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ASSOCIATION BETWEEN ISCHEMIC STROKE AND NEUTROPHIL COUNT. A PROSPECTIVE OBSERVATIONAL STUDY.

Original Research

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ABSTRACT

Background: Ischemic stroke remains a leading cause of mortality and long-term disability worldwide, with inflammation playing a pivotal role in its pathogenesis. Neutrophils, key mediators of the innate immune response, have been increasingly implicated in exacerbating brain injury through proinflammatory mechanisms. This study investigates the prognostic significance of peripheral blood neutrophil counts in relation to stroke severity and functional outcomes in acute ischemic stroke patients.

Objective: To evaluate the association between admission neutrophil counts and the severity of ischemic stroke, and to explore the potential of neutrophil levels as a prognostic biomarker in clinical practice.

Methods: A prospective observational study was conducted at the Department of Neurology, Pakistan Institute of Medical Sciences (PIMS), Islamabad. A total of 114 adult patients with radiologically confirmed acute ischemic stroke were enrolled within 24 hours of symptom onset. Neutrophil counts were obtained from complete blood counts at admission. Stroke severity was assessed using the NIH Stroke Scale (NIHSS), and functional outcomes were measured at discharge using the modified Rankin Scale (mRS). Multivariate logistic regression was performed to evaluate associations while adjusting for age, gender, hypertension, and diabetes.

Results: Severe stroke (NIHSS ≥16) was observed in 71% of patients with high neutrophil counts (≥8000 cells/μL), compared to 39% in those with normal levels ($p = 0.005$). Severe disability (mRS ≥4) was significantly more common in the high neutrophil group (37% vs. 14%, $p = 0.009$). Multivariate analysis confirmed high neutrophil count as an independent predictor of severe stroke (adjusted OR: 3.45, 95% CI: 1.62–7.32, $p = 0.001$).

Conclusion: Elevated neutrophil counts are significantly associated with greater stroke severity and poorer functional outcomes, supporting their role as a potential prognostic biomarker in ischemic stroke. These findings warrant further research into neutrophil-targeted therapies to improve stroke prognosis.

Keywords: Biomarkers, Clinical Outcomes, Inflammation, Ischemic Stroke, Neutrophil Count, Neutrophil-Mediated Inflammation, Stroke Severity.

INTRODUCTION

Stroke remains one of the foremost public health challenges globally, ranking among the leading causes of death and long-term disability. Ischemic stroke, the most prevalent subtype, accounts for nearly 87% of all stroke cases and occurs due to the obstruction of cerebral arteries by a thrombus, leading to impaired blood flow and neuronal damage (1). Despite advances in acute stroke management, the long-term outcomes remain variable, often influenced by a complex interplay of factors such as age, comorbid conditions, genetic susceptibility, and increasingly, systemic inflammatory responses (2). Among the various components of the immune system, neutrophils have emerged as critical mediators in the pathophysiology of ischemic stroke, contributing both to injury and repair mechanisms. The inflammatory cascade following cerebral ischemia is triggered within minutes of arterial occlusion (3). Microglial and astrocytic activation leads to the release of proinflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6), which collectively disrupt the integrity of the blood-brain barrier (BBB) and promote neuronal apoptosis (4,5). Neutrophils are among the first immune cells to infiltrate the ischemic brain parenchyma, and while their role in debris clearance and tissue remodeling via enzymes like myeloperoxidase (MPO) and neutrophil elastase has been acknowledged (6), their excessive accumulation is associated with exacerbation of tissue damage. Through mechanisms such as oxidative stress induction, promotion of vascular permeability, and release of matrix metalloproteinases (MMPs), neutrophils intensify neuroinflammation and contribute to BBB degradation, facilitating further immune cell infiltration (7).

Moreover, neutrophils have been implicated in the formation of neutrophil extracellular traps (NETs), which can occlude microvessels and exacerbate ischemic injury. Several clinical studies have demonstrated a positive correlation between elevated neutrophil counts and stroke severity, as measured by the National Institutes of Health Stroke Scale (NIHSS) and infarct volumes observed on imaging modalities (8). The neutrophil-to-lymphocyte ratio (NLR) has gained attention as a readily available and cost-effective biomarker, with higher values linked to poor functional outcomes, increased mortality, greater infarct size, risk of hemorrhagic transformation, and susceptibility to post-stroke infections (9). Given these associations, neutrophil-mediated inflammation presents a promising target for therapeutic intervention. Pharmacological strategies aimed at modulating neutrophil activity—such as the use of neutrophil elastase inhibitors, MMP blockers, and anti-inflammatory compounds—have shown encouraging results in preclinical models of ischemic stroke (10). Furthermore, advances in molecular and gene-targeted therapies offer new avenues to selectively regulate neutrophil responses, potentially improving recovery and reducing secondary complications (11). In light of the mounting evidence implicating neutrophils in ischemic stroke progression and outcome, the present study aims to investigate the prognostic significance of neutrophil counts and NLR in ischemic stroke patients, with a specific focus on their association with clinical severity and functional recovery.

METHODS

This prospective observational study was conducted in the Department of Neurology at the Pakistan Institute of Medical Sciences (PIMS), Islamabad, to explore the relationship between neutrophil counts and clinical outcomes in patients with acute ischemic stroke. Adult patients aged 18 years and above who presented with radiologically confirmed acute ischemic stroke were consecutively enrolled. Neuroimaging confirmation was established through either computed tomography (CT) or magnetic resonance imaging (MRI) within 24 hours of admission. Ethical approval was granted by the Institutional Review Board (IRB) of PIMS, and written informed consent was obtained from all patients or their legally authorized representatives before enrollment. Participants were included if they presented within 24 hours of symptom onset, were aged 18 years or older, had complete baseline clinical and laboratory data, and were willing to participate. Patients were excluded if they had a hemorrhagic stroke or other non-ischemic stroke variants, chronic inflammatory or autoimmune conditions (such as systemic lupus erythematosus or rheumatoid arthritis), ongoing systemic infections at the time of admission, or a recent history (within 30 days) of immunosuppressive therapy (3,5). Furthermore, patients participating in concurrent clinical trials or those with incomplete medical records were excluded to minimize bias and maintain the study's methodological integrity.

Data collection was carried out by trained research personnel using a structured data form under the supervision of the principal investigator. Demographic data (age, sex), clinical history (comorbidities such as hypertension and diabetes mellitus), and stroke characteristics were recorded. Neutrophil counts were extracted from complete blood count (CBC) tests obtained at admission. Stroke

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severity was assessed using the National Institutes of Health Stroke Scale (NIHSS) at the time of presentation, while functional outcomes at discharge were measured using the modified Rankin Scale (mRS). Radiological findings were reviewed to confirm diagnosis and assess infarct characteristics. To ensure data reliability, a double-entry process was used, and cross-verification was performed by the principal investigator. Statistical analyses were performed using IBM SPSS version 20.0. Continuous variables were summarized as mean ± standard deviation for normally distributed data or median with interquartile range for non-normal distributions. Categorical variables were reported as frequencies and percentages. For comparisons across stroke severity groups based on NIHSS scores, independent t-tests or Mann-Whitney U tests were used, depending on data distribution. Associations between neutrophil levels and functional outcomes (mRS ≥3 indicating poor outcome) were evaluated using chi-square tests. Pearson or Spearman correlation coefficients were applied to assess the strength of relationships between neutrophil counts and NIHSS scores. Multivariate logistic regression models were constructed to adjust for potential confounding variables including age, sex, hypertension, and diabetes mellitus. Sensitivity analyses were conducted using varying neutrophil cut-off thresholds to test the robustness of the results. A two-tailed p-value <0.05 was considered statistically significant, with 95% confidence intervals reported.

RESULTS

The study included a total of 114 patients diagnosed with acute ischemic stroke, with a mean age of 52 ± 13 years. There was a slight male predominance, with 65 male patients (57%) compared to 49 females (43%). Regarding comorbidities, 54% of the patients had hypertension, 25% had both hypertension and diabetes mellitus, 6.1% had diabetes alone, and 3.5% had no significant comorbidities. The most frequently affected territory was the left middle cerebral artery (MCA) in 29% of cases, followed by the right MCA in 21% and the right cerebellum in 8.8%. Infarcts involving the anterior cerebral artery (ACA), posterior cerebral artery (PCA), brainstem, and cerebellum were less common, each representing 2.6–3.5% of cases. Patients were categorized into two groups based on neutrophil count: normal (<8000 cells/μL) and high (≥8000 cells/μL). Comparative analysis revealed that 71% of patients with high neutrophil counts presented with severe stroke, compared to 39% in the normal neutrophil group. Moderate strokes were more prevalent in the normal neutrophil group (42%) than in the elevated group (18%). These differences in stroke severity as assessed by the NIH Stroke Scale were statistically significant ($p = 0.005$). In terms of head injury classification using the Glasgow Coma Scale (GCS), severe head injury was observed in 35% of patients with high neutrophil counts versus 19% in the normal group. Minor head injuries were more common among those with lower neutrophil levels (64% vs. 47%), although this difference did not reach statistical significance ($p = 0.2$). Functional outcomes, evaluated using the modified Rankin Scale (mRS), further supported these findings. Severe disability (mRS ≥4) was significantly more frequent in the high neutrophil group (37%) than in the normal group (14%), with the difference achieving statistical significance ($p = 0.009$). Minor disability (mRS 1–2) was more prevalent in the normal neutrophil group (8.3% vs. 1.3%).

A graphical analysis of neutrophil levels in relation to stroke severity showed a clear trend: mean neutrophil counts increased with the degree of stroke severity. Patients with moderate strokes exhibited the lowest neutrophil counts, while those with severe strokes had the highest values, with a wider range of variability. Logistic regression modeling demonstrated a progressively rising probability of severe stroke with increasing neutrophil counts. The predicted probability curve displayed a steep incline around mid-range neutrophil levels, plateauing at higher concentrations, which suggests a saturation point in the inflammatory response beyond which additional neutrophils confer limited additional risk. After controlling for potential confounders including age, gender, hypertension, and diabetes mellitus, multivariate logistic regression analysis demonstrated that elevated neutrophil counts (≥8000 cells/μL) remained a significant independent predictor of severe stroke. Patients with high neutrophil levels were 3.45 times more likely to present with severe stroke compared to those with normal levels (adjusted OR: 3.45, 95% CI: 1.62–7.32, $p = 0.001$). Hypertension was also independently associated with an increased risk of severe stroke (adjusted OR: 2.13, 95% CI: 1.02–4.46, $p = 0.045$). In contrast, neither age (adjusted OR: 1.01, 95% CI: 0.97–1.06, $p = 0.58$), male gender (adjusted OR: 1.31, 95% CI: 0.66–2.63, $p = 0.43$), nor diabetes mellitus (adjusted OR: 1.26, 95% CI: 0.58–2.75, $p = 0.55$) showed statistically significant associations with stroke severity after adjustment. These findings reinforce the prognostic relevance of neutrophilic inflammation in acute ischemic stroke and support its potential utility as a biomarker for risk stratification at presentation.

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Table 1: Demographic and Clinical Characteristics of the Study Population (N = 114)

Variable	n (%) or Mean ± SD
Age	52 ± 13
Gender	
Female	49 (43%)
Male	65 (57%)
Comorbidities	
Diabetes	7 (6.1%)
HTN, DM	4 (3.5%)
Hypertension	62 (54%)
Hypertension diabetes	29 (25%)
Hypertension, diabetes	8 (7.0%)
Nil	4 (3.5%)
Territory of Infarction	
Brain stem	3 (2.6%)
Cerebellar	4 (3.5%)
Left ACA	4 (3.5%)
Left MCA	33 (29%)
Left PCA	4 (3.5%)
MCA stroke	4 (3.5%)
Right ACA	4 (3.5%)
Right MCA	24 (21%)
Right cerebellum	10 (8.8%)
Right basal MCA	4 (3.5%)

Table 2: Comparison of Clinical Outcomes by Neutrophil Count Categories

Variable	Normal Neutrophil Count (<8000 cells/µL, N = 361)	High Neutrophil Count (≥8000 cells/µL, N = 781)	p-value
GCS Score Categorization			
Severe head injury (coma)	7 (19%)	27 (35%)	0.2
Moderate head injury	6 (17%)	14 (18%)	
Minor head injury	23 (64%)	37 (47%)	
mRs Categories			
No disability	0 (0%)	0 (0%)	0.009
Minor disability	3 (8.3%)	1 (1.3%)	
Moderate disability	28 (78%)	48 (62%)	
Severe disability	5 (14%)	29 (37%)	
Stroke Severity (NIHSS)			
No stroke symptoms/Minor stroke	0 (0%)	0 (0%)	0.005

Variable	Normal Neutrophil Count (<8000 cells/µL, N = 361)	High Neutrophil Count (≥8000 cells/µL, N = 781)	p-value
Moderate stroke	15 (42%)	14 (18%)	
Moderate to severe stroke	7 (19%)	9 (12%)	
Severe stroke	14 (39%)	55 (71%)	

Table 3: Adjusted Odds Ratios for Severe Stroke

Variable	Adjusted OR	95% CI	p-value
High Neutrophil Count (≥8000 cells/µL)	3.45	1.62-7.32	0.001
Hypertension	2.13	1.02-4.46	0.045
Age	1.01	0.97-1.06	0.58
Male Gender	1.31	0.66-2.63	0.43
Diabetes Mellitus	1.26	0.58-2.75	0.55

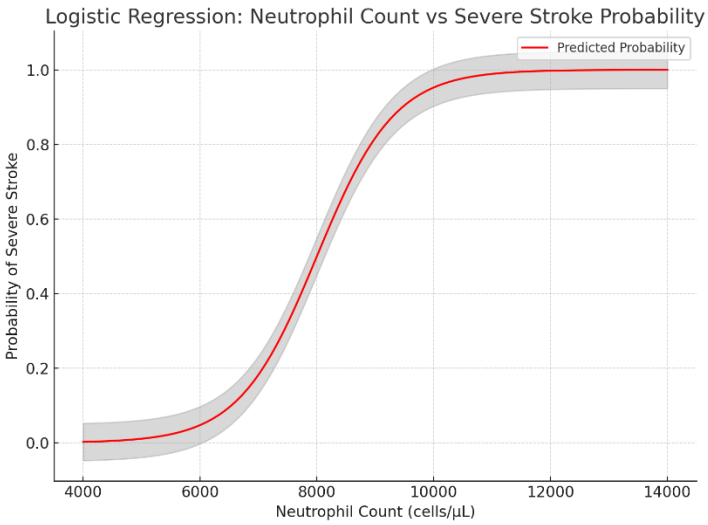

Figure 1 Logistic Regression: Neutrophil Count vs Severe Stoke Probability

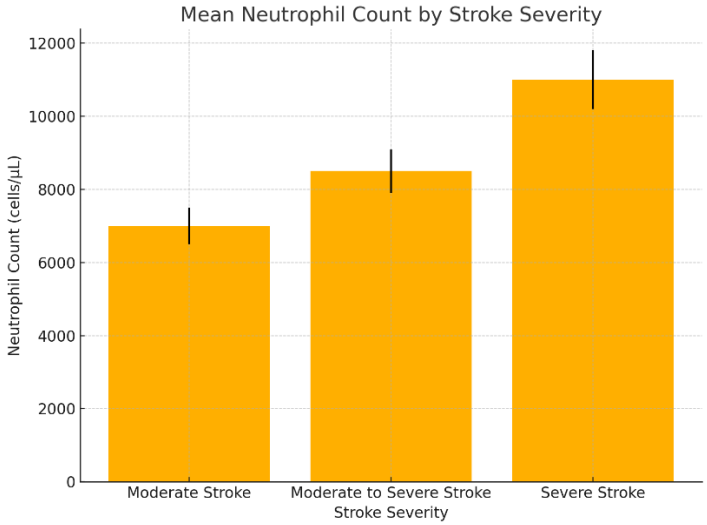

Figure 2 Mean Neutrophil Count by Stroke Severity

DISCUSSION

The findings of this study underscore a robust association between elevated neutrophil counts and increased severity of ischemic stroke, thereby reinforcing the inflammatory basis of cerebrovascular injury. Patients with neutrophil counts equal to or exceeding 8000 cells/µL were significantly more likely to present with severe strokes as assessed by NIHSS scores, with 71% exhibiting severe deficits compared to only 39% in the normal neutrophil group ($p = 0.005$). This observation is consistent with prior literature that has described the central role of neutrophils in mediating early post-ischemic inflammation and subsequent neurological deterioration (12). Neutrophils infiltrate the ischemic brain rapidly and, through degranulation and release of reactive oxygen species, contribute to the disruption of the blood-brain barrier and neuronal injury. The dualistic role of neutrophils in ischemic stroke pathophysiology is well acknowledged. While they may promote tissue recovery through the clearance of necrotic debris and secretion of remodeling enzymes such as myeloperoxidase (MPO), their excessive accumulation has been implicated in amplifying oxidative stress and triggering secondary damage via matrix metalloproteinases and proinflammatory cytokines (13,14). This cascade is closely intertwined with glial cell activation and the subsequent release of mediators like IL-6, TNF-α, and IL-1β, all of which synergistically contribute to exacerbation of brain injury and

functional impairment (15). The neutrophil-to-lymphocyte ratio (NLR) has emerged as a surrogate inflammatory marker with prognostic significance. Its elevation correlates with greater infarct volume and worse outcomes, a finding that was mirrored in the current study where severe disability (mRS ≥4) was observed in 37% of patients with high neutrophil counts compared to only 14% in the normal group ($p = 0.009$) (16,17).

The strength of this study lies in its prospective design and adjustment for confounding variables through multivariate logistic regression, which revealed that elevated neutrophil count remained an independent predictor of severe stroke (adjusted OR: 3.45, 95% CI: 1.62–7.32, $p = 0.001$) (18). These findings highlight the potential of using neutrophil count as a prognostic biomarker in acute stroke triage, enabling clinicians to identify patients at higher risk for poor outcomes. Moreover, the results lend support to emerging therapeutic strategies targeting neutrophil-mediated injury, including the use of neutrophil elastase inhibitors and MMP blockers that have demonstrated efficacy in experimental models of cerebral ischemia (19,20). Despite these strengths, the study had certain limitations. It was conducted at a single tertiary care center, which may limit the generalizability of findings to broader populations. Additionally, the study relied on a single measurement of neutrophil count at admission, without assessing temporal fluctuations that could offer insights into dynamic immune responses. The sample size, although adequate for preliminary associations, may not have been sufficiently powered to detect more subtle interactions between neutrophil count and secondary clinical variables. Moreover, the study did not include longer-term follow-up data beyond discharge, which limits understanding of the lasting prognostic value of admission neutrophil counts on stroke recovery trajectories.

Future research should consider multi-center validation with larger sample sizes and include longitudinal monitoring of inflammatory biomarkers throughout the course of hospitalization and recovery. Integrating neutrophil count trends with other inflammatory indices, neuroimaging biomarkers, and clinical scales could refine risk stratification tools. Additionally, interventional trials evaluating the impact of targeted anti-inflammatory therapies on stroke outcomes would be instrumental in translating these findings into therapeutic protocols. Overall, this study adds to the growing body of evidence supporting the utility of neutrophil-based metrics in prognostication and management of ischemic stroke, while also identifying avenues for advancing individualized stroke care.

CONCLUSION

In conclusion, this study highlights a clear and meaningful association between elevated neutrophil counts and the severity of ischemic stroke, emphasizing the prognostic value of inflammatory markers in acute cerebrovascular events. By demonstrating that higher neutrophil levels are linked to worse neurological outcomes and greater functional impairment, the findings support the role of neutrophils not only as contributors to post-stroke inflammation but also as potential clinical biomarkers for risk stratification. These insights have practical implications for early assessment and tailored management of stroke patients, suggesting that routine monitoring of neutrophil levels could inform prognosis and guide therapeutic strategies. The research also sets a foundation for future exploration into targeted anti-inflammatory interventions aimed at modulating neutrophil activity to improve recovery and long-term outcomes in ischemic stroke care.

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
Nazia Nijat	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
Sayed Rooh Ullah	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Aimal Khan	Contributed to Data Collection and Analysis

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Author	Contribution
	Has given Final Approval of the version to be published
Shabab Hussain	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Adeeb Hussain	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Muhammad Afzal	Contributed to study concept and Data collection Has given Final Approval of the version to be published
Haider Abbas	Writing - Review & Editing, Assistance with Data Curation

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