

Letters

Spontaneous dissection of intracranial vertebral artery: clinical recovery with conservative treatment

Sir: Spontaneous dissection of cerebral arteries is an uncommon, though increasingly recognised cause of cerebrovascular disease. The dissection of the intracranial vertebral-basilar system usually causes death, except in a few recent surgically treated cases.

We describe a case of non-traumatic dissection of the intracranial vertebral artery with a double lumen arteriographic sign. Cervical immobilisation, anti-platelet therapy and absolute rest led to complete recovery.

A 34-year-old man with no past history of illness except mild hypertension and gastric dyspepsia, had suffered a sudden severe neck pain, headache and dizziness of two hours' duration which began during *coitus interruptus*, and was admitted to our hospital. Neurological examination revealed a conscious, alert patient with dysarthric speech, dysphagia, diplopia, hiccups and numbness and weakness in the left limbs. Meningeal signs were absent. Pupils were reactive. Fundi oculi were normal. Right sixth nerve cranial palsy and nystagmus on bilateral horizontal gaze were observed. There was left hemiparesis, left hemi-cerebellar dysfunction and sensory loss in the right limbs. Deep tendon reflexes were normal. Plantar response was flexor on the right and equivocal on the left. CT scan and CSF studies were normal. Two days later, the patient was improving but weakness of the left limbs and sensory loss in the right limbs persisted. Left vertebral arteriogram on the fourth day demonstrated a double lumen from C-1 to the beginning of the basilar artery (fig 1A and B).

Absolute rest, cervical immobilisation with a collar and therapy with dipyridamol (400 mg/day) were initiated. Aspirin was avoided owing to the patient's past history of gastric dyspepsia. Two days later, owing to severe headaches attributed to dipyridamol intolerance, the patient was given ticlopidine (500 mg/day) instead. Biological studies, luetic serology, EKG and a new CT scan were normal. The patient was asymptomatic by the seventh day. A new arteriographic study in the fourth week demonstrated complete disappearance of the left vertebral artery lesions (fig 2). Treatment was continued for ten days more and progressive mobilisation was initiated. Anti-

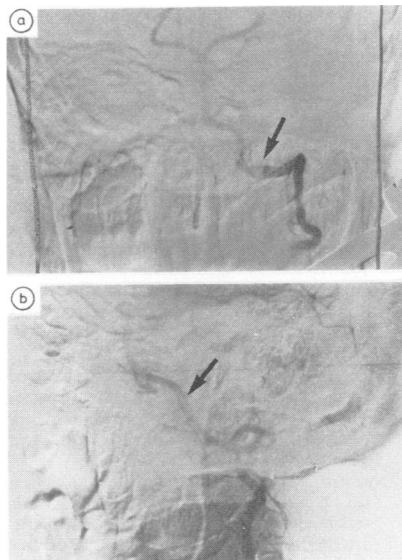


Fig 1 Left vertebral angiogram: the arrow indicates the double lumen pattern inside the vessel. Antero-posterior (A) and lateral (B) views.

platelet therapy was maintained for six months. No further complications have occurred in the first year of follow-up.

Intracranial artery dissection has been associated with fibromuscular dysplasia, cystic medial necrosis, syphilis, atherosclerosis, Marfan's syndrome, hypertension, migraine and head injury. Other factors such as chiropractic manipulation, sneezing, violent coughing and sporting activities can trigger the dissection.^{1,3} However, no association with sexual intercourse has yet been reported.

Of the 39 cases of dissection of the intracranial vertebral-basilar system described in the literature, only five, who were treated surgically, have survived.⁴⁻⁷ Accurate diagnosis is almost never made preoperatively or before death. To our knowledge, our case is the first of its kind to be reported with clinical recovery and subsequent angiography showing re-establishment of a normal lumen with conservative treatment. Arteriographic normalisation has previously been described in carotid artery dissections.⁸ Unfortunately, the demonstration of a double lumen that has been considered the only angiographically diagnostic sign of artery dissection is uncommon. This diagnostic angiographic feature has only been found in two^{10,11} of 39 described cases.^{4-7,9-38}

Anticoagulation and anti-platelet therapies have been employed in cerebral arte-

rial dissection to reduce the chance of deterioration and embolic complications.³ However, the successful evolution in our case should also be attributed to bed rest and cervical immobilisation, which help to prevent continuous minor trauma that might impede the healing of the dissection plane.

We conclude that conservative treatment with the use of anti-platelet drugs, rest and cervical immobilisation may be effective in cases of spontaneous dissection of the intracranial vertebral artery. Nevertheless, further experience of surgical and conservative treatment is necessary for future guidance on the treatment of choice in cases of dissection of the intracranial vertebral-basilar system.

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Fig 2 Cervico-cerebral angiograms: complete recanalisation after conservative management.

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Postinfectious meningoencephalitis complicating *Mycoplasma pneumoniae* in a child

Sir: *Mycoplasma pneumoniae* is the primary non-bacterial pathogen causing lower respiratory tract infections among school-age children.^{1,2} Of the non-respiratory manifestations, neurological disorders are the most common. They include Guillain-Barre's syndrome, meningitis, transverse myelitis and encephalitis.^{3,4} The prognosis is generally favourable which may be illustrated by the following case report.

A 12-year-old, right-handed boy was admitted to hospital because of right-sided hemiparesis. Nine days prior to admission he had stayed home from school with a headache. During the next few days he had suffered mild upper respiratory symptoms with fever, his temperature rising to 38.5°C and he seemed confused at night. The boy was already starting to recover when he noticed a weakness in his right leg which, by the next day, had evolved into a right-sided hemiparesis, resulting in admission. His temperature was 38°C, pulse rate 104/min, blood pressure 110/70 mm Hg. On neurological examination, the boy was bradyphrenic but alert. He had a mild expressive aphasia, and right-sided hemiparesis. Complete blood count, ESR, chemistry profile and urine analysis were normal. CSF contained 102 WBCs/mm³, protein 86 mg/l and glucose 3.8 mmol/l. The CSF cytogram showed polymorphonuclear leucocytes, monocytes, many lymphocytes, plasma cells, and macrophages. A chest radiogram demonstrated increased bronchial markings. A CT scan revealed white matter hypodensity in the left parietal region. An EEG showed diffuse marked slowing of background activity on the left side and no abnormalities on the right side.

During the next three days the aphasia increased, right-sided hemianopia became apparent and the hemiparesis developed into a hemiplegia. Four days after admission the boy started to vomit and became comatose. His left pupil was dilated but still responded to light. Bilateral decorticate rigidity developed suddenly, with equally wide and fixed oval pupils. A repeat CT (fig a) demonstrated marked finger shaped white matter hypodensity in the left hemisphere. An Ommaya device was inserted. Treatment was then continued with dexamethasone, hyperventilation under etomidate, repeated mannitol infusions and external CSF drainage with monitoring of CSF pressure. Serological data indicated active *Mycoplasma pneumoniae* infection: complement fixation 1:64, immuno-