University Heart Journal
Vol. 17, No. 1, January 2021

Assessment of left Ventricular Longitudinal Function in Different Hypertensive Left Ventricular Geometry

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Abstract

Background: *Hypertension remains as the major risk factor for cardiovascular diseases. Hypertensive left ventricular hypertrophy was shown to be associated with increased morbidity and mortality. Left ventricular radial function (Ejection fraction) tends to remain normal in hypertensive patients, particular attention should be given to longitudinal function along with diastolic function. Left ventricular longitudinal function may vary across different hypertensive LV geometry with different prognosis.*

Results: *Of the total 214 study subjects, 109 (50.9%) were Cases and 105 (49.1%) were Controls. The mean ages of cases and controls were 52.66 (± 10.96) and 50.21 (± 10.91) years respectively. Left ventricular ejection function (LVEF) was almost identical in both groups [mean LVEF in case 68.7 % (± 6.9) Vs control 68.7(± 5.4), (p 0.947)]. Among the cases 43% had concentric hypertrophy (CH), 20% had eccentric hypertrophy (EH), 20% had concentric remodeling (CR), while normal geometry constituted the least 16.5%. Mean systolic mitral annular velocity (Vs) and mean early diastolic velocity (Ve) assesed by pulse wave tissue doppler imaging were observed to be significantly decreased in cases compared to their control counterpart (11.46 ± 1.26 vs. 15.41 ± 1.00 cm/sec, p < 0.001 and 13.80 ± 2.37 vs. 16.76 ± 2.67 cm/sec, p < 0.001. There was significant reduction of Vs in concentric hypertrophy and eccentric hypertrophy (11.31 ± 1.41 and 12.27 ± 2.14). (p <0.001 and <0.005). Among cases 55 (50.5%) and among controls 17 (16%) had diastolic dysfunction. Mean systolic mitral annular velocity (Vs) in patients with diastolic dysfunction (12.42 ± 1.90 cm/sec) was significantly lower than that in patients without diastolic dysfunction (13.86 ± 2.30 cm/sec) (p < 0.001).*

Conclusion: *Radial function (LVEF) remains normal in patients with systemic hypertension as compared to controls. LVH is common among hypertensive and concentric hypertrophy is the commonest geometry. LV longitudinal systolic function as assessed by systolic mitral annular velocity (Vs) by DTI was significantly reduced in hypertensives and CH is the most severely affected with EH at intermediate risk. Diastolic dysfunction is also common but almost alaways accompanied by impairment of LV longitudinal systolic function.*

University Heart Journal 2021; 17(1): 31-37

Introduction:

Hypertension is one of the most common worldwide condition afflicting humans and it is the most important modifiable risk factor for coronary heart disease, stroke, congestive heart failure, end-stage renal disease, and peripheral vascular disease.[1] Its high mortality is somewhat related to left ventricular hypertrophy (LVH).[1] Subjects with LVH consistently have 2 to 4 or more fold higher rates of cardiovascular complications.[2] In hypertension, LVH is initially a useful compensatory process that represents an adaptation to increased ventricular wall stress, but it is also the first step toward the development of overt clinical disease.[1] The Framingham study has shown that the prevalence of echocardiographic LVH is 15-20 % in mild hypertensive patients and further increases in patients with more severe hypertension.[3] Several diagnostic criteria for LVH diagnosis can be used. LVH can be defined as a value of LV mass greater than the mean + 2 standard deviation of the value obtained in a "control" general population (i.e. > 134 g/m2 or > 130 g/m2 in male patients and > 110 g/m2 or > 100 g/m2 in female patients, as observed in two different population studies.[4,5] Geometric adaptation of the left ventricle to increased cardiac load may be different among patients. Concentric hypertrophy is characterized by increased mass and increased relative wall thickness, whereas eccentric hypertrophy is characterized by increased mass and relative wall thickness < 0.45; concentric remodelling

Received: 26 July 2020 **Accepted:** 15 November 2020

occurs when there is increased thickness with respect to radius, in the presence of normal LV mass. A large number of studies have reported on the relationship between LVH at baseline examination and the risk of subsequent morbid or mortal events in clinical or epidemiological populations. Concentric hypertrophy appears to carry the highest risk and eccentric hypertrophy an intermediate risk.[3,6,7]

It has been recognised that the fall in cavity volume with left ventricular systole involves longitudinal as well as circumferential shortening, although the latter plays the dominant role.[8] This asymmetry is reflected in myocardial structure—most of the left ventricular fibres are arranged circumferentially, particularly in the mid-wall and the base of the ventricle, however, with the progressive change in fibre angle across the wall, longitudinally directed fibres are found in the subendocardial and subepicardial free wall as well as in the papillary muscles.[9] The long axis passes from the fibrous apex to the fibrous atrioventricular ring. Unlike with the minor axis, the extent of long axis shortening is not modified by changes in the minor axis. Without this longitudinal component, normal sarcomere shortening would lead to a shortening fraction of 12% and an ejection fraction of less than 30%.[10]

Although longitudinally directed fibres comprise only a small portion of the overall myocardial mass, they affect cardiac function in ways that differ significantly from the more abundant circumferential ones. The contribution of long axis function to normal cardiac physiology deserves more attention than it has received in the literature. This study is mainly focused on that.

Materials and Methods:

This was a hospital based cross-sectional study, from January 2005 to December 2005, conducted in the Department of Cardiology, University Cardiac Centre, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka.

Study Population: 109 consecutive patients with essential hypertension who were referred for echocardiography from Cardiac Out Patient Department, Hypertension clinic and other Departments of BSMMU. The control were 105 age and sex matched healthy subjects or patients without cardiovascular disease.

Inclusion criterias:

Patients with essential hypertension (Hypertension is defined as the blood pressure (BP) equal to or greater than 140/90 mm Hg on at least two occasions at two weeks interval or subjects on antihypertensive medication. The duration of hypertension was ascertained to the length of time since diagnosis of hypertension.

Exclusion criterias:

Valvular heart disease, Atrial fibrillation, Chronic renal disease, Diabetes mellitus, Pregnancy, Hypertrophic obstructive cardiomyopathy (HOCM) and subjects with Poor echo window.

Methods:

- All the patients were explained in detail about the study. Informed consent was taken from all patients who were included in the study. On the day of the examination, demographic data and measurements of blood pressure were taken from the patients and control group. Every patient was clinically evaluated by detail history and physical examination performed during entry into the study protocol and recorded in pre-designed proforma. Eelectrocardiogram (ECG), Chest X-ray posteroanterior view, fasting blood sugar, Serum creatinine, were done in all patients. In ECG, the Sokolow-Lyon voltage criteria for LV hypertrophy [Sv1 + (Rv5 or Rv6) > 3.5 mV (35mm)] was used.
- Echocardiographic Evaluation: Echocardiographic examinations were done with the patients in the left semilateral position with Siemens Acuson CV70 Ultrasound Imaging System using 2.5 MHz phased-array transducer with ECG gating. Measurements of the left ventricular internal diameter in end diastole (LVIDd), LV diameter in end systole (LVIDs), interventricular septum (IVS) and posterior wall thickness (PWT) were made from M-mode readings according to the recommendations of the American Society of Echocardiography (ASE). Measurements were averaged over three readings. The ventricular mass was calculated with the modified Devereux formula[5]: $0.80\,[1.04\,(DIVS + DLVPW)^3 - (LVDD)^3] + 0.6$, and the ventricular mass index (LVMI) obtained through correction of mass by the body surface area. The criteria used for the definition of hypertrophy are 134g/m2 for males and 110g/m2 for females[11]. Relative wall thickness (RWT) was calculated using the formula 2PWT/LVIDd. Normal RWT was taken as < 0.45.

According to the LVMI and RWT the patients were classified into 4 ventricular geometry groups, as follows:

1) Normal geometry (NG) – normal LVMI and normal RWT.
2) Concentric remodeling (CR) – normal LVMI and RWT ≥0.45.

3) Concentric hypertrophy (CH) – LVMI > pre-established limits and RWT ≥0.45.

4) Eccentric hypertrophy (EH) - LVMI > pre-established limits and RWT < 0.45.

Doppler Studies: Mitral Valve inflow (MVI) velocities were obtained with sample volume (2.4 mm gate) at the tip of mitral valve leaflets in apical four-chamber (A4C) view. Measurements included peak early left ventricular filling velocity (E), peak atrial filling velocity (A), their ratio (E/A), A duration and deceleration time of early diastolic mitral inflow. Isovolumic relaxation time (IVRT) was obtained with sample volume in between LV outflow tract and tips of mitral valve leaflets. Measurements up to three cycles at end expiration were averaged. Pulmonary vein (PV) flow recordings were obtained from the A4C view directed at the right upper pulmonary vein (RUPV). Doppler Tissue Imaging: Pulsed wave DTI was performed by placing a sample volume (2.4 mm gate) along the mitral annulus. Mitral annular velocities were measured lateral and septal from apical 4-chamber view, anterior and inferior from 2-chamber view. Systolic (Vs), early (Ve) and late (Va) diastolic annular velocities were averaged from the four sites.

Data Analysis:

Data were analysed using SPSS (Statistical Package for Social Sciences). The test statistics used to analyze the data were descriptive statistics, Chi-squared (c^2) Test, Fisher's Exact Probability Test, Student's t-test and Pearson's correlation. The data measured on continuous scale were presented as mean and SD/SEM and compared between groups using Student's t-test, while categorical data were expressed as frequency and corresponding percentages and compared between groups with the help of Chi-squared or Fisher's Exact Probability Test. The relationship between two continuous variables was evaluated with help of Pearson's correlation. The level of significance was 0.05. P-value <0.05 was considered significant. The summarized information was then presented in the form of tables and charts.

Results:

Of the total 214 study subjects, 109 (50.9%) were Cases and 105(49.1%) were Controls. The mean ages of cases and controls were 52.66 ± 10.96 and 50.21 ± 10.91 years respectively. The age categories were observed to be almost identically distributed between the groups (p > 0.05).

Table-I

Age distribution of the subjects between groups: (n=214)

Age (yrs)	Group		p-value#
	Case (n = 109)	Control(n = 105)	
< 30	1(0.9)*	1(1.0)	
30 – 40	8(7.3)	18(17.1)	
40 – 50	31(28.4)	27(25.7)	0.191
50 – 60	34(31.2)	35(33.3)	
≥60	35(32.1)	24(22.9)	
Mean SD	52.66 ± 10.96	50.21 ± 10.91	

Sex: Males were a bit higher in both the groups (56.9% in cases and 55.2% in controls). The groups were almost homogeneous in term of sex (p > 0.05).

Table-II

Sex distribution of the subjects between groups: (n=214)

Sex	Group		p-value#
	Case (n = 109)	Control(n = 105)	
Male	62(56.9)*	58(55.2)	0.809
Female	47(43.1)	47(44.8)	

Pertinent baseline variables:

Pertinent baseline variables like pulse rate, LA-diameters and LVEF (%) between groups were compared between groups. Out of four variables, mean LA-diameter in Parasternal long axis (PLAX) 2-D guided M-mode and LA-diameter in 2-D apical 4 chamber (A4C) were revealed to be significantly greater in cases compared to the control group (p < 0.001). Pulse rate and LVEF were almost identical between groups.

Table-III

Distribution of pertinent baseline variables between groups: (n=214)

Baseline variables#	Group		p-value*
	Case (n=109)	Control (n=105)	
Pulse (per-minute)	72 ± 5	72 ± 6	0.871
LA-diameter (PLAX)	35.98 ± 5.12	32.10 ± 5.02	<0.001
LA-diameter (apical 4-chamber)	47.67 ± 5.17	42.71 ± 7.30	<0.001
LVEF (%)	68.77 ± 6.93	68.71 ± 5.37	0.947

Duration of hypertension: Over 60% of the cases had been suffering from HTN for < 5 years at the time of entry into

the study. Some 23.9% had been suffering for 5 – 10 years and the rest 14.7% for more than 10 years. 60% of the cases were taking anti-hypertensive medications regularly.

LV geometry in hypertensive patients and controls: 43.1% of the cases had concentric hypertrophy. Eccentric hypertrophy and concentric remodeling each consisted of 22.2%, while normal geometry constituted the least 16.5%. Majority of the controls had normal geometry (81%) followed by eccentric hypertrophy 8.6%, concentric remodeling 7.6% and concentric hypertrophy 2.8%.

Table-IV

Distribution of LV geometry in case & control: (n=214)

	Case	Controls
Hypertensive LV geometry	No (%)	No (%)
Concentric hypertrophy (CH)	47 (43.1)	03 (2.8)
Eccentric hypertrophy (EH)	22 (20.1)	09 (8.6)
Concentric remodeling (CR)	22 (20.1)	08 (7.6)
Normal geometry (NG)	18 (16.5)	85 (81)

Association of sex and LVH: Females [35 out of 47 (74.5%)] tend to develop LVH significantly more than their male [34 out of 62 (54.8%)] counterpart. ($p < 0.05$).

Association of duration of hypertension and LVH: Duration of hypertension <10 yers [50 out of 84 (59.5%)] & >10 years [19 out of 35 (76%)] was found to exert some influence on LVH but did not reach statistical significance ($p > 0.05$).

Mitral annular velocities by DTI:

Mean systolic mitral annular velocity (Vs) and mean early diastolic velocity (Ve) were observed to be significantly decreased in cases compared to their control counterpart (11.46 ± 1.26 vs. 15.41 ± 1.00 cm/sec, $p < 0.001$ and 13.80 ± 2.37 vs. 16.76 ± 2.67 cm/sec, $p < 0.001$ respectively). The mean late diastolic velocity (Va), on the other hand, was significantly higher in cases compared to control group (17.52 ± 2.88 vs. 15.86 ± 2.7, $p < 0.001$).

Table-V

Comparison of mitral annular velocities by DTI between groups: (n=214)

Mitral annular velocities# (cm/sec)	Group		p-value*
	Case (n = 109)	Control (n = 105)	
Vs	11.46 ± 1.26	15.41 ± 1.00	<0.001
Ve	13.80 ± 2.37	16.76 ± 2.67	<0.001
Va	17.52 ± 2.88	15.86 ± 2.7	<0.001

LV longitudinal function (Vs) in different LV geometry: There was significant reduction of Vs in concentric hypertrophy and eccentric hypertrophy (11.31 ± 1.41 and 12.27 ± 2.14). ($p < 0.001$ and < 0.005).

Table-VI

Influence of hypertensive LV geometry on Vs: (n=214)

Hypertensive LV geometry	Vs (Mean ± SD)	p-value*
Concentric hypertrophy (CH)		
Present	11.31 ± 1.41	<0.001
Absent	14.04 ± 2.11	
Eccentric hypertrophy (EH)		
Present	12.27 ± 2.14	0.003
Absent	13.59 ± 2.25	
Concentric remodeling (CR)		
Present	12.88 ± 2.04	0.182
Absent	13.48 ± 2.31	
Normal geometry (NG)		
Present	14.90 ± 1.57	<0.001
Absent	12.00 ± 1.92	

Ve in different LV geometry: There was significant reduction of Ve in Concentric hypertrophy (Mean ± SD) (12.86 ± 2.05, $p < 0.001$) and concentric remodeling (14.27 ± 1.82, $p < 0.05$) but no significant change in EH (15.17 ± 2.25, $p > 0.05$).

Mitral valve inflow (MVI) parameters: A-wave duration, decelaration time and IVRT were found to be significantly increased in cases compared to those in controls (165.93 ± 3.14 vs. 154.70 ± 1.99 ms, $p < 0.005$, 155.01 ± 3.84 vs. 134.59 ± 2.01 ms, $p < 0.001$ and 94.70 ± 1.62 vs. 82.81 ± 0.76 ms, $p < 0.001$ respectively).

Diastolic dysfunction in groups:

Abnormal relaxation and pseudonormal diastolic dysfunctions were significantly higher in cases compared to their control counterpart ($p < 0.05$ and $p < 0.001$ respectively). Only 1 case had restrictive filling.

Table VII

Distribution of diastolic dysfunction between groups: (n=214)

Diastolic dysfunction	Group		p-value
	Case (n = 109)	Control (n = 105)	
Normal*	54 (49.5)**	88 (83.8)	<0.001
Abnormal relaxation*	33 (30.3)	15 (14.3)	0.005
Pseudonormal*	20 (18.3)	2 (1.9)	<0.001
Restrictive filling#	1 (0.9)	00	0.509

Systolic mitral annular velocity (Vs) in diastolic dysfunction: Mean systolic mitral annular velocity (Vs) in patients with diastolic dysfunction (12.42 ± 1.90 cm/sec) was significantly lower than that in patients without diastolic dysfunction (13.86 ± 2.30 cm/sec) ($p < 0.001$).

Table-VIII

Comparison of Vs between subjects with and without diastolic dysfunction: (n=214)

Mitral annular velocities# (cm/sec)	Diastolic dysfunction		p-value*
	Present (n = 69)	Absent (n = 145)	
Vs	12.42 ± 1.90	13.86 ± 2.30	<0.001

Discussions:

This study was carried out to see the consequences of hypertension on LV geometry and function, especially the longitudinal systolic function. The study was done at the Department of Cardiology, Bangabandhu Sheikh Mujib Medical University from January 2005 to December 2005. The mean age (± standard deviation) of the cases was 52.66 (± 10.96) and control was 50.21 (±10.51). Highest number of cases (32%) were in > 60 years group followed by 50-60 years group (31%) and only 1 case (0.9%) under the age of 30 years signifying hypertension is the disease of aging. The age categories observed to be almost identically distributed between cases and controls. Among the cases 62 (56.9%) were male and in controls male were 58 (55.2%). The groups were almost homogenous in terms of sex.

As transmitral flow velocity is load dependent so heart rate is an important parameter to be matched in the groups. The pulse rate was almost identical in cases and controls (72 ±5 Vs 72±6). The duration of hypertension (time since first detection) varied among cases but most of them were less than 5 years [67 cases (61.5%)]. 65 cases (59.6%) were taking antihypertensive medications regularly. So over 40% of cases failed to adhere to drug therapy, which is similar to other reports. Conlin et al. (2001) reported that < 50% patients continue taking prescribed antihypertensive drug therapy for 4 years.[12] Absence of symptoms is probably the most important cause of discontinuation of drug.

In ECG, the Sokolow-Lyon voltage criteria for LV hypertrophy [Sv1 + (Rv5 or Rv6) > 3.5 mV (35mm) detected LVH in 18.3% of cases (63.2% by echocardiography) and in 2.9% of controls (11.4% by echocardiography). The finding reaffirms the relative insensitivity of this criterion in detecting LVH. Devereux et al (1987) showed ECG had sensitivities of 10-38% (moderate LVH) and 30-57% (severe LVH).[11] 2.9 % of controls had LVH by this criteria match with the the Framingham Study that shown that the prevalence of LVH, according to echocardiography criteria is quite low in a general population sample (about 3%).[3]

Left atrial (LA) diameters measured from both the parasternal 2-D guided M-mode and apical 4-chamber 2-D views showed significant increase in cases than in controls ($p < 0.001$). The LA size serve as a barometer of chronic loading condition such that patient with increased LA size likely to have significant lasting elevation of filling pressure.[13]

The LV ejection fraction (LVEF) were similar in both the groups with mean (±SD) LVEF among cases is 68.77% (±6.93) and among control is 68.71% (±5.36) [p = 0.947]. So radial function remained normal in patients with systemic hypertension as compared to controls but the reduction in systolic mitral annular amplitude and velocity in the long axis was not compensated for by circumferential shortening. This finding is similar to that of Yip et al. (2002).[14]

The prevalence of LV geometric patterns detected in cases are concentric hypertrophy (CH) in 47 patients (43.1%), Eccentric hypertrophy (EH) in 22 patients (20.1%), Concentric remodeling (CR) in 22 patients (20.1%) and normal geometry (NG) in 18 Patients (16.5%). So prevalence of LV hypertrophy (LVH) (CH+EH) was 63.2% and LV structural Change (CH+EH+CR) was 83.4% among the cases. The use of the same criteria applied in our study (134g/m2 and 110g/m2) in 510 participants of the HOT Study provided a prevalence of hypertrophy of 62%.[6] Using the same criteria, another study in Brazil showed 47.4% of the patients had NG, 25.4% CR, and 27.2% hypertrophy, adding to a total of 52.6% of patients with cardiac structural changes.[16] In the VITAE Study, the echocardiographic assessment of a large population of essential hypertension obtained from reference centers in Spain provided a prevalence of ventricular hypertrophy and of concentric remodeling that ranged from 59.2% to 72.2% and from 6.5% to 11.4%, respectively, depending on the criterion used.[16] Recent St. Petersburg study showed prevalence of LVH ranged from 52.2 to 72.2% by the use of different threshold for LVH definition.[17] So our findings are more or less comparable to others.

The prevalence of LV geometric patterns detected in controls were CH in 3 subjects (2.9%), EH in 9 subjects (8.6%), CR in 8 subjects (7.6%) and NG in 85 subjects (81%). So prevalence of LVH (CH+EH) was 11.4% and LV structural Change (CH+EH+CR) was 20% among the controls. Echocardiographic technique, demonstrated that

the prevalence of LVH in the Framingham population was 5%, in subjects younger then 30 years and 50 % in those older then 70 years.[3]

We also search for the association of age, sex and duration of hypertension with LVH. We found that cases having age 60 years or above were significantly prone to develop LVH compared to those with below 60 years of age ($p < 0.05$). St. Petersburg study also showed that LVH increases proportionately with age. In our study females tend to have more LVH than their male counterpart, this finding differs from St. Petersburg study, which showed male are more prone to develop LVH.[17] In our study, 76% of the cases having hypertension for >10 years developed LVH compared to 60% in cases having hypertension of <10 years duration. But the difference is not statistically significant ($p >0.05$) probably due to fewer cases with > 10 years of hypertension. Verdecchia and associates did not find any association between duration of HT and LVH[18]. Most other studies showed influence of duration of hypertension on LVH.[19]

The mean systolic mitral annular velocity (Vs) (cm/second) by DTI was significantly reduced in patients with hypertension compared to the controls (11.46±1.25 Vs 15.40±0.99) [$p <0.001$]. Reduction of Vs was most marked in cases with CH (11.31 ± 1.41) followed by EH (12.27 ± 2.13), CR (12.88 ± 2.03) and NG (14.90 ± 1.57). So LV longitudinal systolic function is most affected in patients with concentric hypertrophy. This finding is consistent with the findings of a study in Bulgaria.[20] In 132 patients (72 male, Av age 57.2±11.3 years) with essential hypertension they also found most marked reduction of Vs (cm/sec) in CH (6.9 ±2.2) followed by in EH (8.1 ± 2.9), CR (8.9 3.1) and NG (9.8 ± 2.4). So even the radial function (LVEF) is normal in patients with hypertension compared to controls the longitudinal systolic function is significantly reduced.

MVI parameters revealed IVRT and DT were significantly prolonged ($p <0.001$) in cases than in controls. Similarly PV flow revealed significant increase of atrial reversal (AR) velocity into the PV during atrial contraction and also significant lengthening of AR flow duration ($p <0.001$). All of these parameters signify worsening diastolic properties.[13]

The assessment of diastolic function using Doppler interrogation of MVI, PV flow and DTI revealed abnormal relaxation (grade I diastolic dysfunction) among the cases and control were 33 (30.3%) and 15 (14.3%) respectively; pseudonrmal (grade II diastolic dysfunction) among the cases and controls were 20 (18.3%) and 2 (1.9%) respectively; restrictive filling pattern (grade III diastolic dysfunction) among cases and controls were 1 (0.9%) and 0 respectively. So different grades of diastolic dysfunction were present in 55 cases (50.5%) and in 17 controls (16.19%). Yip G et al (2002) showed prevalence of diastolic dysfunction of 52% in hypertensive cases.[14]

As it is also reasonable to suggest that peak early diastolic lengthening rate determined by Doppler tissue imaging (DTI) is an index of early diastolic function.[21] Mean early diastolic mitral annular velocity (Ve or E') (cm/sec) were compared in cases and controls and in various LV geometry. Mean (± SD) Ve in cases were 13.80 (± 2.3) and in controls were 16.76 (2.67) ($p <0.001$). Most marked reduction of mean Ve noticed in cases with CH (12.85 ± 2.05) followed by CR (14.26± 1.81), EH (15.17±2.25) and NG (16.732.84). This finding is consistent with the findings of Marchev, Kuneva & Denchev (2004), who found Ve in CH 7.8 ± 1.7, in EH 9.1±2.3, in CR 9.0±1.9 and in NG 11.2±2.5.[20]

The mean systolic mitral annular velocity (Vs) by DTI in subjects with diastolic dysfunction was significantly reduced compared to those without diastolic dysfunction (12.42 ± 1.90 Vs 13.86± 2.30, $p<0.001$). This is comparable to findings of Yip G et al (2002), who found Vs were lower in patients with diastolic dysfunction (mean±SEM), (4.8±0.2 cm/s) than in the age matched normal controls (6.1±0.14 cm/s) ($p<0.001$). They concluded that in patients with diastolic heart failure and evidence of left ventricular hypertrophy, there is systolic left ventricular impairment as measured by DTI of the longitudinal axis.[14] Thus subtle abnormalities of systolic function are present in patients with heart failure and a normal left ventricular ejection fraction, and there appears to be a continuum of systolic function between those with truly normal, mildly impaired (labelled diastolic heart failure), and obviously abnormal left ventricular systolic function. Isolated diastolic dysfunction is uncommon.

As LVH was shown to be associated with increased morbidity and mortality and regression of LVH was shown to decrease risk by various studies including Framingham study consideration should be given to prevent LVH and to treat those with LVH aggressively especially CH. As the radial function tends to remain normal in hypertensives particular attention should be given to longitudinal function along with diastolic function. Study should be carried out to see the efficacy of antihypertensive drugs in improving longitudinal function.

Conclusions:

Radial function (LVEF) remains normal in patients with systemic hypertension as compared to controls. Left atrial (LA) diameters showed significant increase in hypertensives than in controls. LVH is common among hypertensive and concentric hypertrophy is the commonest geometry. Increasing age, female sex has shown to be associated with LVH but duration of hypertension failed to show significant association. LV longitudinal systolic function as assessed by DTI was significantly reduced in hypertensives and CH is the most severely affected with EH at intermediate risk. Diastolic dysfunction is also common but isolated diastolic dysfunction without impairment of longitudinal systolic function is rare. To reduce the morbidity and mortality of hypertension, prevention and early detection of LVH taking particular attention to LV longitudinal as well as diastolic function is important.

Study limitations:

The study included a small number of patients so firm conclusions could not be made. Though age, sex and heart rate were matched possible associated risk factors were not matched between groups. Majority of the cases were on antihypertesive medications and their influences on LV geometry and function could not be assessed.

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