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Uncomplicated dissection of the right internal carotid artery with coexisting left-sided ischemic stroke - case report

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ABSTRACT

The dissection of the internal carotid artery, although rare, is in five to twenty-five percent a causative agent of acute ischemic stroke in patients up to the age of 45. The aim of this paper is to present the case of ischemic stroke within the right hemisphere with left-sided uncomplicated dissection of the internal carotid artery in the case of a young woman. A 35-year-old patient was admitted to the Department of Neurology on an emergency basis due to the sudden motor dysphasia and paresis of the right upper limb. During the anamnesis, the woman reported a head injury suffered two days earlier as a result of fainting. Diagnostic procedures toward ischemic stroke indicated a presence of hypodense area within the left hemisphere of the brain. Subsequent studies – MRI, CT angiography confirmed the existence of an ischemic lesion in the area of the left middle cerebral artery as a consequence of obstruction of the left internal carotid artery. In addition, a dissection of the right internal carotid artery was revealed. Further differential diagnosis, including systemic diseases, did not reveal the reasons for the coexistence of ischemic stroke and vessel dissection on the opposite side. Carotid dissection is a rare but dangerous condition. It either develops spontaneously or after an injury to the head or neck. Any kind of injury of the carotid artery can cause dissection and should lead to include this condition in the differential diagnosis- the more so since even small dissection of the internal carotid artery can be manifested with a delay.

Keywords: carotid dissection, ischemic stroke

1. INTRODUCTION

The stroke is a set of clinical symptoms associated with the sudden occurrence of focal or generalized brain dysfunction, which arises due to cerebral circulatory disorders and lasts more than a day. In many cases, stroke is a life-threatening condition. It requires hospitalization, preferably in a specialised ward. The symptoms of stroke depend on the location of the lesion site [1,2]. The most common ones are weakness or lack of sensation on one side of the body, paresis or paralysis of half of the body, particularly severely affecting the ability to move the limbs. Other symptoms include speech disorders such as aphasia, illegible handwriting and reading, acalculia, apraxia, amnesia, as well as disturbances in the ability to recognize things or the ability to swallow, and even visual disturbances. A characteristic symptom is the asymmetry of the face resulting from drooping mouth corner, eyelid, and facial paresthesia. Stroke may occur suddenly during the day or after physical exertion [3].

There are two types of strokes. The hemorrhagic stroke is caused by cerebral hemorrhage, whose group includes subarachnoid and intracerebral hemorrhage. The ischemic stroke is caused by the arrest of the blood supply to the brain. Ischemic strokes account for 85-90% of all strokes and are caused by a sudden stop of the blood supply to the brain as a result of narrowing or complete occlusion of arterial vessels. Cardiac arrhythmias, primarily atrial fibrillation, valve defects, acute myocardial infarction, atherosclerotic lesions, inflammatory changes in the vessels and obstruction of the arteries supplying the brain are among causes of ischemic stroke [4, 5].

In 20% of cases stroke develops from a dissection of the internal carotid artery, most often in the subendothelial form, which as a result of embolic mechanisms may lead to stenosis or obstruction of the vessel [3]. The dissection of the internal carotid artery is a rare pathology, however, it is considered to be a significant risk factor for stroke or transient ischemic stroke (TIA) in people at an early age [6].

Literature review indicates that the first case of traumatic dissection of the internal carotid artery was described in 1947 by Dratz and Woodhall, and in 1957, Jentzer, Anderson and Schechter published the first description of the spontaneous dissection of this vessel [7].

Due to the intensive development of modern methods of diagnosis and therapy of acute cerebrovascular disease, it is possible to significantly improve the prognosis of patients affected by stroke. In particular, the introduction of effective therapy for specific stroke, which is thrombolytic therapy [8]. Effective treatment is also facilitated by the development of specialized stroke wards with a neurological intensive care units that allow for the possibility to provide professional assistance to patients in the acute phase of stroke [9].

The described case presents a simultaneous occurrence of two vascular pathologies. Right-sided dissection of the internal carotid artery, which occurred without clinical symptoms and left-sided narrowing of the internal carotid artery with a stroke manifestation, which probably arose on the basis of the primary dissection of this vessel. The aim of this work is to present the diagnostic process and to explain the causes of the clinical situation.

2. CASE REPORT

A 34-year-old female patient was admitted to the Neurology Department due to motor dysfunction and paresis of the right upper limb. Symptoms occurred on the same day, just after awakening. In the anamnesis, the patient informed about the surgery performed 10 days earlier due to an umbilical hernia, and an episode of fainting with loss of consciousness on the first day after surgery, accompanied with a head injury - then consulted in emergency department, a CT scan of the head was performed and no pathology was founded. The neurological examination revealed a medium degree of right-sided paresis (3/5 on the Lovett scale), deep vivid reflexes on the right side and slightly weakened superficial sensation. There were no disturbances of deep sensation and cranial nerve abnormalities. In the anamnesis, the patient also denied smoking, alcohol abuse, and using hormonal contraception, she did not suffer from chronic diseases and taking any medication.

During her stay at the Department of Neurology, basic laboratory tests were performed - blood morphology with smear, coagulation, and biochemistry, among which the following deviations from the standard were observed: RDW-CV - 11.4%, PT - 13 sec, INR - 1.16, CRP - at 6.13. In addition, a test for the presence of alcohol in the blood - 0.03 g / l.

The conducted diagnostics shows the presence of a small hypodense area within the left hemisphere of the brain.

Further imaging examinations during hospitalization - MRI, and angio-CT confirmed the existence of an ischemic lesion in the area of left central cerebral artery supply resulting from obstruction of the left internal carotid artery. In addition, delamination of the internal carotid artery on the right side with a length of 12mm approx. Vascular surgeon consulted the patient condition and decided not to qualify her for surgery, therefore the anticoagulation treatment with low molecular weight heparin was introduced, and a good therapeutic effect was achieved.

Further diagnostic procedures aimed at explaining the cause of bilateral vascular anomalies in a young patient. Vessel ultrasound was performed using the Doppler method, in which no evident atherosclerotic lesions were observed, however, the flow in the left carotid artery, which appeared to be obstructed over the entire length covered by the study, was not visualized. Transcranial Doppler examination (TCD) showed presence of collateral circulation through the anterior cerebral artery with a flow reversal in the initial section of the left anterior cerebral artery. In the remaining arteries included in the study the direction and spectrum of flow was normal. A series of tests for systemic diseases were performed: ANA / ENA antibodies, IgM, IgG, IgA anticardiolipin antibodies and V Leiden factor - all negative. An anamnesis, that could suggest the cause of vascular anomalies, including smoking or hormonal disorders, also came out negatively.

One week after admission, the consultant psychologist stated that the patient had preserved an understanding of speech, maintained dialogue, correctly answered questions and carried out instructions. The tone of the voice was unchanged. The patient had a slight degree of non-fluent speech, dragging and repeating syllables and therefore encountered difficulties in expressing his own thoughts. The speech rate was accelerated.

The patient was discharged home in a good general condition, without complaints, with the recommendation of a preventive intake of dabigatran at a dose of 2×150 mg and a control angio-CT after about 3 months. Currently, the patient stays under neurological supervision.

3. DISCUSSION

The dissection of the internal carotid artery (ICAD) is rare, but it is a serious threat to the patient's health and life. Population studies conducted in Europe have shown that the annual incidence of this pathology is 3 per 100,000 people [10]. It is in the 0.5-4% causative factor of ischemic stroke in the entire population and in 25% is the cause of stroke in young patients (before 45 years of age) [11, 12].

Internal carotid dissection can occur in the extracranial (75% of all cases) and intracranial part [13]. It may occur during any period of life, however, the disease peak is most common in the fifth decade [14]. Cases of ICAD occurrence in infants due to perinatal injury have also been reported [15]. Robertson described a case of dissection of ICA in a 14-year-old boy. The paresis of the right limb found in him subsided after an hour, without leaving any symptoms [16]. The incidence of pathology in both sexes does not show differences, however, the ailment in women is found on average 5 years earlier than in men [17].

ICAD develops spontaneously or after a head or neck injury. Among the risk factors are mainly systemic diseases (fibromuscular dysplasia, Ehler-Danlos syndrome, reduction of collagen type 3 production, osteogenesis imperfecta, Behcet's disease), migraine, invasive neurosurgical and cardiac surgery, intense vomiting, excessive physical effort [18, 19]. Smoking cigarettes, poorly controlled diabetes, hypertension, hypercholesterolemia and oral contraceptives also have some significance in the etiopathogenesis of this disorder [20].

The disease may arise as a result of a blunt neck injury, head bumps with excessive retraction, which causes stress on the carotid artery on the bone structures or as a result of a fracture of the mandible or the base of the skull [21].

Two forms of carotid artery dissection are mentioned: subadventitial and subendothelial. In case of the subendothelial dissection, the blood gets under the layer of damaged endothelium and detaches it from the wall of the vessel, which in turn leads to narrowing or occlusion of the vessel and to an emergence of a dissecting aneurysm. In place of the damaged endothelium, an intramural hematoma is formed together with a thrombus that gradually narrows the lumen, which becomes a source of congestion. This mechanism is mainly associated with the symptoms of ischemic stroke [22]. The second form of dissection - subadventitial - consists in getting blood extravasation between the central membrane and adventitia, which causes a significant dilatation of the artery. In this form, the most common Horner's syndrome or cranial nerve damage [23]. Both forms of ICAD are reported with similar frequency [24].

The dissection of the internal carotid arteries may present in the form of a variety of clinical symptoms. Such a different clinical manifestation makes it difficult to make a correct diagnosis [25]. Local and neurological symptoms are mentioned. Local symptoms usually precede the symptoms of CNS ischemia [26].

The most common symptom is a headache (66%) occurring on the side of dissection, mainly in the frontotemporal region, less frequently in the occiput [27]. In 50% of cases, face pain occurs [28]. Pain usually has a sudden onset, which may mistakenly suggest subarachnoid haemorrhage. Pain complaints result from stimulation of the trigeminal nerve fibers that surround the carotid artery [29]. The second most frequent clinical manifestation (25-58%) is the ipsilateral Horner's syndrome [30]. Symptoms include pupil constriction, ptosis, and a collapse of the eyeball. However, these symptoms are not accompanied by a

disorder of sweat glands on the face, because they are supplied by post-ganglionic sympathetic fibers that run only along the external carotid artery [31]. Another local manifestation of ICAD may be cranial nerve palsy, occurring in 3-12% of cases [32]. It mainly concerns the lower cranial nerves, and the nerve compression behind the styloid process in the thrombosis space is considered the cause. Most often, the sublingual nerve is paralyzed, palsy of nerves IX, X and XI may also occur [33]. Other less common local symptoms include tinnitus, dizziness, fainting, swelling of the neck, and the presence of murmur above the carotid artery.

The neurological symptoms of cerebral ischemia that are the result of ICAD include stroke, TIA, episodic amaurosis fugax, monocular blindness. They occur at a frequency of 30-80% [34]. The most common of them are stroke and TIA which occur in 20% of ICAD cases.

Sturzenegger et al. carried out retrospective studies on patients with spontaneous ICA dissection. They described 44 clinical cases (15 women and 29 men) aged 25-69. All patients had neurological symptoms. The most common symptoms were a unilateral headache (68%), Horner's syndrome (48%), transient ischemic attack (20%) and stroke (9%) [35]. Biousse et al. analyzed cases of 65 patients. The most common symptoms were cerebral ischemia (79%), followed by a headache (74%), Horner syndrome (31%), tinnitus (14%) [36].

Until now, only a few clinical cases describing carotid dissection with a minor-asymptomatic or asymptomatic course can be found in the literature. During the observation of 200 patients by Schievink, in 5% the incident appeared asymptomatic and was detected accidentally [37]. Towned and others described the case of spontaneous dissection of the carotid artery in a young patient. The symptoms were limited only to a headache and minimal loss symptoms [38]. Pace et al., analyzing ICA dissection literature from the last 30 years, detected 28 cases of spontaneous ICAD. They described that half of them showed only minor or transient symptoms [39].

Most often ICAD (in about 66% of cases) is located near the carotid dividing at the C1-C2 level [40]. In 80% of cases, it manifests in one location [41]. However, sometimes it can occur on both sides.

The bilateral location of ICAD usually appears in the traumatic mechanism, and may also be accompanied by dissection of the vertebral arteries. The cause of it can be, for example, a car accident, an example of such cause is described in the work of Croenlein which describes the bilateral dissection of ICA due to participation in a major collision on the motorway with the resulting significant neurological symptoms [42]. Also described is a case of bilateral ICA distension in a drunken woman, who was run over a small vehicle, revealed in an autopsy study [43]. We should also mention the interesting case of bilateral ICA dissection, described by Marangi et al., Which occurred in an elderly patient with an earlier infection of syphilis. The infectious disease could cause degenerative changes in the artery walls, consequently leading to clinically significant ischemic changes in the brain [44]. Carotid dissection without obvious causes is very rare, however, a case report of a 42-year-old woman with extensive ischemic changes in the brain as a result of bilateral, spontaneous ICA dissection has been described in the literature [45].

In the initial diagnostic process of ICAD, the doppler ultrasonography plays a key role, thanks to its high prevalence, ease of performance and lack of invasive action. Typical changes seen in the ultrasound examination are the presence of intimal flap in the lumen of the vessel, the finding of double flow, narrowing of a lumen or complete lack of flow.

The sensitivity of this method is high and can reach up to 95%, however, it should always be verified by angio-CT or angio-MR examination of cervical and intracranial vessels. The sensitivity and specificity of MR angiography is 100% [46].

According to A. Misztal and W. Kwiatkowska, angiography of the capillaries is still the main study and reference method [47]. As an invasive examination is associated with a certain risk of complications, it should be reserved only to verify diagnostic doubts. However, this is a method that allows to implement endovascular treatment and to plan surgical treatment. In the study, you can get a "string sign", narrowing of the artery lumen or the presence of a pseudo-aneurysm. Pathognomonic symptoms include an intimal flap and the presence of double vessel lumen.

Due to the fact that the main cause of ischemic changes in the central nervous system in the course of ICAD is microemboli, the treatment should be aimed primarily at preventing embolism. In most cases, it is sufficient and leads to the withdrawal of changes within a period of up to 6 months. After analyzing the available medical literature, it can be concluded that there are still no randomized studies comparing the efficacy of antiplatelet drugs with anticoagulants in patients with ICAD. The research carried out so far shows that there are no major differences in survival and incidence of side effects by comparing antiplatelet and anticoagulation therapies [48]. Engelter et al. suggest that therapy should begin with the intravenous administration of unfractionated heparin followed by antithrombotic therapy for 3 to 6 months [49]. According to the current AHA / ASA guidelines (American Heart Association / American Stroke Association) on the prevention of stroke in cases of stroke or TIA, warfarin treatment is recommended for 3-6 months or the use of antiplatelet drugs (recommendations IIa B). The next stage of treatment after 3-6 months is long-term antiplatelet therapy (recommendations IIb C). However, in patients with recurrent ischemic events, warfarin therapy is continued.

The indication for surgical treatment is the enlargement of dissection, the presence of significant hemodynamic stenosis and the presence of an aneurysm exceeding the 2-fold dimension of healthy ICA. The endovascular procedure developing in recent years is more preferred in ICAD cases than the classical operation with the opening of the lumen due to the lower rate of complications and technical difficulties. However, currently, only individual descriptions of groups of patients successfully treated with these methods can be found in the literature [50, 51]. One of the studies describes 7 patients with established ICAD who underwent stenosis of the internal carotid artery. In all patients, the clinical efficacy of the procedure was found, and the vessel patency in imaging examinations was 100%. Patients were treated with clopidogrel for six weeks after the procedure and lifelong acetylsalicylic acid [52].

The prognosis for ICAD of the extracranial segment is good unless there is a stroke or irreversible ischemic ocular lesions. Research shows that a headache, neck pain, and cranial nerve palsy usually disappear, although they can evolve into a chronic form.

Schievink et al. report a 7-year follow-up of 200 patients with carotid artery dissections (104 women and 96 men). The average age of patients was 44.9 years (range from 16 to 76). The re-dissection of the artery occurred in 4 patients (2%) during the month and in 12 patients (6%) in the period between 1 and 6 years after the incident. Younger patients had a greater risk of recurrent dissection. It can, therefore, be assumed that the risk of recurrence is small and amounts to about 1% per year [37].

The diagnostic difficulty in our case was to explain the occurrence of bilateral vascular anomalies in a patient at an early age. After extensive diagnostics, we can assume that the patient's head injury caused by falling from his own height led to the dissection of both internal carotid arteries. The dissection of the right ICA proceeded without clinical symptoms, whereas the dissection of the left ICA led to the formation of a dissecting aneurysm, which solidified and narrowed the lumen of the vessel, which resulted in the symptoms of ischemic stroke.

Wiśniewski and Książkiewicz describe the case of internal carotid artery dissection with subsequent intracranial occlusion with a good effect of anticoagulant therapy. The patient was diagnosed with right-sided Horner syndrome and symptoms of pyramidal pathway damage. Angio-CT examination of the cerebral vessels showed right-sided dissection of the wall at the length of approx. 25 mm. within the internal carotid artery. A double flow channel was found, intramural hematoma was not described. On the basis of the entire clinical picture and the results of additional tests, the diagnosis of internal carotid artery dissection was established with subsequent intracranial occlusion of the right internal carotid artery (RICA). The anticoagulant treatment with low molecular weight heparin was initiated at the therapeutic dose and anticoagulant treatment with warfarin with monitoring of the INR index.

4. CONCLUSIONS

The case presented by us is an example of traumatic and bilateral dissection of the internal carotid arteries, in which right-sided dissection occurred without clinical symptoms, while left-sided dissection resulted in a subsequent occlusion in the intracranial section complicated by stroke.

The case report shows that considering the aetiology of stroke in young people, the dissection of the intracerebral arteries should also be taken into account. It is a rare but significant cause of ischemic stroke as a result of the embolism mechanism. Therefore, correct diagnosis and thus faster implementation of treatment can significantly improve the prognosis in these patients.

We would also like to draw attention to the asymptomatic clinical manifestation of the right-sided dissection of ICA. This suggests that if there are risk factors for vascular dissection, even in the absence of symptoms, physicians should have increased vigilance towards this pathology. In addition, any head and neck trauma that may result in ICA damage should prompt this condition to be included in the differential diagnosis, the more so that internal carotid dissection may be manifested with a delay.

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