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CORRELATION OF SERUM URIC ACID LEVEL WITH LEVEL OF URINARY ALBUMIN IN PATIENTS WITH DIABETIC NEPHROPATHY

Original Research

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ABSTRACT

Background: Diabetic nephropathy is a leading cause of chronic kidney disease worldwide, contributing significantly to morbidity and mortality among individuals with diabetes mellitus. Uric acid, a final product of purine metabolism, has been increasingly linked with renal dysfunction and progression of nephropathy. Albuminuria is considered a key marker of renal injury, reflecting glomerular and tubular damage. While extensive research has been conducted globally, limited evidence is available from Pakistan, where genetic diversity and varying environmental exposures may influence disease patterns.

Objective: This study aimed to evaluate the correlation between serum uric acid levels and urinary albumin excretion in patients with diabetic nephropathy.

Methods: This correlational study was conducted in the Department of Medicine, Khyber Teaching Hospital, Peshawar, between 26th September 2024 and 25th March 2025. A total of 84 patients, aged 18–70 years, with a confirmed diagnosis of diabetic nephropathy, were enrolled through non-probability consecutive sampling. Exclusion criteria included liver disease, gout, renal transplant, and medications influencing uric acid levels. Serum uric acid was measured from venous blood samples, and 24-hour urine collections were performed for albumin estimation. Data analysis was conducted using SPSS version 26, with Pearson correlation applied to assess the relationship between serum uric acid and urinary albumin.

Results: The mean age of participants was 50.65 ± 5.29 years, and the mean diabetes duration was 8.27 ± 3.09 years. Males comprised 44 patients (52.4%). Microalbuminuria was present in 46 patients (54.8%), while 24 patients (28.6%) had macroalbuminuria, and 14 patients (16.7%) had normal urinary albumin. Raised serum uric acid (>7.2 mg/dl) was detected in 26 patients (31.0%). The mean urinary albumin was 210.62 ± 197.14 mg/24 hr, and mean serum uric acid was 6.79 ± 1.25 mg/dl. Pearson correlation revealed a strong positive association ($r = 0.788$, $p = 0.000$) between urinary albumin and serum uric acid.

Conclusion: A significant positive linear correlation was established between serum uric acid and urinary albumin in diabetic nephropathy, suggesting that uric acid may serve as an additional marker of renal injury in diabetes.

Keywords: Albuminuria, Chronic Kidney Disease, Diabetes Mellitus, Diabetic Nephropathy, Hyperuricemia, Serum Uric Acid, Urinary Proteins.

INTRODUCTION

Diabetes mellitus (DM) is recognized as one of the most pressing global health problems, with its prevalence rising at an alarming rate. Nearly 200 million new cases are diagnosed annually, highlighting the immense burden this disease poses on healthcare systems worldwide (1). The condition is characterized by chronic hyperglycemia, which significantly increases the risk of cardiovascular complications, particularly coronary artery disease, through mechanisms driven by atherosclerosis (2). In Pakistan, the prevalence of diabetes is estimated at 13.14%, underscoring the magnitude of the issue at the national level (1). Among the metabolic disturbances associated with diabetes, uric acid has gained increasing attention. Uric acid is the end product of purine metabolism and its generation involves enzymatic reactions that yield reactive oxygen species (ROS). These ROS contribute negatively to vascular health and renal function, thereby amplifying the risks of cardiovascular disease and kidney damage (3). Normally, a substantial amount of uric acid is excreted through the gastrointestinal tract, while the majority is eliminated via the kidneys. Consequently, serum uric acid can serve as an indirect marker of oxidative stress produced during purine metabolism (4). Renal dysfunction is often first detected through albuminuria, defined as excessive excretion of albumin in the urine. Albuminuria reflects impaired tubular reabsorption and altered glomerular permeability, making it a sensitive clinical marker of renal pathology (5). Prolonged albuminuria is central to the development of diabetic nephropathy (DN), an irreversible complication characterized by structural and functional nephron damage in individuals with diabetes (6). DN represents the leading cause of advanced renal disease globally, placing a major burden on patients and healthcare systems alike.

The interplay between hyperuricemia and albuminuria in diabetes has been explored in several studies. A study reported that overt proteinuria was significantly more common among patients with type 2 diabetes who also exhibited elevated uric acid levels (7). Similarly, a study demonstrated a significant linear correlation between serum uric acid and urinary albumin excretion, with a correlation coefficient of 0.301 (8). These findings indicate that uric acid may not only reflect oxidative stress but also contribute to the pathophysiological progression of diabetic nephropathy. Despite global efforts, there is limited local data in Pakistan examining the relationship between uric acid and albuminuria in diabetic patients. Previous studies conducted in certain regions were limited by small sample sizes and lacked consideration of genetic diversity within different populations. Understanding this relationship in the Pakistani context is particularly important, as variations in genetic and environmental factors may influence disease patterns. Therefore, this study was designed to investigate the correlation between serum uric acid and urinary albumin levels in individuals with diabetic nephropathy. By addressing this gap, the research aims to provide locally relevant insights into the role of uric acid as a potential marker of renal injury in diabetes, thereby contributing to improved risk stratification and management strategies for affected individuals.

METHODS

This correlational study was conducted in the Department of Medicine, Khyber Teaching Hospital, Peshawar, from 26th September 2024 to 25th March 2025. Male and female patients aged 18 to 70 years with a confirmed diagnosis of diabetic nephropathy were enrolled. Inclusion criteria required evidence of diabetes mellitus along with renal involvement, established by reduced estimated glomerular filtration rate (eGFR <60 ml/min/1.73m² calculated using the Cockcroft–Gault formula) and/or albuminuria on 24-hour urine collection, supported by ultrasonographic findings of shrunken echogenic kidneys. Exclusion criteria included patients with protein-losing enteropathy, chronic liver disease or conditions impairing hepatic synthetic function, a history of gout or use of anti-gout medications, post-renal transplant status, use of drugs known to influence uric acid levels such as thiazide diuretics, and those with endocrine disorders affecting metabolic balance. Hyperuricemia was defined as serum uric acid greater than 7.2 mg/dl. Albuminuria was categorized as normal (<30 mg/24 h), microalbuminuria (30–300 mg/24 h), and macroalbuminuria (>300 mg/24 h). The sample size was calculated as 84 using a sample size calculator for correlational studies, with an anticipated correlation coefficient (r) of 0.301 between serum uric acid and urinary albumin, at 80% power and a 95% confidence interval (9). Participants were recruited through non-probability consecutive sampling from the inpatient department. Ethical approval was obtained from the institutional review board prior to the initiation of the study and written informed consent was secured from all participants. Anonymity and confidentiality were maintained, and no risks were posed to patients through participation.

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Baseline demographic and clinical data were collected, including age, sex, body mass index (BMI), duration of diabetes, residence (urban or rural), occupation, education level, monthly income, and socioeconomic status. Each patient underwent a comprehensive medical history and physical examination. Venous blood was collected from the antecubital vein of the non-dominant arm under aseptic precautions, with a 5 cc sample sent to the hospital laboratory within 30 minutes for serum uric acid estimation. For urinary analysis, participants were instructed to collect all urine voided over a 24-hour period. The first morning urine on the day of collection was discarded, after which all voided urine including the next morning's sample was collected in a provided container. Samples were sent to the laboratory for quantitative measurement of 24-hour urinary albumin, and results were recorded according to operational definitions. Data analysis was performed using IBM SPSS Statistics version 24. Quantitative variables were expressed as mean ± standard deviation, and categorical variables were presented as frequencies and percentages. The relationship between serum uric acid levels and urinary albumin excretion was evaluated using the Pearson correlation coefficient (r), interpreted as mild (r = 0.1–0.3), moderate (r = 0.4–0.5), or strong (>0.5). A p-value of less than 0.05 was considered statistically significant.

RESULTS

The study enrolled 84 participants with diabetic nephropathy. The mean age of the patients was 50.65 ± 5.29 years, and the mean body mass index (BMI) was 21.74 ± 1.12 kg/m². The mean duration of diabetes was 8.27 ± 3.09 years, while the mean duration of chronic kidney disease (CKD) was 3.56 ± 1.41 years. A majority of participants (77.4%) were older than 45 years, and 52.4% were male. More than half of the patients (61.9%) had a BMI greater than 21 kg/m². In terms of residence, 56.0% lived in urban areas, and 44.0% resided in rural areas. The duration of diabetes ranged widely; however, most patients (54.8%) had been diagnosed for 6–10 years, while 32.1% had diabetes for 5 years or less, and 13.1% for more than 10 years. Hypertension was present in 66.7% of the participants. Regarding diabetes treatment, 52.4% were on oral antidiabetic therapy, while 47.6% were on injectable treatment. For CKD management, 69.0% of the patients were receiving hemodialysis, and 31.0% were managed conservatively. The majority were diagnosed with type 2 diabetes mellitus (88.1%), while 11.9% had type 1 diabetes mellitus. Analysis of outcome variables showed that urinary albumin excretion was abnormal in a large proportion of patients. Microalbuminuria was observed in 54.8% of the cohort, while 28.6% had macroalbuminuria. Only 16.7% of patients had normal urinary albumin levels. Raised serum uric acid levels (>7.2 mg/dl) were recorded in 31.0% of patients, while 69.0% had normal uric acid levels. The association between serum uric acid and urinary albumin was statistically significant (p = 0.000). Among patients with normal urinary albumin excretion, none had elevated serum uric acid. In contrast, 17.4% of patients with microalbuminuria and 75.0% of patients with macroalbuminuria exhibited raised uric acid levels, indicating a progressive increase in hyperuricemia with worsening albuminuria.

The mean urinary albumin excretion was 210.62 ± 197.14 mg/24 h, while the mean serum uric acid concentration was 6.79 ± 1.25 mg/dl. Correlation analysis revealed a strong positive relationship between urinary albumin and serum uric acid, with a Pearson correlation coefficient of r = 0.788, suggesting that higher levels of uric acid were associated with increased albuminuria. Subgroup analyses were performed to explore whether the correlation between serum uric acid and urinary albumin remained consistent across different demographic and clinical characteristics. A stronger association was observed in patients older than 45 years (r = 0.81) compared to those 45 years or below (r = 0.72). Males exhibited a slightly higher correlation (r = 0.80) than females (r = 0.77). Patients with a BMI above 21 kg/m² showed a robust correlation (r = 0.82), whereas those with BMI ≤21 kg/m² demonstrated a comparatively lower correlation (r = 0.74). Similarly, patients with a diabetes duration of more than 10 years had the strongest correlation (r = 0.85), followed by those with 6–10 years (r = 0.79) and ≤5 years (r = 0.73). Treatment modalities also influenced the relationship, with patients on hemodialysis showing a stronger correlation (r = 0.83) compared to those managed conservatively (r = 0.76). These findings indicate that the association between serum uric acid and urinary albumin was consistently positive across all subgroups, with greater strength in older patients, those with longer diabetes duration, higher BMI, and advanced renal replacement therapy.

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Table 1: Means and standard deviation of patients according to baseline parameters (n = 84)

Parameters	Mean	Std. Deviation
Age (years)	50.65	5.290
BMI (kg/m2)	21.745	1.1290
Diabetes duration (years)	8.270	3.093
CKD duration (years)	3.562	1.414

Table 2: Frequencies and percentages of patients according to various parameters (n = 84)

Parameters		Frequency	Percent
Age (years)	45 or below	19	22.6
	More than 45	65	77.4
Gender	Female	40	47.6
	Male	44	52.4
BMI (kg/m2)	21.0 or below	32	38.1
	More than 21.0	52	61.9
Residence	Rural	37	44.0
	Urban	47	56.0
Diabetes duration (years)	5 or below	27	32.1
	6 to 10	46	54.8
	More than 10	11	13.1
HTN	Yes	56	66.7
	No	28	33.3
DM Rx	Oral	44	52.4
	Injectable	40	47.6
CKD Rx	Conservative	26	31.0
	HD	58	69.0
DM Type	T1DM	10	11.9
	T2DM	74	88.1

Table 3: Frequencies and percentages according to outcomes variables (n = 84)

Urinary albumin	Normal (<30mg/24hr)	14	16.7
	Micro (30-300mg/24hr)	46	54.8
	Macro (>300mg/hr)	24	28.6
Uric acid level	Normal (<7.2mg/dl)	58	69.0
	Raised (>7.2mg/dl)	26	31.0

Table 4: Contingency table analysis for association between urinary albumin and serum uric acid (n = 84)

		Uric acid level (mg/dl)		Total	Chi square p value
		Normal (<7.2)	Raised (>7.2)		
Urinary albumin (mg/24hr)	Normal (<30)	14	0	14	0.000
		100.0%	0.0%	100.0%	
	Micro (30-300)	38	8	46	
		82.6%	17.4%	100.0%	
	Macro (>300)	6	18	24	
		25.0%	75.0%	100.0%	
Total		58	26	84	
		69.0%	31.0%	100.0%	

Table 5: Correlational analysis between urinary albumin and serum uric acid level (n = 84)

Variables	Mean	S.D	Pearson Correlation
Urinary albumin (mg/24 hr)	210.62	197.139	0.788
Uric acid (mg/dl)	6.786	1.2539	

Table 6: Subgroup Analysis of Correlation Between Serum Uric Acid and Urinary Albumin (n = 84)

Subgroup	**Correlation Coefficient (r)**
Age ≤ 45 years	0.72
Age > 45 years	0.81
Gender	
Female	0.77
Male	0.80
BMI	
≤ 21 kg/m²	0.74
> 21 kg/m²	0.82
Diabetes duration	
≤ 5 yrs	0.73
6–10 yrs	0.79
> 10 yrs	0.85
CKD conservative therapy	0.76
Hemodialysis	0.83

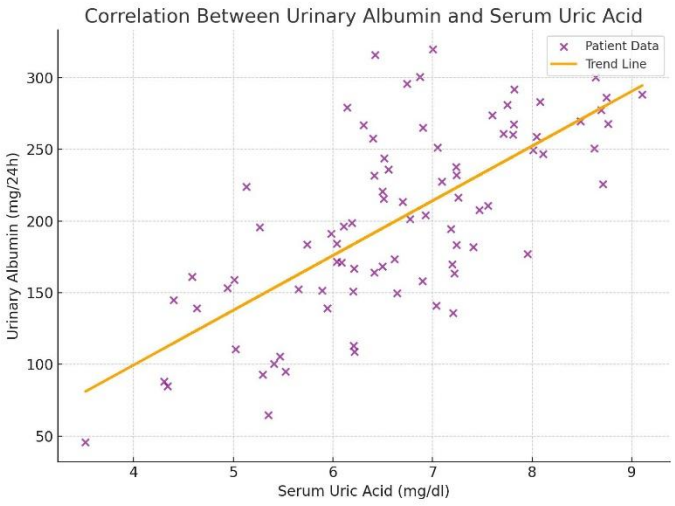

Figure 1 Correlation Between Urinary Albumin and Serum Uric Acid

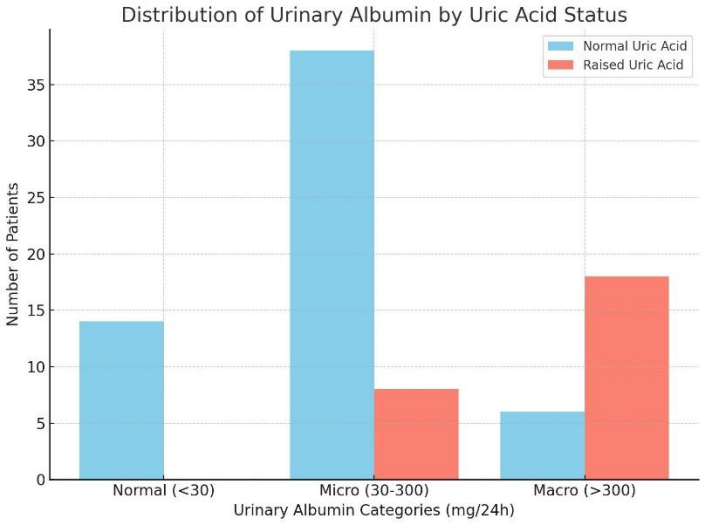

Figure 2 Distribution of Urinary Albumin by Uric Acid Status

DISCUSSION

The present study demonstrated that patients with diabetic nephropathy had a mean age of just above 50 years, with the majority being older than 45 years, and slightly more than half were males. The mean duration of diabetes was over eight years, while the mean duration of chronic kidney disease exceeded three years. More than half of the participants presented with microalbuminuria, nearly one-third with macroalbuminuria, and raised serum uric acid levels were observed in almost one-third of the cohort. A strong positive correlation was found between urinary albumin excretion and serum uric acid levels, with a Pearson correlation coefficient of 0.788 and a statistically significant p value. When compared to prior literature, the mean age in this cohort was slightly higher than some reported studies yet lower than others, reflecting differences in life expectancy and regional demographics across study populations (10-12). The gender distribution observed in this study was broadly consistent with several previous findings, where males were slightly predominant, although certain reports indicated higher female representation (13,14). This variability highlights that gender-related differences in diabetic nephropathy may be influenced by local demographic, cultural, or genetic factors, while evidence regarding the impact of gender on disease progression remains mixed (15).

The mean duration of diabetes in this study was comparable to some published reports, though lower or higher values have been documented depending on the study setting and population characteristics (16,17). Importantly, the finding that the majority of participants had a diabetes duration between six and ten years supports the understanding that longer disease duration predisposes to nephropathy, a finding echoed consistently across different geographic regions. In terms of outcome variables, the mean urinary albumin and serum uric acid levels in this study were broadly aligned with published literature. Several authors have demonstrated that higher serum uric acid levels were associated with worsening albuminuria, and correlations have ranged from mild to strong depending on study design, population, and adjustment for confounders (18-20). The strong positive correlation reported in this cohort is comparable to some international findings that emphasized uric acid as a significant biomarker of renal damage in diabetes. Differences in correlation strength between studies may be attributed to variations in patient characteristics, sample sizes, and methodological approaches. The clinical implications of these findings suggest that monitoring serum uric acid levels may provide additional insight into the risk and severity of albuminuria in patients with diabetic nephropathy. Elevated uric acid could act as an early marker of renal deterioration, complementing traditional markers such as eGFR and urinary albumin excretion (21,22). This has potential utility in risk stratification, early intervention, and possibly guiding therapeutic approaches, especially in resource-limited healthcare settings.

The strengths of this study include the use of standardized diagnostic criteria, robust correlation analysis, and the inclusion of both genders across a broad adult age range. However, there were limitations. The single-center design and relatively small sample size may limit the generalizability of the findings. Cross-sectional methodology precludes causal inference, and potential confounding variables

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such as dietary intake, genetic predisposition, use of nephroprotective drugs, and blood pressure control were not fully explored. Additionally, subgroup analyses revealed important trends but did not adjust for potential effect modifiers in multivariate models, which could have strengthened the conclusions. Future studies with larger, multicenter cohorts and longitudinal designs are warranted to clarify the causal relationship between hyperuricemia and progression of diabetic nephropathy. Incorporation of genetic and environmental factors, as well as detailed assessment of comorbid conditions and therapeutic interventions, may provide a more comprehensive understanding. Interventional trials targeting uric acid reduction in diabetic populations could further establish its role as a modifiable risk factor for slowing renal decline. Overall, this study contributes to the growing body of evidence highlighting the association between uric acid and albuminuria in diabetic nephropathy, reinforcing its potential significance as a clinical marker for renal impairment in patients with diabetes.

CONCLUSION

This study concluded that patients with diabetic nephropathy frequently exhibited hyperuricemia in association with increased urinary protein excretion, particularly in those with more advanced albuminuria. A consistent positive correlation between serum uric acid and urinary albumin highlighted the interlinked progression of metabolic and renal dysfunction in diabetes. These findings underscore the importance of considering serum uric acid not only as a metabolic by-product but also as a potential marker of renal injury, which may aid in the early identification of patients at risk of worsening nephropathy and guide timely preventive strategies in clinical practice.

AUTHOR CONTRIBUTION

Author	Contribution
Mahnoor Khattak*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Hamza Ali Khan	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Muhammad Saeed Jan	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Hammad Naeem	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Sana Rahim Khan	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Nayab Munib	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published

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