Carotid Artery Dissection after Scuba Diving

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Carotid artery dissection is a relatively unrecognized vascular injury, which may occur spontaneously or following various forms of trauma, in young persons without the classic vascular risk factors. Internal carotid artery dissection is one of the etiologies for stroke in this age group and should be considered in every young patient with stroke. This diagnosis should also be suspected in any case with the combination of Horner's syndrome and headache or neck pain [1].

Early diagnosis and anticoagulant treatment of carotid artery dissection are of paramount importance to prevent its complications: carotid artery thrombosis and embolic strokes. It is therefore important to be familiar with ICAD and its various presentations. We describe a patient with internal carotid artery dissection following scuba diving, who presented with headache, dysgeusia and Horner's syndrome and was successfully treated with anticoagulants, resulting in complete recovery within 3 weeks.

Patient Description

A 48 year old physician suffered severe pain in the left side of his neck and face for 3 days. On the second day he began to experience dysgeusia (all foods and beverages tasted bitter), and on the third day became alarmed when he noticed anisocoria (the right pupil was more dilated than the left). He had been scuba diving 2 days before the appearance of the neck pain. He remembered that he had felt some dizziness and stiff neck for about 10 minutes immediately after surfacing and had then carried a heavy bag containing all the wet diving equipment on his left shoulder. The depth of the dive was only 8 meters and the duration was 1.5 hours.

He was examined by a neuro-ophthalmologist who confirmed the diagnosis of left-sided Horner's syndrome (with all its components: ptosis, myosis, anhydrosis). The physical examination was otherwise normal. A computed tomography scan of the head and neck and chest X-rays were normal. Magnetic resonance imaging of the head with gadolinium demonstrated a dissection of the distal part of the extracranial portion of the left internal carotid artery. The patient was admitted to the neurology ward and was put on bed-rest. A second MRA confirmed the diagnosis of carotid artery dissection. Infusion of 25,000 units of heparin per day was begun and after one day he was also given warfarin (Coumadin®). After 5 days on heparin overlapping with warfarin, and without any new neurologic symptoms or signs, the patient was discharged with warfarin treatment for 6 months. The Horner syndrome and the dysgeusia resolved after 4 weeks. Two years after the incident, the patient is healthy and without any neurologic sequelae.

Comment

Internal carotid artery dissection is a relatively unknown entity, yet it is one of the major causes of cerebrovascular accident in young persons [1,2]. One study found ICAD to be the cause for 10–20% of strokes in young patients. While neurologists and vascular surgeons are familiar with this condition, internists, pediatricians and general practitioners – who may be the first to encounter such patients – are far less acquainted with it.

Trauma and primary arterial diseases are the main causes of ICAD. But in many cases of spontaneous dissection the etiology remains unknown. Minor trauma such as hyperextension or rotation of the neck during yoga, sports activity, general anesthesia, coughing and sneezing may be involved in some of these cases. These movements may cause the initial injury to the artery by mechanical stretching. Predisposing factors for ICAD have been described in some patients, including Marfan's syndrome, fibromuscular dysplasia, cystic medial necrosis and Ehler-Danlos syndrome [3]. Although we believe that the ICAD in our patient was caused by hyperextension of the neck during the dive, we cannot exclude the additional role of trauma to the artery caused by the heavy load that he carried on his shoulder after diving.

ICAD is characterized by local signs such as unilateral pain in the neck, headache, facial pain, Horner's syndrome and lower cranial nerve palsies, followed a few hours or days later by signs and symptoms of cerebral ischemia [3,4]. In a series of 44 patients with ICAD [1], cerebral ischemia was preceded by severe pain for more than 3 days in 60% of those patients who eventually suffered a stroke. The headache may resemble migraine, making it difficult to distin-

ICAD = internal carotid artery dissection
MRA = magnetic resonance angiography

guish between the two entities. In the same series [1], Horner's syndrome occurred in 48%, ipsilateral cerebral ischemia in 82%, ocular ischemia in 16%, and cranial nerve palsy in 5%. Cerebral or retinal ischemia has been reported in 50–95% of patients with ICAD. In one series 40% of the patients developed an ischemic stroke either at presentation or following the other presenting symptoms. Stroke is thought to result from embolization, originating from thrombus which is formed at the site of the arterial wall injury. The taste disorders as described in our patient are rare, and it is presumed that they result from injury to the chorda tympani, which is close to the carotid artery [5]. While the gold standard for diagnosing ICAD is conventional arteriography, magnetic resonance techniques are replacing conventional arteriography. Magnetic resonance angiography is both sensitive and accurate in diagnosing ICAD and can demonstrate the intramural hematoma itself. Computerized tomographic angiography, which is more available, may also be used for the diagnosis. Its results are similar to those of MRA but there is less experience in diagnosing ICAD with this technique. Ultrasonography has been increasingly used in the diagnosis of ICAD [3,4], with sensitivity of up to 95% and high accuracy.

The prognosis of ICAD is highly variable. It is excellent in cases that are diagnosed on the basis of local signs, but death or major neurologic sequelae has been reported in 15% of cases. The long-term prognosis for patients who survive the initial dissection is excellent. Within the first 2–3 months after the dissection, most of the cases of stenosis caused by the dissection resolve and two-thirds of the occlusions are recanalized [2,3].

There are no published reports of randomized studies comparing different therapeutic modalities for ICAD. Anticoagulation with heparin followed by warfarin is recommended, and despite the known hazards of this treatment it is believed that anticoagulation prevents thrombosis and embolization of intracranial arteries. The optimal duration of anticoagulation has not been determined, with most authorities recommending 3–6 months of anticoagulation. Surgical treatment and endovascular procedures (such as percutaneous angioplasty with stent implantation) should be considered in patients with persistent symptoms of ischemia despite anticoagulation.

ICAD is a challenging diagnosis that can be missed even by experienced clinicians. It should be suspected in any case of Horner's syndrome, particularly if accompanied by headache or neck pain. Early diagnosis is crucial so that anticoagulant therapy can be administered, thus preventing the development of stroke.

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