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CASE REPORT

An aggressive seizure and behavioural disorder following trivial head injury

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We report on a normal 6-year-old boy in whom a trivial head injury triggered a severe seizure, behavioural and cognitive disorder. Complete recovery occurred within 6 months. An aetiology such as trivial head injury is significant for prognosis as the outcome is invariably excellent.

Key words: trivial head injury; seizures; behaviour.

INTRODUCTION

The role of trauma in the pathogenesis of seizures is well recognized[1,2]. However, most relevant reports refer to patients seeking medical attention for their injury and are biased towards more severe cases[1–5]. Some of these patients may have mild trauma, but trivial injury will be overlooked. We present a child who developed an aggressive seizure and behavioural disorder following trivial head injury.

CASE REPORT

This boy was well with normal development until the age of 6 years. At 8 pm one evening while on his bicycle in his home, he collided with another child, fell off his bicycle and hit his head on the marble floor. There was no laceration or loss of consciousness, but he cried for several minutes. He went to bed normally but on awakening looked unwell and 10 minutes later had a partial seizure. He walked away from his mother 'as if he did not know where he was going', his lips became blue, he salivated and was incontinent. During the next 12 hours he had a further four partial seizures with one progressing to secondary generalization. On admission to a paediatric hospital he received intravenous diazepam and oral carbamazepine was initiated at 10 mg/kg daily. His neurological state, skull x-ray, electroencephalograph (EEG) and computer tomography (CT) brain scan were normal. Comprehensive haematological, biochemical and viral investigations including examination of his cerebrospinal fluid were normal.

His behaviour changed dramatically, his parents described him as 'intolerable', and there was severe impairment of concentration and learning ability. Four days later he started to have numerous nocturnal and diurnal seizures consisting of frightened laughter, impairment of consciousness, rhythmic tremor of the lips and occasionally incontinence of urine. These were video-taped and reviewed. Video EEG of one of the seizures was described as showing fixation of gaze and automatisms accompanied by diffuse slow waves at 2 Hz. The background had an alpha rhythm at 7–8 Hz and bilateral runs of delta with left anterior emphasis. High-resolution magnetic resonance imaging (MRI) was performed twice and was normal. Neither his seizures nor his behaviour changed when carbamazepine was substituted with sodium valproate. However, 2 months later seizures stopped when ox-carbazepine was added to sodium valproate. At this stage, an all-night video EEG showed three electrical seizures of repetitive spikes and spike–slow

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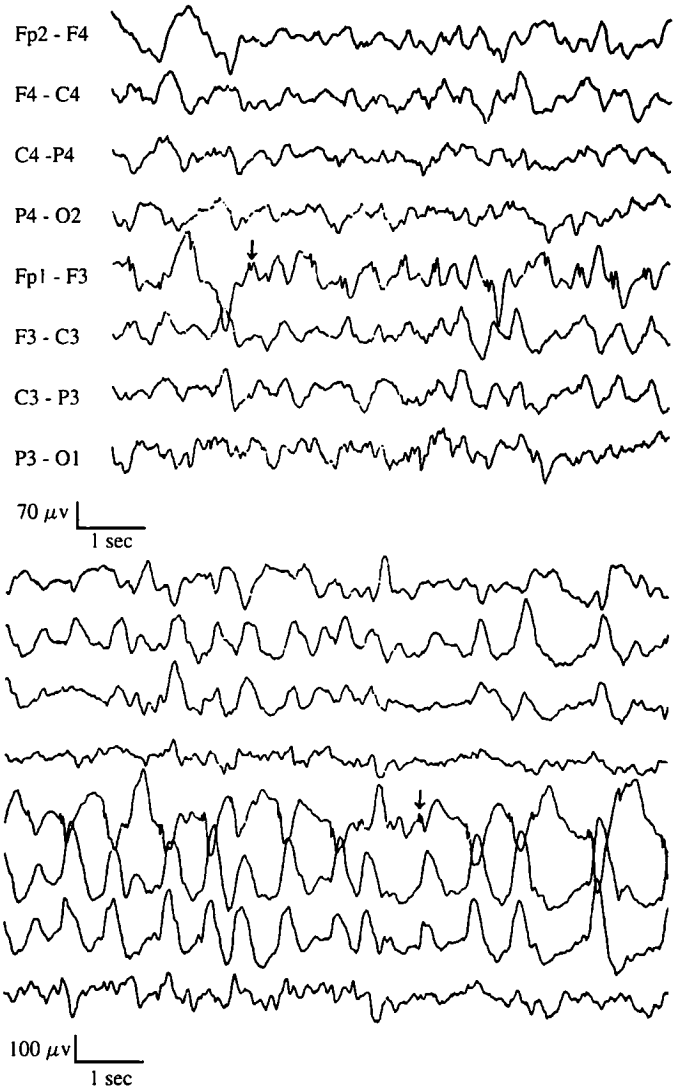

Fig. 1: EEG recording of a 3-minute electrical seizure during sleep, 4 months after injury. Upper: Onset with small spikes in Fp1–F3 (arrow). Lower: Spikes (arrow) and high-amplitude slow waves at 2 Hz in the left fronto-central electrodes, 45 seconds later.

waves in the left frontal electrode lasting for 2–3 minutes (Fig. 1). There were no detectable clinical events.

His abnormal behaviour started to improve 1 month after cessation of seizures and he was normal 4 months later. On review, 2 years after injury he has had no further seizures and his behaviour and school progress are normal. An EEG recorded during natural sleep was well organized with frequent medium amplitude sharp and slow waves localized around the right central electrode and less frequently in the left mid-temporal electrode.

DISCUSSION

Parents often attribute their child's seizures to a trivial head injury. It is our practice to disregard this association. This is the first child in whom such a relation is evident. It cannot be coincidental that an aggressive seizure and behavioural disorder erupted within hours of an injury and remitted within months. One may speculate that the centrotemporal spikes found in his recent EEG explain his susceptibility to a trivial head injury with a response similar to that of the benign childhood epilepsy with affective symptoms[6].

The definition of mild and trivial head injury varies amongst different reports of post-traumatic seizures. Jennet[1] defined trivial injuries as those with 'no post-traumatic amnesia, no depressed fracture and no intracranial hemorrhage', but included cases with linear fractures or loss of consciousness, which cannot be considered trivial. Early onset post-traumatic seizures within 1 week of head injury, had an incidence of 5% in children admitted with all types of head injury and in 80% occurred within 24 hours. There

was continuation of fits in 17% after the first week with a similar incidence in 'trivial and non-trivial' injuries. Children under 5 years were more susceptible than those over 5 years. Using Jennet's definition, Snoek *et al*[3] identified 42 children with 'trivial' head injury followed by a symptom-free period and subsequent neurological deterioration. Thirteen had post traumatic seizures within the first hours, but only one had further seizures beyond the first week following the injury. The remaining children had neurological deterioration without seizures. Oka *et al*[4] identified 34 children with 'mild or trivial' head injury and neurological consequences. Twenty six had post traumatic seizures within 6 hours of injury and none continued for more than 48 hours.

Annegers *et al*[5] reported that only 6 of 614 children with 'mild' head injury had early seizures, but the incidence of late seizures was similar to that in the general population despite the inclusion in 'mild head injury' of cases with loss of consciousness.

Our case fulfills all the criteria of trivial head injury as defined by Aicardi[7] with no immediate symptoms beyond crying. The seizures in this child were associated with the abrupt onset of a severe behavioural and cognitive disorder. Though rare, these disturbances are seen in children for several months following mild head injury[8]. Well-controlled studies have found no lingering deficits after 1 year[9].

Aetiology of the delayed deterioration after head injury has been debated. Leao's[10] spreading wave of neuronal suppression, rapid changes in cerebral blood flow[11], direct contusion of the cortex, focal or generalized brain swelling due to hyperaemia or oedema and diffuse axonal injury due to shearing have all been implicated[12]. Cerebral maturation may make adults more resistant and skull malleability makes infants less resistant to injury[7].

Studies on the role of the EEG and neuroimaging in prediction of post-traumatic seizures vary according to the severity of injuries and anticonvulsant used. Post-traumatic seizures may occur in patients in whom the EEG was normal initially[2], although EEG abnormalities are frequent in head injury. Levin *et al*[13] identified parenchymal lesions in 17 of 20 patients with mild or moderate head injury with MRI. Positron emission tomography has been used in patients with poor neuropsychological outcome following minor head injury and showed a high incidence of temporal-lobe abnormality[14,15].

In conclusion, trivial head injury may rarely trigger a severe seizure, cognitive and behavioural disorder. Such an aetiology is significant for prognosis as outcome is invariably excellent as this child demonstrates.

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