International Journal of Health Sciences and Research
[Indexed, Peer-Reviewed Journal]
Volume 16; Issue: 3; March 2026
Website: www.ijhsr.org
Original Research Article *ISSN: 2249-9571*

Distribution of Elevated Serum Uric Acid Levels among Patients with Acute Ischemic Stroke - A Cross-Sectional Study from Northeast India

Eladmi Siangshai[1], Sarukumar Debbarma[2], Subhrajit Das[3], Subhranil Bhattacharjee[4]

[1]Senior Resident, Department of General Medicine, Agartala Government Medical College and Govind Ballabh Pant Hospital, Agartala, Tripura, India
[2]Associate Professor, Department of General Medicine, Agartala Government Medical College and Govind Ballabh Pant Hospital, Agartala, Tripura, India
[3]Senior Resident, Department of General Medicine, Agartala Government Medical College and Govind Ballabh Pant Hospital, Agartala, Tripura, India
[4]Post Graduate Teacher, Department of General Medicine, Agartala Government Medical College and Govind Ballabh Pant Hospital, Agartala, Tripura, India

Corresponding Author: Dr. Eladmi Siangshai

DOI: https://doi.org/10.52403/ijhsr.20260324

ABSTRACT

Background: Acute ischemic stroke (AIS) remains a major cause of mortality and disability worldwide. Serum uric acid (SUA), a biomarker with dual antioxidant and pro-oxidant properties, has been increasingly investigated for its prognostic relevance in AIS. However, data from Northeast India are limited.
Objective: To evaluate the distribution of demographic, clinical, and biochemical characteristics of AIS patients, with particular emphasis on serum uric acid levels.
Methods: This cross-sectional study enrolled 100 patients with AIS who met the inclusion criteria. Demographic details, comorbidities, and socioeconomic status were documented. Stroke severity was examined using the National Institutes of Health Stroke Scale (NIHSS). SUA levels were measured using uricase-based enzymatic assays.
Results: The majority of patients were aged 51–60 years (41%). The cohort showed a male predominance (76%). Hypertension (43%) and diabetes (25%) were the most common comorbidities. The mean SUA concentration was 5.1 ± 1.4 mg/dL, within the physiological range. Nearly half of the patients presented with mild (45%) or moderate (46%) stroke severity, with a mean NIHSS score of 7.4 ± 5.8. Mortality was observed in 8% of cases, consistent with contemporary hospital-based cohorts.
Conclusion: AIS patients in Northeast India demonstrated SUA levels within normal limits, predominantly mild-to-moderate stroke severity, and relatively low mortality. The results suggest that moderate levels of SUA might help protect the brain, but very high or low levels could be harmful. More large, multicentre studies are needed to better understand how SUA can be used as a marker and treatment in AIS.

Keywords: Acute ischemic stroke, Serum uric acid, Neuroprotection, Prognostic biomarker, Stroke severity, Northeast India

INTRODUCTION

Stroke is still one of the main causes of mortality and long-term disability around the world. Acute ischemic stroke (AIS) makes up about 90% of all stroke cases and occurs when a blood vessel is blocked, leading to reduced cerebral blood flow and damage to nerve cells. Despite advancements in reperfusion therapies, the number of AIS cases is still going up globally, especially in low- and middle-income countries where conditions like hypertension, diabetes, and metabolic syndrome are common.[1] Identifying biomarkers that can predict prognosis or guide therapeutic strategies is therefore of immense clinical importance.

Uric acid physiology

Serum uric acid (SUA), the final product of purine metabolism, has emerged as a biomarker of interest in cerebrovascular disease. SUA is known to have dual biological roles: on the one hand, it acts as a potent antioxidant, scavenging free radicals and mitigating oxidative stress; on the other hand, hyperuricemia has been implicated in endothelial dysfunction, systemic inflammation, and vascular disease.[2] This paradoxical nature has led to conflicting evidence regarding its role in AIS. Several longitudinal investigations have demonstrated that patients with AIS who present with higher baseline serum uric acid levels tend to have a reduced risk of subsequent functional dependence.[3]

Pathophysiological relevance in AIS

During ischemic injury, oxidative stress plays a central role in neuronal damage. SUA, being a major antioxidant in human plasma, may theoretically confer neuroprotection by reducing reactive oxygen species.[4] Conversely, elevated SUA levels are associated with metabolic syndrome, hypertension, and cardiovascular disease - all established risk factors for stroke.[5] Thus, SUA may simultaneously act as a protective factor during acute ischemia and a detrimental factor in long-term vascular health.

Clinical evidence and controversies

Recent studies have reported variable outcomes for SUA and AIS. Some cohort studies have found that higher SUA levels are linked to poor functional recovery and higher mortality,[6] but other studies point to possible neuroprotective effects, especially when SUA is given with thrombolytic therapy.[7] Recent meta-analyses indicate that the debate remains unresolved, since dose–response relationships may demonstrate either neuroprotective or adverse outcomes depending on baseline risk and specific population characteristics.[8,2]

Epidemiological observations

The prevalence of hyperuricemia in AIS patients varies across populations, with estimates ranging from 20% to 40%.[9] Differences in diet, genetics, and other health conditions may account for this. South Asian groups have higher rates of metabolic syndrome and hyperuricemia, so studying SUA in AIS is especially important for them.[10,11]

Research gap and study rationale

Despite extensive research, the precise role of SUA in AIS remains unresolved. Although SUA has been widely studied as a prognostic marker, few studies have systematically quantified the proportion of AIS patients with elevated SUA at admission. Quantifying prevalence helps elucidate SUA's biomarker potential and support its integration into clinical decision-making. The lack of available evidence from Northeast India prompted us to design and conduct this study. The present study aimed to evaluate the distribution of demographic, clinical, and biochemical characteristics among patients with AIS, with particular emphasis on serum uric acid levels.

MATERIALS AND METHODS

Study type and design:

This investigation was structured as an observational, descriptive study employing a cross-sectional design.

Study setting:
The research was carried out in the Department of General Medicine at Agartala Government Medical College and GB Pant Hospital (AGMC & GBPH).

Study period:
Data collection and analysis spanned 18 months, commencing from the date of protocol approval.

Study population and inclusion criteria:
Participants included all patients presenting with AIS to the institution during the study period.

Exclusion criteria:
Patients were excluded from the study if they met any of the following conditions:

1. Failure to provide written informed consent.
2. History of prior transient ischemic attack (TIA) or cerebrovascular accident (CVA).
3. Current use of medications such as loop or thiazide diuretics, aspirin, cyclosporine, or antitubercular agents.
4. Presence of malignancy.
5. Known diagnosis of gout, clinical evidence of gout, or chronic renal failure.
6. Diagnosis of hemorrhagic stroke.

Sample size and sampling technique:
The study subjects were enrolled from the General Medicine ward of AGMC & GBPH during the study period.

To calculate the sample size, the Cochran's formula for cross-sectional studies was used:

$$N = \frac{Z_\alpha^2 \times P \times (1 - P)}{L^2}$$

Where, N = Sample size
Z_α^2: Standard normal deviate at 5% level of significance is= 1.96
P = Prevalence of AIS patients with elevated Serum Uric acid=60%
L = Margin of error at 10%
Therefore, the calculated sample size was:
Sample size (N) =1.96x1.96x0.60[1-0.60]/ $(0.10)^2$ = 92.1984 rounded to 100.

Study tools:
The following tools were employed in the study:

1. **Case record proforma** – Used to document demographic details, medical history, clinical examination findings, and laboratory results.
2. **Serum uric acid estimation** – Measurement of SUA levels in enrolled patients.
3. **Neuroimaging** – Non-contrast computed tomography (NCCT) of the brain and magnetic resonance imaging (MRI) of the brain.
4. **National Institutes of Health Stroke Scale (NIHSS)** – A standardized 11-item scale assessing stroke severity, including level of consciousness, eye movements, visual fields, facial palsy, motor function (arm and leg), limb ataxia, sensory deficits, language, speech, and neglect. Stroke severity was categorised as:[12]

- 0: No stroke symptoms
- 1–4: Minor stroke
- 5–15: Moderate stroke
- 16–20: Moderate to severe stroke
- 21–42: Severe stroke

Study procedure:
Patients admitted with AIS to the Department of General Medicine at Agartala Government Medical College and GB Pant Hospital during the study period (as per inclusion criteria) underwent routine haematological investigations, including serum uric acid estimation (defined as >5.7 mg/dl in women and >7 mg/dl in men). After obtaining informed consent, a detailed history covering demographic details, occupation, education, socioeconomic status, medication and addiction habits, past illnesses, and family history was recorded. General and systemic examinations of the cardiovascular, central nervous, gastrointestinal, and genitourinary systems were performed.

Blood samples were collected in the Department of Biochemistry under aseptic precautions. Five millilitres of fasting venous blood were drawn and placed in pre-labelled

containers containing heparin, EDTA, or clot activator. Samples were allowed to clot at room temperature, and plasma/serum was separated by centrifugation at 3,000 rpm for 5–10 minutes. Proper labelling and coding were ensured; samples were analysed immediately whenever possible or stored at 2–8°C for up to 72 hours if analysis was delayed.

Serum uric acid was measured using standard biochemical techniques. Commonly employed assays included the photometric method (phosphotungstic acid reduction), high-performance liquid chromatography (HPLC), and uricase-based enzymatic methods. Among these, uricase assays were most frequently used, with variations classified as direct or indirect, and further subdivided into kinetic and equilibrium approaches.

Ethical considerations:

The Institutional Ethics Committee (IEC) of AGMC approved the study protocol before the research started. Participants received information in their native language about the study's goals, their rights, and the voluntary nature of participation. Their confidentiality was protected, and they could leave the study at any time. After collecting the data, all records were anonymised, cleaned, and stored securely in a password-protected spreadsheet.

Data analysis:

We checked the data for accuracy and completeness and entered them into Microsoft Excel. Categorical variables were presented as percentages, and continuous variables were reported as mean ± standard deviation. The findings were presented using bar charts and pie diagrams.

RESULTS

The study consisted of 100 participants, and their ages were grouped into three categories. A relatively small proportion of the sample (15%) was aged 50 years or below. The largest segment was found in the 51–60-year range, accounting for 41% of participants. The remaining 44% were aged 61 years or older, making this the most-represented group (Figure 1).

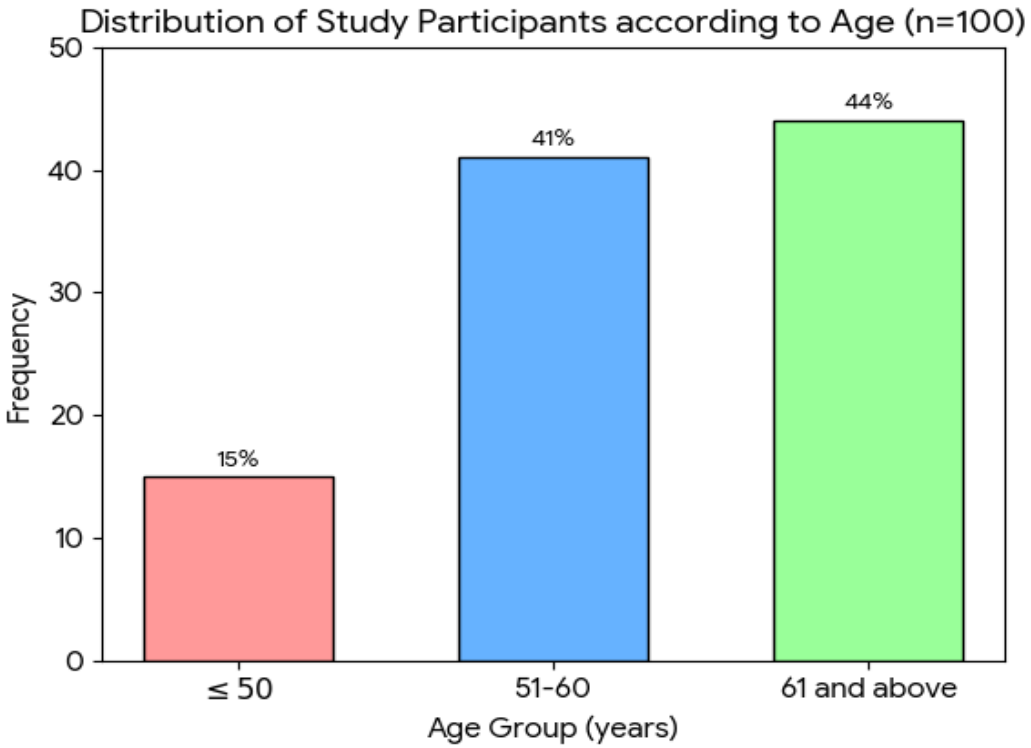

Figure 1: Distribution of Study Participants by Age Group

Among the participants, 76% were male. Females accounted for 24% of the sample. This indicates that the study group was predominantly male, with roughly three out of every four participants being male (Figure 2).

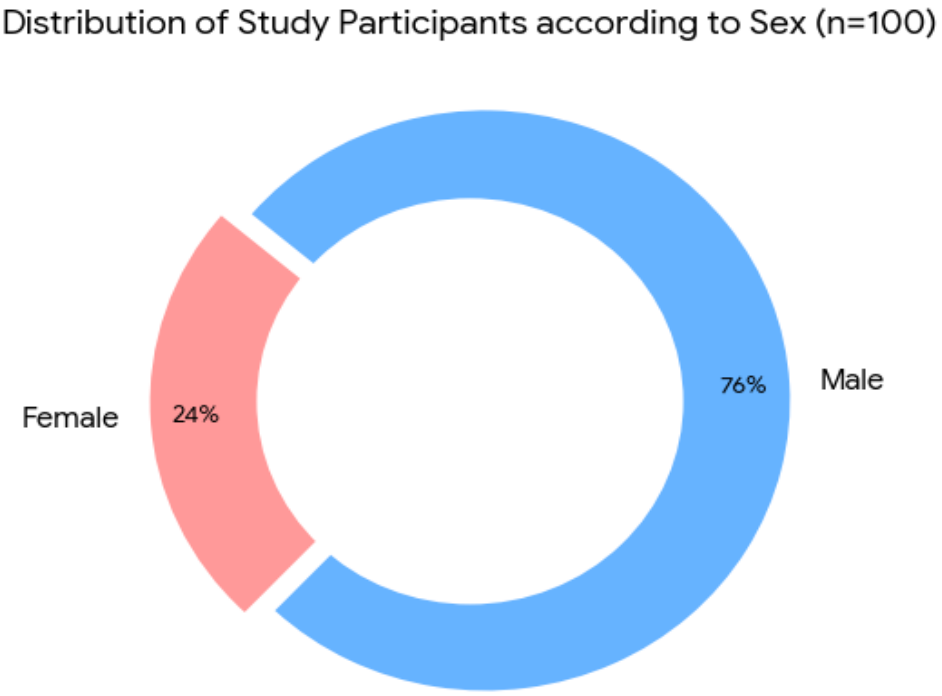

Figure 2: Distribution of Study Participants by Sex

Among the participants, 64% reported living in urban areas, while 36% were from rural areas. This indicates that nearly two-thirds of the study population resided in urban locations, making them the dominant group. In contrast, just over one-third of participants came from rural backgrounds (Figure 3).

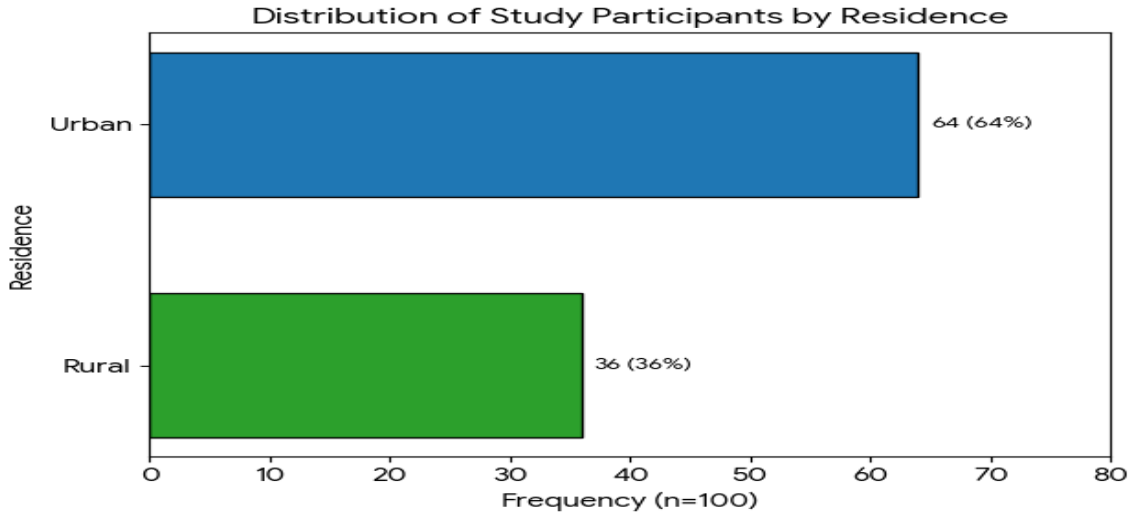

Figure 3: Distribution of Study Participants by Residence

Assessment of socioeconomic status using the B.G. Prasad classification showed that Class II (40%) and Class III (30%) comprised the largest segments of the cohort. A smaller proportion were categorised as

Class IV (18%), and only 12% were in Class I (Figure 4).

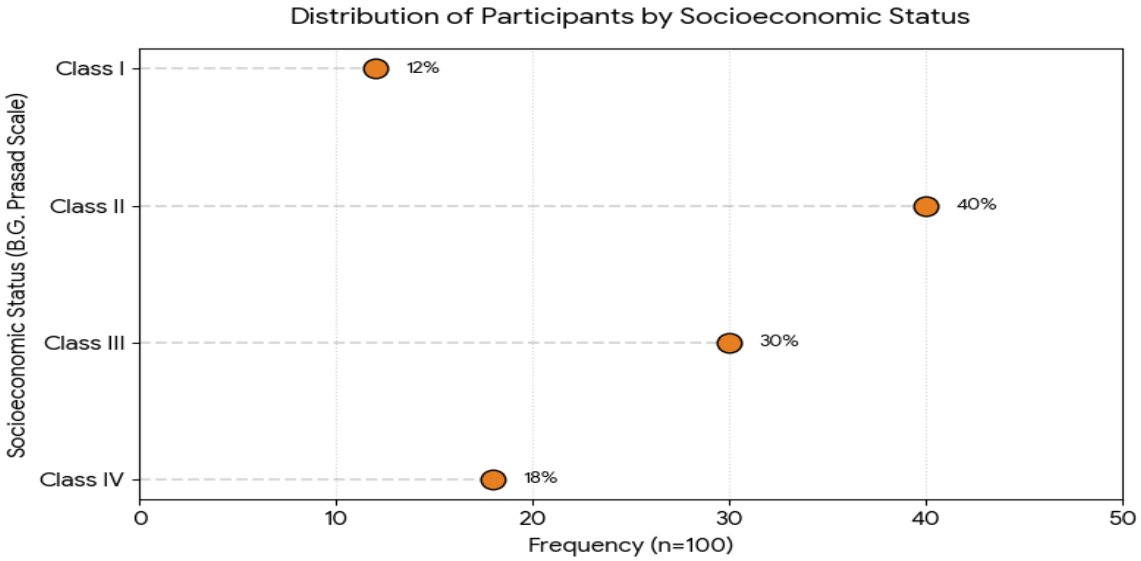

Figure 4: Distribution of Study Participants by Socioeconomic Status

Hypertension was the most common comorbidity, affecting 43% of the study population. Diabetes mellitus was reported in 25% of individuals. This distribution indicates that nearly half of the participants were living with hypertension, while one-fourth had diabetes (Figure 5).

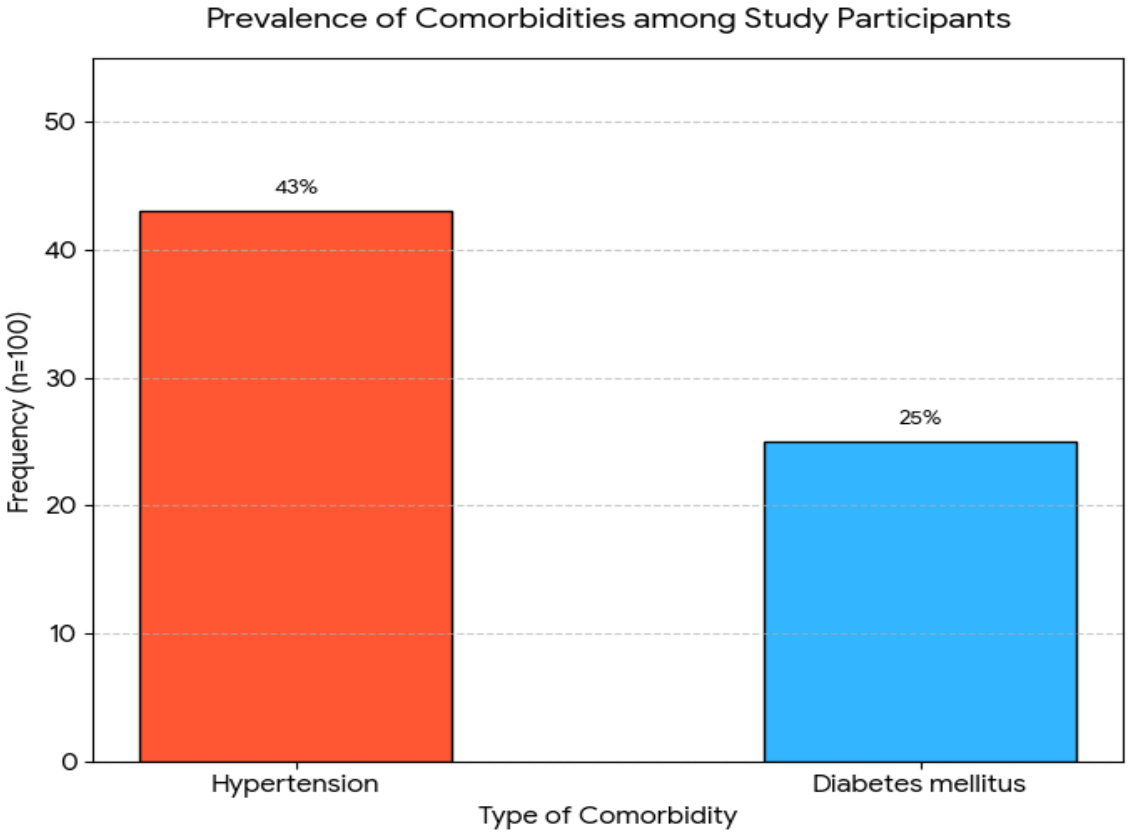

Figure 5: Distribution of Study Participants by Comorbidity

The severity of AIS among participants, assessed using the NIHSS score, showed that nearly half of the study population had mild strokes (45%), while a similar proportion experienced moderate severity (46%). A smaller fraction presented with moderate-to severe strokes (7%), and only 2% had severe strokes. The mean NIHSS score was 7.4 ± 5.8, indicating that, overall, the study group tended toward the mild-to-moderate range of stroke severity (Figure 6).

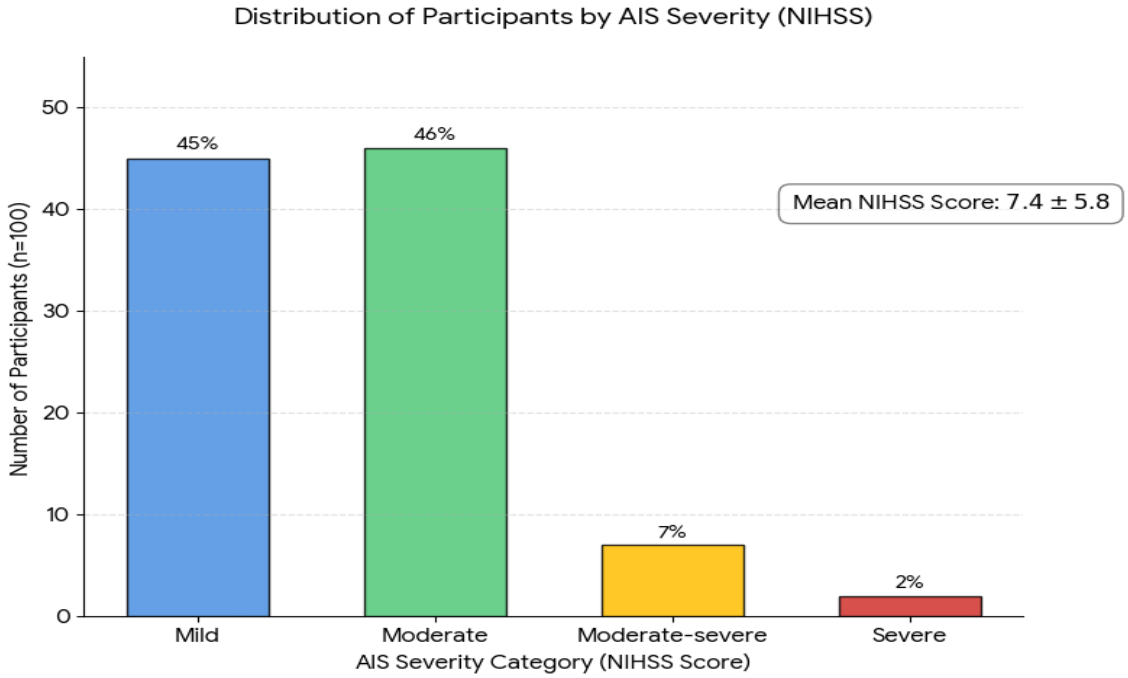

Figure 6: Distribution of Study Participants by AIS Severity

The mean serum uric acid level among the study participants was 5.1 mg/dl, with a standard deviation of 1.4. This indicates that, on average, uric acid values in the sample were within the normal physiological range, though individual levels varied moderately around the mean (Figure 7).

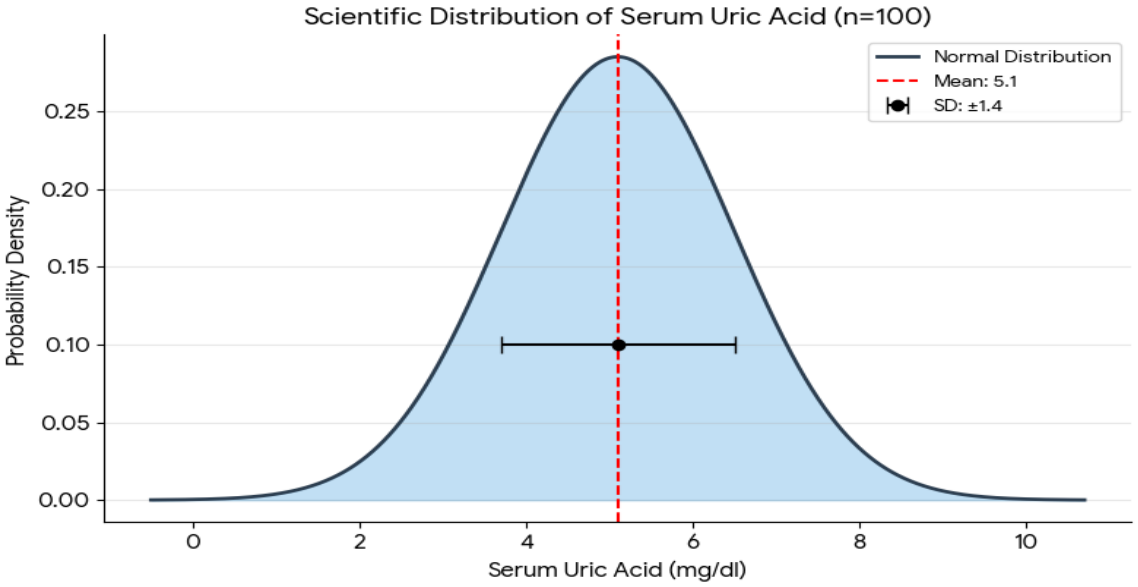

Figure 7: Serum Uric Acid Distribution in Study Participants

Among the participants, 8% experienced mortality during the study period, while the remaining 92% survived. This indicates that most individuals did not succumb, with deaths occurring in less than one-tenth of the sample (Figure 8).

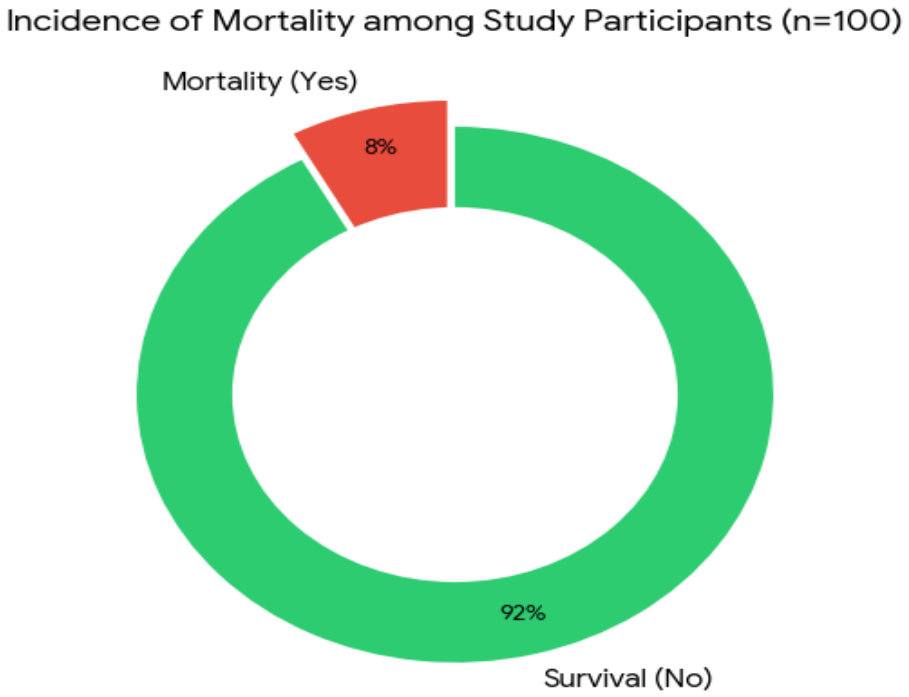

Figure 8: Incidence of Mortality among Study Participants

DISCUSSION

The present study explored the distribution of demographic, clinical, and biochemical characteristics of patients with AIS, with a particular focus on serum uric acid (SUA) levels. The mean SUA concentration in our cohort was 5.1 ± 1.4 mg/dl, which lies within the physiological range. This finding is similar to recent observational studies reporting average SUA values ranging from 4.9 mg/dL (Maharashtra) to 6.2 mg/dL (Punjab) in AIS populations in India.[13,14] Importantly, our study adds evidence from Northeast India, a region where data remain sparse.

Uric Acid and Stroke Severity

Nearly half of our participants presented with mild to moderate stroke severity (mean NIHSS 7.4 ± 5.8). A Turkish cohort study demonstrated that higher SUA levels correlated with greater NIHSS scores, suggesting a potential detrimental effect of hyperuricemia on neurological outcomes.[15] Conversely, a systematic review emphasised SUA's antioxidant properties, proposing that moderate elevations may attenuate oxidative stress during ischemia.[1] Our findings, with SUA levels largely within normal limits, may help explain the predominance of mild-to-moderate severity in this cohort, supporting the hypothesis that balanced SUA concentrations could be neuroprotective.

Mortality and Prognosis

Mortality in our study was relatively low (8%), aligning with contemporary reports of short-term AIS mortality ranging between 6.5–12% in similar hospital-based cohorts.[16,17] Earlier reviews have highlighted hyperuricemia as a risk factor for increased stroke mortality, mediated partly by systemic inflammation and endothelial dysfunction.[18] However, other studies suggest that SUA may improve survival when administered adjunctively with thrombolytic therapy, due to its free-radical-scavenging capacity.[19] The relatively normal SUA levels in our cohort

may have contributed to the favourable survival outcomes observed.

Clinical Controversies

The dual role of SUA remains a central point of contention. On one hand, SUA acts as a major plasma antioxidant, potentially reducing neuronal injury during ischemia. On the other hand, chronic hyperuricemia is linked to hypertension, diabetes, and metabolic syndrome—all prevalent comorbidities in our study population (43% hypertensive, 25% diabetic). This paradox is reflected in recent dose–response meta–analyses, which show that both very low and very high SUA levels are associated with poor outcomes, while intermediate levels may be protective.[20] Our findings, with SUA values clustering around the mid-range, support this nuanced interpretation.

Regional and Epidemiological Relevance

South Asian populations, including those in Northeast India, have higher rates of metabolic syndrome and hyperuricemia compared to Western cohorts. This makes SUA a particularly relevant biomarker in this setting. Our study contributes region-specific data, underscoring the need for larger multicentre studies to clarify whether SUA should be incorporated into routine prognostic assessment in AIS patients from resource-limited regions.

Strengths and Limitations

A main strength is the systematic documentation of demographic, socioeconomic, and comorbidity profiles alongside SUA levels, allowing contextual interpretation. However, the limitations of our study include the cross-sectional design, which limits causal inference, and the relatively small sample size. Longer studies with repeated SUA measurements are warranted to verify and strengthen the present findings.

CONCLUSION

In summary, our study found that AIS patients in Northeast India had mean SUA levels within the normal range, with most cases presenting with mild-to-moderate strokes and relatively low mortality. These findings align with recent studies suggesting that intermediate SUA levels may confer neuroprotection, while extremes are detrimental. Given the ongoing debate, SUA should be considered a context-dependent biomarker, influenced by baseline vascular risk and treatment modalities. Future prospective studies are essential to determine whether SUA can be integrated into prognostic models or therapeutic strategies for AIS.

Declaration by Authors

Ethical Approval: Approved
Acknowledgement: None
Source of Funding: None
Conflict of Interest: The authors declare no conflict of interest.

REFERENCES

1. Roman-Filip I, Roman-Filip C, Morosanu V, Andone S, Bajko Z, Balasa R. Uric Acid in Cerebral Ischemia: A Systematic Review of Its Biomarker Value and Role in Neuroprotection. Int J Mol Sci. 2025;26(21):10268.
2. Zhong J, Cai H, Zhang Z, Wang J, Xiao L, Zhang P, et al. Serum uric acid and prognosis of ischemic stroke: Cohort study, meta-analysis and Mendelian randomization study. Eur Stroke J. 2024;9(1):235-243.
3. Zhang W, Cheng Z, Fu F, Zhan Z. Serum uric acid and prognosis in acute ischemic stroke: a dose-response meta-analysis of cohort studies. Front Aging Neurosci. 2023; 15:1223015.
4. Zhu B, Huang X, Zhang J, Wang X, Tian S, Zhan T, et al. A New Perspective on the Prediction and Treatment of Stroke: The Role of Uric Acid. Neurosci Bull. 2025;41(3):486-500.
5. Wang JJ, Yi JK, Zhou LR, Chen J, Zhang BQ, Huang HM, et al. A systematic review of the connection between serum uric acid levels and the risk of cardiovascular disease. Front Cardiovasc Med. 2025; 12:1577952.
6. Wang Z, Lin Y, Liu Y, Chen Y, Wang B, Li C, et al. Serum Uric Acid Levels and Outcomes After Acute Ischemic Stroke. Mol Neurobiol. 2016;53(3):1753-1759.

7. Chamorro A, Amaro S, Castellanos M, Segura T, Arenillas J, Martí-Fábregas J, et al. Safety and efficacy of uric acid in patients with acute stroke (URICO-ICTUS): a randomised, double-blind phase 2b/3 trial. Lancet Neurol. 2014 May;13(5):453-60. doi: 10.1016/S1474-4422(14)70054-7.
8. Zhang P, Wang R, Qu Y, Guo ZN, Zhen Q, Yang Y. Serum Uric Acid Levels and Outcome of Acute Ischemic Stroke: a Dose-Response Meta-analysis. Mol Neurobiol. 2024;61(3):1704-1713.
9. Haseeb M, Qadri M, Talpur MTH, Hussain MA, Jamil N, Malik F, et al. Prevalence of hyperuricemia in acute ischemic stroke patients: A cross-sectional study. Pak J Health Sci. 2023;4(10):26–30.
10. Pramod Kumar TA, Mohan V. Epidemic of cardio-metabolic disorders in south Asia: need for urgent solutions. Eur Heart J. 2025;46(29):2838-2840.
11. Mahadevan M, Bose M, Gawron KM, Blumberg R. Metabolic Syndrome and Chronic Disease Risk in South Asian Immigrants: A Review of Prevalence, Factors, and Interventions. Healthcare (Basel). 2023;11(5):720.
12. NIH Stroke Scale Training, Part 2. Basic Instruction. Department of Health and Human Services, National Institute of Neurological Disorders and Stroke. The National Institute of Neurological Disorders and Stroke (NINDS); Version 2.0.
13. Shah H, Patange A. The study of serum uric acid levels in ischemic stroke patients. Int J Contemp Med Res. 2019;6(1): A16-A20.
14. Kaur I, Khurana A, Sachdev JK, Mohan G. Evaluation of serum uric acid in acute ischaemic stroke. Int J Adv Med. 2017;4(1):60-65.
15. Kaya Tutar N, Küçükoğlu H, Köksal A. The effect of serum uric acid level on stroke severity. Med J Bakirkoy. 2025;21(4): 343-349.
16. Quinn T, Kitagawa K, Leung T, Molina C, Rabinstein A, Bentley R, et al. A systematic literature review on the burden of disease for patients with moderate to severe acute ischemic stroke. Medicine (Baltimore). 2025;104(3):e41249.
17. Bârsan IC, Iluţ S, Tohănean N, Pop R, Vesa ŞC, Perju-Dumbravă L. Resistin and In-Hospital Mortality in Patients with Acute Ischemic Stroke: A Prospective Study. Journal of Clinical Medicine. 2024;13(16):4889.
18. Padda J, Khalid K, Padda S, Boddeti NL, Malhi BS, Nepal R, et al. Hyperuricemia and Its Association with Ischemic Stroke. Cureus. 2021;13(9): e18172.
19. Liu X, Liu M, Chen M, Ge QM, Pan SM. Serum uric acid is neuroprotective in Chinese patients with acute ischemic stroke treated with intravenous recombinant tissue plasminogen activator. J Stroke Cerebrovasc Dis. 2015;24(5):1080-1086.
20. Crawley WT, Jungels CG, Stenmark KR, Fini MA. U-shaped association of uric acid to overall-cause mortality and its impact on clinical management of hyperuricemia. Redox Biol. 2022; 51:102271.

How to cite this article: Eladmi Siangshai, Sarukumar Debbarma, Subhrajit Das, Subhranil Bhattacharjee. Distribution of elevated serum uric acid levels among patients with acute ischemic stroke - a cross-sectional study from Northeast India. *Int J Health Sci Res.* 2026; 16(3):191-200. DOI: *10.52403/ijhsr.20260324*

******