Case report

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Young Female with Recurrent Spontaneous Cervical Artery Dissection

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Abstract - Previous neurosonological study demonstrated the value of neurosonology in daily monitoring of the cervical artery dissection. This study showed rate of recurrence of 27 % in unaffected arteries. There are two group of recurrences, early and late. Early spontaneous cervical artery dissection (sCAD) recurrences are not uncommon and usually involve arteries previously unaffected by dissection. They are oligosymptomatic (headache or neck pain) or asymptomatic and might be correlated with a transient arterial disorder. Late sCAD recurrences are rare and can occur at site of previous sCAD and might be indicative of an underlying persistent connective tissue weakness. In this case report we present a 31- year old female patient who presented to emergency room because of headache and pain in the neck which started 3 days before. Initial brain CT scan and CT-angiography were normal. Next day she became disoriented and complained of headache, vertigo, nausea. Magnetic resonance of the brain showed little hematoma in quadrigeminal cistern and left vertebral artery dissection in V2 segment. Initial neurovascular ultrasound (nUS) showed dissection in V2 segment left vertebral artery. Next nUS after five days showed new dissection in V1 and V2 segment in right vertebral artery. Following nUS showed initial regression hematoma in both vertebral arteries. To conclude, early sCAD recurrences are not uncommon and usually involve arteries previously unaffected by dissection. Because of that it is important to daily neurosonology monitoring of the dissection.

Key words: dissection; cerebral arteries; diagnostic imaging; recurrence

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Introduction

The cervical arteries are both internal carotid and vertebral arteries. Dissection of one or both can be asymptomatic but can also lead to cerebral ischemia, stroke, subarachnoid haemorrhage or death [1]. Spontaneous cervical artery dissection (sCAD) is common cause of stroke in young and middle-aged patients [2]. Only 1 % to 2 % of all ischemic strokes are caused by sCAD, but in younger patients, sCAD accounts for 10 % to 25 % of strokes [2]. Separation of the arterial wall layers results in dissection and causes that blood collect between layers as an intramural hematoma. The development of intramural hematoma with subintimal dissections causes luminal stenosis or occlusion. This may result in cerebral ischemia due to thromboembolism, hypoperfusion, or a combination of both [3,4]. Extracranial sCAD are more common than intracranial dissection [5]. Pathophysiology of the sCAD is likely multifactorial, with

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both genetic predisposition and minor trauma which is estimated play role in approximately 40 % of cases of spontaneous CAD [6]. Various connective tissue disorder and vascular abnormalities have been associated with dissection, and most common association is with fibromuscular dysplasia. The symptoms of sCAD may vary from asymptomatic to medullary ischemia to ischemic stroke. Most common symptoms of SCAD include headache and neck pain, Horner syndrome, pulsatile tinnitus, and signs of cerebral ischemia or transient ischemic attack [4,7]. Asymptomatic patients or associated with local symptoms only usually are undiagnosed [8]. Early recurrence is not uncommon and usually involves arteries previously unaffected by dissection [9]. Mural hematomas are best detected by axial cervical MR using the T1 fat suppression technique, neurovascular ultrasound (nUS) is most widely and accessible tool for screening and monitoring of the course of the disease [8,10,11]. The hematoma usually resolves within 6 months, and the outcome is mostly good.

Subjects and Methods

In this case report we present a 31 - year old female patient who presented to neurological emergency room because of headache and pain in the neck which started 3 days before she presented to emergency department. At home she had taken analgesics without pain reduction. Her neurological status was normal. Initial brain computer tomography (CT) scan and CT-angiography performed in emergency room were normal. Lumbar puncture was done, and the analysis of cerebrospinal fluid was regular. Laboratory testing were normal. After analgesic she was discharged. The next day she became disoriented and complained of worsening of headache, vertigo, nausea. She was healthy, non-smoker and from medication she was taking only oral contraceptives. She denied recent infection or neck and head trauma. She was admitted to hospital and magnetic resonance was performed. Magnetic resonance of the brain showed little hematoma in quadrigeminal cistern and left vertebral artery dissection in V2 segment (Figure 1). Still her neurological finding was normal, she was normotensive and had no fever. Initial neurovascular ultrasound (nUS) showed dissection in V2 segment left vertebral artery (Figure 2), and ultrasound of both carotid arteries, and right vertebral artery was normal. Therapy with antiplatelet was initiated. Next nUS after five days showed new dissection in V1 and V2 segment in right vertebral artery (Figure 3), and progression of hematoma in left vertebral artery involving V1 and V1 segment. She didn't have any new symptoms or had worsening of headache. Control magnetic resonance of the head and neck confirmed dissection of the left vertebral artery in V2 segment extending throughout V1 and V3 segment, confirmed new dissection in right vertebral artery in V1 and V2 segment. Hematoma in

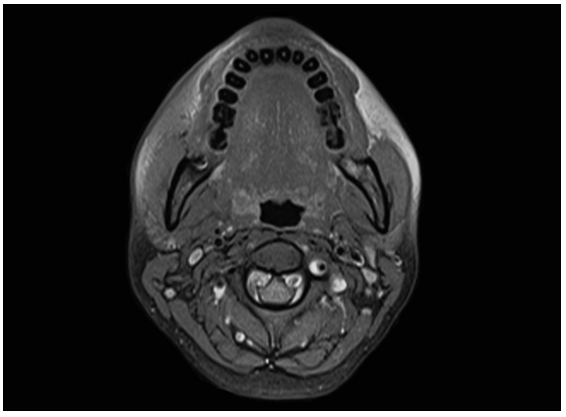

Figure 1. Brain magnetic resonance imaging scan showing left vertebral artery dissection

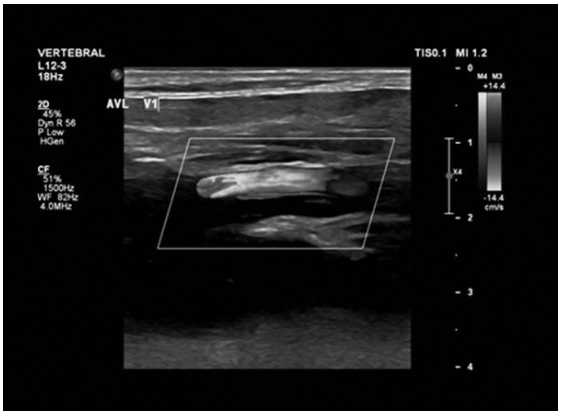

Figure 2. Neurovascular ultrasound showing hematoma in V2 segment in left vertebral artery

quadrigeminal cistern was in regression. During hospitalization repeated neurovascular ultrasound showed initial regression in size of hematoma in both vertebral arteries. Cardiology evaluation was normal, and extensive laboratory testing for hypercoagulable state and autoimmune disease did not show any significant abnormal deviation. Test for monogenetic connective tissue disease and vasculitis was negative. After twenty-two days in hospital, she was discharge with recommendation for antiplatelet therapy. Two months after dissection repeated neurovascular ultrasound showed minimal residual hematoma in left and right vertebral artery (Figure 4) Four months after dissection neurovascular ultrasound showed complete recanalization of both vertebral arteries without hematoma.

Discussion

We present a young woman with recurrent spontaneous bilateral vertebral dissection. Pathophysiology of the CeAd is likely multifactorial, and risk factors include genetic predisposition, minor trauma, migraine headaches, pregnancy and postpartum, previous infection or inflammation, connective tissue disease and others. Medical history and extensive tests during hospitalization did not determine cause of her sCAD. Vertebral artery dissection most often occurs in the cervical transverse processes of C6 to C2 (V2 segment) which was also the case with our patient [12]. Patient presented with most common symptoms, headache, pain in the neck and little hematoma in quadrigeminal cistern. Neurosonology tests were used for assessment and monitoring of the dissection. Magnetic resonance angiography confirmed the diagnosis of dissection. Spontaneous CAD is a highly dynamic process, with a generally benign prognosis. Previous neurosonology studies showed high sensitivity of extracranial colour Doppler for the assessment of sCAD. Barachini and associates demonstrated the value of neurosonology in daily monitoring of the dissection and revealed rate of recurrence of 27 % in unaffected arteries [9]. Spontaneous CAD recurrences can be divided in two groups, early and late one. Early sCAD recurrence is not uncommon and usually involves arteries previously unaffected by dissection, often is oligosymptomatic or asymptomatic and occur in first month [9]. Late sCAD recurrence is rare and can occur at site of previous sCAD and might be indicative of an underlying connective tissue weakness [9]. Because early recurrence is common, we did daily neurosonologic monitoring. Patient has early recurrence, and it was oligosymptomatic. In study Baracini and associates all but one complete recanalization occurred within the first 9 month, most of them

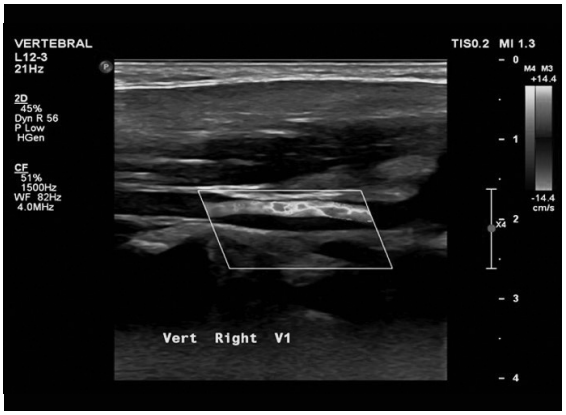

Figure 3. Neurovascular ultrasound showing hematoma in V1 segment in right vertebral artery

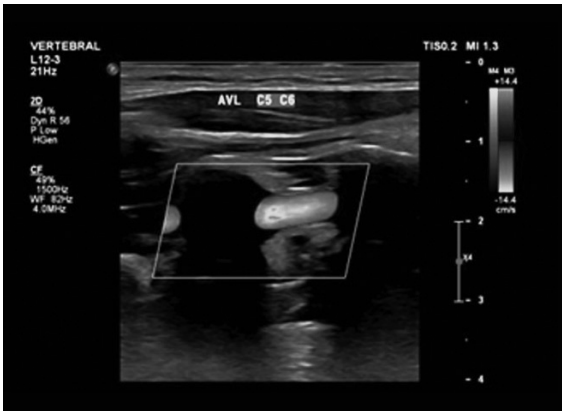

Figure 4. Neurovascular ultrasound showing minimal residual hematoma in left right vertebral artery

within 3 months [9]. Our patient has complete recanalization in both vertebral arteries after 4 months. The Cervical artery Dissection in Stroke Study (CADISS) data was published in early 2015, and the authors found no difference in the efficacy of antiplatelet versus anticoagulant drugs at preventing stroke and death in patients with sCAD [13]. Our patient was treated with antiplatelet medication.

We present a young woman with recurrent spontaneous bilateral vertebral dissection that cause little hematoma in quadrigeminal cistern. Spontaneous CAD is an uncommon yet important cause of stroke, especially in the young. Sometimes it is a difficult to diagnosed because one third of patient present with nonspecific symptoms, such a headache and neck pain. The diagnosis of sCAD should be suspected in patients presenting with acute headache, neck pain, stroke like symptoms and a history of mild trauma, or family history of connective tissue disease. Early spontaneous recurrence is not uncommon. That is why neurosonology is very important in daily monitoring of the dissection.

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Conflict of interest

None to declare.

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None.

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