to the release of vasoactive peptides from trigeminal sensory perivascular fibres.\textsuperscript{18}

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\section*{Occlusion of the basilar artery in a 7 year old boy}

Sir: Occlusion of the basilar artery is uncommon in childhood; a review of the literature disclosed only 29 cases documented in children up to 16 years old;\textsuperscript{1–11} there was a clear male dominance.\textsuperscript{6} The most frequent cause for occlusion of the basilar artery in adults is atherosclerosis,\textsuperscript{12} while in children the reported cases have been associated with congenital malformations,\textsuperscript{2,3} embolism,\textsuperscript{4,5} arterial,\textsuperscript{6} cervical injuries,\textsuperscript{7,11} and idiopathic causes.\textsuperscript{1,4,6,7,10} We describe a 7 year old boy with occlusion of the basilar artery verified by angiography and a locked-in state who had previous craniocervical injury.

A 7 year old boy was admitted to our service because of sudden headache, vomiting and lethargy. Two weeks earlier he had sustained injuries to the head and cervical region, while being violently shaken against a tree trunk by an older child, resulting in malaise and drowsiness lasting a few hours but without loss of consciousness. Ten days before admission he had unsteady gait and cerebellar signs in the right limbs lasting a few minutes, accompanied by headache and vomiting. On admission his temperature was 38°C, blood pressure 120/70 mm Hg and pulse rate 116/minute. He had decerebrate rigidity, oculocephalic deviation toward the left and spastic tetraparesis with bilateral Babinski sign. The pupils were symmetrical with normal reactions. The corneal reflexes were present. He was able to blink voluntarily, reacting to verbal commands by moving the eyes up and down. There was involuntary downward jerking of the eyes with slow upward drift (ocular bobbing), and sucking mouth movements without uttering words and his facial expression at times mimicked crying, either spontaneously or after stimuli. The child was admitted to the intensive care unit. The remainder of the physical examination, cardiologic studies, lumbar puncture and laboratory analyses including clotting tests, antinuclear factor and syphilitis tests all were normal, as were chest films and cranial and spine films. The EEG revealed a slowed basic rhythm of low voltage and occasional bursts of bilateral delta waves in the frontal and occipital areas. A CT scan showed areas of decreased density at the level of the left middle cerebellar peduncle, left lateral half of the pons and left cerebellar hemisphere. Angiography showed normal vertebral arteries and a well-filling supernumerary branch originating in the right subclavian artery. A complete proximal occlusion of the basilar artery was observed (fig). Left carotid arteriography revealed partial and irregular filling of the distal portion of the basilar artery through the left posterior communicating artery.

Treatment was begun with heparin sodium i.v. and because of worsening in ventilation, tracheostomy was performed and a volumetric respirator connected. Three weeks later the patient still opened and closed his eyes, responded to visual stimuli, breathed spontaneously and could perform voluntary proximal movements with the upper limbs, but did not speak or respond to commands and no contact could be established. Four weeks after admission the patient died because of massive haemorrhage as a complication of the tracheostomy. No post-mortem examination could be performed.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{Complete occlusion of the basilar artery at its proximal third (arrow). Left vertebral artery is normal.}
\end{figure}

\section*{References}


Letters

Although occlusion of the basilar artery has been well studied and documented in adult patients, particularly after the work of Kubik and Adams, it is very uncommon in childhood. It is characterised by sudden alterations to the state of consciousness with motor and neuro-ophthalmological anomalies in the majority of cases. In one third of all patients there are prodromal episodes with transient or reversible neurological disturbances. Oclusion of the basilar artery may give rise to the locked-in-syndrome caused by ventral pontine ischaemia, although other sites of origin also have been described. Pontine lesions were confirmed in our patient by ocular bobbing and CT scan images of decreased density at thepons.

The diagnosis of occlusion of the basilar artery must be established through angiography. The examination of the four branches is advisable to rule out congenital vascular anomalies, laminar flow and spasm of the vertebral or basilar arteries. The prognosis for occlusion of the basilar artery generally is poor with a mortality rate reaching 85% in adults and nearly 25% in children, while there may be serious sequelae in survivors. In our patient death was not directly attributable to the occlusion of the basilar artery.

Mild cervical injuries have repeatedly been held responsible for different types of lesions in the vertebral arteries, such as tearing of the intima, thrombosis, aneurysms and dissections, which in turn may be the cause of severe neurological disturbances. In such cases the occlusion of the basilar artery may be produced by the thrombosis extending from the damaged vertebral arteries or through embolisms originating at the site of the thrombosis. The location of the arterial injury is not restricted to the occipitoatlantoaxial level but includes the course through the transverse foramina, with sudden lateral rotating movements either with or without forced extension of the neck being the most frequent cause for this type of lesion.

Our patient had no signs of arteritis, cardiac embolism, congenital malformation or cervical lesions and therefore craniovascular injury appeared to be the most probable cause for the occlusion in spite of the normal angiographic findings for both vertebral arteries.

When occlusion of the basilar artery is suspected in a child, the appropriate angiographic studies should be performed in selected patients to confirm the diagnosis with a complete study to determine its possible aetiological factors. A previous cervical injury must always be looked for.

Multiple plasmacytoma presenting as raised intracranial pressure

Sir: Plasma cell tumours may cause compression of the spinal cord or nerve roots and are sometimes associated with peripheral neuropathy or involvement of the central nervous system. The skull bones are often involved but intracranial complications are rare. We report an adult male with multiple plasmacytoma who presented with raised intracranial pressure.

A 40 year old man presented with severe occipital headache for 10 days and diplopia on looking to the right. He gave a 3 month history of low back pain. Examination revealed bilateral papilloedema, partial right lateral rectus palsy and limited straight leg raising on both sides. A CT scan of the head was normal. Opening pressure at lumbar puncture was over 400 mm CSF; protein, glucose, and gamma globulin content were normal and no cells were seen. The blood urea was elevated at 10 mmol/l with normal electrolytes. Bone alkaline phosphatase, calcium, plasma protein electrophoresis, plasma protein and albumin were normal. The ESR was 10 mm in the first hour, haemoglobin 12-0 g/dl, white cell count was 12 × 10^9/l (normal distribution) with normal platelet count. A 24 hour urine collection revealed normal creatinine clearance with protein loss of 3-2 g.

A radiograph of the sacrum showed a large osteolytic lesion. Small "punched-out" lytic lesions were seen on radiographs of the skull and left clavicle and multiple deposits in sacrum, pelvis, ribs and spine were evident on the bone scan. Two bone marrow aspirates gave normal appearances and bone marrow trephine showed no evidence of metastases or myeloma. Biopsy of the sacrum revealed plasmacytoma. One month after admission, free kappa light chain globulins were found in the urine and accounted for 3-9 g in a 24 hour protein loss of 5-2 g. Soon afterwards, plasma protein electrophoresis revealed kappa chains plus a significant fall in plasma immunoglobulin levels. CSF electrophoresis and plasma viscosity were normal. CT scan of head at this stage revealed no intracranial abnormality; however, there was a large lytic lesion in the left temporal bone.

Despite melphalan, dexamethasone, allopurinol and radiotherapy, he deteriorated over the course of the following year with numerous pathological fractures and infections. He developed severe hypercalcaemia and later succumbed to a chest infection.

The diagnosis of multiple plasmacytoma was based on biopsy and radiological evidence of multiple bone lesions without...
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