

Assessment of Diastolic Dysfunction in Hypertensive patients and its association with Left Ventricular Mass Index

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Objective: Early detection of diastolic dysfunction in hypertensive patients to reduce complications and improve outcome. **Place of Study:** East Medical and Cardiology wards of Mayo Hospital Lahore. **Study Design:** A descriptive cross-sectional study. **Patients and Methods:** 51 hypertensive patients between 50 – 80 years of either sex enrolled through out patient department of Mayo Hospital Lahore underwent Doppler Echocardiography and data was analyzed by using Chi-square and Students 't' tests. **Results:** The percentage of left ventricular hypertrophy (LVH) was 66.66%. On comparing hypertensive patients with and without LVH, E and A wave velocities, E:A ratio, deceleration time and isovolumic relaxation time were similar. **Conclusion:** 33.33% of patients had no LVH in the presence of diastolic dysfunction so LVH was not an independent factor associated with abnormal flow patterns in hypertensive patients with normal systolic contractility. Impaired relaxation was the predominant pattern of diastolic dysfunction and increased further with age. **Key words:** Diastolic dysfunction, hypertension, Left ventricular mass index.

Hypertension is the most prevalent and predictive remediable risk factor for cardiovascular disease. In the past evaluation of the myocardium has been limited to examining systolic function of the heart but recently however investigators have demonstrated that abnormalities of diastolic function of heart provides important contributions to the signs and symptoms experienced by the patients with heart diseases¹. It was also found that some patients having frank congestive heart failure have normal systolic function. In fact abnormalities of diastolic function may precede that of systolic function in the early stages of disease. Factors affecting diastolic function include age², heart rate, preload, after load, hypertension and ischemic heart disease. Even it was found that in young untreated hypertensives without LVH, 22% were found to have abnormal LV filling, clear abnormalities of diastolic dysfunction might antedate overt-LVH. It is found that normotensive relatives of the hypertensive patients also show diastolic dysfunction³. Two basic mechanisms by which hypertension causes diastolic dysfunction are left ventricular hypertrophy^{4,5} and microvascular myocardial ischemia. The most common abnormalities found in hypertensive diastolic dysfunction are the reduction in peak filling rate and increased time to peak filling. It was found that not only persistent hypertension causes cardiac structural abnormalities but also white-coat hypertension causes such abnormalities.

Diastolic dysfunction can be due to an obstruction to left ventricular filling or an external compression of the left ventricle, but it is usually considered to result from left ventricular abnormalities⁶ which may result from increased myocardial stiffness and impaired relaxation. Relaxation can either be slowed, decreasing early diastolic filling or incomplete, which impairs filling throughout the diastole and decreasing end-diastolic distensibility. In the pressure volume plan, reduced distensibility is represented by a leftward and upward shift of the end-diastolic pressure

volume relation (EDPVR). When this occurs, significantly higher pressures are required to distend the left ventricle to achieve the same end diastolic volume. If the shift in the EDPVR is severe enough, filling of the left ventricle to the level sufficient to produce a normal stroke volume can only be achieved with an elevated pulmonary venous pressure that will be associated with pulmonary congestion. Thus an alteration in diastolic distensibility may produce pulmonary congestion and congestive heart failure in the absence of systolic dysfunction. Three abnormal patterns of left ventricular filling are *delayed relaxation*, *pseudonormalization* and *restrictive* in which the E/A ratio (Early left ventricular filling/Atrial filling) is <1, >1 and >2 respectively.

Diastolic dysfunction may present for several years before any symptoms occur and may thus represent first phase of heart failure before systolic dysfunction prevails. Therefore it is of great importance to detect diastolic dysfunction in due time and start treatment before systolic dysfunction with irreversible structural changes occur.

Patients and Methods:

It was a descriptive cross-sectional study in which 51 hypertensive patients both male and female between 50-80 years of age were included through out patient department of Mayo Hospital Lahore while patients less than 50 and more than 80 years of age or having poor visualization on echocardiography or suffering from co-morbid illnesses were excluded.

After enrolment informed consent was taken and complete physical examination was performed to rule out Diabetes mellitus, ischemic heart disease, chronic renal failure, pulmonary disease, valvular and pericardial disease. ECG, X-Ray chest, urine complete examination, blood urea, serum creatinine and blood sugar was analyzed for each patient under study. Then each patient was given time for Echocardiography in the Medicine/Cardiology department where LVIDd (left ventricular internal

diastolic dimension), LVIDs (left ventricular internal systolic dimension), IVSD (interventricular septal dimension), PWT (posterior wall thickness), E/A ratio, IVRT (isovolumetric relaxation time), DT (deceleration time) measured and left ventricular mass (LVM) was calculated by using cube method.

$$LVM = 1.1 [(IVS + LVID + PWT)^3 - LVID^3]$$

Then it was divided by 1000 to convert it into gram and this mass was divided by surface area (BSA) to convert it into left ventricular mass index (LVMI kg/m²)

$LVMI = LVM/BSA \text{ kg/m}^2$ Then percentage of hypertensive patients with diastolic dysfunction was calculated individually by using E/A ratio, IVRT&DT. Hypertensive patients with and without diastolic dysfunction were put into two groups and students 't' test was applied to compare the means of two groups.

Results:

Twenty-one (41.17%) out of total 51 were male patients and 30 (58.82%) were females. 34 patients (66.66%) had left ventricular hypertrophy. The statistical difference between the left ventricular mass index of the patients who had left ventricular hypertrophy to those who has no left ventricular hypertrophy was significant $P < 0.001$. There was a statistically significant difference of the diastolic blood pressure and posterior wall thickness of the patients who had left ventricular hypertrophy than those who had no hypertrophy $P < 0.05$ however hypertrophic group had higher systolic blood pressure (mean value 164.83 mm of Hg) and left ventricular internal dimensions than non hypertrophic group. No correlation between left ventricular mass index and either of the values of Isovolumetric relaxation time, Deceleration time and E/A ratio was noted $P > 0.05$. So there is no difference in any of these filling parameters between hypertrophics and non hypertrophics. Then sensitivity of each of these parameters for diastolic dysfunction was assessed and deceleration time was found as the most sensitive parameter in 45 patients (88.23%) and the least one was E/A ratio found in 29(56.86%) patients. But the mitral flow patterns among patients with and without hypertrophy was almost identical $P > 0.05$.

Discussion:

The Doppler ultrasound data has described the three distinct mitral flow patterns: normal, impaired relaxation and restrictive physiology^{7,8,9}. The latter two represent the extreme of diastolic dysfunction spectrum. In this study standard 2-D directed M-mode measurements of left ventricle were performed on 51 patients and results were compared with previously published reports by Kundi et al⁵ and Memon MA et al⁴. Left ventricular dimensions and left ventricular mass were significantly higher in our study when compared with the study results of Kundi et al⁵ and almost identical when compared with the Memon MA et al⁴. The significant difference of Kundi et al⁵ and our study parameters is probably due to perhaps racial differences. On comparing with Zabalgoita M et al¹⁰ all parameters

were similar except left ventricular internal dimensions and left ventricular mass index which were comparatively lower in our population. These differences are likely to be due to difference in population with different genetic, endocrine factors and physical activity. In our study DT was the most sensible parameter (88.23%) second one was IVRT (82.35%) and least one was E/A ratio identified in 56.86% cases while comparing with Edam E et al¹¹ study, IVRT was the most sensitive parameter, E/A ratio was the second most and DT was the least sensitive. The probable explanation of this difference was perhaps the duration of hypertension and treatment compliance which was very poor on our study because of socioeconomic constraints that was probably responsible for more interstitial fibrosis, and more severe stage of diastolic dysfunction.

Conclusion:

33.33% of cases with diastolic dysfunction had no left ventricular hypertrophy (LVH) so LVH is not an independent factor associated with abnormal flow patterns (diastolic dysfunction) in hypertensive patients with normal systolic function. Diastolic dysfunction increases as the duration of hypertension increases, therefore, it is of great importance to detect diastolic dysfunction in due time and start treatment before systolic dysfunction with irreversible structural changes occurs. Doppler echocardiography is a reliable, cost effective and easily acceptable technique to detect diastolic dysfunction.

References:

1. Mirsky I. Assessment of diastolic function: Suggested methods and future consideration. *Circulation* 1984;69:836.
2. Courtois M, Mechem CJ et al. Factors related to end systolic volume and important determinations of peak early diastolic transmitral flow velocity. *Circulation* 1998; 85:1132-35.
3. Graettinger WF, Neutel JM, Smith. LV diastolic filling alterations in normotensive adolescent with a family history of hypertension. *Am J Cardiol* 1999;68:51-56.
4. Anis M, Ishaq M et al. Echocardiographic correlation of left ventricular mass index in normotensive and hypertensive Pakistani population. *Pak J Cardiology* 2000;11(1):9-21.
5. Kundi A. Echocardiographic Doppler parameters in normal adult Pakistani population: 2-D directed M-mode echocardiographic measurements. *Pak J Cardio* 1993;4:156.
6. Palmieri V, Wachtell K, Gerds E, Bella J et al. Left ventricular function and haemodynamic features of inappropriate left ventricular hypertrophy in patients with systemic hypertension. *Am Heart J* 2001; 141(5):784-91.
7. Little WC et al. Evaluation of left ventricular diastolic function from the pattern of left ventricular filling. *Clin Cardiol* 1998;21:5-9.
8. Garcia MJ et al. An index of early left ventricular filling that combined with pulsed Doppler peak E velocity may estimate capillary wedge pressure. *J Am Coll Cardiol* 1997;29:448.
9. Pinamonti B, Zecchin M, Di LA, Gregori D. Persistence of restrictive left ventricular filling pattern in dialyzed cardiomyopathy: an ominous prognostic sign. *J Am Coll Cardiol* 1997;29:604-12.
10. Zabalgoitia M, Rehman SN et al. Role of left ventricular hypertrophy in diastolic dysfunction in aged hypertensive patients. *J Hypertens* 1997; 15(10): 1175-9
11. Adam E, Schussheim, Joseph A et al. Midwall fractional shortening in an independent predictor of left ventricular diastolic dysfunction in asymptomatic patients with systemic hypertension. *Am J Cardiol* 1998;82:1056-59.