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Serum Uric Acid Levels As A Prognostic Indicator in Acute Ischaemic Stroke

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Conflicts of Interest: Nil

Abstract

Introduction: Stroke remains a major public health concern worldwide, contributing significantly to morbidity and mortality. Among the different types of strokes, ischemic stroke accounts for the majority of cases, occurring due to the occlusion of cerebral arteries and subsequent ischemic injury. Stroke is the second leading cause of death globally, significantly affecting individuals, families and healthcare systems.

Objectives

Primary Objective: To study the serum uric acid levels in acute ischemic stroke and assess its prognostic significance.

Secondary Objective: To study the association between serum uric acid and various risk factors for stroke.

Material and Method:

Study Design: Prospective observational study

Study Period: 12 months from IEC approval (August

2023 to August 2024)

Study Setting: Department of General Medicine, Govt.

Medical College, Ernakulam

Sample Population: Patients admitted to General medicine wards of Government medical college, Ernakulam with diagnosis of acute ischemic stroke satisfying the inclusion criteria and exclusion criteria.

Sampling Method: Consecutive sampling

Study Tool: Semi Structured peer reviewed proforma.

Result: The study involved 120 participants and the findings revealed that the mean age of stroke patients was 63.25 years, with a male predominance (56.7%).

Discussion: Among the study population, hypertension (65%) and dyslipidemia (58.3%) were the most common comorbidities, followed by diabetes (48.3%) and smoking (40.8%).

Keywords: Antioxidant, Cyclosporine, Dyslipidemia, Ischemic Stroke, Serum Uric Acid.

Introduction

Stroke remains a major public health concern worldwide, contributing significantly to morbidity and mortality. Among the different types of strokes, ischemic stroke accounts for the majority of cases, occurring due to the occlusion of cerebral arteries and subsequent ischemic injury. Understanding biochemical and molecular factors influencing the severity and prognosis of ischemic stroke is critical for improving early intervention strategies and patient outcomes. One such potential biomarker that has gained attention in recent years is serum uric acid (SUA). Uric acid is the final product of purine metabolism, primarily excreted by the kidneys and, to a lesser extent, through the gastrointestinal tract ¹. Historically, it has been regarded as a waste product with potential pathological implications, particularly in conditions such as gout, metabolic syndrome, and cardiovascular diseases. However, recent studies suggest that uric acid also plays a significant role as an antioxidant, capable of scavenging reactive oxygen species (ROS) and neutralizing oxidative stress, which is a key contributor to ischemic brain injury ².

Uric acid is a major contributor to plasma antioxidant capacity, with a concentration almost ten times higher than other well-known antioxidants such as Vitamin C and Vitamin E. This suggests that uric acid could play a significant role in mitigating oxidative damage in the brain during ischemic stroke ³.

During an ischemic stroke, the sudden interruption of cerebral blood flow leads to a cascade of metabolic disturbances, including an excessive generation of ROS, mitochondrial dysfunction, and inflammatory responses.

Several experimental models and clinical studies have demonstrated that uric acid can reduce neuronal injury by counteracting oxidative stress. Additionally, uric acid may play a role in maintaining endothelial function and microvascular integrity, which are crucial in limiting post-ischemic damage. Nonetheless, the clinical implications of these findings remain debated, as increased uric acid levels have also been linked to vascular dysfunction and atherosclerosis 4,5 Some hypotheses suggest that increased uric acid levels in acute ischemic stroke may reflect an adaptive response to counteract oxidative stress and inflammation. Conversely, hyperuricemia is often associated with hypertension, diabetes, obesity, and metabolic syndrome, which are well-established risk factors for stroke 6.

Objectives

Primary Objective

1. To study the serum uric acid levels in acute ischemic stroke and assess its prognostic significance.

Secondary Objective

2. To study the association between serum uric acid and various risk factors for stroke (such as Diabetes, Systemic hypertension, Dyslipidemia, Smoking)

Material and Method

Study Design

This study was designed as a prospective observational study, where patients diagnosed with acute ischemic stroke were systematically evaluated for serum uric acid levels and its potential prognostic implications. The study followed a hospital-based cohort approach, focusing on patients admitted to the General Medicine Department of Government Medical College, Ernakulam. The prospective nature of the study ensured

real-time data collection, allowing for a comprehensive analysis of clinical parameters.

Study Duration

The study was conducted over a 12-month period, August 2023 to August 2024, commencing upon approval from the Institutional Ethics Committee (IEC) of Government Medical College, Ernakulam.

Study Setting

This study was carried out in the Department of General Medicine, Government Medical College, Ernakulam, a tertiary care center catering to a wide patient population across Kerala.

Study Population

The study included patients admitted to the General Medicine wards of Government Medical College, Ernakulam, with a confirmed diagnosis of acute ischemic stroke, meeting the predefined inclusion and exclusion criteria.

Inclusion Criteria

- Patients diagnosed with first-ever acute ischemic stroke, confirmed via CT scan within 24 hours of onset.
- 2. Patients aged 18 years or older

Exclusion Criteria

- Patients with a known history of gout or clinical evidence of gout.
- 2. Patients diagnosed with chronic renal failure.
- 3. Patients whose CT scans reveal hemorrhage or other space-occupying lesions (excluding infarcts).
- Patients with hematological disorders, including leukemia and myeloproliferative disorders.
- 5. Patients on medications that may cause hyperuricemia, including:
- Loop diuretics

- Anticancer drugs (Cisplatin, Cyclosporine, Cyclophosphamide)
- Antitubercular drugs (ATT) (Pyrazinamide, Ethambutol)
- Pentamidine, Theophylline, Ketoconazole, Levodopa'

Sampling Method

A consecutive sampling method was used to enroll all eligible patients within the study duration, ensuring a comprehensive representation of stroke cases. This approach allowed for the inclusion of all patients meeting the predefined criteria, reducing selection bias and improving the generalizability of the findings.

Study Tool

Data collection was conducted using a semi-structured, peer-reviewed proforma, specifically designed to capture demographic data, clinical findings, laboratory parameters, and stroke outcomes. The proforma was reviewed and validated by subject matter experts to ensure its reliability and applicability in the study setting.

Sample Size Calculation

The sample size was determined based on a study published in the International Journal of Medical Research and Review (2021), which reported that the prevalence of hyperuricemia in acute ischemic stroke was 77%. This prevalence rate was used to calculate an adequate sample size, ensuring sufficient statistical power to analyze the association between serum uric acid levels and stroke prognosis.

Using the standard formula for sample size calculation:

$$N = \frac{4PQ}{D^2}$$

where

- P = 77 (prevalence of hyperuricemia)
- Q = 100 P = 23
- D = 7.7 (beta error assumed at 10%)

$$N = \frac{4 \times 77 \times 23}{7.7 \times 7.7}$$

The required sample size was 119, rounded off to 120 patients for the study.

Data Management and Statistical Analysis

All collected data were systematically recorded in Microsoft Excel and analyzed using Epi Info software. Various statistical methods were employed to evaluate the relationship between serum uric acid levels and stroke prognosis. Quantitative data were analyzed using the independent sample t-test, while categorical data were expressed as rates, ratios, and percentages, and compared using the Chi-square test. Continuous data were expressed as mean ± standard deviation (SD) and analyzed using the t-test. Additionally, correlation analysis was conducted to examine the association between serum uric acid levels and the MRS score, further assessing its relationship with stroke severity and progression.

Result

Table 1: Age and Gender Distribution of Stroke Patients

Age Group (Years)	Male (n, %)	Female (n, %)	Total (n, %)
45-55	18 (26.5%)	12 (23.1%)	30 (25%)
56-65	25 (36.8%)	17 (32.7%)	42 (35%)
66-75	20 (29.4%)	18 (34.6%)	38 (31.7%)
>75	5 (7.3%)	5 (9.6%)	10 (8.3%)
Total	68 (56.7%)	52 (43.3%)	120 (100%)

This section presents the findings of the study evaluating serum uric acid (SUA) levels as a prognostic indicator in acute ischemic stroke. The data is systematically analyzed according to demographic characteristics, stroke severity, SUA levels, association with risk factors, radiological findings, and prognostic value. The statistical analyses performed include descriptive statistics, correlation tests, and regression analysis to establish the relationship between SUA levels and stroke outcomes.

Age and Gender Distribution

A total of 120 patients diagnosed with acute ischemic stroke were included in the study, all meeting the established inclusion and exclusion criteria. The mean age of the study participants was 63.25 ± 9.53 years, ranging from 45 to 80 years. The distribution of age groups indicated that stroke was more prevalent in the age category of 56-65 years, which accounted for 35% of the total sample. This was followed by the 66-75 years age group, comprising 31.7% of patients. The smallest group consisted of patients older than 75 years (8.3%). The gender distribution of the study population revealed that stroke was more prevalent in males than in females. Out of the 120 patients, 68 (56.7%) were male, while 52 (43.3%) were female.

Age and gender distribution 30 25 25 20 18 18 20 17 12 10 5 5 45-55 56-65 66-75 >75 Age group

■ Male (n, %) ■ Female (n, %)

Figure 1: Bar Chart for Age and Gender Distribution of Stroke Patients

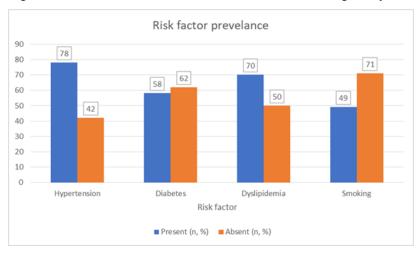
Distribution of Risk Factors

The study further analyzed the prevalence of key risk factors associated with ischemic stroke, including hypertension, diabetes, dyslipidemia, and smoking. Among the participants, hypertension emerged as the most prevalent comorbidity, affecting 65% (n = 78) of patients. This aligns with established findings that hypertension is a significant risk factor for cerebrovascular events due to its role in arterial damage and atherosclerosis. Diabetes mellitus was present in 48.3% (n = 58) of patients, confirming its well-documented contribution to cerebrovascular disease. Dyslipidemia, another significant risk factor, was observed in 58.3% (n = 70) of the study population. Smoking, a modifiable risk factor, was reported in 40.8% (n = 49) of patients, emphasizing the need for targeted interventions to reduce tobacco use as a preventive measure.

Table 2: Prevalence of Risk Factors Among Study Participants

Risk Factor	Present (n, %)	Absent (n, %)
Hypertension	78 (65%)	42 (35%)
Diabetes	58 (48.3%)	62 (51.7%)
Dyslipidemia	70 (58.3%)	50 (41.7%)
Smoking	49 (40.8%)	71 (59.2%)

Figure 2: Bar chart for Prevalence of Risk Factors Among Study Participants



Baseline Blood Pressure and Fasting Blood Sugar Levels

Baseline measurements for blood pressure (BP) and fasting blood sugar (FBS) were obtained for all study participants. The mean systolic blood pressure (SBP) was 148.5 ± 19.2 mmHg, while the mean diastolic blood pressure (DBP) was 87.6 ± 14.8 mmHg. These findings indicate that a large proportion of patients had elevated BP, which is a significant contributor to ischemic stroke due to its effect on cerebrovascular integrity.

The mean fasting blood sugar (FBS) level was 135.92 ± 43.51 mg/dL, with a minimum of 90 mg/dL and a maximum of 280 mg/dL. Elevated blood sugar levels at the time of stroke presentation have been linked to worse outcomes, suggesting that glucose control is an important consideration in stroke management.

Table 3: Mean BP and Fasting Blood Sugar Levels in the Study Population

Parameter	Mean ± SD	Minimum	Maximum
Systolic BP (mmHg)	148.5 ± 19.2	110	190
Diastolic BP (mmHg)	87.6 ± 14.8	70	110
Fasting Blood Sugar (mg/dL)	135.9 ± 43.5	90	280

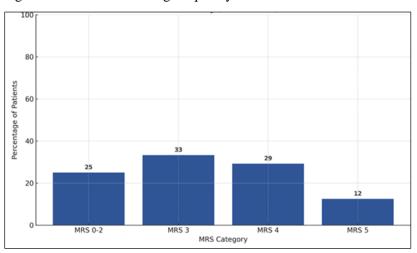
Distribution of MRS Scores on Day 1

Stroke severity was assessed using the Modified Rankin Scale (MRS) at admission, which categorizes stroke severity based on functional impairment. Among the study participants, 25% had mild stroke (MRS 0-2), while 33.3% had an initial MRS score of 3(mod stroke), 29.2% had MRS score 4(moderately severe stroke) and 12.5% had MRS score of 5(severe stroke). The distribution of MRS scores at admission is presented in Table 4.

Table 4: Stroke Severity (MRS Scores) at Admission

MRS Score	Severity Level	Frequency (n, %)
0-2	Mild	30 (25%)
3	Moderate	40 (33.3%)
4	Moderately Severe	35 (29.2%)
5	Severe	15 (12.5%)

Figure 3: Bar Chart showing frequency of initial mRS score at the time of admission



Correlation between Uric Acid and Stroke Outcome

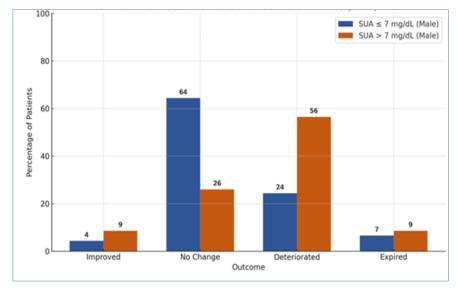
Patients were grouped based on their functional outcome at Day 7. A gender-specific analysis was conducted, defining hyperuricemia as serum uric acid (SUA) >7.0 mg/dL in males and >6.0 mg/dL in females. The analysis revealed a clear association between elevated SUA levels and unfavourable clinical outcomes. Patients with higher SUA levels were significantly more likely to experience neurological deterioration or death.

In males with SUA>7mg/dl, those that deteriorated were 56.5% compared with the males with SUA<7mg/dl, in whom 24.4% deteriorated. Similarly in females with SUA>6mg/dl, 55.6% deteriorated compared to 36% deterioration in those with SUA<6mg/dl. These findings reinforce the potential of SUA as an independent prognostic biomarker in acute ischemic stroke, with its predictive value consistent across both sexes despite the reduced overall mortality

Table 5: Association between Serum Uric Acid and Stroke Outcomes in Male Patients

SUA Level (mg/dL)	Improved (n, %)	No Change (n, %)	Deteriorated (n, %)	Expired (n, %)
≤7 mg/dL	2(4.4%)	29 (64.4%)	11 (24.4%)	3(6.6%)
>7 mg/dL	2 (8.6)	6 (26%)	13 (56.5%)	2 (8.6%)

Figure 4: Bar chart showing Association between Serum Uric Acid and Stroke Outcomes in Male Patients



Mean Serum Uric Acid Levels in Stroke Patients

Serum uric acid (SUA) levels were measured in all 120 stroke patients within 24 hours of admission. The mean SUA level was 6.61 ± 0.92 mg/dL, with a minimum of 4.6 mg/dL and a maximum of 8.9 mg/dL. These findings suggest that a substantial proportion of patients had SUA levels above the standard reference ranges (>7 mg/dL in men, >6 mg/dL in women), which has been linked to poorer stroke outcomes in prior research.

Table 6: Mean Serum Uric Acid Levels in Study Participants

Parameter	Mean ± SD	Minimum	Maximum
SUA (mg/dL)	6.61 ± 0.92	4.6	8.9

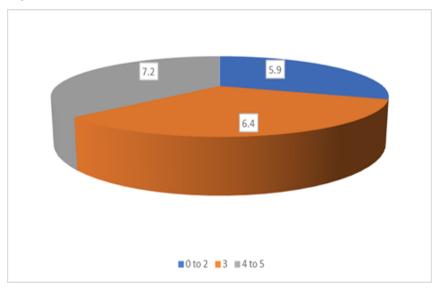
Comparison of Uric Acid Levels Based on Stroke Severity (MRS Score at Admission)

To evaluate the potential role of SUA in determining stroke severity, SUA levels were stratified according to the Modified Rankin Scale (MRS) score at admission. Patients with higher MRS scores (\geq 4) had significantly higher SUA levels (p < 0.05), indicating a possible association between hyperuricemia and more severe strokes.

Table 7: Serum Uric Acid Levels in Patients with Different MRS Scores

MRS Score	Mean SUA (mg/dL) \pm SD
0-2	5.9 ± 0.7
3	6.4 ± 0.8
4-5	7.2 ± 1.1

Figure 5: Pie chart for Serum Uric Acid Levels in Patients with Different MRS Scores



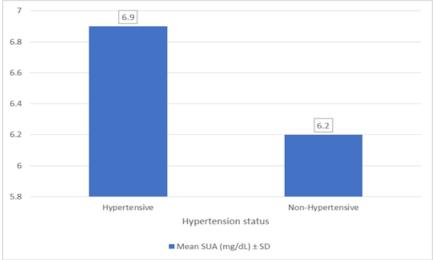
Uric Acid Levels in Hypertensive vs. Non-Hypertensive Patients

Among hypertensive patients (n = 78), the mean SUA level was significantly higher (6.9 \pm 0.8 mg/dL) compared to non-hypertensive patients (6.2 \pm 0.7 mg/dL, p < 0.05). This suggests that hypertension may contribute to increased SUA levels, possibly through renal dysfunction or endothelial damage.

Table 8: Comparison of Serum Uric Acid Levels in Hypertensive and Non-Hypertensive Patients

Hypertension Status	Mean SUA (mg/dL) \pm SD	p-value
Hypertensive	6.9 ± 0.8	<0.05
Non-Hypertensive	6.2 ± 0.7	

Figure 6: Bar chart for Comparison of Serum Uric Acid Levels in Hypertensive and Non-Hypertensive Patients



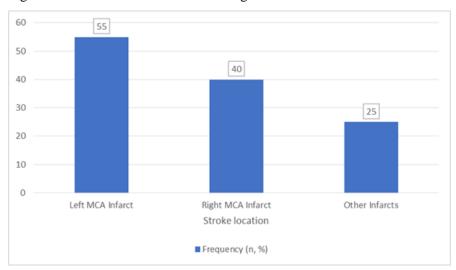
Stroke Distribution Based on CT Brain Findings

The majority of strokes in this study were classified as middle cerebral artery (MCA) infarcts, which accounted for 79.1% of all cases.

Table 9: CT Brain Findings in Stroke Patients

Stroke Location	Frequency (n, %)
Left MCA Infarct	55 (45.8%)
Right MCA Infarct	40 (33.3%)
Other Infarcts	25 (20.9%)

Figure 7: Bar chart for CT Brain Findings in Stroke Patients



Regression Analysis of Uric Acid and Stroke Outcome

A multivariate logistic regression analysis was conducted to determine whether serum uric acid (SUA) levels were an independent predictor of poor stroke outcomes (MRS \geq 4 at Day 7). The model included known risk factors such as age, hypertension, diabetes, dyslipidemia, and smoking status. The analysis revealed that SUA > 6.5 mg/dL was significantly

associated with poor stroke outcomes (p < 0.01), with an adjusted odds ratio (OR) of 2.8. This finding suggests that hyperuricemia independently increases the likelihood of worse functional status following an acute ischemic stroke.

Table 10: Regression Analysis of Uric Acid as a Predictor of Stroke Outcome

Variable	Adjusted OR	95% CI	p-value
SUA > 6.5 mg/dL	2.8	1.5 - 5.3	<0.01
Hypertension	1.9	1.1 - 3.2	0.02
Diabetes	1.4	0.9 - 2.3	0.08
Dyslipidemia	1.6	1.0 - 2.8	0.05
Smoking	1.2	0.8 - 1.9	0.15

The logistic regression model confirmed that SUA levels above 6.5 mg/dL are an independent predictor of functional deterioration, even after adjusting for other risk factors. Additionally, hypertension and dyslipidemia were also found to be significant contributors to worse outcomes, emphasizing the need for better risk factor management in stroke patients.

Discussion

The present study aimed to evaluate the prognostic significance of serum uric acid (SUA) levels in patients with acute ischemic stroke (AIS). The findings indicate that elevated SUA levels are significantly associated with stroke severity, functional outcomes, and risk factor burden, reinforcing the hypothesis that uric acid plays a crucial role in cerebrovascular pathophysiology. This discussion contextualizes the results with existing literature, explores possible mechanisms linking SUA with stroke severity, and considers clinical implications for stroke management.

The study revealed a mean SUA level of 6.61 mg/dL in stroke patients, patients who experienced deterioration had significantly higher SUA levels (7.2 \pm 1.1 mg/dL) compared to those who improved (5.9 \pm 0.7 mg/dL, p < 0.05), suggesting that hyperuricemia may play a role in stroke progression with higher levels observed in

patients with severe neurological deficits and poor functional recovery.

A possible explanation for this association is the dual role of uric acid as both an antioxidant and a prooxidant. While SUA exhibits neuroprotective effects by scavenging free radicals, it can also act as a pro-oxidant under pathological conditions, contributing to endothelial dysfunction, inflammation, and platelet aggregation (Kimura, 2019) ²⁸.

Conclusion

This study provides compelling evidence that serum uric acid (SUA) levels are a significant prognostic marker in acute ischemic stroke (AIS). The findings indicate that higher SUA levels (>6.5 mg/dL) are strongly associated with greater stroke severity, poorer functional recovery, and increased mortality risk. Patients with elevated SUA levels had significantly worse neurological impairment as measured by the Modified Rankin Scale (MRS) at Day 7, and regression analysis confirmed that hyperuricemia is an independent predictor of poor stroke outcomes (adjusted OR = 2.8, p < 0.01). A While most research, including the present study, suggests a strong association between elevated SUA and worse stroke outcomes, a few studies have found no significant correlation with mortality. This discrepancy highlights the need for further investigation into the interaction

between SUA and other metabolic and cardiovascular risk factors.

These findings hold important clinical implications, suggesting that serum uric acid could be used as an accessible, cost-effective biomarker to assess stroke severity and prognosis.

Serum uric acid is significantly associated with stroke severity and functional outcomes, making it a valuable prognostic biomarker in acute ischemic stroke. The findings suggest that monitoring SUA levels upon hospital admission may aid in identifying high-risk patients and optimizing stroke management strategies.

References

- Saceleanu VM, Toader C, Ples H, Covache-Busuioc RA, Costin HP, Bratu BG, et al. Integrative Approaches in Acute Ischemic Stroke: From Symptom Recognition to Future Innovations. Biomedicines. 2023;11(10).
- Wang H, Zhang H, Sun L, Guo W. Roles of hyperuricemia in metabolic syndrome and cardiackidney-vascular system diseases. Am J Transl Res. 2018;10(9):2749-63.
- Gherghina ME, Peride I, Tiglis M, Neagu TP, Niculae A, Checherita IA. Uric Acid and Oxidative Stress-Relationship with Cardiovascular, Metabolic, and Renal Impairment. Int J Mol Sci. 2022;23(6).
- Zhang W, Zhu L, An C, Wang R, Yang L, Yu W, et al. The blood brain barrier in cerebral ischemic injury – Disruption and repair. Brain Hemorrhages. 2020;1(1):34-53.
- Lakhan SE, Kirchgessner A, Hofer M. Inflammatory mechanisms in ischemic stroke: therapeutic approaches. J Transl Med. 2009;7:97.

- 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.
- Sampson AL, Singer RF, Walters GD. Uric acid lowering therapies for preventing or delaying the progression of chronic kidney disease. Cochrane Database Syst Rev. 2017;10(10):Cd009460.
- 8. Kuwabara M, Fukuuchi T, Aoki Y, Mizuta E, Ouchi M, Kurajoh M, et al. Exploring the multifaceted nexus of uric acid and health: a review of recent studies on diverse diseases. Biomolecules. 2023;13(10):1519.
- Maulana S, Nuraeni A, Aditya Nugraha B. The Potential of Prognostic Biomarkers of Uric Acid Levels in Coronary Heart Disease Among Aged Population: A Scoping Systematic Review of the Latest Cohort Evidence. J Multidiscip Healthc. 2022;15:161-73.
- Chugh C. Acute Ischemic Stroke: Management Approach. Indian J Crit Care Med. 2019;23(Suppl 2):S140-s6.
- 11. Sacco RL, Kasner SE, Broderick JP, Caplan LR, Connors JJ, Culebras A, et al. An updated definition of stroke for the 21st century: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2013;44(7):2064-89.
- 12. Fantini S, Sassaroli A, Tgavalekos KT, Kornbluth J. Cerebral blood flow and autoregulation: current measurement techniques and prospects for noninvasive optical methods. Neurophotonics. 2016;3(3):031411.
- 13. Claassen JAHR, Thijssen DHJ, Panerai RB, Faraci FM. Regulation of cerebral blood flow in humans: physiology and clinical implications of

- autoregulation. Physiological Reviews. 2021;101(4):1487-559.
- 14. Clemente-Suárez VJ, Redondo-Flórez L, Beltrán-Velasco AI, Ramos-Campo DJ, Belinchón-deMiguel P, Martinez-Guardado I, et al. Mitochondria and Brain Disease: A Comprehensive Review of Pathological Mechanisms and Therapeutic Opportunities. Biomedicines. 2023;11(9).
- 15. van Putten M, Fahlke C, Kafitz KW, Hofmeijer J, Rose CR. Dysregulation of Astrocyte Ion Homeostasis and Its Relevance for Stroke-Induced Brain Damage. Int J Mol Sci. 2021;22(11).
- Fricker M, Tolkovsky AM, Borutaite V, Coleman M, Brown GC. Neuronal Cell Death. Physiol Rev. 2018;98(2):813-80.
- 17. Elmore S. Apoptosis: a review of programmed cell death. Toxicol Pathol. 2007;35(4):495-516.
- 18. Song M, Yu SP. Ionic regulation of cell volume changes and cell death after ischemic stroke. Transl Stroke Res. 2014;5(1):17-27.
- 19. Kushiyama A, Nakatsu Y, Matsunaga Y, Yamamotoya T, Mori K, Ueda K, et al. Role of Uric Acid Metabolism-Related Inflammation in the Pathogenesis of Metabolic Syndrome Components Such as Atherosclerosis and Nonalcoholic Steatohepatitis. Mediators Inflamm. 2016;2016;8603164.
- 20. Mei Y, Dong B, Geng Z, Xu L. Excess Uric Acid Induces Gouty Nephropathy Through Crystal Formation: A Review of Recent Insights. Front Endocrinol (Lausanne). 2022;13:911968.
- Maiuolo J, Oppedisano F, Gratteri S, Muscoli C, Mollace V. Regulation of uric acid metabolism and excretion. International Journal of Cardiology. 2016;213:8-14.

- 22. Chen C, Lü JM, Yao Q. Hyperuricemia-Related Diseases and Xanthine Oxidoreductase (XOR) Inhibitors: An Overview. Med Sci Monit. 2016;22:2501-12.
- 23. Du L, Zong Y, Li H, Wang Q, Xie L, Yang B, et al. Hyperuricemia and its related diseases: mechanisms and advances in therapy. Signal Transduct Target Ther. 2024;9(1):212.
- 24. Sanchez-Lozada LG, Rodriguez-Iturbe B, Kelley EE, Nakagawa T, Madero M, Feig DI, et al. Uric Acid and Hypertension: An Update With Recommendations. Am J Hypertens. 2020;33(7):583-94.
- 25. McMullan CJ, Borgi L, Fisher N, Curhan G, Forman J. Effect of Uric Acid Lowering on Renin-Angiotensin-System Activation and Ambulatory BP: A Randomized Controlled Trial. Clin J Am Soc Nephrol. 2017;12(5):807-16.
- 26. Liu F, Du G-L, Song N, Ma Y-T, Li X-M, Gao X-M, et al. Hyperuricemia and its association with adiposity and dyslipidemia in Northwest China: results from cardiovascular risk survey in Xinjiang (CRS 2008–2012). Lipids in Health and Disease. 2020;19(1):58.
- 27. Fang Y, Mei W, Wang C, Ren X, Hu J, Su F, et al. Dyslipidemia and hyperuricemia: a cross-sectional study of residents in Wuhu, China. BMC Endocr Disord. 2024;24(1):2.
- 28. Kimura Y, Tsukui D, Kono H. Uric Acid in Inflammation and the Pathogenesis of Atherosclerosis. Int J Mol Sci. 2021;22(22).
- 29. Xu Y, Wong E, Rusli B, Abdul K, Earnest BS, Wong YH. The Association between Uric Acid Level and Ischemic Stroke. OBM Neurobiology. 2024;08:1-27.

30. Liu Z, Zhang D, Zeng L, Guo W, Lu Q, Lei Z, et al. Serum uric acid/creatinine ratio and 1-year stroke recurrence in patient with acute ischemic stroke and abnormal renal function: results from the Xi'an stroke registry study of China. Frontiers in Neurology. 2025;16:1496791.