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Serum Uric Acid and Glycemic Control among Diabetes Mellitus Patients in Semarang, Indonesia: A Hospital-Based Cross-Sectional Study

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ABSTRACT

Introduction: The relationship between serum uric acid (SUA) and glycemic status in established diabetes is biologically plausible but inconsistent across studies because insulin resistance, renal urate handling, glycosuria, obesity, kidney function, and glucose-lowering therapy can influence SUA in different directions. This study evaluated the association of SUA with fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) in a hospital-based sample in Semarang, Indonesia.

Methods: This retrospective cross-sectional study screened 410 medical records from Sultan Agung Islamic Hospital, Semarang, during 2018–2020. Thirty records with complete SUA, FBG, and HbA1c data were available for analysis. Hyperuricemia was defined as SUA >7.0 mg/dL in men and >6.0 mg/dL in women. The revision reanalyzed the available raw dataset using Shapiro–Wilk testing, Spearman correlation, exploratory partial Spearman correlation adjusted for age, sex, and recorded obesity status, and Fisher's exact test. Two-sided $p<0.05$ was considered statistically significant.

Results: The mean age was 54.3±6.5 years; 16/30 (53.3%) were women and 18/30 (60.0%) met the sex-specific definition of hyperuricemia. SUA showed a moderate positive monotonic association with FBG (Spearman $\rho=0.401$, 95% bootstrap CI 0.053 to 0.665, $p=0.028$); this remained similar after adjustment for age, sex, and obesity (partial $\rho=0.396$, $p=0.041$). The association with HbA1c was weak and not statistically significant ($\rho=0.270$, 95% bootstrap CI -0.126 to 0.624, $p=0.149$).

Conclusion: Higher SUA was associated with higher FBG, but not HbA1c. These exploratory findings should be interpreted cautiously because of the small, highly selected sample and incomplete data on renal function, medication use, diabetes duration, and other confounders.

Keywords: Diabetes mellitus; fasting blood glucose; HbA1c; hyperuricemia

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Introduction

Diabetes mellitus (DM) refers to a wide range of metabolic disorders that share underlying commonality with hyperglycemia. It could occur in different ways and impacts the body differently, either through autoimmune destruction of β-cells of the pancreas or progressive resistance to insulin despite a degree of insulin insufficiency. Due to the chronic nature of this condition and because it affects the whole body, DM has to be regularly monitored, observed, and managed throughout the patient's life, given that healthcare systems are significantly strained by the burden of diabetes mellitus and its complications. [1,2] Central to the development of these complications is persistent hyperglycemia, which drives progressive damage due to a wide range of mechanisms most of which include oxidative stress, the inflammatory pathway, the production of advanced glycosylation end products, and alterations in the endothelial function.[3] Due to this, hyperglycemia is considered a hallmark of the disease and can be used for its diagnosis and monitoring. Prolonged elevated blood glucose levels are associated with an increased risk of both microvascular and macrovascular complications, contributing to morbidity and mortality in affected individuals, and are known to damage a range of vital organs including the eyes, kidneys, heart, and nerves.[1,2]

Diabetes mellitus (DM) remains a major global health challenge, with prevalence projected to reach 783 million cases by 2045 [4]. Poor glycemic control, reflected by elevated fasting blood glucose (FBG) and glycated hemoglobin (HbA1c), is strongly associated with increased risks of microvascular and macrovascular complications, making optimization of metabolic control a central goal of diabetes management [5,6] The increasing prevalence of diabetes is driven by a combination of many factors, among them demographic shifts and aging populations, sedentary lifestyles, and increasingly rising rates of obesity. Low- and middle-income countries currently account for a notable and growing share of the total global diabetes burden, where delayed diagnosis and often severely limited access to comprehensive care frequently culminate in suboptimal glycemic control levels. In such settings, the routine use of laboratory markers plays an exceptionally important role in guiding clinical decision-making. Therefore, it is imperative to underscore the necessity for better understanding the precise factors influencing glycemic status, [7–9] In this context, there is an urgent need to identify additional metabolic factors that may influence glycemic control or serve as accessible markers to improve risk stratification and clinical decision-making.

In recent years, the role of serum uric acid levels as a modifiable risk factor of type 2 DM has attracted considerable attention. Hyperuricemia has been widely observed in patients with type 2 DM, especially those with insulin resistance and metabolic syndrome [10]. Clinical trials and experimental studies have suggested a bidirectional relationship, whereby insulin resistance can affect the renal clearance of uric acid, thereby increasing serum uric acid levels, while increased uric acid levels can induce endothelial dysfunction, oxidative stress, and inflammation that further enhance insulin resistance and cardiovascular risk.[11,12]. This immediately raises a pertinent question: whether serum uric acid primarily reflects an individual's glycemic control status or independently contributes to the documented adverse metabolic outcomes.

Despite these uncertainties, data from hospitals in Indonesia itself are still limited. Moreover, the clinically relevant question for a cross-sectional hospital study is therefore not whether uric acid causes adverse metabolic outcomes, but whether a routinely measured SUA value tracks concurrent glycemic status in the local clinical population. Indonesian hospital-based data addressing this narrower question are limited, and previous reports have produced positive, negative, and null associations. This study aimed to estimate the direction, magnitude, and precision of the association between SUA and two glycemic markers—FBG and HbA1c—among patients whose records were available at Sultan Agung Islamic Hospital, Semarang, during 2018–2020.

Methods

This retrospective observational study used a cross-sectional design and medical-record data from Sultan Agung Islamic Hospital, Semarang, Indonesia, during 2018-2020.

A total of 410 records were screened using consecutive record sampling. Records were eligible for the analytic dataset when SUA, FBG, and HbA1c results were available within the study period. Records with incomplete or missing data for one or more of these three laboratory variables were excluded, leaving 30 complete records for analysis. The archived extraction did not permit a more detailed classification of the missing-data pattern among the 380 excluded records. Because this was a retrospective complete-case analysis, the analytic sample was determined by data availability rather than by an a priori sample-size calculation. Additional exclusions based on chronic kidney disease, gout, urate-lowering therapy, diuretics, SGLT2 inhibitors, or other medications could not be consistently applied because these variables were not retained in the available research extraction.

SUA was recorded in mg/dL. Hyperuricemia was defined as SUA >7.0 mg/dL in men and >6.0 mg/dL in women, consistent with the original study protocol. FBG was recorded in mg/dL and categorized as elevated when >126 mg/dL. HbA1c was recorded as a percentage and categorized as elevated when >6.5%. Age, sex, systolic and diastolic blood pressure, and recorded obesity status were also abstracted when available. The source summary categorized 24 patients as hypertensive and 6 as non-hypertensive; exact BMI values, diabetes duration, renal-function indices, and complete medication histories were not available.

SUA, FBG, and HbA1c measurements were extracted from routine hospital laboratory records. The archived research dataset did not preserve the analyzer model, analytical principles, calibration data, or internal and external quality-control records; these assay-specific details therefore could not be reconstructed retrospectively.

Continuous variables were summarized using mean±standard deviation or median and interquartile range, as appropriate, and categorical variables using frequency and percentage. Given the small sample and marginal normality of FBG, Spearman rank correlation was used as the primary association analysis. Ninety-five percent confidence intervals (CIs) for Spearman coefficients were estimated with 20,000 bootstrap

resamples. Exploratory partial Spearman correlations were calculated, adjusting for age, sex, and recorded obesity status. Pearson correlation was retained as a sensitivity analysis to facilitate comparison with the original analysis. Fisher exact tests compared elevated FBG and elevated HbA1c between hyperuricemic and non-hyperuricemic groups. Statistical analyses were performed using Python 3.13 with SciPy and statsmodels. All tests were two-sided and P<.05 was considered statistically significant.

The study was approved by the Bioethics Commission for Medical and Health Research, Faculty of Medicine, Universitas Islam Sultan Agung, Semarang (approval No. 56/EC/KEPK/2020). All patient data were anonymized before analysis.

Results

Of 410 screened medical records, 30 complete records (7.3%) were included in the analysis. The remaining 380 records were excluded because one or more required laboratory variables were incomplete or missing (Figure 1).

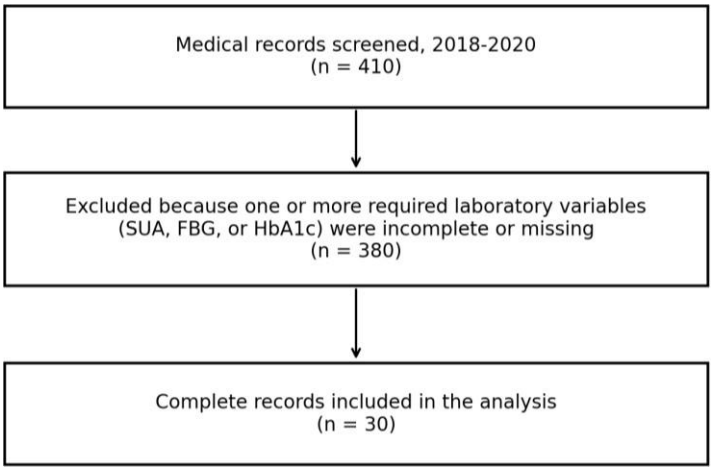

Figure 1. Flow diagram of record selection

The mean age was 54.3±6.5 years; 16 patients (53.3%) were women. Eighteen records (60.0%) met the sex-specific definition of hyperuricemia. Nineteen records (63.3%) had elevated FBG, and 19 (63.3%) had elevated HbA1c. Baseline characteristics are summarized in Table 1.

Table 1. Baseline characteristics of the analyzed records (n=30)

Characteristic	Value
Age, years	54.3 ± 6.5
Women	16 (53.3%)

Characteristic	Value
Men	14 (46.7%)
Serum uric acid, mg/dL	6.95 ± 2.13
Hyperuricemia	18 (60.0%)
Systolic blood pressure, mmHg	154.8 ± 33.5
Diastolic blood pressure, mmHg	89.5 ± 21.1
Hypertension, as reported in source summary	24 (80.0%)
Obesity, recorded	6 (20.0%)
FBG, mg/dL	149 (104.8-189.5)
Elevated FBG (>126 mg/dL)	19 (63.3%)
HbA1c, %	7.12 ± 1.79
Elevated HbA1c (>6.5%)	19 (63.3%)

Values are mean ± standard deviation, median (interquartile range), or n (%). FBG, fasting blood glucose; HbA1c, glycated hemoglobin. Exact BMI values, diabetes duration, renal-function indices, and medication histories were unavailable.

SUA showed a moderate positive monotonic association with FBG (Spearman rho=0.401, 95% CI 0.056 to 0.666; P=0.028). After adjustment for age, sex, and recorded obesity, the association remained similar (partial rho=0.396; P=0.041). The association between SUA and HbA1c was weak and not statistically significant (rho=0.270, 95% CI -0.131 to 0.626; P=0.149) (Table 2).

Table 2. Association between serum uric acid and glycemic parameters

Outcome	Spearman rho	95% CI	P	Partial rho*	P
FBG	0.401	0.056 to 0.666	0.028	0.396	0.041
HbA1c	0.270	-0.131 to 0.626	0.149	0.269	0.174

Exploratory partial Spearman correlation adjusted for age, sex, and recorded obesity status. CI, confidence interval; FBG, fasting blood glucose; HbA1c, glycated hemoglobin.

As a sensitivity analysis, Pearson correlation yielded r=0.317 (P=0.088) for SUA-FBG and r=0.278 (P=0.137) for SUA-HbA1c. Scatter plots are shown in Figure 2.

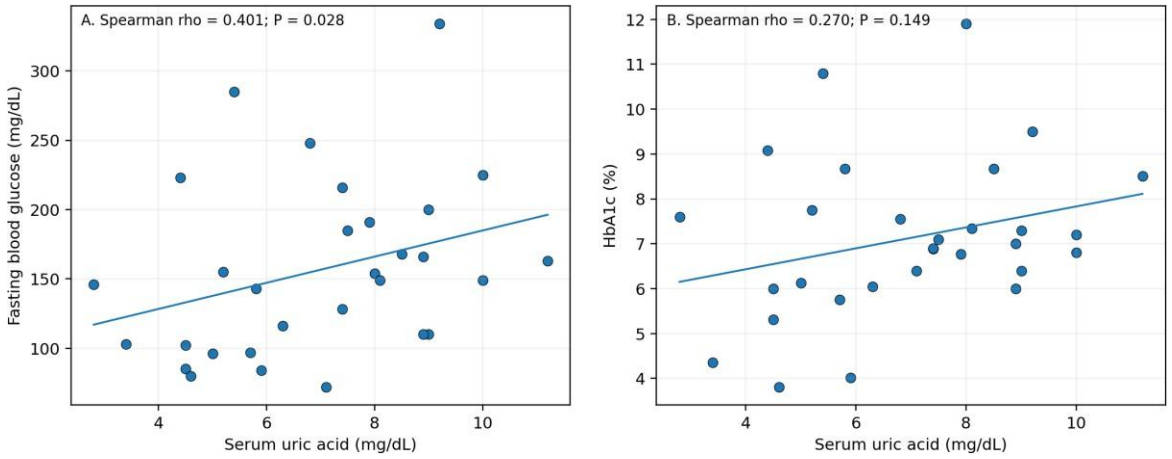

Figure 2. Scatter plots of serum uric acid with (A) fasting blood glucose and (B) HbA1c

Elevated FBG was present in 14 of 18 hyperuricemic patients (77.8%) compared with 5 of 12 non-hyperuricemic patients (41.7%). The same distribution was observed for elevated HbA1c. These categorical differences did not reach the conventional significance threshold (Fisher exact P=.063 for each comparison) (Table 3).

Table 3. Glycemic categories according to hyperuricemia status

Glycemic parameter	Hyperuricemia (n=18)	No hyperuricemia (n=12)	Fisher P
Elevated FBG	14 (77.8%)	5 (41.7%)	0.063
Elevated HbA1c	14 (77.8%)	5 (41.7%)	0.063

FBG, fasting blood glucose; HbA1c, glycated hemoglobin

Discussion

The main finding was a modest positive monotonic association between SUA and FBG, while the association with HbA1c was weak and not statistically significant. The FBG association remained similar after limited adjustment for age, sex, and recorded obesity. The categorical analysis also showed a higher proportion of elevated FBG and HbA1c among hyperuricemic patients, although these comparisons narrowly missed the conventional significance threshold. Because only 30 complete records were analyzed, these estimates should be interpreted as exploratory rather than definitive.

The biological relationship between uric acid and glucose is not expected to be uniform across all stages of dysglycemia. Hyperinsulinemia can increase renal urate reabsorption, whereas marked hyperglycemia can promote glycosuria and uricosuria. Wei et al. reported that SUA and HbA1c may change dynamically during the development of type 2 diabetes, and Shi et al. described an inverted-U

relationship between SUA and fasting glucose across non-diabetic, prediabetic, and diabetic states [13–15]. These mechanisms may partly explain why the present study identified an association with FBG but not with HbA1c.

Previous clinical studies have also reported inconsistent directions of association. Patel et al. observed inverse correlations between SUA and both HbA1c and fasting glucose among participants with diabetes, while a meta-analysis of urate-lowering interventions found a reduction in fasting glucose overall but no clear HbA1c benefit and no consistent glycemic benefit in diabetic subgroups [16,17]. This heterogeneity supports the interpretation that SUA is strongly influenced by metabolic stage and coexisting clinical factors and should not be considered a direct substitute for standard glycemic markers.

Renal function and medication exposure are particularly important potential confounders. Higher blood glucose can promote uric acid excretion, while SGLT2 inhibitors lower SUA through uricosuric mechanisms; impaired kidney function can further modify urate handling [18–20]. The present analysis could adjust only for age, sex, and recorded obesity. Information on renal function, gout, urate-lowering therapy, diuretics, SGLT2 inhibitors, diabetes duration, and other glucose-lowering treatment was not sufficiently available for adjustment. Residual confounding is therefore likely.

This study has several limitations. Only 30 of 410 screened records were complete, creating substantial potential for selection bias. The specific pattern and mechanism of missingness among the 380 excluded records could not be reconstructed. With n=30, the study has approximately 80% power only for relatively large correlations of about $|r|=0.49$ or greater, so smaller true associations may be missed. Exact BMI, renal-function measures, diabetes duration, and medication data were unavailable, and assay-specific analyzer and quality-control information could not be recovered from the archived research extraction. The cross-sectional design also prevents conclusions about temporality or causality.

Clinically, FBG and HbA1c remain the appropriate validated measures of glycemic status. The present findings suggest that SUA may covary with FBG in some hospital-based patients, but the small sample and incomplete confounder information prevent use of SUA as an independent marker of glycemic control. Larger studies with explicit eligibility criteria, complete renal and medication data, and adequately powered multivariable analyses are required.

Conclusion

In this small and highly selected hospital-based sample, higher serum uric acid was modestly associated with higher fasting blood glucose after limited adjustment for age, sex, and recorded obesity, whereas no statistically significant association was found with HbA1c. These findings are exploratory and do not establish serum uric acid as an independent marker of glycemic control. Larger studies with complete renal-function and medication data are needed.

Conflicts of Interest

There is no conflict of interest.

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