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A case report



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Surgical treatment of spontaneous common carotid dissection. A case report

BACKGROUND: Cervical carotid dissection is more common in extracranial vessel: internal carotid artery dissection (ICAD) is typical, vertebral artery dissection is uncommon, common carotid artery dissection (CCAD) is rare and even a more rare cause of ischemic stroke. Cervical artery dissections account up to 20-25% of ischemic strokes in young patients. Isolated and spontaneous common carotid artery dissection without aortic damage is unique. Indeed in the Literature 8 cases were identified. MRI and CTA were the most commonly used for diagnosis and follow-up.

CASE REPORT: A 67-year-old came to our observation reporting burning pain in the right latero-cervical region in supine position, irradiated in the temporal region and recurrent episodes of migraine with aura (scintillating scotoma), in the last 3 months. The last Doppler Ultrasound control, performed after the onset of symptoms, showed an highlighted dissection wall with double lumen at the origin of the bulb and the internal carotid artery on the right. Aortic arch arteriography confirmed the diagnosis. The patient underwent surgery (longitudinal arteriotomy, removing four intimal flaps, fastening the distal common carotid artery with 3 Kunlin's points).

RESULTS: Any neurological or vascular problems after surgery were noticed.

DISCUSSION AND COMMENTS: The pathogenesis can be related to a combination of an intrinsic weakness in the arterial wall and an external trigger. The diagnosis of CAD is made with MRI (78.0%), conventional angiography (31.1%), CTA (14.7%), and ultrasound (11.3%).

CONCLUSION: No evidence-based guidelines exists for treatment of CCAD. In our patient surgical CEA treatment was the optimal solution.

KEY WORDS: Carotid dissection, Vascular emergency

Introduction

Cervical artery dissections account up to 20-25% of ischemic strokes in young patients ¹.

The incidence of vertebral artery dissection (VAD) has been estimated ² to be 1.5 per 100000 population year-

ly whereas of carotid artery dissection has been estimated to be 3 for 1000000 population yearly.

Cervical carotid dissection is more common in extracranial vessel: internal carotid artery dissection (ICAD) is typical, vertebral artery dissection is uncommon, common carotid artery dissection (CCAD) is rare and even a more rare cause of ischemic stroke ^{3,5}.

The mean age of the patients is 40-46 years, rare in children. It occurs earlier in women than in men ⁴.

The dissection begins as a tear or defect of the intimal lining of neck arteries. The intima will usually separate from the medial layer, typically in the direction of blood

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flow and causes a formation of an intramural hematoma. Subintimal dissection turns into formation of a pseudoaneurysm.

In a recent Review of cervical artery dissections, no cases of CCAD causing stroke were described.

Isolated and spontaneous common carotid artery dissection without aortic damage is unique. Indeed in the Literature 8 cases were identified ⁶.

Case Report

A 67-year-old hypertensive with cardiovascular disease family history, dyslipidemic, non smoker, thyrotoxic woman was admitted in our Department.

She came to our observation reporting burning pain in the right latero-cervical region in supine position, irradiated in the temporal region and recurrent episodes of migraine with aura (scintillating scotoma), in the last 3 months. The patient underwent endarterectomy for right internal carotid artery stenosis 10 years ago and aorto-bifemoral graft placement for abdominal aortic aneurysm with bilateral stenosis of the iliac arteries 6 years ago. The last Doppler Ultrasound control, performed after the onset of symptoms, showed an highlighted dissection wall with double lumen at the origin of the bulb and the internal carotid artery on the right (Fig. 1).

CT angiography (Fig. 2) showed at the level of the right common carotid a disconnected intimal extended to about 25 mm to about 3 cm from the bifurcation and two small breaches of entry and exit were observed, without signs of thrombosis of the false lumen.

Aortic arch arteriography confirmed the diagnosis and showed a stenosis of about 30% of the left subclavian artery. The patient underwent surgery (longitudinal arteriotomy, removing four intimal flaps, fastening the distal common carotid artery with 3 Kunlin's points) (Fig. 3).



Fig. 1

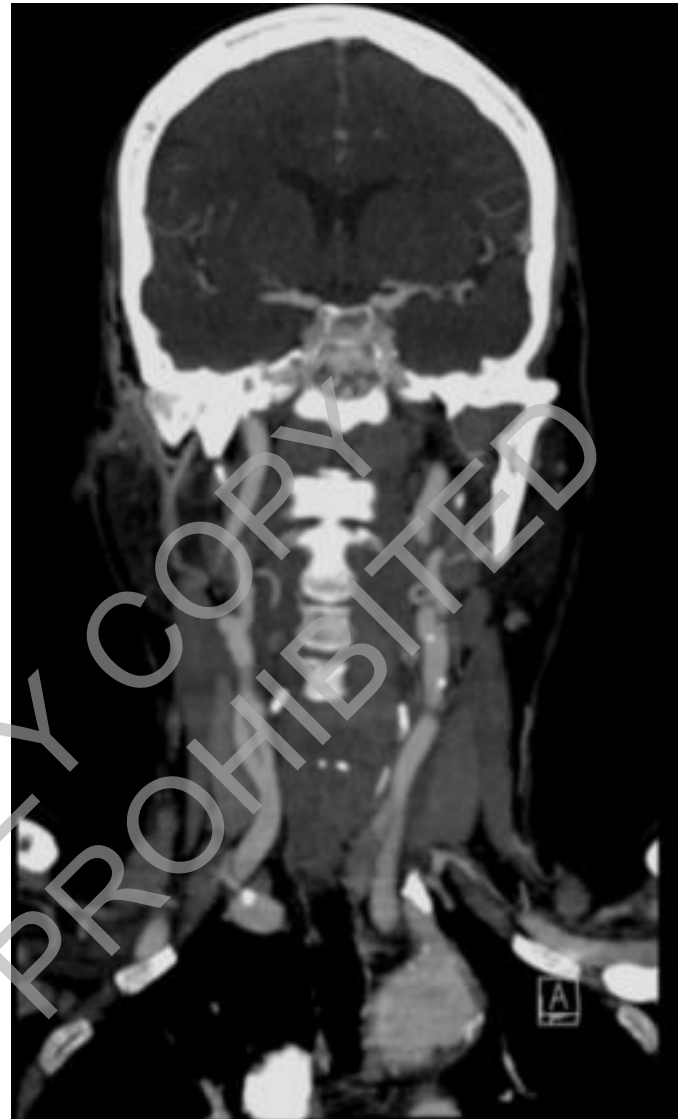


Fig. 2

Results

Any neurological or vascular problems after surgery were noticed. After 6 months the patient didn't show any symptoms and the Ultrasound control showed no evidence of neck vessel defect.

Discussion and Comments

The pathogenesis can be related to a combination of an intrinsic weakness in the arterial wall and an external trigger that occurs in 81% of the cases. In 19% of cases, when no obvious mechanism or trigger can be identified, the dissection is classified as spontaneous ⁷.

Proposed extrinsic factors are mayor neck trauma, or minor trauma including neck manipulation, cervical spine manual therapy, specifically high velocity thrust

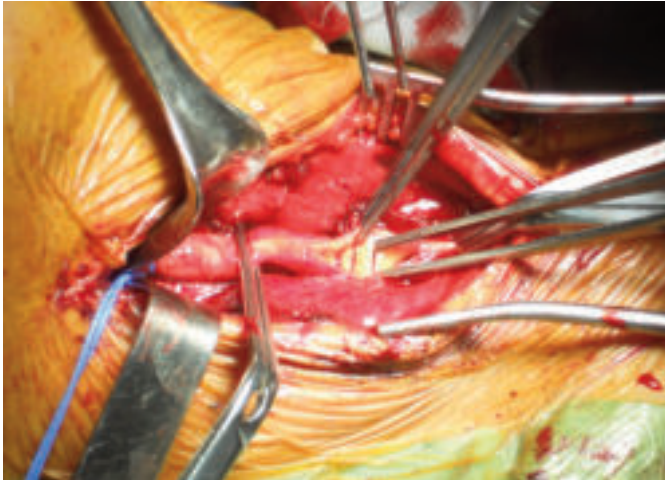


Fig. 3

manipulation or end-range rotational mobilization techniques.

Intrinsic susceptibility is thought to be a generalized arteriopathy, vascular anomaly or genetic predisposition (defect of alpha 1 antitrypsin, hyperhomocysteinemia with MTHFR TT genotype), Connective tissue disorder (Ehlers-Danlos syndrome type IV, Marfan's Syndrome, Cystic medial necrosis, polycystic kidney disease), Muscular fibrous dysplasia, Moyamoya⁸, infections, cardiovascular risk factor (hypertension Diabetes, Hypercholesterolemia, smoking), contraceptive pill use.

ICA dissection presents with local symptoms or signs associated or not to ischemic stroke.

Local signs and symptoms are craniocervical pain, facial pain, ipsilateral frontotemporal headache, tinnitus, cranial nervous paresis (especially for IX, X and XII, rarely XI, V, VI).

Ischemic events include TIA, homolateral amaurosis fugax, distal ischemic stroke in medial cerebral artery area⁹. However, it has been suggested that for dissection to occur there is a contribution from both intrinsic and extrinsic factors. It has been proposed that the mechanism involves both a preexisting intrinsic susceptibility such as hypovolemic events, distal embolic events, several thrombosis caused by a medio-intimal damage or intramural hematoma that causes acute thrombosis.

The diagnosis of CAD is made with MRI (78.0%), conventional angiography (31.1%), CTA (14.7%), and ultrasound (11.3%).

Ultrasound shows a luminal stenosis or flow reduction carotid bifurcation and rarely a double lumen (pathognomonic sign), intraluminal hyperechoic/isoechoic lesion, intimal flap. MRI and CTA were the most commonly used imaging modalities both for diagnosis and follow-up.

An obvious advantage of MRI is the ability to perform diffusion-weighted imaging to diagnose acute ischemia, axial T1 fat-saturation imaging through the neck to elucidate intramural methemoglobin, and time-of-flight and contrast-enhanced MRA to survey the neck vessels in a single study.

Digital subtraction angiography (DSA) is still the gold standard for SCAD diagnosis when a vertebral artery dissection is suspected. Angiographic evidence include luminal irregular stenosis (tie sign), occlusion mouse's tail sign, or evidence of aneurysmatic dilation; medio-intimal flaps and double lumen are rarely observed¹⁰.

Dissection site is still important for the prognosis: mortality rate in intracranial carotid dissection (75%) is more than the extracranial presentation (<10%). Recurring is 2% in the first month's after the treatment, 1% a year for ten year and is most frequent in young patients and patients with a familiar tendency.

Conclusion

Treatment of CCAD varies based on the etiology. Goals include prevention of vessel-to-vessel embolism, prevention of thrombus progression, and maintenance of adequate blood flow to ensure distal perfusion. Options include antiplatelet therapy and anticoagulation therapy for 6 months after the acute episode; endovascular angioplasty and stenting in case of complicated lesion and tight stenosis¹¹.

In our patient surgical CEA treatment was the optimal solution, as antiplatelet therapy was unsuccessful and in consideration of her hostile anatomy. No evidence-based guidelines exists for treatment of CCAD, and prospective, randomized trials are needed to determine optimal treatment.

Riassunto

INTRODUZIONE: La dissecazione dei vasi epiaortici è responsabile del 20%-25% degli ictus ischemici giovanili. L'incidenza annuale riportata in uno studio francese del 2008 di Menon et coll è pari a 3 per 100.000 per la dissecazione carotidea e 1,5 per 100.000 per la dissecazione vertebrale. Dissecazioni isolate e spontanee dell'arteria carotide comune, non associate a dissecazione aortiche sono estremamente rare: dal 1960 al 2003 sono stati riportati solo 8 casi in letteratura.

CASO CLINICO: Donna di 67 anni, affetta da ipertensione arteriosa e dislipidemia in trattamento con terapia medica. Non fumatrice, anamnesi familiare positiva per patologia cardiovascolare. Pregresso intervento di EA carotidea destra ed innesto aorto-bifemorale per patologia aneurismatica dell'aorta addominale e ostruttiva delle arterie iliache comuni. Giungeva alla nostra osservazione con la seguente sintomatologia: dolore urente in regione latero-cervicale destra che compariva in clinostatismo, irradiato in regione temporale ed associato a scotomi scintillanti e tinnito. La paziente veniva sottoposta ad ECD TSA d'urgenza che metteva in evidenza dissecazione parietale con doppio lume in corrispondenza della carotide comune destra. Tale reperto veniva confer-

mato anche all' esame Angio TC. Si procedeva, quindi, ad arteriografia dell'arco aortico e dei vasi epiaortici, per via transascellare sinistra, ma per l'anatomia sfavorevole dell'arco aortico si soprassedeva alla correzione endoluminale. Veniva, quindi, sottoposta ad intervento chirurgico, durante il quale veniva riscontrata la dissecazione intimale completa dell'arteria carotide comune (circa 4 cm) che all'estremo prossimale (origine carotide interna) presentava una placca residua. Si procedeva, quindi ad asportazione di quattro flap intimali e della placca residua e fissaggio con 3 punti di Kunlin dell'estremo distale dell'arteria carotide comune.

RISULTATI: IL decorso post-operatorio è stato regolare. Ad un mese la paziente è stata sottoposta ad ECD vasi epiaortici di controllo che evidenziava la pervietà dell'asse carotideo destro senza segni di ulteriori dissecazioni.

DISCUSSIONE E COMMENTI: L'eziologia della dissecazione dei vasi epiaortici non è ben definita. L'81% dei casi sono correlati a traumi cervicali maggiori o microtraumatismi ripetuti. Il restante 19% dei casi sono dissecazioni spontanee. Il quadro clinico della CCA consiste in sintomi e segni locali a cui possono seguire, dopo un intervallo di tempo variabile da ore a qualche giorno, manifestazioni ischemiche cerebrali. Per la definizione diagnostica e il monitoraggio è indicato l'utilizzo di metodiche sia ultrasonografiche che angiografiche non invasive (angio-TC o angio-RM). In casi selezionati o quando sussistono dubbi, in modo particolare nel sospetto di dissecazione vertebrale, può essere giustificato ricorrere all'angiografia convenzionale. La prognosi varia secondo la sede della dissecazione: la mortalità per dissecazione carotidea intracranica è più elevata (75%) rispetto alla forma extracranica (<10%). Il rischio di recidiva è del 2% durante il primo mese e dell' 1% all'anno per primi dieci anni ed è maggiore nei pazienti più giovani o con anamnesi familiare positiva.

CONCLUSIONE: Il trattamento di una dissecazione sintomatica per TIA o ictus rimane controverso in assenza di studi clinici randomizzati. In fase acuta il trattamento con eparina seguita da anticoagulanti orali (TAO), salvo controindicazioni, è raccomandato. Procedure endovascolari sono state usate per trattare stenosi gravi o lesioni complicate da pseudoaneurismi, ma non vi sono dati da studi randomizzati che ne abbiano definito l'utilità e

l'efficacia nel tempo. Nel nostro caso abbiamo optato per una terapia chirurgica in quanto la paziente era già in trattamento antiaggregante e non è stata percorribile la via del trattamento endovascolare a causa della sfavorevole anatomia.

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