

Leukocytes, platelets, and electrolyte status: correlation with acute ischemic stroke severity at Haji Adam Malik Hospital

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Abstract

Background

Acute ischemic stroke is a major cause of disability and mortality, with inflammatory processes and metabolic stability playing key roles in its progression. This study aimed to analyze the correlation of leukocyte counts, platelet counts, and electrolyte status (sodium, potassium, and chloride) with the severity of acute ischemic stroke based on the National Institutes of Health Stroke Scale (NIHSS) at Haji Adam Malik Hospital, Medan.

Methods

This analytical study used a cross-sectional design involving 90 ischemic stroke patients admitted during the 2023–2025 period. Baseline laboratory values and initial NIHSS scores were retrieved from medical records and analyzed statistically.

Results

The analysis revealed a significant positive correlation between leukocyte count and NIHSS score ($p = 0.002$; $r = 0.315$) and between platelet count and NIHSS score ($p = 0.005$; $r = 0.293$), indicating that higher leukocyte and platelet levels upon admission are associated with more severe neurological deficits. In contrast, no statistically significant correlations were found between NIHSS scores and levels of sodium ($p = 0.272$), potassium ($p = 0.211$), or chloride ($p = 0.487$).

Conclusion

Elevated baseline leukocyte and platelet counts are significantly correlated with greater acute ischemic stroke severity, serving as valuable indicators for early clinical stratification. Conversely, early serum electrolyte levels do not demonstrate a direct relationship with initial neurological severity in these patients.

INTRODUCTION

Ischemic stroke remains one of the leading causes of mortality and long-term disability worldwide, accounting for a substantial global health burden. Recent epidemiological data indicate that stroke is the second leading cause of death and the third leading cause of death and disability combined, with more than 12 million new cases and 6.5 million deaths reported annually.¹ The burden of stroke is disproportionately concentrated in low- and middle-income countries, including Indonesia, where stroke continues to be a major contributor to morbidity and mortality.²

Assessment of stroke severity is crucial for determining prognosis and guiding acute management. The National Institutes of Health Stroke Scale (NIHSS) is the most widely used and validated clinical instrument

for evaluating neurological deficits in acute ischemic stroke. Higher NIHSS scores are associated with larger infarct volumes, poorer functional outcomes, and increased mortality.^{3,4} Consequently, identifying readily available biomarkers associated with NIHSS scores may facilitate early risk stratification and improve clinical decision-making.

Inflammation plays a pivotal role in the pathophysiology of ischemic stroke. Cerebral ischemia triggers activation of inflammatory pathways characterized by leukocyte recruitment, cytokine release, and blood–brain barrier (BBB) disruption, all of which contribute to secondary neuronal injury.⁵ Elevated leukocyte counts have been associated with infarct expansion, worse neurological deficits, and unfavorable clinical outcomes.⁶ Likewise, platelets are critically involved in thrombus formation and inflammatory responses during cerebral ischemia. Alterations in platelet count and platelet-related indices have been linked to stroke severity and prognosis, although existing evidence remains inconsistent.^{7,8}

Electrolyte disturbances are also frequently observed in patients with acute ischemic stroke and may exacerbate neurological injury. Hyponatremia has been associated with cerebral edema, increased NIHSS scores, poor functional recovery, and higher mortality rates.^{9,10} Abnormal potassium levels may contribute to neuromuscular dysfunction, cardiac arrhythmias, and adverse neurological outcomes, while chloride imbalance has been linked to greater stroke severity and increased mortality risk.^{11–13} Despite growing evidence regarding the prognostic significance of leukocyte count, platelet count, and serum electrolyte abnormalities in ischemic stroke, findings across studies remain heterogeneous, and data from Indonesian populations are still limited. Therefore, this study aimed to determine the association between leukocyte count, platelet count, and serum electrolyte levels (sodium, potassium, and chloride) and the severity of ischemic stroke as assessed by the National Institutes of Health Stroke Scale (NIHSS) in patients treated at Adam Malik General Hospital, Medan.

METHODS

Study design and setting

This retrospective analytic observational study employed a cross-sectional design to evaluate the association between leukocyte count, platelet count, serum sodium, potassium, and chloride levels and ischemic stroke severity as assessed by the National Institutes of Health Stroke Scale (NIHSS). The study was conducted at the Department of Neurology, H. Adam Malik General Hospital, a tertiary referral and teaching hospital in Medan, North Sumatra, Indonesia. Data collection was performed between August 10 and August 22, 2025, using medical records of patients diagnosed with ischemic stroke during the period 2023–2025.

Study population and participants

The study population consisted of all adult patients diagnosed with acute ischemic stroke who were admitted to the Department of Neurology during the study period. Eligible participants were identified

through hospital medical records and selected using a consecutive sampling approach.

Inclusion criteria were: (1) age ≥ 18 years; (2) diagnosis of acute ischemic stroke confirmed by clinical evaluation and neuroimaging findings; (3) availability of complete laboratory data including leukocyte count, platelet count, serum sodium, potassium, and chloride levels obtained at admission; and (4) documented National Institutes of Health Stroke Scale (NIHSS) assessment performed during the acute phase of hospitalization.

Patients were excluded if they had: (1) hemorrhagic stroke or transient ischemic attack; (2) recurrent stroke with incomplete baseline data; (3) active systemic infection at admission; (4) hematological disorders affecting leukocyte or platelet counts; (5) chronic kidney disease requiring dialysis; (6) severe hepatic dysfunction; (7) malignancy; (8) autoimmune or inflammatory diseases known to affect hematological parameters; or (9) incomplete clinical, laboratory, or NIHSS data. A total of 90 patients fulfilled the eligibility criteria and were included in the final analysis.

Sample size and sampling technique

The minimum sample size was calculated using the formula for correlation analysis proposed by Dahlan, assuming a significance level (α) of 0.05 and a minimum clinically meaningful correlation coefficient (r) of 0.30. The minimum required sample size was 85 participants.

Participants were recruited using a consecutive sampling method, whereby all eligible patients meeting the inclusion criteria during the study period were included until the required sample size was achieved.

Data Collection

Secondary data were obtained from hospital medical records. Extracted information included demographic characteristics, CT-scan findings confirming ischemic stroke, NIHSS scores, leukocyte count, platelet count, serum sodium level, serum potassium level, and serum chloride level. Although the original protocol planned serial assessments at admission, one week after admission, and at discharge, the present analysis utilized admission data only.

Variables and Measurements

The primary outcome of this study was stroke severity, which was evaluated at hospital admission using the NIHSS. The NIHSS is a well-validated neurological assessment tool with scores ranging from 0 to 42 points, where higher scores indicate a greater degree of neurological impairment. The independent variables comprised various laboratory parameters obtained from routine blood examinations performed at admission, specifically leukocyte count ($10^3/\mu\text{L}$), platelet count ($10^3/\mu\text{L}$), and serum concentrations of sodium (mEq/L), potassium (mEq/L), and chloride (mEq/L). Prior to statistical analysis, data management procedures, including rigorous data editing, coding, entry, cleaning, and storage, were systematically

executed. Data quality was further verified through repeated validation checks to minimize transcription and entry errors.

Statistical analyses were performed using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA). The normality of data distribution was assessed using the Kolmogorov-Smirnov test. Descriptive statistics were employed to summarize demographic and clinical characteristics; continuous variables are presented as mean $\pm$ standard deviation (SD) or median and interquartile range (IQR) according to their distribution, while categorical variables are expressed as frequencies and percentages.

Bivariate associations between NIHSS scores and baseline laboratory parameters were evaluated using correlation analysis. Data distribution was assessed prior to analysis; for variables demonstrating a normal distribution, Pearson's correlation coefficient was employed. Conversely, for variables with a non-normal distribution or those involving the ordinal NIHSS scores, Spearman's rank correlation coefficient was utilized. All tests were two-sided, and a p-value < 0.05 was considered statistically significant.

Ethical Considerations

The study protocol was reviewed and approved by the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sumatera Utara, under ethical approval number 616/KEPK/USU/2025. Because the study utilized retrospective medical-record data, all patient information was anonymized before analysis to ensure confidentiality and privacy. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

RESULTS

Overall baseline data

A total of 90 patients with acute ischemic stroke were included in the analysis (Table 1). Based on NIHSS classification, 29 patients (32.20%) had minor stroke, 42 (46.70%) had moderate stroke, 14 (15.60%) had mod. to sev. stroke, and 5 (5.60%) had severe stroke.

Table 1
Patients' characteristics (N = 90)

Feature		<i>p value</i> ^a
Age, in years, M (IQR)	59 (31–83)	0.003
Male, n(%)	45 (50.00)	0.000
NIHHS, M (IQR)	9 (1–32)	0.003
Leukocyte (10 ³ /μL), M (IQR)	10.280 (3.620–31.180)	0.002
Platelet (10 ⁵ /μL), M (IQR)	2.600 (0.810–5.800)	0.025
Sodium mEq/L, M (IQR)	144.500 (112.000-153.000)	0.000
Potassium mEq/L, χ (s)	3.930 (0.611)	0.091
Chloride mEq/L, M (IQR)	105.000 (92.000-113.000)	0.000
χ : mean; s: standard deviation; M: Median; IQR : Interquartile Range; <i>p value</i> ^a : For normally distributed continuous variables, values were derived from pearson correlation; for non-normally distributed continuous variables, values were obtained from Spearman's rank correlation tests		

Spearman correlation analysis demonstrated a significant positive association between admission leukocyte count and stroke severity (Table 2). Patients with leukocytosis accounted for 41.1% of the study population, and a greater proportion of severe stroke cases occurred in this group. Leukocyte count showed a weak-to-moderate positive correlation with NIHSS score ($r = 0.315$, $p = 0.002$), indicating that higher leukocyte levels were associated with greater neurological impairment.

Table 2
Bivariate Correlation between variables and NIHSS score (N = 90)

Variable	NIHSS score										Total	<i>p</i> value	<i>r</i> value
	Minor		Moderate		Mod. to Sev.		Severe						
n	%	n	%	n	%	n	%	n	%				
Leukocyte	0	0.00	1	1,10	0	0,00	0	0,00	1	1,10	0,002 ²	0,315 ²	
Low	23	25.60	22	24.40	6	6.70	1	1.10	52	57.80			
Normal	6	26.70	19	21.10	8	8.90	4	4.40	37	41.10			
High													
Platelet	3	3.30	2	2.20	0	0,0	0	0.00	5	5,60	0,005 ²	0,293 ²	
Low	26	28.90	40	44.40	12	13.3	4	4.40	82	91.10			
Normal	0	0.00	0	0,00	2	2.20	1	1.1	3	3.30			
High													
Sodium	3	3.30	4	4.40	1	1.1	0	0.00	8	8.80	0,272 ²	0,117 ²	
Low	15	16.80	25	27.80	6	6.7	2	2.20	48	53.50			
Normal	11	12.20	13	14.40	7	7.8	3	3.30	34	37.70			
High													
Potassium	4	4.40	9	10.10	3	3.30	2	2.2	18	20.00	0,211 ¹	-0,133 ¹	
Low	25	27.8	33	36.70	11	12.2	3	3.3	72	80.00			
Normal	0	0.00	0	0.00	0	0.00	0	0.00	0	0.00			
High													
Chloride	2	2.20	3	3.30	0	0.00	0	0.00	5	5.6	0,487 ²	0,074 ²	
Low	24	26.7	30	33.30	11	12.20	5	5.60	70	77.8			
Normal	3	3.3	9	10.10	3	3.30	0	0.00	15	16.6			
High													
¹: Data were analysed using the Pearson correlation test, ²: Data were analysed using Spearman's rank correlation test.													

¹: Data were analysed using the Pearson correlation test, ²: Data were analysed using Spearman's rank correlation test.

Similarly, platelet count was significantly associated with NIHSS score. Patients with thrombocytosis represented 3.3% of the cohort, and all of them were classified as having mod. to sev. or severe stroke. Spearman analysis revealed a weak positive correlation between platelet count and NIHSS score ($r = 0.293$, $p = 0.005$), suggesting that higher platelet counts were associated with increased stroke severity.

In contrast, serum electrolyte parameters were not significantly associated with stroke severity. No statistically significant correlations were observed between serum sodium level and NIHSS score ($p = 0.272$), serum potassium level and NIHSS score ($p = 0.211$), or serum chloride level and NIHSS score ($r = 0.074$, $p = 0.487$).

Overall, leukocyte count and platelet count demonstrated significant positive correlations with NIHSS score, whereas serum sodium, potassium, and chloride levels did not show significant associations with neurological severity at admission.

DISCUSSION

This study investigated the correlation between baseline hematological parameters (leukocytes and platelets), serum electrolytes (sodium, potassium, and chloride), and the initial neurological severity of acute ischemic stroke graded by NIHSS upon admission at H. Adam Malik Hospital. Bivariate analysis revealed a statistically significant, positive correlation between both baseline leukocyte count and platelet count with baseline NIHSS scores. Conversely, serum electrolyte levels, namely sodium, potassium, and chloride did not demonstrate a statistically significant linear correlation with immediate stroke severity in this cohort. These findings underscore the distinct pathophysiological roles of early sterile neuroinflammation and microvascular thrombosis as direct drivers of acute cerebral tissue injury, whereas systemic electrolyte fluctuations may represent subacute homeostatic shifts or secondary metabolic complications rather than primary determinants of initial neurological deficits.

Cerebral ischemia triggers an immediate, sterile inflammatory cascade within the brain parenchyma. The sudden reduction in cerebral blood flow deprives neurons and astrocytes of oxygen and glucose, leading to ATP depletion, cellular depolarization, and subsequent necrotic cell death. These dying cells release danger-associated molecular patterns (DAMPs), such as high-mobility group box 1 (HMGB1), heat shock proteins, and purines, into the extracellular matrix.¹⁴ DAMPs bind to pattern recognition receptors (PRRs), including toll-like receptors (TLRs), on resident microglial cells and perivascular macrophages, activating them toward a pro-inflammatory M1 phenotype.¹⁵

Once activated, these resident immune cells release a surge of pro-inflammatory cytokines, including tumor necrosis factor- α (TNF- α), interleukin-1 beta (IL-1 β), and interleukin-6 (IL-6), alongside chemokines like monocyte chemoattractant protein-1 (MCP-1).¹⁶ This localized chemical gradient alters the endothelial expression of cell adhesion molecules, such as P-selectin, E-selectin, intercellular adhesion molecule-1 (ICAM-1), and vascular cell adhesion molecule-1 (VCAM-1), in the cerebral microvasculature.¹⁷ Simultaneously, this local signaling triggers a systemic stress response via the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system, stimulating the bone marrow to accelerate the release of stored marginated margin pool leukocytes, resulting in the peripheral leukocytosis observed upon hospital admission.¹⁸

Our descriptive data strongly support this pathophysiological framework: 41.1% of the patients presented with explicit leukocytosis ($> 11 \times 10^3/\mu\text{L}$), and notably, 80% of the cases categorized as severe stroke (NIHSS

21–42) were found within this leukocytotic subgroup. Peripheral neutrophils are the first circulating immune cells to cross the compromised BBB into the ischemic penumbra, often within the first few hours of onset.¹⁹ Once inside the parenchyma, neutrophils exacerbate tissue damage through the release of matrix metalloproteinases (particularly MMP-9), reactive oxygen species (ROS), and the formation of neutrophil extracellular traps (NETs).²⁰ MMP-9 degrades endothelial basal lamina proteins, aggravating vasogenic edema, increasing the volume of the ischemic core, and predisposing the brain to hemorrhagic transformation.²¹

Furthermore, a high circulating leukocyte volume can cause mechanical clogging within the compromised cerebral microcirculation, a phenomenon known as the "no-reflow" effect.²² When leukocytes adhere extensively to the endothelial walls of capillarized ischemic zones, they physically obstruct microvascular blood flow, impeding collateral circulation and expanding the ischemic core into tissue that might otherwise have been salvaged as penumbra.²³ This correlation aligns with Semerano et al. and Boehme et al., who demonstrated that early leukocyte elevation independently predicts larger infarct volumes on neuroimaging and corresponds with elevated baseline NIHSS scores.^{24,25}

A key finding in this study is that despite the vast majority of the population (91.1%) maintaining platelet counts within the conventional physiological range (150,000–450,000/ μ L), higher absolute platelet values within or above this normal spectrum correlated linearly with increased neurological impairment. This pattern is exemplified by the fact that 100% of the patients presenting with true thrombocytosis (> 450,000/ μ L) presented with moderate to severe or severe neurological deficits.

Platelets are fundamental cellular components in the initiation and propagation of the ischemic cascade, particularly in stroke subtypes driven by large artery atherosclerosis or small vessel occlusion.²⁶ The rupture or erosion of an intra-arterial atheromatous plaque exposes subendothelial collagen and von Willebrand factor (vWF) to the circulating blood. Platelets rapidly adhere to these exposed substrates via surface glycoprotein receptors (GP Ia/IIa and GP Ib-IX-V, respectively).²⁷ This initial adhesion triggers intracellular signaling pathways that lead to platelet activation, characterized by structural shape changes and the degranulation of alpha and dense storage bodies.²⁸ These granules release potent autocrine and paracrine amplifiers, including adenosine diphosphate (ADP), serotonin, and thromboxane A₂ (TxA₂), which recruit additional circulating platelets to the site of vascular injury.²⁹

The final common pathway of platelet aggregation involves the conformational activation of the integrin $\alpha_{IIb}\beta_3$ (GP IIb/IIIa) receptor, which binds circulating fibrinogen and vWF molecules to form a stable macro-thrombus.³⁰ A higher circulating mass of platelets at the time of arterial occlusion enhances the kinetics of this aggregation process. This increased mass promotes the formation of larger, more stable, and densely packed occlusive thrombi that are highly resistant to endogenous fibrinolysis.³¹ Moreover, activated platelets actively engage in cross-talk with circulating leukocytes. The surface expression of P-selectin on activated platelets binds to P-selectin glycoprotein ligand-1 (PSGL-1) on neutrophils and monocytes, forming platelet-leukocyte aggregates (PLAs).³² These aggregates further stimulate the

release of pro-inflammatory cytokines and tissue factor, linking thrombosis directly to the neuroinflammatory cascade.³³

This finding supports the clinical work of Amalia and Dalimonthe, who demonstrated that altered platelet-to-white blood cell ratios (PWR) are strongly tied to ischemic stroke severity and can differentiate between atherothrombotic and cardioembolic stroke mechanisms.³⁴ While some literature, such as Xu et al., notes that severe thrombocytopenia may complicate stroke presentations by increasing spontaneous micro-hemorrhage risks, our study confirms that within an unselected acute sample, higher absolute numbers of platelets track with greater upfront neurological deficits due to their primary role in thrombo-occlusion.³⁵

One of our most striking findings is that baseline neurological severity, as measured by the NIHSS, appears entirely independent of admission serum electrolyte levels (sodium, potassium, and chloride). This lack of initial statistical correlation presents an interesting divergence from several classic clinical reports that frequently link acute dysnatremia, dyskalemia, or dyschloremia with poorer overall clinical outcomes and elevated mortality rates in stroke units.³⁶ However, this discrepancy can be resolved by separating the indicators of initial neurological severity from subacute prognostic indicators.

Serum sodium concentration is primarily a reflection of systemic water balance rather than localized cerebral perfusion architecture. While severe hyponatremia is known to induce cellular brain swelling via osmotic shifts, and hypernatremia can induce systemic hyperosmolality, these shifts typically take time to develop or reflect chronic baseline pre-morbid volume states (e.g., secondary to chronic diuretic use or poor hydration).³⁷ The baseline NIHSS score recorded immediately upon emergency room admission primarily reflects the anatomic location of the primary vascular occlusion and the efficiency of the patient's intracranial collateral circle.³⁸ Therefore, a patient with a complete proximal middle cerebral artery (MCA) occlusion will present with a high NIHSS score due to immediate hemiparesis and aphasia, regardless of whether their serum sodium is precisely 136 mEq/L or 144 mEq/L at the moment of admission.

This finding is consistent with Liu et al., who noted that while electrolyte imbalances are common among acute stroke admissions, multivariable logistic regressions demonstrate that baseline electrolyte values fail to act as independent predictors of initial neurological scores when adjusted for age, history of hypertension, and vascular territory.³⁹ Similarly, Kazancı et al. observed that while early dysnatremia is a strong predictor of 30 day mortality in intensive care settings, it exhibits no meaningful linear correlation with baseline NIHSS scores upon admission.⁴⁰ This indicates that abnormal sodium values primarily impact survival by predisposing patients to secondary subacute complications, such as severe cerebral edema, cardiac arrhythmias, or central pontine myelinolysis, later during their hospital stay.⁴¹

A similar mechanism applies to our potassium and chloride findings. Although potassium is essential for maintaining cardiac rhythm and neuromuscular transmembrane potentials, peripheral serum potassium measurements do not accurately reflect localized extracellular fluid shifts within the ischemic brain parenchyma.⁴¹ In the hyperacute phase, localized intracellular potassium efflux occurs rapidly within the core area due to the failure of the Na^+ , K^+ -ATPase pump.⁴³ However, this localized extracellular accumulation is well isolated by the surrounding architecture and does not immediately affect systemic

peripheral vein samples.⁴⁴ Our findings match Koren et al., who reported that systemic potassium values upon admission do not correlate with upfront NIHSS scales, even though they remain predictive of long-term cardiac safety profiles.⁴⁵

Similarly, serum chloride did not demonstrate a significant correlation in our study. This is notable given recent studies by Wang et al. and Haller et al. linking hyperchloremia to acute kidney injury (AKI) and poor stroke recovery.^{46–47} Our data indicate that while chloride abnormalities can reflect severe metabolic acidosis or heavy fluid resuscitation, they do not directly dictate immediate neurovascular deficits upon presentation.

In conclusion, elevated admission leukocyte and platelet counts were significantly associated with greater stroke severity as measured by the NIHSS. Both biomarkers demonstrated weak positive correlations with neurological impairment, suggesting their potential utility as readily available indicators for early clinical risk stratification. In contrast, serum sodium, potassium, and chloride levels were not significantly associated with NIHSS score at admission. These findings indicate that inflammatory and hematologic responses may play a more prominent role in the initial neurological severity of ischemic stroke than electrolyte disturbances. Further prospective studies incorporating serial laboratory measurements and multivariable analyses are warranted to clarify the prognostic value of these biomarkers and their relationship with long-term clinical outcomes.

Several limitations should be considered when interpreting the findings of this study. First, the retrospective cross-sectional design precludes the establishment of causal relationships between hematological or electrolyte parameters and ischemic stroke severity. Second, the study was conducted at a single tertiary referral center with a relatively limited sample size, which may restrict the generalizability of the findings to broader populations. Third, laboratory measurements were obtained only at hospital admission, preventing evaluation of temporal changes in inflammatory markers and electrolyte status during the acute and recovery phases of stroke. In addition, potential confounding factors, including infarct volume, stroke subtype, comorbid conditions, medication use, and acute treatment strategies, were not comprehensively assessed and may have influenced the observed associations.

Future investigations should employ prospective multicenter designs with larger and more diverse patient populations to improve external validity. Serial assessments of leukocyte count, platelet count, and electrolyte levels throughout hospitalization may provide further insight into their dynamic relationship with neurological deterioration and recovery. Moreover, incorporating neuroimaging characteristics, vascular risk profiles, and functional outcome measures such as the modified Rankin Scale would help clarify the prognostic significance of these biomarkers. Such studies may contribute to the identification of reliable, readily accessible biomarkers for risk stratification and individualized management of patients with acute ischemic stroke.

Declarations

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DISCLOSURE

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