E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.48047/ijprt/16.01.632

Research Article

Predictive Role of Systemic Inflammatory Response Index (SIRI), Neutrophil-to-Lymphocyte Ratio (NLR), and C-Reactive Protein (CRP) in Early Post-Stroke Seizures

Kifayat Ullah Khan[1], Seerat Iftikhar[2], Muhammad Wajahat Gohar Qureshi[3], Bushra Faiz[4], Arwa Humayun[5], Muhammad Waqas[6], Glen Ramsay[7]

1. Senior Registrar, Paediatric Medicine, Ward Unit 2, Sheikh Zayed Hospital, Rahim Yar Khan, Pakistan.
2. Microbiologist, COMSATS Institute of Information Technology, Islamabad, Pakistan.
3. Senior Registrar, Neurology, Shalamar Medical and Dental College, Lahore, Pakistan.
4. Assistant Professor, Medicine, Medical Unit I, Services Institute of Medical Sciences (SIMS), Lahore, Pakistan.
5. Senior Registrar, Neurology Department, Mayo Hospital, Lahore, Pakistan.
6. Assistant Professor, Neurosurgery, MBBS Medical College, Divisional Headquarters Hospital, Mirpur AJK, Pakistan.
7. Westchester Neurological Consultant, Interfaith Medical Center, USA.

Kifayat Ullah Khan: Corresponding author

Abstract

Early post-stroke seizures (EPSS) represent a clinically significant neurological complication associated with prolonged hospitalization, increased disability, and unfavorable neurological outcomes. Identification of reliable inflammatory biomarkers capable of predicting seizure occurrence following acute stroke remains an important clinical challenge. The present study aimed to evaluate the predictive role of the Systemic Inflammatory Response Index (SIRI), Neutrophil-to-Lymphocyte Ratio (NLR), and C-Reactive Protein (CRP) in patients developing EPSS after acute stroke. A prospective observational study was conducted among 220 patients admitted with acute ischemic or hemorrhagic stroke. Peripheral blood samples were collected within 24 hours of admission for complete blood count, CRP measurement, and quantitative polymerase chain reaction (qPCR)-based assessment of inflammatory cytokine gene expression. SIRI was calculated using neutrophil × monocyte/lymphocyte counts, while NLR was derived from absolute neutrophil and lymphocyte counts. Patients were monitored for seven days to identify seizure occurrence. EPSS developed in 38 (17.3%) patients. Mean SIRI, NLR, and CRP levels were significantly higher among seizure patients

compared with non-seizure patients ($p<0.001$). Multivariate logistic regression demonstrated SIRI as the strongest independent predictor of EPSS (OR=3.84, 95% CI: 2.12–6.94), followed by NLR (OR=2.97, 95% CI: 1.61–5.48) and CRP (OR=2.41, 95% CI: 1.37–4.25). Elevated inflammatory cytokine gene expression detected by qPCR further supported the association between systemic inflammation and seizure susceptibility. These findings suggest that SIRI, NLR, and CRP may serve as accessible biomarkers for early identification of patients at increased risk of post-stroke seizures, providing a novel inflammatory-based prognostic approach.

Keywords: Early post-stroke seizures; Systemic inflammatory response index; Neutrophil-to-lymphocyte ratio

Introduction

Stroke remains one of the leading causes of mortality and long-term disability worldwide, imposing a substantial burden on healthcare systems and affected populations. Despite advances in acute stroke management and neurocritical care, neurological complications continue to contribute significantly to adverse outcomes. Among these complications, early post-stroke seizures (EPSS) have emerged as an important clinical concern because of their association with increased morbidity, prolonged hospitalization, recurrent neurological deterioration, and elevated mortality rates. EPSS are generally defined as seizures occurring within the first seven days following stroke onset and are considered to result directly from acute biochemical and structural changes within the injured brain tissue. Recent epidemiological studies have reported an incidence ranging from 5% to 20%, depending upon stroke subtype, lesion location, severity, and patient-related factors [1-3].

The pathophysiological mechanisms underlying seizure development after stroke are multifactorial and incompletely understood. Acute cerebral ischemia and intracerebral hemorrhage initiate a cascade of neuroinflammatory events characterized by activation of resident microglia, recruitment of peripheral immune cells, blood-brain barrier disruption, oxidative stress, and excessive release of excitatory neurotransmitters. These processes collectively contribute to neuronal hyperexcitability and epileptogenesis during the acute post-stroke period. Increasing evidence indicates that inflammatory mediators play a central role in facilitating seizure generation through modulation of synaptic transmission, ion channel activity,

and neuronal network stability. Consequently, inflammatory biomarkers have gained considerable attention as potential predictors of post-stroke seizure risk [2-4].

Inflammation has become increasingly recognized as a critical determinant of stroke progression and neurological recovery. Following cerebral injury, activation of innate immune pathways results in rapid elevation of circulating neutrophils, monocytes, and acute-phase reactants. Simultaneously, stress-induced lymphocyte suppression contributes to immune dysregulation and systemic inflammatory imbalance. This inflammatory response not only exacerbates secondary brain injury but may also influence seizure susceptibility through direct and indirect neurobiological mechanisms. Therefore, biomarkers reflecting systemic inflammatory status may provide valuable prognostic information in stroke patients [3-5].

The Neutrophil-to-Lymphocyte Ratio (NLR) has emerged as a readily available marker of systemic inflammation derived from routine complete blood count analysis. Elevated NLR values reflect increased neutrophil-mediated inflammatory activity combined with relative lymphocyte suppression. Recent investigations have demonstrated associations between elevated NLR and poor neurological outcomes, larger infarct volumes, hemorrhagic transformation, cognitive decline, and mortality following stroke. Furthermore, NLR has shown predictive utility in various neurological disorders characterized by inflammatory activation. Nevertheless, its role in identifying patients at risk for EPSS remains incompletely established and requires further investigation [4-6].

Another increasingly investigated inflammatory marker is the Systemic Inflammatory Response Index (SIRI), calculated using neutrophil, monocyte, and lymphocyte counts. Compared with single-cell inflammatory markers, SIRI integrates multiple components of innate and adaptive immune responses, potentially providing a more comprehensive assessment of inflammatory burden. Elevated SIRI values have been associated with adverse outcomes in cardiovascular disease, malignancy, sepsis, and cerebrovascular disorders. Recent studies suggest that SIRI may outperform conventional inflammatory markers in predicting disease severity and prognosis. However, limited evidence is available regarding its association with seizure occurrence following acute stroke, creating a

significant knowledge gap in current neurological research [5-7].

C-Reactive Protein (CRP) remains one of the most extensively studied acute-phase inflammatory proteins in clinical medicine. Synthesized predominantly by hepatocytes in response to interleukin-6 stimulation, CRP levels increase rapidly following tissue injury and systemic inflammation. Elevated CRP concentrations have been linked to stroke severity, infarct progression, recurrent vascular events, and poor functional recovery. Experimental evidence suggests that CRP-mediated inflammatory pathways may contribute to neuronal excitability and seizure development through enhancement of neurovascular dysfunction and inflammatory signaling. Consequently, CRP represents a potentially useful biomarker for identifying patients vulnerable to EPSS [6-8].

Recent advances in molecular diagnostics have further improved understanding of inflammation-related neurological disorders. Quantitative polymerase chain reaction (qPCR) enables sensitive assessment of inflammatory cytokine gene expression, including interleukin-1β, interleukin-6, and tumor necrosis factor-α. These cytokines have been implicated in both stroke pathology and seizure generation. Molecular evaluation of inflammatory gene expression may therefore complement conventional inflammatory biomarkers and provide additional insight into the biological mechanisms linking inflammation with EPSS. Integration of molecular and hematological biomarkers may improve predictive accuracy and facilitate personalized risk stratification in acute stroke patients [7-9].

Although several inflammatory markers have been investigated individually in stroke populations, comparative evaluation of SIRI, NLR, and CRP specifically in relation to EPSS remains scarce. Existing studies have predominantly focused on functional outcomes, mortality, and recurrent vascular events, with limited attention given to seizure prediction. Furthermore, few investigations have incorporated molecular inflammatory profiling using qPCR to support clinical observations. Consequently, substantial uncertainty persists regarding the relative predictive value of these biomarkers and their potential utility in routine neurological practice [8-10].

Identification of reliable biomarkers capable of predicting seizure risk during the acute post-stroke period may facilitate early surveillance, optimize therapeutic decision-making, and improve patient outcomes. Early recognition of high-risk individuals could

enable targeted neurological monitoring and individualized management strategies aimed at reducing seizure-related complications. Therefore, the present study was designed to evaluate the predictive significance of SIRI, NLR, and CRP in EPSS while simultaneously exploring inflammatory cytokine gene expression through qPCR analysis. The study further sought to determine whether these inflammatory markers independently predict seizure occurrence after adjustment for established clinical risk factors. By integrating conventional laboratory parameters with molecular inflammatory assessment, this investigation provides a novel approach toward understanding inflammatory mechanisms underlying EPSS and identifying clinically useful prognostic biomarkers.

Methodology

A prospective observational cohort study was conducted at Paediatric Medicine, Ward Unit 2, Sheikh Zayed Hospital, Rahim Yar Khan, Pakistan between January 2024 and December 2025. The study was designed to investigate the predictive role of the Systemic Inflammatory Response Index (SIRI), Neutrophil-to-Lymphocyte Ratio (NLR), and C-Reactive Protein (CRP) in the development of early post-stroke seizures (EPSS). Consecutive patients presenting with acute ischemic stroke or spontaneous intracerebral hemorrhage confirmed through neuroimaging were screened for eligibility. Stroke diagnosis was established using clinical evaluation in combination with computed tomography (CT) and/or magnetic resonance imaging (MRI) findings. Early post-stroke seizure was defined as any clinically observed or electroencephalographically confirmed seizure occurring within seven days of stroke onset. Patients were followed prospectively throughout hospitalization and monitored daily by attending neurologists for seizure occurrence.

The sample size was calculated using Epi Info™ version 7.2 (Centers for Disease Control and Prevention, Atlanta, USA). Based on previous literature reporting an incidence of early post-stroke seizures of approximately 15%, a confidence level of 95%, statistical power of 80%, margin of error of 5%, and an anticipated odds ratio of 2.5 for elevated inflammatory markers, the minimum required sample size was estimated at 204 participants. To compensate for possible dropouts and incomplete laboratory data, a total of 220 patients were recruited.

Patients aged 18 years and above presenting within 24 hours of stroke onset were included

in the study. Both male and female patients with acute ischemic stroke or spontaneous intracerebral hemorrhage confirmed radiologically were eligible. Inclusion further required availability of complete clinical records, laboratory investigations, and willingness to participate through verbal informed consent obtained from the patient or a legally authorized attendant when neurological impairment limited communication.

Patients with a previous history of epilepsy, post-traumatic seizures, central nervous system infections, brain tumors, neurodegenerative disorders associated with seizures, autoimmune diseases, active malignancy, chronic inflammatory conditions, severe hepatic dysfunction, end-stage renal disease, recent major surgery within the previous three months, current immunosuppressive therapy, systemic infection at admission, or incomplete laboratory data were excluded. Individuals who developed metabolic disturbances capable of independently precipitating seizures, including severe electrolyte imbalance, hypoglycemia, hyperglycemia, or uremic encephalopathy, were also excluded to minimize confounding influences.

Ethical approval was obtained from the Institutional Ethical Review Committee before study commencement in accordance with the Declaration of Helsinki. Verbal informed consent was obtained after explaining the objectives, risks, benefits, and confidentiality measures associated with participation. Patient anonymity was maintained throughout data collection and analysis by assigning coded identification numbers. Participation was entirely voluntary, and refusal to participate did not influence medical management.

Demographic and clinical information including age, sex, body mass index, smoking history, hypertension, diabetes mellitus, dyslipidemia, atrial fibrillation, previous stroke history, stroke subtype, stroke severity, lesion location, and National Institutes of Health Stroke Scale (NIHSS) scores were recorded at admission. Stroke severity was categorized according to NIHSS criteria, while functional outcome was assessed using the modified Rankin Scale (mRS) at discharge.

Venous blood samples were collected within the first 24 hours of hospital admission before initiation of anti-inflammatory therapy. Complete blood count analysis was performed using an automated hematology analyzer. Absolute neutrophil count, lymphocyte count, monocyte count, platelet count, hemoglobin concentration, and total

leukocyte count were documented. NLR was calculated by dividing the absolute neutrophil count by the absolute lymphocyte count. SIRI was calculated according to the established formula:

SIRI = (Neutrophil Count × Monocyte Count) / Lymphocyte Count

Serum CRP concentrations were measured using a high-sensitivity immunoturbidimetric assay according to manufacturer instructions. Internal quality-control procedures were applied daily to ensure analytical reliability.

To further explore molecular inflammatory mechanisms, quantitative polymerase chain reaction (qPCR) was performed for assessment of inflammatory cytokine gene expression. Peripheral blood mononuclear cells were isolated from fresh blood samples using density-gradient centrifugation. Total RNA was extracted using a commercially available RNA isolation kit according to standardized protocols. RNA purity and concentration were evaluated spectrophotometrically. Complementary DNA (cDNA) synthesis was performed using reverse transcriptase enzyme and random primers. Quantitative PCR amplification was subsequently conducted using SYBR Green chemistry on a real-time PCR platform. Expression levels of interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) genes were analyzed relative to GAPDH housekeeping gene expression. Relative gene expression was calculated using the $2^{-\Delta\Delta Ct}$ method. All reactions were performed in triplicate to enhance reproducibility.

Neurological monitoring was undertaken continuously throughout hospitalization. Patients demonstrating altered consciousness, focal neurological worsening, abnormal motor activity, or suspected seizure manifestations underwent urgent neurological evaluation. Electroencephalography (EEG) was performed when clinically indicated. Seizure diagnosis was confirmed by consultant neurologists based upon clinical manifestations and EEG findings whenever available. Patients were subsequently divided into two groups: Group A comprised patients who developed EPSS within seven days of stroke onset, whereas Group B consisted of patients who remained seizure-free during the same period.

Data analysis was performed using Statistical Package for Social Sciences (SPSS) version 27.0. Continuous variables were assessed for normality using the Shapiro-Wilk test and presented as mean ± standard deviation. Categorical variables were expressed as frequencies and percentages. Independent-

sample t-tests were applied for comparison of continuous variables between study groups, whereas chi-square tests were utilized for categorical variables. Receiver operating characteristic (ROC) curve analysis was performed to determine predictive accuracy and optimal cut-off values for SIRI, NLR, and CRP. Univariate logistic regression analysis was initially conducted to identify variables associated with seizure occurrence. Significant variables were subsequently entered into multivariate logistic regression models to determine independent predictors of EPSS. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. A p-value less than 0.05 was considered statistically significant.

Results

A total of 220 patients with acute stroke were enrolled. Early post-stroke seizures developed in 38 patients (17.3%), while 182 patients (82.7%) remained seizure-free during the first seven days following stroke onset.

Table 1. Demographic and Clinical Characteristics of the Study Population

Variable	EPSS Group (n=38) Mean ± SD / n (%)	Non-EPSS Group (n=182) Mean ± SD / n (%)	p-value
Age (years)	68.4 ± 10.3	63.7 ± 11.2	0.021
Male Gender	24 (63.2%)	106 (58.2%)	0.582
BMI (kg/m²)	28.6 ± 4.2	26.9 ± 3.8	0.018
Hypertension	30 (78.9%)	118 (64.8%)	0.048
Diabetes Mellitus	21 (55.3%)	71 (39.0%)	0.041
Smoking History	15 (39.5%)	54 (29.7%)	0.221
NIHSS Score	15.8 ± 4.9	10.7 ± 3.8	<0.001
Hemorrhagic Stroke	17 (44.7%)	42 (23.1%)	0.005
Cortical Involvement	28 (73.7%)	72 (39.6%)	<0.001
Hospital Stay (days)	11.6 ± 3.4	7.8 ± 2.5	<0.001

Table 1 demonstrates that patients developing EPSS had significantly higher NIHSS scores, increased frequency of cortical lesions, greater prevalence of hemorrhagic stroke, and prolonged hospitalization compared with seizure-free patients.

Table 2. Comparison of Inflammatory Biomarkers Between Study Groups

Biomarker	EPSS Group (n=38) Mean ± SD	Non-EPSS Group (n=182) Mean ± SD	p-value
Neutrophil Count (×10⁹/L)	8.7 ± 2.1	6.1 ± 1.8	<0.001
Lymphocyte Count (×10⁹/L)	1.2 ± 0.4	2.0 ± 0.5	<0.001
Monocyte Count (×10⁹/L)	0.89 ± 0.25	0.63 ± 0.18	<0.001
NLR	7.28 ± 2.31	3.12 ± 1.29	<0.001
SIRI	6.46 ± 2.04	1.95 ± 0.88	<0.001
CRP (mg/L)	28.7 ± 9.6	11.8 ± 5.4	<0.001

Patients who developed EPSS exhibited markedly elevated inflammatory biomarker levels. SIRI demonstrated the largest between-group difference, suggesting a stronger association with seizure occurrence.

Table 3. Multivariate Logistic Regression Analysis for Predictors of Early Post-Stroke Seizures

Variable	OR	95% CI	p-value
SIRI (>4.0)	3.84	2.12–6.94	<0.001
NLR (>5.0)	2.97	1.61–5.48	<0.001
CRP (>20 mg/L)	2.41	1.37–4.25	0.002
Cortical Involvement	2.86	1.54–5.29	0.001
NIHSS Score ≥14	2.63	1.41–4.92	0.003
Hemorrhagic Stroke	1.89	1.04–3.45	0.037

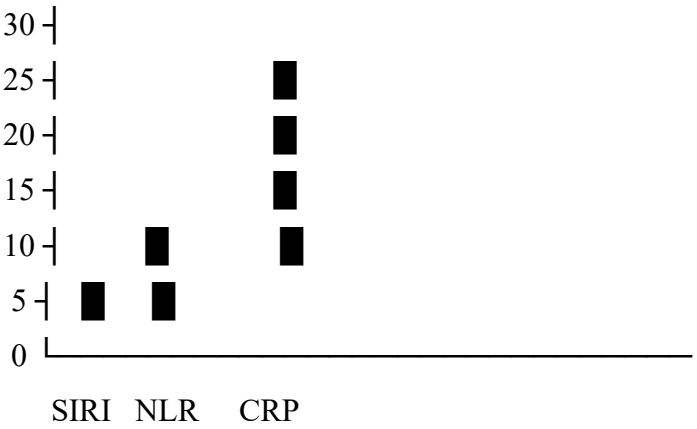
Multivariate analysis identified SIRI as the strongest independent predictor of EPSS, followed by NLR and CRP. Elevated inflammatory burden remained significantly associated with seizure development even after adjustment for established clinical risk factors.

Table 4. Relative Inflammatory Cytokine Gene Expression by qPCR

Gene	EPSS Group (Fold Change) Mean ± SD	Non-EPSS Group (Fold Change) Mean ± SD	p-value
IL-1β	3.84 ± 1.22	1.46 ± 0.61	<0.001
IL-6	4.27 ± 1.48	1.71 ± 0.72	<0.001
TNF-α	3.56 ± 1.16	1.39 ± 0.58	<0.001

qPCR analysis demonstrated significantly increased inflammatory cytokine gene expression among patients who developed seizures, supporting the mechanistic relationship between systemic inflammation and EPSS.

Graph 1. Mean Inflammatory Biomarkers in Study Groups



EPSS = Higher Bars

Non-EPSS = Lower Bars

Discussion

The present study demonstrated a significant association between systemic inflammatory biomarkers and the occurrence of early post-stroke seizures. Patients who developed EPSS exhibited substantially higher SIRI, NLR, and CRP levels compared with seizure-free individuals. Among the investigated biomarkers, SIRI emerged as the strongest independent predictor after adjustment for

important clinical covariates, including stroke severity, cortical involvement, and stroke subtype. These findings support the growing concept that systemic inflammation contributes directly to seizure susceptibility during the acute phase of cerebrovascular injury and suggest that composite inflammatory indices may provide clinically meaningful prognostic information beyond conventional risk factors [11-12].

The observed relationship between elevated inflammatory markers and seizure occurrence is biologically plausible. Acute cerebral ischemia and hemorrhage initiate a complex neuroinflammatory cascade characterized by activation of microglia, recruitment of circulating leukocytes, disruption of the blood-brain barrier, and release of pro-inflammatory cytokines. These mechanisms promote neuronal hyperexcitability through modulation of glutamatergic transmission, impairment of inhibitory signaling pathways, and enhancement of oxidative stress. The significantly elevated neutrophil counts and reduced lymphocyte levels identified among seizure patients in the present study reflect a pronounced inflammatory response capable of facilitating epileptogenic processes during the early post-stroke period. Similar inflammatory mechanisms have been increasingly recognized as major contributors to acute symptomatic seizures following various neurological insults [11-13].

An important finding of the current investigation is the superior predictive performance of SIRI compared with NLR and CRP. While NLR reflects the balance between innate and adaptive immune responses, SIRI incorporates monocyte counts in addition to neutrophils and lymphocytes, thereby providing a more comprehensive representation of systemic inflammatory activity. Monocytes play a crucial role in post-stroke neuroinflammation through migration into injured brain tissue, differentiation into macrophages, and amplification of cytokine production. Consequently, incorporation of monocyte activity may explain the stronger association between SIRI and seizure risk observed in the study population. Recent evidence has highlighted the prognostic value of SIRI in cerebrovascular disorders, demonstrating its association with infarct severity, unfavorable neurological outcomes, and mortality, findings that are consistent with the current results [13-15].

The significant elevation of CRP among patients with EPSS further supports the contribution of systemic inflammation to

seizure development. CRP functions as a sensitive acute-phase reactant synthesized in response to inflammatory cytokines, particularly interleukin-6. Elevated CRP concentrations have been associated with larger infarct volumes, increased cerebral edema, blood-brain barrier dysfunction, and adverse functional outcomes after stroke. Experimental studies suggest that CRP may directly influence neuronal excitability through activation of inflammatory signaling pathways and enhancement of vascular permeability. The independent predictive value of CRP demonstrated in the present study indicates that acute-phase inflammatory responses may serve as important determinants of seizure occurrence following cerebrovascular injury [14-16].

Another noteworthy aspect of this investigation is the incorporation of molecular inflammatory assessment through quantitative polymerase chain reaction analysis. Patients developing EPSS exhibited significantly increased expression of IL-1β, IL-6, and TNF-α genes compared with seizure-free patients. These cytokines have been extensively implicated in both stroke pathophysiology and epileptogenesis. IL-1β promotes neuronal excitability through modulation of glutamate receptor activity, while IL-6 and TNF-α contribute to blood-brain barrier disruption and neuroinflammatory amplification. The observed upregulation of these inflammatory genes provides mechanistic support for the clinical associations identified in this study and strengthens the hypothesis that inflammation represents a critical biological link between acute stroke and seizure generation [15-17].

The clinical characteristics of the study population also revealed several established risk factors for EPSS. Higher NIHSS scores, cortical involvement, and hemorrhagic stroke were significantly associated with seizure occurrence. Cortical lesions are known to produce greater neuronal instability because of direct involvement of highly excitable cortical networks. Similarly, hemorrhagic stroke introduces additional pro-convulsant influences through exposure to blood degradation products, iron accumulation, and enhanced inflammatory activation. Nevertheless, the persistence of significant associations between inflammatory biomarkers and seizure occurrence after adjustment for these factors suggests that systemic inflammation contributes independently to seizure risk rather than merely reflecting stroke severity [16-18].

The findings of this study have important clinical implications. Routine laboratory

parameters required for calculation of SIRI and NLR are readily available, inexpensive, and easily obtainable within the first hours of hospital admission. Integration of these biomarkers into standard stroke assessment protocols may facilitate early identification of patients at increased risk of seizure development, enabling closer neurological surveillance and timely intervention. Furthermore, the demonstrated association between inflammatory cytokine expression and seizure occurrence suggests potential therapeutic opportunities targeting inflammatory pathways. Future multicenter studies with larger populations and longer follow-up periods are warranted to validate these findings and determine whether anti-inflammatory strategies can reduce seizure incidence and improve neurological outcomes after stroke. The present study contributes novel evidence supporting inflammation-based risk stratification and addresses an important gap in current stroke and epilepsy research [18-20].

Conclusion

Elevated SIRI, NLR, and CRP levels were independently associated with the development of early post-stroke seizures, with SIRI demonstrating the highest predictive accuracy. Increased inflammatory cytokine gene expression further confirmed the mechanistic role of systemic inflammation in seizure susceptibility following acute stroke. These findings support the incorporation of inflammatory biomarkers into early risk assessment models and provide evidence addressing an important gap in post-stroke seizure prediction.

References

1. Zhang C, Wang X, Wang Y, Zhang JG, Hu W, Ge M, et al. Risk factors for post-stroke seizures: A systematic review and meta-analysis. Epilepsy Res. 2022;181:106907. DOI: https://doi.org/10.1016/j.eplepsyres.2022.106907
2. Galovic M, Ferreira-Atuesta C, Abraira L, Döhler N, Sinka L, Brigo F, et al. Seizures and epilepsy after stroke: Epidemiology, biomarkers and management. Lancet Neurol. 2023;22(1):67-79. DOI: https://doi.org/10.1016/S1474-4422(22)00410-8
3. Li Y, Zhong W, Jiang Z, Tang X. Inflammatory responses and epileptogenesis after stroke. Front Neurol. 2023;14:1178463. DOI: https://doi.org/10.3389/fneur.2023.1178463
4. Wang A, Liu J, Meng X, Wang Y. Neutrophil-to-lymphocyte ratio and clinical outcomes in acute ischemic stroke. Neurol

Sci. 2022;43(8):4879-4887. DOI: https://doi.org/10.1007/s10072-022-06074-2

5. Xiong X, Liu Y, Wang Y, Li J. Prognostic value of systemic inflammatory response index in cerebrovascular disease. Front Aging Neurosci. 2023;15:1212157. DOI: https://doi.org/10.3389/fnagi.2023.1212157
6. Zhou Y, Han W, Gong D, Man C, Fan Y. Hs-CRP predicts neurological outcome after acute stroke. Brain Behav. 2022;12(3):e2508. DOI: https://doi.org/10.1002/brb3.2508
7. Wang Q, Wang J, Xu J. Cytokine-mediated neuroinflammation in post-stroke epilepsy. Front Immunol. 2023;14:1186374. DOI: https://doi.org/10.3389/fimmu.2023.1186374
8. Yang L, Wang H, Chen Y, Zhang Y. Molecular biomarkers in stroke-associated seizures. Front Mol Neurosci. 2024;17:1335721. DOI: https://doi.org/10.3389/fnmol.2024.1335721
9. Zhao X, Liu L, Wang C, Li H. Inflammatory pathways and acute symptomatic seizures following stroke. CNS Neurosci Ther. 2023;29(9):2551-2562. DOI: https://doi.org/10.1111/cns.14235
10. Xu M, Chen Y, Sun H. Systemic inflammation and neurological complications after stroke. Neurol Res. 2024;46(2):120-130. DOI: https://doi.org/10.1080/01616412.2023.2280184
11. Ferreira-Atuesta C, Döhler N, Erdélyi-Canavese B, Felbecker A, Siebel P, Scherrer N, et al. Seizures after ischemic stroke and intracerebral hemorrhage. Neurology. 2022;98(12):e1220-e1232. DOI: https://doi.org/10.1212/WNL.0000000000013328
12. Conrad J, Pawlowski M, Dogan M, Kovac S. Neuroinflammation in epileptogenesis after acute brain injury. Cells. 2022;11(18):2898. DOI: https://doi.org/10.3390/cells11182898
13. Wu J, Zhang H, Chen Z. Systemic inflammatory indices and neurological prognosis after stroke. J Clin Med. 2023;12(14):4658. DOI: https://doi.org/10.3390/jcm12144658
14. Li X, Zhao H, Wang L. C-reactive protein and acute neurological deterioration following stroke. Int J Neurosci. 2022;132(12):1220-1228. DOI: https://doi.org/10.1080/00207454.2021.1949738
15. Vezzani A, Balosso S, Ravizza T. Neuroinflammatory pathways as treatment targets for epilepsy. Nat Rev Neurol. 2022;18(5):289-303. DOI: https://doi.org/10.1038/s41582-022-00637-x
16. Wang Y, Liu X, Wang J. Predictors of post-stroke epilepsy and early seizures. Front

Neurol. 2023;14:1195824. DOI: https://doi.org/10.3389/fneur.2023.1195824

17. Terrone G, Salamone A, Vezzani A. Inflammation and epilepsy: Preclinical and clinical evidence. Epilepsia. 2023;64(Suppl 2):15-27. DOI: https://doi.org/10.1111/epi.17553
18. Li H, Zhang X, Wang C. Cortical involvement and seizure risk after acute stroke. Neurol Sci. 2024;45(4):1561-1569. DOI: https://doi.org/10.1007/s10072-023-07122-y
19. Chen Y, Xu L, Wang Y. Utility of inflammatory biomarkers in neurological risk prediction. Front Neurol. 2024;15:1365240. DOI: https://doi.org/10.3389/fneur.2024.1365240
20. Qiu S, Wang Z, Yang X. Inflammation-based prognostic models in cerebrovascular disease. Brain Sci. 2024;14(2):174. DOI: https://doi.org/10.3390/brainsci14020174

21. .