

Research Article

Evaluation of diastolic function in hypertensive using echocardiography

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ABSTRACT

Background: Heart failure is one of the most common causes of cardiovascular morbidity along with mortality and hypertension is the most common cause of cardiac failure. Recent studies have shown that isolated diastolic dysfunction very often accompanies hypertensive heart disease. Aims: The present study was conducted to identify the risk of cardiac failure in hypertensive individuals by evaluating diastolic function using mitral inflow velocities of echocardiography.

Methods: Subjects attending cardiology outpatient department for master health check up with age between 25 to 80 years were selected. The procedure was explained and consent was obtained from the subjects. All the subjects underwent a detailed clinical examination. Height, weight and blood pressure of the subjects was measured and BMI was calculated. Two-dimensional Doppler echocardiographic examinations were performed with an ultrasonographic system (Philips iE 133 Echo Doppler). The student t test, chi square test and logistic regression analysis were used to find the influence of ejection fraction and heart rate on diastolic function.

Results: The trans-mitral inflow parameters on Doppler echocardiography such as E wave deceleration time (DT), isovolumetric relaxation time (IVRT) and early to late transmitral peak flow velocities ratio (E/A) were significantly different in hypertensive subjects on treatment having normal ejection fraction, when compared to normotensive subjects. We observed a decrease in E/A ratio and increase in DT, IVRT with hypertension.

Conclusions: From the present study it can be concluded that hypertension is significantly associated with diastolic dysfunction of heart, even in subjects with normal systolic function.

Keywords: Cardiac failure, Diastolic dysfunction, Echocardiography, Ejection fraction, Hypertension

INTRODUCTION

High blood pressure is more prevalent in the general population. Hypertension significantly contributes to cardiovascular (CV) morbidity and mortality by causing substantial structural and functional adaptations, including diastolic dysfunction (DD), left ventricular hypertrophy (LVH), ventricular and vascular stiffness. Chronic hypertension is the most common cause of diastolic dysfunction and failure. Abnormalities of ventricular relaxation and the consequences of diastolic

dysfunction may signify myocardial end-organ damage in patients who have hypertension which precede ventricular hypertrophy.^{1,2}

Isolated diastolic dysfunction is impairment of isovolumetric ventricular relaxation and decreased compliance of the left ventricle. Symptomatic diastolic dysfunction is called diastolic heart failure. With diastolic dysfunction, heart meets the body's metabolic needs, whether at rest or during exercise, but at a higher filling pressure's. With mild dysfunction, late filling increases

until the ventricular end-diastolic volume returns to normal. In severe cases, the ventricle becomes stiff and the atrial muscle fails, end-diastolic volume cannot be normalized even with elevated filling pressure. This process reduces stroke volume and cardiac output, causing effort intolerance. Moreover a small increase in left ventricular end-diastolic volume causes a marked increase in left ventricular end-diastolic pressure as a result of changes in the passive stiffness of the myocardium or in the process of active myocardial relaxation. This is indicated as a shift upward and to the left in a graph of the end diastolic pressure volume relationship reflecting enhanced sensitivity of intraventricular filling pressures to even small changes in filling volume. The consequential limitations on myocardial reserve narrow the window for clinical compensation, enhancing vulnerability to the development of heart failure symptoms as a consequence of rapid changes in afterload (e.g.: during bouts of uncontrolled hypertension).³

Among structural alterations, changes in the cardiac myocyte cytoskeleton and extracellular matrix (ECM) have been implicated as potential underlying causes of diastolic dysfunction.⁴⁻⁶ In hypertension, both synthesis and degradation of collagen increase greatly which aggravates disequilibrium resulting in rapid increase in collagen synthesis along with increased age related formation of advanced glycosylation end products. These structural changes in the ECM result in fibrosis of the myocardium, leading to increased ventricular stiffness, impaired relaxation, and diastolic dysfunction.⁷

This study was undertaken to estimate the prevalence of diastolic dysfunction in asymptomatic hypertensive population. Assessment of diastolic function can be performed with several non-invasive (2D- and Doppler-echocardiography, color Doppler M-mode, Doppler tissue imaging, MR-myocardial tagging, radionuclide ventriculography) and invasive techniques (micromanometry, angiography, conductance method). Doppler-echocardiography is the most useful tool to routinely measure diastolic function. Grading of diastolic dysfunction: The grading scheme is mild or grade I (impaired relaxation pattern), moderate or grade II (pseudo normal) and severe or grade III (restrictive filling). This scheme was an important predictor of all-cause mortality in a large epidemiologic study.⁸

METHODS

Subjects were selected from the hospital attending cardiology outpatient department for master health checkup, aged between 25 to 80 years, who volunteered to take part in the study. The procedure was explained and consent was obtained from the subjects. All the subjects underwent a detailed clinical examination before being included in the study as per the study protocol. The approval of department of cardiology and medical ethics committee was taken for this study.

Subjects were divided into two groups i.e., study and control group. Study group: 50 hypertensive individuals between 25-80 years of age with well controlled hypertension of more than 2 years duration. Control group: 50 healthy individuals between 25-80 years of age with no history of hypertension.

Inclusion criteria:

Study group: Controlled Hypertension for more than 2 years ; Males and females aged 25-80 years ; Normal body mass index (BMI: 19-25 Kg/m²) ; Normal Cardiovascular and Respiratory system on clinical examination; No history of Diabetes mellitus; No history of any acute infectious diseases; No history of any chronic diseases.

Control group: Normotensive population; males and females aged 25-80 years; Normal body mass index (BMI: 19-25 Kg/m²); Normal cardiovascular and respiratory system on clinical examination; No history of diabetes mellitus; No history of any acute infectious diseases; No history of any chronic diseases.

Exclusion criteria:

Subjects with age < 25 years and > 80 years; H/o diabetes, connective tissue disorders, congenital heart diseases, coronary artery disease, pericardial disease, valvular heart disease, cardiac arrhythmias and patients where it was technically difficult to perform echocardiography, subjects with abnormal cardiovascular and respiratory system on clinical examination.

Two-dimensional and Doppler echocardiographic examinations were performed with an ultrasonographic system (Philips iE 133 echo Doppler) equipped with multifrequency transducer. Left ventricular ejection fraction (LVEF) was calculated from apical two- and four-chamber views. Left ventricular inflow velocities were measured at the level of the mitral annulus in 4 chamber view.^{9,10} Early diastolic peak velocity (MF-E wave) and Late diastolic peak velocity (MF-A wave), and ratios of early and late diastolic peak velocities (E/A), deceleration time of E wave (DT), were computer derived. Isovolumetric relaxation time (IVRT), the time interval between aortic valve closure and mitral valve opening was measured.

Normal values for the trans-mitral Doppler echocardiographic parameters: Left ventricular ejection fraction - 0.55 to 0.75; Peak mitral early diastolic flow velocity E - in < 41 yrs. - 76 +/- 13 cm / sec; in > 55 yrs. - 63 +/- 11 cm / sec. Peak mitral late diastolic flow velocity A - in < 41 yrs. - 38 +/- 8 cm / sec; in > 55 yrs. - 52 +/- 9 cm / sec; Mitral flow E / A ratio - in < 45 yrs. - 2.1 +/- 0.6; in > 55 yrs. - 1.3 +/- 0.3; Iso volumetric Relaxation time (IVRT): Normal upper limits are 90, 100 and 105 ms in ages <30, 30-50 and more than 50 years respectively. Deceleration Time (DT): It is the time interval measured

from the peak of e - wave velocity till it reaches base line. Normal = 160 – 220 milli seconds.¹¹

Statistical analysis:

The results were analyzed using SPSS software. The data was presented as mean \pm standard deviation. Student t test was used to evaluate the differences between filling parameters among hypertensive and normotensive subjects. Chi square test was used for statistical analysis of gender distribution in cases and controls. Logistic regression analysis was used to find the influence of ejection fraction and heart rate on diastolic function as these two variables were found to be significantly different in cases and controls.

RESULTS

In our study, the cases and controls were comparable in terms of body mass index (p value = 0.210), age (p value = 0.979) and gender distribution (p value > 0.05).

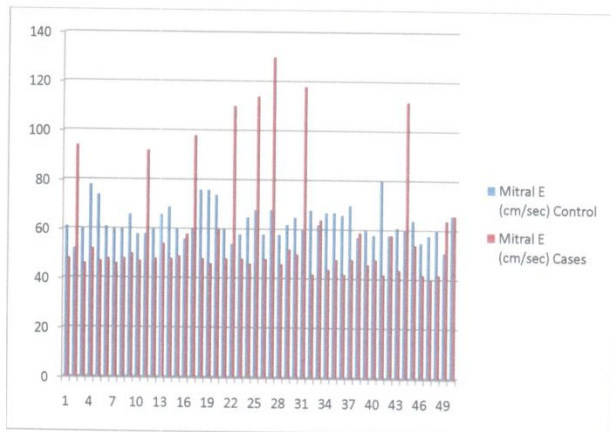


Figure 1: Comparison of mitral early diastolic peak flow velocity (E) between control and case group.

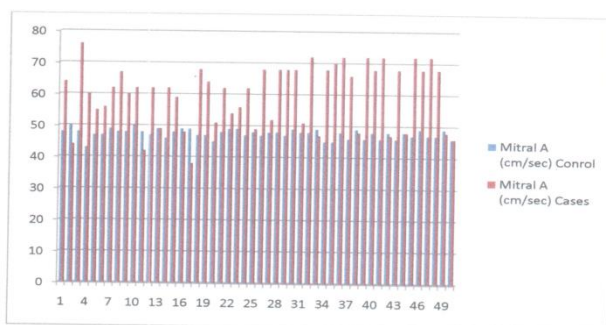


Figure 2: Comparison of mitral late diastolic peak flow velocity (A) between control and case group.



Figure 3: Comparison of deceleration time between control and case group.

All the subjects revealed a normal ejection fraction (> 45%) on Doppler echocardiography. The mean heart rate in cases (63.52 bpm) was less significantly (p < 0.0001) than that of controls (72.56 bpm). The mean mitral E value of cases (58.84 cm/s) was not decreased significantly (p = 0.227) when compared to controls (62.98 cm/s).



Figure 4: Comparison of isovolumetric relaxation time between control and case group.

The mean mitral A value of cases (59.98 cm/s) was increased significantly (p < 0.0001) when compared to controls (47.5 cm/s). The mean E/A ratio of cases (1.0546) was decreased significantly (p = 0.003) when compared to controls (1.3236).

The mean DT of cases (229.8 milli sec) was increased significantly when compared to controls (193.28 milli sec) (p < 0.0001).

The mean IVRT of cases (105.54 milli sec) was increased significantly when compared to controls (99.2 milli sec) (p = 0.016). The results of the study showed a decrease in E/A ratio and an increase in DT in hypertensive group compared to normotensive group.

Table 1: Different cardiac parameters with mean and S.D. along with p value.

Parameter	Control (Mean \pm SD)	Cases (Mean \pm SD)	P Value
E value cm/s	62.98 \pm 6.607	58.84 \pm 23.17	0.1147
A value cm/s	47.56 \pm 1.402	59.98 \pm 9.832	< 0.001
E/A ratio	1.323 \pm 0.168	1.054 \pm 0.59	0.03
DT (milli sec)	193.28 \pm 17.75	229.8 \pm 37.5	0.0001
IVRT (milli sec)	99.2 \pm 4.37	105.54 \pm 17.76	0.016
EF %	67.44 \pm 1.808	66.4 \pm 2.94	0.036

**Figure 5: Comparison of ejection fraction between control and case group.**

DISCUSSION

Arterial hypertension is a major risk factor for congestive heart failure. The progression of hypertensive cardiomyopathy towards heart failure includes serial left ventricular changes such as left ventricular concentric remodeling and left ventricular hypertrophy.¹²⁻¹⁴ In the presence of these left ventricular geometric abnormalities, significant modifications of left ventricular diastolic properties occur like increased stiffness and decreased active relaxation of myocardium. These modifications are globally defined as left ventricular diastolic dysfunction (diastolic dysfunction), which can precede alterations of left ventricular systolic function and per se be the main determinants of symptoms and signs of heart failure.^{15,16} Clinical presentation of heart failure with preserved ejection fraction (HFPEF) is indistinguishable from that of heart failure with reduced ejection fraction, with Doppler echocardiography being the only reliable discriminator.¹⁷ The diagnostic importance of this tool raises from the high feasibility of trans-mitral Doppler indices of diastolic function as shown by population studies.¹⁸

The results of our study indicate that there was an impaired left ventricular diastolic function in hypertensive when compared to normotensives which correlate with the results of Cesare Russo et al.¹⁹ who studied the independent effect of diabetes and

hypertension on left ventricular diastolic function in high risk population, without evidence of overt heart disease.

Decrease in E/A ratio in cases compared to controls suggests that, in chronic hypertensive, a greater proportion of end diastolic volume is the result of late filling, which is the function of atrial contraction (A) rather than the early filling (E) which happens to be due to active ventricular relaxation. This decrease in E/A ratio and increase in E-wave deceleration time suggests impaired myocardial relaxation during diastole in hypertensive population which in accordance with the results of Azad Akkoc et al.²⁰, Verdecchia et al.¹² and Akintunde et al.²¹ Increased IVRT i.e., the time taken by the left ventricular pressure to fall below left atrial pressure, suggests impaired myocardial relaxation.²² The increase in IVRT in our study correlates with the study done by Akintunde et al.²¹ Hypertension exerts pressure overload on the heart. Cardiac myocytes cannot proliferate once they have matured to their adult form. However, there is a constant turnover of the contractile proteins that make up the sarcomere. In response to the hemodynamic stresses associated with heart failure, angiotensin II, tumor necrosis factor α (TNF α), norepinephrine and other molecules induce protein synthesis via intranuclear mediators of gene activity such as c - fos, c - jun and c - myc. This causes myocyte hypertrophy with an increase in sarcomere number and a re-expression of fetal and neonatal forms of myosin and troponin.²³ The excessive workload imposed by increased systemic pressure is at first sustained by concentric left ventricular hypertrophy, characterized by an increase in wall thickness.²⁴ Myocardial relaxation is markedly influenced by pressure overload, which is greater in the presence of concentric left ventricular geometry. Pressure overload dependent abnormalities of coronary microcirculation and fibrosis related extracellular matrix disarray might also interfere with normal left ventricular relaxation. In addition concentric left ventricular geometry may also be associated with unfavorable metabolic abnormalities, which may influence cellular mechanisms of active relaxation.²⁵ Diastolic dyssynchrony, may be a probable mechanism contributing to hypertensive heart disease.²⁶

The prevalence of diastolic dysfunction in our study was 84%. This is correlating with the studies done by Akintunde et al.²¹ and Ike et al.²⁷ who studied the prevalence of diastolic dysfunction in adult hypertensive in Nigeria. However, this is higher than the 46% - 48% as reported by Angiomachalelis et al.²⁸ and De Mora et al.²⁹ in European population. This may be explained by the prevalence of left ventricular hypertrophy in 78% of our study group. Also, the prevalence of left ventricular hypertrophy is more in blacks, when compared to Caucasians, as demonstrated by Mayet et al.³⁰ Sixty eight percent of the subjects in our case study group were found to have early diastolic dysfunction. The decreased prevalence of late diastolic dysfunction (16%) can be explained by the fact that 34% of our study populations

were using ACE inhibitors. There is evidence that treatment with angiotensin converting enzyme inhibitors and angiotensin receptor blockers reduces interstitial collagen deposition and fibrosis, which directly improves myocardial relaxation and compliance as demonstrated by Angiomachalelis et al.²⁸ and Mitsunami et al.³¹ in their studies.

The mean heart rate in study group (63.52 ± 5.718 bpm) was significantly lower than controls (72.56 ± 8.61 bpm). This could be due to the effect of β adrenergic receptor blockers being taken as anti-hypertensive medication by 56% our cases. β adrenergic receptor blockers do not directly affect myocardial relaxation. However, they improve the diastolic filling by preventing tachycardia.³² The mean value of ejection fraction for controls was 67.44 ± 1.80 % and that for cases was 66.40 ± 2.94 %. The difference in the distribution of this variable among both groups was statistically significant ($p = 0.036$). Hence logistic regression analysis was done to find out whether this low ejection fraction was influencing the diastolic dysfunction. However on analysis it was found that ejection fraction did not have any significant influence on diastolic dysfunction.

CONCLUSION

From the present study it can be concluded that hypertension is significantly associated with diastolic dysfunction of heart, even in subjects with normal systolic function. The transmitral inflow parameters on Doppler echocardiography such as E wave deceleration time (DT), isovolumetric relaxation time (IVRT) and early to late transmitral peak flow velocities ratio (E/A) were significantly different in hypertensive subjects on treatment having normal ejection fraction, when compared to normotensive subjects. Among these three variables, E wave deceleration time (DT) was more significantly altered in cases compared to controls, followed by E/A ratio and IVRT. DT is the most important transmitral inflow parameter in determining diastolic dysfunction, when compared to IVRT and E/A ratio.

Summary: A cross sectional study was undertaken to analyze diastolic function in 50 asymptomatic, chronic, well controlled hypertensive population (case group) compared to 50 age, gender and body mass index matched normotensive population (control group). The results of the study showed a decrease in E/A ratio and an increase in DT in hypertensive group compared to normotensive group. This suggests that in chronic hypertensive, a greater proportion of end diastolic volume is the result of late filling rather than early filling, reflecting impaired active ventricular relaxation. IVRT is prolonged in hypertensive group compared to normotensive group indicating that, the time taken by left ventricular pressure to fall below left atrial pressure is increased which also suggests that there is impaired myocardial relaxation in hypertensive group.

Prevalence of diastolic dysfunction in hypertensive group was 84%. Among these people only 16% had late diastolic dysfunction. This decreased prevalence of late diastolic dysfunction could be attributed to improvement of diastolic function by usage of angiotensin converting enzyme inhibitors (ACEI), angiotensin receptor blockers (ARB) and β adrenergic receptor blockers, by majority of subjects of the hypertensive group (study group).

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Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

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