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SERUM ALBUMIN LEVELS AND ITS CORRELATION WITH THE SEVERITY OF ACUTE ISCHEMIC STROKE

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ABSTRACT

Introduction: Acute ischemic stroke (AIS) significantly contributes to global neurological morbidity and mortality. The prompt identification of prognostic indicators is crucial for predicting disease severity, optimizing early therapy, and enhancing clinical outcomes. Serum albumin, a protein produced by the liver, is essential for sustaining oncotic pressure, binding both endogenous and exogenous substances, and providing antioxidant and anti-inflammatory properties. Its involvement in stroke has been subject to heightened scrutiny. Hypoalbuminemia is correlated with poorer outcomes in critically sick patients and is suggested as a possible indicator of stroke severity.

Aims: This study aimed to evaluate the correlation between serum albumin levels at admission and the severity of acute ischemic stroke.

Methods: A cross-sectional observational study was conducted at a tertiary care hospital over a period of 12 months. Patients aged 18 years and above who were admitted within 72 hours of symptom onset and diagnosed with acute ischemic stroke based on clinical examination and neuroimaging (CT/MRI) were included. Detailed history, neurological examination, and relevant laboratory investigations were performed. Serum albumin levels were measured within 24 hours of admission. Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS) at presentation and the functional outcome was evaluated as modified Rankin Scale (mRS). Statistical analysis involved Pearson's correlation and regression analysis to determine the association between serum albumin, NIHSS scores and mRS

Results: A total of 85 patients (mean age 64.2 ± 10.8 years; 60% male) were included. The mean serum albumin level was 3.4 ± 0.5 g/dL. Based on NIHSS, 34.1% had mild, 44.7% moderate, and 21.2% severe strokes. A statistically significant inverse correlation was observed between serum albumin levels and NIHSS scores and mRS ($p < 0.001$). Patients with lower albumin levels were more likely to present with severe neurological deficits. Multivariate regression analysis confirmed that serum albumin was an independent predictor of stroke severity even after adjusting for age, sex, comorbidities, and other laboratory parameters.

Conclusion: Acute ischemic stroke, Hypoalbuminemia, NIHSS score, Prognostic marker, Stroke severity.

INTRODUCTION

Stroke is a primary contributor to morbidity, death, and prolonged disability globally. Acute ischemic stroke (AIS) constitutes roughly 85% of all stroke cases and arises from the obstruction of cerebral arteries, resulting in diminished cerebral blood flow and neuronal damage.[1]

The global incidence of stroke is escalating, especially in low- and middle-income nations, attributed to the growing prevalence of risk factors including hypertension, diabetes mellitus, dyslipidemia, and aging demographics. Timely evaluation of stroke severity is essential for establishing suitable treatment approaches, forecasting outcomes, and distributing resources for rehabilitation and extended care.[2,3]

Historically, clinical scoring systems such as the National Institutes of Health Stroke Scale (NIHSS) have been employed to assess neurological impairments and forecast outcomes in Acute Ischemic Stroke (AIS). There is an increasing interest in developing dependable and economical biochemical indicators that can aid in the early evaluation of stroke severity and prognosis. One marker now under examination is serum albumin.[4]

Albumin, the predominant plasma protein produced by the liver, is crucial for sustaining oncotic pressure, facilitating the movement of hormones, fatty acids, and pharmaceuticals, and providing anti-inflammatory, antioxidant, and neuroprotective benefits.[5] It has been suggested that the biological features of albumin may affect the pathophysiological mechanisms in acute ischemic stroke, including the control of blood-brain barrier permeability, the decrease of oxidative stress, and the attenuation of inflammation.[6] Numerous experimental and clinical investigations indicate that low blood albumin levels, known as hypoalbuminemia, may correlate with adverse neurological outcomes, increased infarct volume, heightened stroke severity, and a raised risk of sequelae, including cerebral edema and infections.[7]

Furthermore, the neuroprotective and volume-expanding characteristics of albumin have prompted the concept that it may directly enhance cerebral perfusion and mitigate ischemia injury. Nonetheless, despite encouraging correlations, the connection between serum albumin and stroke severity remains well comprehended and inconsistently documented across various groups.[8]

The simplicity, cost-effectiveness, and extensive availability of blood albumin testing make it a desirable tool for assessing its relationship with stroke severity at presentation, particularly in resource-constrained environments. Comprehending this link may facilitate the early identification of high-risk individuals, inform clinical decision-making, and potentially strengthen future treatment trials concerning albumin supplementation.

AIMS

To evaluate the correlation between serum albumin levels at admission and the severity of acute ischemic stroke.

MATERIALS AND METHODS

A cross-sectional observational study was conducted over a period of 12 months at Sree Mookambika Institute of Medical Sciences, Kulasekharam. All patients aged 18 years and above, admitted to the hospital with a diagnosis of acute ischemic stroke within 72 hours of symptom onset, were screened for eligibility. The diagnosis of AIS was confirmed through clinical evaluation and neuroimaging (computed tomography or magnetic resonance imaging of the brain). A total of 85 patients were included only after informed consent was obtained from them or their legally authorized representatives.

Inclusion Criteria

- Age ≥18 years
- Presentation within 72 hours of stroke onset
- Diagnosis of acute ischemic stroke confirmed by CT/MRI
- Availability of serum albumin measurement within 24 hours of admission

Exclusion Criteria

- Hemorrhagic stroke or transient ischemic attack
- Chronic liver disease, nephrotic syndrome, malignancy, or any condition affecting serum albumin levels
- Severe infections or sepsis at admission
- Patients who received albumin supplementation before measurement

Upon admission, a detailed medical history was recorded, including demographic information, comorbidities (e.g., hypertension, diabetes, atrial fibrillation), medication use, and lifestyle factors. A comprehensive neurological examination was conducted by a trained neurologist. Laboratory investigations included complete blood count, serum electrolytes, renal and liver function tests, blood glucose levels, lipid profile, and serum albumin, which was measured using a standard automated biochemical analyzer within 24 hours of admission. serum albumin levels at admission were categorized into three groups for analysis:

- Normal albumin (≥3.5 g/dL)
- Mild hypoalbuminemia (3.0–3.49 g/dL)
- Moderate to severe hypoalbuminemia (<3.0 g/dL)

Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS) at the time of admission. The NIHSS is a validated 15-item scoring system that quantifies neurological impairment; higher scores indicate more severe stroke.[9] Functional outcome was evaluated using the modified Rankin Scale (mRS)**,** a widely used measure of disability or dependence in daily activities, with scores ranging from 0 (no symptoms) to 6 (death).[10]

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 20.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. The correlation between serum albumin levels and stroke severity (NIHSS) and functional outcome (mRS) was evaluated using Pearson's correlation coefficient. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 85 patients with confirmed acute ischemic stroke were included. The mean age was 63.7 ± 11.2 years, and 52 (61.2%) were male. The most prevalent comorbid condition was hypertension, found in 58 (68.2%) patients, followed by diabetes mellitus in 39 (45.9%) patients.

Parameter	**n (%) / Mean ± SD**
Gender – Male	52 (61.2%)
Gender – Female	33 (38.8%)
Hypertension	58 (68.2%)
Diabetes Mellitus	39 (45.9%)
Smoking	27 (31.8%)
Dyslipidemia	21 (24.7%)
Time from symptom onset to admission (hours)	18.5 ± 9.4

Table 1: Demographic and Clinical Characteristics

The mean serum albumin level of 3.42± 0.46 g/dL g/dL, though close to the lower limit of the normal range, suggests a high prevalence of hypoalbuminemia among patients presenting with acute ischemic stroke. The majority of patients (n = 37; 43.5%) had mild hypoalbuminemia, followed by 33 patients (38.8%) with normal albumin levels. A significant portion, 15 patients (17.6%)**,** presented with moderate to severe hypoalbuminemia.

Further analysis showed a trend of increasing stroke severity with decreasing albumin levels. Among patients with normal albumin levels, most presented with mild to moderate stroke severity, while severe strokes (NIHSS ≥15) were more common in those with albumin levels <3.0 g/dL**.** Patients with more severe strokes had significantly lower serum albumin levels, suggesting a strong inverse relationship between albumin and neurological deficit severity. (Table 2)

Stroke Severity (NIHSS)	**n (%)**	**Mean Albumin (g/dL) ± SD**	**p-value**
Mild (≤5)	29 (34.1%)	3.73 ± 0.32	< 0.001
Moderate (6–14)	38 (44.7%)	3.39 ± 0.35	
Severe (≥15)	18 (21.2%)	3.04 ± 0.38	

Table 2: Stroke Severity (NIHSS Score) and Serum Albumin

Functional outcome was assessed using the mRS at discharge. Patients with favorable outcomes had significantly higher mean serum albumin levels compared to those with unfavorable outcomes (3.65 ± 0.36 g/dL vs. 3.22 ± 0.41 g/dL; $p < 0.001$). A favorable functional outcome was significantly associated with higher serum albumin levels, suggesting its prognostic utility in AIS recovery.

Outcome (mRS)	**n (%)**	**Mean Albumin (g/dL) ± SD**	**p-value**
Favorable (0–2)	41 (48.2%)	3.65 ± 0.36	< 0.001
Unfavorable (3–6)	44 (51.8%)	3.22 ± 0.41	

Table 3: Functional Outcome (mRS Score) and Serum Albumin

There was a moderate, statistically significant inverse correlation between serum albumin and both NIHSS and mRS scores. After adjusting for confounders (age, sex, hypertension, diabetes, and time to admission), serum albumin remained an independent predictor of both stroke severity and functional outcome. Lower serum albumin levels were associated with higher NIHSS and mRS scores, indicating more severe neurological deficits and poorer functional outcomes. (Table 4)

Outcome Variable	Correlation Coefficient (r)	p-value	Regression Coefficient (β)	95% Confidence Interval	Adjusted p-value
NIHSS Score	–0.51	<0.001	–0.42	–0.67 to –0.23	<0.001
mRS Score	–0.43	0.002	–0.36	–0.59 to –0.14	0.002

Table 4: Correlation and Regression Analysis Between Serum Albumin, NIHSS, and mRS

DISCUSSION

The demographic distribution was consistent with previously reported epidemiological data, with a higher prevalence of stroke among males (61.2%) and a mean age of 63.7 years. Hypertension (68.2%) and diabetes mellitus (45.9%) emerged as the most common comorbidities, highlighting their critical role as risk factors for stroke.

The mean serum albumin level in the cohort was 3.42 ± 0.46 g/dL, which lies near the lower boundary of the normal physiological range. Notably, more than 60% of patients exhibited some degree of hypoalbuminemia, suggesting that reduced albumin levels may be a common finding in patients presenting with AIS.

A significant trend was observed between albumin levels and stroke severity. Patients with severe strokes (NIHSS ≥15) had markedly lower albumin levels (mean 3.04 ± 0.38 g/dL) compared to those with mild strokes (NIHSS ≤5, mean 3.73 ± 0.32 g/dL), with a statistically significant difference ($p < 0.001$). This inverse relationship suggests that low serum albumin may reflect either a marker of poor systemic health or may actively contribute to greater vulnerability of the brain to ischemic injury due to its roles in antioxidant defense, anti-inflammatory activity, and maintenance of endothelial integrity.

Functional outcomes at discharge, assessed by the mRS, further reinforced the prognostic significance of serum albumin. Patients with favorable outcomes (mRS 0–2) had significantly higher serum albumin levels (3.65 ± 0.36 g/dL) compared to those with unfavorable outcomes (mRS 3–6, 3.22 ± 0.41 g/dL; $p < 0.001$). This reinforces earlier findings that albumin may serve not only as a severity marker but also as a predictor of short-term recovery and prognosis in AIS patients.

Correlation and regression analyses supported these observations. A moderate, statistically significant inverse correlation was found between serum albumin and NIHSS ($r = –0.51$) as well as mRS ($r = –0.43$), with both correlations maintaining significance even after adjusting for potential confounders like age, sex, hypertension, diabetes, and time from

symptom onset. Regression analysis confirmed that serum albumin was an independent predictor of both neurological deficit severity and functional outcome.

These findings are consistent with previous studies which reported that hypoalbuminemia was associated with increased infarct volume, worse neurological outcomes, and higher mortality in stroke patients. The mechanisms may include increased blood-brain barrier permeability, heightened oxidative stress, and impaired vascular reactivity. The role of albumin as a neuroprotective agent has also been explored in clinical trials, such as the ALIAS (Albumin in Acute Stroke) trial, although larger trials are needed to confirm any therapeutic implications.

Several studies have consistently demonstrated that low serum albumin levels are significantly associated with poor outcomes in patients with AIS. Babu et al.[11] reported that reduced albumin levels were linked with worse functional outcomes, as indicated by a modified Rankin Scale (mRS) score >3. The adjusted odds ratio (OR) was 1.972 (95% CI, 1.103–4.001; $p < 0.001$), and patients with lower albumin levels also exhibited higher rates of stroke recurrence and mortality. This association remained significant across all stroke subtypes.

Similarly, Gao et al.[12] found that individuals in the lowest tertile of serum albumin had an OR of 2.43 (95% CI, 1.18−5.01; $p = 0.046$) for poor functional outcome, even after adjusting for demographic and clinical variables. Their use of restricted cubic spline regression indicated a linear inverse relationship between albumin levels and poor outcomes (p for linearity = 0.017), suggesting that serum albumin is an independent predictor of prognosis in AIS.

Kasundra et al.[13] further supported these findings by demonstrating that higher serum albumin levels were associated with lower NIH Stroke Scale (NIHSS) scores at admission and better functional recovery, as measured by the Barthel Index at one week post-admission ($P < 0.001$ for both).

Zhou et al.[14] extended these observations to longer-term outcomes. Over a 3-month follow-up, patients with albumin levels <35 g/L had significantly higher risks of poor functional outcome (adjusted OR 1.37; 95% CI, 1.12–1.67) and mortality (adjusted hazard ratio [HR] 2.13; 95% CI, 1.41–3.23), compared to those in the 40–44.9 g/L range. Notably, each 10 g/L decrement in serum albumin was associated with worsened prognosis (adjusted OR 1.17; HR 1.86), with these trends persisting up to one year.

Yang et al.[15] highlighted the role of albumin in post-stroke complications, particularly pneumonia. They observed an inverse dose-dependent relationship between albumin levels and

the risk of pneumonia in AIS patients, especially among those with milder strokes (OR 0.84; 95% CI, 0.77–0.93). Albumin also showed moderate predictive value for pneumonia development (AUC = 0.661; 95% CI, 0.620–0.701), with an optimal threshold of 42.6 g/L.

Finally, Bielewicz et al.[16] noted a decline in albumin levels during the acute phase of ischemic stroke and identified significant correlations between albumin deficits and various markers of injury severity, including ischemic lesion volume ($r = 0.39$, $p < 0.05$), S100BB serum levels ($r = 0.36$, $p < 0.05$), and neurological status on day 10 ($r = 0.59$, $p < 0.001$).

CONCLUSION

This cross-sectional observational study demonstrated a significant inverse correlation between serum albumin levels and both the severity and functional outcome of acute ischemic stroke. Patients with lower serum albumin levels at admission were more likely to present with higher NIHSS scores and experience worse functional outcomes as assessed by the modified Rankin Scale. The findings suggest that serum albumin, a routinely available and inexpensive biochemical parameter, may serve as a useful prognostic biomarker in the early evaluation of stroke severity and outcome.

Incorporating albumin level assessment into the initial workup of acute ischemic stroke may help in risk stratification, guiding clinical decision-making and resource allocation. However, further large-scale prospective and interventional studies are warranted to validate these results and to explore whether correction of hypoalbuminemia can improve clinical outcomes in stroke patients

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CONFLICTS OF INTEREST:

There are no conflicts of interest

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