NEUROLOGICAL PICTURE

Vertebral artery dissection presenting as cerebellar infarction

(A) Cerebral CT showing left cerebellar hemispheric low density with distortion of the fourth ventricle. There is moderate dilatation of the third (small arrow) and lateral ventricles (large arrows). (B) Axial cerebral T2 weighted MRI showing an area of high intensity involving the left cerebellar hemisphere with moderate swelling. The abnormal area extends to the midline and involves both the cortex and white matter. The fourth ventricle is distorted. (C) Parasagittal cerebral T1 weighted MRI showing an area of low intensity involving the inferoposterior aspect of the left cerebellar hemisphere. (D) Cervical T1 weighted fat saturated MRI showing an absence of the expected flow void in the left vertebral artery (large arrow) and high signal apparently in its walls. Normal flow void in right vertebral artery (small arrow).

A seven year old boy presented with a 4 day history of frontal headache, vomiting, dizziness, and tiredness. He had sustained a minor head injury 6 days before admission by falling from his bicycle. This was not associated with loss of consciousness and he initially made a full recovery. On examination, he was drowsy with no focal neurological signs and no papilloedema.

Cerebral CT showed a low density region and swelling of the left cerebellar hemisphere with distortion of the fourth ventricle and hydrocephalus (figure A). Cerebral MRI (figure B and C) showed an area of high signal on T2 weighted images and low signal on T1 weighted images in the inferoposterior aspect of the left cerebellar hemisphere. The lesion involved both cortex and white matter, extended to the midline and did not enhance, the appearances being typical of an acute left posterior inferior cerebellar artery infarct. T1 weighted fat saturated MRI of his neck was performed and demonstrated an absence of the expected flow void in the left vertebral artery up to the level of C1 suggesting occlusion (figure D). In addition, there was extensive high signal in the walls of the middle and lower vertebral artery, typical of dissection. Flow was detected in the intradural portion of the left vertebral artery and the right vertebral artery appeared normal. Further questioning of his mother disclosed that he had had neck pain since his bicycle injury.

The patient was managed conservatively with intravenous fluids, paracetamol, and dexamethasone. Anticoagulation was not commenced due to the risk of causing haemorrhage into the infarct. Full blood count, urea and electrolytes, liver function tests, coagulation screen, B₁₂, folate, C reactive protein, and complement C3 and C4 concentrations were normal. The erythrocyte sedimentation rate was 19. Antinuclear antibody concentrations were negative and no cause for the vertebral dissection was established other than the history of minor trauma. He made a good recovery with no neurological deficit and was discharged home on aspirin after seven days.

Cerebral or cerebellar infarction is a rare event in children but in previously well children dissection is a common cause and should be actively looked for. Magnetic resonance angiography of the neck arteries may show vascular occlusion or narrowing. T1 weighted fat saturated MRI, in which fat signal is nulled, may show acute haematoma in the walls of the dissected vessel as a crescent of high signal, this appearance probably obviating the need for conventional angiography in the proper clinical setting.

PETER J A HUTCHINSON
JOHN D PICKARD

Department of Neurosurgery

J NICHOLAS P HIGGINS

*Department of Radiology, University of Cambridge,
Addenbrooke's Hospital, Cambridge, UK*

Correspondence to: Mr Peter Hutchinson, Department of Neurosurgery, University of Cambridge, Box 167, Addenbrooke's Hospital, Cambridge CB2 2QQ, UK.