

Main Clinical and Neurological Risk Factors and Pathogenetic Predictors Participating in the Formation of Post-Stroke Convulsion Syndrome

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Abstract Post-stroke seizure syndrome is one of the most serious and prognostically unfavorable complications of acute cerebrovascular disorders, occurring in 6-18% of stroke patients and significantly worsening the functional outcome of the disease. The development of epileptic seizures in the post-stroke period not only exacerbates neurological deficit and slows down neurorehabilitation processes, but also significantly increases the risk of recurrent vascular disorders and death.

Keywords Post-stroke epilepsy, Seizure syndrome, Cerebrovascular diseases, Epileptogenesis, risk factors, Neural inflammation, Ischemic stroke, Hemorrhagic stroke, Neurorehabilitation, Prognostic factors, Perinfarction zone, Synaptic plasticity

1. Introduction

The pathogenesis of post-stroke epileptogenesis is a complex multi-component process that includes primary ischemic or hemorrhagic damage to brain tissue, secondary neurochemical and structural changes, the formation of pathological neural networks, and a disruption of the balance of excitatory and inhibitory neurotransmitter systems [1,6]. Neuro-inflammatory processes, glial scarring, changes in ion channels, and synaptic plasticity in the peri-infarction zone play a special role in the development of convulsive readiness [2,7]. The clinical polymorphism of post-stroke seizures, ranging from early symptomatic seizures in the acute period of stroke to late epileptic seizures developing months or years after a vascular accident, necessitates a comprehensive study of risk factors and predictors for the development of this complication. Modern neuroimaging technologies, including diffusion-weighted MRI, functional MRI, and positron emission tomography, open up new possibilities for identifying morphofunctional predictors of epileptogenesis at the preclinical stage [3,9]. Identifying reliable clinical, neurological, and paraclinical predictors of post-stroke seizure syndrome is crucial for developing personalized prevention and early treatment strategies, optimizing neuroprotective therapy, and improving the long-term prognosis in patients with cerebrovascular diseases. Analysis of modern literature indicates insufficient systematization of data on post-stroke epilepsy risk factors

and the absence of validated prognostic models for clinical use [4,10]. The multifactorial nature of post-stroke epileptogenesis requires a comprehensive approach to studying the interaction of various pathogenetic mechanisms, including the localization and size of the lesion, stroke type, patient age, comorbid pathology, and genetic predisposition factors.

Purpose of the study: to identify the main clinical and neurological risk factors and pathogenetic predictors of the formation of post-stroke seizure syndrome to develop personalized approaches to the prevention and treatment of this complication.

2. Materials and Methods of Research

In order to study the neurological system in depth, patients participating in the study were subjected to objective and clinical-neurological examination methods according to a standard scheme. A total of 143 patients participated in the study, who had a stroke for more than 1 month and had a stroke in the early and late recovery, as well as in the residual period. Of these, 111 were men and 32 were women. 30 patients with a history of stroke without pain were taken as a control group.

NIHSS was used to assess the severity of stroke during clinical and neurological examinations. The Barthel index was used to assess the functional state and daily life activity of patients, and the modified Rankin Scale (mRS) was used to determine the degree of disability.

The proportion of women and men (%) in the group with seizures accompanied by ischemic and hemorrhagic stroke,

as well as seizure-free stroke, and the average age in each group, were determined. Comparison allows for the assessment of demographic differences between clinical groups and age-sex factors influencing the development of epilepsy.

The development of seizures in stroke patients is observed in the older age group depending on age: in all three groups, the average age is around 60 years. In groups with ischemic and hemorrhagic seizures, the proportion of women and men is almost equal, while in the group without seizures, men predominate (71%). These demographic differences correspond to the nature of the clinical process: seizures occur equally in both sexes, but seizure-free stroke is more common in men. The similarity of age indicators confirms that stroke and post-stroke epilepsy occur mainly in older age, to identify the main clinical and neurological risk factors and pathogenetic predictors of the formation of post-stroke seizure syndrome to develop personalized approaches to the prevention and treatment of this complication.

3. Results

In order to study the neurological system in depth, patients participating in the study were subjected to objective and clinical-neurological examination methods according to a standard scheme. A total of 143 patients participated in the study, who had a stroke for more than 1 month and had a stroke in the early and late recovery, as well as in the residual period. Of these, 111 were men and 32 were women. 30 patients with a history of stroke without pain were taken as a control group. NIHSS was used to assess the severity of stroke during clinical and neurological examinations. The Barthel index was used to assess the functional state and daily life activity of patients, and the modified Rankin Scale (mRS) was used to determine the degree of disability. The proportion of women and men (%) in the group with seizures accompanied by ischemic and hemorrhagic stroke, as well as seizure-free stroke, and the average age in each group, were determined. Comparison allows for the assessment of demographic differences between clinical groups and age-sex factors influencing the development of epilepsy.

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Seizures were classified according to the international classification ILAE (2017) as early (within 7 days after a stroke) and late seizure syndrome. In order to objectively assess the clinical severity of seizures, the National Hospital Seizure Severity Scale (NHS3) was used. Using this scale, the duration of the attack, loss of consciousness, automatisms,

physical injuries, and the severity of the post-sexual state were assessed. To predict the risk of epilepsy development after stroke, the SeLECT scale was used in patients with ischemic stroke, and the CAVE scale in patients with hemorrhagic stroke. Using these prognostic scales, patients were divided into low, medium, and high-risk groups. The Montreal Cognitive Assessment (MoCA) scale was used to assess the cognitive state of patients. The Hospital Anxiety and Depression Scale (HADS) was used to determine the level of mental and emotional state, including anxiety and depression. These scales were also used in the analysis of gender characteristics.

4. Conclusions

1. The clinical, epidemiological, and psycho-emotional characteristics of post-stroke seizure syndrome indicate that early seizures were recorded in 71 patients (50.3%), in whom the frequency of seizures increased by 1-2 times/month, and in late seizures by 3-4 times/month ($U < 0.001$). Focal seizures predominated in patients with early attacks (54.9%), while generalized seizures were more common in late attacks (32.8%). The worsening of the psycho-emotional state and the presence of signs of cognitive impairment in patients with late seizures indicated a worsening of the clinical course.
2. The main pathogenetic predictors of seizures in ischemic and hemorrhagic stroke were identified, cortical damage ($\chi^2=11.2$; $p < 0.01$), frontal-temporal localization ($\chi^2=9.6$; $p < 0.01$), NIHSS ≥ 16 (OR ≈ 3.2), generalized seizures ($\chi^2=14.3$; $p < 0.001$) and PLEDs activity ($\chi^2=8.12$; $p < 0.05$) are the strongest signs of severe seizures.
3. A strong correlation was found between the S100B level, calcium $\ll 2.1$ mmol/l, and EEG epileptiform activity. In patients with a high level of S100B, epileptiform activity in the EEG was noted in 70% of cases ($\chi^2=19.2$; $p < 0.001$), cortical damage was 73.8% ($\chi^2=13.1$; $p < 0.001$). A low level of calcium was noted as an independent biochemical predictor of seizure severity ($\chi^2=16.4$; $p < 0.001$).

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