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Original Research

# Serum Galectin-3 as a Prognostic Biomarker in Acute Ischemic Stroke: A Comparative Cross-Sectional Study

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## Abstract

**Background:** Stroke is a major cause of morbidity and mortality globally, with a particularly high burden in low- and middle-income countries such as Nigeria. Early identification of biomarkers, such as serum Galectin-3, which plays a role in inflammation and tissue remodeling, may improve diagnostic accuracy and patient outcomes in acute ischemic stroke. This study aimed to compare serum Galectin-3 levels between patients with acute ischemic stroke and age- and sex-matched stroke-free controls.

**Methodology:** A prospective comparative cross-sectional study was conducted at the University of Ilorin Teaching Hospital, Ilorin, Nigeria. A total of 100 first-ever acute ischemic stroke patients and 100 apparently healthy controls were recruited. Serum Galectin-3 levels were measured using an enzyme-linked immunosorbent assay (ELISA). Statistical analysis was performed to compare Galectin-3 levels between groups using the Statistical Package for the Social Sciences (SPSS), version 24.0.

**Results:** The median serum Galectin-3 level was significantly higher in stroke patients compared to controls [60 (55.00–63.00) vs 56 (49.00–62.00), p = 0.003]. The two groups were comparable in age and sex distribution, but hypertension and diabetes were significantly more prevalent among stroke patients. Elevated serum Galectin-3 levels were positively associated with stroke diagnosis.

**Conclusion:** Serum Galectin-3 is significantly elevated in acute ischemic stroke patients compared to stroke-free individuals. Galectin-3 could serve as an important biomarker for acute ischemic stroke, aiding in timely diagnosis and management.

**Keywords:** Stroke, Galectin-3; Biomarkers; Ischemic Stroke; Prognosis; Inflammation.

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## Introduction

Stroke is a rapidly developing clinical syndrome characterized by a disturbance of cerebral function lasting more than 24 hours or leading to death, with no apparent cause other than vascular origin [1]. It represents a major cause of morbidity, mortality, and disability worldwide [2]. According to the World Health Organization (WHO), stroke is the second leading cause of death globally [3].

Ischemic stroke, resulting from an obstruction of blood flow to the brain, is the predominant type, accounting for up to 87% of stroke cases in the Western world [4]. However, studies from Nigeria show variability in the prevalence of ischemic stroke. The large multicenter SIREN study demonstrated that ischemic stroke remains the dominant subtype in Nigerian and Ghanaian populations [5], while more recent hospital-based data from South-Southern Nigeria reported that cerebral infarction accounted for the majority of stroke admissions [6]. A continental review further confirmed that ischemic stroke constitutes most cases across Africa [7]. Stroke continues to represent a substantial proportion of hospital admissions in Nigeria, with contemporary reports showing rates comparable to earlier estimates [6, 8]
Stroke fatality rates remain high in Nigeria. A seven-year retrospective review from Benin-City, Southern Nigeria, reported case fatalities of 21.2% at 7 days, 25.5% at 14 days, and 30.8% at 30 days, with nearly 83% of deaths occurring within the first month of admission. Mortality was significantly higher in patients with intracerebral hemorrhage (68.8%) compared to those with ischemic stroke (24.0%) [9]. Sanya et al. found a 30-day in-hospital fatality rate of 21.2% in the Middle Belt region [10]. Therefore, improving early diagnosis and prognosis is critical for stroke management in resource-limited settings [11].

Galectin-3, a β-galactoside-binding lectin, has emerged as a significant molecule involved in inflammation, fibrosis, and tissue remodeling [12]. It is expressed by several cell types, including macrophages, microglia, and endothelial cells [12]. In response to cerebral ischemia, Galectin-3 expression increases, mediating processes such as angiogenesis, microglial activation, and inflammatory responses. [13]

Animal studies have shown that Galectin-3 expression peaks during early reperfusion after cerebral ischemia and contributes to post-ischemic brain remodeling [13, 14]. Human studies suggest that elevated serum Galectin-3 levels are associated with stroke severity and poor outcomes [15]. However, there is limited data regarding serum Galectin-3 in African stroke populations [16].

Thus, this study aimed to compare serum Galectin-3 levels between acute ischemic stroke patients and age- and sex-matched stroke-free controls in Ilorin, Kwara State, Nigeria, and to provide evidence for its potential use as a diagnostic biomarker.

## Methods

This was a prospective, comparative, cross-sectional hospital-based study conducted at the University of Ilorin Teaching Hospital (UITH), Ilorin, Kwara State, Nigeria [17]. UITH is a 500-bed tertiary healthcare facility that serves as a referral center for Kwara State and adjoining states[17]. The study population consisted of 100 first-ever acute ischemic stroke patients and 100 apparently healthy, age- and sex-matched stroke-free controls recruited consecutively over the study period[17]. Stroke was defined according to the World Health Organization criteria as a focal or global disturbance of cerebral function lasting more than 24 hours or leading to death, with no apparent cause other than vascular origin [1]. Diagnosis was confirmed using cranial computed tomography (CT) or magnetic resonance imaging (MRI) scans. Controls were verified to be stroke-free using the Questionnaire for Verifying Stroke-Free Status (QVSFS) [16]. Inclusion criteria for stroke patients included adults aged 18 years and above presenting within 72 hours of stroke symptom onset and confirmed diagnosis of ischemic stroke. Exclusion criteria included a previous history of stroke, chronic kidney disease, recent acute coronary

syndrome, major trauma, or surgery within one month prior to admission [17]. Controls were excluded if they had a history of stroke or active infections, or inflammatory diseases at the time of enrollment [17]. The minimum required sample size was determined using the formula for estimating a single proportion, with adjustments for the population size at UITH being less than 10,000 stroke cases annually. A 10% attrition rate was incorporated to account for non-responses, leading to a final target sample size of 100 participants in each group [16]. Ethical approval was obtained from the UITH Ethical Review Committee with the protocol number ERC/PAN/2018/09/0691, and informed consent (both verbal and written) was obtained from all participants or their legally authorized representatives before enrollment [16]. Data were collected through structured interviewer-administered proforma capturing socio-demographic and clinical characteristics. The stroke-free status of control participants was verified using the Questionnaire for Verifying Stroke-Free Status (QVSFS), a previously validated 8-item tool**.** Anthropometric measurements such as weight, height, and waist circumference were taken using standardized procedures.

Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters ($kg/m^2$). Blood pressure was measured using a mercury sphygmomanometer with an appropriately sized cuff, following standard guidelines [16]. The average of three readings taken two minutes apart after a five-minute rest was used [17]. Venous blood samples were collected from all participants. The blood was allowed to clot at room temperature and centrifuged at 3000 rpm for 10 minutes to obtain serum. The serum samples were then stored at -20°C until batch analysis. Serum Galectin-3 levels were measured using commercially available enzyme-linked immunosorbent assay (ELISA) kits (Calbiotech Inc., Spring Valley, California, USA) following the manufacturer's protocol [17]. Stroke severity on admission was assessed using the National Institutes of Health Stroke Scale (NIHSS) by a trained investigator certified in the administration of the NIHSS tool. For this study, NIHSS scores were categorized as follows: 0 = no stroke symptoms, 1–4 = minor stroke, 5–15 = moderate stroke, 16–20 = moderate-to-severe stroke, and 21–42 = severe stroke [16]. Thirty-day functional outcomes were determined using the modified Rankin Scale (mRS), categorizing patients into good (mRS 0–2) and poor (mRS 3–6) functional outcomes [16]. Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) version 24.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized using means and standard deviations or medians and interquartile ranges as appropriate, while categorical variables were presented as frequencies and percentages. The normality of continuous variables was assessed using the Shapiro-Wilk test to determine the appropriate use of parametric (Student's t-test) or non-parametric (Mann–Whitney U) tests**,** and Chi-square tests were used for categorical variables; however, Fisher's exact test was applied in instances where the expected cell frequencies were fewer than five. A p-value of less than 0.05 was considered statistically significant [16].

## Results

A total of 200 participants were recruited into the study, comprising 100 acute ischemic stroke patients and 100 stroke-free controls. There were no significant differences between the two groups regarding age (mean age: Stroke group = 64.48±9.46years; Control group = 62.87±10.31 years; p = 0.806) and sex distribution (male: Stroke group = 48%; Control group = 41%; p = 0.345)

**Table 1: Sociodemographic Characteristics of Study Participants**.

| Variable | Subjects<br>n (%)<br>n = 100 | Control<br>n (%)<br>n = 100 | Total<br>N (%)<br>N = 200 | Df | $\chi^2$- test | *p*-value |
|---|---|---|---|---|---|---|
| **Age (years)** | | | | | | |
| 18 – 45 | 2 (2.0) | 4 (4.0) | 6 (3.0) | 4 | 1.616 | 0.806 |
| 46 – 65 | 58 (58.0) | 60 (60.0) | 118 (59.0) | | | |
| > 65 | 40 (40.0) | 36 (36.0) | 76 (38.0) | | | |
| Mean±SD | 64.48±9.46 | 62.87±10.31 | | | | |
| **Sex** | | | | | | |
| Male | 48 (48.0) | 41 (41.0) | 89 (40.5) | 1 | 0.891 | 0.345 |
| Female | 52 (52.0) | 59 (59.0) | 111 (59.5) | | | |

**$\chi^2$: Chi-square test; $^t$: Paired T-test; df: degree of freedom; SD: standard deviation**

Table 2 shows the comparison of serum galectin-3 between the patients and controls. The median value of serum galectin-3 was higher in patients 60(55-63) pg/ml compared to controls 56(49-62) pg/ml. The highest median serum galectin-3 value among the stroke patients, 61(58.25-63.75) pg/ml was demonstrated among the older age group (> 65 years). A sharp rise in median serum galectin-3 value was observed [47(45) pg/ml versus 60(54.75-63.0) pg/ml] with increasing age across age groups (18-45years versus 46-65years) of the stroke participants, respectively. The median serum galectin-3 value was very slightly higher in female stroke patients 61(57.25-64.00) pg/ml, compared to the male stroke patients, 58.5(54.00- 62.00).

**Table 2: Comparison of Median Serum Galectin-3 Levels between Stroke Cases and Controls**

| Variable | Serum Galectin-3 (pg/mol) | | | | | |
|---|---|---|---|---|---|---|
| | | Subjects | | Control | Z | *p-value* |
| | | Median (IQR) | | Median (IQR) | | |
| | N=100 | | N=100 | | | |
| **Age** | | | | | | |
| 18 – 45 | 2 (2%) | 47 (45.00) | 4 (4%) | 53(47.5 - 66.75) | | |
| 46 – 65 | 58 (58%) | 60 (54.75-63.00) | 60 (60%) | 57(49.00-61.75) | | |
| > 65 | 40 (40%) | 61(58.25-63.75) | 36 (36%) | 56 (48.00-62.00) | | |
| **Sex** | | | | | | |
| Male | 48 (48%) | 58.5(54.00-62.00) | 41 (41%) | 56(47.50-61.50) | | |
| Female | 52 (52%) | 61(57.25-64.00) | 59(59%) | 57(51.00-62.00) | | |
| **Median (IQR)** | | 60 (55.00-63.00) | | 56 (49.00-62.00) | -2.978$^z$ | **0.003**** |

**IQR: Interquartile range;pg: picogram; ml: millilitres: **99% CI; $^z$: Wilcoxon signs ranked test.**

A graphical representation of the distribution of serum Galectin-3 levels between stroke patients and controls is shown.

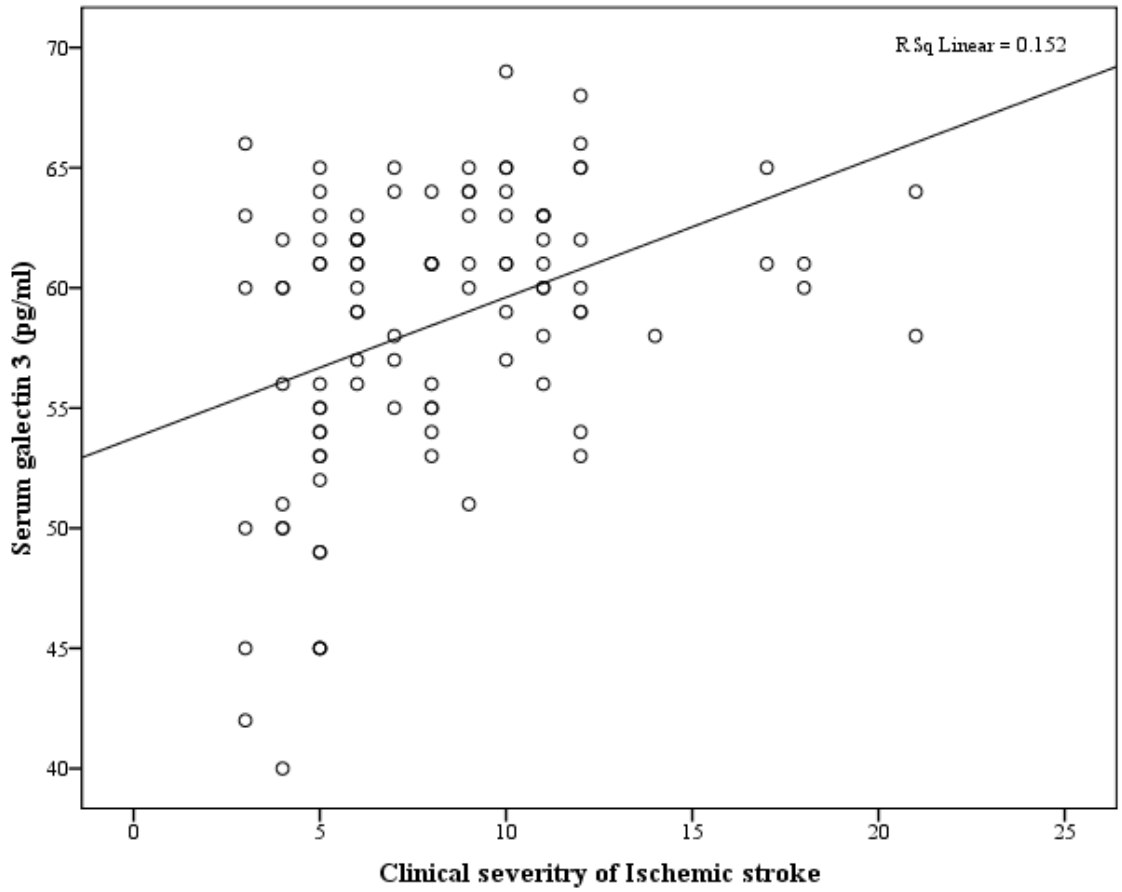


**Figure 1: Boxplot of Serum Galectin-3 Levels among Stroke Cases and Controls**

Table 3 shows there was a significant positive correlation between admission serum Galectin-3 levels and stroke severity as measured by the NIHSS (Spearman's rho = 0.15, $p < 0.001$, $R^2 = 0.526$). However, in the multivariate logistic regression model, serum Galectin-3 was not an independent predictor of 30-day mortality (OR = 0.989, 95% CI: 0.814–1.146, p = 0.984). In contrast, male sex (OR = 1.603, 95% CI: 1.016–2.340, p = 0.049), age >65 years (OR = 3.929, 95% CI: 1.066–14.488, p = 0.040), and stroke severity (NIHSS: OR = 1.129, 95% CI: 1.034–2.196, p = 0.001) were significant independent predictors of poor 30-day outcome (Table 4). Level of consciousness and infarct volume were not statistically significant.

**Table 3: Logistic Regression Analysis Showing Predictors of Poor 30-day Functional Outcome**

| **Outcome:**<br>**30-day Mortality** | **Multiple logistic regression**<br>**OR (95% C.I.)** | ***p*-value** |
|---|---|---|
| **Sex (male)** | 1.603(1.016 – 2.340) | **0.049** |
| **Age (> 65 years)** | 3.929 (1.066 – 14.488) | **0.040** |
| **Level of consciousness** | 1.214 (0.226 – 6.528) | 0.829 |
| **Serum Galectin-3** | 0.989(0.814 – 1.146) | 0.984 |
| **Infarct volume** | 0.687(0.968 – 1.012) | 0.352 |
| **Stroke severity (NIHSS)** | 0.129 (1.034 – 2.196) | 0.999 |

## Discussion

This study demonstrated that serum Galectin-3 levels were significantly higher in acute ischemic stroke patients compared to age- and sex-matched stroke-free controls. This finding suggests that Galectin-3 may play a role in the pathophysiology of acute ischemic stroke and could serve as a potential biomarker for its diagnosis and prognostication.

The elevated serum Galectin-3 levels observed among stroke patients in this study are consistent with previous reports indicating that Galectin-3 is upregulated in response to cerebral ischemia and may contribute to the inflammatory processes associated with stroke [12, 14]. Galectin-3 has been implicated in microglial activation and post-ischemic tissue remodeling, processes that are critical in the progression of neuronal injury after stroke [12, 13].

Furthermore, a positive correlation was found between admission serum Galectin-3 levels and stroke severity, as measured by the National Institutes of Health Stroke Scale (NIHSS). This aligns with experimental findings suggesting that elevated Galectin-3 levels are associated with greater neuronal damage and worse neurological deficits following ischemic injury [12, 13]

The association between higher admission Galectin-3 levels and poor 30-day functional outcomes, assessed using the modified Rankin Scale (mRS), further supports the prognostic significance of Galectin-3 in stroke outcomes. Elevated Galectin-3 levels may reflect ongoing inflammatory responses and secondary injury mechanisms that hinder functional recovery [12, 13].

Compared to other biomarkers studied in stroke, Galectin-3 offers the advantage of being a stable molecule measurable with standard laboratory techniques, making it suitable for use in resource-limited settings such as Nigeria [17]. The findings from this study provide additional evidence supporting the utility of Galectin-3 as a simple, cost-effective adjunct to clinical and radiological assessments in acute stroke management [17].

However, certain limitations must be considered. The study was conducted at a single tertiary hospital, which may limit the generalizability of the findings to other populations [17]. Additionally, serial measurements of Galectin-3 were not performed, limiting the ability to assess dynamic changes over time [16]. Despite these limitations, the study's strengths include the use of standardized diagnostic criteria, validated outcome measures, and a relatively large sample size for a biomarker study in an African population [16].

## Conclusion

This study demonstrated that serum Galectin-3 levels were significantly elevated in patients with acute ischemic stroke compared to stroke-free controls. Higher admission serum Galectin-3 levels were associated with greater stroke severity and poorer 30-day functional outcomes. These findings suggest that Galectin-3 may serve as a useful diagnostic and prognostic biomarker in the management of acute ischemic stroke. Routine measurement of serum Galectin-3 could enhance early risk stratification and clinical decision-making, particularly in resource-limited settings. Further studies are recommended to validate these findings across broader populations and assess the potential of Galectin-3 as a therapeutic target.

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