Imaging techniques in aortic valve and root surgery

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PART II

Postoperative evaluation of patients

after aortic valve and root surgery

Effects of aortic valve and root surgery on left ventricular performance

Chapter 7:

Left ventricular reverse remodeling after aortic valve surgery for acute versus chronic aortic regurgitation

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Echocardiography. 2016;33:1458-1464

Abstract

Background: Extent of left ventricular (LV) reverse remodeling after aortic valve repair or replacement (AVR) may differ between patients operated for acute aortic regurgitation (AR) and chronic AR. The aim of this study was to compare changes in LV volumes and function between patients with acute and chronic AR who underwent AVR.

Methods: A total of 98 patients (54±15 years, 61% men) with acute (n=21) or chronic AR (n=77) were included in the present retrospective evaluation. LV volumes, LV ejection fraction and global longitudinal strain indexed for LV end-diastolic volume (GLSi) were assessed preoperatively and after a median follow-up of 28 months (interquartile range: 17-66 months).

Results: Patients with acute AR tended to have smaller preoperative LV end-diastolic volume compared to chronic AR (156±15 vs. 183±6 ml; p=0.070). Both in patients with acute and chronic AR, significant LV reverse remodeling with sustained reduction in LV volumes occurred during follow-up with a significant smaller LV end-diastolic volume in acute AR compared to chronic AR (106±8 vs. 128±5 ml; p=0.032). Preoperative and postoperative LV ejection fractions were not significantly different between groups. In contrast, GLSi was better in patients with acute AR compared to chronic AR before AVR (-1.34±0.20 vs. -0.96±0.07 %/10 ml; p=0.042) and during follow-up (-1.65±0.16 vs. -1.29±0.07 %/10 ml; p=0.017).

Conclusion: After AVR, LV reverse remodeling occurs both in patients with acute and chronic AR. However, LV end-diastolic volume was more reduced and GLSi was more preserved during follow-up in acute AR than in chronic AR.

Introduction

The prevalence of aortic regurgitation (AR) in the general population is 10% and is often mild.[1,2] Severe chronic AR results in a combination of left ventricular (LV) volume overload (due to the regurgitant volume) and LV pressure overload (due to systolic hypertension as a result from an increase in total aortic stroke volume).[3] In early stages of the disease, the LV adapts to the volume overload with eccentric LV hypertrophy to preserve LV systolic function.[4] Progressive LV dilation and systolic hypertension increase LV wall stress leading to ultrastructural myocardial changes that may not reverse after aortic valve repair or replacement (AVR).[5-7] In severe acute AR, these changes may not take place and therefore the remodeling process after emergent AVR may be different to that observed in patients with chronic AR.

The studies evaluating the presence and clinical implications of LV reverse remodeling after AVR have focused on patients with severe chronic AR.[8-11] However, little is known about the LV reverse remodeling process in patients with severe acute AR. Accordingly, the aim of the current study was to characterize changes in LV volumes and function after AVR in patients with acute AR and chronic AR.

Methods

Patients

The present retrospective study included adult patients who underwent aortic valve and root surgery for AR or aortic root pathology from 1998 to 2013 at the Leiden University Medical Center, The Netherlands. Patients with moderate to severe and severe AR and no more than mild aortic stenosis were selected from a large echocardiographic database. Patients with available echocardiograms at baseline (before surgery) and at least 6 months after aortic valve replacement were included. If patients underwent reoperation on the aortic valve and/or aortic root during follow-up, the last transthoracic echocardiography before reoperation was considered in the analysis. Concomitant mitral or tricuspid valve disease was not an exclusion criterion.

Baseline clinical characteristics, EuroSCORE II and surgical procedures were recorded. In addition, LV volumes and function and valvular hemodynamics were assessed with 2-dimensional transthoracic echocardiography preoperatively and at 6 months or longer follow-up. All data were prospectively collected in the departmental Cardiology Information System (EPD-Vision, Leiden University Medical Center, Leiden, The Netherlands) and retrospectively analyzed. Patients were divided into two groups: patients with acute AR developed ≤6 weeks before AVR and patients with chronic AR lasting for >6 weeks before AVR.

The institutional ethical committee approved the retrospective analysis of clinical and echocardiographic data and waived the need for patient written informed consent for patients

followed-up at the Leiden University Medical Center, The Netherlands. For retrospective analysis of postoperative echocardiographic data of patients followed-up in referral centers, written informed consent was obtained.

Two-dimensional transthoracic echocardiography

Transthoracic echocardiography was performed at rest with patients in the left decubitus position using commercially available ultrasound systems (System 5, Vivid 7 and E9, General Electric Healthcare, Vingmed, Horten, Norway) equipped with 3.5-MHz or M5S transducers. Two-dimensional and Doppler data were acquired at the parasternal, apical, subcostal and supra-sternal views according to current recommendations.[12] The echocardiographic data were digitally stored in cine-loop format and data analysis was retrospectively performed using EchoPac (112.0.1, GE Medical Systems, Horten, Norway). LV end-diastolic (LVEDV) and end-systolic (LVESV) volumes were quantified in the apical two- and four-chamber views using the Simpson's biplane method and LV ejection fraction (LVEF) was calculated.[12] Further evaluation of LV systolic function was performed with two-dimensional speckle-tracking longitudinal strain analysis. LV longitudinal strain was measured from apical two-, three- and four-chamber views and averaged to obtain LV global longitudinal strain (GLS). To take into consideration changes in LV volumes over time after AVR, GLS was corrected for LVEDV and expressed as percentage of deformation per 10 ml of LVEDV (GLSi).

Aortic valve function was evaluated using color, continuous- and pulsed-wave Doppler. AR grade was assessed using a multiparametric approach including the measurement of the jet width relative to the LV outflow tract width and the vena contracta width in the parasternal long-axis view and apical three- or five-chamber views and the measurement of the pressure half time (if feasible) with continuous wave Doppler on the apical three- or five-chamber views. AR was graded as 0 (absent), 1 (mild), 2 (mild-moderate), 3 (moderate-severe) or 4 (severe).[13] Aortic stenosis grade was assessed measuring the aortic jet velocity and transaortic mean pressure gradient on continuous-wave Doppler recordings of the valve obtained in the apical three- or five-chamber views. Left ventricular outflow tract velocity was measured using the pulsed-wave Doppler in the apical three- or five-chamber views with the sample volume placed 5 mm below the aortic annulus plane into the left ventricle. Aortic valve area was calculated using the continuity equation. Patients with more than mild aortic stenosis were excluded as per inclusion and exclusion criteria of the study.

Surgery

After median sternotomy, arterial cannulation was performed of the distal ascending aorta (in elective non-dissected aortic pathology and in the absence of dilatation of the distal part of the ascending aorta) or the subclavian or femoral artery (in patients with ascending aorta

dissection or dilatation). Patients underwent aortic valve and root replacement or valve-sparing aortic root reconstruction. In aortic valve and root replacement techniques, the coronary buttons were mobilized and the sinuses of Valsalva and the aortic valve were excised. A mechanical or biological prosthesis was then implanted. Thereafter the coronary buttons were reattached.[14,15] For valve-sparing aortic root reconstruction, either a supracoronary ascending aorta replacement was performed with restoration of the sinotubular junction or the native sinuses of Valsalva were resected and a graft was implanted using the reimplantation technique (modified David procedure) or the remodeling technique (Yacoub technique), as previously described.[16-18] Leaflet repair procedures (leaflet triangular resection, leaflet resuspension and plication of the free edge of the leaflet) were performed if needed. In addition, concomitant aortic arch replacement was performed if the luminal diameter at this level was >45 mm or – in cases of dissection – a (re)entry tear was present in the arch.

Follow-up

Patients underwent two-dimensional transthoracic echocardiography during follow-up at the discretion of the treating cardiologist. The median follow-up duration was 28 months (interquartile range: 17-66 months).

Statistical analysis

All data analyses were performed using the SPSS software version 20 (SPSS, Chicago, IL, USA). Categorical variables are reported as numbers and percentages. Continuous variables are reported as mean ± standard deviation or median and interquartile range. Differences between acute and chronic AR were analysed using the chi-square test (for categorical variables) and the unpaired Student's *t*-test or Mann-Whitney U test (for normally or non-normally distributed continuous variables, respectively). Linear mixed model analysis was used to assess the differences in change in LV dimensions and function over time between the two groups. Onset of AR (acute or chronic) and time of transthoracic echocardiography (preoperative or follow-up) were incorporated in the model as fixed variables. An unstructured covariance matrix was applied. The estimated marginal mean ± standard error of the mean was presented. All statistical tests were two-sided. A p-value<0.05 was considered statistically significant.

Results

A total of 98 patients (mean age 54±15 years, 61% men) were evaluated: acute AR was present in 21 (21%) patients and chronic AR in 77 (79%) patients. Acute AR was classified as moderate-severe in 6 (29%) patients and severe in 15 (71%) patients and the etiology was endocarditis in

14 (66%) patients, acute type A aortic dissection in 5 (24%) and acute pulmonary edema associated with aortic root dilation in the remaining 2 (10%) patients. Chronic AR was classified as moderate-severe in 49 (64%) patients and severe in 28 (36%) patients. The clinical and surgical characteristics of both groups are displayed in Table 1. Cardiovascular risk factors were comparable between groups. However, the creatinine clearance was significantly lower and New York Heart Association class IV heart failure symptoms were more frequently reported in patients with acute AR compared with chronic AR. Patients with acute AR had higher EuroSCORE II compared with patients with chronic AR. Among patients with acute AR, 6 (29%) patients underwent emergent surgery and 15 (71%) patients underwent urgent surgery. In the entire cohort, the aortic valve and root were replaced by a biological prosthesis in 60 (61%) patients and by a mechanical prosthesis in 10 (10%) patients.

Table 1. Baseline and surgical characteristics.

	Acute AR (n=21)	**Chronic AR (n=77)**	**p-value**
Age (years)	56 ± 14	53 ± 15	0.380
Male	10 (48%)	50 (65%)	0.234
Body surface area (m^2)	1.85 ± 0.18	2.03 ± 0.41	0.059
Smoking	5 (24%)	18 (23%)	1
Diabetes mellitus	3 (14%)	2 (3%)	0.110
Hypertension	8 (38%)	29 (38%)	1
Dyslipidaemia	2 (10%)	7 (9%)	1
NYHA functional class			0.002
I	11 (52%)	29 (38%)	
II	3 (14%)	26 (33%)	
III	2 (10%)	20 (26%)	
IV	5 (24%)	2 (3%)	
Creatinine clearance (ml/min)	78 ± 36	104 ± 37	0.008
EuroSCORE II (%)	7.3 (3.3 – 22.4)	2.5 (1.6 – 4.2)	<0.001
Bicuspid aortic valve	2 (10%)	27 (35%)	0.042
Aortic valve and root technique			0.414
Replacement	17 (81%)	53 (69%)	
Valve-sparing restoration	4 (19%)	24 (31%)	
Mitral valve surgery	6 (29%)	15 (19%)	0.549
Tricuspid valve surgery	4 (19%)	10 (13%)	0.725
CABG	2 (10%)	6 (8%)	1

Data are presented as mean ± standard deviation, as median (interquartile range) or as number (percentage). AR: aortic regurgitation. CABG: coronary artery by-pass grafting. EuroSCORE: European System for Cardiac Operative Risk Evaluation. NYHA: New York Heart Association.

In the remaining 28 (29%) patients, a valve sparing procedure was performed with isolated aortic valve repair in 3 patients, supracoronary ascending aorta replacement with restoration of the sinotubular junction in 6 patients, reimplantation technique of David in 17 patients and

the remodeling technique of Yacoub in 2 patients. There were no differences in the number and type of concomitant surgeries performed in both groups.

LV reverse remodeling after AVR for acute versus chronic AR

The median echocardiographic follow-up duration was 34 months (interquartile range: 18-66 months) in patients with acute AR and 26 months (interquartile range: 16-64 months) in patients with chronic AR (p=0.491). Recurrence of moderate-severe or severe AR at follow-up was present in 5 (5%) patients, all in chronic AR patients. At follow-up, the mean aortic valve gradient was similar between patients with acute and patients with chronic AR (9.3±10.3 vs. 7.3±5.3 mmHg; p=0.236).

Figure 1 shows the LV volumes preoperatively and at follow-up. The preoperative LVEDV was slightly smaller in patients with acute AR compared with chronic AR (156±15 vs. 183±6 ml, p=0.070). The preoperative LVESV was comparable between acute and chronic AR (73±10 vs. 85±4 ml; p=0.162). At follow-up, in both groups, LV reverse remodeling occurred with a significant reduction in LVEDV and LVESV. However, the LVEDV was significantly smaller at follow-up in patients with acute AR than in patients with chronic AR (106±8 vs. 128±5 ml; p=0.032).

The change in LV function is displayed in figure 2. The preoperative LVEF was similar in patients with acute AR and chronic AR (55±2 vs. 54±1%; p=0.595) and increased during follow-up with no difference between groups (acute AR: 57±2% vs. chronic AR: 59±1%; p=0.444). In patients with acute AR, GLS was −15.8±1.3% before and −15.5±0.9% after surgery (p=0.874). In patients with chronic AR, GLS was −15.0±0.8% before and −14.3±0.4% after surgery (p=0.373). Because GLS represents shortening of the LV, which is dependent on the size of the LV, GLS was indexed for LVEDV. The GLSi was significantly better in patients with acute AR compared with chronic AR at baseline (−1.34±0.20 vs −0.96±0.07%/10 mL; P=.042) as well as during follow-up (−1.65±0.16 vs −1.29±0.07; p=0.017).

Discussion

The main findings of the present study include the observation that patients with acute AR had slightly smaller pre-operative LVEDV compared with patients with chronic AR suggesting the lack of LV remodeling to compensate the volume and pressure overload in the former group. Despite this observation, significant LV reverse remodeling after AVR was observed in both patients with acute AR and patients with chronic AR, with significantly smaller LVEDV and more preserved LV systolic function (based on speckle tracking echocardiography measurements) at follow-up among patients with acute AR compared with patients with chronic AR.

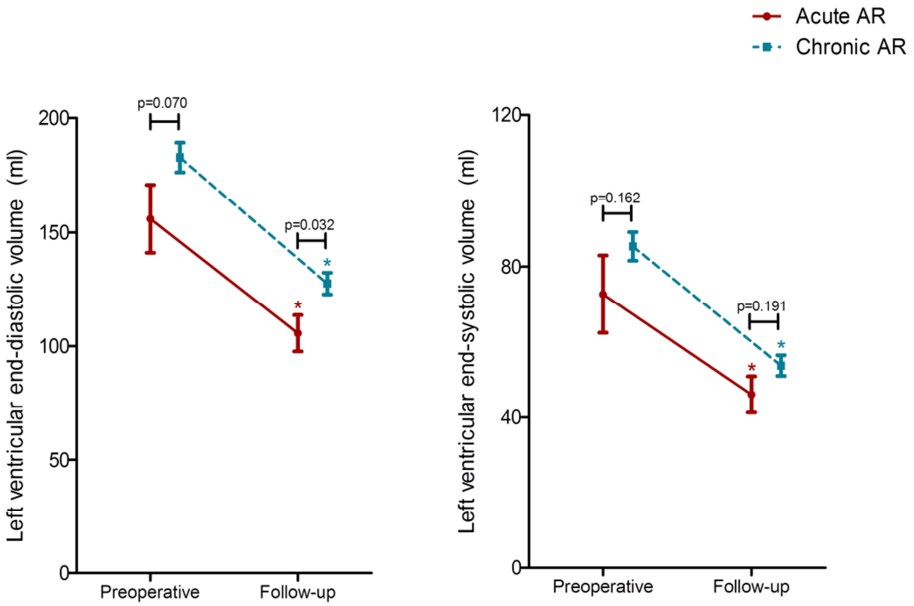

Figure 1. Left ventricular volumes over time in acute and chronic AR.
*Data are displayed as estimated marginal means ± standard error of the mean. *p<0.05 compared with preoperative. AR: aortic regurgitation. LVEDV: left ventricular end-diastolic volume. LVESV: left ventricular end-systolic volume*

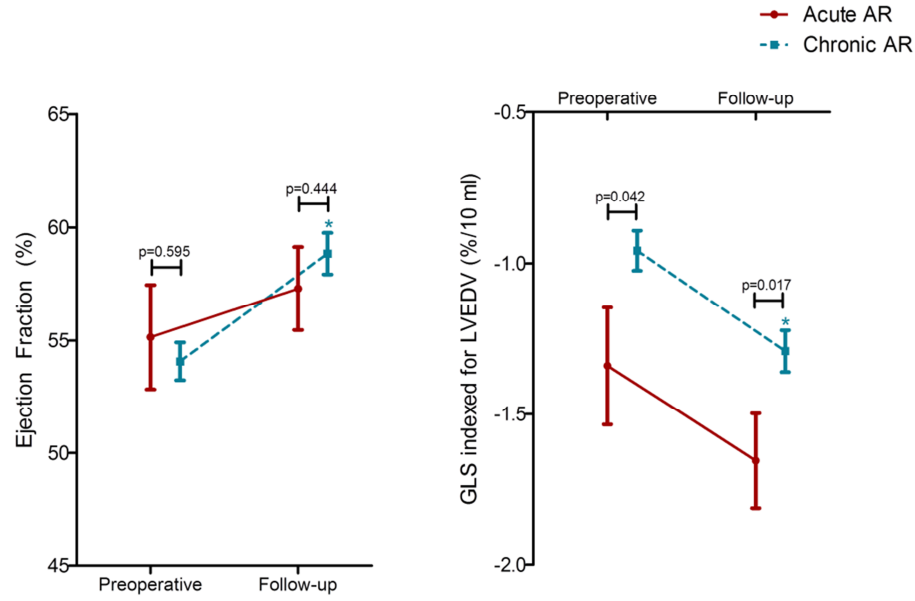

Figure 2. Left ventricular function over time in acute and chronic AR.
*Data are displayed as estimated marginal means ± standard error of the mean. *p<0.05 compared with preoperative. AR: aortic regurgitation. GLSi: global longitudinal strain indexed for left ventricular end-diastolic volume. LVEF: left ventricular ejection fraction.*

Acute versus chronic aortic regurgitation

Timing of aortic valve repair/replacement for AR depends on the presence of symptoms or documentation of LV systolic function impairment or LV dilatation.[19] Acute severe AR is usually associated with abrupt onset of heart failure symptoms, signs of low cardiac output and initial signs of LV remodeling, including LV dilatation and hypertrophy that cannot compensate the abrupt increase in volume and pressure overload.[20] Experimental models have shown changes in extracellular matrix, perivascular fibrosis and increased cardiomyocyte cross-sectional area early after onset of acute severe AR.[20] In contrast, in chronic severe AR, the adaptive changes in LV structure to compensate the gradual increase in volume and pressure overload result in larger LV dilatation and hypertrophy (compared with acute AR) that normalize wall stress and preserve LV systolic function.[4] When the AR progresses and LV wall stress increases, LV systolic dysfunction occurs.[21-23]

In acute AR immediate surgical intervention is usually necessary and can be performed with good outcome,[24,25] whereas in chronic AR patients, surgery is not recommended until symptoms develop or when LVEF<50% or LV end-diastolic diameter >75 mm or LV end-systolic diameter >55mm.[19]

Aortic valve and root surgery restoring the competence of the aortic valve reduces the LV volume and pressure overload inducing LV reverse remodeling. Previous studies have shown that LV reverse remodeling occurs, both after aortic valve and root replacement and after valve-sparing aortic root reconstruction in patients with chronic AR.[8-10] In contrast, comparison of postoperative LV reverse remodeling and change in systolic function between patients operated for acute AR and patients operated for chronic AR have not been described extensively. Kumpuris et al. compared three patient groups with severe AR who underwent aortic valve surgery; chronic AR with postoperative LV reverse remodeling, chronic AR without postoperative LV reverse remodeling and acute AR.[26] Patients operated for acute AR and patients operated for chronic AR who showed postoperative LV reverse remodeling had similar LV end-diastolic and LV end-systolic diameters preoperatively and postoperatively (median 30 days after surgery).[26] The present evaluation also showed LV reverse remodeling in both groups of patients which was more pronounced among patients with acute AR than in patients with chronic AR. Probably, more advanced microscopic remodeling with increased myocardial fibrosis in patients with chronic AR may preclude normalization of LV volumes after AVR.

Furthermore, recovery or normalization of LV systolic function is an important surgical outcome with prognostic implications. In chronic AR patients with preoperative LV systolic dysfunction or severe dilation who underwent aortic valve and root surgery, the LVEF improved significantly.[8,27] Particularly, patients with LV reverse remodeling early after surgery experienced an increase in LVEF from 47±9% to 56±6% during follow up, whereas in patients

without LV reverse remodeling after surgery, the LVEF remained low (from 32±6% to 26±9%).[11] However, LVEF may not be a sensitive marker of LV systolic function in these patients. As previously demonstrated, patients with chronic severe AR may have impaired GLS despite having normal LVEF suggesting the presence of ultrastructural changes of the myocardium.[23] GLS may be also a better reflector of the LV performance after AVR. A previous study including 47 chronic AR patients showed significant postoperative improvement in GLS normalized for LVEDV (from about -0.9 %/10 ml to -1.2 %/10 ml; $p<0.01$).[5] We found similar results in chronic AR patients (from -0.96 %/10 ml to -1.29 %/10 ml; $p<0.001$). In addition, we demonstrated that patients with chronic AR had more impaired GLSi compared to acute AR patients before and after surgery suggesting more myocardial dysfunction in the former patients.

Clinical implications

The present study provides further insight into the effect of AR on LV performance before and after AVR. Chronic AR was associated with less LV reverse remodeling and less improvement in LV function after aortic valve and root surgery compared with acute AR, which might be attributed to increased myocardial fibrosis in patients with chronic AR. Although LVEF was not significantly different between patients with acute and chronic AR before as well as after surgery, GLSi was less affected in patients who underwent surgery for acute severe AR suggesting a lesser degree of microscopic remodeling (myocardial fibrosis and extracellular matrix changes) in these patients.

Current guidelines recommend surgery in asymptomatic patients with chronic severe AR when LV dilation (LV end-diastolic diameter >75mm or LV end-systolic diameter >55mm) or LV dysfunction (LVEF <50%) occurs. The present study indicates that chronicity of AR is associated with diminished postoperative improvement in LV performance and normalization of LV volumes. Therefore, more sensitive parameters of LV dysfunction or early remodeling may be helpful to improve the outcomes of aortic valve surgery for chronic AR. GLSi is able to detect myocardial dysfunction in an earlier stage compared to LVEF.[5,23] Studies including GLSi in surgical decision-making might help in the optimal timing of surgery in patients with chronic AR. Perhaps surgery at an earlier stage can prevent the development of irreversible changes of the myocardium.

Limitations

This was a retrospective study with a limited number of patients. Only patients with preoperative and follow-up echocardiography were included in the present analysis which might have introduced a selection bias. Clinical data for patients who did not meet the inclusion criteria were not collected and therefore comparisons between included and excluded patients are not feasible to investigate the differences and selection bias.

Furthermore, there was no prospective follow-up protocol, thus echocardiographic follow-up was performed at the discretion of the treating cardiologist. The exact duration of AR was unknown in the majority of patients with chronic AR; therefore no further analysis could be performed relating the duration of AR to the extent of LV reverse remodeling and LV function improvement.

Conclusion

LV reverse remodeling occurs after aortic valve surgery both for acute and chronic AR. However, LVEDV was more reduced and GLSi was more preserved during follow-up in patients operated for acute AR than in patients with chronic AR.

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