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Continuous Electroencephalography in Pediatric Traumatic Brain Injury: Seizure Characteristics and Outcomes

Jarin Vaewpanich, MD[1] **[Instructor, Pediatric Critical Care Medicine]** and **Karin Reuter-Rice, PhD, CPNP-AC, FCCM, FAAN**[2,3,4] **[Associate Professor]**

[1]Department of Pediatrics, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand, 270 Rama VI Rd, Thung Phaya Thai, Ratchathewi, Bangkok, Thailand 10400, jarivaew@yahoo.com, +6681-170-6180, (Former Pediatric Intensive Care Fellow, Duke University Children's Hospital, 2013-2015)

[2,3,4]Duke University, School of Nursing, School of Medicine, Dept. of Pediatrics, Duke Institute for Brain Sciences, 307 Trent Drive, DUMC 3322, Durham, NC 27710, karin.reuter-rice@duke.edu, 919-681-7647

Abstract

Background—Traumatic injury (TBI) is a major cause of pediatric morbidity and mortality. Secondary injury that occurs as a result of a direct impact plays a crucial role in patient prognosis. The guidelines for the management of severe TBI target treatment of secondary injury. Posttraumatic seizure, one of the secondary injury sequelae, contributes to further damage to the injured brain. Continuous electroencephalography (cEEG) helps detect both clinical and subclinical seizure, which aids early detection and prompt treatment.

Objective—To examine the relationship between cEEG findings in pediatric traumatic brain injury and neurocognitive/functional outcomes.

Methods—This study focuses on a subgroup of a larger prospective parent study that examined children admitted to a level-1 trauma hospital. The subgroup included sixteen children admitted to the pediatric intensive care unit (PICU) who received cEEG monitoring. Characteristics included demographics, cEEG reports, antiseizure medication. We also examined outcome scores at time of discharge and 4–6 weeks post-discharge using the Glasgow Outcome Scale-Extended Pediatrics and a center-based speech pathology neurocognitive/functional evaluation scores.

Results—Sixteen patients were included in this study. Patients with severe TBI made up the majority of those that received cEEG monitoring. Non-accidental trauma was the most frequent TBI etiology (75%) and subdural hematoma was the most common lesion diagnosed by CT scan (75%). Fifteen patients received anti-seizure medication and levetiracetam was the medication of choice. Four patients (25%) developed seizures during PICU admission and 3 patients had subclinical seizures that were detected by cEEG. One of these patients also had both a clinical and

Correspondence to: Karin Reuter-Rice.

subclinical seizure. Non-accidental trauma was an etiology of TBI in all seizure patients. Characteristics of a non-reactive pattern, severe/burst suppression and lack of sleep architecture, on cEEG were associated with poor neurocognitive/functional outcome.

Conclusion—Continuous electroencephalography demonstrated a pattern that associated seizures and poor outcomes in patients with moderate to severe traumatic brain injury, particularly in a subgroup of non-accidental trauma patients. Best practice should include institutional based TBI cEEG protocols, which may detect early of seizure activity and promote outcomes. Future studies should include examination of individual cEEG characteristics to help improve outcomes in pediatric TBI.

1. Introduction

Traumatic brain injury (TBI) is a major cause of morbidity and mortality in the United States.[1] An average of 53,014 deaths occur as a result of a TBI annually.[1] In children and adolescents, TBI is one of the leading causes of mortality and morbidity, with 475,000 cases per year occurring in children younger than 14 years of age.[2] Pediatric TBI is responsible for more than $1 billion in TBI-associated hospitalizations costs[3] and multidisciplinary care costs of $60.4 billion over their lifetime.[4]

Traumatic brain injury occurs in two phases. First is the primary injury phase, which occurs immediately after mechanical forces directly impact the brain tissue, causing parenchymal and vascular damage. This phase is followed by a secondary phase, which is associated with failure of auto-regulation leading to cerebral ischemia, an increase in brain metabolism, posttraumatic cerebral ischemia, release of excitatory neurotransmitters, and cellular energy failure.[5] Additionally, secondary injury can be exacerbated by difficulty regulating external insults such as hypoxia, hypotension and hyperthermia that lead to further damage. The secondary injury phase has a significant impact on mortality and morbidity after TBI.[6–7]

Predictive factors that influence the outcome of pediatric TBI are related to the severity of primary brain injury and the occurrence of secondary insults.[8–9] Glasgow Coma Scale (GCS) at admission, evidence of hypotension and hypoxia, maintenance of cerebral perfusion pressure and decreasing cerebral metabolism by preventing the development of posttraumatic seizure (PTS) require vigilance and aggressive management for favorable pediatric TBI outcomes.[10–12]

The guidelines for the management of severe TBI focus on how to prevent and treat secondary injury to avoid further brain damage.[13] Aggressive treatment of increased intracranial pressure, hypotension, hypoxia, hypercarbia, and fever are major concerns in the guidelines.[13] One other therapy that has significant impact to the outcome of pediatric TBI is antiseizure prophylaxis. There are insufficient data in pediatric TBI to consider antiseizure prophylaxis as a level I recommendation.[13] However early detection and treatment of a PTS has a tremendous effect on the mortality and morbidity rate in pediatric TBI.[14–15]

A posttraumatic seizure is classified as either early or late. Early PTS occurs in first 7 days after injury, whereas late PTS develops after 7 days of injury. Studies examining early PTS in pediatric TBI report increased mortality rates and worse neurodevelopmental

outcomes.[16–18] Early PTS has been reported in up to 42.5% of pediatric TBI with possible mechanisms associated with increased intracranial pressure and increased cerebral metabolism.[19] However, diagnosis of PTS is challenging in a patient who does not exhibit symptoms of clinical seizures. Subclinical, as well as clinical, seizures have shown to increase secondary injury.[19–21] Arndt and his team reported 16.1% of pediatric TBI patients had subclinical or electrographic seizures and that they contributed to poor functional outcome with an increased length of hospital stay.[18] Therefore, early detection and judicious management of clinical and subclinical PTS can lead to decreased morbidity and mortality in pediatrics TBI. Continuous electroencephalography (cEEG) is considered the best diagnostic study for all seizure diagnosis in TBI.[22] Continuous EEG also provides additional information in the prognosis of TBI patients.[23]

Diagnosis of early PTS requires both clinical observation for overt seizure activity and cEEG monitoring to detect subclinical seizures, particularly for those patients with severe TBI, and who are comatose, as they have increased risk of early PTS occurrence.[24] Continuous EEG is also the most sensitive bedside diagnostic study used to diagnose subclinical seizures, and it is recommended for all TBI patients with a Glasgow Coma Score (GCS) lower than 8 and an unexplained or persistent altered consciousness.[25] This monitoring modality has been used to improve seizure detection and guide therapy. One study of pediatric patients with acute encephalopathy and TBI found that cEEG led to management changes that included starting or escalating antiseizure medication to terminate seizure activity.[26] Furthermore, the detection of background reactivity on cEEG, present in the sleep stage, has been associated with poor outcomes in TBI patients.[27–28]

Because PTS impacts patient's functional outcomes and mortality, cEEG has been used with increasing frequency in both pediatric and adult TBI patients for early detection and therapy. This study focuses on describing cEEG findings consistent with PTS, which may be associated with poor outcomes in pediatric TBI. We also identify individual characteristics associated with developing early PTS. By identifying associations of PTS in pediatric TBI we can consider changes in management strategies to improve outcomes.

2. MATERIALS AND METHODS

2.1 Population and Setting

This study is a subgroup analysis of 16 children who were part of a larger single-center, prospective, longitudinal observational parent study of children (n=60) admitted to a level-1 trauma hospital for a TBI during the months of December 2012 to July 2015. The parent study included previously healthy children ages 10 days to 15 years admitted with Glasgow Coma Scale (GCS) scores of mild (13–15), moderate (9–12) and severe (3–8) at time of study enrollment.[29] The subgroup included only those TBI patients that were admitted to the PICU and had cEEG monitoring. An injury severity score (ISS) was calculated based on injured body region.[30] Patients received standard-of-care TBI management. The study had institutional review board approval by the hospital and informed consent was obtained from the parent or legal guardian of all participants.

2.2 Data collection

Data was collected by accessing the patients' electronic health records (EHR) and the parent study's REDCap database. Demographic data included age, sex, injury severity score (ISS), and type and mechanism of injury. The GCS was recorded in the Emergency Department and during study participation in the PICU. Daily physiologic variables, such as vital signs, laboratory and diagnostic studies, seizure activity, and antiseizure medication were recorded. Also collected at time of discharge from hospital and upon follow-up (4–6 weeks post hospitalization) were several outcome measures.

2.3 Continuous Electroencephalography (cEEG) data

We collected information about all seizure events observed in the field, in the emergency department, and prior to, and after start of cEEG in the PICU. The decision for starting cEEG monitoring was dependent on the attending physician's judgment. The attending physician would order a cEEG based on history and clinical presentation such as, clinical seizure, severity of injury, abnormal vital signs or abnormal GCS. Continuous EEG monitoring then followed institutional protocol using a standard international 10/20 system placement of scalp electrodes. The duration of monitoring depended on the identification of seizure activity and the healthcare team's clinical decision to continue monitoring. We collected cEEG data immediately after start of cEEG and recordings were interpreted by a pediatric epilepsy fellow and board certified pediatric electrographer.

2.4 Seizure classification

Seizures were classified as clinical seizures, subclinical seizures, status epilepticus and subclinical status epilepticus. Subclinical seizures were identified by rhythmic sharp/spike waves from cEEG that lasted longer than 10 seconds without clinical correlation. Status epilepticus was defined by an ongoing seizure for more than 30 minutes.[31]

2.5 Outcome measures

We examined outcome measures such as the speech pathology neurocognitive/functional evaluations (SPNFE) and the Glasgow Outcome Scale-Extended, Pediatric (GOS-E Peds) to examine if a relationship existed between the patient's outcome at time of discharge and on their first follow-up visit (approximately 4–6 weeks post discharge).

The pediatric speech pathology neurocognitive/functional evaluation (SPNFE) is a site-specific evaluation. The SPNFE is derived from multiple standard measures used by speech pathologists in pediatric assessments. These cognitive and functional tests give raw and standardized scores that are compared to normative data. The SPNFE is performed either upon admission, or when a child is determined medically stable to evaluate, with all admitted pediatric patients with TBI. The speech pathologists are trained to regularly evaluate TBI patients and their evaluation is adjusted based upon the age of the patient and determines the plan for necessary care. The neurocognitive/functional categories include: 1) cognitive-communication; 2) auditory; 3) visual; 4) motor; 5) speech integrity; and 6) feeding. These categories address the developmental neurocognitive/functional abilities of the patient at the time of evaluation. Therefore, depending on the TBI severity, multiple evaluations may occur during time of hospitalization.

The SPNFE is also performed upon follow-up after discharge from the hospital for those patients who require outpatient follow-up therapies. The discharge and follow-up evaluations address level of delay, if one exists. The levels are documented as no delay, mild, moderate or severe delay. For the purposes of this study, those levels were assigned a numeric ordinal scale, which included: 0 = no delay, 1 = mild delay, 2 = moderate delay and 3 = severe delay. These numeric values allowed for the research team to compare delay over two outcome time points: discharge from hospital, and at follow-up.

The GOS-E Peds scale categorizes traumatic brain injuries based on the injured child's outcome using an 8-point numerical scale.[32] This scale was adapted from the adult version (Glasgow Outcome Scale-Extended [GOS-E]) that does not account for the differences in developmental activities specific to children under the age of 17 years. The scale ranges from 1 to 8 (Upper Good Recovery to Death) and is ranked as followed: 1 - Upper good recovery, 2 -Lower good recovery, 3 - Upper moderate disability, 4 - Lower moderate disability, 5 - Upper severe disability, 6 - Lower severe disability, 7 - Vegetative state, and 8 – death. The outcome score is determined using a questionnaire that accounts for the child's independence in and outside the home, ability to function in a school environment, participation in leisure and social activities, and maintenance of healthy friendships and relationships.[32] The questionnaire may be satisfied by telephone interview, by in-person assessment, or by data extraction from the patient's EHR.[32] The GOS-E Peds is used in research to quantify a patient's recovery, and is not specifically used in the clinical management of a patient.[28]

3. Results

The analysis was conducted using Microsoft Excel 2007as follows. First, the general characteristics of demographic and injury severity were analyzed using descriptive statistics; categorical variables were expressed as numbers and percentages; and continuous variables as means ± SDs. A total of 16 patients were included in this study (Table 1). The study had an equal number of male and female patients with an average age of 3.1 years. Two thirds of the patients met the classification of severe TBI. There were 5 patients with mild TBI who received cEEG monitoring due to clinical suspicions of seizure during their admission. The mean ISS was 27.6 (± 9). Etiology of injury was predominantly (75%) non-accidental trauma (NAT). Patients also had a 56% reported incidence of pre-hospital seizures and 25% experienced pre-hospital cardiac arrest. The most common initial CT findings were subdural hematoma (75%), followed by skull fracture (37.5%). Two patients died as a result of their brain injury while on study.

Table 2 reviews cEEG findings and outcome in TBI patients who had seizures. Seizures occurred in four patients (25%). The mean onset of cEEG monitoring was 0.5 days and 1.42 days from admission in the seizure and non-seizure groups respectively. The mean duration of cEEG monitoring in patients with seizures was 143.91 hours, which was longer than the non-seizure group. Initial cEEG findings in patients with seizures indicated an absent or abnormal sleep architecture (75%), nonreactive (50%), and burst suppression (50%). The four patients with severe TBI and seizure activity demonstrated poor outcome in both the GOS-E Peds and the SPNFE scores at time of discharge.

Table 3 demonstrates seizure characteristics and treatment. Fifteen patients (93%) received antiseizure medication within the first day of admission. First line medication for seizure prophylaxis was levetiracetam and was used in all patients who received antiseizure medication. Other antiseizure medications included phenytoin, phenobarbital, pentobarbital and benzodiazepine. In the four patients who had a documented prehospital seizure each received one dose of lorazepam, a benzodiazepine, by emergency medical responders prior to hospital admission. Once admitted, four severe TBI patients were identified to have seizures. There were 2 patients who developed status epilepticus, with one requiring aggressive seizure management prior to cEEG monitoring. In addition to levetiracetam, phenobarbital, phenytoin, pentobarbital and benzodiazepine were used in both patients that had status epilepticus. Three patients experienced subclinical seizures detected by cEEG. Of those three, one was found to have both clinical and subclinical seizures, whereas the other two only had subclinical seizure activity. The two patients with subclinical seizures were recorded within 24 hours of admission and sporadic subclinical seizure activity was seen in both patients up to 72 hours after the first seizure.

We also examined individual patients to better describe their individual clinical characteristics in association with cEEG finding and outcome (Table 4). There were 8 patients who had cEEG monitoring for more than 1 day. Within this group the cEEG findings were categorized into initial (results in 24 hours after established cEEG), subsequent (results day after established cEEG) and last cEEG report. Initial cEEG report findings showed a nonreactive EEG pattern in 3 patients (19%). Of those three patients, one patient's cEEG pattern was read as active on the final cEEG report. This patient's GOS-E Peds score reflected a 4 at discharge and a score of 2 at follow-up. Congruently, the SPNFE score also showed a change from 2 at discharge to 1 upon follow-up. In the non-reactivity pattern group, patients (n=2) demonstrated a non-reactivity pattern throughout cEEG monitoring and showed severe neurological disability (GOS-E Peds score >5) upon discharge. Five children had severe depressed or burst suppression on the background cEEG. All of them had unfavorable outcomes including death and severe disability on GOS-E Peds and SPNFE. Continuous EEG demonstrated absent sleep architecture in 5 patients and abnormal sleep architecture in 2 patients. Patients with sleep architecture disturbance had high GOS-E Peds and SPNFE scores after discharge and upon follow-up. With the exception of one patient, sleep architecture returned to normal on last cEEG report.

4. Discussion

Traumatic brain injury has significant impact to pediatric neurological development and PTS aggravated the secondary injury, causing further injury. Pediatric TBI patients with PTS had severe neurocognitive and functional impairment at discharge and at follow up. Although a small sample size, the incidence of PTS was 25% in our study, which is higher than previously report.[33]

Non-accidental trauma, also known as abusive head trauma, was a frequent etiology of TBI in our study. The NAT patients were of a younger age (< 1 year) than patients who were admitted for an accidental TBI, which is consistent with the literature.[34] Radiologic finding in our study found a higher incidence of subdural hematoma injury in NAT patients, which is

also consistent with studies that report similar findings.[35] Of the four patients who developed seizures during PICU admission, all were diagnosed with NAT. These patients were younger in age (< 1 year) and had poorer outcomes without improvement upon discharge. Three patients with NAT also had had subclinical seizures, which is consistent with a previous study that found NAT children had higher incidence of electrographic seizure as well as a higher morbidity compared to accidental TBI patients.[36–38] Therefore, clinical observation of seizures alone in severe NAT TBI patients is not sensitive enough to appreciate seizure activity. However this study demonstrated that subclinical seizures can occur within 24 hours of admission. In non-seizure group cEEG monitoring was typically started a day later than seizure group. Therefore, the possibility exists that the patients who received cEEG monitoring a day later could have had subclinical seizures that were missed. In this patient population the use of cEEG monitoring for seizure detection would be highly recommended and should be started within 24 hours of admission.

Levetiracetam was prescribed in all patients who received antiseizure medication. Previous studies reported the efficacy of levetiracetam was equal to phenytoin for seizure prophylaxis after brain injury.[39] However, phenytoin and phenobarbital require blood sampling to monitor for therapeutic medication level, thereby supporting the use of levetiracetam for PTS prophylaxis.[40] In our study, there were 7 patients who did not have prehospital seizures but still received levetiracetam for seizure prophylaxis. One patient developed a PTS while on the medication. The use of levetiracetam for seizure prophylaxis was consistent with a recent report that found 17% of pediatric traumatic brain injury patients who were on levetiracetam prophylaxis developed seizures.[32] This suggests that cEEG monitoring plays a crucial role while on antiseizure prophylaxis.

There were several cEEG characteristics that appeared to correlate with outcome in pediatric TBI. Ramachandrannair and his team found that a reactive encephalographic pattern could determine neurological outcome and mortality in both adult and pediatric TBI patients.[41] In their study, a nonreactive EEG pattern was associated with poor neurological outcome especially if there was no recovery of the EEG pattern throughout the period of EEG recording. Effects from sedation and antiseizure medication can be interpreted as a nonreactive electrographic pattern in the initial cEEG. However, after stabilization, the subsequent cEEG activity can assist with determining the actual patient's brain wave activity. In addition, when burst suppression was seen on the cEEG, it was associated with poor outcomes in anoxic brain injury.[42] The presence of burst suppression was associated with poor outcome and mortality in pediatric TBI patients.[42] The presence of sleep architecture on cEEG has also been reported as a predictor of clinical outcome in pediatric patients.[43] In our study we found that the persistent abnormal or absence of sleep architecture during cEEG monitoring in pediatric TBI was associated with poor neurological outcome.

In this subgroup the timing and initiation of cEEG monitoring varied between patients. Our results demonstrated that patients with moderate to severe TBI tended to develop seizure within 24 hours of admission and the majority of seizures were subclinical. This data argues the point that cEEG monitoring should be initiated as soon as possible to support the early detection of seizure activity. The initiation of cEEG monitoring within the first 24 hours

after injury is also consistent with the American Clinical Neurophysiology Society's recommendation.[44] Moreover in pediatric TBI, continuation of cEEG monitoring to detect subsequent seizures promotes best practice and can promote neurological outcomes

4.1 Study Limitations

This single center study with a small sample size allowed us to easily collect continuous data in a group of patients who had a standard approach to TBI care. This study focused on patients who had cEEG and who were at high risk of seizure, such as patients with moderate to severe TBI and/or history of prehospital injury-related seizure. Although this may explain the incidence of PTS in our sample of children who experienced a severe TBI, it aligns with the literature. These results might not be generalizable, but they provide insight and support for the use of cEEG as a diagnostic study to identify subclinical seizures and direct seizure management of pediatric traumatic brain injury. Although outcomes where limited to 6 weeks, we appreciate that longer-term outcome evaluations would be beneficial and should be consider in future work.

5. Conclusion

Continuous EEG monitoring demonstrated a pattern that associated seizures and poor outcomes in pediatric patients with moderate to severe traumatic brain injury, particularly in a subgroup of NAT patients. Best practice should include the consistent use of cEEG in TBI patients such as preset institutional protocols that could potentially prevent missed seizure activity. Future examination of individual cEEG characteristics in a large sample size would help determine other best practices and outcomes in pediatric traumatic brain injury.

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Highlights

- Posttraumatic seizure was related to poor neurocognitive and functional outcomes.
- Non-accidental trauma/abusive head trauma had a higher rate of subclinical seizures.
- Patterns in continuous electroencephalography provided insight into detecting subclinical seizures and patient outcomes.

Table 1

Patient Characteristics (N=16)

Characteristics	Results
Race, n (%)	
White/Caucasian	6 (37.5)
Black/African American	8 (50)
Other	2 (12.5)
Gender, n (%)	
Male	8 (50)
Female	8 (50)
Age (year), mean (SD)	3.1 (5)
GCS injury severity, n (%)	
Mild	5 (31.3)
Moderate	0 (0)
Severe	11 (68.7)
Injury Severity Scores, mean (SD)	27.6 (9)
Etiology (n, %)	
- Non-accidental trauma (NAT)- abusive	12 (75)
- Motor vehicle collision	2 (12.5)
- Other	2 (12.5)
Prehospital seizure, n (%)	9 (56)
Prehospital cardiac arrest, n (%)	4 (25)
CT scan findings, n (%)*	
Subdural hematoma	12 (75)
Epidural hematoma	2 (12.5)
Intraparenchymal hemorrhage	1 (6.3)
Intraventricular hemorrhage	1 (6.3)
Subarachnoid hemorrhage	4 (25)
Diffuse axonal injury	2 (12.5)
Skull fracture	6 (37.5)
PICU length of stay, mean (SD)	9.9 (10)
Hospital length of stay, mean (SD)	23.8 (26.6)
Death, n (%)	2(12.5)

Emergency Department (ED); Glasgow Coma Scale (GCS); Computed Tomography Scan (CT scan)

*Most patients had more than one finding on CT scan

Table 2

Continuous Electroencephalography Results in No-Seizure and Seizure Groups and Outcome Scores

Characteristics	Results	
	No seizures (n=12)	Seizures (n=4)
Onset of cEEG (day), mean (SD)	1.42 (0.9)	0.5 (0.57)
Duration of cEEG (hr), mean (SD)	48.75 (37.38)	143.91 (33.79)
Initial EEG finding, n (%)		
Nonreactive	1 (8)	2 (50)
Burst suppression	2 (17)	2 (50)
Absent or abnormal sleep architecture	4 (33)	3 (75)
SPNFE at discharge, (n=14)		
No impairment	4 (33)	0
Mild	1 (8)	0
Moderate	3 (25)	0
Severe	2 (16)	4 (100%)
GOS-E Peds at discharge, (n=16)		
Good recovery	4 (33)	0
Moderate disabled	3 (25)	0
Severe disabled	3 (25)	4 (100)
Death	2 (17)	0

Continuous electroencephalography (cEEG); Glasgow Coma Scale (GCS); Glasgow Outcome Scale-Extended, Pediatrics (GOS-E Peds); Speech Pathology Neurocognitive/Functional Evaluation (SPNFE)

Table 3

Seizure Characteristics and Antiseizure Medication

Seizure Characteristics	**Results (n=16)**
Seizure, n (%)	4 (36.4)
Seizure type, n = 4	
clinical	1 (25)
subclinical	2 (50)
clinical + subclinical seizure	1 (25)
Status epilepticus	2 (50)
Patients received antiseizure medication, n (%)	15 (93%)
Onset day 1 of antiseizure medication[b]	15/15
Type of antiseizure medication[c], n (%)	
- Phenobarbital	3 (19)
- Phenytoin	4 (25)
- Pentobarbital	3 (19)
- Levetiracetam	15 (100)
- Benzodiazepine	3 (19)

[a,b] Day 0 indicate the day of injury;

[c] Some participants had more than one antiseizure medication;

[d] Indicates GCS at the beginning of EEG

Table 4

Individual Patient Characteristics, cEEG Finding and Outcomes (n=16)

Pt.	Age (yrs)	GCS	CT findings	Initial EEG				Subsequent EEG				Last EEG				GOS-EPED d/c, f/u score	SPNFE d/c, f/u score
				Reactivity	Severe/ Burst suppression	Sleep architecture	Seizure	Reactivity	Severe, Burst suppression	Sleep architecture	Seizure	Reactivity	Severe/ Burst Suppression	Sleep architecture	Seizure		
1.	4	5	SDH	Non-reactive	No	Abnormal	No	Non-reactive	No	Normal	No	Reactive	No	Normal	No	4, 2	2, 1
2	0.25	14	SDH	Reactive	No	Normal	No	NA	NA	NA	NA	NA	NA	NA	NA	2, 2	0,1
3	0.33	14	SDH, SAH	Reactive	No	Normal	No	NA	NA	NA	NA	NA	NA	NA	NA	1, na	0, na
4	1.33	15	SDH, Skull fracture	Reactive	No	Normal	No	NA	NA	NA	NA	NA	NA	NA	NA	1, 1	0, 0
5	0.17	6	SDH, infarction	Reactive	No	Normal	Yes	Reactive	No	Absent	Yes	Reactive	No	Normal	No	6, 6	3, 3
6	0.25	3	DCI, infarction	Non-reactive	Yes	Absent	No	Non-reactive	Yes	Absent	No	Non-reactive	No	Absent	No	5, na	3, na
7	1.33	15	SDH	Reactive	No	Normal	No	NA	NA	NA	NA	NA	NA	NA	NA	3, 1	0, 0
8	0.17	3	SDH, DCI, Skull fracture	Non-reactive	Yes	Absent	Yes	Non-reactive	No	Absent	Yes	Non-reactive	No	Absent	No	6, 6	3, 3
9	0.58	15	SDH	Reactive	No	Awake	No	NA	NA	NA	NA	NA	NA	NA	NA	2, 1	1, 0
10	15	4	EDH, ICH, DAI Skull fracture	Reactive	Yes	Absent	No	Reactive	yes	Absent	No	Non-reactive	Yes	Absent	No	8, na	na, na
11	0.17	3	SDH, SAH, DAI	Reactive	Yes	Absent	No	NA	NA	NA	NA	NA	NA	NA	NA	8, na	na, na
12	9.83	3	IVH, SAH, Contusion	Reactive	Yes	Absent	No	Non-reactive	Yes	Absent	No	Reactive	No	Absent	No	6, na	3, na
13	0.25	6	SDH	Reactive	No	Abnormal	No	Reactive	No	Abnormal	Yes	Reactive	No	Abnormal	No	6, 5	3, 2
14	0.67	7	SDH, Skull fracture	Reactive	No	Normal	No	NA	NA	NA	NA	NA	NA	NA	NA	6, 6	2, 2

Pt.	Age (yrs)	GCS	CT findings	Initial EEG				Subsequent EEG				Last EEG				GOS-EPED d/c, f/u score	SPNFE d/c, f/u score
				Reactivity	Severe/ Burst suppression	Sleep architecture	Seizure	Reactivity	Severe, Burst suppression	Sleep architecture	Seizure	Reactivity	Severe/ Burst Suppression	Sleep architecture	Seizure		
15	1.17	3	EDH, SAH, Infarction, Contusion	Reactive	No	Normal	No	Reactive	No	Normal	No	Reactive	No	Abnormal	No	6, 6	3, 1
16	2	6	SDH,SAH, Skull fracture Contusion	Reactive	No	Normal	No	NA	NA	NA	NA	NA	NA	NA	NA	3, 2	2, 0

Discharge from hospital (D/C); Follow-up visit (F/U); Glasgow Outcome Scale-Extended, Pediatrics (GOS-E Peds); Not performed or no data (NA); Speech Pathology Neurocognitive/Functional Evaluation (SPNFE)