E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.48047/ijprt/15.02.566

Research Article

Correlation Between Glycemic Control and Microalbuminuria in Type 2 Diabetic Patients.

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Received: - 30/11/2025 ***Accepted: - 20/12/2025*** ***Published: - 27/12/2025***

ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycemia, which can lead to microvascular complications such as diabetic nephropathy. Microalbuminuria, the early excretion of albumin in urine, is a sensitive marker of kidney damage and a predictor of progression to overt nephropathy and cardiovascular disease. correlation between glycemic control and microalbuminuria in T2DM patients is essential for early detection, timely intervention, and preservation of kidney function.

Material and Methods: A case-control study was carried out in the Biochemistry Department of Nootan Medical College to assess renal biomarkers in T2DM patients. The study involved 125 T2DM patients and 125 healthy controls, with ethical approval and informed consent obtained. Participants aged 18–70 years with confirmed T2DM were included, while major systemic illnesses and certain drug use were excluded. Fasting, postprandial blood, and urine samples were collected. Data were analyzed using SPSS, with $p \leq 0.05$ considered significant and $r \geq 0.5$ indicating strong correlation.

Result: Our study showed a significant increase ($p = 0.0001$) in FBS, PPBS, and HbA1c in the diabetic group compared to controls. Serum creatinine and urine microalbumin were also markedly higher ($p = 0.0001$) in the study group, indicating impaired renal function.

Conclusion: Early microalbuminuria screening and maintaining optimal HbA1c levels are essential for protecting kidney function and improving diabetes management.

Keywords: Type 2 Diabetes, Glycemic Control, Microalbuminuria,

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]

An elevated blood glucose, is the hallmark of diabetes mellitus, is a group of metabolic disorders resulting from impaired insulin secretion, insulin action, or both. In type 1 diabetes, autoimmune destruction of pancreatic β-cells leads to insulin deficiency and persistent hyperglycemia, whereas type 2 diabetes arises from insulin resistance combined with a gradual decline in β-cell function [2]. Worldwide, the prevalence of type 2 diabetes mellitus (T2DM) is increasing at a concerning pace, with significant implications for economies, living standards, public health, and healthcare infrastructure. According to the International Diabetes Federation (IDF), about 589 million adults aged 20–79 was living with diabetes in 2024; this number is projected to rise to approximately 852.5 million by 2050. In India alone, an estimated 89.8 million individuals had diabetes in 2024, a figure expected to hike to 156.7 million by 2050 [3,4].

Diabetes leads to a range of vascular complications including microvascular including neuropathy, nephropathy, and retinopathy and macrovascular conditions such as cardiovascular disease, stroke, and peripheral vascular disease. These vascular changes are associated with anatomical, structural, and functional alterations that contribute to dysfunction across multiple organ systems [5,6].

Both type 1 and type 2 diabetes can lead to diabetic nephropathy (DN), a serious and progressive renal complication. DN is the leading cause of end-stage renal disease (ESRD). It typically begins with microalbuminuria, which gradually advances to overt proteinuria and ultimately results in kidney failure. Approximately 25% of individuals with type 2 diabetes develop microalbuminuria or a more advanced stage of DN, with deterioration occurring at an annual rate of about 2–3%. Characteristic pathological features of DN include glomerular hyperfiltration, thickening of the glomerular basement membrane, and expansion of the mesangial extracellular matrix. These structural changes increase urinary albumin loss and eventually lead to renal failure, accompanied by glomerulosclerosis and tubular damage. Risk factors contributing to DN include obesity, hypertension, smoking, dyslipidemia, older age at onset, longer disease duration, and poor glycemic control. Diabetic individuals are particularly prone to dyslipidemia and DN [7,8].

Monitoring kidney function in diabetic patients is crucial for early detection and prevention of renal deterioration. Renal biomarkers such as serum creatinine, estimated glomerular filtration rate (eGFR), blood urea nitrogen (BUN), and urinary albumin excretion provide valuable insights into the structural and functional status of the kidneys. These biomarkers are known to be influenced by glycemic levels, making the assessment of their correlation essential for understanding the progression of diabetic nephropathy. Glycated hemoglobin (HbA1c) serves as a reliable indicator of long-term glycemic control and reflects the average plasma glucose concentration over the preceding 2–3 months. Identifying the relationship between HbA1c levels and renal biomarkers can help clinicians predict early renal involvement, tailor therapeutic

strategies, and improve patient outcomes [9,10].

This study aims to evaluate the correlation between glycemic control and renal biomarkers in patients with T2DM.

Material and Method

A case-control study was carried out in the Biochemistry Department of Nootan Medical College and Research Centre to assess renal biomarkers in individuals with T2DM. The study enrolled 125 confirmed T2DM patients and an equal number of healthy controls from the General Medicine OPD at Nootan General Hospital, Visnagar. Ethical clearance and written informed consent were obtained prior to participation. 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.

Blood sample collection: 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 (serum 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 was calculated. p-value ≤ 0.05 is considered significant and r – value ≥ 0.5 is considered significantly correlated

Result

Table 1: Comparison of Blood sugar, HbA1c, S. Creatinine 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
Urine Microalbumin (mg/24hr)	35.50±11.52	15.87±3.85	0.0001

Table 1 shows average blood glucose, HbA1C, serum creatinine, 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 and urine microalbumin ($p<0.05$) as compared to the control subjects.

Table 2: Correlation of HbA1c with S. Creatinine, Urine Microalbumin in study group

Study group		r value
HbA1c	Serum Creatinine	0.48
	Urine microalbumin	0.56
	T. cholesterol	0.68

There is ($r > 0.5$) significant positive correlation between HbA1c level and urine microalbumin. There is a moderate correlation between HbA1c level and serum creatinine ($r = 0.48$) in T2DM subjects.

Table 3: Correlation of HbA1c with S. Creatinine, Urine Microalbumin in control group

Control group		r value
HbA1c	Serum creatinine	0.25
	Urine microalbumin	0.21

There is no significant correlation between HbA1c level and serum creatinine, urine microalbumin in the control group.

Discussion

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.

The present 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.[11] C. C. Hsu et al in their study concluded that higher HbA1c is associated with the development of microalbuminuria in type 2 diabetes patients.[12] Persistent hyperglycemia is a key driving factor in the development of diabetic nephropathy (DN). Elevated blood glucose activates alternative metabolic pathways, such as the polyol and sorbitol pathways, leading to metabolic disturbances. This excessive metabolic activity causes biochemical changes in vital tissues including the kidneys, resulting in irreversible structural and functional damage. In DN, glomerular endothelial cells, mesangial cells, podocytes, and other endothelial cells are particularly vulnerable to hyperglycemia-induced injury. Because podocytes are highly specialized and prone to apoptosis, DN is often described as a "podocyte-centered" disorder, reflecting their critical

role in increasing glomerular permeability and advancing disease progression [13,14].

Advanced glycation end products (AGEs), which play a significant role in the development of diabetic nephropathy, accumulate under conditions of chronic hyperglycemia. These AGEs form through a non-enzymatic reaction between glucose and proteins, particularly those that are long-lived structural components [15,16]. Advanced glycation end products (AGEs) activate key transcription factors such as nuclear factor κB (NF-κB) and protein kinase C (PKC), both of which contribute to the progression of diabetic nephropathy. Their activation occurs through direct binding of AGEs to their receptors as well as indirectly through increased production of reactive oxygen species, which in turn stimulates the release of cytokines, adhesion molecules, and chemokines [17,18].

CONCLUSION

The connection between glycemic control and renal biomarkers in patients with Type 2 diabetes mellitus (T2DM) is complex yet vital for effective disease management. A strong correlation between HbA1c levels and indicators such as urine microalbumin and serum creatinine suggests that poor glycemic regulation contributes to the onset and progression of diabetic nephropathy. This underscores the importance of early screening for microalbuminuria in T2DM patients. Such insights highlight the critical role of maintaining optimal blood glucose levels to protect kidney function and improve overall diabetes care.

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