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A Study Of High Serum Calcium Level In Diabetes Mellitus And Its Association With Left Ventricular Hypertrophy

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Abstract:
Background*: Diabetes alters the metabolic components of the body. Because of this metabolic abnormalities, many internal structures which depend on the metabolic products are also affected. Internal organelle dysfunction finally leads to organ dysfunction and death. Hypercalcemia is one of the important complication of diabetes mellitus. Hypercalcemia occurs in diabetes due to many mechanisms including insulin resistance. The heart is damaged by many conditions like hypertension, diabetes, dyslipidemia etc.Even though hypercalcemia is not defined as a risk factor for cardiac complications in older days, many trials and studies have proved that hypercalcemia will produce severe cardiac abnormality including systolic and diastolic dysfunction based on morphological changes in the heart. The heart is damaged by many conditions like hypertension, diabetes, dyslipidemia etc.Even though hypercalcemia is not defined as a risk factor for cardiac complications in older days, many trials and studies have proved that hypercalcemia will produce severe cardiac abnormality including systolic and diastolic dysfunction based on morphological changes in the heart. Hypercalcemia produces both pressure and volume overload. This change finally progresses into ventricular dysfunction and death. These hypertrophic changes can be easily detected by echocardiography. Hypercalcemia can cause left ventricular hypertrophy by producing vascular calcification and an increase in the calcineurin pathway. By the activation of signalling pathways, they promote the growth and proliferation of cardiac muscle cells, leading to an increase in muscle mass and wall thickness. It can cause direct deposition of calcium in the heart muscle, which can cause structural changes and impair the contractility of the heart. Apart from this, hypercalcemia also induces cardiac hypertrophy by altering lipid metabolism. Hypercalcemia itself produces a dyslipidemic effect by inhibiting lipid catabolism in the initial steps. Alteration in the cytosolic calcium levels determines ventricular systolic and diastolic dysfunction.*
Materials and Methods*.*
After obtaining clearance from Institution Ethical Committee, SNMC and HSK, the study was conducted in the Medicine ward of SNMC. This study was a hospital-based case-control study. All the cases and controls which satisfy the inclusion criteria and exclusion criteria were included in the study. Informed consent was taken for participation in the study. Following investigations, Serum calcium, fasting blood glucose, PPBS, Hba1c and 2D echo was done. Serum calcium was compared between the 2 groups (10)
Results
According to our serum calcium was main Determinator of left ventricular remodeling by many mechanisms. Serum calcium was > 10.2 in LVH group people but in non-LVH group people serum calcium level was within the normal limit that was given as < 10.2 mg/dl. 1 patient in non-LVH group was having high serum calcium but does not make statistical changes in that group. The mean serum calcium of LVH group was 10.6mg/dl (10)
Conclusion:
Normal calcium mandatory for excitation-contraction coupling but high calcium adversely affect the ECC and produces ventricular dysfunction and through neurohormonal mechanism it produces cardiac muscle hypertrophy. According to this study increased serum calcium in diabetes has strong correlation with occurrence of cardiac remodeling.Hence always check the serum calcium level in diabetic patient which will predict the development of LVH. Unnecessary calcium supplementation in diabetic patients produces many adverse effects including left ventricle remodeling (10)
Key Word*: LVH, Remodelling,Diabeted mellitus*

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I. Introduction

Diabetes alters the metabolic components of the body. It produces the metabolic abnormality in both extreme like hypocalcemia as well as hypercalcemia, hyponatremia and hypernatremia. Because of this metabolic abnormalities many internal structures which depends the metabolic products are also affected. Internal organelle dysfunction finally leads the organ dysfunction and death. Hypocalcemia is one of the important complication of diabetes mellitus. It occurs when the patient develops renal insufficiency. But hypercalcemia occurs in diabetes due to many mechanisms including insulin resistance. Meanwhile hypercalcemia itself produces insulin resistance To avoid this things this study is conducted to evaluate the cardiac complication of calcium by excluding the conditions which produces hypoalbuminemia. By using the serum calcium (spot) we can able to identify the cardiac complications. Heart is damaged by many conditions like hypertension, diabetes, dyslipedemia and also hypercalcemia. Eventhough the hypercalcemia is not defined as risk factor for cardiac complication in older days, many trials and studies are proved that hypercalcemia will produce severe cardiac abnormality including systolic and diastolic dysfunction. is based on morphological changes of the heart. The morphological changes include ventricular cavity diameter, wall thickness, scarred area. So the remodeling is defined as it is the morphological changes of the heart after injury. Apart from MI many cardiac conditions are benign and some are significant. But some benign condition will progresses into significant myocardial abnormality. But a middle aged man or women develops the same hypertrophy because of hypertension or diabetes or hypercalcemia which in turn leads to hypertrophic changes finally leading to cardiac failure, arrhythmias and death. Cardiac hypertrophy will develops in many forms like concentric or eccentric.. Hypercalcemia produces both pressure and volume overload. This changes finally progresses into ventricular dysfunction and death. This hypertrophic changes can be easily detected by echocardiography. We can also predicts the left ventricular remodelling by assessing serum calcium level in diabetic patients which can be used to save the patient from cardiac failure. And many newer drugs are also identified to prevent the left ventricular remodeling. Hypercalcemia can cause left ventricular remodeling by producing vascular calcification and increases the calcineurin pathway. Apart from this way hypercalcemia also induces cardiac remodeling by altering the lipid metabolism. Hypercalcemia itself produces dyslipidemic effect by inhibiting the lipid catabolism in initial steps. Alteration in the cytosolic calcium levels determines the ventricular systolic and diastolic dysfunction. Generally 8.5- 10.2 mg/dl is considered as normal serum calcium as per standard text books. Symptoms of hypercalcemia will develops if serum calcium increases more than 12. So the level 10.2 to 12 is the asymptomatic hypercalcemic level. This study is designed to identify the association of elevated serum calcium with left ventricular remodeling in diabetic patients..

II. Material And Methods

After obtaining clearance from Institution Ethical Committee, SNMC and HSK, study will be conducted in Medicine ward of SNMC. This study will be a hospital based Case-Control study. All the cases and controls which satisfy the inclusion criteria and exclusion criteria will be included in the study.Informed consent will be taken for participation in the study.Following investigations,Serum calcium,fasting blood glucose, PPBS,Hba1c and 2D echo was done.Serum calcium was compared between the 2 groups.
Inclusion criteria :

Cases :

- Age group: 21- 80 years
- Patients with type 2 diabetes.Diabetes in this study will be defined by the american diabetes association as Either Fasting plasma glucose (FBS) of >125 mg/dl or Post prandial blood sugars at 2 hr (PPBS) >200 mg/dl
- Echo showing LVH

CONTROLS
age 21 to 80 yrs
patients with Diabetes

Exclusion criteria:
CASES
Patients with hypertension
On treatment with sulfonylurease

A h/o myocardial infarction, coronary artery bypass or angioplasty, atrial fibrillation, moderate to severe valvular heart disease, stroke or occlusive peripheral vascular disease, heart failure
A history of parathyroid disease or vitamin D related disorder. Medication history including vitamin D, bisphosphonate, estrogen replacement therapy and diuretics .

Patient with chronic liver diseases

CONTROLS
Same as above with LVH
Study Location: This was a tertiary care teaching hospital based study done in Department of General Medicine, at SNMC ,Bagalkot ,karnataka. (10)

Study Duration: march2021 to march2023.

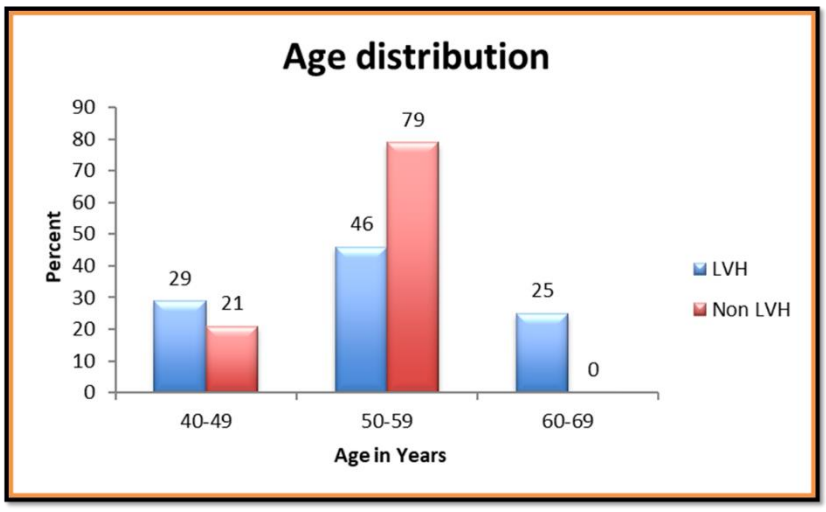
Statistical analysis (10 Bold)

Data was analyzed using SPSS version 20 (SPSS Inc., Chicago, IL). Student's *t*-test was used to ascertain the significance of differences between mean values of two continuous variables and confirmed by nonparametric Mann-Whitney test. In addition, paired *t*-test was used to determine the difference between baseline and 2 years after regarding biochemistry parameters, and this was confirmed by the Wilcoxon test which wasa nonparametric test that compares two paired groups. Chi-square and Fisher exact tests were performed to test for differences in proportions of categorical variables between two or more groups. The level $P < 0.05$ was considered as the cutoff value or significance. (10)

III. Result

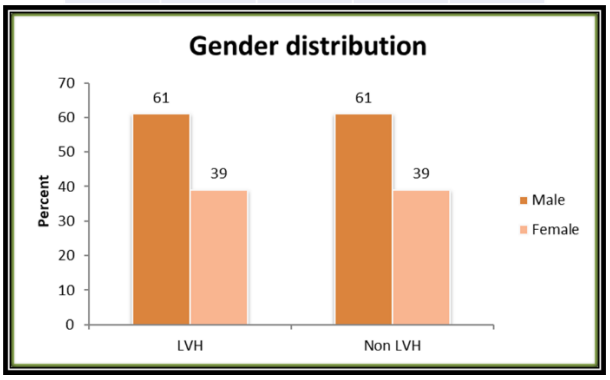
Our study has 30 cases and 30 controls all of whom were diabetic. Most of the patients were within age limit of 50 to 59 yrs.

Age	LVH		NON LVH	
	Count	%	Count	%
40-49	9	29	7	21
50-59	13	46	23	79
60-69	8	25	0	0
Total	30	100	30	100
Chi Square test P<0.009, Sig				

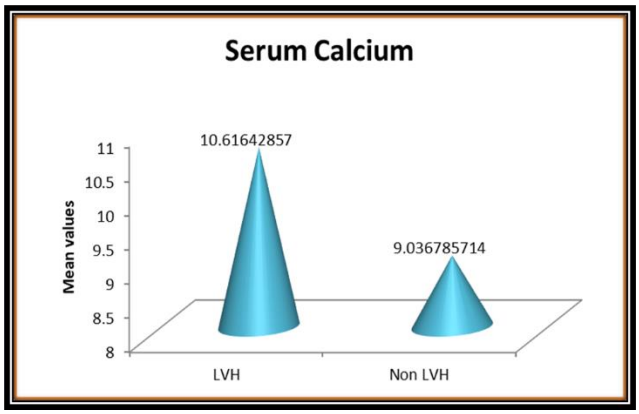


No of males are higher in both groups compared to females

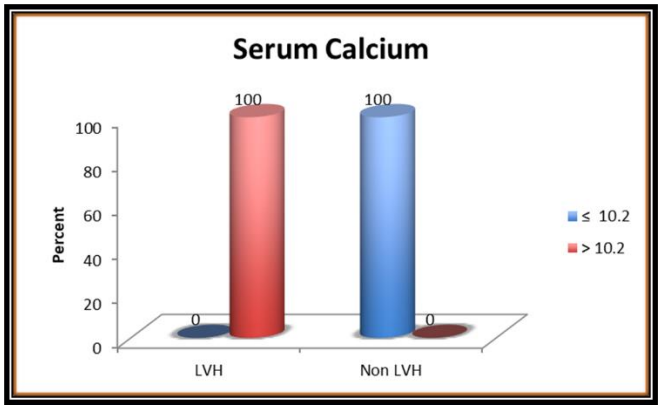
Gender	LVH		NON LVH	
	Count	%	Count	%
Male	18	61	18	61
Female	11	39	11	39
Total	30	100	30	100



The mean serum calcium value in LVH was higher than NON LVH group and it was statistically significant.



Serum hypercalcemia (>10.2) is present in the LVH group and not in NON-LVH group.



IV. Discussion

It is now clear that impaired calcium hemostasis is the key factor for cardiac hypertrophy, arrhythmias and heart failure. Other than direct calcium, dysfunction of calcium channels, and proteins also plays a role in the development of cardiac failure.

Hypercalcemia causes hypertrophy in many ways. The most important mechanism is calcium calmodulin-dependent calcineurin activation that leads translocation of NFAT protein into the nucleus.

In myocardial cells mitochondria also regulate the calcium levels in dyadic space. The calcium overload condition leads to increased myocardial calcium uptake that leads to increased oxidative stress followed by mitochondrial dysfunction and death. Myocardial calcifications because of hypercalcemia impairs ventricular relaxation and causes diastolic dysfunction

In our study calcium has a high positive predictive value for the development of cardiac muscle hypertrophy. Increased serum calcium >10.2 is the margin for the development of cardiac hypertrophySo it is clear that LVH in diabetic patients is due to increased calcium levels. Increased calcium level has a positive correlation with LVH occurrence. In our study, we checked the albumin-adjusted serum calcium levels. This study only considered 1 simple test for LVH prediction that is serum calcium which is highly cost-effective

V. Conclusion

Calcium is the important ion for cardiac and skeletal muscle contraction, maintain blood coagulation and bone mineralization.

Increased serum calcium will produce insulin resistance and vise versa.

Serum calcium also increases the diabetic prevalence by interacting with GLUT 4 receptors.

Normal calcium is mandatory for excitation contraction coupling(ECC) but high calcium adversely affects the ECC and produces ventricular dysfunction and through neurohormonal mechanism, it produces cardiac muscle hypertrophy.

According to this study increased serum calcium in diabetes has strong correlation with occurrence of left ventricular hypertrophy.

Hence always check the serum calcium level in diabetic patient which will predict the development of LVH.

Unnecessary calcium supplementation in diabetic patients will produce many adverse effects including left ventricle hypertrophy

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