e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2022; 14(11); 741-750

Original Research Article

Effects of Preoperative Pulmonary Arterial Hypertension on Patients Receiving Valve Surgery for Rheumatic Heart Disease

Pragateshnu Das

Assistant Professor, Neurology Department, KIMS Bhubaneswar

Received: 22-09-2022 / Revised: 13-10-2022 / Accepted: 29-11-2022
Corresponding author: Dr. Pragateshnu Das
Conflict of interest: Nil

Abstract

Background: Ankle fracture fixation postoperative pain has a significant impact on both the **Background and Aims:** The data on the negative effects of pulmonary arterial hypertension (PAH) on outcomes following cardiac surgery for rheumatic heart disease is mixed (RHD). The researchers looked at Indian individuals with RHD and preoperative PAH who underwent cardiac surgery to see if they had poor short- and long-term results.

Methods: This was a 407-patient retrospective observational study. The patients were classified as having no or mild PAH (pulmonary artery systolic pressure (PASP) <30 mm of Hg), moderate PAH (PASP 31-55 mm of Hg), or severe PAH (PASP >55 mm of Hg) based on PAH as measured by echocardiography. In-hospital mortality and significant morbidities were the primary endpoints, whereas long-term survival was the secondary objective.

Results: In-hospital mortality was 24.9%, and there was no difference between patients with severe (9.1%), moderate (4.5%), or mild PAH (2.8%) (P = 0.09). Prolonged breathing was more common in patients with severe PAH (P = 0.007). >2-packed cell transfusion, extended breathing, and acute renal injury were all independently linked to mortality, but not moderate or severe PAH. On long-term follow-up [81.37 percent (mean duration 19.40±14.10 months)], patients with MS and severe PAH had significantly higher mortality (P = 0.03) than those with no or moderate PAH (P = 0.03). Mortality was 8% and not statistically different (P = 0.25) across PAH categories.

Conclusion: Patients receiving valve surgery for RHD with moderate or severe PAH have similar short and long-term outcomes. Patients with MS who had severe PAH died sooner than those who did not have PAH.

Keywords: Indian, pulmonary hypertension, rheumatic heart disease

Introduction

Rheumatic heart disease (RHD) is a serious health problem in poor nations, where it accounts for the majority of cardiovascular morbidity and mortality in young people, with an estimated 250,000 fatalities each year. [1] Symptomatic patients with left-sided valvular abnormalities are frequently reported to have pulmonary arterial hypertension (PAH). The European System for Cardiac Operative Risk Evaluation II (EuroSCORE II), one of the most widely used scoring systems for predicting mortality after cardiac surgery, identifies

moderate [pulmonary artery systolic pressure (PASP) 31–55 mmHg] and severe [PASP more than 55 mmHg] PAH as risk factors for mortality. [2]

In comparison to prior research, recent trials have shown improved outcomes in patients with severe PAH, with lower perioperative mortality rates. [3] The evidence on this topic appears to be mixed, with some research identifying PAH as an independent risk factor and others concluding that severe PAH has no effect on mortality. There are very few research on this vital topic in India. [4,5]

The authors describe how preoperative PAH affects short- and long-term results in patients undergoing mitral ± aortic valve surgery for RHD.

Methods

This was a retrospective examination of observational data. Adult patients who had four years of consecutive cardiac surgery at our institute for RHD were considered eligible for the study. Isolated aortic valve replacement was ruled out because it has a completely different pathophysiology than PAH, as did the lack of preoperative PAH data and incomplete records.

In-hospital mortality was defined as all-cause mortality in the same admission of cardiac surgery, as well as major morbidities such as re-exploration for bleeding, prolonged ventilator support (for more than 24 h), cardiac surgery-related acute kidney injury (CSAKI) (defined as a 50 percent increase or doubling of preoperative creatinine during 7 days or the need for renal replacement therapy), and neurologic deficit of more than 24 h. Long-term survival was the secondary goal.

Demographic and procedural data were abstracted from medical records. It included age, sex and pre-existing comorbidities, including, anaemia as per world health oragnisation (WHO) definition, New York Heart Association (NYHA) functional classification, atrial fibrillation (AF), chronic obstructive pulmonary disease (COPD) that was defined as per X-ray and clinical findings, diabetes mellitus, renal disease (abnormal creatinine level >1.5 mg/dL and/or reduced creatinine clearance <50 ml/min), previous cerebrovascular accident (CVA) as defined as neuro-deficit of >24 h, peripheral vascular disease (PVD) and previous cardiac surgery. The primary predominant lesion and associated tricuspid regurgitation (TR) with its severity were also noted.

The American Society of Echocardiography's guidelines for preoperative echocardiography imaging were followed. [6] Doppler echocardiography was used to calculate the right ventricular to right atrial pressure gradient during systole, which was approximated by the modified Bernoulli equation as 4v2, where v represents the tricuspid regurgitation jet velocity in m/s. The patients were classified into three groups based on PASP, which was calculated using the most recent echocardiography before surgery and was defined as (1) no or mild PAH (PASP less than 30 mmHg), (2) moderate PAH (PASP 31–55 mmHg), and (3) severe PAH (PASP greater than 55 mmHg) (PASP more than 55 mmHg). These cut-offs were determined using the EuroSCORE II system's definitions. [2] No right heart catheterization (RHC) or pulmonary vascular resistance (PVR) measurements were performed on any of the patients.

In addition to standard American Society of Anesthesiology Guidelines, intraoperative monitoring included femoral arterial catheter-based arterial blood pressure monitoring, arterial blood gas monitoring, central venous pressure monitoring, and temperature monitoring at two sites: the nasopharynx and the skin. Midazolam, fentanyl, and propofol were used to induce general anaesthesia. Vecuronium was used to relax muscles. All patients received a 10-mg/kg bolus of

intravenous tranexamic acid after induction. For anaesthetic maintenance, titrated doses of sevoflurane or isoflurane, as well as titrated boluses of fentanyl and midazolam, were employed according to haemodynamic parameters. All of the patients had surgery via a median sternotomy. After appropriate heparinisation (ACT >400 seconds), cardiopulmonary bypass (CPB) was achieved with aortic and bicaval cannulation. During CPB, moderate hypothermia (32–34°C) was maintained. Membrane oxygenator, roller pump, tubing, and crystalloid prime solution made up the extracorporeal circuit. To achieve cardiac arrest and give myocardial protection during aortic clamping, blood cardioplegia was used. Protamine reversal was given at the end of CPB to restore normal ACT. The intensive care unit received all of the patients. Patients were extubated according to hospital guidelines. CPB duration, aortic cross-clamping time (ACC), concurrent procedure, and prosthetic valve selection were all reported as operational parameters.

Long-term follow-up data was extracted from hospital records; if the records were not available, patients were contacted by phone. Mortality and readmission data were gathered for cardiac (prosthetic-valve thrombosis, congestive heart failure, arrhythmias causing substantial haemodynamic disturbances, etc.) and non-cardiac (anticoagulation-related bleeding, neurological problems, respiratory reasons, etc.) causes.

According to the EuroSCORE II system, patients were divided into three categories based on the severity of their PAH. The mean and standard deviation of continuous variables were calculated and compared between PAH groups using analysis of variance. The percentages of categorical data were compared using the Chi-square test. The median was used to characterise ordinal data (interquartile range, range). To find factors linked to mortality, researchers conducted univariate and multivariate regression analysis. Crude survival curves were estimated using the nonparametric Kaplan–Meier method. The log-rank test was used to compare survival among groups. All P values <0.05 were considered as significant. The Statistical Package for Social Sciences (SPSS) version 16.0.0 for Windows (SPSS Inc., Chicago, IL, USA) was used for analysis.

Results

During the research period, 482 patients underwent surgery. Patients with isolated aortic valve replacement (42; 8.7%), non-availability of preoperative PAH data (23; 4.8 percent), and incomplete records were all removed (20; 4.2 percent). 407 patients were included in the final analysis. A total of 278 patients had isolated mitral valve replacement (MVR), 50 had MVR + tricuspid valve repair (TV rep), 79 had double valve replacement (DVR), and 7 had DVR + TV rep.

Table 1 summarises the patients' baseline clinical characteristics. The cohort had 32.7 percent (133/407) and 40.5 percent (165/407) preoperative moderate and severe PAH, respectively. Except for the fact that there were more patients with severe PAH in the NYHA III and IV classes, more patients with severe PAH had severe TR, and fewer patients in the severe PAH category underwent simultaneous aortic valve surgery, the groups had similar demographics and comorbidities. In the moderate and severe PAH groups, Euro SCORE II was considerably higher. Table 1 shows that a considerably higher number of patients in the severe PAH category had TV repair procedures, despite the fact that overall CPB and ACC durations were similar in all categories.

Post-operative outcomes are summarised in Table 2. Overall incidence of in-hospital mortality was 5.9% (n = 24), although the incidence of mortality was

higher in severe PAH patients (9.1%; *n* = 15) as compared to patients with moderate PAH (4.5%; *n* = 6) and no or mild PAH (2.8%; *n* = 3), but it was not statistically significant(*P*=0.09). As Far as Postoperative Morbidities are concerned, patients with severe PAH had higher incidence of prolonged ventilation (*P* = 0.007) but incidence of other morbidities such as re-exploration for bleeding (*P* = 0.97), CSAKI (*P* = 0.53) and neurologic deficit (*P* = 0.9) was similar in all categories of PAH. Results of univariate and multiple regression analysis for mortality are shown in Table 3. Factors independently associated with mortality were more than two-packed cell transfusion, prolonged ventilation and CSAKI but not moderate (*P* = 0.37) and severe PAH (*P* = 0.43). When we compared mortality rates as per predominant lesion [mitral stenosis (MS) compared with mitral regurgitation (MR)] and PAH category, we found that patients with MS and severe PAH had significantly higher mortality as compared to no or mild PAH (*P* = 0.03); mortality rates were similar in all categories of PAH in patients with MR as predominant lesion (*P* = 0.98) [Table 4].

The long-term follow-up was available in 81.37% patients. Mean long-term follow-up was 19.40 ± 14.10 months. The total patient-years of follow-up were 350 patient-years. The readmission rates for both cardiac and non-cardiac reasons were similar across all PAH categories (*P* = 0.88). The overall mortality of the entire cohort over the follow-up period was 8% and not statistically different (*P* = 0.25) in patients with no or mild PAH (8%), moderate PAH (11%) and severe PAH (6%), as shown in Table 5. Also, long-term survival was not significantly different in the moderate and severe PAH groups compared with the normal and mild PAH groups using the Kaplan–Meier curve analysis (Chi-square value 2.47; *P* = 0.29, log-rank test) [Figure 1].

Discussion

The study's major findings are that individuals with moderate or severe PAH who have cardiac surgery for RHD have equal short- and long-term mortality and morbidity as those who had no or mild PAH. Prolonged post-operative ventilation is more common in patients with severe PAH. Patients with MS as the primary lesion and severe PAH have significantly higher mortality (P = 0.03) than patients with no or mild PAH; death rates in patients with MR as the primary lesion were identical in all categories of PAH (P = 0.98).

Table 1: Preoperative characteristics and intra-operative variables

	Total (*n*=407)	**None or mild PAH (*n*=109; 26.8%)**	**Moderate PAH (*n*=133; 32.7%)**	**Severe PAH (*n*=165; 40.5%)**	***P***
Preoperative characteristics					
Age (mean±SD) (years)	38±12	38±13	39±12	38±12	0.85
Sex (female %)	231 (56.8%)	56 (51.4%)	82 (62.1%)	93 (56.4%)	0.24
Height (mean±SD) (cm)	158±9	159±9	158±9	159±9	0.78
Weight (mean±SD) (kg)	48±10	48±11	48±10	47±10	0.37
Haemoglobin (mean±SD) (g%)	12.6±1.7	12.7±1.7	12.3±1.5	12.7±1.9	0.09
Anaemia (yes) (%)	192 (47.2%)	47 (44%)	72 (54%)	73 (44%)	0.17
Creatinine (mean±SD) (mg%)	0.96±0.2	0.96±0.25	0.92±0.2	0.99±0.3	0.09
COPD (%)	8 (2%)	2 (1.9%)	1 (0.8%)	5 (3%)	0.37
DM (%)	7 (1.7%)	2 (1.9%)	3 (2.3%)	2 (1.2%)	0.78
CVA (%)	20 (4.9%)	6 (5.6%)	4 (3%)	10 (6.1%)	0.45
PVD (%)	2 (0.5%)	1 (0.9%)	0	1 (0.6%)	0.57
Redo surgery	31 (7.8%)	5 (4.7%)	11 (8.3%)	15 (9.1%)	0.39

EF (mean±SD) (%)	60±7	60±7	60±8	61±6	0.77
NYHA					0.003
II (%)	115 (28%)	41 (37.6%)	29 (22%)	33 (20%)	
III	279 (67%)	41 (37.6%)	63 (47%)	109 (66%)	
IV	13 (3.2%)	2 (2%)	6 (5%)	5 (3%)	
AF (%)	201 (49%)	46 (42%)	78 (59%)	77 (47%)	0.02
Primary lesion					0.01
MS (%)	301 (74%)	70 (64%)	99 (75%)	132 (80%)	
MR (%)	106 (26%)	39 (36%)	34 (25%)	33 (20%)	
More than mild TR	59 (14.5%)	8 (7.3%)	17 (13%)	34 (20.6%)	0.001
Concomitant aortic valve surgery (%)	79 (19.4%)	24 (22%)	37 (28%)	18 (11%)	0.00
EuroSCORE II	2.2±1.3	1.9±1.1	2.2±1.1	2.4±1.5	0.002
Intraoperative variables					0.000
Surgery	278 (68.3%)	81 (74.3%)	82 (61.7%)	115 (69.7%)	
MVR	50 (12.3%)	4 (3.7%)	14 (10.5%)	32 (19.4%)	
MVR+TV repair	72 (17.7%)	22 (20.2%)	34 (25.6%)	16 (9.7%)	
DVR	7 (1.7%)	2 (1.8%)	3 (2.3%)	2 (1.2%)	
DVR+TV repair					
CPB time (min)	108±45	110±48	117±44	110±42	0.33
ACC time (min)	77±37	83±39	89±34	81±34	0.18
Prosthesis					
Mechanical/Bio-prosthesis (%)	89.4%/10.6%	97%/3%	88.7%/11.3%	90.3%/9.7%	0.89

PAH – Pulmonary arterial hypertension; COPD – Chronic obstructive pulmonary disease; DM – Diabetes mellitus; CVA – Cerebrovascular accidents; PVD – Peripheral vascular disease; EF – Ejection fraction; NYHA – New York Heart Association; AF – Atrial fibrillation; MS – Mitral stenosis; MR – Mitral regurgitation; TR – Tricuspid regurgitation; EuroSCORE II – European System for Cardiac Operative Risk Evaluation II; MVR – Mitral valve replacement; DVR – Double valve replacement; CPB – Cardiopulmonary bypass; ACC – Aortic cross clamp

Table 2: Early post-operative outcomes

Variables	**Total (*n*=407)**	**No or mild PAH (*n*=109; 26.8%)**	**Moderate PAH (*n*=133; 32.7%)**	**Severe PAH (*n*=165; 40.5%)**	***P***
Re-exploration(%)	5 (1.2%)	1 (0.9%)	2 (1.5%)	2 (1.8%)	0.97
Prolonged ventilation (%)	55 (13.5%)	6 (5.5%)	18 (13.5%)	31 (18.3%)	0.007
CSAKI (%)	198 (24.1%)	21 (19.3%)	34 (25.7%)	43 (26.1%)	0.38
Neurologic deficit (%)	6 (1.5%)	1 (0.9%)	2 (2.2%)	3 (2.1%)	0.82
Mortality (%)	24 (5.9%)	3 (2.8%)	6 (4.5%)	15 (9.1%)	0.09

PAH – Pulmonary arterial hypertension; CSAKI – Cardiac-surgery-related acute kidney injury

RHD is a huge health-care burden in poor nations, affecting primarily young people. [1] PAH is a common complication of left-sided valve disease, and it is most common in people who have had rheumatic mitral valve involvement for a long time. PAH is the result of a combination of pulmonary vasoconstriction and obliterative alterations in the pulmonary vascular bed, as well as passively transmitted backpressure from left atrial (LA) hypertension. [7] The pressure in a thin-walled chamber like the LA is determined by several factors including its stiffness, mechanical effects of rhythmic contraction and relaxation, diastolic period, the amount of blood entering and exiting it, and net atrio-ventricular compliance, which includes compliance of the LA and left ventricle (LV), as well as compliance of the pulmonary venous system. In a study of 20

patients, Schwammenthal et al. discovered a significant increase in PA pressure after exercise in a subgroup of individuals with impaired atrioventricular compliance. [8] There appears to be a wide range of atrio-ventricular compliance in MS patients, including individuals with low compliance and patients with normal compliance. On exercise or in situations of increased cardiac output, patients with low compliance develop significant PAH, a severe increase in LA pressure, and symptoms of MS, whereas patients with normal compliance remain asymptomatic as the increased RV stroke volume is accommodated in the compliant pulmonary venous bed. [9] This explains why, despite the fact that all of our patients had severe mitral valve disease, only 40% of our sample acquired severe PAH.

Table 3: Univariate and multiple logistic regression analysis for in-hospital mortality

Variables	Univariate analysis			Multivariate analysis		
	OR	CI	*P*	OR	CI	*P*
Creatinineclearance	1.01	0.96-1.06	0.64			
Anaemia	0.74	0.12-4.39	0.74			
NYHA III	0.84	0.13-5.34	0.86			
NYHA IV	6.78	0.27-168.8	0.24			
Mod PAH	0.36	0.003-3.4	0.37			
Severe PAH	2.26	0.13-5.34	0.43			
Lesion MS vs. MR	5.0	0.59-42	0.14			
EuroSCORE II	1.11	0.6-1.9	0.69			
CPB time	0.97	0.9-1.03	0.36			
ACC time	1.03	0.95-1.1	0.41			
>2 PC transfusion	13.47	1.7-106.5	0.01	10.76	1.97-58.6	0.006
Re-exploration	0.89	0.09-8.75	0.92			
CSAKI	11.89	2.5-54.1	0.001	8.26	2.2-30.93	0.002
Prolongedventilation	31.57	6.1-161	0.000	20.86	5.75-75.55	0.000

NYHA – New York Heart Association; MS – Mitral stenosis; MR – Mitral regurgitation; CPB – Cardiopulmonary bypass; ACC – Aortic cross clamp; PC – Packed cell; CSAKI – Cardiac surgery-related acute kidney injury; PAH – Pulmonary arterial hypertension

Table 4: Mortality rates as per predominant lesion andseverity of PAH

Predominant lesion	PAH category	Mortality (%)	*P*
Mitral stenosis (*n*=301)	No or mild	1/70 (1.4%)	0.03
	Moderate	4/99 (4%)	
	Severe	13/132 (9.8%)	
Mitral regurgitation (*n*=106)	No or mild	2/39 (5.1%)	0.98
	Moderate	2/34 (5.9%)	
	Severe	2/33 (6.1%)	

PAH – Pulmonary arterial hypertension

Table 5: Long-term outcomes

Parameter	Total (*n*=331)	No/Mild PAH (*n*=84)	Moderate PAH (*n*=111)	Severe PAH (*n*=136)	*P*
No readmissions	191 (58%)	50 (60%)	60 (54%)	81 (60%)	0.88
Readmissions for cardiac cause	42 (13%)	11 (13%)	15 (14%)	16 (12%)	
Readmission for non-cardiac cause	70 (21%)	16 (19%)	23 (21%)	31 (23%)	
Mortality	28 (8%)	7 (8%)	13 (11%)	8 (6%)	0.25

According to RHC, PAH is defined as a rise in mean pulmonary arterial pressure of more than 25 mmHg at rest. [10] Echocardiography is a quick and easy way to check for PAH. [11] PAH is identified as a risk factor for mortality in EuroSCORE II as moderate (PASP 31–55 mmHg) and severe (PASP more than 55 mmHg). [2] 2604 Doppler–RHC pairs were evaluated in a recent meta-analysis of 32 research. The authors concluded that there is a good correlation between echo-based and RHC assessments of PAH, especially in patients with left-sided cardiac disease and high PA pressures. [12] Echocardiography is also a non-invasive, practical, and convenient method. As a result, the authors employed EuroSCORE II cut-offs and values based on the most recent preoperative echocardiogram. Previous research have also used echo-based data to estimate PAH, similar to the current study. [3,13]

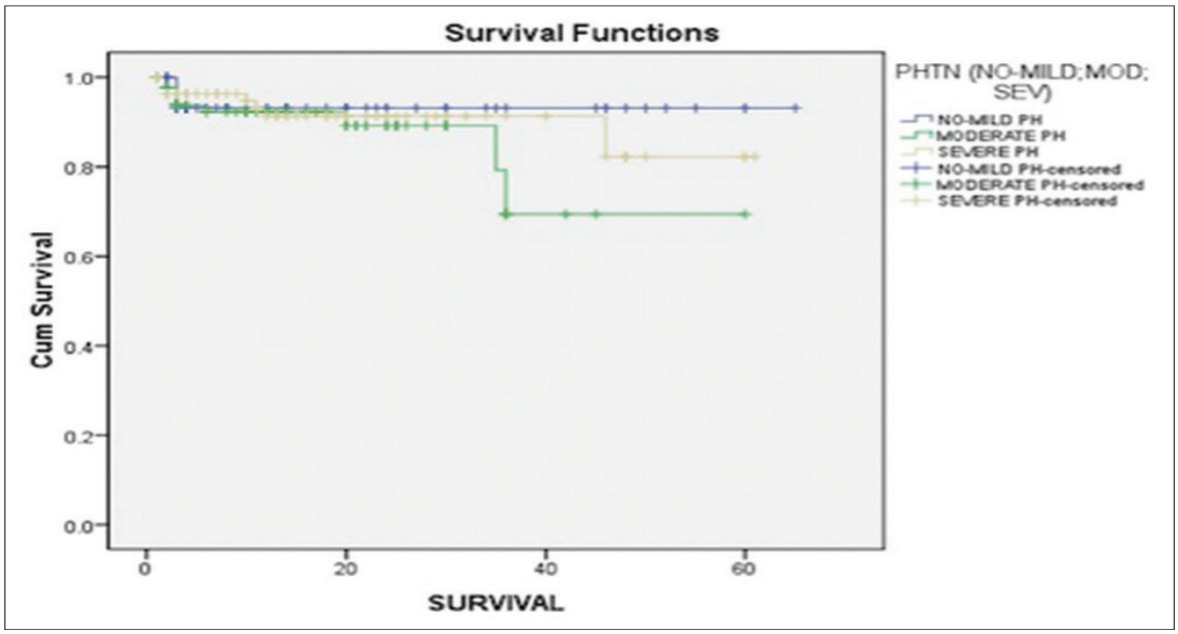

Figure 1: Long term Survival by Kaplan-Meier curve analysis

There is a large corpus of research on the impact of PAH on valve surgery results. These studies looked at a variety of patient demographics and assessed PAH using multiple methodologies with varying cut-offs and follow-up [3-5,13-25]. [Table 6]. The current study looked at how PAH, as determined by Doppler echocardiography, affected patients undergoing cardiac surgery for RHD only, using EuroSCORE II cut-offs. In our study, the incidence of severe PAH was 40%, which is consistent with earlier research. [13] In-hospital death rates of 5.9% and 9.1% in mild and severe PAH patients, respectively, are comparable to newly published evidence on cardiac surgery in RHD patients. [13,14,22] The mortality of patients with severe PAH was not statistically different from those with moderate, no, or mild PAH. More than two-packed cell transfusion, extended breathing, and CSAKI were all related with a higher risk of death. The majority of prior studies in diverse patient groups have concluded that mitral valve surgery can be conducted in patients with severe PAH with acceptable perioperative mortality.[3-5,22-24] Our findings are similar to those of earlier research. In a recent large research of 1639 RHD patients undergoing cardiac surgery, PASP >70 mmHg was found to be an independent risk factor for in-hospital

mortality (OR 2.93, 95 percent CI 1.61–5.32, P 0.001), while patients with PASP >52 mmHg had a greater 1-year mortality .[13] Another intriguing finding in our study was that when patients were classified as having MS or MR, those with MS and severe PAH had considerably higher mortality than those with no or moderate PAH, whereas those with MR had similar mortality in all categories of PAH.

Improved survival rates in severe PAH patients may be due to a variety of factors. Improved anaesthetic, surgical, and CPB techniques and materials have resulted from a greater knowledge of the pathophysiological alterations caused by PAH. Patients with PAH require special anaesthetic considerations in order to avoid right ventricular collapse. Balanced anaesthetic approach, prevention of hypoxia, hypercarbia, avoidance of nitrous gas, cautious use of vasodilator and inotrope therapy, and use of post-operative ventilator support are all part of it. [4] PAH diminishes dramatically after repair or replacement of a defective mitral valve, it has been discovered. [4,20] One of the important findings of our study was that patients with severe PAH experienced higher rates of post-operative mechanical ventilation. The exact reasons for prolong ventilation were not available for detailed analysis. This appears to be more likely an association rather than causative effect.

There have been numerous examples of severe PAH returning to normal after MVR. [4,20,26] This could be one of the main reasons why patients with severe and mild PAH have similar long-term results. All types of PAH have comparable long-term survival rates. This finding contradicts certain previously published studies that claimed severe PAH patients had a worse survival rate. [12,14] We also looked at the rates of readmissions for both cardiac (prosthetic valve thrombosis, congestive heart failure, arrhythmias causing significant haemodynamic disturbances, and so on) and non-cardiac (anticoagulation-related bleeding, neurological complications, respiratory causes, and so on) reasons and found no significant differences.

The current study has several significant limitations, the most significant of which being its retrospective nature and single-center investigation. Because the trial lasted so long, it is likely that management techniques for PAH may alter. We used Doppler echocardiography to assess PA pressure rather than RHC, which is the gold standard. Certain patients who did not return to our facility were contacted via telephonic call, which can lead to mistakes.

Conclusion

Short- and long-term results of patients receiving mitral ± aortic valve surgery for RHD are unaffected by moderate or severe PAH. Patients with MS who had severe PAH had a greater mortality rate than those who did not have PAH, although mortality in patients with MR as the major pathology did not vary.

References

1. Marijon E, Mirabel M, Celermajer DS, Jouven X. Rheumatic heart disease. Lancet. 2012;379:953-64.
2. Nashef SAM, Roques F, Sharples LD, Nilson J, Smith C, Goldstone AR, *et al.* EuroSCORE II. Eur J Cardiothorac Surg. 2012; 41:734-45.
3. Enter DH, Zaki A, Duncan BF, Kruse J, Anderi AC, Li Z, *et al.* A contemporary analysis of pulmonary hypertension in patients undergoing mitral valve surgery: Is this a risk factor? J Thorac Cardiovasc Surg. 2016; 151:1288-99.
4. Tempe DK, Hasija S, Datt V, Tomar AS, Virmani S, Banerjee A, *et al.* Evaluation and comparison of early hemodynamic changes after elective mitral valve replacement in patients with severe and mild pulmonary arterial hypertension. J Cardiothorac

Vasc Anesth. 2009;23:298-305.
5. Mubeen M, Singh AK, Agarwal SK, Pillai J, Kapoor S, Srivastava AK. Mitral valve replacement in severe pulmonary arterial hypertension. Asian Cardiovasc Thorac Ann. 2008; 16:37-42.
6. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, *et al*. Recommendations for chamber quantification: A report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quanti cation Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr. 2005;18:1440-63.
7. Groves P. Surgery of valve disease: Late results and late complications. Heart. 2001;86:715-21.
8. Schwammenthal E, Vered Z, Agranat O, Kaplinsky E, Rabinowitz B, Feinberg MS. Impact of atrioventricular compliance on pulmonary artery pressure in mitral stenosis: An exercise echocardiographic study. Circulation. 2000;102:2378-84.
9. Neema PK, Rathod RC. Pulmonary artery hypertension in mitral stenosis: Role of right ventricular stroke volume, atrio-ventricular compliance, and pulmonary venous compliance**.** J Anaesthesiol Clin Pharmacol. 2012; 28:261-2.
10. Galiè N, Hoeper MM, Humbert M. Guidelines for the diagnosis and treatment of pulmonary hypertension: The Task Force for the diagnosis and treatment of pulmonary hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society(ERS), endorsed by the International Society of Heart and Lung Transplantation (ISHLT). Eur Heart J. 2009; 30:2493-537.
11. McGoon M, Gutterman D, Steen V, Barst R, McCroy DC, Fortin TA, *et al*. Screening, early detection, and diagnosis of pulmonary arterial hypertension: ACCP evidence- based clinical practice guidelines. Chest. 2004;126:14S-34S.
12. Finkelhor RS, Lewis SA, Pillai D. Limitations and strengths of Doppler/Echo pulmonary artery systolic pressure–right heart catheterization correlations: A systematic literature review Echocardiograpahy. 2015;32:10-8.
13. Jiang L, Wei XB, He PC, Feng D, Liu YH, Liu J, *et al*. Value of pulmonary artery pressure in predicting in-hospital and one- year mortality after valve replacement surgery in middle-aged and aged patients with rheumatic mitral disease: An observational study. BMJ Open. 2017;7:e014316.
14. Yang B, DeBenedictus C, Watt T, Farley S, Salita A, Hornsby W, *et al*. The impact of concomitant pulmonary hypertension on early and late outcomes following surgery for mitral stenosis. J Thorac Cardiovasc Surg. 2016; 152:394-400.
15. Gosain P, Larrauri-Reyes M, Mihos CG, Escolar E, Santana O. Aortic and/or mitral valve surgery in patients with pulmonary hypertension performed via a minimally invasive approach. Interact CardioVasc Thorac Surg. 2016; 22:668-70.
16. Castillo-Sang M, Guthrie TJ, Moon MR, Lawton JS, Maniar H, Damiano RJ, *et al*. Outcomes of repeat mitral valve surgery in patients with pulmonary hypertension. Innovations. 2015;10:120-4.
17. Coutinho GF, Garcia AL, Correia PM, Branco C, Anyunes MJ. Negative impact of atrial fibrillation and pulmonary hypertension after mitral valve surgery in asymptomatic patients with severe mitral regurgitation: A 20-year follow-up. Eur J Cardiothorac Surg. 2015;

48:548-56.
18. Song X, Zhang C, Chen X, Chen Y, Shi Q, Niu Y, *et al*. An excellent result of surgical treatment in patients with severe pulmonary arterial hypertension following mitral valve disease. J Cardiothorac Surg. 2015; 10:70.
19. Paras I, Azam H, Khaki AB. Effects of pulmonary hypertension on early outcomes after mitral valve replacement. Pak Heart J. 2015; 48:96-100.
20. Bayat F, Aghdaii N, Farivar F, Bayat A, Valeshbabad AK. Early hemodynamic changes after mitral valve replacement in patients with severe and mild pulmonary artery hypertension. Ann Thorac Cardiovasc Surg. 2013; 19:201-6.
21. Kumar N, Sevta P, Satyarthy S, Agrawal S, Betigeri VK, Satsangi DK. Early results of mitral valve replacement in severe pulmonary artery hypertension—An institutional prospective study. World J Cardiovasc Surg. 2013; 3:63-9.
22. Elwany SE, Mohamed AH, Abu El-Hussain AK. Outcome after mitral valve replacement in patients with rheumatic mitral valve regurgitation and severe pulmonary hypertension. Egypt J Cardiothorac Anesth. 2013;7:74-8.
23. Cesnjevar RA, Feyrer R, Walther F. High-risk mitral valve replacement in severe pulmonary hypertension—30 years experience. Eur J Cardiothorac Surg. 1998; 13:344–52.
24. Vincent J, Dogan T, James R. Long term outcome of cardiac surgery in patients with mitral stenosis and severe pulmonary hypertension. Circulation. 1995;92:137-42.
25. Camara ML, Aris A, Padro JM. Long term results of mitral valve surgery in patients with severe pulmonary hypertension. Ann Thorac Surg. 1988; 45:133-6.
26. Parvathy UT, Rajan R, Faybushevich AG. Reversal of abnormal cardiac parameters following mitral valve replacement for severe mitral stenosis in relation to pulmonary artery pressure: A retrospective study of noninvasive parameters – Early and late pattern. Interv Med Appl Sci. 2016; 8:49-59.