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To study left ventricular structural abnormalities in patients of diabetes mellitus with chronic kidney disease

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Abstract

Background: Macro vascular as well as micro vascular complications are the predominant cause of mortality in Diabetes Mellitus. Cardiovascular complications of diabetics largely account for the morbidity and mortality among diabetic patients. The most common cardiovascular complication of diabetics in ESRD is left ventricular (LVH) hypertrophy and it is also an independent risk factor for survival. These patients have higher proportions of abnormal LV geometry, with LV concentric remodeling and LV concentric hypertrophy as the most frequent pattern. The current study attempted to study prevalence and pattern of left ventricular hypertrophy in diabetic patients with chronic kidney disease.

Methods: Data was examined from sixty patients of 20-60 years after thoroughly examining and applying exclusion criteria. These sixty patients divided into two groups of thirty each:

Group I consisted of DM with CKD stage 3-4 (GFR > 15)

Group II consisted of DM with CKD stage 5 (GFR ≤ 15)

Echocardiography was done in all the cases. Measurements of interventricular septal wall thickness (IVSD), LV end diastolic dimension (LVEDD) and posterior wall thickness in diastole (PWTD) were recorded in accordance with the American Society of Echocardiography recommendations using M-mode. Left ventricular mass index was calculated. Left ventricular mass index (LVMI) was used to characterize LV geometry.

Result: The prevalence of various left ventricular geometry according to GFR in present study were as –in diabetic patients in Group I, the proportion of patients having concentric hypertrophy was 10%, concentric remodeling 13.3%, eccentric hypertrophy 16.7%. in Group II , the prevalence of patient having concentric hypertrophy was 43.3%, concentric remodeling 23.3%, eccentric hypertrophy 10 %. There was significant difference in proportions of various LV geometry between two groups (p = 0.006; significant).

Conclusion: This depicted that patients with severe CKD had high proportions of various LV geometry as compared to patients with moderate CKD.

Keywords: Diabetes Mellitus, ESRD, LVEDD, PWTD, LV geometry.

Introduction

In Diabetes Mellitus, Chronic hyperglycemia is a major initiator of diabetic micro- and macro complications such as retinopathy, cardiovascular disease and nephropathy. Hyperglycemia-induced mechanisms lead to vascular dysfunctions, which include increased polyol pathway flux, altered cellular redox state, increased formation of diacylglycerol (DAG), subsequent activation of protein kinase C (PKC) isoforms and accelerated non-enzymatic formation of advanced glycated end products. These mechanisms contribute to the pathophysiologic features of diabetic complications.[1] These complications largely account for the excess morbidity and mortality. Routine screening of asymptomatic patients with diabetes for retinopathy, nephropathy and neuropathy is recommended.[2]

ESRD (end stage renal disease) develops in 50% of diabetic individuals with overt nephropathy within 10 years and in more than 75% of diabetic patients by 20 years. CKD have higher rates of cardiovascular morbidity and mortality than would be predicted by Framingham models of cardiovascular risk. Once patients progress to GFR <45 ml/min, then cardiovascular disease burden is increased as compared to individuals with more preserved renal function.[3]

Left ventricular hypertrophy (LVH) is documented as the most frequent cardiac alteration in ESRD and is an independent risk factor for survival in ESRD.[4,5] Insulin resistance itself stimulates left ventricular mass growth as supported by a study of medical scientist of the medical research council of Canada.[6] Regression of LVH by pharmacological intervention is associated with an improvement inprognosis.[7,8]

In clinical practice the most reliable tool for quantifying left ventricular mass and diagnosing LVH is transthoracic echocardiography.[9] Left ventricular hypertrophy is defined as an increase in the mass of the left ventricle, which can be secondary to an increase in wall thickness, an increase in cavity size or both. LVH as a consequence of hypertension usually presents with an increase in wall thickness with or without an increase in cavity size. This increase in mass predominantly results from a chronic increase in after load of the left Ventricle.[10] To improve the clinical outcomes in End Stage Renal Disease (ESRD), it is essential to prevent LVH and its complications.[11]The prevalence of LVH increases as kidney function worsens and may be as high as 70%–80% before initiation of dialysis.[12]

Geometric patterns of left ventricular hypertrophy are very important. Left ventricular mass index (LVMI) is used to characterize LV geometry as normal geometry when LVMI is normal and relative wall thickness is <0.45, left ventricular concentric remodeling when normal LVMI is combined with RWT >0.45, concentric left ventricular hypertrophy when LVMI is increased with RWT >0.45, eccentric left ventricular hypertrophy when LVMI is increased and RWT <0.45.[13]

Patients having LV concentric remodeling have the same adverse risk as patients having concentric hypertrophy. The elevation in risk with either finding is same for both cardiovascular disease, death and is independent of the level of blood pressure.[14]

Desai et al in 2016 concluded that left ventricular (LV) mass and geometry was associated with risk of cardiovascular disease (CVD) and LV mass, geometry contributed to 10 years risk prediction for CVD in adults aged more than 65 years in the Cardiovascular Health Study.[15] Earlier detection of left ventricular hypertrophy in diabetic patients with CKD definitely improve the morbidity, mortality of patients as left ventricular hypertrophy leads to major cardiovascular complications.[16] It will improve the prognosis of patients by early detection of left ventricular hypertrophy and early management.

Materials and Methods

This cross-sectional study was conducted on sixty patients attending OPD & indoor of Guru Nanak Dev Hospital, Amritsar during period of July 2016 to July 2017. These sixty patients were having Diabetes Mellitus with chronic Kidney disease. Exclusion criteria included patients

having congenital or rheumatic valvular heart disease, patients with acute kidney injury and ischemic heart disease. After obtaining informed consent form patients, detailed history, clinical examination, echocardiography and other investigations were carried out. These sixty patients divided into two groups of thirty each:

Group I consisted of DM with CKD stage 3-4 (GFR > 15)
Group II consisted of DM with CKD stage 5 (GFR ≤ 15)

Echocardiography was done in all the cases. Measurements of interventricular septal wall thickness (IVSD), LV end diastolic dimension (LVEDD) and posterior wall thickness (PWTD) in diastole were recorded in accordance with the American Society of Echocardiography recommendations using M-mode.[17] The left ventricular mass (LVM) was calculated on Philips i E 33 machine using Devereux modified ASE cube formula and indexed for height to obtain the LVM index (LVM)·. LV diastolic diameter and posterior wall thickness simultaneously was used for reading Relative wall thickness (RWT)which was measured in study as 2 x posterior wall thickness/ LV diastolic diameter. It was considered increased when >0.45.[17] The data from present study was systematically collected, complied and statistically analyzed using software IBM SPSS 17.0 to draw relevant conclusions. Chi square and t test was used for comparing categorical variable. p value of <0.05 was considered statistically significant and p value of <0.001 was considered as highly significant.

Results

Study group comprised of total 60 diabetic patients with CKD, age ranging from 30-70 years. The mean age of study population was 55.900 years. Out of total 60 patients, 29 were males &rests 31 were females. Mean HbA1C was 7.828±0.584 in the present study.

The left ventricular hypertrophy was present in 35 patients out of total 60 patients. Mean value of left ventricular end diastolic dimension in Group I was 4.694±0.537 and mean value of left ventricular end diastolic dimension was5.218±0.472 in Group II. Mean value of inter ventricular septal wall thickness in Group I was1.010±0.356 and mean value of inter ventricular septal wall thickness was 1.296±0.272 in Group II. There was significant difference in inter ventricular septal wall thickness between Group I and II (p = 0.001; significant).

Mean value of posterior wall thickness was 0.982±0.234 in Group I and mean value of posterior wall thickness was 1.391±0.252 in Group II. There was significant difference in posterior wall thickness between Group I and II (p = 0.000; highly significant).Mean value of relative wall thickness in Group I was 0.410± 0.084 and mean value of relative wall thickness was 0.518±0.095 in Group II. There was significant difference in relative wall thickness between Group I and II (p = 0.000; highly significant).

Mean value of left ventricular mass in Group I was166.333±89.572 and mean value of left ventricular mass was 295.533±93.548 in Group II. There was significant difference in left ventricular mass between Group I and II (p = 0.000; highly significant).Mean value of left ventricular mass index in Group I was 66.200±34.296 and mean value of left ventricular mass index was 114.000±37.243 in Group II. There was significant difference in left ventricular mass index between Group I and II (p = 0.000; highly significant).

The prevalence of increased value of LVEDD was 16.7% in present study. This depicted that there was increased prevalence of high value of LVEDD in diabetic patient with CKD. The prevalence of increased value of IVSD was 43.3% in present study. This depicted that there was increased prevalence of high value of IVSD in diabetic patient with CKD. The prevalence of increased value of PWTD was 58.3% in present study. This depicted that there was increased prevalence of high value of PWTD in diabetic patient with CKD. The prevalence of increased value of RWT was 45% in present study. This depicted that there was increased prevalence of high value of RWT in diabetic patient with CKD.

Number of patients having left ventricular concentric hypertrophy, concentric remodeling, eccentric hypertrophy were 16,11,8 respectively in total sixty patients. The prevalence of various left ventricular geometry according to GFR in present study were as - in Group I, proportion of patient having concentric hypertrophy was 10%, concentric remodeling 13.3%, eccentric hypertrophy 16.7%. In Group II, prevalence of patient having concentric hypertrophy was 43.3%, concentric remodeling 23.3%, eccentric hypertrophy 10 %. This depicted that patients with severe CKD had high proportions of LVH as compared to patients with moderate CKD.

Main clinical characteristics profile of all patients

Total number	60
Sex (M/F)	29/31
Age (years; mean± SD)	55.9000 ±10.82480
LVH (Present/absent)	35/25
Hb1AC (percentage/;mean±SD)	7.8283±0.58457
LVEDD (cm; mean±SD)	4.9565±0.56678
IVSD (cm; mean±SD)	1.1533±0.34606
PWTD (cm; mean±SD)	1.1868±0.31748
RWT (ratio; mean±SD)	0.4643±0.10434
LVM (grams; mean±SD)	230.93±111.736
LVMI(g/m^2;mean±SD)	90.100± 42.904
N/concentric-remodeling/concentric-hypertrophy/eccentric-hypertrophy	25/11/16/8

Prevalence of LVH according to GFR

LVH	Group I		Group II		Total
	No.	%age	No.	% age	
Present	12	41.0	23	76.6	24
Absent	18	59.0	7	23.4	36
Total	30	100	30	100	60

Prevalence of increased LVMI according to GFR

LVMI	Group I		Group II		Total
	No.	%age	No.	% age	
Increased	8	26.7	16	53.3	24
Normal	22	73.3	14	46.7	36
Total	30	100	30	100	60

LV Goemetry according to GFR

LV Geometry	Group I		Group II		Total
	No.	%age	No.	%age	
Concentric Hypertrophy	3	10	13	43.3	16
Concentric remodeling	4	13.3	7	23.3	11
Eccentric remodeling	5	16.7	3	10	8
Normal	18	60	7	23.3	25
TOTAL	30	100	30	100	60

Discussion

In present study, left ventricular hypertrophy was present in 58.3 % of patients. This was comparable to study conducted by Foley in 2001. He followed hemodialysis patients with no prior history of cardiac disease and reported that 62% of the patients had an increased left ventricular hypertrophy.[18] The occurrence of LVH was 41% in patients of Group I and 76.6% in patients of Group II in present study. It was comparable to study conducted by Vito M Campese in 2014.[19] The prevalence of LVH was 76.6% in patients with end stage renal disease. This result was comparable to Clinical journal of the American society of Nephrology (2011) which established that the prevalence of LVH in stage 5 CKD was 70-80%.[20]

In our study, mean Left ventricular end diastolic dimension was 4.694 in Group I and 5.218 in Group II. It was comparable to a study published in cardiovascular journal of Africa in 2002 where mean LVEDD was 4.675±0.572 in patients with moderate CKD and 4.675±0.679 in patients with severe CKD. Mean value of inter ventricular septal wall thickness dimension was 1.010 in Group I and was 1.296 in Group II. This was comparable to a study published in cardiovascular journal of Africa in 2002 where mean IVSD was 0.945±.019 in patients with moderate CKD and 1.230±0.308 in patients with severe CKD.[16]

In present study, mean value of posterior wall thickness in Group I was 0.982 and was 1.391 in Group II. This was comparable to a study published in cardiovascular journal of Africa in 2002 where mean IVSD was 0.952±0.177 in patients with moderate CKD and 1.16±0.278 in patients with severe CKD. [16] In our study, mean value of relative wall thickness was 0.410 in Group I and 0.518 in Group II. This result was comparable to a study published in cardiovascular journal of Africa in which mean RWT was 0.40±0.07 in patients with moderate CKD and 0.52 ± 0.17 in patients with severe CKD.[16]

The Mean value of left ventricular mass in Group I was 166.333 and 295.533 in Group II in present study. Dormandy et al concluded that with decline in GFR, left ventricular mass was increased.[21] Our result was comparable to this study. Krishnamurthy et al in 2016 concluded that mean left ventricular mass was increased in both males and females of diabetic, hypertensive group as compared to hypertension only.[22]

The Mean value of left ventricular mass index in Group I was 66.200±34.296 and 114.000±37.243 in Group II. This result was comparable to a study in The Institute of Endocrinology and Diabetology in China (2014) were mean value of LVMI was 127.7 in patients with severe chronic kidney disease and was 65.7 in patients with moderate CKD.[22] Prospective investigation of the vasculature in uppsala seniors (PIVUS) in 2004 and uppsala longitudinal study of adult Men (ULSAM) in 1970 concluded that LVMI increased with progressive decline in renal function.[5]

The prevalence of left ventricular geometry according to GFR in present study were as, in Group I, percentage of patients having concentric hypertrophy was 10%, concentric remodeling 13.3%, eccentric hypertrophy 16.7%. In Group II, percentage of patient having concentric hypertrophy was 43.3%, concentric remodeling 23.3%, eccentric hypertrophy 10%.These results were comparable to previous study published in cardiovascular journal of Africa in 2002.[16]

In patients with severe CKD, concentric remodeling and concentric hypertrophy were the geometric patterns most frequently encountered. Earlier detection of left ventricular hypertrophy in diabetic patient with CKD improves the morbidity and mortality of patients.

Summary

The most common cardiovascular complication of diabetics with ESRD is left ventricular (LVH) hypertrophy and it is also an independent risk factor for survival. Patients with severe CKD had increased values of IVSD, LVEDD, RWT and PWTD as compared to moderate CKD. Concentric remodeling and concentric hypertrophy were the geometric patterns most frequently encountered in patients with severe CKD. Earlier detection of left ventricular

hypertrophy in diabetic patient with CKD improve the prognosis of patients by early management.

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