

Transplant-TR: A Few Points

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Citation

B Phillips. *Transplant-TR: A Few Points*. The Internet Journal of Cardiology. 2003 Volume 2 Number 2.

Abstract

The reported incidence of tricuspid valve regurgitation in the heart transplant population varies widely, occurring in 20-83% of patients [1, 2]. Regardless of the actual incidence, most tricuspid regurgitation after transplantation is mild (either asymptomatic or easily-controlled with diuretics). On occasion, however, the degree of regurgitation may be of such severity as to warrant tricuspid valve surgery [3, 4]. The etiology of TR in this select population has been attributed to a number of theoretical mechanisms: anastomotic technique; ischemic reperfusion injury; iatrogenic damage from endocardial biopsy; size mismatch between donor and recipient hearts; and chronic rejection. While deciphering the mechanism responsible may be difficult, it should be noted that the onset of tricuspid valve disease may have profound consequences in this vulnerable population.

INTRODUCTION

The technique of orthotopic heart transplantation commonly performed until the 1990's was that of Lower and Shumway's [5, 6] in which a right biatrial anastomosis was created. Although this preserved a portion of the recipient's right atrium, the final geometry of the amalgamated product was not infrequently distorted. Furthermore, this arrangement led to asynchronous atrial contraction and atrial dilatation. Atrial systolic contraction, which normally contributes up to 20% of cardiac output, was thought to be compromised with this technique. Before long, reports of tricuspid regurgitation and mitral regurgitation surfaced (in the context of a normal valve apparatus) [7, 8] Sievers, et al [9] are credited with popularizing the modern technique of bicaval anastomosis. Today, this is the most common method used in orthotopic heart transplantation. In their report of this procedure, 24 patients were prospectively randomized to receive either conventional versus bicaval anastomoses. Echocardiographic follow-up, 2 years later, demonstrated a larger right atrium and a higher incidence of tricuspid regurgitation during exercise in those undergoing biatrial-conventional anastomoses. In a similar report, Sarsam, et al also found a lower incidence of post-operative right heart failure and right atrial pressure (4.9 mmHg vs. 9.6mm Hg, $p < .01$) in those transplanted with bicaval anastomoses, despite a lack of difference in pulmonary artery pressures [10].

DISCUSSION

Percutaneous transvenous endomyocardial biopsy of the transplanted heart is the preferred method to detect allograft

rejection post-operatively [11]. While patients undergo 10 - 15 biopsies in the first post-operative year, major complications, such as cardiac perforation and tamponade are unusual, and occur in less than 0.4% of cases [12]. Less severe complications include dysrhythmias, heart block, endocarditis, and tricuspid regurgitation. Tricuspid regurgitation in the post-biopsy heart transplant patient is frequently the result of inadvertent iatrogenic injury of individual chords by the biopsy biopome [13]. In one of the earliest reports of this complication, Braverman, et al. noted a 6.2% incidence of iatrogenically induced post-biopsy tricuspid regurgitation as echocardiographically documented by posterior leaflet chordal prolapse into the right atrium during systole. In a report of 193 transplant patients undergoing some 2960 biopsies, Huddleston, et al also found a 6% incidence of post biopsy tricuspid regurgitation. However, two-thirds of these patients were either without or had mild symptoms and most were treated medically with diuretics. Only 1% of all patients developed iatrogenically induced severe tricuspid regurgitation that was refractory to medical treatment necessitating surgical correction [14]

In addition to the aforementioned etiologies, some authors have also attributed the higher incidence of tricuspid regurgitation in the transplant population to pre-existing pre-operative elevated pulmonary arterial pressures. For example, Lewen et al found that pulmonary artery systolic pressure (greater than 55mmHg) one month pre-operatively was a predictor of moderate to severe tricuspid regurgitation post-operatively (Mean, 17 months). In addition, he reported that PVR was significantly elevated in patients with

moderate or severe TR [15]. Rees et al also found that all post-transplant patients with moderate regurgitation had pulmonary artery systolic pressures of greater than 30mmHg, in addition to a higher incidence of right ventricular enlargement [2]. They did not, however, find a correlation between post-operative pulmonary artery pressures and tricuspid regurgitation. When tricuspid regurgitation does occur in the transplant-patient, medical management is usually effective. When severe enough to warrant surgical correction (i.e. the existence of signs and symptoms of right-sided failure), valve replacement is most commonly performed to avoid failed repair and the need for further operative intervention.

Figure 1

Important Literature

Chan et al 2001 (<i>Stanford</i>) 336 patients, 5 yr review	-	90 pts, Moderate TR 23 pts, Severe TR 6 pts eventually required Replacement
Williams et al 1996 (<i>Mass Gen</i>) 72 patients, 29 month f/u	-	23 pts, Moderate/Severe MR 10 pts – Flail Tricuspid Leaflet (7 had severe TR)
Rees et al 1993 (<i>Ochsner</i>) [2] 48 patients, 5 month mean f/u	-	41 pts with TR (35 Mild, 6 Moderate)
Aziz et al 1999 (<i>Manchester</i>) 249 patients, 10 yr. review	-	102 pts with Moderate-Severe TR

	161 pts - Conventional		88 pts - Bicaval	
	No/Mild TR	Mod-Severe TR	No/Mild TR	Mod-Severe TR
1 month	93/156	63/156	72/84	12/84
12 months	73/146	73/146	62/81	19/81
24 months	67/138	71/138	56/79	23/79
Recent	38/119	81/119	49/70	21/70

* Risk factors for Early TR: conventional technique, rejection episodes > grade 2, and increased pulmonary resistance

* Risk Factors for Late TR: conventional technique, rejection episodes > grade 2, and the total number of heart biopsies

Clinically, 85% of the transplant recipients with Moderate-Severe TR

presented with Classic signs of Right Heart Failure:

- Progressive Fatigue (35%)
- Chronic Fluid Congestion (61%)
- LE Edema (78%)
- Hepatomegaly (29%)
- JVD (59%)

91% of these pts required massive doses of furosemide to control symptoms (greater than 120 mg/day)... unknown if any required valve replacement ?

CONCLUSION

The reported incidence of tricuspid valve regurgitation following orthotopic transplantation is highly variable amongst institutions. Fortunately, most patients have mild tricuspid regurgitation. On occasion, however, the TR may be of such severity as to require intervention. While deciphering the actual etiology of TR may be difficult, the onset of symptoms following heart transplant has profound consequences in the overall management of these patients.

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References

1. Yankah, A.C., et al., Tricuspid valve dysfunction and surgery after orthotopic cardiac transplantation. *European Journal of Cardio-Thoracic Surgery*, 2000. 17(4): p. 343-8.
2. Rees, A.P., et al., Valvular regurgitation and right-sided cardiac pressures in heart transplant recipients by complete Doppler and color flow evaluation. *Chest*, 1993. 104(1): p. 82-7.
3. Votapka, T.V., R.S. Appleton, and D.G. Pennington, Tricuspid valve replacement after orthotopic heart transplantation. *Annals of Thoracic Surgery*, 1994. 57(3): p. 752-4.
4. Stahl, R.D., et al., Tricuspid valve dysfunction in the transplanted heart. *Annals of Thoracic Surgery*, 1995. 59(2): p. 477-80.
5. Lower, R.R. and N.E. Shumway, Studies of the orthotopic homotransplantation of the canine heart. *Annals of Thoracic Surgery*, 1960. 11: p. 18-23.
6. Shumway, N.E., R.R. Lower, and R.C. Stofer, Transplantation of the heart. *Advances in Surgery*, 1966. 2: p. 265-84.
7. Angermann, C.E., et al., Anatomic characteristics and valvular function of the transplanted heart: transthoracic versus transesophageal echocardiographic findings. *Journal of Heart Transplantation*, 1990. 9(4): p. 331-8.
8. Stevenson, L.W., et al., Mitral regurgitation after cardiac transplantation. *American Journal of Cardiology*, 1987. 60(1): p. 119-22.
9. Sievers, H.H., et al., An alternative technique for orthotopic cardiac transplantation, with preservation of the normal anatomy of the right atrium. *Thoracic & Cardiovascular Surgeon*, 1991. 39(2): p. 70-2.
10. Sarsam, M.A., et al., An alternative surgical technique in orthotopic cardiac transplantation. *Journal of Cardiac Surgery*, 1993. 8(3): p. 344-9.
11. Caves, P.K., et al., Diagnosis of human cardiac allograft rejection by serial cardiac biopsy. *Journal of Thoracic & Cardiovascular Surgery*, 1973. 66(3): p. 461-6.
12. Fowles, R.E. and J.W. Mason, Endomyocardial biopsy. *Annals of Internal Medicine*, 1982. 97(6): p. 885-94.

13. Braverman, A.C., et al., Ruptured chordae tendineae of the tricuspid valve as a complication of endomyocardial biopsy in heart transplant patients. *American Journal of Cardiology*, 1990. 66(1): p. 111-3.

14. Huddleston, C.B., et al., Biopsy-induced tricuspid

regurgitation after cardiac transplantation. *Annals of Thoracic Surgery*, 1994. 57(4): p. 832-6; discussion 836-7.

15. Lewen, M.K., et al., Tricuspid regurgitation by Doppler echocardiography after orthotopic cardiac transplantation. *American Journal of Cardiology*, 1987. 59(15): p. 1371-4.

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