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RESEARCH ARTICLE

Association between Serum Uric Acid levels and Cardiovascular risk factors in type 2 diabetes mellitus

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ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) is a prevalent and growing health concern in Bangladesh, contributing significantly to the burden of cardiovascular diseases (CVD). Elevated serum uric acid (SUA) levels have been linked to several metabolic disorders, including diabetes and cardiovascular complications. However, the relationship between SUA levels and cardiovascular risk factors in T2DM patients in Bangladesh is underexplored. This study aimed to assess the association between SUA levels and key cardiovascular risk factors in T2DM patients.

Methods: This analytical cross-sectional study was conducted from January to December 2023 with 120 participants (90 T2DM patients and 30 controls). Sociodemographic and clinical data, including BMI, waist-hip ratio (WHR), blood pressure, lipid profile, and diabetes duration, were collected. Serum uric acid levels were measured using the phosphotungstic acid method. Data were analyzed with IBM SPSS version 21, using independent t-tests and chi-square tests for comparisons between groups. Results: The mean SUA level was significantly higher in the diabetic group (5.88 ± 1.04 mg/dL) compared to controls (4.13 ± 0.85 mg/dL). A positive correlation was found between SUA levels and BMI ($r = 0.45$) and WHR ($r = 0.38$). Diabetic patients with hypertension had significantly higher SUA levels (6.60 ± 1.10 mg/dL) than non-hypertensive patients (4.79 ± 1.18 mg/dL, $P < 0.05$). SUA levels also increased with diabetes duration, with the highest levels in patients with diabetes for 9-12 years (6.47 ± 1.07 mg/dL).

Conclusion: This study found that serum uric acid levels were significantly higher in T2DM patients and positively correlated with BMI, WHR, hypertension, and dyslipidemia. SUA levels increased with the duration of diabetes, highlighting its potential role in predicting cardiovascular risk. Regular monitoring of SUA can be useful in managing T2DM patients to prevent cardiovascular complications.

Keywords: Type 2 Diabetes Mellitus; Serum Uric Acid; Cardiovascular risk factors; Bangladesh; hypertension

1. Introduction

Non-communicable diseases (NCDs), particularly type 2 diabetes mellitus (T2DM) and cardiovascular diseases (CVDs), are among the leading causes of morbidity and mortality globally[1]. According to the World

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Health Organization, over 77% of all NCD-related deaths occur in low- and middle-income countries[2], with South Asia bearing a disproportionate share of this burden[3]. In Bangladesh, the prevalence of T2DM and CVDs has been steadily rising due to rapid urbanization, sedentary lifestyles, high-calorie diets, and limited access to preventive healthcare[4]. Current estimates suggest that more than 10% of Bangladeshi adults are living with diabetes, and this figure is expected to double within the next two decades if no interventions are made[5].

T2DM is a chronic metabolic disorder characterized by insulin resistance and hyperglycemia, both of which are central to the development of metabolic syndrome. This syndrome encompasses a cluster of interrelated risk factors—abdominal obesity, hypertension, hypertriglyceridemia, low HDL cholesterol, and elevated fasting blood glucose—that collectively increase the risk of atherosclerosis, coronary artery disease (CAD), stroke, and other cardiovascular complications[6,7]. These risk factors often coexist and act synergistically, accelerating the progression of vascular damage and increasing overall cardiovascular risk.

In recent years, growing attention has been directed toward serum uric acid (SUA) as a novel, modifiable biomarker of cardiovascular risk. Hyperuricemia, defined as elevated SUA levels, has traditionally been linked to gout[8]. However, emerging evidence suggests that SUA may be involved in the pathogenesis of insulin resistance, endothelial dysfunction, oxidative stress, and inflammation—key mechanisms contributing to cardiovascular disease. Studies have demonstrated that elevated SUA may independently predict adverse cardiovascular outcomes, including myocardial infarction, heart failure, stroke, and renal impairment in patients with T2DM[9,10]. Biochemically, SUA is the final breakdown product of purine metabolism, primarily excreted by the kidneys. In individuals with insulin resistance, renal handling of uric acid may be impaired, resulting in its accumulation[11]. Additionally, increased adiposity and systemic inflammation common in diabetic populations further contribute to elevated SUA levels[12]. SUA has also been shown to correlate positively with body mass index (BMI), waist-hip ratio (WHR), low-density lipoprotein (LDL), and triglycerides, and negatively with high-density lipoprotein (HDL), reinforcing its role as both a consequence and contributor to metabolic dysregulation[13].

While many international studies have examined the link between SUA and cardiovascular risk in diverse populations, data specific to South Asian and Bangladeshi cohorts remain limited[14]. South Asians, including Bangladeshis, are known to develop T2DM and CVD at lower BMI thresholds compared to Western populations, with more prominent central obesity and genetic predisposition to insulin resistance. This underlines the importance of region-specific research to identify culturally and clinically relevant risk factors[15,16].

In Bangladesh, existing studies on SUA have primarily focused on its association with gout or renal disease, with limited exploration of its role in cardiometabolic risk among diabetic individuals. Given the country's rising prevalence of diabetes and cardiovascular conditions, and the potential of SUA to serve as an early and accessible marker of cardiovascular risk, this area warrants urgent investigation. Therefore, the present study was undertaken to evaluate the association between serum uric acid levels and cardiovascular risk factors

2. Materials and methods

2.1. Study settings and design

This analytical cross-sectional study was conducted at a tertiary care hospital in Dhaka, Bangladesh, within the Department of General Medicine and the Department of Biochemistry. The study aimed to explore

the relationship between serum uric acid levels and cardiovascular risk factors in individuals diagnosed with Type 2 Diabetes Mellitus (T2DM). The study took place over the course of one year, from January 2023 to December 2023. A total of 120 participants were enrolled, consisting of 90 patients with Type 2 Diabetes Mellitus and 30 healthy controls.

2.2. Study procedure

Upon obtaining informed written consent from each participant, data were systematically collected through a structured proforma. Socio-demographic data including age, gender, occupation, and socioeconomic status were recorded. Clinical parameters such as the duration of diabetes, family history of diabetes, smoking habits, and the presence of other cardiovascular risk factors were also documented. A comprehensive physical examination was performed, including anthropometric measurements: body weight, height, body mass index (BMI), and waist-to-hip ratio (WHR). Blood pressure measurements (both systolic and diastolic) were taken using a standard mercury sphygmomanometer. A 10 ml venous blood sample was collected after an overnight fast for laboratory investigations. These included the measurement of fasting blood glucose (FBG), postprandial blood glucose (PPBG), serum lipid profile (total cholesterol, HDL, LDL, and triglycerides), blood urea, and serum creatinine.

2.3. Estimation of Serum Uric Acid

Serum uric acid levels were determined using the phosphotungstic acid method. In the first step, the serum sample was deproteinized by adding 1 ml of distilled water, 0.2 ml of serum, 0.4 ml of 2/3 N sulfuric acid (H_2SO_4), and 0.4 ml of 10% sodium tungstate. The mixture was centrifuged for 5 minutes to separate the proteins. In the second step, 1 ml of the filtrate was mixed with 0.75N sodium hydroxide and 0.5 ml of saturated picric acid. After an incubation of 15 minutes, the absorbance was measured at 490 nm using a colorimeter. The uric acid concentration was calculated based on the color intensity[17].

2.4. Operational definitions

❖ Type 2 Diabetes Mellitus was defined according to the American Diabetes Association (ADA) criteria as[18]:

- Fasting Plasma Glucose (FPG) ≥ 126 mg/dL (7.0 mmol/L) after at least 8 hours of fasting, or
- Casual Plasma Glucose ≥ 200 mg/dL (11.1 mmol/L) with symptoms of hyperglycemia (e.g., polyuria, polydipsia, and unexplained weight loss), or
- Oral Glucose Tolerance Test (OGTT): 2-hour plasma glucose ≥ 200 mg/dL (11.1 mmol/L) after a 75g glucose load.

❖ Hyperuricemia was defined as serum uric acid levels exceeding:

- >7.0 mg/dL in men, and
- >6.0 mg/dL in women, based on established clinical criteria.

2.5. Hyperuricemia

Hyperuricemia refers to elevated levels of uric acid in the blood. In this study, hyperuricemia was considered a potential risk factor for cardiovascular disease, gout, and metabolic syndrome. Uric acid, a product of purine metabolism, can form crystals when present in excessive amounts, which may lead to inflammation and tissue damage. Elevated levels of uric acid are often associated with chronic conditions such

as Type 2 diabetes and hypertension. As per the established criteria, hyperuricemia was defined as serum uric acid levels greater than 7.0 mg/dL in men and greater than 6.0 mg/dL in women[19].

2.6. Inclusion and exclusion criteria

Inclusion Criteria:

- Patients diagnosed with Type 2 Diabetes Mellitus (regardless of the duration or glycemic status).
- Age >40 years.
- Both male and female participants.

Exclusion Criteria:

Patients were excluded from the study if they met any of the following conditions:

- Renal failure (serum creatinine > 2.0 mg/dL).
- Long-term use of diuretics or steroids.
- Regular alcohol consumption (more than 3-4 drinks per week).
- Use of chemotherapy or antimetabolite drugs.
- Hepatic disorders.
- Peripheral vascular disease, cerebrovascular disease, or pulmonary tuberculosis.
- Renal transplant recipients.
- Pregnant or lactating women.

2.7. Statistical analysis

Descriptive statistics were used to summarize the data. For continuous variables, mean and standard deviation (SD) were used, while for categorical variables, frequency and proportions were calculated. To compare the data between the diabetic group and control group, independent sample t-tests were used for continuous variables, and Chi-square tests were used for categorical variables. A p-value < 0.05 was considered statistically significant. All data were analyzed using IBM SPSS version 21.

3. Results

A total of 90 diabetic subjects and 30 healthy controls were included in the study. Both the diabetic and control groups were comparable in terms of sociodemographic and anthropometric parameters. The mean age of the participants was 58.5 years in the diabetic group and 56.0 years in the control group. No significant differences were observed in the age, gender distribution, or baseline anthropometric parameters (Body Mass Index, Waist-Hip Ratio) between the two groups.

This **Table 1** compares the baseline sociodemographic and clinical parameters between the diabetic and control groups. As seen, the diabetic group had significantly higher values for BMI, Waist-Hip Ratio (WHR), Fasting Blood Sugar (FBS), Postprandial Blood Sugar (PBS), and serum uric acid (SUA) compared to the control group. The mean SUA for diabetic subjects ranged from 3.1 to 8.5 mg/dL, while it was significantly lower in controls (2.5 to 5.0 mg/dL).

Table 1. Comparison of cases and controls with respect to baseline parameters

Parameter	Cases (N=90)	Controls (N=30)
Gender	M=54, F=36	M=14, F=16
Mean Age (years)	58.5 ± 7.2	56.0 ± 8.0
BMI (kg/m²)	26.1 ± 4.1	23.0 ± 3.4
WHR	0.77 ± 0.06	0.72 ± 0.05
FBS (mg/dL)	120.4 ± 28.3	94.2 ± 15.1
PBS (mg/dL)	192.6 ± 45.1	141.3 ± 27.5
SUA (mg/dL)	3.1 to 8.5	2.5 to 5.0

This **Table 2** highlights the comparison of cardiovascular risk factors between the diabetic and control groups. The family history of CAD and hypertension did not show a significant difference between the two groups ($P > 0.05$). However, smoking was more prevalent in the diabetic group (42.59%) compared to the control group (20.00%), with a statistically significant P-value of 0.032. This could indicate a higher risk of smoking as a comorbidity in diabetic patients.

Table 2. Analysis of cases and controls in relation to Selected Cardiovascular risk factors

Risk Factor	Cases (N=90)	Controls (N=30)	P-value
Family History of CAD	15 (16.67%)	7 (23.33%)	0.343
Smoking (among males)	23 (42.59%)	6 (20.00%)	0.032
Hypertension	18 (20%)	8 (26.67%)	0.391

This **Table 3** shows the prevalence of hyperuricemia in the diabetic and control groups. Hyperuricemia was present in 11.11% of the diabetic patients, with serum uric acid levels exceeding 7.0 mg/dL for men and 6.0 mg/dL for women. Interestingly, no control subjects had hyperuricemia. This emphasizes the higher likelihood of hyperuricemia among individuals with diabetes.

Table 3. Association between Hyperuricemia and Diabetes Mellitus

Hyperuricemia	Cases (N=90)	Controls (N=30)
	Frequency (%)	Frequency (%)
Positive	10 (11.11%)	0 (0%)
Negative	80 (88.89%)	30 (100%)

This **Table 4** illustrates the association between duration of diabetes and serum uric acid levels. As the duration of diabetes increased, there was a significant rise in serum uric acid levels. Diabetic subjects with 9-12 years of diabetes had a significantly higher mean SUA (6.60 ± 1.08 mg/dL) compared to those with 2-4 years and 5-8 years of diabetes. The differences were statistically significant ($P = 0.001$), indicating that prolonged duration of diabetes is associated with higher serum uric acid levels (Tables 4).

Table 4. Duration of diabetes and hyperuricemia

Duration of Diabetes (Years)	No. of Patients	Hyperuricemia (mg/dL)		P-value
		Mean	SD	
2-4	15	4.35	0.76	0.356
5-8	40	4.68	1.02	0.001
9-12	35	6.60	1.08	0.001

This **table 5** presents the association between serum uric acid levels and various cardiovascular risk factors in diabetic patients. The mean SUA was significantly higher in diabetic subjects with hypertension (6.58 ± 1.10 mg/dL) and lipid profile abnormalities (6.45 ± 1.05 mg/dL), both showing highly significant differences (P = 0.0001). However, no significant difference was observed between those with ischemic heart disease and those with myocardial infarction in terms of their serum uric acid levels (P = 0.391).

Table 5. Association between Serum Uric Acid levels and other Cardiovascular risk factors among diabetic subjects

Parameter	No. of Cases	SUA (mg/dL)		P-value
		Mean	SD	
Hypertension	Yes (18)	6.58	1.10	0.0001
	No (72)	4.83	1.19	
Lipid Profile Abnormality	Yes (30)	6.45	1.05	0.0001
	No (60)	4.70	1.13	
Ischemic Heart Disease	Yes (16)	6.78	1.19	0.391
Myocardial Infarction	Yes (7)	7.12	0.92	

This **Table 6** demonstrates that serum uric acid has a moderate positive correlation with BMI, WHR, total cholesterol, LDL, and triglycerides among diabetic patients. There is a negative correlation with HDL cholesterol, suggesting that higher SUA is associated with adverse lipid profiles and obesity-related parameters.

Table 6. Correlation of Serum Uric Acid with Cardiovascular risk parameters

Parameter	Pearson's Correlation Coefficient (r)	P-value
Body Mass Index (BMI)	0.45	0.002
Waist-Hip Ratio (WHR)	0.38	0.004
Total Cholesterol	0.41	0.003
LDL	0.36	0.006
Triglycerides	0.42	0.002
HDL	-0.28	0.031

This **table 7** shows that serum uric acid levels increase progressively with higher BMI categories. Obese patients had significantly higher SUA levels compared to normal-weight individuals (P = 0.001), indicating a strong link between obesity and hyperuricemia in diabetic populations.

Table 7. Serum Uric Acid levels by BMI categories in diabetic patients

BMI Category	No. of Patients	Mean SUA (mg/dL)	Standard Deviation	P-value
Normal (<24.9)	25	4.21	0.85	
Overweight (25–29)	40	5.35	0.92	0.001
Obese (≥30)	25	6.49	1.10	

4. Discussion

The findings demonstrate a strong and statistically significant association between elevated SUA levels and key metabolic and cardiovascular risk markers, including body mass index (BMI), waist-hip ratio (WHR), lipid abnormalities, hypertension, and duration of diabetes. These associations suggest that SUA may be a relevant and independent biomarker for assessing cardiovascular risk in diabetic patients. A notable finding of the study was that hyperuricemia was present in 11.11% of the diabetic subjects, while none of the controls (0%) exhibited elevated SUA levels (Table 3). The SUA range in diabetics was 3.1 to 8.5 mg/dL, compared to a significantly lower range of 2.5 to 5.0 mg/dL in controls (Table 1). This aligns with previous studies where hyperuricemia has been found more prevalent in T2DM populations[20]. For example, a multi-country pooled analysis reported hyperuricemia rates ranging from 10% to 25% among diabetic patients, depending on the population and diagnostic criteria used. These consistent patterns underscore the role of uric acid dysregulation in the pathophysiology of diabetes[21].

The diabetic group in this study also showed significantly higher mean BMI (26.1 ± 4.1 kg/m²) and WHR (0.77 ± 0.06) compared to controls (BMI: 23.0 ± 3.4 kg/m², WHR: 0.72 ± 0.05) (Table 1). Additionally, serum uric acid demonstrated a moderate positive correlation with BMI ($r = 0.45$, $p = 0.002$) and WHR ($r = 0.38$, $p = 0.004$) (Table 6). SUA levels also increased progressively across BMI categories: 4.21 ± 0.85 mg/dL in normal-weight, 5.35 ± 0.92 mg/dL in overweight, and 6.49 ± 1.10 mg/dL in obese diabetic patients (Table 7). This supports the established link between obesity and hyperuricemia, where excessive adipose tissue may impair uric acid excretion due to chronic low-grade inflammation and insulin resistance. Other studies have shown comparable patterns, with obese diabetic patients often recording SUA levels exceeding 6.5 mg/dL[22].

Dyslipidemia was another significant factor associated with elevated SUA. Diabetic subjects with abnormal lipid profiles had a mean SUA of 6.45 ± 1.05 mg/dL, compared to 4.70 ± 1.13 mg/dL in those without dyslipidemia (Table 5). Furthermore, SUA showed a positive correlation with total cholesterol ($r = 0.41$), LDL ($r = 0.36$), and triglycerides ($r = 0.42$), while negatively correlating with HDL ($r = -0.28$, $p = 0.031$) (Table 6). These findings are in line with global data suggesting that elevated SUA may contribute to or result from lipid metabolism disturbances, potentially via hepatic steatosis or oxidative stress pathways[23,24].

A significant association was also observed between hypertension and elevated SUA levels. Diabetic patients with hypertension had a mean SUA of 6.58 ± 1.10 mg/dL, significantly higher than 4.83 ± 1.19 mg/dL in non-hypertensives ($p = 0.0001$) (Table 5). Elevated SUA may contribute to hypertension through mechanisms involving renal sodium retention, vascular smooth muscle proliferation, and endothelial dysfunction. These findings support earlier studies that have consistently found hypertensive diabetic patients to have SUA levels 0.8–1.5 mg/dL higher on average than normotensive counterparts[25-27].

The duration of diabetes had a clear effect on SUA levels in this study. Diabetic patients with 9–12 years of disease had significantly higher mean SUA (6.60 ± 1.08 mg/dL) compared to those with 5–8 years (4.68 ± 1.02 mg/dL) and 2–4 years (4.35 ± 0.76 mg/dL) (Table 4). This trend suggests a cumulative impact of prolonged metabolic stress on uric acid handling, possibly due to progressive insulin resistance or declining

renal function. Similar trends have been observed in international studies where patients with diabetes duration ≥10 years had SUA levels reaching 6.5–7.0 mg/dL, with increased risk of macrovascular complications[28-30].

Interestingly, despite higher SUA levels in patients with ischemic heart disease (6.78 ± 1.19 mg/dL) and myocardial infarction (7.12 ± 0.92 mg/dL), the differences did not reach statistical significance (Table 5). This may be attributed to the smaller sample size in these subgroups or the influence of additional confounders such as medication use or renal function. However, these elevated values still support the hypothesis that hyperuricemia contributes to a pro-inflammatory and pro-atherogenic state[31].

Smoking was significantly more prevalent in diabetic males (42.59%) compared to controls (20%), with a p-value of 0.032 (Table 2). While this study did not directly link smoking to SUA levels, smoking is a well-established contributor to oxidative stress and vascular inflammation, which may amplify the cardiovascular burden in hyperuricemic individuals[32].

5. Conclusion

This study demonstrated that serum uric acid (SUA) levels were significantly higher among diabetic individuals compared to healthy controls in the Bangladeshi population. Elevated SUA showed a clear association with key cardiovascular risk factors, including increased BMI, waist-hip ratio, hypertension, and lipid profile abnormalities. Additionally, SUA levels were found to rise progressively with longer duration of diabetes, indicating a cumulative risk over time. These findings underscore the potential of SUA as a valuable marker for identifying patients at increased cardiovascular risk. Given the growing burden of diabetes and heart disease in Bangladesh, routine monitoring of SUA in diabetic patients may aid in early detection and more comprehensive risk management strategies.

6. Limitations of the study

Despite providing valuable insights into the association between serum uric acid levels and cardiovascular risk factors in type 2 diabetic patients, this study has several limitations:

1. **Cross-sectional design** – The study's cross-sectional nature prevents the establishment of causal relationships between elevated serum uric acid and cardiovascular outcomes.
2. **Single-center setting** – Data were collected from a single healthcare facility, which may limit the generalizability of the findings to the broader Bangladeshi population.
3. **Small sample size** – Although the sample size was adequate for preliminary analysis, a larger cohort would provide more robust and representative results.
4. **Lack of longitudinal follow-up** – Long-term cardiovascular outcomes were not assessed, which limits the ability to determine whether elevated SUA directly contributes to future cardiovascular events.
5. **Potential confounders** – Lifestyle factors such as dietary habits, physical activity, medication use (e.g., antihypertensives, diuretics, or statins), and kidney function were not comprehensively accounted for, which could influence serum uric acid levels.

Limited biochemical profiling – Some additional cardiovascular biomarkers (e.g., C-reactive protein, HbA1c, or eGFR) were not included, which could have enhanced the assessment of metabolic and renal influences on SUA.

Conflict of interest

The authors declare no conflict of interest.

Author contributions

All authors contributed equally to the conception, design, data collection, analysis, and writing of this manuscript. All authors have read and approved the final version.

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