Original Article

Efficacy of valve replacement surgery in patients with severe pulmonary hypertension

Umer Darr, Tabish Jabeen, Shakaib Chughtai
Tabba Heart Institute, Karachi, Pakistan.

Abstract

Objectives: To evaluate the safety of valve replacement surgery in rheumatic heart disease patients with severe pulmonary hypertension (SPH); defined as pulmonary artery systolic pressure (PASP ≥ 60 mmHg) on patients operated for valve replacement at Tabba Heart Institute, Karachi.

Method: From July 2005 to September 2007, total of 112 patients underwent valve replacement (AVR, MVR, AVR+MVR) at our institution. We retrospectively examined the patients with SPH.

Results: The male:female ratio was 8:16, age range 18 to 70 years. Data was entered in SPSS version 16. Student t test was used for analyzing the qualitative data and chi-square for the quantitative data.

Each case was reviewed on its merit and patient safety maintained by the cardiology and anaesthesia team.

Twenty four patients had SPH (range; 60 to 120 mmHg, mean 77.38). Fifteen underwent MVR; AVR one and 7 had AVR+MVR and one MVR + CABG. Three bioprosthetic and 21 mechanical prostheses were implanted. LVEF ranged from 47% to 75 %. Left atrium size ranged from 35 mm to 160mm. Out of 24 patients 10 patients had giant left atria (>6.5 cm). Four patients had dilated RV (range; 17mm to 31 mm).

We observed no operative mortality, one patient developed post-operative pulmonary hypertensive crisis and one developed acute renal failure. The incidence of post-op atrial fibrillation was 12.5%. All patients were NYHA class IV pre-operatively and NYHA class I or II post-operatively. There were no neurological or pulmonary complications in our series and none of the patients had re-exploration for bleeding.

Conclusion: Cardiac surgery can be successfully performed with an acceptable morbidity and very low mortality in patients with long standing valvular disease and SPH.

Keywords: Valve, Hypertension, Pulmonary, Rheumatic heart disease, Karachi (JPMA 61:893; 2011).

Introduction

Rheumatic heart disease (RHD) is the most serious complication of rheumatic fever (RF). Acute rheumatic fever follows 0.3% of cases of group A beta-haemolytic streptococcal pharyngitis.[1] As many as 39% of patients with acute rheumatic fever may develop varying degrees of pancarditis with associated valve insufficiency, heart failure, pericarditis, and even death.[2] With chronic rheumatic heart disease, patients develop valve stenosis with varying degrees of regurgitation, atrial dilation, arrhythmias, and ventricular dysfunction making it the leading cause of valve replacement in adults.[3]

Increasing pulmonary arterial systolic pressure (SPAH) is directly associated with a progressive decline in survival for patients.[4] The prognosis of patients with SPAH is variable and depends on the severity of haemodynamic derangement and the underlying primary disorder. Untreated, patients with severe pulmonary hypertension (PASP ≥ 60 mmHg) or right heart failure survive approximately 1 year.[5,6] Patients with moderate elevations in pulmonary artery pressure (mean pressure <55 mm Hg) and preserved right heart function have a median survival of 3 years from diagnosis.[7]

In many patients with chronic RHD both the mitral and aortic valves may be involved, often with mixed lesions in one or both locations.[8] In general, management should be predicated on the identification of the dominant valve lesion and location, though it is recognized that the proximal valve lesion(s) may mask the presence and significance of the more distal valve lesion(s).

This study was undertaken to evaluate the safety of valve replacement surgery in rheumatic heart disease patients.

Patients and Methods

We retrospectively analyzed the clinical records of 112 patients who underwent valve replacement at the Tabba Heart Institute from July 2005 to September 2007. All patients were referred by various cardiologists in and around Karachi. The WHO classification was used for pulmonary hypertension staging (1). Clinical stages were:

WHO Group I — Pulmonary arterial hypertension (PAH):

◆ Idiopathic (IPAH)

◆ Familial (FPAH)

◆ Associated with other diseases (APAH): collagen vascular disease (e.g. scleroderma), congenital shunts between the systemic and pulmonary circulation, portal hypertension, HIV infection, drugs, toxins, or other diseases or disorders

◆ Associated with venous or capillary disease.

WHO Group II — Pulmonary hypertension associated with left heart disease:

◆ Atrial or ventricular disease

◆ Valvular disease (e.g. mitral stenosis)

WHO Group III — Pulmonary hypertension associated with lung diseases and/or hypoxemia:

◆ Chronic obstructive pulmonary disease (COPD), interstitial lung disease (ILD)

◆ Sleep-disordered breathing, alveolar hypoventilation

◆ Chronic eposure to high altitude

◆ Developmental lung abnormalities

WHO Group IV — Pulmonary hypertension due to chronic thrombotic and/or embolic disease:

◆ Pulmonary embolism in the proximal or distal pulmonary arteries

◆ Embolization of other matter, such as tumor cells or parasites

WHO Group V — Miscellaneous:

Each case was reviewed on its own merit. Evaluation was done by the cardiology and anaesthesia team to maintain patient safety.

Statistical analysis was done by entering the data in SPSS version 16. Qualitative data was analyzed by student t test and quantitative data by the Chi Square test.

Results

Of the 112 valve replacement patients, we identified 24 with advanced rheumatic heart disease and severe pulmonary hypertension (PASP ≥ 60 mmHg). Our report focused on the safety and efficacy of valvular surgery in such patients. Patient demographics were extracted from the medical files. All patients were regularly monitored in hospital and in postoperative clinics. The variables studied were the time of diagnosis, clinical presentation, type of surgery, immediate and late complications, mortality and improvement. Data sets were not adjusted for risks.

Table-1: Echocardograpic Findings.

	PRE OP (n)	POST OP (≤ 6months)
Severity of Tricuspid Regurge		
Trace	0	1
Mild to Moderate	10	5
Severe	8	4
No Regurgitation	6	14
Left Atrial Size		
Enlarged (≤ 40mm)	20	7
Not enlarged	4	17
Left Venricular Size And Function		
Normal	22	23
Hypokinetic	1	1
Dilated	1	0
Right Atrial Size		
Normal	16	22
Dilated	8	2
Right Venticular Size		
Normal (<25mm)	18	21
Enlarged (>25mm)	6	3
Pulmonary Artery Pressures		
Severe (≥ 60mmHg)	24	4
State of Interventricular Septum		
No abnormality	18	24
Paradoxical	1	0
Flattening	5	0
Septal Thickness		
Normal (<11mm)	23	23
Abnormal (>11mm)	1	1

** Post operative echocardiographic analysis ranges from 6 months to 2 years.

Of the 24 patients with SPH (range; 60 to 120 mmHg, mean 77.38 ± 15.28 mmHg), 06 patients had hypertension (4 controlled and 2 uncontrolled), 03 had diabetes (2 controlled and 1 uncontrolled), 1 was a case of hyperthyroidism, 2 had previous MI and 1 was in chronic renal failure. Operatively 15 underwent MVR, AVR 01 and 07 had AVR+MVR and 01 MVR+CABG. Three bioprosthetic and 21 mechanical prostheses were implanted. Five of the 24 patients had undergone previous Percutaneous Transverse Mitral Commissurotomy (PTME). The time interval between PTMC and Valvular surgery ranged from a minimum of 17 years to a maximum of 21 years. Four of 24 patients also underwent tricuspid ring annuloplasty.

Patients ages ranged from 18 to 70 (mean 42.6±12.85 years). Fifteen (62.5%) were more than 40 years old and nine (37.5%) were younger than 40 years of age. Sixteen (66.6%) patients were women. The left ventricular ejection fraction (LVEF) ranged from 47 % to 75% (mean 59.29 and standard deviation 9.02). Left atrium size ranged from 35 mm to 160mm (mean 56.66 and standard deviation 18). Out of 24 patients 10 had giant left atria (>6.5 cm). Four patients had dilated RV (range; 17mm to 31 mm).

Table-2: Mortality and morbidity.

Complications	No. of patients (n)		
	Month 6	Year 1	Year 2
Congestive heart failure	1	0	0
Atrial fibrillation	2	1	0
Digoxin toxicity	0	1	0
Brady arrest	1	0	0
Sepsis	2	0	0
Pericardial effusion	0	1	0
Renal failure	1	0	0
Prosthetic valve endocarditis	1	0	0

No post-operative mortality was observed till 2 years.

Reviewing each individual's echocardiographic finding, the severity of tricuspid regurge was no regurge in 6 patients, trace in 0, mild to moderate in 10 and severe in 8, this changed to no regurge in 14 patients, trace in 1, mild to moderate in 5 and severe in 4. Left atrial (LA) size was enlarged (≤ 40mm) in 20 of 24 individuals while post operatively only 7 patients had enlarged LA. Regarding left ventricular (LV) size and function 22 patients had normal LV size and function pre operatively while 1 had hypo kinetic and 1 dilated LV, post op that changed to 23 with normal size and function while 1 remained hypo kinetic. Eight of 24 patients had dilated left atria while 6 had dilated right ventricle pre op, that changed to 2 and 3 respectively. Of the 24 patients with severe pulmonary artery pressures (≥ 60mmHg) only 4 demonstrated pressures more than 60 mmhg post operatively. The state of septal thickness (23 normal [<11mm] and 1 abnormal [>11mm]) pre and post operatively remained constant.

No operative mortality was encountered, regarding operatively morbidity (less than 1 month after the operation) one (4.16%) patient developed an acute pulmonary hypertensive crisis and one (4.16%) went into acute renal failure. The incidence of atrial fibrillation was 12.5% risk factors being advanced age, mitral stenosis, and left atrial enlargement. All the patients were NYHA class III or IV pre-operatively and improved to either NYHA class I or II post-operatively. None of the patients underwent neurological or pulmonary complications. The mean duration of stay for patients in the hospital was 8.7 days. It was found to be higher in patients aged 40 or above (9.4 days) than in patients less than 40 (7.6 days).

All patients were followed up to 2 years. Of these 24, presented with congestive heart failure, 3 atrial fibrillation, and 1 digoxin toxicity. 1 patient developed Brady arrest, 2 went into sepsis, 1 pericardial effusion, 1 renal failure and developed prosthetic valve endocarditis.

Discussion

Delaying an operation for valve replacement in advanced rheumatic heart disease can lead to fatal outcomes. When surgery is postponed, increased pulmonary artery pressure contributes to an increase in right sided pressures unfavourably affecting mortality and morbidity rates.[9] Therefore optimal timing is crucial for reducing mortality rates in candidates for valve replacement.

Despite a documented decline in the incidence of RHD in industrialized countries during the past three decades, no such decrease could be appreciated in the developing countries. Retrospective studies have documented RF incidence rates as high as 206/100 000 and RHD prevalence rates as high as 18.6/1000 in the developing world, necessitating aggressive control measures and management.[10]

Initially, heart failure in RHD generally responds to bed rest and steroids, but in patients with severe symptoms, diuretics, angiotensin converting enzyme inhibitors and digoxin may be used.[11] When heart failure persists or worsens after aggressive medical therapy, particularly in patients with advanced rheumatic valvular heart disease, surgery to decrease valve insufficiency may be life-saving.[12]

In general terms, the necessity for surgical treatment is determined by the severity of the patient's symptoms and evidence that cardiac function is significantly impaired.[13] It is particularly important to prevent irreversible damage to the left ventricle and irreversible pulmonary hypertension, since both considerably increase the risk of surgical treatment, impair long-term results and render surgery as an elevated risk.[14]

It is important to emphasize that medical management of chronic rheumatic heart disease must defer to operative intervention when clinical or echocardiographic criteria are met, and when surgery is both accessible and feasible. In many cases, the development of heart failure, particularly when attributable to left ventricular systolic dysfunction, implies that surgery has been inappropriately delayed.[15]

Nowadays it is no longer necessary or even advisable to delay surgery until advanced symptoms are present, and thus surgery is timed earlier today than it was even a decade ago. On the other hand, many but not all patients with far advanced disease, once considered inoperable, are now often helped substantially by valve surgery. However, selection of which of these very ill patients will or will not benefit from valve surgery

remains a challenge.[16]

Generally patients with enlarged hearts, especially with advanced pulmonary hypertension are regarded as high risk candidates for open heart surgery.[17] Appropriate pre-op medical optimization and patient selection, play a large role in improving outcomes. The treatment algorithm for this subset of patients must be individualized.

References

1. Infectious disease emergencies. Part II: patients presenting with cardiac decompensation or circulatory insufficiency (shock). West J Med 1976; 125: 119-36.
2. Chin TK, Chin EM, Siddiqui T, Sundell AK. Rheumatic Heart Disease. (Online) (Cited 2006 May 19). Available from URL: http://.emedicine.medscape.com/article/891897-overview.
3. Roberts WC, Farooq H. Facts and principles learned at the 32nd annual Williamsburg Conference on Heart Disease. Proc (Bayl Univ Med Cent) 2005; 18: 141-56.
4. Sharma S. Pulmonary Hypertension Secondary. (Online) (Cited 2010 Jun 1). Available from URL: http://emedicine.medscape.com/article/303098-overview.
5. Karki DB, Neopane A, Regmi S. An update on pulmonary arterial hypertension; Kathmandu Univ Med J 2007; 5: 574-82.
6. Raiesdana A, Loscalzo J. Pulmonary arterial hypertension. Ann Med 2006; 38: 95-110.
7. Thenappan T, Shah SJ, Rich S, Maitland MG. A USA-based registry for pulmonary arterial hypertension: 1982-2006. Eur Respir J 2007; 30: 1103-10.
8. Rheumatic Fever and Rheumatic Heart Disease: Report of a WHO Expert Consultation, 2001 Oct 29 - Nov 1, Geneva, Switzerland: World Health Organization, 2004; pp 64.
9. Giant Left Atrium with Rheumatic Mitral Stenosis; Ates M, Sensoz Y, Abay G, Akcar M. Tex Heart Inst J 2006; 33: 389-91.
10. Rheumatic heart disease in the developing world: prevalence, prevention, and control EISENBERG. Eur Heart J 1993; 14: 122-8.
11. Rheumatic fever and rheumatic heart disease; Report of a WHO Expert Consultation Geneva, 29 October-1 November 2001; World Health Organization, Geneva 2004; pp 70-1.
12. Dr. Jeyamalar Rajadurai. Clinical Practice Guidelines on heart failure. (Online) (Cited 2007 April 4). Available from URL: http://www.acadmed.org.my/cpg/heart_failure.doc.
13. Rheumatic Fever and Rheumatic Heart Disease: Report of a WHO Expert Consultation, 2001 Oct 29 - Nov 1, Geneva, Switzerland: World Health Organization, 2004; pp 70-1.
14. Carl VL. A heart failure specialist's perspective on cardiac surgery for heart failure. Curr Heart Failure Rep 2005; 2: 46-53.
15. Rheumatic Fever and Rheumatic Heart Disease: Report of a WHO Expert Consultation, Geneva, 2001 Oct 29 - Nov 1, World Health Organization, 2004; pp 56.
16. Carabello BA, Is it ever too late to operate on the patient with valvular heart disease? J Am Coll Cardiol 2004; 44: 376-83.
17. Other Techniques in Special Circumstances: Pulmonary Thromboendarterectomy in Right Heart Failure. Book title: RNA-Protein Interaction Protocols, Publisher: Springer London, 2008; pp 251-61.