Left Atrium Function in Systemic Hypertension

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Abstract: Objective: Assessment of left atrium (LA) deformation by 2-Dimensional Speckle Strain Imaging (2DSI) has been recently proposed as an alternative method of exploring LA function. The purpose of this study was to assess the relationship of LA function, particularly reservoir function, with LA structural remodeling related to left ventricular hypertrophy (LVH) in patients with hypertension (HTN) using conventional, tissue Doppler, and 2-Dimensional speckle-tracking echocardiography (STE).

Methods: Two hundred patients with systemic hypertension and fifty age and gender matched controls underwent routine transthoracic echocardiography and speckle tracking study of the left atrium. LA volume was calculated by the biplane method of disks (modified Simpson’s rule). Peak early diastolic transmitral flow velocity/peak early diastolic mitral annular motion velocity (E/Em)/peak systolic LA strain was defined as LA diastolic stiffness.

Results: Hypertensive patients were classified to group I with Left ventricle hypertrophy (LVH) (75% n = 150) and group II without LVH (25% n=50). LV Mass was 132.7 +/- 16.5g in control and 218.2 +/- 60.4 grams in study population. Left atrium volume indexed to body surface area was 19.4 +/- 3.4 versus 27.4 +/- 3.3 ml/m^2 in controls and hypertensive subjects respectively. (P value 0.000). Left atrium anteroposterior diameter was higher in the hypertensive group 4.22 +/- 0.56 versus 3.4 +/- 0.26 cm in control group (P value 0.000) Using STE left atrial longitudinal strain was 26.05 +/- 3.08 in hypertensive patients and 33.7 +/- 2.1 among controls ( P < 0.000).

Conclusion: Speckle tracking imaging might be required to detect early impairment of LA function in hypertension.

Keywords: systemic hypertension, speckle tracking, left atrium, longitudinal strain

1. Introduction

Although often referred to as “the forgotten chamber” left atrium (LA) plays a critical role in the clinical expression and prognosis of patients with heart and cerebrovascular disease. Together with conventional echocardiography parameters, new techniques such as two dimensional STE and three-dimensional echocardiography, allow early detection of LA dysfunction before anatomical alterations occur. LA dysfunction and its important prognostic implications maybe detected sooner by LA strain than by volumetric measurements. [1]

Left atrium anatomy: The LA cavity is located in the mediastinum, oriented leftward and posterior to the right atrium. LA structure is characterized by pulmonary venous component, a lateral finger-like appendage, an inferior vestibular component, which surrounds the mitral valve orifice, and a prominent body that shares the septum with the right atrium. The pulmonary venous component with venous orifices at each corner is situated posterior and superiorly, and directly confluent with the body. [2] The walls of the LA can be described as superior (roof) posterior (infero-posterior), left lateral, septal, and anterior [3]

2. Standard Echocardiography Methods

The LA size is measured at the end-ventricular systole when the LA chamber is at its greatest dimension, in long-axis view (antero-posterior diameter) and in 4-chamber view (Longitudinal and transverse diameters) [4] (fig1)

The anterior-posterior diameter, calculated with M-mode or 2D echocardiography, is no longer considered as adequately representative of the true LA dimension. For these reasons, the American Society of Echocardiography, in conjunction with the European Association of Echocardiography, recommend the measurement of LA volumes with either an ellipsoid model or the Simpson’s method in four- and two-chamber apical views [5].

During the cardiac cycle, the LA acts as a reservoir, receiving pulmonary venous return during LV systole; as a conduit, passively transferring blood to the LV during early diastole; and as pump in late diastole. In normal subjects, the reservoir, passive conduit, and pumping phases account for approximately 40, 35, and 25%, of the atrial contribution to stroke volume, respectively [6] (Fig 2)
3. Patients and Methods

The research ethical committee of Cairo University hospitals approved the research. From February 2014 to February 2015, a total of 200 patients (aged 55±13 years) with systemic hypertension were recruited from outpatient clinics at Cairo University hospitals. Exclusion criteria included a history or evidence of atrial arrhythmia, history of congenital heart disease, rheumatic heart disease, valvular surgery, coronary artery disease, or poor echocardiography window. All subjects gave their written informed consent for participation in the study. Fifty age-matched healthy individuals (aged 53±6.4 years) were recruited as controls.

Hypertension was defined as the use of antihypertensive therapy or the persistent elevation of blood pressure above 140/90 mmHg on two or more occasions with the patient in a sitting position for at least five minutes.

4. Echocardiography

Trans-thoracic echocardiography performed using available ultrasound system (IE 33, Philips Medical System). LV mass was calculated using Devereux formula = 0.8 \((1.04([LVID+PWT+IVST]^3-[LVID]^3]) + 0.6 \text{ g}\) where LVID is the left ventricle internal dimension, PWT is the posterior wall thickness, IVST is the interventricular septal thickness, 1.04 is the specific gravity of the myocardium, and 0.8 is the correction factor.

5. Measurements of LA Deformation by STE

The LA endocardial border was manually defined using a point-and-click technique. The software divided the region of interest into 6 segments and generated the averages of the values and curves of velocity, strain, and strain rate for each segment. Global peak LA strain during the whole cardiac cycle was calculated. To derive a noninvasive dimensionless parameter, the ratio of E/Em to LA peak strain was used to estimate the LA stiffness.

6. Statistical Analysis

All statistical calculations were done using computer program SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) release 15 for Microsoft Windows (2006) All Data were statistically described in terms of mean ± standard deviation (± SD). P values less than 0.05 was considered statistically significant.

7. Results

All patients were subjected to history taking and physical examination. Demographic parameters including waist circumferences (WC) and Body mass index were obtained. Patients were classified to group I with Left ventricle hypertrophy (LVH) (75% n= 150) and group II without LVH (25% n=50) (fig 3). Females constitutes 66.6% of group I and eighty % of group II (P value 0.693)
Hypertensive subjects had higher body mass index than controls (33.3+-7.4 versus 25.9+-7.2) (p< 0.001). Systolic blood pressure was significantly higher among the hypertensive subjects compared to controls 152.70+-9.7 versus 119.4+-8.06 mmhg respectively (p value 0.000) Diastolic blood pressure was 70.3+-6.9 in controls versus 95.23+-5.04 mmhg in hypertensive group (p value 0.000)

Conventional Echocardiography Study

Left ventricle end diastolic diameters 4.6+- 0.4 and 5.2 +-0.6 cm in normotensives and hypertensive subjects respectively (P value 0.075).Septum wall thickness (SWT) in normotensives and hypertensive was 0.83+-0.13 and 1.18 cm respectively (P value0.000 ). LV Mass was 132.7 +-16.5g and 218.2+-60.4 grams in controls and study population respectively

Left atrium anteroposterior diameter was higher in the hypertensive group 4.22+-0.56 versus 3.4+-0.26 cm in control group (P value0.000). Peak left atrial longitudinal strain was significantly reduced in study population (26.05+-3.08 % versus 33.7+-2.1 among controls ) ( P<0.000) (Fig4)

Figure 4: Left atrium longitudinal strain in a representative control subject (a) and hypertensive patient (b)

8. Discussion

It was recommended that echocardiographic evaluation of a patient with HTN should include assessment LV dimensions, mass, left ventricular function, LA volume, thoracic aorta and coronary artery patency [11] The current study showed that SWT and PWT were significantly thicker among the hypertensive subjects than in the controls. LV end-diastolic dimensions were higher among the hypertensive subjects than in the controls.

In a cohort of patients who were mainly hypertensive LA enlargement was a frequent finding in patients with preserved systolic function seen in clinical practice; this abnormality was found to be strongly related to LVH and to diastolic dysfunction, [12] Concordant with these findings we found that LA diameter was 3.4+-0.2 in controls and 4.2+-0.2 cm in hypertensive patients (p value0.000 ). Moreover it was 3.6+-0.15 cm in patients without LVH and 4.4 +-0.5 cm in patients with LVH (P value 0.0000)

Recent study found that prevalence of LA enlargement was 49.4% and 6.5% in hypertensive patients and normotensive subjects, respectively (P<0.0001) [13]. Moreover, The current study showed that left atrium volume indexed to body surface area in controls and hypertensive subjects was 19.4+-3.4 versus 27.4+- 3.3 ml/m² respectively. (P value 0.000)

During the reservoir phase, strain increases as a consequence of stretching in response to LA filling, reaching a positive peak just before mitral valve opening, the so-called peak atrial longitudinal strain . In the current study, it was significantly reduced in hypertensive group (26.05+-3.08 % versus 33.7+-2.1 among controls) (P<0.000)
9. Conclusion

- LA function by STE is significantly reduced in hypertensive patients compared to normal controls.
- It might be required to detect early impairment of LA function in hypertension, especially in the absence of LA enlargement because it is particularly sensitive in assessing LA function in hypertension.

References


