An Inverse Relationship between Serum Uric Acid and Glycated Hemoglobin in Type 2 Diabetes Mellitus: A Cross-Sectional Analysis

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Abstract

Background: Glycated hemoglobin (HbA1c) is a critical indicator of glycemic control in Type 2 Diabetes Mellitus (T2DM). Serum uric acid (SUA), traditionally a marker of gout, is increasingly recognized for its role in metabolic dysfunction. The interplay between SUA and glycemic control remains complex. **Objective:** To evaluate the association between serum uric acid levels and HbA1c among patients with T2DM. **Methods:** A cross-sectional study of 100 T2DM patients was conducted over 18 months. Patients were categorized by glycemic control based on HbA1c levels: good (<7%), fair (8–9%), and poor (>9%). SUA was measured using the uricase enzymatic method and compared across these categories. **Results:** Mean SUA levels were highest in patients with HbA1c <7% (6.2 ± 1.1 mg/dL), lower in those with HbA1c 8–9% (5.4 ± 0.9 mg/dL), and lowest in those with HbA1c >9% (4.1 ± 0.7 mg/dL). There was a statistically significant inverse correlation ($r = -0.42$, $p < 0.01$) between SUA and HbA1c. **Conclusion:** SUA levels decline with worsening glycemic control in T2DM, suggesting that elevated SUA may be more prominent in early or well-controlled diabetes. These findings highlight SUA's potential as a marker for insulin resistance rather than advanced hyperglycemia.

Keywords: Serum uric acid, HbA1c, type 2 diabetes mellitus, insulin resistance, metabolic marker

Introduction

Type 2 diabetes mellitus (T2DM) is a multifaceted metabolic disorder marked by chronic hyperglycemia and insulin resistance. HbA1c is an established long-term marker of glycemic control, while SUA has emerged as a novel biomarker linked

with insulin resistance and cardiovascular disease risk. Interestingly, the relationship between SUA and HbA1c is not linear. Studies suggest that SUA levels may be elevated in pre-diabetic and early diabetic states, but decline as glycemic control worsens. This study evaluates the association between SUA and HbA1c in T2DM patients, aiming to clarify whether SUA may be used to monitor metabolic disturbances beyond glucose levels alone.

Materials and Methods

Design: Cross-sectional observational study
Setting: General Medicine Department, SGT Medical College & Hospital, Gurugram
Duration: 18 months
Sample Size: 100 patients with T2DM

Inclusion Criteria: Diagnosed T2DM, on treatment for at least 6 months
Exclusion Criteria: Renal dysfunction, anemia, gout, infections, cardiovascular/cerebrovascular diseases

Investigations: SUA measured using uricase method; HbA1c measured using latex agglutination assay Patients were categorized into three groups based on HbA1c levels: <7% (good), 8–9% (fair), and >9% (poor)

Statistical Analysis: ANOVA and Pearson correlation performed using SPSS v21. A p-value <0.05 was considered significant.

Results

Table 1. Mean Serum Uric Acid across HbA1c Groups

HbA1c Group	No. of Patients	Mean HbA1c (%)	Mean SUA (mg/dL)
<7%	32	6.3 ± 0.4	6.2 ± 1.1
8–9%	38	8.4 ± 0.3	5.4 ± 0.9
>9%	30	9.8 ± 0.5	4.1 ± 0.7

Correlation Analysis: Pearson correlation coefficient between HbA1c and SUA: $r = -0.42$, $p < 0.01$

Discussion

This study confirms a significant inverse relationship between SUA and HbA1c. Patients with better glycemic control had higher SUA levels. As glycemic control

deteriorated, SUA levels dropped, supporting the hypothesis of glucose-induced uricosuria and reduced renal reabsorption of urate in hyperglycemic states. These findings are consistent with the results of previous studies, which observed similar trends and proposed oxidative stress and altered renal handling of urate as mechanisms for this pattern.

Conclusion

SUA levels are inversely correlated with HbA1c in patients with T2DM. Elevated SUA may signal early metabolic dysregulation and insulin resistance, while lower SUA in poorly controlled diabetes reflects altered renal excretion mechanisms. Monitoring SUA can thus provide metabolic insights in diabetic management.

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