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RESEARCH ARTICLE

The Role of Serum Uric Acid in Ischemic Stroke

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Abstract: Ischemic stroke is considered as a leading cause of mortality and long-term disability worldwide. Several potential biomarkers were previously studied in relation to stroke, among them; serum uric acid (SUA) that have attracted a significant attention owing to its dual role seen as an antioxidant and a pro-oxidant. The current study was designed as a cross-sectional observational study that was conducted at Baquba Teaching Hospital, Diyala, Iraq, over a period from November 2024 to April 2025. The study was aimed to investigate the association between SUA levels and the incidence of ischemic stroke, severity and the disease outcomes. The current study included 56 Iraqi patients were diagnosed with stroke at Baquba Teaching Hospital, Diyala, Iraq and 44 healthy subjects recruited as controls. Blood samples were collected and SUA levels were measured using enzymatic methods. Statistical analysis revealed no significant difference in uric acid levels between healthy and abnormal subjects. Furthermore; no significant correlations were recognized between each of; SUA and glucose, blood pressure, age, or body weight. However, the study revealed a moderate positive correlation between SUA and serum urea levels. The obtained findings proposed that SUA may not serve as a direct risk factor for incidence of ischemic stroke, despite its role still complex and may vary depending on accompanying comorbid conditions. Further longitudinal with larger sample size studies are required to elucidate its predictive and therapeutic significance.

Keywords: Serum uric acid,stroke, SUA, cormorbid condition.

INTRODUCTION

1.1. Stroke

Globally, stroke is considered as the second leading cause of death and the third to that of disability (Khalil MI, Salwa M, 2020). It constitutes a substantial financial and economic burden on the patients and their families. Furthermore, new incidence of the stroke has reached 13.7 million in 2016, with nearly 87% of all cases being ischemic stroke worldwide (Saini V, Guada L, 2021). Moreover, stroke is reported to be the fifth prevalent cause of the death in United States with an annual figure of about 142,000 deaths, while approximately 795,000 subjects experiences a newly diagnosed or recurrent stroke. The roughly estimated annual expense of treating the condition reaches $34 billion for United States government (Saini V, Guada L, 2021).

Uric acid; a weak organic acid; is the final catabolite from purine nucleotide catabolism. It stands out as the most essential antioxidant in the circulation possessing a concentration exceeding tenfold in comparison to other antioxidants. Uric acid in blood is found to be functioning as a scavenger for each of; superoxide, the hydroxyl, and the oxygen radicals (King, R.D. et al, 2023). On the other hand, hyperuricemia is defined as uric acid serum concentration being higher than 6.8 mg/dl (Saini V, Guada L, 2021).

Uric acid levels homeostasis is regulated by the equilibrium that created between its production and excretion by the kidneys. Sources of uric acid is either originated from exogenous dietary high purines intake such as red meat, seafood or from endogenous source such as tissues catabolism and the de novo synthesis of purine from both RNA and DNA molecules bases (Bansal BC, Gupta RR, 1975). In regard to hyperuricemia; it involved in pathophysiology of diverse conditions such as gout, chronic kidney disease, cardiovascular conditions like coronary artery disease, insulin resistance syndrome, obesity, metabolic syndrome, hyperlipidemia and hypertension (Sridharan R. 1992; Chamorro A. et al, 2002).

Ameliorated renal excretion of uric acid is reported to be accountable for about 90% of gouty cases, while the elevated synthesis of uric acid comprises only 10% of cases (Weir CJ. et al 2003). Nevertheless, it still questionable whether the state of hyperuricemia is major risk factor to the onset and incidence of stroke. During last few decades, various prospective trials has traced the relationships of serum levels of uric acid and the stroke coincidence, but the study findings were found to be conflicting. multiple studies were reported that the uric acid is prescribed as a free radical scavenger and as an antioxidant protecting the brain tissue from an oxidative damage, so protecting against post-stroke terrible neurological outcomes (Hozawa A. et al, 2006), (Amaro S. et al, 2007).

Meanwhile, various other studies were observed that the state of hyperuricemia is found to be in charge of an exaggerated stroke occurrence and mortality (Amaro S, Planas AM. 2008). The reported discrepancy can be attributed to the variation in the sample sizes as well as, the geographic differences, population ethnicity, sample gender, the age, individual's socio-economic effectors and methodologies manipulated to perform the study (Zhang B. et al, 2010).

The present study attempts to arouse the interest of uric acid in regard to ischemic stroke condition and achieving better understanding of its role and completing the efforts of various former academic studies in addition to versatile clinical trials to finally presents a better managements and optimum care in events of the stroke condition (Hong JM. Et al, 2010).

Aims of the study:

The primary aim of this study is to evaluate the potential relationship between serum uric acid (SUA) levels and ischemic stroke. Specifically, the study seeks to:

1. Assess whether serum uric acid levels differ significantly between patients with ischemic stroke and healthy individuals.
2. Investigate the correlation between SUA and clinical parameters such as blood pressure, glucose, age, weight, and urea levels.
3. Determine whether SUA levels are associated with the severity of ischemic stroke.
4. Provide local data from Iraqi patients that may support future clinical and preventive strategies in stroke management.

MATERIAL AND METHODS

2.1. Study Design and Setting:

This study was designed as a cross-sectional observational study conducted at [Baquba Teaching Hospital], Diyala, Iraq, over a period of [from November 2024 to April 2025].

The aim was to assess the serum uric acid levels in patients diagnosed with ischemic stroke and to investigate their possible association with stroke severity.

Sampling and sample size : Convenience sampling technique employed. A convenience sample of 100 participants voluntarily agreed to join the study.

Ethical issue: This study was anonymous, voluntary, beneficial, and harmless.

Human subject protection: Taken information will be secured and names and they will be well informed about the purposes of the study.

Study Population:

The study included a total of 100 participants, comprising ischemic stroke patients who were admitted to the neurology ward and diagnosed based on clinical examination and neuroimaging (CT or MRI). A control group of healthy individuals, matched for age and gender, was also included for comparison.

Inclusion Criteria:

- Patients aged ≥18 years.
- Confirmed diagnosis of acute ischemic stroke via CT or MRI.
- Consent to participate in the study.

Exclusion Criteria:

- Patients with hemorrhagic stroke.
- History of chronic kidney disease, gout, or malignancy.
- Use of medications that affect uric acid levels (e.g., allopurinol, diuretics).
- Recent infection or inflammatory conditions.

2.2. Demographic data:

Patients demographic data such as (age, gender, weight, address, …. etc.), in addition to other clinical and biochemical parameters and treatment the patients were receiving before and during hospitalization period mentioned in the current study were recruited from patient's files and from direct questionnaire either from the patients or by their companions or the medical staff.

2.3. Serum Uric Acid Measurement – Procedures

1. Blood Sample Collection (Venipuncture Procedure):

• Patient Preparation:

The patient should ideally fast for 4–8 hours prior to blood collection to avoid any dietary interference with serum uric acid levels.

• Materials Required:

• Tourniquet

• 70% isopropyl alcohol swab

• Sterile needle and syringe or vacuum blood collection system

• Plain vacutainer tube (red-top or yellow-top without anticoagulant)

• Gloves, gauze, and bandage

• Procedure:

1. Confirm the patient's identity and explain the procedure.
2. Apply the tourniquet 3–4 inches above the venipuncture site.
3. Clean the site with an alcohol swab using circular motion and allow it to dry.
4. Insert the needle into the vein and collect 3–5 mL of blood into a plain vacutainer tube.
5. Remove the tourniquet once blood starts flowing.
6. After collection, withdraw the needle, apply pressure to the site with gauze, and cover it with a bandage.
7. Label the tube and transport it to the laboratory.

• Sample Handling:

Allow the blood to clot at room temperature for 15–30 minutes. Then centrifuge at 3000 rpm for 10 minutes to separate the serum. Transfer the clear serum to a clean tube for analysis.

2. Laboratory Measurement of Serum Uric Acid:

• Method:

The most common method used is the enzymatic colorimetric method using uricase.

• Principle:

Uric acid is oxidized by uricase enzyme to produce allantoin and hydrogen peroxide. The hydrogen peroxide then reacts with a chromogen in the presence of peroxidase to form a colored compound. The intensity of the color, measured spectrophotometrically at 520–550 nm, is proportional to the uric acid concentration.

• Procedure (General Steps):

1. We prepared reagents and calibrators according to the kit manufacturer's instructions.
2. We added specific volumes of serum sample, reagent 1 (uricase), and reagent 2 (peroxidase + chromogen) into cuvettes or reaction wells.
3. Then we incubated the mixture at 37°C for 5–10 minutes.
4. measured the absorbance using a spectrophotometer at 520–550 nm.
5. Finally we calculated the uric acid concentration using a standard calibration curve [Tetik V, Tetik S, Kurban S, et al. 2022.]

2.4. Statistical analysis

The statistical analysis for this study was conducted using IBM SPSS Statistics Version 29.0.1.0. The analysis process began with descriptive statistics to provide an overview of the continuous variables in the dataset, including measures such as mean, standard deviation, minimum, and maximum values. This helped in understanding the general characteristics of the study population, such as age, weight, and biochemical parameters.

Frequencies and percentages were calculated for categorical variables such as gender, patient status (normal vs. abnormal), level of consciousness, and disease severity (mild, moderate, severe), allowing us to describe the distribution of these categories in the sample.

To assess whether there were statistically significant differences in uric acid levels between groups (e.g., normal vs. abnormal patients), an Independent Samples t-test was applied. When assumptions of normality and equal variances were not met, the Kruskal-Wallis H test, a non-parametric alternative, was used to compare uric acid levels across more than two independent groups such as categories of disease severity or consciousness level.

Furthermore, Pearson correlation analysis was used to examine the linear relationships between uric acid levels and other continuous variables including urea, glucose, systolic and diastolic blood pressure, age, and patient weight. This helped to explore whether increases in uric acid were associated with changes in these physiological measures.

Pairwise comparisons with Bonferroni adjustments were conducted following the Kruskal-Wallis tests to identify which specific groups differed from each other when overall significance was found.

RESULTS AND OBSERVATIONS:

3.1.Patient Status

This part shows the distribution of patient cases categorized as normal or abnormal, providing an overview of case severity in the sample.

Table (1): Patient Status

Patient Status			
		Frequency	Percent%
Valid	abnormal	56	56.0%
	normal	44	44.0%
	Total	100	100.0%

Abnormal: 56 participants (58.0% of the total sample)
Normal: 44 participants (42.0% of the total sample)
Total: 100 participants (100.0%)

3.2.Gender

Here we explore the gender distribution in the sample to determine the balance between male and female participants.

Table (2): gender distribution in the sample

Gender				
		Frequency	Percent	Valid Percent
Valid	Female	45	45.0	45.0
	Male	55	54.0	54.0
	Total	100	100.0	100.0

Female: 45 participants (45.0% of the total sample)

Male: 55 participants (54.0% of the total sample)
Total: 100 participants (100.0%)
The gender distribution is fairly balanced, with slightly more males (55%) than females (45%).

3.3.Descriptive Statistics
This section provides a summary of the main continuous variables such as age, weight, glucose, urea, and uric acid to understand the central tendency and variability among patients.

Table (3): Descriptive Statistics of participants

Descriptive Statistics				
	Minimum	Maximum	Mean	Std. Deviation
Age	10	86	52.46	16.655
Patient weight	34.0	175.0	74.693	17.4883
Urea	18.90	128.00	42.7986	19.15211
Glucose	76.8	278.0	132.705	44.9320
Uric Acid	2.1	11.2	5.816	2.5062

1. Age:
Mean = 52.46, Min = 10, Max = 86, Std. Deviation = 16.655.
The sample has a wide age range, with an average age of 52.46 years.
2. Weight:
Mean = 74.69, Min = 34.0, Max = 175.0, Std. Deviation = 17.49.
Weight varies significantly among the participants, with an average of 74.69 kg.
3. Urea:
Mean = 42.80, Min = 18.90, Max = 128.00, Std. Deviation = 19.15.
Urea levels show considerable variation, with an average level of 42.80.
4. Glucose:
Mean = 132.71, Min = 76.8, Max = 278.0, Std. Deviation = 44.93.
Blood glucose levels vary greatly, with an average of 132.71.
5. Uric Acid:
Mean = 5.82, Min = 2.1, Max = 11.2, Std. Deviation = 2.51.
Uric acid levels are less variable compared to glucose and urea, with an average of 5.82.

General Interpretation:
Age and weight show significant variation in the sample.
Urea, glucose, and uric acid also exhibit wide variation, indicating diverse health conditions among participants.
The standard deviations suggest that these values are spread out around their means.

Independent Samples Test
We test whether uric acid levels significantly differ between normal and abnormal patient groups using an independent samples t-test.
1. Levene's Test for Equality of Variances:
F = 25.872, Sig. < .001:
Since the Sig. value is less than 0.05, this indicates that the assumption of equal variances between the two groups (normal and abnormal) is violated. Therefore, we will use the row labeled "Equal variances not assumed" for interpretation.

2. t-test for Equality of Means (using Equal variances not assumed):
t = 1.444, df = 83.475, Sig. (2-tailed) = 0.076:
The p-value (0.076) is greater than 0.05, which indicates that there is no statistically significant difference in the mean levels of Uric Acid between the two groups (normal vs abnormal).
This means that, based on this data, the difference in Uric Acid levels between the normal and abnormal groups is not significant.

3. Mean Difference:

Mean Difference = 0.152:
The difference in the mean levels of Uric Acid between the two groups is 0.152.

Table (4): Independent Samples Test

Independent Samples Test											
	Levene's Test for Equality of Variances		t-test for Equality of Means								
	F	Sig.	t	df	Significance		Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference		
					One-Sided **p**	Two-Sided **p**			Lower	Upper	
Equal variances assumed	25.872	<.001	1.444	98	.076	.152	.7200	.4985	-.2693	1.7093	
Equal variances not assumed			1.444	83.475	.076	.152	.7200	.4985	-.2714	1.7114	

4. Standard Error Difference:
Std. Error Difference = 0.4985:
This is the standard error of the mean difference, which indicates the precision of the estimate.
5. 95% Confidence Interval of the Difference:
Lower = -0.2693 , Upper = 1.7093:
The 95% confidence interval for the difference in means ranges from -0.2693 to 1.7093.
Since 0 is within this range, it further supports the finding that there is no statistically significant difference in Uric Acid levels between the two groups.

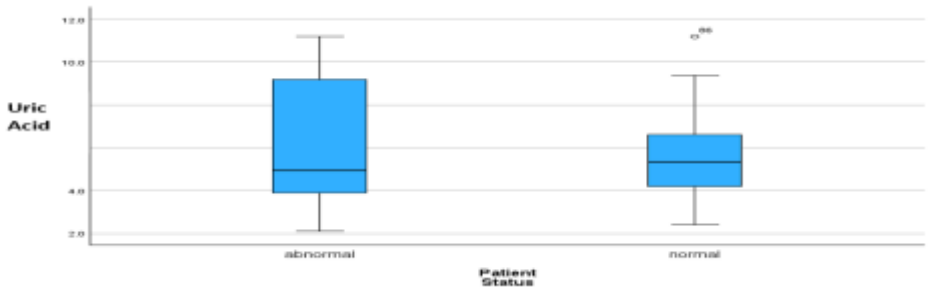


Figure (3): Uric Acid levels between the two groups

Based on the Independent Samples t-test, we can conclude that there is no significant difference in Uric Acid levels between the normal and abnormal groups, as the p-value is greater than 0.05.

Independent-Samples Kruskal-Wallis Test (Disease Severity)

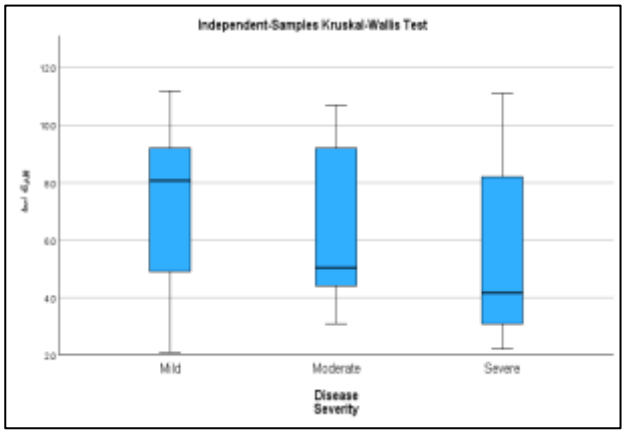
A non-parametric Kruskal-Wallis test is used to examine differences in uric acid levels across different disease severity levels (mild, moderate, severe).

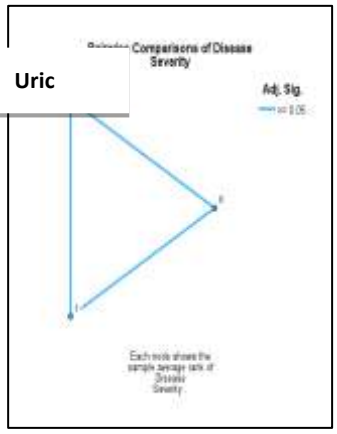
Table (5): Independent-Samples Kruskal-Wallis Test

Independent-Samples Kruskal-Wallis Test Summary	
Total N	50
Test Statistic	2.255[a]
Degree Of Freedom	2
p-value	.324

The Kruskal-Wallis test was conducted to determine whether there are statistically significant differences in Uric Acid levels across the three categories of Disease Severity (e.g., Mild, Moderate, Severe). The test yielded a chi-square value of 2.255 with 2 degrees of freedom and a p-value of 0.324.
Since the p-value is greater than 0.05, we fail to reject the null hypothesis. This indicates that there is no statistically significant difference in uric acid levels between the different disease severity groups.

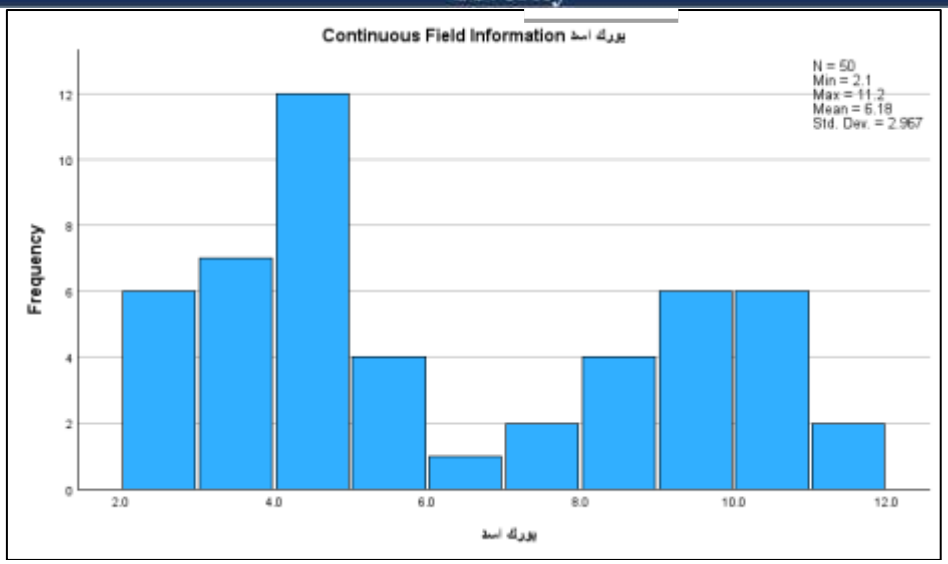
Table (5): Pairwise Comparisons of Disease Severity
Adjusted p-values (Adj. Sig.) are all **greater than 0.05** for all comparisons between the severity levels (Severe,Moderate, Mild) regarding Uric Acid.
Since **all adjusted p-values are above 0.05**, this indicates **no statistically significant dikfferences** between these groups.





Sample 1-Sample 2	Test Statistic	Std. Error	Std. Test Statistic	Sig.	Adj. Sig.[a]
Severe-Moderate	5.732	4.630	1.238	.216	.647
Severe-Mild	6.955	5.556	1.252	.211	.632
Moderate-Mild	1.222	5.745	.213	.832	1.000

Pairwise Comparisons of Disease Severity



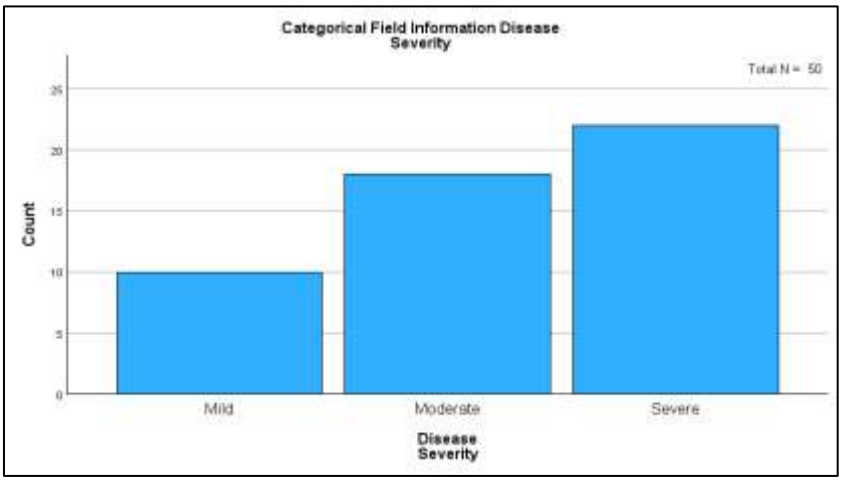


Figure (4):Pairwise Comparisons of Disease Severity

The statistical analysis revealed that there are **no significant differences** in Uric Acid levels between patients with **Severe**, **Moderate**, or **Mild** disease severity.

3.4.Correlation between Uric Acid and Urea

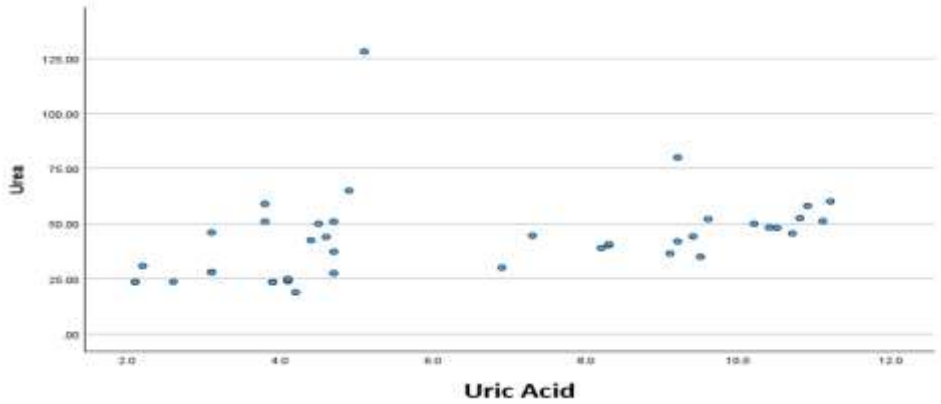
This section examines whether there's a linear relationship between uric acid and urea levels in the blood.

Table (6): Correlation between Uric Acid and Urea

Correlations			
		Uric Acid	**Urea**
Uric Acid	Pearson Correlation	1	.435*
	p-value		.043
	N	30	22
Urea	Pearson Correlation	.435*	1
	p-value	.043	
	N	22	22
*. Correlation is significant at the 0.05 level (2-tailed).			

- The **Pearson correlation coefficient (r = 0.435)** indicates **a moderate positive relationship** between **Uric Acid** and **Urea** levels.
- The **p-value = 0.043 < 0.05**, which means the correlation is **statistically significant.**
- The analysis was based on **22 abnormal cases** (where both Uric Acid and Urea were available).

Figure (5): Correlation between Uric Acid and Urea



There is a statistically significant moderate positive correlation between **Uric Acid** and **Urea** levels among abnormal patients **(r = 0.435, p = 0.043**). As Urea levels increase, Uric Acid levels tend to increase as well.

3.5.Correlation between Uric Acid and Glucose

We analyze the association between uric acid and glucose levels to check for any potential metabolic link.

Table (7): Correlation between Uric Acid and Glucose

Correlations			
		Uric Acid	**Glucose**
Uric Acid	Pearson Correlation	1	.144
	p-value		.523
	N	30	22
Glucose	Pearson Correlation	.144	1
	p-value	.523	
	N	22	22

- The **Pearson correlation coefficient (r = 0.144)** indicates a **very weak positive relationship** between **Uric Acid** and **Glucose.**
- The **p-value = 0.523 > 0.05**, meaning the correlation **is not statistically significant**.
- The analysis was based on **22 abnormal patients**.

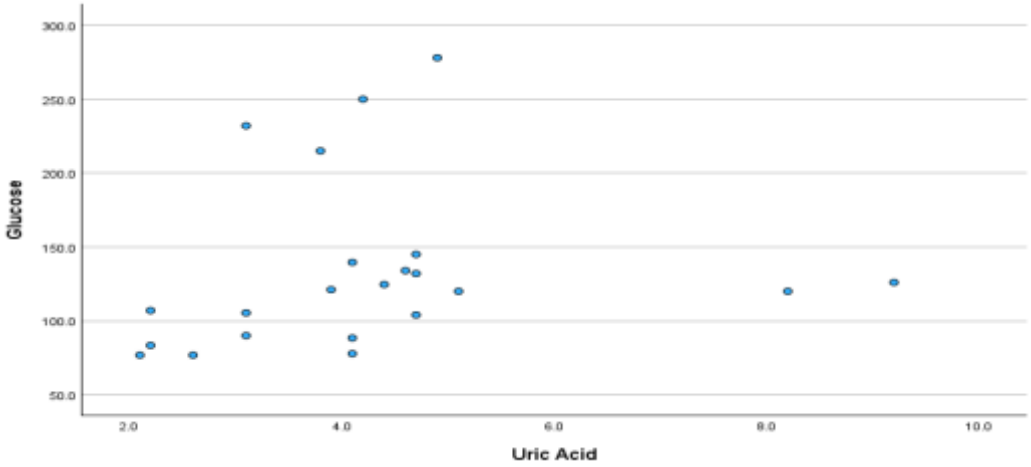


Figure (6): Correlation between Uric Acid and Glucose

There is no statistically significant relationship between **Uric Acid** and **Glucose** among abnormal patients **(r = 0.144, p = 0.523)**. This suggests that variations in Glucose levels are not associated with changes in Uric Acid levels in this group.

3.6. Correlation between Uric Acid and Blood Pressure

This part investigates the relationship between uric acid levels and both systolic and diastolic blood pressure.

- There is **no significant relationship** between **Uric Acid** and either **Systolic** or **Diastolic Blood Pressure** among abnormal patients.
- Both correlations are weak (r ≈ 0.1 or less), and **p-values > 0.05**, indicating **no statistical significance**.

However, there is a **strong and significant positive correlation** between Systolic and Diastolic pressure **(r = 0.667, p < 0.001**), which is expected physiologically

Table (8): Correlation between Uric Acid and Blood Pressure

Correlations				
		Uric Acid	Systolic_BP	Diastolic_BP
Uric Acid	Pearson Correlation	1	-.078	.114
	p-values		.490	.309
	N	98	81	81
Systolic_BP	Pearson Correlation	-.078	1	.667**
	p-value	.490		<.001
	N	81	82	82
Diastolic_BP	Pearson Correlation	.114	.667**	1
	p-value	.309	<.001	
	N	81	82	82

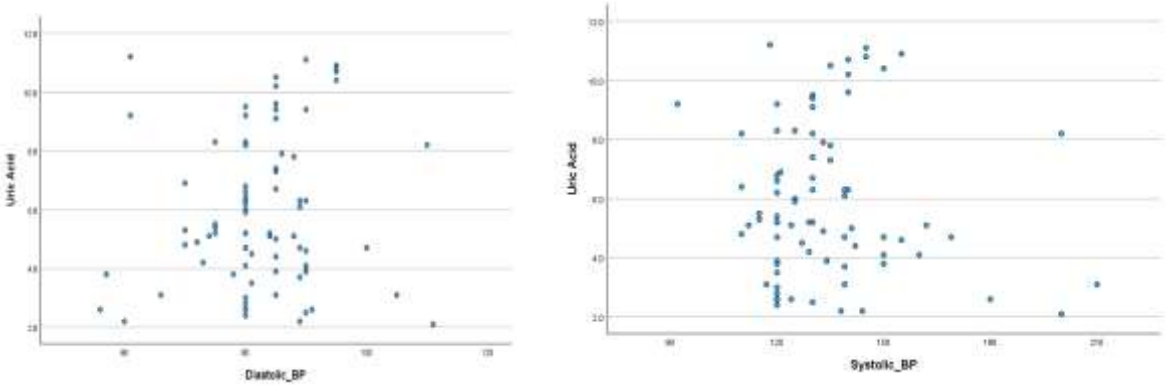


Figure (7): Correlation between Uric Acid and Blood Pressure

Uric Acid levels do not show a statistically significant association with either systolic or diastolic blood pressure among abnormal patients. This suggests that variations in Uric Acid are independent of blood pressure values in this group.

3.7.Correlation between Uric Acid and Age

We assess whether age has any influence on uric acid levels among the studied patients.

Table (9): Correlation between Uric Acid and Age

Correlations			
		Uric Acid	Age
Uric Acid	Pearson Correlation	1	.013
	p-value		.901
	N	98	96
Age	Pearson Correlation	.013	1
	p-value	.901	
	N	96	98

Pearson Correlation Coefficient (r) = 0.013**:** **This value is very close to** 0**, indicating a** very weak correlation **between** Uric Acid **and** Age**.**

– **Significance (p-value) = 0.901**: The **p-value is much greater than 0.05**, indicating that the correlation between **Uric Acid** and **Age** is **not statistically significant**.

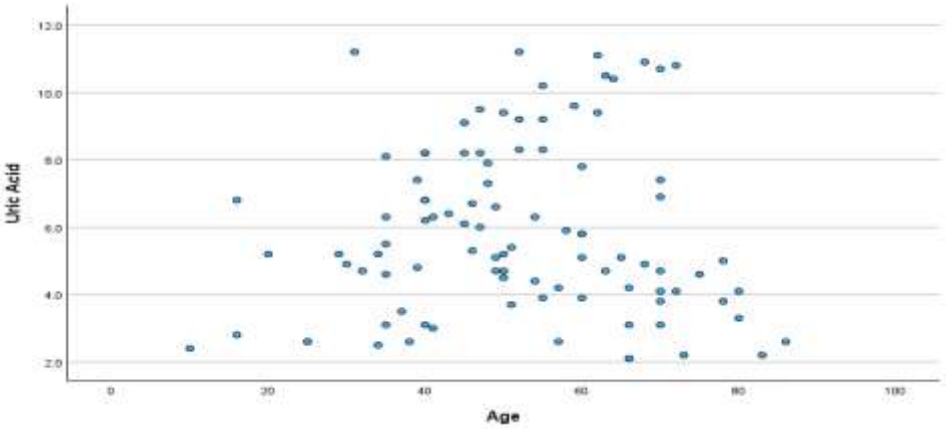


Figure (8): Correlation between Uric Acid and Age

There is **no significant correlation** between **Uric Acid** levels and **Age** in the sample. The **very weak correlation** (r = 0.013) and **non-significant p-value** (p = 0.901) suggest that age does not influence or correlate with uric acid levels in this sample.

3.8. Correlation between Uric Acid and Patient Weight

This section analyzes if there's a relationship between a patient's body weight and uric acid concentration.

Table (10): Correlation between Uric Acid and Patient Weight

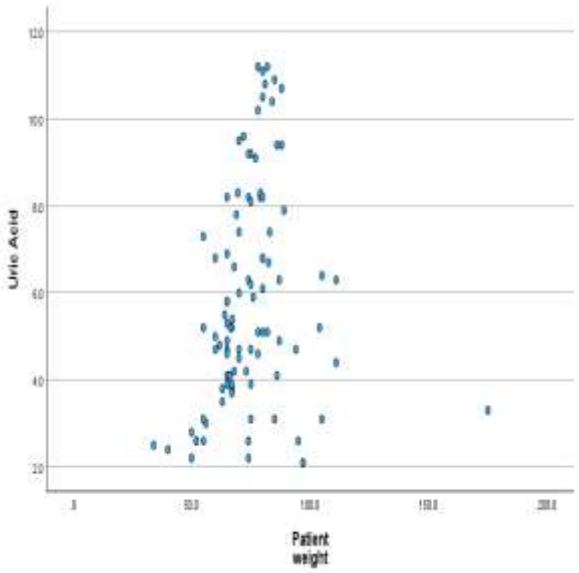
Correlations			
		Uric Acid	Patient weight
Uric Acid	Pearson Correlation	1	.165
	p-value		.103
	N	98	98
Patient weight	Pearson Correlation	.165	1
	p-value	.103	
	N	98	100

– **Pearson Correlation Coefficient (r) = 0.165**: This is a **weak positive correlation** between **Uric Acid** and **Weight**, indicating that there is a very slight tendency for **higher weight** to be associated with **higher levels of uric acid**. However, this correlation is weak.

– **Significance (p-value) = 0.103**: The **p-value** is **greater than 0.05**, meaning that the correlation is **not statistically significant**. Therefore, we cannot conclude that there is a meaningful relationship between **weight** and **uric acid** levels in the sample

Figure (9): Correlation between Uric Acid and Patient Weight

There is a **weak and non-significant correlation** between **Uric Acid** levels and **Weight** in this sample. The p-value (0.103) suggests that any relationship observed between the two variables could likely be due to random chance.



DISCUSSION

The current study traced the relationship between serum uric acid (SUA) levels and ischemic stroke among a sample of 56 patients and 44 participants as a controls. Through conducting comprehensive statistical analyses, multiple findings were elucidated in regard to the role of SUA in ischemic stroke.

Independent samples t-test revealed no statistically significant difference in the SUA levels between ischemic stroke patients and those in normal group ($p = 0.076$). This finding suggests that SUA independently may not contribute to onset of the ischemic stroke. This obtained observation aligns with other several other former findings such as those performed by (Hong et al, 2010 and Li et al, 2023) which revealed no causal definitive link between SUA levels and stroke outcomes markers after adjusting for the confounding variables.

Correlation analyses didn't reveal a significantly reported relationships between the SUA and the key physiological parameters including glucose ($r = 0.144$, $p = 0.523$), the systolic and the diastolic blood pressure ($r = -0.078$ and 0.114, respectively), age ($r = 0.013$, $p = 0.901$), or body weight ($r = 0.165$, $p = 0.103$). This propose that serum uric acid variations are likely to be an independent of these common clinical markers in the enrolled population study.

However, we noticed a pronounced exception after examining the statistics of our data shown as a positive moderate correlation between the SUA and serum urea levels ($r = 0.435$, $p = 0.043$), which indicates that the integrity of kidney function plays a crucial role in the determination SUA level. Given that kidneys are considered as the primary organ for uric acid excretions, so, any amelioration in renal clearance that resulted in an elevated SUA, can indirectly be a predisposing factor for ischemic stroke outcomes.

Furthermore, the present data didn't reveal a statistically significant difference in the SUA levels across different categories of patients after were distributed according to disease severity ($p = 0.324$). This indicate that the SUA level cannot be considered as predictive of the disease severity, a finding which questions utility of the serum uric acid levels as a biomarker in assessing disease clinical progression or the prognosis in the ischemic stroke.

Despite the prevailed lack of data statistical significance in the present study; the existing literature within this issue offers a conflicting evidence. Chamorro and colleagues (Chamorro et al, 2002) and Amaro and colleagues (Amaro et al, 2007, 2008) were concluded a potential neuroprotective role of the SUA, and linked this observation to its antioxidant capacity. On the hand, other studies such as those conducted by Zhang et al. (2010) and Hozawa et al. (2006) associate hyperuricemia levels with worsening of stroke outcomes and was accompanied with increased mortality rates. These discrepancies among various studies findings could be attributed to the methodological variations, the differences in studied populations, other geographic factors, and the associated comorbidities.

Despite what revealed by the findings of the present study, the role of the SUA in ischemic stroke still controversial owing to the conflicting results prevailed by previous literature. So, further research required involving larger sample size and studying more diverse populations, conducting a longitudinal design, in addition to comprehensive control aiming to confounding the variables is crucial to reveal the real nature of involvement of SUA in the stroke pathology.

In summary, despite SUA level does not look to serve as a direct risk factor or revealed obviously as a prognostic indicator for the ischemic stroke in the present study; its contribution in the stroke pathophysiology cannot be completely ignored.

CONCLUSION

1.The present study didn't disclose a direct role of serum uric acid levels in incidence of the ischemic stroke in our studied population.

2.Furthermore, there was no direct link was elucidated between serum uric acid levels and the severity of ischemic stroke status of the patients.

3.There was an obvious correlation between serum uric acid levels and renal function integrity, highlighting the clinical importance of this marker in evaluating kidney function and follow up in those patients.

4.Overall, the current study confirms the metabolic role of uric acid and consequently its predicting importance of entire patient health status and the effectiveness of treatment in in- and out-patients diagnosed with ischemic stroke.

REFERENCES

1. Khalil MI, Salwa M, Sultana S, Al Mamun MA, Barman N, Haque MA. Role of serum uric acid in ischemic stroke: A case-control study in Bangladesh. PLoS One. 2020; 15: e0236747. doi:10.1371/journal.pone.0236747.
2. Saini V, Guada L, Yavagal DR. Global epidemiology of stroke and access to acute ischemic stroke interventions. Neurology. 2021; 97: S6-S16. doi:10.1212/WNL.0000000000012781
3. Bansal BC, Gupta RR, Bansal MR, Prakash C. Serum lipids and uric acid relationship in ischemic thrombotic cerebrovascular disease. Stroke. 1975; 6: 304-307.
4. Sridharan R. Risk factors for ischemic stroke: A case control analysis. Neuroepidemiology. 1992; 11: 24-30. doi:10.1159/000110902.
5. Chamorro A, Obach V, Cervera A, Revilla M, Deulofeu R, Aponte JH. Prognostic significance of uric acid serum concentration in patients with acute ischemic stroke. Stroke. 2002; 33: 1048 1052.
6. Weir CJ, Muir SW, Walters MR, Lees KR. Serum urate as an independent predictor of poor outcome and future vascular events after acute stroke. Stroke. 2003; 34: 1951-1956.
7. Hozawa A, Folsom AR, Ibrahim H, Nieto FJ, Rosamond WD, Shahar E. Serum uric acid and risk of ischemic stroke: The ARIC study. Atherosclerosis. 2006; 187: 401-407.
8. Amaro S, Soy D, Obach V, Cervera A, Planas AM, Chamorro A. A pilot study of dual treatment with recombinant tissue plasminogen activator and uric acid in acute ischemic stroke. Stroke. 2007; 38: 2173-2175.
9. Amaro S, Planas AM, Chamorro Á. Uric acid administration in patients with acute stroke: A novel approach to neuroprotection. Expert Rev Neurother. 2008; 8: 259-270.
10. Zhang B, Gao C, Yang N, Zhang W, Song X, Yin J, et al. Is elevated SUA associated with a worse outcome in young Chinese patients with acute cerebral ischemic stroke? BMC Neurol. 2010; 10: 82.
11. Hong JM, Bang OY, Chung CS, Joo IS, Gwag BJ, Ovbiagele B. Influence of recanalization on uric acid patterns in acute ischemic stroke. Cerebrovasc Dis. 2010; 29: 431-439.
12. Feigin VL, et al. "Global and regional burden of stroke during 1990–2010: findings from the Global Burden of Disease Study 2010." The Lancet, 2014. https://pubmed.ncbi.nlm.nih.gov/24449944
13. Donnan GA, Fisher M, Macleod M, Davis SM. Stroke. Lancet. 2008;371(9624):1612-1623. doi:10.1016/S0140-6736(08)60694-7 https://pubmed.ncbi.nlm.nih.gov/17707117
14. Caplan LR. Types of ischemic stroke. In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. Accessed [insert access date]. 2023.
15. Maiuolo J, Oppedisano F, Gratteri S, Muscoli C, Mollace V. Regulation of uric acid metabolism and excretion. Int J Cardiol. 2016;213:8-14. doi:10.1016/j.ijcard.2015.08.086 https://pubmed.ncbi.nlm.nih.gov/26610459
16. Glantzounis GK, et al. "Uric acid and oxidative stress." Current Pharmaceutical Design, 2005. https://pubmed.ncbi.nlm.nih.gov/16178772
17. Sautin YY, Johnson RJ. "Uric acid: the oxidant-antioxidant paradox." Nucleosides Nucleotides Nucleic Acids, 2008.
18. https://pubmed.ncbi.nlm.nih.gov/1905370
19. https://www.uptodate.com/contents/types-of-ischemic-stroke
20. Milionis HJ, et al. "Serum uric acid levels and risk of ischemic stroke in elderly individuals: The EPIC–Norfolk Study." Stroke, 2005.
21. https://pubmed.ncbi.nlm.nih.gov/16239005
22. Amaro S, et al. "Uric acid levels and clinical outcomes in patients with acute ischemic stroke." Stroke, 2015.
23. https://pubmed.ncbi.nlm.nih.gov/25744569
24. King, R.D.; Kelley, E.E. Urate Biology and Biochemistry: A Year in Review 2022. Gout Urate Cryst. Depos. Dis. 2023, 1, 115-121.
25. https://doi.org/10.3390/gucdd1030011
26. Li J, et al. "Serum uric acid and prognosis of ischemic stroke: cohort study and meta-analysis." Frontiers in Neurology, 2023.
27. https://pubmed.ncbi.nlm.nih.gov/37905729
28. Tetik V, Tetik S, Kurban S, et al. "Evaluation of Serum Uric Acid Levels in Patients with Ischemic Stroke and Its Association with Stroke Severity." Journal of Stroke and Cerebrovascular Diseases,

vol. 31, no. 5, 2022, 106488. DOI: 10.1016/j.jstrokecerebrovasdis.2022.106488

29. https://doi.org/10.1016/j.jstrokecerebrovasdis.2022.106488