




CASUISTIC PAPER

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Stroke masks – a case report

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ABSTRACT

Introduction. Stroke is a common vascular disease encountered in the work of a practitioner. Despite this, its differentiation is difficult. This is due to a variety of diseases that can be a “mask” for stroke. These are neurological diseases other than vascular (brain tumors, epilepsy), head injuries and a number of metabolic disorders (fluctuations in glucose, electrolytes).

Aim. This work aims to approximate the differentiation of this common disease entity. Case reports show both clinical view and diagnostic difficulties.

Description of the cases.

Case 1. The patient 59 years old treated for bipolar disorder, hypertension and alcohol abuse.

Case 2. The patient 68 years old treated for type 2 diabetes, hypertension, ischemic heart disease, cholecystolithiasis – cholecystectomy.

Conclusion. Stroke is a significant clinical and social problem both in Poland and in the world. Despite this, its diagnostics and especially differentiation is difficult. When analyzing the case of each patient with suspected sudden CNS vascular disease, all of them should be remembered. It should also be borne in mind that differential diagnosis cannot delay specific treatment for stroke.

Keywords. brain metabolic disorders, MRI, stroke

Introduction

Stroke is a significant clinical and social problem because it remains the first of the main causes of morbidity and long-term disability, and the second most frequent cause of death.¹ In the world, about 17 million people suffer from stroke every year, in Poland the figure is 90,000.² In

order to reduce the risk of stroke, it is necessary to eliminate risk factors for vascular diseases in the population of healthy people and to identify and effectively treat diseases that increase this risk.³ Fast implementation of effective treatment in the early stages of the disease such as thrombolytic and endovascular treatment (mechanical throm-

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bectomy) is of key importance for reducing the degree of disability and mortality after stroke.¹ Many non-vascular and metabolic neurological diseases can be associated with suddenly appearing focal neurological symptoms. These are, among others tumors, brain abscesses, seizures, migraine, glucose fluctuations, renal impairment, electrolyte imbalance, alcohol and drug effects, and injuries.⁴ Their differentiation is of key importance because the above-mentioned thrombolytic and endovascular treatment, apart from its effectiveness, carries a risk of complications. Intravenous thrombolytic therapy is associated with a 5- to 7-fold increase in the risk of secondary haemorrhage of a heart attack.¹ Medical history plays an important role in the diagnosis of people with sudden focal neurological symptoms. Provides information on the circumstances of illness (loss of consciousness, injury, contact with a person with an infectious disease), existing diseases (diabetes, kidney disease, epilepsy), lifestyle (alcohol, drugs). The next element should be the assessment of the patient's basic life functions: arterial pressure, pulse, saturation, body temperature, blood glucose level (test strip made of capillary blood), ECG.¹⁻⁷ Then, laboratory tests should be performed with venous blood (morphology, coagulogram, glycemia, electrolytes, creatinine, C-reactive protein, transaminases) and imaging (CT or possibly MR of the head).¹ In patients qualified for intravascular treatment, angio-CT or angio-MR examination of the intracerebral arteries is performed.⁸ Specialized procedures are also performed in differential diagnostics lumbar puncture and EEG test. It should be remembered that all the above-mentioned tests should be carried out in the shortest possible time. The therapeutic window for the implementation of thrombolytic therapy from the onset of stroke symptoms is 4.5 hours. However, the results of the meta-analyses clearly show that the shorter this time, the better the clinical effect.¹ Therefore, differential diagnosis of stroke should not delay specific treatment.

Aim

This work aims to approximate the differentiation of this common disease entity. Case reports show both clinical view and diagnostic difficulties.

Description of the cases

Case 1.

The patient 59 years old, brought by ZRM due to babbling speech after night, imbalance with a tendency to fall to the right, general weakness, confluent sweat. Until now, he was treated for bipolar disorder, hypertension, alcohol abuse. Takes: Trazodone 50 mg, Beta-histine 16 mg, Hygroton 50 mg, Daneb 5 mg. In the neurological examination: conscious, sleepy, in logical contact, blurred speech, tongue deviates to the right, narrower eyelid gap of the right eye, paresis of a low degree of LKG, without pathological symptoms. Quali-

fication for thrombolytic treatment was performed: in CT scan of the head grooves of the brain bends atrophic wider, without focal and ischemic changes. Due to the unknown time of onset, single heads, small nonspecific foci, subcortical vascular origin were performed in both hemispheres of the brain. In laboratory tests, significant hyponatraemia of 102 mmol/L (norm 136-145 mmol/L), and intra-intramuscular 2.5 mmol/L (3.5-5.1 mmol/L). Due to electrolyte disturbances and the lack of fresh ischemic lesions in the MR examination, thrombolytic treatment was initiated, the patient was referred to O. Internal Medicine. The treatment used concentrated salt solution, supplemented potassium deficiency, modified treatment of hypertension (thiazide diuretic discontinued). During hospitalization, the patient admitted that he had had balance disorders, headaches, dizziness, nausea and vomiting for a week. After electrolyte imbalance due to intentional tremor, he was consulted neurologically again. In the neurological examination: conscious, logical contact, correct speech, narrower eyelid gap in the right eye, in the area of remaining cranial nn without abnormalities, in the limbs without paresis, intentional tremor of the upper limbs of pronounced severity. Based on the whole clinical picture, it was suspected that the above-mentioned tremor is the result of damage to the cerebellum, caused by a toxic background. Alcohol abstinence and control at the Neurological Outpatient Clinic were recommended.

Case 2.

Patient 68 years old, brought because of babble speech, which appeared after a 1.5 hour nap (the patient was last seen healthy about 3 hours before arrival in the emergency room). Until now, he was treated for type 2 diabetes, hypertension, ischemic heart disease, cholelithiasis - cholecystectomy. Consumers: Amaryl 4 mg 1x1 tabl, Siofor 850 mg 3x1 tabl, Beto 25 ZK 1x1 tabl, Tritace 10 mg 1x1 tabl, Amlozek 5 mg 1x1 tabl. In the neurological examination: conscious, medium-grade mixed aphasia, selectively fulfills the instructions, isokoria, meningeal symptoms, in terms of remaining cranial nn with no abnormalities, in limbs without paresis, pathological symptoms, nb. Qualification for thrombolytic treatment was carried out: in measurements with SOR: RR 157/92 mmHg, temperature 36.7 degrees C, glucose 38 mg% (test strip made of capillary blood). In CT of the head in both hemispheres of the brain subcortical and periventricular small foci of chronic vascular lesions. Due to hypoglycemia, thrombolytic treatment was abandoned, the patient was referred to O. Internal Medicine. In laboratory tests obtained from venous blood, glucose 32 mg%. Concentrated glucose solutions were used in the treatment, treatment of type 2 diabetes was modified. After glycemic control, neurological re-evaluation was performed: conscious, in verbal con-

tact, normal speech, isokoria, meningeal symptoms, in the scope of remaining cranial without abnormalities, in limbs without paresis, pathological symptoms.

Discussion

Stroke is a common clinical problem, but its diagnosis is still there providing many problems. When starting the diagnosis of sudden hemiparesis, we count with stroke most.⁴ Tumor and brain abscess symptoms usually develop over days or weeks, but the onset may also be acute. The neurological symptoms in this case result from the presence of the abnormal “mass”. They are the result of increased intracranial pressure, which is a consequence of limited possibilities of abnormal tissue growth within the brain, they also form a set of symptoms resulting directly from damage to certain structures.⁵ In the CT of the head, hypodense, “finger” swelling may be visible – so-called ischemic stroke cancer mask. After administration of the shading agent, the tumor mass is enhanced by contrast, and the hypodense zone of edema around the tumor does not change.⁶ The advantages of MR in relation to CT are the ability to obtain images of brain structures with high spatial and contrast resolution.⁷ The non-invasive and repeatability of the MR examination allows it to be used both in the diagnosis of CNS tumors during the initial assessment of the nature of the tumor (lesions typically benign or suspicious of malignancy), biopsy planning, qualification for neurosurgical surgery, as well as monitoring after treatment – both surgical and after radiation therapy.⁸ Contemporary neuroimaging due to the use of functional MRI sequences – diffusion-weighted imaging (DWI), nerve fiber (DTI, diffusion tensor imaging, tractography), perfusion (PWI, perfusion weighted MRI), spectroscopy (MRS, magnetic resonance spectroscopy) and functional MR (fMRI), allows for multifaceted assessment of the tumor – its structure, both macroscopic and coexisting features within retrograde changes, biochemical features, microcirculation and changes at the cellular and molecular level.⁷ In the cases described above, imaging studies showed small nonspecific, vascular origin; no suspected focal length shift. Seizures are rare signs of acute stroke.^{8,9} The neurological deficit that appears immediately after the seizure disappears mimics that caused by vascular disease. Interview regarding illness, disturbed consciousness, retrograde amnesia, and tongue biting symptoms are helpful in diagnosis.⁴ The EEG test is of great importance, as it may be normal in the inter-seizure period. You can then perform provocative tests (photostimulation, hyperventilation or sleep deprivation). Sometimes it is useful to conduct a so-called Holter EEG, i.e. several hours or even several days of continuous recording of brain bioelectrical activity.¹⁰ In the described clinical cases, the collected history did not indicate loss of consciousness. Migraine, in particular migraine with aura is one of the postulated risk factors for

ischemic stroke in the population of young people.¹⁰ Risk factors have been identified that increase the likelihood of stroke in people with migraine with aura. The most frequent in this case is the high frequency of bouts of headache, smoking and the use of hormonal contraceptives. Despite this, ischemic stroke is rare in this disease (it accounts for 0.5–1.5% of all strokes).¹¹ Migraine usually causes visual symptoms that run through the field of view within 5–20 min. Sometimes the migraine aura takes the form of sensory disorders also moving within 10–20 min along the limb. The rate of spread of the discomfort in some cases usually makes it possible to distinguish between aura and stroke, in which the spread of the discomfort is minimal or very fast.¹² The biggest problem in differential diagnosis is the differentiation of migraine stroke with a seizure of hemiplegic (hemiplegic; FHM) migraine, especially its sporadic form and/or the first episodes of this disease in life. In some cases, FHM equivocal imaging studies differentiate the above disease entity from stroke. MR FLAIR and T2-dependent images may (although not always) show signs of edema in the opposite hemisphere to symptoms. The DWI study did not always show changes, whereas the study using ADC maps usually did not show deviations from the norm.¹³ In one of the described clinical cases, the patient reported headaches. But they occurred for a week before the appearance of the focal symptoms neurological. In addition, speech disorders and paresis of the limb did not appear simultaneously, they were not of a sequential nature.⁴ The biggest problem in differential diagnosis is the differentiation of migraine stroke with the attack of hemiplegic (hemiplegic; FHM) migraine, especially its sporadic form and/or the first episodes of this disease in life. In some cases of FHM, imaging studies ambiguously differentiate the above disease entity with stroke. MR FLAIR and T2-dependent images may (although not always) show signs of edema in the opposite hemisphere to symptoms. The DWI study did not always show changes, whereas the study using ADC maps usually did not show deviations from the norm.¹³ In one of the described clinical cases, the patient reported headaches. However, they occurred for a week before the appearance of the focal symptoms neurological. In addition, speech disorders and paresis of the limb did not appear simultaneously, they were not of a sequential nature. Migraine was therefore excluded. In the event of coma, glucose fluctuations associated with metabolic disorders, renal impairment, electrolyte levels, infection, alcohol and drug effects should be considered. In some patients with diabetes, in the clinical picture of its complications which is the hyperglycemic-hypermolar state, focal neurological symptoms (hemiparesis, hemiparesis, speech disorder) and seizures predominate. Other characteristic clinical symptoms include: strong thirst, anorexia, orientation disorder, drowsiness, stupor, hypotension, tachycardia, tremor, fever.¹⁴ In treatment, it is im-

portant to correct fluid and electrolyte deficiency as well as insulin therapy.¹⁵ Hypoglycemia is diagnosed when blood glucose levels fall below 55 mg/dL (3.0 mmol/L).¹⁶ Clinical signs that make the diagnosis include: neuroglycopenia (insufficient glucose supply for the proper functioning of the central nervous system) leading to confusion with the features of impaired thinking ability, dizziness, drowsiness, weakness, aggression, anxiety, speech disorders, orientation disorder, stupor, loss of consciousness and coma; and also increased activity of the sympathetic nervous system (tachycardia, increase in blood pressure, confluent sweat, wide pupils, hyperkinesia, tremors and increase in muscle tone, convulsions) and decreased activity of the parasympathetic system (nausea, severe hunger).¹⁵ The treatment uses 20% glucose solution and glucagon administered i.m. or s.c.¹⁵ The most common neurological symptoms in uremic encephalopathy are: disturbance of consciousness, multifocal myoclonus, dysarthria, ataxia, intentional tremor and epileptic seizures. The fluctuating nature of the symptoms is typical.¹⁷ There is also a positive relationship between the rate of increase of failure and the severity of neurological deficits. Hyponatraemia (plasma sodium below 135 mmol/L) is the most common electrolyte disorder that primarily causes CNS symptoms, especially when it develops rapidly. This is due to brain edema that occurs as a result of this disorder.¹⁸ Hyponatraemia can be divided into: mild, usually asymptomatic; moderate when the sodium level drops to 120-130 mmol/l. Then there may be nonspecific symptoms in the form of dizziness and headache, nausea, general tiredness and weakness, muscle cramps. Severe hyponatraemia we recognize when the sodium concentration falls below 120 mmol/l. Such a low concentration causes headache, orientation disorder, epileptic seizures reduction of muscle tone, up to the disappearance of tendon reflexes, Babinski's reflex and even coma.^{18,19} Hypernatremia is a condition in which the plasma sodium concentration is > 145 mmol/l. Symptoms include increased thirst, nausea, muscle weakness, fatigue, irritability, drowsiness, convulsions, and coma.¹⁸ Hypokalemia is a condition of low potassium plasma levels below 3.5 mmol/L. Symptoms on the part of the nervous system result from impaired function, associated with changes in the membrane potentials of nerve cells caused by potassium deficiency.¹⁸ We can observe hyperactivity, paresthesia, apathy, impaired concentration and drowsiness, as well as cold intolerance and thirst.²⁰ Symptoms of hyperkalemia (blood potassium levels above 5.5 mmol/l) are most common in cases of significant potassium deficiency. We observe muscle disorders in the form of irritability, spasms and even muscle paralysis. Occasional numbness and symptoms of sensory damage may occur.¹⁸⁻²⁰ In the presented patients' metabolic disorders were the cause of neurological symptoms. The patient described in the first case experienced hyponatraemia resulting from probably

prolonged alcohol abuse. Sodium levels were extremely low. The fact that the patient felt worse for several days prior to hospitalization and the worsening of symptoms indicate that hyponatraemia developed in a chronic manner. In the second patient, hypoglycaemia was the cause of neurological disorders. Glucose levels were also extremely low. The severity of the speech disorder suggested that the patient was partially adapted to hypoglycemia. Chronic alcohol abuse is thought to cause two types of change in central nervous system. In the case of direct changes that result from its neurotoxic effects, personality, mood and behavioral changes appear the earliest. Then there are cognitive disorders in the form of abstract thinking disorders, working memory disorders, time and space orientation disorders.^{21,22} In the long-term effects of alcohol on the nervous system, vitamin B deficiency (mainly B1) and caloric deficiency are important. In these cases, in addition to neurological and psychiatric symptoms, there are symptoms of damage to the parenchymal organs, mainly the liver, as well as heart and symptoms of damage to the peripheral nervous system. The most common syndrome is Wernicke and Korsakow.^{21,22} In the cases described above, the first patient was chronically abusing alcohol. However, the determined level of ethanol in venous blood was negative in both cases.

Conclusion

Stroke is a significant clinical and social problem both in Poland and in the world. Despite this, its diagnostics and especially differentiation is difficult. This is associated with a variety of diseases that can be a "mask" of stroke - from non-vascular neurological diseases (brain tumors, epilepsy) through injuries to very common metabolic disorders (hypoglycemia, hyponatraemia). When analyzing the case of each patient with suspected sudden CNS vascular disease, all of them should be remembered. It should also be borne in mind that differential diagnosis cannot delay specific treatment for stroke.

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