

Improvement in Mitral Regurgitation after Aortic Valve Replacement

Sorel Goland MD, Gaby Loutaty MD, Alexander Arditi MD, Eitan Snir MD, Itzhak Abend MD and Abraham Caspi MD

Institute of Cardiology, Kaplan Medical Center, Rehovot, Israel

Key words: aortic stenosis, aortic valve replacement, mitral valve regurgitation, concomitant mitral valve regurgitation

Abstract

Background: Concomitant mitral valve regurgitation is often present in patients with aortic stenosis. The additional MV replacement is associated with high operative risk. Previous studies have shown an amelioration of MV regurgitation after aortic valve replacement but most of the patient groups were heterogenous.

Objectives: To determine whether AV replacement for aortic stenosis has any effect on MV regurgitation.

Methods: We reviewed two-dimensional echocardiography and color flow Doppler assessment of both aortic stenosis and MV regurgitation severity in 30 patients. Patients with previous MV surgery, organic MV disease, occlusive carotid artery disease, ejection fraction < 50%, and coexisting significant AV regurgitation were excluded. Preoperatively, MV regurgitation was mild in 23 patients (77%) and moderate in 7 (23%); in no patient was the condition severe. All patients had severe aortic stenosis (peak average aortic gradient 86 ± 22 mmHg in the mild MV regurgitation group and 83 ± 26 mmHg in the moderate group). The patients were divided into two groups according to the severity of MV regurgitation (associated mild, and moderate). Group 2, with moderate MV regurgitation, was the most problematic in terms of decision making for concomitant MV surgery. Therefore, additional assessment of several parameters was required.

Results: There was a significant decrease in MV regurgitation area (7.6 ± 1.9 vs. 3.0 ± 1.2 cm², $P \leq 0.012$) and percent ($28 \pm 5\%$ vs. $12 \pm 6\%$, $P \leq 0.001$) between pre- and postoperative evaluation. Thus, the severity of the condition in all patients with moderate MV regurgitation decreased after AV replacement; in the mild group it remained unchanged in 53% or improved in 47%. There was no association between the preoperative gradient on the aortic valve and the degree of MV regurgitation.

Conclusions: In our population of patients with severe aortic stenosis there were no patients with coexisting severe MV regurgitation. The decision to repair or replace a severely leaking mitral valve is an easy one, as in mild MV regurgitation. The clinical problem often presents in patients with severe aortic stenosis and moderate MV regurgitation. We believe that additional MV surgery is not necessary, at least in patients with preserved left ventricular function and without organic MV disease.

IMAJ 2003;5:12-14

Mitral valve regurgitation is a common finding in patients with congenital or degenerative aortic stenosis. The additional mitral valve replacement in patients undergoing surgery for aortic valve replacement is associated with twice the operative mortality [1]. Several studies have evaluated the amelioration of MV regurgitation in patients who had undergone AV replacement for aortic

stenosis, but all these studies included patients with concomitant occlusive carotid artery disease, AV regurgitation or varying degrees of left ventricular dysfunction. The aim of this study was to determine whether there is an improvement in MV regurgitation after AV replacement for aortic stenosis in patients with normal LV function, without OCAD or organic mitral valve disease.

Materials and Methods

We studied 30 patients (13 women and 17 men, mean age 72 ± 6.5 years) who underwent AV replacement for severe aortic stenosis over a 3 year period (1996–1999) and had coexisting MV regurgitation on preoperative echo study. (The total number of patients who underwent AV replacement in our hospital was 200.) MV regurgitation was moderate in 7 patients and mild in 23. We included only patients with adequate preoperative echocardiographic study who were also available for postoperative echo follow-up. Exclusion criteria were: concomitant OCAD, more than mild coexisting AV regurgitation, varying degrees of LV dysfunction, and organic MV disease.

Echocardiography

M-mode, two-dimensional pulsed, continuous wave and color Doppler studies were performed with Hewlett-Packard Sono 1000 machines for assessment of both aortic stenosis and MV regurgitation, at 2.5 months (range 1–3.5) before and 13 months (range 6–18) after AV replacement. The aortic gradient was calculated using the modified Bernoulli equation. MV regurgitation was assessed by means of color Doppler and obtained in any view as a maximal jet area (MV regurgitation area) and also as a percentage of left aortic area (MV regurgitation percentage) [2]. MV regurgitation was graduated as follows: MV regurgitation% < 20% (mild), 20–40% (moderate), more than 40% (severe). Mitral annular diameter was obtained during end-diastole. Mitral annulus calcification was defined as: mild – few calcifications in small (mostly posterior) area of mitral annulus; moderate – small calcium deposits, detected circumferentially, including also the anterior aspect of mitral annulus; and severe – large masses of calcium deposits involving the entire area of mitral annulus. Left ventricular function and morphology was obtained off-line using two-dimensional guided M-mode, applying standard American Society of Echocardiology criteria [3].

MV = mitral valve
AV = aortic valve

LV = left ventricular
OCAD = occlusive carotid artery disease

Statistical analysis

Data are expressed as mean \pm SD. Pre- and post-AV replacement data were compared using the paired *t*-test. The unpaired *t*-test was used between group comparisons. *P* value < 0.05 was considered significant.

Results

Patients

All patients ($n = 30$) were considered to have pre- and postoperative adequate echo studies and were divided into two groups: Group 1 – with coexisting mild MV regurgitation (23 patients, mean age 71 ± 1.3), and Group 2 – with moderate disease (7 patients, mean age 74 ± 1.3). All patients had severe aortic stenosis preoperatively (the peak gradient average in the mild MV regurgitation group was 87 ± 22 mmHg, and 83 ± 26 mmHg in the moderate group, $P = 0.78$) and preserved LV function (Group 1: ejection fraction $70 \pm 10\%$, Group 2: EF $59 \pm 12\%$ with borderline statistical significance between the two groups, $P = 0.05$). All but one patient received a mechanical prosthesis.

MV regurgitation

Preoperatively, the condition was mild in 23 patients (77%) and moderate in 7 (23%); in no patient was it severe. No patient had abnormalities of papillary muscles or chordae; all patients had normal mitral leaflet morphology. Mitral annulus calcification was present in 13 patients (70%) with mild MV regurgitation and in 7 patients (100%) in the moderate group. In patients with mild MV regurgitation, 11 (84%) had mild MAC and 2 patients (16%) had moderate MAC. In the group with moderate MV regurgitation, 5 patients (72%) had mild MAC and 2 (28%) had moderate MAC.

In Group 1 (mild MV regurgitation), the condition improved in 47% and remained unchanged in 53%. In no patient did it increase in severity. All patients (100%) in Group 2 (moderate MV regurgitation) showed improvement [Figure 1]. Additional assessment of several parameters was conducted in this group [Table 1], including maximum MV regurgitation area, left atrium area, ratio

MV regurgitation area/LA area, and mitral annulus diameter. There was a significant improvement in MV regurgitation area (7.6 ± 1.9 vs. 3.0 ± 1.2 cm², $P \leq 0.002$) and MV regurgitation % ($28\% \pm 5$ vs. $12\% \pm 6$, $P \leq 0.001$) [Figure 2]. There was no statistically

LA = left atrium

Table 1. Echocardiographic changes after AV replacement in moderate MV regurgitation group

	Pre-AV replacement	Post-AV replacement	<i>P</i>
Peak aortic gradient (mmHg)	83 ± 26	24 ± 10	0.0003
LA area (cm ²)	27.7 ± 4	26.5 ± 7	0.6
Mitral annulus diameter (cm)	3.43 ± 0.25	3.24 ± 0.58	0.4
MV regurgitation jet area (cm ²)	7.7 ± 1.9	3.0 ± 1.2	0.0012
% MV regurgitation/LA area	28 ± 5	12 ± 6	0.001
LVEDd (mm)	54 ± 9	53 ± 12	0.4
LVESd (mm)	35 ± 10	34 ± 12	0.9
EF (%)	59 ± 12	62 ± 14	0.3

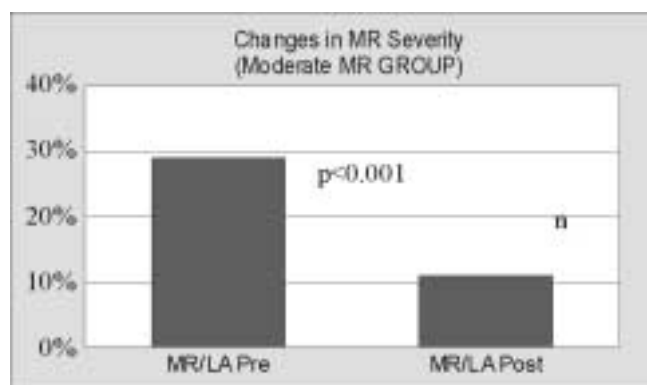


Figure 2. Graph showing reduction of MV regurgitation: % MV regurgitation jet area (cm²)/LA area (cm²) before and after AV replacement.

EF = ejection fraction

MAC = mitral annulus calcification

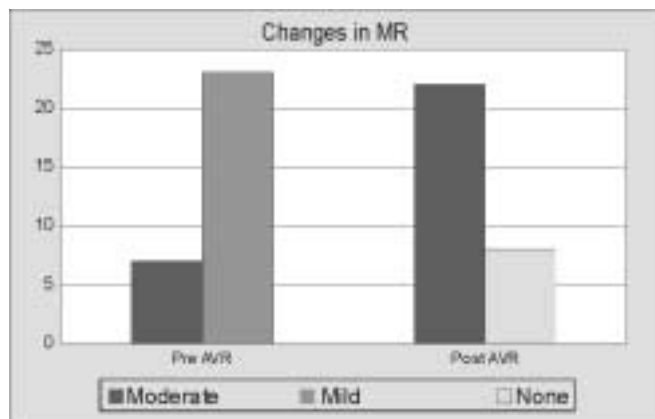


Figure 1. Graph showing improvement of MV regurgitation after aortic valve replacement.

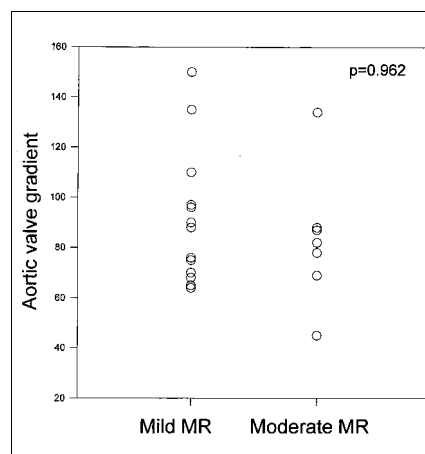


Figure 3. Relation between pre- and postoperative peak aortic valve gradient and degree of MV regurgitation.

significant reduction of mitral annulus diameter (3.48 ± 0.25 cm, $P = 0.4$), left atrium diameter (27 ± 4 vs. 26 ± 7 cm², $P = 0.6$), left ventricle end-diastolic diameter, (54.5 ± 9 vs. 52.7 ± 9 mm, $P = 0.4$), left ventricle end-systolic diameter (35 ± 10 vs. 34.6 ± 12 mm, $P = 0.9$), and EF (59 ± 15 vs. $63\% \pm 14$, $P = 0.3$).

Aortic stenosis

The average preoperative peak aortic gradient was 83 ± 26 mmHg, which decreased to 24 ± 10 mmHg after AV replacement [Table 1]. In group 1 (mild MV regurgitation), aortic valve peak gradient decreased from 86 ± 22 to 33 ± 9 mmHg ($P < 0.0001$), and in Group 2 (moderate) from 83 ± 26 to 27 ± 10 ($P < 0.0003$). There was no association between preoperative gradient on the aortic valve and degree of MV regurgitation [Figure 3].

Discussion

This study differs from previous reports in that we evaluated a homogenous population of patients with severe aortic stenosis and coexisting mitral valve regurgitation both before and after aortic valve replacement, performing in addition semiquantitative analysis. We excluded all patients with OCAD who underwent concomitant coronary artery bypass graft, patients with varying degrees of LV dysfunction, coexisting AV replacement and organic MV disease.

In previous reports, Tunick et al. [4] showed correction of MV regurgitation after AV replacement, and Harris et al. [5] not only determined the clear improvement in MV regurgitation in patients with severe aortic stenosis after surgery but also suggested that the reduction was due to decreased intraventricular pressure as well as changes in LV morphology. The degree of MV regurgitation in patients with severe aortic stenosis depends on the regurgitant orifice and driving pressure across the aortic valve. The latter significantly decreases after AV replacement but the regurgitant orifice is also related to changes in ventricular morphology, as demonstrated by Harris's report [5]. The opposite conclusion was reached by Adams and Otto [6], who showed a reduction of MV regurgitation in only 40% of patients. The significant limitation in these studies was the heterogeneity of the patient population.

Our study confirms the results of Tunick [4] and Harris [5], which demonstrated a significant reduction in MV regurgitation after AV replacement in aortic stenosis patients. The greatest improvement was observed in the moderate MV regurgitation group and remained unchanged or improved in the mild group. No patient experienced a worsening in degree of the condition after AV replacement. There was no association between the aortic preoperative gradient and degree of the condition. In our population with severe aortic stenosis, no patient had coexisting severe MV regurgitation. There were no statistical significantly changes in mitral annulus diameter, LVEDd, LVESd or EF after AV

replacement. MAC was present in most patients with associated MV regurgitation. We suggest that this finding is due to degenerative disease in our elderly population.

Our patients, by study design, had no intrinsic mitral valve disease and we considered that the etiology was functional MV regurgitation. We suggest, nevertheless, that the main mechanism in the amelioration of mitral regurgitation in our patients is related to a significant reduction in driving pressure across the aortic valve. The absence of changes in left ventricle and left atrium geometry may be related to the short duration of follow-up, and further studies with longer follow-up should be performed to confirm these changes.

Our study had several limitations. The study was retrospective and included only a relatively small number of patients with moderate MV regurgitation. Incomplete data were found in reviewed echo studies for measurements such as proximal isovelocity surface area or regurgitant volume. During the study we used mostly semiquantitative assessment of mitral regurgitation.

Conclusions

Patients with both severe aortic stenosis and mild and moderate mitral valve regurgitation showed amelioration of the regurgitation after aortic valve replacement. In this population of patients there was a low incidence of severe MV regurgitation.

We suggest that in patients with severe aortic stenosis and coexisting MV regurgitation – but without significant structural MV disease or OCAD and with normal LV function – there can be a marked improvement following AV replacement. Thus, mitral valve surgery with its concomitant morbidity and mortality at the time of AV replacement may not be necessary.

References

1. Kirklin JW. Combined aortic and mitral valve disease with or without tricuspid valve disease. In: Marklin JW, ed. *Textbook of Cardiac Surgery*. New York: Churchill Livingstone, 1993:573–87.
2. Helmike F, Nanda NC, Hsinzing MC, et al. Color Doppler assessment of mitral regurgitation with orthogonal planes. *Circulation* 1987;75: 175–83.
3. Schiller NB, Shah PM, Crawford M, et al., and the American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-dimensional Echocardiograms. Recommendation for quantitation of the left ventricle by two-dimensional echocardiography. *J Am Soc Echo* 1989;2:358–67.
4. Tunick PA, Gindea A, Kronzon I. Effect of aortic valve replacement for aortic stenosis on severity of mitral regurgitation. *Am J Cardiol* 1990;65:1219–21.
5. Harris KM, Malenka D, Haney MF, et al. Improvement in mitral regurgitation after aortic valve replacement. *Am J Cardiol* 1997;80:741–5.
6. Adams PB, Otto CM. Lack of improvement in coexisting mitral regurgitation after relief of valvular aortic stenosis. *Am J Cardiol* 1990;66(1):105-7.

Correspondence: Dr. S. Goland, Institute of Cardiology, Kaplan Medical Center, Rehovot 76100, Israel.

Phone: (972-8) 944-1376

Fax: (972-8) 944-1883

email: sorel6@012.net.il

LVEDd = left ventricle end-diastolic diameter

LVESd = left ventricle end-systolic diameter