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LOW SERUM CALCIUM PREDICTS HIGHER STROKE SEVERITY: A HOSPITAL-BASED STUDY OF NIHSS SCORE DETERMINANTS

Original Research

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ABSTRACT

Background: Ischemic stroke is a leading cause of global mortality and long-term disability, accounting for the majority of all stroke cases. The severity of neurological impairment in acute stroke directly influences clinical outcomes and recovery potential. Recent evidence suggests that systemic calcium dysregulation may contribute to ischemic brain injury. As an accessible and cost-effective laboratory marker, serum calcium has the potential to aid early prognostic assessment in acute stroke.

Objective: To assess the association between serum calcium levels and stroke severity, as measured by the NIH Stroke Scale (NIHSS), in patients with acute ischemic stroke.

Methods: This cross-sectional study was conducted at the Department of Neurology, Pakistan Institute of Medical Sciences (PIMS), Islamabad, from August 2024 to February 2025. A total of 1201 adult patients (≥18 years) with CT or MRI-confirmed acute ischemic stroke presenting within 24 hours of symptom onset were enrolled. Serum calcium levels and other biochemical parameters were assessed within 12 hours of hospital admission. NIHSS scores were assigned by certified neurologists within 24 hours of presentation. Multivariate linear regression analysis was used to determine the independent association between serum calcium and NIHSS scores, adjusting for age, serum albumin, blood pressure, serum electrolytes, and length of hospital stay.

Results: The mean serum calcium level was 8.71 ± 0.54 mg/dL, while the average NIHSS score was 15.27 ± 5.83. Among the cohort, 36% had calcium levels ≤8.5 mg/dL. A significant inverse correlation was found between serum calcium and stroke severity ($r = –0.31$, $p = 0.00047$). In multivariate regression, serum calcium remained an independent predictor of NIHSS score ($\beta = –3.3$, 95% CI: –5.2 to –1.4, $p = 0.001$).

Conclusion: Lower serum calcium levels are independently associated with greater neurological impairment in acute ischemic stroke, supporting its utility as a prognostic biomarker in clinical practice.

Keywords: Biomarkers, Clinical Outcomes, Ischemic Stroke, Pathophysiology, Risk Factors, Serum Calcium, Stroke Severity.

INTRODUCTION

Stroke remains a leading cause of mortality and long-term disability worldwide, imposing a profound burden on public health systems and affected individuals alike. Each year, approximately 15 million people experience a stroke, with an estimated 5 million dying and another 5 million living with permanent disabilities that compromise functional independence and quality of life (1). Ischemic stroke, the most prevalent type, accounts for nearly 87% of all stroke cases and arises due to an obstruction in the cerebral blood supply. The acute and long-term consequences of stroke have fueled the ongoing search for reliable prognostic biomarkers that can inform early clinical decisions and improve therapeutic outcomes (2). Among the various emerging biomarkers, serum calcium has drawn growing interest for its potential role in predicting stroke severity. While classically associated with skeletal integrity and neuromuscular transmission, calcium also governs critical intracellular processes, including synaptic activity, neuronal excitability, and membrane stabilization. Disruption in calcium homeostasis is a well-recognized feature of cerebral ischemia, contributing to neurodegeneration through excitotoxicity, mitochondrial failure, and oxidative stress (3,4). In this context, serum calcium levels—particularly when decreased—may reflect systemic and neuronal vulnerability, amplifying the detrimental effects of ischemic injury. The complex interplay between calcium dynamics and ischemic pathophysiology is underscored by a paradox: whereas excessive intracellular calcium influx promotes neuronal death, low extracellular or serum calcium levels may signify an impaired physiological state that exacerbates ischemic damage (5). Recent clinical evidence supports this notion, with several studies indicating that hypocalcemia is associated with greater neurological impairment, larger infarct volumes, and worse functional outcomes as measured by the NIH Stroke Scale (NIHSS) and Modified Rankin Scale (mRS) (6–8). In particular, patients with serum ionized calcium levels below 4.5 mg/dL have been found to present with significantly higher NIHSS scores, indicating more severe neurological deficits (4).

Moreover, low serum calcium may serve as a surrogate for broader systemic dysregulation. Conditions frequently coexisting with hypocalcemia—such as diabetes, renal impairment, and cardiovascular disease—are themselves well-established contributors to both stroke incidence and poor prognosis (9,10). Consequently, hypocalcemia may represent not only a modifiable factor influencing stroke severity but also a marker of compounded clinical risk. Furthermore, findings suggest that calcium deficiency may heighten the risk of post-stroke complications, including infections, thereby extending its clinical significance beyond the initial ischemic insult (7). Despite these compelling associations, considerable gaps remain in the current understanding of calcium's prognostic utility. Differences in measurement techniques—whether assessing total, ionized, or albumin-corrected calcium—may affect clinical interpretations. Additionally, it is unclear whether low calcium precedes stroke as a predisposing factor or merely reflects the acute physiological stress response to cerebral ischemia (11,12). This ambiguity underscores the need for well-designed, prospective investigations to clarify temporal relationships and explore whether calcium-targeted interventions might offer therapeutic benefit. Given its accessibility, cost-effectiveness, and minimal invasiveness, serum calcium measurement represents an attractive candidate for incorporation into routine stroke assessment protocols. Should its prognostic value be confirmed, it could inform risk stratification, guide acute management, and optimize rehabilitation planning (10). This study aims to investigate the relationship between low serum calcium levels and stroke severity in patients with acute ischemic stroke, using the NIHSS score as a standardized clinical measure. By exploring this association, the study seeks to contribute meaningful insights to the growing body of literature on serum biomarkers and their role in enhancing stroke care.

METHODS

This prospective cross-sectional observational study was conducted in the Department of Neurology at Pakistan Institute of Medical Sciences (PIMS), Islamabad, between August 2024 and February 2025. The study aimed to evaluate the association between serum calcium levels and stroke severity among patients presenting with acute ischemic stroke. Ethical clearance for the study was granted by the Institutional Review Board (IRB) of PIMS and informed consent was obtained from all participants or their legally authorized representatives prior to enrolment. The study conformed to the principles outlined in the Declaration of Helsinki and adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cross-sectional studies. Adult patients aged 18 years and above were enrolled if they presented within 24 hours of symptom onset and had a confirmed diagnosis of acute ischemic stroke based on clinical evaluation and neuroimaging—either computed tomography (CT) or magnetic resonance imaging

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(MRI) of the brain. Eligible patients were also required to have complete laboratory data, including serum calcium levels, and undergo neurological evaluation using the National Institutes of Health Stroke Scale (NIHSS) within 24 hours of admission. Only hemodynamically stable individuals who could tolerate laboratory testing were included. Patients were excluded if they had evidence of hemorrhagic stroke on neuroimaging, a prior history of stroke with residual deficits, known disorders affecting calcium metabolism such as hypo- or hyperparathyroidism, or chronic kidney disease with an estimated glomerular filtration rate (eGFR) below 30 mL/min/1.73m². Additional exclusion criteria included active malignancy or malignancy within the preceding five years, current use of calcium supplements or medications that influence calcium levels, incomplete biochemical profiles, missing NIHSS data, or death within the first 24 hours of hospital admission (13,14).

Data collection followed a standardized protocol. Certified neurologists administered the NIHSS to quantify stroke severity within 24 hours of admission. Demographic and clinical variables—including age, sex, comorbid conditions, and medication history—were obtained from patient records and structured interviews. Blood pressure was measured after a 5-minute rest in the supine position, with three readings averaged for documentation. Venous blood samples were collected within 12 hours of admission using aseptic technique, and serum calcium was measured using an automated colorimetric method with a standard reference range of 8.5–10.5 mg/dL. Additional biochemical markers such as serum albumin, glucose, sodium, and potassium were assessed using validated laboratory protocols. All tests were performed in a centrally accredited diagnostic facility by technicians blinded to the clinical status of the patients. Internal quality control measures were rigorously applied, including regular calibration of analyzers and participation in external proficiency assessments. Data entry was carried out electronically using pre-structured forms, with a subset of entries cross-verified through double entry to minimize transcription errors. Statistical analysis was performed using R software. The normality of continuous variables was evaluated using the Shapiro-Wilk test alongside histogram and Q-Q plot inspection (15,16). Descriptive statistics were presented as means ± standard deviation for normally distributed data or median with interquartile range for non-normal data. Categorical variables were reported as frequencies and percentages. Serum calcium levels were dichotomized into low (≤8.5 mg/dL) and normal/high (>8.5 mg/dL) categories based on the reference range. To assess the relationship between serum calcium and stroke severity, Pearson correlation coefficients and scatter plots were employed. A multiple linear regression model was constructed with NIHSS score as the dependent variable. Independent variables included serum calcium, serum albumin, systolic blood pressure, serum sodium, potassium, and duration of hospital stay. Diagnostic tests ensured model validity, including checks for linearity, normality of residuals, homoscedasticity using the Breusch-Pagan test, and multicollinearity assessed through variance inflation factors (VIF < 5). A p-value of less than 0.05 was considered statistically significant for all tests, and all analyses were two-tailed.

RESULTS

A total of 1201 patients with acute ischemic stroke were included in the study. The mean age of the participants was 64.28 ± 11.89 years. Stroke severity, assessed using the NIHSS, had a mean score of 15.27 ± 5.83, indicating a moderate to severe degree of neurological impairment. The mean serum calcium level was 8.71 ± 0.54 mg/dL, and serum albumin averaged 3.75 ± 0.38 g/dL. Random blood glucose levels were elevated, with a mean of 164.28 ± 34.68 mg/dL. Systolic and diastolic blood pressure measurements were 152.40 ± 18.96 mmHg and 89.16 ± 12.37 mmHg, respectively. Electrolyte levels were within physiological ranges, with serum sodium at 136.80 ± 2.84 mmol/L and potassium at 4.14 ± 0.28 mmol/L. The average length of hospital stay was 9.01 ± 3.50 days. When categorized by serum calcium levels, 36% of the cohort (n = 433) had low or borderline-normal calcium (≤8.5 mg/dL), while 64% (n = 768) had calcium levels above 8.5 mg/dL. A statistically significant inverse correlation was observed between serum calcium and NIHSS score ($r = –0.31$, $p = 0.00047$). Multiple linear regression confirmed that serum calcium remained an independent predictor of NIHSS score ($\beta = –3.3$, 95% CI: –5.2 to –1.4, $p = 0.001$) after adjusting for confounders including serum albumin, blood pressure, serum electrolytes, and length of hospital stay. Model diagnostics showed residuals to be normally distributed (mean = 0.00, SD = 5.49), with no violation of linear regression assumptions. Other variables in the model did not exhibit statistically significant associations with stroke severity.

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Table 1: Descriptive Statistics of Clinical and Biochemical Characteristics in Patients with Acute Ischemic Stroke (N = 1201)

Characteristic	Mean ± SD
Age (years)	64.28 ± 11.89
NIHSS Score	15.27 ± 5.83
Serum Calcium (mg/dL)	8.71 ± 0.54
Serum Albumin (g/dL)	3.75 ± 0.38
Random Blood Glucose (mg/dL)	164.28 ± 34.68
Systolic BP (mmHg)	152.40 ± 18.96
Diastolic BP (mmHg)	89.16 ± 12.37
Serum Sodium (mmol/L)	136.80 ± 2.84
Serum Potassium (mmol/L)	4.14 ± 0.28
Length of Stay (days)	9.01 ± 3.50
Serum Calcium Category	n (%)
Low/Normal (=8.5)	43 (36%)
High (>8.5)	77 (64%)
Residuals	0.00 ± 5.49

Table 2: Multivariate Linear Regression Analysis of Factors Associated with Stroke Severity (NIHSS Score)

Characteristic	Beta	95% CI	p-value
Serum Calcium (mg/dL)	-3.3	-5.2, -1.4	0.001
Serum Albumin (g/dL)	-0.5	-3.3, 2.3	0.7
Systolic Blood Pressure (mmHg)	-0.01	-0.07, 0.04	0.7
Diastolic Blood Pressure (mmHg)	-0.02	-0.11, 0.07	0.6
Serum Sodium (mmol/L)	0.1	-0.27, 0.46	0.6
Serum Potassium (mmol/L)	1.5	-2.2, 5.2	0.4
Length of Stay (days)	-0.2	-0.50, 0.10	0.2

Note: CI = Confidence Interval

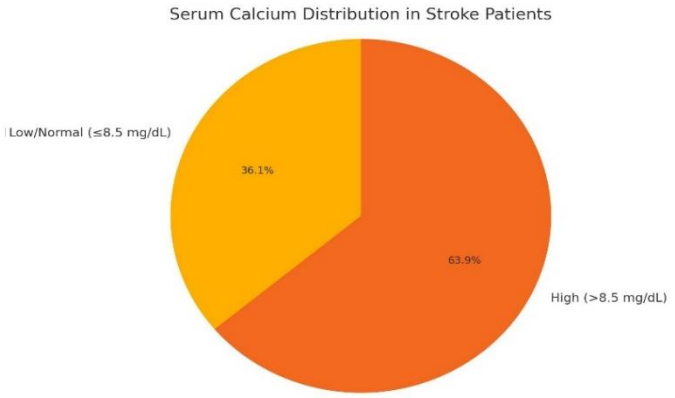

Figure 1 Serum Calcium Distribution in stoke Patients

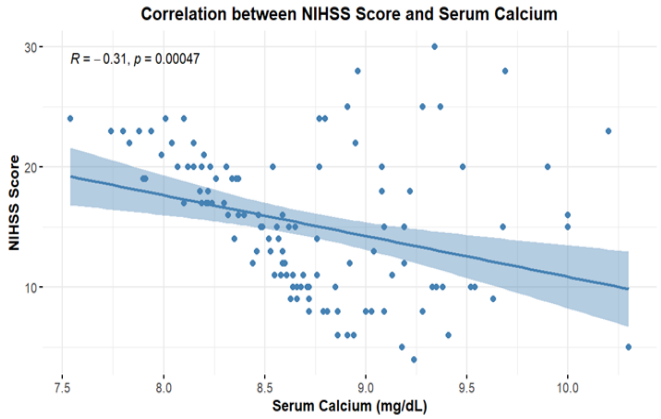

Figure 2 Correlation Between NIHSS Score and Serum Calcium

DISCUSSION

The present hospital-based cross-sectional study explored the association between serum calcium levels and stroke severity in patients diagnosed with acute ischemic stroke, revealing a significant inverse relationship between these variables. Lower serum calcium levels were found to be independently associated with higher NIHSS scores, reflecting more severe neurological deficits. This aligns with existing evidence indicating that disturbances in calcium homeostasis play a central role in the pathophysiological cascade of cerebral ischemia. Calcium ions serve as vital second messengers in neuronal function, and in the setting of ischemic injury, their dysregulation triggers a series of neurotoxic events, including excitotoxicity, mitochondrial dysfunction, oxidative stress, and ultimately, neuronal death (17,18). While most research has focused on intracellular calcium overload as a driver of neuronal injury, this study adds to the growing body of evidence suggesting that systemic hypocalcemia may also serve as an external marker of increased vulnerability to ischemic damage. Findings from this study are consistent with previous reports that have demonstrated a link between low serum calcium levels and increased stroke severity, infarct volume, and adverse short-term outcomes (19,20). Importantly, the association persisted even after controlling for potentially confounding factors such as serum albumin, blood pressure, serum electrolytes, and hospital length of stay, supporting the independent prognostic value of serum calcium. Contrary to initial expectations, variables such as serum albumin and other electrolytes did not demonstrate statistically significant associations with NIHSS scores, suggesting that serum calcium may offer a unique and more specific prognostic signal in the acute stroke setting. The clinical implications of these findings are noteworthy, especially in resource-constrained environments (21). Serum calcium is a low-cost, widely accessible, and minimally invasive parameter, and its inclusion in the initial diagnostic panel for acute ischemic stroke could enable early risk stratification and inform individualized care plans. In settings lacking advanced neuroimaging or molecular diagnostics, serum calcium could serve as a pragmatic tool for estimating stroke severity and prioritizing treatment pathways (22). Additionally, incorporating serum calcium into existing stroke severity scales or predictive algorithms could enhance their sensitivity and prognostic accuracy.

Nevertheless, certain limitations warrant careful consideration. The cross-sectional design inherently limits the ability to establish causality, as temporal changes in calcium levels post-stroke were not captured. The study relied solely on total serum calcium measurements, which may not accurately reflect biologically active calcium, particularly in the presence of altered albumin levels. Ionized and albumin-corrected calcium levels offer a more precise representation of calcium status and should be prioritized in future research. Moreover, potential confounders such as dietary calcium intake, vitamin D levels, magnesium status, and parathyroid hormone concentrations were not assessed, which could influence systemic calcium regulation and stroke outcomes. Another limitation involves the lack of functional outcome measures beyond the acute phase, such as the Modified Rankin Scale (mRS) at discharge or follow-up, which could have provided additional insight into the long-term implications of calcium levels. Furthermore, subgroup analyses based on comorbidities, age groups, infarct location, or stroke subtype were not conducted, which may have yielded important nuances in calcium's prognostic value across diverse clinical profiles. Despite these limitations, this study possesses several strengths, including the use of standardized protocols for clinical assessment, centralized laboratory testing with blinded analysis, and comprehensive multivariate adjustment for relevant clinical variables. These methodological features enhance the internal validity of the findings and

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contribute valuable evidence to an emerging area of stroke research. Future studies with longitudinal designs, larger sample sizes, and detailed biochemical profiling are warranted to validate these observations and explore mechanistic links between serum calcium and stroke pathogenesis (23). In summary, this study supports the potential of serum calcium as an independent biomarker of stroke severity in patients with acute ischemic stroke. Its routine assessment may offer an accessible and informative addition to the early evaluation process, guiding clinical decision-making and contributing to more personalized approaches in stroke care.

CONCLUSION

This study concludes that lower serum calcium levels are significantly associated with increased stroke severity in patients with acute ischemic stroke, reinforcing its potential utility as a prognostic biomarker. As a readily available, cost-effective, and minimally invasive laboratory parameter, serum calcium offers promising value for early risk assessment and clinical stratification in acute stroke care. These findings highlight the need for further exploration of serum calcium not only as a diagnostic adjunct but also as a possible therapeutic consideration in future stroke management strategies.

AUTHOR CONTRIBUTION

Author	Contribution
Ahmad Hussain*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Sayed Saad Ali	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Rahim Abbas	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Bushra Ishaq	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Khubaib Khan	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Nazia Nijat	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Muhammad Iqbal	Contributed to study concept and Data collection Has given Final Approval of the version to be published

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