

Case Report

Identification of internal carotid artery dissection in patients with Migraine — Case report and literature review

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Abstract

Although patho-physiology of spontaneous internal carotid artery dissection (sICAD) is largely unknown, an association with migraine has been suggested but not proven.

Migraine is a condition which is worth considering while one is hunting a possible cause for internal carotid artery dissection (ICAD) and it may be found more often than expected.² To date it remains a diagnosis of exclusion in patients with migraine. As opposed to migraine with aura, migraine without aura is significantly more frequent among patients with SICAD. It has been suggested that ICAD produces stroke in 36-68% of patients as a result of occlusion of the artery at or near the site of the dissection, or embolization occurring distally from a dislodged fragment of thrombus. We report a 31-year old woman with headache and ptosis as initial symptoms. Magnetic resonance imaging

(MRI) confirmed the diagnosis. Prompt treatment was instituted with anti-platelet agents and the patient had complete resolution of symptoms. Our case report highlights the importance of identifying the patients with ICD with history of migraine, in the absence of other risk factors and adds to the sparse literature currently available on the subject.

Introduction

Although dissections of the aorta is known to physicians and cardiologists for over a century, dissections of the neck arteries are rarely diagnosed by non-neurologists and hence, diagnosis is often delayed for this potentially treatable cause. The internal carotid artery (ICA) in the neck is the most commonly affected artery and is usually involved in distal extra cranial segments; well above the ICA origin.¹

Many of the diagnosed or labelled 'spontaneous dissections' may be triggered by minor physical events

considered inconsequential by the patient. There are many other causes of ICA dissection including congenital and acquired abnormalities of the connective tissue elements, e.g. Marfan's syndrome, cystic medial necrosis, fibro-muscular dysplasia, atherosclerosis etc. In the absence of these risk factors or aetiologies, it is worth considering possibility of migraine as a possible cause of ICA dissection. A recent published retrospective study has concluded the association of CAD with male sex, and possible association with smoking and migraine.³

Case Report

A previously healthy, 31-years-old white female with background history of migraine was admitted to us with drooping of left eyelid and moderately severe diffuse throbbing headache in June 2007. In addition she had mild retro-orbital pain on the same side. She also had transient 'floaters' in her left eye which settled within few minutes. The headache was different from her normal migraine headaches. There were no associated symptoms of limb weakness, speech or swallowing disturbances. She was an ex-smoker of three years (previously 10 cigarettes per day for 5 years) and consumed alcohol socially. She was not on any medication apart from oral contraceptive pill. Her family history was insignificant.

Neurological examination revealed left sided partial ptosis, small pupil, and no disturbance of sweating.

Her Laboratory investigations including routine bloods, coagulation profile, and auto-antibody screen including anti-cardiolipin Antibody, c-ANCA and p-ANCA were all normal. Urine for proteins and Homogenistic acid were also negative.

She had computed tomography (CT) scan of the brain which was normal. Magnetic resonance imaging (MRI) of the brain and magnetic resonance angiography (MRA) for neck arteries was organised which confirmed dissection of left ICA.

She was commenced on aspirin as she was planning for family and therefore wasn't considered for anticoagulation. Repeat MRA showed recanalized ICA but calibre was slightly reduced when compared to the right sided ICA. However she made full recovery within eight weeks of onset of her symptoms. She was initially followed up within one month, followed by a three monthly and six monthly follow up in the same year. Her follow up with Neurologist is on a yearly basis for five years. However, the patient has been advised to get in earlier, if symptomatic.

Discussion

The pathophysiology of carotid artery dissection can be either traumatic or spontaneous. The true incidence of spontaneous dissection is unknown. Once considered rare, increased awareness, combined with non-invasive evaluation by ultrasonography and MRA, has shown a more frequent

occurrence. Our Case-report highlights the fact that any focal neurology, especially in a young patient which can't be explained by CT scan findings needs further extensive investigations especially if there is background of migraine. This may help to prevent missing a potentially treatable condition. The following reveals the diagnostic confusion that one can come across while dealing with ICAD.

Headache and neck pain is the most common presenting symptoms of ICAD and is typically sudden in onset. The location of pain varies amongst patients, but most common sites are around the eye, front, or upper neck.³ Headache was reported to be presenting symptom in more than 90% of the patients in a series of 36 cases by Mokri B et al.¹ and headache was different in character than what they had experienced in the past. Silverman and Wityk described three cases of ICAD whose presentation suggested migrainous aura.⁴ Other common clinical manifestations are neurological ischaemic signs and a symptom, including stroke or transient ischaemic attacks.⁵ Another common sign of ICAD is an incomplete Horner's Syndrome that includes miosis and ptosis, but no anhidrosis,⁶ as was the case in our patient. Thus sudden onset painful Horner's Syndrome may suggest arterial dissection and should alert the clinician. Local symptoms consisting of head or neck pain preceded by Horner's Syndrome in 96% patients were described by Biousse et al in his study.⁷ The other symptoms which can be seen in this condition are tinnitus and visual scintillations. Some patients experience cranial nerve palsies as a result of a disruption of blood supply by either mechanical compression or by embolization.

Another diagnostic difficulty is the interval between the initiation of dissection and onset of the symptoms which may be hours to days and even few weeks in some cases. In general, focal cerebral ischaemic symptoms are the most common manifestations in traumatic group. Conversely, Horner's syndrome with headaches is a common clinical findings in spontaneous ICAD.⁸ The diagnosis of ICAD can be very difficult in patients with migraine especially before the onset of ischaemic signs,⁹ rather it may go undiagnosed.

In summary, based on our review, spontaneous internal carotid artery dissection is more common than most clinicians appreciate. The diagnosis can be missed by experienced physicians of all specialities. The clinical picture varies from mild cerebral and/or cranial nerve dysfunction to a completed stroke. Clinical appearance is primarily characterized by local signs such as headache or facial pain, Horner's syndrome, lower cranial nerve palsies, and pulsatile tinnitus, followed a few hours or days later by signs of cerebral or retinal ischaemia. Diagnosis of carotid artery dissection can be delayed, since the symptoms may be attributed to migraine, especially with transient symptoms resembling migraine with aura but in patients with history of migraine there is increased frequency of ICD, which we need to consider in such a clinical setting.

Angiography was traditionally considered to be the gold standard diagnostic test but has been recently replaced with MRA which is non-invasive and has similar diagnostic yield.

There is no single consensus regarding the treatment. Traditional medical intervention is anticoagulation or in selected cases anti-platelets. Surgical intervention is generally reserved for patients who failed to respond to anticoagulation and is usually in the form of percutaneous endovascular balloon angioplasty and stent placement. Most reports advocate immediate heparinization after diagnosis, followed by oral anticoagulation therapy for at least six months. However, few studies have shown good clinical outcome without anticoagulation therapy. Regardless of the treatment strategy patients should be monitored with serial sonography or MRA.

Surgical treatment of arterial dissections is warranted only if other measures do not prevent progressive ischaemic events. Also, surgical management of these dissections is warranted for patients without symptoms who have aneurysmal changes. These aneurysms may evolve and become symptomatic many months after a dissection. Although direct surgical access has been attempted by interposition grafting or even extra-cranial to intracranial bypass grafting and resultant exclusion of the involved segment, the technology for treatment today involves primarily balloon dilatation and stenting of the region involved, thereby excluding the aneurysm from the circulation. The prognosis of these arterial dissections is highly variable, excellent in cases limited to local signs but poor and leading to death or major sequelae in about 15% of cases.

Conclusion

In young women with a history of migraine, a careful study of the extra cranial and intracranial arteries is warranted especially if they have a different character of headache such as migraine which may represent a predisposing condition for sICAD. This is particularly true in the absence of other risk factors. Also painful Horner syndrome should alert clinicians to the possibility of a silent carotid dissection. Magnetic resonance imaging of neck arteries is the imaging modality of choice for suspected ICAD.

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