Basilar artery dissection

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SUMMARY Dissection of the basilar artery caused sudden coma and death in a 40-year-old man. Atypical clinical features were explained at necropsy. A ventral dissection of the artery within its outer layers resulted in destruction of the pontine tegmentum with sparing of the basis pontis. An unsuspected defect in the internal elastic lamina in the left internal carotid artery was also found suggesting a more generalised disorder of arterial walls. Basilar artery dissection should be considered in the diagnosis of coma in young people.

Basilar artery occlusion is a common form of stroke that presents clinically as combinations of quadriplegia, upper cranial nerve signs and coma.^{1,2} Destruction of the basis pontis is usually the major pathological finding, with variable involvement of the tegmentum of the mid-brain and pons.¹ Dissection of the basilar artery is a rare cause of this clinicopathologic picture.³

An initially baffling case of sudden coma was found to be due to dissection of an ectatic basilar artery. The atypical clinical features were explained at necropsy. Massive infarction of the brain stem tegmentum was found with sparing of the basis pontis, the reverse of the usual situation.

Case report

A 40-year-old male was found slumped behind the wheel of his parked car 2½ hours after leaving his workplace for lunch. He had been in good health apart from borderline hypertension detected 4 weeks previously. There was no history of headache or of transient neurological disturbance. On examination he was deeply unconscious. He was afebrile, pulse rate was 60/min and blood pressure was 140/70 mm Hg. Shallow spontaneous respirations were present at a rate of 6/min. The optic fundi were normal and the pupils were fixed in mid-position. There was a slight left divergent squint and oculocephalic reflexes were absent. Ice-water caloric irrigation caused slight ipsilateral tonic deviation of the eyes on stimulating the right ear but there was no response after stimulating the left ear. Corneal reflexes were symmetrically diminished and there was bilateral facial weakness. The gag reflex was absent but tracheal stimulation provoked a cough. There was no neck

stiffness. The limbs were flaccid with normal deep tendon reflexes and equivocal plantar responses. All four limbs showed appropriate flexor withdrawal responses to deep pain but intense supraorbital pressure did not elicit any response. Investigations revealed that plasma biochemistry, blood sugar, full blood count, drug screen, and radiographs of skull, cervical vertebrae, and chest were normal. Arterial blood gases revealed a mild respiratory acidosis. CT scan was interpreted as normal but showed a small enhancing opacity in the suprasellar cistern (fig 1). Lumbar puncture yielded clear fluid under normal pressure containing 180 red cells and 3 lymphocytes per cmm with normal protein and sugar; culture and viral isolation were negative. Nasotracheal intubation was performed and he was ventilated mechanically. Transient increases in blood pressure (up to 200 mm Hg systolic) were treated with intravenous hydralazine. Nutrition was maintained by intravenous fluids and nasogastric feeding. The temperature rose to 39° over 12 hours, in the absence of evidence of infection. Repeated detailed neurologic examination revealed no significant change in his clinical state except that the caloric responses were totally absent by the 6th hospital day. On the 10th day vertebral angiography revealed gross abnormalities of the basilar artery (figs 2 and 3). This was interpreted as basilar ectasia with atheroma, but in retrospect clear evidence of dissection was present (fig 3). A left carotid angiogram was normal. The left posterior cerebral and left superior cerebellar arteries did not fill on either the left carotid or vertebral studies. The patient died on the 15th hospital day.

Post mortem examination

General examination was normal apart from bronchopneumonia. The heart weighed 385 g and showed no evidence of left ventricular hypertrophy. There was minimal coronary atherosclerosis. The carotid arteries, aorta and its major branches were macroscopically normal. The brain weighed 1480 g. A large haemorrhagic infarct was found in the left cerebellar hemisphere with small areas of spotty infarction in the right cerebellar hemisphere. In the cerebrum there was bilateral infarction of the posterior part of the thalamus as well as a large occipital infarct on the left.

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Fig 1 CT scan showing an enhancing lesion in the region of the supra-sella cistern slightly displaced to the right.

Fig 2 Vertebral angiogram (Towne's view) showing slight ectasia of the vertebro-basilar system. The left posterior cerebral artery does not fill.

Fig 3 Vertebral angiogram (lateral view) showing a double lumen of the basilar artery. The false lumen is ventral, lying on the clivus and extending into the supra-sellar area.

In the brain stem there was extensive infarction of the pontine and mid-brain tegmentum extending up to the subthalamus. The basis pontis was spared (fig 4). The basilar artery and right vertebral artery showed scattered atheroma. The basilar artery was compressed 1 cm above its origin by blood in the adventitia. Distal to this point the artery was contracted and there was complete occlusion of the superior cerebellar and posterior cerebral arteries on

the left. The internal carotid arteries and their branches were normal. Sections of the basilar artery showed a dissection between the adventitia and the outer media, and the origin of the dissection was demonstrated (fig 5). Sections of the left internal carotid artery showed a defect in the internal elastic lamina (fig 6); similar defects could not be found in sections of the vertebral, coronary and other arteries.

Fig 4 Section of the upper pons. The basis pontis is almost entirely preserved while there is extensive destruction of the pontine tegmentum. Luxol fast blue $\times 2.6$.

Fig 6 Left internal carotid artery. An unsuspected defect in the internal elastic lamina is shown. Aldehyde fuchsin Gomori $\times 30$.

Fig 5 Basilar artery. Dissection between media and adventitia. Communication between the true and false lumen is clearly shown. Aldehyde fuchsin Gomori $\times 30$.

Discussion

Dissection of the cervico-cerebral arteries is a rare but probably underdiagnosed cause of stroke. Alexander⁴ found approximately 60 cases in a review of the world literature cases to 1977 and Fisher⁵ has added another 21 personal cases. Less than one third of these cases have been basilar artery dissections^{3,4,6-22} and there have been only four cases of this entity published in English over the last 20 years.^{4,12,16}

In this case the clinical presentation was not typical of basilar occlusion. The deep coma with symmetrical cranial nerve abnormalities and unimpressive long tract signs led to an extensive search for causes of a metabolic or toxic encephalopathy. Basilar dissection should have been suspected after the CT scan (fig 1) and confirmed by the angiographic appearance (fig 3). However, the diagnosis was not fully appreciated until necropsy.

Occlusion of the basilar artery usually occurs in patients over 50 years of age.^{1,2} Dissection of the basilar artery affects a younger age group; most cases were aged 20-45 years.⁶⁻²² Both conditions usually cause destruction of the basis pontis with variable involvement of the brainstem tegmentum.^{1,2,18,19} In the present case the basis pontis was spared (fig 4), accounting for the absence of impressive long tract signs. Presumably the dissection occluded the long circumferential branches of the basilar artery supplying the tegmentum whilst the paramedian and short circumferential branches were largely spared.¹

Dissections usually occur in the plane between the internal elastic lamina and the media.^{4,7-11,13,16-19} Rarely, as in this case, the dissection is within the media or adventitia (fig 5).^{12,20} Direct communication between the true and false lumen was demonstrated (fig 5), a finding recorded only once before.²⁰

Various causes of cervico-cerebral arterial dissection have been postulated including congenital medial defects,²⁰ intimal fibrodysplasia,²¹ homocystinuria,¹⁴ trauma,¹⁸ syphilis,³ migraine,⁴ arteritis,¹⁹ and cystic medial necrosis⁶ but usually no cause is demonstrated. The presence in this case of a medial defect in the left carotid artery (fig 6) raises the possibility of multiple congenital arterial defects but no similar defect was found in other arteries.

Basilar artery dissection should be considered in patients, especially young adults, presenting with hind-brain stroke or unexplained coma. CT scan may suggest the diagnosis but angiography is required for confirmation. Whereas carotid dissection⁵ may resolve spontaneously without severe clinical deficits, all reported cases of basilar dissection have been fatal.

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