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Original article

Usefulness of atrial function for risk stratification in asymptomatic severe aortic stenosis

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ABSTRACT

Aim: We aimed to evaluate the usefulness of left atrial (LA) mechanics and stiffness over global left ventricular (LV) longitudinal strain (GLS) for risk stratification in severe aortic stenosis (AS).
Methods: From a cohort of 89 prospective asymptomatic patients with severe AS and normal LV ejection fraction, 82 (32 men, mean age 73 ± 10 years) truly asymptomatic patients, scheduled after a negative exercise echocardiogram, were enrolled. Forty age- and gender-matched prospective, asymptomatic subjects served as controls. Predefined end points were the occurrence of symptoms (dyspnea, angina, syncope), and death during follow-up.
Results: At study entry, patients had: impaired LV GLS ($p = 0.001$), reduced LA reservoir ($p < 0.001$), high LA stiffness ($p < 0.001$), and increased valvulo-arterial impedance ($p < 0.001$) compared to controls. During follow-up [16 ± 14.9 months (ranging from 1 month to 4.2 years)], 53 patients (64.6%) reached one of the endpoints. Patients with events showed lower LV GLS ($p > 0.001$), lower LA reservoir ($p < 0.001$), and greater LA stiffness ($p < 0.001$) than those asymptomatic. On univariate Cox regression analysis, LV GLS ($p < 0.001$), LA reservoir ($p < 0.001$), and LA stiffness ($p = 0.004$) were strong predictors of adverse events. Kaplan–Meier curves showed that event-free survival was significantly higher in patients with a LV GLS ≥16.8% [$p < 0.001$; area under the curve (AUC) = 0.922; sensitivity = 86%, specificity = 80%], a LA reservoir ≥19.8% ($p = 0.001$; AUC = 0.860, sensitivity = 71%, specificity = 84%), and a LA stiffness <0.78 ($p < 0.001$; AUC 0.819, sensitivity 70%, specificity 89%). On multivariate analysis, only LV GLS remained significantly associated with patients' prognosis (hazard ratio = 1.49, 95% CI = 1.11–2.01, $p = 0.008$).
Conclusions: In asymptomatic patients with severe AS, an efficient cardiovascular system is based on an effective atrial-ventricular interplay. LA function assessment is useful for early identification of risk in these patients. LV GLS however was confirmed to be the best predictor of patients' outcome.
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Introduction

Aortic stenosis (AS) represents the most common valvular heart disease in the general population [1]. The occurrence of symptoms is crucial as a prognostic marker of this condition, which identifies patients that will require a short-term aortic valve replacement [2,3]. In contrast, the management of asymptomatic patients, particularly of those with preserved ejection fraction (EF), remains

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a controversial issue. Rest and exercise echocardiography play a pivotal role in risk stratification of asymptomatic patients with severe AS, providing essential information on left ventricular (LV) function, valve anatomy, and hemodynamics [4–7]. Furthermore, calcium score of the aortic valve, an increased global LV afterload, as well as subclinical LV longitudinal dysfunction are currently gaining relevant roles in the decision-making for asymptomatic patients [8–14].

Nonetheless, it has been clearly demonstrated that in AS, particularly in hypertensive patients, beyond the well-known consequences on ventricles and arteries, the chronically increased afterload is also accompanied by a progressive left atrial (LA) enlargement and dysfunction [15–17]. Moreover, an increased LA volume appears as a marker of poor long-term prognosis in severe AS [18].

However, the prognostic implication that an early detection of LA functional impairment might have in these patients has not been fully elucidated. Therefore, we sought to explore LA strain and stiffness in a cohort of asymptomatic patients with severe AS and preserved EF, with the aim to evaluate whether this additional analysis of cardiac function may provide useful information in the context of an accurate patients' risk stratification.

Methods

From June 2009 to January 2014, atrial mechanics were prospectively evaluated in 89 consecutive asymptomatic patients (36 men, mean age 76 ± 11 years) with severe AS who were referred to the echocardiographic laboratory for regular follow-up of their valve disease. Severe AS was defined as an aortic valve area (AVA) $<1\ cm^2$ (or indexed AVA $<0.6\ cm^2/m^2$), a trans-valvular peak velocity ≥4 m/s on continuous wave Doppler, and a mean gradient ≥40 mmHg[2]. To better clarify symptom status, we used exercise stress echocardiography, according to modified Bruce protocol, which has demonstrated an additive prognostic value over clinical findings, resting echocardiography and exercise testing for unmasking symptoms. An exercise echocardiogram was considered normal if the patient remained asymptomatic (no angina, limiting dyspnea at low workload, syncope, or near-syncope) during the test, with an adequate increase in systolic blood pressure (>20 mmHg), absence of complex ventricular arrhythmias and/or ≥2 mm horizontal or down sloping ST segment depression, and/or wall motion alterations [19,20]. Patients unable to perform exercise ($n = 4$) and those with a positive response ($n = 3$) were excluded from the study. Thus, the final population consisted of 82 truly asymptomatic patients (32 men, mean age 73 ± 10 years) with severe AS. No patient with paradoxical (low flow-low gradient) AS was enrolled.

Moreover, to exactly know the LA functional status in AS, mean values of LA strain and stiffness from the patients' cohort were compared to those from a control group without AS, including 40 age-gender matched asymptomatic subjects (15 men, mean age 73 ± 10 years), undergoing standard echocardiogram for global cardiovascular risk profile evaluation, which were prospectively and consecutively enrolled.

Both patients and controls all were in sinus rhythm. All had normal LVEF (≥50%), no more than mild associated heart valve disease, no kidney failure, no previous myocardial infarction, and optimal image quality for endocardial border detection and speckle tracking analysis.

At study entry, the following clinical data were collected: age, gender, body surface area (BSA), history of hypercholesterolemia (total cholesterol ≥190 mg/dl or patients receiving lipid-lowering therapy), diabetes mellitus (fasting blood glucose ≥126 mg/dl on two occasions or patients currently receiving oral hypoglycemic medication or insulin), systemic arterial hypertension (blood pressure ≥140/90 mmHg or patients receiving antihypertensive treatment), and overweight (body mass index ≥25 kg/m[2]).

The regional ethics committee approved the protocol, and all subjects gave written informed consent.

Standard echocardiographic evaluation

Echocardiographic examinations were performed using a VIVID-7 ultrasound machine (GE Vingmed Ultrasound, Horten, Norway), equipped with a phased-array transducer. M-mode, two-dimensional, color Doppler, pulsed-wave, and continuous wave Doppler data were stored on a dedicated workstation (EchoPAC, version 8.0.0; GE Medical Systems), for offline analysis. The measurements were made for three cardiac cycles, and the average value was calculated. The LV diameters, wall thickness, and outflow tract (LVOT) diameter were measured according to the recommendations of the American Society of Echocardiography [21].

The transvalvular aortic velocity time integral, mean pressure gradient, and peak aortic velocity were obtained using continuous wave Doppler ultrasonography. The right parasternal view was used whenever possible. The aortic valve area was determined using the continuity equation method and indexed to the BSA, using the Du Bois and Du Bois formula [22]. The stroke volume (SV) was calculated using the Doppler method as follows: $0.785 \times (\text{LVOT diameter})^2 \times \text{LVOT velocity time integral}$. The LVEF was derived using the biplane Simpson disk method [23]. The LV mass was determined with the area-length method [21], and the mass index was calculated as the LV mass/BSA (g/m[2]) ratio. The diagnosis of LV hypertrophy was determined using a LV mass index ≥102 g/m[2] in men and ≥81 g/m[2] in women [21]. A cut-off value of ≥0.45 for the relative wall thickness was considered to define a concentric remodeling [21]. The mitral flow peak velocities (E and A) and E/A ratio were measured using pulsed wave Doppler. Furthermore, from stored color tissue Doppler imaging loops, the value of E′ was obtained by averaging the peak early-diastolic velocities calculated at the level of mitral annulus. The E/E′ ratio was also included as an estimate of LV filling pressure.

Systemic arterial pressure was measured using an arm cuff sphygmomanometer before the Doppler echocardiographic examination. To estimate the global LV afterload, we calculated the valvulo-arterial impedance (Zva; mmHg/ml/m[2]) as the sum of systemic arterial pressure and the mean transvalvular pressure gradient divided by the SV index [24]. Left atrial areas and volumes were measured at end-LV systole, when the LA chamber is at its largest size, using the biplane method of disks (modified Simpson's rule) in the apical four- and two-chamber view and average value was obtained [21]. Mean areas and volumes of the LA were also indexed to BSA to correct for body size variability. Care was taken to exclude the pulmonary veins and LA appendage from the LA tracing and the plane of the mitral annulus was used as inferior border [21].

Speckle-tracking echocardiography

The analyses of both LA and LV mechanics were performed offline by a single experienced and independent echocardiographer from the recordings of two-dimensional (2D) gray-scale imaging, using the EchoPAC semi-automated two-dimensional strain software.

Three consecutive heart cycles were recorded and averaged, using a frame rate of 60–80 frames/s.

LA strain and LA stiffness

Left atrial function was studied using 2D speckle-tracking echocardiography performed from the apical four- and two-

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