Original Article

Relation between Serum Uric Acid and Anthropometric Measures in Diabetic Nephropathy Patients

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Abstract

Background: Diabetic nephropathy is a major challenge of diabetes mellitus, leading to significant morbidity and mortality. Raised serum uric acid (SUA) tier is a potential cause of deterioration of kidney disease, such as diabetic nephropathy. Anthropometric measures for instance Body Mass Index (BMI) and waist circumference (WC) are crucial markers of adiposity, which is intimately linked to both progression of diabetic nephropathy and hyperuricemia.

Objective: To evaluate the relation between SUA and anthropometric measures in diabetic nephropathy.

Methods: A cross-sectional study was executed involving 150 patients by convenience sampling diagnosed with diabetic nephropathy at the Diabetic Clinic of Tertiary care Hospital in Lahore. SUA levels were measured using enzymatic methods, while anthropometric measures, including BMI and waist circumference, were recorded. The relation between SUA and these anthropometric measures was analyzed using Spearman correlation coefficient.

Results: The median (IQR) SUA in diabetic nephropathy patients was 6.9 (5.4-8.6) mg/dl and in healthy group was 5.2 (4.6-6.2) mg/dl. In diabetic nephropathy group, a significant direct relation of SUA was found with BMI (rho = 0.296, $p < 0.001$) and also with waist circumference (rho = 0.435, $p < 0.001$). In healthy group, significant direct relation was found with waist circumference only (rho = 0.212, $p = 0.001$). SUA, Waist circumference and BMI were higher considerably in diabetic nephropathy when measured against the control.

Conclusion: The study demonstrates a significant relationship between elevated SUA levels and adverse anthropometric measures in diabetic nephropathy patients. These findings suggest that managing obesity through lifestyle modifications and pharmacotherapy could play a critical role in controlling SUA and potentially slowing the progression of diabetic nephropathy.

Key words: Uric acid, waist circumference, diabetic nephropathy.

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Introduction

Diabetic nephropathy is a foremost cause of terminal kidney disease universally, posing a major challenge in diabetes management. With the increasing burden of diabetes mellitus, especially type 2 diabetes, the need to comprehend the mechanisms driving the progression

of diabetic nephropathy is very much required for developing effective interventions.[1]

Serum uric acid (SUA) has emerged as a prime factor in this context. Traditionally considered a marker of renal dysfunction, recent research suggests that elevated SUA levels may play a leading role in the pathology of diabetic nephropathy.[2] Hyperuricemia can induce reactive oxidative stress, inflammation, and vascular dysfunction, all of which contribute to the progression of kidney damage.[3] SUA has been associated with glomerular hypertension, promoting glomerulosclerosis and interstitial fibrosis—hallmarks of diabetic nephropathy. Furthermore, it can impair renal autoregulation, exacerbating damage in individuals with compromised kidney function due to diabetes.[4]

Anthropometric adiposity markers, as Body Mass Index (BMI), waist circumference (WC), waist-to-hip ratio (WHR), body fat percentage, are closely linked to both diabetes mellitus and diabetic nephropathy. Obesity, particularly central (visceral) obesity has a prime role in the pathogenesis of insulin impediment, type 2 diabetes and metabolc syndrome, which are main risk factors for diabetic renal disease.[5,6]

Elevated serum uric acid (SUA) levels are increasingly recognized as both a marker and a potential contributor to the advancement of diabetic nephropathy, particularly in the context of adiposity-related diabetes. The interplay between obesity, uric acid, and diabetic nephropathy can be described through several key mechanisms mediated through insulin resistance.[7] Insulin resistance leads to decreased renal uric acid clearance, resulting in hyperuricemia. Higher uric acid exacerbates insulin resistance by impairing endothelial function and promoting oxidative stress, creating a vicious cycle that contributes to hyperglycemia and accelerates the onset of diabetic nephropathy.[8]

Understanding SUA's role in diabetic nephropathy offers potential therapeutic avenues, such as using urate-lowering agents, to slow disease progression and improve renal outcomes in diabetic patients.[5,6,8]

Current study aims to evaluate the relation between SUA and anthropometric measures in patients with diabetic nephropathy, with the goal of providing insights into potential strategies for slowing disease progression.

Methods

This was cross-sectional observational study, executed at the University of Health Sciences, Lahore over a span of 1 year from March 2010 to February 2011. Approval was taken from Ethical Board of the University of Health Sciences (No: UHS Education/126-091228/20-10-2009). A total 150 patients of diabetic nephropathy were included in the study. Sample size was determined by WHO calculator using the formulation for study design. Convenience sampling technique was employed, and patients were recruited from Services Institute of Medical Sciences, Lahore.[9]

Inclusion criteria were as follows: age between 30-60 years, diagnosed with type 1 or 2 diabetes mellitus, and evidence of nephropathy (microalbuminuria or proteinuria, with or without reduced glomerular filtration rate). Exclusion criteria included patients with gout, chronic inflammatory diseases, or those on urate-lowering therapy.

Paticipants provided written informed consent before recruitment. Demographics, as age, gender, duration of diabetes, and medication history, were gathered through structured questionnaire and previous records.

BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Waist circumference was measured at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest, using a flexible measuring tape.[5]

Blood samples were collected after an overnight fast. Serum uric acid levels were measured using an enymatic calorimetric method on an automated analyzer. Serum creatinine and glucose levels were also measured to assess renal function and glycemic control.[10]

Data were scrutinized with SPSS version 22. As data distribution was not normal Mann Whitney U statistical test was employed for comparing the Medians of quantitative variables between diabetic nephropathy and healthy groups. Spearman's correlation coefficient was used to evaluate the relationship between SUA levels and anthropometric measures. A p-value <0.05 was of statistical significance.

Results

The population of current study consisted 150 male patients of diabetic nephropathy and 75 healthy male controls. Median (IQR) age of Group 1 subjects was 49.01 (43-55) years and that of the other was 38.5 (35-45) years. The Median (IQR) SUA in diabetic nephropathy patients was 6.7 (5.6-8) mg/dl and in healthy group was 5.2 (4.6-6.2) mg/dl. SUA, Waist circumference and BMI were higher considerably in diabetic nephro-

pathy group as compared to the healthy one (Table- I). In the group with diabetic nephropathy, a significant direct relation was found between SUA and BMI (rho = 0.296, $p < 0.0001$) also waist circumference (rho = 0.435, $p < 0.0001$). In healthy group, significant direct/ positive correlation was found with waist circumference only (rho = 0.212, p = 00.001), (Table-II, Figures I & II).

Table I: *Comparison of variables between study groups.*

Variables	Diabetic Nephropathy (N=150)	Healthy Group (N=75)	p-value
Age in years	49.01 (43-55)	38.5 (35-45)	0.000*
Systolic blood pressure	130 (110-140)	115 (90-130)	0.000
Diastolic blood pressure	80 (70-90)	75 (70-80)	0.000
Waist circumference in cm	102 (95-106)	88.5 (72-90)	0.000
Body mass index in kg/m^2	28.72 (25.91-31.67)	27.86 (22.97-30.56)	0.55
Uric acid	6.9 (5.4-8.8)	5.2 (4.6-6.2)	0.000

Mann Whitney U stats is employed to compare study groups. A p-value less than 0.05 is of statistical significance.

Table II: *Correlation of Study variables.*

Correlation of variables by Spearman Test		Waist circumference (cm)	Body mass index (Kg/meter square)
Diabetic nephropathy	Serum	0.435 0.000*	0.296 0.000*
Healthy group	uric acid	0.212 0.001*	0.31 0.285

Spearman stats is employed to determine correlation between the variables in each group. A p-value less than 0.05 is of statistical significance.

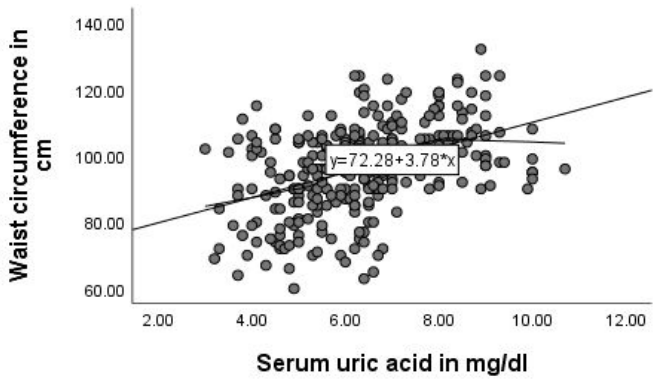

Figure-I: *Scatter plot showing significant relationship between waist circumference and serum uric acid in diabetic nephropathy group.*

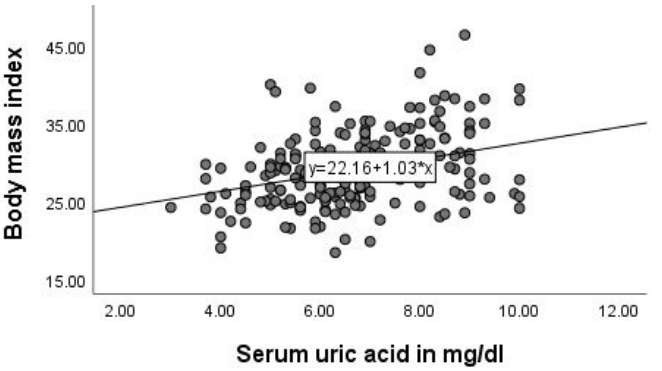

Figure-II: *Scatter plot showing significant relationship between serum uric acid and body mass in diabetic nephropathy group.*

Discussion

The current study evaluated the relationship between serum uric acid (SUA), anthropometric measures of obesity, and diabetic nephropathy in male patients of diabetic nephropathy and healthy male controls. A key observation from the study is the significantly higher median SUA in diabetic nephropathy patients as compared to the healthy subjects. The median level of SUA l in the diabetic nephropathy group was 6.8 mg/dl, while in the control healthy group was 5.2 mg/dl. This observation is in harmony with previous studies that have pinned hyperuricemia as not only a marker but also a contributor to the pathophysiology of diabetic nephropathy.[10-12] Elevated SUA levels are thought to promote renal injury by inducing oxidizing stress, inflammation, vascular dysfunction, and fibrosis, all of which contribute to the worsening of kidney function in diabetic patients.[13-15] The significantly higher levels of SUA in the nephropathy group highlights the importance of managing uric acid levels in this population to slow down the progression of renal damage.[16]

The study also found that waist circumference and BMI were higher significantly in the group with diabetic nephropathy as to healthy ones, suggesting a strong link between obesity and diabetic nephropathy.[17,18] Obesity, particularly central or visceral obesity is crucial in the evolution of insulin resitance and related states, either of which are closely linked with the onset and progress of type 2 diabetes mellitus and nephropathy.[19] Increased waist circumference and BMI are markers of excess adiposity, which promotes chronic low-pace inflammation, oxidation stress, and dysregulated lipid metabolism.[15] These factors contribute to kidney damage, further

exacerbating diabetic nephropathy in individuals with obesity.

A key finding of this study is the significant direct relation between SUA levels and anthropometric obesity measures, particularly in the diabetic nephropathy group. SUA showed a strong direct relation with waist circumference (rho = 0.435, $p < 0.001$) and a moderate correlation with BMI (rho = 0.296, $p < 0.001$). This suggests that individuals with higher levels of central obesity are likely to have elevated SUA tier, further increasing their risk of renal complications.[20] Central obesity, measured by waist circumference, predicts metabolic risk more than BMI alone, as it reflects the accumulation of visceral fat, which is metabolically active and secretes pro-inflammatory cytokines that promote both insulin resistance and kidney damage.[21]

The bridge between SUA and adiposity is well-documented in the literature. Adipose tissue, especially visceral fat, contributes to uric acid production through increased breakdown of purines. Additionally, insulin resistance associated with obesity impairs renal excretion of uric acid, leading to hyperuricemia.[21,22] This creates a bidirectional relationship in which obesity contributes to elevated SUA levels, and hyperuricemia, in turn, exacerbates the metabolic and inflammatory burden of obesity, further promoting renal dysfunction.[22]

Interestingly, in the healthy control group, SUA levels showed a considerable direct correlation with waist circumference (rho = 0.212, $p = 0.001$) but not with BMI. This finding suggests that even in individuals without diabetic nephropathy, central obesity is a more important determinant of SUA levels than overall body mass. It highlights the potential importance of waist circumference as a risk factor for hyperuricemia and its associated complications, even in seemingly healthy individuals.[23]

Overall, the findings of this study emphasize the complex interplay between SUA, obesity, and diabetic nephropathy. Elevated SUA levels, particularly in the context of obesity, may serve as an early indicator of kidney dysfunction in patients with diabetes. The strong correlation between SUA and central obesity (waist circumference) highlights the need for targeted interventions aimed at reducing visceral fat and managing hyperuricemia to mitigate the risk of diabetic nephropathy.[22-24] The positive correlations between SUA and anthropometric measures of obesity underscore the potential role of hyperuricemia as a modifiable risk factor in the progress of diabetic nephropathy. These findings suggest that managing both obesity and SUA levels may be key strategies in prevention or postponement of renal complications in diabetic patients.[24]

Conclusion

There is a significant relation between elevated SUA levels and adverse anthropometric measures, particularly BMI and waist circumference, in diabetic nephropathy patients. These indicate that addressing obesity may be a key target in controlling SUA levels and slowing the progression of diabetic nephropathy. Further studies are required to explore the causal relationships and to evaluate the effect of weight management strategies on SUA levels and renal outcomes in this patient population.

Conflict of Interest: None

Funding Source: None

Ethical Consideration: The study was approved by the ethical review boarcd. Informed written consent was obtained from the participants, and the confidentiality of their data was clearly explained.

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Authors Contribution

All the authors contributed equally in accordance with ICMJE guidelines and are accountable for the integrity of the study.

MI: Study design and concept, Acquisition of data and final approval

TT: Data Collection, analysis and final revision

AA: Data Collection, statistical analysis and final revision

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