

Original Research Article

CLINICAL SIGNIFICANCE OF SERUM SIALIC ACID IN DIABETIC KIDNEY DISEASE AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Diabetic nephropathy is one of the most common microvascular complications of type 2 diabetes mellitus (T2DM) and a leading cause of chronic kidney disease. Chronic low-grade inflammation and endothelial dysfunction play a central role in its pathogenesis. Serum sialic acid, an acute-phase reactant and marker of endothelial injury, has been proposed as a potential biomarker for diabetic microvascular complications. This study aimed to evaluate the relationship between serum sialic acid concentration and diabetic nephropathy in patients with T2DM.

Materials and Methods: This hospital-based cross-sectional observational study included 93 patients with type 2 diabetes mellitus. Participants were categorized into groups based on the presence and severity of diabetic nephropathy using urinary albumin-to-creatinine ratio and estimated glomerular filtration rate. Clinical details, anthropometric measurements, and biochemical parameters including serum sialic acid, HbA1c, serum creatinine, and urinary albumin excretion were assessed. Serum sialic acid levels were compared between groups, and correlations with renal and glycaemic parameters were analyzed using Spearman's rank correlation coefficient. A p value <0.05 was considered statistically significant.

Results: Diabetic nephropathy was present in 52 (55.9%) patients. Serum sialic acid levels were significantly higher in patients with diabetic nephropathy compared to those without nephropathy (72.6 ± 11.4 mg/dL vs 54.2 ± 8.1 mg/dL; $p < 0.001$). A progressive and significant increase in serum sialic acid levels was observed with increasing severity of nephropathy, from normoalbuminuria to macroalbuminuria (ANOVA $p < 0.001$). Serum sialic acid showed a significant positive correlation with duration of diabetes ($r = 0.48$), HbA1c ($r = 0.42$), urinary albumin-to-creatinine ratio ($r = 0.61$), and serum creatinine ($r = 0.55$), and a significant negative correlation with eGFR ($r = -0.58$) (all $p < 0.001$).

Conclusion: Serum sialic acid concentration is significantly associated with the presence and severity of diabetic nephropathy in patients with type 2 diabetes mellitus. Its strong correlation with renal dysfunction and glycaemic control suggests that serum sialic acid may serve as a simple and cost-effective adjunctive biomarker for early detection and assessment of diabetic nephropathy. Further prospective studies are required to establish its prognostic utility.

Keywords: Type 2 diabetes mellitus; Diabetic nephropathy; Serum sialic acid; Inflammation; Albuminuria; Renal dysfunction.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycaemia and is a leading cause of microvascular complications worldwide. Among these, diabetic nephropathy (DN) remains one of the most common and serious complications, affecting approximately 30–40% of individuals with long-standing T2DM.^[1] DN is a major contributor to chronic kidney disease and end-stage renal disease, significantly increasing cardiovascular morbidity and mortality.^[2] The pathophysiology of DN is complex and involves sustained hyperglycaemia-induced metabolic and haemodynamic changes, oxidative stress, activation of inflammatory pathways, and endothelial dysfunction, leading to progressive structural and functional renal damage.^[3]

Chronic low-grade inflammation plays a pivotal role in the development and progression of diabetic nephropathy. Hyperglycaemia promotes the formation of advanced glycation end products, activation of protein kinase C, and increased production of pro-inflammatory cytokines, which collectively contribute to endothelial injury and increased vascular permeability within the renal microcirculation.^[4] These inflammatory processes precede overt clinical manifestations of nephropathy and may not be adequately reflected by conventional markers such as serum creatinine or estimated glomerular filtration rate in early disease stages.

Serum sialic acid (SSA) is a terminal monosaccharide component of glycoproteins and glycolipids present on cell membranes and circulating acute-phase proteins.^[5] It serves as an important marker of systemic inflammation and endothelial activation. Elevated SSA levels have been reported in various inflammatory and metabolic conditions, including diabetes mellitus, where increased synthesis and shedding of sialic acid-rich glycoproteins occur as a consequence of vascular endothelial damage and inflammatory stimulation.^[6] In T2DM, persistent hyperglycaemia is believed to enhance endothelial turnover and acute-phase protein production, resulting in higher circulating SSA concentrations.^[7] Evidence suggests that serum sialic acid levels are significantly higher in patients with T2DM compared to non-diabetic individuals, with further elevations observed in those with microvascular complications such as nephropathy, retinopathy, and neuropathy.^[8] In the context of diabetic nephropathy, increased SSA levels have been associated with the presence and severity of albuminuria and with declining renal function, indicating a possible role of SSA as a marker of glomerular endothelial injury and inflammatory burden.^[9] These findings support the hypothesis that SSA reflects ongoing renal microvascular damage before advanced renal impairment becomes clinically apparent.^[10]

Despite these observations, the relationship between serum sialic acid concentration and diabetic

nephropathy has not been extensively studied across different stages of renal involvement, particularly in patients with type 2 diabetes mellitus. Available studies are limited by heterogeneous populations, variable definitions of nephropathy, and lack of region-specific data. Given the rising prevalence of T2DM and diabetic kidney disease in the Indian population, further evaluation of SSA as a potential biomarker is warranted.^[11,12] Elucidating the relationship between serum sialic acid levels and diabetic nephropathy may provide insight into its utility as a simple, cost-effective marker for early renal involvement and disease progression in T2DM. Such an approach could aid in early risk stratification and timely intervention, ultimately contributing to improved renal and cardiovascular outcomes in patients with type 2 diabetes mellitus.

MATERIALS AND METHODS

Study design and setting: This was a hospital-based, cross-sectional observational study conducted in the Department of Medicine in collaboration with the Department of Biochemistry at a tertiary care teaching hospital for a period of 2 years between June 2023 to June 2025. The study was carried out over a defined study period after obtaining approval from the Institutional Ethics Committee. All procedures were performed in accordance with the ethical standards of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment.

Study population: The study included adult patients diagnosed with type 2 diabetes mellitus (T2DM) based on the American Diabetes Association diagnostic criteria. Patients aged ≥ 30 years with a minimum duration of diabetes of one year were considered eligible. Both male and female patients attending the outpatient department or admitted to the medical wards were consecutively recruited during the study period. Patients with known non-diabetic kidney disease, acute kidney injury, urinary tract infection, chronic inflammatory or autoimmune disorders, active infections, hepatic dysfunction, malignancy, or cardiovascular events within the preceding three months were excluded. Patients receiving drugs known to influence inflammatory markers, such as systemic corticosteroids or immunosuppressive agents, were also excluded to avoid confounding effects on serum sialic acid levels.

Grouping and definition of diabetic nephropathy: Participants were categorized into groups based on the presence or absence of diabetic nephropathy. Diabetic nephropathy was defined using urinary albumin excretion and/or albumin-to-creatinine ratio (ACR). Patients with normoalbuminuria (ACR < 30 mg/g) were considered as having no nephropathy, while those with microalbuminuria (ACR 30–299 mg/g) or macroalbuminuria (ACR ≥ 300 mg/g) were classified as having diabetic nephropathy. Where applicable, estimated glomerular filtration rate

(eGFR) was calculated using the CKD-EPI formula to assess the degree of renal function impairment.

Clinical and anthropometric assessment: Detailed clinical history was obtained from all participants, including duration of diabetes, treatment details, and presence of diabetic complications. Blood pressure was measured using a standardized protocol, and hypertension was defined as systolic blood pressure ≥ 140 mmHg, diastolic blood pressure ≥ 90 mmHg, or current use of antihypertensive medication. Anthropometric measurements such as height, weight, and body mass index (BMI) were recorded using calibrated instruments.

Laboratory investigations: After an overnight fast of at least 8–10 hours, venous blood samples were collected under aseptic conditions. Fasting plasma glucose, serum creatinine, and lipid profile were analyzed using standard enzymatic methods on an automated analyzer. Glycaemic control was assessed using glycated hemoglobin (HbA1c), measured by high-performance liquid chromatography or an equivalent standardized method. Serum sialic acid concentration was estimated using a validated biochemical method, such as the thiobarbituric acid assay or enzymatic colorimetric method, depending on laboratory availability. Results were expressed in mg/dL. Internal quality control measures were followed throughout the study period to ensure analytical accuracy and reproducibility. A spot urine sample was collected for estimation of urinary albumin and creatinine, and the albumin-to-creatinine ratio was calculated. In selected cases, 24-hour urinary protein estimation was performed where clinically indicated.

Outcome measures: The primary outcome measure was the serum sialic acid concentration and its relationship with the presence and severity of diabetic nephropathy. Secondary outcomes included the correlation of serum sialic acid levels with urinary

albumin excretion, eGFR, duration of diabetes, HbA1c, and other clinical parameters.

Statistical analysis: Data were entered into a spreadsheet and analyzed using SPSS (20.0). Continuous variables were tested for normality using the Shapiro–Wilk test. Normally distributed continuous variables were expressed as mean \pm standard deviation, while non-normally distributed data were expressed as median with interquartile range. Categorical variables were expressed as frequencies and percentages. Comparisons between two groups were performed using Student’s t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. One-way analysis of variance (ANOVA) was used for comparison of more than two groups, followed by appropriate post-hoc analysis. The association between serum sialic acid levels and renal as well as glycaemic parameters was assessed using Spearman’s rank correlation coefficient, considering the non-normal distribution of urinary albumin-to-creatinine ratio and biochemical variables. A two-tailed p value of <0.05 was considered statistically significant.

RESULTS

A total of 93 patients with type 2 diabetes mellitus were included in the study. The mean age of the study population was 54.6 ± 8.9 years, with a male predominance (62.4%). The mean duration of diabetes was 9.8 ± 4.6 years. More than half of the participants (52.7%) had coexisting hypertension. The mean BMI was 26.1 ± 3.4 kg/m², indicating an overweight population. Mean glycaemic control was suboptimal, with an average HbA1c of 8.2 ± 1.4 %. Renal function parameters showed a mean serum creatinine of 1.32 ± 0.48 mg/dL and a mean eGFR of 72.4 ± 18.6 mL/min/1.73 m² [Table 1].

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (n = 93)

Variable	Frequency (%) / mean \pm SD
Age (years)	54.6 ± 8.9
Gender	
Male	58 (62.4)
Female	35 (37.6)
Duration of diabetes (years)	9.8 ± 4.6
BMI (kg/m ²)	26.1 ± 3.4
Hypertension	49 (52.7)
Systolic BP (mmHg)	136.8 ± 14.2
Diastolic BP (mmHg)	84.6 ± 8.9
HbA1c (%)	8.2 ± 1.4
Serum creatinine (mg/dL)	1.32 ± 0.48
eGFR (mL/min/1.73 m ²)	72.4 ± 18.6

BMI: body mass index; HbA1c: glycated hemoglobin; eGFR: estimated glomerular filtration rate; BP: blood pressure.

Of the 93 participants, 52 (55.9%) had diabetic nephropathy, while 41 (44.1%) did not show evidence of nephropathy. Patients with diabetic nephropathy were significantly older and had a longer duration of diabetes compared to those without nephropathy ($p = 0.018$ and $p < 0.001$,

respectively). Glycaemic control was significantly poorer in the nephropathy group, with higher HbA1c levels ($8.7 \pm 1.3\%$ vs $7.6 \pm 1.2\%$, $p < 0.001$). Renal parameters showed markedly higher serum creatinine levels and significantly lower eGFR in patients with nephropathy ($p < 0.001$ for both). Urinary albumin-

to-creatinine ratio was substantially elevated in the nephropathy group ($p < 0.001$). Notably, serum sialic acid levels were significantly higher in patients with

diabetic nephropathy compared to those without nephropathy (72.6 ± 11.4 mg/dL vs 54.2 ± 8.1 mg/dL, $p < 0.001$) [Table 2].

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levels ($8.7 \pm 1.3\%$ vs $7.6 \pm 1.2\%$, $p < 0.001$). Renal parameters showed markedly higher serum creatinine levels and significantly lower eGFR in patients with nephropathy ($p < 0.001$ for both). Urinary albumin-to-creatinine ratio was substantially elevated in the nephropathy group ($p < 0.001$). Notably, serum sialic acid levels were significantly higher in patients with diabetic nephropathy compared to those without nephropathy (72.6 ± 11.4 mg/dL vs 54.2 ± 8.1 mg/dL, $p < 0.001$) [Table 2].

Table 2: Comparison of Clinical and Biochemical Parameters Between Patients with and Without Diabetic Nephropathy

Parameter	Without DN (n = 41)	With DN (n = 52)	p value
	Frequency (%) / mean \pm SD / median (IQR)		
Age (years)	52.1 \pm 8.3	56.6 \pm 9.1	0.018
Duration of diabetes (years)	7.1 \pm 3.2	11.9 \pm 4.3	<0.001
HbA1c (%)	7.6 \pm 1.2	8.7 \pm 1.3	<0.001
Serum creatinine (mg/dL)	0.98 \pm 0.21	1.59 \pm 0.46	<0.001
eGFR (mL/min/1.73 m ²)	86.3 \pm 14.7	61.4 \pm 15.8	<0.001
Urine ACR (mg/g)	18 (12–24)	186 (64–412)	<0.001
Serum sialic acid (mg/dL)	54.2 \pm 8.1	72.6 \pm 11.4	<0.001

DN: diabetic nephropathy; ACR: albumin-to-creatinine ratio; eGFR: estimated glomerular filtration rate

Serum sialic acid levels demonstrated a progressive and statistically significant increase with worsening severity of diabetic nephropathy. Patients with normoalbuminuria had the lowest mean serum sialic acid levels (54.2 ± 8.1 mg/dL), followed by those with microalbuminuria (66.8 ± 9.2 mg/dL), while the highest levels were observed in patients with

macroalbuminuria (81.4 ± 10.6 mg/dL). One-way analysis of variance revealed a highly significant difference in serum sialic acid concentrations across the three groups (ANOVA $p < 0.001$). Post-hoc analysis confirmed that the differences between each pair of groups were statistically significant [Table 3].

Table 3: Serum Sialic Acid Levels According to Severity of Diabetic Nephropathy

Nephropathy category	Serum sialic acid (mg/dL)
	mean \pm SD
Normoalbuminuria (n=41)	54.2 \pm 8.1
Microalbuminuria (n=29)	66.8 \pm 9.2
Macroalbuminuria (n=23)	81.4 \pm 10.6
p value	<0.001

Serum sialic acid levels showed a statistically significant positive correlation with duration of diabetes ($r = 0.48$, $p < 0.001$), glycated hemoglobin (HbA1c) levels ($r = 0.42$, $p < 0.001$), urinary albumin-to-creatinine ratio ($r = 0.61$, $p < 0.001$), and serum creatinine ($r = 0.55$, $p < 0.001$). A significant

negative correlation was observed between serum sialic acid levels and estimated glomerular filtration rate (eGFR) ($r = -0.58$, $p < 0.001$), indicating an inverse relationship between inflammatory burden and renal function [Table 4].

Table 4: Correlation Between Serum Sialic Acid and Renal and Glycaemic Parameters

Parameter	Correlation coefficient (r)	p value
Duration of diabetes	0.48	<0.001
HbA1c (%)	0.42	<0.001
Urine ACR	0.61	<0.001
Serum creatinine	0.55	<0.001
eGFR	-0.58	<0.001

Correlation analysis was performed using Spearman's correlation coefficient. eGFR: estimated glomerular filtration rate; HbA1c: glycated hemoglobin.

DISCUSSION

The present study demonstrates a significant association between serum sialic acid (SSA) concentration and diabetic nephropathy in patients with type 2 diabetes mellitus, supporting the role of SSA as a marker of inflammation and endothelial dysfunction in diabetic renal disease. In our cohort of 93 patients, more than half had evidence of diabetic nephropathy, reflecting the high burden of microvascular complications among Indian patients with long-standing and suboptimally controlled diabetes in studies by Manhas et al., and Wani et al.^[13,14] Patients with nephropathy were older, had a longer duration of diabetes, poorer glycaemic control, and significantly impaired renal function compared to those without nephropathy, findings that are consistent with established epidemiological patterns reported in Indian and global studies by Vasu et al., and Mattiottiet al.^[15,16] A key finding of this study is the markedly higher serum sialic acid levels observed in patients with diabetic nephropathy compared to those without nephropathy (72.6 ± 11.4 mg/dL vs 54.2 ± 8.1 mg/dL, $p < 0.001$). This observation aligns with earlier reports by El Badawy et al., and Ramana et al., that have shown elevated SSA levels in diabetic individuals with microvascular complications.^[17,18] The increase in SSA in nephropathy likely reflects enhanced hepatic synthesis of acute-phase glycoproteins and increased shedding of sialic acid-rich components from damaged glomerular endothelial cells under chronic hyperglycaemic and inflammatory stress.^[19] Since the glomerular endothelium plays a central role in maintaining the charge-selective barrier of the kidney, its injury may directly contribute to albuminuria and progressive renal dysfunction.^[20] Importantly, the present study demonstrates a stepwise and statistically significant rise in serum sialic acid levels with increasing severity of diabetic nephropathy, from normoalbuminuria to microalbuminuria and macroalbuminuria (ANOVA $p < 0.001$). This graded increase suggests that SSA not only differentiates patients with and without nephropathy but may also reflect the severity of renal involvement.^[21] Similar trends have been reported in previous studies by Prajna et al., and Singh et al., where SSA levels were significantly higher in patients with macroalbuminuria compared to those with microalbuminuria, supporting its potential utility as a marker of disease progression.^[22,23] The progressive rise in SSA parallels the escalating

inflammatory burden and endothelial injury that accompany worsening nephropathy.^[24] Correlation analysis further strengthens the biological plausibility of these findings. Serum sialic acid showed a strong positive correlation with urinary albumin-to-creatinine ratio ($r = 0.61$, $p < 0.001$) and serum creatinine ($r = 0.55$, $p < 0.001$), and a significant negative correlation with eGFR ($r = -0.58$, $p < 0.001$). These associations indicate that higher SSA levels are closely linked to worsening renal function and increased proteinuria.^[25] Comparable correlations have been reported in earlier studies by Shahvaliet al., and Prajna et al., suggesting that SSA reflects both structural and functional renal impairment in diabetes.^[25,26] The inverse relationship between SSA and eGFR highlights the role of systemic inflammation in accelerating renal decline. Additionally, serum sialic acid demonstrated significant positive correlations with duration of diabetes and HbA1c levels, underscoring the influence of chronic hyperglycaemia on inflammatory activation.^[27] Persistent poor glycaemic control promotes oxidative stress, formation of advanced glycation end products, and activation of pro-inflammatory pathways, all of which contribute to endothelial damage and increased circulating SSA levels.^[28,29] These findings emphasize that SSA integrates the cumulative effects of metabolic dysregulation and vascular injury over time.^[30]

Limitations

The present study has certain limitations that should be considered while interpreting the findings. First, the cross-sectional design limits the ability to establish a causal relationship between serum sialic acid levels and the development or progression of diabetic nephropathy. Temporal changes in serum sialic acid and their predictive value for incident nephropathy could not be assessed. Second, the study was conducted at a single tertiary care centre with a relatively modest sample size, which may limit the generalizability of the findings to the broader diabetic population. Third, although major confounding conditions influencing inflammatory markers were excluded, residual confounding due to unmeasured inflammatory or metabolic factors cannot be completely ruled out. Fourth, serum sialic acid was measured at a single time point, and intra-individual variability over time was not evaluated. Finally, other inflammatory biomarkers such as C-reactive protein or interleukins were not assessed, which could have provided a more comprehensive understanding of the

inflammatory milieu associated with diabetic nephropathy.

CONCLUSION

This study demonstrates a significant association between serum sialic acid concentration and diabetic nephropathy in patients with type 2 diabetes mellitus. Serum sialic acid levels were markedly higher in patients with nephropathy and showed a progressive increase with worsening severity of renal involvement. The strong correlations observed between serum sialic acid and urinary albumin excretion, renal function parameters, duration of diabetes, and glycaemic control underscore its close relationship with inflammatory burden and endothelial dysfunction in diabetic kidney disease. These findings suggest that serum sialic acid may serve as a simple and cost-effective adjunctive biomarker for identifying patients at increased risk of diabetic nephropathy and for assessing disease severity. Future prospective and longitudinal studies with larger, multi-centre cohorts are warranted to validate these findings and to explore the potential role of serum sialic acid in predicting disease progression and therapeutic response.

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