Caveats in the management and diagnosis of cerebellar infarct and vertebral artery dissection

N Ramphul, U Geary

Early MRI, available 24 h a day and at weekends, will make a timely diagnosis in cases of cerebellar infarct and a normal initial CT scan does not exclude it. In many emergency departments MRI is not as routinely available out of hours as CT. It is important to appreciate the varied symptomatology and signs of cerebellar infarcts in order to avoid misdiagnosis or delayed diagnosis and to remember that, apart from requiring treatment with anticoagulation for the cerebellar infarct itself, the complications of cerebellar infarct may require surgical intervention. Vertebral artery dissection as a cause of cerebellar infarct may require anticoagulation or endovascular therapy.

Mortality from cerebellar infarcts has been reported to be as high as 11%. Surgical intervention may be required in the form of external ventricular drainage with or without decompressive craniotomy and removal of infarcted cerebellum. The initial CT scan of the brain in the acute phase may be reported as normal, and a wrong diagnosis such as gastroenteritis or labyrinthitis may be attributed to the cerebellar infarct owing to the varied symptomatology. We emphasise the importance of recognising the possible need for surgical intervention for the infarct, as well as the possibility of medical management of the vertebral artery dissection in the form of anticoagulation or therapeutic endovascular methods of treatment. We present the case of a patient in whom the diagnosis of acute cerebellar infarct as a likely consequence of vertebral artery dissection was only made at MRI following a normal CT scan of the brain.

CASE REPORT
A 58-year-old man developed sudden onset of vertigo while going downstairs about 20 min after waking up in the morning. There was no history of head or neck trauma. He then had gradual onset of an occipital headache which radiated down his neck and he began to vomit. The patient was on treatment for hypertension (25 mg atenolol once daily), had a history of hypercholesterolaemia, was an ex-smoker for 4 years and had no significant family history. On examination, the patient’s Glasgow Coma Score was 15/15 and the initial blood pressure was 182/121 mm Hg. Pulse rate was 115 bpm and aural temperature was 34.6°C. The pupils were small and reactive and there was nystagmus, maximal on looking to the left. The cranial nerves were intact. Power was decreased at 4/5 in the left upper limb. Plantars were downgoing on the right and equivocal on the left. An ECG showed normal sinus rhythm and the chest radiograph was normal, as were blood tests, except for mild hypokalaemia (3.2 mmol/l) secondary to vomiting. The differential diagnosis was subarachnoid haemorrhage or brainstem infarct. A CT scan of the brain was reported as normal and an urgent MRI scan of the brain was requested. Intramuscular chlorpromazine and intravenous ondansetron were used to control the emesis and intravenous morphine to control the headache. The patient was also administered 1 litre of normal saline intravenously. The MRI brain scan demonstrated a fresh left posterior inferior cerebellar artery distribution infarct (fig 1). There was an absence of flow within the left vertebral artery, as seen on magnetic resonance angiography, which was suggestive of thrombosis complicating an acute dissection. The patient was admitted under the care of the medical team.

Following a neurosurgical and neurological consultation, a decision was made not to commence heparin anticoagulation and antiplatelet therapy with aspirin was prescribed instead. The patient underwent a transoesophageal echocardiogram which demonstrated mild atheroma in the descending aorta but was otherwise normal. The patient also had a normal thrombophilia screen. The patient’s symptoms of headache and vertigo were controlled with oral tramadol, paracetamol, ondansetron and zopiclone. He was discharged 9 days after admission and was asymptomatic at review in the outpatient department at 1 and 2 months.

DISCUSSION
Cerebellar infarcts make up just over 2% of all strokes. They can occur at any age and have even been described in neonates. They can lead to oedema of the cerebellum with consequent obstructive hydrocephalus and brainstem compression resulting in coma, respiratory arrest and death. This may take days to develop. One cause is vertebral artery dissection as a source of thrombotic embolus. The signs and symptoms of cerebellar infarct are of sudden onset and include vertigo, nausea, vomiting, headache, nystagmus, deafness, tinnitus and postural instability. Sometimes there are no cerebellar signs. Vertebral artery dissection can cause neck and occipital pain, as well as headache. Cerebellar infarcts can be misdiagnosed based on an initial normal CT scan of the brain. MRI is the preferred investigative tool for stroke, particularly to detect acute infarct. Savitz et al analysed 15 cases of misdiagnosed cerebellar infarctions and found that almost all of the cases had a normal CT brain scan that led to incorrect diagnoses. The overall mortality of their cohort was 40%, with 50% of the survivors having disabling deficits. Delay in diagnosis or erroneous diagnosis may lead to morbidity and mortality. Vertebral artery dissection can occur spontaneously or after even minor or trivial trauma such as coughing. The intima or media of the artery is disrupted leading to haematoma formation in the subintimal, medial or subadventitial layers, resulting in disrupted blood flow followed by luminal thrombosis and distal embolism. Diagnosis can be made by catheter angiography or magnetic resonance angiography.
Aetiological factors have been postulated such as fibromuscular dysplasia, giant cell arteritis, atherosclerosis, Marfan’s disease and cystic medial necrosis. Stroke following vertebral artery dissection can be delayed, even by up to 2 months. The onset of dissection is usually accompanied by acute onset of neck or occipital pain and headache. Anticoagulation may prevent progression of symptoms. Dissection should be diagnosed as early as possible as recanalisation can occur within a few days. Dissection may resolve spontaneously. Anticoagulation usually involves heparin and, even though there is a dearth of controlled studies that support this form of management, there are not—to the best of our knowledge—any cases in the literature reporting adverse effects secondary to anticoagulation. However, adverse reactions have been reported following failure to anticoagulate. If anticoagulation is contraindicated or medical treatment is not successful, therapeutic endovascular treatment may be considered on a case by case basis.

Department of Emergency Medicine, St James’s Hospital, Dublin, Republic of Ireland

Correspondence to: Dr N Ramphul, Department of Emergency Medicine, St James’s Hospital, Dublin, Republic of Ireland; npramphul@eircom.net

Accepted: 14 December 2007

Competing interests: None.

REFERENCES

Retinal detachment diagnosed by magnetic resonance imaging

A 42-year-old nurse noted a sudden loss of vision in her right eye associated with periorcular numbness. Suspecting multiple sclerosis she consulted a neurologist who documented counting fingers vision with a normal examination. A magnetic resonance imaging (MRI) scan performed the same day clearly demonstrated a macular off-retinal detachment (fig 1), which was subsequently repaired with a scleral buckle.

This unusual case, in which a retinal detachment was diagnosed with an MRI scan, illustrates a valuable point. A hand-held ophthalmoscope, with its high magnification, small field of view and lack of stereopsis, does not permit sufficient retinal views to rule out ocular pathology. Shallow retinal detachments, macular oedema, central serous retinopathy and macular degeneration, for example, are difficult to identify with a direct ophthalmoscope and MRI. We recommend that anyone presenting with loss of vision without an obvious cause should be referred to an ophthalmologist.

S Harsum, C Clark, P Fison
Department of Ophthalmology, Epsom and St Helier NHS Trust, Sutton, UK

Figure 1 A T1 weighted magnetic resonance imaging scan showing a right intraocular temporal elevation, with its base at the optic nerve, indicating a retinal detachment.

Correspondence to: Mr S Harsum, Department of Ophthalmology, Epsom and St Helier NHS Trust, Cotswold Road, Sutton SM2 5NF, UK; harsum@doctors.org.uk

Competing interests: None.

Patient consent: Obtained.