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CLINICAL, IMMUNOLOGICAL, NEUROPHYSIOLOGICAL, AND GENETIC PREDICTORS OF SYMPTOMATIC EPILEPSY FOLLOWING STROKE

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Abstract. Background: Post-stroke epilepsy is a common complication of cerebrovascular events, significantly affecting patients' neurological outcomes and quality of life. Its development is influenced by multiple factors, including stroke type, lesion location, neuroinflammatory processes, and genetic predisposition. **Objective:** To evaluate the prognostic significance of clinical-neurological, immunological, neurophysiological, and genetic factors in the development of symptomatic epilepsy following ischemic or hemorrhagic stroke. **Methods:** A prospective observational study was conducted on 82 patients aged 45–85 years who had experienced ischemic or hemorrhagic stroke. Clinical evaluation included neurological deficit assessment using the NIHSS, imaging studies, and risk factor analysis. Neurophysiological assessment was performed using EEG, immunological markers were measured through cytokine levels (IL-1β, IL-6, TNF-α), and genetic polymorphisms associated with neuroinflammation and neuronal excitability were analyzed. Patients were followed for 12 months for seizure occurrence and neurological changes. **Results:** Symptomatic post-stroke epilepsy developed in 18 patients (21.9%). Significant predictors included cortical lesion location, hemorrhagic stroke type, high NIHSS scores, early epileptiform activity on EEG, elevated IL-6 and TNF-α levels, and specific genetic polymorphisms. A combined analysis of clinical, neurophysiological, immunological, and genetic factors allowed accurate risk stratification. **Conclusions:** Post-stroke epilepsy is a multifactorial condition influenced by clinical, neurophysiological, immunological, and genetic factors. Comprehensive assessment of these parameters improves prognostic accuracy and may guide personalized preventive and therapeutic strategies.

Keywords: post-stroke epilepsy, ischemic stroke, hemorrhagic stroke, neuroinflammation, cytokines, EEG, genetic polymorphisms, NIHSS, prognostic factors.

Introduction. Stroke remains one of the leading causes of disability and mortality worldwide. According to the World Health Organization (WHO), millions of new cases of cerebrovascular diseases are registered annually, with the consequences of stroke significantly reducing patients' quality of life and placing a substantial burden on healthcare systems. Stroke can lead to various complications, among which post-stroke (symptomatic) epilepsy occupies a particularly important place.

Post-stroke epilepsy is defined as recurrent seizure episodes that occur following a stroke. According to the literature, its incidence ranges from 5% to 20%, depending on the type of stroke (ischemic or hemorrhagic), the location of the lesion, patient age, and accompanying risk factors. Seizure activity after stroke is associated with more severe neurological deficits, an increased risk of recurrent stroke, and a reduction in quality of life.

Modern research indicates that the pathogenesis of post-stroke epilepsy is multifactorial, involving the interaction of clinical-neurological factors, neuroinflammatory processes, neurophysiological changes, and genetic predisposition. Elevated levels of pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) may contribute to neuronal hyperexcitability, while certain polymorphisms in genes involved in the regulation of inflammatory processes and neuronal membrane excitability may increase the risk of seizure development.

Aim of the Study. The aim of this study was to evaluate the prognostic significance of clinical-neurological, immunological, neurophysiological, and genetic factors in the development of symptomatic epilepsy following stroke.

Materials and Methods. The study included 82 patients aged 45 to 85 years who had experienced either ischemic or hemorrhagic stroke. All participants provided informed consent for participation. Patients with a history of epilepsy, traumatic brain injury, or oncological or infectious diseases of the central nervous system were excluded. Clinical assessment involved the collection of medical history, identification of risk factors such as hypertension, diabetes mellitus, smoking, and dyslipidemia, as well as evaluation of the severity of neurological deficits using the National Institutes of Health Stroke Scale (NIHSS). The localization and nature of the stroke were confirmed through computed tomography (CT) and magnetic resonance imaging (MRI).

Neurophysiological assessment was performed using electroencephalography (EEG), which allowed for the detection of epileptiform activity and cortical-level dysfunction. Immunological evaluation included measurement of pro-inflammatory cytokine levels (IL-1β, IL-6, and TNF-α) using enzyme-linked immunosorbent assay (ELISA). Genetic analysis was conducted to identify polymorphisms in genes associated with neuroinflammation and neuronal excitability, including IL-6, TNF-α, and BDNF, using polymerase chain reaction (PCR) and sequencing techniques.

All patients were followed for a period of 12 months after the stroke, during which the occurrence of seizure activity, frequency of episodes, and changes in neurological status were recorded. Statistical analysis included descriptive statistics, t-tests for quantitative variables, chi-square tests for categorical variables, and regression analysis to assess the prognostic significance of various factors. Differences were considered statistically significant at $p<0.05$.

Results. Out of the 82 patients included in the study, symptomatic post-stroke epilepsy developed in 18 individuals, accounting for 21.9% of the cohort. Analysis of clinical-neurological factors revealed that cortical localization of the lesion and hemorrhagic type of stroke were significantly associated with a higher risk of seizure development ($p=0.01$ and $p=0.02$, respectively). Additionally, patients with higher NIHSS scores at the time of admission were more likely to develop post-stroke epilepsy, indicating that greater neurological deficit is an important predictor ($p=0.03$).

Neurophysiological evaluation demonstrated that the presence of epileptiform activity on EEG during the early post-stroke period was strongly associated with subsequent seizure episodes ($p=0.01$). Immunological assessment showed that elevated levels of pro-inflammatory cytokines IL-6 and TNF-α were significantly correlated with the development of post-stroke epilepsy, whereas IL-1β levels did not demonstrate a statistically significant effect.

Genetic analysis identified certain polymorphisms in the IL-6 and BDNF genes that were associated with an increased risk of post-stroke seizure activity. Combining clinical, neurophysiological, immunological, and genetic factors into a prognostic model allowed for the identification of patients at high risk of developing post-stroke epilepsy with an accuracy of up to 85%.

These findings indicate that post-stroke epilepsy is a multifactorial condition, with its development influenced by a combination of lesion characteristics, severity of neurological deficit, neurophysiological abnormalities, inflammatory responses, and genetic predisposition.

Discussion. The results of this study confirm the multifactorial nature of post-stroke epilepsy. Cortical lesion location and hemorrhagic stroke were found to significantly increase the likelihood of seizure development, which is consistent with previous studies. Higher NIHSS scores, reflecting more severe neurological deficits, were also associated with a greater risk, indicating that extensive cortical damage plays a key role in epileptogenesis.

Neurophysiological findings demonstrated that early epileptiform activity on EEG serves as a strong predictor of subsequent seizures, highlighting the importance of early neurophysiological monitoring in post-stroke patients. Immunological analysis revealed that elevated levels of IL-6 and TNF-α contribute to neuronal hyperexcitability, suggesting that neuroinflammation plays a central role in the pathogenesis of post-stroke epilepsy. In contrast, IL-1β levels did not show a statistically significant effect in this cohort, which may reflect the complexity of inflammatory mechanisms in seizure development.

Genetic predisposition also appears to be a critical factor. Certain polymorphisms in IL-6 and BDNF genes were associated with an increased risk of seizures, supporting the concept that individual genetic variability in neuroinflammatory and neuroplastic pathways can influence epileptogenesis. These findings underscore the value of a comprehensive approach that integrates clinical, neurophysiological, immunological, and genetic data to improve risk stratification and guide personalized preventive strategies.

1. ReferenceSymptomatic post-stroke epilepsy developed in 21.9% of the studied patients, highlighting its clinical significance.

2. Key predictors of post-stroke epilepsy include cortical lesion location, hemorrhagic stroke type, severity of neurological deficit (NIHSS), early epileptiform activity on EEG, elevated IL-6 and TNF-α levels, and specific genetic polymorphisms.

3. A comprehensive evaluation of clinical-neurological, neurophysiological, immunological, and genetic factors enhances the accuracy of predicting post-stroke epilepsy and enables optimization of preventive and therapeutic strategies.

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