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ORIGINAL ARTICLE

Analyzing The Correlation of Uric Acid and Fasting Blood Sugar in Type 2 Diabetes Patients: A Study from ODSK Hospital, North Sulawesi, Indonesia

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Abstract

Type 2 diabetes mellitus (T2DM) is a major non-communicable disease in Indonesia and is commonly diagnosed using fasting blood glucose (FBG) levels. Recent studies have suggested a potential association between fasting blood sugar (FBS) and uric acid, which plays a role in oxidative stress and glucose metabolism, although findings have been inconsistent. This study aimed to examine the association between blood uric acid levels and FBG levels in T2DM patients at ODSK Regional Hospital, North Sulawesi, Indonesia, given the rising prevalence of T2DM and related metabolic disorders in the region. A cross-sectional study was conducted using secondary data from medical records of T2DM patients between July 2023 and July 2024. Among male patients, 25 percent had uric acid levels between 3.0 and 7.4 mg/dL, while 28.8 percent had levels above 7.4 mg/dL. Among female patients, 32.7 percent had uric acid levels between 2.1 and 6.3 mg/dL, and 13.5 percent had levels above 6.3 mg/dL. Additionally, 84.6 percent of all patients had FBG levels of 126 mg/dL or higher. Statistical analysis showed no significant correlation between blood uric acid levels and FBG levels ($\rho = 0.541$; $r = 0.541$), suggesting that there is no significant association between these two variables in this population.

Introduction

Non-communicable diseases (NCDs) are chronic diseases influenced by genetic, physiological, environmental, and behavioral factors, such as hypertension, obesity, and unhealthy lifestyles [1]. Diabetes mellitus (DM), one of the NCDs with a high mortality rate, occurs due to a deficiency or the inability of insulin to function effectively [2–4].

The International Diabetes Federation (IDF) reported that 10.5% of the adult population aged 20–79 years suffer from diabetes, with more than 90% of cases being type 2 diabetes mellitus (T2DM) [5]. Diabetes is a major public health problem worldwide, affecting more than 537 million people, and the burden of DM is increasing globally. The prevalence in adults is projected to increase by 25% by 2030 and 51% by 2045 [6].

The 2023 Indonesia Health Survey reported a national prevalence of DM at 1.7%, with type 2 DM being the most common type [7]. In North Sulawesi, the prevalence was ranked 5th nationally at 2.1%, exceeding the national average. The 2018 Basic Health Research reported a physician-diagnosed DM prevalence of 2.27% in North Sulawesi, with the highest cases in Manado City at 3.45% [8]. Furthermore, data from the Central Statistics Agency of Manado City showed that DM ranked 6th among the 10 most common diseases, with a total of 6,804 cases [9].

This condition is worsened by the typical Manadonese diet, which tends to be high in carbohydrates and purines. Rice as a staple food has the potential to increase blood glucose levels, while consumption of certain meats and fish can trigger increased uric acid levels [10]. In addition, irregular eating patterns, low physical activity, and very rare exercise, as is often found in T2DM patients at Pancaran Kasih Hospital, Manado, further worsen blood glucose control [11].

The diagnosis of T2DM primarily uses fasting blood glucose (FBG) as the primary indicator [12]. Blood glucose levels in patients with T2DM can be influenced by various factors [7], one of which is uric acid levels. Based on previous research by Husen and Ratnaningtyas regarding the relationship between uric acid levels and blood glucose in elderly adult women in Mandiraja Wetan Village using the Pearson correlation analysis test, it was revealed that there was a significant relationship with a positive correlation between uric acid and blood glucose (r=0,418, ρ=0,022) [8].

Studies in Indonesia have shown a high prevalence of hyperuricemia in T2DM patients, with 56% affected, particularly women (62.5%). Research at Sanglah General Hospital, Denpasar, found hyperuricemia in 54.1% of uncontrolled and 59.0% of controlled HbA1C cases, indicating a complex relationship between uric acid and glucose metabolism [13]. Hyperuricemia frequently coexists with DM risk factors like obesity, high fructose consumption, and alcohol intake, but whether it serves as a mere risk indicator or a direct contributor to DM is still unclear. In the general population, it has been linked to prediabetes, while in individuals with type 2 DM, it is associated with disease progression and complications [14].

Uric acid is the final product of purine metabolism in the body [15] and serves as a potent antioxidant by scavenging singlet oxygen, peroxide radicals, and hydroxyl radicals [16]. However, excessive uric acid levels can mediate oxidative stress and increase the production of reactive oxygen species (ROS) [17]. In pancreatic β-cells, oxidative stress activates key pathways like AMPK, mTOR, and JNK, leading to complex and often paradoxical effects on insulin secretion and cell survival. Initially, AMPK activation can be protective by promoting autophagy and reducing oxidative stress, but prolonged activation disrupts β-cell proliferation and impairs insulin secretion. Similarly, while mTOR signaling is essential for β-cell growth and survival, oxidative stress can lead to both suppression and dysregulation of mTOR activity, contributing to β-cell failure. The JNK pathway, activated by oxidative stress, disrupts insulin signaling and promotes β-cell apoptosis, further exacerbating dysfunction in diabetes progression [18,19]. The low levels of antioxidant enzymes in β-cells make them more vulnerable to mitochondrial damage, exacerbated by elevated uric acid levels, which trigger inflammation, β-cell death, and insulin resistance through the expression of IL-1β, IL-6, TNF-α, and CRP [20,21].

Elevated uric acid levels are also closely associated with glucose metabolism. The pentose phosphate pathway enhances nucleotide synthesis and purine catabolism through the production of ribose-5-phosphate, which generates uric acid [22]. High blood glucose levels can worsen uric acid production, creating a cycle in which uric acid and blood glucose levels influence each other [17,21,23].

An increase in uric acid is associated with diabetic microvascular complications, one of which is diabetic nephropathy (DN), a long-standing microvascular complication of diabetes and a leading cause of end-stage kidney disease in developed countries. Both excessively high and low levels of uric acid are linked to the development of albuminuria (increased protein in the urine) in patients with type 2 diabetes. Additionally, higher uric acid levels are associated with an increased likelihood of developing DN [21,24].

Previous studies have also demonstrated a varying relationship between FBG and blood uric acid. In the study by Praditya and Maslim using the Pearson correlation test, the results showed

an insignificant relationship between fasting blood sugar and serum uric acid in T2DM patients at Atma Jaya Hospital (r=-0.101, ρ=0.563) [25].

Prior research has shown varying relationships between FBG and serum uric acid levels in patients with T2DM. Some studies suggest a significant correlation, while others demonstrate no significant relationship. These variations may be attributed to differences in study populations, measurement methods, and research designs. The study by Praditya and Maslim utilized medical record data with a limited sample size and excluded patients with impaired kidney function or those taking medications affecting uric acid levels, which may have limited the generalizability of their findings [25]. Meanwhile, Ratnaningtyas and Husen used a portable device (EasyTouch GCU-Meter) to measure blood glucose and uric acid levels, which may differ in accuracy compared to standard laboratory methods [26].

These differing findings highlight inconsistencies in the relationship between uric acid levels and FBG, presenting an opportunity for further research to achieve a more consistent and comprehensive understanding of this relationship. This study contributes to bridging these differences by examining the relationship between uric acid levels and FBG in a T2DM population in North Sulawesi. By employing laboratory-based measurement methods and considering the characteristics of the local population, which may have distinct dietary patterns and lifestyle factors, this research aims to provide a more comprehensive understanding of this association. Furthermore, the findings of this study can help clarify whether population-specific factors and methodological differences influence the inconsistencies observed in previous research, offering new insights relevant to the Indonesian population.

Thus, this study hypothesizes that there is a significant relationship between blood uric acid levels and FBG levels in patients with type 2 diabetes mellitus (T2DM) at ODSK Regional Hospital, North Sulawesi Province, Indonesia. Therefore, this study aims to investigate the relationship between blood uric acid levels and FBG levels in patients with T2DM at ODSK Regional Hospital in North Sulawesi Province, Indonesia. This research focuses on the local population of North Sulawesi, providing insights into the region's health conditions. The relevance of this study is significant, given the increasing prevalence of T2DM and uric acid-related issues in Indonesia.

Materials and Methods

The research method used is an observational analytic study with a cross-sectional design. This study is a retrospective analysis, utilizing secondary data from the medical records of T2DM patients at ODSK Regional Hospital in North Sulawesi Province from July 2023 to July 2024. The sampling method used is total sampling. The data to be collected include identity, diagnosis, FBG results, and blood uric acid levels.

The period from July 2023 to July 2024 was selected to ensure the study captures recent and relevant patient data while maintaining consistency in clinical protocols. A one-year timeframe allows for an adequate sample size, ensuring more reliable statistical analysis. Aligning with hospital record-keeping practices, this timeframe also ensures data collection remains within the scope of ethical and administrative approvals.

The research sample consists of data from all T2DM patients at ODSK Regional Hospital in North Sulawesi Province who meet the research criteria. The inclusion criteria for this study are all patients with T2DM at ODSK Regional Hospital in North Sulawesi Province between July 2023 and July 2024, who underwent blood uric acid laboratory tests, and patients aged 19 years or older. The exclusion criteria for this study include patients diagnosed with type 1 diabetes mellitus (DM), maturity-onset diabetes of the young (MODY), gestational diabetes, and neonatal diabetes, as well as patients with incomplete, damaged, or unreadable FBG and blood uric acid data.

In this study, serum uric acid and FBG levels were measured using medical records obtained from a chemistry analyzer (Mindray BS-430). Serum uric acid levels are categorized with normal ranges of 3.0–7.4 mg/dL for males and 2.1–6.3 mg/dL for females. FBG levels are classified, where a value of ≥126 mg/dL is considered indicative of T2DM. Both variables are measured on a ratio scale, ensuring precise quantification for correlation analysis.

Univariate analysis will be used to describe the distribution of data and sample frequencies, such as age, gender, uric acid levels, and FBG, without associating them with other variables. Bivariate analysis will be conducted to assess the relationship between blood uric acid levels and FBG levels. Normality testing for data distribution will use the Kolmogorov-Smirnov test, followed by Spearman correlation analysis. A p-value > 0.05 for the normality test indicates a normal data distribution, while a p-value < 0.05 for the correlation test indicates statistically significant results.

Results and Discussion

The characteristics of T2DM patients at the ODSK Hospital, North Sulawesi Province, from July 2023 to July 2024 are presented in Table 1. The data includes 52 samples that meet the inclusion and exclusion criteria, categorized by gender, age, fasting blood sugar levels, and blood uric acid levels.

Table 1. Characteristics of T2DM patients.

Variables	Frequency	Percentage (%)
Gender		
Male	28	53.8
Female	24	46.2
Age		
19 – 24	1	1.9
25 – 34	0	0
35 – 44	3	5.8
45 – 54	13	25
55 – 64	21	40.4
65 – 74	11	21.2
≥ 75	3	5.8
Fasting Blood Sugar Levels (mg/dL)		
< 126	8	15.4
≥ 126	44	84.6
Blood Uric Acid Levels (mg/dL)		
Male: 3,0 – 7,4	13	25
Male: > 7,4	15	28.8
Female: 2,1 – 6,3	17	32.7
Female: > 6,3	7	13.5

Based on the distribution of samples by age category according to the 2023 Indonesia Health Survey, the majority of T2DM patients were aged 55–64 years, accounting for 40.4% of the total. According to the gender distribution data presented in Table 1, the majority of patients with T2DM in this study were male, comprising 28 patients (53.8%), compared to 24 female patients (46.2%).

Based on the results presented in Table 1, the majority of T2DM patients, comprising 44 individuals (84.6%), had FBG levels ≥ 126 mg/dL, while eight individuals (15.4%) had FBG levels < 126 mg/dL. Among these patients, blood uric acid levels in females were most commonly in the range of 2.1–6.3 mg/dL, with 17 individuals (32.7%) falling into this category, as shown in Table 1. In male patients, the highest uric acid levels were recorded in the >7.4 mg/dL category, with 15 individuals (28.8%).

The results of the normality test for unstandardized residuals (Table 2), based on the Kolmogorov-Smirnov test with 52 samples, showed a mean of 0 and a standard deviation of

2.81. The most extreme difference between the sample distribution and the normal distribution was 0.166 for absolute and positive values and -0.104 for negative values. The test produced a significance value (Asymp. Sig. 2-tailed) of 0.001, indicating that the residuals did not follow a normal distribution at the 5% significance level ($p < 0.05$). In addition, the Monte Carlo significance test confirmed the results, with a p-value of 0.002 and a 99% confidence interval ranging from 0.001 to 0.002.

Table 2. Data normality test results.

			Unstandardized Residual
N			52
Normal Parameters	Mean		.0000000
	Std. Deviation		2.81003622
Most Extreme Differences	Absolute		.166
	Positive		.166
	Negative		-.104
Test Statistic			.166
Asymp. Sig. (2-tailed)			.001
Monte Carlo Sig. (2-tailed)	Sig.		.002
	99% Confidence Interval	Lower Bound	.001
		Upper Bound	.002

Based on the Spearman-Rank correlation test analysis, the ρ-value was 0.541, and the correlation coefficient (r) was -0.087 for blood uric acid levels and FBG levels (Table 3). Since the ρ-value > 0.05, it can be concluded that there is no significant relationship between blood uric acid levels and FBG levels in T2DM patients at ODSK Regional General Hospital, North Sulawesi Province (Table 3).

Table 3. Correlation test of uric acid levels with fasting blood sugar levels.

	Blood Uric Acid Levels	
	Correlation coefficient (r)	**Significance (ρ)**
Fasting Blood Sugar Levels	-0.087	0.541

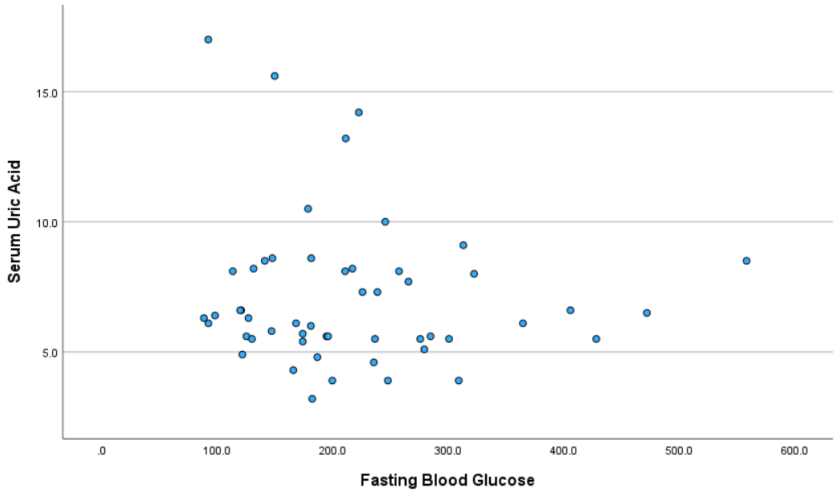

Figure 1. Scatter plot of blood uric acid and fasting blood sugar.

The scatter plot illustrates the relationship between FBG levels (x-axis) and serum uric acid levels (y-axis), as shown in Figure 1. The data points exhibit a wide dispersion, suggesting a complex or weak correlation between these two variables. Most observations cluster around

lower glucose levels (below 250 mg/dL) and moderate uric acid concentrations (approximately 5–10 mg/dL). The pattern suggests variability in uric acid levels among individuals with both normal and elevated glucose concentrations.

The results obtained in this study for the age category are in line with the research findings conducted by Komariah and Rahayu who reported that the majority of T2DM patients in their study at the Proklamasi Outpatient Clinic, Depok, West Java, were aged 46–65 years (69.4%) from a total of 134 samples [27]. The prevalence of T2DM tends to increase with advancing age. The causes of T2DM in older adults include a decline in β-cell function and increased insulin resistance, which can be attributed to reduced muscle mass (sarcopenia) and the accumulation of visceral fat. These factors contribute to greater glucose intolerance in elderly individuals [28,29].

The gender distribution data in Table 1 is in line with the research of Aprilio et al. on the characteristics of type 2 DM patients at the Simpang Periuk Community Health Center, Lubuklinggau City, which reported that of the 36 patients, 26 patients (72.2%) were male [30]. This result aligns with the theory of fat distribution in males, who tend to store fat around internal organs (visceral fat), which is metabolically more active than subcutaneous fat. Additionally, a decline in testosterone levels in elderly men may further increase the risk of developing T2DM [31].

The fasting blood sugar level category in diabetic patients obtained in this study is in line with the study conducted by Kusdiantini and Istiqomah, which reported that from a total sample of 30 people, 21 people (70%) had FBG levels ≥ 126 mg/dL [32]. In this study, there were T2DM patients with FBG levels of less than 126 mg/dL. The results of this study still show that many diabetic patients have uncontrolled fasting blood sugar levels. This may be because the population in North Sulawesi Province has its characteristics that affect the manifestation of T2DM. Factors such as food consumption patterns and lifestyle contribute to variations in the study's results. People in North Sulawesi are known to consume foods high in carbohydrates and animal protein, which can contribute to uncontrolled fasting blood sugar levels. In addition, the level of physical activity that tends to be low can also affect the incidence of T2DM [11,33].

The results of this study also revealed fasting blood sugar levels in controlled conditions. This finding is related to the pathophysiology of early-stage T2DM, where glucose tolerance remains near normal despite the presence of insulin resistance. This is because pancreatic β-cells compensate by increasing insulin production. However, as insulin resistance and compensatory hyperinsulinemia progress, the pancreatic islets of Langerhans in certain individuals may fail to sustain the hyperinsulinemic state [34].

Blood uric acid levels in T2DM patients in this study have results that are in line with the findings of a study conducted by Haque et al., which involved 310 blood samples and found that male T2DM patients had higher uric acid levels compared to female patients [35]. The findings of this study may be related to the theory that estrogen levels in males are lower than those in females, leading to a tendency for higher uric acid levels in males. This is because estrogen can influence uric acid transporter proteins by reducing the activity of solute carrier family two member 9 (SLC2A9), also known as glucose transporter 9 (GLUT9). This reduction in activity decreases the reabsorption of uric acid in the kidneys [36].

A study by Tsai et al. investigating the relationship between serum testosterone levels and hyperuricemia in men found that men with testosterone levels <400 ng/dL were at higher risk of developing hyperuricemia (serum uric acid levels ≥7 mg/dL) [37]. This is because low testosterone levels in men are associated with increased insulin resistance, which negatively impacts uric acid clearance by the kidneys [37,38].

The gender distribution findings in this study are also linked to genetic theories, specifically mutations in the hypoxanthine-guanine phosphoribosyltransferase (HPRT) gene, located on the human X chromosome. These mutations can cause defects in HPRT enzyme activity, primarily affecting males, leading to an accumulation of its substrates, hypoxanthine and guanine. These excess purines are subsequently converted into uric acid by the enzyme xanthine oxidase (XO), resulting in elevated uric acid levels in the body [39].

Based on the results of this study, it does not prove that uric acid levels affect FBG levels, nor does it confirm that FBG levels directly affect uric acid levels. These correlation test results align with the study by Praditya and Maslim, which used Pearson's correlation test and found no significant relationship between FBG and serum uric acid levels ($r = -0.101$, $\rho = 0.563$) [25]. Similarly, Rangareddy's study on serum uric acid levels in T2DM patients in urban South India reported no significant relationship between blood uric acid levels and FBG levels ($r = 0.058$, $\rho = 0.0691$) [40].

Conversely, previous research by Husen and Ratnaningtyas using Pearson's correlation analysis revealed a significant relationship between uric acid and blood glucose levels ($r = 0.418$, $\rho = 0.022$) [26]. A study by Pandey et al. also found that FBG levels were significantly correlated with blood uric acid levels ($r = 0.170$, $\rho = 0.006$) [41]. Furthermore, Haque et al's study of 310 samples reported a significant relationship between uric acid levels and FBG levels through Pearson's correlation test ($\rho < 0.01$) [35].

These differences may be attributed to variations in study design, sample characteristics, and measurement methods. For instance, Husen and Ratnaningtyas' study focused specifically on elderly female participants and used the EasyTouch GCU-Meter for glucose and uric acid measurements, which may differ in accuracy from laboratory-based methods [26]. Meanwhile, Pandey et al. and Haque et al. employed a broader sample that included both diabetic and non-diabetic individuals, which may have influenced their findings. Additionally, variations in genetic factors, dietary habits, and lifestyle choices across study populations may have contributed to the inconsistent results [35,41].

The findings of this study are related to the theory that high blood glucose levels in diabetic patients interfere with the reabsorption of uric acid in the kidney, since glucose and uric acid are reabsorbed at the same site. As a result, more uric acid is excreted in the urine, leading to lower blood uric acid levels. Transporters such as GLUT9, which facilitate the reabsorption of uric acid into the bloodstream, may also be affected by glucose levels, further reducing uric acid reabsorption. This mechanism may explain the negative association between uric acid levels and diabetes [25,35] suggests the possibility of a reverse causal relationship, where high blood glucose levels in diabetic patients may contribute to lower blood uric acid levels due to increased uric acid excretion through the kidneys.

Dietary factors are closely associated with the increase in exogenous purines that the body metabolizes. Foods rich in purines, such as seafood, meat, organ meats, and certain vegetables and legumes, can significantly influence uric acid levels in the body. This is because purines are present in the nucleic acids contained within nucleoproteins. When purine-containing foods are consumed, digestive enzymes break down nucleoproteins into proteins and nucleic acids. The nucleic acids are then degraded into nucleotides, releasing nitrogenous bases such as purines (adenine and guanine) and pyrimidines. In the final stage, purines are broken down by the enzyme xanthine oxidase, resulting in the production of uric acid [42].

Other factors may also influence the results of this study, including dietary habits, lifestyle, genetics, and anthropometric variables. High uric acid levels are often found in individuals with diabetes risk factors, such as obesity, fructose consumption, and alcohol intake. Among newly diagnosed diabetes patients, low uric acid levels are more frequently observed, possibly due to glucosuria, renal hyperfiltration, low-calorie diets, osmotic diuresis, and fluid accumulation [14].

These confounding factors underscore the complexity of the relationship between uric acid levels and fasting blood glucose (FBG).

In addition to the factors mentioned above, several other aspects may influence the results of this study. The data utilized in this research are derived from patient medical records at ODSK Regional Hospital, North Sulawesi Province, which constitute secondary data. Reliance on secondary data necessitates access permission and depends on the completeness of medical records, which can potentially limit the availability of detailed information. Furthermore, the relatively small sample size restricts the generalizability of the findings to a broader population. A limited sample size also increases the risk of bias, which may impact the validity of the study's conclusions.

These findings have significant clinical implications, particularly in the management of T2DM. While uric acid levels do not appear to have a direct impact on fasting glucose levels, monitoring both parameters remains crucial due to their shared metabolic risk factors. Clinicians should consider comprehensive metabolic assessments rather than focusing solely on individual biomarkers.

For future research, it is important to consider various factors that may influence the results, such as lifestyle, medical history, and genetic factors, while also utilizing a larger sample size to ensure more detailed and comprehensive findings. Longitudinal studies should be conducted to investigate the long-term relationship between uric acid and glucose metabolism, along with mechanistic studies to elucidate the biological pathways that link insulin resistance, glucose metabolism, and uric acid regulation. Additionally, investigating the impact of lifestyle interventions, such as dietary modifications and physical activity, on both uric acid and fasting glucose levels could provide valuable insights.

Conclusions

This study aims to investigate the relationship between blood uric acid levels and FBG levels in T2DM patients at ODSK Regional Hospital, North Sulawesi. The results showed no significant correlation between these variables, rejecting the initial hypothesis. While hyperglycemia can impact kidney function and uric acid metabolism, other factors, such as diet, obesity, and genetics, may play a more significant role. These findings suggest that the relationship between blood glucose and uric acid levels is not universal and may vary across different populations. Differences in sample size, demographics, and confounding factors could explain discrepancies with previous research. Further studies should investigate long-term metabolic interactions and refine clinical assessments for patients with T2DM.

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Ethical Clearance: This study was ethically approved by the Health Research Ethics Committee of Poltekkes Kemenkes Manado, meeting the seven WHO 2011 standards. The approval is valid from December 10, 2024, to December 10, 2025, under reference number KEPK.01/12/484/2024.

Informed Consent Statement: Not applicable.

Data Availability Statement: Data from this study are derived from medical records and cannot be shared publicly due to confidentiality and privacy regulations.

Conflicts of Interest: The authors declare that they have no conflicts of interest.

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