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TO EVALUATE THE UTILITY OF SERUM URIC ACID IN THE PROGNOSIS OF ACUTE ISCHEMIC STROKE

General Medicine

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ABSTRACT

Acute ischemic stroke carries significant morbidity, disability, and mortality rates, imposing a substantial burden on patients and society. Uric acid (UA) is the end product of purine metabolism. The role of uric acid (UA) in health and disease remains complex due to its dual nature as both a pro-oxidant and an antioxidant. UA can contribute to inflammatory responses, vascular endothelial cell damage, platelet aggregation, and oxidative stress. However, it also has neuroprotective effects and preserves vascular endothelial cell function. The relationship between UA and stroke prognosis remains inconclusive in observational studies, prompting further investigation.

KEYWORDS

Uric Acid, Stroke Prognosis, Registry, Mendelian Randomizatio

INTRODUCTION

Globally, stroke is the third most common cause of mortality and the fourth leading cause of disease burden. In India, stroke was the largest contributor to disability adjusted life years (DALYs), and a chief contributor to deaths caused by neurological disorders, accounting for 37.9% of total neurological disorder DALYs, as estimated by the Indian Global Burden of Disease Study 1990-2019. A stroke or cerebrovascular accident is defined as an abrupt onset of a neurologic deficit that is attributable to a focal vascular cause. Stroke is the third common cause of death in the world after coronary heart disease and cancer especially in the elderly.[1,2] The mortality rate of stroke in the acute phase is as high as 20% and it remains higher for several years after the acute event in stroke patients than in the general population.[1] Cerebral ischemia initiates a complex cascade of metabolic events, generating nitric oxide and free oxygen radicals.[3] These free radicals and reactive oxygen species (ROS) mediate a great part of injuries appearing after a transitory ischemic attack or during permanent ischemia, modifying macromolecules especially DNA , initiating apoptosis and necrosis.

Serum uric acid being one of the major aqueous antioxidant in human beings should have a protective role in stroke patients. Several large studies have provided conflicting results regarding the clinical significance of elevated serum uric acid levels in cerebrovascular diseases. Many studies including the National Health And Nutrition Examination Survey (NHANES) study concluded that uric acid is an independent risk factor for development of cardiovascular and cerebrovascular diseases.[4] By contrast a prospective hospital-based study involving 881 patients found that higher level of serum urate predicted better outcomes following stroke, suggesting that serum urate may be beneficial and protect against poor outcomes.[5] In addition an experimental study showed that uric acid administered early after thromboembolic stroke is neuroprotective in the rat brain and it extends the benefits of recombinant tissue plasminogen activator (rtPA).[6]

Types of Stroke

Acute stroke is the rapid onset of focal neurological findings in a vascular territory due to cerebrovascular disease. The two main types are:

- **Ischemic Stroke:** Caused by the interruption of blood flow to a certain area of the brain and accounts for 85% of all acute strokes. The most common mechanism is embolism, often generated from thrombi in the heart due to cardiac diseases like atrial fibrillation, valvular heart disease, and cardiomyopathy.

- **Hemorrhagic Stroke:** Caused by the bursting of a blood vessel (acute hemorrhage) and accounts for 15% of all acute strokes.

A transient ischemic attack (TIA) is a transient episode of neurologic dysfunction due to focal brain, spinal cord, or retinal ischemia, without acute infarction or tissue injury, and is considered a medical emergency.

Uric Acid and Ischemic Stroke

The progression of ischemic stroke is closely related to oxidative stress, a component of ischemia-reperfusion injury, which involves excessive production of reactive oxygen species (ROS).

- The brain is more susceptible to oxidative stress damage due to fewer endogenous antioxidants.
- **Uric Acid (UA)** is an endogenous antioxidant derived from purine metabolism. It is involved in almost 70% of free radical scavenging reactions in humans, neutralizing peroxynitrite, hydroxyl, and superoxide radicals, and playing a major role in preventing lipid peroxidation.
- Xanthine oxidase activity and UA synthesis increase under ischemic conditions, suggesting elevated serum UA may mark underlying tissue ischemia.
- UA presents a paradox: it is an antioxidant but also contributes to local tissue inflammation and plaque progression by accumulating as crystals within atherosclerotic plaques.

In the central nervous system, beneficial antioxidant actions seem to predominate over harmful pro-oxidant effects.

Materials and Methods

It was a hospital based analytical cross sectional study conducted in the department of medicine during the period of 2 yea
Patients more than 18 years of age and Patient with NCCT head/MRI brain confirmed case of acute ischemic stroke were included in the study.

Old CVA (Cerebrovascular Accident), Hemorrhagic stroke, CVT (Cerebral Venous Thrombosis), Known case of valvular heart disease, Patients receiving drugs that alter serum uric acid level (e.g., Diuretics, Allopurinol, Losartan, Fenofibrate, Febuxostat), Known case of malignancy, Renal dysfunction, Gout and More than moderate alcohol consumption were exc;ided frp, the study.

Data collection:

After approval from Ethical committee and obtaining informed consent form Complete history and examination were conducted for patients presenting with symptoms/signs of stroke or diagnosed with stroke within 7 days of onset. The patient was sent for NCCT head or MRI brain, and the report was collected. For eligible patients, 2ml of venous blood was collected in a plain vial for serum uric acid level analysis. Severity of stroke was assessed using the NIH Stroke Severity scale (NIHSS).

NIHSS Scoring System

(The NIHSS maximum score is 42. Scoring is as follows):

- Score 0: No Stroke
- Score 1–4: Minor Stroke

- Score 5–15: Moderate Stroke
- Score 15–20: Moderate to Severe Stroke
- Score 21–42: Severe Stroke

Statistical Analysis

Data was expressed as percentage and mean \pm S.D.. Kolmogorove-Smirnove analysis was performed for checking data linearity. Chi-square test was used to analyze the significance difference between two independent groups. Pearson correlation was used to find the correlation between the two variables. A **P-value < 0.05** was considered statistically significant.

Observations and Results

The study included a total of **125 patients** with acute ischemic stroke.
Demographic Profile

Mean Age: 57.68 \pm 11.23 years. The majority of patients (32.0%) were in the 56-65 age group. * **Gender:** Male: 85 patients (68.0%). Female: 40 patients (32.0%).

.0%). 69.6% of patients suffered from dyslipidemia (87 out of 125). 81.6% of patients were hypertensive (102 out of 125). 42.4% of patients suffered from diabetes mellitus (53 out of 125). 42.4% of patients had a smoking history (53 out of 125).

NIHSS Distribution

The majority of patients were categorized as Moderate to Severe Stroke based on the NIHSS (National Institutes of Health Stroke Scale):

- Moderate Stroke (Score 5–15): 31.2% (39 patients).
- Moderate to Severe Stroke (Score 15–20): 41.6% (52 patients).
- Severe Stroke (Score 21–42): 27.2% (34 patients).

Correlation and Association Findings

Correlation between Age and Serum Uric Acid (SUA) Levels

- There was a **significant correlation** between age groups and SUA levels (R value = 0.780, P value = 0.001).
- Mean SUA levels generally increased with age, from 4.6000~\text{mg}/\text{dL} in the \le 25 group to 10.7500~\text{mg}/\text{dL} in the 76-85 age group.

Correlation between NIHSS and SUA Levels

- There was a **significant correlation** between NIHSS and SUA levels (R value = 0.923, P value = 0.001).
- The mean SUA levels increased with stroke severity:
- Moderate Stroke: 5.9005~\text{mg}/\text{dL}
- Moderate to Severe Stroke: 7.7635~\text{mg}/\text{dL}
- Severe Stroke: 10.7121~\text{mg}/\text{dL}

Association of Risk Factors with SUA Levels

All associations showed a statistically significant difference (P<0.01) in SUA levels between groups:

Risk Factor	Group	Mean SUA (mg/dL)	P value
Hypertension	No	6.4835	0.005^{**}
	Yes	8.3226	
Diabetes Mellitus	No	6.9875	0.007^{**}
	Yes	9.3383	
History of Smoking	No	6.79	0.008^{**}
	Yes	9.60	
Dyslipidemia	No	5.9426	0.002^{**}
	Yes	8.8760	

Association between Sex and SUA Levels

There was a **statistical significant difference** in SUA levels between sexes (P value = 0.003). Female Mean SUA: 7.2400~\text{mg}/\text{dL}. Male Mean SUA: 8.3345~\text{mg}/\text{dL}.

Discussion

The present study was conducted to evaluate the role of uric acid in assessing the severity of acute ischemic stroke, addressing the conflicting results in human studies and the scarcity of data in the Indian scenario.[7]

Males were the majority (68%) with a male-to-female ratio of 2.1:1. Mean SUA levels were higher in males (8.33\pm1.99~\text{mg}/\text{dL}) than females (7.24\pm1.70~\text{mg}/\text{dL}) (P<0.003). This finding is consistent with other studies. The mean age was 57.68\pm11.23 years. The majority of patients were in the 56-65 age group (32%). The comparison of mean SUA levels showed a positive correlation between age and SUA.[8] Mean SUA levels were higher in the elderly, for example, cases over 75 years had a mean SUA level of .75\pm1.96~\text{mg}/\text{dL}. This aligns with studies that found SUA levels associated with an increasing risk for acute ischemic stroke in elderly patients.[9]

The study demonstrated a **positive correlation** between the National Institutes of Health Stroke Scale (NIHSS) score and the mean Serum Uric Acid (SUA) levels (Correlation coefficient, r=+0.923). Mean SUA levels increased with stroke severity, from 5.9\pm0.57~\text{mg}/\text{dL} for Moderate Stroke to 10.7\pm1.0~\text{mg}/\text{dL} for Severe Stroke.[10] This finding aligns with the results of a study by Shah H. et al. (2019) which concluded that the severity of acute ischemic stroke is directly proportional to the mean SUA levels (Correlation coefficient, r=+0.6979).[7] Conversely, some studies suggest a protective role, such as a meta-analysis by Wang Z et al. (2016), which concluded that high SUA level was associated with a better outcome after acute ischemic stroke. Another study found that for each \text{mg}/\text{dL} increase of SUA, there was a 12\% increase in the odds of good clinical outcome.[11]

The mean SUA level was found to be **directly proportional** to the presence of risk factors like hypertension, diabetes mellitus, dyslipidemia, and smoking. The majority of patients (81.6%) were hypertensive. SUA levels were significantly higher in hypertensive patients (8.32\pm1.95~\text{mg}/\text{dL}) compared to normotensive patients (6.48\pm1.15~\text{mg}/\text{dL}) (P<0.005). SUA levels in diabetic patients (9.33\pm1.85~\text{mg}/\text{dL}) were significantly higher than in non-diabetic subjects (6.98\pm1.36~\text{mg}/\text{dL}) (P<0.007). A positive correlation was found between dyslipidemia and SUA (P<0.002). The majority of patients (69.6%) were dyslipidemic. Mean SUA levels were higher in smokers(9.6\pm1.69~\text{mg}/\text{dL}) compared to non-smokers (6.79\pm1.12~\text{mg}/\text{dL}) (P=0.008), showing an association between smoking history and SUA levels.[12]

The present study suggests that SUA can be used as a marker for increased risk of stroke and for risk measurement after a stroke.

CONCLUSIONS

The study concluded that the serum uric acid (SUA) level is an important **biomarker for the prognosis of acute ischemic stroke**. Specifically, the findings support the conclusion that SUA level is associated with both:

- The **severity of acute ischemic stroke** (directly proportional to mean SUA levels).
- The **presence of other risk factors** for acute ischemic stroke (SUA levels are directly proportional to risk factors like hypertension, diabetes mellitus, dyslipidemia, and smoking).

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