Early posttraumatic seizures in non-accidental head injury: relation to outcome

Karen M Barlow MB ChB MRCP, Paediatric Neurology Research Fellow;
Jacqui J Spowart, Senior Neurophysiology Technician;
Robert A Minns* PhD FRCP(Edin), Consultant Paediatric Neurologist, Department of Paediatric Neurosciences, Royal Hospital for Sick Children, Edinburgh, Scotland.

**Correspondence to last author at* Department of Paediatric Neurosciences, Royal Hospital for Sick Children, 9 Sciences Road, Edinburgh EH9 1LF, Scotland.
E-mail: Robert.Minns@ed.ac.uk

To document the characteristics of early posttraumatic seizures (EPTS) in non-accidental head injury (NAHI), and examine their relation with outcome, a retrospective study was carried out. All children with NAHI admitted to the Royal Hospital for Sick Children, Edinburgh, since 1981 were identified. The characteristics of EPTS, EEG, and outcome were noted. Forty-four cases were identified. The average age of children at presentation was 5.9 months. Thirty-two of these children had EPTS. The median length of follow-up was 3 years. The mortality rate was six in 44 (14%). The neurodevelopmental outcome correlated significantly with the presence and severity of EPTS (Tau=0.317, p=0.017). Of survivors, 22% developed late posttraumatic epilepsy; the outcome in those with epilepsy was significantly worse than those without (p<0.0001). It was concluded that the severity of the primary brain injury dictates the severity of the EPTS and neurodevelopmental status at follow-up.

Seizures are a common complication of traumatic encephalopathies. Early posttraumatic seizures (EPTS) are defined as occuring within 1 week of the trauma (Jennett and Teasdale 1973). In accidental head injury of all severity in children, the overall incidence of EPTS is between 5 and 6.5%, but can be up to 30% in children with severe head injuries.

In accidental head injury the incidence of EPTS is known to be higher in children than adults (4.2% compared with 0.9%, respectively; Kollevold 1976); higher in younger than older children (15.7% in children <2 years, 11.6% in children <3 years); more frequent in the presence of an intracranial haematoma (Kollevold 1976); and increased with increasing severity of the injury (all injury severity 5 to 6.5% compared with up to 30% in severe head injury only; Annegers et al. 1980).

Although EPTS are seen frequently in non-accidental head injury (NAHI), there are only incidental reports of their incidence ranging between 65 and 74% (Bonnier et al. 1995, Ewing-Hobbs et al. 1998). There are no studies that examine the nature of EPTS in detail or their relation with outcome in NAHI.

The aim of this study was to document the frequency and clinical features of EPTS in the acute encephalopathy of NAHI and the EEG abnormalities, and to examine their relation with outcome.

Method

This is a retrospective study. All children admitted to the Royal Hospital for Sick Children in Edinburgh with an acute encephalopathy due to a NAHI between 1981 and 1998 were entered into the study. As the perpetrator rarely confesses to causing the injury, this diagnosis is often one of suspicion. Diagnostic criteria were to reduce the possibility of false positives. All cases had to have an acute encephalopathy plus at least two of the following: retinal haemorrhages; skeletal fractures (e.g. rib fractures, metaphyseal avulsions); a history which was inconsistent or inconsistent with the examination; subdural haemorrhage; or other malicious injury (e.g. burns, bite marks; Goldstein et al. 1993). Using these criteria, a child who simply has an isolated subdural effusion without an adequate explanation would be excluded from the study.

IDENTIFICATION OF CHILDREN

The children were identified by various methods: ICD-9 and ICD-10 criteria with a combination of head injury plus child abuse, ward and intensive care admission records, social work records, and hospital death records.

CLINICAL DETAILS

Hospital records were reviewed and clinical details noted. Information about early posttraumatic seizures was obtained using both medical and nursing notes. It is not always possible to assess precisely the time from injury to seizure onset in these cases, as often the history is unreliable with the occurrence of any injury sometimes being denied. In only 15 cases were we able to document with certainty the time interval between injury and first seizure. Therefore, we took the fixed point as the time of admission (to any hospital) to the first seizure. The number, length, and duration of seizures was noted together with the presence of status epilepticus. Status epilepticus was defined as a seizure or

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repeated seizures without return of consciousness lasting more than 30 minutes. Seizures were classified as (1) responsive to medication without episodes of status epilepticus or (2) unresponsive to medication (failure to respond to at least two antiepileptic medications) with or without episodes of status epilepticus.

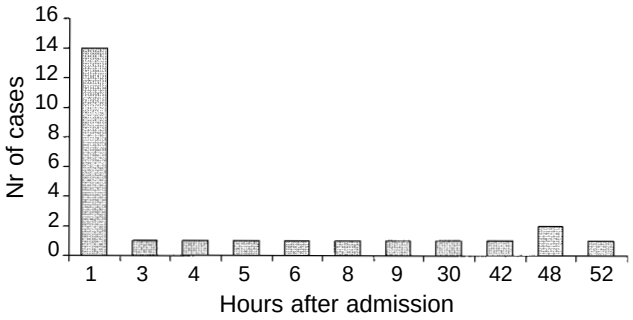

Figure 1: *Early onset of EPTS in non-accidental head injury. EPTS affects 56% on arrival or within first hour after admission, 85% within first 24 hours, and all by 52 hours.*

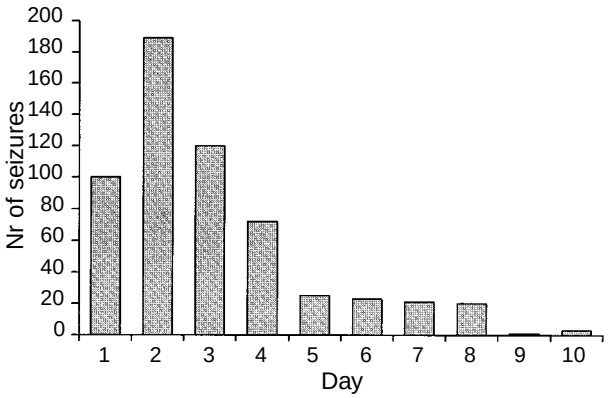

Figure 2: *Greatest frequency of seizures occurred in second 24-hour period after admission, and all seizures had resolved after day 10.*

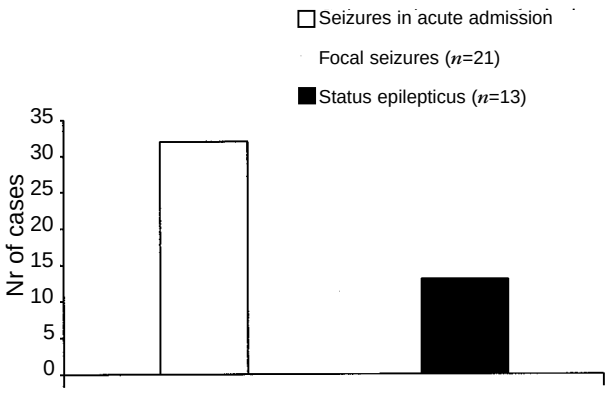

Figure 3: *EPTS occurred in 74% of cases of NAHI. Of these, focal seizures were present in 66% and 41% had one or more episodes of status epilepticus.*

EEG

The EEGs performed during the acute encephalopathy were reviewed. These recordings used standard 10 to 20 international head measurement system with polygraphy. The patient's age, state of arousal, and details of medication at the time of the EEG were noted.

OUTCOME

An outcome was awarded based on the last hospital follow-up. Seshia's 6-point coma outcome classification score was used as this includes both neurological and developmental outcome measures. The abnormal outcome categories are classified as mild, moderate, severe, and vegetative based on increasing deficits in muscle tone, cranial nerve, and other focal neurological deficits. Where there is a mismatch between the neurological and the developmental score, the worst outcome category is taken (Seshia et al. 1983, Seshia 1994). Information about the presence and severity of late posttraumatic epilepsy at follow-up was also noted.

STATISTICAL ANALYSIS

Non-parametric statistical analysis to examine the relation of the presence and severity of EPTS and the development of late posttraumatic epilepsy with outcome was performed using the Kendall rank correlation method. Fisher exact test was used to examine the frequency of status epilepticus and EPTS in the groups with and without late posttraumatic epilepsy at follow-up.

Results

Forty-four children (29 males,13 females) with NAHI were identified. Forty-two children had follow-up. The average age at presentation was 5.9 months (range 1 to 34 months). Thirty-two children had early posttraumatic seizures (23 males, nine females). In six of the 32 children, seizures commenced before presentation at hospital. Sixteen of the 32 children were transferred from referring hospitals. In 21 of the 32 children the onset was within 24 hours and all had an onset within the first 3 days of hospital admission (Fig.1). The seizures reached a peak frequency on the second day, and had stopped in all cases by day 10 (Fig. 2).

Sixteen children had intractable seizures, which failed to respond to at least two antiepileptic medications. Usual antiepileptic medications used included boluses and infusions of benzodiazepines (diazepam and midazolam), phenytoin, paraldehyde, phenobarbitone, and thiopentone. Twenty-one children with EPTS had focal clinical seizures; the focus frequently varied. Thirteen had one or more episodes of status epilepticus, all of which occurred in the first 48 hours of admission (Fig. 3).

EEG

Twenty-eight of the 32 children with EPTS had EEG performed during the acute encephalopathy; most were performed with 72 hours of the acute admission.

Background

Marked background asymmetry was seen in eight cases. In six the background activity was of very low voltage. Three had very high amplitude slow waves (300 μV). In three cases the EEGs were isoelectric.

Seizure activity

Seizure activity was seen in 14 children; 10 had multifocal seizure activity, four had focal seizure activity. Subclinical seizures were noted in four children. An example of an EEG in NAHI is shown in Figure 4.

OUTCOME

Six children died in the acute admission. Two children moved to another region. Twenty-five children were seen by KMB at a special hospital follow-up clinic. The remaining 17 had their outcome assessments based on the most recent hospital and community health records. The median length of follow-up in the survivors was 3 years (range 3 months to 18 years). At follow-up 14 children were developing normally, while neurodevelopmental abnormalities were mild in six, moderate in eight, and severe in eight.

The presence of seizures and whether those seizures were intractable was significantly related to the outcome, $n=42$, Tau$=0.317$, $p=0.017$ (Fig. 5). The number of children without seizures is overly represented in the group with a normal outcome. However, most of those with intractable seizures had a poor outcome and only one with intractable seizures

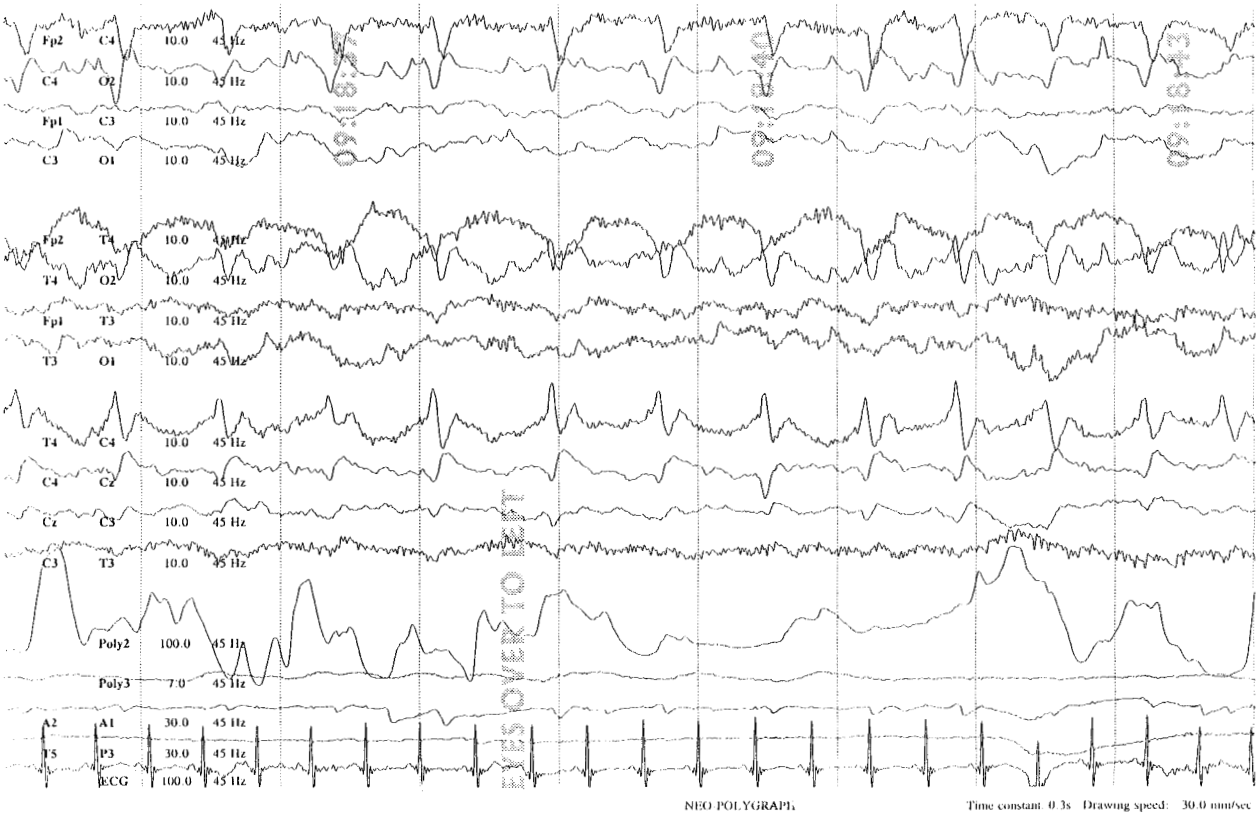

Figure 4: *EEG performed on day 2 on a 5-month-old girl with confirmed NAHI during early posttraumatic seizure. Multifocal epileptic discharges can be seen over right hemisphere (leads Fp2:C4:O2; Fp2:T4:O2; T4:C4:Cz). Respiratory pattern (lead Poly2) is markedly irregular, eyes deviated to left with fine nystagmus seen clinically and is shown on lead A2:A1.*

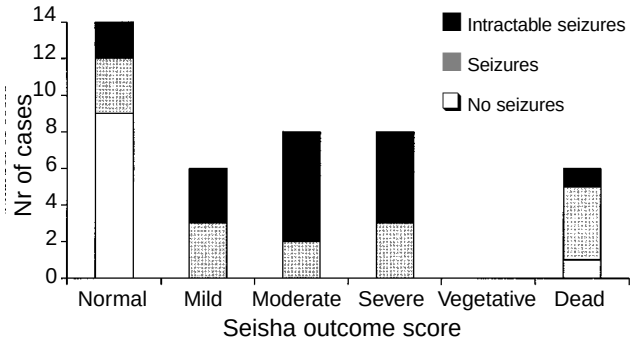

Figure 5: *EPTS occurred in only five of children with NAHI who were developing normally at follow-up.*

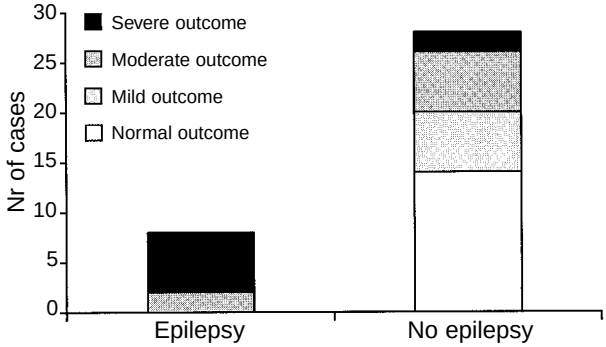

Figure 6: *Relation between neurodevelopmental status and presence of late posttraumatic epilepsy at follow-up.*

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had a normal outcome. One of the six children who died did not have any seizures, but had fixed dilated pupils and an isoelectric EEG within hours of arrival.

POSTTRAUMATIC EPILEPSY

At follow-up eight children had epilepsy, five of whom had intractable epilepsy. Intractable epilepsy is defined as at least one seizure a day despite medical treatment. The severity of the EPTS was not related to the development of posttraumatic epilepsy ($p=0.107$) and one or more episodes of status epilepticus during the acute encephalopathy was not related to posttraumatic epilepsy at follow-up ($p=0.103$). At follow-up, those with epilepsy had significantly greater disabilities than the group without epilepsy, Tau=–0.593, $p<0.0001$ (Fig. 6).

Discussion

The most common form of traumatic head injury in infancy resulting in death is inflicted or NAHI. NAHI occurs most frequently in the first 6 months of life and rarely in children older than 2 years (Duhaime et al. 1987, Brown and Minns 1993). The most common mechanism of injury is a whiplash-shaking injury with or without impact. This results in marked rotational acceleration/deceleration forces within the cranium causing widespread parenchymal damage, subdural haematomas, and diffuse axonal injury. Other mechanisms of injury include pure impact injuries, compression injuries, and penetrating brain injuries. NAHI is important as it has a high mortality rate ranging between 13% and 30% and a high morbidity rate (Duhaime et al. 1987, Brown and Minns 1993, Bonnier et al. 1995, Barlow et al. 1998).

Our results show the natural history of EPTS in NAHI. EPTS can occur before admission (6/44, 14%), and when they do occur they have mostly commenced within 24 hours of admission (27/32, 85%), reached a peak severity and frequency by day 2 (see Fig. 2), and have usually resolved by 1 week (29/32, 91%).

In accidental head injury in children, posttraumatic epilepsy occurs in <5% of cases and is related to the severity of the trauma (Annegers et al. 1980). In our cohort of children with NAHI, eight of 36 (22%) had posttraumatic epilepsy; we found this not to be significantly correlated with EPTS severity nor status epilepticus in the acute encephalopathy. There was, however, a strong statistical relation between the presence of posttraumatic epilepsy and severe degrees of outcome category. We have also shown that the presence of EPTS and intractable seizures was significantly related to poor outcome (see Fig. 5). It can be deduced, therefore, that the acute brain damage responsible for the disability and posttraumatic epilepsy is the most likely cause of the EPTS and status epilepticus than the converse (i.e. EPTS and status epilepticus responsible for the resultant brain injury, although we recognize the potential for this to occur). Laminar grey matter shearing is one component of the severe parenchymal damage from the primary injury, evident on MRI in the early encephalopathy (Barlow et al. 1999) and this shearing isolates the cortex from reticular control and results in intractable seizures. Intractable seizures are also a common feature of other laminar dissociations, i.e. laminar necrosis or laminar dysplasia. GABAergic inhibition is also affected because anticonvulsant drugs such as benzodiazepines are ineffective. While the intractable fits will generally remit after 3 to 4 days, it is important to avoid attempting to control them by polypharmacy because of the consequences of reduced cerebral perfusion and secondary brain injury.

The mortality rate of six out of 44 (14%) is considerably lower than earlier studies have reported. Whether this represents a real decline in mortality, due to better intensive care and neurosurgical management, other factors related to the accuracy of diagnosis, different mechanisms of primary injury, etc. will no doubt be debated in subsequent publications if the lower mortality rate is borne out in other epidemiological studies. In any case, the morbidity of survivors with severe neurodevelopmental abnormalities and epilepsy remains a major problem.

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