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Original article

Associations of residual left ventricular and left atrial remodeling with clinical outcomes in patients after aortic valve replacement for severe aortic stenosis

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ABSTRACT

Background: Aortic valve replacement (AVR) is currently the standard therapy for severe aortic stenosis (AS), and regression of left ventricular (LV) hypertrophy after AVR has been reported. However, data regarding a temporal relation between LV mass and left atrial (LA) volume are limited, and their prognostic impacts have not been fully elucidated. We aimed to clarify the temporal patterns of LA and LV reverse remodeling and their associations with clinical outcomes.

Methods: We retrospectively reviewed 198 consecutive patients who underwent AVR for severe AS. After excluding patients with prior cardiac surgery, atrial fibrillation, concomitant moderate to severe aortic regurgitation, or concurrent mitral valve surgery, 83 patients with echocardiographic LV mass index (LVMI) and LA volume index (LAVI) data before and 1 year after AVR were eligible for the outcome analysis and 29 patients with these 2 measures before surgery, 1 month, 1 year, and 3 years after surgery were eligible for the analysis of time-dependent change of LVMI and LAVI.

Results: Significant reductions in LVMI and LAVI (both $p < 0.001$) after surgery were observed over time. LA dilatation improved and reached a plateau 1 month after surgery, whereas LV hypertrophy improved more gradually and reached a plateau at 1 year. The presence of both LV hypertrophy and LA dilatation 1 year after surgery was associated with significantly higher mortality (patients with both conditions vs. patients with neither or one condition = 22.6% vs. 7.3% at 3 years; $p = 0.031$) and major adverse cardiac and cerebrovascular events (38.9% vs. 12.6% at 3 years; $p = 0.021$).

Conclusions: LA reverse remodeling occurred rapidly after AVR for severe AS, and regression of LV hypertrophy was more gradual. The presence of both residual LV hypertrophy and LA dilatation 1 year after AVR was associated with poor long-term outcomes.

Introduction

Concentric left ventricular (LV) hypertrophy is a common finding in patients with chronic pressure overload, including hypertensive patients and those with aortic stenosis (AS). LV diastolic dysfunction and an increase in LV filling pressure occur in LV hypertrophy and subsequently cause left atrial (LA) dilatation [1–3]. Thus, LV hypertrophy and LA dilatation are manifestations and prognostic markers of left-sided heart failure, and there is a significant positive correlation between these two parameters in patients with preserved ejection fraction [4,5]. LV mass index (LVMI) calculated using echocardiography or cardiac magnetic resonance is a standard measure for quantitative evaluation of LV hypertrophy [6,7]. Increased LVMI as well as LA volume index (LAVI) measured on echocardiograms is related to adverse events in many cardiac diseases including isolated severe AS [8–13].

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Although significant regression of LV hypertrophy is seen in patients undergoing aortic valve replacement (AVR) for severe AS [14], a significant portion of these patients do not show favorable left heart reverse remodeling, assessed by LVMI and LA dimension, and such patients may have poor clinical outcomes [15,16]. In AS patients, LA dilatation is caused, at least in part, by LV hypertrophy and is related to the magnitude of LV hypertrophy. However, the time course of LA reverse remodeling and its temporal relationship with LV reverse remodeling have not been clarified. In addition, the prognostic impact of LA reverse remodeling has not been fully elucidated. Because it is well recognized that variations in LA configuration cause inaccuracy of LA dimension measurement in the evaluation of LA dilatation [6,17–19], we demonstrate the time course of LVMI and LAVI after AVR for severe AS. The association between residual LV hypertrophy and LA dilatation at 1 year, with long-term clinical outcomes, was also assessed.

Methods

Study patients

We retrospectively reviewed the medical charts of 381 consecutive patients who underwent AVR at our institution from 2006 to 2011. Of these, 198 patients underwent AVR for severe AS. Nine patients who had prior cardiac surgery and 35 patients who had concomitant moderate or severe aortic regurgitation were excluded. Twenty-six patients with concomitant moderate or severe mitral valve regurgitation or stenosis who underwent mitral valve plasty or mitral valve replacement, two patients with concomitant hypertrophic obstructive cardiomyopathy who underwent myomectomy, and 15 patients with documented atrial fibrillation on electrocardiogram before or after AVR were also excluded. Of the remaining 111 patients, echocardiographic LVMI and LAVI data before and 1 year after AVR were available in 83 patients, who were considered eligible for the outcome analysis. These two measures were available for all four time points (before surgery, 1 month, 1 year, and 3 years after surgery) in 29 patients, who were considered eligible for the analysis of time-dependent change of LVMI and LAVI (Fig. 1).

The study protocol was approved by the institutional review board of Kobe City Medical Center General Hospital.

Echocardiographic measurements

Data from two-dimensional transthoracic echocardiography performed preoperatively and 1 month, 1 year, and 3 years postoperatively were used for the analyses. LV end-diastolic dimension (LVDd) and LV end-systolic dimension (LVDs) were measured from the left parasternal long-axis view. Likewise, diastolic interventricular septal wall thickness (IVSTd) and diastolic left ventricular posterior wall thickness (LVPWTd) were measured. LV end-diastolic volume (LVEDV), LV end-systolic volume (LVESV), and LA volume were calculated from the apical four- and two-chamber views using the biplane method of disks, and LV ejection fraction and LAVI were calculated [6]. LV mass was calculated using the formula recommended by the American Society of Echocardiography (ASE) [6]: LV mass = $0.8(1.04[LVDd + LVPWTd + IVSTd]^3 - [LVDd]^3) + 0.6$.

LV hypertrophy was defined as LVMI $\geq$116 g/m^2 for male and $\geq$96 g/m^2 for female patients, and LA dilatation was defined as LAVI $\geq$40 mL/m^2 according to the ASE recommendation [6].

Severe AS was defined as an aortic valve area $\leq$1.0 cm^2, mean trans-aortic valve gradient $\geq$40 mmHg, or peak trans-aortic valve jet velocity $\geq$4.0 m/s [20].

Data collection and definition

Patients' demographic, clinical, and follow-up data were obtained from medical charts or by contacting patients or referring physicians. Atrial fibrillation was defined by documentation on the electrocardiogram. Median follow-up period was 1128 days [interquartile range (IQR): 693–1802 days]. Residual LV hypertrophy and LA dilatation were assessed by echocardiography performed 1 year after surgery.

Outcome measures

Outcome measures were all-cause mortality and the incidence of major adverse cardiac and cerebrovascular events (MACCE). In

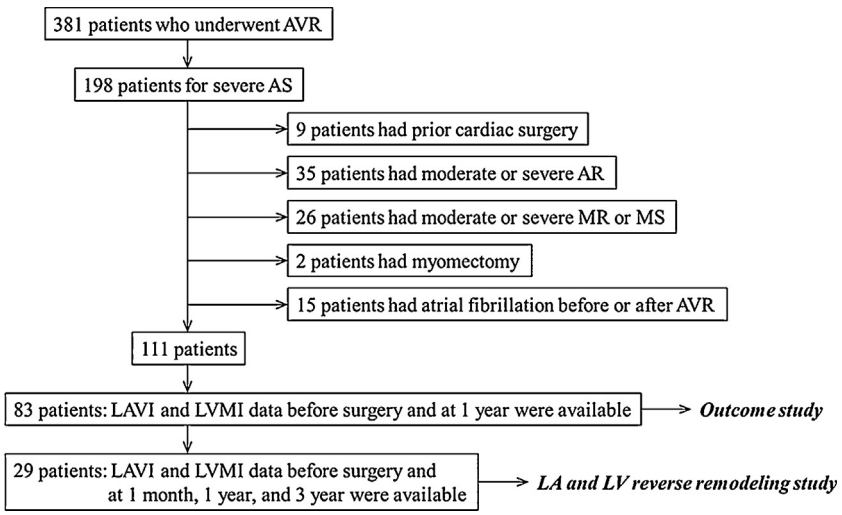

Fig. 1. Patient eligibility diagram. AR, aortic regurgitation; AS, aortic stenosis; AVR, aortic valve replacement; LA, left atrial; LV, left ventricular; MR, mitral regurgitation; MS, mitral stenosis; LVMI, LV mass index; LAVI, LA volume index.