Original Article

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Evaluation of Left Ventricular Strain and Deformation in Hypertensive Patients by Speckle Tracking Echocardiography

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Abstract

Background: Chronic hypertension (HTN) leads to variouspathophysiological and hemodynamic changes that ultimately culminates into left ventricular hypertrophy (LVH) and heart failure. Two-dimensional speckle tracking echocardiography (2D-STE) is novel technique that allows rapid and accurate analysis of incipient stages of systolic and diastolic left ventricular(LV) dysfunction. *Objective:* The objective of this study was to assess the ability of 2D-STE in the assessment regional and global LV strains to characterize features of subclinical LV dysfunction in patients withsystemic hypertension and relatively preserved ejection fraction (EF). *Material and Methods:* Two-dimensional echocardiographic (2DE) images of the LV were acquired in apical four-chamber and parasternal short-axis at the basal levels in 60 subjects, including 15 healthy controls and 45 patients with systemic hypertension. Longitudinal strain (LS) and circumferential strain (CS) were quantified in 13-segment model using Philips IE-33 x matrix machine. *Results:* In comparison with normal controls, global longitudinal LV systolic strain (LVGLS) and global circumferential LV systolic strain (LVGCS) was significantly attenuated in the patients with chronic systemic hypertension. Regional strain in longitudinal axis was significantly reduced at Apex(Ap) and Apicolateral (Apl) and Mid inferior septum (MIS) segment of LVin hypertensive population compared to normotensive group while the regional strain in circumferential axiswas significantly reduced at all six segments (basal anterior, basal anterior septum, basal inferior septum, basal inferior, basal inferior lateral wall and mid anterolateralwall). Strain rate (SR) values were insignificantly reduced in both hypertensive groups with and without LVH in early diastole (at peak E). *Conclusion:* 2D-STE detected substantial impairment of LV strain and regional deformation in chronic hypertensive patients with apparently preserved LVEF, which identifies higher risk subgroups for earlier pharmacolgical intervention in patients with HTN.

Keywords: Hypertension (HTN); Left Ventricular Strain (LVS); Speckle Tracking Echocardiography (STE).

Introduction

Hypertension is animportant identifiablerisk factor for a spectrum of cardiovascular diseases including heart failure, stroke, coronary heart disease andperipheral vascular disease. In the recent years the incidence of HTN and its associated mortality has increased dramatically. The current globally estimated prevalence of HTN in adults is 26% and is expected to increase to 29% by 2025 [1]. Systemic hypertension increases LV wall stress that triggers activationof various neurohormonal pathways leading toexpression of genes regulating structural remodelling of myocardium and extra cellular matrix leading to increasein LV Mass and sets the stage for progression of systolic and diastolic dysfunction [2]. Chronic HTN ultimately causesmaladaptive leftventricular hypertrophy which sets the stage for irreversible deterioration of LV function and eventually results in progression to congestive heart failure [3,4]. Henceforth, it is of utmost importance to detect early stages of LV impairment in patients with chronic arterial hypertension, in order toidentify patients at high risk for developing heart failure and commence appropriate medical intervention.

Ejection fraction (EF) is the current gold standard in echocardiographicallyassessingthe global left ventricular (LV) systolic function [5]. Calculation of LVEF by M mode and biplane method is a sophisticated approach but it does not measure the complex three-dimensional spatial deformation of LV in the longitudinal and circumferential plane. Thus, the assessment of EF only detects impairmentin LV function in a relatively advanced state when the heart failure has already settled in [6]. These subclinical and subtle changes in myocardial deformation, that occur prior to extensive LV impairment remain undetected by conventional EF assessment [7].

Tissue deformation imaging by speckle tracking echocardiography is a recently introduced novel technique, which quantifies regional myocardial deformation assessed byultrasound-based strain and strain rate (SR) usingmyocardial Doppler data or B-mode imaging. This is a precisetechnique with rapid offline analysis to assess regionalleft and right ventricular function and has an added value in quantifyingovert dysfunction or unravelling cardiac-pathology [8]. Strain is a dimensionless quantitative measure of tissue deformation. During the ventricular systole, muscle contracts and shortens in the longitudinaland circumferential dimensions (a negative strain) andthickens or lengthens in the radial direction (a positivestrain) [9]. Strain is calculated as fractional or percent change from the original value and can be measured in longitudinal, radial and circumferential directions . SR measures the time relationship of tissue deformation. SR is expressed as the strain per unit time [10]. In contrast to Tissue Doppler imaging, speckle-tracking echocardiography (STE) is an angle independent technique that is based on the concept of grey-scale-based imaginganalysis and interference patterns frame by frame, that calculates segmental myocardial deformation. The lack of angledependency is of great advantage because regional myocardialstrain (ε) could be tracked in two-dimensional echoimaging, along the direction of the wall and not alongthe ultrasound beam [11].

Material and Methods

The study was a prospective observational study in a tertiary care centre, Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow in patients with chronic hypertension carried out from September 2016 to August 2017. Thestudy was done on 45 hypertensive patients and 15 age- and sex-matched healthy subjects as a control group. The control subjects had no identifiable cardiovascular risk factors and were not receiving any medications. The controls were healthy volunteers recruited from the hospital staff, medical and nursing students, and members of the local community. Written informed consent was obtained from all patients, and the study protocol was approved local ethics committee.

Inclusion Criteria

Inclusion criteria were any adult patient with chronichypertension with or without ECG criteria of LVHin sinus rhythm and normal ejection fraction (EF).

Exclusion Criteria

We excluded patients with ejection fraction <50% or with symptoms or signs of heart failure, diabetes mellitus, patients with known coronary artery disease, regional wall motion abnormalities on echocardiography, patients with significantvalvular disease, hypertrophic cardiomyopathy and patients with atrial fibrillation or other rhythm disturbances.

Methods

Patients visitingcardiology OPD with the diagnosis of systemic hypertension were thoroughly examined after taking detailed clinical history and were subjected to routine hematological and biochemical investigations. Measurement of blood pressure (BP) was taken according to American Heart Association and American College of Cardiology 2013 guidelines for the management of BP [12]. Resting 12-lead surface electrocardiogram was performed with special attention paid towards ECG voltage criteria of LVH. Conventional 2D echocardiography and 2D speckle tracking imaging was performed using Philips IE 33 X matrix machine. All images were obtained in patients and controls in the left lateral decubitus position at end-expiration according to the recommendations of the American Society of Echocardiography (ASE) and connected to single-lead ECG [13]. All standard measurements were obtained in the parasternal long and short-axis views, apical four-chamber view by the single echocardiographer. Quantification of the LV dimensions was done using M-mode echocardiog-raphic method.

Strain imaging was analyzed using QLAB commercial software of Phillips for 2D strain imaging with speckle tracking method. After manual tracing of the endocardial border of 2D tomographic images of LV in longitudinal and short axis plane at mid cavity level at the endsystolic frame and selecting

the appropriate region of interest, including the entire transmural wall, the software automatically determined six segments in each view. Each segmental strain curve was generated by frame-by-frame tracking of the acoustic markers in the myocardial tissue. The marker tracking quality was scored as optimum or poor. Segments with poor acoustic marker tracking despite manual readjustments of the region of interest were deleted from the analysis. Peak systolic longitudinal strain (LS) was measured in 7 segments (the apex, basal anterolateral wall, mid anterolateral, apicolateral wall,apical septum,mid inferior septum,and basal inferior septum. The analysis was done in three cardiac cycles and averaged value was taken as mean of regional and global longitudinal strain. Peak circumferential strain (CS) were measured in six segments (mid anterior, mid anterior septum, mid inferior septum, mid inferior, mid inferior lateral wall and mid anterolateral wall) from a mid-LV short-axis view. Again three cardiac cycles were analyzed and values were averaged and taken as the mean CS. For parasternal short axis imaging the transducer was placed in the left parasternal region at the third and fourth intercostal space. The apical views were recorded with the transducer in the fifth intercostal space in the anterior axillary line close to or over the point of maximal impulse. Images were recorded at frame rates between 60 and 80 frames per second for optimal post processing analysis. All examinations were recorded for offline measurement.

Statistical Methods

Continuous variables were expressed as mean ± SD. The differences between cases and control were analyzed by independent samples student t tests (SPSS 16 software, SPSS Inc., USA). For the categorical variables chi square test was used. A two tailed p-value of <0.05 was considered to be significant.

Results

Regarding demographics and risk factors of the studypopulation, there were no significant differences between the hypertensive subjects and healthy controls. No significant differences were found between the two groups in terms of age or sex (Table 1). ECG voltage criteria of LVH were present in 26.7% of hypertensive patients but they were absent in control group (Table 2). There was a substantially higher systolic and diastolic bloodpressure recorded in patients with systemic hypertension than in the control group (p < 0.001).

Table 1: Baseline demographic, clinical and echocardiographic characteristics in two groups.

Parameters	Cases (n=45)	Controls (n=15)	P value
Age in years(Mean±SD)	54.38±12.38	46.80±14.07	0.06[1]
Male	30(66.7%)	6(40%)	0.06[2]
Female	15(33.3%)	9(60%)	
SBP	168.67±14.20	118.53±6.90	0.0001*
DBP	94.27±6.83	81.33±2.79	0.0001*
LVEF(Mean±SD)	64.31±5.36	62.40±3.64	0.20
LVEDD (mm)(Mean±SD)	39.38±4.92	37.20±3.59	0.12
IVSDd(mm)(Mean±SD)	12.94±5.87	9.87±1.59	0.06
LVPWDd (mm)(Mean±SD)	11.98±2.35	10.57±1.34	0.03*
LA diameter(mm)(Mean±SD)	29.62±5.07	28.13±3.64	0.30
LV mass index(g/m2)	76.05 ± 12.53	75.58 ± 14.52	0.26

Table 2: Comparison between the study groups regarding presence of ECG voltage criteria of LVH.

ECG	Cases(n=45)	Controls(n=15)	P-value[1]
LVH	12(26.7%)	0(0.0%)	0.07
Normal	33(73.3%)	15(100%)	

Conventional echocardiography

Conventional echocardiographic data of patients with systemic hypertension and controls are listed in Table 1, which revealed no significant difference in the global LV ejec-tion fraction (LVEF), left ventric-ular end diastolic dimension (LVEDD), interventricular-septal dimension in diastole (IVSDd) and LA diame-ter between the two groups. However, the left ventricular posterior wall dimension in diastole (LVPWDd) was significantly higher in the hyperten-sive group. There was no significant difference in LV mass between two groups.

2D speckle tracking(2D-STE) Strain/Strain rate

In comparison with normal con-trols, LVGLS was significantly attenuated in patients with

systemic hypertension. LVGLS was found to be significantly (p=0.0001) lower among cases (-17.31±3.82) compared to controls as shown in Figure 1 and Table 3. LVGLS was significantly (p=0.003) lower among cases with LVH on ECG (-14.58±2.50) compared to cases without LVH on ECG (-24.67±2.49). Similarly LVGLS was found to be significantly (p=0.03) lower among cases with LVH on 2decho(-16.52±3.53) compared to cases without LVH (-19.07±3.97).LVLS at Apl (-18.29±6.53 vs -23.73±5.78, p-0.006), ApS (-20.71±8.34 vs -25.73±5.31, p-0.03) and MIS(-18.47±8.15 vs -23.13±6.06, p-0.04) were found to be significantly (p<0.05) lower among cases compared to controls. (Table 4 & Figure 3). LVGCS was found to be significantly (p=0.0001) lower among cases (-21.82±5.10) compared to controls (-30.53±7.44) (Table 3 & Figure 2). All the LVCS parameters in different LV segments were found to be significantly (p<0.05) lower among cases compared to controls. (Table 4 & Figure 4). LVGCS was found to be insignificantly (p=0.52) lower among cases with LVH on ECG (-21.00±4.45) compared to cases without LVH (-22.12±5.34). Similarly LVGCS was found to be insignificantly (p=0.47) lower among cases with LVH on 2d Echo (-21.45±5.81) compared to cases without LVH on 2d echo (-22.64±2.97).

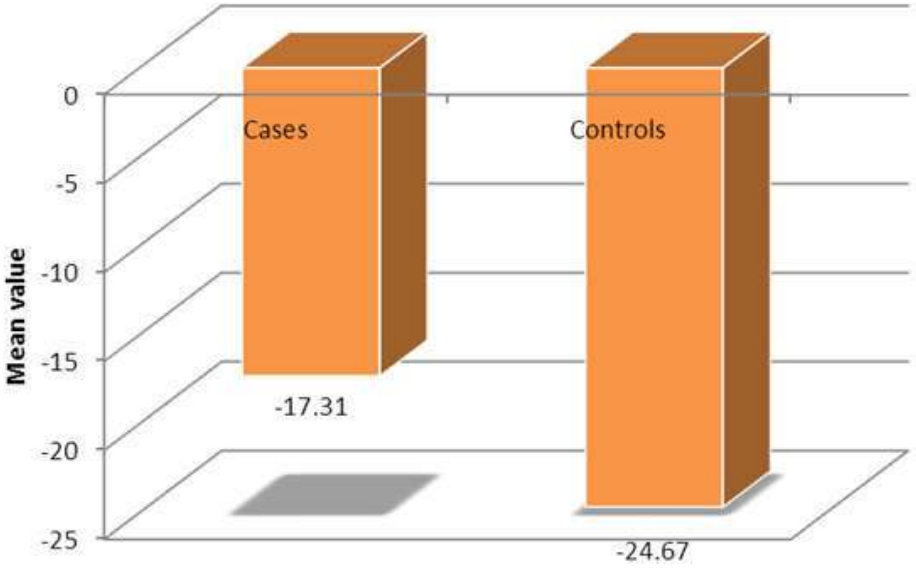

Fig. 1: Comparison of LVGLS between hypertensive subjects and healthy controls

Fig. 2: Comparison of LVGCS between hypertensive subjects and healthy controls

Table 3: Comparison between two groups regarding Left Ventricle global longitudinal and circumferential strain.

Parameters	Cases (n=45)	Controls (n=15)	p-value[1]
LVGLS (Mean±SD)	-17.31±3.82	-24.67±2.49	0.0001*
LVGCS (Mean±SD)	-21.82±5.10	-30.53±7.44	0.0001*

Table 4: Comparison between two groups regarding regional systolic strain in longitudinal and circumferential axis.

LVLS & LVCS parameters	Cases (n=45)	Controls (n=15)	P-value[1]
LVLS-BAL	-22.76±9.09	-24.33±7.29	0.54
LVLS-MAL	-17.02±7.25	-20.60±17.56	0.26
LVLS-Apl	-18.29±6.53	-23.73±5.78	0.006*
LVLS-Apex	-19.58±6.35	-22.20±12.61	0.29
LVLS-ApS	-20.71±8.34	-25.73±5.31	0.03*
LVLS-MIS	-18.47±8.15	-23.13±6.06	0.04*
LVLS-BIS	-15.44±10.51	-20.60±4.20	0.07
LVCS-BAS	-24.51±6.20	-31.47±7.54	0.001*
LVCS-BA	-22.51±7.05	-26.67±9.46	0.07
LVCS-BAL	-19.80±9.76	-28.00±7.28	0.004*
LVCS-BIL	-22.64±8.20	-28.64±13.34	0.04*
LVCS-BI	-21.20±7.93	-31.60±8.21	0.0001*
LVCS-BIS	-23.09±8.05	-31.00±9.79	0.03*

The results of this study indicated that the strain rate (SR) values were insignificantly reduced in both hypertensive groups with and without LVH in early diastole (at peak E). However, there was no significant reduction in SR except LVLSR at Apex. LVLSR-Apex was found to be significantly (p=0.005) lower among cases (1.24±0.46) compared to controls (1.70±0.68). (Table 5 & Figure 5 & 6)

Table 5: Comparison between two groups regarding regional strain rate in early diastole (at peak E) in longitudinal and circumferential axis.

LVLSR & LVCSR parameters	Cases (n=45)	Controls (n=15)	P-value[1]
LVLSR-BAL	1.35±0.67	1.60±0.51	0.19
LVLSR-MAL	1.08±0.64	1.37±0.58	0.13
LVLSR-Apl	1.31±0.57	1.50±0.59	0.26
LVLSR-Apex	1.24±0.46	1.70±0.68	0.005*
LVLSR-ApS	1.29±0.58	1.61±0.66	0.08
LVLSR-MIS	1.25±0.79	1.55±0.70	0.21
LVLSR-BIS	1.01±0.64	1.35±0.54	0.07
LVCSR-BAS	1.20±0.51	1.09±0.43	0.45
LVCSR-BA	1.18±0.45	1.09±0.59	0.54
LVCSR-BAL	1.27±1.11	1.02±0.55	0.41
LVCSR-BIL	1.18±0.61	1.00±0.58	0.30
LVCSR-BI	1.14±0.50	1.04±0.64	0.53
LVCSR-BIS	1.15±0.53	0.99±0.57	0.35

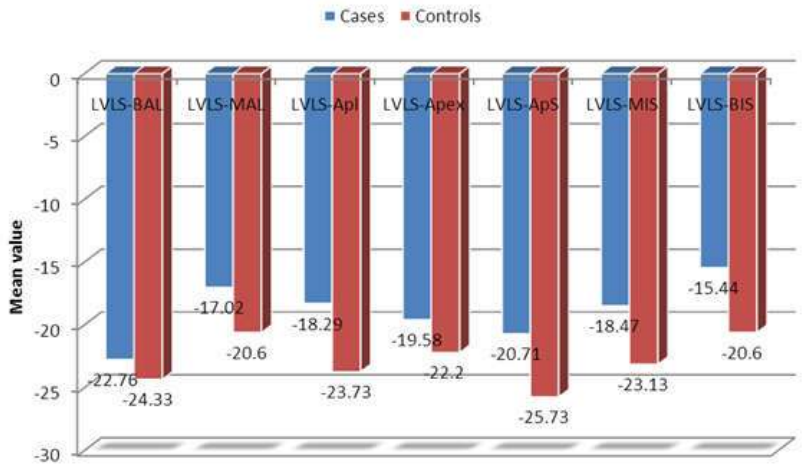

Fig. 3: Comparison of regional LVLS parametersbetween hypertensive subjects and healthy controls

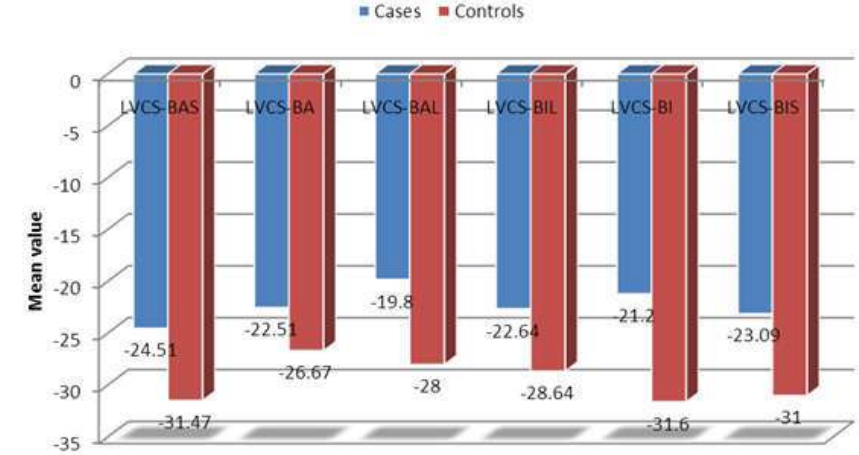

Fig. 4: Comparison of regional LVCS parameters between hypertensive subjects and healthy controls

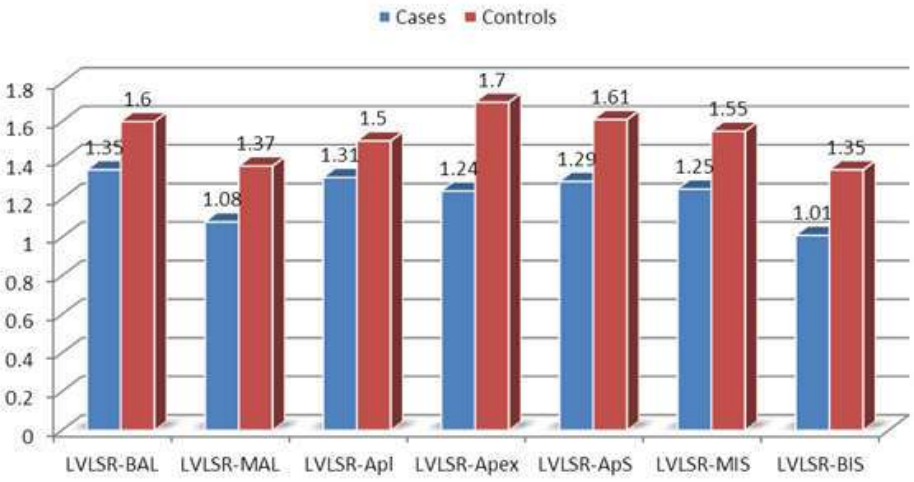

Fig. 5: Comparison of regional LVLSR between hypertensive subjects and healthy controls.

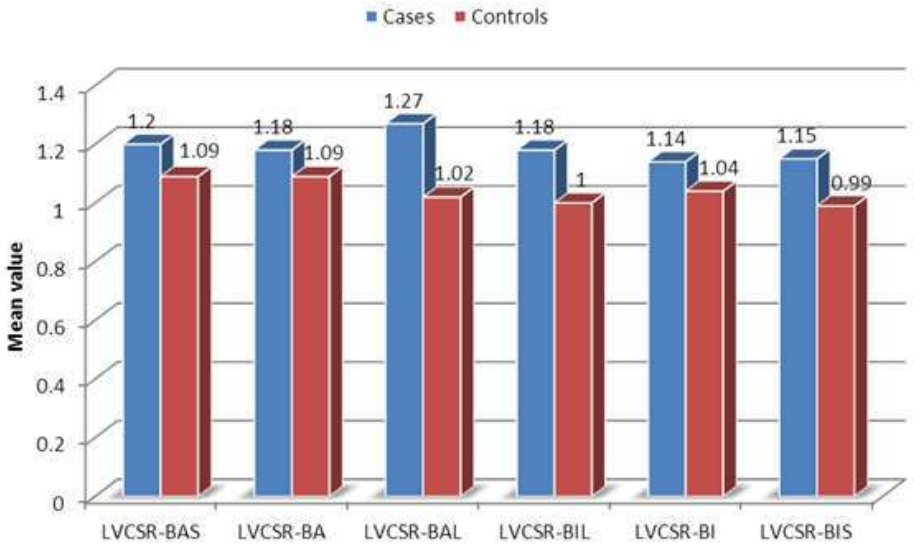

Fig. 6:

Discussion

The abnormal diastolic filling pattern is the most common abnormality of LV function in hypertensive patients.In these patients, LV systolic function is commonly considered normal if the global EF is preserved. However, the EF is a crude measure of global cardiac contractile function and does not take into account the regional and segmental systolic abnormalities [14]. The substantial early subclinical LV systolic dysfunction usually remain undetected by traditional measures of LV systolic function but often may be responsible for symptomatology of hyperten-sive patients.

2D-STE appears more sensitive than both conven-tional echocardiographyand DTI in identifying the reduction of intrinsicmyocardial contractility, evident in hypertensive patients even before LV hypertrophy becomes detectable.It wasobserved from the present study that an impairment of longitudinalLV systolic function is present as detected by a significant reduction of LV systolic strain and systolic SR values inhypertensive patients even without LVH. Moreover, a significant reduction in strain values wasfound in hypertensive patients with LVH in comparison to hypertensive patients without LVH. These findings highlightsthe advantages of 2D-STE over DTI in the detection of earlyimpairment of LV systolic function.

The results of the present study showed that global strain in longitudinal and circumferential axis was significantly different in hypertensive and normoten-sive groups. LVGLS was found to be

significantly (p=0.0001) lower among cases (-17.31±3.82) compared to controls (-24.67±2.49). Similarly LVGCS was also found to be significantly (p=0.0001) lower among cases (-21.82±5.10) compared to controls (-30.53 ±7.44). Among cases with LVH on ECG and on 2D echo compared to cases without LVH,LVGLS was found to be significantly (p=0.003) lower but LVGCS was insignificantly lower.

Our study results shows that earliest impairment of regional myocardial contractile performance involves longitudinal myocardial fibres with subsequent involvement of circumferential and mid myocardial fibres. The present study results are in concordance with the findings from the previous studies like Kouzuet et al. study [15], who found that longitudinal strain was significantly reduced in those with LV hypertrophy compared with healthy control subjects. Additionally, Imbalzano et al. [16] revealed that 2DSTE showed an impairment of systolic longitudinal strain in all hypertensive patients including those without LVH. Again, a study done by Saghiret et al. [17] revealed that hypertensive individuals with LVH had significantly decreased systolic longitudinal strain and strain rate values compared with control subjects. Di Bello et al. [18], also demonstrated that longitudinal two dimensional strain in prehypertensive (-18.9±3.4%) and in hypertensive (-18.0±3.3%) was significantly lower than in normotensive (-23.9±3.0%) (P< 0.002) subjects.

The present study also compared the peak Left ventricular longitudinal strain(LVLS) in different segments. LVLS at – Apl (-18.29±6.53 vs -23.73±5.78, p-0.006), ApS (-20.71±8.34 vs -25.73±5.31, p-0.03) and MIS (-18.47±8.15 vs -23.13±6.06, p-0.04) were found to be significantly (p<0.05) lower among cases compared to controls.While peak Left ventricular circumferential strain (LVCS) in different LV segments were found to be significantly (p<0.05) lower among cases compared to controls.

The results of the present study are compatible with those of Tulika et al. [19] on 72 hypertensive patients with preserved EF which revealedthat the regional strain in longitudinal axis was significantly reduced at Apex and Apico lateral segment of LV in hyperten-sive population compared to normotensive group (-17.99± 5.21 Vs-19.77±4.17; p<0.01 and -14.78 ±5.69 Vs -17.40± 5.23; p<0.01) respectively.

Left ventricular diastolic function assessment by strain rate

The results of the present study indicated that the SR values were insignificantly reduced in both hypertensive groups with and without LVH in early diastole (at peak E). However, there was no signifi-cant reduction in SR except LVLSR at Apex. LVLSR-Apex was found to be significantly (p=0.005) lower among cases (1.24±0.46) compared to controls (1.70 ±0.68).

Goebel et al. [20], also found that, in the patient group with LVH, systolic SR and early diastolic SR quantified in longitudinal and circumferential direction, were lower compared with the group without LVH. Huang et al. [21] also compared 88 patients with essentialhypertension in addition to age-matched andsex-matched 30 normotensive healthy volunteersserving as normal controls. His analysis showedthat in the essential hypertension group early diastoliclongitudinal SR and circumferential SR were significantly lowerthan normal controls.

The major findings of the present study was that impairment of LV deformation is common in patients with isolated hypertension as evidenced by reduced magnitudes of LV longitudinal and circumferential strain despite almost preserved LVEF in the all of the patients. The assessment of LV systolic longitudinal function and circumferential contraction strain analysis reveals new concepts in myocardial function assessment in hypertension that can identify the high risk subgroups in HTN who can derive substantial benefit from anintensive antihypertensive treatment program with aim to ameliorate this early subclinical LV dysfunction along with regression of LV hypertrophy. These abnormal patterns of longitudinal and circumferential systolic contraction and abnormal diastolic LV filling may play animportant role in the development of acute and chronic heart failure in patients with chronic hypertension and thus can be targeted by appropriate pharmacotherapy.

The limitations of the present study include small sample size, so the trends of reduced regional systolic strain observed in longitudinal axis might be truly reduced but due to the limited power of the study the results can not be generalized to a broader segment of population with HTN. We did not check the reproducibility of obtaining estimates of regional systolic strain by different operators; thus, there could have been measurement bias, especially in the small sample size study population. There was no blinding of the operator regarding hypertensive patients and control subjectsand this study was a single centre non randomized study.

Conclusion

A substantial impairment of LV systolic and diastolic functionsis detected by 2D STE in chronic

hypertensive patients with apparently preserved LV systolic function as compared to normal population. It also provides insights into pattern of early LV dysfunction with important pathophysiological considerations. The extent of impairment was found to be more especially if associated withLVH.

Disclosure: None

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