and volume overload leads to irreversible RV deterioration, tricuspid regurgitation could be a compensatory mechanism and that its correction may result in acute RV decompensation [7]. Filsoufi et al. have proposed that a prospective series be undertaken to correlate preoperative echocardiogram, MRI, haemodynamic, clinical and biochemical findings with postoperative outcome to try and identify those hearts that will not cope with surgical correction [6].

Our study documents a long-term survival following TVR that is consistent with other reports. Those that were alive at the time of review have survived a mean of 13.9 years since surgery thus far and for the entire cohort there was a 50% probability of surviving 12.5 years. It has been suggested that the aetiology of the valve lesion plays an important role in long-term survival [8]. Several groups have reported 10 year survival in excess of 70% for TVR for Ebstein valves [7,16], compared with 41% for rheumatic disease [7] although no difference in survival was found between aetiological groups in our study.

Although the perioperative mortality is substantial, a proportion of patients in whom surgery is successful go on to survive 10 years or more, as reported in this study and others [7,8]. For this reason, some authors advocate mechanical valve replacement of the tricuspid valve [8]. Others, in contrast, recommend bioprostheses as this avoids the requirement for anticoagulation, arguing that patients undergoing TVR are unlikely to survive long enough to risk valve degeneration [12]. Most studies comparing mechanical to biological tricuspid prostheses report similar survival and re-operation rates [1,10,12].

Greater BSA was found to be statistically significantly associated with late mortality in our study. This is unexpected, as there have been several studies which have reported small body size to be associated with worse outcomes after cardiac surgery [17,18]. We are uncertain of the significance, if any, of this finding in our study.

Given the findings in our study, particular attention is paid to the state of the right ventricle and the clinical evidence of heart failure in patients referred for tricuspid surgery. While we have demonstrated that isolated tricuspid replacement carries a high operative mortality, we are encouraged by the long term results in this study.

Limitations

This study is limited by its relatively small number of patients and retrospective nature. Concomitant left sided valve surgery was excluded in order to identify those patients for whom the decision to operate was due to the ticuspid valve. However, nearly half the group underwent other associated cardiac procedures which may have influenced both the decision to operate and outcomes. Nine patients had had previous left sided vale surgery, and it is possible that late survival may have been influenced by these coexisting cardiac lesions. Analysis of outcome as related to aetiology was carried out as congenital versus other due to the small numbers in each aetiological subcategory. Nonetheless this series is one of the largest reported experiences of valve replacement performed where the tricuspid valve was the dominant clinical lesion.

Conclusion

This study demonstrates a high perioperative risk associated with isolated tricuspid valve replacement. Long term survival was consistent with other reports. Ascites and higher Frusemide dose were associated with worse outcomes, suggesting the importance of pre-operative clinical status. Discriminating between right ventricles which are capable of recovery and those which have irreversibly failed remains difficult.

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