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RESEARCH ARTICLE

Relationship of Glycemic Control with Kidney Function Tests and Lipid Profile in Type 2 Diabetes Mellitus Patients in a Tertiary Care Hospital

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Abstract: ***Background:*** Diabetes causes both microvascular complications, which comprise neuropathy, nephropathy and retinopathy and macrovascular complications, which comprise cardiovascular disease, stroke, and peripheral vascular disease. The aim of this prospective research study is to investigate the relationship between blood glucose levels, kidney function, and lipid profile in patients diagnosed with type 2 diabetes mellitus (T2DM). The variations in blood glucose levels significantly influence renal function and lipid metabolism in individuals with T2DM. ***Material and Methods:*** This study was conducted at Santosh Medical College and Hospital, Ghaziabad, Uttar Pradesh. 125 pre-diagnosed type 2 diabetic patients were taken as the study group and the same number of healthy individuals were taken as the control group. Inclusion criteria will include a confirmed diagnosis of Type 2 diabetes mellitus, 18 to 70 years of age, and willingness to participate in the study. Fasting and postprandial blood sugar, glycated haemoglobin (HbA1c), serum creatinine, urine microalbumin, T.Chol., TG, HDL, LDL and VLDL were estimated in both groups. ***Result:*** In our study, we found that there is a significant (p-value = 0.0001) rise in FBS, PPBS and HbA1c in the study group as compared to the control group. Serum creatinine and urine microalbumin in the study group were found to be significantly (p-value = 0.0001) elevated in the study group as compared to the control group. In Lipid Profile parameters, Total cholesterol, triglycerides, LDL and VLDL were significantly (p-value = 0.0001) higher in the study group as compared to the control group but there was no significant (p-value = 0.0361) difference in HDL cholesterol level between these two groups. ***Conclusion:*** A significant association of HbA1c with urine microalbumin and serum creatinine indicates that impaired blood glucose level causes nephropathy. This emphasizes the need for early detection of urine microalbuminuria in type 2 diabetic patients. Also, elevated blood glucose causes dyslipidemia which may cause cardiovascular disorders. The study suggests the need for effective diabetes control and the prevention of associated renal damage and cardiovascular risk factors. These findings may have implications for optimizing diabetes management strategies, including the importance of glycemic control in preserving kidney health and managing cardiovascular risk factors in this population.

Keywords: Type 2 diabetes; Glycemic control; Renal profile; Lipid parameters.

INTRODUCTION

Hyperglycemia, or increased blood glucose levels, is a hallmark of diabetes mellitus, which is described as "a group of metabolic diseases characterized by defects in insulin secretion, insulin action, or both." Type 1 diabetes is caused by an autoimmune-mediated destruction of beta cells of pancreas, causing insulin insufficiency and hyperglycemia; type 2 diabetes is caused by a diminished responsiveness to insulin (insulin resistance) and reduced insulin production by beta cells.(1)

Globally, the incidence of type 2 diabetes mellitus (T2DM) is rising at an alarmingly high rate, impacting the economy, living standards, public health, and healthcare system.(2)The International Diabetes Federation (IDF) found that 589 million adults with age between 20-79 years worldwide were suffering from diabetes in 2024; by 2050, that number is supposed to increase to 852.5 million.In India 89.8 milllion people are suffering from diabetes in 2024 and it expected to rise to 156.7 million by 2050.(3)

Elevated blood glucose levels brought on by a gradual impairment in insulin secretion or tissue resistance to insulin are the hallmarks of T2DM which is a complex, multisystemic metabolic disease. Insulin resistance and beta-cell dysfunction vary in severity in T2DM which is a widespread and diverse condition. A strong link exists between obesity and T2DM, mediated by central nervous system pathways. These pathways integrate data from the environment and peripheral organs to regulate appetite and energy expenditure.(4)

Diabetes results in macrovascular problems including peripheral vascular disease, cardiovascular disease, and stroke as well as microvascular problems like neuropathy, nephropathy, and retinopathy.(5)Anatomical, structural, and functional abnormalities that result in multiorgan dysfunction are among the vascular modifications linked to diabetes.(6)

The etiologic features of diabetic macrovascular (including big vessels, like arteries and veins) and microvascular (involving tiny vessels, like capillaries) abnormalities are similar. Through a variety of metabolic and structural abnormalities, such as the synthesis of AGE, aberrant initiation of signaling cascades (like PKC), increased synthesis of ROS, and abnormal activation of hemodynamic regulation systems (like RAS), chronic hyperglycemia plays a major part in the onset of vascular complications of diabetes.(7)

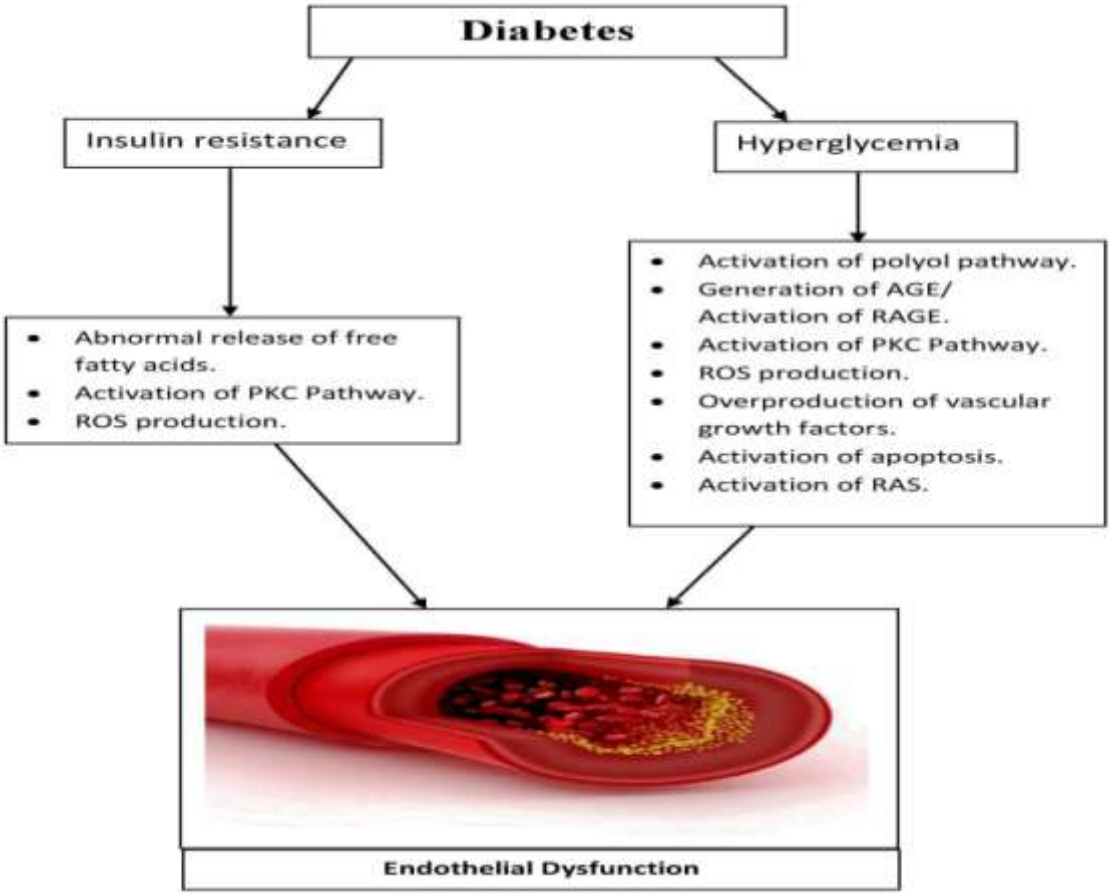

Fig 1: mechanisms for diabetes-associated endothelial dysfunction.

T1DM and T2DM lead to the development of diabetic nephropathy (DN) which is a critical and advancing complication of diabetes. DN being the main cause of ESRD, usually starts as microalbuminuria, which progress into overt albuminuria and finally renal failure. About 25% of type 2 diabetic patients suffer from microalbuminuria or a more severe stage of DN that deteriorate at a speed of 2% to 3% annually. Additional defining characteristics of DN include glomerular hyperfiltration and thickening of glomerular basement membranes, which cause mesangial extracellular matrix expansion and further increased excretion of albumin in urine, which progresses to renal failure and glomerular and tubular sclerosis. The risk factors for DN are obesity, hypertension, tobacco use, dyslipidemia, age of onset, duration of diabetes, and hyperglycemia.(7) Diabetic patients are at risk for the development of dyslipidemia and Diabetic nephropathy. Hyperglycemia causes lipid derangement and nephropathy. Elevated levels of serum cholesterol, triglycerides and lipoproteins are the main lipid derangements in diabetes which are common risk factors for coronary artery disease. It is found that elevated blood glucose levels and duration of diabetes are responsible for nephropathy and microalbuminuria.(8)

MATERIALS AND METHODS

A Case-control study was planned to analyze the Relationship of Glycemic control with Kidney function tests and Lipid profile in Type 2 diabetes mellitus Patients (T2DM). This study was conducted on patients attending the OPD of the Department of General Medicine and the Department of Biochemistry of Santosh Medical College & Hospital, Ghaziabad, Uttar Pradesh. 125 pre-diagnosed type 2 diabetic patients were taken as the study group and the same number of healthy individuals were taken as the control group. Inclusion criteria will include a confirmed diagnosis of T2DM, 18

to 70 years of age, and willingness to participate in the study. Before registering for the study, informed written consent was taken from the participants, expressing their willingness to participate in the study. Individuals suffering from Type 1 Diabetes mellitus, hemochromatosis, chronic kidney disease, chronic liver disease, thyroid disorders, malignancies and patients taking lipid-lowering drugs were excluded from the study.

5 ml blood sample was collected from each subject after overnight fasting and transferred into EDTA (for glycated hemoglobin), sodium fluoride and potassium oxalate tube (for FBS) and plain tube (for T. Chol., TG, HDL and creatinine). 2 ml postprandial sample was collected in sodium fluoride and potassium oxalate tube (for PPBS). Spot urine sample was also collected from each subject for urine microalbumin estimation.
SPSS software was used for statistical analysis. Mean ± Standard deviation, p-value and r – value were calculated. p-value ≤ 0.05 is considered significant and r – value ≥ 0.5 is considered significantly correlated.

RESULTS AND OBSERVATIONS:

Table 1: Comparision of Blood sugar, HbA1c, S. Creatinine, T. Chol, HDL, TG, LDL, VLDL and Urine Microalbumin between study and control group

Parameter	Study group	Control group	p value
FBS (mg/dl)	163.53±69.28	87.60±10.52	0.0001
PPBS (mg/dl)	234.15±98.95	118.18±13.52	0.0001
HbA1c (%)	8.00±1.26	4.84±0.38	0.0001
S. Creatinine (mg/dl)	1.45±0.64	0.80±0.2	0.0001
T. Cholesterol (mg/dl)	210.65±40.80	142.51±24.52	0.0001
HDL (mg/dl)	44.68±5.52	46.05±4.77	0.0361
TG (mg/dl)	200.36±67.15	126.03±27.29	0.0001
LDL (mg/dl)	125.90±35.18	71.25±23.10	0.0001
VLDL(mg/dl)	40.07±13.42	25.20±5.46	0.0001
Urine Microalbumin (mg/24hr)	35.50±11.52	15.87±3.85	0.0001

Table 1 shows average blood glucose, HbA1C, serum creatinine, Total cholesterol (T. Chol.), high density lipoproteins (HDL) and triglycerides (TG) and urine microalbumin levels both in the diabetic patients (the study group) and the control group. The T2DM subjects presented significantly higher concentrations of FBS, PPBS, HbA1c, serum creatinine, T. Chol., TG and urine microalbumin ($p<0.05$) as compared to the control subjects. But there is insignificant higher ($p>0.05$) level of HDL in the control group as compared to the study group.

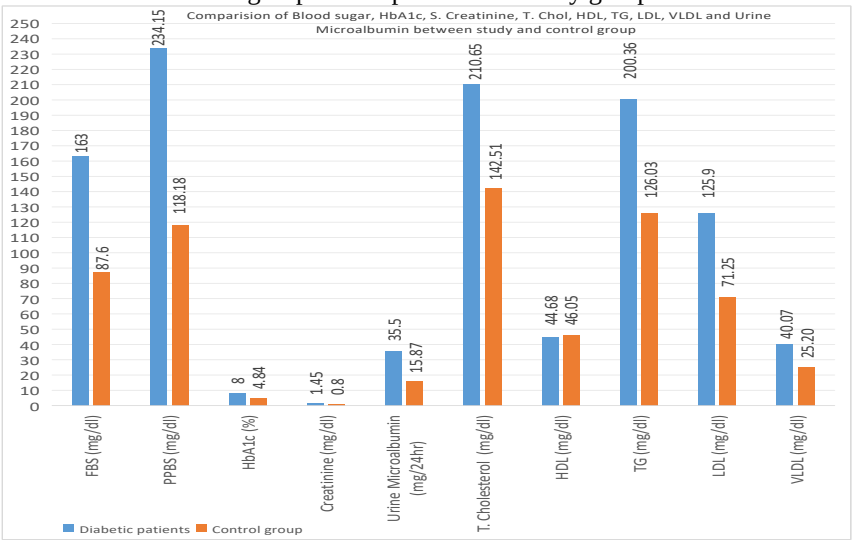

Chart 1: Shows the comparisons of Comparision of Blood sugar, HbA1c, S. Creatinine, T. Chol, HDL, TG, LDL, VLDL and Urine Microalbumin between study and control group.

Table 2: Correlation of HbA1c with S. Creatinine, Urine Microalbumin, T. Chol, HDL, TG, LDL and VLDL in study group

Study group		r value
HbA1c	Serum Creatinine	0.480150406
	Urine microalbumin	0.560574465
	T. cholesterol	0.681446997
	HDL	-0.11896668
	TG	0.728454744
	LDL	0.530812046
	VLDL	0.728454744

There is (r > 0.5) significant positive correlation between HbA1c level and urine microalbumin, Total cholesterol (T. Chol.) and triglycerides (TG) in T2DM subjects. There is a moderate correlation between HbA1c level and serum creatinine (r = 0.48) in T2DM subjects. However there is no correlation between HbA1c and HDL in the same group.

Table 3: Correlation of HbA1c with S. Creatinine, Urine Microalbumin, T. Chol, HDL, TG, LDL and VLDL in control group

Control group		r value
HbA1c	Serum creatinine	0.25245638
	Urine microalbumin	0.216060412
	T. cholesterol	0.234952021
	HDL	-0.28593647
	TG	0.073688756
	LDL	0.291055547
	VLDL	0.073688756

There is no significant correlation between HbA1c level and serum creatinine, urine microalbumin, T. Chol., HDL, TG and LDL in the control group.

DISCUSSION

In the study group our results show a significant positive correlation of HbA1c with TG (r=0.728) and total cholesterol (r=0.681). Arshad Hussain et al in their study in 2015 on 401 type 2 diabetes patients and Houda et al in their study conducted between 2017 and 2018 found a similar result to the present study.(9,10) Higher HbA1c levels have been linked to dyslipidemia, which is represented by raised LDL cholesterol and triglyceride levels. The lipid profile may deranged as a result of poor glycaemic control's impact on lipid metabolism. Moreover, hyperglycemia can exacerbate inflammation and oxidative stress, which can lead to dyslipidemia.(11,12) The hydrolysis of triglyceride-rich lipoproteins is greatly hampered by the strong inhibition of lipoprotein lipase synthesis that occurs due to diminished insulin activity. This result in rise in Triglyceride-rich lipoproteins level in blood and delayed removal of chylomicron and VLDL from blood. Another consequence of insulinopenia is a significant rise in lipolysis in adipose tissue that results in the influx of free fatty acids into the bloodstream. Increased production and release of VLDL are all results of enhanced TG synthesis in the liver which is a consequence of a higher fatty acid supply to the liver. In type 2 diabetic individuals, a decline in glycemic control will exacerbate their underlying dyslipidemia, resulting in more marked increases in blood triglyceride levels. If fresh VLDL is generated at a significantly higher rate, LDL levels may increase subsequently.(13)Compared to other lipid abnormalities, hypertriglyceridemia is more prevalent in

2 diabetes mellitus patients.(14)However Alzahrani et al in their study found that HbA1c has no correlation with total cholesterol.(2) In a study conducted in 2017 it was suggested that insulin resistance has a major part in the development of diabetic dyslipidemia.(9)

Our study also showed a significant correlation of HbA1C with urine microalbumin (r=0.56) and serum creatinine (r=0.48). Similar results were found by Khadka et al.(15) C. C. Hsu et al in their study concluded that higher HbA1c is associated with the development of microalbuminuria in type 2 diabetes patients.(16) One of the main causes of diabetic nephropathy (DN) is persistent hyperglycemia. The polyol and sorbitol pathways, among other collateral metabolic processes, are stimulated in the presence of elevated blood glucose.(17,18) Metabolic imbalance brought on by excessive collateral metabolism results in biochemical abnormalities in important tissues, including the kidney, which alter structure and function irreversibly. Glomerular endothelial cells (GECs), mesangial cells (MC), podocytes, and endothelial cells are the primary cellular targets of hyperglycemia injury in DN. DN is frequently called a "podocyte centrality" disease because of the high apoptotic susceptibility of highly specialized podocytes, which raises glomerular permeability.(18)

Advanced glycation end products (AGE), which are also implicated in the pathophysiology of DN, can accumulate when blood glucose levels are elevated.(19) AGE is synthesized from the spontaneous reaction between glucose and proteins predominantly on long-lived structural proteins.(20) Nuclear factor κB (NF-κB) and PKC are two transcription factors linked to the development of DN that are expressed and activated as a result of AGE. Both direct (via AGE receptors) and indirect (by the creation of free oxygen radicals, which results in the production of cytokines, adhesion molecules, and chemokines) effects are possible.(21,22)

CONCLUSIONN

The relationship between glycemic control, kidney function tests, and lipid profiles in Type 2 diabetes mellitus (T2DM) patients are multifaceted and crucial for managing the conditions effectively. A significant association of HbA1c with urine microalbumin and serum creatinine indicate that impaired blood glucose level causes nephropathy. This emphasizes the need for early detection of urine microalbuminuria in type 2 diabetic patients. Also elevated blood glucose causes dyslipidemia which may cause cardiovascular disorders. The study suggests the need for effective diabetes control and the prevention of associated renal damage cardiovascular risk factors. These findings may have implications for optimizing diabetes management strategies, including the importance of glycemic control in preserving kidney health and managing cardiovascular risk factors in this population.

Abbreviation: T. Chol. =Total cholesterol, TG= triglycerides, HDL=high density lipoproteins, LDL= low density lipoproteins, VLDL= very low density lipoproteins, DN=diabetic nephropathy, RAS=renin-angiotensin system, AGE=advanced glycation end products, FFA=free fatty acid, RAGE= receptors for AGE, ROS=reactive oxygen species, PKC=protein kinase C, ESRD= end-stage renal disease.

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