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Physiology

CORRELATION OF LEFT VENTRICULAR REMODELING IN DIABETICS ALONE, HYPERTENSIVE, BOTH DIABETICS AND HYPERTENSIVE AND NORMAL

Dr Bhanu Priya H* Assistant Professor, Department of Physiology, Akash Institute of Medical Science and Research Centre. *Corresponding Author

Dr Kusuma Devi MS Professor, Depatrment of Physiology, BANGALORE Medical College and Research Centre.

ABSTRACT Diabetes mellitus (DM), a worldwide epidemic disease, affects the heart not only through the intensification of the classic predisposing factors for the development of heart failure, but also through the direct involvement of the myocardial tissue. The aim of our study was to evaluate the effect of DM on Left Ventricle dimensions, wall thickness and mass in both normotensive and hypertensive adults.

Subjects and methods: The study included 53 patients (mean age 45.5 ± 9.5 years) divided into 3 groups. I: included patients with DM only, II: included patients with hypertension only and III: included patients with both. Additional 15 normal subjects were added as control group. Left ventricle dimensions, wall thickness, systolic function and mass were assessed by 2 - Dimensional Echocardiography.

Results: LV thickness and mass was significantly higher in diabetics compared to normal. LV wall thickness,mass was significantly higher in hypertensives compared to diabetics.

Conclusion: DM is an independent risk factor for the increased LV mass and impaired systolic function regardless of association with hypertension or normal.

KEYWORDS : Diabetes Mellitus. Hypertension,2D ECHO

INTRODUCTION:

India is the diabetic capital of the world with nearly 50 million diabetics.It has been suggested that impairment of left ventricular (LV) function in patients with DM is due to concomitant risk factors such as arterial hypertension (HTN) or to diffuse peripheral and coronary atherosclerosis.However, actual mechanisms remain unclear and evidence has accumulated for the existence of a distinct diabetic cardiomyopathy[1].

Diabetic cardiomyopathy is currently defined as a diastolic dysfunction, and several studies of DM patients have identified Left Ventricular diastolic dysfunction as the earliest functional alteration in the course of diabetic cardiomyopathy,and also established it as an important prognostic parameter[2,3].

In addition, recent investigations have found that Left ventricuar longitudinal myocardial systolic dysfunction, rather than Left ventricular diastolic dysfunction, should be considered the first marker of a preclinical form of diabetic cardiomyopathy in DM patients with preserved Left ventricular ejection fraction without overt Heart Failure[4].

M – mode ECHO remains relatively inexpensive diagnostic tool in assessment of Left Ventricular structure. Fractional fiber shortening is used as a determinant of systolic performance in Left Ventricular Hypertrophy. Increased Left Ventricular mass thickness exihibits reduced shortening velocity. Significant impairment in FFS is due to alterations in dimensions of Left ventricle wall thick,Left Ventricle cavity,Left Ventricle geometry and fibrous changes in Left ventricle myocardium[5].

Several epidemiological investigators described the association between Left Ventricular hypertrophy and impaired glucose tolerance. Relation between DM and Left venticle mass are inconsistent,hence the present study was undertaken to evaluate the effect of DM on Left ventricle dimensions, wall thickness and mass in both normotensive and hypertensive patients.

SUBJECTS AND METHODS

Study was carried out in Bnagalore Medical College and Research Centre during the eriod January 2016 – March 2016.53 patients were referred to cardiology clinic for cardiac assessment. Depending on the history, the patients were divided into 4 groups:

Group I: included 13 patients with DM only.

Group II: included 13 patients with hypertension only.

Group III: included 12 patients with DM and hypertension.

Group IV: included 15 normal individuals with no hypertension and DM as control group

Clinical examination

Detailed history and through clinical examination were performed. Hypertension and DM was defined.

Transthoracic 2DE examination

Examination was performed by echo-technician while the patient in the left lateral decubitus position using both apical and parasternal views. The following M-mode parameters were measured: 1) LV systolic (LVSD) and diastolic (LVDD) internal dimensions, 2) Thickness of interventricular septum (IVS) and LV posterior wall (PW) at diastole, 3) LV fractional shortening (FS) by the standard equation: (LVDD – LVSD)/ LVDD, 3) LV mass using the LV mass (g) = 0.8 {1.04 x (LVDD + IVS + PW)3 - (LVDD)3} + 0.6, 4)

Statistical Analysis

All data were presented as mean ± SD. Independent sample t-test was used for comparison between the three patients groups and normal group. Comparison between the groups was performed by analysis of variance (ANOVA) test. Frequencies are expressed by percentage with 95% confidence intervals. A single and multiple linear regression analysis with stepwise elimination were performed. LV mass was considered as the dependent variable and the independent variables included in the model were gender, age and the presence or absence of hypertension and DM. The level of significance was set to $P < 0.05$.

RESULTS

Baseline criteria

Parameter	Control	Only DM	Only HTN	Both
Age	46.7±1.8	45.7±1.8	44.7±1.8	48.7±1.8
LA (cm)	26.7±1.8	40.08±2.41	40.3±1.92	42.83±4.8
LVDD (cm)	42.5±5.8	52.5±6.5	52.5±6.6	54.16±6
LVSD(cm)	28.5±2.5	39.9±5.8	39.8±4.8	41.08±3.9
FS(%)	35.2±3	27.6±2.6	28.5±1.8	28.2±1.87
EF(%)	64.6±3.08	51.6±5.9	52.6±5.3	49.2±6.1
IVS(cm)	10.3±2.02	13±1.7	13.±1.5	13.1±1.6
PW(cm)	9±0.29	12.3±0.5	12.2±0.58	12.3±0.51
LV Mass(gm)	202.2±20.2	407.2±77.1	450.2±33.2	416.7±56.8

Effect of DM in normotensive patients

Compared to normal's, Group I patients showed significant increase of LVDD and LVSD (52.7 ± 6.5cm, 28.5 ± 2.8 cm vs 42.5 ±5.8cm, 39.9 ± 5.5cm respectively; $p<0.0001$). Values of LV wall thickness (IVS and PW) and mass were significantly higher in Group I than Group IV (all $p<0.0001$).

DM versus hypertension

In patients with hypertension only (Group II), thickness of IVS and PW were significantly higher than in patients with DM only (Group I) (13.5 ± 1.2 cm and 12.3 ± 0.58 cm vs.13 ± 0cm and 12.2 ± 0.1cm; $p<0.001$ respectively). LV mass were significantly higher also in Group II than in Group I (452.1 ± 33.3 g vs.407.6 ± 77.1 g; p= 0.001). LVDD and

LVSD showed no significant difference between both patient groups.

Effect of DM in hypertensive patients

Compared to normal's, Values of LVDD, LVSD, wall thickness (IVS and PW) and mass were significantly higher in Group III than Group IV (all $p<0.0001$).Comparison between Group I and Group III showed no significant difference in all values except for IVS thickness which was significantly higher in Group III than Group I.

DISCUSSION

It is widely acknowledged that increased LV mass is thought to increase cardiovascular risk through a series of unfavorable metabolic, functional and structural cardiac changes. This explained the facts that an increased LV mass is a premier risk factor for cardiac events e.g. myocardial infarction and heart failure.[5]

Accumulating data from experimental, pathological, epidemiological and clinical studies have shown that DM affects the cardiac function (systolic and diastolic) and structure independent of hypertension, coronary artery disease or any other known risk factors [7-12].

The current study aimed to evaluate the effect of DM on LV internal dimensions, systolic function and LV mass using conventional echocardiography. The study included 3 equal patients groups (DM without hypertension, Hypertension without diabetes and DM with hypertension) and described the changes in echo parameters in each group in comparison with the other groups and with normal individuals.

Struthers and Morris reported that LV hypertrophy was present in 30% of patients with type 2 DM independent of blood pressure or use of antihypertensive medication. The prevalence of LVH increases with the severity of hypertension ranging from 38% to 72% [12].

Our results showed that the prevalence of increased LV wall thickness and mass in patients with DM (Group I) was comparable to those with hypertension (Group II). Compared to normal individuals, LV mass was significantly higher in all 3 patient groups. When the 3 patient groups were compared with each other, no significant differences in LV wall thickness, systolic function and LV mass were detected.

The impaired glucose metabolism may cause electrical instability of the myocardium. Insulin may act as a growth hormone for cardiomyocytes, mainly through the extracellular signal regulated kinase (ERK) and/or protein kinase C (PKC) pathways.

Insulin also stimulates collagen synthesis in cardiac fibroblasts. Hyperglycemia may in itself promote left ventricular hypertrophy, mainly involving transforming growth factor beta1 and collagen synthesis by cardiac fibroblasts, likely via the phosphatidylinositol 3-kinase and PKC beta pathways. Insulin interacts with neurohormonal systems and activates the sympathetic nervous system, increases the pressor response to angiotensinII and increases the stimulating effects of angiotensin II on ERKs involved in cellular proliferation and extracellular matrix deposition[13]. In short, insulin resistance can influence cardiac structure through several mechanisms: increased cellular lipids, non-enzymatic glycated end products, altered myocardial protein degradation, insulin-like growth factors mediated effects, altered matrix remodeling, altered vascular compliance, sympathetic activation , increased renal sodium reabsorbtion. Also, know days, there are evidence, based more on experimental data, for the role of oxidative stress on cardiac remodeling. ROS stimulate myocardial growth, matrix remodeling, and cellular dysfunction. ROS activate a broad variety of hypertrophy signaling kinases and transcription factors [10].

Cardiomyocyte apoptosis is another important contributor to hypertrophic remodeling and cell dysfunction. Apoptosis is inhibited in cells at low levels of ROS stimulation, whereas the opposite occurs at higher levels. Mechanisms include DNA and mitochondrial damage and activation of proapoptotic signaling kinases[15,16].

Oxidative stress and metabolic shift are intimately linked with myocardial hypertrophy, but their interrelationship is not clearly understood.

Our findings may have clinical impact in treating and following up of patients with DM. It will be of great value for the treating physician to assess the parameters of LV hypertrophy and systolic function with the start of treatment and during follow up of diabetic patients with or without associated hypertension.

CONCLUSIONS

LV fractional shortening is significantly reduced in diabetics. Hence DM is an independent risk factor for the increased LV mass and impaired systolic function regardless of association with hypertension

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