Original Article

Association of Serum Uric Acid with Urine Albumin Among Patients with Type -2 Diabetes Mellitus at Tertiary Care Hospital

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Abstract

Background: Microalbuminuria is recognized as an early marker of diabetic nephropathy and cardiovascular risk in patients with type 2 diabetes mellitus (T2DM). Identifying its prevalence and correlates in newly diagnosed diabetic patients is essential for timely intervention and prevention of long-term complications. **Material and Methods:** A cross-sectional study was conducted over one year in the Outpatient Department of Medicine at Government Medical College, Baramulla. A total of 100 patients with type 2 diabetes mellitus (T2DM) aged 30–70 years were enrolled after applying the inclusion and exclusion criteria. Demographic details, medical history, body mass index (BMI), and blood pressure data were collected. Fasting blood glucose, lipid profile, serum creatinine, hemoglobin, uric acid, and glomerular filtration rate were measured. Microalbuminuria was assessed by the immunoturbidimetric method in 24-hour urine samples, with levels of 30–300 mg/24 h considered significant. **Results:** The prevalence of microalbuminuria was found to be [insert % from your results]. Patients with microalbuminuria had significantly higher mean ages, durations of diabetes, systolic and diastolic blood pressures, fasting blood sugars, and serum creatinines compared to those without microalbuminuria ($p < 0.05$). A strong association was also observed between microalbuminuria and hypertension, dyslipidemia, and poor glycemic control. **Conclusion:** Microalbuminuria is common among T2DM patients and shows significant correlation with glycemic status, hypertension, and renal function parameters. Early detection and management of microalbuminuria in diabetic patients can help prevent progression to overt nephropathy and reduce cardiovascular morbidity.

Keywords: Microalbuminuria; Type-2 Diabetes; Serum Uric Acid; Diabetic Nephropathy; Glycemic Controls; Cardiomascular Risk.

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Introduction

Diabetes, often referred to as the "silent killer," presents an increasingly complex pathophysiological challenge due to its close associations with numerous other risk factors. Alongside cardiovascular disease, cancer, and respiratory illnesses, diabetes is recognized as one of the most pressing global health concerns of the 21st century.[1,2]

Type 2 diabetes mellitus (T2DM) is characterized by key features, including hyperglycemia, insulin resistance, and impaired insulin production. While the disease's pathophysiology is multifactorial—shaped by genetic factors that affect insulin dynamics and environmental contributors such as obesity—its mechanisms remain incompletely understood.

The prevalence of diabetes has escalated to pandemic levels, particularly in developing nations such as India and China. According to the World Health Organization (WHO), the prevalence of diabetes is increasing most rapidly in low- and middle-income countries.[1,2] The International Diabetes Federation (IDF) reported in 2019 that China (116.4 million cases), India (77.0 million cases), and the United States (31.0 million cases) accounted for the highest numbers of people with diabetes. Furthermore, projections indicate that by 2030 and 2045, China will bear the burden of 140.5 and 147.2 million cases, while India will have 101.0 and 134.2 million cases, respectively.[3] Historically, diabetes mellitus was recognized for its association with hyperglycaemia, but it is now established as a significant risk factor for atherosclerotic vascular diseases, including stroke. This heightened risk is only partially explained by traditional risk factors such as hyperinsulinemia, elevated triglyceride levels, reduced HDL cholesterol, hypertension, and glucose intolerance. Emerging markers, such as serum uric acid (SUA) levels, have garnered attention as additional risk indicators. Another well-established marker is microalbuminuria, defined as urinary albumin excretion (UAE) between 30 and 300 mg in a 24-hour period, which reflects subclinical microvascular damage to the glomerular filtration barrier preceding diabetic nephropathy.[3] Even UAE levels

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below the microalbuminuria threshold are linked to increased cardiovascular morbidity and mortality.[4]

Hypertension is another significant contributor to elevated UAE, and the coexistence of hypertension and T2DM amplifies the likelihood of microalbuminuria. In fact, most individuals with T2DM also present with hypertension, further compounding the risk.[5] Arterial stiffness, along with interactions between microvascular and macrovascular.

Dysfunction has been proposed as another pathway contributing to the development of microalbuminuria. Multiple studies have demonstrated a relationship between arterial stiffness and microalbuminuria in patients with type 2 diabetes mellitus (T2DM) and other populations. Notably, these studies often focus on individuals with long-standing or indeterminate durations of diabetes, which raises the possibility that disease progression exacerbates microalbuminuria and vascular stiffness.[6–8]

A critical knowledge gap remains regarding the individual contributions of hyperglycaemia, elevated blood pressure, and macrovascular dysfunction to the development of microalbuminuria in the early stages of T2DM—before the cardiovascular complications associated with the disease become evident. Consequently, the present study aimed to investigate the prevalence of hyperuricemia in the Indian population and its potential association with other cardiovascular risk factors in individuals with diabetes.

Materials and Methods

This study was conducted in the outpatient setting of the Department of Medicine, Government Medical College, Baramulla. It was designed as a cross-sectional study with a duration of one year. Ethical approval was obtained from the Institutional Ethics Committee of the institution before the commencement of the study. Written informed consent was taken from all participants, and complete confidentiality of the collected information was maintained. The study population consisted of 100 patients diagnosed with type 2 diabetes mellitus. Patients aged 30 to 70 years were included. Exclusion criteria were patients with diabetic nephropathy, acute or chronic kidney disease, rheumatological diseases, neurological or psychiatric disorders, myeloproliferative disorders, substance abuse, alcohol intake, and pregnant or lactating women. The sample size was estimated using the formula $n = Z^{2*} P (1 - P)/d^2$, where the Z value at a 95% confidence interval was 1.96, P denoted the expected proportion, and d represented the absolute precision. Data was collected using a structured case study form. The diagnosis of type 2 diabetes mellitus was established according to the American Diabetes Association (ADA) criteria, which included fasting blood glucose levels of ≥126 mg/dL and/or a 2-hour post-load glucose level of ≥200 mg/dL during an oral glucose tolerance test with 75 g of glucose. A detailed medical history, with an emphasis on comorbidities and medication use, was recorded, followed by a physical examination. Body mass index (BMI) was calculated in kg/m². Blood pressure was Measured in the sitting position using a validated oscillometric device (Microlife Exact BP, Microlife AG, Switzerland), and hypertension was defined as an ambulatory daily blood pressure ≥135/85 mmHg and/or current use of antihypertensive medication.

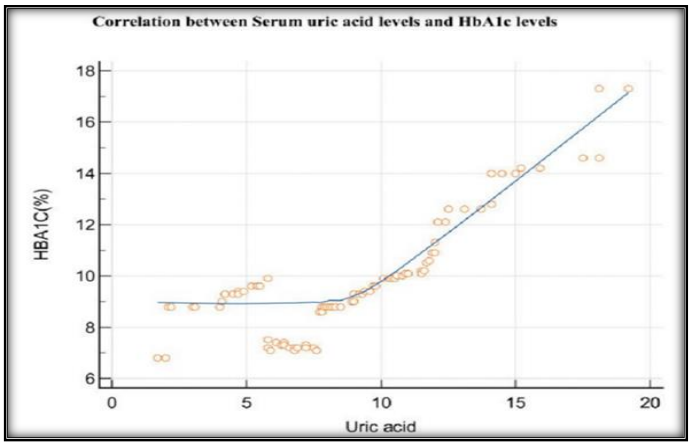
Blood samples were collected to determine fasting glucose, lipid profile, renal function tests, hemoglobin levels, serum uric acid, creatinine levels, and a complete blood count. Glomerular filtration rate (GFR) was estimated using the Cockcroft–Gault formula. Urinary albumin excretion (UAE) was measured using the immunoturbidimetric method on 24-hour urine samples, which is considered the gold standard for diagnosing microalbuminuria. Microalbuminuria was defined as UAE between 30 and 300 mg/24 hours. Participants were instructed to avoid strenuous exercise and maintain their usual diet before sample collection.

The variables studied included laboratory parameters such as fasting blood glucose, mean arterial blood pressure, serum uric acid, urinary albumin, total cholesterol, triglycerides, leukocyte count, erythrocyte count, platelet count, hemoglobin, and creatinine levels. Additional variables analyzed were type and duration of diabetes mellitus, treatment modalities, presence of comorbidities including hypertension, adherence to treatment, and behavioral factors such as alcohol intake, dietary habits, and physical activity. Mean arterial blood pressure was calculated using the formula: MABP = (SBP + 2 × DBP)/3.

For statistical analysis, data were entered in Microsoft Excel and analyzed using IBM SPSS version 16. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as percentages. Student's t-test was applied for continuous variables, while the Chi-square test and Fisher's exact test were used for categorical variables. Spearman's correlation was applied to assess the relationship between serum uric acid and urinary albumin levels. A p-value of less than 0.05 was considered statistically significant.

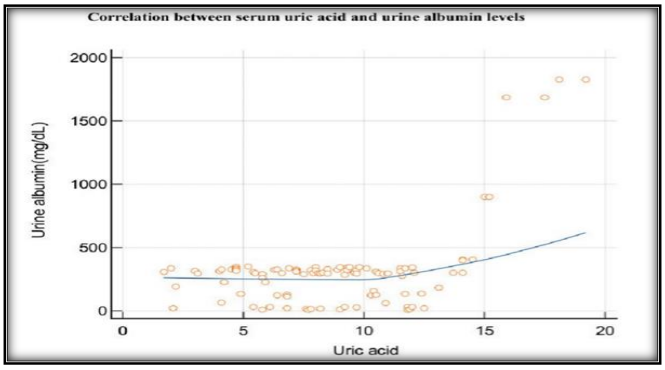
Results

The study population consisted of 100 participants, stratified into four age groups. Seventeen individuals (17%) were younger than 40 years, 27 participants (27%) were between 41 and 50 years, 34 participants (34%) were in the 51–60 age group, and 22 participants (22%) were between 61 and 70 years of age. The overall mean age was 52.01 years with a standard deviation of 10.36 years. In terms of gender distribution, 47 participants (47%) were male and 53 (53%) were female.

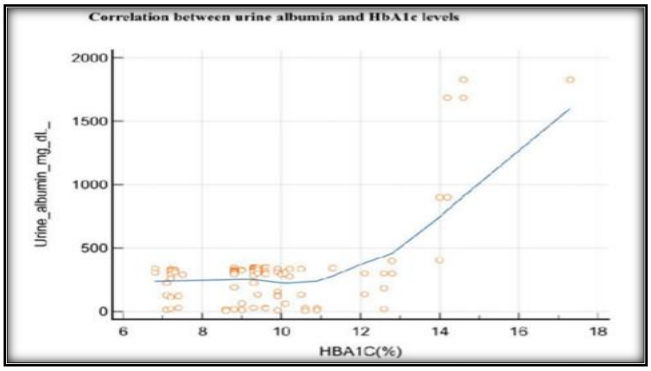


Based on body mass index (BMI), 27 individuals (27%) had values between 18.6 and 24.9 kg/m², 50 participants (50%) fell into the overweight category with a BMI of 25–29.9 kg/m², and 23 participants (23%) were obese with a BMI greater than 30 kg/m². The mean BMI was 27.80 ± 4.20 kg/m². Analysis of blood glucose values showed that fasting blood sugar (FBS) ranged from 85 to 245 mg/dL, with a mean of 151.36 ± 26.94 mg/dL, while postprandial blood sugar (PPBS) values ranged from 110 to 398 mg/dL, with a mean of 213.32 ± 60.61 mg/dL.

The duration of diabetes varied across the study population. Thirty-five participants (35%) had diabetes for less than five years, 46 participants (46%) for six to ten years, and 19 individuals (19%) for more than eleven years. The mean duration of diabetes was 7.04 ± 3.57 years. Triglyceride levels ranged from 104 mg/dL to 343 mg/dL, with a mean of 181.92 ± 50.29 mg/dL. A large proportion of participants, 74 individuals (74%), were found to have dyslipidaemia, while 26 (26%) did not.



When glycaemic control was assessed using HbA1c levels, 19 participants (19%) had good control with HbA1c <7.5%, whereas the majority, 81 individuals (81%), exhibited poor control with HbA1c >7.6%. The mean HbA1c level across the study population was 9.83 ± 2.22%. Urinary albumin analysis revealed a wide variation, with levels ranging from 6.0 mg/dL to 1826.40 mg/dL, and a mean of 331.08 ± 371.11 mg/dL. Categorically, 17 participants (17%) had normal albumin excretion (<30 mg/dL), 31 (31%) had microalbuminuria (30–300 mg/dL), and 52 (52%) had macroalbuminuria (>300 mg/dL). Serum uric acid levels also showed wide variability, ranging from 1.70 to 19.20 mg/dL, with a mean value of 8.99 ± 3.83 mg/dL.



Correlation analyses revealed significant associations between metabolic and renal parameters. Serum uric acid showed a strong positive correlation with HbA1c ($r = 0.8431$, $p < 0.0001$, 95% CI: 0.7750–0.8918) and an even stronger correlation with the duration of diabetes ($r = 0.9873$, $p < 0.0001$, 95% CI: 0.9811–0.9914). A moderate positive correlation was observed between serum uric acid and urinary albumin levels ($r = 0.5683$, $p < 0.0001$, 95% CI: 0.4185–0.6879). Similarly, urine albumin demonstrated significant correlations with HbA1c ($r = 0.6638$, $p < 0.0001$, 95% CI: 0.5374–0.7610) and with duration of diabetes ($r = 0.5743$, $p < 0.0001$, 95% CI: 0.4260–0.6926).

A comparison of serum uric acid levels between patients with good and poor glycemic control revealed significant differences. The mean serum uric acid among those with good control was 6.16 ± 1.61 mg/dL, while in those with poor control it was 9.66 ± 3.91 mg/dL. This difference was statistically significant with a p-value of 0.0002.

Discussion

In the present study, most participants were aged 51–60 years (34%), followed by 41–50 years (27%), 61–70 years (22%), and <40 years (17%), with a mean age of 52.01 ± 10.36 years. This distribution aligns with Albert et al,[9] (2022), who reported a peak at 51–60 years, and Chalak Mehrdad et al,[10] (2023), who found a mean age of 56.31 ± 7.84 years. Aladag et al,[11] (2024). Reported a slightly higher mean age of 57.8 ± 11.6 years. In this study, the sample consisted of 47% males and 53% females, which is similar to the findings of Aladag et al,[11] (38.7% male, 61.3% female) and Chalak Mehrdad et al,[10] (37.8% male, 62.2% female). Albert et al,[9] also reported a comparable gender balance within albuminuria subgroups.

Body mass index analysis revealed that 27% had a BMI of 18.6–24.9, 50% had a BMI of 25–29.9, and 23% had a BMI of>30, with a mean of 27.80 ± 4.20 kg/m². Chalak Mehrdad et al,[10] documented a higher mean BMI of 30.23 ± 4.19, while Albert et al,[9] reported a lower mean of 24.96 ± 2.06. In contrast, Aladag et al,[11] observed severe obesity with mean values of 35.1 and 40.0. Fasting blood sugar averaged 151.36 ± 26.94 mg/dL and postprandial 213.32 ± 60.61 mg/dL, values comparable to Albert et al,[9] (155.50 ± 55.13 mg/dL) and slightly higher than Chalak Mehrdad et al,[10] (142.98 ± 37.36 mg/dL). Aladag et al,[11] linked elevated glucose levels with albuminuria, although without direct FBS/PPBS values.

Duration of diabetes was <5 years in 35%, 6–10 years in 46%, and >11 years in 19%, with a mean of 7.04 ± 3.57 years. Chalak Mehrdad et al,[10] reported an average duration of 8.28 years, Aladag et al,[11] noted a range of 7.8–9.5 years across groups, and Albert et al,[9] confirmed that a longer duration correlated with worsening albuminuria. Triglycerides in this study averaged 181.92 ± 50.29 mg/dL, higher than Albert et al,[9] (129.13 ± 28.47 mg/dL) but lower than Aladag et al,[11] (229.0 ± 60.3 mg/dL in microalbuminuria). Dyslipidemia was present in 74%, consistent with other reports, as noted by Aladag et al,[11] and Chalak Mehrdad et al,[10] who both described dyslipidemic profiles, particularly reduced HDL-C.

Poor glycemic control (HbA1c >7.6%) was seen in 81%, with a mean HbA1c of 9.83 ± 2.22%, notably higher than Albert et al,[9] (6.85 ± 1.24%) and Chalak Mehrdad et al,[10] (7.45–8.09%), but

similar to Aladag et al,[11] (8.5–10.6%). Mean urine albumin was 331.08 ± 371.11 mg/dL, with 17% normoalbuminuria, 31% microalbuminuria, and 52% macroalbuminuria. Albert et al,[9] reported 49.4% normoalbuminuria, 31.3% microalbuminuria, and 19.3% macroalbuminuria, while Chalak Mehrdad et al,[10] found proteinuria values in the macroalbuminuric range. Aladag et al,[11] confirmed a significant correlation between uric acid and urinary protein. Serum uric acid averaged 8.99 ± 3.83 mg/dL, markedly higher than Albert et al,[9] (5.79 ± 1.65), Chalak Mehrdad et al,[10] (5.04–5.14), and Aladag et al,[11] (4.0–4.7). Correlation analysis showed uric acid was strongly associated with HbA1c ($r = 0.8431$, $p < 0.0001$), duration of diabetes ($r = 0.9873$, $p < 0.0001$), and urine albumin ($r = 0.5683$, $p < 0.0001$). These results mirror Albert et al,[9] who observed rising HbA1c and uric acid with albuminuria severity, Chalak Mehrdad et al,[10] who reported significant uric acid–proteinuria correlations (R = 0.738 baseline, R = 0.491 follow-up), and Aladag et al,[11] who found positive associations between HbA1c, urinary protein, and uric acid.

Urine albumin correlated with HbA1c in the present study ($r = 0.6638$, $p < 0.0001$), in line with Albert et al,[9] and Aladag et al.[11] The duration of diabetes correlated with albuminuria ($r = 0.5743$, $p < 0.0001$), consistent with Albert et al.[9] Finally, uric acid was significantly higher in poorly controlled patients (9.66 vs 6.16 mg/dL, $p = 0.0002$), confirming trends described by Albert et al,[9] Chalak Mehrdad et al,[10] and Aladag et al,[11] thereby highlighting the interconnected roles of hyperuricemia, glycemic control, and albuminuria in T2DM.

Conclusion

In conclusion, the present study highlights a strong association between elevated serum uric acid levels and both albuminuria and glycemic control in patients with type 2 diabetes mellitus (T2DM). These findings emphasize the multifactorial nature of diabetic nephropathy and suggest that hyperuricemia could serve as a potential marker for renal involvement and poor metabolic control in diabetic individuals.

Early identification and management of elevated uric acid levels, along with strict glycemic control, may help mitigate the progression of nephropathy and associated cardiovascular risks.

This study emphasizes the importance of a comprehensive metabolic evaluation in T2DM patients, focusing not only on glucose levels but also on renal biomarkers, such as albuminuria and uric acid, to improve long-term outcomes.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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