IJCNP, Volume 3, Issue 6, 2025 ISSN: 2995-536X
https://medicaljournals.eu/index.php/IJCNP

INTERNATIONAL JOURNAL OF COGNITIVE NEUROSCIENCE AND PSYCHOLOGY

Atypical Manifestations of Cerebral Strokes

Kilichev Nurbek Boliyor ogli
Clinical Resident, Department of Neurology
Samarkand State Medical University

Abdiqakhhorov Elnur Sodiq ogli
Clinical Resident, Department of Neurology
Samarkand State Medical University

Ergashev Mukhriddin Boriboj ogli
Clinical Resident, Department of Neurology
Samarkand State Medical University

Ergashev Dilshod Utkir ogli
Clinical Resident, Department of Neurology
Samarkand State Medical University

Kasimov Arslanbek Atabaevich
PhD, Associate Professor, Department of Neurology
Samarkand State Medical University

Abstract: Cerebrovascular diseases are an urgent problem of modern medicine and define an independent direction - angioneurology. In turn, cerebral strokes (CS) occupy a leading place in the structure of cerebrovascular pathology. This is associated with the persistent global trend of their constant growth.

Key words: cerebral strokes, atypical clinical manifestations.

Introduction.

Annually, approximately 15 million people worldwide suffer from cerebral stroke, of which 5 million die, and 5 million patients remain with persistent neurological deficit. In many countries worldwide, cerebral stroke is the most common cause of disability and the third most frequent cause of death, according to WHO data. The development of acute neurological symptoms observed in the clinic of cerebral stroke necessitates that practicing physicians conduct differential diagnosis with other similar diseases of the central nervous system (CNS). Timely diagnosis of CS is extremely important, as it determines the timing of emergency drug 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 who suddenly develop aphasia and hemiparesis. The second group includes patients who raise doubts regarding acute cerebrovascular pathology, for example, patients without vascular risk factors with unilateral weakness of facial muscles combined with hyperacusis and persistent taste changes in the presence of paresis of facial muscles of the lower half of the face. The third group includes patients with presumed cerebral stroke, however, other vascular diseases are also possible, such as conversion disorders, Todd's paralysis (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 frequent are hypoglycemia, syncope, lipothymia, Meniere's syndrome [1, 3, 9]. When conducting thrombolysis, special attention is paid to differential diagnosis of stroke. In this regard, a group of patients with acute cerebral stroke having unusual or atypical clinical manifestations is of great practical interest. D. Huff called such cases of cerebral strokes "chameleon strokes".

Much less attention is paid to missed or untimely diagnosed cerebral strokes, therefore it is quite justified to generalize literature data on the features of diagnosis and clinical course of atypical forms of CS, which is the focus of this review.
Atypical symptoms observed in CS arise for various reasons. First, in the first minutes or hours of CS development at the prehospital stage, complete medical information necessary for making the correct clinical diagnosis is often absent. In addition, it is necessary to consider the fact that neurological symptoms in patients with CS may develop prolonged over time ("stroke in progress"). Second, the development of non-classical manifestations of CS is often associated with significant (anatomical) variability of classical blood supply basins of the brain. It is clinically more difficult to establish a diagnosis of lacunar stroke in patients with small lesion sizes, in the early stages of cerebral stroke, in young people, when circulatory disorders are localized in the vertebrobasilar system and in the absence of lateralization of motor or speech deficit.
This literature review analyzes the causes of diagnostic difficulties of non-classical manifestations with the aim of accurate and timely diagnosis of the acute period. Although the main attention in the literature review is devoted to the acute period of ischemic stroke, certain aspects of diagnostic errors in hemorrhagic strokes are also addressed, including intracerebral hemorrhages (ICH), subarachnoid hemorrhages (SAH) and hemorrhages resulting from cerebral venous and sinus thrombosis (CVST).
Cerebral stroke is usually characterized by sudden development of focal neurological deficit in the form of hemiparesis, aphasia or hemianopsia depending on the localization of brain lesions or the involved vascular basin. In individual cases, clinical manifestations of cerebral strokes may be expressed by unclear focal deficit, as well as scattered neurological symptoms.
Such manifestations include primarily neuropsychiatric disorders.
According to literature data, these neuropsychiatric symptoms are differentiated into the following clinical forms: acutely developing confusion, quantitatively altered level of consciousness [4, 8]. In 3% of patients, cerebral stroke in the acute period manifests as psychiatric disorders in the form of delirium, delusions, acutely developed dementia or mania, mimicking the clinic of psychiatric diseases. Focal neurological disorders are often absent or mild and transient, so they are easily missed. Such symptoms are usually observed in patients with focal stroke in the frontal or parietal region of the right (non-dominant) hemisphere. Some focal symptoms related to CS, such as anosognosia, aphasia, akinetic mutism, abulia and aprosody, may be incorrectly interpreted by practicing physicians as manifestations of depression. For example, patients with CS localization in the right frontal or parietal region are unable to correctly perceive and express appropriate emotional intonations due to aprosody, their speech is monotonous, in connection with which such patients are erroneously diagnosed with affective disorders.
Cerebral stroke in the area of the caudate nucleus in the blood supply basin of the anterior lenticulostriate arteries often manifests only with subtle neuropsychiatric or behavioral disorders, such as abulia, mental and emotional inertia, decreased or absent initiative (motivation) of motor activity in conversation and usual daily activities. Similar signs are observed in patients with isolated CS in the frontal lobes and subcortical structures; they are caused by damage to limbic-frontal pathways and their connections with the thalamus. In patients with right-sided focal lesions of the orbitofrontal cortex, thalamus and temporo-parietal region, manic states often develop, accompanied by psychosis. Complex partial epileptic seizures caused by CS localization in the temporal lobes are often accompanied by psychotic disorders in many patients.
Pathological laughter and crying, as well as inappropriate situations of uncontrolled attacks of laughter and crying are common consequences of CS, although they are observed relatively rarely. These symptoms usually occur in CS associated with bilateral damage to supranuclear motor pathways, in the area of the pons, basal ganglia or periventricular subcortical areas, 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 with involvement of subcortical parts of the entire hemisphere).
Acutely developing confusion often accompanies the clinic of delirium. In individual patients, the initial manifestation of cerebral stroke may be precisely delirium, especially with its hemispheric localization. These psychiatric disorders are more often observed in hemorrhagic than in ischemic stroke. CS with localization in the right temporal gyrus, right inferior parietal lobe or occipital lobe manifest as acute psychotic states, confusion, agitation, restlessness and subtle neurological symptoms, consequently delirium is often erroneously diagnosed in such patients. Acute ischemia in the vertebrobasilar basin, leading to thalamic damage, especially its paramedian nuclei, sometimes manifests as inexplicably rapid development of consciousness depression with subsequent semantic amnesia and minimal neurological deficit, which often gives reason to suspect acute psychiatric pathology. This form of amnesia should be

distinguished from transient global amnesia (sudden transient loss of memory for recent events and impaired ability to retain new information with normal neurological examination results) [5, 6].

Stroke in the area of the corpus callosum manifests with symptoms of interhemispheric disconnection, consequently only confusion is diagnosed in patients. Patients with CS who have predominantly receptive-type aphasia are also quite often erroneously perceived as patients with confusion. Patients with semantic aphasia due to the complexity of speech contact sometimes give the impression of having confusion. In such cases, it is difficult to identify the presence of hemianopsia in the patient, especially without special testing, to thoroughly examine speech function, and to perform perimetry. The presence of vascular history, clear consciousness in such patients, and acute development of neurological disorders undoubtedly facilitate making the correct clinical diagnosis in favor of the vascular nature of the process.

Acute cerebrovascular disorders accompanied by bilateral damage to the primary visual associative zone often manifest as visual agnosia, prosopagnosia or anosognosia. These visual disorders are difficult to diagnose with insufficient experience of the practicing physician and can be erroneously taken for confusion. A classic example of such disorders is Anton's syndrome, occurring with bilateral occipital brain infarction, manifesting as cortical blindness and characterized by denial of blindness with fantastic answers. The literature describes Balint's syndrome, also caused by bilateral occipito-parietal CS, which is characterized by impaired visual perception and inability to recognize more than one object simultaneously [10].

Altered level of consciousness in patients with CS in the form of rapid decrease in consciousness level and absence of reaction to external stimuli is the initial manifestation of extensive cerebral strokes, especially hemorrhagic ones, caused by rapid increase in intracranial pressure. These signs may be manifestations of ictal or postictal unresponsiveness that developed after an epileptic seizure [3]. Two unique pathological syndromes observed in cerebral strokes localized in the vertebrobasilar basin deserve attention. 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 thalamus or rostral perforating arteries are affected), causing infarction of these areas, patients arrive in a state of cerebral coma, other neurological disorders are often absent in them. The second syndrome, described in the literature as distal basilar artery syndrome, is caused by embolic occlusion of the distal part of the basilar artery where it branches into the posterior cerebral arteries. Patients with CS in this zone upon admission to the hospital, as a rule, lack consciousness, have quadriplegia, sometimes urinary and fecal incontinence. Diagnostic significance is provided by such signs as pupillary pathology (severe miosis) or oculomotor disorders (floating eye movements, often bilateral), which are detected in more than 40% of patients [2].

It is traditionally believed that cerebral stroke is most often accompanied by motor function loss. Nevertheless, in a small number of observations in the initial period of CS, patients often have various dyskinesias (hyperkinesias, hypokinesias or motor disorders of the convulsive type).

The literature describes various types of dyskinesias observed in the acute period of CS. These include dystonia, chorea, athetosis, tremor, myoclonus, convulsive twitches, limb trembling and asterixis. In the stroke registry of Lausanne (Switzerland), the prevalence of movement disorders in 2500 patients with acute stroke was 1%, with hemichorea, hemiballism 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 dyskinesias, there is no connection between dyskinesia and the affected vascular basin, stroke side, or its subtype. The development of dyskinesias at disease onset occurs against the background of atherosclerosis with involvement of large intracerebral vessels, cardiogenic embolism, intracerebral hemorrhages, involvement of the thalamus, cerebellum and brainstem [2, 4].

J. Handley et al. analyzed 2942 works devoted to the study of post-stroke movement disorders from 1966 to 2008 and concluded that dystonia, chorea and hemiballism are most often caused by CS in the area of the basal ganglia, tremor most often develops with involvement of the posterior parts of the thalamus or dentatorubrothalamic pathways, CS in the area of the striatum or lentiform nuclei cause parkinsonism. M. Ghika-Schmid et al. (2007) reported that the syndrome characterized by muscle dystonia, jerky movements, feeling of "clumsy" hand is specifically associated with small strokes in the blood supply zone of the posterior choroidal artery. Myoclonus is most often observed with stroke localization in the vertebrobasilar basin. Segmental myoclonus is also described in strokes in the area of the midbrain and pons, palatal myoclonus (regular rhythmic contractions of the soft palate) is the only manifestation of lacunar stroke in the pons area [10].

Often, in the presence of involuntary, repetitive hyperkinesias of the limbs, practical physicians diagnose partial motor

epileptic seizures and do not timely diagnose CS. Small cerebral strokes in the area of the pons base clinically manifest as involuntary tonic spasms and contralateral hemiparesis. Such clonic limb movements resembling convulsions or freezing states are also observed in deep strokes localized in the brainstem and thalamus [1, 5]. These abnormal movements are associated with dysfunction of the corticospinal tract (descending inhibitory fibers that influence anterior horn motor neurons, motor neurons of affected limbs). Such movement disorders are sometimes observed in patients with distal basilar artery syndrome and are often erroneously interpreted as status epilepticus. In such cases, the presence of concomitant oculomotor disorders, more often bilateral, absence of typical epileptiform discharges on EEG during dyskinesia paroxysms serve as additional support in making the correct clinical diagnosis. Knowledge of such motor disorders will undoubtedly facilitate early and timely diagnosis of CS, as well as adequate therapy.

Epileptic seizures are also frequent in the acute period of cerebral stroke, occurring with a frequency of 1.5 to 5.7% of observations, according to conducted studies. Epileptic seizures as the debut of cerebral stroke are usually observed in young people, more often against the background of intracerebral hemorrhages, with cortical infarcts, as well as when localized in the zone of adjacent vascularization in the internal carotid artery basin [3, 5]. As mentioned earlier, it is very important for clinicians to differentiate motor deficit caused by cerebral stroke and accompanied by convulsions at the beginning of the disease from postictal Todd's paralysis. It is quite difficult to conduct such differential diagnosis based on clinical examination alone in the first minutes and hours of disease development. It is necessary to use modern neuroimaging methods - magnetic resonance angiography, positron emission tomography, perfusion magnetic resonance imaging (MRI).

The prevalence of epileptic seizures is especially high in cerebral venous and sinus thrombosis, venous infarcts. Thus, in an international study of patients with cerebral venous and dural sinus thrombosis, epileptic seizures were recorded in 40% of cases. In the history of such patients, constant headaches and other signs of increased intracranial pressure (optic disc edema, etc.) were observed.

One of the most unusual and atypical manifestations of CS is the so-called alien hand syndrome, in which one hand acts independently of the patient's voluntary control. This syndrome can be observed with CS localization in the area of the corpus callosum, frontal lobes or posterolateral part of the parietal lobe [13]. Alien hand syndrome results from disruption of connections between the primary motor cortex zone where the hand is projected and the premotor cortex. Patients retain the ability to perform movements. Physicians who do not know about this unusual syndrome interpret it as psychiatric dysfunction. In the presence of the above-described syndrome, it is advisable to conduct a test for left- or right-handedness.

There are also reports of lacunar infarction with isolated involvement of vestibular nuclei. In this case, vestibular disorders are accompanied by more pronounced gait changes and other neurological manifestations (sensory, cochlear disorders), which is taken into account when differentiating from acute vestibular syndrome of peripheral origin. For differentiation of oculomotor disorders, determining the true cause of the disease can help, combination of negative head thrust test results with simultaneous eye deviation and nystagmus (changing direction or vertical), confirming central origin of oculomotor dysfunction.

Isolated or almost isolated involvement of cranial nerves due to infarction in the area of nuclei or fiber damage at nerve exit from the brainstem is rare but still occurs. This relates to involvement of 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 impairment and systemic dizziness suggest peripheral origin of these symptoms; in CS in the anterior inferior cerebellar artery basin, both hearing and vestibular function may be affected. Acute hearing loss is often associated with labyrinth infarction with involvement of the labyrinthine artery (thrombosis) [6, 7].

Acute monoparesis (isolated unilateral weakness of facial muscles, upper or lower limb) is also another atypical manifestation of CS. In two large studies with a total of 6805 patients, the frequency of monoparesis occurrence (to which one study also included isolated facial muscle weakness) varied from 2.5 to 4.1%. Some of these patients had subcortical CS localization.

"Cortical" hand syndrome is a classic but rarely encountered syndrome in CS. Since the anatomical formation known as the "cortical hand knob" is quite large (relative to the number of anatomical formations served), a stroke in this area of the precentral gyrus with small size can cause very limited deficit, affecting only the hand, several fingers or even one thumb. Considering the fact that in the clinic the radial or ulnar side predominates, in such situations discogenic involvement of the cervical spine with radial or ulnar nerve neuropathy is often erroneously diagnosed. Thorough examination of complex types of sensitivity, including stereognosis, two-dimensional spatial sense and kinesthetic

sensitivity, has diagnostic significance. CS manifesting as "cortical" hand syndrome often arise as a result of artery-to-artery embolism against the background of ipsilateral carotid artery atherosclerosis or due to cardiogenic embolism [2].

Arm or hand paresis occurs much more frequently, while in one-third of cases of recurrent CS, lower limb monoparesis is observed. A classic example of this is stroke localization in the anterior cerebral artery basin, when the medial surface of the precentral gyrus is affected. Most such patients develop barely distinguishable weakness of the ipsilateral gastrocnemius muscle; in some cases, sensitivity is impaired in the paretic limb, which is detected during targeted sensitivity examination. With subcortical CS localization, monoparesis of both upper and lower limbs is often observed. Involvement of predominantly the lower limb in the pathological process is also characteristic of cerebral strokes with localization in the middle cerebral artery basin, ICH and hemorrhages due to CVST. Finally, similar to "cortical" hand syndrome, in some cases with ischemic and hemorrhagic strokes, "cortical" foot syndrome develops. Patients with this syndrome have isolated foot drop, which mimics peroneal nerve involvement.

References:

1. Ilkhomovna, K. M., Eriyigitovich, I. S., & Kadyrovich, K. N. (2020). Morphological Features of microvascular Tissue of the Brain at hemorrhagic stroke. The American Journal of Medical Sciences and Pharmaceutical Research, 2(10), 53-59.
2. Kadyrovich, K. N., Erkinovich, S. K., & Ilhomovna, K. M. (2021). Microscopic Examination Of Postcapillary Cerebral Venues In Hemorrhagic Stroke. The American Journal of Medical Sciences and Pharmaceutical Research, 3(08), 69-73.
3. Камалова, М. И., & Хайдаров, Н. К. (2020). Prevention and risk factors for brain infarction (literature review). Журнал неврологии и нейрохирургических исследований, 1(2).
4. Ismoilov, O. I., Murodkosimov, S. M., Kamalova, M. I., Turaev, A. Y., & Mahmudova, S. K. (2021). The Spread Of SARS-Cov-2 Coronavirus In Uzbekistan And Current Response Measures. The American Journal of Medical Sciences and Pharmaceutical Research, 3(03), 45-50.
5. Shomurodov, K., Khaidarov, N., & Kamalova, M. (2021). The formation and eruption of baby teeth in children. Збгрник наукових праць SCIENTIA.
6. Khodjieva D. T., Khaydarova D. K., Khaydarov N. K. Complex evaluation of clinical and instrumental data for justification of optive treatment activites in patients with resistant forms of epilepsy //American Journal of Research. USA. – 2018. – №. 11-12. – С. 186-193.
7. Kamalova M. I., Khaidarov N. K., Islamov S. E. Pathomorphological Features of hemorrhagic brain strokes //Journal of Biomedicine and Practice. – 2020. – С. 101-105.
8. Ershov V. I. The acute period of ischemic stroke: abstract of the dissertation of the Doctor of Medical Sciences/ V. I. Ershov.— Orenburg, 2007. — 88 p.
9. Odinak M. M. Cerebral ischemia. Neuroprotective therapy. Differential approach/M. M. Odinak, I. A. Voznyuk, S. N. Yanishevskii.— St. Petersburg, 2002. — 250 p.
10. Ilkhomovna K. M., Kadyrovich K. N., Eriyigitovich I. S. Clinical and demographicquality of life for patients with ischemic stroke in Uzbekistan //ACADEMICIA: An International Multidisciplinary Research Journal. – 2020. – Т. 10. – №. 10. – С. 883-889.
11. Kamalova M., Khaidarov N. Assessment of quality of life in ischaemic stroke patients //Збірник наукових праць Scientia. – 2021