Research Article

Clinical Significance of Preoperative Pulmonary Artery Hypertension in Patients Undergoing Mitral Valve Replacement for Rheumatic Heart Disease and Its Prognostic Implications

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Background: Rheumatic heart disease (RHD) is a leading cause of mitral valve pathology globally, frequently complicated by pulmonary arterial hypertension (PAH). The impact of preoperative PAH severity on postoperative outcomes following mitral valve replacement (MVR) needs to be evaluated.

Objectives: To follow the course of preoperative PAH following MVR for RHD, and to assess the effect of PAH severity on postoperative outcomes.

Methods: A single-centre, prospective observational study was conducted at a tertiary care centre. Eighty consecutive patients with RHD undergoing MVR with preoperative PAH (sPAP ≥ 20 mmHg) were enrolled between April 2024 and December 2025. Clinical, echocardiographic, intraoperative and postoperative parameters were recorded at baseline, before discharge, 1 month and 3 months postoperatively. Statistical analysis was performed using repeated measures ANOVA and logistic regression.

Results: The study population had a mean age of 42.8 ± 12.4 years with female predominance (61.2%). Mean baseline sPAP was 43.1 ± 6.0 mmHg, which progressively decreased to 32.5 ± 1.3 mmHg at 3-month follow-up ($p < 0.001$). Overall in-hospital mortality was 5% (n = 4). Mortality was significantly associated with severe preoperative PAH, advanced right ventricular dysfunction, severe tricuspid regurgitation, NYHA Class III/IV, higher BMI and prolonged operative times ($p < 0.001$ for all).

Conclusion: MVR effectively reduces PAH in patients with rheumatic mitral valve disease. However, PRE operative right ventricular dysfunctions, rather than PAH severity alone, was the primary determinant of postoperative outcome. Early surgical referral before irreversible right ventricular dysfunction is crucial for optimal outcomes.

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INTRODUCTION

Rheumatic heart disease (RHD) leads to valvular heart disease and affects an estimated 40 million individuals worldwide, predominantly children and young adults in low- and middle-income countries. It constitutes one of the most significant sources of cardiovascular morbidity and premature mortality in developing nations. The mitral valve is the most frequently affected, and left-sided valvular disease is commonly accompanied by pulmonary arterial hypertension (PAH)[1].

The pathophysiological mechanism involves progressive transmission of elevated left atrial pressure, triggering pulmonary vascular remodeling and ultimately leading to fixed pulmonary hypertension.[2]

Mitral valve replacement (MVR) or repair remains the definitive treatment for symptomatic patients. Severe PAH is a major risk factor for perioperative mortality, with mortality rates as high as 30% reported in the 1970s. Contemporary series have demonstrated substantially improved outcomes with mortality rates ranging from 6–12%. Role of medical therapies like pulmonary vasodilator therapy targeting endothelin pathways and sildenafil for PAH complicating left heart disease is limited and surgical correction of underlying valvular

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pathology is the definitive treatment in current practice[3]. The present study seeks to systematically evaluate the changes in preoperative PAH after MVR for RHD and to identify preoperative echocardiographic and clinical predictors of postoperative outcomes.[4]

Previous studies show that mild-to-moderate PAH is largely reversible following successful MVR, but severe and long-standing PAH may persist postoperatively due to established pulmonary vascular remodeling. The degree of right ventricular (RV) adaptation to chronically elevated afterload is a critical determinant of perioperative outcome, with advanced RV dysfunction and tricuspid regurgitation (TR) being consistently identified as major predictors of postoperative mortality and morbidity.[5]

Echocardiography parameters are the important surrogates in the preoperative assessment of patients with RHD and PAH. Key parameters including Tricuspid Annular Plane Systolic Excursion (TAPSE), right ventricular fractional area change (RVFAC), TR jet velocity, and estimated right ventricular systolic pressure (eRVSP) provide non-invasive surrogates of pulmonary haemodynamics and RV functional reserve. Serial echocardiographic assessment following MVR offers a valuable window into the dynamics of PAH regression and its temporal relationship with improvements in RV function and clinical status.[6]

In India and other developing nations, where RHD continues to impose a disproportionate burden of cardiovascular disease, patients frequently present at an advanced stage of disease with NYHA Class III or IV symptoms, severe mitral stenosis or regurgitation, and established PAH. Delayed surgical referral — attributable to limited healthcare access, socioeconomic constraints, and under-recognition of early disease — results in a substantial proportion of patients undergoing MVR with suboptimal haemodynamic profiles. Understanding the preoperative predictors of PAH regression and adverse outcomes in this high-risk population is therefore of paramount clinical importance.[7,8]

Several prospective and retrospective studies have evaluated the relationship between preoperative PAH and postoperative outcomes following MVR; however, data from South Asian cohorts remain sparse, and the echocardiographic thresholds that identify patients at highest risk are not uniformly defined. The present prospective observational study was conducted at a tertiary care centre in New Delhi with the objective of characterising the natural history of PAH regression following MVR for RHD, quantifying its association with postoperative outcomes, and identifying echocardiographic and clinical parameters that independently predict perioperative mortality and morbidity in this population.[9,10]

Methods

Study Design and Setting

This was a single-centre, prospective observational study conducted at the Department of Cardiothoracic and Vascular Surgery (CTVS), Atal Bihari Vajpayee Institute of Medical Sciences & Dr. Ram Manohar Lohia Hospital (ABVIMS & Dr. RML Hospital), New Delhi. The study was conducted over a period of 18 months, from April 2024 to December 2025. Ethical approval was obtained from the Institutional Ethics Committee (IEC) prior to commencement. All consecutive adult patients (aged ≥18 years) with a confirmed diagnosis of rheumatic heart disease (RHD) presenting for mitral valve replacement (MVR). Inclusion required: (i) echocardiographically confirmed rheumatic mitral valve disease (stenosis, regurgitation, or mixed) as the primary indication for surgery; (ii) presence of pulmonary arterial hypertension defined as an estimated systolic pulmonary artery pressure (sPAP) ≥20 mmHg on preoperative transthoracic echocardiography; and (iii) provision of written informed consent. Patients were excluded if they had: (i) non-rheumatic aetiology of mitral valve disease (e.g., degenerative, ischaemic, congenital); (ii) active infective endocarditis at the time of surgery; (iii) prior cardiac surgery; (iv) concomitant significant aortic valve disease requiring intervention; Enrolment was performed using total enumeration (consecutive) sampling; all eligible patients presenting during the study window were enrolled. A total of 80 patients fulfilled the criteria and constituted the final study cohort.

Data Collection and Clinical Assessment

A structured, pre-validated data collection proforma was used to prospectively record clinical, echocardiographic, intraoperative, and postoperative parameters at each time point. Preoperative data included: patient demographics (age, sex, body mass index [BMI]); symptom duration and severity classified by the New York Heart Association (NYHA) functional classification; relevant comorbidities (systemic hypertension, type 2 diabetes mellitus, chronic kidney disease [defined as eGFR <60 mL/min/1.73 m^2]); history of prior percutaneous balloon mitral valvotomy (PBMV); current medications including anticoagulation,

diuretics, beta-blockers, and vasodilators; and history of tobacco use or other addictions. Drug allergy history was also documented. Intraoperative data recorded included: total cardiopulmonary bypass (CPB) time (minutes), aortic cross-clamp (ACC) time (minutes), type of prosthetic valve implanted (mechanical vs. bioprosthetic), and whether concomitant tricuspid valve procedures were performed. Postoperative data included duration of mechanical ventilation (hours), inotropic support (days), intensive care unit (ICU) stay (days), and occurrence of complications.

Echocardiographic Assessment Protocol

Transthoracic echocardiography (TTE) was performed at four standardised time points: (T0) preoperatively within 48 hours of surgery; (T1) before hospital discharge (typically on postoperative day 7–10); (T2) at one month postoperatively; and (T3) at three months postoperatively. All studies were performed by a single experienced cardiologist blinded to intraoperative findings, using a standardised protocol on a Philips EPIQ 7 ultrasound system with a 2.5–5 MHz phased-array transducer, in accordance with the guidelines of the American Society of Echocardiography (ASE) and the European Association of Cardiovascular Imaging (EACVI).

Pulmonary artery systolic pressure (sPAP) was estimated as the right ventricular systolic pressure (RVSP) using the peak tricuspid regurgitant (TR) jet velocity measured by continuous-wave Doppler, applying the simplified Bernoulli equation with an assumed right atrial pressure (RAP) of 5 mmHg in the absence of clinical evidence of elevated RAP:

$$eRVSP = 4 \times (TRV)^2 + RAP \text{ (assumed = 5 mmHg)}$$

PAH severity was classified per the 2022 ESC/ERS guidelines as: normal (sPAP <30 mmHg), borderline (30–39 mmHg), mild (40–49 mmHg), moderate (50–59 mmHg), and severe (≥60 mmHg). Right ventricular (RV) function was assessed using the following parameters: (i) Tricuspid Annular Plane Systolic Excursion (TAPSE) measured by M-mode, with RV dysfunction defined as TAPSE <17 mm; (ii) RV fractional area change (RVFAC) from the apical four-chamber view, with dysfunction defined as RVFAC <35%; (iii) tissue Doppler-derived tricuspid lateral annular systolic velocity (S′), with dysfunction defined as S′ <9.5 cm/s; (iv) right atrial volume index (RAVI), calculated as right atrial volume indexed to body surface area; and (v) right ventricular hypertrophy (RVH) graded as mild, moderate, or severe based on end-diastolic RV wall thickness. Mitral valve morphology and the severity of tricuspid regurgitation (TR) were graded according to current ASE criteria (mild, moderate, severe). Left ventricular ejection fraction (LVEF) was calculated by the modified Simpson biplane method.

Surgical Technique

All patients underwent elective MVR via a median sternotomy. Cardiopulmonary bypass (CPB) was established using aorto-bicaval cannulation. Myocardial protection was achieved with antegrade cold blood cardioplegia delivered at 10 mL/kg initially, followed by repeat doses every 20 minutes. A left lateral atriotomy (Sondergaard's groove approach) was used in all cases to expose the mitral valve. The diseased mitral valve was excised, with posterior leaflet preservation attempted wherever technically feasible to maintain annulo-ventricular continuity. Prosthetic valve selection — mechanical (St. Jude Medical bileaflet) or bioprosthetic (Carpentier-Edwards pericardial) — was determined jointly by the operating surgeon and the patient following discussion of the risks and benefits of each valve type, patient age, and feasibility of long-term anticoagulation. Valve sizing was performed with standard sizers, and prostheses were implanted with interrupted pledgeted mattress sutures. Concomitant tricuspid valve annuloplasty (De Vega or ring annuloplasty) was performed in patients with moderate-to-severe functional tricuspid regurgitation confirmed on preoperative echocardiography and intraoperative assessment. All patients were separated from bypass with standard vasopressor and inotropic support as required, and transferred to the cardiac ICU for postoperative monitoring.

Postoperative Management and Outcome Assessment

The primary outcome was PAH regression, defined as a statistically significant and clinically meaningful reduction in serial sPAP measurements across the four assessment time points. Secondary outcomes included: all-cause in-hospital mortality; postoperative renal failure (new requirement for renal replacement therapy or serum creatinine ≥2× baseline); respiratory failure (re-intubation or tracheostomy requirement); re-exploration for bleeding or cardiac tamponade; duration of mechanical ventilation; total ICU stay; and duration of inotropic support. Follow-up echocardiographic assessments at one month (T2) and three months (T3) were performed in the outpatient clinic during scheduled review visits.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Shapiro-Wilk test and are presented as mean ± standard deviation (SD) where normally distributed, or as median with interquartile range (IQR) where non-normally distributed. Categorical variables are expressed as absolute frequencies and percentages. Serial changes in mean sPAP across the four time points (T0–T3) were analysed using one-way repeated measures Analysis of Variance (ANOVA) with sphericity assessed by Mauchly's test; where sphericity was violated, the Greenhouse-Geisser correction was applied. Post-hoc pairwise comparisons were performed using the Bonferroni correction to account for multiple testing. Independent predictors of PAH regression and postoperative adverse outcomes were identified using binary logistic regression with stepwise variable selection; results are reported as adjusted odds ratios (OR) with 95% confidence intervals (CI). Comparison of continuous variables between mortality groups (survivors vs. non-survivors) was performed using the independent samples t-test for normally distributed variables and the Mann-Whitney U test for non-normal distributions. Categorical comparisons were made using the chi-square test or Fisher's exact test as appropriate. A two-tailed p-value of <0.05 was considered statistically significant throughout.

RESULTS

Cohort Characteristics

A total of 80 patients fulfilling inclusion and exclusion criteria were enrolled. The cohort was predominantly middle-aged (mean age 42.8 ± 12.4 years; median 43.5 years) with female predominance (61.2%), consistent with the established sex predilection of rheumatic mitral valve disease. Overweight and obesity were prevalent (82.5% with BMI ≥ 25 kg/m²). All patients presented with breathlessness; comorbidities (hypertension, diabetes mellitus, chronic kidney disease) were present in 60%. Prior balloon mitral valvotomy had been performed in only 6.3% of patients.

Echocardiographic and Preoperative Profile

At baseline, the majority had mild tricuspid regurgitation (61.3%), though 10.0% had severe TR. RVH was absent in 40.0% and severe in 10.0%. Most procedures were elective (95%). Mean aortic cross-clamp time was 97.6 ± 10.6 minutes and mean cardiopulmonary bypass time was 119.1 ± 12.0 minutes.

Table 1: Cohort Characteristics (n = 80)

Characteristic	*n / Mean ± SD*	*%*
Age < 30 years	13	16.3
Age 31–40 years	18	22.5
Age 41–50 years	30	37.5
Age 51–60 years	15	18.8
Age > 60 years	4	5.0
Mean Age (years)	42.8 ± 12.4	—
Gender: Female	49	61.2
Gender: Male	31	38.8
BMI < 22.9 (Underweight)	6	7.5
BMI 23.0–24.9 (Normal)	8	10.0
BMI 25.0–30.0 (Overweight)	26	32.5
BMI > 30.0 (Obese)	40	50.0
Mean BMI (kg/m²)	23.5 ± 1.7	—
Comorbidities (HTN/DM/CKD)	48	60.0
Smoking / Addiction	27	33.8
Prior Balloon Valvotomy	5	6.3

HTN = hypertension; DM = diabetes mellitus; CKD = chronic kidney disease; BMI = body mass index.

Table 2: Baseline Echocardiographic and Preoperative Clinical Profile

Parameter	*Category*	*n (%)*
TR Severity: Mild	—	49 (61.3%)
TR Severity: Moderate	—	23 (28.7%)
TR Severity: Severe	—	8 (10.0%)
RVH Grade: Normal	—	32 (40.0%)
RVH Grade: Mild	—	18 (22.5%)
RVH Grade: Moderate	—	22 (27.5%)
RVH Grade: Severe	—	8 (10.0%)
All patients: Breathlessness	100%	80 (100%)

TR = tricuspid regurgitation; RVH = right ventricular hypertrophy.

Prevalence of Pulmonary Hypertension Post-MVR

Serial echocardiographic assessment of systolic pulmonary artery pressure (sPAP) by repeated-measures ANOVA demonstrated a highly significant and progressive reduction in pulmonary hypertension following mitral valve replacement ($p < 0.001$ at all time points). Mean sPAP declined from 43.1 ± 6.0 mmHg at baseline to 32.5 ± 1.3 mmHg at three months — a total reduction of 10.6 mmHg. The most substantial decrement (7.6 mmHg) occurred within the first postoperative month.

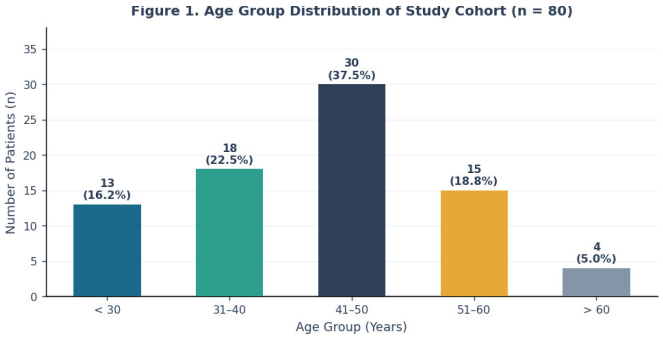


Figure 1: Age group distribution of the study cohort (n = 80). The largest proportion (37.5%) belonged to the 41–50-year age group. Mean age 42.8 ± 12.4 years.

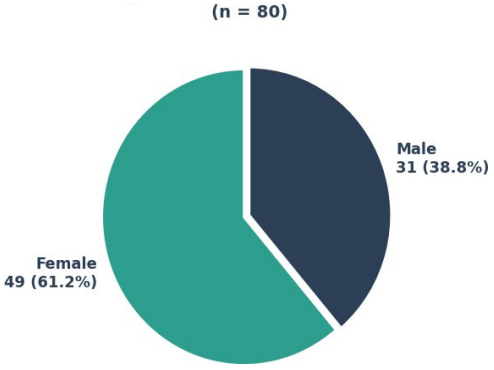


Figure 2: Gender distribution. Female patients constituted 61.2% (n = 49), consistent with the well-established female predominance in rheumatic mitral valve disease.

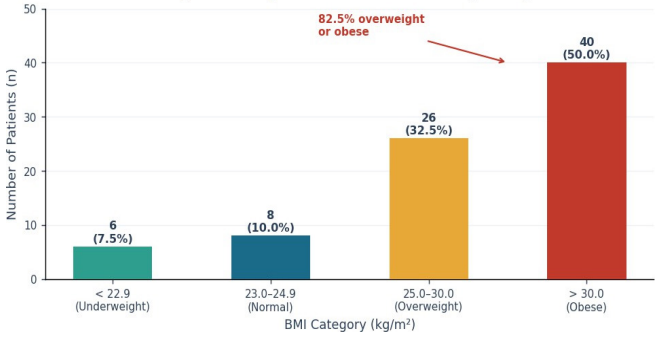


Figure 3: BMI distribution. A significant majority (82.5%) were overweight or obese — an important determinant of cardiopulmonary reserve and surgical risk.

At baseline, 80.0% of patients had mild PAH (sPAP 40–49 mmHg). By three months, 27.5% had normalised their pulmonary artery pressure (sPAP < 30 mmHg) and a further 66.2% had regressed to the borderline range (30–39 mmHg), representing a dramatic redistribution toward lower severity categories. Persistent moderate or severe PAH at three months was observed in only 2.4% of patients.

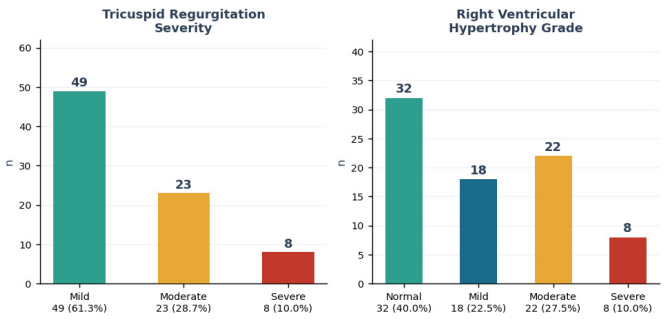


Figure 4: Preoperative echocardiographic profile showing TR severity (left) and RVH grading (right). Severe-grade disease in both parameters was confined to 8 patients (10.0%) each, and all four in-hospital deaths occurred exclusively within these severe-grade subgroups.

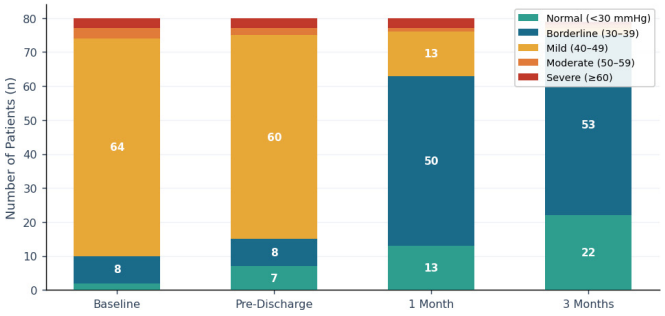


Figure 5: PAH severity distribution across follow-up time points. Baseline mild PAH (80.0%) progressively normalised, with 27.5% reaching normal sPAP and 66.2% in the borderline range by 3 months post-MVR.

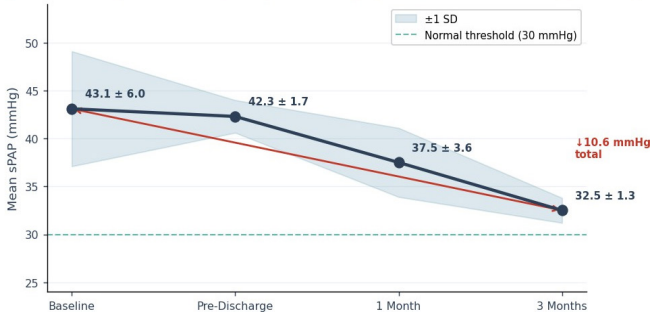


Figure 6: Mean sPAP trajectory post-MVR (±1 SD shading). Repeated-measures ANOVA, $p < 0.001$. The steepest decline occurred between discharge and 1 month (↓ 7.6 mmHg); total reduction was 10.6 mmHg at 3 months. Dashed line = normal PAP threshold (30 mmHg).

Survival According to Presence and Severity of Pulmonary Hypertension

In-hospital mortality was 5.0% (4/80). All four deaths occurred exclusively in patients with the most adverse preoperative profile: severe PAH, severe RVH, severe TR, and NYHA functional Class III/IV. No mortality

Table 3: Prevalence and Severity of Pulmonary Hypertension Across Follow-Up Time Points (Repeated Measures ANOVA)

PAH Grade	*Baseline n (%)*	*Pre-Discharge n (%)*	*1 Month n (%)*	*3 Months n (%)*
Normal (<30 mmHg)	2 (2.5)	7 (8.8)	13 (16.3)	22 (27.5)
Borderline (30–39 mmHg)	8 (10.0)	8 (10.0)	50 (62.5)	53 (66.2)
Mild (40–49 mmHg)	64 (80.0)	60 (75.0)	13 (16.2)	2 (2.5)
Moderate (50–59 mmHg)	3 (3.8)	2 (2.5)	1 (1.2)	1 (1.2)
Severe (≥60 mmHg)	3 (3.8)	3 (3.7)	3 (3.7)	1 (1.2)
Mean sPAP (mmHg ± SD)	43.1 ± 6.0	42.3 ± 1.7	37.5 ± 3.6	32.5 ± 1.3
Mean Reduction from Baseline	—	2.8 mmHg	7.6 mmHg	10.6 mmHg
p-value (vs Baseline)	—	< 0.001*	< 0.001*	< 0.001*

*sPAP = systolic pulmonary artery pressure; * p < 0.001 vs baseline. PAH grading: Normal < 30 mmHg; Borderline 30–39 mmHg; Mild 40–49 mmHg; Moderate 50–59 mmHg; Severe ≥ 60 mmHg.*

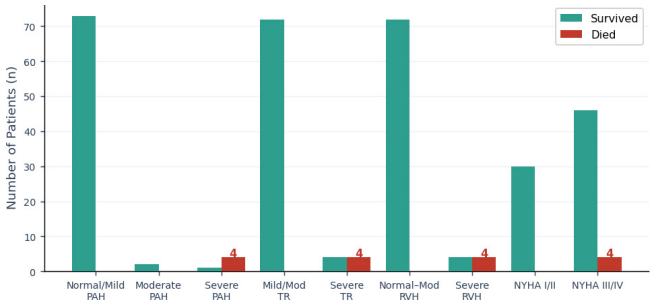


Figure 7: In-hospital mortality stratified by PAH severity, TR severity, RVH grade, and NYHA class. All four deaths (5.0%) were confined exclusively to severe-grade subgroups in every parameter, with no mortality in milder disease categories.

was observed in patients with milder grades of these conditions, suggesting a threshold effect whereby the combination of severe pulmonary and right-heart disease confers markedly elevated operative risk.

Independent t-test analysis identified significantly higher BMI, prolonged aortic cross-clamp and CPB times, extended mechanical ventilation, and greater inotropic requirement in non-survivors compared with survivors (all $p < 0.001$). These findings implicate both preoperative haemodynamic burden and intraoperative ischaemic duration as critical determinants of outcome.

Predictors of Outcome

Repeated-measures ANOVA identified multiple independent predictors of elevated PAH levels across all follow-up time points (Table 5). Although all subgroups demonstrated progressive PAH reduction following MVR, patients with higher baseline risk maintained relatively elevated sPAP throughout the observation period. Gender and smoking were not statistically significant predictors.

The most potent predictors — NYHA class III/IV, low TAPSE (< 17 mm), and dilated RV — were associated with significantly higher baseline sPAP and attenuated regression, highlighting the importance of right ventricular reserve and functional status in determining

Table 4: Comparison of Operative Parameters and Severity Markers: Non-Survivors vs Survivors (Independent t-test)

Variable	*Died (n=4)*	*Survived (n=76)*	*p-value*	*Clinical Context*
BMI (kg/m²)	28.7 ± 0.2	23.3 ± 1.5	< 0.001*	Higher obesity
Aortic Clamp Time (min)	114.0 ± 1.8	96.7 ± 10.1	< 0.001*	Prolonged ischaemia
CPB Time (min)	140.5 ± 2.3	118.0 ± 11.2	< 0.001*	Prolonged bypass
ICU Stay (days)	2.1 ± 0.1	2.6 ± 0.8	< 0.001*	Complex recovery
Mechanical Ventilation (hrs)	14.0 ± 0.0	7.9 ± 2.0	< 0.001*	Prolonged ventilation
Inotropic Support (days)	3.1 ± 0.2	3.1 ± 1.1	< 0.001*	Haemodynamic instability
PAH Grade at Baseline	Severe (all 4)	Mild–Moderate	—	All deaths: severe PAH
TR Severity	Severe (all 4)	Mild–Moderate	—	All deaths: severe TR
RVH Grade	Severe (all 4)	Normal–Moderate	—	All deaths: severe RVH
NYHA Class	III/IV (all 4)	I/II or III/IV	—	All deaths: NYHA III/IV

*CPB = cardiopulmonary bypass; ICU = intensive care unit; PAH = pulmonary arterial hypertension; RVH= right ventricular hypertrophy; TR = tricuspid regurgitation; NYHA = New York Heart Association; * p < 0.001.*

Table 5: Predictors of PAH Levels Across Follow-Up: Repeated Measures ANOVA by Risk Factor Subgroup

Parameter	Category	Baseline sPAP	1 Month sPAP	3 Months sPAP	p-value
Age	< 50 yrs	44.8 ± 1.4	36.8 ± 1.6	33.6 ± 1.6	0.03*
	> 50 yrs	43.6 ± 1.8	37.6 ± 1.7	32.4 ± 1.4	
BMI	< 25 (Normal)	42.7 ± 3.4	36.7 ± 1.4	33.5 ± 1.1	0.01*
	≥ 25 (Overweight/Obese)	43.5 ± 3.8	37.5 ± 1.8	32.3 ± 1.6	
Comorbidities	Present	43.4 ± 4.7	37.4 ± 2.2	32.2 ± 1.7	0.04*
	Absent	44.8 ± 3.5	36.8 ± 1.5	33.6 ± 1.1	
NYHA	III/IV	46.8 ± 4.2	38.8 ± 2.7	33.6 ± 1.2	< 0.001*
	I/II	44.8 ± 1.4	36.8 ± 3.7	33.6 ± 3.4	
TAPSE	< 17 mm (Low)	46.9 ± 3.4	38.9 ± 4.4	33.7 ± 3.9	< 0.001*
	17–20 mm (Borderline)	43.2 ± 5.6	37.2 ± 0.9	34.0 ± 1.5	
	> 20 mm (Normal)	43.8 ± 2.5	33.8 ± 1.4	32.6 ± 2.1	
RV Size	Dilated	43.8 ± 3.3	37.8 ± 1.3	34.6 ± 1.9	< 0.001*
	Normal	44.6 ± 4.2	34.6 ± 2.2	33.4 ± 3.1	
Gender	Male / Female	—	—	—	NS
Smoking	Present / Absent	—	—	—	NS

*TAPSE = tricuspid annular plane systolic excursion; RV = right ventricle; NS = not significant; * statistically significant.*

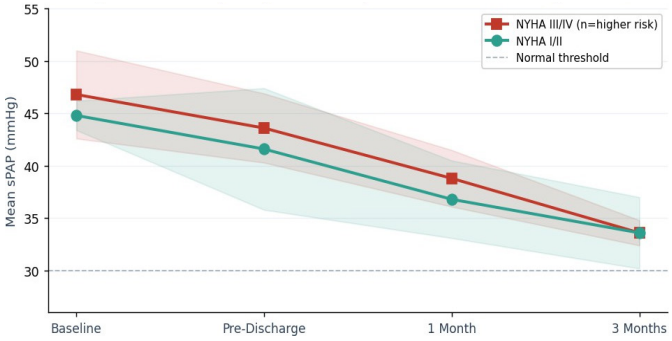


Figure 8: sPAP trajectory stratified by NYHA functional class ($p < 0.001$). NYHA Class III/IV patients demonstrated persistently higher sPAP across all time points, though both groups showed significant progressive reduction post-MVR.

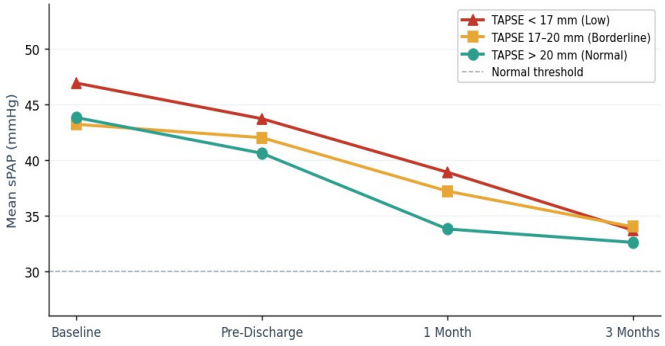
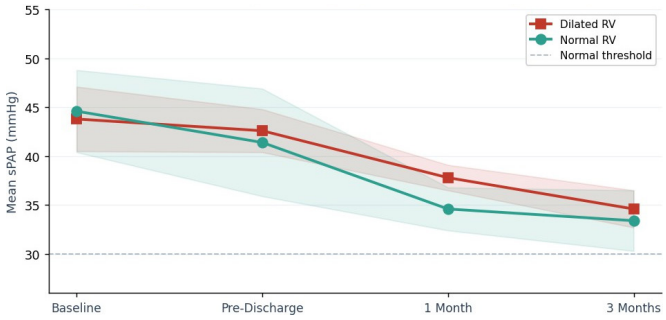


Figure 9: sPAP trajectory by preoperative TAPSE category ($p < 0.001$). Low TAPSE (< 17 mm) was associated with persistently elevated sPAP, reflecting impaired right ventricular contractile reserve and limited haemodynamic improvement after MVR.



Abbreviations: MVR = mitral valve replacement; sPAP = systolic pulmonary artery pressure; PAH = pulmonary arterial hypertension; NYHA = New York Heart Association; TR = tricuspid regurgitation; RVH = *right ventricular hypertrophy; BMI = body mass index; CPB = cardiopulmonary bypass; TAPSE = tricuspid annular plane systolic excursion; RV = right ventricle; SD = standard deviation; CI = confidence interval; NS = not significant.*

Figure 10: PAH regression stratified by right ventricular size ($p < 0.001$). Dilated RV was associated with higher residual sPAP at 3 months (34.6 ± 1.9 vs 33.4 ± 3.1 mmHg), confirming RV dimensions as an independent predictor of incomplete PH resolution.

the extent of pulmonary pressure normalisation after successful valve correction. Older age (> 50 years), higher BMI (≥ 25 kg/m²), and comorbidities also independently predicted higher PAH values at all time points.

Discussion

The present prospective study provides comprehensive data on the clinical significance and prognostic

implications of preoperative pulmonary arterial hypertension in patients undergoing MVR for rheumatic mitral valve disease. The findings are consistent with and extend the existing literature in several important respects.[11]

Demographic Profile

The study cohort was predominantly middle-aged (mean 42.8 ± 12.4 years) with female predominance (61.2%), reflecting the well-established epidemiology of rheumatic mitral valve disease. Similar demographics were reported by Farooq et al. (mean age approximately 40 years) and Ayyildiz et al. (mean age 56 years, 71.4% female). The younger age at presentation in the present study compared to Ayyildiz et al. likely reflects the greater disease burden and delayed surgical referral characteristic of developing nations, where repeated streptococcal infections accelerate valve damage. The high prevalence of comorbidities (60%) and obesity (50% with BMI >30) further underscores the multifactorial perioperative risk in this population.[12,13]

PAH Regression Following MVR

One of the principal findings of this study is the significant, progressive and sustained reduction in pulmonary artery pressure following MVR. The total mean reduction of 10.6 mmHg over three months (from 43.1 to 32.5 mmHg, $p < 0.001$) represents a clinically and statistically meaningful improvement. This is consistent with the established pathophysiological understanding that PAH secondary to rheumatic mitral valve disease, particularly when it is predominantly reactive (passive backpressure-mediated), is largely reversible upon relief of the hemodynamic obstruction. The observation that the greatest reduction occurred within the first postoperative month (7.6 mmHg) suggests rapid reversal of the reactive pulmonary vascular component, while progressive improvements thereafter reflect ongoing structural vascular remodeling.[14,15]

These findings align with those of Farooq et al., Kim et al., Song et al. and Bayat et al., all of whom demonstrated significant postoperative reductions in PAP following MVR. The shift in PAH grade distribution, from 80% mild PAH at baseline to 27.5% normalised and 66.2% borderline at 3 months, reinforces the therapeutic effectiveness of timely surgical intervention.[16,17,18,19,20]

Predictors of PAH Regression

Subgroup analysis revealed that NYHA Class III/IV, TAPSE <17 mm, dilated right ventricle, elevated TR jet velocity (>3.5 m/s), moderate-to-severe LVEF dysfunction, advanced age (>50 years), comorbidities and higher BMI were all independently associated with higher residual PAP levels at all follow-up time points (all $p < 0.05$). While all patient subgroups demonstrated some degree of PAH reduction, those with more severe baseline disease parameters maintained persistently elevated PAP, indicating incomplete regression. This finding is of particular clinical significance as it identifies a high-risk subset for whom early surgical referral — before the development of irreversible right ventricular dysfunction — is critically important.[21,22]

Mortality and Adverse Outcomes

The overall in-hospital mortality rate of 5% (4/80) compares favourably with contemporary published series. Abdelbaki et al. reported 10.6% operative mortality in severe PAH patients, while Farooq et al. and Kew et al. reported rates in the 5–10% range. In the present study, all deaths occurred in patients with the highest disease burden — severe PAH, severe RVH, severe TR and NYHA Class III/IV — underscoring that it is the overall right heart substrate, rather than PAH alone, that drives perioperative mortality. Logistic regression demonstrated that advanced age (>65 years), underweight BMI, NYHA Class III/IV, severe preoperative PAH, low TAPSE and dilated RV all significantly predicted postoperative mortality, renal failure and respiratory failure.[23,24]

Notably, prolonged CPB time and aortic cross-clamp time in non-survivors (CPB 140.5 vs 118.0 minutes; clamp time 114.0 vs 96.7 minutes) likely reflect the technical complexity of operating in the setting of advanced right ventricular dysfunction and severe tricuspid disease, requiring concomitant tricuspid valve interventions that prolong operative time.[25,26]

Clinical Implications

The findings of this study have direct clinical implications. PAH alone should not be considered a contraindication to MVR in rheumatic heart disease patients; rather, right ventricular functional reserve is the critical determinant of outcome. The current ESC/ACC guidelines recommend surgery when PAH develops in the context of mitral valve disease, as this signals hemodynamic decompensation. The present study provides prospective observational evidence supporting early surgical intervention, before irreversible right ventricular remodeling occurs, as the key to optimal surgical outcomes.[27,28]

Limitations

This study has several limitations. As a single-centre investigation, the findings may not be universally

generalisable. The sample size of 80 patients, while adequate for primary analysis, limited statistical power for mortality subgroup analyses given the low event rate. The follow-up period of three months, while sufficient to document early PAH regression and RV remodeling, does not allow assessment of long-term outcomes. The absence of a control group precludes direct comparison with medical management. Future multi-centre randomised controlled trials with longer follow-up are warranted.

Conclusion

This prospective study demonstrates that pulmonary arterial hypertension secondary to rheumatic mitral valve disease is largely reversible following surgically successful mitral valve replacement. A statistically significant and progressive reduction in sPAP was observed from baseline through three months of follow-up, accompanied by substantial improvement in right ventricular structure and function, regression of tricuspid regurgitation and improvement in NYHA functional class.

Crucially, PAH severity alone did not independently predict early postoperative mortality. Adverse outcomes were predominantly driven by advanced preoperative right ventricular dysfunction, severe tricuspid regurgitation, NYHA Class III/IV functional status, left ventricular dysfunction, elevated BMI and prolonged operative times. Non-regression of PAH was observed almost exclusively in patients with severe baseline pulmonary hypertension and advanced right ventricular impairment.

Overall, MVR is safe and effective in reducing PAH in rheumatic mitral valve disease, including patients with elevated pulmonary pressures. The right ventricular functional and structural status — not PAH severity per se — is the primary determinant of postoperative outcome. These findings strongly advocate for early surgical referral before the onset of irreversible right ventricular dysfunction to achieve optimal outcomes in patients with rheumatic mitral valve disease and evolving pulmonary hypertension.

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