CASE REPORT

Carotid artery dissection after treadmill running

D J M Macdonald, E C A McKillop

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The case is presented of carotid artery dissection causing Horner's syndrome in a 25 year old woman after treadmill running. She presented with characteristic eye signs and mild pain. Early diagnosis and treatment was associated with no long term, serious complications. It is now recognised that carotid artery dissection is more common than previously thought. Better imaging of the carotid artery has facilitated the diagnosis, and it is now essential to rule out this potentially fatal condition in patients presenting with post-ganglionic Horner's syndrome. It is accepted that relatively trivial trauma can cause a carotid artery dissection, and many sporting activities may place young, fit, healthy people at risk.

A 25 year old, previously healthy, woman developed Horner's syndrome with neck pain after running 10 km on a treadmill. She did not recall any injury during the run, although she had slightly lost her balance at one point but had not actually fallen. Very mild neck pain on the left side started soon after the run. Several hours later she noticed a constricted left pupil and a drooping eyelid (fig 1). The eye symptoms prompted her to present to an ophthalmologist, who diagnosed idiopathic Horner's syndrome probably secondary to a viral infection. The next day she trained again and noticed that she did not sweat over her left forehead. She remained concerned and re-presented to hospital. Further investigation of her Horner's syndrome was performed to exclude sinister causes. A chest radiograph and Doppler ultrasound of her left common and internal carotid arteries were normal, but a magnetic resonance angiogram (MRA) revealed a dissection of her extracranial internal carotid artery. Transcranial Doppler ultrasound examination of the cerebral circulation was performed, which showed no evidence of microemboli. She was therefore treated conservatively with aspirin for anticoagulation. Follow up MRA six months later showed complete resolution of the dissection with a normal internal carotid artery. The Horner's syndrome remained. A full systemic examination and investigation failed to find any underlying condition that might have predisposed her to carotid artery dissection.

DISCUSSION

Horner's syndrome is named after the physician who first described the signs in 1869.¹ It includes ptosis of the upper and lower eyelids with narrowing of the palpebral aperture and ipsilateral miosis (constricted pupil) and can be associated with ipsilateral anhidrosis (loss of sweating) and apparent enophthalmos (retracted eye).¹ The clinical signs depend on the location of the lesion within the sympathetic pathway. The sympathetic chain is anatomically divided into central, preganglionic, and postganglionic segments.² The affected part can be determined clinically.² Traditionally it has been taught that the causes of postganglionic Horner's syndrome are relatively benign.² In recent years, however, it

Figure 1 Left Horner's syndrome with ptosis and miosis. Reproduced with permission from the patient.

has been recognised that a leading cause is internal carotid artery dissection (ICAD) due to the involvement of the pericarotid sympathetic plexus which is stretched or damaged by inflammation following the dissection.³ It is now clear that ICAD has been under-diagnosed as a cause of Horner's syndrome, mainly because of the difficulty in accurately imaging the carotid arteries.^{2,4} Imaging of the carotid arteries with percutaneous angiography is invasive and expensive, but the advent of MRA has provided a far more desirable method.² This is now considered the "gold standard" for diagnosis of ICAD.² It is suspected that unrecognised ICAD has been the cause of many cases of Horner's syndrome previously labelled as idiopathic.²

ICAD can occur spontaneously or secondary to trauma.⁵ Many classed as spontaneous, however, may be due to relatively minor neck trauma or physical effort.⁵ Documented cases include ICAD secondary to coughing, vomiting, body building, and hair washing.⁵ The two main mechanisms of injury are hyperextension and rotation or full flexion of the neck.⁵ It is important to recognise that insignificant trauma may cause ICAD because it is a recognised cause of ischaemic stroke in young adults.³ A retrospective review of 200 carotid dissections³ showed the mean age to be 45 (range 16–74). Ischaemic events occurred

What is already known on this topic

- Internal carotid artery dissection can be caused by mild trauma to the neck and can present with Horner's syndrome

What this study adds

- Trivial trauma in young, fit, healthy subjects can cause internal carotid artery dissection, and sporting activities may place participants at risk
- This diagnosis should be considered in subjects presenting with Horner's syndrome

Abbreviations: ICAD, internal carotid artery dissection; MRA, magnetic resonance angiogram

in 145 cases, resulting in 116 strokes (three deaths), 22 transient ischaemic attacks, five retinal infarcts, and two cases of amaurosis fugax.

A key therapeutic issue is the latency period between the onset of dissection and the ischaemic event. About 80% of ischaemic events arise within the first seven days, but latency periods of up to five months have been recorded.⁶ The recommended treatment, because of this, is anticoagulation with systemic heparin and then warfarin or antiplatelet treatment for six months.⁷

This case highlights that dissection of the internal carotid artery can occur in young, fit, healthy subjects following relatively trivial trauma. Many sporting activities may therefore place participants at risk. In this patient, pain was not a prominent feature, as might be expected, therefore ICAD must be considered in the differential diagnosis of all patients presenting with Horner's syndrome. Prompt diagnosis of ICAD is essential to avoid associated morbidity and mortality. Horner's syndrome therefore merits urgent hospital referral for full investigation and treatment.

Authors' affiliations

D J M Macdonald, Orthopaedics Department, Western Infirmary, Glasgow, Scotland, UK

E C A McKillop, Gartnavel General Hospital, Glasgow

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Figure 1 is reproduced with the patient's permission.

Correspondence to: Mr Macdonald, Orthopaedics Department, Western Infirmary, Glasgow G3 8SB, Scotland, UK; djmmacd@hotmail.com

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COMMENTARY

The authors have done an excellent job at describing a rare but very important condition which may present to sports medicine physicians. For clinicians who may deal predominantly with young, otherwise healthy patients, this case report is an excellent reminder about carotid artery dissection. This condition is often overlooked and can be missed on initial presentation, as it was in this case. Physicians need to have a high index of suspicion when dealing with a suspected case of carotid artery dissection, as delay in diagnosis can result in considerable morbidity and mortality for the patient.

J S Delaney

McGill Sport Medicine Clinic, 475 Pine Ave W, Montreal, Quebec, Canada, QC H2W 1S4; j.delaney@mcgill.ca