VIA MEDICA

ORIGINAL ARTICLE

Cardiology Journal
2009, Vol. 16, No. 1, pp. 68–72

ISSN 1897–5593

Sustained benefit of left ventricular remodelling after valve replacement for aortic stenosis

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Abstract

Background: *Valve replacement for aortic stenosis (AS) determines negative ventricular remodelling. We used cross sectional and Doppler echocardiography to check how rapidly it occurs and to assess if these changes are sustained over time.*

Methods: *We evaluated in 34 patients subjected to aortic valve replacement for AS morphological and functional (ejection fraction and E:A ratio) left ventricular data by echocardiography prior to surgery and 2 postoperative studies: early after surgery (pQ1) and at mid-term evolution (pQ2).*

Results: *Left ventricular mass index was reduced at pQ1 (from 152 ± 47 g/m^2 to 113 ± 31 g/m^2; $p < 0.01$) as well as end-diastolic (from 51.3 mm to 48.3 mm; $p < 0.03$), end-systolic (from 32.2 mm to 29.4 mm; $p < 0.02$), interventricular septum (from 12.9 mm to 10.3 mm; $p < 0.01$), and posterior wall (from 12.5 mm to 11 mm; $p < 0.01$) dimensions. Left ventricular ejection fraction (from 61.2% to 65.2%; $p < 0.04$) and E:A ratio (from 0.94 to 0.98; $p < 0.01$) increased significantly at pQ1. There were no significant differences in measurements between pQ1 and pQ2.*

Conclusions: *Aortic valve replacement surgery leads to a rapid negative left ventricular remodelling during the first 7 months, including a decrease in myocardial hypertrophy and an improvement in systolic and diastolic function. These beneficial hemodynamic changes are sustained for at least 3 years.* (Cardiol J 2009; 16: 68–72)

Key words: aortic stenosis, valve replacement, ventricular remodelling

Introduction

Aortic valve stenosis (AS) is a common disorder and the most frequently acquired valvular disease in developed countries. Left ventricular systolic pressure overload due to progressive AS leads to a marked myocardial hypertrophy and changes in left ventricular relaxation parameters (diastolic function) without left ventricular ejection fraction disturbances up to advanced evolutive stages.

Prosthetic aortic valve replacement (AVR) dramatically reduces this left ventricular pressure overload immediately after surgery, determining a marked left ventricular mass index decrease [1], a left ventricular ejection fraction (LVEF) [2], and diastolic function improvement [3]. Literature shows contradictory information about the chronological course of these changes [3, 4]. In this study we used cross-sectional and Doppler echocardiography to check this rapidly ventricular remodelling

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Received: 29.07.2008 Accepted: 29.10.2008

and to assess if these changes are sustained over time.

Methods

The study population included retrospectively consecutive survivor patients with isolated AS undergoing surgical aortic valve replacement between January 2003 and December 2006. All patients underwent cross-sectional and Doppler echocardiography 3 months before surgery (mean: 3 months, range 2–4 months), and were evaluated 2 times after AVR depending on clinical follow up indication in the outpatient department. Each patient underwent standard left-sided heart catheterization and hemodynamic study that confirmed severe isolated AS, prior to surgery.

All operations were performed by the same surgical team at another reference hospital.

All echocardiographic studies were performed by the same experienced work team (each team member with experience of more than 1000 previous studies) using the same equipment (Philips Envisor), both pre-operatively and during follow up. End-diastolic and end-systolic measurements of left ventricle internal dimensions, interventricular septal thickness, and posterior wall thickness were obtained according to the guidelines of the American Society of Echocardiography [5] in millimetres. The left ventricular mass index (LVMI) was calculated according to body surface area (m^2) as described by Deveroux et al. [6]. Left ventricular ejection fraction (LVEF) was estimated by Teicholz method [7]. Flow velocity profiles were obtained from apical projections of pulsed (transmitral flow) and continuous (transaortic flow) wave Doppler. We evaluate the E:A ratio measuring the maximum peak early (E) and atrial (A) transmitral flow velocities (cm/s) [8]. Peak instantaneous gradient and mean transaortic gradient (mm Hg) were measured by peak velocity and perimetry of the left ventricular outflow tract waveform, respectively.

The study was approved by the local bioethical committee and all patients gave their informed consent.

Statistical analysis

The statistical analysis was performed by computing the data (SPSS 13.0 software). All data were normally distributed by the normality test (Mann Whitney U test). Values are expressed as mean ± ± standard deviation for continuous variables. Paired comparisons between preoperative and postoperative studies were performed using the two-tailed Student-*t* test. The level of significance was established at $p < 0.05$.

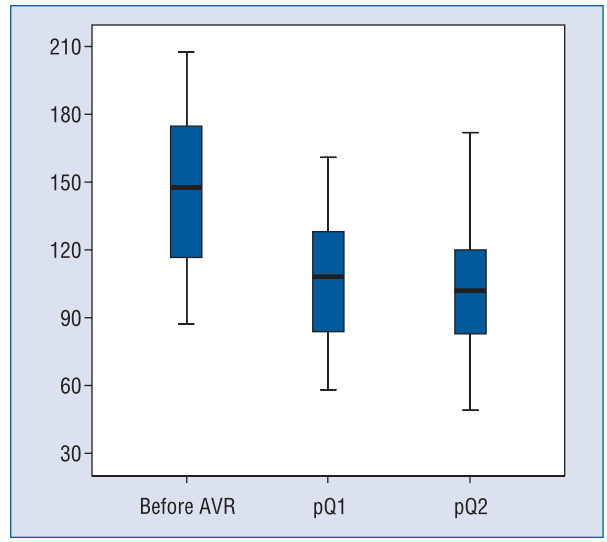

Figure 1. Early significant regression in left ventricular mass index [g/m^2] after aortic valve replacement (AVR), and sustained benefit in a long term observation (pQ1: 7 ± 6 months after surgery; pQ2: 32 ± 14 months after surgery).

Results

Thirty-four patients were enrolled (17 men and 17 women), with a mean age of 67.9 ± 8.8 years. Peak instantaneous gradient and mean transaortic gradient were 94.9 ± 28.4 mm Hg and 59.9 ± ± 17.1 mm Hg, respectively. Twelve patients received bioprostheses and 22 received mechanical prostheses. The early postoperative study (pQ1) was performed 7 ± 6 months after surgery, and the second postoperative study (pQ2) was performed 32 ± 14 months after surgery (Fig. 1). The main pre- and postoperative (pQ1 and pQ2) measurements are summarized in Table 1. Peak instantaneous and mean transaortic gradient showed a significant reduction. There were no significant changes in patient body surface area during the consecutive echocardiograms, and the LVMI calculation was undisturbed. With respect to baseline measurements, 64.7% (n = 22) normalized their LVMI, considering it as a decrease down to 135 g/m^2 in men and to 115 g/m^2 in women. LVMI was reduced markedly at pQ1 (from 152 ± 47 g/m^2 to 113 ± ± 31 g/m^2; $p < 0.01$) owing to a significant reduction in end diastolic (from 51.3 mm to 48.3 mm; $p < 0.03$), end systolic (from 32.2 mm to 29.4 mm; $p < 0.02$), interventricular septum (from 12.9 mm to 10.3 mm; $p < 0.01$), and posterior wall (from

Table 1. Results in consecutive echocardiographic studies and statistical significance of differences between them; AVR — aortic valve replacement; pQ1 — first echocardiogram after surgery; pQ2 — second echocardiogram after surgery; p_1 — statistical significance of differences between "Before AVR" and "pQ1"; p_2 — statistical significance of differences between "pQ1" and "pQ2".

	Before AVR	pQ1	p_1	pQ2	p_2
Body surface [m^2]	1.76 ± 0.16	1.76 ± 0.14	0.48	1.75 ± 0.14	0.92
Left ventricle end-diastolic diameter [mm]	51.3 ± 7.8	48.3 ± 6.9	0.03	49.3 ± 7.3	0.39
Left ventricle end-systolic diameter [mm]	32.2 ± 8.6	29.4 ± 4.9	0.02	28.6 ± 8.2	0.70
Interventricular septum [mm]	12.9 ± 2.4	10.3 ± 1.7	< 0.01	10.4 ± 2.2	0.90
Posterior wall [mm]	12.5 ± 1.8	11 ± 1.5	< 0.01	10.3 ± 1.9	0.02
Left ventricular ejection fraction (%)	61.2 ± 11.5	65.2 ± 8.7	0.04	62.9 ± 9.4	0.26
Left ventricular mass index [g/m^2]	152 ± 47	113 ± 31	< 0.01	112 ± 41	0.82
Peak gradient [mm Hg]	94.9 ± 28.4	25.8 ± 6.8	< 0.01	29.6 ± 11.2	0.04
Mean gradient [mm Hg]	59.9 ± 17.1	14.7 ± 4.4	< 0.01	16.1 ± 6	0.16
E:A ratio	0.94 ± 0.24	0.98 ± 0.3	< 0.01	0.99 ± 0.43	0.86

12.5 mm to 11 mm; $p < 0.01$) dimensions. Only 3 patients presented reduced LVEF (< 50%) before surgery. LVEF increased significantly at pQ1 (from 61.2% to 65.2%; $p < 0.04$). Abnormal left ventricular relaxation was found in 79.5% of patients with a E:A ratio < 1. E:A ratio increased at pQ1 (from 0.94 to 0.98; $p < 0.01$).

There were no significant differences in measurements between pQ1 and pQ2 (Table 1) except a sustained left ventricular posterior wall regression (11 mm a 10.3 mm; $p < 0.02$), and a trend towards higher peak instantaneous and mean transaortic gradients.

Discussion

Left ventricular pressure overload due to aortic valve stenosis leads to a marked hypertrophic response of the myocardium. Prosthetic aortic valve replacement immediately reduces the left ventricular pressure overload, conditioning a marked decrease in wall tension. This comes out into a negative left ventricular remodelling with evident LVMI regression with regard to a rapid ventricular dimension and myocardial thickness reduction, well-known during the first 12 months [1, 9–10] and sustained until 18 months after surgery [2, 4], independent of gender [4, 11], age (elderly patients) [12, 13], or ventricular functional status (non-preserved LVEF) [14]. However, this negative ventricular remodelling is not well referred over the first 18 months following AVR, and only evaluated by Lund et al. [15] for a long period (more than 10 years), suggesting a progressive increase in LVMI strongly associated with chronic high blood pressure. In this sense we can see how, in the midterm period (3 years) LVMI, left ventricular diameter and wall thickness remain stable after the regression shown during the first months following surgery. Similar results have been described by Waszyrowski et al. [16] in a similar number of patients with aortic stenosis, in which the greatest hemodynamic improvement is observed 1 year after valve replacement without further changes during follow-up. The absence of further changes could be explained by no significant changes in transprosthetic pressure gradient after the first year. Our study demonstrates that a significant increase in transprosthetic peak pressure gradient during the follow up (25.8 ± 6.8 mm Hg *vs*. 29.6 ± 11.2 mm Hg; $p = 0.04$) does not modify the sustained hemodynamic benefit.

Most patients who undergo AVR surgery present a preserved LVEF due to myocardial hypertrophy. Although this fact has not been evaluated as well as LVMI regression, we can find agreement with our results in other studies [17], which describe an increase of about 5% in LVEF with respect to the baseline [1]. In our observations, LVEF improvement after AVR is sustained during the third year of follow up. Some studies emphasize that patients at an advanced stage of AS presenting with left ventricular systolic dysfunction show a greater benefit. We cannot find similar results due to the short number of patients presenting with non-preserved LVEF. A short follow-up study did not indicate LVEF as a factor influencing LVMI regression [18], and there were no differences in preoperative LVEF between the patients with normal or abnormal LVMI after a longer follow-up [19].

Progressive left ventricular myocardial hypertrophy in AS evolution determines disturbances in ventricular relaxation parameters and, finally, diastolic dysfunction [11], as seen in most part of our patients. Improvements in these parameters have been evaluated by many authors with similar results [9]: left ventricular diastolic function improves early after surgery in parallel with a reduction in the aortic transprosthetic gradient. Ikonomidis et al. [3] evaluated diastolic dysfunction parameters four years after AVR surgery, defining a sustained improvement, but with an absence of improvement with incomplete ventricular hypertrophy regression or progressive transprosthetic gradient enlargement. Recently, an observation ten years after AVR surgery was published [4] in which a trend toward moderate to severe stages of diastolic dysfunction independent of hypertrophy regression magnitude was noted. In our observation the E:A ratio improved immediately after valve replacement, and remained stable for at least 3 years, although this time series did not allow a comparison with the long-term results described before. It is remarkable that the mean E:A index was abnormal during the three consecutive studies in our observation.

Although left ventricular hypertrophy late after aortic valve replacement for aortic stenosis has not been connected with increased mortality, it is associated with increased morbidity (impaired exercise capacity, higher New York Heart Association dyspnoea class, a tendency for more frequent chest pain expressed as higher Canadian Cardiovascular Society class, and more rehospitalisation) [20]. Considering the sustained benefit of left ventricular remodelling, our study could suggest a systematic left ventricular morphology evaluation by echocardiography after AVR, regardless of transprosthetic pressure gradient, and a thorough clinical follow up in patients who develop a significant increase in left ventricular mass index.

Limitations of the study

Because pure aortic stenosis is not as prevalent as associations with other valvulopaties, a low number of patients were enrolled, although the number was great enough to find a strong statistical significance. Regardless of their extensive work experience, the subjectivity of the echocardiography operator should be taken into account.

Conclusions

Aortic valve replacement surgery leads to a rapid negative left ventricular remodelling during the first 7 months, including a decrease in left ventricular hypertrophy and improvement in systolic and diastolic function. These beneficial hemodynamic changes are sustained during at least 3 years, independently of transprosthetic gradient increase.

Acknowledgements

The authors do not report any conflict of interest regarding this work.

We would like to thank Ms. Eva Lacambra for her support in statistical analysis, and to the Department of Cardiac Surgery of Miguel Servet University Hospital for their professional and valuable work day by day.

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