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Case report

VERTEBRAL ARTERY DISSECTION: CASE REPORT

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Abstract

Case report: A 36-year-old female patient with no cardiovascular risk factors was admitted as an emergency due to a disturbance in the gait and balance, dizziness, headache, which started suddenly approximately one day before admission to the neurology department. Nuclear magnetic resonance imaging studies showed an ischemic event in the left segment of the medulla oblongata, associated with a history of neck injury. NMR of the brain with FS sequence found a dissection of the left vertebral artery in proximal intracranial segment, approximately 1 cm in length. After conservative treatment with antiplatelet and antilipemic therapy, neurological improvement was observed. After one and a half years of follow-up, there was no recurrence of the same or similar neurological symptomatology. Conclusion: This paper highlights the importance of neck trauma, even after some time latency following the initial injury, as a cause of vertebral artery dissection and subsequent ischemic brain damage. This enables timely diagnostic and therapeutic actions, which certainly contribute to a better final outcome, which is a basic goal in our daily work.

Keywords: vertebral artery, dissection, neck trauma

Introduction

Vertebral artery dissection occurs when blood enters or accumulates within the wall of an artery, most commonly as a result of a disruption of the endothelial surface[1]. Although not a common pathological event, vertebral artery dissection can lead to ischemia and, less commonly, subarachnoid hemorrhage[1]. Dissection of cervical artery is a rare cause of cerebral ischemia in the general population, but it is a common cause of non-atherosclerotic vasculopathy which leads to ischemic cerebral shock and transient ischemic attack (TIA) in young patients, most often younger than 45 years, with an average onset at 46.5 years. Most patients with vertebral artery dissection do not have symptoms and do not seek medical help until symptoms of a stroke or TIA appear, which can be often delayed by several days after the acute dissection[2].

Vertebral artery dissection occurs more often in younger patients compared to carotid artery dissection.

The cumulative incidence of cervical artery dissections is 2.6 cases per 100,000. The incidence of vertebral artery dissections is 0.97 cases per 100,000, and the incidence of carotid artery dissections is 1.72 cases per 100,000[3].

Intracranial vertebral dissections are more common in men, while women are 2.5 times more likely to be affected by extracranial vertebral dissections[4].

Intracranial dissections are often associated with subarachnoid hemorrhage in half of the cases [4] due to rupture of the dissecting aneurysm[5].

It has been reported in the literature that 1-4 cases per year may be documented from large tertiary hospitals[6].

Arterial dissections occur either spontaneously, as part of genetic diseases (fibromuscular dysplasia, Marfan syndrome, Moy-Moy disease, Ehlers-Danlos syndrome type IV, Williams syndrome, Turner syndrome, osteogenesis imperfecta type I) or after craniocervical trauma. The most common association is with fibromuscular dysplasia[7]. Cases of cervical dissection after neck extensions and chiropractic maneuvers, which are temporally distant from the acute event, have been described in the literature.

Spontaneous vertebral artery dissections are known to cause stroke in young and middle-aged populations[4]. When spontaneous, they are most often etiologically correlated with genetic or environmental factors, although no consistent cause has been found[8]. When traumatic, they are most often associated with head, neck, and cervical spine injuries. If undetected or untreated, they can potentially lead to significant infarction[9].

Case report

We present the case of a 36-year-old patient without cardiovascular risk factors, with dizziness and instability with a cerebrovascular ischemic event in the left segment of the medulla oblongata, associated with evidence of neck injury and neuroradiological findings of dissection of both vertebral arteries.

This case report was carried out after obtaining informed consent from the patient. Consent was also sought and obtained from the department for radiology for the use of neuroradiological examination data.

The patient admitted to the emergency department with disturbances in gait and balance, dizziness, and headache, which had started suddenly approximately one day before admission to the neurology department. On admission, she was in good somatic status (heart action rhythmic, tones clearly audible, without pathological murmurs, TA 100/60 mmHg, HR 76 beats/min; lungs on auscultation with vesicular breathing; remaining organ system findings were normal). Neurological examination on admission revealed mild left-sided semi-ptosis and horizontal nystagmus with a fast component directed to the right, which quickly become weakened. The patient did not report diplopia. Upon phonation, the uvula was slightly deviated to the right, with mild right-sided palatal lowering, and a slightly decreased reflex of vomiting. The remaining findings of the cranial nerves were normal. Speech and swallowing were normal. MTRs with orderly and symmetrical reflex responses. During bilateral examination on the plantar-cutaneous reflex, the response was asymmetrical, the right one was with sluggish reaction with weaker tonic phase; at examination with Chedok modification bilateral extensor contractions were induced, which were discrete on the left, and on the right, a clear extension of the fingers and an episode of short-term fan spreading of the fingers was registered. Positional, coordination tests (P-N, P-K) and GMS were normal. Sensitivity: tactile, thermal sensation on the corpse and limbs and proprioception sensitivity for kinesthesia and paresthesia was bilateral and symmetrically preserved. Negates spontaneous sensitive syndrome. Meningeal signs were negative. Romberg clearly positive, with a predilection for falling to the left. Ataxic gait, unable to walk independently, only with the support of another person. Initially, a CT scan was performed with normal findings, but due to clinical findings highly

suggestive of a cerebrovascular event in the vertebrobasilar arterial basin, more precisely in the brainstem, an emergency NMR of the brain was performed on the same day with the finding of an ischemic lesion in the area of the left segment of the medulla oblongata.

From the patient's medical history, it was noted that she had headaches over the previous two weeks, which had been treated with analgesics prescribed by her - general practitioner, but without any significant therapeutic benefit. She also had a history of dizziness, described as short-lasting attacks, less than one minute, with spontaneous remission. Additional information was obtained from the patient's medical history - less than two months ago, she had sustained a neck injury when a heavy object (the upper movable part of a lifting bed) fall onto her neck. However, she did not complain of pain or other symptoms at the site of impact, and, therefore, she did not consult a doctor.

From comorbidity, she provided information about thrombophilia (MTHFR heterozygous, the information was anamnestic, there was no medical documentation attached). She also informed about a history of low serum iron levels and borderline sugar values, for which she was not receiving therapy. Three pregnancies: the first one (2019) with placental abruption and a stillborn child; the second pregnancy (2020) - a child was born at 32 weeks; the third pregnancy (2022) - a child was born at 34 weeks. All pregnancies were managed as pathological, for which she received low-molecular heparin, without a recommendation from the prescribing specialists to continue therapy after delivery.

The personal history revealed that the patient completed higher education, was employed, married, a mother of two children (from three pregnancies), did not smoke cigarettes nor consume alcohol, was predominantly right-handed. She was vaccinated for SARS-CoV-2. She denied allergies to food and drugs.

The patient was not taking any chronic therapy. The family history was negative for diseases of neurological interest.

During the hospital stay, a series of additional examinations were performed in order to resolve the etiological nature of the presented neurological clinical picture (CT, CDDS of the carotid and vertebral arteries, CT angiography of the neck and brain vessels, NMR of the brain with standard pulse sequences, TOF and FS sequence, TCCD Bubble test, echocardiography and cardiac examination). NMR of the brain with standard pulse sequences (Figure 1a) and TOF sequence (Figure 1b) was performed with delineation of an infarct lesion with dimensions of 5x3mm in the left segment of the medulla oblongata. It was neuroradiologically monitored with control NMR of the brain with description of a small elongated hypersignal change in the left segment of the medulla oblongata - postischemic. On the CDDS of the carotid and vertebral arteries, at the transition of V2 to V3 segment of both vertebral arteries, turbulent flow with increased flow velocities and narrowing of the lumen with a diameter measuring 2.4 mm on the right and 2.6 mm on the left was observed. CT angiography of the neck and brain blood vessels was performed, revealing normal finding. Additionally, NMR of the brain with FS sequence (Figure 2) showed a segmental irregular lumen of the left vertebral artery at the level of the proximal intracranial segment, extending approximately 1 cm. A slight tortuosity and a hypersignal ring on the FS sequence were observed that might be suitable for dissection. The remaining segment of the left vertebral artery displayed an orderly lumen and flow, slightly dominant on the right. Echocardiography was performed, revealing normal findings, TCCD Bubble test - transtemporal approach - in the ACM bilaterally after contrast injection, about 4-5 microembolic signals were obtained. Over 50 microembolic signals with curtain effect were obtained with the Valsalva maneuver. The test was positive for right-to-left shunt grade 4. A cardiological examination was performed with the findings from echocardiography and TCCD Bubble test. Therapeutic recommendations were given and TEE and cardiac surgery consultation were recommended.

Additionally, due to laboratory verified elevation of anti-TPO, an endocrinologist was consulted who gave therapeutic recommendations.

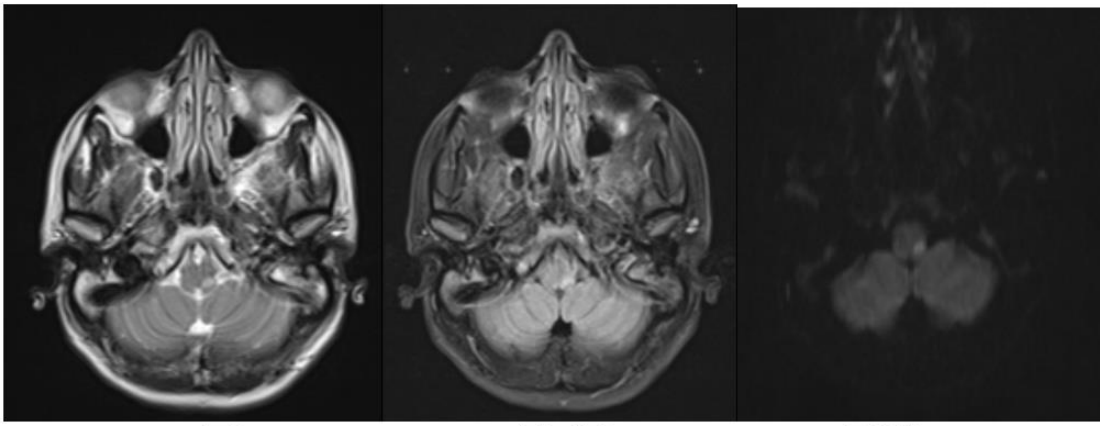

Fig. 1a. NMR of the brain with TOF sequences

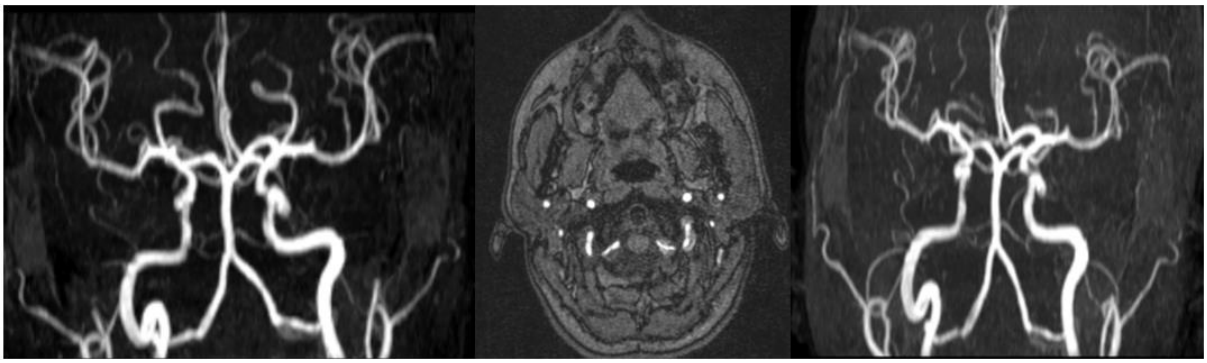

Fig.1b. NMR of the brain with T1FS sequence

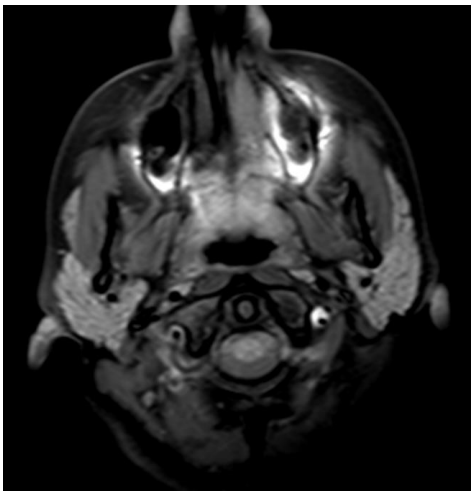

Fig. 2. NMR of the brain with FS sequence

The patient was treated with antiedematous, antiplatelet, low preventive thromboprophylactic doses of low molecular weight heparin, and antilipemic and symptomatic therapy. Under prescribed therapy, improvement was noted in the clinical neurological plan with complete withdrawal of the subjective feeling of dizziness, complete exhaustion of the

nystagmus; Romber was negative; gait was regular; discrete left-sided semi-ptosis persisted; during phonation the uvula was slightly deviated to the right, with a slightly lowered right palatine arch and a mildly weakened gag reflex. When testing the plantar-cutaneous reflex, flexor responses were obtained bilaterally, which were asymmetrical; the right one was with a slower reaction and a weaker tonic phase. When testing the modification according to Chedok, extensor courses were caused bilaterally, which were discrete on the left and on the right, and a clear extension of the fingers as well as a brief episode of fan-like spreading were registered.

The patient was discharged in a stable somatoneurological condition with a recommendation for antiaggregation and antilipemic therapy. After discharge from the hospital, the patient had a cardiological consultation, and underwent a transesophageal echocardiography, confirming the existence of a PFO, but without the need for its closure. She was advised to take antiaggregation and antilipemic therapy. The repeated tests for vasculitis showed normal findings as well as those for thrombophilia (MTHFR heterozygous).

The patient is regularly monitored in outpatient cardiology and neurology settings, without recurrence of the same or similar neurological symptomatology. She is functionally independentl within her family and has completely returned to her professional duties. Regular follow-ups with CDDS of the neck blood vessels show improvement in local findings, with normal hemodynamic indicators during insonation in both vertebral arteries.

Discussion

The vascular anatomy of the vertebrobasilar arterial system is quite consistent, and the vertebral arteries arise from the subclavian artery on each side respectively, and ascend to the base of the skull, uniting to form a single basilar artery[10]. In 70% of the population, the left vertebral artery is dominant. The vertebral arteries have their greatest mobility and vulnerability to mechanical injury at the level of C1-C2 vertebrae[11] at the exit point from the transverse foramen of the axis and its entry into the skull.

Dissection can occur at any point along the vertebral artery, although it most commonly affects its distal third[10].

Vertebral artery dissection can be spontaneous or traumatic. Traumatic vertebral artery dissection is most commonly associated with head, neck, and cervical spine injuries[12]. Dissection usually occurs at the time of traumatic injury, but as in our case, clinical presentation may occur with a time delay, some time after the injury.

Although most dissections heal spontaneously, a small proportion of patients have disease progression with complications and pseudoaneurysm formation.

Vertebral dissection can potentially lead to cerebral infarction[13].

The most common mechanism by which ischemia occurs in cases of extracranial arterial dissections is arterial embolism from an intraluminal thrombus, hence the rationale for the use of antithrombotic drugs (antiplatelet or anticoagulant) in secondary prevention to reduce the risk of ischemic stroke.

Digital subtraction angiography, which was considered the “gold standard” in the past, is no longer routinely used for the diagnosis of dissection.

Brain magnetic resonance imaging with DWI/ADC detects ischemic lesions with high specificity and sensitivity from the first minutes (hypersignal on the DWI sequence, hyposignal on the ADC sequence). Patients with acute ischemic stroke detected by DWI but not on the FLAIR sequence are likely to be eligible for thrombolysis (so-called DWI/FLAIR mismatch), if they meet the inclusion criteria.

Vertebral artery dissection is detected with high accuracy by the so-called “crescent sign” on T1 FS NMR sequences, and MRA sequences can reveal the absence of a “signal gap” in the affected vessel.

CT scans detect early signs of ischemia (parenchymal hypoattenuation, loss of gray/white matter differentiation), but several hours must elapse after the acute event before the infarcted area can be visualized.

Therapeutic options are still debatable in patients with acute ischemic stroke and vertebral or carotid artery dissection.

The CADISS clinical trial[14] included 250 patients, of whom 118 patients with carotid artery dissection and 132 patients with vertebral artery dissection. A total of 128 patients received antiplatelet therapy, while 124 patients received oral anticoagulant therapy. In this study, no statistical difference was found between the two groups in terms of recurrence of ischemic events or recanalization rate at one year.

According to the AHA guideline for secondary prevention of acute stroke[15], in patients with acute stroke or TIA less than 3 months after the acute event, it is reasonable to use either antiplatelet therapy (aspirin) or oral anticoagulant therapy (warfarin).

The European guidelines for the management of intra- and extracranial arterial dissections[16] recommend, in the case of extracranial arterial dissection complicated by stroke or TIA (transient ischemic attack), antiplatelet agents that have a better risk-benefit ratio than oral anticoagulants.

If there are neurological or other comorbid conditions that require oral anticoagulant therapy, it is recommended to use a direct anticoagulant (DOAC) instead of vitamin K antagonists.

In the case of a minor stroke or TIA caused by extracranial arterial dissection, the guideline suggests the use of dual antiplatelet therapy with aspirin and clopidogrel limited to a few weeks.

In exceptional cases, in which recurrent ischemic events occur despite optimal antithrombotic therapy or in case of extension of the dissection, endovascular or surgical treatment may be considered after evaluation by a multidisciplinary team consisting of a neuroradiologist, a neurologist and a neurosurgeon.

Dissection can be a clinical challenge to detect and diagnose due to its diverse presentation and the lack of uniform screening/guidelines[12]. This was the case in our patient, where the findings on the initial CT scan as well as CT angiography were normal. However, a thorough history, clinical experience, and clinical instinct were a crucial starting point for the diagnostic and therapeutic management of this case. Vertebral artery dissection is more common in polytrauma, especially if there are injuries to the head, neck, or cervical spine. Signs of spinal injury may mimic or mask the clinical features of vertebral artery dissection, and therefore early CT angiography and/or color Doppler and duplex ultrasonography should be used as a noninvasive screening tool in patients with significant traumatic injuries[13].

Conclusion

Neuroradiological examinations are essential for establishing the diagnosis of extracranial arterial dissection and quantifying the size of the lesion and the progression of the disease.

Our case report highlights the importance of a well-taken history and anamnestic clarification of all potential etiological factors, which serve as a foundation for further investigations. Neuroradiological imaging techniques play a key role in establishing an accurate and timely diagnosis and identification of the etiology of cerebral ischemic lesions. Each additional neuroradiological examination provides valuable information that helps in choosing the most appropriate therapeutic treatment.

There is no “gold standard” in the diagnosis of cervical dissections.

MRI with T1, T2, FLAIR and DWI/ADC sequences with cranial and cervical T1 FS can be used, which is useful for identifying small intramural hemorrhages. MRI should be complemented with MRA with contrast and TOF sequence of MRI.

As an alternative to MRI, CT with cerebral and cervical CT angiography can be used. The choice between these two methods is primarily based on the availability and experience of each center.

Therapeutic options are still debatable in patients with acute ischemic stroke and vertebral or carotid artery dissection.

This paper highlights the importance of neck trauma, even after some time latency following the initial injury, as a cause of vertebral artery dissection and subsequent ischemic brain injury. This enables timely diagnostic and therapeutic actions, which certainly contribute to a better final outcome, which is a basic goal in our daily work.

Conflict of interest statement. None declared.

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