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Neurovascular Dynamics and Hemodynamic Instability in Acute Ischemic Stroke: A Clinical and Biochemical Investigation

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ABSTRACT

Cerebrovascular diseases, particularly acute ischemic stroke (AIS), represent a major public health concern due to their high morbidity and mortality. This study investigates the neurovascular and hemodynamic parameters in AIS patients, evaluating their correlation with functional outcomes and infarct size. A prospective observational approach was employed to measure cerebral blood flow (CBF), systemic inflammatory markers, and neurochemical biomarkers within 72 hours of symptom onset. Findings indicated that early cerebral hypoperfusion and elevated C-reactive protein (CRP) levels were significantly associated with poor neurological recovery. These results underscore the importance of early neurovascular assessment in guiding stroke management and improving patient prognosis.

KEYWORDS:

Cerebrovascular disease, ischemic stroke, cerebral blood flow, hemodynamics, biomarkers, CRP, neurological outcome

INTRODUCTION:

Cerebrovascular diseases, primarily ischemic and hemorrhagic strokes, account for significant neurological disability and mortality worldwide. Among them, acute ischemic stroke (AIS) constitutes approximately 85% of all cases. Despite advancements in reperfusion therapies, including intravenous thrombolysis and mechanical thrombectomy, many patients continue to experience adverse outcomes due to delayed diagnosis and limited understanding of early cerebrovascular dynamics.

Emerging evidence highlights the critical role of cerebral perfusion and systemic inflammation in influencing stroke severity and recovery. Reduced cerebral blood flow (CBF) following arterial occlusion initiates a cascade of metabolic and neurochemical changes, including excitotoxicity, oxidative stress, and blood-brain barrier (BBB) disruption. Concurrently, inflammatory markers such as CRP and interleukins rise systemically, further aggravating neuronal damage.

The objective of this study was to analyze the interplay between neurovascular parameters and systemic inflammatory responses in the early stages of AIS. We hypothesized that early identification of hemodynamic compromise and biochemical abnormalities could predict long-term neurological outcomes

MATERIALS AND METHODS

Study Design and Population

A prospective observational study was conducted between March 2022 and February 2023 at three tertiary care hospitals. A total of 124 adult patients (aged 45–82 years) diagnosed with first-ever AIS confirmed by neuroimaging (MRI or CT) were included. Exclusion criteria included prior stroke, hemorrhagic stroke, active infection, or comorbid neurodegenerative diseases.

Data Collection

All participants underwent detailed clinical assessment, including the National Institutes of Health Stroke Scale (NIHSS) at admission and modified Rankin Scale (mRS) at 90 days post-stroke. Blood samples were drawn within 24 hours of admission to assess CRP, interleukin-6 (IL-6), and neuron-specific enolase (NSE). CBF was measured using transcranial Doppler ultrasonography and CT perfusion.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation. Correlation between CBF, CRP, and functional outcome (mRS) was evaluated using Pearson's correlation

coefficient. Multivariate logistic regression was employed to identify predictors of poor outcome (mRS ≥ 3). A p-value < 0.05 was considered statistically significant.

RESULTS

Among the 124 patients, 78 were male and 46 female. The mean age was 65.4 ± 9.1 years. The majority of strokes involved the middle cerebral artery territory. Reduced mean CBF (<30 mL/100g/min) was observed in 69% of participants. Elevated CRP (>5 mg/L) and IL-6 levels were significantly associated with larger infarct volume and higher NIHSS scores on admission. Statistical analysis revealed a moderate negative correlation between CBF and mRS at 90 days ($r = -0.48$, $p < 0.001$), and a positive correlation between CRP levels and poor outcome ($r = 0.52$, $p < 0.001$). Multivariate analysis showed that low CBF (OR: 3.21, 95% CI: 1.78–5.81) and high CRP (OR: 2.76, 95% CI: 1.49–5.09) were independent predictors of unfavorable recovery.

DISCUSSION

The present study reinforces the hypothesis that early neurovascular and inflammatory markers play a pivotal role in the prognosis of acute ischemic stroke. Cerebral blood flow, as a dynamic parameter reflecting the extent of perfusion deficit, closely correlates with neurological impairment and infarct size. The observed inverse relationship between CBF and mRS suggests that restoration of perfusion within the first critical hours may mitigate long-term disability.

CRP, an acute-phase reactant, has been increasingly recognized as a surrogate marker of neuroinflammation. Elevated levels reflect systemic immune activation, which exacerbates ischemic injury via endothelial dysfunction, leukocyte adhesion, and increased BBB permeability. Our findings are consistent with prior studies that link high CRP with stroke recurrence and mortality.

Furthermore, the integration of hemodynamic and biochemical profiles may offer a more holistic approach to risk stratification. Future studies should consider longitudinal tracking of these markers and their responsiveness to intervention, such as anti-inflammatory agents or neuroprotective therapies.

CONCLUSION

This study demonstrates that reduced cerebral blood flow and elevated CRP levels in the acute phase of ischemic stroke are significant predictors of poor functional outcomes. Early detection of neurovascular compromise and systemic inflammation could enhance risk assessment and guide targeted therapeutic strategies. Incorporating these biomarkers into clinical practice may improve recovery trajectories and reduce stroke-related burden.

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