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Clinical And Etiological Factors Of Atopic Stroke (Literature Review)

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Abstract. Vascular diseases of the brain are an urgent problem of modern medicine and determine its independent direction - angioneurology. In turn, in the structure of cerebrovascular pathology, the leading place is occupied by cerebral strokes (MI). This is due to the continuing trend towards their constant growth in the world.

Keywords. symptoms, cerebral stroke, atypical forms, hyperacusis.

Introduction

Every year in the world about 15 million people suffer a cerebral stroke, of which 5 million die, and 5 million patients remain with persistent neurological deficits. In many parts of the world, stroke is the most common cause of disability and the third most common cause of death, according to WHO. The development of acute neurological symptoms observed in the clinic of cerebral stroke dictates the need for practitioners to carry out differential diagnostics with other similar diseases of the central nervous system (CNS). Timely diagnostics of MI is extremely important, since it is it that determines the period of urgent medical correction [4, 6].

According to a number of researchers, all patients with acute vascular pathology admitted to a neurological hospital can be conditionally divided into four groups. The first group includes patients with obvious cerebral stroke, for example, elderly people with untreated atrial fibrillation, in the diagnosis and clinical course of atypical forms of MI, which is the focus of this review.

Atypical symptoms observed with MI, who suddenly develop aphasia and hemiparesis. The second group includes patients who raise doubts about acute cerebrovascular pathology, for example, patients without vascular risk factors with unilateral weakness of the facial muscles in combination with hyperacusis and persistent taste changes in the presence of paresis of the facial muscles of the lower half of the face. The third group includes patients with suspected cerebral stroke, but other vascular diseases are possible, such as conversion disorders, Todd's palsy (a syndrome of prolonged motor

disorders after an epileptic seizure, with or without aphasia) or hemiplegic migraine. Among other pathological conditions similar to cerebral stroke, the most common are hypoglycemia, syncope, lipotimia, Meniere's syndrome [1, 3, 9, 19]. When carrying out thrombolysis, special attention is paid to the differential diagnosis of stroke. In this regard, a group of patients with acute cerebral stroke with unusual or atypical clinical manifestations is of great practical interest. D. Huff called such cases of cerebral strokes "chameleon strokes" [12]. Much less attention is paid to missed or untimely diagnosed cerebral strokes; therefore, it is quite reasonable to generalize the literature data on the issues of diagnostics and clinical course of atypical forms of MI, which is the focus of this review.

The atypical symptoms observed with MI arise for various reasons. Firstly, in the first minutes or hours of the development of MI at the prehospital stage, all the complete medical information necessary for the correct clinical diagnosis is often lacking. In addition, it is necessary to take into account the fact that neurological symptoms in patients with MI can develop for a prolonged period of time ("stroke in progress"). Second, the development of nonclassical manifestations of MI is often associated with significant (anatomical) variability of the classical basins of cerebral blood supply. It is clinically more difficult to diagnose lacunar stroke in patients with small lesions, in the early stages of cerebral stroke, in young people, with localization of cerebrovascular accidents in the vertebrobasilar system and in the absence of lateralization of motor or speech deficits.

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).

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, 26].

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 [13].

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 [23]. 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 [22].

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, 14, 21].

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].

Often, in the presence of involuntary, recurring hyperkinesis of the extremities, practical doctors diagnose partial motor epileptic seizures and do not diagnose MI in a timely manner. Minor cerebral strokes in the area of the base of the pons are clinically manifested by involuntary tonic spasms and contralateral hemiparesis. Similar clonic limb movements, reminiscent of convulsions or freezing states, are also observed in deep strokes with localization in the region of the brain stem and the optic hillock [15, 24, 27]. These abnormal movements are associated with dysfunctions of the corticospinal tract (descending inhibitory fibers that affect the motor neurons of the anterior horns, motor neurons of the affected limbs). Such movement disorders are sometimes observed in patients with the syndrome of damage to the distal basilar artery and are often mistakenly interpreted as status epilepticus. In such cases, the presence of concomitant oculomotor disorders, more often bilateral, the absence of typical epileptiform discharges on the EEG during paroxysms of dyskinesias serve as additional help in making the correct clinical diagnosis. Knowledge of such motor disorders will undoubtedly facilitate the early and timely diagnosis of MI, as well as adequate therapy.

In the acute period of cerebral stroke, epileptic seizures are also common, which occur with a frequency of 1.5 to 5.7% of observations, according to studies. Epileptic seizures, as the onset of cerebral stroke, are usually observed in young people, more often against the background of intracerebral hemorrhages, with cortical infarctions, as well as with their localization in the zone of adjacent vascularization in the basin of the internal carotid artery [3, 5]. As indicated earlier, it is very important for clinicians to differentiate motor deficits caused by stroke and accompanied by seizures at the onset of the disease from Todd's postictal palsy. It is rather difficult to carry out such a differential diagnosis on the basis of a single clinical examination in the first minutes and hours of the development of the disease. It is necessary to use modern methods of neuroimaging - magnetic resonance angiography, positron emission tomography, perfusion magnetic resonance imaging (MRI).

The prevalence of epileptic seizures is especially high with thrombosis of cerebral veins and sinuses, venous infarctions. So, in the course of an international study of patients with thrombosis of the cerebral veins and sinuses of the dura mater, epileptic seizures were recorded in 40% of cases. In the history of such patients, constant headaches and other signs of increased intracranial pressure (edema of the optic discs, etc.) were observed. One of the most unusual and atypical manifestations of MI is the so-called alien hand syndrome, in which one hand acts independently of the patient's voluntary control. This syndrome can be observed when MI is localized in the corpus callosum, frontal lobes or posterolateral part of the parietal lobe [13, 25]. Alien hand syndrome is the result of a disruption in the connections between the primary motor cortex, where the hand projects, and the premotor cortex. At the same time, the patients retain the ability to perform movements. Doctors who are unaware of this unusual syndrome interpret it as mental dysfunction. In the presence of the above syndrome, it is advisable to test for left- or right-handedness.

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.

Isolated or nearly isolated lesions of the cranial nerves due to an infarction in the area of the nuclei or damage to fibers when the nerve leaves the brainstem is rare, but still occurs. This refers to damage to the III and VII pairs of cranial nerves, which is observed in connection with systemic angiopathy against the background of diabetes mellitus, arterial hypertension, hyperlipidemia, complex vasculitis.

Simultaneous hearing loss and systemic dizziness suggest a peripheral genesis of these symptoms; with MI in the basin of the anterior inferior cerebellar artery, both hearing and vestibular function may suffer. Acute hearing loss is often associated with a labyrinth infarction when the labyrinth artery is damaged (thrombosis) [6, 7].

Acute monoparesis (isolated one-sided weakness of the muscles of the face, upper or lower limb) is also another atypical manifestation of MI. In two large studies with a total of 6805 patients, the incidence of monoparesis (to which one study also included isolated facial weakness) ranged from 2.5 to 4.1%. In some of these patients, the subcortical localization of MI was established.

Cortical hand syndrome is a classic but rare MI syndrome. Since the anatomical formation known as the "cortical tubercle of the hand" is large enough (relative to the number of anatomical structures served), a stroke in this area of the precentral gyrus, with its small size, can cause a very small deficit in volume, affecting only to a hand, several fingers or even one thumb. Given the fact that the clinic is dominated by the lesion of the radial or ulnar side, it is often in a similar situation that a dysogenous

lesion of the cervical spine with neuropathy of the radial or ulnar nerves is mistakenly diagnosed. In this case, a thorough study of complex types of sensitivity, including stereognosis, two-dimensional spatial feeling and kinesthetic sensitivity, is of diagnostic value. MI, manifested by the “cortical” hand syndrome, often result from arterio-arterial embolism against the background of ipsilateral atherosclerosis of the carotid artery or as a result of cardiogenic embolism [12, 18, 25].

Paresis of the hand or hand is much more common, while in a third of cases of repeated MI, monoparesis of the lower limb is observed. A classic example of this is the localization of a stroke in the anterior cerebral artery, when the medial surface of the precentral gyrus suffers. In most of these patients, a subtle weakness of the ipsilateral gastrocnemius muscle occurs, in some cases, the sensitivity on the paretic limb is impaired, which is revealed by a targeted sensitivity study. With subcortical localization of MI, monoparesis of both the upper and lower extremities is often observed. Involvement in the pathological process mainly of the lower limb is also characteristic of cerebral strokes with localization in the basin of the middle cerebral artery, IUD and hemorrhages due to CVT [16].

Finally, similar to the “cortical” hand syndrome, in a number of cases with ischemic and hemorrhagic strokes, the “cortical” foot syndrome develops. In patients with this syndrome, isolated sagging of the feet is observed, which mimics the defeat of the peroneal nerve.

We present the following clinical observations of MI with pseudoperipheral movement disorders.

Clinical observation

Patient M., 51 years old, was admitted to the clinic of nervous diseases on 01/20/11 with complaints of headaches, dizziness associated with an increase in blood pressure, weakness in the left hand, especially in the IV and V fingers, restriction of movement in them, inability to squeeze hand in a fist, numbness along the outer edge of the hand.

Has been sick for 1.5 months. He fell ill acutely, woke up in the morning and noticed weakness in the fingers of his left hand. The disease does not associate with anything, however, the history of hypertension for more than 10 years, high blood pressure was also in the patient's mother, who died from intracerebral hemorrhage. I went to the doctor at the place of residence, and was diagnosed with neuropathy of the left radial nerve. After the course of treatment, the patient did not notice any improvement in his condition and was subsequently referred for a consultation to the regional polyclinic, where he was admitted to the neurological department of OKB No. 2. He smokes. In 1986, he was involved in emergency rescue operations at the Chernobyl nuclear power plant. From the transferred diseases notes chronic gastroduodenitis, chronic non-obstructive bronchitis, ischemic heart disease.

On admission: average height, decreased nutrition, blood pressure 160/90 mm Hg, pulse 92 beats / min, rhythmic.

In the neurological status: asthenized, frequent insomnia, cerebral microsymptomatics in the form of flattening of the left nasolabial fold, positive reflexes of oral automatism. Tendon and periosteal reflexes from the upper and lower extremities are revived, S> D, movements in the fourth and fifth fingers on the left are limited - difficulty in clenching the hand into a fist, positive Venderovich test on the left, cannot move the left little finger on the table, hypesthesia along ulnar type on the left, muscle strength in the fourth and fifth fingers of the left hand is reduced to 3 points.

Examinations: clinical blood and urine tests: no pathology.

ECG left ventricular hypertrophy, diffuse changes in the myocardium, rhythm 88 / min.

Doppler ultrasound of extra-, intracranial vessels: signs of angioedema in the carotid basin on the right, obstructed venous outflow.

Ophthalmologist: fundus angiopathy.

ENMG of the upper extremities: weakening of supraspinal influences at the level of the cervical thickening. There are no signs of peripheral nerve damage.

MRI of the cervical spine: moderate manifestations of osteochondrosis.

MRI of the brain: multiple lacunar strokes in the basal parts of the frontal lobes, pronounced ventriculomegaly.

Clinical diagnosis: ischemic lacunar stroke (November, 2010) in the basal regions of the right frontal lobe, severe paresis of the left ulnar hand as a manifestation of grade III hypertension; hypertensive encephalopathy of the III degree, pronounced venous-cerebrospinal fluid circulation, pronounced asthenovegetative syndrome.

Our patient had systemic angiopathy, which developed against the background of arterial hypertension and possible radiation sclerosis, since the patient was in the exclusion zone at the Chernobyl nuclear power plant, which led to the formation of a lacunar state, according to MRI data. The acute development of the disease with the appearance of paresis of the fingers of the left hand testifies in favor of an acute cerebrovascular syndrome in the form of a lacunar stroke. Differential diagnosis was carried out at the initial stage of the disease with ischemic neuropathy of the left ulnar nerve. An increase in physiological reflexes on the diseased side, the presence of multiple subcortical foci on MRI, and the absence of signs of damage to the peripheral motor neuron, according to the ENMG data of the upper extremities, testify against peripheral monoparesis. Thus, in our clinical observation, a lacunar stroke in the basal region of the right frontal lobe clinically manifested itself as isolated paresis of the fourth and fifth fingers of the left hand.

Conclusions

The issues raised in this review indicate that almost any neurological symptom can be explained by acute cerebrovascular accident. The cited literature data facilitate the differential diagnosis of cerebral

strokes and diseases similar to them. They indicate a discrepancy between the classic symptoms of cerebral stroke, confirming the diagnosis, and atypical ones.

A valuable clinical feature of MI is the concept of comparing the symptoms of "irritation" and symptoms of "prolapse". Most often, with cerebral strokes, symptoms of "loss" prevail (that is, loss of motor functions, sensitivity, vision). For example, with regard to the motor sphere, hyperkinesis will be symptoms of "irritation", while muscle weakness and the resulting decrease or cessation of movements are symptoms of "prolapse". In relation to the visual sphere, photopsies are symptoms of "irritation" of the occipital cortex, and blindness is a symptom of its "loss".

Thus, the manifestations of cerebral strokes are heterogeneous, as are their causes. Under the right clinical circumstances, four principles can help the practitioner make a correct diagnosis. First, MI should be suspected in any case of sudden onset of neurological symptoms. Secondly, it is necessary to be aware of these rare and atypical manifestations of cerebral strokes. Thirdly, one should take it as a rule to conduct the most deep neurological examination of patients with acute neurological symptoms. Fourth, the mandatory use of neuroimaging methods.

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