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Association of Hyperuricaemia with Urinary Albumin Excretion in Patients with Type II Diabetes Mellitus

Amit Sharma, Praveen Kumar Malik**, Rajesh Manocha***, Ravi Bhargav*****

Abstract

***Background:** Diabetes mellitus (DM) is a major global health concern, leading to numerous complications, including retinopathy, neuropathy and nephropathy. Hyperuricaemia and glycated haemoglobin (HbA1c) have been identified as potential contributors to diabetic nephropathy, yet their combined impact on urinary albumin excretion (UAE) is not fully understood. The interplay between these factors may provide critical insights into early detection and management strategies for diabetic nephropathy.*

***Objective:** This study aims to investigate the relationship between serum uric acid and UAE in patients with type II diabetes mellitus (T2DM), and to determine whether hyperuricaemia and glycaemic control influence the severity of albuminuria.*

***Methods:** A hospital-based cross-sectional study was conducted among 150 T2DM patients, measuring serum uric acid, HbA1c, and urinary albumin levels. Statistical analyses, including correlation and regression models, were used to assess relationships between these variables.*

***Results:** Higher serum uric acid level was significantly associated with increased UAE. Patients with elevated uric acid levels had a higher risk of moderately and severely increased albuminuria. A combined effect of hyperuricaemia and poor glycaemic control was observed, leading to a greater likelihood of progressive kidney dysfunction.*

***Conclusion:** Both hyperuricaemia and poor glycaemic control independently contribute to increased UAE, highlighting their importance as early indicators of diabetic nephropathy. Proactive management of uric acid levels and HbA1c may help in reducing the risk of renal complications in T2DM patients.*

***Key words:** Diabetes mellitus, hyperuricaemia, HbA1c, urinary albumin excretion, diabetic nephropathy.*

Introduction

Diabetes Mellitus (DM) is a chronic metabolic disorder characterised by persistent hyperglycaemia due to insulin resistance, insufficient insulin secretion, or both. It has emerged as a global health crisis, with increasing prevalence and significant morbidity and mortality. Among the myriad complications associated with DM, diabetic nephropathy is a leading cause of end-stage renal disease (ESRD), imposing a substantial burden on healthcare systems worldwide. Early detection and intervention are paramount to slowing disease progression and mitigating long-term renal damage[1].

One of the most commonly used markers for diabetic nephropathy is urinary albumin-creatinine ratio (UACR), which serves as an early indicator of renal dysfunction. Moderately increased albuminuria, defined as UACR between 30 and 300 mg/g, represents the initial stage of kidney damage, whereas severely increased albuminuria, exceeding 300 mg/g, suggests advanced renal impairment. Understanding the factors that contribute to increased UACR in diabetics is crucial for implementing timely and effective treatment strategies[2].

Hyperuricaemia, or elevated serum uric acid levels (>7.0 mg/dL in men and >5.7 mg/dL in women), has gained attention as a potential contributor to renal dysfunction. Uric acid has been implicated in endothelial dysfunction, oxidative stress, and inflammatory responses, all of which can exacerbate kidney damage in diabetics. Additionally, poor glycaemic control, as indicated by elevated HbA1c levels, is strongly associated with renal impairment due to the damaging effects of prolonged hyperglycaemia on the renal microvasculature. However, the combined impact of hyperuricaemia and glycaemic control on UACR remains underexplored[3].

This study aims to investigate the association between serum uric acid and urinary albumin excretion in T2DM patients. By identifying these relationships, we hope to contribute valuable insights into the early detection and management of diabetic nephropathy, ultimately improving patient outcomes.

**Junior Resident, **Professor and Head, ***Professor, ****Assistant Professor, Department of General Medicine, ESIC Medical College and Hospital, Faridabad - 121 012, Haryana.*

***Corresponding Author:** Dr Praveen Kumar Malik, Professor and Head, Department of General Medicine, ESIC Medical College and Hospital, Faridabad - 121 012, Haryana. Tel: 9811827454, E-mail: praveenmalik@gmail.com*

Methods

Study Design and Participants

This hospital-based cross-sectional study was conducted at ESIC Medical College and Hospital, Faridabad, involving T2DM patients aged 18 - 65 years. The study included individuals diagnosed with T2DM for at least one year, as confirmed by their medical history and laboratory reports. Patients with chronic kidney disease (CKD), acute infections, or those on medications influencing uric acid metabolism, such as diuretics or uricosuric agents, were excluded to minimise confounding variables.

A structured questionnaire was used to collect demographic and clinical data, including age, gender, duration of diabetes, lifestyle habits, medication history, and co-morbid conditions such as hypertension and dyslipidaemia. A thorough physical examination was performed, including measurements of blood pressure, and body mass index (BMI).

Data Collection and Laboratory Analysis

Blood and urine samples were collected. Laboratory tests were conducted using standardised protocols:

- Serum Uric Acid: Measured using reflectance spectrophotometry. Hyperuricaemia was defined as >7.0 mg/dL in men and >5.7 mg/dL in women.
- HbA1c: Assessed by using immunoturbidimetry, with a cut-off value of >6.5% for poor glycaemic control.
- Urinary Albumin Creatinine Ratio: Determined via reflectance spectrophotometry. Spot urine samples were collected. Patients were classified as normoalbuminuric (<30 mg/g), moderately increased albuminuric (30 - 300 mg/g), or severely increased albuminuric (>300 mg/g).
- Serum Creatinine and eGFR: Measured to assess baseline renal function and estimated glomerular filtration rate (eGFR).

Statistical Analysis

Descriptive statistics were used to summarise baseline characteristics. Pearson's correlation was employed to assess the association between uric acid, HbA1c, and UACR. A multiple regression model was used to adjust for confounders such as age, BMI, hypertension, and dyslipidaemia. Statistical significance was set at $p < 0.05$.

Results

Table I categorises the age distribution of a group of 150 participants. The majority of the participants were between the age of 31 - 60 years, accounting for 63.4% of the study cohort. The second largest age group was ≥60 years of age, representing 28% of the sample. Those aged ≤30 years made up 8.6% of the study group. Overall, the mean age of the group was 40.3 ± 14.5 years.

Table I: Distribution of age

Age (Years)	Frequency	% of Total
≤30	13	8.6%
31 - 60	95	63.40%
≥60	42	28%
Total	150	100.0%
Mean ± SD	40.3 ± 14.5	

$X^2 = 25.1$, $p = <0.001$

Table II: Distribution of gender

Gender	Frequency	% of Total
Male	67	44.7%
Female	83	55.3%
Total	150	100.0%

$X^2 = 1.71$, $p = 0.191$

Table II displays the gender distribution of patients with type II diabetes mellitus, showing a slight predominance of females over males. Out of 150 participants, 55.3% were females (n = 83), while males accounted for 44.7% (n = 67). This distribution indicated a minor gender imbalance, with females being more represented in the study sample. A chi-square test was conducted to examine whether this distribution was significantly different from an expected equal proportion of genders. The chi-square value ($\chi^2 = 1.71$) and the associated p-value ($p = 0.191$) indicated no statistically significant difference in gender distribution, suggesting that the observed variation could likely be due to random chance rather than a true gender disparity in the sample.

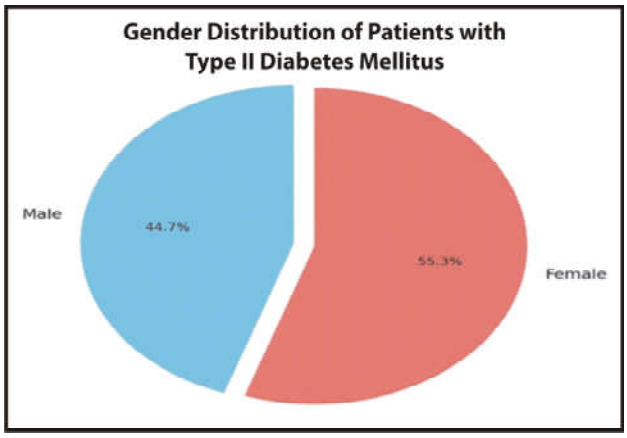

Fig. 1:

Table III: Distribution of chief complaints

Chief Complaints	Frequency	% of Total
Fatigue	31	20.66%
Weight loss	43	28.7%
Increased thirst	36	24.0%
Frequent urination	25	16.64%
Others	15	10.0%
Total	150	100.0%

$X^2 = 3.33, p = 0.343$

In the study analyzing chief complaints among 150 patients, the most common complaint reported was weight loss, affecting 43 individuals and constituting 28.7% of the total complaints. Increased thirst followed closely, reported by 36 patients, accounting for 24.0% of the complaints. Fatigue was another significant complaint, experienced by 31 patients, making up 20.66% of the total. Frequent urination was noted in 25 patients, representing 16.64% of the complaints. Other less common complaints were grouped under 'Others,' totaling 15 cases and covering 10.0% of the complaints (Table III).

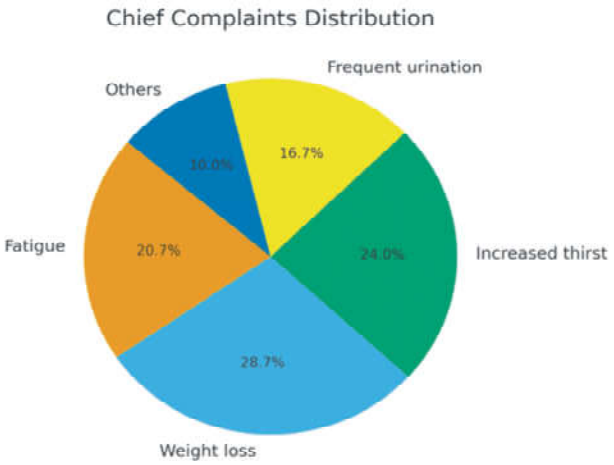

Fig. 2:

Table IV: Descriptive statistics of vital signs

	95% Confidence Interval					
Vital signs	Mean	Lower	Upper	SD	t-value	p-value
SBP (mmHg)	135.7	124	158	17.12	97.1	0.00238
DBP (mmHg)	85.1	82	98	7.15	145.9	0.00515
Pulse (/min)	79.3	73	109	12.83	75.7	0.00434
Respiratory Rate (/min)	15.6	13	16	2.34	81.3	0.00276

Table IV data represents vital signs with their respective mean values, 95% confidence intervals, standard deviations, t-values, and p-values. The systolic blood pressure (SBP) had a mean of 135.7 mmHg with a 95% confidence interval ranging from 124 to 158 mmHg, a standard deviation of 17.12, a t-value of 97.1, and a highly significant p-value of 0.00238. Diastolic blood pressure (DBP) showed a mean of 85.1 mmHg, a confidence interval from 82 to 98 mmHg, a standard deviation of 7.15, a t-value of 145.9, and a p-value of 0.00515. The pulse rate was observed at a mean of 79.3 beats per minute, with a confidence interval between 73 and 109, a standard deviation of 12.83, a t-value of 75.7, and a p-value of 0.00434. Lastly, the respiratory rate was reported with a mean of 15.6 breaths per minute, a confidence interval from 13 to 16, a standard deviation of 2.34, a t-value of 81.3, and a p-value of 0.00276. This statistical data indicates highly significant differences for all measured vital parameters.

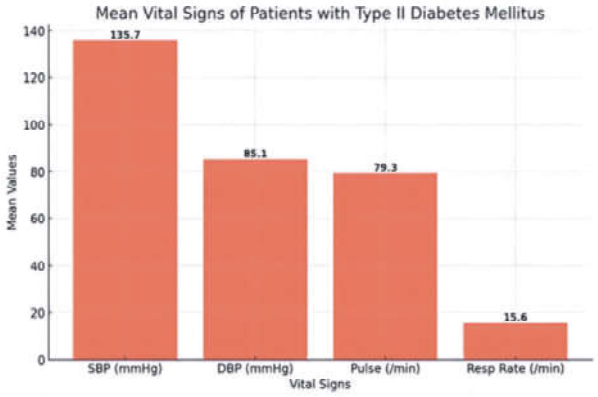

Fig. 3:

Table V: Descriptive statistics of Renal Function Tests (RFT)

	95% Confidence Interval					
RFT	Mean	Lower	Upper	SD	t-value	p-value
Blood Urea (mg/dl)	33.673	22.20	48.52	9.861	41.8	0.00532
Creatinine (mg/dl)	0.897	0.6	0.98	0.186	59	0.0012
Uric Acid (mg/dl)						
– Male	10.532	7.1	11.2	1.392	60.9	0.053
– Female	9.231	6.2	10.7	1.235	58.2	0.00123

Table V depicts the assessment of renal functions through blood tests, and the results show the following statistics within a 95% confidence interval. For blood urea, the mean level was 33.673 mg/dL, ranging from 22.20 to 48.52 mg/dL with a standard deviation (SD) of 9.861, a t-value of 41.8, and a p-value of 0.00532, indicating statistical significance. Creatinine level had a mean of 0.897 mg/dL, with a lower limit of 0.6 mg/dL and an upper limit of 0.98 mg/dL, an SD of 0.186, a t-value of 59, and a highly significant p-value of 0.0012. Uric

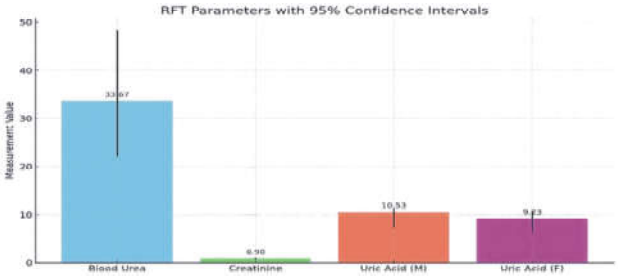

Fig. 4:

acid levels varied by gender; males had a mean of 10.532 mg/dL with a range of 7.1 to 11.2 mg/dL, an SD of 1.392, a t-value of 60.9, and a p-value of 0.053, suggesting marginal significance. In contrast, females had a mean uric acid level of 9.231 mg/dL, ranging from 6.2 to 10.7 mg/dL, an SD of 1.235, a t-value of 58.2, and a p-value of 0.00123, also indicating statistical significance.

Table VI: Descriptive statistics of glycaemic control

		95% Confidence Interval				
Glycaemic Control	**Mean**	**Lower**	**Upper**	**SD**	**t-value**	**p-value**
HbA1C (%)	7.64	7.6	9.8	1.53	53.2	0.0017
RBS (mg/dl)	295.53	286	347	56.6	63.9	0.0053
FBS (mg/dl)	190.85	180	221	33.38	70	0.0062

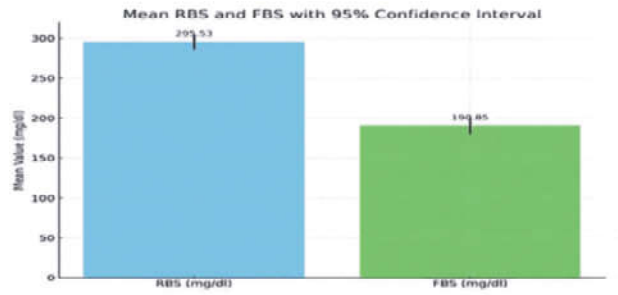

Fig. 5:

Table VI describes the glycaemic control parameters, as measured in the study, exhibiting significant differences. The mean haemoglobin A1C (HbA1C) was 7.64% with a 95% confidence interval (CI) ranging from 7.6% to 9.8%, standard deviation (SD) of 1.53%, and a highly significant t-value of 53.2 (p - 0.0017). Random blood sugar (RBS) levels had a mean of 295.53 mg/dL, with a 95% CI of 286 to 347 mg/dL, SD of 56.6 mg/dL, and a t-value of 63.9 (p 0.0053). Fasting blood sugar (FBS) levels were averaged at 190.85 mg/dL, with a 95% CI from 180 to 221 mg/dL, SD of 33.38 mg/dL, and a t-value of 70 (p-0.0062), indicating strong statistical significance in all measurements of glycaemic control within the study.

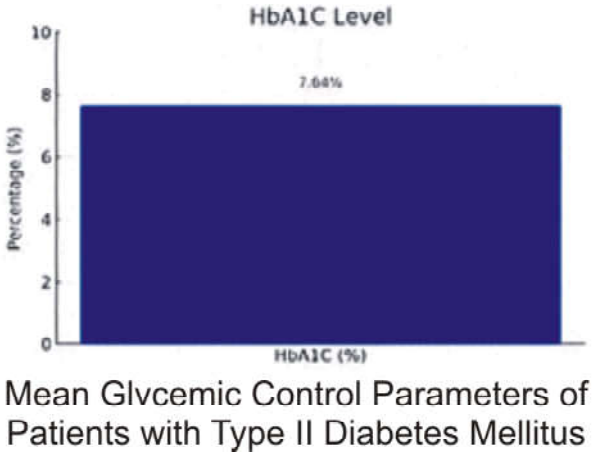

Fig. 6:

Table VII: Distribution of UACR category with age

Age (years) with frequency	**Normal Albuminuria**	**Moderately Increased Albuminuria**	**Severely Increased Albuminuria**
<30 (13)	7	4	2
30 - 60 (95)	22	44	29
>60 (42)	2	4	36
Total = 150	31	52	67

$X^2 = 29.0$, $p = <0.001$.

Table VII categorises albuminuria into three UACR categories: normal albuminuria, moderately increased albuminuria, and severely increased albuminuria. Normal albuminuria was observed in 31 patients, accounting for 20.66% of the total. Moderately increased albuminuria was more frequent, observed in 52 patients, comprising 34.67% of the total. The highest frequency was seen in severely increased albuminuria, with 67 patients, making up 44.67% of the total. among the studied group. The highest frequency of

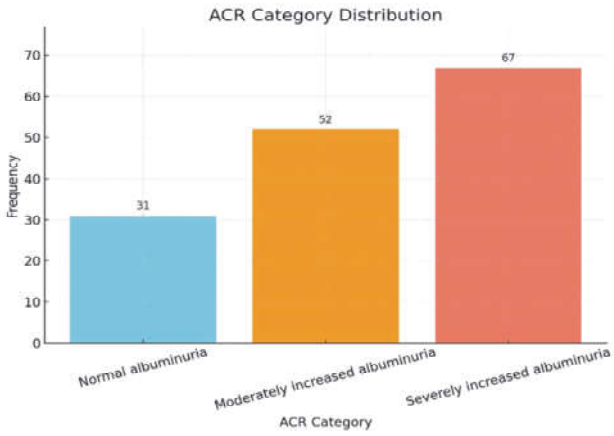

Fig. 7:

moderately and severely increased albuminuria was observed in age group 30 - 60 yrs and >60 yrs, respectively.

Table VIII depicts the logistic regression analysis, and the impact of various predictors on the urinary albumin creatinine ratio (UACR). The haemoglobin A1c (HbA1C) levels had a positive association with an increase in the UACR, with an odds ratio (OR) of 1.57 (95% CI: 1.22, 2.02) per percentage increase, indicating a statistically significant effect (p <0.001). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) also showed positive effects with ORs of 1.02 (95% CI: 1.00, 1.04) and 1.03 (95% CI: 1.01, 1.05) per mmHg increase respectively, achieving significance at p-values of 0.045 and 0.021. Age demonstrated a positive effect on UACR category with an OR of 1.05 per year increase (95% CI: 1.01, 1.09; p = 0.012). Creatinine levels were strongly associated with UACR category, with an OR of 2.41 (95% CI: 1.43, 4.05) per mg/dL increase (p <0.001). Uric acid levels also contributed positively to the UACR category with an OR of 1.36 (95% CI: 1.02, 1.82) per mg/dL increase (p = 0.038).

Table VIII: Impact of various predictors on UACR using logistic regression

Model Co-efficients - UACR Category

						95% Confidence Interval	
Predictor	**Estimate**	**SE**	**Z**	**P**	**Odds ratio**	**Lower**	**Upper**
HbA1C (%)	0.45	0.12	3.75	<0.001	1.57	1.22	2.02
SBP (mmHg)	0.02	0.01	2.00	0.045	1.02	1.00	1.04
DBP (mmHg)	0.03	0.01	2.30	0.021	1.03	1.01	1.05
Age	0.05	0.02	2.50	0.012	1.05	1.01	1.09
Creatinine (mg/dL)	0.88	0.26	3.38	<0.001	2.41	1.43	4.05
Uric Acid (mg/dL)	0.31	0.15	2.07	0.038	1.36	1.02	1.82

SE: Standard Error, Z: Z-Value, p = p-Value

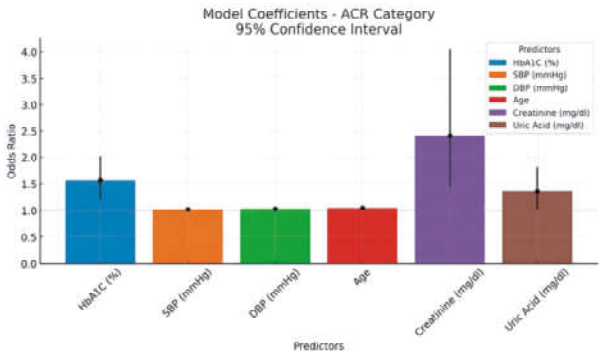

Fig. 8:

Discussion

The present study demonstrated a significant association between age and urinary albumin excretion in type II diabetes mellitus patients. The highest frequency of severely increased albuminuria was observed in the >60 year age group, accounting for 36 out of the 150 participants. Additionally, the 30 - 60 year age group also showed considerable moderately increased albuminuria, i.e., 44 out of 150 patients. The mean age of participants was 40.3 ± 14.5 years. Statistical analysis highlighted this age-related trend in albumin excretion, evidenced by a significant chi-square value of 29.0 and a p-value of less than 0.001. Neupane *et al*[4] reported a mean age of 58.94 years with a standard deviation of 13.80 years among their diabetic cohort. Their study primarily focused on older individuals compared to our study, where a broader age range was considered. Despite the differences in age distribution, both studies underscore the prevalence of kidney-related complications in older diabetic patients. Kaushal *et al*[5] analysed 100 patients with a mean age of 57.64 years and a standard deviation of 10.07, with age ranging from 40 to 80 years. This narrower age range and higher mean age compared to our study indicates focus on a relatively old demography. Similar to our findings, Kaushal *et al* study observed the increased renal stress among older diabetic patients, though direct comparisons on urinary albumin excretion were limited without specific albuminuria data from their study.

The present study identified a gender distribution among type II diabetes mellitus patients with 55.3% female (83/150) and 44.7% male (67/150). The chi-square analysis showed no significant gender difference in urinary albumin excretion, with a value of 1.71 and a p-value of 0.191. Neupane *et al*[4] reported a gender distribution of 58% male (29 participants) and 42% female (21 participants) in their diabetic cohort. This distribution differs slightly from our study, where a higher proportion of females was noted. Kaushal *et al*[5] analysed a cohort where males comprised

52% and females 48%, which is more balanced compared to our study but still shows a slight male predominance, contrasting with our female-majority findings.

The present study investigated chief complaints among 150 type II diabetes mellitus (T2DM) patients, revealing that increased thirst (24.0%) and weight loss (28.7%) were the most common, followed by fatigue (20.66%), frequent urination (16.64%) and other less common were grouped under "others" covering 10% of the complaints. A chi-square test yielded a value of 3.33 with a p-value of 0.343, indicating no significant variation in the distribution of these symptoms among the participants. Wongkongkam *et al*[6] assessed clinical presentations in T2DM patients with and without peripheral arterial disease (PAD). Among their participants, those with PAD often reported intermittent claudication (70.4%), while a significant majority of those without PAD had no symptoms (90%). This contrasts starkly with the frequent general diabetic symptoms like fatigue and weight loss noted in our study. Their chi-square analysis showed a highly significant difference in symptom presentation ($p \leq 0.001$), emphasizing the impact of PAD on symptom severity and type in diabetic patients. Hamiel and Zeitler[7] detailed the clinical presentations of type 1 and type 2 diabetes in children. Common symptoms for both T1DM and T2DM included polydipsia, polyuria, and polyphagia. T2DM in children was often diagnosed incidentally and associated with obesity, acanthosis nigricans, and more commonly, co-morbidities like hypertension and dyslipidaemia. While their study focuses on a younger demography and differentiates by diabetes type, the presence of symptoms such as polydipsia and polyuria align with our findings in an adult T2DM population.

The present study assessed the vital parameters of patients with type II diabetes mellitus, establishing significant findings for systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse rate, and respiratory rate. The mean SBP was reported at 135.7 mmHg with a confidence interval (CI) of 124 to 158 mmHg, and a standard deviation (SD) of 17.12 mmHg. DBP averaged 85.1 mmHg with a CI of 82 to 98 mmHg and an SD of 7.15 mmHg. The pulse rate was 79.3 per minute with a CI of 73 to 109 per minute and an SD of 12.83 per minute, while the respiratory rate was 15.6 per minute with a CI of 13 to 16 per minute and an SD of 2.34 per minute. Each parameter showed a highly significant t value (SBP: 97.1, DBP: 145.9, Pulse: 75.7, Resp Rate: 81.3) with p-values of 0.00238, 0.00515, 0.00434, and 0.00276, respectively, indicating robust statistical significance across all measures. Kaushal *et al*[5] reported mean SBP and DBP values of 128.96 mmHg (SD 14.62) and 77.1 mmHg (SD 7.53), respectively, in a cohort of 100 participants. The maximum and minimum reported SBP values were 160 and 110 mmHg, while for DBP, they were 90 and 60 mmHg. These results show lower average blood pressures compared to our findings, possibly indicating differences in demographic characteristics or disease management strategies between the cohorts. Lai *et al*[8] examined systolic and diastolic blood pressures in participants with and without albuminuria. Their findings showed mean SBP values of 133.91 mmHg (SD 16.20) for those without albuminuria and 134.80 mmHg (SD 15.77) for those with albuminuria, with a hazard ratio of 1.01 (95% CI: 0.98 - 1.04, $p = 0.40$). For DBP, the means were 77.57 mmHg (SD 10.85) without albuminuria and 76.45 mmHg (SD 11.06) with albuminuria, with a hazard ratio of 0.99 (95% CI: 0.96 - 1.04, $p = 0.72$). These blood pressure values are also lower than those observed in our study, and the statistical analysis did not demonstrate significant differences associated with albuminuria, contrasting with the significant trends observed in our data.

The present study assessed renal function tests (RFT) in patients with type II diabetes mellitus, showing significant results for blood urea, creatinine, and uric acid levels. The mean values were 33.673 mg/dL for blood urea with a confidence interval (CI) of 22.20 to 48.52 and a standard deviation (SD) of 9.861, creatinine was 0.897 mg/dL with a CI of 0.6 to 0.98 and an SD of 0.186, and uric acid in males 10.532 mg/dL with a CI of 7.1 to 11.2, SD of 1.392 and in females mean 9.231 with a CI of 6.2 to 10.7 and a SD of 1.235. All parameters showed high statistical significance with t-values exceeding 40 and p-values of 0.00532, 0.0012, 0.053, and 0.00123, respectively. Qin *et al*[9] examined serum uric acid (SUA) levels in a cohort differentiated by gender, reporting mean SUA of 303.6 μmol/L in total, with males at 321.4 μmol/L and females at 275.8 μmol/L. The study highlighted significant differences between genders ($p < 0.0001$). The uric acid level reported by Qin *et al*[9] when converted to mg/dL (divide by 59.48 to convert μmol/L to mg/dL) would be approximately 5.10 mg/dL for the total group, which is lower compared to the uric acid levels found in our study. Nikolaidou *et al*[10] analysed urea, creatinine, and uric acid in diabetic patients with recent onset, where urea averaged 31.2 mg/dL (± 6.9 SD), creatinine 0.88 mg/dL (±0.16 SD), and uric acid 5.4 mg/dL (± 1.7 SD). Their findings are comparable to ours, although the creatinine and uric acid levels in our study are slightly higher. No significant differences were found in urea and creatinine levels between hypertensive and normotensive diabetic patients, nor in uric acid levels (p-values: urea = 0.927, creatinine = 0.114, uric acid = 0.458).

The present study evaluated glycaemic control in patients with type II diabetes mellitus, achieving significant findings in HbA1C, random blood sugar (RBS), and fasting blood sugar (FBS). The study reported a mean HbA1C of 7.64%

with a confidence interval (CI) of 7.6% to 9.8% and a standard deviation (SD) of 1.53%, RBS at 295.53 mg/dL (CI: 286 to 347, SD: 56.6), and FBS at 190.85 mg/dL (CI: 180 to 221, SD: 33.38). All parameters indicated highly significant statistical values with t-values over 50 and p-values 0.0017, 0.0053 and 0.0062, respectively. Bonakdaran *et al*[11] reported higher mean values in their diabetic cohort with a fasting blood glucose of 191.32 mg/dL (SD: 66.25) and an HbA1C of 8.68% (SD: 1.96), both higher than in our study. This suggests worse glycaemic control in their sample as compared to ours, where the mean HbA1C was notably lower at 7.64%. Neupane *et al*[4] also found a higher average of HbA1C at 8.12% with an SD of 2.14%, which again indicates less effective glycaemic control compared to our study's findings. The higher variability in their data suggests a broader range of control among participants. Sabzghabaei and Rajabian[12] reported mean fasting blood glucose at 139.84 mg/dL (SD: 38.4) and HbA1C at 7.41% (SD: 1.41), which are both lower than in our study. Their lower FBS and HbA1C readings indicate better average glycaemic control among their participants compared to our study. These comparative insights underline the variability in diabetes management effectiveness across different populations, stressing the importance of tailored treatment approaches to optimise glycaemic control.

In the present study, we analysed the distribution of albuminuria among 150 patients with type II diabetes mellitus, categorizing them based on albumin creatinine ratio (ACR). The results demonstrated that 20.66% (31 patients) had normal albuminuria, while 34.67% (52 patients) exhibited moderately increased albuminuria and severely increased albuminuria was in 44.67% (67 patients). The chi-square statistic was significant at 29.0 with a p-value of less than 0.001, indicating a significant distribution of albuminuria categories among the participants. Kaushal *et al*[5] categorised diabetic patients into groups based on their ACR levels. They reported a mean urinary ACR of 22.3 µg/mg (SD: 4.53, range: 14 - 30) for the normoalbuminuria group (46 patients), 144.6 µg/mg (SD: 71.11, range: 56.2 - 380) for the moderately increased albuminuria group (33 patients), and 421.3 µg/mg (SD: 150.33, range: 51.8 - 750) for the severely increased albuminuria group (21 patients). The higher ACR values in their moderately increased albuminuria and severely increased albuminuria groups compared to our study highlight more pronounced renal involvement. These studies collectively underscore the significance of assessing albuminuria in diabetes management, demonstrating variations in kidney health across different diabetic cohorts and reinforcing the importance of early detection and intervention.

The present study investigated the association between several predictors and albuminuria category in patients with type II diabetes mellitus using logistic regression analysis. Significant findings were observed for the intercept and HbA1C, with the latter showing a substantial influence on the odds of having albuminuria: for each percentage increase in HbA1C, the odds of albuminuria increased by approximately 1.57 times (Odds Ratio: 1.57, CI: 1.22 - 2.02, p <0.001). SBP and DBP also showed positive effects with ORs of 1.02 (95% CI: 1.00, 1.04) and 1.03 (95% CI: 1.01, 1.05) per mmHg increase respectively, achieving significance at p-values of 0.045 and 0.021. Age demonstrated a positive effect on ACR category with an OR of 1.05 per year increase (95% CI: 1.01, 1.09; p = 0.012). Creatinine levels were strongly associated with ACR category, with an OR 2.41 (95% CI: 1.43, 4.05) per mg/dL increase (p <0.001). Uric acid levels also contributed positively to ACR Category with an OR of 1.36 (95% CI: 1.02, 1.82) per mg/dL increase (p = 0.038). These predictors showed significant associations with albuminuria in this cohort.

In patients with elevated levels of uric acid (UA), there is often an associated increase in urinary albumin-to-creatinine ratio (UACR), indicating a potential for kidney damage or disease progression. Similarly, heightened levels of HbA1C, a marker for long-term glycaemic control, are linked to increased UACR, reflecting a higher risk of diabetic kidney disease. When both uric acid and HbA1C levels are elevated, the impact on UACR is even more pronounced. This dual elevation exacerbates the stress on the kidneys, potentially accelerating the pathogenesis of kidney damage. Therefore, monitoring and managing both uric acid and HbA1C levels in patients are crucial for mitigating the risk of renal complications, especially in those with predisposing conditions such as diabetes and hyperuricaemia.

Conclusion

This study highlights a significant association between hyperuricaemia, glycaemic control, and urinary albumin excretion in T2DM patients. Elevated serum uric acid and HbA1c independently contribute to increased UAE, suggesting their potential role as early biomarkers of diabetic nephropathy. The combined effect of hyperuricaemia and poor glycaemic control accelerates renal dysfunction, emphasizing the need for comprehensive management strategies targeting both metabolic and renal parameters.

From a clinical perspective, early screening for serum uric acid and HbA1c levels in diabetic patients could facilitate timely interventions, potentially delaying or preventing the onset of diabetic nephropathy. Given that hyperuricaemia has been linked to endothelial dysfunction and oxidative stress, uric acid-lowering therapies such as allopurinol or febuxostat may serve as adjuncts to conventional

nephroprotective strategies. Additionally, strict glycaemic control through lifestyle modifications and pharmacologic interventions remain paramount in mitigating kidney damage.

Despite these promising findings, certain limitations should be acknowledged. The study's cross-sectional design precludes establishing causality, and the relatively small sample size necessitates larger-scale longitudinal studies for validation. Moreover, factors such as dietary patterns, genetic predisposition, and inflammatory markers warrant further exploration to elucidate their role in the observed associations.

In conclusion, integrating serum uric acid and HbA1c measurements into routine diabetic care may enhance early detection and personalised management of diabetic nephropathy. Future research should aim to establish causative relationships and evaluate the efficacy of targeted interventions in improving renal outcomes in T2DM patients.

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