Section: General Medicine

Original Research Article

STUDY OF SERUM URIC ACID IN UNCONTROLLED T2DM CO-RELATION TO CAROTID INTIMAL MEDIA THICKNESS

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Received : 07/01/2026
Received in revised form : 24/02/2026
Accepted : 10/03/2026

Keywords:
Diabetes mellitus, Hyperuricemia, Hyperlipidemia, Hypertension, Cardiovascular disease, Serum uric acid, Carotid intimal media thickness.

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DOI: 10.47009/jamp.2026.8.2.119

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (2); 641-644

ABSTRACT

Background: The rising global prevalence of type 2 diabetes mellitus (T2DM) highlights the urgent need for comprehensive insight into its associated cardiovascular risks. This study aimed to assess the correlation between SUA levels and CIMT in T2DM patients across varying states of glucose metabolism. **Materials and Methods:** This prospective observational study included 60 adult patients who had been diagnosed with T2DM. The participants were categorised into group 1, FBG < 100 mg/dL (normal glucose tolerance); group 2: FBG 100-125 mg/dL (impaired fasting glucose); and group 3, FBG ≥ 126 mg/dL (T2DM). Basic demographic data including age, gender, and clinical history were collected. Fasting blood glucose (FBG) and serum uric acid (SUA) levels and carotid intimal media thickness (CIMT) were measured. **Result:** The mean age of the participants was 62.33 ± 5.48 years, with 65% being male and 35% female. Both SUA levels and CIMT were higher in groups with more severe glucose dysregulation. The mean SUA level increased progressively from 6.2 ± 1.1 mg/dL in the normal glucose group to 8.9 ± 1.7 mg/dL in the T2DM group. CIMT measurements increased from 0.89 ± 0.12 mm in the normal glucose group to 1.15 ± 0.18 mm in the T2DM group. A moderate positive correlation was observed between serum uric acid levels and CIMT, with a correlation coefficient (r) of 0.589 and a p-value of <0.0001. **Conclusion:** High SUA levels are associated with increased risks of cardiovascular diseases and atherosclerosis in patients with uncontrolled T2DM, highlighting the importance of managing uric acid and blood glucose levels to mitigate these health risks.

INTRODUCTION

Obesity has become a pervasive global issue due to lifestyle changes, such as high-fat, high-calorie diets and reduced physical activity. Consequently, the prevalence of type 2 diabetes mellitus (T2DM), hyperuricaemia, hyperlipidaemia, hypertension, and cardiovascular disease (CVD) has been increasing. These conditions often interact, making it essential to understand their associations for valuable clinical insights.[1-3] T2DM presents a high morbidity rate, with hyperuricaemia and vascular atherosclerosis being common in these patients. Additionally, patients with diabetes face a high risk of CVD, which is the leading cause of mortality in this population. Previous reports have shown that hyperuricaemia is closely associated with CVD. However, it remains unclear whether high levels of serum uric acid (SUA) directly induce CVD in T2DM patients.[3-5]

Elevated uric acid levels are associated with several well-documented risk factors for CVD, including age, obesity, hyperlipidaemia, hypertension, and diabetes. Specifically, elevated SUA levels have been linked to atherosclerosis, a significant concern in uncontrolled T2DM. Hyperuricemia is commonly associated with hyperinsulinemia or DM, as aberrant renal function and faulty glucose metabolism impair the body's ability to excrete uric acid, leading to hyperuricemia.[1,2,4,6]

Carotid atherosclerosis and plaques are expected to affect 267.25 million and 199.83 million people, respectively, by 2024. Uric acid has been implicated in cardiovascular and cerebrovascular disorders, and several studies have demonstrated that SUA levels independently predict these conditions, particularly myocardial infarction and stroke. Thus, the SUA level is a powerful predictor of cardiovascular disease. Recent studies have also found that hyperuricemia is associated with increased carotid intima-media thickness (CIMT).[7,8]

Hyperuricaemia has been suggested as a risk factor for CVD in the general population. However, this association may be confounded by established CVD risk factors. Data on this relationship in the Indian

ISSN (O): 2687-5365; ISSN (P): 2753-6556

population are scarce, as most studies have been conducted in Japan and China, with few focusing on patients with diabetes mellitus (DM). In the 21st century, hyperuricemia has been observed to play a role in the development of metabolic syndrome, coronary artery disease, and DM.[2,3]

This study aimed to correlate SUA levels with CIMT in T2DM patients across varying states of glucose metabolism. In addition, we evaluated the potential of SUA monitoring as a marker of atherosclerosis severity in patients with uncontrolled T2DM.

MATERIALS AND METHODS

This prospective observational study included 60 adult patients who had been diagnosed with diabetes mellitus in a tertiary college hospital.

Inclusion criteria:

Adults aged ≥ 18 years who were diagnosed with diabetes mellitus were included.

Exclusion criteria:

Patients with severe comorbid conditions that could influence carotid intimal thickness, patients on medications known to significantly affect SUA levels, and pregnant or lactating women were excluded.

Participants were categorised into three groups based on their FBG levels: group 1, FBG < 100 mg/dL (normal glucose tolerance); group 2: FBG 100-125 mg/dL (impaired fasting glucose); and group 3, FBG ≥ 126 mg/dL (diabetes mellitus).

Data collection: Basic demographic data, including age, sex, and clinical history, were collected from each participant. Fasting blood samples were collected from each participant to measure the SUA levels. SUA levels were determined using an enzymatic colourimetric method. Fasting blood glucose levels were measured using standard laboratory procedures.

The CIMT was measured using real-time B-mode ultrasound. A high-resolution ultrasound system equipped with a 7.5 MHz linear array transducer was utilised for this purpose. Measurements were performed by a trained sonographer who was blinded to the participants' glucose levels and SUA results.

Data analysis: The data were analysed using SPSS version 25.0. Descriptive statistics were computed for the baseline demographic and clinical characteristics. Categorical data were summarised as frequencies and percentages, whereas continuous data were summarised as means and standard deviations. Chi-square tests were used to compare categorical variables, and independent t-tests were used to compare continuous variables. Statistical significance was set at $p < 0.05$.

RESULTS

In this prospective study involving 60 participants, the mean age was 62.33 ± 5.48 years, with 65% being male (n=39) and 35% female (n=21). The mean duration of diabetes among the participants was 6.45 ± 5.12 years. The participants were categorised into three groups based on their glucose metabolism status: normal fasting blood glucose, impaired fasting glucose, and diabetes mellitus [Table 1].

Table 1: Serum uric acid levels and carotid intimal media thickness (CIMT) across glucose metabolism groups

Glucose metabolism group		No of participants	Mean SD	P value
Age		60	62.33 ± 5.48	-
Serum uric acid level (mg/dl)	Normal fasting blood glucose	20	6.2 ± 1.1	0.1524
	Impaired fasting glucose	20	7.5 ± 1.3	0.0274*
	Diabetes mellitus	20	8.9 ± 1.7	
CIMT (mm)	Normal fasting blood glucose	20	0.89 ± 0.12	0.1453
	Impaired fasting glucose	20	0.98 ± 0.15	0.0421*
	Diabetes mellitus	20	1.15 ± 0.18	

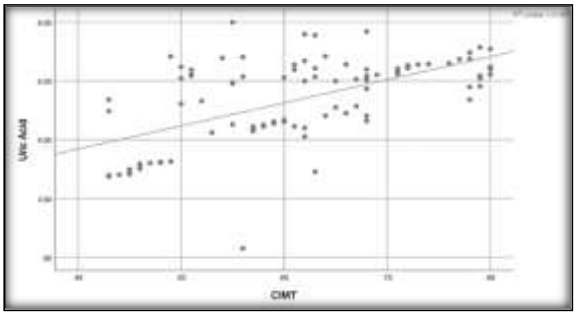

Figure 1: Correlation between serum uric acid and carotid intimal media thickness

Our findings indicate that both serum uric acid (SUA) levels and carotid intimal media thickness (CIMT) were higher in groups with more severe glucose dysregulation. The mean SUA level increased progressively from 6.2 ± 1.1 mg/dL in the normal glucose group to 8.9 ± 1.7 mg/dL in the diabetes mellitus group. Similarly, CIMT measurements increased from 0.89 ± 0.12 mm in the normal glucose group to 1.15 ± 0.18 mm in the diabetes mellitus group. The mean SUA levels and CIMT differed significantly between the impaired fasting glucose and diabetes mellitus groups [Table 1].

A moderate positive correlation was observed between serum uric acid levels and CIMT, with a correlation coefficient (r) of 0.589 and p-value of <0.0001 [Figure 1].

DISCUSSION

Our study demonstrated a significant association between SUA levels and CIMT in patients with varying glucose metabolic states. Our data indicated that both SUA levels and CIMT increased with the

ISSN (O): 2687-5365; ISSN (P): 2753-6556

severity of glucose dysregulation. Patients with diabetes mellitus exhibited the highest SUA levels and CIMT measurements, followed by those with impaired fasting glucose and those with normal fasting blood glucose levels. The progressive increase in SUA levels and CIMT measurements across the groups suggests a potential link between hyperuricaemia and atherosclerosis progression in patients with diabetes mellitus. This finding is in line with a previous study that highlighted the role of uric acid as a risk factor for cardiovascular diseases including atherosclerosis.

The findings of Gao et al. align with the results of this study, demonstrating that patients with high uric acid levels exhibit higher CIMT and thickening rates than those with normal uric acid levels. In the hyperuricaemia group, both CIMT and thickening rate progressively increased with FBG levels. Even after controlling for variables such as age, blood pressure, cholesterol, and FBG level, a significant association between SUA level and CIMT was observed in the cross-sectional population data. Additionally, it was found that reducing uric acid levels led to a significant decrease in CIMT, suggesting a potential therapeutic target for cardiovascular risk reduction.[1]

Researchers generally agree that several mechanisms, including elevated uric acid, promote low-density lipoprotein oxidation and lipid peroxidation, thus aiding atherosclerosis. Subsequently, increased uric acid causes uric acid microcrystals to deposit in the vascular intima, triggering local inflammation and endothelial damage, leading to lipid deposition.[9] Elevated uric acid is also associated with reactive oxygen species, causing vascular inflammation. Furthermore, uric acid directly promotes platelet aggregation and thrombosis, with platelet-released cytokines causing vascular smooth muscle hyperplasia.[10,11] Thus, lowering uric acid is a primary treatment to enhance its excretion, shown to reduce blood pressure, especially in young patients without prolonged hypertension.12 A study by Rajpoot et al. conducted a study in Madhya Pradesh, India, and showed a statistically significant but weak negative linear correlation between glycated haemoglobin and uric acid. Additionally, there was a statistically significant positive linear correlation between SUA and average CIMT.[3]

A recent two-step Mendelian randomisation study performed by Chen et al. found that SUA was genetically correlated with BMI (OR = 1.080, 95% CI: 1.024-1.139, p = 0.005). SUA had a positive causal effect on atrial fibrillation (OR = 0.892; 95% CI: 0.804-0.990, p = 0.032), coronary artery disease (OR = 0.942, 95% CI: 0.890-0.997, p = 0.037), and essential hypertension (OR = 1.080, 95% CI: 1.024-1.139, p = 0.005). They found a causal relationship between high SUA and increased obesity risk, as well as a higher risk of various CVDs.[13] Moreover, Mendelian randomized analysis revealed that elevated SUA levels lead to higher blood pressure, subsequently increasing the risk of CVD like coronary heart disease and stroke. Diabetes, which is associated with a higher risk of atherosclerotic disease, also increases the risk of stroke compared to non-diabetics.[14]

Previous genome-wide studies by Tin et al. demonstrated a significant association between impaired glucose tolerance and CIMT. Additionally, a genome-wide study on uric acid found that transcription factors involved in urate metabolism might influence various metabolic processes, such as blood glucose regulation and other cardiovascular risk factors, complicating the determination of causality.[15]

Our findings indicate that SUA is an independent risk factor for carotid atherosclerosis, with elevated SUA levels promoting carotid intimal thickening influenced by FBG patterns. The observed moderate correlation between SUA levels and CIMT further supports the hypothesis that elevated SUA levels contribute to vascular changes associated with atherosclerosis. This relationship underscores the importance of monitoring SUA levels in patients with diabetes mellitus as it could serve as a marker for the severity of atherosclerosis and help guide therapeutic interventions.

CONCLUSION

In conclusion, managing serum uric acid levels in patients with diabetes could potentially mitigate the risk of atherosclerosis and related cardiovascular complications. Further investigation using a specific model is warranted to validate the predictive value of SUA monitoring in guiding personalised management strategies and improving cardiovascular outcomes in this patient population. This study provides important insights into optimising cardiovascular risk management in patients with uncontrolled T2DM by managing uric acid levels to improve patient outcomes.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556

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ISSN (O): 2687-5365; ISSN (P): 2753-6556