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Original Research Article

Evaluation of Renal Complications in Uncontrolled Type 2 Diabetes Mellitus: A Tertiary-Care Cross-sectional Perspective

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Abstract:

Background: Diabetic nephropathy (DN) is one of the most serious microvascular complications of type 2 diabetes mellitus (T2DM), leading to end-stage renal disease (ESRD) and increased cardiovascular risk. In India, delayed diagnosis and poor glycemic control significantly contribute to early-onset renal deterioration. This study aimed to assess the pattern and severity of diabetic nephropathy in patients with poorly controlled T2DM and identify associated clinical factors.

Methods: This cross-sectional study was conducted over a 12-months period from March 2024 to February 2025 at Dr. BSK Institute of Medical Sciences, Kanpur. A total of 140 patients with known T2DM and poor glycemic control (HbA1c ≥8%) were enrolled. All patients underwent clinical evaluation, biochemical testing including serum creatinine, estimated glomerular filtration rate (eGFR), and urinary albumin-to-creatinine ratio (UACR). Staging of diabetic nephropathy was based on KDIGO guidelines.

Results: Among the 140 patients, 64 (45.7%) had microalbuminuria and 32 (22.9%) had overt proteinuria, indicating progression of nephropathy. Stage 1 and 2 DN was present in 36.4% of patients, stage 3 in 42.1%, and stage 4 or higher in 21.4%. Duration of diabetes >10 years, presence of hypertension, and HbA1c ≥10% were significantly associated with advanced nephropathy stages ($p<0.01$). Additionally, 28.6% of patients with macroalbuminuria had elevated serum creatinine levels (>1.5 mg/dL), while 14.3% had eGFR <30 mL/min/1.73 m^2.

Conclusion: A substantial proportion of patients with uncontrolled type 2 diabetes mellitus exhibit progressive stages of diabetic nephropathy. Poor glycemic control, prolonged disease duration, and coexisting hypertension significantly contribute to renal function decline. Regular screening using UACR and eGFR is essential for early detection and intervention to prevent irreversible renal damage in diabetic patients.

Keywords: Diabetic nephropathy, Type 2 diabetes mellitus, Albuminuria, eGFR, Proteinuria, Glycemic control, chronic kidney disease.

Introduction

Diabetes mellitus, particularly type 2 diabetes mellitus (T2DM), has emerged as a major global health concern, with India being home to one of the largest diabetic populations in the world. Among the numerous complications arising from uncontrolled diabetes, diabetic nephropathy (DN) stands out as the leading cause of chronic kidney disease (CKD) and end-stage renal disease (ESRD) worldwide [1]. It is estimated that up to 30–40% of patients with T2DM will eventually develop some degree of nephropathy, with a significant proportion progressing silently to irreversible renal impairment if not detected early [2].

Diabetic nephropathy is a progressive microvascular complication characterized initially by hyperfiltration, followed by increasing urinary albumin excretion, a decline in glomerular filtration rate (GFR), and ultimately the development of ESRD [3]. The earliest clinical indicator of nephropathy is microalbuminuria, defined by urinary albumin excretion of 30–300 mg/day. If left unchecked, this progresses to overt proteinuria and a decline in renal function measurable by rising serum creatinine and decreasing estimated glomerular filtration rate (eGFR). Unfortunately, these pathological changes often occur asymptomatically until significant renal damage has occurred [4].

The pathogenesis of diabetic nephropathy is multifactorial and involves chronic hyperglycemia, systemic hypertension, oxidative stress, activation of the renin–angiotensin–aldosterone system (RAAS), and genetic predisposition. Among these, persistent hyperglycemia remains the principal modifiable fac-

tor [5]. Hemoglobin A1c (HbA1c) levels are a validated measure of long-term glycemic control, and elevated values (typically ≥8%) are closely linked with accelerated microvascular damage. Prolonged duration of diabetes and coexisting hypertension further compound the risk and severity of nephropathy [6].

In India, the challenge is magnified by delayed diagnosis, inadequate routine screening, financial constraints, and inconsistent follow-up, especially in semi-urban and rural populations. Despite the known burden, many patients with poorly controlled diabetes are not regularly monitored for nephropathy markers such as urinary albumin-to-creatinine ratio (UACR) and eGFR. Consequently, patients often present at advanced stages of renal disease, when preventive interventions have limited efficacy.

Given this background, the current study was undertaken at a tertiary care teaching hospital in Kanpur to evaluate the pattern and progression of diabetic nephropathy in patients with poorly controlled T2DM. By correlating clinical and biochemical parameters including HbA1c, duration of diabetes, blood pressure status, albuminuria, and eGFR this study aims to highlight the magnitude of renal involvement in uncontrolled diabetics and underscore the critical need for early detection and management strategies tailored to the Indian healthcare setting.

Materials and Methods

Study Design and Setting: This was a hospital-based cross-sectional observational study conducted in the Department of General Medicine at Dr. BSK Institute of Medical Sciences, Lakhanpur Kalyanpur, Kanpur, Uttar Pradesh, India. The study was carried out over a period of 12 months, from March 2024 to February 2025

Study Population and Sample Size: A total of 140 patients with a confirmed diagnosis of type 2 diabetes mellitus and documented poor glycemic control were enrolled. Patients attending the outpatient department or admitted for routine evaluation or diabetic complications were screened and included based on inclusion criteria. The sample size was determined based on regional diabetic nephropathy prevalence estimates and institutional caseload.

Inclusion Criteria

- Age ≥30 years
- Known case of type 2 diabetes mellitus for at least one year
- HbA1c ≥8.0% at the time of evaluation
- Willingness to provide informed consent and undergo laboratory testing

Exclusion Criteria

- Patients with type 1 diabetes mellitus
- Known cases of chronic kidney disease due to non-diabetic causes (e.g., glomerulonephritis, obstructive uropathy, polycystic kidney disease)
- History of urinary tract infection within the last three months
- Patients on nephrotoxic medications or recent radiocontrast exposure
- Pregnant women
- Patients unwilling to provide consent

Data Collection and Clinical Assessment

All eligible patients underwent detailed clinical assessment, including:

- Demographic information (age, gender, duration of diabetes)
- Anthropometric data (BMI, blood pressure)
- Comorbid conditions such as hypertension and dyslipidemia
- Medication history (including use of ACE inhibitors/ARBs, insulin, or oral hypoglycemics)

Laboratory Investigations

- **Glycemic control:** HbA1c was measured using standardized HPLC method
- **Renal function markers:**
    - Serum creatinine (mg/dL)
    - eGFR calculated using the CKD-EPI equation
    - Urinary albumin-to-creatinine ratio (UACR) from spot urine sample
- **Other parameters:** fasting and postprandial glucose, lipid profile

Classification of Diabetic Nephropathy

Staging of diabetic nephropathy was performed based on KDIGO (kidney disease: Improving Global Outcomes**)** 2020 guidelines using a combination of eGFR and albuminuria categories:

- **Albuminuria staging:**
    - A1: <30 mg/g (normal to mildly increased)
    - A2: 30–300 mg/g (moderately increased / microalbuminuria)
    - A3: >300 mg/g (severely increased / macroalbuminuria)
- **eGFR staging:**
    - G1: ≥90 mL/min/1.73 m²
    - G2: 60–89 mL/min/1.73 m²
    - G3a: 45–59
    - G3b: 30–44
    - G4: 15–29
    - G5: <15

Outcome Measures

The primary outcome was the proportion of patients in each stage of diabetic nephropathy based on

UACR and eGFR. Secondary objectives included analysis of associations between DN stage and clinical factors such as:

- Duration of diabetes
- HbA1c level
- Presence of hypertension
- Use of nephroprotective drugs

Statistical Analysis: Data were recorded using Microsoft Excel and analyzed using SPSS version 27.0. Descriptive statistics were used for baseline characteristics. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequency and percentages. Pearson's Chi-square test was used for comparison of categorical variables, and t-tests or ANOVA for continuous variables. Correlation analysis was performed between nephropathy severity and risk factors. A p-value <0.05 was considered statistically significant.

Results

A total of 140 patients with poorly controlled type 2 diabetes mellitus (HbA1c ≥8%) were evaluated for the presence and severity of diabetic nephropathy. The mean age of participants was 56.4 ± 9.2 years, with a slight male predominance (58.6%). Most patients (62.9%) had diabetes for more than 10 years. Microalbuminuria was observed in 64 patients (45.7%) and macroalbuminuria in 32 patients (22.9%), while 44 patients (31.4%) had normal UACR levels. Reduced eGFR (<60 mL/min/1.73 m^2) was noted in 48 patients (34.3%). Patients with macroalbuminuria had significantly higher serum creatinine and lower eGFR. Duration of diabetes, HbA1c level, and presence of hypertension were significantly associated with advanced stages of diabetic nephropathy.

Table 1: Age-wise distribution of patients

Age Group (years)	Number of Patients	Percentage (%)
31–40	9	6.4
41–50	28	20.0
51–60	52	37.1
61–70	39	27.9
>70	12	8.6
Total	**140**	**100.0**

Table 2: Gender distribution

Gender	Number of Patients	Percentage (%)
Male	82	58.6
Female	58	41.4
Total	**140**	**100.0**

Table 3: Duration of diabetes mellitus

Duration (years)	Number of Patients	Percentage (%)
<5	18	12.9
5–10	34	24.2
>10	88	62.9
Total	**140**	**100.0**

Table 4: HbA1c levels among study population

HbA1c (%) Range	Number of Patients	Percentage (%)
8.0–8.9	29	20.7
9.0–9.9	47	33.6
≥10.0	64	45.7
Total	**140**	**100.0**

Table 5: Urinary albumin-to-creatinine ratio (UACR) categories

UACR Category	Number of Patients	Percentage (%)
Normal (<30 mg/g)	44	31.4
Microalbuminuria	64	45.7
Macroalbuminuria	32	22.9
Total	**140**	**100.0**

Table 6: eGFR distribution (KDIGO G-stage)

eGFR Stage	Number of Patients	Percentage (%)
G1 (≥90)	23	16.4
G2 (60–89)	69	49.3
G3a (45–59)	19	13.6
G3b (30–44)	16	11.4
G4 (15–29)	13	9.3
Total	**140**	**100.0**

Table 7: Serum creatinine levels

Serum Creatinine (mg/dL)	Number of Patients	Percentage (%)
≤1.0	64	45.7
1.1–1.5	36	25.7
>1.5	40	28.6
Total	**140**	**100.0**

Table 8: Association of albuminuria with duration of diabetes

Duration (years)	Normal	Microalbuminuria	Macroalbuminuria
<5	11	6	1
5–10	17	14	3
>10	16	44	28

Table 9: Association of albuminuria with HbA1c levels

HbA1c (%) Range	Normal	Microalbuminuria	Macroalbuminuria
8.0–8.9	21	8	0
9.0–9.9	17	25	5
≥10.0	6	31	27

Table 10: Association of albuminuria with hypertension

Albuminuria Status	Hypertensive Patients	Percentage (%)
Normal	12	27.3
Microalbuminuria	39	60.9
Macroalbuminuria	29	90.6

Table 11: Use of nephroprotective drugs (ACEi/ARBs)

Drug Use	Normal	Microalbuminuria	Macroalbuminuria	Total
On ACEi/ARBs	29	32	8	69
Not on ACEi/ARBs	15	32	24	71

Table 12: Correlation analysis (r values)

Parameter	Correlation Coefficient (r)	p-value
Duration of diabetes	+0.61	<0.001
HbA1c	+0.53	<0.001
Hypertension	+0.48	0.003
Serum creatinine	+0.66	<0.001
eGFR	-0.59	<0.001

Table 1 and Table 2 describe the demographic profile, with a predominance of patients aged 51–70 years and a slight male majority. Table 3 and Table 4 indicate that most patients had longstanding diabetes (>10 years) with poor glycemic control (HbA1c ≥9% in nearly 80%). Table 5 shows that 45.7% had microalbuminuria and 22.9% had macroalbuminuria, confirming significant renal involvement. Table 6 and Table 7 highlight the decline in eGFR and elevated serum creatinine, especially in macroalbuminuric patients. Tables 8 and 9 confirm strong associations between severity of nephropathy with longer disease duration and higher HbA1c levels. Table 10 underscores the role of hypertension in advancing nephropathy, while Table 11 shows that ACEi/ARB use correlated with less severe disease. Table 12 presents statistically significant correlations between albuminuria and multiple clinical parameters, reaffirming that diabetic nephropathy progression is multifactorial and largely preventable with timely intervention.

Discussion

This study provides valuable insights into the pattern and progression of diabetic nephropathy (DN) in patients with poorly controlled type 2 diabetes mellitus (T2DM) in a tertiary care setting. Our findings indicate that a substantial proportion of patients with elevated HbA1c levels exhibit early to advanced stages of nephropathy, even in the absence of overt clinical symptoms. This supports the growing body of evidence that microvascular complications begin to manifest silently in uncontrolled diabetes and emphasizes the importance of early screening and risk stratification [7].

A striking finding in this study was the high prevalence of albuminuria. Nearly 69% of patients demonstrated abnormal urinary albumin excretion—45.7% had microalbuminuria and 22.9% had macroalbuminuria [8]. This aligns with previous Indian studies and international literature that report microalbuminuria as an early and frequent finding in diabetics with poor glycemic control. Additionally, 34.3% of patients had eGFR below 60 mL/min/1.73 m^2, indicating stage 3 or higher chronic kidney disease (CKD). These findings underscore that renal function compromise begins even before patients become symptomatic or show a significant rise in serum creatinine levels [9].

The current study reaffirms that duration of diabetes plays a central role in the progression of DN. Patients with diabetes of more than 10 years' duration were significantly more likely to have moderate to severe nephropathy. Similarly, glycemic control, as reflected by HbA1c, showed a strong positive correlation with both albuminuria severity and decline in eGFR. Patients with HbA1c ≥10% had disproportionately higher rates of macroalbuminuria and renal impairment, suggesting that prolonged poor control accelerates glomerular injury [10].

Hypertension, present in a large majority of patients with macroalbuminuria (90.6%), also emerged as an independent contributor to DN progression. This highlights the interplay between metabolic and hemodynamic stressors in diabetic renal injury. It is well known that elevated intraglomerular pressure from uncontrolled hypertension exacerbates proteinuria and accelerates renal fibrosis, particularly in patients with pre-existing endothelial dysfunction due to hyperglycemia [11].

The role of ACE inhibitors and ARBs in renoprotection was evident in our analysis. Patients on these agents had significantly lower rates of macroalbuminuria, even among those with elevated HbA1c. This supports existing guidelines that advocate for early initiation of RAAS blockade in diabetics with microalbuminuria to delay nephropathy progression. Unfortunately, nearly half of our patients were not on these drugs, possibly due to lack of awareness, suboptimal prescribing practices, or patient non-adherence [12,13].

Another notable observation was the significant association between albuminuria and rising serum creatinine. Although serum creatinine is a late marker of nephropathy, 28.6% of our patients already had creatinine >1.5 mg/dL, suggesting missed opportunities for earlier detection. This further emphasizes the need for routine UACR and eGFR assessments in all diabetics, particularly those with longstanding disease or poor glycemic control [14].

From a public health perspective, our findings are alarming. A large proportion of patients had already entered stage 3 or 4 CKD, with a considerable risk of progression to end-stage renal disease requiring dialysis or transplant. Given the resource constraints and late presentation patterns in Indian settings, it becomes critical to integrate DN screening into routine diabetes care, supported by structured follow-up and aggressive risk factor management [15].

Despite being a cross-sectional study, the strength of this research lies in its systematic evaluation of both functional (eGFR) and structural (albuminuria) markers of nephropathy across multiple strata of glycemic control, duration of diabetes, and comorbidity burden. Limitations include the lack of renal biopsy confirmation (as this was not ethically or clinically indicated in most cases) and absence of long-term follow-up data to assess the trajectory of decline. However, the study offers a practical and representative snapshot of the burden of nephropathy in uncontrolled diabetics attending tertiary care centers in northern India.

Conclusion

The present study demonstrates that diabetic nephropathy is highly prevalent among patients with poorly controlled type 2 diabetes mellitus, even in the absence of clinical symptoms. Microalbuminuria and declining eGFR were common findings, especially in those with longer disease duration, elevated HbA1c levels, and associated hypertension. Macroalbuminuria and raised serum creatinine were significantly more likely in patients not receiving nephroprotective therapy. These results emphasize the urgent need for routine screening using UACR and eGFR in all diabetics, particularly those with HbA1c ≥9% or disease duration >10 years. Early detection, timely initiation of RAAS blockade, and strict glycemic and blood pressure control are essential to delay progression to end-stage renal disease. Public health strategies must prioritize diabetic renal screening and physician education to curb the growing burden of CKD in India's diabetic population.

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