

Case report

An Unusual Case of Traumatic Internal Carotid Artery Dissection during Snowboarding

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Abstract

The presentation of Horner's syndrome following blunt trauma is uncommon, but is of important clinical significance. Identification of the constellation of signs of Horner's syndrome should, therefore, prompt urgent neuro-radiologic imaging. Early diagnosis and initiation of appropriate treatment can lead to excellent outcomes in the majority of cases and prevent devastating cerebral ischaemic damage. A progressive case of Horner's syndrome following blunt injury to the neck in an amateur snowboarder is presented.

Key words: Horner's syndrome, snowboard, anisocoria, amaurosis fugax.

Introduction

Oculosympathetic palsy or Horner's syndrome is the triad of meiosis, ptosis, and anhidrosis that results from disruption of the sympathetic pathways between the brain and the eye. Numerous aetiologies underlay Horner's syndrome and although the individual signs of the syndrome do not constitute an emergency, their presence makes any Horner's syndrome a potential vascular emergency due to the proximity of the internal carotid artery to the sympathetic ganglia. A progressive case of Horner's syndrome following blunt injury to the neck in an amateur snowboarder is presented.

Case report

A 39-year-old male fell during snowboarding. He landed on his back and right thumb and was treated for a Bennett's fracture. Two days later, he presented to Emergency Department for follow up of his fracture. His wife had noted his unequal pupil size prompting referral to the ophthalmic department (Figure 1).

He complained of a mild left sided periocular headache and a heavy feeling on the left side of his face. On examination, he was alert and systemically well. Snellen visual acuity was 6/6 bilaterally. He had partial left-sided eyelid ptosis with apparent enophthalmos of the left globe. The right pupil was larger than the left by 1mm. Both pupils were reactive to light. There was possible left sided anhidrosis, but this finding could not be ascertained firmly. The remainder of the ocular and neurological examination was normal. Both carotid arteries were palpable, and no thrill or bruits were noted. He had no prior ocular or vascular history.

The diagnosis of Horner's syndrome was made on the constellation of signs noted on examination. Of particular note was the presence of periocular and neck pain. The diagnosis was pharmacologically confirmed using 4% Cocaine drops to both eyes. The left pupil was not dilated compared to the contralateral side 30 minutes later. The presence of a painful Horner's syndrome prompted urgent liaison with the radiologist, and Computerised tomography (CT) scans of his head and neck were normal. Due to strong clinical suspicion, magnetic resonance imaging (MRI) with magnetic resonance angiography (MRA) was requested and demonstrated a dissection of the left internal carotid artery in the neck without occlusion of the lumen. The vertebral arteries were normal and no other intracranial abnormalities were noted. Warfarin anticoagulation was initiated, and the patient had no neurological sequelae. No complications were noted at four months review and the patient was subsequently maintained on antiplatelet therapy.



Figure 1. Photograph of patient, demonstrating left-sided Horner's syndrome (with permission).

Discussion

Horner's syndrome comprises ipsilateral partial ptosis, pupillary meiosis, apparent enophthalmos, facial anhidrosis, heterochromia of the irides (if before age two or congenital), and transient decrease in intraocular pressure. (Worthington and Snape, 1998) The syndrome arises from a lesion that interrupts the sympathetic neuronal pathways from the hypothalamus to the eye.

Sympathetic innervation to the eye consists of a three-neuron arc. The first order neuron originates in the dorsolateral hypothalamus. It descends through the reticular formation of the brainstem and travels to the ciliospinal centre of Budge between the levels of the eighth cervical and fourth thoracic vertebrae (C8-T4). There, it synapses with second order neurons whose pre-ganglionic cell bodies give rise to axons, which exit the white rami communicantes of the spinal cord via the anterior horn

and enter the sympathetic chain in the neck, synapsing in the superior cervical ganglion. Here, cell bodies of third order neurons give rise to post-ganglionic axons that course to the eye with internal carotid artery via the cavernous sinus. Fibres from these axons form the long and short posterior ciliary nerves. These sympathetic nerve fibres innervate the dilator of the iris. Post-ganglionic sympathetic fibres also innervate the muscle of Müller, responsible for the initiation of eyelid retraction during eyelid opening. Post-ganglionic sympathetic fibres responsible for facial sweating follow the external carotid artery to the sweat glands of the face. Interruption at any location along this pathway (pre-ganglionic or post-ganglionic) will induce an ipsilateral Horner's syndrome (Chan et al., 2001).

The most common aetiology of Horner's syndrome is neoplasia (35% - 60%) followed by trauma (4% - 13%) (Bell et al., 2001). The underlying mechanism in traumatic carotid artery dissection is usually blunt trauma and includes road traffic accidents, trampolining, chiropractic manipulation of the neck, treadmill running, skiing and birth injuries (Bell et al., 2001; Demetriades et al., 2009; Fletcher et al., 1995; Macdonald and McKillop, 2006). Cervical rotation and compression of the arterial wall is postulated to cause a small intimal tear that leads to intra-medial haemorrhage compromising the vascular supply to the superior cervical ganglion. (Fletcher et al., 1995) The signs of a Horner's syndrome may present up to 5 days after the injury. In 50% of dissections, there will be no signs of neck trauma (Fletcher et al., 1995).

Cervicocephalic dissections were once considered uncommon for neurological/neuro-ophthalmic disorders such as stroke or transient ischaemic attacks (TIA), and Horner's syndrome (Bell et al., 2001). However, with the advent of MRA and computerised tomographic angiography (CTA), carotid and vertebral dissections are being recognised more often. Stringaris et al reported 12 cases of carotid dissection in which MRI with MRA was superior to conventional angiography (Stringaris et al., 1996). Computerised tomographic angiograms have a comparable efficacy to MRA. Vertinsky et al. (2008) conducted a study on 18 patients with 25 dissections and showed that CT/CTA identified more intimal flaps, pseudo-aneurysms and significant stenosis than MRI/MRA. Ultrasound techniques are also a valuable modality. Intimal tears on Duplex scan are pathognomonic and have 79% sensitivity for detection of carotid dissections.

Conclusion

Treatment of Horner's syndrome depends on the cause. Early diagnosis and expedient referral is the key. Internal carotid artery dissection (ICAD) is a potentially life-threatening condition and carries a substantial risk of disabling stroke (Dziewas et al., 2003). Carotid dissection is under-recognised as a cause of Horner's syndrome and can be missed. (Chan et al, 2001) We present a rare case report of ICAD following blunt trauma in the neck in an amateur snowboarder. This is the second reported such case (Mukhopadhyay and Iorwerth, 2010). Our case highlights the importance of diagnosing ICAD because anti-

coagulation can prevent carotid thrombosis and embolism (Chan et al, 2001). The treatment advocated for dissection is anticoagulation for 3-6 months (McCorry and Bamford, 2004).

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Key points

- Blunt injury to the neck can result in Horner's syndrome.
- Horner's syndrome should alert clinicians to the possibility of a silent ICAD.
- MRI and MRA of the head and neck constitute the imaging modality of choice to look for ICAD.
- The treatment of choice for ICAD is anticoagulation for 3-6 months.

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