



Continuous EEG Monitoring in Critically Ill Children and Prognostic Factors for Short-term Outcome: An Observational Study

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ABSTRACT

Aim: To evaluate the association of etiology, continuous electroencephalography (cEEG) findings and neuroimaging findings with short-term outcomes for patients admitted to a pediatric intensive care unit (PICU) for acute encephalopathy.

Materials and Methods: A total of 24 children admitted to a PICU for acute encephalopathy were enrolled into this study. The etiology, treatment, duration of stay in the PICU, their demographic information and their past medical history were recorded. cEEG was initiated as quickly as possible following admission to the PICU and continued for at least 24 hours. Their short-term prognosis was evaluated by the Pediatric Cerebral Performance Category score (PCPC) at PICU discharge.

Results: The most common cause was traumatic brain injury comprising 25% (n=6) of all cases. Other common causes were asphyxia (hanging, foreign body aspiration, drowning) (n=4, 16.67%) and intoxication (n=3, 12.5%). Twenty-two patients underwent cranial imaging. The most common findings in CT were hemorrhage (n=6, 30%) and ischemia/edema (n=6, 30%). Fourteen patients had unfavorable PCPC outcome scores. There was a tendency for poorer outcomes in those patients with hemorrhage/fracture or ischemia/edema in the imaging and for those patients who needed either pre-hospital CPR or had non-convulsive seizures but without statistical significance.

Conclusion: cEEG in critically ill children is useful for detecting both epileptic and non-epileptic events. The use of cEEG in PICUs can be helpful for the better management of cases.

Keywords: Acute encephalopathy, children, continuous EEG monitoring, intensive care, prognosis

Introduction

Acute encephalopathy is responsible for 2-3% of pediatric emergency department visits and 1-11% of pediatric intensive care unit (PICU) admissions (1,2). Acute encephalopathy is an important cause of morbidity and mortality. Prompt

and proper evaluation and treatment are of the utmost importance. Even though it has a low sensitivity for defining the underlying etiology, electroencephalography (EEG) is a valuable tool in determining the extent of cerebral injury and long-term prognosis.

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Non-convulsive seizures (NCS) and non-convulsive status epilepticus (NCSE) are common in critically ill children, occurring in 10-50% of patients with acute encephalopathy (3). Pediatric patients may be at higher risk of NCS when compared to adults due to lower seizure thresholds, limited communication, and various behavioral disturbances, making the diagnosis of NCS more challenging and suggesting the need for closer monitoring in this population (4). Additionally, there is increasing data that electrographic seizures and electrographic status epilepticus are associated with worse outcomes (3,5).

Continuous EEG (cEEG) monitoring can lead to changes in the clinical management of patients in PICUs, including the initiation, escalation or discontinuation of anti-seizure drugs or urgent neuroimaging (6).

The aim of this study was to evaluate the association of factors such as etiology, cEEG findings and neuroimaging findings with the short-term outcome of those patients admitted to a PICU with acute encephalopathy.

Materials and Methods

Twenty-four patients admitted to Ege University Faculty of Medicine, PICU due to acute encephalopathy with a Glasgow Coma Scale score of eight or less, between 01.05.2018 and 01.10.2018 were included in this study prospectively. Their etiology, treatment, duration of stay in the PICU, demographic information, neuroimaging results and past medical history were recorded. cEEG was initiated as quickly as possible following admission to the PICU and continued for at least 24 hours. All recordings were carried out as outlined by the international 10-20 system with an electrode cap. Electrodes were applied using a conductive paste. All acquisitions were performed by the same technician using the same device (Brain quick clinical EEG line Micromed s.p.a. Italy). cEEG evaluation was made in accordance with the American Clinical Neurophysiology Society's (ACNS) Standardized Critical Care EEG Terminology (7).

EEG traces were evaluated in terms of background activity (predominant frequency, symmetry, voltage, continuity), and the presence of stage II sleep transients (K-complexes and spindles), periodic discharges (PDs), rhythmic delta activity (RDA) and interictal discharges. Electrographic seizures are defined as abnormal, paroxysmal electroencephalographic events that differ from the background activity, last longer than ten seconds (unless associated with clinical signs), have a plausible electrographic field, an evolution in frequency, morphology

and spatial distribution. Electrographic status epilepticus is defined as uninterrupted electrographic seizures lasting 30 minutes or longer, or repeated electrographic seizures totaling more than 30 minutes in any one-hour period (8).

cEEG recordings were reviewed every eight to twelve hours by a child neurologist. In case where frequent NCS were identified, more frequent interpretations were provided until seizures were controlled. If clinical events were recorded, cEEG was interpreted as soon as possible whether that event was ictal or non-ictal. In all cases, cEEGs were recorded for at least 24 hours. In case of convulsive/ NCS or abnormal background activity (discontinuous or burst suppression), cEEG monitoring was extended to 72 hours. cEEG recording was continued for at least six hours following the last seizure. The first hour of the cEEG (cEEG 1 h) and rest of the recording (cEEG 24 h) were evaluated separately.

Clinical and electrographic seizures were treated with intravenous midazolam or levetiracetam.

EEGs were evaluated by six independent readers (three senior child neurologists, two junior child neurologists, one pediatric resident) in accordance with the ACNS Standardized Critical Care EEG Terminology (7). Interrater agreement was assessed as a percentage of perfect agreement and Fleiss' kappa among the six reviewers (9).

The Pediatric Cerebral Performance Category score (PCPC) at PICU discharge was determined in every case. The PCPC is a validated six-point scale categorizing degrees of functional impairment. PCPC categories are: 1= normal, 2= mild disability, 3= moderate disability, 4= severe disability, 5= coma and vegetative state, and 6= death (10). PCPC scores of 3-6 (moderate/severe disability, vegetative, death) were accepted as unfavorable short-term outcomes.

Statistical Analysis

Data analysis was performed in Ege University Faculty of Medicine Department of Biostatistics. The SPSS (Statistical Package for Social Sciences) for Windows 25.0 package program was used for statistical analysis. Numerical variables are presented as arithmetic mean \pm standard deviation or median (minimum-maximum); categorical variables are presented as summary statistics as numbers and percentages. The normality of the data in numerical variables was tested with the Shapiro-Wilk test. For numerical variables without normal distribution, Wilcoxon analysis was used in paired groups. In case of materiality, binary comparisons were made with Dunn's test. For categorical data, the comparison between groups was made

by creating cross tables using chi-square analysis. All data analysis was performed at the 0.05 significance level.

This study was reviewed and approved by Ege University Faculty of Medicine Ethics Committee (date: 08.05.2018, approval no: 18-5/49). The purpose of this study was explained to the legal guardian of every patient. Written informed voluntary consent was obtained from all individual participants included in this study. This study adhered to the ethical guidelines and was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki.

Results

Patient Characteristics & cEEG Findings

Of the 24 patients included in this study, 14 were female (58%) and 10 were male (42%). The mean age of the patients was 92.24 ± 69.5 months (4-216 months).

The average time to reach an emergency setting was 142.5 ± 93.3 minutes (40-270 minutes). The average time to start cEEG acquisition after PICU admission was 124.8 ± 61.07 minutes (30-240 minutes). The mean cEEG monitoring duration was $1,620 \pm 907.01$ minutes (1,080-4,320 minutes). cEEG recordings lasted 24 hours for 21 patients. In two patients with a burst suppression pattern (BSP) and NCS (case 7 and case 21) and one patient with convulsive and NCS (case 23) cEEG monitoring was extended to 72 hours. All the patients were orotracheally intubated and mechanically ventilated during the monitoring period. The length of stay in the PICU was between two and 46 days (mean 13.75 ± 11.6 days).

Cardiopulmonary resuscitation (CPR) was applied in four cases before admission to the hospital. Four patients presented with seizures before admission to the PICU. Only one patient (case 4) had a seizure during the first hour of cEEG monitoring, which lasted for two minutes. The patient had three more focal onset myoclonic seizures lasting 1-2 minutes in cEEG 24 h. The seizures were successfully treated with intravenous midazolam.

The most common cause for admission was traumatic brain injury (TBI) comprising 25% of all cases (n=6). Other common causes were asphyxia (hanging, foreign body aspiration, drowning) (n=4, 16.67%) and intoxication (n=3, 12.5%). The etiology and clinical information regarding the cases are presented in Table I.

Twenty patients underwent cranial computed tomography (CT). Cranial magnetic resonance imaging (MRI) was also obtained in 10 out of the 20 cases with CT

scan. Two patients were evaluated with MRI alone and no neuroimaging was possible in two patients. The most common findings in CT were hemorrhage (n=6, 30%) and ischemia/edema (n=6, 30%). Hemorrhage (33.33%), ischemia/edema (25%), diffuse axonal injury (16.67%) and T2 hyperintensities with restricted diffusion (16.67%) were the common findings on cranial MRI. The demographic, radiological and cEEG findings are presented in Table I.

The cEEG data of the 24 patients included in this study were analyzed in two different categories: The first hour of monitoring (cEEG 1 h) and after the first hour (cEEG 24 h). The cEEG 1 h and 24 h findings are presented in Table II.

Slow background activity (theta and/or delta) was observed in 15 cases (63%) in cEEG 1 h and 17 out of 24 cases (70%) in cEEG 24 h. The rate of slow background activity during the first hour and after the first hour recordings were similar (63% vs. 70%) (p=0.54).

NCSs were detected in two patients (8%) within the first hour of monitoring. Case 21 with suspected metabolic encephalopathy had an NCS lasting 50 seconds. Case 23 who also had a myoclonic seizure had an NCS lasting 60 seconds in the first hour of cEEG. Both seizures terminated spontaneously. Four patients had NCSs in cEEG 24 h. Two of these (case 21 and case 23) also had NCSs in the first hour of recording. Case 23 had four NCSs in cEEG 24 h, none of which were longer than two minutes. Case 21 had one NCS during the recording after the first hour. Case 7, admitted for drowning, had one NCS during cEEG 24 h. Case 17, admitted for head trauma, had three NCSs in cEEG 24 h lasting 1-2 minutes. None of the patients with NCS received paralytic medication infusion. When all of the patients were evaluated, two (18%) of all the NCSs were detected in cEEG 1 h, while 9 (82%) were detected in cEEG 24 h.

Two out of four patients with NCS were less than six months of age, one was eight years old and the other was 11 years old. NCS was not observed in the patient with a previous epilepsy diagnosis. Two of the four patients with NCS were admitted for head trauma, one for asphyxia and one for metabolic disease.

NCSE was not detected in any of the patients.

Short-term Outcome (PCPC)

In terms of PCPC scores at PICU discharge, 10 cases had favorable short-term outcomes (score ≤ 2) and 14 cases had unfavorable outcomes (score ≥ 3). One patient (case 7) was lost on the 13th day of his admission to the PICU. This patient had a history of CPR for 20 minutes after drowning.

Patient	Age (months)	Etiology	Imaging finding	EEG finding (0-1 hour)	EEG finding (2-24 hours)	Prognosis (PCPC score)
1	147	Asphyxia (hanging)	Ischemia/edema (CT)	*	RDA IED	Unfavorable (4)
2	49	Hepatic encephalopathy	Not performed	TWs RDA	*	Favorable (2)
3	22	TBI	Hemorrhage (CT) Hemorrhage, DAI (MRI)	IED	RDA IED	Unfavorable (3)
4	166	Epilepsy	Normal (CT, MRI)	IED	*	Favorable (2)
5	98	TBI	Hemorrhage (CT) Hemorrhage, DAI (MRI)	Amplitude asymmetry (mild)	Amplitude asymmetry (mild)	Favorable (2)
6	125	Intoxication (butane abuse)	Ischemia/edema (CT, MRI)	*	*	Unfavorable (4)
7	98	Asphyxia (drowning)	Ischemia/edema (CT)	BSP	BSP NCS	Unfavorable (6)
8	27	Asphyxia (foreign body aspiration)	Normal (CT)	RDA	IED	Unfavorable (3)
9	11	Metabolic encephalopathy	Ischemia/edema (CT, MRI)	*	RDA	Unfavorable (4)
10	34	Hypertensive encephalopathy (PRES)	Cerebral atrophy (CT) T2 hyperintensities with restricted diffusion (MRI)	Discontinuous activity IED	BSP	Favorable (2)
11	36	TBI	Hemorrhage, fracture, ischemia/edema (CT) Hemorrhage, ischemia/edema, subfalcine herniation (MRI)	*	IED	Unfavorable (4)
12	126	ADEM	Demyelinating lesions (MRI)	*	*	Favorable (1)
13	190	Intoxication (attempted suicide)	Normal (CT)	IED	IED	Favorable (1)
14	40	Asphyxia (drowning)	Not performed	Amplitude asymmetry (marked)	*	Unfavorable (4)
15	216	Intracranial mass	Hemorrhage (CT)	*	*	Unfavorable (4)
16	216	Fanconi anemia (septic shock, MOF)	Normal (CT)	BSP	Discontinuous activity	Unfavorable (4)
17	134	TBI	Hemorrhage, fracture (CT)	*	IED NCS	Unfavorable (3)
18	40	Uremic encephalopathy (HUS)	Extrapontine myelinolysis (CT, MRI)	Discontinuous activity	Discontinuous activity	Favorable (2)
19	187	Intoxication (attempted suicide)	Normal (CT)	*	*	Favorable (1)
20	138	TBI	Normal (CT)	Frequency asymmetry (mild)	*	Favorable (1)
21	5	Metabolic encephalopathy	T2 hyperintensities suggestive for metabolic disease (MRI)	NCS	Frequency asymmetry (mild) IED NCS	Unfavorable (3)
22	76	Hepatic encephalopathy	Normal (CT)	Amplitude asymmetry (mild)	Amplitude asymmetry (mild)	Favorable (2)

Table I. Continued

Patient	Age (months)	Etiology	Imaging finding	EEG finding (0-1 hour)	EEG finding (2-24 hours)	Prognosis (PCPC score)
23	4	TBI	Hemorrhage, ischemia/edema (CT, MRI)	Amplitude asymmetry (marked) LPD RDA IED CS, NCS	Discontinuous activity LPD, BIPD RDA CS, NCS	Unfavorable (4)
24	11	CINCA syndrome	Cerebral atrophy (CT, MRI)	IED	*	Unfavorable (3)

ADEM: Acute disseminated encephalomyelitis, BIPD: Bilateral independent periodic discharges, BSP: Burst suppression pattern, CINCA syndrome: Chronic infantile neurologic cutaneous and articular, CS: Convulsive seizure, CT: Computed tomography, DAI: Diffuse axonal injury, IED: Interictal epileptiform discharges, LPD: Lateralized periodic discharges, MOF: Multi organ failure, MRI: Magnetic resonance imaging, NCS: Non-convulsive seizure, PCPC: Pediatric cerebral performance category, PRES: Posterior reversible encephalopathy syndrome, RDA: Rhythmic delta activity, TBI: Traumatic brain injury, TWs: Triphasic waves
*: No pathological finding

The scores of all four cases with a history of CPR before the hospitalization was 3 or above.

There were not enough subjects in all groups to reliably test the relationship between etiology and prognosis statistically. However, those patients with asphyxia, head trauma or metabolic disease tended to have worse prognosis. The etiology and PCPC scores of the cases are outlined in Table I.

When the relationship between cranial imaging findings and short-term outcome was evaluated, all of the patients with "ischemia/edema" in cranial imaging and 83.3% (5/6) of those patients with "hemorrhage±fracture" displayed unfavorable outcome. The difference was statistically significant for the ischemia/edema group but not for the hemorrhage±fracture group ($p=0.046$, $p=0.333$). Additionally, having a normal cranial imaging was not related to favorable outcome ($p=0.074$). The cranial imaging findings and short-term outcome results are outlined in Table III.

When the short-term outcome was evaluated in relationship to the frequency of cEEG background activity, the outcomes of all patients with alpha (including beta) frequency was good and the outcomes of those patients with a slowing of background activity was variable. In the latter group, five out of 13 patients (38%) displayed good outcome.

Out of four patients with discontinuous background activity, two had favorable and two had unfavorable outcomes. Two of the three patients with BSP had unfavorable outcomes. One patient (case 7) with burst suppression was lost.

When evaluated in terms of background properties (frequency, continuity, presence of sleep characteristics,

presence of episodic or rhythmic activity) and short-term prognosis, the data was either not suitable for statistical analysis or did not reach statistical significance.

All four patients with NCS had unfavorable outcomes but the difference did not reach statistical significance ($p=0.115$).

Four patients had NCS. Two of these patients had NCS in both cEEG 1 h and cEEG 24 h. Whereas two patients had NCS only in cEEG 24 h. Nine out of 11 NCS in the study were recorded in cEEG 24 h. Patients with ischemia/edema in cranial imaging had significantly worse prognosis. Although those patients with asphyxia, head trauma and metabolic disease and those patients who received CPR before hospitalization tended to have worse prognosis, the differences were not statistically significant.

Discussion

Acute encephalopathy can be traumatic or non-traumatic. Common etiological causes and prognosis vary according to age and the quality of care provided. In a study by Löhr Junior et al. (11), the causes of acute coma in children were as follows; central nervous system (CNS) infection in 30%, status epilepticus in 23%, hypoxic ischemic encephalopathy in 22%, toxic metabolic causes in 18%, and other causes in 8% of the patients. In the study by Schreiber et al. (12) which included 94 children, the most common causes for acute encephalopathy were prior neurologic problems in 27.7%, TBI in 17%, meningitis/encephalitis in 16% and hypoxia/anoxia in 11.7% of the children. In our study, TBI was found to be the cause for acute encephalopathy in 6 cases (25%). Other causes were asphyxia (16.67%), intoxication (12.25%), hepatic encephalopathy (8.33%), metabolic encephalopathy (8.33%), uremic encephalopathy (4.17%), hypertensive encephalopathy (4.17%), septic shock

(4.17%), chronic infantile neurologic cutaneous articular syndrome (4.17%), intracranial mass (4.17%), acute disseminated encephalomyelitis (4.17%), and epilepsy (4.17%). In our study, TBI was the most common etiological reason ($p=0.024$). This might be related to our PICU being a tertiary trauma center which serves a wide region. None of the patients included in this study had CNS infection, which is common in other studies.

NCS and NCSE have been shown to develop in 10-40% of patients admitted to PICUs with acute encephalopathy. Approximately half of electrographic seizures develop within the first hour and 80-90% within the first 24 hours (5,13-15). Jette et al. (15) reported the incidence of NCS as 39% in a four-year study in 117 children who were under the age of 18 and hospitalized in a PICU. While 15% of these seizures developed in the first hour of recording,

Table II. Cranial imaging and short-term outcome findings in patients

		cEEG 1 h (number of patients)	cEEG 24 h (number of patients)
Slow background activity*		15	17
Continuity*	Continuous	20	19
	Discontinuous	2	3
	Burst suppression pattern	2	2
Sleep structure	Stage 2 sleep transients present	9	15
Asymmetry	Amplitude	Mild 2 Marked 2	2 -
	Frequency	Mild 1 Marked -	1 -
Periodic & rhythmic discharges	Lateralized periodic discharges	1	2
	Rhythmic delta activity	3	4
	Triphasic waves	1	
IED	Spike	1	1
	Sharp	6	7
Seizure	Convulsive seizure	1	1
	Non-convulsive seizure	2	4

IED: Interictal epileptic discharge
*: Some patients had more than one pattern throughout the acquisition

Table III. Cranial imaging and short-term outcome findings in patients

Imaging finding	Patients (n, %)	Favorable prognosis (n, %)	Unfavorable prognosis (n, %)
Hemorrhage/fracture	6 (27.27)	1 (16.67)	5 (83.33)
Ischemia/edema	6 (27.27)	-	6 (100)
Cerebral atrophy	2 (9.09)	1 (50)	1 (50)
Diffuse axonal injury	2 (9.09)	1 (50)	1 (50)
Demyelinating lesions	1 (4.54)	1 (100)	-
Extrapontine myelinolysis	1 (4.54)	1 (100)	-
Subfalcine herniation	1 (4.54)	-	1 (100)
T2 hyperintensities with restricted diffusion	1 (4.54)	1 (100)	-
T2 hyperintensities suggesting metabolic disease	1 (4.54)	-	1 (100)
Normal	7 (31.81)	5 (71.43)	2 (28.57)

*: Twenty-two patients underwent cranial imaging. Some patients had more than one finding

80% developed within 24 hours. In a prospective study conducted by Schreiber et al. (12) with 94 children with acute encephalopathy, NCSs were detected in 30% and NCSE in 18% of patients. Ninety-seven percent of these seizures were detected during 24 hour monitoring. In the study by Abend et al. (14), 101 children and infants who were admitted to a PICU for altered states of consciousness were evaluated. Thirty-two percent of the patients had NCSs and 19% had NCSE. Fifty-two percent of these seizures were detected in the first hour and 87% of them were detected in 24 hour cEEG monitoring (6). Similar to previous studies, only 18% (2/11) of NCSs developed within the first hour of recording in our group. The rest of the NCSs were observed within the 23 hours after the first hour. In three cases, monitoring continued for 72 hours and no seizures were detected after 24 hours. In a retrospective study which included 625 adult patients monitored in a tertiary center, 72 h risk of seizures were found to be lower than 5% if no epileptiform abnormalities were present in the first two hours of recording (16). Children may be at increased risk for NCS compared to adults (17). Younger age, prior convulsive status epilepticus or seizures and acute neuroimaging abnormalities were reported as risk factors for having NCS in critically ill children (3,18-20). In our study, TBI was found to increase the risk of NCS significantly ($p=0.032$).

Acute brain injury is reported to be associated with worse outcome in the literature (5,12). The study by Fink et al. (21) included 130 children with infectious encephalopathy or TBI in Africa. Patients with TBI were reported to have worse outcome than infectious encephalopathy. In the present study, the short-term outcome was unfavorable in asphyxia, TBI, and inborn metabolic diseases groups for 75%, 66.6% and 100% of cases, respectively. However, the correlation between the etiology and short-term outcome did not reach statistical significance. This might be due to the small sample size of our group.

It is well established that cranial imaging is useful for diagnosis in acute encephalopathy. Additionally, it may help in predicting the prognosis (22,23). In this study, we found that having ischemia/edema in cranial imaging to be a significant risk factor for unfavorable short-term outcome ($p=0.046$). Although those patients with hemorrhage/fracture in cranial imaging also had unfavorable outcome, the difference did not reach statistical significance ($p=0.333$). This was thought to be due to our small sample size.

Some studies have demonstrated that patients with severe EEG background abnormalities such as discontinuous activity and BSP tend to have worse prognosis than those

patients with mild/moderate background abnormalities (5,24). Topjian et al. (24) evaluated 128 patients who underwent EEG monitoring within one day of return of spontaneous circulation in terms of cEEG background activity, EEG reactivity and PCPC scores. EEG background pathologies including slow-disorganized, discontinuous-burst suppression, attenuated-flat activity and an absence of EEG reactivity were associated with mortality and unfavorable neurologic outcome. It is plausible to expect worse prognosis in those patients with severe background abnormalities such as BSP. However, due to the small sample size and low number of patients in each group, we could not make a statistical statement.

Some studies have shown an association between electrographic seizures/ electrographic status epilepticus and unfavorable neurodevelopmental outcome in critically ill children (5,12,25-27). Kirkham et al. (25) performed cEEG (one to three channels) in 204 children aged ≤ 15 years and determined their outcome at 1 month. They reported that unfavorable outcome (29/111 survivors; 26%) was independently predicted by the presence of electroencephalographic seizures. A recent study which included data from the PANGAEA study stated that receiving anti-seizure medication was associated with worse neurological outcomes (27). In our study, although there was a tendency for poorer outcome in those patients with NCS, the difference did not reach statistical significance. This might be due to the small sample size of our study.

Study Limitations

There are limitations in our study. First, we had a small sample size. Additionally, our study was not sufficiently powered. This may have caused us to report some factors as being not significantly related with outcome which may actually be seen to be significant with a larger sample size. The strength of this study is that all EEG recordings were evaluated by six interpreters and interrater agreement rates were assessed with statistical methods. We think that this data may be more useful when pooled with other data found in the literature.

Conclusion

In conclusion, cEEG in critically ill children is useful not only for detecting NCS but also for defining other non-epileptic events. The use of cEEG in PICUs can contribute to the more exact management of cases. Optimum collaboration between intensive care and neurology units is needed.

Ethics

Ethics Committee Approval: This study was reviewed and approved by Ege University Faculty of Medicine Ethics Committee (date: 08.05.2018, approval no: 18-5/49).

Informed Consent: Written informed voluntary consent was obtained from all individual participants included in this study.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Design: Ö.Ö.B., Data Collection and/or Processing: Ö.Ö.B., P.Y.Ö., Analysis and/or Interpretation: Ö.Ö.B., P.Y.Ö., Revising the Article: P.Y.Ö., S.K., İ.D., H.M.S., S.Y., G.A., H.T., B.K., S.G., Writing: Ö.Ö.B., E.Ş., S.G.

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References

1. Bazaraa HM, El Houchi S, Rady HI. Profile of patients visiting the pediatric emergency service in an Egyptian university hospital. *Pediatr Emerg Care* 2012; 28:148-52.
2. Lanetzi CS, de Oliveira CA, Bass LM, Abramovici S, Troster EJ. The epidemiological profile of Pediatric Intensive Care Center at Hospital Israelita Albert Einstein. *Einstein (Sao Paulo)* 2012; 10:16-21.
3. Sánchez Fernández I, Sanseverè AJ, Guerriero RM, et al. Time to electroencephalography is independently associated with outcome in critically ill neonates and children. *Epilepsia* 2017; 58:420-8.
4. Wilson CA. Continuous electroencephalogram detection of non-convulsive seizures in the pediatric intensive care unit: review of the utility and impact on management and outcomes. *Transl Pediatr* 2015; 4:283-9.
5. Topjian AA, Gutierrez-Colina AM, Sanchez SM, Berg RA, Friess SH, Dlugos DJ, Abend NS. Electrographic status epilepticus is associated with mortality and worse short-term outcome in critically ill children. *Crit Care Med* 2013; 41:215-23.
6. Abend NS, Topjian AA, Gutierrez-Colina AM, Donnelly M, Clancy RR, Dlugos DJ. Impact of continuous EEG monitoring on clinical management in critically ill children. *Neurocrit Care* 2011; 15:70-5.
7. Hirsch LJ, LaRoche SM, Gaspard N, et al. American Clinical Neurophysiology Society's Standardized Critical Care EEG Terminology: 2012 version. *J Clin Neurophysiol* 2013; 30:1-27.
8. Abend NS, Chapman KE, Gallentine WB, et al. Electroencephalographic monitoring in the pediatric intensive care unit. *Curr Neurol Neurosci Rep* 2013; 13:330.
9. McHugh ML. Interrater reliability: the kappa statistic. *Biochem Med (Zagreb)* 2012; 22:276-82.
10. Fiser DH. Assessing the outcome of pediatric intensive care. *J Pediatr* 1992; 121:68-74.
11. Löhr Junior A, Liberalesso PB, Luzzi GC, de Faria AC, Bugallo MJ, Santos ML. Etiologia e a morbi-letalidade do coma agudo em crianças [Acute coma in children: etiology, morbidity and mortality]. *Arq Neuropsiquiatr* 2003; 61:621-4.
12. Schreiber JM, Zelleke T, Gaillard WD, Kaulas H, Dean N, Carpenter JL. Continuous video EEG for patients with acute encephalopathy in a pediatric intensive care unit. *Neurocrit Care* 2012; 17:31-8.
13. Herman ST, Abend NS, Bleck TP, et al. Consensus statement on continuous EEG in critically ill adults and children, part II: personnel, technical specifications, and clinical practice. *J Clin Neurophysiol* 2015; 32:96-108.
14. Abend NS, Wusthoff CJ, Goldberg EM, Dlugos DJ. Electrographic seizures and status epilepticus in critically ill children and neonates with encephalopathy. *Lancet Neurol* 2013; 12:1170-9.
15. Jette N, Claassen J, Emerson RG, Hirsch LJ. Frequency and predictors of nonconvulsive seizures during continuous electroencephalographic monitoring in critically ill children. *Arch Neurol* 2006; 63:1750-5.
16. Westover MB, Shafi MM, Bianchi MT, et al. The probability of seizures during EEG monitoring in critically ill adults. *Clin Neurophysiol* 2015; 126:463-71.
17. Claassen J, Mayer SA, Kowalski RG, Emerson RG, Hirsch LJ. Detection of electrographic seizures with continuous EEG monitoring in critically ill patients. *Neurology* 2004; 62:1743-8.
18. Abend NS, Gutierrez-Colina AM, Topjian AA, et al. Nonconvulsive seizures are common in critically ill children. *Neurology* 2011; 76:1071-7.
19. McCoy B, Sharma R, Ochi A, et al. Predictors of nonconvulsive seizures among critically ill children. *Epilepsia* 2011; 52:1973-8.
20. Williams K, Jarrar R, Buchhalter J. Continuous video-EEG monitoring in pediatric intensive care units. *Epilepsia* 2011; 52:1130-6.
21. Fink EL, von Saint Andre-von Arnim A, Kumar R, et al. Traumatic Brain Injury and Infectious Encephalopathy in Children From Four Resource-Limited Settings in Africa. *Pediatr Crit Care Med* 2018; 19:649-57.
22. Weiss N, Galanaud D, Carpentier A, Naccache L, Puybasset L. Clinical review: Prognostic value of magnetic resonance imaging in acute brain injury and coma. *Crit Care* 2007; 11:230.
23. Khipal J, Sankhyan N, Singhi SC, Singhi P, Khandelwal N. Clinical Utility of MRI Brain in Children with Non-traumatic Coma. *Indian J Pediatr* 2017; 84:838-42.
24. Topjian AA, Sánchez SM, Shults J, Berg RA, Dlugos DJ, Abend NS. Early Electroencephalographic Background Features Predict Outcomes in Children Resuscitated From Cardiac Arrest. *Pediatr Crit Care Med* 2016; 17:547-57.
25. Kirkham FJ, Wade AM, McElduff F, et al. Seizures in 204 comatose children: incidence and outcome. *Intensive Care Med* 2012; 38:853-62.
26. Abend NS, Arndt DH, Carpenter JL, et al. Electrographic seizures in pediatric ICU patients: cohort study of risk factors and mortality. *Neurology* 2013; 81:383-91.
27. Snooks KC, Yan K, Farias-Moeller R, Fink EL, Hanson SJ. Continuous Electroencephalogram and Antiseizure Medication Use in an International Pediatric Traumatic Brain Injury Population. *Neurocrit Care* 2022; 36:573-83.