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Brainstem ischemic stroke without permanent sequelae during the course of spontaneous internal carotid artery dissection – case report

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Summary

Background:

Internal carotid artery dissection (ICAD) is a frequent cause of a stroke in young patients. Risk factors which can lead to dissection include neck injury and diseases of the inner wall of the artery. Common symptoms in ICAD are cervical pain and headache, Horner's syndrome, paralysis of the cranial nerves and subsequently cerebral and retinal ischemia.

MR angiography in TOF technique and brain MRI in T1- and T2-weighted images, FLAIR and DWI sequences are the method of choice in patients with ICAD but contrast-enhanced multislice computed tomography remains the fastest and the most available diagnostic method.

Case Report:

A 39-year old woman, previously healthy, presented to the Hospital Emergency Department because of increasing neck pain on the right side and difficulty in swallowing. The neurological examination revealed: drooping of the right eyelid with narrow palpebral fissure, dysarthria, anisocoria (narrower pupil on the right side), unilateral hypoesthesia on the left side, weak palatal and pharyngeal reflexes on both sides, paresthesia within the left half of the body. Seven days before, the patient felt a sudden, severe neck pain radiating to the temporal apophysis.

CT angiography revealed a defect in contrast filling within the left internal carotid artery and right vertebral artery. MRI of the head with MR angiography showed internal carotid artery dissection on the left side and dissection of the right vertebral artery and no ischemic changes within the brain.

Conclusions:

CT and MR angiography are methods characterized by high sensitivity in detecting dissection of the cervical arteries.

Key words:

dissection • MR • CT angiography • ischemic stroke

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Background

Internal carotid artery dissection as a cause of ischemic stroke in the general population accounts for only a small percentage of cases (approx. 5%), whereas in the population of young adults – for 10–25% [1]. In approximately 20% of patients, the dissection occurs bilaterally or coexists with the dissection of one of the vertebral arteries.

Factors predisposing to vascular dissection include intimal damage and hemorrhage within the vessel wall. A false lumen (in case of preserved blood flow in the distal part of the dissection) and/or intramural hematoma with subsequent vessel stenosis or obstruction may form [2]. More profound damage of the vessel wall with hemorrhage between the middle and the outer (adventitial) layer occurs rarely. In such cases, vessel dilation appears and the narrowing of the lumen is less severe.

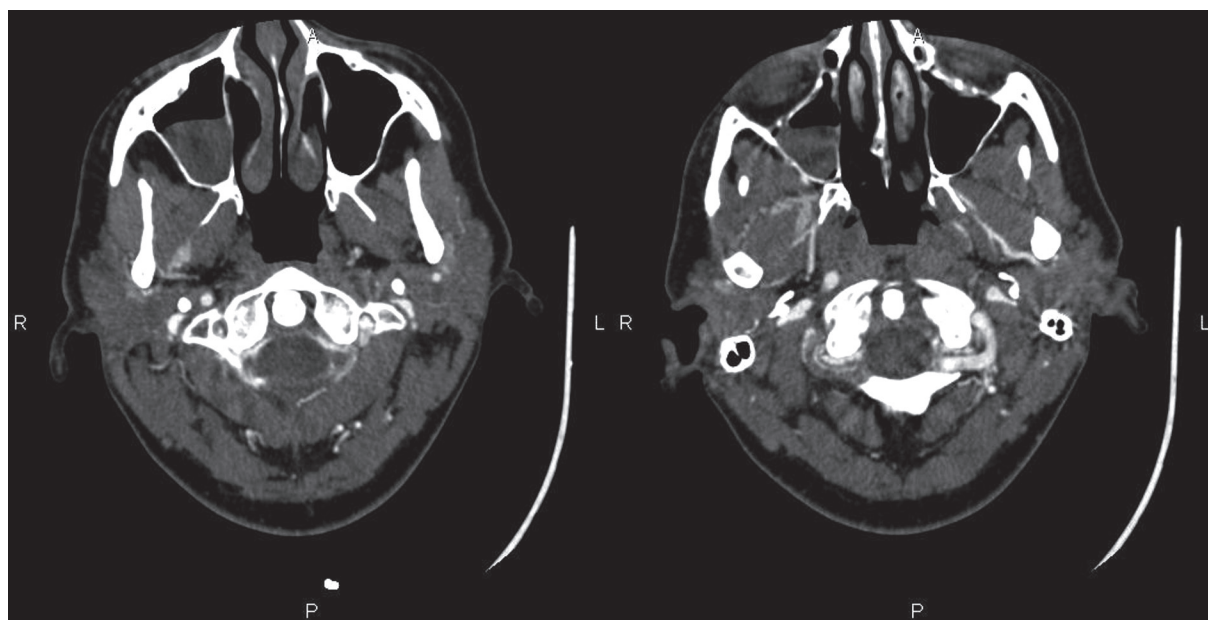


Figure 1. CT angiography revealed a lack of contrast enhancement in the left internal carotid artery and the right vertebral artery.

The causes of dissection include trauma (blunt injury in the area of the neck, iatrogenic injuries) and more common idiopathic dissections occurring due to pathologically changed vessel wall (fibromuscular dysplasia, Marfan syndrome, Behcet's disease and other connective tissue diseases, Ehlers-Danlos syndrome, α -1-antitrypsin deficiency, drug abuse, infections).

Clinical symptoms include facial and neck pain (both acute or gradually progressing), Horner syndrome (most often associated with an ophthalmic pain), palsy of cranial nerves IX–XII [3], tinnitus and, less often, ischemic stroke (most commonly – an embolic occlusion of the middle cerebral artery) or retinal ischemia due to arterial embolism [4].

Diagnostic procedure in patients with suspected carotid artery dissection depends on the pathomechanism of dissection, patient's clinical state and the availability of diagnostic methods.

Ultrasonography is a widely available diagnostic method. However, carotid arteries are tortuous and therefore cannot be entirely visualized by this technique, which limits its usefulness. Typical changes in artery dissection include the presence of two separate blood flow channels, lumen restriction, high-resistance blood flow or occlusion [5].

In angiography (DSA), the dissection can be diagnosed basing on indirect findings such as vessel narrowing.

Multislice contrast-enhanced CT scanning still remains the quickest and the most available diagnostic method. It allows a simultaneous assessment of vascular structures, soft tissues, bones and cerebral structures. CT scans usually show a smooth narrowing of arterial lumen without typical atherosclerotic changes and a dissected intimal flap.

Despite its obvious advantages, this method not always allows to visualize the false lumen. For a more precise

assessment of the dissection (its extension and severity), as well as to visualize the intramural hematoma, TOF MRA is used along with brain MRI in T1- and T2-weighted sequences, FLAIR and DWI, which is the method of choice in patients with carotid artery dissection [6]. Unfortunately, the MRI is of limited availability in many health care centers and therefore its immediate implementation in acute dissection is not always possible.

The management of carotid artery dissection includes anti-coagulant therapy or, alternatively, stenting [2].

Case Report

A 39-year-old woman, previously healthy, presented to the Emergency Department due to a progressing right-sided neck pain and swallowing difficulty. Neurological examination revealed right-sided ptosis with palpebral fissure narrowing, dysarthria, anisocoria (right-sided miosis), left-sided hypoesthesia, bilaterally decreased palatal and pharyngeal reflexes and left-sided paresthesias. Seven days earlier she felt a sudden, intense pain in the neck area radiating towards the right mastoid process.

Laboratory tests were performed (complete blood count, electrolytes, lipid profile, urinalysis) as well as the classic chest x-ray. Test results were normal except for the elevated cholesterol level.

Single-phase CT of the brain revealed a hypodense lesion in deep brain structures in the right hemisphere (diameter of 11 mm) and a lipoma in the right temporal lobe (12×9 mm). CT showed no other abnormalities.

CT angiography of cephalad arteries (from the level of the aortic arch to the level of the circle of Willis) showed an 18-mm-long section with absence of contrast enhancement in C1 segment of the left internal carotid artery (slit-like lumen in that section), which could correspond to parietal

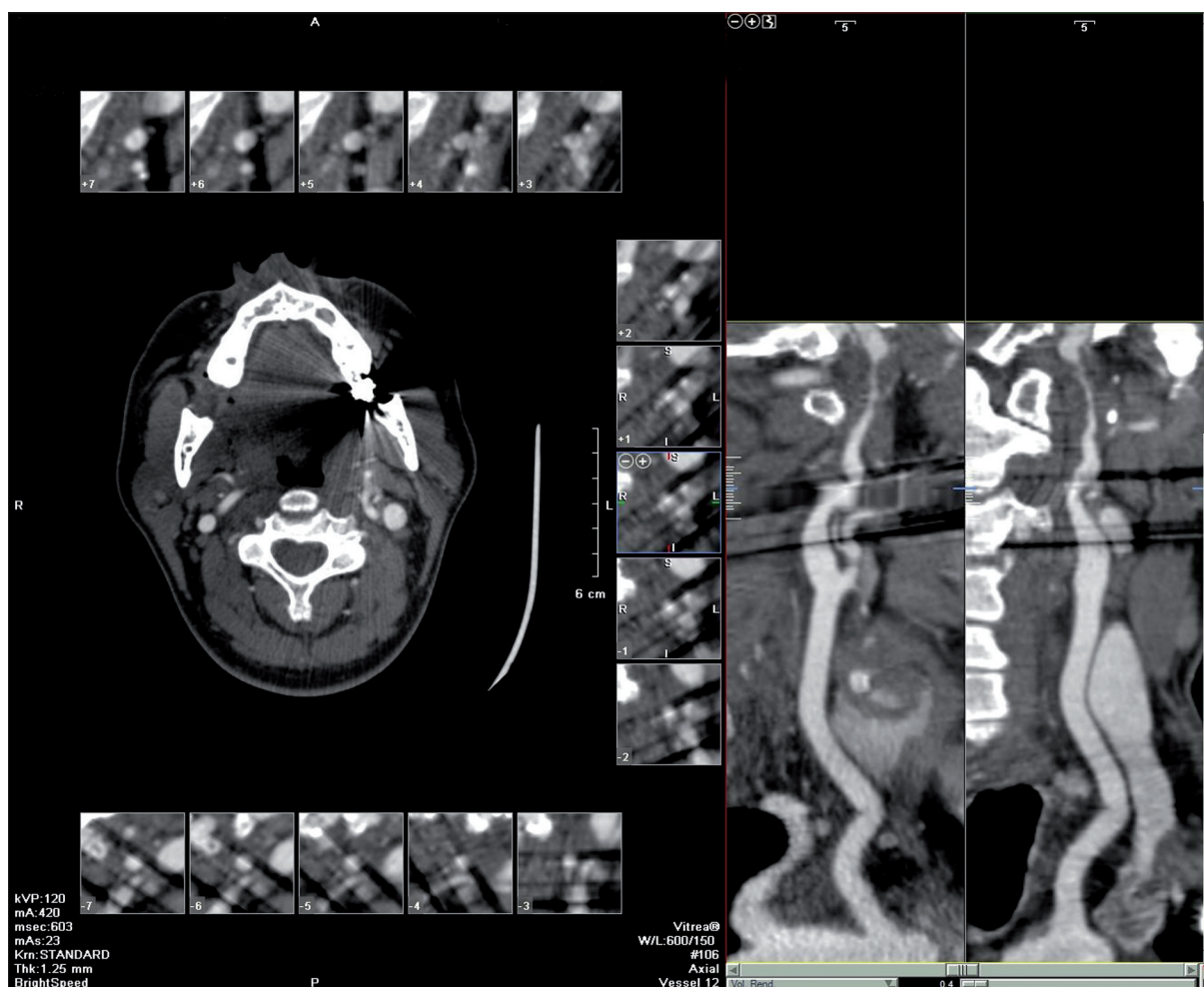


Figure 2. Reconstructed image of the left common carotid artery and the left internal carotid artery with a lack of contrast enhancement.

thrombus or dissection (Figures 1, 2). In segment V3 of the right vertebral artery, a round-shaped 28-mm-long section lacking contrast enhancement was found (the entire section with slit-like lumen as well), which could correspond to an annular thrombus or dissection. The left vertebral artery was the predominant one. The other findings were: asymmetric larynx and right vocal fold thickening. No other abnormalities were visualized.

Patient was transported to the referential centre. The specialist in interventional neuroradiology disqualified the patient from stenting. Treatment with anticoagulant was implemented.

Diagnostics included:

- echocardiography (normal)
- ultrasound of intracranial arteries, showing high-resistance blood flow in the right vertebral artery suggesting vessel stenosis/dissection. No pathology in carotid vessels was found.
- brain MRI with MR angiography, revealed a dissection of the left internal carotid artery with thrombosis in the false lumen and a narrow true lumen (Figure 3), as well as the right vertebral artery dissection (dissection limited to the extracranial portion but the intracranial portion was narrow).

No features of restricted diffusion or acute ischemic lesions were found on DWI in the brain stem or in other parts of the cerebrum (Figure 4).

A hyperintense lesion at the base of the temporal lobe in T1- and T2-weighted sequences and FLAIR (probably the previously described lipoma). Enlarged Virchow-Robin space in deep brain structures on the right. No other abnormalities.

Tests for autoimmune diseases were performed. The patient was discharged from hospital in a good general condition. Oral anticoagulant therapy was prescribed. A follow-up visit was appointed in three months' time. Brain stem stroke was diagnosed (symptoms persisting for several days); neurological examination on discharge did not reveal any deficits apart from a slight paresthesia of left extremities.

After three months, the patient presented for the appointed hospitalization. No neurological signs were found on examination. Patient's only complaints were periodic paresthesias in left lower limb. Previously performed tests for autoimmune diseases (anti-nucleic antibodies, lupus anticoagulant, and IgG, IgM, IgA anticardiolipin antibodies, as well as c-ANCA and p-ANCA) were negative.

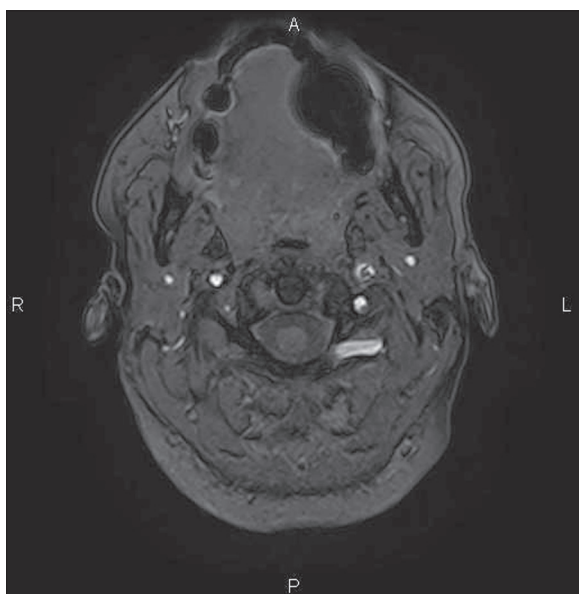


Figure 3. TOF MRA revealed a dissection within the common and internal carotid arteries.



Figure 4. No features of stroke were found within the brain stem (FLAIR (ax) and T2-weighted (sag)).

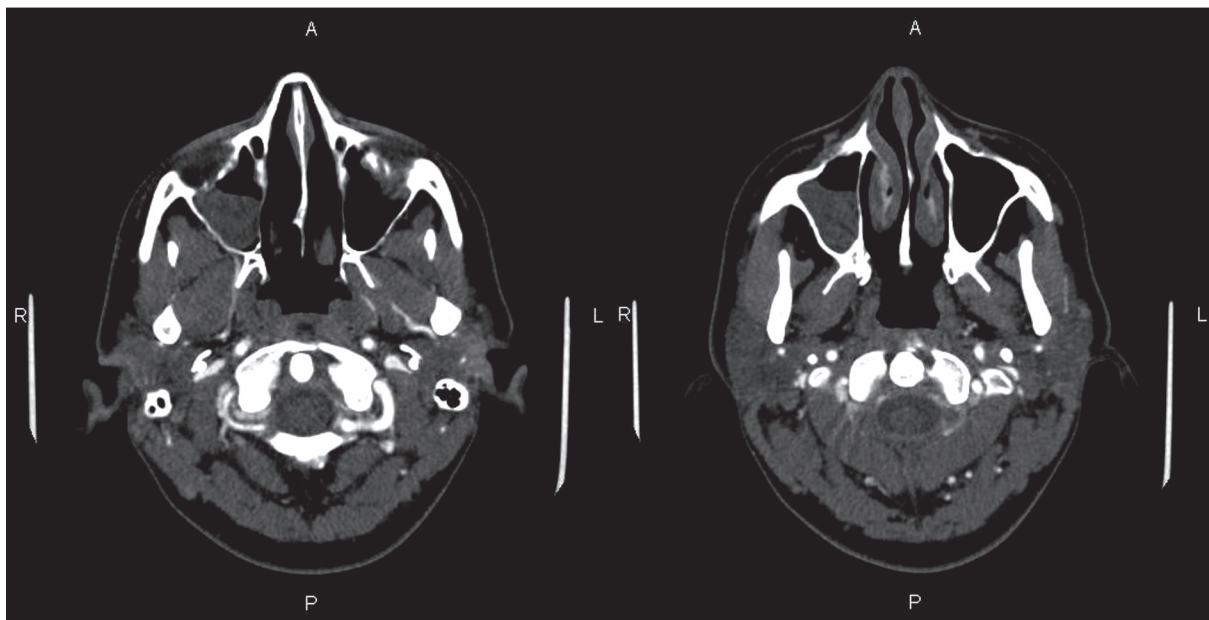


Figure 5. Follow-up CT angiography after 6 months revealed normal contrast enhancement of left carotid artery and features of residual dissection within the right vertebral artery.

The ultrasound of carotid and renal arteries revealed no aberrations and no features of fibromuscular dysplasia.

Follow-up CT angiography of carotid arteries showed residual dissection in the left internal carotid artery only in a short segment within the petrous part of the temporal bone. The remaining part (previously dissected) demonstrated normal blood flow in the entire vessel diameter (Figure 5). Blood flow in the right vertebral artery was normal.

The next follow-up CT was appointed in three months' time. The patient continued anticoagulant therapy.

Discussion

The presented case of a young woman, never subjected to any chronic pharmacological therapy before the described incident, demonstrates the effects of immediately implemented and precise diagnostics and administration of appropriate treatment on the resolution of neurological deficits and revascularization.

In young patients with an ischemic event of CNS, a detailed differential diagnosis is essential. With the absence of the risk factors of ischemic stroke, the dissection of cephalad arteries should be taken into consideration, especially when pain in the neck area is one of the symptoms.

After making a diagnosis of carotid artery dissection, special attention should be paid to find the potential cause of that vascular pathology, including connective tissue diseases, fibromuscular dysplasia, possible trauma. Apart from detailed history-taking, several additional specialist examinations have to be performed.

In patients disqualified from stenting, anticoagulant treatment is implemented, usually for the period of 3–6 months.

Conclusions

The differential diagnosis of ischemic stroke, especially in a young patient, should always include carotid artery dissection, even with a negative history of trauma.

References:

1. Rummeny EJ, Reimer P, Heindel W: MR Imaging of the Body. Georg Thieme Verlag KG, Stuttgart, 2009; 564–75
2. Walecki J: Postępy neuroradiologii. Polska Fundacja Upowszechniania Nauki, 2007; 91–113 [in Polish]
3. Kasravi N, Leung A, Silver I et al: Dissection of the internal carotid artery causing Horner syndrome and palsy of cranial nerve XII. *CMAJ*, 2010; 182: E373–77
4. Özkan Arat Y, Volpi J, Arat A et al: Bilateral internal carotid artery and vertebral artery dissections with retinal artery occlusion after a roller coaster ride – case report and a review. *Turkish Journal of Trauma & Emergency Surgery. Ulus Travma Acil Cerrahi Derg*, 2011; 17(1): 75–78
5. Misztal M, Kwiatkowska W, Ohly P et al: Rozwarstwienie tętnicy szyjnej wewnętrznej – symptomatologia, diagnostyka i leczenie. *Kardiologia Polska*, 2011; 69(9): 958–62 [in Polish]
6. Taschner CA, Leclerc X, Lucas C et al: Computed tomography angiography for the evaluation of carotid artery dissections. *Front Neurol Neurosci*, 2005; 20: 119–28