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Original Research Article

A STUDY OF ASSOCIATION OF CLINICAL SEVERITY OF ACUTE ISCHEMIC STROKE WITH SERUM CALCIUM LEVELS AT THE TIME OF ADMISSION: A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Acute ischemic stroke is a leading cause of mortality and disability worldwide. Dysregulation of calcium homeostasis is a key mechanism in the ischemic injury. This study aimed to investigate whether serum calcium levels at the time of hospital admission correlate with the clinical severity of acute ischemic stroke as assessed by the National Institutes of Health Stroke Scale (NIHSS).

Materials and Methods: A cross-sectional observational study was conducted at T.S. Misra Medical College & Hospital, Lucknow, enrolling 50 consecutive patients diagnosed with acute ischemic stroke by CT imaging, presenting within 24 hours of symptom onset. Serum total calcium, albumin-corrected calcium, and ionized calcium were measured at admission. Stroke severity was assessed using NIHSS at admission, day 4, and discharge. Patients were classified as mild (NIHSS 1–4), moderate (NIHSS 5–15), or severe (NIHSS 16–42). Infarct size was categorized on CT imaging. Statistical correlation and group comparisons were performed across severity strata and discharge status.

Results: The mean age was 61.66 ± 13.88 years, with a male predominance of 70%. Mean serum total calcium was 9.09 ± 0.90 mg/dl; corrected calcium was 9.28 ± 0.98 mg/dl; and ionized calcium was 1.15 ± 0.12 mmol/L. At admission, 68% had moderate strokes. A statistically significant inverse relationship was found between total serum calcium and stroke severity ($p = 0.035$), with mild stroke patients having the highest calcium (9.45 ± 0.61 mg/dl) and severely affected patients the lowest (8.80 ± 0.85 mg/dl). Ionized calcium also correlated significantly with stroke severity ($p = 0.044$). Patients who deteriorated clinically had significantly lower total calcium (7.84 ± 0.58 mg/dl) compared to those who improved (9.44 ± 0.64 mg/dl; $p < 0.001$). The NIHSS improved from 7.16 at admission to 5.82 at discharge. No significant association was found between ionized calcium and infarct size ($p = 0.565$).

Conclusion: Lower admission serum calcium was significantly associated with greater stroke severity and poorer short-term outcomes. Serum total calcium may serve as a simple, cost-effective, and widely available prognostic biomarker in acute ischemic stroke. Larger prospective studies are needed.

Keywords: Acute ischemic stroke, serum calcium, corrected calcium, NIHSS score, stroke severity, infarct size, prognostic biomarker.

INTRODUCTION

Acute ischemic stroke constitutes one of the most serious neurological emergencies globally and remains a leading cause of mortality and long-term disability. The pathophysiology of cerebral ischemia is complex and involves multiple interrelated cellular and molecular mechanisms that collectively

determine the extent of neuronal injury and clinical outcome. Among the biochemical derangements that accompany an acute ischemic event, disturbances in calcium ion homeostasis have been identified as among the most pivotal contributors to the ischemic cascade — the sequence of biochemical reactions leading to irreversible neuronal death. The association between serum calcium levels and the clinical severity of acute ischemic stroke is therefore an area of active scientific inquiry, with significant implications for outcome prediction and potential therapeutic targeting.[1]

Calcium ions serve fundamental roles in the central nervous system, including neurotransmitter release, synaptic signal transmission, and maintenance of cellular membrane integrity. Under normal physiological conditions, intracellular calcium concentrations are maintained at extremely low levels relative to extracellular concentrations — a gradient essential for neuronal signalling. During cerebral ischemia, however, this tight regulation is lost. The ischemic event triggers a rapid and massive influx of calcium from the extracellular into the intracellular space, and the resulting intracellular calcium accumulation acts as the primary initiator of cell death pathways. This calcium overload activates destructive enzymes including proteases, lipases, and endonucleases, culminating in neuronal malfunction and eventual cell death.[1] The result is activation of excitotoxic cascades, oxidative stress, mitochondrial dysfunction, and apoptotic pathways — all of which are calcium-mediated processes that amplify ischemic injury.

The relationship between serum calcium levels and acute ischemic stroke outcomes has been explored in multiple studies, though findings have not always been consistent. Several key studies demonstrated that higher serum calcium levels at admission were associated with milder neurological deficits and better functional outcomes. Ovbiagele et al. found that patients with elevated admission calcium had less severe strokes and better discharge function.[2] Similarly, Buck et al. demonstrated that higher serum calcium was associated with smaller cerebral infarct volumes on neuroimaging, suggesting a possible neuroprotective role.[3] Conversely, Chung et al. reported that elevated serum calcium was associated with poorer short-term outcomes and higher long-term mortality, suggesting the relationship may be complex and influenced by the stage and context of measurement.[4] The timing of calcium measurement has also been shown to matter; Ovbiagele et al. found that calcium measured at 72–96 hours post-stroke had a stronger correlation with outcome than very early measurements.[5] In addition to timing, the specific calcium fraction being measured is important — since only ionized (free) calcium is physiologically active, direct measurement of ionized calcium or correction of total calcium for albumin levels has been advocated for more clinically meaningful interpretation.[6,7] Demographic and geographic factors further complicate interpretation, with studies in Asian populations demonstrating trends different from those in Western cohorts, reinforcing the need for population-specific research.[8,9]

Standardized clinical tools such as the NIH Stroke Scale (NIHSS) allow systematic quantification of neurological deficits, guide treatment decisions, and facilitate outcome assessment. The NIHSS has been extensively used in studies correlating admission serum calcium with stroke severity. Given that measuring serum calcium is a routine, low-cost laboratory investigation available in virtually all healthcare settings — including resource-limited environments — the potential for this biomarker to augment existing prognostic tools is clinically significant.[10] The present cross-sectional study was therefore designed to investigate the association between admission serum calcium levels and the clinical severity of acute ischemic stroke using a standardized, rigorous methodological approach, with the aim of generating evidence to guide both clinical practice and future research.

MATERIALS AND METHODS

This cross-sectional observational study was conducted in the Department of General Medicine at T.S. Misra Medical College & Hospital, Lucknow, over a period of 18 months. Fifty consecutive patients diagnosed with acute ischemic stroke by non-contrast CT (NCCT) brain — with MRI performed when clinically indicated — who presented within 24 hours of stroke onset were enrolled using non-probability convenience sampling. The sample size was calculated using the standard formula for cross-sectional study design ($n = Z^2 \times p \times q / d^2$), yielding a minimum of 49 patients, which was rounded to 50 to account for data loss. Ethical clearance was obtained from the Institutional Ethics Committee, and written informed consent was obtained from all participants or their legally authorized representatives.

Patients above 18 years of age with confirmed ischemic stroke presenting within 24 hours of symptom onset were included. Those with non-ischemic strokes, patients on calcium supplements or with pre-existing hypocalcemic conditions, pregnant patients, and those unwilling to consent were excluded. At admission, a comprehensive clinical history and neurological examination were performed. Stroke severity was assessed using the NIH Stroke Scale (NIHSS) at admission, on the 4th day, and at discharge. Patients were stratified into mild (NIHSS 1–4), moderate (NIHSS 5–15), severe (NIHSS 16–20), and very severe (NIHSS 21–42) categories. Discharge status was classified as improved, unchanged, or deteriorated.

Blood samples were obtained within 24 hours of admission for measurement of serum total calcium, albumin-corrected calcium (using the formula: corrected calcium = measured total calcium + 0.8 × [4.0 − measured albumin in g/dL]), and ionized calcium by ion-selective electrode methodology.

Infarct size was classified on neuroimaging as small (< 1.5 cm, lacunar), medium (> 1.5 cm but < 1/3 of a major arterial territory), or large (> 1/3 of MCA territory or complete infarction of a major territory).

Data were entered into structured Case Record Forms and analyzed using Pearson's correlation coefficient, one-way ANOVA, and descriptive statistics.

RESULTS

Table 1: Demographic, Clinical, and Laboratory Profile of Study Participants (N = 50)

Parameter	Finding
Age (Mean ± SD)	61.66 ± 13.88 years
Age group 51–70 years	40%
Age group 71–90 years	36%
Age group 30–50 years	24%
Male : Female	35 (70%) : 15 (30%)
BMI (Mean ± SD)	25.69 ± 5.74 kg/m²
Time to Admission (hours) ± SD	12.45 ± 6.10
Mean Serum Calcium (mg/dl) ± SD	9.09 ± 0.90
Mean Corrected Calcium (mg/dl) ± SD	9.28 ± 0.98
Mean Ionized Calcium (mmol/L) ± SD	1.15 ± 0.12
Mean Serum Albumin (g/dl) ± SD	3.76 ± 0.39
Mean Haemoglobin (g/dl) ± SD	12.81 ± 1.56
Mean Random Blood Sugar (mg/dl) ± SD	134.08 ± 39.50
Mean Hospital Stay (days) ± SD	6.58 ± 3.75
Hypertension	32 (64%)
Diabetes Mellitus	20 (40%)
Current Smokers	19 (38%)
Non-Smokers	22 (44%)
Ex-Smokers	9 (18%)

Table 2: Distribution of Stroke Severity and NIHSS value (N= 50)

Parameter	Category	Admission	4th Day	Discharge
Stroke Severity	Mild	13 (26%)	18 (36%)	23 (46%)
	Moderate	34 (68%)	29 (58%)	24 (48%)
	Severe	2 (4%)	2 (4%)	2 (4%)
	Very Severe	1 (2%)	1 (2%)	1 (2%)
NIHSS (Mean ± SD)		7.16 ± 4.82	6.20 ± 3.80	5.82 ± 3.53

Table 3: Anatomical Location, Infarct Size and discharge status (N = 50)

Variables		Frequency	Percent (%)
Stroke Location	MCA Territory	11	22.0
	Cerebellar	10	20.0
	PCA Territory	10	20.0
	ACA Territory	8	16.0
	Lacunar	6	12.0
	Brainstem	5	10.0
Infarct Size	Small	31	62.0
	Medium	16	32.0
	Large	3	6.0
Discharge Status	Improved	32	64.0
	Deteriorated	11	22.0
	Same	7	14.0

Table 4: Association of Serum Calcium and Ionized Calcium Levels with Stroke Severity at Admission (Mean ± SD)

Calcium Parameter	Mild (N = 13)	Moderate (N = 34)	Severe (N = 2)	Very Severe (N = 1)	p-value
Serum Calcium (mg/dl)	9.45 ± 0.61	9.04 ± 0.92	8.80 ± 0.85	6.90	**0.035***
Ionized Calcium (mmol/L)	1.20 ± 0.07	1.14 ± 0.11	0.99 ± 0.38	1.30	**0.044***

Statistically significant

Table 5: Association of Serum and Ionized Calcium Levels with Discharge Outcome, and Ionized Calcium with Infarct Size (Mean ± SD)

A) Calcium Levels and Discharge Outcome

Calcium Parameter	Deteriorated (N = 11)	Improved (N = 32)	Same (N = 7)	p-value
Serum Calcium (mg/dl)	7.84 ± 0.58	9.44 ± 0.64	9.47 ± 0.50	**< 0.001***
Ionized Calcium (mmol/L)	1.20 ± 0.13	1.14 ± 0.09	1.15 ± 0.21	0.42

Statistically significant

B) Ionized Calcium Levels and Infarct Size

Parameter	Small (N = 31)	Medium (N = 16)	Large (N = 3)	p-value
Ionized Calcium (mmol/L)	1.148 ± 0.103	1.150 ± 0.158	1.227 ± 0.075	0.565

Table 1 presents the demographic, clinical, and laboratory profile of the 50 enrolled patients. The mean age was 61.66 ± 13.88 years, with the largest group (40%) in the 51–70 year age range. A clear male predominance was observed (70% males). The mean BMI was 25.69 ± 5.74 kg/m², placing the cohort in the normal-to-overweight range. Hypertension was the most prevalent comorbidity (64%), followed by diabetes mellitus (40%). Mean admission serum calcium was 9.09 ± 0.90 mg/dl, corrected calcium was 9.28 ± 0.98 mg/dl, and ionized calcium was 1.15 ± 0.12 mmol/L, all within or near normal limits.

Table 2 presents stroke severity distribution at three time points, anatomical distribution of stroke location, infarct size, and discharge status. At admission, the majority of patients (68%) presented with moderate strokes (NIHSS 5–15), with 26% mild and 6% severe or very severe. Progressive improvement was observed: by discharge, 46% had mild strokes, reflecting a meaningful reduction in mean NIHSS from 7.16 at admission to 5.82 at discharge.

The MCA territory was most frequently affected (22%), and 62% of infarcts were small. Sixty-four percent of patients improved at discharge [Table 3].

Table 4 demonstrates a statistically significant inverse relationship between serum calcium level and stroke severity (p = 0.035). Patients with mild strokes had the highest mean total serum calcium (9.45 ± 0.61 mg/dl), which decreased progressively with increasing severity — moderate (9.04 ± 0.92 mg/dl), severe (8.80 ± 0.85 mg/dl), and markedly reduced in the very severe stroke patient (6.90 mg/dl). Ionized calcium similarly showed a statistically significant association with stroke severity (p = 0.044), with mild stroke patients having the highest values (1.20 ± 0.07 mmol/L) and severe stroke patients the lowest (0.99 ± 0.38 mmol/L).

Table 5A reveals a highly significant association between admission total serum calcium and discharge outcome (p < 0.001). Patients who deteriorated had markedly lower mean total calcium (7.84 ± 0.58 mg/dl) compared to those who improved (9.44 ± 0.64 mg/dl) or remained unchanged (9.47 ± 0.50 mg/dl). Ionized calcium, however, did not differ significantly across discharge status groups (p = 0.42). Table 5B demonstrates that ionized calcium levels were not significantly different across small, medium, and large infarct size categories (p = 0.565), indicating that ionized calcium at admission does not significantly vary with the anatomical extent of the infarct.

DISCUSSION

This cross-sectional study of 50 patients with acute ischemic stroke found that lower admission serum calcium levels were significantly associated with greater clinical severity and poorer short-term outcomes. The study population had a mean age of 61.66 ± 13.88 years, with the majority in the 51–70 year age group, consistent with the well-established increase in stroke incidence with advancing age and the accumulation of vascular risk factors over time — findings corroborated by Nayak RR et al,[11] and Chinnammanavar PKB et al,[12] A clear male predominance (70%; ratio 2.33:1) was observed, in keeping with the ratio of 2.25:1 reported by both Chinnammanavar PKB et al,[12] and Pavan Kumar et al,[13] reflecting the greater prevalence of traditional vascular risk factors such as smoking and hypertension among men. Hypertension was the dominant comorbidity (64%), consistent with its established role as the leading modifiable risk factor for both ischemic and hemorrhagic stroke, causing structural arterial changes including lipohyalinosis and accelerated atherosclerosis that predispose to lacunar and territorial infarcts. Diabetes mellitus was present in 40% of patients, consistent with the findings of Nayak RR et al,[11] who demonstrated that diabetes and dyslipidemia show increasing association with age. The MCA territory was the most commonly affected (22%), followed by cerebellar and PCA territories (20% each), consistent with the predominance of anterior circulation strokes in the existing literature. Small infarcts predominated (62%), suggesting a majority of patients with lacunar or partial territorial infarcts where collateral circulation limited expansion.

The principal finding of this study was a statistically significant inverse relationship between admission total serum calcium and stroke severity as measured by the NIHSS (p = 0.035) [Table 4], with mild stroke patients having the highest mean calcium (9.45 ± 0.61 mg/dl) and severe patients the lowest (8.80 ± 0.85 mg/dl; very severe patient: 6.90 mg/dl). Ionized calcium similarly correlated with severity (p = 0.044) [Table 4]. These findings are supported by multiple prior studies. Vijay Prabhu SN et al,[14] demonstrated that lower serum ionized calcium was associated with greater stroke severity on NIHSS. Pavan Kumar et al,[13] and Chinnammanavar PKB et al,[12] both reported that among several serum biomarkers, only calcium showed a positive correlation with NIHSS severity — albumin and uric acid were not significant. Guven et al,[15] demonstrated that NIHSS scores were significantly higher in patients with lower calcium levels. Amudapalli et al.[16] found that low blood calcium occurred significantly more in patients with severe stroke categories (p < 0.05), and Gupta et al,[17] emonstrated a dose-response relationship between calcium quartiles and severity. Regarding infarct size, no statistically significant association between ionized calcium and infarct size was found (p = 0.565) [Table 5B], consistent with Bawiskar N et al,[18] who also found no correlation

between ionized calcium and infarct size (r = 0.082), though they identified significant negative correlations with total and corrected calcium. Nayak RR et al,[11] and Prabhakar K et al,[19] demonstrated significant negative correlations between total calcium and infarct size, and Borah M et al.[20] showed significant negative correlations for all three calcium forms with infarct size (r = −0.58, −0.54, −0.53 respectively). The discrepancy between our ionized calcium findings and some studies may reflect differences in sample size, timing of measurement, and method of infarct size determination.

Regarding discharge outcomes [Table 5A], patients who deteriorated had significantly lower total calcium (7.84 ± 0.58 mg/dl) compared to those who improved (9.44 ± 0.64 mg/dl; p < 0.001), whereas ionized calcium did not significantly differentiate outcome groups (p = 0.42). These findings align with Amudapalli et al,[16] who demonstrated a significant difference in calcium values between poor and good outcome groups using the modified Rankin Scale. Gupta et al,[17] showed a compelling dose-response relationship, with poor functional outcomes (mRS > 3) in 91.66% of the lowest calcium quartile versus only 7.14% in the highest quartile, persisting at three months follow-up. Wu et al.[21] analyzed 1,773 patients and found the lowest ionized calcium quartile was independently associated with increased 30-day, 90-day, and 365-day mortality; notably, they also observed that the highest ionized calcium quartile was associated with increased short-term mortality — suggesting a U-shaped relationship that cautions against simple calcium supplementation.[21] The NIHSS improved from 7.16 at admission to 5.82 at discharge [Table 2], with the proportion of mild strokes increasing from 26% to 46%, consistent with the pattern described by Nayak RR et al,[11] and supporting the concept of early spontaneous neurological recovery through penumbral rescue, edema resolution, and collateral circulation. The biological plausibility for these associations is well-established: adequate serum calcium supports membrane stability and reduces pathological intracellular calcium influx during ischemia; calcium-dependent enzymes including calpains, phospholipases, and endonucleases are activated by cellular calcium overload; and low serum calcium may indicate compromised physiological reserves or reflect the systemic metabolic disruption accompanying severe cerebrovascular injury. The low cost, universal availability, and ease of serum calcium measurement make it an attractive candidate for incorporation into routine stroke assessment pathways, particularly in resource-limited settings.[10]

CONCLUSION

This study demonstrates that lower admission serum total calcium levels are significantly associated with greater clinical severity of acute ischemic stroke (p = 0.035) and with poorer short-term clinical outcomes at discharge (p < 0.001). There was also a significant correlation between ionized calcium and stroke severity on NIHSS (p = 0.044). However, ionized calcium did not significantly differ across discharge status groups (p = 0.42) or infarct size categories (p = 0.565), suggesting that while total serum calcium is the more reliable prognostic indicator, the relationship between different calcium fractions and stroke outcomes warrants further investigation. Given its simplicity, cost-effectiveness, and universal availability, serum total calcium has the potential to serve as a valuable, readily available prognostic biomarker in acute ischemic stroke care — especially in resource-limited settings. These findings contribute to the growing body of evidence supporting the importance of calcium homeostasis in cerebrovascular pathology. Large-scale, multicenter prospective studies with serial calcium measurements, long-term functional follow-up, and multivariate analyses incorporating calcium-regulatory hormones and confounding variables are required to confirm these associations and to explore whether targeted modulation of calcium homeostasis may improve outcomes in acute ischemic stroke.

REFERENCES

1. Guven H, Cilliler AE, Koker C, Sarikaya SA, Comoglu SS. Association of serum calcium levels with clinical severity of acute ischemic stroke. Acta Neurol Belg. 2011;111(1):45–9.
2. Ovbiagele B, Liebeskind DS, Starkman S, Sanossian N, Kim D, Razinia T, et al. Are elevated admission calcium levels associated with better outcomes after ischemic stroke? Neurology. 2006;67(1):170–3.
3. Buck BH, Liebeskind DS, Saver JL, Bang OY, Starkman S, Ali LK, et al. Association of higher serum calcium levels with smaller infarct volumes in acute ischemic stroke. Arch Neurol. 2007;64(9):1287–91.
4. Chung JW, Ryu WS, Kim BJ, Yoon BW. Elevated calcium after acute ischemic stroke: association with a poor short-term outcome and long-term mortality. J Stroke. 2015;17(1):54–9.
5. Ovbiagele B, Starkman S, Teal P, Lyden P, Kaste M, Davis SM, et al. Serum calcium as prognosticator in ischemic stroke. Stroke. 2008;39(8):2231–6.
6. Nayak RR, Narayanaswamy S. Association of serum calcium levels with infarct volume and stroke scores in acute ischemic stroke patients: An observational study. Caspian J Intern Med. 2022;13(4):780–5.
7. Vijay Prabhu SN, Tripathi BK, Agarwal Y, Kabi BC, Kumar R. Association of serum calcium levels with clinical severity of ischemic stroke at the time of admission as defined by NIHSS score: A cross-sectional, observational study. J Family Med Prim Care. 2022;11(10):6427–32.
8. Zhang JF, Meng X, Jing J, Pan Y, Wang YL, Zhao XQ, et al. Serum calcium and long-term outcome after ischemic stroke: results from the China National Stroke Registry III. Atherosclerosis. 2021;325:24–9.
9. Lu Y, Ma X, Tazmini K, Yang M, Zhou X, Wang Y. Admission Serum Calcium Level and Short-Term Mortality After Acute Ischemic Stroke: A Secondary Analysis Based on a Norwegian Retrospective Cohort. Front Neurol. 2022;13:889518.
10. Appel SA, Molshatzki N, Schwammenthal Y, Merzeliak O, Toashi M, Sela BA, et al. Serum calcium levels and long-term mortality in patients with acute stroke. Cerebrovasc Dis. 2011;31(1):93–9.
11. Nayak RR, Narayanaswamy S. Association of serum calcium levels with infarct volume and stroke scores in acute ischemic stroke patients: An observational study. Caspian J Intern Med. 2022 Fall;13(4):780–785.

12. Chinnammanavar PKB, Somannavar VG, Mohan PB. Role of Serum Calcium, Serum Albumin, and Serum Uric Acid as Markers of Initial Neurological Severity and Short-term Outcome Indicators in Acute Ischemic Stroke. J Assoc Physicians India. 2024 May;72(5):41–44.
13. Bc PK, Somannavar VG. A Study on the Role of Serum Calcium, Serum Albumin and Serum Uric Acid as Markers of Initial Neurological Severity and Short Term Outcome Indicators in Acute Ischemic Stroke. J Assoc Physicians India. 2022 Apr;70(4):11–12.
14. Vijay Prabhu SN, Tripathi BK, Agarwal Y, Kabi BC, Kumar R. Association of serum calcium levels with clinical severity of ischemic stroke at the time of admission as defined by NIHSS score: A cross-sectional, observational study. J Family Med Prim Care. 2022;11(10):6427–6432.
15. Guven H, Cilliler AE, Koker C, Sarikaya SA, Comoglu SS. Association of serum calcium levels with clinical severity of acute ischemic stroke. Acta Neurol Belg. 2011;111(1):45–9.
16. Amudapalli L, Alekhya A, Indira Devi T, et al. Serum calcium as a prognostic marker in acute ischemic stroke. Journal of Medical Science and Research. 2020;08(11):431–434.
17. Gupta A, Dubey U, Kumar A, et al. Correlation of serum calcium levels with severity and functional outcome in acute ischemic stroke patients. Int J Res Med Sci. 2017;3:3698–702.
18. Bawiskar N, Kumar S, Acharya S, Kothari N, Gemnani RR. Association of Serum Calcium With Infarct Size and Severity in Acute Ischemic Stroke: A Rural Hospital-Based Cross-Sectional Study. Cureus. 2023;15(8):e43015.
19. K P, N PS, S PBB. Correlation of serum calcium levels with infarct size and severity of stroke using NIHSS score in patients with acute ischemic stroke. International Journal of Research in Medical Sciences. 2019;8(1):55–59.
20. Borah M, Dhar S, Gogoi DM, Ruram AA. Association of Serum Calcium Levels with Infarct Size in Acute Ischemic Stroke: Observations from Northeast India. J Neurosci Rural Pract. 2016 Dec;7(Suppl 1):S41–S45.
21. Wu C, Li X, Li J, Huo R, Zhao H, Ying Y. Association between serum calcium and prognosis in patients with acute ischemic stroke in ICU: analysis of the MIMIC-IV database. BMC Anesthesiol. 2024;24(1):139.