Clinical Neurological and Immunological Features of Vascular Seizures in Hemorrhagic Stroke

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ABSTRACT

This is due to the etiological, pathogenetic and clinical heterogeneity of ischemic stroke, as well as the presence of patients with concomitant diseases that aggravate the course of stroke and prevent the directed action of neuroprotective agents. 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, Arterial Hypertension, Epilepsy, Vascular Factor, Risk Factor, Neuroclinical Signs

This literature review analyzes the reasons for the diagnostic difficulties of nonclassical manifestations in order to accurately and timely diagnose the acute period. Although the literature review focuses on the acute period of ischemic stroke, certain aspects of diagnostic errors in hemorrhagic strokes, including intracerebral hemorrhages (IUDs), subarachnoid hemorrhages (SAH) and hemorrhages as a result of venous thrombosis and cerebral sinuses, are also discussed. (TCVS).

According to recent epidemiological studies, the incidence of epilepsy in patients over 60 years of age in 2006 reached 104, and in 2020 - 127.2 cases per 100,000 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].

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 mainly of the 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, DM 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 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 ischemic focus, their effects on intact functioning neurons, in which critical depolarization shifts and discharge activity are formed, and stop immediately after stabilization. ¬zatsii 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.

Cerebral stroke is usually characterized by the sudden development of focal neurological deficits in the form of hemiparesis, aphasia or hemianopsia, depending on the localization of foci of brain damage or the involved vascular basin. In some cases, the clinical manifestations of cerebral strokes can be expressed by fuzzy focal deficits, as well as diffuse neurological symptoms.

These manifestations include, first of all, neuropsychiatric disorders.

According to the literature, these neuropsychic symptoms are differentiated into the following clinical forms: acutely developing confusion of consciousness, quantitatively altered level of consciousness [4, 8, 11]. In 3% of patients, cerebral stroke in the acute period is manifested by mental disorders in the form of delirium, delirium, acutely developed dementia or mania, imitating the clinic of mental illness. In this case, focal neurological disorders are often absent or are mild, transient, so they are easy to miss. Similar symptoms are usually observed in patients with focal stroke in the frontal or parietal region of the right (non-dominant) hemisphere. Some focal MI-related symptoms, such as anosognosia, aphasia, akinetic mutism, abulia, and aprosody, may be misinterpreted by medical practitioners as manifestations of depression. For example, patients with MI localization in the right frontal or parietal region are unable to correctly perceive and express the appropriate emotional intonations due to aprosody, their speech is monotonous, and therefore an erroneous diagnosis of affective disorders is established in such a patient.

Cerebral stroke in the area of the caudate nucleus in the pool of blood supply to the anterior lenticulostriatal arteries is often manifested only by worn out neuropsychic or behavioral disorders, such as abulia, mental and emotional inertia, decreased or lack of initiative (motivation) of motor activity in conversation and normal daily activities. Similar signs are observed in patients with isolated MI in the frontal lobes and subcortical structures; they are caused by damage to the limbic-frontal pathways and their connections with the optic tubercle. Patients with right-sided focal lesions of the orbitofrontal cortex, visual tubercle and temporoparietal region often develop manic states accompanied by psychosis. Complex partial epileptic seizures caused by the localization of MI in the temporal lobes are often accompanied by psychotic disorders in many patients. Violent laughing and crying, as well as inappropriate situations of uncontrolled fits of laughing and crying, are common consequences of MI, although they are relatively rare. These symptoms usually occur

in MI associated with bilateral lesions of the supranuclear motor tracts, in the area of the pons, basal ganglia or periventricular subcortical regions, basal parts of the frontal or parietal lobes. Such psychoemotional disorders as despair and hopelessness, anxiety, aggression and refusal of treatment are also not uncommon in patients with carotid stroke (especially when the subcortical parts of the entire hemisphere are affected).

Acutely developing confusion of consciousness often accompanies the clinic of delirium. In some patients, delirium may be the initial manifestation of cerebral stroke, especially when it is hemispheric. These mental disorders are more often observed with hemorrhagic than with ischemic stroke. MI localized in the right temporal gyrus, right inferior parietal lobe or occipital lobe are manifested by acute psychotic states, confusion, agitation, anxiety and erased neurological symptoms, as a result of which delirium is often mistakenly diagnosed in such patients. Acute ischemia in the vertebrobasilar basin, leading to damage to the optic hillock, especially its paramedian nuclei, sometimes manifests itself in an inexplicably rapid development of depression of consciousness, followed by semantic amnesia and minimal neurological deficit, which often suggests an acute mental pathology. This form of amnesia should be distinguished from transient global amnesia (sudden transient loss of memory for recent events and impairment of the ability to store new information with normal results of neurological examination) [5, 16].

A stroke in the corpus callosum is manifested by symptoms of interhemispheric dissociation, as a result of which patients are diagnosed only with a state of confusion. MI patients with predominantly receptive aphasia are also often mistaken for confusion. Patients with semantic aphasia sometimes give the impression of confusion due to the difficulty of verbal communication. In such cases, it is difficult to identify the presence of hemianopsia in a patient, especially without special testing, to study in detail the function of speech, to conduct perimetry. The presence of a vascular history, clear consciousness in such patients, acute development of neurological disorders undoubtedly facilitate the establishment of the correct clinical diagnosis in favor of the vascular nature of the process.

Acute disorders of cerebral circulation, accompanied by bilateral damage to the primary visual associative zone, are often manifested by visual agnosia, prosoagnosia or anosognosia. These visual disturbances are difficult to diagnose with insufficient experience with a medical practitioner and can be mistaken for a state of confusion. A classic example of such disorders is Anton's syndrome, which occurs with bilateral occipital cerebral infarction, manifested by cortical blindness and is characterized by a denial of the fact of blindness with fantastic responses. The literature describes Balint's syndrome, which is also caused by bilateral occipital-parietal MI, which is characterized by impaired visual perception and inability to recognize more than one object at a time [3].

The altered level of consciousness in patients with MI in the form of a rapid decrease in the level of consciousness and lack of response to external stimuli is the initial manifestation of extensive cerebral strokes, especially hemorrhagic ones, caused by a rapid increase in intracranial pressure. These signs can be a manifestation of ictal or postictal unresponsiveness that developed after an epileptic seizure [13]. Noteworthy are two unique pathological syndromes observed in cerebral strokes localized in the vertebrobasilar basin. In the first case, with embolic occlusion of the central artery of Percheron (a variant of arterial blood supply, in which the medial perforating arteries of the optic tubercle or rostral perforating arteries are affected), causing infarction of these areas, patients are admitted in a state of cerebral coma, other neurological disorders they are often absent. The second syndrome, described in the literature as a syndrome of the distal part of the basilar artery, is caused by embolic occlusion of the distal part of the basilar artery in the place where it branches into the posterior cerebral arteries. In patients with MI in this area, upon admission to the hospital, as a rule, there is no consciousness, there is

quadriplegia, and sometimes urinary and fecal incontinence. In this case, such signs as pathology on the part of the pupils (gross miosis) or oculomotor disorders (floating movements of the eyeballs, often bilateral), which are detected in more than 40% of patients, are of diagnostic value [12].

Traditionally, it is believed that a cerebral stroke is most often accompanied by a loss of motor functions. Nevertheless, in a small number of observations in the initial period of MI, patients often have various dyskinesias (hyperkinesias, hypokinesis, or seizure-type motor disorders).

Various types of dysinesia observed in the acute period of MI are described in the literature. These include dystonia, chorea, athetosis, tremor, myoclonus, convulsive twitching, tremors, and asterixis. In the registry of cerebral strokes in Lausanne (Switzerland), the prevalence of movement disorders in 2500 patients with acute stroke was 1%, with hemichorea, hemibalism and dystonia being the most common extrapyramidal symptoms. Small subtentorial cerebral strokes with involvement of the basal ganglia in the pathological process were more often associated with dyskinesias. According to other researchers, in patients with cerebral stroke and dyskinesia, there is no connection between dyskinesia and the affected vascular system, the side of the stroke, or its subtype. The development of dyskinesias at the onset of the disease occurs against the background of atherosclerosis with damage to large intracerebral vessels, cardiogenic embolism, intracerebral hemorrhage, damage to the optic hillock, cerebellum and brain stem [2, 11].

J. Handley et al. From 1966 to 2008, we analyzed 2942 works devoted to the study of post-stroke movement disorders, and came to the conclusion that dystonia, chorea, and hemibalism are most often caused by MI in the area of the basal ganglia, tremor most often develops with damage to the posterior parts of the optic tubercle or dentorubrothalamic tract, MI in the area of the striatum or lenticular nuclei cause parkinsonism. M. Ghika-Schmid et al. (2007) reported that a syndrome characterized by muscular dystonia, abrupt movements, feeling of a “clumsy” hand, is specifically associated with minor strokes in the area of blood supply to the posterior choroidal artery. Myoclonus is most often observed with the localization of strokes in the vertebrobasilar basin. Segmental myoclonus has also been described in strokes in the midbrain and pons; palatine myoclonus (regular rhythmic contractions of the soft palate) is the only manifestation of lacunar stroke in the pons [10].

There are also reports of lacunar infarction with isolated lesions of the vestibular nuclei. In this case, vestibular disorders are accompanied by a more pronounced change in gait and other neurological manifestations (sensory, cochlear disorders), which is taken into account when differentiating with acute vestibular syndrome of peripheral genesis. To differentiate oculomotor disorders, determining the true cause of the disease, a combination of negative results of the head push test with simultaneous deviation of the eyeballs and nystagmus (changing direction or vertical), confirming the central genesis of oculomotor dysfunction can help.

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