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Outcome after Mitral Valve Replacement in Patients with Pulmonary Hypertension

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ABSTRACT

Background: Mitral valve complications can lead to the serious consequence of pulmonary arterial hypertension (PAH). It has long been believed that the onset of PAH increases the chance of a poor outcome in individuals having mitral valve replacement surgery (MVR).

Objective: The aim of the present study was to assess and improve the early outcome after MVR in patients with pulmonary hypertension.

Patients and methods: This study included forty-eight patients undergoing MVR at the Cardiothoracic Surgery Department, Suez Canal University Hospital. Patients were divided into: group (A) 25 patients: with mild to moderate pulmonary hypertension; and group (B) 23 patients: with severe pulmonary hypertension. Transthoracic Doppler echocardiography, systolic pulmonary arterial pressure (sPAP), left atrium diameter (LAD), left ventricular end-diastolic dimensions (LVEDD), left ventricular end systolic dimensions (LVESD), fraction shortening (FS) and left ejection fraction (LEF), were evaluated and compared in all patients.

Results: Mitral valve pathology was non-significantly different between the two groups. However, number of patients who had grade III IV tricuspid regurge was significantly higher in group B. The ventilation duration in group B was significantly longer than group A. The duration of weaning from inotropic support was significantly longer in group B. We found no significant difference between the two groups regarding postoperative complications and mortality with postoperative mortality of 4.3% in group B.

Conclusion: Pulmonary arterial systolic pressure decreased near normal value in most patients after surgery.

Keywords: Mitral Valve Replacement; Pulmonary Hypertension; Outcomes.

INTRODUCTION

Rheumatic heart disease is still a major global health concern, despite a significant decline in its frequency in affluent nations. When the mitral valve is involved, mitral regurgitation and/or stenosis occur. In most cases, mitral valve replacement (MVR) is required when surgery is necessary. A mechanical or bioprosthetic valve is placed in place of the patient's damaged mitral valve during MVR heart surgery [(1)].

Pulmonary hypertension is categorized by the World Health Organization into five groups: (1) PAH, defined as elevated PA pressure with a wedge pressure less than or equal to 15 mm Hg; (2) pulmonary venous hypertension; (3) pulmonary hypertension linked to parenchymal lung disease or chronic hypoxia; (4) pulmonary hypertension caused by emboli; and (5) miscellaneous [(2)].

Rise of the left atrial (LA) pressure and LV filling pressure, passively raises the pulmonary vein's backward pressure. Continuously high pulmonary venous pressure lead to alveolar capillary stress failure, which is followed by acute alveolar edema and capillary leakage [(3)].

PAH has a significant prognostic impact in left-sided valve heart disease (VHD). Because of the significant implications for risk management and risk stratification, the evaluation of PAH is essential for these patients [(4)].

The goal of this study was to assess early outcome after mitral valve replacement in patients with pulmonary hypertension. Also, to evaluate the triggers for further complications of postoperative mitral valve replacement.

PATIENTS AND METHODS

A descriptive prospective study was done at the Cardiothoracic Surgery Department, Suez Canal University Hospitals in the period between July 2017 and June 2018. It included forty-eight patients undergoing mitral valve replacement divided into two groups according to their preoperative pulmonary artery pressure: **Group (A):** 25 patients with mild to moderate pulmonary hypertension (25 ≤ sPAP < 55 mm Hg), and **Group (B):** 23 patients with severe pulmonary hypertension (sPAP ≥55 mm Hg).

Inclusion criteria:

All patients had pulmonary hypertension with baseline mean pulmonary artery pressure (mPAP) of at least 25 mmHg (as measured by transthoracic echocardiography). Age >18 years, and both sexes were included.

Exclusion criteria:

The following patients were excluded from the study; including coexisting coronary artery disease, congestive heart failure, aortic valve disease, renal dysfunction (serum creatinine ≥ 2.0 mg/dl), hepatic dysfunction (serum bilirubin ≥ 3.0 mg/dl), tricuspid valve endocarditis, redo mitral valve replacement, pregnant females, and patients who presented initially with cardiopulmonary arrest.

A. Preoperative preparation:

A thorough clinical cardiac examination was conducted along with a full history. After the patients' stable or unstable conditions were evaluated, the

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necessary investigations and management strategy were identified. Numerous laboratory tests were performed, including arterial blood gas (ABG), complete blood count (CBC), blood type and cross matching, coagulation profile, serum electrolytes, and random blood sugar testing if necessary.

- **Radiographic investigations as:**

- A posteroanterior and lateral image of a plain chest X-ray taken while the patient was upright were used to assess the pulmonary vasculature, the various heart chambers, and the cardiothoracic ratio.
- Echocardiography (M mode, two dimension) was done. The measurements included the various dimensions of the heart chambers, the mitral valve, fractional shortening, pulmonary artery pressure, assessment of other valves, the functions of the RV, and the existence of left atrial thrombus.
- Echocardiography was performed on the initial visit, before surgery, during the procedure using transesophageal echocardiography (TEE), after discharge, and at 3-month follow-up.
- Coronary angiography was recommended for patients with a positive history of ischemic vascular events, postmenopausal women, and males older than 40.

- **Medical treatment**:

Patients in group (B) who had a mPAP greater than 55 mm Hg were prescribed with 25 mg of sildenafil three times a day as a preoperative care for six weeks before to surgery. The patient's condition determines the appropriate dosage and diuretics. Rate control as required.

Every patient received their heart medicine dose in the morning. Prior to being transferred to the operating room, every patient received a 10-mg intramuscular dose of morphine sulphate. Local anaesthetic was used to introduce a 14-gauge peripheral intravenous cannula once the patient arrived in the preparation room. The ideal dosage for sedation was 0.03-0.07 mg/kg of midazolam. Under local anaesthesia, two arterial line blood samples were taken: one for baseline active clotting time (ACT) preoperatively and the other for baseline ABG analysis. Preoperative monitoring included direct arterial blood pressure, pulse oximetry, and a five-lead ECG.

(B) Intraoperative Assessment:

- **Anaesthetic technique:**

All patients received the same intraoperative anaesthetic approach, which included fentanyl 5–10 mic/kg and pancuronium 0.02 mg/kg as well as 0.5–1 mg/kg of propofol as a supplemental hypnotic. 100–200 microgram of fentanyl was administered as a further dose. An appropriate-sized endotracheal tube was used to intubate the trachea orally once the patient's muscles had fully relaxed. Isoflurane 0.5–1% inhalation was used to maintain anaesthesia in every patient.

Following induction, the right internal jugular vein was punctured to install a single lumen (Angio 16 gauge) and three lumen central venous catheters. Additionally placed were a nasogastric tube, a urethral catheter, and a nasopharyngeal temperature probe. It was necessary to implant TEE probe.

- **Surgical technique:**

The patient was placed on supine position then draped, and the sternum was exposed to the midclavicular line. A sternal retractor was positioned and opened after a median sternotomy. Aorto-bicaval cannulation and aortic root cannula insertion were done. Encircling the venae cavae with umbilical nylon tapes secured with tourniquet may be tightened after cardioplegia arrest to minimize blood return into the left atrium.

The inter-atrial groove was dissected in front of the right superior pulmonary vein. The mitral valve was accessed by opening the left atrium by an incision made posteriorly and parallel to the interatrial groove.

Following mitral valve replacement, a single layer 3/0 polypropylene suture was used to seal the left atriotomy. Ventricular vent de-airing was accomplished by passing the vent between the valve's orifice and the suture line. In order to allow the right heart to fill with blood, the venae caval tapes were released, and the perfusionist was given instructions to limit venous return to the pump.

The cardiopulmonary circuit was then permitted to empty fully of the venous return. Following the resumption of cardiac contraction, the aortic root suction was maintained while the heart was allowed to fill. Cardiopulmonary bypass was stopped once heart function returned and the patient was fully recovered. Air was still being expelled long after the heart started beating again. Before administering the protamine, decannulation was done and the suture line was fastened. The pericardium was then closed by continuous sutures across the aortic root. The sternotomy was then gradually closed in stages.

Cardiopulmonary Bypass (CPB):

There was use of the membrane oxygenators. During CPB, hematocrit was maintained at about 28%. The main methods used to protect the heart were antegrade cold crystalloid cardioplegia, topical cooling with iced saline slush. Every 30 minutes, a dose of 15-20 ml/kg of cardioplegia was administered.

Technique of mitral valve replacement:

The procedure involved securing the valve prosthesis to the annulus firmly using dependable suturing procedures, all while avoiding tissue interference with valve performance and causing no harm to surrounding structures or the heart.

Implantation was needed to protect the surrounding anatomical tissues of the mitral valve

annulus from harm. Maintaining LV function required maintaining the chordal attachments to the annulus and the papillary muscle.

Everting sutures (atrium to ventricle to sewing ring) were used to ensure proper bileaflet valve function. The prosthetic valve was forced into the centre of the orifice and the prosthetic valve leaflets' tissue interference was reduced. This was especially crucial if the annular chordal attachments were kept intact.

Repairing the tricuspid valve typically came after replacing the mitral valve. In these situations, a transseptal incision and the right atrium were frequently used to access the mitral valve. The tricuspid valve surgery was performed after the aortic cross-clamp was removed, the septum was closed, and the mitral valve prosthesis was in place.

(C) Postoperative evaluation:

During their stay in the ICU and in the hospital (4 days after being discharged from the ICU), every patient received a complete evaluation. During ICU stay; hemodynamics of the patients and ventilator support were estimated. Weaning from inotropic if needed was recorded. During hospital stay; morbidity, mortality, period of hospital stay, follow up echocardiography before discharge to assess the prosthetic mitral valve, left ventricular function and dimensions, and pulmonary artery pressure were evaluated.

Ethical Consideration:

The Suez Cana University Academic and Ethical Committee granted clearance for the study (IRB#3366/2018). Written informed permission was acquired from each participant. Throughout the examination, adherence to the Helsinki Declaration was maintained.

Statistical analysis

SPSS version 20.0 was used to statistically analyze the data. Descriptive statistics were used, such as mean±SD and percentage (%). The Student's t-test, the Mann-Whitney test, and the X^2-test were used for analytical statistics. P value if < 0.05 was deemed significant.

RESULTS

The current study showed that both groups were matched regarding basic demographic and clinical characteristics, regarding age, sex, BMI, presence of HTN, DM, AF and smoking **(Table 1)**. All echo parameters were insignificantly different between both groups preoperatively except for PAP, which was significantly higher in group B compared to group A **(Figure 1)**.

Table (1): Distribution of the studied groups regarding their demographic data and clinical characteristics

Characteristics	Groups				P value
	A (N=25)		B (N=23)		
	no	%	no	%	
Age (years) Mean ±SD	39.48 ± 10.3		43.52± 11.8		0.212 (NS)
Sex					
Male	7	28	7	30.4	0.853(NS)
Female	18	72	16	69.6	
BMI Mean ±SD	25.72 ±2.2		28.02 ±5.2		0.049*
Hypertension (g/dL)	8	32	9	39.1	0.606(NS)
Diabetes Mellitus	4	16	4	17.4	0.897(NS)
Atrial Fibrillation	10	40	11	47.8	0.585(NS)
Smoking	1	4	4	17.4	0.129(NS)

NS: insignificant difference, *: Significant

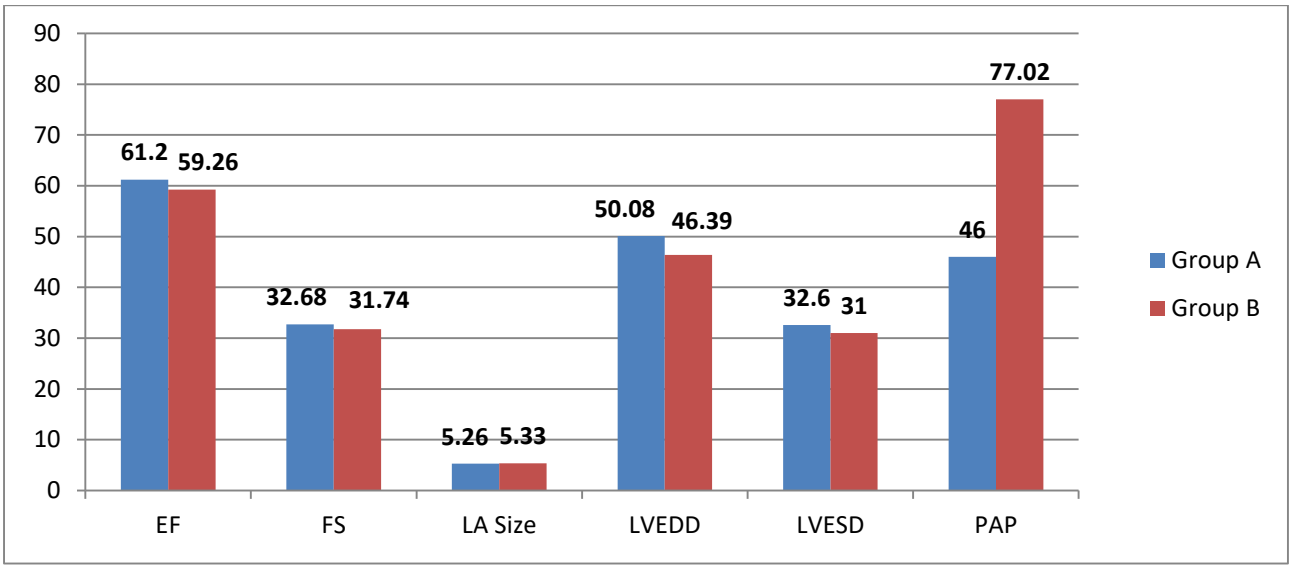

Figure (1): Preoperative echocardiographic data among the studied patients.

Mitral valve pathology was insignificantly different between the two groups. However, number of patients who had tricuspid regurgitation grade III -IV was significantly higher in group B than group A **(Table 2)**.

Table (2): Preoperative assessment of mitral valve status and the tricuspid valve regurgitation

	Groups				P value
	A (N=25)		B (N=23)		
	no	%	no	%	
Mitral valve status					
Stenosis	17	68.0	16	69.6	0.907(NS)
Regurgitation	8	32.0	7	30.4	
Tricuspid regurgitation					
Grade I and II	15	60.0	5	21.7	0.0001 *
Grade III and IV	5	20.0	18	78.3	

NS: Insignificant difference, *: Significant

Duration of aortic cross clamp, incidence of tricuspid valve repair, total cardiopulmonary bypass time, post bypass PAP by TEE, weaning from bypass by inotropes, and number of units of packed RBCs transfused during operation were significantly higher in group B than group A **(Table 3)**.

Table (3): Intraoperative assessment for ACCT and TVR

	Groups		P value
	A (N=25)	B (N=23)	
	Mean ±SD	Mean ±SD	
ACCT	54.56 ±16.9	73.70 ±17.1	0.0001*
Tricuspid repair			
Yes	8 (32%)	20 (87%)	0.048*
No	17 (68%)	3 (13%)	
Total cardiopulmonary bypass time/ Minutes (Mean ±SD)	86.80 ± 18.2	111.39 ± 23.4	0.0001*
Weaning from bypass by inotropes	17 68%	21 91.3%	0.047*
Blood transfusion per unit (Mean ±SD)	1.72 ±0.7	2.04 ±0.7	0.474(NS)
TEE PAP (Mean ±SD)	33.36±4.2	46.65±10.6	0.001*

ACCT: aortic cross clamp time; TVR: tricuspid valve repair *: Significant; NS: Insignificant difference.

The duration of weaning from ventilator was significantly higher in group B than group A. However, number of patients who needed inotropic support was insignificantly different between the two groups and duration of weaning from inotropic support was significantly higher in group B **(Table 4)**.

Table (4): Intensive care course among the studied groups

	Groups				P value
	A (N=25)		B (N=23)		
Weaning from ventilator (hours) Mean ±SD	6.00±1.4		10.22±4.7		0.001*
Need for inotropic support	19	76%	20	87%	0.331(NS)
Weaning from inotropic support/hours Mean ±SD	25.84 ±16.5		46±20.0		0.001*

*: Significant, NS: Insignificant difference

Regarding postoperative course**,** there was no significant difference between the 2 studied groups regarding mediastinal bleeding, arrhythmia, CNS complications, mortality, and sternal wound infection. No patients had renal or hepatic impairment in both groups **(Table 5)**.

Table (5): Postoperative course among the studied groups

	Groups				P value
	A (N=25)		B (N=23)		
	no	%	no	%	
Mediastinal bleeding	1	4	2	8.7	0.502(NS)
Arrhythmia	4	16	7	30.4	0.235(NS)
Renal impairment	0	0	0	0	1.00
Hepatic impairment	0	0	0	0	1.00
CNS complications	2	8	0	0	0.166(NS)
Mortality	0	0.0	1	4.3	0.292(NS)
Sternal wound Infection	3	12	2	8.7	0.708(NS)

NS: Insignificant difference

The duration of ICU and hospital stay was insignificantly different between the two groups **(Table 6)**.

Table (6): Postoperative stay among the studied groups

			P value
	Group A (N=25)	B Group (N=23)	
	Mean ±SD	Mean ±SD	
ICU stay/days	3.44±3.8	3.26±0.7	0.828(NS)
Hospital stay/days	7.92±6.2	6.78±1.2	0.394(NS)

NS: Insignificant difference

The echocardiographic data on discharge regarding EF was insignificantly different between the two groups; however, PAP was significantly higher in group B than group A **(Table 7)**.

Table (7): Postoperative echocardiographic data among the studied groups

	Groups		P value
	A (N=25)	B (N=23)	
	Mean ±SD	Mean ±SD	
Ejection fraction (EF)	58.68±4.6	55.35±12.9	0.236(NS)
Pulmonary arterial pressure (PAP)	31.60±4.9	37.43±9.5	0.010*****

NS: Insignificant difference, *: Significant

The mPAP significantly decreased immediately after mitral valve replacement compared to baseline values. Moreover, mPAP was significantly lower after 3 months postoperatively compared to baseline values **(Figure 2)**.

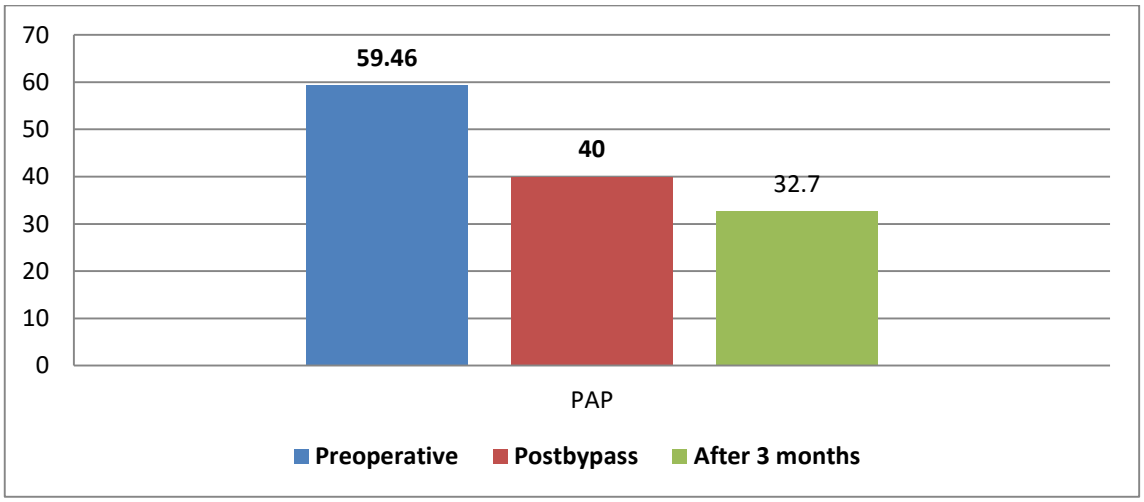

Figure (2): PAP difference among preoperative, post bypass, and 3-months postoperative values.

DISCUSSION

In developing nations, the leading cause of valvular heart disease is rheumatic heart disease. Patients with mitral valve disease are often within their second or third decade of life when they are scheduled for surgery. Many patients, particularly those from rural areas, emerge at a very late stage of the disease, with a very high pulmonary artery pressure (PAP) [(5)].

In this prospective study, 48 patients with pulmonary hypertension undergoing mitral valve replacement at Suez Canal University Hospitals were included. Our goal was to evaluate the early results of mitral valve replacement in pulmonary hypertension patients. When compared to baseline values, we discovered that the mPAP dropped considerably as soon as the mitral valve was replaced. In addition, three months following surgery, the mPAP was considerably lower than baseline levels.

Our results are in line with those of **Elwany *et al.*** [(6)]**,** who performed a prospective study on thirty patients who had baseline sPAP values of at least 40 mmHg and underwent elective MVR for rheumatic mitral valve regurgitation. They discovered that the median sPAP significantly dropped following bypass and remained low for the duration of the study. Moreover, **Cámara *et al.*** [(7)] observed 88 patients with severe pulmonary hypertension who had mitral valve surgery over a ten-year period, with an average systolic pulmonary artery pressure of 94 mm Hg (range: 70-180 mm Hg). PAPS was seen to have a considerable postoperative regression, falling from 101 mmHg to 40.5 mmHg.

Also, **Bendjaballah *et al.*** [(8)] performed operations on 201 patients with PAH and mitral or mitro-tricuspid illness. Depending on the degree of preoperative PAH, these individuals were split into two groups: (102 patients in Group A had moderate PAH, with 40 ≤ sPAP<60 mmHg; 99 patients in Group B had severe PAH, with sPAP > 60 mmHg. Following the surgery, the mean PAP significantly decreased.

In contrast to our findings, **Figuero *et al.*** [(9)] involved 111 patients having MVR with sPAP of more than 40 mmHg. The percentage of patients with PAH decreased from 64.9% to 43.2% just following mitral valve replacement. The majority of patients with persistent PAH following MVR had much higher PAP for prolonged periods of time, which may help to explain this. They discovered that the degree of PAH and TR severity prior to surgery, the size of the prosthesis implanted, and the requirement for tricuspid repair were all related to persistent PAH following mitral valve replacement. Prior to surgery, the mean gradients across the mitral valve were larger in their older patients.

In the current study, patients were divided into two groups according PH severity: Group A mild to moderate pulmonary hypertension (25 ≤ sPAP < 55 mmHg) and Group B with severe pulmonary hypertension (sPAP ≥ 55 mmHg. Both groups were matched regarding basic demographic and clinical characteristics regarding age, sex, BMI, presence of HTN, DM, AF and smoking. We found preoperative RVSP was significantly lower in group A compared to group B (P <0.05). However, preoperative assessment showed that the mean LVEDD, LVES, EF, FS, and LAD were insignificantly different between both groups.

Our finding agree with **Pande *et al.*** [(10)] who studied seventy patients with predominant rheumatic mitral stenosis receiving mitral valve replacement. Group A (RVSP < 40 mmHg) and Group B (RVSP >41 mmHg) were created based on RVSP. According to their report, group A had a much lower RVSP than group B. When group A was compared to group B, the mean LVEDD and mean LVES were considerably greater (P <0.05). EF and LAD, however, did not differ

significantly between the two groups. In addition, **Bendjaballah *et al.*** [8] revealed that group A had significantly lower left atrial diameter and significantly higher LVEDD and LVESD. But there was no discernible difference in EF between both groups. Moreover, **Figuero *et al.*** [9] showed no significant difference in mean left atrial diameter or mean ejection fraction between group B (who had preoperative PAH) and group A (who did not).

In our study, sildenafil was given in a dose 25 mg three times daily for six weeks for all patients in group B with preoperative severe pulmonary hypertension, and transthoracic echocardiography was done prior to surgery. It was found that pulmonary artery pressure was insignificantly different before and after sildenafil administration for 6 weeks.

Similar to our finding, **Alassal *et al.*** [11] who performed a retrospective study on 680 patients who had mitral valve replacement. The patients were divided into two groups: PASP 50-80 mmHg, classified as severe pulmonary hypertension (group A), and those with PASP > 80 mmHg, classified as extreme pulmonary hypertension (group B). After taking sildenafil orally for six weeks, 66% of cases had an insignificant decrease in PAH. It resulted in a negligible decline in exercise tolerance, functional class, and pulmonary hemodynamics.

On the contrary, **Salem *et al.*** [12] studied forty patients with valvular heart disease, idiopathic dilated cardiomyopathy, chronic thromboembolic disease, chronic obstructive pulmonary disease, and interstitial lung fibrosis who were known to have symptomatic secondary pulmonary hypertension. The patients were tasked with either a placebo or sildenafil for a period of six weeks. The baseline and 6-week follow-up included assessments of haemodynamic parameters, functional class determined by the NYHA classification, and echocardiographic measurements of pulmonary artery systolic pressure and left ventricular ejection fraction. At six weeks, the sildenafil group's mean NYHA class was 2.05±0.4, while the placebo group's was 2.6 ± 0.6, with a statistical significance of p = 0.02. The mean sPAP of the sildenafil group was significantly lower (43 ± 4 mmHg) than that of the placebo group. The sildenafil group's mean systolic pulmonary artery pressure was considerably lower (43 ± 4 mmHg) than the placebo patients (53±7 mm Hg), with a p-value of 0.02. The sildenafil group's ejection fraction was higher (59±12 %) compared to 54±14% in the placebo group but the difference was not statistically significant.

In our study, preoperative evaluation revealed that 8 patients (32%) had mitral valve regurgitation and 17 patients (68%) had mitral valve stenosis in group A. The study found that there was no statistically significant difference between the two groups as in group B, 16 patients (69.6%) had mitral valve stenosis and 7 patients (30.4%) experienced regurgitation. As opposed to this, in group (A), while five patients (20%) had tricuspid valve regurgitation grade III–IV, fifteen patients (60%) had grade I–II, and five patients (20%) had no tricuspid valve regurgitation. There was a statistically significant difference between the two groups in group (B), where 18 patients (78.3%) had tricuspid valve regurgitation grade III–IV and 5 patients (21.7%) had grade I–II.

Similarly, **Kumar *et al.*** [13] performed a prospective study on 68 patients who had mitral valves replaced due to significant PAH (PAP > 50 mmHg) and severe rheumatic mitral valve disease. Preoperative PAP was used to divide the patients into two groups. Twelve patients (17.65%) had supra-systemic PAP with a mean of 82.4 mmHg, while 56 patients (82.35%) had sub-systemic or systemic PAP with a mean of 58.4 mmHg (group I). Twelve patients (21.4%) had mitral valve regurgitation, twenty patients (35.7%) had mixed lesions, and twenty patients (42.8%) had mitral valve stenosis in group (I). Eight patients (66.6%) in group (II) had mitral valve stenosis, one patient (8%) had regurgitation of the mitral valve, and three patients (25%) had mixed lesions. The two groups did not exhibit a statistically significant difference in the severity of tricuspid valve regurgitation.

Moreover, **Elwany *et al.*** [6] documented that in their study seven patients (23.3%) had severe tricuspid regurgitation and twenty patients (66.7%) had mild-to-moderate regurgitation. **Figuero *et al.*** [9] revealed that 22 patients (34.9%) with mitral valve stenosis, 27 patients (42.8%) with mitral valve regurgitation, and 14 patients (22.2%) with mixed lesion were reported to have been part of group A. Twenty-two patients (45.8%) in group B had mitral valve stenosis, thirteen patients (27.1%) had regurgitation of the mitral valve, and thirteen patients (27.1%) had mixed lesion. However, group B had a noticeably greater proportion of individuals with severe tricuspid regurgitation.

In our study, group B underwent a longer cardiopulmonary bypass and aortic cross clamp procedure overall than Group A. There was a statistically significant difference between the two groups as 17 patients (68%) in group A and 21 patients (91.3%) in group B weaned from cardiopulmonary bypass with inotropic support. The quantity of transfused packed red blood cell units used during the procedure did not, however, differ much between the two groups. Furthermore, group A had a considerably less percentage of patients (32%) who required tricuspid valve replacement than group B (87%). Similar to our finding **Paras *et al.*** [14] revealed significantly higher CPB times in patients belonging to group B compared to group A. Furthermore, a greater percentage of patients in group B needed high inotropic assistance.

On the contrary, **Bendjaballah *et al.*** [8] disagreed with our finding. The number of patients who required blood transfusions, frequency of inotropic support use, length of total cardiopulmonary bypass, and aortic cross clamp time did not differ statistically between the two groups.

In addition, **Kumar *et al.*[13]** revealed that 8 patients in group A and 4 patients in group B underwent De Vega tricuspid annuloplasty ($p = 0.203$). The aortic cross clamp time did not differ substantially between the two groups ($p = 0.078$), however the mean CPB time was found to be significantly shorter in group A than in group B ($p < 0.001$).

In our study, it was found that ventilation duration in group B was significantly longer than group A. Although the proportion of patients who needed inotropic support was not significantly different between both groups, the duration of weaning from inotropic support was significantly longer in group B.

Similarly, **Paras *et al.*[14]** revealed that 8 patients (53.3%) in group B required prolonged ventilation duration > 6 hours, while 3 patients (20%) in group A required it ($p < 0.05$). Moreover, **Kumar *et al.*[13]** found that postoperative mechanical ventilation was required for a substantially longer period of time (32.2 ± 21.4 hours vs. 21.5 ± 11.7 hours) ($p = 0.04$) for patients in group B. The length of time that both groups used vasodilators and inotropic support, however, did not differ appreciably. The tiny sample size in group B (non-representative) may help to explain this.

In contrast to the findings in our study, **Bayat *et al.*[15]** found that there was no significant difference in the two groups' inotropic require or the amount of time it took to wean off of the ventilator. Also, **Bendjaballah *et al.* [8]** discovered that there was no discernible difference in the ventilation time between the two groups.

In our study, following surgery, there was no significant difference between groups A and B regarding postoperative course. In group A, one patient (4%) experienced mediastinal bleeding, four patients (16%) experienced arrhythmia, two patients (8%) experienced CNS complications, three patients (12%) developed sternal wound infections, but there was no mortality. In group B, on the other hand, two patients (8.7%) experienced mediastinal bleeding, seven patients (30.4%) experienced arrhythmia, two patients (8.7%) experienced sternal wound infections, and one patient (4.3%) passed away. Furthermore, the length of hospital stays and intensive care unit stays did not differ significantly between the two groups.

In consistence with our findings, **Bendjaballah *et al.* [8]** revealed that the majority of the ICU problems, affecting 6 patients in group A and 4 patients in group B, involved mediastinal haemorrhage. The early death rate worldwide was 0.49%; on day 40, renal failure claimed the life of just one patient in group B. Furthermore, they stated that there was no discernible difference in the length of hospital and intensive care unit stays between the two groups.

On the contrary, **Kumar *et al.* [13]** reported that group B experienced a substantially longer ICU stay than Group (A), although the length of hospital stay was similar ($p > 0.05$). A total of 5.8% of patients died in the first postoperative period, including two patients (3.5%) in group A and two patients (16.6%) in group B. The tiny sample size of group B prevented this difference from reaching statistical significance. Right ventricular dysfunction resulting in chronically reduced cardiac output was the cause of both deaths in group B. Sepsis and low cardiac output syndrome were the causes of death for the two patients in group A. Renal impairment was noted in three patients both at follow-up and during the hospital stay of the surviving patients, and bleeding resulted in re-exploration in two of them.

Moreover, **Song *et al.* [16]** stated that postoperatively, one patient showed bleeding that required an emergency reoperation, and that another patient required a second operation on the eighth day following heart surgery due to a pericardial effusion. Six patients with respiratory insufficiency received noninvasive positive pressure ventilation in an effort to prevent re-intubation, while one patient experienced acute respiratory failure following left heart failure and required re-intubation after the endotracheal tube was removed. The rate of perioperative death was 3.1%.

In addition, **Alassal *et al.* [11]** revealed that the death rate varied between groups (A and B) at 3.7% (20/540) and 14.3% (20/140), with a significant increase in group II patients' mortality rates ($p=0.03$). Hemodynamic instability and abrupt heart failure accounted for the majority of deaths in both groups, which happen in the first postoperative week. The statistical analysis showed that the rate of mortality increased with increasing age with the severity of pulmonary hypertension ($p=0.031$). Twelve cases out of the twenty (60%) of group A mortality were over fifty years old, and sixteen cases out of the twenty (80%) of group B mortality were over fifty.

In our study, group A showed an immediate decrease in pulmonary artery pressure following mitral valve replacement, from 46.00 to 33.36 post-bypass, to 31.60 upon discharge, and to 30.56 at the 3-month follow-up. Group B showed an immediate decrease in pulmonary artery pressure following mitral valve replacement, from 74.09 to 46.65 post-bypass, to 37.43 upon discharge, and to 33.57 at the 3-month follow-up. There was no discernible decrease in PAP in all but two individuals whose preoperative PAP was greater than 90 mmHg; this could be because the patients were elderly and had chronic illnesses.

Paras *et al.*[14] who matched our findings, stated that patients in both Groups (A and B) showed a considerable drop in PAP right after mitral valve replacement. But in Group (B) patients, this drop in pressure was more noticeable. After surgery, the PAP was reduced to approximately 30 mmHg in patients in Group B, while group A showed an average decrease in pressure of approximately 15 mmHg. Following MVR, two patients whose preoperative PAP was greater than 80 mmHg did not exhibit an observable decrease in PAP. This can be the result of a patient-prosthesis mismatch following surgery. Also, **Bendjaballah *et al.*[8]** revealed that PAPs in both groups had significantly

decreased after surgery as compared to preoperative values. Thirteen patients in group A had mild persistent PAH, ten in group B had moderate PAH, and two in group A had severe PAH.

In concordance with our findings, **Kumar *et al.*[13]** revealed that group A showed a considerable drop in PAP to levels close to normal following mitral valve replacement. Group B exhibited a considerable drop in the same period; however, there was still a large amount of residual PAH. **Elwany *et al.*[6]** stated that after the bypass, there was a noticeable drop in PAP, and that this alteration remained for 48 hours after surgery. Moreover, **Bayat *et al.* [15]** observed that, in individuals with severe PAH, PAP did not considerably drop immediately following MVR, although it did so throughout the first 24 hours.

Alassal *et al.* [11] revealed a significant decrease in PASP, which varied from 35 to 65 mmHg with a mean of 42.3 ±11.6 mmHg (p=0.001) in patients in group A with severe PHT. However, there was a notable drop in PASP which varied from 40 to 70 mmHg with a mean of 55±8 mmHg, in group B with high PHT. They stated that getting older and delaying the surgical treatment shortened the time it took to reverse the rise in pulmonary pressure following detection of the lesion.

CONCLUSION

We conclude that following surgery, the majority of patients' pulmonary arterial systolic pressure dropped to a number that was close to normal based on our experience and the experiences of others. The results of mitral surgery have been improved by appropriate preoperative care, the development of cardiopulmonary bypass, myocardial protection, and anaesthetic technique; hence, severe pulmonary hypertension need not be an absolute contraindication.

The procedure should ideally be carried out on a younger patient, ideally prior to any anatomical alterations in the pulmonary vasculature or myocardium. In order to reduce the danger of an increased surgical risk as a result of chronic PHT, early referral of patients with mitral valve disease and PHT to a surgeon is advised.

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