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Echocardiographic Comparison in Diastolic Failure Patients with and without Left Ventricular Hypertrophy

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Abstract

Introduction: Left ventricular diastolic dysfunction (LVDD) is suspected when a patient presents with a clinical diagnosis of heart failure despite a preserved ejection fraction. Left ventricular hypertrophy (LVH) has been associated with poor clinical outcomes in patients of LVDD. In this study, we aimed to compared the clinical characteristics and echocardiographic findings of LVDD patients with and without LVH. *Methodology:* LVDD patients at our centre diagnosed with and without LVH were included in the study. LVDD was defined by the abnormal relaxation patterns of Doppler mitral inflow and tissue Doppler. Demographic, clinical, laboratory and echocardiographic parameters were compared between LVH and non-LVH patients with LVDD. *Results:* 50 patients of LVH and non-LVH LVDD were included. Age and systolic blood pressure were found to be significantly higher among the LVH group, while mean heart rate and total cholesterol were found to be significantly lower among LVH patients. Mean left ventricular mass index (121.46±19.32 vs 74.93±11.54 gm/m2), p value <0.001), left atrium size (3.82±0.82 vs 3.57±0.34 cm, p value < 0.001), relative wall thickness (0.59±0.11 vs 0.53±0.16, p value < 0.05), filling pressure (16.85±5.21 vs 15.01±4.32 mm of Hg, p value < 0.05) and Tei index (0.59±0.16 vs 0.51±0.11, p value < 0.05) were found to be significantly higher among patients with LVH. *Conclusions:* Coexistence of LVH and LVDD can increase the mortality manifold and thus early identification by echocardiography may prompt close monitoring and aggressive management.

Keywords: Left Ventricular Hypertrophy; Left Ventricular Diastolic Dysfunction; Tei Index.

Introduction

Diastolic heart failure is a clinical syndrome in which patients have symptoms and signs of heart failure, normal or near normal left ventricular (LV) ejection fraction (EF), normal or near normal LV volume, and evidence of diastolic dysfunction [1]. From the physiological point of view, left ventricular diastolic dysfunction (LVDD) may be used to describe the presence of impaired LV relaxation and/or increased passive LV stiffness in the presence of normal or abnormal LV systolic function. A clinician may suspect LVDD when a patient presents with a clinical diagnosis of heart failure despite a preserved ejection fraction. Strong associations of LVDD with aging, obesity, hypertension, diabetes mellitus and left ventricular hypertrophy have been suggested previously [2]. Furthermore, LVDD is significantly associated with the development of heart failure and high mortality even when it is asymptomatic.[3]Left ventricular hypertrophy (LVH) in particular has been associated with poor clinical outcomes in patients of LVDD [4]. In this study, we aimed to compared the clinical characteristics and echocardiographic findings of LVDD patients with and without LVH.

Methodology

Study design and sampling

The present study reviewed the clinical parameters of all patients who were diagnosed with LVDD at our centre between March 2017 till March 2018. Equal

number of patients with and without LVH were included in the study (50 each). We excluded patients who had a history of valvular heart disease, atrial fibrillation and myocardial infarction in the past 6 months. So that LV systolic failure does not have a confounding effect on the filling pressures, patients with ejection fraction (EF) less than 50% were excluded as well.

The study protocol was approved by the institutional ethics committee and informed written consents were obtained from all patients. The study was conducted according to the principles expressed in the Declaration of Helsinki and the treatment of the patients was not affected in any way by being included or excluded from the study.

Echocardiography procedure and measurements

Transthoracic echocardiographic examinations were performed by the same experienced technician using the General Electric (machine name and manufacturer). From standardized views, M-mode guided two dimensional images were taken and stored digitally. To assess the diastolic parameters, the mitral inflow and the mitral annular motion velocity were measured by the Doppler studies. Transmitral E wave velocity (E), transmitral A wave velocity (A), early diastolic mitral velocity (Ea) and the deceleration time from the peak of the early diastolic wave to baseline (DT) were assessed for all patients. For this study, LVDD was diagnosed in patients with impaired relaxation and pseudonormal/restrictive mitral flow pattern. A normal mitral inflow pattern was recognized if E/A ratio was 0.75 or higher, Ea 8 cm/s or higher, and E/Ea 10 or lower. Patient was classified with impaired relaxation mitral inflow pattern if the E/A ratio was less than 0.75, and with pseudonormal/restrictive mitral inflow pattern if the E/A ratio was 0.75 or higher, Ea less than 8 cm/s or E/Ea greater than 10 [5].

LVH was defined as suggested by the the American Society of Echocardiography/European Society of Echocardi-ography chamber quantification guidelines left ventricular mass index more than 115 g/m^2 in men and more than 95 g/m^2 in women [6].

The Tei index combines both systolic and diastolic cardiac performance and can be practically be used to assess overall cardiac performance [7]. It is calculated by the following formula: (isovolumic contraction time+isovolumic relaxation time)/ ejection time, and in adults, a left ventricle Tei index of less than 0.4 is considered normal. Filling pressure (FP) was calcu-lated by the following formula: [1.24 (E/e') + 1.9] where E and e' are the early filling velocities of the mitral inflow and the tissue Doppler, respectively [8].

Data Collection and Data Analysis

Demographic data including age, gender and past medical history of diabetes mellitus, hypertension, and coronary artery disease, body mass index (BMI), systolic BP, diastolic BP, pulse pressure, and heart rate were obtained from medical records or interviews with patients. Results of laboratory data including fasting glucose, triglyceride, total cholesterol, and hematocrit were also noted for each patient. Additionally, past history of any medication like angiotensin converting enzyme inhibitors (ACEIs), angiotensin II receptor blockers (ARBs), b- blockers, calcium channel blockers (CCBs), diuretics, and nitrates was also noted. Data were entered in SPSS software (version 23) for statistical analysis. Quanti-tative data were expressed as means (and standard deviation) and qualitative data as percentages. After checking for normality, continuous variables were compared using the student's t test and and categorical by chi-square. All tests were 2-sided and the level of significance was established as $p < 0.05$.

Results

During the study period, we included a total of 100 patients with a diagnosis of LVDD, of which LVH and non-LVH patients were 50 each. Comparison of baseline characteristics of patients in both the groups is as described in Table 1. Age (72.44± 11.29 vs 69.16±9.45 years, p value <0.05) and systolic blood pressure (134±16 vs 131±15 mm of Hg, p value <0.05) were found to be significantly higher among the LVH group as compared to the non-LVH group, while, mean heart rate (72±12 vs 76±16 beats/min, p value < 0.05) and total cholesterol (184.8±51.7 vs 210.3±44.6 mg/dl, p value <0.05) were found to be significantly lower among LVH patients. On comparison of echocardiographic parameters, mean left ventricular mass index (121.46±19.32 vs 74.93± 11.54 gm/m^2), p value <0.001), left atrium size (3.82 ± 0.82 vs 3.57±0.34 cm, p value < 0.001), relative wall thickness (0.59±0.11 vs 0.53±0.16, p value < 0.05), filling pressure (16.85±5.21 vs 15.01±4.32 mm of Hg, p value < 0.05) and Tei index (0.59±0.16 vs 0.51± 0.11, p value < 0.05) were found to be significantly higher among patients with LVH as compared to patients without LVH. (Table 2).

Table 1: Baseline characteristics of the patients.

Variable	Group I (LVH patients, n=50)	Group II (non-LVH patients, n=50)	P Value
Demography and past history			
Age	72.44 ± 11.29	69.16 ± 9.45	<0.05
Gender (M/F)	29/21	28/22	0.218
BMI (kg/m2)	27.43 ± 7.21	28.03 ± 6.92	0.102
Systolic blood pressure (mm of Hg)	134 ± 16	131 ± 15	<0.05
Diastolic blood pressure	73 ± 13	72 ± 11	0.231
Pulse pressure (mm of Hg)	61 ± 11	59 ± 13	0.072
Heart rate (beats/min)	72 ± 12	76 ± 16	< 0.05
DM (%)	21 (42%)	24 (48%)	0.122
Hypertension (%)	39 (78%)	32 (64%)	0.083
Coronary artery disease (%)	19 (38%)	23 (46%)	0.342
Medication history			
ACE inhibitors/ Angiotensin receptor blockers	29 (56%)	26 (52%)	0.523
Calcium Channel Blockers	23 (48%)	28 (56%)	0.091
b blockers	28 (56%)	31 (64%)	0.711
Diuretics	23 (48%)	28 (56%)	0.629
Nitrates	15 (30%)	19 (38%)	0.023
Laboratory parameters			
Creatinine (mg/dl)	1.29 ± 1.02	1.18 ± 1.42	0.092
Fasting glucose (mg/dl)	108.4 ± 29.4	115.6 ± 32.9	0.183
Triglyceride (mg/dl)	173 ± 53	160 ± 85	0.062
Total cholesterol (mg/dl)	184.8 ± 51.7	210.3 ± 44.6	<0.05

Values described either as mean ± standard deviation or as numbers (percentage)

Table 2: Findings of echocardiography of the patients.

Echocardiography parameter	Group I (LVH patients, n=50)	Group II (non-LVH patients, n=50)	P Value
Left Ventricular mass index (gm/m2)	121.46 ± 19.32	74.93 ± 11.54	<0.001
Left Atrial size (cm)	3.82 ± 0.82	3.57 ± 0.34	< 0.001
Relative wall thickness	0.59 ± 0.11	0.53 ± 0.16	< 0.05
Ejection Fraction %	62.31 ± 7.42	66.82 ± 8.14	0.061
Transmitral E wave velocity / Transmitral A wave velocity	0.84 ± 0.33	0.82 ± 0.28	0.102
Filling Pressure (mm of Hg)	16.85 ± 5.21	15.01 ± 4.32	< 0.05
Tei index	0.59 ± 0.16	0.51 ± 0.11	<0.05

Values described as mean ± standard deviation

Discussion

This study evaluated and compared LVDD patients with and without LVH, which is the most commonly associated condition with LVDD. In our patient population, LVDD was diagnosed among aged population; LVH patients were significantly older than non-LVH patients. More than two thirds of the patients were hypertensive. In a population based survey, diastolic abnormalities were frequently associated with other pathologic conditions such as arterial hypertension, LV hypertrophy, and coronary artery disease [9]. The authors of that survey also found diastolic dysfunction to be related to obesity and diabetes mellitus. In the backdrop of a high prevalence of hypertension, finding significantly high systolic blood pressure in the LVH group than the non-LVH group points us towards the expected relationship between LVH and hypertension [10]. A mechanism explaining this association is the neuro-hormonally mediated maladaptive LVH, resulting in diastolic dysfunction. Hypertension results in the activation of the renin–angiotensin–aldosterone axis and increased transforming growth factor-beta levels, which stimulate extracellular matrix deposition, resulting in perivascular fibrosis in the heart [11].

Over the years the technical capabilities of echocar-diographic and Doppler studies have improved significantly [12]. The non-invasive nature and the widespread availability of these techniques have resulted in their widespread use in diagnosing diastolic heart failure. These studies provide a valuable insight into the dynamics of LV relaxation and filling and a significant prognostic information as well. The finding of

echocardiographic evidence of diastolic dysfunction in an asymptomatic patient is a risk factor for the development of heart failure and by identifying such patients early, their progres-sion can be prevented [13]. The basic mechanisms that result in diastolic dysfunction may either be intrinsic to the cardiomyocyte or may be a consequence of abnormalities in the extracellular matrix. Ventricular relaxation is also affected by neurohormonal and cardiac endothelial activity [14]. The common causes of LVDD are hypertrophy and ischemia, but several other conditions may cause heart failure in the presence of a normal LV EF.

For diagnosing diastolic heart failure, evidence of heart failure in the presence of normal LV EF is necessary. While some authors have supported the usefulness of echocardiogenic evidence of abnormal LV relaxation or diastolic stiffness [15], some have argued including the results of cardiac catheterization to document presence of diastolic failure [16]. Though, blood BNP levels can diagnose heart failure, it cannot differentiate between systolic and diastolic heart failure. Echocardiography is currently the most commonly used modality to assess LV function. In addition to the ejection fraction, the echocardiogram provides other information on LV function, LV geometry and wall thickness, regional wall motion abnormalities, valvular disease, pericardial disease, and left atrial size. Tei Index was high in all patients included in the study, but was significantly higher in LVH patients. Impaired relaxation evidenced by prolonged isovolumic relaxation time can be induced by both LV systolic as well as diastolic dysfunction. Therefore, hemodynamic indices of relaxation can be reliably identified with the Tei Index as both phases of LV function are reflected in Tei Index [17].

There are a few limitations of this study. Firstly, all measurements were not confirmed by cardiac catheterization, which is the gold standard for such estimations. Secondly, this being a single centre study, the results might not be generalizable to other centres as the technique and expertise of echocardiography can vary. Lastly, the impact of LVH on the clinical outcome of LVDD patients could not be ascertained in this study. This would require studying the survival analysis of LVDD patients by following them for a long time.

Conclusion

The results of this study show that LVDD patients with LVH had higher filling pressure and Tei Index as compared to those without LVH. Hypertension was common in LVDD patients and higher systolic blood pressure in LVH group could have directly caused higher filling pressures. Coexistence of LVH and LVDD can increase the mortality manifold and thus early identification by echocardiography may prompt close monitoring and aggressive management.

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Conflict of interest: None

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