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Karpuz H, Ayan F, Hacıoglu Y, Koldas L

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Short-Term Regression of Left Ventricular Mass After Aortic Valve Replacement in Aortic Stenosis With Left Ventricular Concentric Hypertrophy

H. Karpuz, L. Koldas, F. Ayan, Y. Hacıoglu

After relief of aortic valve obstruction by valvular replacement, the left ventricular mass is supposed to regress. In this study, we assessed the early evolution of left ventricular mass 3 months after operation in patients with aortic stenosis and concentric hypertrophy. Echocardiographic study and measurements were performed in 27 patients (8 women; mean age 62 ± 12 years), according to the recommendations of the American Society of Echocardiography. Hypertrophy was defined as concentric when the ratio of left ventricle wall thickness to left ventricle short axis radius was more than 0.45. After surgery, we observed a significant reduction in left ventricular end-diastolic diameter, interventricular and posterior wall thickness; the left ventricular mass index reduction was also significant (from 141 ± 36 g/m² to 107 ± 29 g/m², $p < 0.05$).

These results already show a significant regression of left ventricular mass 3 months after aortic valve replacement in patients with aortic stenosis accompanied by left ventricular concentric hypertrophy. *J Clin Basic Cardiol* 2002; 5: 101–102.

Key words: aortic stenosis, aortic valve replacement, left ventricular mass

In aortic stenosis (AS), left ventricular hypertrophy (LVH) is one of the fundamental mechanisms of adaptation as a result of an increased afterload [1]. Even though initially this is an useful compensatory process, it may lead to progressive myocardial failure [2] due to the increased susceptibility of the hypertrophied myocardium to ischaemia [3]. Aortic valve replacement (AVR) has produced a dramatic change in the natural history of AS. In this context, measurement of the degree of LVH in patients with AS, particularly the assessment of the regression of such hypertrophy after valve replacement is of major importance [4]. In the previous studies [5–7], it was found that LVH regression is a time-consuming process and occurs over a period of several months.

The aim of the present study is to clarify the degree of short-term reversal of LVH after 3 months of AVR. To this end we designed a prospective study in which we planned to examine the left ventricular mass index (LVMI) pre-operatively and after 3 months of AVR in patients with aortic stenosis and echocardiographically confirmed LVH.

Materials and Methods

The patient population comprised 52 consecutive patients with aortic stenosis who were admitted to the Division of Cardiovascular Surgery at Centre Hospitalier Universitaire Vaudois (CHUV) for aortic valve replacement. 25 patients were excluded from the study because of the technically difficult echocardiographic study, abnormality of regional wall motion, asymmetrical LVH, severe left ventricular dilatation (> 32 mm/m²), evidence of hypertension or concomitant therapy. The remaining 27 patients with concentric LVH (8 women, 19 men, mean age 62 ± 12 years) were enrolled to the study.

Echocardiography

Two-dimensional echocardiographic studies were performed using a Hewlett-Packard Sonos 1500 (Hewlett-Packard company, Andover, MA, USA) equipped with 2.5 or 3.5 MHz probes for imaging. Studies were recorded on 0.5-inch videotape (Panasonic 750 D) for storage and review. The patients

were examined at the left lateral position with a 30° elevated bed-head. Special care was taken to avoid misleading angulations of the left ventricular long axis. M-mode measurements of the internal dimensions of LV (left ventricular end-diastolic diameter, LVEDD) and of the interventricular septum thickness (IVST) and the posterior wall thickness (PWT) were made according to the recommendations of the American Society of Echocardiography [8]; only readable echocardiographic tracings were accepted.

All measurements were taken as a mean of 3 consecutive cardiac cycles if the patient was in sinus rhythm or as the mean of 6 consecutive cardiac cycles if in atrial fibrillation. To avoid interobserver variability, all echocardiographic measurements were performed by the same physician.

Concentric LVH was defined as the ratio of LV septum to LV radius > 0.45 ; M-mode measurements were made at mid-papillary muscle level, in the short axis view.

The left ventricular mass (LVM) was estimated by the formula of Devereux as $\{1.04 [(IVST + PWT + LVEDD)^3 - (LVEDD)^3]\} \times 0.8 + 0.6$ [9], and was divided by the body surface to drive the LVM index (LVMI). There were no significant changes in body weight that would reduce LVMI and influence the results.

Surgery

All the patients had a successful aortic valve replacement with bileaflet mechanical valves (21–29 mm), St. Jude Medical or ATS (Advancing The Standard).

Statistical analysis

Changes in mass and the other parameters over time were assessed by the paired *t*-test. All values are expressed as mean \pm SD. A $p < 0.05$ was used to identify significant results.

Results

The values of the parameters before and after the aortic valve replacement (AVR) are shown in Table 1. The changes in LV wall thickness were more obvious than those seen in LV diameters. There was a reduction of approximately 11 % in

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From the Department of Cardiology, Cerrahpasa Medical School, University of Istanbul, Turkey

Correspondence to: Dr. Hakan Karpuz, Istanbul University Cerrahpasa School of Medicine, Department of Cardiology, Aksaray/Istanbul, Turkey; e-mail: y_hac@hotmail.com

Table 1. Changes in the parameters of LV in 27 patients before and after 3 months of AVR

	IVST (mm)	PWT (mm)	LVEDD (mm)	LVM (g)	LVMi (g/m ²)
Before AVR	12.9 ± 1.6	12.1 ± 1.5	50.1 ± 7	254 ± 71	141 ± 36
After AVR	11.5 ± 1.6	10.6 ± 1.6	47.2 ± 5	194 ± 61	107 ± 29
p-value	< 0.005	< 0.001	< 0.001	< 0.001	< 0.001

IVST: end-diastolic interventricular septum thickness; LVEDD: left ventricular end-diastolic diameter; LVM: left ventricular mass; LVMi: left ventricular mass index; PWT: end-diastolic posterior wall thickness

IVST (from 12.9 ± 1.6 mm to 11.5 ± 1.6 mm, $p < 0.005$) and 13 % in PWT (from 12.1 ± 1.5 mm to 10.6 ± 1.6 mm, $p < 0.001$). LVEDD was reduced by 6 % (from 50.1 ± 7 mm to 47.2 ± 5, $p < 0.001$). In all patients, except one, LVM was reduced by approximately 25 %. Moreover, the mean values of LVMi, being above the normal limits defined by Devereaux et al. [9] before AVR, were found to be within the normal limits after 3 months of AVR. As it was mentioned above, an increased LVM (from 197 g to 548 g) was found in one patient only. In this patient, while the wall thickness remained constant, a remarkable increase in LVEDD (from 46 mm to 56 mm) was seen and the ejection fraction (EF) decreased from 50 % to 30 %. However, no abnormal wall motion or prosthetic valve dysfunction was detected.

Discussion

There are not many studies in the literature focused on the early results of AVR in isolated populations of patients with aortic stenosis accompanied by concentric LV hypertrophy. In their study, Monrad et al. [6] reported a 21 % reduction in LVM after 1.6 years of AVR. However, their study group was not homogeneous and included patients with aortic regurgitation (AR), as well. Although different types of LVH may have different effects on the outcome in most of the studies performed up to date, the type of LVH was not taken into consideration. Nevertheless, according to these studies it was postulated that the decrease in LVH was most apparent in the first 6 months after the AVR and after 12 months the reduction was very slight. Similar results were reported in patients undergoing AVR due to aortic regurgitation.

Our results show correlation with those of some previous studies. For example, Hess et al. [10] reported a 35 % reduction in LVMi in their 21 patients after a follow-up of 17.5 months. At the end of the study, mean LVMi was found to be 115 g/m² (within the normal limits), similar to the results of our study. Pantley et al. [11], followed up the patients with AS or AR after AVR for 18 months and observed a decrease of 44 % in LVM. Kennedy et al. [12], similarly, found a 32 % reduction in LVM in patients with AS and/or AR after a follow-up of 19 months.

A more comparable study to ours, according to the length of the follow-up, was conducted by Henry et al. [5], in which 42 patients with isolated AS were followed up for 6 months after AVR. Moreover, they had the opportunity to analyze 13 of the patients earlier after AVR and observed that the maximal regression in LVM was seen in the first 10–23 days. While after the first 6 months there was a very slight regression, after the 31st month no change was observed.

Montalescot et al. [13], who found that the most important preoperative abnormality in their 12 patients with AS was the abnormal increase in LVM, reported that LVM mostly decreased within the first 3 months after AVR and during the following 5 years no additional regression was observed.

In a more recent study, Christakis et al. [14] reported a significant reduction in LVMi from 141.4 ± 45.2 g/m² to 127.5 ± 32.8 g/m² very soon after AVR (approximately 5 days). In several other studies comparing the regression of LVH in different types of implanted prosthetic valves early regression in LVH was confirmed [15–17].

It was interesting to find that in the study performed by Lund et al. [17] only in 18 % of the patients were the values of LVMi reduced to normal after 3 months of AVR. However, in most of our patients LVMi was detected to be within normal limits at the same period of time after AVR.

In relation to our patient in whom a progression in LVM was detected, the reason for that outcome was explained by the finding of LVEDD being markedly increased after AVR. Since an apparent enlargement in LV makes LV look more spherical than it used to be, an overestimation in the calculation of the LVM may occur.

As may be concluded from the results of all these studies, in patients with AS, AVR causes a significant reduction in LVH very early, within a period of several weeks or months. Our study confirms this finding, emphasizing the importance of the regression in the concentric hypertrophy that plays an important role in the process of “remodeling” of the myocardium. However, the clinical influence of this regression on the prognosis of the patients is still uncertain and remains to be investigated further.

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