

Left ventricular diastolic dysfunction in congestive heart failure due to systemic hypertension: A study in national institute of cardiovascular diseases and hospital, Dhaka, Bangladesh

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Abstract

Background: This was a prospective observational study was carried out in National Institute of Cardiovascular Diseases and Hospital (NICVD), Dhaka, Bangladesh during the period from January 2017 to December 2018. Our aim was to evaluate left ventricular diastolic dysfunction in congestive heart failure resulting from systemic hypertension.

Methods: 50 patients with systemic hypertension who had recently experienced CHF with normal ejection fraction ($\geq 50\%$) and no clinical history of ischemic cardiomyopathy were studied. The patients were divided into two groups according to the degree of echocardiographic hypertrophy: group-I (26 patients) with a ventricular mass/volume ratio >1.8 and group-II (24 patients) with a ratio <1.8 .

Results: Group I patients had a higher ejection fraction (67.62 ± 3.14 vs 55.33 ± 4.13 , $P < 0.001$), smaller ventricular diameter (28.88 ± 2.46 vs 34.38 ± 4.37 , $P < 0.001$), higher LV mass (154.42 ± 6.80 vs 123.38 ± 5.58 , $P < 0.001$), lower ETT positivity (15% vs 75% , $P < 0.001$). Clinically, group I had more frequent audible fourth heart sound (57% vs 20% , $P < 0.001$), low incidence of audible third heart sound (15% vs 70% , $P < 0.001$), ECG evident LVH mass (96% vs 16% , $P < 0.001$), cardiomegaly (23% vs 70% , $P < 0.001$).

Conclusion: There were no significant differences between groups for NYHA class, age, sex, heart rates and systolic blood pressure but significantly associated with Diastolic BP, ECG (LVH) and Cardiomegaly.

Keywords: diastolic dysfunction, CHF, ECG evident LVH mass, higher LV mass

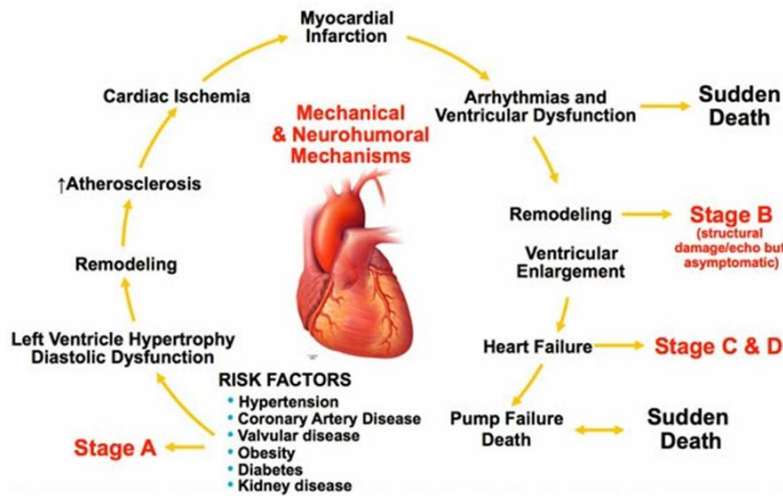
1. Introduction

Cardiac cycle consists of both systole and diastole, and the classical syndrome of congestive heart failure, may be either due to systolic dysfunction or diastolic dysfunction or both. Congestive heart failure (CHF) with normal left ventricular systolic function and an abnormal diastolic function is a common clinical entity. Typically, signs and symptoms are indistinguishable from those of heart failure related to systolic dysfunction (Paul *et al.* 1996) [1]. Coronary artery disease, systemic hypertension and aging are all associated with diastolic congestive heart failure (DCHF) (Spencer and Lang 1997). In cases of CHF in elderly patients, the prevalence of left ventricular failure with normal ejection fraction is as high as 30-40 percent (Echeverria *et al.* 1983; Dougherty *et al.* 1984; Sourer *et al.* 1985; Kessler 1988) [17] with arterial hypertension being the most frequent etiologic cause (Echeverria *et al.* 1983; Dougherty *et al.* 1984) [15]. Several factors predispose to increased diastolic stiffness in a left ventricle with normal systolic performance. These include fibrosis, pericardial constriction and myocardial restriction (Bonow *et al.* 1992) [22]. Diastolic dysfunction is caused by at least two distinct, yet interrelated. Properties of the heart: the passive elastic properties and active relaxation of the myocardium (Paul *et al.* 1996) [1]. With the loss of elastic properties of the heart, there is reduction in compliance and with impairment of relaxation; there is an

increase in myocardial wall tension during diastole, both of which cause increased pulmonary venous pressure. The most common condition in which these factors conspire to elevate filling pressure is hypertension. Loss of elastic properties in hypertension leading to abnormal diastolic function results from the effect of hypertrophy and increase in collagen network of the myocardium. Weber *et al.* (1994) described the dynamic collagen turnover in the myocardium. Collagen. With a high tensile strength, is a major determinant of chamber stiffness. Factors that appear to contribute to the appearance of myocardial fibrosis through their effects as collagen turnover include hormones of the renin-angiotensin-aldosterone system, endothelial and bradykinin (Weber and Brilla 1991). Besides this, impaired active relaxation also contributes to the pathophysiology of ventricular diastolic dysfunction, resulting from systemic hypertension, coronary artery disease and aging. Congestive heart failure caused by abnormal diastolic function may be far more common than previously recognized. The diastolic disorder must be distinguished from systolic abnormalities because treatment is significantly different. A history of myocardial infarction, cardiomegaly, Q-wave on the electrocardiogram and an S₄ gallop favour systolic dysfunction. However, an S₄ gallop, left ventricular hypertrophy and normal cardiac size favour diastolic function. However, there is overlap since the disease may

produce either or both' Clinical assessment without cardiac imaging is, therefore, incomplete and inaccurate in many cases (Goldsmith and Dich 1993). To accurately distinguish between systolic and diastolic dysfunction, left ventricular

function must be assessed. This can be accomplished with echocardiography, radionuclide angiography or radiographic ventriculography.



Source: Google

Fig 1

Echocardiography seems nice swashbuckling attributable to its immovableness and lack of radiation likewise as its ability to guage controller operate, serosa standing, wall motion and chamber hypertrophy. Ideally the diagnosing ought to be confirmed by documenting elevation of left cavum blood pressure, however usually this can be often impractical. Therefore, noninvasive procedures like radionuclide and sonogram studies to be wide used. Whereas radionuclide roentgenography could be a powerful tool for excluding left cavum pulse dysfunction, its use for designation pulse disfunction is proscribed (Spencer and Lang 1997) Christian Johann Doppler diagnostic technique, a noninvasive and straightforward procedure, provides insight into left cavum pulse disfunction (Appleton *et al.* 188; Appleton 1993; Pai and Beech 1996). The foremost normally used Christian Johann Doppler parameters of pulse disfunction square measure derived from left cavum influx and pulmonic blood vessel influx. A PW Christian Johann Doppler sample volume is placed at the atrioventricular valve leaflet tips to guage left cavum pulse operate that reveals prolonged isovolumic time constant, prolonged retardation time, attenuated E-wave to A- wave magnitude relation on mitral influx. A PW Christian Johann Doppler study at vein reveals multiplied S-wave to D-wave magnitude relation and vein 'a'-wave reversal period prolonged and rate multiplied (Klein *et al.* 1994) [9]. as a result of the trail physical mechanism in pulse cardiopathy includes a high pulse pressure-volume relationship, the medical aid has been directed at reducing filling pressure by decreasing end-diastolic volume, reducing rate and treating cardiovascular disease with medicine that conjointly cause regression of left cavum hypertrophy (Paul *et al.* 1996) [1]. The Yankee school of Cardiology/American Heart Association (ACA/AHA) task force divides pharmacological treatment into 3 classifications for the management of pulse cardiopathy. Category I visit medicine that square measure perpetually indicated, like diuretics and nitrates and medicines suppressing auriculoventricular conductivity. Category II agents area unit acceptable;

however, their effectiveness is unsure. These embrace medication B-blocker, vasoconstrictive changing accelerator (ACE) matter. Category III medicine aren't indicated and embrace medicine with positive isotropous effects. Though prognosis is best with beat disfunction than with pulsation disfunction, morbidity and mortality still be high in older patients. The rumored annual rate for patients with failure and preserved left cavity pulsation failure varies from three to twenty five p.c (Vasan *et al.* 1995). The syndrome of beat failure is common however neglected event within the analysis of hypertensive patients. The signs and Symptoms is also like those in patients with pulsation failure. The prognosis is also completely different because of heterogeneousness within the parent population. There's no uniformity in the way to diagnose and the way to tear patients with failure with beat disfunction. Therefore, the current study was undertaken to judge the left cavity beat disfunction in symptom failure ensuing from general cardiovascular disease in our population.

2. Objectives

General Objective

1. To assess the prevalence of diastolic dysfunction in congestive heart failure resulting from systemic hypertension.

Specific objectives

2. Assessment of diastolic dysfunction in congestive heart failure due to hypertension by deferent echocardiography methods.
3. See the correlation between hypertrophy and ischemia in diastolic dysfunction.

3. Materials and methods

The present prospective observational study was Congestive heart failure resulting from systemic hypertension during the period from January 2017 to December 2018. 50 randomly selected hypertensive patients of congestive heart failure (CHF) with diastolic dysfunction were taken from indoor

departments of the above centers; Informed consents were taken from the patients. Detailed history and physical examination findings were recorded in study report sheet (Appendix-I). ECG, chest X-ray two-dimensional and M-mode echocardiography reports were recorded on the same sheet. Doppler echocardiography was done by a cardiologist and evaluation involved pulse-wave Doppler sampling of mitral inflow (tips of leaflets) and pulmonary vein inflow (right upper vein 1-2 cm deep). Flow patterns across the mitral inflow are E/A ratio, deceleration time (m/s), isovolumic relaxation time (m/s) and flow patterns across pulmonary inflow S/D ratio, atrial reversal (AR) velocity, Ad/ARd ratio (Fig. 6 and 7). Eligible patients underwent stress testing and coronary angiography. Initially 320 cases were selected for the study with CHF and 210 were eliminated on the basis of ejection fraction (EF) <0.5. Of the 110 remaining cases, 32 were excluded because of rheumatic heart disease (RHD), hypertrophic cardiomyopathy (HCM) and dilated cardiomyopathy (DCM). The rest 78 were hypertensive, of whom 28 were rejected because of poor echocardiogram, or because they were incompatible of stress testing due to unfitness (Fig. 9). Data have been expressed in frequency, percentage and mean ±SD as applicable. Comparison between groups was done by Chi-square test, Student's t test and Fisher exact test, as applicable. Data were analyzed by computer-based statistical software. P value <0.05 was taken as significant.

Inclusion criteria

- a) Patients with history of hypertension for >35 years
- b) Patients with dyspnea of cardiac origin (NYHA class II, III and IV)
- c) Pulmonary edema verified by CXR-P/A view
- d) Echocardiographic ally determined ejection fraction ^50%
- d) Sinus rhythm.

Exclusion criteria

- a) Secondary hypertension
- b) History of angina or myocardial infarction
- c) History of diabetes mellitus, chronic renal failure and hypertrophic
- d) History of rheumatic fever and rheumatic heart disease, congested heart disease and any systemic diseases that causes left ventricular hypertrophy
- e) Any endocardial, myocardial or pericardial disease
- f) Poor echo-windows.

4. Results

A total 320 patients were screened with congestive heart failure, among them 210 were rejected because of ejection fraction <50 i.e. systolic dysfunction (65%), 32 were excluded due to rheumatic heart disease, ischemic heart disease, hypertrophic and dilated cardiomyopathy and 28 patients were eliminated because of poor echo-window and physical unfitness and thus 50 patients with diastolic dysfunction (16%) with hypertensive were included in the study [Figure 9], though total number of diastolic dysfunction were 110 (35%). [Table 2] shows the age and sex distribution with highest frequency in the age group 61-65 years, comprising 32 percent and 51-60 years 28 percent, 40-50 year 24 percent, 65 + years 8 percent and 30-40 years 6 percent in descending order. The patients were classified

into two groups according to the degree of echocardiographic hypertrophy in group I (26 patients with a mass/volume ration > 1.8) and group II (24 patients with M/V ration < 1.8). Table shows age 50 were 76 percent of which 18 from group I (69.23%) and 20 from group II (83.33%). Diastolic dysfunction of group I and group II patients according to sex, presented male were 31 (62%) of which 19 from group I (73.08%) and 12 from group II (50%), female were 19 (38%) of which 7 from group I and 12 from group II (50%). Table shows types of diastolic dysfunction, with sample volume position at mitral inflow with tips of leaflets. There are 3 types of diastolic dysfunction -impaired relaxation (IR), pseudo normalization (PN) and restricted filling (RF). By E/A ratio, cases of various types are 20 (IR), 10 (PN) and 20 (EF); by deceleration time (Tdec), 19 (IR), 10 (PN) and 21 (RF), by IVRT are 19 (IR), 11 (PN) and 20 (RF). Table also shows types of diastolic dysfunction with sample volume position at right upper pulmonary vein inflow - impaired relaxation are easily diagnosed by S/D ratio > 1, but the differentiation between pseudo normalization and extracted filling is difficult unless compared with mitral flow PWD, especially tdec and IVRT. By S/D ratio, cases of various types are 20 (IR), 10 (PN) and 20 (RF); by AR velocity are 19 (IR), 10 (PN), 21 (RF) and by Ad/ARd are 19 (IR), 11 (PN) and 20 (RF). Cardiac catheterization has been done in 22 cases out of 50 cases of diastolic dysfunction. Among 22 cases, diastolic dysfunction was found in 18 cases (LVEDP > 15 mmHg) and left ventricular end-diastolic pressure was normal in 4 cases, all of them were of impaired relaxation variety. Therefore, Doppler echocardiographic estimation is comparable (significant) with cardiac catheterization estimation. ETT were positive in 4 patients in group I and 18 patients of group II, whereas ETT were negative in 22 patients in group I and 6 patients of group II. So, group I show more negativity than group II. Among 22 ETT positive patients, group I consists of 4 patients, of which 2 are CAG positive and 2 are negative. Group II consists of 18 patients of which 16 are positive and 2 are negative. According to degree of hypertrophy, NYHA class II dyspnoea in 26 cases of group I and in 24 cases of group II. Mean age is 53.65 years with SD 11.28 in group I and 57.29 with SD 9.44 in group II. Males were 31 and female 19. Mean heart rate ±SD in group I is 80.92±8.03 and 78.75±6.18 in group II. S3 were mainly found in group II, whereas S4 i group I. Both systolic and diastolic blood pressure is higher in group II, LVH were more positive in group I, cardiomegaly in group II, ejection fraction more in group I and duration of hypertension were almost equal.

Table 1: Age and sex distribution of the study patients (n=50)

| Age | Male | Female | Total |
|---------------|------------|------------|-------------|
| Group (Years) | | | |
| >35 | 2 (6.45) | 1 (5.26) | 3 (6.00) |
| 36-40 | 8 (25.81) | 4 (21.05) | 12 (24.00) |
| 41-45 | 8 (25.81) | 6 (31.58) | 14 (28.00) |
| 56-50 | 10 (32.26) | 6 (31.58) | 16 (32.00) |
| >50 | 3 (9.68) | 2 (10.53) | 4 (8.00) |
| Total | 31(62.00) | 19 (38.00) | 50 (100.00) |

Table 2: Distribution of diastolic dysfunction of group 1 (M/V >1.8) and group 11(M/V >1.8) patients according to age (n=50)

| Sex | Group 1 (n=26) | Group 11 (n=24) | Total No (%) |
|-----|----------------|-----------------|--------------|
| | MV>1.8 Range | MV>1.8 Range | |

| | | | |
|-------|------------------|------------------|-------------|
| | (1.9-2.9) No (%) | (1.2-1.7) No (%) | |
| <35 | 8 (30.77) | 4 (16.67) | 12 (24.00) |
| >50 | 18 (69.23) | 20 (83.33) | 38 (76.00) |
| Total | 26 (52.00) | 24 | 50 (100.00) |

P>0.05(not significant)

Table 3: Distribution of diastolic dysfunction of group I and group II patients according to age (n=50)

| Sex | Group I (n=26) No (%) | Group II (n=24) No (%) | Total No (%) |
|--------|-----------------------|------------------------|--------------|
| Male | 19 (73.08) | 12 (50.00) | 31 (62.00) |
| Female | 7 (26.92) | 12 (50.00) | 19 (38.00) |
| Total | 26 (52.00) | 24 (48.00) | 50 (100.00) |

P>0.05(not significant)

Table 4: Classification of patients according to left ventricular hypertrophy (echocardiography findings) (n=50)

| Variables | Group I (n=26) Mean±SD | Group II (n=24) Mean±SD | P Value |
|-----------|------------------------|-------------------------|-----------|
| LVPWT mm | 15.04±0.82 | 11.50±1.10 | <0.001*** |
| IVST mm | 15.15±0.92 | 11.50±0.98 | <0.001*** |
| LVIDd | 48.65±2.95 | 51.25±3.67 | <0.01** |
| LVIDs | 28.88±2.46 | 34.38±4.37 | <0.001*** |
| LV mass | 154.42±6.80 | 123.38±5.58 | <0.001*** |
| LVVd | 66.7±73.37 | 83.17±6.37 | <0.001*** |
| EF | 67.62±3.19 | 55.334±.13 | <0.001*** |

** =significant, *** = highly significant

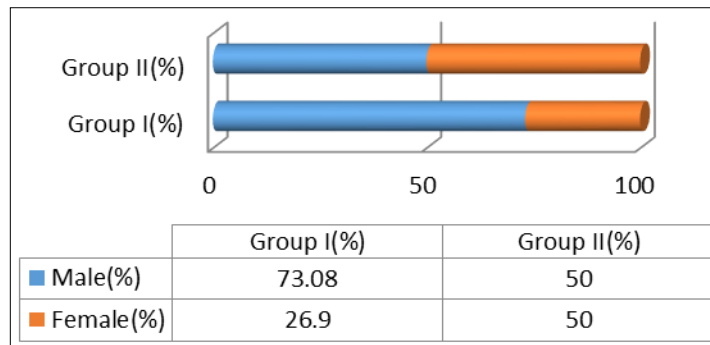


Fig 1: Age and sex distribution of the study patients (n=50)

Table 5: Duration of hypertension and diastolic dysfunction (n=50)

| Duration of hypertension (years) | Group I n=26 No (%) | Group II n=24 No (%) | Total No (%) |
|----------------------------------|---------------------|----------------------|--------------|
| >35 | 7 (26.92) | 12 (16.67) | 31 (62.00) |
| 36-40 | 7 (26.92) | 12 (16.67) | 19 (38.00) |
| 41-45 | 5 (19.23) | 24 (37.50) | 50 (100.00) |
| 46-50 | 5 (19.23) | 3 (12.50) | |
| >50 | 2 (7.69) | 4 (16.66) | |
| Total | (52.00) | 24 (48.00) | 50 (100.00) |
| Mean±SD | 11.31±5.76 | 12.96±5.49 | |

P>0.05(Not significant)

Table 6: Distribution of types of diastolic dysfunction by Doppler test (mitral inflow) (n=50)

| Sampl volume position Mitral flow | Impaired relaxation value Cases | Pseudonormaliza tion value Cases | Restricted filing value Cases |
|--------------------------------------|------------------------------------|--|----------------------------------|
| E/A Ratio | <1 20 | 1.0-1.5 10 | >1.5 20 |
| tdec (m/sec) | >240 19 | 180-220 10 | <180 21 |
| IVRT (m/sec) | >110 19 | 80-100 11 | <70 20 |

Table 7: Distribution of types of diastolic dysfunction by Doppler test (pulmonary venous inflow) (n=50)

| Sample volume position Pulmonary venous flow | Impaired relaxation value Cases | Pseudonormaliza tion value Cases | Restricted filing Value Cases |
|---|------------------------------------|--|----------------------------------|
| S/D Ratio | <1 20(1.0-2.0) | 1.0-1.5 10 (.3-1.0) | >1.5 20 (0.1-0.5) |
| AT velocity (cm/sec) | >25 19 | >25 10 | >35 21 |
| Ad/ARd (m/sec) | >1 19 | <1 11 | <1 20 |

Table 8: Correlation between Doppler echocardiographic findings and cardiac catheterization (n=50).

| Total number =50 | Diastolic dysfunction present | Absent | No % | No % |
|------------------------------------|-------------------------------|--------|------|------|
| Doppler echocardiography (n=50) | 50 (100.0) | 0 | | |
| Cardiac catheterization (n=22) | 18 (81.82) | 4 | | |
| done only in ETT positive patients | (18.18) | | | |

Fishers exact test = $P < 0.01$ (significant)

Table 9: ETT findings among groups of the study participants (n=50)

| Total number =50 | ETT positive | ETT Negative |
|------------------|--------------|--------------|
| Group 1 (n=26) | No (%) | No (%) |
| | 4 (15.38) | 22 (84.62) |
| Group 2 (n=24) | 18 (75.00) | 6 (25.00) |

Table 10: CAG findings of ETT positive patients among study participants (n=22)

| Total number =22 | CAG positive No (%) | CAG Negative No (%) |
|------------------|---------------------|---------------------|
| Group 1 (n=4) | 2 (50.00) | 2(50.00) |
| Group 2 (n=18) | 16 (88.89) | 2 (11.11) |

Table 11: Clinical findings according to degree of hypertrophy (n=50)

| Variables | Group 1 (Mean± SD) | Group II (Mean± SD) | P Value |
|-----------------------|-----------------------|------------------------|------------|
| Dyspnea (NYHA II-IV) | 26(100%) | 24(100%) | NS |
| Age(years)(Mean± SD) | 55.65±11.28 | 57.29±9.44 | >0.10 |
| Sex | | | NS |
| Male | 19(73.08) | 12(50.00%) | |
| Female | 7(26.92%) | 12(50.00%) | |
| Heart Rate | 80.92±8.03 | 78.75±6.18 | >0.10NS |
| Heart Sound | | | |
| S3 | 4(15.38%) | 17(70.83%) | |
| S4 | 15(57.69%) | 5(20.83%) | |
| Systolic BP | 169.23±24.81 | 176.67±23.34 | >0.10NS |
| Diastolic BP | 93.85±8.87 | 98.96±9.09 | <0.05* |
| ECG(LVH) | | | |
| Positive | 25(23.08%) | 4(16.67%) | <0.01* * |
| Negative | 1(3.85%) | 20(83.33%) | |
| Cardiomegaly | | | |
| Positive | 6(23.08%) | 7(21.17%) | <0.01* * |
| Negative | | 20(76.92%) | 17(70.83%) |

* =significant, * * =highly significant, NS= Not significant

5. Discussion

This prospective observational study was undertaken to evaluate left ventricular diastolic dysfunction in congestive heart failure resulting from systemic hypertension. As the sample size was not very large, they may not be representative of all hypertensive heart diseases with congestive heart failure in the community. In the present study, age of the study population ranged from 30 to 75 years, with incidence in 61-70 years comprising 32 percent, followed by 51-60 years 28 percent, 41-50 years 24 percent, 71-75 years 8 percent and 30-40 years 6 percent. Paul and Gheorghideet al. [1] also found diastolic dysfunction more in elderly (50 to 60 years) group. Although by standard echocardiographic criteria is reversal of E/A ratio favour the diagnosis of diastolic dysfunction, Marantz et al. (1994) [2] have shown that this inversion may be normal in older subjects. So, it is important to use the other parameters hi

elderly subjects. In the series of 50 hypertensive patients with LV failure with normal ejection fraction, it is possible to divide into two groups: group I with a mass/volume ratio > 1.8 whose dominant character is the high degree of reactive hypertrophy more in the elderly; and group II with a mass/volume ration<1.8 whose dominant character is high rate of ischemia, also more in elderly group. Left ventricular mass (LVM) is estimated using M-mode at end-diastole, using the measurement of IV septal thickness (IVST), posterior wall thickness (PWT) ar, left ventricular diameter (LVID) as follows: $LVM = 0.8 (1.04 f(ST + PWT + LVID)^3 - LVID^3) + 0.6 g$ where, 1.04 is the specific gravity of myocardium; 0.8 is the correction factor added since anatomic weight would otherwise be overestimated by approximately 20 percent (Lima 1998). LV volumes were calculated on the basis of end-systole and end-diastole diameter. To evaluate the Influence of the ventricular mass

and rate of regional ischemia, the cutting point is mass/volume ratio is 1.8 with maximal level of differentiation between hypertrophy and ischemia (Miguel *et al.* 1993). [3] Diastolic dysfunction was common in male (62%) than female (38%). Less number of female patients was involved in the study as small number of female patients attends the hospital for treatment. High incidence of hypertrophy were found in male patients in comparison to female (73.08% vs 26.92%) but incidence of ischemia were equal in both male and female (50% vs 50%). The most significant findings according to echocardiographic left ventricular hypertrophy were increased left ventricular posterior wall thickness and interventricular septal thickness, decreased LV end-diastolic and end-systolic diameter, increased LV mass and higher ejection fraction in group I (hypertrophic group) than in group II (ischemic group). These results are somewhat similar to the results described by Topol and Araill (1985) [4] and Miguel *et al.* (1993) [3-4]. Duration of hypertension did not correlate with the incidence of diastolic dysfunction and there was no significant difference between group I and group II. There was no published data both at home and abroad to compare the relationship between duration of hypertension and incidence of diastolic dysfunction with the present study. Doppler patterns of diastolic dysfunction include normal diastolic function, impaired relaxation, pseudo normal filling and restricted filling. These patterns evolve from one to another in a single individual with changes in disease evaluation, treatment and loading condition as described by Gerald *et al.* (1996) [5]. Impaired relaxation is typically manifested by E/A ratio <1, an increased deceleration time (T/Dec) and an increased isovolumetric relaxation time (IVRT) with sample volume position at mitral flow. Pulmonary venous flow is also abnormal characterized by increased S/D ratio, decreased atrial reversal (AR) velocity and increased ratio of atrial duration (Ad) to atrial reversal duration (ARd). At this state, patient is usually mildly symptomatic with exceptional activities with normal filling pressure and normal LA dimension. Pseudo normalization refers to normal appearance of mitral flow (E: A ratio between 1.0 and 1.5, tdec 180-220, IVRT 80-100) and pulmonary venous flow (S: D ratio <1, AR velocity >25, Ad/ARd <1). At stage II, the effects of impaired relaxation on early diastolic filling become opposed by the elevated left atrial pressure and the early diastolic transmitral pressure gradient and mitral flow velocity pattern return to normal. This phenomenon is called pseudo normalization to indicate that although left ventricular filling (appears normal, significant abnormalities of diastolic functions are present. In most cases, left atrial and left ventricular and diastolic filling pressure are elevated, the left atrium is increased in size and patients often complain of exertional dyspnoea (Appleton *et al.* 1988; Klein *et al.* 1994; Ishida *et al.* 1986) [6-10]. The restricted filling pattern is characterized by increased E: A ratio, decreased tdec, decreased IVRT on mitral flow, and decreased S: D ratio, increased AR velocity and Ad/ARd ratio is decreased on pulmonary venous flow. At stage III, it represents a severe decrease in LV chamber compliance. Diastolic filling pressures are elevated and patients are markedly symptomatic and demonstrate a severely reduced functional capacity. The left atrium is dilated and hypo contractile (Appleton *et al.* 1988) [6]. Cardiac catheterization were done only in 22 cases, of which 18 showed elevated LV end-diastolic pressure more

than 15 mmHg and 4 showed normal LV end-diastolic pressure, all of them showed diastolic dysfunction by Doppler assessment. Thus, Doppler evaluation provides a noninvasive, safe and rapid bedside alternative to cardiac catheterization for the assessment of LV diastolic dysfunction (Kaul 1991) [11]. Exercise tolerance tests were done in all cases that were physically active. Out of 50 cases, 22 were positive for provokable myocardial ischemia. Among 22, group I showed 4 positive cases and group II showed 18 positive cases. Thus, there were two distinct subgroups: group I characterized by high degree of reactive hypertrophy and low incidence of ischaemia and group II with only moderate hypertrophy and high risk of ischaemia. These findings correlated well with the results of Iriate *et al.* (1993) [12]. Coronary angiogram (CAG) was performed in 22 ETT positive cases, out of which, 2 patients of group I was positive, 2 were normal. And 16 patients of group II were positive, 2 were normal. Four patients, 2 from group I and 2 from group II were found to have provokable myocardial ischaemia but CAG were normal. The ETT positive but CAG negative cases constitute patients of syndrome X and their ischaemia may have been caused by hypertensive microangiopathy (Fragasno *et al.* 1997) [13]. There was no significant difference between groups for NYHA class, age, sex, heart rate, systolic blood pressure. A fourth sound was more common in group II, whereas the third sound was more frequent in group II. ECG evidenced LHV were more frequent in group I than group II, CXR evidenced cardiomegaly in group II than group I. The findings correlate well with the study of Iriate *et al.* (1993). [12] our findings indicate that patients with diastolic dysfunction in hypertensive heart disease with CHF with normal ejection presented in two ways: one characterized by severe hypertrophy and the other by a high rate of ischaemia. But their clinical profile was uniform and indistinguishable from CHF due to depressed LV systolic function as reported by Echeverria *et al.* (1983) [14], Dougherty *et al.* (1984) [15], Soufer *et al.* (1985) [16] and Kessheret *et al.* (1988). [16-17] Our study showed the possible influence of degree of hypertrophy or of regional myocardial ischaemia on the pathophysiologic or clinical characteristics of hypertensive heart disease results in CHF with normal ejection fraction. Therapy should be aimed at pathophysiologic regression of the hypertrophy in the first case and at improvement of ischaemia in the second case. Doppler echocardiography is an important tool in the case of such patients. It provides a noninvasive, safe and rapid bedside alternative to cardiac catheterization for the assessment of ventricular diastolic function.

6. Limitations of the Study

Present study conducted in one centre with small sample size, which can't reflect the scenarios of the whole country.

7. Conclusion

Congestive coronary failure from left chamber heartbeat dysfunction in hypertensive patients presents with 2 totally different profiles: one characterized by severe hypertrophy and low incidence of anemia and therefore the different by high rate of heart muscle regional ischemia and low incidence of hypertrophy. They give the same clinical pattern, and that they are indistinguishable from CHF because of low ejection fraction. Medical aid ought to be aimed toward pathophysiologic regression of the

hypertrophy within the 1st case and at improvement of the anemia within the second.

8. Acknowledgement

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Conflict of interest

The Author no conflict of interest.

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