



CLINICAL CORRELATION OF SERUM URIC ACID AND LIPID PROFILE IN ACUTE CEREBROVASCULAR EVENTS WITH RADIOLOGICAL STUDIES

General Medicine

Dr Gaurav Saini Post Graduate-General Medicine

Dr Niyaz Ahmad MD Medicine (Professor)

Dr Rohitash Sharma DM Neurology (Ass. Professor)

ABSTRACT

This study aimed to investigate the correlation between serum uric acid (SUA) levels, lipid profiles, and acute cerebrovascular events (CVEs) in patients with radiological evidence. A total of 50 patients admitted to the General Medicine wards with first-ever acute stroke within 24 hours of symptom onset were included in the study. The baseline characteristics of the participants, including age, fasting blood sugar (FBS), systolic blood pressure (SBP), diastolic blood pressure (DBP), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL), total cholesterol (TC), and uric acid (UA) were recorded. The results revealed that 68.2% of female participants and 57.1% of male participants had elevated SUA levels. The mean SUA level was found to be higher than the normal range, indicating a potential association between SUA and acute stroke. Furthermore, a significant positive correlation was observed between SUA levels and TC and TG levels, suggesting a possible link between SUA and abnormal lipid profiles. However, no significant correlation was found between SUA levels and dyslipidemia or the size of the stroke. It is important to note that this study had some limitations, including a small sample size and the inability to examine SUA levels across different subtypes of acute stroke. In conclusion, this study provides preliminary evidence of elevated SUA levels in patients with acute stroke and suggests a potential association between SUA and abnormal lipid profiles. Further research with larger sample sizes and a more diverse range of acute stroke subtypes is needed to elucidate the precise role of SUA and lipid profiles in acute cerebrovascular events. Such studies may contribute to a better understanding of the underlying mechanisms and potential therapeutic implications for stroke prevention and management.

KEYWORDS

INTRODUCTION:

A stroke is sudden cessation of blood flow to a part of the brain, following which neurological function is lost. The two forms of stroke are ischemic and hemorrhagic. Acute ischemic stroke, which is more common than hemorrhagic stroke, is a stroke brought on by thrombosis or embolism.

In worldwide after cancer and coronary artery disease, stroke is a third most prevalent cause of death, especially among the elderly [1,2]. In persons above 65 years old, the second most frequent cause of impairment is stroke and dementia; around 25% of stroke survivors go on to develop dementia [3] Stroke is a significant contributor to morbidity and long-term impairment; up to 40% of survivors are not expected to regain their ability to care for themselves or be independent[2].

Stroke incidence varies greatly among different countries. It was determined that India had a prevalence of stroke of 203 per 100,000 people over the age of 20, or roughly 1 million instances. The cause of up to 85% of strokes is ischemia. Atherosclerosis of small and major arteries supplying the brain is the most common cause of ischemic stroke. Normal perfusion is impeded, typically by a severe stenosis of arteries or blockage brought on by atherosclerosis [4]. It has variety of risk factors, like hypertension, smoking, hyperlipidemia, and diabetes [4].

In humans and higher animals, uric acid is the ultimate catabolite in the purine metabolism [5]. The kidney removes it from the plasma and stores it there as sodium urate [6]. Age and sexual orientation have an impact on uric acid levels. Prior to puberty, both males and girls typically have blood uric acid levels of 3.6 mg/dl Value reaches adult levels after puberty, with women having a value 1 mg/dl less than men. Oestrogen-induced increases in renal urate clearance are thought to be the cause of this lower quantity in women [5].

The established cardiovascular risk factors high blood pressure, high BMI, resistance to insulin, Mets, and elevated serum triglyceride have all been associated to increased uric acid levels [6,7]. Uric acid, has a property scavenger for free radical scavenger and having neuroprotection effects [6,8]. In humans, uric acid makes up around 50% of the plasma's antioxidant capability [8,9]. It is unclear how urate contributes to ischemic stroke. Higher serum urate upon admission was associated with worse outcomes (death or in-care) and a higher

rate of vascular events following ischemic stroke, according to a retrospective review of hospitalization data for 2495 patients in Glasgow [10]. On the other hand, a prospective hospital-based study comprising 881 patients discovered that a greater level of blood urate predicted better results after a stroke, indicating that serum urate may be advantageous and guard against bad outcomes [11].

The current study's objectives were to evaluate the blood uric acid levels of patients who had ischemic cerebrovascular accidents and to examine the relation between uric acid levels and lipids in this population.

MATERIALS AND METHODS:

The study was conducted in the Department of Medicine at Shri Guru Ram Rai Institute of Medical & Health Sciences and SMIH (Shri Mahant Indresh Hospital), Dehradun. The aim of the study was to investigate the correlation between acute stroke and serum uric acid (SUA) levels, as well as the connection between different lipid profile elements and stroke.

The research also aimed to assess the relationship between radiological tests, serum uric acid levels, and lipid profile levels. It was a prospective cross-sectional study carried out over a period of 1½ years in the General Medicine wards of SMIH, Dehradun, with a sample size of 50.

Inclusion Criteria-

Patients are admitted to our center with a first ever acute stroke, whether or not there is evidence of infarction or hemorrhage on an MRI or CT scan within 24 hours of the stroke's start.

Exclusion Criteria-

Patients who have previously experienced TIAs or CVAs

Patients receiving treatment with losartan or statins. Patients with known heart conditions that potentially be emboli sources or whose echocardiograms revealed sources of emboli. Patients with known gout cases, those who display clinical gout symptoms, or those who are using uricosuric medications.

Patients with myeloproliferative diseases or other hematological abnormalities like leukemia.

- Patients CT scan show space occupying lesions.
- Patients who take thiazide diuretics.

- Patients who suffer from chronic kidney failure.

Statistical Analysis:

The data thus obtained was analysed by appropriate statistical tests. Quantitative data was expressed by mean and standard deviation and difference of means was observed by t test and qualitative data was expressed as percentages and difference between proportions was observed using appropriate test. 95% confidence level was used to quantify at risk values and factors. Significant factors in study was considered using multivariate logistic regression $p < 0.05$ was considered significant. To calculate the significance of results we used- Pearsons correlation, Anova test and Z- Square test.

RESULTS:

Table1: Baseline characteristics of the study participants (N=50)

Characteristic	Cases (Mean $\pm$ SD)
Age	60.64(11.01)
FBS	152.44(68.38)
SBP	146.84(28.61)
DBP	86.80(17.52)
TG	150.43(59.08)
HDL-C	38.10(12.44)
LDL	122.62(54.40)
TC	209.51(51.94)
Uric Acid	7.23(1.76)

Table2: Correlation between Stroke & Other Parameters (N=50)

Parameter	Stroke		P Value
	Hemorrhagic Mean (SD)	Ischemic Mean (SD)	
Age (in Years)	71.8(15.2)	63.8(12.4)	0.182
SBP	176.0(18.2)	143.8(27.8)	0.015
DBP	106.0(23.0)	84.4(15.7)	0.008
FBS	137.6(31.1)	156.5(70.5)	0.557
TC	195.96(72.7)	211.01(50.0)	0.544
TG	132.4(88.4)	152.4(56.0)	0.478
HDL	44.5(15.9)	37.4(12.0)	0.228
LDL	121.9(51.1)	122.7(55.3)	0.976
Uric Acid	6.46(1.79)	7.29(1.75)	0.316
Size	34.94(38.44)	27.21(33.77)	0.634

Table3: Correlation between Lipid Profile and Uric Acid (N=50)

Lipid Profile	Uric Acid Correlation Coefficient	P Value
TC	0.624	<0.001
TG	0.484	<0.001
HDL	0.061	0.673
LDL	0.294	0.038

A positive correlation is seen between uric acid and TC, TG which was significant ($p < 0.001$).

DISCUSSION

There is limited information available regarding the potential risk that uric acid poses for vascular disease and acute stroke. As a result, it was intended for the current study to evaluate Serum uric acid levels in patients with ischemic cerebrovascular accident and to find whether there was any correlation, if any, between Serum uric acid levels and lipids with cerebrovascular accident which was ischemic in nature. The current cross-sectional study was conducted from 2020 to 2022 on 50 patients with cerebrovascular accidents at, Shri Guru Ram Rai Institute of Medical and Health Sciences, Department of medicine Dehradun, Uttarakhand.

In this current study, 36% of a patient were between the ages of 51 and 60, 66% were men, 20% had smoked, and 42% had drank alcohol in the past. 56% of people had hypertension and 54% had diabetes. MCA (86% of individuals with stroke was the most prevalent area.

In this current study, elevated blood uric acid levels were observed in 68.2% of female participants and 57.1% of male participants.

The mean serum uric acid level, which was elevated over a normal range at 7.21(1.75), were also found to be elevated. Acute stroke has serum uric acid levels that are on the higher side. These data imply that patients.

In this current study, 62%, 46%, 72%, and 72% of patients had abnormal levels of Total Cholesterol, Triglyceride, High density lipid, and Low-density lipid, respectively. A high majority of patients with elevated TC and TG were also found to have elevated blood uric acid ($p < 0.001$). Additionally, it was noted that there was no relationship ($P > 0.05$) between increased serum uric acid, dyslipidemia and the size of the stroke.

Further analysis of the relationship between serum uric acid and lipids revealed that, even while patients with increased LDL and aberrant HDL did not see a statistically significant change, patients with abnormal levels of lipid—i.e., elevated TC, HDL, LDL, and triglycerides—had significantly high mean serum uric acid levels. This study did have some limitations, including a limited sample size and the inability to examine serum uric acid levels throughout the range of acute stroke subtypes. Precise roles of serum uric acid and lipid profile would be further explored in research with bigger sample sizes and diverse acute stroke subtypes.

It will take further research to demonstrate a connection between lipid profile or serum uric acid and size of stroke.

CONCLUSION:

In this current study, 62% of acute stroke patients showed elevated serum uric acid levels. Additionally, it was discovered, the average serum uric acid levels were higher than expected (7.23 ± 1.76). MCA territory was the region with the highest rate of stroke.

Furthermore, the quantitative examination of lipids revealed considerably higher mean serum uric acid levels in patients with aberrant cholesterol, LDL, HDL, and triglycerides as well as significantly higher serum uric acid levels in individuals with elevated TC and triglycerides.

The relative mean values for TC, TG, HDL, and LDL were 209.5, 150.4, 38.11, and 122.6 mg/dL. Neither the lipid profile nor the SERUM URIC ACID was related to the size of the stroke.

REFERENCES

1. Hariklia VD, Apostolos H, Haralambosk The Role of Uric Acid in Stroke. The Issue Remains Unresolved. The Neurologist 2008;14:238-42.
2. Feigin VL, Lawes CM, Bennett DA, Barker-Collo SL, Parag V. Worldwide stroke incidence and early case fatality reported in 56 population-based studies: a systematic review. Lancet Neurol 2009;8(4):355-69.
3. Llibre J, Valhuerdi A, Fernandez O. prevalence of stroke and Associated Risk factors in Older Adults in Havana city and Matanzas Provinus, Cuba (10/66 Population-Based study) MEDICC Review 2010;12:20-24.
4. Sethi PK. Stroke - Incidence in India and Management of Ischaemic stroke. Neuroscience 2002;6(3): 139-43.
5. Barr WG. Uric acid. In: Walker HK, Hall WD, Hurst JW, editors. Clinical Methods. The History, Physical, and Laboratory Examination. 3rd ed. Boston: Butter worth; 1990. p. 760-763.
6. Heo SH, Lee SH. High levels of serum Uric acid are associated with silent brain infarctivn. Journal of the Neurol Scien 2010;297:6-15.
7. Hariklia VD, Apostolos H, Haralambosk The Role of Uric Acid in Stroke. The Issue Remains Unresolved. The Neurologist 2008;14:238-42.
8. Amaro S, Urrea X, Gomez-Choco M. Uric Acid Levels Are Relevant in Patients With Stroke Treated with Thrombolysis. Stroke 2011;42:28-32.
9. Ogbera A, Azenabor A. Hyperuricemia and the metabolic syndrome in type 2 DM. DiabetolMetabSyndr 2010;2:24-30.
10. Weir CJ, Muir SW, Walters MR, Lees KR. Serum Urate as an independent predictor of poor outcome and future vascular events after acute ischemic stroke. Stroke 2003;34:19516
11. Chamorro A, Obach V, Cerrera A, Revilla M, Deulofeu R, Aponte JH. Prognostic significance of uric acid serum concentration in patients with acute ischemic stroke. Stroke 2002;33:1048-52.