The Dissections of Craniocervical Arteries

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ABSTRACT

Dissection of craniocervical arteries internal carotid artery (ICA), or vertebral artery (VA) is an increasingly recognized entity and infrequent cause of stroke. We investigated 8 patients (4 women and 4 men) with dissections of the craniocervical arteries. Diagnostic procedures for detection of craniocervical dissection included: extracranial ultrasound-color Doppler flow imaging (CDFI) of carotid and vertebral arteries, transcranial Doppler sonography (TCD) and radiological computed tomography (CT) and digital subtractive angiography (DSA) examinations. Ultrasound findings (CDFI of carotid and vertebral arteries) were positive for vessel dissection in seven patients (or 87.5 per cent) and negative in one patient. DSA was consistent with dissection in five patients (or 62.5 per cent), negative in one, while in two patients the examination was not performed due to known allergy to contrast media. Five patients (62.5 per cent) were treated with anticoagulants, one with suppressors of platelet aggregation, and two patients were operated. Six patients (75 per cent) after the treatment showed partial recovery of neurological defects, and an improvement of ultrasound finding of dissected arteries. In one patient, following operation, stroke developed with deterioration of motor deficit, and one patient was readmitted three months later due to a newly developed stroke and soon died. The diagnosis should be suspected in any young or middle-age patient with new onset of otherwise unexplained unremitting headache or neck ache, especially in association with transient or permanent focal neurological deficits.

Key words: craniocervical arterial dissection, color Doppler flow imaging, digital subtractive angiography, stroke

Introduction

The dissections of craniocervical arteries (carotid and vertebral) are sudden lesions of the arterial wall. The early clinical manifestations are often subtle, but permanent neurological disability and death can result if the diagnosis is delayed^{1,2,3}. They are infrequent cause of cerebral stroke (0.4–2.5 per cent) in general population, more often in younger age (5–20 per cent) and in women.

In majority of cases, the cause and the pathogenesis of dissections are not clarified 1,2,3,4 .

The dissections of arterial wall are initiated by the rupture of vasa vasorum of the media leading to the hemorrhage within the wall and separating its layers, or by the primary lesion of the intima. If the process is progressing from separated layers of the media towards the lumen, secondary stenosis or occlusion by the thrombosis of dissected portion of the vessel could occur. Further embolic accidents rising from a thrombotic vessel are also possible. The dissection progressing towards the adventitia results in a pseudo aneurysm of the wall and threatens with the rupture¹.

The dissections are divided in: 1. traumatic (head and neck trauma with concomitant lesions of the arteries), and 2. spontaneous. The commonest disorders that predispose dissection are; fibromuscular dysplasia, cystic medial necrosis, Marfan's syndrome^{1,2,3,4}. Increased predisposition to dissection was found in patients with decreased level of alpha 1 antitripsin^{5,6}. In some subjects dissections were repeated, in others there was a familiar predisposition^{1–10}.

Even some insignificant »trivial« traumas (coughing, sneezing, vomiting, excessive exercise, sudden rotation of head and neck, awkward sleeping position, chiropractic grips) could result in spontaneous dissection of

Received for publication November 8, 2002

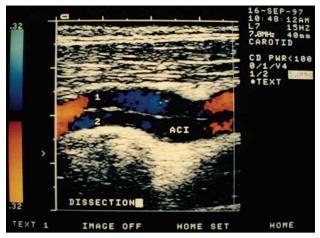


Fig. 1. Extracranial ultrasound color Doppler flow imaging showing dissection of the internal carotid artery. ACI – arteria carotis interna.

arteries^{1,11}. In 1/3 of patients, dissections could be multiple involving more than one artery¹¹. In respect with localization, dissections of craniocervical arteries could be extra or intracranial.

Common symptoms are abrupt headache, neck ache and focal neurological signs. An additional asymptomatic dissection of another artery is found in about 20 per cent of cases and appears to be more frequent when symptomatic dissection involves the vertebral arte $ry^{2,4,6}$.

It is important to distinguish dissection from atheromatous disease, which may have a very similar angiographic appearance, because the treatments are different for these two processes. Internal carotid artery (ICA) dissection can usually be distinguished from atherosclerotic narrowing by location, shape, and associated clinical and angiographic features. ICA dissection usually occurs at sites that are atypical for atheromatous disease. They are frequently located a few centimeters distal to the common carotid bifurcation, while atheromatous changes are usually located just at the ICA origin¹.

Diagnostic procedures include ultrasound (CDFI (Figure 1) and TCD)¹²⁻¹⁶ and radiological [DSA (Figure 2), MRI – magnetic resonance imaging, MRA – magnetic resonance angiography, CT] methods¹⁷⁻¹⁹.

Treatment generally consists of administration of anticoagulants or suppressors of platelet aggregation. Operative treatment is occasionally performed¹.

Patients and Methods

Eight patients (four males and four females) aged 41-66 years with dissection of craniocervical arteries (internal carotid and vertebral) (Figure 3), admitted to the University Department of Neurology, Sestre Milosrdnice University Hospital, Zagreb, between May 1998 and June 1999 are reported. In that period, there were 697 stroke patients admitted to neurology department and in eight patients stroke was caused by craniocervical dissections. Patient history was taken and clinical neurological examinations performed immediately after the admission. Diagnostic procedures included ultrasound (CDFI and TCD) and radiological (CT and DSA) examinations. The patients were analyzed immediately after the admission. For extracranial Doppler examination we used real-time duplex ultrasound system (Acuson 128 XP), consisting of a 7.5 MHz probe for morphologic evaluation and 5 MHz probe for haemodinamic evaluation of the craniocervical arteries. The extracranial carotid and vertebral arteries were imaged on both sides, and blood flow velocities were recorded. A pulsed-wave Doppler device operating at 2 MHz was used for all transcranial Doppler ultrasound examinations (TCD DWL Multi Dop L). Middle cerebral, ante-



Fig. 2. Digital subtractive angiography imaging showing dissection of the internal carotid artery.

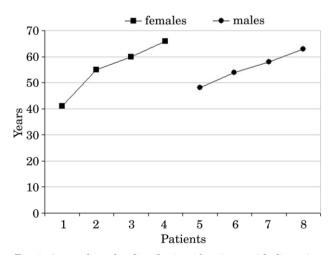


Fig. 3. Age and gender distribution of patients with dissection of craniocervical arteries.

rior cerebral, posterior cerebral artery, and ICA bifurcation were analyzed at different depths by transtemporal approach, and intracranial VA and basilar artery through the occipital foramen. DSA was performed by the percutaneous femoral approach. CT examinations were performed with five-millimeter imaging (Siemens). Risk factors, treatment and outcome of the disease were analyzed.

Results

There were 697 stroke patients admitted to neurology department between May 1998 and June 1999 and in 8 patients stroke was caused by craniocervical dissections (1.1%). We evaluated 8 patients with dissections of the craniocervical (carotid and vertebral) arteries. Spontaneous dissections were found in six and posttraumatic in two subjects. A dissection of internal carotid artery was found in four patients and vertebral artery dissection was found in two patients. One patient had dissections of both internal carotid and vertebral arteries on ipsilateral side and one patient, together with internal carotid artery dissection showed an intracranial aneurysm (Figure 4). Leading symptoms were focal neurological defects, head - and neck ache. CDFI of carotid and vertebral arteries was positive for vessel dissection in seven and negative in only one patient. Patient with negative CDFI was admitted to department of neurology due to subarachnoidal hemorrhage. Subject underwent conventional DSA procedure and craniocervical dissection was detected. Afterwards, subject was scheduled for follow-up by means of CDFI. The finding of CDFI was negative, probably due to vessel wall restitution. DSA was consistent with dissection in five patients, negative in one, while in two the examination was not performed due to known allergy to contrast me-

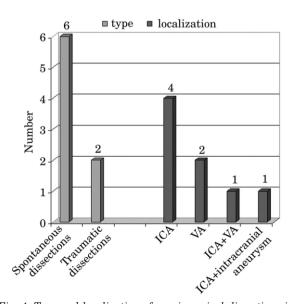


Fig. 4. Type and localization of craniocervical dissections in subjects. ICA – internal carotid artery, VA – vertebral artery.

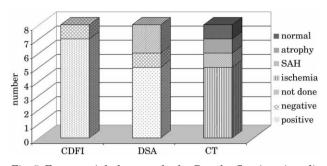


Fig. 5. Extracranial ultrasound color Doppler flow imaging, digital subtractive angiography and computed tomography findings in patients with dissection of craniocervical arteries. CDFI – Color Doppler Flow Imaging, DSA – Digital Subtractive Angiography, CT – Computed Tomography, SAH – subarachnoidal hemorrhage.

dia. In a patient with negative DSA finding, diagnosis of craniocervical dissection was first recognized by means of CDFI. Afterwards, patient underwent conventional angiography procedure, which is considered the gold standard for diagnosis of dissection. We assume that negative DSA probably resulted from blood vessel wall restitution that occurred in the meanwhile. Ischemic lesion on CT was reported in five patients, subarachnoidal hemorrhage in one, atrophy in one, while in one patient CT finding was normal (Figure 5). When risk factors were examined, hypertension was found in seven patients, hyperlipidaemia and/or cardiac disease in three, diabetes mellitus and smoking in two patients. Two patients were operated, five treated with anticoagulants and one with suppressors of platelet aggregation. Following treatment six patients (one operated and five treated with anticoagulants) showed partial recovery of neurological defects together with an improvement of ultrasound finding of dissected arteries. In one patient, following operation, stroke developed with deterioration of motor deficit. Last patient, treated only with suppressors of platelet aggregation, was readmitted three months later due to a newly developed stroke and soon died.

Conclusion

The dissections of craniocervical arteries are infrequent but important cause of stroke, especially in younger population. It is prerequisite to recognize this disease by clinical examination, to perform non-aggressive ultrasound and than radiological examinations to verify clinical diagnosis. Noninvasive ultrasound methods have high temporal resolution and practical features for bedside monitoring of the patients Although DSA is considered to be the gold standard for detection of dissection, vascular ultrasound has also proven to be sensitive for craniocervical dissections. Risk factors should be minimized, anticoagulant treatment started as soon as possible in order to decrease the risk of heavy stroke and lethal outcome.

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DISEKCIJE KRANIOCERVIKALNIH ARTERIJA

SAŽETAK

Disekcije kraniocervikalnih (karotidnih i vertebralnih) arterija, su sve češće prepoznate kao uzrok cerebrovaskularnog inzulta. Prikazali smo 8 bolesnika (4 žene i 4 muškarca), sa disekcijama kraniocervikalnih arterija. Dijagnostičke metode kojima su bili podvrgnuti uključivale su ultrazvučne (CDFI – ekstrakranijski kolor doppler karotidnih i vertebralnih arterija, TCD – transkranijsku doppler sonografiju) te radiološke pretrage (CT – kompjutoriziranu tomografiju mozga i DSA – digitalnu subtrakcijsku angiografiju). U 7 slučajeva (87.5%) CDFI karotidnih i vertebralnih arterija pokazao je pozitivan nalaz disekcije žile. Pet bolesnika imalo je pozitivan nalaz DSA (62.5%), jedan negativan, a u dva bolesnika pretraga nije učinjena radi alergije na kontrast. Pet bolesnika (62.5%) liječeno je antikoagulancijama, jedan antiagregacijskom terapijom, a dvoje operativno. Kod šest bolesnika (75%) je nakon terapije došlo do djelomičnog kliničkog oporavka i poboljšanja UZV nalaza na diseciranim krvnim žilama. Jedan je bolesnik nakon operativnog zahvata doživio recidiv moždanog udara uz pogoršanje neurološkog deficita, a jedan je nakon tri mjeseca doživio ponovni moždani udar sa smrtnim ishodom. Na dijagnozu se mora posumnjati u mlađih ili bolesnika srednje dobi sa naglim neobjašnjivim glavoboljama ili bolovima u vratu straga, osobito ako su tegobe udružene sa prolaznim ili trajnim neurološkim deficitom.