International Journal of
Health Systems and Medical Sciences

ISSN: 2833-7433
Volume 2 | No 2 | February -2023

Prediction of Epileptic Symptoms after Hemorrhagic Stroke

Khaydarova Dildora Kadirovna [1], Sharopova Munisa Mukhsinovna [2]

[1] Docent of the Department of Neurology, Tashkent Medical Academy, Doctor of Medical Sciences

[2] Tashkent Medical Academy, Faculty of medical pedagogy, 6 courses

Abstract: Vascular diseases of the brain are one of the most important problems of modern neurology and cardiology. Increasing prevalence, disability, high mortality of patients with very limited prospects for the restoration of impaired functions and ability to work determine the medical and social nature of their consequences.

Keywords: hemorrhagic stroke, epilepsy, vascular factor, risk factor, neuroclinical signs.

Epilepsy is a polyetiological disease characterized by polymorphism of clinical symptoms with a variety of neurobiological, neuropsychological, pathopsychological, and social manifestations. Worldwide, there is a high prevalence of this disease, which is 0.5-2%. Annually, 50-70 cases are recorded per 100,000 population. Important features of this disease include a combination of both psychotic and non-psychotic registers of disorders [1-3], various complaints and multiple repeated requests for medical help. According to the available data from a number of studies [4-6], various factors contribute to the development of this disease: genetic, perinatal, environmental, personal. A large number of works are devoted to the study of psychological aspects in epilepsy [7-9]. Moreover, over the past years, in the structure of psychopathological disorders in epilepsy, there has been a decrease in the proportion of epileptic manifestations proper, with a simultaneous increase in the proportion of non-psychotic affective disorders, in particular, the depressive pole, which is a reflection of clinical pathomorphosis.

The prevalence of DR among patients with epilepsy is extremely high and, according to different authors, ranges from 10% to 55% (7). Despite the high incidence of DR, these disorders often remain unrecognized in 50% of patients with epilepsy (BE), due to both atypical manifestations and misdiagnosis (8). The presence of depression reduces the quality of life to a greater extent than the frequency and severity of epileptic seizures (6). Depression not only worsens the quality of life, but also negatively affects the dynamics of neurological symptoms during treatment (3). Depression claims the lives of about 1 million people annually due to suicide, and the incidence of suicide in combination with epilepsy of depression is 5-10 times higher than in the general population (4).

The problem of epilepsy remains at the center of attention of neuroscientists. This is due not only to the diversity of neurophysiological features of the disease, the versatility of the clinical picture, but also to various complications that develop against the background of regular use of antiepileptic drugs. Cognitive impairments, along with seizures, are among the main characteristics of patients with epilepsy [1-3, 6]. In this regard, there is an opinion that it is the cognitive defect that is one of the reasons for the violation of social adaptation and disability of such patients. In fact, as literature sources show, a history of more than 100 generalized tonic-clonic seizures in most cases leads to the

development of cognitive impairment or pre-dementia disorders [4, 5, 9]. At the same time, attention disorders prevail in the structure of the cognitive defect. The situation is somewhat different in temporal lobe epilepsy with complex partial seizures. In this case, for the occurrence of a defect and dementia, it is not the number of seizures that matters, but the duration of the disease. So, according to a number of authors, irreversible changes are detected after 5 years of continuous occurrence of complex partial seizures. At the same time, other sources indicate a longer period of 20 years [10, 11]. And in this case, the cognitive defect mainly concerns the function of memory. Based on these data, it can be concluded that cognitive disorders in epilepsy depend on many factors and differ in the polymorphism of clinical manifestations. Cognitive impairment in epilepsy, as a rule, manifests itself in the form of impaired memory, speech, attention, and thinking. As literature sources show, it is of scientific interest to study the relationship between cognitive impairments and personality and psychopathological characteristics of patients with different forms of epilepsy and different types of seizures [14, 15]. In addition, little has been studied about the effect of antiepileptic drugs on the aggravation of cognitive impairment, which is of great importance for clinical neurology.

The relevance of studying the clinical and psychological aspects of the functioning patterns of patients with epilepsy is determined not only by the fact that this disease is highly common among the entire group of neuropsychiatric diseases [10, 11], but also by the influence of the chronification of this disease on the social adaptation of patients, their system relationships in micro- and macrosociety [12, 13]. A comprehensive assessment of functioning disorders in patients with epilepsy reflects the particular importance of timely and targeted therapeutic and rehabilitation measures for this group of patients. The concept of "pattern" (eng. "pattern" - form, example) in this study, we consider as a form, a model of behavior of living individuals. The traditional notion that epilepsy is a chronic disease characterized by a gradual increase in severity, frequency of seizures, and personality changes has now been revised. So, along with severe cases of the course of the disease, there are also enough positively favorable. This principle was put by us as the basis for the division of patients into 2 groups: with favorable and unfavorable types of flow.

The purpose of the study was to study disorders of functioning patterns in patients with epilepsy, taking into account the types of the course of the disease.

In patients with arterial hypertension, destructive changes occur in the walls of cerebral vessels, mainly in the muscular wall of arterioles: the ion permeability of smooth muscle cell membranes increases, followed by their hypoxia, anoxia and death. The number of smooth muscle cells in the muscular membrane of the proximal, distal lenticulostriate arteries, enveloping cortical arterioles decreases by more than 50% of the initial number. These changes lead to diffuse accumulation of cell decay products in the vascular wall, fibrinoid necrosis, formation of microaneurysms in arterioles with a diameter of 70-700 microns, which are rupture sites [3].

Of great importance in the development of non-traumatic intracerebral hemorrhages are the features of the blood supply to the subcortical nodes: the lenticulostriate arteries depart from the middle cerebral artery at a right angle, the number of anastomoses between them is small, which does not provide sufficient depreciation in hypertensive cerebral crises and leads to their rupture [10].

Currently, there are two leading mechanisms for the development of stroke intracerebral hemorrhages: rupture of a pathologically altered vessel with the formation of a hematoma and hemorrhagic impregnation. Hemorrhages in the form of a hematoma, according to autopsy materials, are found in 85% of cases, have typical localization, are located in the subcortical nodes, in the lobes of the brain (lobar) and in the cerebellum. In the acute period of the disease, with this type of hemorrhage, there is no significant destruction of the brain substance, since the outflowing blood pushes the nerve fibers apart. In 15% of cases, hemorrhagic impregnation of the brain substance develops, which most often develops in the thalamus, in the pons and is accompanied by significant destruction of the surrounding brain tissue [5].

The formation of intracerebral hematomas leads to vascular-reflex, parabiotic and necrotic reactions in the brain tissue. The volume of hemorrhage in most cases increases within 2-3 hours after the disease. Subsequently, local and distant vascular reflex reactions develop, leading to a cascade of

pathophysiological reactions in brain structures, to their ischemia, edema, and swelling [6].

The time parameters of reversible ischemia of the brain substance, the causes of this ischemia, its effect on the surrounding brain structures, as well as the time of occurrence and the degree of growth of irreversible cerebral changes are still being discussed [7].

The prevalence of epilepsy is from 1.5 to 31 cases per 1000 population, the incidence is from 11 to 134 cases per 100 thousand population. The second peak in the incidence of epilepsy after childhood occurs in the elderly due to an increase in the incidence of chronic cerebral ischemia (CCI) and stroke among them. The risk of developing epilepsy in people older than 70 years is even higher than in the first 10 years of life [1, 2].

According to recent epidemiological studies, the incidence of epilepsy in patients older than 60 years in 2006 reached 104, and in 2020 - 127.2 cases per 100 thousand of the population. This is due to an increase in the number of persons of older age groups in the population, as well as an increase in the prevalence of cerebrovascular pathology, which is one of the leading risk factors for epilepsy in elderly patients [2]. According to the International Antiepileptic League, in the general structure of the causes of symptomatic locally caused epilepsy, vascular diseases of the brain account for 6-8%. The prevalence of this epilepsy increases from 15 cases (after 50 years) to 45–50 (after 60–75 years) cases per 100,000 population [3].

According to V.A. Manukovsky (2006), when determining the tactics of resuscitation treatment of patients with dislocation syndrome, it is advisable to divide them into groups - with compensated, subcompensated and decompensated condition. The author proves that in the compensated state of patients, the treatment is based on the generally accepted principles of intensive care, in the subcompensated state, it is necessary to urgently ensure the optimal flow of oxygen-enriched blood to the brain, and in the decompensated state, effective treatments have not been found [8].

One of the reasons for severe disability and high mortality in patients with hemorrhagic stroke is the development of recurrent hemorrhage. The frequency of postoperative hemorrhages reaches 50% and is primarily associated with insufficient intraoperative hemostasis, intractable high blood pressure, concomitant diseases of internal organs in the stage of sub- and decompensation [9].

Among patients with cerebrovascular pathology suffering from epilepsy, in 27% of cases, this disease was associated with a previous stroke, in the rest - with signs of CCI, manifested by "silent" strokes in the blood supply basin of the predominantly middle cerebral artery, hypodense foci of hemispheric localization.

Vascular diseases of the brain are one of the most important problems of modern neurology and cardiology. Increasing prevalence, disability, high mortality of patients with very limited prospects for the restoration of impaired functions and ability to work determine the medical and social nature of their consequences [11].

The study of cerebral hemodynamics in patients with arterial hypertension (AH) seems to be relevant due to the fact that this disease is a risk factor for acute and chronic disorders of cerebral blood supply, having a complex and multifaceted effect on the state of many organs and functional systems of the body. [13]

This circumstance makes it necessary to study cerebral hemodynamics in persons with chronic disorders of cerebral circulation in order to timely and differentially treat hemodynamic disorders. In the diagnosis of chronic disorders of cerebral circulation, a significant place is occupied by neuroimaging methods, in particular, computer and magnetic resonance imaging. However, some authors believe that non-invasive transcranial Dopplerography is highly informative here, which makes it possible to determine the severity and nature of structural changes in intracranial vessels, as well as their localization, and to quantify the state of blood flow in the basin of the main cerebral arteries [3, 5].

Conclusion. Thus, in chronic cerebrovascular disease, a steadily progressing atherosclerotic process is accompanied by a decrease in blood flow velocity in the main arteries of the head. At the same

time, changes in LBF are detected by transcranial Doppler sonography on earlier Doppler sonography. Both at the extra- and intracranial levels, depression of blood flow initially occurs in the arteries of the vertebrobasilar basin, and later LBF decreased in the carotid bed. The detection of changes in the Doppler study generally preceded the increase in symptoms of an organic lesion of the nervous system, and in the vast majority of cases, the localization of neurological symptoms corresponded to the pool of reduced blood flow velocity parameters. This pattern is more pronounced in the II stage of DCE, when the leading symptom complex is isolated in the clinical picture.

References

1. Ammirati F. Diagnosing syncope in clinical practice // Eur. Heart J. 2000. Vol. 21, No. 11. P. 935-940.
2. Baumbach G.L., Heistad D. Cerebral circulation in chronic arterial hypertension // Hypertension. 1998. Vol. 12. P. 89-95.
3. Graham G.H. Tissue plasminogen activator for acute ischemic stroke in clinical practice. A Metaanalysis of safety date // Stroke. 2003. Vol. 34, No. 12. P. 2847-2850.
4. Shetty S.H., Lev M.H. CT perfusion // Acute Ischemic Stroke. Imaging and intervention / Gonsales R.G., Hirsch J.A., Koroshetz WJ. et al. (eds.). Berlin-Heidelberg: SpringerVertag, 2006.
5. Wintermark M., Sesay M., Barbier E. et al. Comparative overview of brain perfusion imaging techniques // Stroke. 2005. Vol. 36. P. 8399.
6. Ходжиева Д.Т., Хайдаров Н.К. Хайдарова Д.К. Коррекция астеноневротического синдрома энергокорректоромцитофлавином // Неврология. – Ташкент, 2013. - №3. С.16-19.,
7. Ходжиева Д.Т., Хайдарова Д.К., Хайдаров Н.К., Самадов А.У. Дифференцированная терапия в остром периоде ишемического инсульта // Неврология – 2011. - № 4. - С. 34.
8. Рахматова С. Н., Саломова Н. К. Қайта Такрорланувчи Ишемик Ва Геморрагик Инсультли Беморларни Эрта Реабилитация Қилишни Оптималлаштириш //Журнал Неврологии И Нейрохирургических Исследований. – 2021. – Т. 2. – №. 4.
9. Рахматова С. Н., Назарова Ж. А., Аликулова Н. А. Особенности когнитивного дефицита у больных с хронической ишемией мозга в зависимости от пола //Электронный инновационный вестник. – 2018. – №. 4. – С. 59-61.
10. Рахматова, С. Н. "Особенности клиники и течения дисциркуляторной энцефалопатии у мужчин и женщин." *Психическое здоровье* 12.5 (2014): 114-116.
11. Уринов М. Б., Ахророва Ш. Б., Рахматова С. Н. Сроки восстановления лицевого нерва при параличе Белла в зависимости от стороны поражения и пола //Психическое здоровье. – 2014. – Т. 12. – №. 10. – С. 67-69.
12. Cordonnier C., Sprigg N., Sandset E.C. et al. Women Initiative for Stroke in Europe (WISE) group, Nature Reviews // Neurology. 2017. Vol. 13 (9). P. 521-532.;
13. Coutts S.B. Diagnosis and Management of Transient Ischemic Attack // Continuum (MinneapMinn). – 2017. – Vol. 23 (1, Cerebrovascular Disease). – P. 82-92.
14. Spirin A.L. Supratentorial intracerebral hemorrhages: pathophysiological aspects and treatment tactics / A.L. Spirin, A.P. Trashkov, N.V. Gypsy // Pediatrics. 2015. T 6. No. 1. S. 96.
15. Bogoslovsky A.G. Influence of surgical treatment of hypertensive intracranial hematomas on the regression of secondary cerebral ischemia and the outcome of hemorrhagic stroke / A.G. Bogoslovsky // Proceedings of the All-Russian Scientific and Practical Conference “Polenov Readings”. SPb., 2005. S. 167.
16. Gafurov B.G. Modern principles of treatment of acute cerebral stroke / B.G. Gafurov //

Neurology. 2008. No. 3-4. pp. 50-51.

17. Dzhamgyrchieva A.A. Analysis of the outcomes of hypertensive intracerebral hemorrhages / A.A. Dzhamgyrchieva, K.B. Yrysov // Vestnik AGIUV. 2013. Special issue. No. 3. S. 23-25.Sembill J.A., Kuramatsu J.B., Gerner S.T. Hematoma enlargement characteristics in deep versus lobar intracerebral hemorrhage // Ann Clin Transl Neurol. 2020 Mar; 7 (3): 363-374. DOI:10.1002/acn3.51001.