Letters

Conjugate deviation of gaze in hepatic encephalopathy

Sir: Transient ocular motor disturbances such as oculobobbing,1 dysconjugate gaze,2 skew deviation3 and absent horizontal responsiveness to oculovestibular testing4 have been described in patients with hepatic encephalopathy. Sustained conjugate deviation of the eyes, however, is usually considered indicative of a contralateral irritative lesion or an ipsilateral destructive lesion of the cortex or a lesion of the contralateral pontine gaze centre.5 Here we describe a patient with fulminating hepatic failure in whom conjugate deviation of gaze developed two days before death. No structural lesion could be demonstrated on CT scan or at necropsy.

A 52-year-old man with a long history of ethanol abuse and biopsy-proven hepatic cirrhosis was admitted in an obtunded state. On examination he had tense ascites and pedal oedema and heavily jaundiced sclerae. He responded to only simple verbal commands and had bilateral asterixis; neurological assessment was otherwise unremarkable. Laboratory data at that time were: total bilirubin 273μmol/l; serum albumin 16g/l; serum aspartate transaminase 518u/l, lactic dehydrogenase 452u/l; alkaline phosphatase 915u/l and γ-glutamyltransferase 430u/l; haemoglobin 9·3 g/dl. Treatment with low protein diet, lactulose, and spironolactone was instituted. The patient’s mental state deteriorated progressively over the next week and he became unresponsive to verbal stimuli but continued to respond purposefully to pain. At this time it was noticed that his eyes were deviated to the left in a conjugate fashion and failed to respond to caloric and oculovestibular stimuli. Pupils were equal and reactive to light and there was no papilloedema. Plantar responses were flexor and tone of the limbs was normal. Total bilirubin was now 316 μmol/l and serum ammonia 116 μmol/l (normal 17-47 μmol/l).

A structural cortical or brainstem lesion was suspected. CT scan was normal and EEG showed nonspecific slowing in the theta range. CSF analysis showed only the presence of bilirubin in the fluid. The eyes remained deviated until two days later when the patient died suddenly. The last total bilirubin level a few hours before death was 522 μmol/l.

A necropsy was performed. The brain (wet weight 1380 g) was jaundiced, but the most careful macroscopic and microscopic examination failed to reveal any structural lesion in any area. In keeping with the clinical picture, the liver was shrunked (840 g) and cirrhotic.

Stupor in this patient was due to hepatic encephalopathy. Cirrhosis with ascites proved by biopsy and necropsy, pedal oedema, abnormal liver function tests with elevated serum ammonia, diffuse EEG slowing and normal CT scan and CSF make this conclusion inescapable. The presence of sustained conjugate deviation of gaze, not previously described in hepatic stupor, was confusing and suggested a structural lesion. No such lesion could be demonstrated, either on CT scan or at necropsy. The occurrence of the conjugate deviation in this case must, presumably, be explicable on the basis of selective vulnerability to metabolic insult of centres associated with control of conjugate eye movements.

There is no doubt that toxic-metabolic conditions may affect brain-stem tegmental function as well as motor pathways and hemispheric structures.2 3 In such patients, systemic and laboratory evidence of severe metabolic disturbance and preservation of other functions at the same level should suggest a metabolic explanation for the signs.

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References


Recurrent meningitis due to labyrinthine fistula

Sir: We wish to describe two children who had recurrent meningitis. In children recurrent meningitis may have various causes.1-3 In the absence of an immunological defect, it may be the result of anatomical abnormalities, such as skull fracture, tumour and congenital defects. In a few cases, the pathology responsible for the recurrent meningitis may be in the middle ear.4-8 The triad of recurrent meningitis, severe unilateral or bilateral sensori-neural deafness and opaque tympanic membrane or other evidence of fluid in the middle ear should raise the possibility of middle ear pathology. The two children we describe, both presented with this triad arising from labyrinthine fistula.

A 5-year old girl with a normal birth history and development presented initially at 7 months of age with Haemophilus influenzae meningitis. She recovered quickly but developed severe bilateral sensori-neural deafness. A few weeks later, she had an attack of pneumococcal meningitis which recurred at the age of 23 months. Radiological examination revealed a defect at the apex of the petrous temporal bone. A left lateral cisternotomy with dural repair ± the middle fossa was performed at 2 years of age. However, she had five further attacks of meningitis which included three due to pneumococcus and two due to Haemophilus influenzae at between 2 years 3 months and 5 years 6 months. At the age of 5 years 8 months she was admitted to the Hospital for Sick Children, London. Examination showed the left tympanic membrane to be normal, but the right was bluish and a fluid level was seen. She had bilateral sensori-neural deafness. The rest of the neurological examination, including full haematological examination, was normal. The results of investigations were not diagnostic: haemoglobin level was 11·9 g/dl; white cell count was 18.7 ×10⁹/l (neutrophils 71%; lymphocytes 21%; reticulocytes 2%; eosinophils 2%; basophils 1%). Complement C3 was 108% of standard. Nitroblue tetrazolium test was normal. Radiographs of the sinuses showed minor mucosal thickening in the antrum. Chest radiograph was normal. Tomograms of the mastoids showed the lateral part of the right internal auditory meatus wider than that on the left, and the vestibule was slightly larger. The basal coil of the right cochlea was normal. Computed tomography (CT) showed an opaque right middle ear. A right exploratory transmeatal mastoidectomy showed an intratympanic fistula. The hearing was not improved. The parents consented to hearing aids. She was discharged with no further attacks.

A 12-year-old boy who had recurrent attacks of meningitis with Haemophilus influenzae meningitis was admitted aged 3 months. He had recurrent meningitis at the age of 7-9 months with Haemophilus influenzae meningitis. He recovered quickly. He had recurrent meningitis at the age of 2 years 9 months with Haemophilus influenzae meningitis. He recovered quickly but developed severe bilateral sensori-neural deafness. A left lateral temporal cisternotomy with dural repair was performed at 3 years 4 months. Examination showed a right tympanic membrane to be normal, but the left ear was cloudy with fluid level. She had bilateral sensori-neural deafness. The rest of the neurological examination, including full haematological examination, was normal. The results of investigations were not diagnostic: haemoglobin level was 10·5 g/dl; white cell count was 17×10⁹/l (neutrophils 70%; lymphocytes 24%; reticulocytes 1%). Complement C3 was 107% of standard. Nitroblue tetrazolium test was normal. Radiographs showed minimal mucosal thickening in the antrum. Chest radiograph was normal. Tomograms of the mastoids showed the lateral part of the right internal auditory meatus wider than that on the left, and the vestibule was slightly larger. The basal coil of the right cochlea was normal. Computed tomography (CT) showed an opaque right middle ear. A right exploratory transmeatal mastoidectomy showed an intratympanic fistula. The hearing was not improved. The parents consented to hearing aids. She was discharged with no further attacks.
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