ORIGINAL ARTICLE

The impact of severe pulmonary hypertension on the outcome of mitral valve surgery and its regression after surgery

Muhammad Sohail Chaudhri [1], Faisal Ali[2], Asif Rashid Alamgir[3], Irfan Rasheed[1], Azka Usman[1], Zulfiqar Haider[1]

[1] Department of Cardiac Surgery, Faisalabad Institute of Cardiology, Faisalabad, Pakistan
[2] Department of Cardiology, Faisalabad Institute of Cardiology, Faisalabad, Pakistan
[3] Department of Anesthesia, Faisalabad Institute of Cardiology, Faisalabad, Pakistan

Received: 4-December-2017.
Accepted 18-December-2017

Correspondence:
Muhammad Sohail Ch. Assistant Professor of Cardiac Surgery, Faisalabad Institute of Cardiology, Serena Road, Faisalabad, Pakistan. Email: msohaildr@yahoo.com | Tel: +923215236076 | Fax: +92419201518

ABSTRACT

Objective

To find out if severe pulmonary hypertension is a major contributory factor to in-hospital mortality and its regression after isolated mitral valve.

Methods

This retrospective observational study was conducted at cardiac surgery department, Faisalabad Institute of Cardiology, Faisalabad from May 2016 to July 2017. Forty six patients who fulfilled the inclusion criteria were included in the study. Mitral valve replacement was performed with a mechanical or bioprosthetic valve as indicated. Preoperative and postoperative echocardiography was done to determine the severity of pulmonary hypertension (PTH). Data consisting of preoperative and postoperative variables was recorded in Excel spreadsheet.

Results

Mean age of the patients was 33.8 years. Isolated mitral valve replacement was performed in 27 patients (58.7%), while 19 patients (41.3%) underwent mitral valve replacement along with tricuspid valve repair. There was no perioperative mortality and all patients were successfully discharged home. The change in pre-operative and post operative pulmonary artery pressures was analyzed in 31 patients where both pressure were available. Mean preoperative pulmonary artery pressure was 77mmHg and mean postoperative pulmonary artery pressure was 36.74 mmHg. A paired sample t-test was used to find out the significance of change in pressure which had a p-value of <0.0001 making it highly significant.

Conclusions

Mitral valve replacement in the presence of severe PHT is a safe procedure. Significant reduction of PHT postoperatively strongly justifies surgery in such cases.

Key Words:
Mitral Valve, Mitral Stenosis, Pulmonary hypertension, Mitral Valve Replacement, Mitral Valve Repair, Rheumatic Heart Disease

INTRODUCTION

Pulmonary hypertension (PHT) has been reported in 78% patients of rheumatic mitral valve disease and often has its impact on left atrial pressure as well as remodeling of pulmonary vascular resistance [1]. The effect of pulmonary venous hypertension on pulmonary circulation is, however, highly variable ranging from no effect to severe pulmonary vascular resistance [2]. Long-standing PHT increases after-load on the right ventricle (RV) leading to hypertrophy and eventually cor-pulmonale. Long standing right ventricular failure is associated with dilatation of right ventricle and tricuspid regurgitation which in turn exacerbates the RV dysfunction [3].

In the early 1950's cardiac catheterization and histological investigations characterized the physiology and microvascular changes provoked by PVH. The PH is likely to resolve to some degree with the relief of left atrial hypertension even in the presence of long-standing and severely increased pulmonary vascular resistance [1,4]. However, residual or persistent PHT after Mitral valve replacement or repair may give rise to symptoms of exertional dyspnea, dizziness, chest pain and pedal edema [5]. These clinical features have a striking similarity with those of mitral valve disease and may adversely affect the quality of life of a patient.

The rheumatic heart disease is quite prevalent in our population despite its lower incidence in developed countries. It usually affects the younger age group and is by far the most common reason for mitral valve replacement or repair. Mitral stenosis is more common than mitral regurgitation and both can present with pulmonary hypertension due to long standing disease [6].

Severe PTH has been implicated as a risk factor for increased operative mortality rates in patients undergoing mitral valve replacement or repair. The fears of high predicted mortality, poor regression of pulmonary hypertension and persistence of symptoms after successful mitral valve surgery have always raised questions of the suitability surgical treatment. Therefore, any evidence contrary to these fears may favorably influence the surgeons in their decision making. Resultantly many patients my get a dramatic improvement in the quality of their life [7] . We conducted this study to assess effect of PHT on operative mortality and its pattern of the regression after mitral valve surgery.

METHODS

It is a retrospective Observational Study conducted at Faisalabad Institute of Cardiology, Faisalabad. The study period extended over 14 months starting from May 2016 to July 2017. All patients who underwent mitral valve surgery with or without tricuspid valve repair and had severe pre-operative pulmonary hypertension (i.e. systolic PAP >60mm Hg) were included in the study. Patients undergoing redo surgery or requiring concomitant coronary bypass, aortic valve surgery of congenital heart surgery were excluded from the study.

The operations were performed by consultant surgeons with more than 2 years experience of working independently. All operations were done through median sternotomy using standard protocol of cardiopulmonary bypass. Intermittent cold blood cardioplegia was used along with systemic hypothermia at 320C. Mitral valve replacement was performed with a mechanical or bioprosthetic valve using either continuous or interrupted sutures. Tricuspid valve repair was always augmented with an annuloplasty ring.

The Pre-operative and post-operative echocardiography was used to determine the severity of PHT. For echocardiography Vivid E9 (GE) machine was used, with M5Sc probe which operates at 1.5-4.6 MHz. To quantify pulmonary hypertension with echocardiography, the maximal Tricuspid Valve Regurgitation velocity was measured with CW Doppler. To derive Pulmonary artery pressure from TR signal, following equation was used:

$$\text{Systolic Pulmonary Artery Pressure} = 4 \times (\text{TR Velocity})^2 + \text{RAP}$$

A systematic search of our dedicated Electronic database (Cascade Databases Lahore, Pakistan) identified Forty six (46) patients who fulfilled the above mentioned criteria. The data consisting of pre-operative, and post-operative variables was recorded in Excel spreadsheet. The data was summarized using mean $\pm$ standard deviation for the numeric variables and frequency and percentages for categoric variables. The comparison of preoperative and postoperative hypertension was done using paired sample t-test and p-values were determined. A p-value of less than 0.05 was considered significant.

RESULTS

Majority of the patients were below the age of 35 years (mean=33.8, SD=11.992). Eleven out of 46 (23.9%) patients were male and the remaining 35 (76.1%) were female. The mean body weight was 51.61 kg (SD=14.794) and mean BMI was 21.75 (SD=5.89). The mean pre-operative left ventricular ejection fraction of the patients was 58.67 (SD=5.816) with the minimum being 40% in 1 patient. All patients had good right ventricular function preoperatively. Thirty-three (71.7%) patients had severe mitral stenosis while 13 patients (28.3%) had severe mitral regurgitation. Isolated mitral valve replacement was performed in 27 patients (58.7%), while 19 patients (41.3%) underwent mitral valve replacement along with tricuspid valve repair. Mean cardiopulmonary bypass and aortic cross-clamp times were 89.87 (SD=23.15) and 62.48 (SD+18.746) minutes respectively. Mean ventilation time was 7.13 (SD=5.647) hours while the mean inotropic requirement was 30.22(SD=23.12) hours. There was no peri-operative mortality and all patients were successfully discharged home. Mean preoperative PHT was 77mm Hg (SD+20.203). Post-operative PHT was 36.74 (SD+8.805) in 31 patients. In 10 patients there was no TR so PHT could not be calculated while record of postoperative echocardiography was not available in 5 patients.

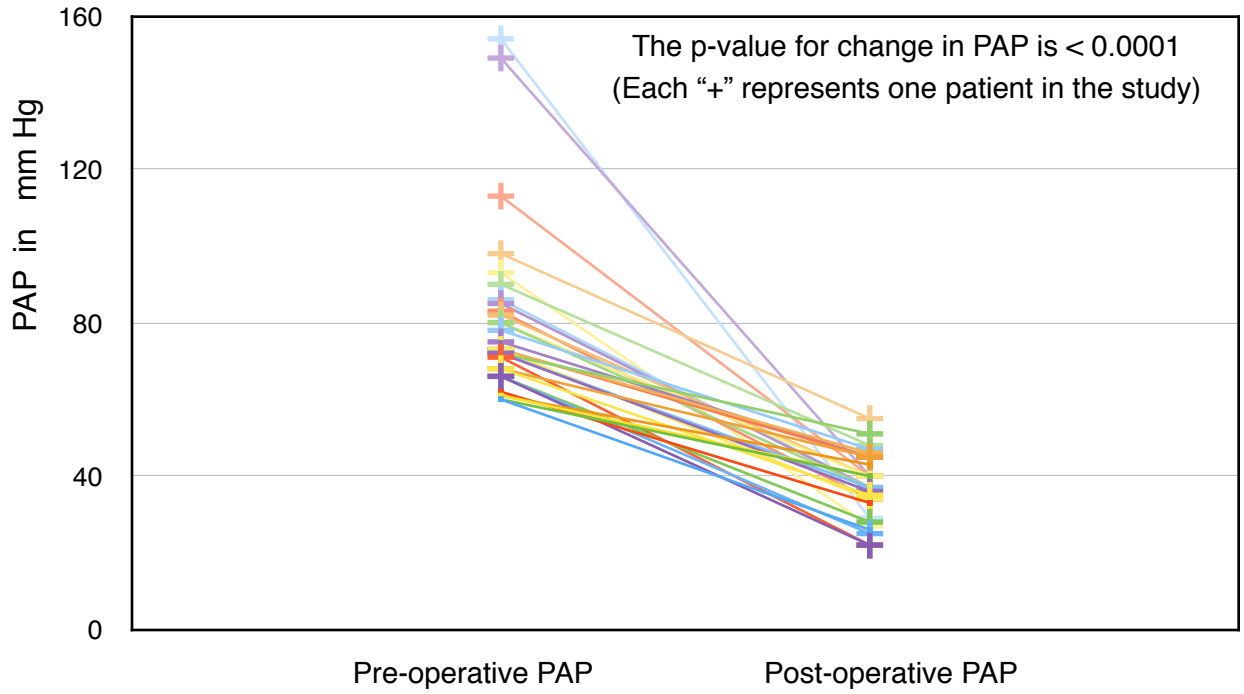
DISCUSSION

Surgery of the mitral valve is the commonest valve surgery being performed by cardiac surgeons in our country. The PH has been a part and parcel of the disease. The mechanism by which PH develops in patients with MVD is driven by an increase of LA pressure, which leads to pulmonary venous hypertension, and subsequently pulmonary arterial hypertension. With mitral stenosis (MS), there are two well-characterized associated hemodynamic states. Initially, with progression of MS, the mitral valve area (MVA) and cardiac output decrease with a concomitant rise in the LA pressure. Later as disease progresses, with a continued reduction in MVA and elevation of LA pressures, changes in the pulmonary bed occcur, increasing the pulmonary vascular resistance (PVR) and PH. Finally overt right heart failure occurs from right ventricular pressure overload. The major symptom of PH is dyspnea on exertion [1]. Other symptoms include fatigue, weakness, angina, syncope, and abdominal distention. Symptoms at rest are reported only in very advanced cases [8].

The increase in pulmonary arterial pressure is often out of proportion to the degree of left atrial hypertension, reflecting a major increase in pulmonary vascular resistance [9]. In mitral stenosis, the small pulmonary arteries exhibit distal extension of smooth muscle, medial hypertrophy and fibrosis, and adventitial thickening. Reports have also described substantial intimal thickening and even fibrosis, including in young patients. The small veins usually have thickened and fibrotic media, intimal fibrosis and may be arterialized. The capillaries are engorged and possess thickened basement membranes. The lymphatics are dilated and thick walled than normal [2]. Surgical decompression of the left atrium through mitral commissurotomy or mitral valve replacement has often yielded marked regression of pulmonary hypertension. Similar decrease in PVR was reported after percutaneous trans-mitral commissurotomy (PTMC). Levine and co-workers demonstrated an immediate decrease in PVR from 630±570 to 447±324 dynes-sec/cm5 in 14 patients who underwent PTMC. Repeat catheterization 7±4 months after procedure showed further improvement of pulmonary hypertension in 12 of the 14 patients, with a mean pulmonary vascular resistance for the group as 280±183 dynes-sec/cm [9,10]. The acute hemodynamic response to bileaflet mechanical MVR was reported in 60 patients with PH from the modern era. Evaluation of 30 patients with severe PH (mean PA pressure of 54 mm Hg) showed that mean PA pressure fell to 23 mm Hg at 48 hours postoperatively [1]. Our study has also demonstrated significant reduction in the PH to less than half of the preoperative levels. For the 10 patients in whom PH could not be calculated due to absence of tricuspid regurgitation, we believe that lack of tricuspid regurgitation in itself suggests a significant decrease in the PH.

Without relief of mitral valve obstruction, observational data from several decades ago demonstrated that mean survival was 2.4 years when severe PH was present with severe MS. Mitral valve repair and replacement have dramatically improved the outlook in patients with mitral valve disease and severe PH. Results from the early days of cardiac surgery carried significant mortality. Nichols et al. (1964) reported surgical mortality of 23 percent for all

FIGURE 1



types of mitral valve surgery in those with a pulmonary arterial pressure greater than 50 mmHg [4]. This still represents a major improvement in survival and quality of life as compared to patients in the medically treated group. However, owing to advancements in postoperative care, results have steadily improved. Ghoreishi (2011) reported operative mortality of 12% in 148 patients with severe PH undergoing MV surgery for mitral regurgitation [11]. Other studies have also reported similar results. In 475 patients with severe PH undergoing MV surgery, Enter et al (2016) have shown a 30-day mortality of 4.6% with 5-year survival of 86.2% in patients with severe PH and 75.4% in patients with extreme PH (pulmonary artery systolic pressure > 80 mm Hg). They concluded that mortality in MV surgery is unaffected by severe PH, but extreme PH remains a risk factor. The 30-day mortality risk effect of PH has been included in the EuroSCORE (European System for Cardiac Operative Risk Evaluation) II model [3,12]. Our study is different in this regard from the above mentioned studies as there was no mortality in our study population, even in 14 patients with extreme PH. We believe the main reason for this difference in mortality is the mean age, which was 33 years in our study group and nearly 60 years or more in the other studies.

Severe PH (and especially extreme PH) has been considered a relative, if not absolute, contraindication to mitral valve surgery particularly in those requiring mitral valve replacement. The findings presented here suggest that the reverse should be the case. Extreme pulmonary hypertension should be regarded as a positive indication for early operation in many of these patients. Mitral valve surgery performed at any time during follow-up is safe and independently associated with a reduced risk of total mortality and CVD in every subset of patients [12]. The most recent ACC/AHA management guidelines also recommend surgical intervention for mitral valve in asymptomatic patients who have a resting PA systolic pressure greater than 50 mm Hg, or a PA systolic pressure greater than 60 mm Hg with exercise.

CONCLUSION

The mitral valve surgery can be performed safely in patients with severe pulmonary hypertension. There is significant regression of pulmonary hypertension after mitral valve surgery. We are justified to conclude that no

patient should be refused mitral valve surgery because of extreme pulmonary hypertension.

ACKNOWLEDGMENT

The authors acknowledge the guidance and support of Professor Anjum Jalal in preparation of manuscript and its statistical analysis.

REFERENCES

1. Kiefer TL, Bashore TM. Pulmonary hypertension related to left-sided cardiac pathology. Pulm Med 2011;2011:381787 ! doi: 10.1155/2011/381787
2. Kulik TJ. Pulmonary blood flow and pulmonary hypertension: Is the pulmonary circulation flowophobic or flowophilic? Pulm Circ 2012; 2: 327–339 doi: 10.1086/678471
3. Enter DH, Zaki A, Duncan BF, Kruse J. A contemporary analysis of pulmonary hypertension in patients undergoing mitral valve surgery:Is this a risk factor? J Thorac and Cardiovasc Surg 2016;151:1288-99 http://dx.doi.org/10.1016/j.jtcvs.2015.12.063
4. Ward C, Hancock BW. Extreme pulmonary hypertension caused by mitral valve disease Natural history and results of surgery. Br Heart J 1975;37:74-78
5. Fuster V, Steele PM, Edwards WD, Gersh BJ, McGoon MD. Primary pulmonary hypertension: natural history and the importance of thrombosis. Circulation 1984;4:580-87
6. Scott WC, Miller DC, Haverich A, Mitchell RS, Oyer PE, Stinson EB, Jamieson SW, Baldwin JC, Shumway NE. Operative risk of mitral valve replacement: discriminant analysis of 1329 procedures. Circulation. 1985;72:108-19
7. Cevese PG, Gallucci V, Valfre C, Giacomin A, Mazzucco A, Casarotto D. Pulmonary hypertension in mitral valve surgery. J Cardiovasc Surg 1980;21:7-10
8. Badesch DB, Champion HC, Sanchez MA. Diagnosis and assessment of pulmonary arterial hypertension. J Am Coll Cardiol 2009; 54:S55-66 doi: 10.1016/j.jacc. 2009.04.011
9. Levine MJ, Weinstein JS, Diver DJ, Berman AD, Wyman M, Cunningham MJ. Progressive Improvement in Pulmonary Vascular Resistance After Percutaneous Mitral Valvuloplasty. Circulation 1981;79:1061-67
10. Zener JC, Hancock EW, Shumway NE, Harrison DC. Regression of extreme pulmonary hypertension after mitral valve surgery. Am J Cardiol 1972;30:820-826
11. Ghoreishi M, Evans CF, DeFilippi CR, Hobbs G, Young CA, Griffith BP. Pulmonary hypertension adversely affects short- and long-term survival after mitral valve operation for mitral regurgitation: implications for timing of surgery. J Thorac Cardiovasc Surg 2011;142:1439-52. doi: http://dx.doi.org/10.1016/j.jtcvs.2011.08.030
12. Barbieri A, Grigioni F, Tribouilloy C, Avierinos JF. Prognostic and therapeutic implications of pulmonary hypertension complicating degenerative mitral regurgitation due to flail leaflet: A Multicenter Long-term International study. Eur Heart J 2011;32:751-9. doi:10.1093/eurheartj/ehq294

Conflict of Interest: None declared
Source of Funding: Nil
Ethical Approval: From Ethical Review Committee of Faisalabad Institute of Cardiology

Cite this article as:

Chaudhri MS, Rasheed I, Alamgir AR. The impact of severe pulmonary hypertension on the outcome of mitral valve surgery and its regression after surgery
Pakistan Journal of Cardiovascular & Thoracic Surgery 2018;13(1):5-9