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ORIGINAL ARTICLE

Aortic Prosthesis-Patient Mismatch as a Risk Factor on Regression of Left Ventricular Mass

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ABSTRACT

Objective: Prosthesis-patient mismatch (PPM) of the aortic prosthesis is frequent trouble following aortic valve replacement (AVR). The residual high transprosthetic pressure gradient (PG) could interrupt left ventricular mass regression (LVMR). The purpose of this study was to assess the frequency of PPM as well as its impact on LVMR postoperatively. **Methods:** 106 patients were prospectively evaluated after AVR for aortic stenosis (AS) from Jan 2016 to Dec 2021. Patients were classified into three groups based on the effective orifice area index (EOAI) (cm^2/m^2); Group A (≥0.85), B (0.8-0.84), and C (< 0.8). Follow-up of LVMR was performed postoperatively on all patients after six months. **Results**: We didn't record significant changes in basic data among studied groups away from the basal surface area, which was substantially higher in group C (Mean 2.4, P<0.001). The PPM incidence was observed in 30 survivors (28.3%); 12 survivors in group B and 18 survivors in group C. All groups showed a substantial reduction in posterior wall thickness (P<0.001). However, a significant decrease in peak and mean PG, LVM index, and diameter of the interventricular septum was only observed in groups A and B. **Conclusions:** PPM with EOAI below 0.85 cm^2/m^2 leads to high residual transprosthetic PG and subsequent impairment in LVMR. Therefore, meticulous choosing of the valve size with the availability of alternative options could prevent PPM squeal.

Keywords: Aortic valve replacement, Patient prosthesis mismatch, Prosthesis, LV, PPM

INTRODUCTION

Aortic valve stenosis (AS) is the most frequent valvular illness **[1]**. The current prevalence of AS is 4-7% in patients more than 65 years of age **[2]**. Aortic valve progressive narrowing and left ventricular hypertrophy (LVH) are the hallmarks of AS. LVH has been linked to a great risk of arrhythmia, heart failure, and cardiovascular mortality **[3]**.

Aortic valve replacement (AVR) is still the golden key for symptomatic AS. AVR relieves mechanical stenosis with subsequent normalization of the pressure gradient (PG) and regression of LVH **[4]**. It is a crucial goal to normalize LV mass (LVM) to avoid the increased risk of death **[5]**.

Valve prosthesis-patient mismatch (PPM) is considered if the prosthetic effective orifice area (EOA) is excessively narrow compared to the body surface area (BSA). If EOA index (EOAI) is below 0.85 cm^2/m^2, the PPM will develop **[6]**. PPM is a prevalent obstacle with AVR, with an incidence of 44% **[7]**. PPM increases PG across the replaced valve despite functioning well **[8]**.

There is a debate whether PPM is tolerable or not. However, failure of LV mass regression (LVMR) remains a major concern, mainly caused by the residual high-pressure gradient **[9]**. Predictors of adverse events associated with PPM involve severe LVH, old age, LV dysfunction, and concomitant coronary artery **[10,11]**. Our goal in

this study was to estimate the PPM prevalence and the impact of EOAI on LVM.

METHODS

This cohort study prospectively evaluated 106 survivors with pure stenosis of the aortic valve who underwent AVR at Zagazig University between Jan 2016 to December 2021. All participants provided written informed consent, and the study was approved by the Faculty of Medicine, Zagazig University's ethical research committee. The study was conducted by the World Medical Association's Code of Ethics (Declaration of Helsinki) for human studies.

The study ruled on all patients with AVR for severe AS; pure or concomitant less than mild AR with normal LV function, the normal average pressure of pulmonary (≤ 25 mm Hg) **[12]**, and normal sinus rhythm.

We excluded severe AS patients with decreased flow decreased PG; defined as mean systolic pressure gradient (SPG) < 40 mmHg with aortic valve area < 1 cm^2 **[13]**, previous cardiac surgery, concomitant other valve or coronary surgery, aortic root enlargement requiring replacement defined as aortic root diameter more than forty-five mm in Marfan syndrome, fifty mm in the bicuspid aortic valve, or fifty-five mm in the remaining patients **[13]**, systolic dysfunction (left ventricular ejection fraction (LVEF) below fifty %) **[13]**, pulmonary hypertension defined as mean more than 25 mmHg(12), postoperative mortality within six months, atrial fibrillation, or patients with resistant hypertension (systolic > 140 mmHg and/or diastolic > 90 mmHg despite a maximally tolerated dose of three anti-hypertensive drugs including diuretics **[14]**.

We recorded age, gender, basal surface area (BSA), and risk profile, including diabetes mellitus, hypertension, chronic kidney disease, dyslipidemia, and smoking. Pre-operative echocardiographic evaluation confirmed severe AS (mean SPG > 40 mmHg with aortic valve area < 1 cm^2 [(13)]). We recorded the following echocardiographic parameters for all patients preoperative and six months after AVR: LV dimensions (end-systolic and end-diastolic diameters; LVESD and LVEDD), LVEF, end-diastolic interventricular septum diameter (IVSD), end-diastolic posterior wall thickness (PWT), mean and peak SPG across the aortic valve, LVM [LVM= 1.04 ([LVEDD + PWTD + IVSTD]^3- [LVEDD]^3) -13.6 g**[15]**], and LVM index (LVMI) [LVM/ BSA **[16]**].

Preoperative coronary angiography was performed to exclude epicardial coronary artery stenosis > 50%, requiring a coronary artery bypass graft **[13]**.

Bileaflet St. Jude Medical valve prosthesis (Regent or Master) was used for all patients, and the valve size was recorded. Postoperative echocardiography was performed to calculate the EOA of the aortic prosthesis as follows **[17]**:

EOA = A (LVOT) x VTI (LVOT) / VTI (prosthetic valve)

LVOT is the LV outflow tract calculated by; (LVOTdiameter2 x 0.78540). VTI is the LVOT velocity-time integral measured by tracking pulsed wave doppler signal at LVOT at apical 5 chamber view. VTI (valve prosthesis) calculated by tracking continuous-wave Doppler signal across the aortic prosthesis in apical 5 chamber view. EOAIis measured by; EOA / BSA.

Hospital survivors were divided into three groups regarding EOAI as measured by postoperative echocardiography in the 1st outpatient clinic visit. Group A; EOAI ≥ 0.85 cm^2/m^2. Group B; EOAI 0.8 - 0.84 cm^2/m^2. Group C; EOAI < 0.80 cm^2/m^2. All survivors were booked for the postoperative outpatient clinic, where the echocardiographic data in the 6th month were collected. LVMR, LVMI, delta change in LVMI [DLVMI; the difference between preoperative and postoperative], and other echocardiographic data were studied in the 3 groups.

Statistic analysis:

Records were evaluated using SPSS (v. 18, USA). The parametric data were presented as mean ± SD. The comparisons between baseline and six-month follow-up data were performed using Paired student's t-test for parametric. Comparison between the three groups was assessed using one-way ANOVA. Categorical data were shown by frequency and incidence. Categorical data were compared using the chi-square test. The level of significance will be identified at P ≤ 0.05.

RESULTS

The baseline presentations of 106 survivors are listed in table 1. The study revealed a PPM incidence of 28.3%; most PPM patients (18/30, 60%) were severe, and moderate PPM was observed in 18 patients (40%). There was no substantial discrepancy among the three groups regarding age, EF, and NYHA class. The mean BSA was significantly bigger in group C (2.4 Kg/m2) compared to the other two groups (A; 1.9 Kg/m2, B; 2.2 Kg/m2).

The size distribution of the valve prosthesis in the three groups has a significant P-value of less than 0.001, which is presented in table 2.

The correlation between EOAI with valve size is shown in table 3. All prosthetic sizes (from 19 mm to 25 mm) had a significant association with EOAI with a P-value of less than 0.001.

The perioperative echocardiographic impact of PPM on LVM was reported in table 4. Significant mean LVMR was recorded in groups A and B (128 g and 66 g; P<0.001), but this reduction was nonsignificant in group C (15 g; P=0.8). The mean LVMI showed a significant drop postoperatively in groups A and B to become 139.8 g/m^2 and 113.6 g/m^2; P<0.001 with a nonsignificant drop-in group C (128 g/m^2; P=0.08). DLVMI showed a significant difference between groups A and C. The average peak and mean PG and IVSD presented a significant P-value of < 0.001 in groups A and B, without substantial change in group C. The mean PWT regression was significant after AVR in the three groups. We did not report any substantial change regarding LV dimensions or EF postoperatively.

Postoperative improvement in NYHA functional class for the three groups was scheduled in table 5. We recorded a significant clinical improvement built upon slopped NYHA class in the three groups.

Table (1): Baseline characteristic of 106 patients undergoing aortic valve replacement:

Variables	Group A (n=76)	Group B (n=12)	Group C (n=18)	P
Age (years)	39.7 ± 9.8 (23-59)	38 ± 9.3 (24-54)	35.1 ± 10.2 (23-59)	0.196
Male	33 (43.4%)	6 (50%)	5(27.7%)	P=0.39
Female	43 (56.6%)	6 (50%)	13 (72.3%)	
BSA (Kg/m^2)	1.9 ± 0.27	2.2 ± 0.32 a	2.4 ± 0.18 a	<0.001*
DM (n, %)	40 (52.6%)	5 (41.7%)	13 (72.2%)	0.2
HTN (n, %)	28 (36.8%)	3 (25%)	5 (27.8%)	0.6
CKD	15 (19.7%)	1 (8.3%)	3 (16.7%)	0.6
Dyslipidemia	24 (31.6%)	5 (41.7%)	6 (33.3%)	0.8
Smoking	45 (59.2%)	8 (66.7	12 (66.7%)	0.8
Preoperative EF	57.3 ± 5.1 (50-69)	55 ± 3.8 (50-64)	55.5 ± 4.7 (50-68)	0.15
Preoperative NYHA	2.1 ± 3.2 (2-3)	2.3 ± 0.52 (2-3)	2.2 ±0.46 (2-3)	0.89

* significant. [a] significant difference with group A BSA basal surface area. BM diabetes mellitus. HTN hypertension. CKD chronic kidney disease. . EF ejection Fraction. NYHA New York Heart Association.

Table (2): Operative data of 106 patients undergoing aortic valve replacement:

Valve size	Group A (n=76)	Group B (n=12)	Group C (n=18)	P
19 mm	28 (36.8%)	2 (16.7%)	1 (5.5%)	P<0.001*
21 mm	35 (46.1%)	4 (33.3%)	5 (27.8%)	
23 mm	11 (14.5%)	4 (33.3%)	9 (50%)	
25 mm	2 (2.6%)	2 (16.7)	3 (16.7)	

* significant.

Table (3): Indexed effective orifice area according to the size of aortic valve prosthesis:

Valve size	Group A (n=76)	Group B (n=12)	Group C (n=18)	P
19 mm	0.96 ± 0.1 (0.85 – 1.21)	0.81 ± 0.0 [a] (0.81 – 0.81)	0.74 ± 0.0 [a] (0.74)	<0.001*
21 mm	1.09 ± 0.16 (0.87 – 1.34)	0.8 ± 0.01 [a] (0.8 – 0.83)	0.78 ± 0.007 [a] (0.78 -0.79)	<0.001*
23 mm	1.1 ± 0.07 (1.04 – 1.24)	0.82 ± 0.01 [a] (0.81 – 0.84)	0.71 ± 0.02 [ab] (0.7 – 0.75)	<0.001*
25 mm	0.98 ± 0.16 (0.86 -1.1)	0.8 ± 0.0 [a] (0.8 – 0.8)	0.75 ± 0.03 [a] (0.72 -0.79)	<0.001*

[a] significant difference with group A. [b] significant difference with group B. * significant.

Table (4): Impact of prosthesis-patient mismatch on left ventricular function and left ventricular mass regression:

Variables	Group A (n=76)			Group B (n=12)			Group C (n=18)		
	Preoperative	Postoperative	P	Preoperative	Postoperative	P	Preoperative	Postoperative	P
LVM (g)	315 ± 54.5	257 ± 50.3	<0.001*	319.5 ± 99.5	268.2 ± 81.9	<0.001*	386.9 ± 83	341 ± 73.6	0.07
LVMI ($g.m^{-2}$)	167 ± 30.6	134.5 ± 25	<0.001*	143 ± 35	119.9 ± 26.3	<0.004*	160.4 ± 55.2	141.4 ± 28.7	0.07
DLVMI	32.8 ± 14.4			23.2 ± 22			13.4 ± 22.4[a]		0.001*
Mean PG (mmHg)	54.3 ± 11.4	14.9 ± 4.2	<0.001*	84.4 ± 12.5	27.5 ± 1.8	<0.001*	90.9 ± 7.2	33.9 ± 2.3	0.07
Peak PG (mmHg)	75.8 ± 15.2	23.2 ± 6.7	<0.001*	111.7 ± 15.7	41.5 ± 2.4	<0.001*	118 ± 6.5	43.3 ± 3.6	0.06
LVEDD (mm)	52.1 ± 6.9	49.7 ± 6.5	0.08	51.08 ± 5.6	49.2 ± 5.7	0.1	47.8 5.6	46.9 ± 5.2	0.8
LVESD (mm)	32.8 ± 5.8	30.5 ± 5.8	0.07	31.5 ± 4.4	29.1 ± 4.4	0.7	29.3 ± 4.5	28.5 ± 4.5	0.7
EF %	56.1 ± 5.1	58.2. ± 4.9	0.09	54 ± 1.8	56.2 ± 3.6	0.09	55.5 ± 4.7	57.6 ± 4.6	0.08
PWT(mm)	14.1 ± 2.4	9.2 ± 1.4	<0.001*	14.5 ± 1.8	11.8 ± 1.7	<0.001*	15.6 ± 2.3	12.4 ± 1.6	<0.001*
IVSD(mm)	14.2 ± 2.7	9.3 ± 1.4	<0.001*	15 ± 2.04	12.1 ± 1.9	<0.001*	16.1 ± 2.4	13.8 ± 2.06	0.07

* significant. [a] significant difference with group A. LVM left ventricular mass. LVMI left ventricular mass index. DLVMI delta-change in LVMI. PG pressure gradient. LVEDD left ventricular end-diastolic diameter. LVESD left ventricular end-systolic diameter. EF ejection fraction. PWT posterior wall thickness. IVSD interventricular septal diameter.

Table (4): NYHA class correlation between the 3 groups; preoperative and postoperative.

Groups	Preoperative	Postoperative	P
Group A (n=76)	Class II 49 (64.5%) Class III 16 (21%) Class IV 11 (14.5%)	Class I 68 (89.5%) Class II 8 (10.5%)	0.001*
Group B (n=12)	Class II 7 (58.3%) Class III 3 (25%) Class IV 2 (16.7%)	Class I 8 (66.7%) Class II 4 (33.3%)	0.002*
Group C (n=18)	Class II 9 (50%) Class III 7 (38.9%) Class IV 2 (11.1%)	Class I 11 (61.1%) Class II 7 (38.9%)	0.05*

NYHA New York Heart Association. * Significant

DISCUSSION

The most common valvular disease is AS, and LVH is typically observed as a compensatory mechanism of pressure overload **[1]**. AVR is the standard treatment that could unload the left ventricle, permit LVMR, and prolong survival **[18]**. Transcatheter AVR is a current alternative in patients with high surgical risk with comparable efficacy **[19]**.

PPM occurs after AVR when the prosthetic EOA is small enough regarding the BSA, a term primarily defined by Rahimtoola in 1978 **[20]**. PPM is presented by EOAI, formulated by prosthetic EOA / BSA of the patient, where each valve type has its EOA **[21]**.

Hernández-Vaquero and his associates recorded that PPM has no impact on the major adverse outcomes of patients in their 20s and 30s **[22]**. However, decreased LVMR remains the principal complication detector after AVR **[23]**. Therefore, we evaluated the impact of PPM on LVMR after a six-month follow-up.

We recorded an overall PPM incidence of 28.3% in the current study. Of them, 40% had EOAI between 0.8 and 0.85 cm^2/m^2 and 60% had EOAI below 0.8 cm^2/m^2. Failure of LVMR was reported in patients with EOAI < 0.8 cm^2/m^2. We recorded a substantial decrease in PWT in the three groups. But only survivors with EOAI > 0.8 cm^2/m^2 demonstrated a substantial decrease in SPG (peak and mean), LVM, LVMI, and IVSD. However, all groups showed a significant improvement in postoperative NYHA class. Regarding basic data and risk factors, patients in the three groups are comparable.

Regarding the incidence of PPM, Kim et al. reported significant PPM in 27.6% of patients and severe PPM in (1.3%) **[24]**. Furthermore, Alassal et al. recorded that one-quarter of the patients had a moderate degree of PPM, and none had severe PPM **[25]**. Also, Zhang et al. demonstrated that The majority of PPM survivors (83/91, 91.2%) had moderate PPM (EOAI 0.65-0.85 cm2/m2), and eight survivors had severe PPM (EOAI <0.65 cm2/m2) (8.8 %) **[26]**.

In concordance with our study, Tao et al. demonstrated that age did not vary between the PPM group and non-PPM group, but most of the PPM group were females **[27]**. Zhang et al. showed that PPM-positive patients were considerably younger than PPM-negative patients **[26]**. However, Kim et al. reported that PPM patients were older, had elevated BSA, increased body mass index, and raised hypertension **[24]**.

In terms of LVMR, Tasca, Alassal, Kim, Tao, Iqbal, and their coworkers reported a reduction of IVSD, PWT, LVM, and LVMI and a drop in SPG in all groups but the lower reduction was observed in patients with PPM. In multivariate evaluation, higher EOAI (i.e., a lower PPM level), women incidence, and baseline LVM were independent prediction models of more LVMR. However, in concordance with our study, most studies recorded a substantial improvement in NYHA class even with severe PPM **[24,25,27,29]**.

We can summarise that PPM is a frequently complex challenge that often complicates the implantation of the aortic prosthesis for AS. It yields an elevated PG across the prosthesis and disrupted LVMR. So we have to suspect PPM with persistent manifestations, especially if a small EOA is detected and elevated PG or velocity across the prosthesis postoperatively. Insertion of a prosthesis with a small size will not frequently deliver PPM, and it can do well in a patient with small BSA. Whereas with bigger BSA patients, the PPM will be more suspected as they require more stroke volume, special attention should be given to them as they are

at the risk of PPM. The projected IEOA must be routinely measured intraoperative to detect the PPM risk. If PPM is suspected, we should shift to alternative measures such as a bigger-sized prosthesis and/or enlargement of the annulus guided by clinical presentation and benefit/risk ratio.

Our study had many strong points; it was a prospective cohort study that was performed by one type of aortic prosthesis. Confounders of LVMR failure were matched among our study groups. However, our study limitations are the relatively small sample size, only six months of follow-up, and two dimensions of echocardiographic use. We recommend larger-scale studies with longer follow-ups depending on cardiac magnetic resonance (CMR) to verify the true impact of PPM on LVMR.

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