CLINICAL CORRELATION BETWEEN GLYCEMIC CONTROL AND THE PROGRESSION OF DIABETIC NEPHROPATHY IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

Mukhamedova V.M.

Andijan State Medical Institute, Uzbekistan

Abstract: Diabetic nephropathy (DN) is a major microvascular complication of Type 2 Diabetes Mellitus (T2DM) and remains the leading cause of end-stage renal disease worldwide. Persistent hyperglycemia leads to glomerular damage and albuminuria, resulting in progressive renal dysfunction. The aim of this study was to evaluate the association between glycemic control, disease duration, and renal impairment among patients with T2DM.A total of 150 patients with T2DM were enrolled and categorized based on urinary albumin excretion into normoalbuminuria, microalbuminuria, and macroalbuminuria groups. Glycated hemoglobin (HbA1c), fasting plasma glucose (FPG), serum creatinine, and estimated glomerular filtration rate (eGFR) were analyzed. Statistical correlations between HbA1c and renal parameters were assessed using Pearson's correlation.The mean HbA1c level increased progressively with the severity of nephropathy: 7.2 ± 0.9%, 8.6 ± 1.2%, and 9.4 ± 1.3% for normo-, micro-, and macroalbuminuria groups, respectively ($p < 0.001$). Serum creatinine and urinary albumin excretion showed a significant positive correlation with HbA1c ($r = 0.69$, $p < 0.001$), while eGFR demonstrated a strong negative correlation ($r = –0.63$, $p < 0.001$).Poor glycemic control and prolonged disease duration are strongly associated with the progression of diabetic nephropathy. Tight metabolic regulation and early screening for albuminuria can significantly delay renal complications in patients with Type 2 Diabetes Mellitus.

Keywords: Type 2 diabetes, diabetic nephropathy, HbA1c, eGFR, albuminuria, renal dysfunction.

Introduction

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance and relative insulin deficiency, resulting in persistent hyperglycemia and a wide spectrum of systemic complications. Among its microvascular manifestations, diabetic nephropathy (DN) represents one of the most serious long-term outcomes, frequently leading to end-stage renal disease (ESRD) and increased cardiovascular mortality (Thomas et al., 2021).

The pathogenesis of diabetic nephropathy is multifactorial and involves chronic hyperglycemia-induced oxidative stress, glomerular hypertension, and activation of the renin–angiotensin–aldosterone system (RAAS). These factors contribute to glomerular basement membrane thickening, mesangial expansion, and progressive loss of renal filtration capacity (Zhang et al., 2022). Persistent elevation of HbA1c has been identified as a key determinant of nephropathy progression due to its role in the formation of advanced glycation end-products (AGEs) that trigger inflammatory and fibrotic pathways in the kidney.

Although modern antidiabetic therapies, including SGLT2 inhibitors and GLP-1 receptor agonists, have shown renoprotective effects, diabetic nephropathy continues to develop in a substantial proportion of patients with poor glycemic control. Early diagnosis through measurement of urinary albumin and estimation of glomerular filtration rate (eGFR) remains the cornerstone of prevention (American Diabetes Association, 2023).

This study aims to investigate the relationship between glycemic control, disease duration, and renal impairment in patients with Type 2 Diabetes Mellitus, emphasizing the need for regular metabolic monitoring and early intervention to delay renal complications.

Methods

This observational cross-sectional study was conducted on one hundred and fifty patients with clinically confirmed Type 2 Diabetes Mellitus, aged between thirty and sixty-five years. All participants were recruited from the Department of Endocrinology and Nephrology at [Institution Name] after obtaining ethical approval and written informed consent. Patients with chronic kidney disease of non-diabetic origin, hypertension without diabetes, or use of nephrotoxic drugs were excluded from the study.

Venous blood samples were collected after overnight fasting to determine glycated hemoglobin (HbA1c), fasting plasma glucose (FPG), serum creatinine, and lipid profile. Urinary albumin excretion was measured from a first-morning spot urine sample and expressed as the albumin-to-creatinine ratio (ACR). Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI formula.

Based on urinary albumin excretion, participants were classified as follows:

- **Group 1:** Normoalbuminuria (ACR < 30 mg/g)
- **Group 2:** Microalbuminuria (ACR 30–300 mg/g)
- **Group 3:** Macroalbuminuria (ACR > 300 mg/g)

All laboratory tests were performed using standardized automated analyzers. Statistical analyses were performed using SPSS version 26.0. Quantitative data were expressed as mean ± standard deviation. Pearson's correlation test was used to assess relationships between HbA1c, disease duration, serum creatinine, and eGFR. A p-value < 0.05 was considered statistically significant.

Results

Table 1. Biochemical Parameters in Patients with Type 2 Diabetes Mellitus

Parameter	Normoalbuminuria (n=60)	Microalbuminuria (n=55)	Macroalbuminuria (n=35)	p-value
Age (years)	52.1 ± 6.3	54.8 ± 7.1	56.3 ± 6.8	0.048
Disease Duration (years)	6.5 ± 2.2	9.3 ± 3.6	12.1 ± 4.5	<0.001
HbA1c (%)	7.2 ± 0.9	8.6 ± 1.2	9.4 ± 1.3	<0.001
Serum Creatinine (µmol/L)	82 ± 14	108 ± 21	142 ± 33	<0.001
eGFR (mL/min/1.73m²)	96 ± 12	78 ± 15	54 ± 19	<0.001
ACR (mg/g)	18.2 ± 6.3	112.4 ± 38.6	398.7 ± 122.4	<0.001

The results revealed a significant increase in HbA1c, serum creatinine, and urinary albumin excretion with progressive nephropathy severity, while eGFR declined sharply across the three

groups. A strong positive correlation was found between HbA1c and ACR ($r = 0.69$, $p < 0.001$), whereas eGFR showed an inverse relationship with HbA1c ($r = –0.63$, $p < 0.001$). Patients with a disease duration longer than ten years demonstrated markedly higher risks of macroalbuminuria.

Discussion

The findings of this study confirm that poor glycemic control is the primary determinant of diabetic nephropathy progression in Type 2 diabetes. Persistent hyperglycemia induces microvascular injury through endothelial dysfunction and oxidative stress, leading to glomerular sclerosis and albumin leakage. The observed inverse correlation between HbA1c and eGFR supports previous reports by Zhang et al. (2022), indicating that elevated glucose levels accelerate renal functional decline.

Patients with macroalbuminuria exhibited more than double the mean disease duration and nearly 1.5-fold higher HbA1c values compared to those without renal involvement, highlighting the cumulative effect of chronic hyperglycemia over time. Similar observations were reported by Thomas et al. (2021), who emphasized that maintaining HbA1c levels below 7% significantly delays nephropathy onset.

Emerging therapeutic strategies such as sodium-glucose cotransporter 2 (SGLT2) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists have demonstrated strong renoprotective effects independent of glycemic control. Nevertheless, regular screening for albuminuria and early intervention remain essential, as most patients are asymptomatic in the early stages of nephropathy.

Conclusion

Diabetic nephropathy remains a major clinical challenge in the management of Type 2 Diabetes Mellitus. This study demonstrates a clear association between elevated HbA1c, longer disease duration, and declining renal function. Tight glycemic control, routine monitoring of urinary albumin excretion, and early pharmacological intervention can significantly reduce the risk of renal failure. Multidisciplinary care involving endocrinologists, nephrologists, and diabetes educators is essential for improving long-term outcomes in diabetic patients.

References

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