CURRENT MEDICAL STATUS OF PSYCHOSOMATIC CONDITIONS AFTER HEMORRHAGIC STROKE. (Literature review)

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Summary. Stroke is one of the leading causes of seizures in adults. The study of factors included screening surgery, the risk of early and late post-stroke epileptic seizures, and a study of 300 patients aged 41-94 years. Data were studied on disease history and neuroimaging. This study was conducted on the basis of statistical data showing an increased risk of early epileptic seizures with hemorrhagic stroke.

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

Early diagnosis and treatment of cerebral strokes is one of the most important problems in modern neurosurgery and neurology. Non-traumatic intracerebral hemorrhages occur in 70-90% of cases and can develop in any phase of hypertension. Supratentorial non-traumatic hemorrhages occur in 85%, and subtentorial - in 15% of cases. Hemorrhages have a typical localization: in the subcortical nodes in 25-30% of cases, in the visual tubercle - in 10-20%, in the frontal, parietal, temporal and occipital lobe - in 20%, in the cerebellum - in 8-10%, in the bridge - in 5-7% and in the head of the caudate nucleus - in 5% of cases. In some cases, non-traumatic intracerebral hemorrhages develop in both hemispheres of the brain [1].

With the development of non-traumatic intracerebral hemorrhages, it is necessary to take into account a combination of three groups of factors: anatomical (acquired changes and / or congenital malformations of the blood vessels of the brain); hemodynamic (arterial hypertension); factors affecting blood clotting (taking anticoagulants and antiaggregants, blood diseases) [2].

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 [4].

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 [28].

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 [29].

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 [4] .

As you know, diabetes mellitus (DM) is one of the leading causes of CCI and stroke. At the same time, diabetes is one of the central places among the risk factors for stroke. Meanwhile, to date, the question of the effect of DM on the incidence of post-stroke epilepsy remains open. According to some authors, epileptic seizures in DM occur 2 times more often than in people with normal carbohydrate metabolism. Others indicate that there is no effect of DM on the incidence of epilepsy.

Early post-stroke seizures (EP) that occur in the first 7 days of a stroke develop as a result of cytotoxic and metabolic disorders in the focus of ischemia, their effects on intact functioning neurons, in which critical depolarization shifts and discharge activity are formed, and stop immediately after stabilization of metabolic processes [5]. A certain role more often as predictors of impaired activity of antiepileptic systems is played by secondary changes: the severity of edema and plethora, the suddenness factor [6]. Late post-stroke seizures (PP) are based on similar pathogenetic mechanisms, but they are more often the result of spontaneous activity of damaged neurons located in the area adjacent to the post-ischemic cyst, gliosis, or cortical atrophy. They occur within 1 month to 1 year (sometimes later) after a cerebrovascular accident and often indicate the onset of post-stroke epilepsy (PIE).

Both RP and PP significantly worsen the prognosis, increase the risk of death and disability in stroke [1]. Various aspects of the ratio of RP and PP in patients with stroke, risk factors for their occurrence and prognostic significance are currently the subject of active study.

A study was conducted, the purpose of which was to identify risk factors for the development of convulsive seizures in the acute period of stroke.

Indicators of the frequency of repeated non-traumatic intracerebral hemorrhages are not large. Some authors found recurrent hemorrhage in 53 (5.4%) of 989 patients within 1.5-72 months after the primary one, and in 3 of them (5.7%) after secondary

hypertensive hemorrhage. In 34% of cases, repeated hemorrhage occurred during the first year and in 32.1% - during the second year after the primary hemorrhage. The localization of repeated intracerebral hematomas in all patients differed from the primary hemorrhage: in 26.8% of cases, the hemorrhage was in the thalamus, in 21.4% - in the subcortical nuclei, in 3.7% - in the lobe of the cerebral hemispheres. The hematoma volume in all patients was larger than the primary one [30].

Conclusion. Thus, summing up the analysis of modern scientific literature, we can conclude that the problem of surgical treatment of intracerebral hemorrhages is still far from being completed. In this regard, the issue of developing new minimally invasive methods for removing intracerebral hematomas aimed at improving the results of treatment of patients remains relevant.

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