Impact of Prosthesis-Patient Mismatch after Mitral Valve Replacement

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Background and aim of the study: **The study aim was to determine the impact of prosthesis-patient mismatch (PPM) on early and late clinical outcomes, left atrial and ventricular remodeling, late tricuspid valve regurgitation and pulmonary hypertension (PH) in patients after mitral valve replacement (MVR).**

Methods: **A total of 46 patients (mean age 66 ± 9.3 years) with mitral valve diseases and undergoing isolated MVR was enrolled in the study. The mitral valve effective orifice area (EOA) was determined using the continuity equation and indexed for the patient's body surface area (EOAi). PPM was defined as EOAi ≤1.2 cm^2/m^2. PH was defined as a systolic pulmonary artery pressure (sPAP) >40 mmHg. Both, clinical and echocardiographic follow up were performed.**

Results: **PPM was identified in 25% of patients, but no significant differences were observed in baseline and operative characteristics when comparing patients with and without PPM. The NYHA class was improved in most cases after surgery. Indeed, significant decreases in mean transvalvular gradient (from 8.6 ± 2.8 mmHg to 5 ± 1.3 mmHg, p = 0.001), left atrial dimension (LAD) (from 31.9 ± 9.8 mm to 29.5 ± 7 mm, p = 0.011), left ventricular end-systolic diameter (from 42.6 ± 18.1 mm to 35.5 ± 6.6 mm, p = 0.044) and left ventricular end-diastolic diameter (from 55.8 ± 19.2 mm to 48.7 ± 6.1 mm, p = 0.024) were observed over time when comparing preoperative and postoperative echocardiographic data. In addition, at follow up (mean 6.9 ± 1.8 years) there were significant decreases in LAD (from 31.9 ± 9.8 mm to 28 ± 11.1 mm, p = 0.001), left ventricular end-diastolic volume (from 106.9 ± 32.9 ml to 92.3 ± 21.9 ml, p = 0.024), tricuspid regurgitation (TR) (from 87% to 27%, p = 0.002) and PH (from 78.3% to 58.7%, p = 0.043) in all patients. No significant differences were observed in hemodynamic, clinical outcome and atrial natriuretic peptide levels of patients with and without PPM.**

Conclusion: **Mitral PPM does not appear to have any negative effect on ventricular and atrial remodeling, TR and PH during the early and late postoperative periods.**

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Prosthesis-patient mismatch (PPM) occurs when the effective orifice area (EOA) of the prosthetic valve is too small relative to the patient's body surface area, resulting in an increased postoperative transvalvular gradient (1). Whilst previous extensive reports have investigated the role of PPM following aortic valve replacement (2,3), PPM following mitral valve replacement (MVR) has been examined to a much lesser degree (4). Mitral PPM is not uncommon, and has been observed in 30-85% cases after MVR (5,6). In the light of these observation, a pilot study was performed to investigate the impact of this phenomenon on clinical outcome, including left atrial and ventricular remodeling, late tricuspid valve regurgitation and pulmonary hypertension (PH), during the early and late postoperative periods in patients with mitral valve disease and who had undergone isolated MVR.

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Clinical material and methods

Patients

A consecutive series of 117 patients who underwent isolated MVR to treat mitral valve disease at the Unit of Cardiac Surgery of Palermo University between August 2003 and June 2011 was retrospectively reviewed. All patients who had undergone emergency surgery, previous cardiac surgery, concomitant coronary artery bypass grafting (CABG) or other valvular procedures were excluded. Subsequently, 46 patients who underwent echocardiographic follow up (at a mean of 6.9 ± 1.8 years) were included in the study. All data were collected prospectively and recorded in an electronic database; the clinical follow up was completed at routine out-patient clinics.
Approval to conduct the study was granted by the Institutional Review Board of the University of Palermo, and the need for patients to provide their consent to participate was waived.

Surgical technique

All surgical procedures were performed via a standard midline sternotomy and full cardiopulmonary bypass. Antegrade crystalloid cardioplegia was used as conventional myocardial protection strategy. The mitral valve was approached via a left atrial incision, with the native posterior leaflet of the subvalvular apparatus, or both, being preserved in all cases. All prostheses were implanted using interrupted everting 2-0 Ethibond pledget-supported sutures (Ethicon Inc., Somerville, NJ, USA) (except under specific conditions). The prostheses used in the present series were Carpentier-Edwards stented bioprostheses, Hancock bioprostheses, and St. Jude Medical Standard Mechanical prostheses.

Doppler echocardiographic assessment

Clinical and echocardiographic assessments were performed prior to MVR, at hospital discharge, and at follow up. At each visit, all patients underwent a complete M-mode, bi-dimensional and Doppler transthoracic echocardiographic assessment using a Sonos 7500 system (Phillips Medical Ultrasound). All echocardiographic studies were reviewed in a core laboratory and the results independently reviewed by two echocardiographers. The left ventricular ejection fraction (LVEF) was calculated using Simpson's biplane method. Tricuspid regurgitation (TR) was diagnosed using color Doppler measurements of the regurgitant jet flow area (7). Non-equivocal findings at color Doppler measurement of the tricuspid regurgitant jet flow area were assessed by means of vena contracta width and proximal isovelocity surface area (PISA) radius, and the TR degree was adjudicated consequently as TR >2+ if the vena contracta width was >6 mm and PISA radius >5 mm (7). The intra- and inter-observer variabilities for measurements of functional TR in the echocardiography core laboratory were 11 ± 6% and 10 ± 9%, respectively. The in-vivo prosthetic valve effective orifice area (EOA) was calculated using the continuity equation, with the stroke volume measured in the left ventricular outflow

Table I: Demographic and clinical characteristics of the patients (n = 46).

Variable	Value
Follow up (years)*	6.9 ± 1.8
Age (years)*	66 ± 9.3
Body weight (kg)*	69.5 ± 12.7
Height (cm)*	163 ± 9.5
BMI (kg/m^2)*	26.1 ± 4.2
BSA (m^2)*	1.7 ± 0.2
Creatinine (mg/dl)*	0.9 ± 0.2
Male gender	19 (41.3)
Female gender	27 (58.7)
Mortality	0 (0)
Emergency	2 (4.4)
Dilated cardiomyopathy	1 (2.2)
NYHA class	
I-II	16 (35)
III-IV	30 (65)
Smoker	
Ex-smoker	8 (17)
Yes	7 (15)
No	31 (68)
Hypertension	25 (54.4)
Diabetes	4 (8.7)
Renal failure	2 (4.4)
COPD	1 (2.2)
Atrial fibrillation	20 (43.5)
Cardiac failure	1 (2.2)
Cerebrovascular disease	7 (15.2)
Mitral lesion	
Regurgitation	22 (47.8)
Stenosis	6 (13.0)
Mixed	18 (39.1)
Tricuspid regurgitation	40 (87.0)
Tricuspid regurgitation grade	
Faint	3 (9)
Moderate	14 (35)
Severe	23 (56)
Pulmonary hypertension	36 (78)
Mitral dysfunction	
Prolapse	1 (2)
Myxomatous degeneration	11 (25)
Rheumatic	23 (51)
Calcific degeneration	5 (11)
Chordal rupture	5 (11)

*Values are mean ± SD.
Values in parentheses are percentages.
BMI: Body mass index; BSA: body surface area; COPD: Chronic obstructive pulmonary disease.

tract divided by the integral of the mitral valve transprosthetic velocity-time integral during diastole. The calculated values of EOA were compared with normal reference values of EOA provided for each type and size of prosthetic valve in the guidelines of the American Society of Echocardiography (8). The indexed EOA (EOAi) was calculated by dividing the measured EOA by the patient's body surface area (in m^2) at the time of follow up (9). PPM was defined as not clinically significant if the EOAi was >1.2 cm^2/m^2. A value of 1.2 cm^2/m^2 was selected as the threshold for PPM because other previous numerical and clinical studies have suggested that such a level of PPM is associated with moderate/severe PH at rest, and severe PH on mild/moderate physical exercise (10). The systolic pulmonary artery pressure (sPAP) was calculated by adding the systolic right ventricular pressure derived from the TR to the estimated right atrial pressure. The latter was estimated from the diameter and the degree of collapse of the inferior vena cava during inspiration. Pulmonary arterial hypertension was defined as a sPAP >40 mmHg (11).

Atrial natriuretic peptide (ANP) analysis

Blood samples were collected into tubes and centrifuged to obtain serum samples; the latter were stored at -80°C until undergoing enzyme-linked immunosorbent assay (ELISA) to determine ANP levels using a commercial kit (Cusabio, China). All assays were performed according to the manufacturer's instructions. In order to standardize results, reference preparations of ANP were employed in all assays. The results were expressed as pg/ml, with a detection limit of 15.6 pg/ml. Quantitative ANP concentrations were expressed as mean ± SD.

Statistical analysis

Continuous data were expressed as mean ± SD, and categorical data as percentages or frequencies. Differences between subgroups were compared using the chi-squared test or Fisher's exact test (if the expected cell frequencies were less than five) for categorical variables, and the *t*-test or Welch test or Wilcoxon rank-sum test, as appropriate, for continuous variables. Variables were checked for normality and homoscedasticity, and assumptions accepted when $p > 0.05$. To compare preoperative and postoperative characteristics a univariate analysis was performed using a paired *t*-test for quantitative variables and a test for proportions in case of qualitative variables. All reported p-values were two-sided, and a p-value <0.05 was considered to be statistically significant. All analyses were conducted using R 3.0.3 software.

Results

Demographic and clinical characteristics

A total of 46 patients (19 males, 27 females; mean age 66 ± 9.3 years) with mitral valve disease (rheumatic pathology 51%, myxomatous degeneration 25%, calcific degeneration 11%, chordal rupture 11%, prolapsed 1%) who had undergone isolated MVR was investigated. The mean body surface area was 1.7 ± 0.2 cm^2, and the mean body mass index 26.1 ± 4.2 kg/m^2. With regards to co-morbidities, most patients (68%) were non-smokers, 54.4% had hypertension, 8.7% had diabetes, 4.4% had renal failure, 15.2% had cerebrovascular disease and 2.2% had chronic obstructive pulmonary disease. At the time of surgery, 65% of patients were in NYHA classes III-IV and 35% were in NYHA classes I-II. Atrial fibrillation was present in 43.5% of patients. Only one patient had cardiac failure (see Table I).

Intraoperative data and early and late outcomes

A bioprosthesis was implanted in nine cases, and a mechanical prosthesis in 35. The mean valve size was 26.1 ± 1.8 mm. Among the patients, 25% had PPM (EOAi ≤1.2 cm^2/m^2) postoperatively. During the early postoperative period eight patients developed arrhythmias, and one patient had a cerebrovascular complication. The mean hospital length of stay was 10 ± 4.8 days. During the late postoperative period three patients had pulmonary failure, two had cerebrovascular complications, and one patient developed cardiac failure. Postoperatively, the NYHA class was improved in most cases (Table II).

Postoperative echocardiographic data

Significant decreases were observed over time by comparing preoperative and postoperative echocardiographic data. The mean transvalvular gradient was reduced from 8.6 ± 2.8 mmHg to 5 ± 1.3 mmHg ($p = 0.001$), the left atrial dimension (LAD) from 31.9 ± 9.8 mm to 29.5 ± 7 mm ($p = 0.011$), the left ventricular end-systolic diameter from 42.6 ± 18.1 mm to 35.5 ± 6.6 mm ($p = 0.044$), and the left ventricular end-diastolic diameter from 55.8 ± 19.2 mm to 48.7 ± 6.1 mm ($p = 0.024$). In addition, at follow up (mean 6.9 ± 1.8 years) there were significant decreases in the LAD (from 31.9 ± 9.8 mm to 28 ± 11.1 mm; $p = 0.001$), left ventricular end-diastolic volume (from 106.9 ± 32.9 ml to 92.3 ± 21.9 ml; $p = 0.024$), TR (from 87% to 27%; $p = 0.002$) and PH (from 78.3% to 58.7%; $p = 0.043$) in all patients (Table III).

There were no significant differences in baseline and operative characteristics between patients with and without PPM. Neither were any significant differences observed between patients with and without PPM

Table II: Intraoperative data and early and late outcomes of the patients (n = 46).

Parameter	Value
Intraoperative data	
Valve size (mm)	26.1 ± 1.8
Bioprosthesis (n)	9 (19.57)
Mechanical prosthesis (n)	37 (80.43)
Type of prosthesis (n)	
Carpentier-Edwards	4 (9)
Hancock	3 (6)
St. Jude Medical	37 (85)
Transfusion	28 (62.2)
Early postoperative outcomes	
Days in intensive care unit	2.7 ± 1.8
Days in hospital	10 ± 4.8
Creatinine (mg/dl)	0.8 ± 0.2
Arrhythmias (n)	8 (17.4)
Pulmonary failure (n)	0 (0.0)
Renal failure (n)	0(0.0)
Cerebrovascular complications (n)	1 (2.2)
Cardiac failure (n)	0 (0.0)
Thromboembolism (n)	0 (0.0)
Infections (n)	0 (0.0)
Late postoperative outcomes	
Pulmonary failure (n)	3 (6.5)
Renal failure (n)	0(0.0)
Cerebrovascular complications (n)	2 (4.3)
Cardiac failure (n)	1 (2.2)
Thromboembolism (n)	0 (0.0)
Infections (n)	0 (0.0)
Mortality (n) 0 (0.0)	
NYHA class I-II	42 (92)
NYHA class III-IV	4 (8)

*Values are mean ± SD.
Values in parentheses are percentages.

during the follow up assessment in terms of hemodynamic, clinical outcome and ANP levels (Table IV).

Discussion

Currently, clinical results relating PPM to the mitral valve remain controversial, with some research groups suggesting that mitral valve patients with PPM have a high recurrence of heart failure, their survival rate is worse, and their postoperative sPAP is higher (10-14). Others (15) have proposed that mitral valve-related PPM occurs less frequently, and that a smaller prosthesis has no negative effects on hemodynamic performance. Accordingly, it was observed in the present study that PPM does not appear to mediate any negative consequences on ventricular and atrial remodeling, TR and PH following MVR. In addition, PPM does not appear to have any negative effects on early and late postoperative outcomes. Thus, the consideration is that hemodynamic alterations after MVR are not completely related to the phenomenon of PPM. Other factors need to be considered. In all probability, a crucial role is evocated by the histological structure of the mitral valve apparatus, which reflects the genetic background of each patient. Another factor involved might also be the type of surgical technique used. In contrast, atrial and ventricular remodeling are significantly associated with the induction of molecular and cellular mechanisms determining fibrosis, and changes in the dimensions and function of the heart. Such mechanisms are related to the individual genetic assessment and the different mitral valve pathology and pathophysiology. This aspect is not considered in the majority of studies highlighting the negative effects of PPM. Likewise, the type of mitral valve

Table III: Preoperative and postoperative echocardiographic data.

Variable	Preoperative	Postoperative	p-value	Follow up	p-value
MPG (mmHg)	8.6 ± 2.8	5.0 ± 1.3	<0.001	5.1 ± 1.8	<0.001
EF	57.7 ± 8.6	55.5 ± 7.4	0.007	56.1 ± 9.1	0.206
LAD (mm)	31.9 ± 9.8	29.5 ± 7.0	0.011	28.0 ± 11.1	<0.001
LVEDD (mm)	55.8 ± 19.2	48.7 ± 6.1	0.024	51.3 ± 6.3	0.091
LVEDV (ml)	106.9 ± 32.9	88.7 ± 24.7	<0.001	92.3 ± 21.9	0.005
LVESD (mm)	42.6 ± 18.1	35.5 ± 6.6	0.015	40.6 ± 7.1	0.484
LVESV (ml)	40.8 ± 7.9	42.1 ± 4.9	0.300	42.8 ± 16.0	0.368
sPAP (mmHg)	44.3 ± 12.9	32.9 ± 7.6	<0.001	32.0 ± 6.1	<0.001
TR (n)	40 (87)	23 (50)		12 (27)	0.002
PH (n)	36 (78.3)	24 (52.2)	0.529	27 (58.7)	0.043

Values are mean ± SD.
Values in parentheses are percentages.
EF: Ejection fraction; LAD: Left atrial diameter; LVEDD: Left ventricular end-diastolic diameter;
LVESD: Left ventricular end-systolic diameter; MPG: Mean prosthetic gradient; PH: Pulmonary hypertension; sPAP: Systolic pulmonary artery pressure; TR: Tricuspid regurgitation.

Table IV: Preoperative and intraoperative characteristics of patients (n = 46) stratified according to patient-prosthesis mismatch (PPM) severity.

Variable	Moderate PPM	Severe PPM	p-value
Clinical variables			
Creatinine (mg/dl)*	0.9 ± 0.2	0.9 ± 0.3	0.624
Ejection fraction (%)*	62.8 ± 5.5	56.1 ± 8.8	0.024
Left atrial diameter (mm)*	30.7 ± 6.1	32.3 ± 10.8	0.887
LVEDD (mm)*	53.8 ± 16.9	56.5 ± 20	0.478
LVEDV (ml)*	109.5 ± 25.4	106.1 ± 35.2	0.767
LVESD (mm)*	42.2 ± 19.3	42.7 ± 18	0.651
LVESV (ml)*	41.4 ± 6.9	40.6 ± 8.3	0.785
sPAP (mmHg)*	39.9 ± 10.9	45.7 ± 13.3	0.345
Dilated cardiomyopathy (n)	0 (0)	1 (2.9)	1.000
Hypertension (n)	5 (45.5)	20 (57.1)	0.730
Diabetes (n)	1 (9.1)	3 (8.6)	1.000
Renal failure (n)	0 (0.0)	2 (5.7)	1.000
COPD (n)	0 (0.0)	1 (2.9)	1.000
Cerebrovascular disease	0 (0.0)	7 (20.0)	0.171
Atrial fibrillation (n)	5 (45.5)	15 (42.9)	1.000
Cardiac failure (n)	0 (0)	1 (2.9)	1.000
Mitral lesion (n)			
Regurgitation	6 (54.6)	16 (45.7)	
Mixed	4 (36.4)	14 (40.0)	
Stenosis	1 (9.1)	5 (14.3)	1.000
Tricuspid regurgitation (n)	10 (90.9)	30 (85.7)	1.000
Tricuspid regurgitation grade			0.588
Faint	5 (50)	17 (57)	
Moderate	4 (44)	9 (31)	
Severe	1 (10)	4 (12)	
Pulmonary hypertension (n)	8 (72.7)	28 (80)	0.682
Intraoperative variables			
Valve size (mm)*	27 ± 2	25.8 ± 1.6	0.022
Type of prosthesis (n)			0.268
Bioprosthesis	0 (0.0)	7 (20.0)	
Mechanical prosthesis	11 (100.0)	26 (74.3)	
Mitral repair	0 (0.0)	2 (5.7)	
Tricuspid valve surgery (n)	2 (18.2)	8 (22.9)	1.000
Late postoperative outcomes			
Pulmonary failure (n)	1 (9.1)	2 (5.7)	1.000
Renal failure (n)	0 (0)	0 (0)	-
Cerebrovascular complications (n)	0 (0)	2 (5.7)	1.000
Cardiac failure (n)	0 (0)	1 (2.9)	1.000
Thromboembolism (n)	0 (0)	0 (0)	-
Infections (n)	0 (0)	0 (0)	-
ANP (pg/ml)*	0.67 ± 0.98	0.43 ± 0.44	0.7231

*Values are mean ± SD.
Values in parentheses are percentages.
Abbreviations as Table III.

disease (stenosis or regurgitation) could lead to different changes in left ventricular function. Patients with mitral regurgitation often experience severe left ventricular enlargement and systolic dysfunction. Furthermore, each type of lesion may also be associated with specific risk factors, such as coronary artery disease, dilated cardiomyopathy and rheumatic myocarditis. Based on these observations, it is imperative that the pathophysiological status of the patients be considered, and consequently the deleterious effects of PPM might vary.

From a surgical point of view, it must be stressed that during MVR it is very important to preserve the subvalvular apparatus; indeed, this is crucial in order to maintain left ventricular function and long-term survival after MVR. Among the present patients, preservation of the subvalvular apparatus was performed many times, whereas most reported studies on PPM after MVR do not indicate whether the subvalvular apparatus was preserved, or not (12-14).

A further crucial point was the evaluation of PPM-related effects on early and late outcome. The present authors' opinion is that in patients undergoing MVR, in order to analyze the effects of PPM, other risk factors (e.g. reoperation, concomitant CABG, other valvular operations, emergency) that may affect long-term survival should also be taken into account, as recently noted by Aziz and colleagues (11). For example, in the present study PPM was associated with deleterious outcomes in older patients who underwent bioprosthetic valve replacement. However, almost one-quarter of the patients had undergone previous cardiac operations, and 52% had received concomitant CABG and/or aortic valve replacement in addition to PPM. These findings suggest that poor outcomes in patients with PPM could be explained by confounding factors other than PPM. For these reasons, in the present study patients who had undergone emergency operations, previous cardiac surgery, concomitant CABG or other valvular procedures, were excluded.

In two recent meta-analyses, discordant data were reported (15,16). In particular, in the study conducted by Zhang et al. (16), an analysis of eight retrospective cohort studies underlined that current evidence is insufficient to derive a definitive conclusion as to whether mitral PPM would affect long-term survival because of the biases and confounding factors that interfere with late clinical outcomes. Clearly, additional high-quality prospective studies are warranted to evaluate the impact of mitral PPM after MVR in the future.

Study limitations

The main limitations of the present study were its single-center, non-randomized nature, and the small number of patients included. As the retrospective nature of this evaluation would be suspect to all observer and selection biases inherent in such a study, further investigations will be required to confirm and extend these findings.

In conclusion, the results of the present pilot study suggest that PPM does not have any negative effects on clinical outcome after MVR. In particular, PPM did not seem to influence early and late clinical outcome after MVR. Based on these findings, it should be emphasized that it is not prosthesis size per se that determines PPM and subsequent pathological alterations, but rather its relation to the histological structure of the tissue, the individual genetic assessment, and the nature of the characteristic mitral disease of each patient. As a consequence, the use of body surface area as a standard guideline criterion for selecting the surgical approach and prosthesis size might appear inadequate. Clearly, further studies are needed to confirm and extend these findings, to modify this standard guideline criterion, and to consider the hypothesis to add among the guideline criteria for the management of mitral valve disease.

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