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Research Article

PRINCIPLES OF MANAGEMENT OF PATIENTS WITH POST-STROKE SEIZURES

Submission Date: August 06, 2023, Accepted Date: August 11, 2023,

Published Date: August 16, 2023 |

Crossref doi: <https://doi.org/10.37547/TAJMSPR/Volume05Issue08-09>

Dilbar Tadzhievna Khodzhieva

Doctor Of Medical Sciences, Professor Bukhara State Medical Institute, Uzbekistan

Sitora Bakhramovna Barnaeva

Bukhara State Medical Institute, Uzbekistan

ABSTRACT

Statistically, in 1 in 10 adult patients, first-onset epilepsy is associated with a history of stroke, and this aetiology occurs in 1 in 4 patients aged 65 years and older with epilepsy. Incidence rates are higher in patients with intracranial, that is, intracerebral or subarachnoid haemorrhage. Haemorrhage increases the risk of seizures by up to 10- 16%. The risk of subsequent development of epilepsy is highest in patients with remote and sequelae of ischaemic stroke. Classification of seizures in stroke and their frequency Seizures develop in different stages of stroke. Depending on the time of onset, they can be divided into the following types: precursor seizures, early and late seizures. Unfortunately, there is currently no consensus among neurologists about the timing of the onset of these seizures . The most common definitions are that early attacks are those that occurred in the first 24-48 h, the first week, the first 2 weeks, and 1 month from the onset of stroke.

KEYWORDS

Convulsive seizures, stroke, epilepsy.

INTRODUCTION

Stroke is the second most common cause of death and is the leading cause of disability worldwide. Although recent advances in acute stroke therapy have improved life expectancy, there has been a consistent increase in the prevalence of stroke-related epilepsy [1,

2]. The principles of management of patients with post-stroke convulsive seizures have not evolved as rapidly as other aspects of post-stroke management. To date, there are insufficient clinical practice guidelines addressing most of the fundamental issues in the

management of post-stroke seizures [3, 4]. The management of patients with post-stroke epilepsy is of great clinical importance because patients with seizures after stroke have higher mortality and disability than those without seizures [5]. In addition, epilepsy worsens long-term functional outcomes in those who have had a stroke [6]. Seizures worsen the quality of life of patients, may delay the recovery of functions damaged by stroke, and exacerbate cognitive impairment. The social consequences of post-stroke epilepsy play an important role. Therefore, prevention and treatment of epileptic seizures are important in patients with stroke [7]. Frequency of epilepsy in stroke. Stroke is an acute disease that is accompanied by the death of brain cells due to acute cerebral circulatory failure. It is manifested by general cerebral and localised symptoms. The development of stroke is possible under two scenarios: either after 24 hours from the onset of the disease, its signs persist, or there is a lethal outcome. The study of cerebral circulatory disorders (CCD) of the haemorrhagic type is an urgent task because of the tendency to increase the frequency of the pathology, as well as the high level of mortality and disability associated with CCD. The frequency of epileptic seizures in stroke patients varies in a wide range, according to different authors, from 2-33% to 50-78% for early seizures. The frequency of late post-stroke epileptic seizures also varies in different studies from 3-4.5% to 67% according to the literature [1,8]. It has been found that the majority of early convulsive seizures occur within the first 24 h of stroke onset [9]. According to some authors, the average time from the onset of stroke to the first epileptic seizure is 18 months (range 0-7 years). According to other data, most epileptic seizures occur in the 2nd-10th month of ischaemic stroke. Late poststroke epileptic seizures are more likely to occur between 6 months and 2 years after stroke [10]. According to different authors, the frequency of poststroke epilepsy

development ranges from 2.3 to 43%. Analysing age and sex peculiarities, it is worth noting that the majority of convulsive seizures in men occur at the age of 50-69 years, and in women - at the age of 60-79 years [21]. Seizures are more likely to develop in relatively young and middle-aged patients. To give an example, in people under 40 years of age in 30% of cases, and in those under 50 years of age in 23.1% of men and 20% of women (22.6% of cases in total). In patients of older age groups (over 60 years of age), there is a tendency to decrease attacks after ischaemic stroke. At the same time, the development of more severe seizures was found in patients aged 40 to 60 years compared to the group of patients aged 60-80 years. Pathogenesis of epilepsy in stroke Epileptic seizures, divided according to the time of their occurrence, have different pathogenetic mechanisms. Precursor attacks may occur before the onset of clinical symptoms of stroke and are often the only clinical symptom of a transient cerebral circulation disorder or so-called "silent" stroke, the diagnosis of which can be established retrospectively by computed tomography or magnetic resonance imaging [3]. Early attacks are thought to result from local cellular biochemical dysfunction. Disruption of the blood-brain barrier caused by acute ischaemia leads to dysfunction of ion channels and disturbance of neurotransmitter homeostasis [4]. In areas of hypoxic-ischaemic damage, the extracellular concentration of glutamate increases, which may lead to secondary neuronal damage and discharge of epileptiform activity. It is also assumed that the seizures observed in severe stroke are the result of depolarisation of the peri-infarct region [5]. The latter was further confirmed in an animal model of mechanical occlusion of the middle cerebral artery, which demonstrated altered membrane properties and increased excitability of neuronal populations in the neocortex and hippocampus. In rodent models, stroke induces changes in ion channel function that

result in an increase in extracellular potassium and intracellular calcium and sodium [2, 6]. These local ionic shifts may reduce the neurotransmitter capture threshold for depolarisation. In addition to focal ischaemia, global hypoperfusion can induce seizure activity, especially when highly epileptogenic regions such as the hippocampus are involved. Furthermore, extravasated thrombin may also contribute to epileptic seizures by causing prolonged enhancement of reactivity to afferent stimulation. Finally, haemosiderin deposition after intracerebral haemorrhage or haemorrhagic transformation may lead to increased neuronal excitability [7]. Late seizures occur when the occurrence of epileptogenesis is observed. Seizures after stroke in men and women are more likely to occur if the lesion is localised in the frontal lobe of the brain. The susceptibility to seizures is due to gliosis, deafferentation, selective neuronal loss, chronic inflammation, angiogenesis, neurodegeneration, collateral synaptic sprouting and altered synaptic plasticity [3,8]. Seizures lead to destruction of the blood-brain barrier and promote local inflammation, which is involved in the formation of a nidus for the development of late seizures [2,9]. In addition, vasogenic cerebral oedema, collapse of cellular ionic gradients and mitochondrial dysfunction may contribute to secondary irreversible brain damage (gliosis) and lower seizure threshold. Persistent seizure activity in cerebral ischaemia significantly increases infarct size and impairs functional recovery [3]. The latter suggests that stroke-related seizures and stroke share common pathogenic mechanisms and influence each other. The association between stroke and epileptic seizures or epilepsy is bidirectional [4]. Interestingly, middle-aged and elderly patients with newly diagnosed epilepsy have a 2- to 3-fold increased risk of subsequently developing stroke within the next two years [11]. The hypothesis behind this is that epilepsy in these patients may be caused by

microangiopathic changes predisposing to later cerebrovascular changes, making seizures an early biomarker of subsequent stroke [12].

Risk factors: One important issue is the risk factors leading to the development of post-stroke seizures and, in particular, to the further development of post-stroke epilepsy. Much attention is currently being paid to finding risk factors for the development of vascular epilepsy. Most studies in haemorrhagic stroke have revealed a higher incidence of epileptic seizures and their early onset compared to ischaemic stroke. Haemorrhagic transformation is a risk factor for the development of early seizures, as well as an independent predictor of status epilepticus in the acute period of ischaemic stroke [3]. Some studies have found a higher incidence of epileptic seizure development after cardioembolic stroke compared with other subtypes of ischaemic stroke. At the same time, there are a large number of studies in which the relationship between epileptic seizures and cardioembolic stroke subtype has not been confirmed [14]. The role of stroke severity in the development of epilepsy has also been discussed. Population-based and prospective multicentre studies have demonstrated that severe stroke is an important predictor of both early seizures and post-stroke epilepsy [15]. A population-based study described by M. Lofthouse, showed that severe stroke is an important predictor of post-stroke epilepsy. A similar result was obtained in a study by L. Kammersgaard, T. Olsen [8]. The results of the prospective Copenhagen study suggest that severe stroke may be an independent predictor of early seizures, and an association of stroke severity with the development of late epileptic seizures was also found. Currently, there is a mixed attitude of different researchers to the volume of the ischaemic focus as a risk factor for the development of epileptic seizures. There are

supporters of the viewpoint that a large stroke centre volume is a predictor of seizure development [4]. There are studies in which it is shown that the size of the focus more than 1/2 of the cerebral hemisphere may be an independent predictor of late poststroke seizures. In the study of A. Alberti, M. Paciaroni, and V. Caso in a single-factor analysis, large focal volume was a significant predictor of early seizures, but was insignificant in a multivariate analysis [2]. Many risk factors for post-stroke seizures and epilepsy remain poorly understood and the evidence is mixed. Among the new factors, the role of parieto-temporal cortex, supratentorial and superior temporal gyrus involvement in post-stroke epileptogenesis has been confirmed [9]. It has also been found that the spread of ischemic foci to the large hemispheric cortex may serve as a predictor of both early and late epileptic seizures [5]. A retrospective population-based study showed that ethnicity and localisation of the stroke focus are not risk factors for early seizures [4].

CONCLUSIONS

Thus, stroke at the moment remains one of the most important medical and social problems. This is due to their high share in the structure of morbidity and mortality of the population. In addition, cerebrovascular diseases frequently lead to the development of structural epilepsy. Given that in most cases of post-stroke epilepsy occurs at a young age and that there is conflicting data regarding risk factors for the development of post-stroke epilepsy, it is necessary to conduct research to better understand the pathophysiological mechanisms of post-stroke epilepsy, which will ensure timely prevention and diagnosis of post-stroke epilepsy and improve the quality of life of patients..

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