hyperreflexia would not exclude a diagnosis of botulism.

JC MARTÍNEZ-CASTRILLO
MA DEL REAL
A HERNANDEZ GONZALEZ
G DE BLAS*
JC ALVAREZ-CERMEÑO
Servicio de Neurología,
*Servicio de Neurofisiología,**
Hospital Ramón y Cajal,
28034 Madrid, Spain

Correspondence to Dr Martinez Castrillo.

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Herpes simplex encephalitis following a skull fracture

Herpes simplex encephalitis is an uncommon and severe infection, in which early detection and treatment significantly improve the outlook.[1] We report a case following a depressed skull fracture.

A nine year old boy with diabetes since the age of two was admitted with a head injury following a fall from his bicycle. Examination revealed a right frontal laceration. Skull radiographs showed an extensive bifrontal fracture, depressed on the left. At operation brain and cerebrospinal fluid was seen oozing from the laceration. A bicoronal scalp flap was turned. The depressed area was elevated and cleaned, and a burr hole was made to aid exposure and repair of a dural tear on the right. Ampicillin and flucloxacillin were given intravenously for three days and subsequently orally for four more days. He was discharged on day 5 with no neurological deficit, and was given sodium valproate prophylactically.

On day 20 he was readmitted with a two day history of moodiness and staring episodes culminating in a generalised convulsion. Neurological examination was normal and he was apyrexial. Blood sugar was 8 mmol/l. Diazepam was given rectally when a generalised convulsion was observed on the ward. A diagnosis of post traumatic epilepsy was made. An electroencephalogram showed moderate amplitude irregular waves in the frontal leads compatible with the recent injury. By day 23 he had had further episodes preceded by sensations of odd tastes and manifesting as altered consciousness with jaw clenching, drooling and post-ictal left sided weakness. Carbamazepine was added, and the frequency of these episodes decreased. On day 25 a cranial CT scan was normal.

On day 27 he developed a pyrexia and right sided earache. Examination of the ear was normal. The fever recurred the following day, and intermittent seizures progressed to focal status epilepticus which was controlled with an infusion of chlormethiazole. Lumbar puncture revealed 38 white cells, predominantly lymphocytes, and one red cell per high power field. CT scan showed an area of mixed density with enhancement in the right temporal region (figure). This raised the possibility of either an evolving bacterial abscess (although the CT scan lesion was contralateral from the site of the depressed part of the fracture), or a focal encephalitis. He was accordingly treated with chloramphenicol 100 mg/kg/day, benzylpenicillin 200 mg/kg/day, metronidazole 22 mg/kg/day, and acyclovir 10 mg/kg/day. On day 29 he deteriorated with diabetic ketoacidosis. A small ulcer was seen on his lip. A further electroencephalogram showed diffuse severe abnormality, with very high amplitude irregular delta waves occurring in all leads. Despite his increasingly critical condition, a right craniotomy was performed on day 31 to exclude the possibility of an abscess, and this revealed soft inflamed brain with no abscess. Histology of the biopsy specimen showed cerebral cortex containing foci of necrosis with oedema. Perivascular cuffing with lymphocytes was observed; occasional polymorphs and eosinophilic cells were seen. Gram stain for microorganisms was negative. Herpes simplex virus was identified on electron microscopy, and was also subsequently cultured from the biopsy specimen. He was given ventilation for four days postoperatively. Acyclovir was continued for 10 days. Recovery was complicated by a dense left hemiparesis and a pseudobulbar palsy. Subsequently, he made an impressive recovery, and is currently able to run, but has dysarthria, and has little use in his left hand.

Intracerebral bacterial infection is a well recognised but, with appropriate management, a relatively uncommon complication of compound skull fractures, occurring at a rate of 4% in one large series.[2] There are no previous reports of herpes simplex encephalitis (HSE) following a skull fracture. Contamination of the wound is the route of spread of bacterial infection, but our case presumably represents a reactivated latent infection (from viral deoxyribonucleic acid present in the brain) in response to the considerable stress of the injury, rather than innoculation during the original injury.

Absence of a fever and an initially normal CT scan delayed the identification of infection as the cause until 10 days after the onset of symptoms, during which time the diagnosis was of post traumatic epilepsy. Craniotomy and brain biopsy are not felt to be mandatory to reach a diagnosis of HSE in children in whom there is little to suggest an alternative diagnosis,[3] but in this case it was felt important to exclude the possibility of a cerebral abscess.

Focal seizures with altered consciousness should always alert the clinician to the possibility of HSE, even in the absence of a fever or CT scan changes.

P D CAMERON
P BLACKBURN
S J WALLACE
Department of Paediatrics,
University Hospital of Wales,
Heath Park, Cardiff and
Department of Neurosurgery,
The Hospital for Sick Children,
Great Ormond Street, London

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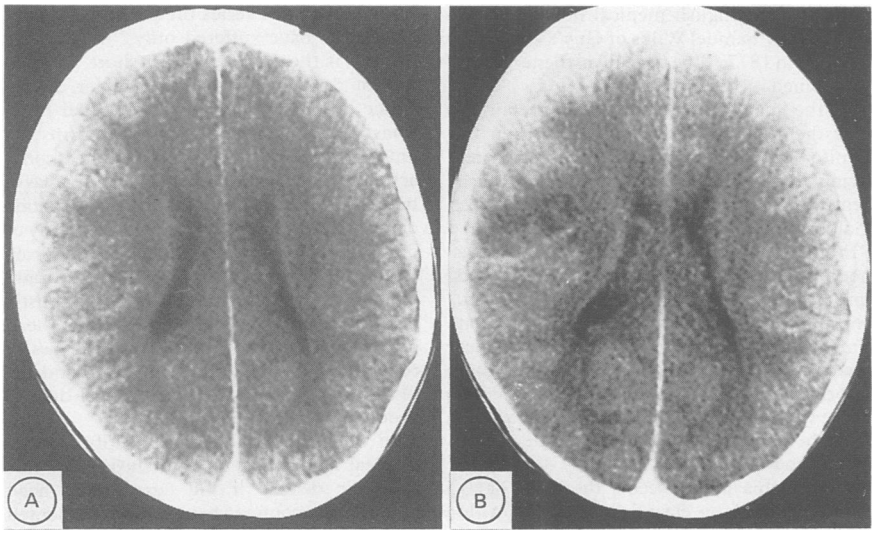

Figure Evolution of the right temporal lesion shown in CT scans; normal scan on day 25, lesion apparent on day 28.

Could lamotrigine be useful in status epilepticus? A case report

Lamotrigine, a new putative antiepileptic drug[1] may have been effective in a case of status epilepticus. The patient was a 17 year old, mentally handicapped, right handed girl who had experienced epilepsy of unknown aetiology from the age of nine months and had been attending our clinic since 1981. At the time of her latest admission to hospital, the patient had been taking a combination of carbamazepine and phenobarbitone at daily doses of 1200 and 200 mg, respectively for about a year, with partial control of seizures (nocturnal tonic fits: 10–30 per month; atypical absences: 3–5/month; atonic seizures: 3–5/month). Interictal EEG recording was characterised by diffuse slow waves, occasionally accompanied by spikes or polyspikes isolated or grouped in symmetrical, generalised bursts. She was admitted to hospital for an unexplained sudden increase in tonic seizures (up to 4–6/hour without recovery of consciousness between fits). A