Lesson of the Week

Subdural empyema: an important diagnosis not to miss

ANDREW G FARKAS, J CHARLES MARKS

Subdural empyema is a rare condition, seen only two or three times a year in the average neurosurgical unit in patients usually referred by general physicians. Overall mortality remains as high as 28%[1] despite surgical drainage and treatment with antibiotics. We describe three patients seen in the past year, illustrating how delay in making this difficult diagnosis leads to poor outcome.

Subdural empyema should be considered in any patient with signs of meningitis who has a suspected source of infection outside the central nervous system; CT with contrast should be requested as a matter of urgency and done before lumbar puncture

Case histories

Case 1—A 23 year old man presented with a four week history of sinusitis treated with various antibiotics. After seven days of occipital headache and three days of vomiting, drowsiness, and confusion he was seen by a general practitioner and sent to hospital with a suspected diagnosis of meningitis. Neck stiffness and a fever of 39·5°C were found. Lumbar puncture yielded cerebrospinal fluid at a pressure of 500 mm water and containing 2·5 g protein per l, 3200 white blood cells, but no organisms. This was taken as confirmation of meningitis. Despite antibiotics his condition deteriorated and next day he was referred to the neurosurgical unit, by which time he was unresponsive to command and had a left hemiplegia. A computed tomography (CT) brain scan with contrast (fig 1) showed an opaque right frontal sinus, midline shift to the left, and an isodense subdural collection over the right hemisphere with no enhancement. Craniotomy and drainage of the empyema and sinus with bone flap removal was performed immediately. Despite antibiotics he died nine days postoperatively from severe brain swelling.

Case 2—A 38 year old woman with a ventricular septal defect and Eisenmenger's syndrome presented with seven weeks of persistent, severe left frontal headache and pronounced left frontal tenderness. Three weeks after the onset of headache examination by a neurologist had shown nothing abnormal. Radiographs of sinuses were normal but the erythrocyte sedimentation rate was 44 mm in the first hour. In the absence of other abnormalities temporal arteritis was diagnosed and high dose prednisolone started four days before admission. The headache worsened and she became drowsy and feverish with vomiting. The differential diagnosis on admission was meningitis and cerebral abscess. Peripheral white cell count was $32{\cdot}2\times10^3/mm^3$. Lumbar puncture yielded a cerebrospinal fluid protein concentration of 0·79 g/l, normal glucose concentration, and scanty lymphocytes but no firm diagnosis was made. After deterioration overnight a CT scan with contrast at the referring hospital showed swelling in the left hemisphere with pronounced midline shift. During transfer to the neurosurgical unit the same day she became unconscious with a dilated, unreactive right pupil and right hemiplegia. Despite immediate craniotomy with drainage of an empyema and removal of the bone flap, adequate antibiotic treatment, and ventilation she died two days postoperatively. Microaerophilic streptococci were cultured from the pus. Necropsy showed no evidence of sinus or middle ear disease, nor of intracerebral abscess with rupture into the subdural space. There was extensive pus over the tenorium cerebelli, in the middle fossa, and in the parafalcine area.

Case 3—This 58 year old woman with Parkinson's disease had a three week history of severe left sided headaches. For a week her left eye had been swollen and her general practitioner in concurrence with an ophthalmologist had diagnosed either cellulitis or early shingles and prescribed erythromycin and acyclovir. She became aphasic over four days but on admission to hospital had no other focal neurological signs and was not feverish. A CT scan with contrast at the neurosurgical unit the same day showed swelling of the left cerebral hemisphere. An incorrect diagnosis of encephalitis was made on the clinical and CT appearances without an electroencephalogram. She was returned to the referring hospital. She rapidly developed a fever of 39·0°C, focal seizures, and spastic right hemiparesis. Lumbar puncture at the referring hospital yielded cerebrospinal fluid at a pressure of 290 mm water and containing protein 1·1 g/l, 47 white blood cells (all lymphocytes), but no organisms. After review of the CT scan next day by a consultant radiologist, who noted a hypodense area in the left temporal area as well as the swelling (fig 2), she was immediately transferred back to the neurosurgical unit. A craniotomy was performed and a subdural empyema drained and bone flap removed. Three months later she remained hemiparetic and incapacitated though alert and talking. We found no sign of disease in sinuses or the middle ear.

Discussion

All three patients were seen by several doctors before the correct diagnosis was made, the delay possibly contributing to the poor outcome. The clinical features of headache progressing to impaired consciousness, hemiparesis, and fever are typical,[2] but given the rarity of subdural empyema the diagnosis was missed.

Can estimation of the white cell count in the cerebrospinal fluid make the diagnosis of meningitis acceptable, so avoiding the need to investigate for an intracranial abscess? The counts in the cerebrospinal fluid in these three cases were 3200, "scanty," and 47 respectively. A high white cell count (case 1) is not specific to meningitis. The indications for further investigations to exclude abscess are discussed below. Lumbar puncture carries hazards. Each patient had a lumbar puncture which was followed by deterioration in clinical state, even though the history was suggestive of raised intracranial pressure and an abscess might have been suspected. Coning after lumbar puncture may be delayed for up to 12 hours from continuing leakage of cerebrospinal fluid through the perforated dura.[3]

In patients with a provisional diagnosis of meningitis CT is indicated when focal neurological signs are present. What, then, are the indications for CT in patients who do not have focal signs? It is in these patients that early diagnosis of abscess may most substantially improve prognosis. We suggest that any patient with signs suggestive of meningitis in the presence of a suspected source of infection outside the central nervous system, most commonly in the middle ear, paranasal sinus, or scalp, should have a CT scan with contrast. If this criterion had been adhered to in case 2 the patient, with a potential source of infection in the heart, would have had an earlier

Department of Neurosurgery, Frenchay Hospital, Bristol BS16 1LE
ANDREW G FARKAS, MB, BS, senior house officer in neurosurgery
J CHARLES MARKS, FRCS, registrar in neurosurgery

Correspondence to: Mr Marks.

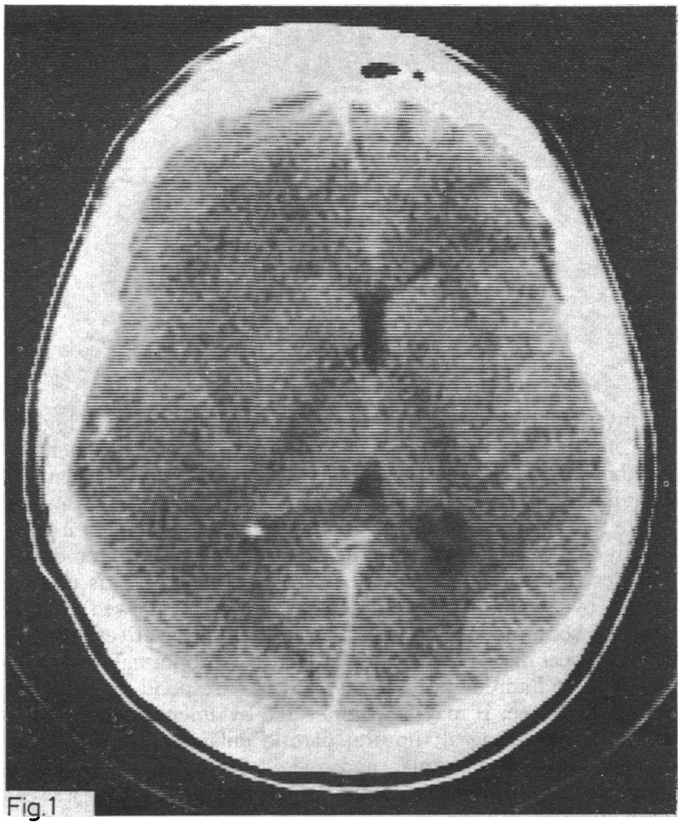

FIG 1—Case 1. CT scan in subdural empyema showing midline shift to left and an isodense subdural collection over right hemisphere.

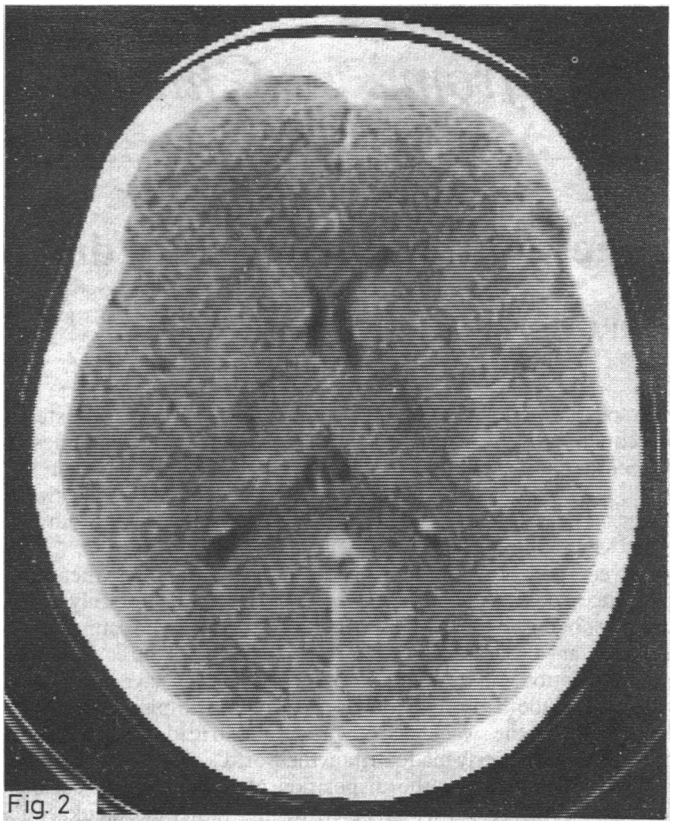

FIG 2—Case 3. CT scan in subdural empyema showing slight midline shift and hypodense area in left temporal region.

CT scan. CT does not always provide an initial, specific radiological diagnosis of pus, which may have the same radiodensity as brain tissue, but some radiological changes are detected in virtually all cases.[2]

What are the indications for transferring patients with a provisional diagnosis of meningitis to a neurosurgical unit? We suggest that these rest on the presence of focal neurological signs (cases 1 and 3) or, in their absence, abnormalities in the CT scan. If scanning is not available in the referring hospital the need for a scan is an indication for transfer. Such CT scan abnormalities include swelling in a hemisphere with midline shift even without other definite signs (cases 2 and 3).

x Ray pictures can now be transmitted along an ordinary telephone line by British Telecom's new technique of image transmission (IMTRAN). This permits discussion of CT scans in conjunction with the clinical picture by senior, experienced staff in specialised centres. The ability of this technique to provide an image of diagnostic quality in suspected cases of subdural empyema, however, is yet to be proved.

The large majority of subdural empyemas are secondary to paranasal or otogenic infections.[2] Only one of our three patients had obvious sinus infection. In case 2 the patient had Eisenmenger's syndrome with right to left shunt, a condition associated with intracerebral abscess but not subdural empyema. The origin of infection in case 3 remains unknown. A microaerophilic streptococcus was isolated in only one patient; this organism is associated with a poor prognosis.[2] The other two patients had previously received antibiotics. The importance of culturing the organism—and in particular anaerobic organisms—in subdural empyema has been emphasised,[4] since anaerobes are common pathogens in sinus infections.

In conclusion, the mortality of this condition remains distressingly high despite radical craniotomy and antibiotics. It is clear that craniotomy gives an improved chance of survival,[1] but only if the disease is recognised before deterioration of conscious level or the appearance of neurological signs or fits. Subdural empyema should be suspected where there is clinical evidence of intracranial infection together with a suspected extracerebral source of infection. Such evidence or focal neurological signs makes CT with contrast obligatory. This should be performed before lumbar puncture.

References

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Is the use of a beta-blocking drug for tinnitus a recognised line of treatment, and what can be its mode of action?

Betablockers do not constitute a generally recognised treatment for tinnitus. Indeed they are becoming increasingly recognised as having some ototoxic effects[1] and hence as being a potential cause of tinnitus. The problem stems in part from a lack of definition of what is being treated in tinnitus. Only intravenous lignocaine has been shown to be significantly effective in actually abolishing the symptom—and then only for very short periods. Recent studies, however, have shown that tinnitus complaint behaviour is not unidimensional, but rather comprises several different orthogonal factors including "distress," "intrusiveness," "sleep disturbance," and "interference with passive auditory entertainment."[2] One of the major components of "tinnitus distress" is anxiety. By relieving the psychophysiological aspects of this, certain betablockers may offer some relief from distress provoked by tinnitus.—S D G STEPHENS, consultant in audiological medicine, Cardiff.

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