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Fasting plasma glucose and serum uric acid levels in the peri-urban region

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Abstract

Background: The prevalence of hyperuricemia has increased, yet there is a gap in empirical studies regarding the association between hyperuricemia and diabetes mellitus in Indonesia.

Purpose: To determine the baseline association between serum uric acid and fasting blood glucose in the peri-urban population.

Method: A quantitative approach was employed, with particular emphasis on a formative method. The study was conducted in Sampangan, Semarang, from January to April 2023. Uric acid levels served as the independent variable, while fasting blood glucose was designated as the dependent variable. A total of 44 participants were selected through total sampling, based on predetermined inclusion and exclusion criteria. Data analysis involved univariate testing to assess the frequency distribution of participant characteristics, followed by bivariate analysis utilizing one-way ANOVA and Kruskal-Wallis tests.

Results: Significant differences were observed in fasting blood glucose, serum uric acid, and systolic blood pressure levels across the healthy, prediabetic, and type 2 diabetic groups, as well as between individuals with normal uric acid levels and those with hyperuricemia. Conversely, no significant differences were found for age, diastolic blood pressure, body mass index (BMI), or cholesterol levels.

Conclusion: This study offers a deeper understanding of serum uric acid levels among healthy individuals, prediabetic patients, and individuals with type 2 diabetes mellitus. Factors such as uric acid concentration, presence of hypertension, and patient initiative contribute to decisions regarding the continuation of screening.

Keywords: Fasting Blood Glucose; Metabolic Health; Peri-urban Population; Uric Acid.

INTRODUCTION

Serum uric acid (SUA) is the final oxidation product of purine catabolism in humans with the loss of uricase. Overproduction and decreased excretion through kidneys lead to hyperuricemia. In recent decades, the prevalence of hyperuricemia has increased substantially worldwide, with a rising trend both in developed and developing nations. The prevalence of hyperuricemia in the general population is estimated at 10–25% (Skoczyńska, Chowaniec, Szymczak, Hetmańczyk, Chyra, & Wiland, 2020).

In a global ethnic group comparison, male Polynesians, such as people in Indonesia, displayed a more significant prevalence of hyperuricemia (Kurniasari, Karwur, Rayanti, Huruta, Lin, Weng, & Tsai, 2022). The prevalence of hyperuricemia in Indonesia is estimated at 18%. A survey of 4,683 rural adult populations in Java Island showed that gout and hyperuricemia prevalence in men was 1.7% and 24.3%, respectively. Elevated SUA is an independent predictor of vascular complications and is associated with excess risk for the development of

diabetes mellitus (Xiong, Liu, & Xu, 2019). These findings highlight the growing public health concern surrounding hyperuricemia in Indonesia, particularly among rural populations. The significantly higher prevalence of hyperuricemia compared to clinically diagnosed gout suggests that many individuals may remain asymptomatic yet at risk for serious metabolic and cardiovascular consequences (Boxhammer, Hammerer, Scherthaner, Prinz, Brandt, Lichtenauer, & Kopp, 2024). Elevated SUA levels, as shown in various studies, are not only a marker of metabolic dysfunction but also contribute independently to the development of insulin resistance, endothelial dysfunction, and systemic inflammation. This reinforces the importance of early screening and management of hyperuricemia, even in the absence of gout symptoms, to mitigate long-term complications such as type 2 diabetes and cardiovascular disease (Wu, Lai, Chou, Liao, You, Bai, & Sun, 2020).

Uric acid may interfere with the stimulation process of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase in the human body. Under elevated concentrations, uric acid enters cells and contributes to glucose and insulin intolerance by inducing oxidative stress through the production of reactive oxygen species (ROS), thereby functioning as an independent risk factor and predictor for the development of type 2 diabetes mellitus (T2DM) (Wardhana & Rudijanto, 2018). This mechanism highlights the pathological role of uric acid that extends beyond its conventional association with gout. Once inside the cell, elevated uric acid levels impair insulin signaling by activating NADPH oxidase and increasing ROS production (Hussain, Elahi, Hussain, Iqbal, Akhtar, & Majid, 2021). The resulting oxidative environment disrupts normal cellular metabolism, diminishes insulin sensitivity, and contributes to pancreatic beta-cell dysfunction. As such, uric acid acts not simply as a metabolic byproduct, but as a pro-oxidant agent that directly disrupts glucose regulation (Han, Han, Yin, Cao, Di, Wu, & Zeng, 2022).

A previous study found a significant negative association between serum uric acid and fasting blood glucose concentrations when participants in all groups were combined in Pearson's coefficient test ($p < 0.01$) (Haque, Rahman, Islam, Molla, & Ali, 2019).

However, the relationship between T2DM and hyperuricemia remains subject to controversy. Although several studies have reported hyperuricemia as a risk factor for the development of diabetes mellitus because of insulin resistance, others have suggested that patients recently diagnosed with DM tend to have lower serum uric acid than prediabetic or normoglycemic (Ni, Lu, Chen, Du, & Zhang, 2019). Elevated SUA levels have been considered not only an independent risk factor for cardiovascular disease but also a factor in the development of metabolic disease. Many studies report that SUA is causally involved in the pathogenesis of DM (Freilich, Arredondo, Zonnoor, McFarlane, & McFarlane, 2022).

RESEARCH METHOD

A quantitative approach was employed, with a particular emphasis on a formative method. The study was conducted in Sampangan, Semarang, between January and April 2023. Uric acid served as the independent variable, while fasting blood glucose (FBG) functioned as the dependent variable. A total of 44 participants were recruited using total sampling, consisting of 27 healthy individuals, 6 prediabetic patients, and 11 patients with type 2 diabetes mellitus, all of whom met specific inclusion and exclusion criteria.

The inclusion criteria required participants to be between 18 and 65 years of age, residents of the peri-urban area for at least one year, willing to participate by signing informed consent, not undergoing regular treatment for metabolic disorders (such as diabetes mellitus or hyperuricemia), and having fasted for a minimum of 8 hours prior to blood sampling. The exclusion criteria involved individuals with a history of chronic kidney or liver disease, pregnant or lactating women, those who had taken medications affecting glucose or uric acid levels (e.g., corticosteroids, allopurinol) within the past two weeks, individuals with cognitive or mental impairments affecting communication, and participants with incomplete or invalid laboratory data.

Fasting blood glucose levels were assessed using the Fingerstick Test, where capillary blood from the fingertip was applied to a test strip and analyzed using a digital device, yielding results

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within three minutes. FBG values <100 mg/dL were categorized as normal, 100–125 mg/dL as prediabetes, and ≥ 126 mg/dL as indicative of type 2 diabetes mellitus.

Uric acid levels were similarly measured using the Fingerstick Test. Blood samples were applied to test strips and analyzed by a digital device within three minutes. Hyperuricemia was defined as uric acid levels exceeding 6 mg/dL in women and 7 mg/dL in men.

Body Mass Index (BMI) was calculated by dividing body weight in kilograms by the square of height in meters (kg/m^2). BMI values were classified as follows: underweight (<18.5), normal (18.5–24.9), overweight (25–29.9), and obese (≥ 30).

Blood pressure measurements were taken using a sphygmomanometer. The results were categorized as normal (systolic <120 mmHg and diastolic <80 mmHg), pre-hypertension (systolic 120–139 mmHg or diastolic 80–89 mmHg), stage 1 hypertension (systolic 140–159 mmHg or diastolic 90–99 mmHg), and stage 2 hypertension (systolic ≥ 160 mmHg or diastolic ≥ 100 mmHg).

Data analysis began with univariate analysis to determine the frequency distribution of participants' characteristics, followed by bivariate analysis employing one-way ANOVA and Kruskal-Wallis tests.

RESEARCH RESULTS

Table 1. Characteristic of the Participants (N=44)

Variable	Group			p-value
	Healthy(n=27) (mean $\pm$ SD)	Prediabetic (n=6) (mean $\pm$ SD)	Type-2 DM (n=11) (mean $\pm$ SD)	
Age	(68.48 $\pm$ 10.67)	(67 $\pm$ 12.21)	(69 $\pm$ 9.26)	0.920 [§]
Sex: M/F	4/23	1/5	7/4	-
SBP	(134.25 $\pm$ 23.9)	(133.16 $\pm$ 14.91)	(129.27 $\pm$ 18.71)	0.815 [§]
DBP	(82.51 $\pm$ 12.11)	(79 $\pm$ 8.98)	(77.09 $\pm$ 5.76)	0.600 [‡]
BMI	(24.54 $\pm$ 5.09)	(23.95 $\pm$ 4.89)	(22.12 $\pm$ 4.53)	0.209 [‡]
SUA (mg/dL)	(5.43 ($\pm$ 1.07)	(6.90 $\pm$ 1.12)	(7.53 $\pm$ 2.00)	0.002[‡]*
Total Cholesterol (mg/dL)	(242.77 $\pm$ 59.62)	(253.5 $\pm$ 53.73)	(214.9 $\pm$ 64.34)	0.361 [§]
Fasting Blood Glucose (mg/dL)	(84.62 $\pm$ 10.50)	(10.8 $\pm$ 6.44)	(205.8 $\pm$ 132.75)	0.005[‡]*

*Significant ($p < 0.05$); [§] One Way Anova; [‡] Kruskal-Wallis; M: male; F: female; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; BMI: Body Mass Index; SUA: Serum Uric Acid.

Table 1 indicates no significant differences in age, systolic blood pressure (SBP), diastolic blood pressure (DBP), body mass index (BMI), or total cholesterol levels among the healthy, prediabetic, and type 2 diabetes groups. The average age across the three groups was similar, approximately 68–69 years. SBP was highest in the healthy group (134.25 $\pm$ 23.9) and lowest in the diabetes group (129.27 $\pm$ 18.71), while DBP decreased from healthy (82.51 $\pm$ 12.11) to diabetic individuals (77.09 $\pm$ 5.76). BMI also tended to decline from the healthy group (24.54 $\pm$ 5.09) to the diabetes group (22.12 $\pm$ 4.53). Total cholesterol was highest in the prediabetic group (253.5 $\pm$ 53.73) and lowest in the diabetic group (214.9 $\pm$ 64.34). Has been significant differences were observed in serum uric acid (SUA) and fasting blood glucose levels, with p-values of 0.002 and 0.005, respectively. SUA levels increased from the healthy group (5.43 $\pm$ 1.07) to the diabetes group (7.53 $\pm$ 2.00), as did fasting blood glucose levels, which rose sharply from 84.62 $\pm$ 10.50 (healthy) to 205.81 $\pm$ 132.75 (diabetic). These findings suggest that SUA and fasting blood glucose are key parameters in distinguishing metabolic status among the groups.

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Table 2. Correlation between Uric Acid Levels and Hyperuricemia in T2DM Patients (N=44)

Variable	Uric Acid	Mean ($\pm$ SD)	p-value
Age	Normal	66.59 ($\pm$ 10.43)	0.252 [§]
	Hyperuricemia	70.24 ($\pm$ 9.62)	
SBP	Normal	127.37 ($\pm$ 18.89)	0.030^{§*}
	Hyperuricemia	141.59 ($\pm$ 22.75)	
DBP	Normal	78.81 ($\pm$ 10.19)	0.158 [‡]
	Hyperuricemia	83.65 ($\pm$ 10.77)	
BMI	Normal	22.96 ($\pm$ 4.57)	0.095 [§]
	Hyperuricemia	25.38 ($\pm$ 4.61)	
FBG	Normal	136.63 ($\pm$ 100.66)	0.005^{‡*}
	Hyperuricemia	88.71 ($\pm$ 21.57)	
Cholesterol	Normal	228.04 ($\pm$ 58.79)	0.239 [§]
	Hyperuricemia	250.65 ($\pm$ 63.42)	

*Significant ($p < 0.05$); [§] Independent-t; [‡] Mann-Whitney

Table 2 shows significant differences between those with normal uric acid levels and those with hyperuricemia in terms of systolic blood pressure (SBP) and fasting blood glucose (FBG). The hyperuricemia group had a higher SBP (141.59 ± 22.75 vs. 127.37 ± 18.89 ; $p=0.030$) and a lower FBG (88.71 ± 21.57 vs. 136.63 ± 100.66 ; $p=0.005$). Other variables, including age, diastolic blood pressure (DBP), BMI, and total cholesterol, showed no significant differences. These findings suggest a correlation between hyperuricemia and elevated SBP, as well as reduced FBG levels, in T2DM patients.

DISCUSSION

This study demonstrates significant differences in serum uric acid (SUA) and fasting blood glucose (FBG) levels among healthy individuals, prediabetic patients, and those with type 2 diabetes mellitus (T2DM). Additionally, in T2DM patients, both systolic blood pressure (SBP) and FBG differ significantly between those with normal uric acid levels and those with hyperuricemia. Serum uric acid is known to be associated with various cardiovascular and

metabolic conditions, including obesity, heart failure, metabolic syndrome, impaired fasting glucose, and elevated fasting plasma glucose (Soeatmadji, Rosandi, Saraswati, Sibarani, & Tarigan, 2023). Biologically, uric acid contributes to the worsening of insulin resistance by reducing the bioavailability of nitric oxide, a key molecule involved in insulin-stimulated glucose uptake (Shirasawa, Ochiai, Yoshimoto, Nagahama, Watanabe, Yoshida, & Kokaze, 2020).

The reduction of nitric oxide (NO) bioavailability by elevated uric acid levels plays a critical role in impairing vascular and metabolic functions. Nitric oxide is essential for maintaining endothelial function and facilitating insulin-mediated glucose uptake in peripheral tissues such as skeletal muscle. When uric acid diminishes NO production, endothelial dysfunction occurs, leading to impaired vasodilation and reduced glucose delivery to tissues (Benn, Dua, Gurrell, Loudon, Pike, Strorer, & Vangieli, 2018). This condition exacerbates insulin resistance and creates a pro-inflammatory and pro-atherogenic environment. The interference of uric acid with nitric oxide pathways not only contributes to metabolic

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dysregulation but also links hyperuricemia to the development of cardiovascular complications commonly seen in individuals with type 2 diabetes mellitus (Mei, Dong, Geng, & Xu, 2022).

Some researchers hypothesize that hyperuricemia might be a risk factor for the development of T2DM. However, since higher SUA levels are often associated with established T2DM risk factors, such as metabolic syndrome and alcohol consumption, it is still unclear whether SUA is an independent risk factor for diabetes or just a risk marker (Aversa, Wondimnew, Welde, & Husen, 2020). Our finding shows increased uric acid levels are associated with fasting blood glucose levels. It aligns with previous studies that high uric acid levels are significantly associated with prediabetes and diabetic patients, indicating that SUA has a significant role in disturbing glucose metabolism. Uric acid may affect blood glucose levels, disturb metabolism, and cause insulin resistance (Cassano, Crescibiene, Hribal, Pelaia, Armentaro, Magurno, & Sciacqua, 2020).

The present study points out a significant difference in FBG in healthy persons, prediabetic patients, and T2DM patients. Prediabetes is a precursor before the diagnosis of diabetes mellitus. Adults with prediabetes often may show no signs or symptoms of diabetes but will have blood glucose levels higher than healthy individual. The normal category of blood glucose level ranges from 70 mg/dL to 99 mg/dL. In patients with prediabetes, fasting blood glucose levels between 110 mg/dL to 125 mg/dL and 126 mg/dL or higher indicate diabetes (Galvão, Beleigoli, Vidigal, Duncan, Schmidt, Appleton, & Diniz, 2021).

It was indicated that the body resists insulin or doesn't produce enough to maintain normal blood sugar levels. Prediabetes is also impaired fasting glucose, glucose tolerance, glucose tolerance, or borderline diabetes (Rahman, Hossain, Das, Kundu, Adegoke, Rahman, & Pang, 2021). During digestion, the pancreas produces insulin, which binds glucose in the blood and takes it into cells as a source of energy. If a person has prediabetes, glucose accumulates in the bloodstream instead of fueling the cells. Then, it affects insulin resistance, becoming a leading cause of prediabetes (Yuan, Liu, Zhou, Gu, Wu, Huang, & Wu, 2023).

These conditions reflect a state of metabolic dysregulation in which the body begins to lose its ability to regulate blood glucose effectively. In prediabetes, insulin resistance develops when cells in muscles, fat, and the liver become less responsive to insulin, leading to elevated blood glucose levels despite normal or even increased insulin production (Ali, Miah, Hasan, Barman, Mou, Hafsa, & Islam, 2020). This stage is often marked by impaired fasting glucose (IFG) and impaired glucose tolerance (IGT), which are key indicators of deteriorating glucose metabolism. Although blood sugar levels have not yet reached the diagnostic threshold for type 2 diabetes, individuals with prediabetes are at significantly increased risk of progressing to full-blown diabetes and developing cardiovascular complications. Early detection and intervention during this stage are critical to prevent or delay disease progression through lifestyle modification and, if necessary, pharmacological therapy (Cano, Abellán, Luque, Jiménez, Recio, & Saldaña, 2022).

Our findings also revealed a significant difference between SBP in normal and hyperuricemia patients. Previous studies have found an association between SUA and hypertension. Systolic blood pressure is associated with risk factors for occurring hyperuricemia (Wang, Qin, Liu, Wang, Xu, Chen, & Lou, 2023). Uric acid may play an essential role in the development of hypertension through activation of the renin-angiotensin-aldosterone system (RAAS). Other studies suggested that the SUA level positively correlated with the incidence of hypertension in middle-aged and elderly adults (Piani, Cicero, & Borghi, 2021).

Hyperuricemia is associated with increased cardiovascular risk, including the risk of developing hypertension. Our study also highlights a significant difference in FBG in normal levels and hyperuricemia patients. It aligns with the previous research in T2DM patients, which found that SUA levels were significantly lower than in healthy and prediabetic individuals and showed an inverse association with FBG concentration (Xu, Huang, Wu, Ji, Guo, & Huang, 2021). This finding indicates that a decreasing trend of SUA is associated with increased blood glucose concentration. In contrast, there is a significant negative relationship between SUA and FBG among healthy and prediabetic

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patients. SUA levels were decreased gradually in the prediabetes and diabetes groups compared to the healthy in both genders (Stewart, Langlois, & Noone, 2019).

CONCLUSION

This study provides a better understanding of serum uric acid levels in healthy individuals, prediabetics and T2DM patients. Factors such as uric acid levels, hypertension, and patient initiative contribute to decisions regarding the continuation of screening.

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