Vertebral artery dissection presenting as isolated vertigo

N J Rane, D McAuley

Vertigo is not an uncommon presentation to the emergency department. It is most commonly caused by benign peripheral processes, such as inner ear or vestibular nerve dysfunction, but can be due to central brain lesions. In this report, we present a central cause of isolated vertigo: brainstem infarct secondary to vertebral artery dissection (VAD). VAD is increasingly being recognised as an important cause of stroke in young people. We discuss the important features that need to be elicited to distinguish central from peripheral disease and the relevance of VAD.

A 24-year-old female student presented to our emergency department with a 3 h history of sudden onset vertigo after singing. She denied all other neurological symptoms including headache, tinnitus, and deafness. She had no relevant past medical history or risk factors for cerebrovascular disease. Examination was significant for bilateral vertical nystagmus and there was a suspicion of left sided facial weakness.

After a normal computed tomographic (CT) scan she was referred to the neurology team for magnetic resonance imaging with angiography (MRA). This showed right-sided thalamic ischaemia and evidence of right vertebral artery dissection (VAD) with mural haematoma (fig 1). On closer questioning the patient revealed she had right-sided neck pain since cycling 5 days previously. The patient was treated with an anticoagulant and her vertigo resolved over 3 days. No evidence of thrombophilia was found. She was presumed to have had a spontaneous VAD with associated thalamic infarct.

Figure 1 Magnetic resonance angiography (axial, T1 weighted) showing dissection of the right vertebral artery.

DISCUSSION

Vertigo is the illusion of rotation caused by asymmetry in the neural activity between left and right vestibular nuclei; it is always temporary and always made worse by head movements. Vertigo is not an uncommon presentation to the emergency department but can be difficult to distinguish from other dizziness symptom complexes. Of primary importance in the emergency department is to distinguish between the common peripheral causes, such as vestibular neuritis and benign positional paroxysmal vertigo, and the rarer but more serious central causes such as cerebellar or brainstem infarction. A single, severe attack of vertigo is most likely due to acute vestibular neuritis or a brainstem infarct.

Vestibular neuritis ("neuronitis", "labyrinthitis") leads to nausea, unidirectional horizontal torsion nystagmus, often suppressed by visual fixation, and a positive (abnormal) head impulse test. Also, the patient can probably stand with their eyes open but becomes very unsteady when the eyes are closed. Features that suggest a central cause include lack of inner ear symptoms (tinnitus, hearing loss or nausea), non-fatigable nystagmus that may be bilateral or may be vertical and will not be suppressed by visual fixation, and any concomitant brainstem or cerebellar signs. A patient with cerebellar infarction usually cannot stand steady even with their eyes open. The head impulse test (testing the integrity of the vestibulo-ocular reflex) involves turning the patient's head quickly 15° and looking for their ability to maintain fixation on a distant object; a normal test makes vestibular neuritis very unlikely and should raise suspicion of a cerebellar or brainstem infarct.

VAD is increasingly being implicated as a cause of ischaemic stroke, especially in young people. It may be involved in up to 40% of all posterior

fossa infarction. There is a well-known association with neck manipulation and sporting activities.¹ Its overall incidence has been placed at 1–1.5 per 100 000 per year.² The diagnosis of VAD can be made using non-invasive MRA, although angiography is still used in equivocal cases. Missing a diagnosis of VAD could have dire consequences, with possible sequelae of intracranial dissection leading to extensive posterior infarction and subarachnoid haemorrhage.

Most authorities advocate treatment of VAD with anticoagulation for 6 months, when there is evidence of vessel recanalisation,³ although this remains controversial. There is currently no randomised control trial on efficacy or safety of antithrombotics. A recent systematic review of carotid artery dissection found no significant difference in the odds of morbidity and mortality between treatment with antiplatelets and anticoagulants. Few intracranial haemorrhages (0.5%) were reported on anticoagulants, none with antiplatelets.⁴

This case highlights the need for an appropriate neurological examination, including some specific simple tests and some knowledge of the different disease entities when dealing with acute vertigo. Also, in any patient with a suspected posterior fossa cerebrovascular accident, a history of neck injury or manipulation is suspicious for a VAD, warranting investigation with MRA.

Emerg Med J 2007;**24**:732.

doi: 10.1136/emj.2007.046946

Authors' affiliations

N J Rane, D McAuley, Cambridge University Hospitals, Cambridge, UK

Correspondence to: Dr Neil J Rane, Christ's College Cambridge, Cambridge, CB2 3BU, UK; nr230@cam.ac.uk

Accepted 29 April 2007

Competing interests: None declared.

Informed consent was obtained for publication.

REFERENCES

- Chen WL, Chern CH, Wu YL, et al. Vertebral artery dissection and cerebellar infarction following chiropractic manipulation. *Emerg Med J* 2006;**23**:e1.
- Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. *N Engl J Med* 2001;**344**:898–906.
- Schievink WI. The treatment of spontaneous carotid and vertebral artery dissections. *Curr Opin Cardiol* 2000;**15**:316–21.
- Lyrer P, Engelter S. Antithrombotic drugs for carotid artery dissection. *Stroke* 2004;**35**:613–4.