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Serum Inflammatory Cytokines (IL-6, TNF-α, and IL-1β) as Predictors of Disease Severity in Acute Ischemic Stroke Patients: A Clinical Study

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ABSTRACT

Background: Acute ischemic stroke (AIS) is a major cause of mortality and long-term disability worldwide. Inflammation plays a critical role in the pathogenesis and progression of cerebral ischemic injury. Following arterial occlusion, several pro-inflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β), are released and may reflect the severity of neuronal damage.

Objective: To evaluate the relationship between serum IL-6, TNF-α, and IL-1β levels and clinical severity in patients with acute ischemic stroke.

Methods: This cross-sectional study was conducted at the Department of Medicine, Nishtar Medical University, Multan, from June 2024 to June 2025. A total of 100 patients with acute ischemic stroke confirmed by CT or MRI within 24 hours of symptom onset were included. Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS). Serum IL-6, TNF-α, and IL-1β levels were measured using enzyme-linked immunosorbent assay (ELISA). Statistical analysis included one-way ANOVA, Pearson correlation, and multivariate logistic regression.

Results: Patients with severe stroke had significantly higher mean serum levels of IL-6, TNF-α, and IL-1β compared to mild and moderate stroke groups ($p < 0.001$). IL-6 showed the strongest positive correlation with NIHSS score ($r = 0.68$), followed by TNF-α ($r = 0.61$) and IL-1β ($r = 0.55$). Multivariate analysis identified IL-6 (AOR = 3.2), TNF-α (AOR = 2.8), and IL-1β (AOR = 2.3) as independent predictors of severe stroke.

Conclusion: Elevated inflammatory cytokines are significantly associated with AIS severity and may aid in early prognostic assessment.

Keywords: Acute ischemic stroke, IL-6, TNF-α, IL-1β, inflammation, NIHSS, biomarkers

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INTRODUCTION

Acute ischemic stroke (AIS) is one of the most common causes of death and long-term disability across the world, as well as a significant burden on the health care system, especially in low- and middle-income nations [1]. It happens because cerebral arteries suddenly become blocked, causing blood stoppage, oxygen deficiency, and consequent damage to neurons. Even with improvements in reperfusion interventions, including thrombolysis and mechanical thrombectomy, a significant percentage of patients have poor neurological outcomes. A primary concern in the clinical treatment of stroke is the need to identify the severity of the disease and prognostic indicators as early as possible [2,3].

Over the last few years, there has been an increasing body of evidence on the central role of inflammation in the pathophysiology of acute ischemic stroke [4]. The repercussions that occur following cerebral ischemia are multifaceted and involve proliferation of resident microglial cells, peripheral immune cell recruitment, and pro-

inflammatory mediators. Among them, interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β) are critical regulators of the inflammatory response and contribute substantially to neuronal destruction [5].

One of the first cytokines to be released following ischemic injury is IL-1β, which is considered to facilitate level I enhanced inflammatory signaling, increase leukocyte infiltration, and amplify neuronal apoptosis [6]. TNF-α is involved in endothelial dysfunction, blood–brain barrier disruption, and elevated oxidative stress, thus worsening ischemic damage. The pleiotropic cytokine IL-6 has been extensively examined regarding its duality in pro-inflammatory and anti-inflammatory activities, but high levels of IL-6 during acute stroke have been repeatedly linked to a larger infarct size, worse neurological outcome, and poor functional recovery [7].

It has been demonstrated that the severity of stroke and clinical outcome relate to the magnitude of the inflammatory response. This is due to elevated levels of IL-6, TNF-α, and IL-1β in the circulation within the first 24 hours after the onset of stroke, reflecting tissue damage and subsequent inflammatory injury [8]. These biomarkers have thus continued to attract more focus as those that may be used to stratify risk early, predict prognosis, and target therapy. In comparison to neuroimaging methodologies, which mainly give structural data, biochemical markers can reveal more about ongoing pathophysiological processes and can be assessed more dynamically during disease progression [9].

Though some international studies have shown that the relationship between inflammatory cytokines and stroke severity is evident, there is still a lack of evidence in South Asian populations, specifically in Pakistan, where rates of hypertension, diabetes, and physical inactivity are increasing, leading to a higher burden of stroke cases. To support the clinical utility of these biomarkers, population-specific studies are required to control genetic, environmental, and healthcare-related variability [10].

Thus, the study aimed to determine whether serum IL-6, TNF-α, and IL-1β could be used as predictors of disease severity in acute ischemic stroke patients. This study attempts to present evidence for integrating inflammatory biomarkers into standard clinical practice and prognostic assessment of stroke patients by establishing their correlation with clinical severity scores.

MATERIALS AND METHODS

This analytical cross-sectional study was conducted in the Department of Medicine, Nishtar Medical University and Hospital, Multan, Pakistan. The study was completed over a 12-month period extending from June 2024 to June 2025. It was designed to examine whether circulating inflammatory mediators, specifically interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β), were associated with the neurological severity of acute ischemic stroke (AIS) at the time of presentation.

A total of 100 patients with a confirmed diagnosis of acute ischemic stroke were enrolled. Recruitment was carried out through consecutive non-probability sampling, and eligible cases were selected from patients presenting to the relevant clinical unit during the defined study interval. Only adult patients aged 18 years and older, regardless of sex, were eligible. Inclusion was restricted to those presenting within 24 hours of onset of neurological symptoms and having radiological confirmation of ischemic stroke by either computed tomography (CT) or magnetic resonance imaging (MRI) of the brain.

Several exclusion criteria were used to reduce measurement distortion of inflammatory biomarkers. Patients were not enrolled if they had hemorrhagic stroke, transient ischemic attack, chronic inflammatory diseases, autoimmune disorders, ongoing infections, malignancy, recent trauma or operative intervention, chronic hepatic disease, renal insufficiency, or a history of immunosuppressive drug use. Cases with refusal to participate or incomplete required data were also omitted from the final sample.

After obtaining written informed consent, each participant underwent detailed clinical assessment at admission. Data collection was performed using a standardized structured proforma developed for the study. The recorded variables included age, sex, smoking history, presence of hypertension, diabetes mellitus, interval since symptom onset, and other clinically relevant background information. Neurological impairment was quantified using the National Institutes of Health Stroke Scale (NIHSS), a recognized and validated scoring system for stroke-related neurological dysfunction. Patients were grouped into three categories based on NIHSS score: mild stroke (≤5), moderate stroke (6–14), and severe stroke (≥15).

For biochemical evaluation, venous blood was obtained from all participants within the first 24 hours after hospital admission using sterile aseptic technique. Approximately 5 mL of venous blood was collected into plain sterile tubes. After allowing adequate clotting time, the samples were centrifuged at 3000 rpm for 10 minutes for serum separation. The isolated serum was then transferred into labeled aliquots and stored at −80 °C until laboratory testing was performed.

Serum levels of IL-6, TNF-α, and IL-1β were quantified using commercial enzyme-linked immunosorbent assay (ELISA) kits according to the procedures provided by the respective manufacturers. All laboratory analyses were completed under controlled and standardized conditions. To improve analytical precision and reproducibility, each sample was tested in duplicate, and the mean value of both readings was used in the final analysis.

The main outcome of the study was to determine the relationship between serum inflammatory cytokine

concentrations and the severity of acute ischemic stroke as assessed by NIHSS score. In addition, the study aimed to determine whether these biomarkers could function as independent indicators of severe stroke after controlling for major clinical confounders.

All study data were coded and entered into Statistical Package for Social Sciences (SPSS), version 26.0, for statistical processing. Continuous variables, including age and cytokine concentrations, were summarized as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. Differences in mean cytokine concentrations among the defined stroke severity groups were examined using one-way analysis of variance (ANOVA). The degree of association between inflammatory marker levels and NIHSS scores was assessed by Pearson correlation analysis. Furthermore, multivariable logistic regression analysis was employed to evaluate whether IL-6, TNF-α, and IL-1β independently predicted severe stroke after adjustment for potential confounders such as age, sex, hypertension, and diabetes mellitus. The p-value of less than 0.05 was regarded as statistically significant.

The Institutional Ethical Review Board of Nishtar Medical University, Multan, approved the study under the reference number 9478/NMU. The study was carried out in complete compliance with the principles established by the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment, and confidentiality of all patient-related information was preserved throughout the study period.

RESULTS

The study involved 100 patients with acute ischemic stroke. The average age of patients was 61.3 ± 10.5 years, and most of the participants were males (62%). Most of the patients had pre-existing vascular risk factors, 68% were hypertensive, and 52% had diabetes mellitus, with 34% of them being active smokers. According to the NIHSS scores during admission, 28 percent of patients were classified as mildly stroked, 42 percent as moderate, and 30 percent as severely stroked. These results denote that a considerable percentage of patients were presented with moderate to severe neurological impairment, which indicates a delayed presentation and severity of burden of disease in the investigated group (Table 1).

Comparison of the serum cytokine levels of the various stroke severity groups showed that there was a significant statistical rise in inflammatory markers with an increase in the severity of a stroke. The patients who suffered severe stroke exhibited significantly higher levels of IL-6 (41.2 ± 7.3 pg/mL), TNF-α (39.5 ± 8.1 pg/mL), and IL-1β (29.4 ± 6.5 pg/mL) than moderate and mild stroke cases. The same case could be said of the patients in the moderate category, as their cytokine levels were also significantly higher than those of the mild category. The disparities between all groups were very significant ($p < 0.001$), which means that there is a strong correlation between the extent of inflammatory response and the extent of neurological severity (Table 2).

Subsequent statistics showed that there was a positive correlation between serum cytokine levels and admission NIHSS scores. The correlation coefficient (r) of IL-6 was 0.68 with TNF-α ($r = 0.61$) and IL-1β ($r = 0.55$) following it, and the correlations are statistically significant ($p < 0.001$). This implies that elevated concentrations of inflammatory cytokines are positively linked with deterioration of neurological impairment and severity of stroke (Table 3).

The multivariate logistic regression analysis was carried out to determine the independent predictors of severe stroke after controlling for the confounding factors such as age, gender, hypertension, and diabetes mellitus. The result indicated that IL-6 (Adjusted Odds ratio (AOR) = 3.2, $p < 0.001$), TNF-α (AOR = 2.8, $p = 0.002$), and IL-1β (AOR = 2.3, $p = 0.004$) were all independent predictors of severe stroke. Among them, IL-6 proved to be the most predictive of them, which means that it is more efficient in prognostic testing of the severity of the disease in patients with acute ischemic stroke (Table 4).

Comprehensively, the findings indicate clearly that high serum concentrations of IL-6, TNF-α, and IL-1β are strongly correlated with higher levels of stroke severity, with IL-6 exhibiting the best correlation and predictive ability. These results demonstrate the possible clinical value of the biomarkers of inflammatory cytokines as clinical markers of early risk stratification and severity in acute ischemic stroke.

Table 1: Baseline Demographic and Clinical Characteristics of Patients (n = 100)

Variable	Value
Age (years)	61.3 ± 10.5
Male	62 (62%)
Female	38 (38%)
Hypertension	68 (68%)
Diabetes Mellitus	52 (52%)
Smokers	34 (34%)
Mild Stroke (NIHSS ≤5)	28 (28%)
Moderate Stroke (NIHSS 6–14)	42 (42%)
Severe Stroke (NIHSS ≥15)	30 (30%)

Table 2: Comparison of Serum Cytokine Levels Across Stroke Severity Groups

Cytokine	Mild (n=28)	Moderate (n=42)	Severe (n=30)	p-value
IL-6 (pg/mL)	12.5 ± 3.2	24.8 ± 5.6	41.2 ± 7.3	<0.001
TNF-α (pg/mL)	15.3 ± 4.1	27.6 ± 6.2	39.5 ± 8.1	<0.001
IL-1β (pg/mL)	10.2 ± 2.9	18.7 ± 4.3	29.4 ± 6.5	<0.001

Table 3: Correlation Between Cytokine Levels and NIHSS Score

Cytokine	Correlation Coefficient (r)	p-value
IL-6	0.68	<0.001
TNF-α	0.61	<0.001
IL-1β	0.55	<0.001

Table 4: Multivariate Logistic Regression Analysis for Predictors of Severe Stroke

Variable	Adjusted Odds Ratio (AOR)	p-value
IL-6	3.2	<0.001
TNF-α	2.8	0.002
IL-1β	2.3	0.004
Age	1.2	0.08
Hypertension	1.4	0.12
Diabetes Mellitus	1.3	0.15

DISCUSSION

The current study reveals that there is a significant and clinically relevant relationship between high levels of serum inflammatory cytokines (IL-6, TNF-α) and the severity of the illness in patients with acute ischemic stroke [11]. The results reveal that patients who came with increased NIHSS scores had significantly increased levels of these cytokines, confirming the hypothesis that the scales of the inflammatory response are directly linked with the degree of neuronal damage and the neurological disability [12].

The onset of acute ischemic stroke triggers a complicated sequence of neuroinflammatory processes that play a role in primary and secondary brain damage [13]. The release of pro-inflammatory cytokines occurs as a result of activation of microglial cells and recruitment of peripheral immune cells, which is a consequence of ischemia and aggravates tissue damage by means of oxidative stress, excitotoxicity, and disruption of the blood-brain barrier [14]. The IL-6 was the most correlated cytokine in this study with the stroke severity, with the highest correlation coefficient with the NIHSS score. This finding aligns with past studies that have shown that the levels of IL-6 dramatically increase after the onset of stroke and correlate well with the size of the infarcts, neurological impairments, and adverse clinical outcomes. The high predictive capacity of IL-6 in the multivariate analysis also helps to support the idea that IL-6 is a major mediator of the post-ischemic inflammation [15].

On the same note, higher levels of TNF-α were highly present in patients with the more severe stroke. TNF-α has been noted to be at the center-stage of endothelial dysfunction and facilitates adhesion and infiltration of leukocytes into ischemic brain tissue [16,17]. It is also involved in apoptotic processes and enhances the destruction of neurons. The results of this paper are consistent with previous data that a higher-than-usual level of TNF-α is linked to poorer neurological impairment and poor functional recovery. The other important pro-inflammatory cytokine IL-1β was also identified to have significant relationships with stroke severity. One of the most immediate cytokines emitted following the ischemic trauma is IL-1β, which plays a role in the intensification of the inflammatory cascade, which in turn leads to the additional damage of neurons and the development of edema [18].

This study demonstrated a dose-dependent relationship between inflammation and disease severity by the gradual rise in the level of cytokines among mild to severe stroke populations. This observation indicates that the inflammatory cytokines not only respond to the degree of brain damage but can also be involved in brain damage. Consequently, the biomarker measurements in the first hours after the stroke indicate a beneficial understanding of the pathophysiological mechanisms and may be used to perform the preliminary risk stratification [19,20].

The fact that several cytokines have been tested together, as opposed to using one biomarker, is also an advantage of this study. The combination of the evaluation of IL-6, TNF-α, and IL-1β will offer a better insight into the inflammatory environment in acute ischemic stroke and will increase the predictability of disease severity. Clinically, this practice can help in making superior decisions in relation to patient surveillance, treatment interventions, and prognostication [1,3].

Nevertheless, some weaknesses should be admitted. This was a one-center study that had a fairly small sample size, which could restrict the generalizability of the results [5-7]. Also, the measure of cytokines was done on a single time point during 24 hours of admission; serial measurements may further show how the changes of inflammation change with time and how it is correlated with the outcome. In addition, there was no long-term functional outcome measurement, and this would have been useful in determining the prognostic score of these biomarkers beyond the degree of severity [13,17].

Further studies in the future are supposed to be conducted on large-scale studies, involving multicentric

studies, having longitudinal follow-ups, and serial cytokine observations. The possible opportunity of anti-inflammatory therapeutic measures aimed at these cytokines is also worth researching, because the adjustment of the inflammatory reaction can become a promising way of successful treatment of acute ischemic stroke [18-20].

CONCLUSION

This study shows that the serum levels of IL-6, TNF-α, and IL-1β are highly elevated in the case of acute ischemic stroke and are closely related to the severity of the disease as measured by the NIHSS score. The best predictive biomarker of severe stroke is IL-6. The results indicate that inflammatory cytokines might be an effective medium of early warning of the severity of the stroke and can be a useful tool in clinical risk stratification and management. Cytokine profiling should be included in routine evaluation as it could improve prognostic performance and promote more tailored stroke care.

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Authors' Contributions: M.R.M. contributed to conceptualization, study design, and manuscript drafting. S.S. was responsible for data collection, patient assessment, and data acquisition. H.T. performed data analysis, interpretation of results, and manuscript editing, and also contributed to supervision and critical revision of the manuscript. All authors approved the final version of the manuscript.

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Data Availability: The datasets generated and analyzed during this study are available from the corresponding author upon reasonable request.

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