Left ventricle function in systemic hypertension from M-Mode to speckle tracking echocardiography

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Abstract

Background: Hypertension results in hemodynamic changes affecting the left ventricle .Two-dimensional echocardiography detect early diastolic dysfunction and left ventricle hypertrophy However ,the systolic function assessed by ejection fraction in most cases is not affected .Speckle tracking Echocardiography(STE) can detect early systolic dysfunction by longitudinal strain and strain rates even with normal ejection fraction

Objective: The aim of the study to assess subtle LV dysfunction in hypertensive patients with and without left ventricular hypertrophy (LVH) using speckle tracking echocardiography

Patients and Methods: Eighty hypertensive patients were recruited from outpatient clinics at Cairo University Hospitals from April 2014 to April 2015. Patients were classified into Left ventricle hypertrophy group (group I) and non-LVH group (group II). Twenty sex and age-matched healthy individuals were recruited as controls

Results: In spite of normal ejection fraction, LV systolic dysfunction was unmasked using the speckle tracking imaging that revealed reduced left ventricle global longitudinal systolic strains in hypertensive participants

Conclusion: In this group of hypertensive patients, myocardial strain parameters identified group of individuals with subclinical left ventricular systolic dysfunction despite preserved ejection fraction even before LVH occurs

Key words: Left ventricle, arterial hypertension, Tissue Doppler imaging, Pulsed Wave Doppler, speckle tracking echocardiography

Introduction

Hypertension is a major risk factor for a number of cardiovascular diseases including stroke, atherosclerosis, type II diabetes, coronary heart disease, and renal disease. It affects 26%of adults worldwide, and its prevalence is predicted to increase to 29% by 2025 **(1)**

The heart responds to long-term pressure overload in an attempt to stabilize cardiac output by means of LVH through an increase in myocyte thickness and increased deposition of extracellular matrix, adrenergic stimulation of the heart, and moving to a higher position on the Frank–Starling curve by volume expansion**(2)**

Echocardiograph evaluation of the hypertensive patient : Prevalence of LVH by echocardiography is estimated at between 20% and 60% for individuals with hypertension. LVH has been shown to be an independent risk factor for predicting myocardial infarction, sudden cardiac death, and heart failure **(3)**

The threshold for defining LVH by M-mode and two-dimensional echocardiography is accepted LVH thickness exceeding 11 mm **(4)**

Early detection of LV dysfunction before the development of LVH may represent a clinical finding that would justify aggressive treatment aimed at reducing cardiovascular morbidity and mortality; therefore it has to be considered in the assessment of global cardiovascular risk **(5)**

Speckle-tracking echocardiography (STE) is an angle-independent technique that may allow an accurate assessment of segmental myocardial deformation by grey-scale-based imaging analysis frame by frame. Moreover, the lack of angle dependency is of great advantage because myocardial strain could be tracked in two-dimensional echo imaging, along the direction of the wall and not along the ultrasound beam **(6)**

Patients and methods:

The study protocol was approved by the Ethics Committee Kasr Aliny Hospital at Cairo University. Eighty adult hypertensive participants aged 54.80+-14 years old were enrolled. Twenty age and gender matched normotensive subjects were recruited as controls. Patient written consent was given by all the participants. 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. **(7)** Patients were subjected to history taking, physical examination and demographic parameters including waist circumferences (WC) and Body mass index

Echocardiography: Echocardiography studies were performed using a commercial scanner (iE33; Philips Medical System, N.A., Bothell,WA, USA) according to the recommendations of the American Society of Echocardiography

M-mode Echocardiography: The left ventricular dimensions, ejection fraction and mass were calculated . LV mass was calculated using Devereux formula (ASE method) = 0.8 x1.04x[LVID+PWT+IVST]^3 – [LVID]^3 + 0.6 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 **(8)**

Pulsed wave Doppler: LV global diastolic function was determined: E and A wave peak velocities (cm/sec) and E/A ratio

Tissue Doppler Imaging TDI: Lateral mitral annular Tissue Velocities using TDI revealed myocardial peak systolic velocity Sm (cm/s) , myocardial early (Em) and late atrial (Am) velocities Em/Am ratio <1 indicates ventricular compliance impairment much more than the reversal of inflow E/A ratio which is affected by several confounders **(9)**

Speckle-tracking echocardiography STE: Three cardiac cycles were acquired and averaged for conventional measurement. Then Aortic valve closure time AVC was defined .Tracking was then performed automatically. Systolic function was assessed by measuring the global longitudinal peak systolic strain GLSS %. **(Fig.1)**

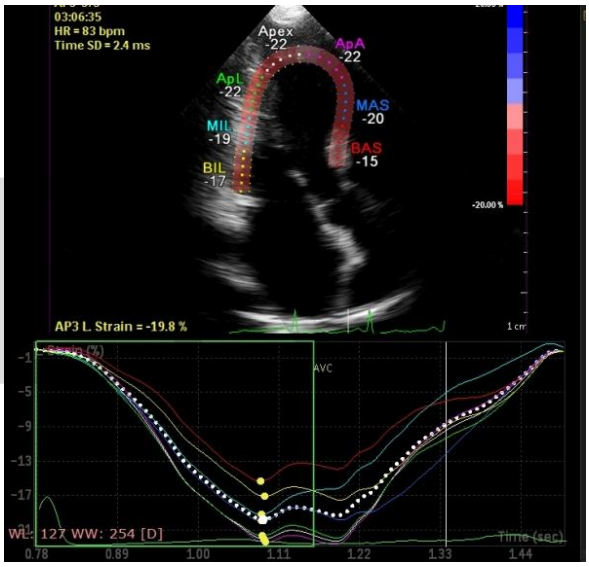

Fig. 1: Global LV longitudinal systolic strain by STE

Results:

Patients were grouped to two groups group I with Left ventricle hypertrophy (LVH) and group II without LVH **(Fig. 2)** 75 % of patients have LVH (n=60) and 25% (n=20) are hypertensive without LVH detected by echocardiography

Female constitute 66.6% and 80% of group I and II respectively (P value 0.693) **(Fig. 3)**

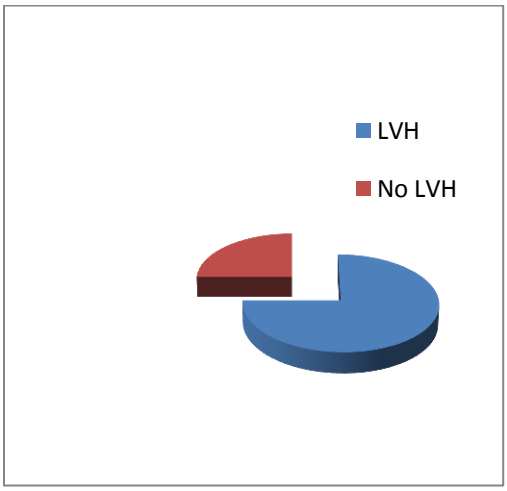

Fig.2: The two groups of the study

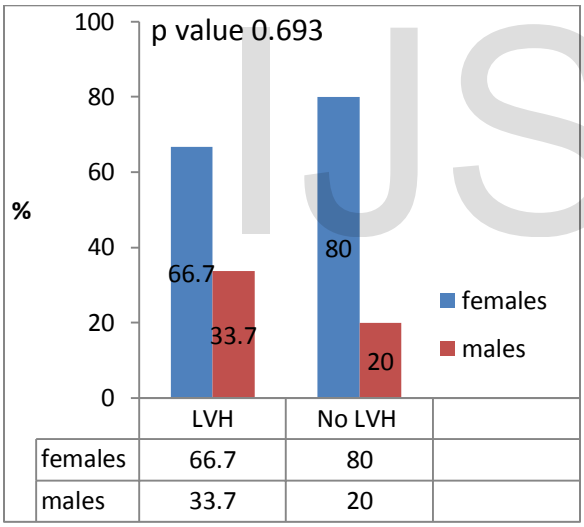

Fig. 3: Gender distribution in the study population

Patients with LVH are heavier with large waist circumference (WC). Mean BMI was 34.5+- 7.9 in group I versus 29.8+-3.7 in group II (P value: 0.086) .WC in group I and II was 117.8+-14.1 and 106.4+-12.7 cm respectively (P value 0.029).

Systolic blood pressure SBP was significantly higher among the hypertensive subjects compared to controls 152.7+-9.7 mmhg versus 129.4+-8.06

(P value < 0.001). SBP in group I and II was 156+-9.0 and 142.8+-1.3 mmhg respectively while DBP was 96.8+-4.2 and 90.4+-4.1 mmhg in Group I and II respectively.

Echocardiography parameters: left ventricle diastolic dimensions LVED was 4.6+- 0.4cm and 4.8+- 0.6 cm in normotensives and hypertensive respectively (P value 0.075). Septum wall thickness (SWT) was 0.8+-0.13in normotensives versus 1.1cm in hypertensive subjects (P value 0.000). Moreover , it was 1.18+-0.1 in LVH group versus 1.0+-0.16 in hypertensive subjects without LVH (P value 0.79) Echo derived LV mass using Devereux formula was 132.7+-16.5 in control group and 244.1+-44.5 versus 140.40+-21.6 grams in group I and II respectively

Pulsed Wave Doppler echocardiography: Mitral E/A ratio 1.2+-0.05 in control and 0.7+-0.1 in study population (P value< 0.001). **(Fig. 4)**

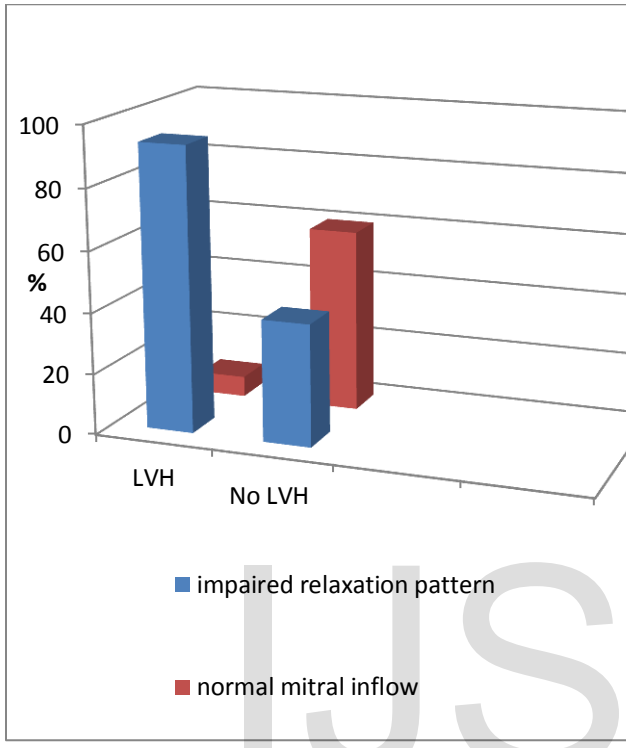

Fig. 4: Mitral inflow pattern in study population

Tissue Doppler imaging of lateral mitral annulus: Em/Am was 1.3 +-0.1 in controls and 0.9+--0.3 in hypertensive (P value 0.000). Estimated pulmonary capillary wedge pressure (PCWP) was calculated as follows: (1.25x (E/Em) + 1.9) it was 7+-0.8 and 10 +-3 mmhg in control and hypertensive groups respectively (P value 0.000)

Speckle-tracking Imaging STE: Global longitudinal systolic strains GLSS -22.45+-0.43in controls and -17.53+-1.3 among hypertensive (P value 0.000). Moreover, global longitudinal systolic strain rate GLSR was -1.13+-0.00 among control group and -1.00+-0.07 among hypertensive subjects (P value 0.000). Early diastolic strain rate in normotensives and hypertensive were 1.44+-0.14 and 1.06+-0.15 respectively (P value 0.000). Late diastolic strain rates were 0.70+ -0.11 and 1.012+-0.15 in normotensives and hypertensive respectively

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) Comparison of numerical variables between the study groups was done using Student t test for independent samples P values less than 0.05 was considered statistically significant.

Discussion:

It was reported that in middle-aged men central distribution of body fat is associated with increased BP, independently of body mass index and insulin resistance, thus suggesting a key role of central adiposity in the full expression of the "metabolic syndrome **(10)**

Concordant with other studies **(11)** we found that BMI in normotensive was 25.9 +-7.2 vs.34.5+-7.9 in group I and 29.8+-3.2 in group II (p value 0.086).

Echocardiography: Many studies pointed to the role of echocardiography for evaluation of patients with hypertension **(12)**.The current study showed that SWT and PWT are significantly thicker among the hypertensive subjects than in the controls. LV end-diastolic dimensions were higher among the hypertensive subjects than in the controls. Left atrial diameter was3.4+-0.2 in controls vs 4.2+-0.2 cm in hypertensive group (P value<0.001). Moreover it was 3.6+-0.15 cm in patients without LVH and 4.4 +-0.5cm in patients with LVH (P value 0.000). This is in agreement with other studies **(13)** that found in a cohort of patients who were mainly hypertensive left atrial 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.

Concerning LV systolic function by ejection fraction EF using Simpson method, it has been found from the present study that there were no significant differences between the three studied groups. Our results are compatible with those of Narayanan et al (**14)** who studied 52 hypertensive patients and 52 control individuals of similar age; they found that EF and endocardial shortening were similar in both groups. Mitral E/A ratio 1.22+-0.05 in control and 0.736+-0.11 in study population (P value< 0.001)

Moreover, E/Em ratio was higher in hypertensive group versus control (6.4 versus 4.1 respectively) (P value <0.001). This is in agreement with **Dekleva et al (15)** who studied 30 patients with hypertension (19 men/11 women, aged 55 +-8 years). They demonstrated that all patients had preserved systolic function (EF = 58 +-15%) and impaired LV relaxation (E/A = 0.79 +- 0.15).

The current study revealed that TDI derived myocardial performance index (MPI) was 0.344+-0.017 in control and 0.347+-0.037 in hypertensive group with non significant P value. Discordant with others who found that MPI remained significantly impaired in persons with hypertension compared to participants without. **(16)**

It was found that hypertensive patients with a preserved LVEF, impaired longitudinal strain is associated with serum levels of the tissue inhibitor of matrix metalloproteinase 1, a marker of myocardial fibrosis, which represents the main determinant of LV

diastolic dysfunction. These findings suggest that change in collagen turnover and the myocardial fibrotic process may cause contractile dysfunction when the LVEF is still normal **(17)**.

The current study showed significantly impaired longitudinal strain in hypertensive group compared to control using STE
(-17.53+-1.3 versus 22.45+-0.43) (P value 0.000). Moreover it was -17+-1.15 and -19+-0.27 in group I and II respectively so it identifies some early LV mechanic changes in hypertension even before LVH occurs

Conclusion: In hypertensive patients, STE reveals a systolic dysfunction before hypertrophy occurs (Stage A of ACC/AHA classification of heart failure) and identifies some early LV mechanic changes that might improve the clinical management of these patients.

Conflict of interest: None

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