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ASSESSMENT OF REGIONAL LEFT VENTRICLE SYSTOLIC FUNCTION BY DOPPLER STRAIN IMAGING IN HYPERTENSIVE PATIENTS

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All the authors contributed significantly to the research that resulted in the submitted manuscript.

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ABSTRACT

Objective: The objective of study was to evaluate regional myocardial function by strain (S) echocardiography in patients with or without diastolic dysfunction and healthy individuals with normal left ventricular ejection fraction.

Methodology: This crossectional comparative study was conducted at department of cardiology, Jinnah Hospital Lahore, from January 2012 to October 2012. Thirty hypertensive and thirty healthy individuals were enrolled in study. Strain Doppler echocardiography was used to evaluate 30 patients with hypertension and 30 age matched control subjects. With pulsed-wave Doppler, mitral inflow velocities were obtained to measure diastolic E and A waves, E/A ratio, and E-wave deceleration time(DT); with tissue doppler imaging early diastolic (Ea), and late diastolic velocities(Aa) and longitudinal peak systolic strain (S) values were measured.

Results: Mean age of study population in control subjects (Group A) 45±6 years and in hypertensive subjects (Group B) 53±7 years. Among control subjects, 18 were males (60%) and 12(40%) were females. In hypertensive subjects 16(53.3%) were males while 14(46.6%) were females. No difference was noted regarding age, sex, body surface area and heart rate. Among the hypertensive patients, 14(46.6%) were having diastolic dysfunction while 16(53.3%) patient were without diastolic dysfunction. Peak strain values were significantly decreased in patients with hypertension with or without diastolic dysfunction as compared to healthy individuals.

Conclusion: Hypertensive patients with or without diastolic dysfunction have reduced left ventricular systolic longitudinal strain.

Key Words: Hypertension, Echocardiography, Diastolic Dysfunction, Peak Systolic Strain

INTRODUCTION

Usually Ejection Fraction (EF) and Fractional Shortening (FS) performed in routine echocardiography is taken as criteria for assessing the systolic function of the heart. EF does not take in to account the longitudinal and circumferential shortening of the myocardium. Doppler tissue imaging, which measures the velocity of myocardium (in the longitudinal direction from apical windows and in the radial direction from short-axis scans) during systole and diastole is used to quantify ventricular function and is more sensitive to subtle changes in contractility than ejection fraction.[1] However, tissue velocities are affected by translational movement and tethering, making it difficult to discriminate akinetic segments that are pulled (or tethered) from actively contracting segments. In addition, velocities are not uniformly distributed across the myocardium, decreasing from base to apex, making difficult the establishment of reference values.

Measurements of myocardial strain and strain rate (SR) are newer indices that have the potential to overcome these limitations. Strain and SRs represent the magnitude and rate, respectively, of myocardial deformation, which is an energy-requiring process that occurs in both systole and diastole.[2] Abnormalities of myocardial deformation are seen early in the development of many pathophysiologic states, including ischemia, and thus provide a sensitive means for detecting regional myocardial dysfunction.[3,4]

Strain images are new echocardiographic modalities on basis of tissue doppler imaging to assess the regional systolic shortening of LV. Strain analysis evaluate whether regional myocardium is stretching or shortening.[5,6] Clinical studies have reported that large number of patients with congestive heart failure may even have normal Ejection Fraction (EF).[7,8] The majority of patients have arterial Hypertension (HTN) in which presence of congestive heart failure has been attributed to isolated Left Ventricle (LV) diastolic dysfunction. According to this concept doppler echocardiographic studies in patients with hypertension have shown high prevalence of diastolic filling abnormalities.[9,10]

We hypothesized that the longitudinal LV systolic contraction might be decreased in patients with hypertension as compared to healthy individuals without hypertension. The peak systolic strain values may be further reduced in hypertensive patients, who have concomitant abnormal LV diastolic filling despite the presence of normal LVEF. The objective of study was to evaluate regional myocardial function by strain (S) echocardiography in patients with or without diastolic dysfunction and healthy individuals with normal left ventricular ejection fraction.

METHODOLOGY

This crossectional comparative study was conducted at department of cardiology, Jinnah Hospital Lahore, from January 2012 to October 2012.

Hypertensive patient included in study were (Systolic BP>140mmHg and Diastolic BP>90mmHg) taken as cases while non-hypertensive (Systolic BP<130mmHg and Diastolic BP<80mmHg) age matched healthy individuals were taken as control. Patients with known ischemic heart disease, valvular heart disease, arrhythmias, heart blocks were excluded.

Our study population consisted of 60 subjects. They were divided into two groups, group A and group B. Group B included 30 patients that were hypertensive and were referred from outdoor patient department Jinnah Hospital Lahore for evaluation and treatment of arterial hypertension. Group A included 30 age matched healthy normotensive individuals that served as control subjects. The hypertensive patients in group B were further divided into two subgroups; hypertensive patients with diastolic dysfunction (B1) and hypertensive patients without diastolic dysfunction (B2).

All patients were examined by performing transthoracic echocardiography on GE-VIVID-7 machine. Left ventricular function was evaluated by conventional Doppler ,tissue Doppler and strain echocardiography. Longitudinal peak systolic strain(S) was measured from the basal , mid and apical segments of left ventricle walls. Doppler measurements like LV diastolic filling (E), peak atrial filling (A) and their ratio (E/A),and mitral deceleration (DT) and tissue derived indices were recorded at mitral annulus,early diastolic velocities Ea ,and late diastolic velocities(Aa).

The data was analyzed using SPSS 16 and results were presented as: Age of patients was presented as mean ±S.D. (Standard Deviation). We were looking for left ventricular longitudinal strain which is a quantitative variable so we applied student t test as the test of significance. A p-value<0.05 was taken as statistical significant.

RESULTS

Conventional Doppler findings were similar in patients and control groups. The two groups were similar with respect to systolic ejection fraction. However peak systolic strain was significantly less in hypertensive patients than non-hypertensive patients. Thirty healthy individuals without hypertension (Group A) and thirty patients with hypertension (Group B) were examined. Mean age of study population in control subject 45±6 years and in hypertensive subjects 53±7 years. The control subjects were further divided into males 18(60%) and 12(40%)

Table 1: Demographics and Clinical Characteristics of Patients and Control Group

Variables	Control Subjects	HTN subjects
Age (Years)	45±6	53±7
Men	18(60%)	16(53.3%)
Systolic BP	120±8mmHg	150±15mmHg
Diastolic BP	70±8 mmHg	90±10mmHg

Values expressed as mean±SD

females. Among hypertensive subjects 16(53.3%) were males while 14(46.6%) were females (Table 1). No difference was noted regarding sex, body surface area, heart rate. Systolic and diastolic blood pressures were comparable between the patients who were hypertensive with diastolic dysfunction and hypertensive patients without diastolic dysfunction. Among the hypertensive patients 14 (46.6%) were having diastolic dysfunction while 16(53.3%) patients were without diastolic dysfunction. The peak systolic strain in all segments was significantly reduced in hypertensive patients as compare to healthy individuals as evident from Table 2. Peak systolic strain was significantly decreased in hypertensive patients with diastolic dysfunction as compared to hypertensive patients with normal diastolic function (Table 3).

DISCUSSION

Strain is a measure of tissue deformation and is defined as the change in length normalized to the original length.[11] When corrected for apico-basal distance, this may indicate expected values for longitudinal systolic shortening of ~12-20%.[12] LV systolic function is commonly considered normal in the presence of a normal EF and fractional shortening despite the fact that neither measures reflect all

Table 2: Echocardiography Findings of the Study Population

Parameters	Normotensive Group(A)	Hypertensive Group(B)	P-value
E/A ratio	1.02±0.53	1.04±0.48	0.87
Deceleration time (ms)	15.27±0.16	192.50±-69.2	0.002
Ejection fraction(%)	65.30±4.88	63.37±-7.89	0.259
Strain anterior wall	-20.0±-3.92	-15.40±-5.84	0.001
Strain lateral wall	-19.47±-3.30	-15.90±-5.03	0.002
Strain inerior wall	-18.93±-2.9	-15.53±-5.20	0.003
Strain posterior wall	-19.10±-2.29	-15.93±-4.43	.002

Table 3: LV Strain Values in Hypertensive Patients with (B1) and without Diastolic Dysfunction (B2)

Strain	Group B1	Group B2	P-value
Strain anterior wall	-11.13±-3.69***	-20.86± -3.86	<0.001
Strain lateral wall	-11.88±-2.09***	-20.50± -2.82	<0.001
Strain inferior wall	-11.13±-2.28***	-19.79± -5.29	<0.001
Strain posterior wall	-11.13±-3.03***	-19.36± -3.34	<0.001

*** P<0.001

aspects of the LV contractile function.[13] This study demonstrates that patients with arterial hypertension with normal EF and fractional shortening have decreased longitudinal LV systolic function, especially in hypertensive patients with diastolic dysfunction. In a study by Ceyhan et al, a significantly decreased peak systolic strain was seen in prediabetics and diabetics when comparing with the healthy individuals.[14] We studied in hypertensive patients with or without diastolic dysfunction. Impaired mid wall myocardial shortening is most pronounced in patients with LV hypertrophy with hypertension. Investigators have previously shown decreased LV peak systolic velocities in patients with hypertension and hypertrophy.[15] The calculation of local velocity gradients as strain, which measures regional contractility independent of the effect of tethering and translation.

In a study by Atilqan et al, which included 57 hypertensive patients and 48 normotensive controls, Hypertensive patients had significantly decreased longitudinal myocardial function compared to control subjects determined by septal (-1.25±-0.30 vs. -1.02±-0.33, p<0.001) and lateral(-1.20±-0.28 vs. 1.02±-0.41, p<0.01) SR (1/s) measurements. This result is in accordance with the results of our study.[16]

Poulsen et al, in their study showed significantly decreased peak systolic stain rate in different segments of left ventricle in patients of hypertension and diastolic dysfunction which is in accordance with our study. Furthermore, the peak systolic strain was significantly lower in all segments of the LV in patients with hypertension and diastolic dysfunction compared with healthy subjects.[17]

The subendocardial longitudinal-oriented myocardial fibers have shown to be particularly vulnerable to ischemia leading to a dominant decrease in shortening in the longitudinal axis. Hypertension and associated LV hypertrophy are important risk factors for developing chronic congestive heart failure and although these patient with congestive heart failure and a normal LVEF have a lower mortality risk than patients with reduced EF, the mortality risk is still significantly increased over control patients.[18]

In a study by Saghir et al, showed individuals with

hypertensive left ventricular hypertrophy has significantly reduced peak systolic strain (-16±-3.2%) compared with control subjects (-21.7±-3.5%). This is in accordance with our study, where there is significantly decreased peak systolic strain in hypertensive patients with diastolic dysfunction[-20.86±-3.86 vs. -11.13±-3.96 ($p<0.001$)].[19]

The assessment of LV systolic longitudinal contraction strain analysis gives new insight in myocardial function in hypertension that might improve our patho-physiological understanding and identify patients at high risk who might benefit from regression of LV hypertrophy following a more aggressive anti hypertensive treatment program. The combination of depressed longitudinal systolic contraction and abnormal diastolic LV filling may play a key role in the development of acute and chronic heart failure in patients with hypertension. However, the clinical significance of these findings is unknown and additional clinical correlation studies are required to evaluate the significance of decreased longitudinal LV function.

STUDY LIMITATIONS

Myocardial ischemia because of the presence of coronary artery disease cannot be ruled out, as no angiography was performed. However, all patients were free of angina and` without signs of ischemia or prior infarction. The relative small number of patients and their different medication regimens makes it difficult not only to rule out, but also to demonstrate any effects of a given type of anti hypertensive medication.

CONCLUSION

Patients with hypertension with or without isolated diastolic dysfunction were shown to have reduced LV systolic longitudinal function assessed by strain doppler imaging although with normal ejection fraction.

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