LETTERS TO THE EDITOR

Misinterpretation of neuroradiological appearances of an epidermoid cyst

Epidermoid cysts have been described as the most frequent tumourous malformation of the CNS.[1 2] The favourable outcome after total removal[1] make an early correct diagnosis highly necessary. We describe a 48 year old male technician who was healthy until April 1988, but who developed thoracic pain and sciatica-like backache on the right side which disappeared a few weeks later. In July neck stiffness and double vision occurred. He was admitted to our hospital in August 1988.

Nuchal rigidity, paralysis of both abducent nerves, weakness of the right superior oblique muscle and the left facial nerve, as well as papilloedema were found. There was a slight displacement of the palatopharyngeal arch to the left. He was afebrile. Routine laboratory data were normal. In September, episodes of confusion and a left-sided ptosis occurred. There was now plegia of both abducent nerves and a weakness of both recti superiores muscles. The cerebrospinal fluid (CSF) showed 40 cells per cubic millimetre. The protein content was slightly elevated to 52·5 mg%. A differential CSF cell count revealed 3% tumour cells, 60% monocytes, 4% macrophages, 4% granulocytes, 15% large lymphocytes, and 16% small lymphocytes. The tumour cells were three times as large as monocytes; the cytoplasms were broad and basophilic. Immunocytochemistry showed heavy labelling of tumour cell cytoplasms and membranes with the epithelial membrane antigen (EMA) indicating their epithelial origin.

Computerised tomography (CT) and magnetic resonance imaging (MRI) revealed an asymmetrically widened interpeduncular cistern and a cerebellopontine cistern on the left side without contrast media or Gd DPTA enhancement. No Hounsfield numbers were measured. MRI sequences were sagittal SE 700/15 T1 weighted and coronal SE 2500/15/90 T2 weighted. Using these "standard" sequences, no differences in the intensity of CSF and the widened cisternae were indicated. Radiographs of the skull did not show any destruction, nor calcifications. As a result of these findings and the typical localisation, an arachnoid cyst was suspected (fig). Finally, the patient developed an acute phase of hepatitis B. He died on 17 September after repeated cardiac arrests.

A complete necropsy showed multiple acute liver necroses, fibrinous endocarditis, subpleural petechiae, pulmonary oedema and the morphological signs of shock. The neuropathological investigation revealed a typical epidermoid cyst at the base of the brain extending from the interpenduncular fossa to the upper medulla oblongata with a mean diameter of 1 cm. The whole left aspect of the pons and the left cerebellopontine angle were covered by the tumour. Both oculomotor nerves, the left trigeminal, facial and the vestibulocochlear nerves as well as both abducent and glossopharyngeal nerves were covered with pearly masses. The tumour lacked calcifications. There was no bone destruction at the base of the skull. The brainstem was slightly displaced to the right.

We conclude that in cases with cranial nerve lesions and cerebellar signs or even meningeal signs an epidermoid cyst at the base of the brain should be excluded. As our observations show, routine performance of CT and MRI may fail to reveal the tumour.[6 7] In most cases, epidermoid cysts have T1 MRI signals intermediate between brain and CSF whereas arachnoid cysts are identical with CSF.[8] This characteristic MRI feature was not present in our case. However, the correct diagnosis might have been suggested by characteristic Hounsfield numbers on CT. Furthermore, a possible distinction between arachnoid and epidermoid cyst could have been achieved by additional MRI sequences. A multi-echo sequence with a TR of around 3000 and 6–9 echoes ranging from TE 26-TE 156 will nearly always show a difference between epidermoid cysts and CSF at least in some part of the series.[9] Hadley and Patterson[10] recommended a sequence sensitive to T1 changes, depending on field strength that is, at 0,35 Tesla an SE 300–500/15–30 or an inversion recovery 1600/400/40 to distinguish between both lesions. Using suitable sequences at 1,5 Tesla in our case, the epidermoid cyst might have been hyperintense to CSF. A displacement of basal brain structures suggesting a cystic process,[6 7] may be missed in epidermoid cysts of small sizes. CSF examination may indicate aseptic meningitis or epithelial tumour cells after spontaneous rupture[1 3-5] but gives negative results in cases with an intact cyst wall. Full use of modern neuroradiological techniques and CSF investigation is necessary to establish the diagnosis of an epidermoid cyst.

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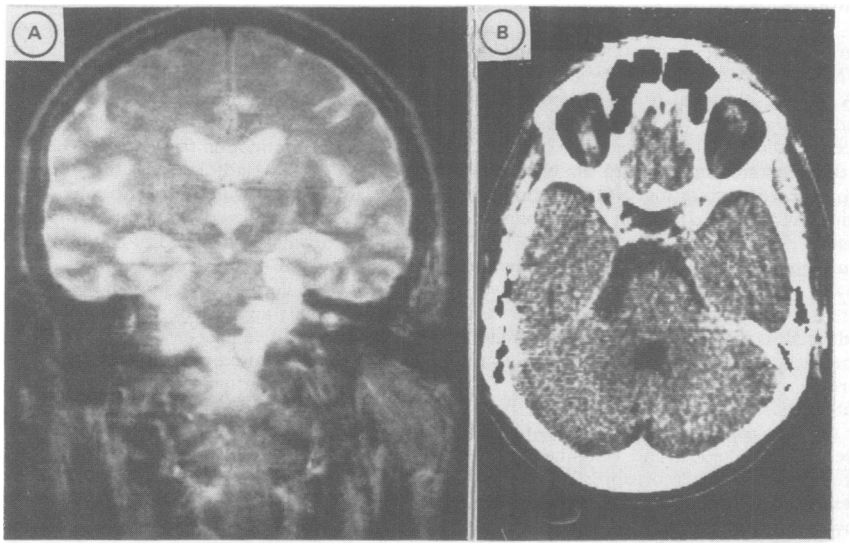

Figure a) MRI (coronal plane, SE 2500/15/90 T2 weighted); b) CT (transverse plane). Neuroradiological findings only disclosed an asymmetrical widened interpeduncular cistern and a cerebellopontine cistern on the left side without contrast media or Gd DPTA enhancement.

Gingival hyperplasia due to sodium valproate

Gingival hyperplasia is a common disorder seen in patients with epilepsy treated with phenytoin.[1] The incidence is approximately 33% in all epileptic patients receiving phenytoin, and higher in institutionalised patients and those with poor oral hygiene.[2] Gingival hyperplasia is a rare side effect of sodium valproate therapy, and only a single case has been reported in a 15 month old child.[3] A patient with epilepsy receiving sodium valproate is reported who developed gingival hyperplasia during therapy.

At the age of 12 years the patient developed myoclonic epilepsy, attacks being frequent in the early morning. She did not receive any anticonvulsant drugs. A year later she developed generalised tonic-clonic seizures. When first seen at the age of 14 years, she was alert, intelligent without any dysmorphic features. General and neurological examination was normal. She was advised to take sodium valporate 600 mg daily. Blood count, liver and renal function tests were normal.