Impact of aortic valve prosthesis-patient mismatch on left ventricular mass regression

Original Article

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ABSTRACT

Background: Aortic valve replacement (AVR) aim to allow left ventricular mass (LVM) regression by relieving valve stenosis and lowering LV pressure. When the effective orifice area (EOA) of the prosthetic valve that has been placed is too small in comparison to the body surface area, it is known as valve prosthesis-patient mismatch (PPM). Thus, the aim of this investigation is to determine if PPM and the degree of LVM regression following AVR are related.

Patients and Methods: The study was a prospective cohort study of 100 patients with isolated aortic stenosis who underwent AVR for 2 years and were placed in two groups (50 patients in each group); group A consisted of patients with no PPM [indexed effective orifice area (IEOA) > 0.85 cm^2/m^2], and group B consisting of patients with PPM (IEOA ≤ 0.85 cm^2/m^2). The main outcomes of interest are type, size, EOA, and IEOA of the prosthetic aortic valve used, follow-up echocardiography after 12 months including gradient across the prosthetic valve, LVM, and LVM regression.

Results: Significant differences were observed between the two groups. In the PPM group, there were fewer reductions in both mean and peak aortic valve gradients compared to the no PPM group. LVM showed a marked absolute regression in the no PPM group (87.0 ± 30.6 g) compared to the PPM group (39.1± 15.9 g, $P < 0.001$).

Conclusion: This study shows that PPM may hamper the regression of LVM after AVR.

Key Words: Aortic stenosis, hypertrophy, prosthesis-patient mismatch.

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INTRODUCTION

One of the most prevalent valvular heart disorders is aortic stenosis. Usually, left ventricular (LV) hypertrophy is seen as the illness progresses. Relieving valve stenosis and lowering LV pressure or volume load is the primary goal of aortic valve replacement (AVR) since this will enable left ventricular mass (LVM) regression. A two-to-three-fold increase in cardiovascular-related mortality is linked to the existence of LV hypertrophy[1]. Both in patients with systemic arterial hypertension and in individuals with normotension, increased LVM is an independent predictor of death[2].

Regretfully, LVM regression can differ greatly in degree and is frequently insufficient among patients. These results emphasize how crucial it is to recognize and if possible, steer clear of risk factors that might lead to persistent LV hypertrophy after AVR[3].

When the effective orifice area (EOA) of the prosthetic valve that has been placed is too small in comparison to the body surface area (BSA), it is known as valve prosthesis-patient mismatch (PPM). Indexed effective orifice area (IEOA) less than or equal to 0.85 cm^2/m^2 is the definition of PPM[4]. The primary hemodynamic effect of this common issue, which affects 20 – 70 % of patients following AVR, is the creation of large transvalvular gradients via prosthetic valves that are typically functioning[5].

It is important to consider residual transprosthetic pressure gradients as they may pose a risk to the regression of LVM following AVR. This is because an elevated gradient would inevitably lead to an increased burden on the LV[6].

Much research have been done on the effect of PPM on LVM regression and there is still debate on this topic[2, 4, 7]. Thus, the aim of this investigation is to determine if PPM and the degree of LVM regression following AVR are related.

PATIENTS AND METHODS:

This research was performed at the Department of General Surgery, Ain Shams University Hospitals. Ethical Committee approval and written, informed consent were obtained from all participants.

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This prospective cohort study was done in Ain Shams University Hospitals and Sheikh Zayed Specialized Hospital from January 2021 to January 2023. The study included patients with isolated pure aortic stenosis who underwent AVR during the study period. Exclusion criteria of the study consisted of patients with more than mild aortic regurgitation, previous myocardial infarction, previous cardiac surgery, patients undergoing concomitant surgical procedures such as mitral valve surgery or coronary artery bypass with AVR, patients with preoperative comorbidities; hepatic impairment (elevated liver enzymes > double normal value) or renal impairment (serum creatinine < 2 mg/dl, estimated glomerular filtration rate > 60 ml/min/1.73 m^2). The study's sampling method was simple random sampling. The study included 100 patients placed in two groups (50 patients in each group); group A included patients who underwent AVR with no PPM (IEOA > 0.85 cm^2/m^2), group B included patients who underwent AVR with PPM (IEOA ≤ 0.85 cm^2/m^2). PPM is defined as an IEOA that is less than or equal to 0.85 cm^2/m^2 as reported by many studies[4, 7]. Using STATA program, setting alpha error at 5 % and power at 85 %. Results from the previous study[2] showed that the mean LVM regression in PPM group and no PPM group was - 48 ± 47 and - 77 ± 49 g, respectively. Based on this, 50 cases per group will be needed.

Methods

Preoperative clinical assessment of the cases, which included history taking and investigating risk factors that included rheumatic heart disease, diabetes mellitus, hypertension, smoking, family history of similar cardiac disease, previous operations and previous admission to CCU with heart failure, fainting attacks, chest pain or decline in daily activities.

Full general examination and searching for findings of low cardiac output or heart failure (cool peripheries, edema, pulmonary rales and elevated jugular venous pressure). BSA of patients is calculated.

Full cardiac examination; auscultation of heart sounds and murmur if present in different cardiac areas and palpation of aortic thrill if present.

Laboratory investigations, which included complete blood picture, blood sugar, coagulation profile, kidney function tests and liver function tests.

Chest radiograph, 12 leads ECG and echocardiography were done; the dimensions of the LV were assessed (left ventricular end-diastolic and left ventricular end-systolic), the end-diastolic interventricular septum thickness [septal wall thickness (SWT)] and posterior wall thickness (PWT) is recorded, LVM and LVM index are calculated based on BSA and LV systolic performance is evaluated using the ejection fraction (EF). Coronary angiography and carotid Doppler and duplex were done.

The operative assessment included type of cardioplegia solution used, type, size, EOA and IEOA of the prosthetic aortic valve used for replacement, cross-clamp time, total bypass time, amount of blood loss and blood transfusion.

The postoperative assessment included ICU stay duration, mechanical ventilation (MV) duration, amount of bleeding and blood transfusion and hospital stay duration.

Follow-up echocardiography after 12 months; the dimensions of the LV are assessed (left ventricular end-diastolic and left ventricular end-systolic), the end-diastolic interventricular septum thickness (SWT) and PWT are recorded, LVM and LVM index are calculated based on BSA and LV systolic performance is evaluated using the EF. Absolute and relative LVM regression are also calculated.

Statistical analysis

Numerical continuous data are evaluated by the mean ± SD, Student t test. Nominal or ordinal are evaluated by χ^2 test. Significance is indicated if P value less than 0.001. The SPSS program was used to evaluate the data.

Statistical analysis was done using IBM SPSS statistics for windows, Version 23.0. Armonk, NY: IBM Corp.

RESULTS:

Comparing operative characteristics based on prosthesis-patient mismatch

The type of cardioplegia used was uniform across both groups, with all patients receiving cold cardioplegia. The cross-clamp duration revealed no significant difference between the no PPM and PPM groups, with a mean of 62.0 ± 15.7 and 61.2 ± 15.4 min (P = 0.9). The total bypass time was also comparable between groups (mean ± SD, 85.3 ± 19.0 min for no PPM vs. 85.1 ± 19.2 min for PPM; P > 0.9). Blood loss during surgery showed no statistically significant difference; however, the PPM group tended to have a higher mean ± SD of blood loss (191.0 ± 78.7 ml for no PPM vs. 217.0 ± 83.1 ml for PPM; P = 0.13).

Comparing prosthetic characteristics based on prosthesis-patient mismatch

All patients in both groups received a St Jude Regent valve. The valve size distribution differed significantly between the groups (P < 0.001); in the no PPM group, 4 % received a 19 mm valve, 50 % a 21 mm valve and 46 % a 23 mm valve. Conversely, in the PPM group, 90 % received a 19 mm valve and 10 % received a 21 mm valve, with no patients receiving a 23 mm valve (Figure 25). The

EOA and IEOA both demonstrated significant reductions in the PPM group compared with the no PPM group (EOA mean ± SD: no PPM, 2.1 ± 0.2 cm^2 vs. PPM, 1.6 ± 0.1 cm^2; IEOA mean ± SD: no PPM, 1.1 ± 0.1 cm^2/m^2 vs. PPM, 0.8 ± 0.0 cm^2/m^2; both $P < 0.001$) (Table 1).

Table 1: Comparing prosthetic characteristics based on prosthesis-patient mismatch (N = 100):

Characteristic	No PPM (N = 50) [n (%)]	PPM, (N = 50) [n (%)]	*P* value
Type			NA
St. Jude	50 (100)	50 (100)	
Size			< 0.001[a]
19 mm	2 (4.0)	45 (90)	
21 mm	25 (50)	5 (10)	
23 mm	23 (46)	0	
EOA (cm^2)			< 0.001[b]
Mean ± SD	2.1 ± 0.2	1.6 ± 0.1	
Median (IQR)	2.0 (2.0, 2.3)	1.6 (1.6, 1.6)	
Range	1.6, 2.3	1.6, 2.0	
IEOA (cm^2/m^2)			< 0.001[b]
Mean ± SD	1.1 ± 0.1	0.8 ± 0.0	
Median (IQR)	1.1 (1.0, 1.2)	0.8 (0.8, 0.8)	
Range	0.9, 1.4	0.7, 0.8	

EOA, effective orifice area; IEOA, indexed effective orifice area; IQR, interquartile range; PPM, patient-prosthesis mismatch.

aPearson's χ^2 test.

bWilcoxon rank sum test.

Evaluation of postoperative outcomes

We found no significant differences in the immediate postoperative course regarding postoperative outcomes. MV duration, ICU stay, blood loss and hospital stay did not differ significantly between the groups. The mean MV duration was 12.1 ± 8.8 h for the no PPM group and 10.3 ± 5.2 h for the PPM group ($P = 0.4$). The ICU stay was 2.3 ± 2.6 days for no PPM and 2.2 ± 1.0 days for PPM patients ($P = 0.13$). Blood loss was comparable between the groups (mean ± SD: no PPM, 284.0 ± 134.2 ml vs. PPM, 268.0 ± 108.7 ml; $P = 0.7$). Similarly, the length of hospital stay was not significantly different (mean ± SD: no PPM, 7.2 ± 3.1 days vs. PPM, 6.9 ± 1.8 days; $P = 0.9$).

Evaluating the changes in echocardiographic parameters

Prosthesis-patient mismatch cases

In patients with PPM, significant echocardiographic changes were observed 1 year following AVR. The left ventricular end-diastolic dimension (LVEDD) decreased from 50.2 ± 7.0 mm before surgery to 49.1 ± 6.3 mm after surgery ($P < 0.001$). Left ventricular end-systolic dimension (LVESD) remained relatively unchanged, with measurements of 33.8 ± 7.2 mm before and 33.3 ± 6.6 mm after ($P = 0.3$). Significant reductions were noted in SWT, from 13.1 ± 1.2 to 11.9 ± 1.2 mm and PWT, from 12.8 ± 1.2 to 11.7 ± 1.2 mm (both $P < 0.001$). LVM showed a significant reduction from 265.0 ± 64.0 to 225.9 ± 57.9 g ($P < 0.001$), as did the LVM index, which decreased from 128.4 ± 31.2 to 109.4 ± 28.0 ($P < 0.001$). There were also substantial reductions in both mean and peak aortic valve gradients; the mean gradient decreased from 57.4 ± 13.4 to 17.5 ± 3.2 ($P < 0.001$) and the peak gradient from 95.6 ± 18.9 to 29.6 ± 4.3 ($P < 0.001$).

Non-prosthesis-patient mismatch cases

In patients with non-PPM, echocardiographic parameters demonstrated significant improvements 1 year after AVR. The LVEDD decreased from 50.8 ± 7.0 mm before surgery to 47.1 ± 5.7 mm after surgery ($P < 0.001$). LVESD also decreased from 34.6 ± 7.2 to 32.6 ± 5.5 mm ($P < 0.001$). Similarly, significant reductions were observed in SWT, decreasing from 13.0 ± 1.2 to 10.5 ± 0.9 mm and in PWT, which decreased from 12.6 1.5 to 10.4 ± 1.0 mm (both $P < 0.001$). Notably, there was a substantial reduction in LVM, from 265.4 ± 64.0 to 178.3 ± 42.9 g and in LVM index, from 136.0 ± 31.7 to 91.4 ± 21.3 g/m^2 (both $P < 0.001$). Additionally, there were marked decreases in the mean and peak aortic valve gradients, from 57.7 ± 12.4 to 10.9 ± 2.7 mmHg and from 99.0 ± 17.3 to 21.1 ± 4.2 mmHg, respectively (both $P < 0.001$).

Overall differences after 1 year of surgery

In the comparative analysis of echocardiographic parameters 1-year post-AVR, significant differences were observed between patients without PPM and those with PPM (Table 2). LVM showed a marked absolute regression in the no PPM group (87.0 ± 30.6 g) compared to the PPM group (39.1 ± 15.9 g, $P < 0.001$). Relative LVM regression also differed significantly between groups, with the no PPM group exhibiting a mean regression of 32.3 ± 8.4 % compared to 14.8 ± 5.7% in the PPM group ($P < 0.001$). Changes in LVEDD post-AVR were greater in the no PPM group (3.7 ± 3.0 mm) versus the PPM group (1.1 ± 1.7 mm, $P < 0.001$). The reduction in LVESD followed a similar pattern (no PPM: 2.0 ± 3.7 mm vs. PPM: 0.5 ± 3.4 mm, $P< 0.001$). SWT differences were more pronounced in the no PPM group (2.4 ± 1.1 mm) compared to the PPM group (1.2 ± 0.8 mm, $P < 0.001$) (Figure 33), as were the changes in PWT (no PPM: 2.2 ± 1.2 mm vs. PPM: 1.0 ± 0.7 mm, $P < 0.001$). The differences in mean aortic valve gradient and peak gradient 1-year postsurgery were statistically significant (mean gradient: no PPM 46.8 ± 12.2 mmHg

vs. PPM 39.9 ± 13.4 mmHg, P=0.008; peak gradient: no PPM 77.9 ± 16.9 mmHg vs. PPM 66.0 ± 18.8 mmHg, $P = 0.002$).

Table 2: Comparing overall differences in echocardiographic parameters after 1 year of surgery in patients without prosthesis-patient mismatch versus prosthesis-patient mismatch patients (N = 50):

Characteristic	No PPM (N = 50)	PPM (N = 50)	*P* value[a]
LVM absolute regression (g)			< 0.001
Mean ± SD	87.0 ± 30.6	39.1 ± 15.9	
Median (IQR)	87.7 (79.4, 99.1)	40.3 (29.2, 47.1)	
Range	5.9, 142.9	7.6, 84.9	
LVM relative regression %			< 0.001
Mean ± SD	32.3 ± 8.4	14.8 ± 5.7	
Median (IQR)	34.0 (30.5, 36.9)	15.1 (12.4, 17.8)	
Range	3.4, 44.8	4.4, 35.7	
Difference in LV mass index			< 0.001
Mean ± SD	44.6 ± 15.3	19.0 ± 8.0	
Median (IQR)	44.4 (40.4, 53.3)	19.3 (14.4, 23.4)	
Range	2.7, 78.7	3.8, 43.4	
Difference in LVESD (mm)			< 0.001
Mean ± SD	2.0 ± 3.7	0.5 ± 3.4	
Median (IQR)	2.0 (1.0, 3.0)	0.0 (- 1.0, 1.0)	
Range	- 8.0, 22.0	- 13.0, 11.0	
Difference in SWT (mm)			< 0.001
Mean ± SD	2.4 ± 1.1	1.2 ± 0.8	
Median (IQR)	3.0 (2.0, 3.0)	1.0 (1.0, 1.8)	
Range	- 2.0, 5.0	0.0, 5.0	
Difference in PWT (mm)			< 0.001
Mean ± SD	2.2 ± 1.2	1.0 ± 0.7	
Median (IQR)	2.0 (2.0, 3.0)	1.0 (1.0, 1.0)	
Range	- 1.0, 3.0	- 1.0, 3.0	
Difference in EF%			0.10
Mean ± SD	1.4 ±.6	4.0 ± 5.8	
Median (IQR)	2.5 (- 2.0, 5.8)	4.0 (1.0, 6.0)	
Range	- 35.0, 24.0	- 5.0, 29.0	
Difference in AV gradient (mean)			0.008
Mean ± SD	46.8 ± 12.2	39.9 ± 13.4	
Median (IQR)	47.0 (36.3, 54.0)	40.0 (29.8, 49.0)	
Range	26.0, 75.0	13.0, 80.0	
Difference in AV gradient (peak)			0.002
Mean ± SD	77.9 ± 16.9	66.0 ± 18.8	
Median (IQR)	77.0 (65.3, 89.5)	67.5 (55.5, 81.3)	
Range	44.0, 111.0	15.0, 103.0	

AV, aortic valve; EF, ejection fraction; IQR, interquartile range; LVESD, left ventricular end-systolic dimensions; LVM, left ventricular mass; PPM, patient-prosthesis mismatch; PWT, posterior Wall Thickness; SWT, septal wall thickness.

aWilcoxon rank sum test.

Regression analysis predicting the postoperative left ventricular mass regression

In univariate analyses, PPM was significantly associated with less postoperative LVM regression, with a beta coefficient of - 48 [95 % confidence interval (CI), - 58 to - 38; $P < 0.001$). No significant associations were found with sex, with males showing a beta of 7.8 (95 % CI, - 5.7 to 21; $P = 0.3$) or with age, with a beta of - 0.11 per year (95 % CI, - 0.72 to 0.51; $P = 0.7$). Similarly, height and mean aortic valve gradient were not predictive of LVM regression, with betas of 0.17 cm (95 % CI, - 0.88 to 1.2; $P = 0.8$) and - 0.06 mmHg (95 % CI, - 0.59 to 0.47; $P = 0.8$), respectively. However, weight and BSA were inversely related to LVM regression, with betas of - 0.67 kg (95 % CI, - 1.2 to - 0.16; P = 0.011) and - 56 m^2 (95 % CI, - 101 to - 11; $P = 0.016$), respectively. Wall thickness measurements, both SWT and PWT, were also significant predictors, with betas of 8.0 mm (95 % CI, 2.7 – 13; $P = 0.003$) and 7.0 mm (95 % CI, 2.2 – 12; $P = 0.005$).

In the multivariate analysis, LVM, LVEDD and IEOA were significant predictors of LVM regression, with beta coefficients of 0.37 (95 % CI, 0.26 – 0.48; P < 0.001), - 1.5 mm (95 % CI, - 2.5 to - 0.43; $P = 0.006$) and 129 cm^2/m^2 (95 % CI, 105–154; $P < 0.001$), respectively. When standardized, these factors remained significant, with LVM showing a beta of 0.69 (95 % CI, 0.48 – 0.90), LVEDD - 0.30 (95 % CI, - 0.51 to - 0.09) and IEOA 0.65 (95 % CI, 0.53 – 0.77). The adjusted R2 value for the model was 63.52 %, indicating that these variables accounted for approximately two-thirds of the variance in postoperative LVM regression ($P < 0.001$).

DISCUSSION

Our study included 100 patients, who underwent baseline demographic, prosthetic and echocardiographic assessments. They were then stratified into two groups based on the presence or absence of PPM, defined by an IEOA less than or equal to 0.85 cm^2/m^2. Each group comprised 50 patients, allowing for a balanced comparative analysis of the impact of PPM on postoperative outcomes.

The study population of Tasca *et al.*[2] includes 109 patients with pure aortic stenosis who underwent AVR between September 1997 and July 2002, while the prospective study of Alassal *et al.*[7] consisted of 67 consecutive patients with isolated pure aortic stenosis, who underwent AVR between February 2007 and March 2011 at two cardiac centers. On the other hand, the study of Kato *et al.*[8] included 165 consecutive patients who underwent AVR for aortic stenosis – divided into two groups according to the presence of PPM – between October 1990 and January 2005. The study population of Roscitano *et al.*[9] comprised of 88 patients over 65 years of age with pure aortic stenosis who underwent mechanical AVR between September 1991 and April 2000.

In our study, the comparison of baseline characteristics between patients with and without PPM (each group N = 50) shows no significant differences in sex distribution ($P = 0.42$) and age (mean ± SD; no PPM: 51.9 ± 11.6, PPM: 52.8 ± 10.7; $P = 0.83$), while the study of Tasca *et al.*[2] showed patients with PPM and those with no PPM were similar in age distribution but differs in sex distribution, sex (male %) (no PPM: 68, PPM: 34; $P < 0.001$). On the other hand, the study of Alassal *et al.*[7] showed the modest difference in sex distribution sex (male %) (no PPM: 37.5, PPM: 41.7; $P < 0.001$). The study of Kato *et al.*[8] showed significant differences in sex distribution between groups sex (male %) (no PPM: 64, PPM: 48; $P < 0.064$). The study of Roscitano *et al.*[9] showed no significant differences in sex distribution ($P = 0.46$).

Significant lower BSA (mean ± SD; no PPM: 2.0 ± 0.1 m2, PPM: 2.1 ± 0.1 m^2; $P < 0.0013$) were observed in the no PPM group in our study, similarly the study of Roscitano *et al.*[9] showed lower BSA in no PPM group (mean ± SD; no PPM: 1.65 ± 0.22 m^2, PPM: 2.01 ± 0.15 m^2; $P < 0.0003$). On the other hand, the study of Tasca *et al.*[2] showed no difference in BSA between the two groups (mean ± SD; no PPM: 1.73 ± 0.17 m^2, PPM: 1.79 ± 0.18 m^2; NS). The relation between BSA and PPM is due to the requirement of higher prosthetic valve size in patients with high BSA, which – higher valve size – is not always applicable to be inserted in small aortic annulus without aortic root enlargement.

In our study, the operative data of two groups was comparable between two groups. The type of cardioplegia used was uniform across both groups, with all patients receiving cold cardioplegia. The cross-clamp duration revealed no significant difference between the no PPM and PPM groups, with a mean of 62.0 ± 15.7 and 61.2 ± 15.4 min ($P = 0.9$). The total bypass time was also comparable between groups (mean ± SD, 85.3 ± 19.0 min for no PPM vs. 85.1 ± 19.2 min for PPM; $P > 0.9$). Blood loss during surgery showed no statistically significant difference. The studies of Tasca *et al.*[2] and Alassal *et al.*[7] showed no differences between the two groups according to previous parameters.

All patients in our study in both groups received a St Jude Regent valve. The valve size distribution differed significantly between the groups ($P < 0.001$); in the no PPM group, 4 % received a 19 mm valve, 50 % a 21 mm valve and 46 % a 23 mm valve. Conversely, in the PPM group, 90 % received

a 19 mm valve, 10 % received a 21 mm valve and no patients received 23 mm valve. In the study of Tasca *et al.*[2], patient distribution regarding prosthesis size was 19 mm: 38 (34.8 %) patients, 21 mm: 39 (35.8 %) patients, 23 mm: 29 (26.6 %) patients and 25 mm: three (2.7 %) patients. On the other hand, the study of Alassal *et al.*[7] showed equal distribution of prosthesis size in the two groups. On the contrary, the mechanical valves that were used in the study of Kato *et al.*[8] consisted of Medtronic Hall (n = 16), SJM Standard (n = 79), SJM Hemodynamic Plus (n = 5) and SJM Regent valves (n = 1). The stented tissue bioprostheses consisted of Hancock II porcine valves (n = 7), Medtronic Mosaic porcine valves (n = 5) and Carpentier-Edwards pericardial valves (n = 26). Freestyle stentless valves were used in seven patients. The study of Roscitano *et al.*[9] showed prosthestic valves distribution as follows. The implanted prostheses were Carbomedics bileaflet (20 cases), St Jude Medical bileaflet (64 cases) and Sorin Bicarbon bileaflet (four cases). Prosthesis sizes were 17 mm in four patients, 19 mm in 20 patients, 21 mm in 48 patients and 23 mm in 16 patients.

The EOA and IEOA in our study both demonstrated significant reductions in the PPM group compared with the no PPM group (EOA mean ± SD: no PPM, 2.1 ± 0.2 cm^2 vs. PPM, 1.6 ± 0.1 cm^2; IEOA mean ± SD: no PPM, 1.1 ± 0.1 cm^2/m^2 vs. PPM, 0.8 ± 0.0 cm^2/m^2; both $P < 0.001$), in a subgroup analysis stratified by valve size, IEOA differed significantly among patients without PPM ($P < 0.001$). Among no PPM patients, those with a 19 mm valve (N = 2) had an IEOA mean of 0.89 ± 0.02, while those with a 21 mm valve (N = 25) had a mean of 1.02 ± 0.06. Patients with a 23 mm valve (N = 23) had a mean of 1.18 ± 0.08. In the PPM cohort, there was also a significant difference in IEOA between the 19 and 21 mm valve sizes ($P = 0.010$). For the 19 mm valve size (N = 45), the mean IEOA was 0.79 ± 0.03. For the 21 mm valve size (N = 5), the mean IEOA was 0.82 ± 0.01.

On the other hand, the study of Alassal *et al.*[7] showed that the overall incidence of PPM was 25 % (24/96) and a moderate degree of PPM was noted in these cases (IEOA between 0.65 and 0.85 cm^2/m^2). The mean IEOA in patients with a 19-mm valve prosthesis was similar in both groups, whereas, in patients with a 21-mm valve prosthesis or larger, the IEOA was significantly higher in those with no PPM (group A) compared to group B. The mean IEOA increased with valve size in group A ($P < 0.0001$). The study of Tasca *et al.*[2] showed lower projected and postoperative IEOAs and higher postoperative peak and mean transprosthetic pressure gradients. The average postoperative IEOA tended to be slightly lower than the projected IEOA.

We found no significant differences in the immediate postoperative course regarding postoperative outcomes. MV duration, ICU stay, blood loss and hospital stay did not differ significantly between the groups. The mean MV duration was 12.1 ± 8.8 h for the no PPM group and 10.3 ± 5.2 h for the PPM group ($P = 0.4$). The ICU stay was 2.3 ± 2.6 days for no PPM and 2.2 ± 1.0 days for PPM patients ($P = 0.13$). Blood loss was comparable between the groups (mean ± SD: no PPM, 284.0 ± 134.2 ml vs. PPM, 268.0 ± 108.7 ml; $P = 0.7$). Similarly, the length of hospital stay was not significantly different (mean ± SD: no PPM, 7.2 ± 3.1 days vs. PPM, 6.9 ± 1.8 days; $P = 0.9$).

In the comparative analysis of echocardiographic parameters 1-year post-AVR in our study, changes in LVEDD post-AVR were greater in the no PPM group (3.7 ± 3.0 mm) versus the PPM group (1.1 ± 1.7 mm, $P < 0.001$). The reduction in LVESD followed a similar pattern (no PPM: 2.0 ± 3.7 mm vs. PPM: 0.5 ± 3.4 mm, $P < 0.001$). SWT differences were more pronounced in the no PPM group (2.4 ± 1.1 mm) compared to the PPM group (1.2 ± 0.8 mm, $P < 0.001$), as were the changes in PWT (no PPM: 2.2 ± 1.2 mm vs. PPM: 1.0 ± 0.7 mm, $P < 0.001$). In the study of Tasca *et al.*[2], the preoperative and postoperative values and the absolute and relative changes in LVM and function, are shown. Overall, interventricular septum thickness, LV PWT, LV internal dimension, LVM and LVM index all decreased significantly after AVR. However, the pattern of LV remodeling was different in the two groups, with a lesser decrease in LV internal dimension in patients with PPM.

The transvalvular gradient is still widely used as a guide to AVR in aortic stenosis and the hemodynamic advantage of AVR arises from its ability to minimize postoperative gradients and favor the normalization of LVM and function. The increased transvalvular gradient associated with PPM has resulted in increased LV work, which in turn influences the regression of LV hypertrophy.

Based on previous data, we investigated the changes in transvalve gradient post-AVR. In our study, the mean aortic valve gradient and peak gradient 1-year postsurgery were statistically significant (mean gradient: no PPM 10.9 ± 2.7 mmHg vs. PPM 17.5 ± 3.2 mmHg, $P = 0.001$; peak gradient: no PPM 21.1 ± 4.2 mmHg vs. PPM 29.6 ± 4.3 mmHg, $P = 0.001$), while the study of Tasca *et al.*[2] showed significant differences in mean aortic valve gradient and peak gradient 1-year postsurgery (mean gradient: no PPM 13.5 ± 4.4 mmHg vs. PPM 19.8 ± 5.9 mmHg, P=0.04; peak gradient: no PPM 23.8 ± 7.5 mmHg vs. PPM 33.2 ± 9.7 mmHg, $P = 0.01$). On the

other hand, the study of Alassal *et al.*[7] showed a significant difference in transvalve gradient between the two groups as follows (mean gradient: no PPM 12.20 ± 6.12 mmHg vs. PPM 20.15 ± 3.46 mmHg, $P = 0.001$; peak gradient: no PPM 19.21 ± 9.45 mmHg vs. PPM 31.43 ± 11.42 mmHg, $P = 0.001$).

Our study observed significant differences between patients without PPM and those with PPM. LVM showed a marked absolute regression in the no PPM group (87.0 ± 30.6 g) compared to the PPM group (39.1 ± 15.9 g, $P < 0.001$). Relative LVM regression also differed significantly between groups, with the no PPM group exhibiting a mean regression of 32.3 ± 8.4 % compared to 14.8 ± 5.7 % in the PPM group ($P < 0.001$). In the study of Tasca *et al.*[2], overall, interventricular septum thickness, LV PWT, LV internal dimension, LVM and LVM index all decreased significantly after AVR. However, the pattern of LV remodeling was different in the two groups, with a lesser decrease in LV internal dimension in patients with PPM. LVM showed a marked absolute regression in the no PPM group (77.0 ± 49 g) compared to the PPM group (48 ± 47 g, $P < 0.001$). The study of Kato *et al.*[8] showed a significant difference between the two groups. LVM showed a marked absolute regression in the no PPM group (91 ± 86 g) compared to the PPM group (57 ± 60 g, $P < 0.001$). On the contrary, the study of Roscitano *et al.*[9] showed a trend for lower postoperative LVM, postoperative LMVI, LVM index relative and absolute regression, but these differences were not statistically significant. LVM regression in the no PPM group (132.5 ± 62.6 g) compared to the PPM group (112.9 ± 78.5 g, $P = 0.51$).

Based on the previous data, the results of our study agree with the findings of other studies showing significant LVM regression 1-year postoperatively. From this study, we can conclude that PPM is a frequent problem in patients undergoing AVR due to aortic valve stenosis. It leads to a higher transprosthetic gradient and impaired LVM regression. A small-sized valve prosthesis does not necessarily result in PPM and may be perfectly adequate in a patient with a small body size.

CONCLUSION

Our study shows that in patients with pure aortic stenosis, PPM may hamper the regression of LVM after AVR. These findings may have important clinical implications given that PPM is frequent in these patients and, as opposed to other risk factors, can be avoided with a preventive strategy at the time of operation.

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Authors' contributions: Amr A. Ibrahim has participated in the conception and design of study, acquisition of data, analysis and interpretation of data; and has been involved in drafting of the manuscript. Mohamed Attia agrees to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Ayman Ammar has participated in the conception and design of study. Tamer Hikal have been involved in revising the manuscript critically for important intellectual content and in the interpretation of data.

CONFLICT OF INTEREST

There are no conflicts of interest.

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