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ORIGINAL ARTICLE

Increased left atrial stiffness in patients with atrial fibrillation detected by left atrial speckle tracking echocardiography

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KEYWORDS

Atrial fibrillation;
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Abstract *Objective:* This study was designed to determine the contribution of left atrial (LA) stiffness to atrial fibrillation (AF) progression by comparing AF patients with normal control subjects, and to evaluate whether LA mechanical function is related to the structural changes of LA.
Background: Abnormalities in the LA structure and function usually develop in patients with AF.
Patients and methods: Twenty paroxysmal and 20 persistent AF patients, were included and studied, using standard, tissue Doppler and speckle tracking echocardiography, and were compared with 20 matched controls. LA maximal volume, mitral annular velocities, and global longitudinal LA strain were measured. The ratio of E/e′ to LA strain was used as an index of LA stiffness.
Results: Study groups were comparable. Paroxysmal and persistent AF patients showed increased LA maximal volume (24.3 ± 3.2, 20.1 ± 5.2 vs. 18.3 ± 1.6, $p < 0.001$) and LV filling pressure (E/e′ = 9.5 ± 1.1, 7.9 ± 1.2 vs. 6.8 ± 1.1, $p < 0.001$), but decreased mitral annular velocities and LA strain (18.1 ± 2.8, 21.5 ± 4.3 vs. 25.9 ± 2.9, $p < 0.001$). LA stiffness was increased in patients with persistent and paroxysmal AF than in the control subjects (0.55 ± 0.12, 0.36 ± 0.09 vs. 0.26 ± 0.02, $p < 0.001$), and was related with LA diameter and LV filling pressure.
Conclusions: Patients with AF have increased LA stiffness in comparison with that of the control subjects and it is higher in persistent than paroxysmal AF. LA volume and LV filling pressure are independent predictors of LA stiffness.

1. Introduction

Atrial fibrillation (AF) is an independent risk factor for mortality across a wide age range in both men and women.[1] It is the most frequently detected arrhythmia in the clinical setting, and it is associated with increased cardiovascular risk.[2]

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AF is a progressive condition that begins with hemodynamic and/or structural changes in the left atrium (LA) and evolves through the paroxysmal and persistent stage.[3]

Better understanding of LA structure and function could lead to improvements in our ability to predict the risk of developing AF and the response to treatment in patients with this arrhythmia. LA enlargement has been widely related to AF, both in patients with persistent and paroxysmal AF.[4]

LA function can be estimated by two-dimensional echocardiography, Doppler analysis of transmitral flow, and tissue Doppler assessment of LV diastolic myocardial velocities. Speckle tracking echocardiography allows the quantification of LA function, and recently, noninvasive estimate of LA strain by speckle tracking has been proposed.[5]

Several studies have shown that LA stiffness increases with atrial remodeling and reflects a deteriorated reservoir function.[6,7] However, to date, little is known with regard to the change in LA mechanical function, including LA stiffness, in patients with AF.[8]

The present study was designed to compare the LA mechanical function, including LA stiffness, in patients with paroxysmal and persistent AF with normal control subjects, and to evaluate whether LA mechanical function, LV filling pressure and LA stiffness are related with the structural changes in LA.

2. Methods

2.1. Study population

We conducted a single center, case-control, cross-sectional study. The study population included 40 patients arranged into two groups: Twenty paroxysmal AF patients (51 ± 13 years, 60% male) and 20 persistent AF patients (49 ± 8 years, 65% male), who underwent echocardiography for the evaluation of cardiac structure and function, between April 2013 and December 2013. Inclusion criteria were as follows; (1) age older than 18 years, (2) recent onset and documented paroxysmal AF on an electrocardiogram or Holter recording in the previous 2 months, and (3) Patients with persistent AF or prior history of AF. Patients were excluded from the study if any of the following were present; (1) LV ejection fraction $< 50\%$, (2) history of ischemic heart disease, (3) dyspnea of New York Heart Association functional class $\geqslant$ II, (4) valvular stenosis or regurgitation $\geqslant$ moderate, (5) hyperthyroidism, acute illness, post-operative status and (6) age > 70 years.

The study protocol was approved by the ethical committee of Assuit faculty of medicine. A written informed consent was obtained from all participants. The consent form was designed with an explanation on the purpose and conduction of this research project. This form was to be explained to each participant; then a written consent was given. Participation was only proceeded after written consent of the participant. The full text of the form was approved by the Ethical Review Committee of Assuit faculty of medicine.

In all subjects, a full medical history was taken, and a complete physical examination was performed. Cardiovascular risk factors were recorded in detail, and all patients underwent standard 12 lead ECG.

Twenty healthy subjects with similar age, gender, and LV systolic function (mean age, 46 ± 8 years; male, 60%; mean LV EF, 64 ± 5.5%) were randomly selected from the subjects who volunteered for general routine health evaluation and underwent echocardiography. None of the controls had any cardiovascular or systemic disease and had sinus rhythm.

2.2. Transthoracic echocardiography

An iE33ultrasound system (Philips xMATRIX echo system, Eindhoven, The Netherlands) was used for the transthoracic echocardio graphic examination. All images and measurements were acquired from the standard views, according to the guidelines of the American Society of Echocardiography[9,10] and were digitally stored for offline analysis with QLAB 9. In the parasternal long-axis views, LA maximum anterior–posterior (A–P) diameter was measured (Fig. 1). In the apical 4-chamber view, LV end-diastolic and end-systolic volumes were measured and LV ejection fraction was calculated by the Simpson method. Pulsed-wave Doppler at the tip of mitral valve leaflets allowed us to measure the early (E) wave velocity and E deceleration time. The LV tissue velocity (e′, a′, s′) was measured by tissue Doppler imaging of the medial mitral annulus and E/e′ was calculated. From the apical 4- and 2-chamber view, the LA maximum volume (before

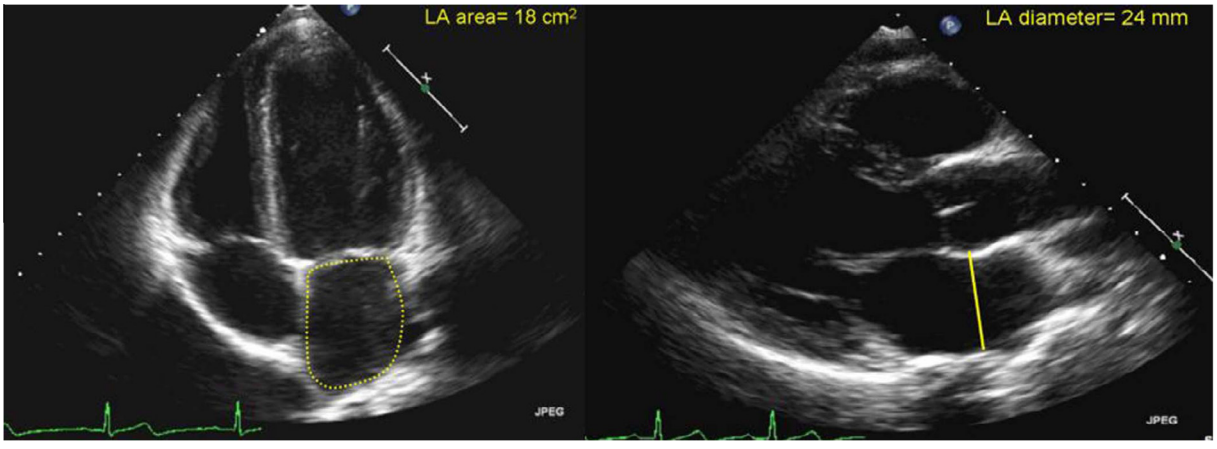

Figure 1 Measurements of left atrial area (before mitral valve opening) in the apical 4 chamber view (on the left side) and left atrial anterior–posterior diameter in the parasternal long axis view (on the right side).