Emergency Medicine: Open Access

Liu et al., Emerg Med (Los Angel) 2017, 7:3
DOI: 10.4172/2165-7548.1000354

Research Article OMICS International

Analysis of 247 Children with Status Epilepticus: Clinical Features and Prognosis

Tian-tian Liu[1], **Ji Wang**[1], **Guo-ping Lu**[2] **and Yi Wang**[1*]

[1]*Department of Neurology, Children's Hospital of Fudan University, Macao SAR, China*

[2]*Intensive Care Unit, Children's Hospital of Fudan University, Macao SAR, China*

[*]**Corresponding author:** Yi Wang, Department of Neurology, Children's Hospital of Fudan University, Macao SAR, China, Tel: +86-18818211513; E-mail: yiwang@shmu.edu.cn

Received date: October 14, 2015; **Accepted date:** May 31, 2017; **Published date:** June 07, 2017

Abstract

Background: Status epilepticus (SE) is one of the most common neurological emergencies in childhood. However, the clinical features and prognosis are poorly characterized in China.

Objectives: To summarize the clinical features and outcomes among Chinese children.

Methods: Children aged from 29 days to 18 years with SE were included in Children's Hospital of Fudan University. The demography, etiology, seizure types, treatment and outcome were analysed. The statistics were conducted by SPSS19.

Results: There were 247 SE patients (F:M=1.25:1). The mean age was 2.4 years. Most of them had acute symptomatic SE (53.06%) including viral encephalitis (48.46%). Most of them had convulsive status epilepticus (83.4%), others had non-convulsive status epilepticus (16.6%). Most of them had combination therapy (74.89%). Lengths of follow-up ranged from 1 month to 3 years after onset of SE. Most of cases were bad outcome (73.80%). Etiology and neurological image were significant risk predictors by multiple logistic regression analysis. Mortality rate was 16.95%. Recurrence rate was 20.76%. Most of cases died within 1 month after onset of SE (65.0%). Most of cases also recurred within 1 month (81.63%). Duration of SE and mechanical ventilation were related to death by multiple logistic regression analysis. The number of antiepileptic drugs (AED) and anaesthetic treatment were related to recurrence of SE by Cox survival analysis.

Conclusion: The proportion of SE was highest in children younger less than 1 year. The most common etiology of SE in children was acute symptomatic, especially viral encephalitis. The clinical features between CSE and NCSE were different. Risk factors between recurrence and death were different. The death rate was higher in cases with duration of SE ≥ 3 hours and mechanical ventilation. Maintenance treatment ≥ 3 AEDs and anaesthetic treatment were related to higher recurrence rate. These results suggested we can improve the prognosis by terminating seizure in time

Keywords: Status epilepticus; Clinical features; Outcome

Introduction

Status epilepticus (SE) is one of common medical emergencies of the paediatric age group. It will lead to serious and even irreversible neurological abnormalities including death if not diagnosis and treatment properly. The traditional epidemiologic definition of SE as continuous or intermittent seizures lasting for more than 30 min with incomplete recovery of consciousness may be impractical [1]. However, more recent definitions advocate a much shorter operational definition: either continuous seizures lasting at least 5 min or two or more discrete seizures between which there is incomplete recovery of consciousness [2,3]. SE has been classified based on clinical features, such as convulsive SE (CSE) or non-convulsive SE (NCSE).

The incidence of CSE in children is approximately 10–27/100,000 per year, and is higher than the 4-6/100,000/year reported in epidemiologic studies of convulsive status epilepticus [4,5]. NCSE have provided overall population incidence rate of between 5.6 and 18.3 per 100000 individuals per year. NCSE often follow CSE, accounting for 26% of the NCSE. The present study focuses on adults, but its research data may not be able to provide children SE reliable basis for clinical work. The children as a special group, the incidence, mortality, etiology, SE type, treatment and prognosis are different from adults [3]. To recognize etiology, seizure type, treatment, rise factors, outcome and the correlation between each other of children with SE is significant for management. Unfortunately, there are few published data from population-based and hospital-based studies of paediatric groups in China.

Methods

Study population

This is an observational study. In this study, we examined patients 29 days to 18 years who were diagnosed with SE in our hospital between 1 January 2012 and 31 December 2014. SE is defined as

continuous or intermittent seizures lasting for more than 30 min without complete recovery of consciousness, including CSE and NCSE. Exclusion criteria: a seizure is less than 30 minutes; the new-born and more than 18 years old.

Data acquisition

We queried cases with a diagnostic code of SE via usage of the International Classification of Diseases, 10th revision (ICD-10) code G41.900. We collected detailed information from medical record, including demography, etiology, seizure types, EEG, VEEG, MRI/CT and treatment. Lengths of follow-up ranged from 1 month to 3 years after onset of SE by electronic medical record (EMR) and telephone. The outcome include neurological sequelae, recurrence and death following SE.

The seizure types were classified as CSE and NCSE. The etiology types are listed and types in Table 1 [6,7]. Outcomes of SE patients and related factors were evaluated and described using the Glasgow Outcome Score (GOS), including good (GOS=1-4) and bad (GOS=5).

Types	Definition
Febrile SE	SE occurring when the only provocation is a febrile (38.0°C) illness and in the absence of defined CNS infection.
Acute-symptomatic	SE in a previously neurologically normal child, within a week of an identified acute neurological insult.
Remote-symptomatic	SE in a child with a history of a pre-existing CNS abnormality in the absence of an identified acute insult.
Acute-on-remote-symptomatic	SE occurring during an acute neurological insult or febrile illness in a child with a remote symptomatic etiology.
Cryptogenic/idiopathic epilepsy-related	SE that occurred in children with a previous diagnosis with idiopathic/cryptogenic epilepsy, or SE that is the child's second unprovoked seizure.
Unclassified	SE that cannot be classified into any other group.

Table 1: The etiology types of SE.

Statistical analysis

SPSS19.0 software was used for statistical analysis with p-values of <0.05 and was considered significant. A chi-square test was conducted. We used logistic regression and Cox survival analysis to analyse the clinical outcome.

Results

In this study, 247 children were included. Details are shown in Table 2.

Item	Inpatient n (%)	Outpatient n (%)	Total n (%)
General condition			
Sex			
Male	135(57.20)	2(18.18)	137(55.47)
Female	101(42.80)	9(81.82)	110(45.53)
Age			
<1y	69(29.24)	4(36.37)	73(29.55)
1-3y	69(29.24)	2(18.18)	71(28.75)
3-6y	39(16.52)	2(18.18)	41(16.60)
6-12y	43(18.22)	2(18.18)	45(18.22)
≥ 12y	16(6.78)	1(9.09)	17(6.88)
History			
CNS disease			
Yes	85(36.02)	4(50.00)	89(36.48)
No	151(63.98)	4(50.00)	155(63.52)
Asphyxia			
Yes	12(5.29)	0(0.00)	12(5.13)
No	215(94.71)	7(100.00)	222(94.87)
Family history			
Yes	24(10.62)	1(16.67)	25(10.78)
No	202(89.38)	5(83.33)	207(89.22)
Clinical feature			
First ever episodes			
Yes	208(91.23)	6(85.71)	214(91.06)
No	20(8.77)	1(14.29)	21(8.94)
SE types			
CSE	195(82.63)	11(100.00)	206(83.40)
NCSE	41(17.37)	0(0.00)	41(16.60)
SE duration			
30 min-1 h	92(38.98)	6(75.00)	98(40.33)
1-3 h	46(19.49)	2(25.00)	48(19.74)
≥ 3 h	98(41.53)	0(0.00)	98(40.33)
Fever			
≥ 38	135(57.20)	5(62.50)	140(57.38)
<38	101(42.80)	3(37.50)	104(42.62)
Auxiliary examination			
MRI/CT			
Normal	77(33.48)	4(100.00)	81(34.62)
Abnormal	153(66.52)	0(0.00)	153(63.38)
Cerebrospinal fluid (CSF)			
Normal	112(65.88)	1(100.00)	113(66.08)

Abnormal	58(34.12)	0(0.00)	58(33.92)
Metabolic acidosis			
YES	104(52.53)	6(85.71)	110(53.66)
NO	94(47.47)	1(14.29)	95(46.34)
EEG			
Normal	20(8.97)	2(50.00)	22(9.69)
Abnormal	203(91.03)	2(50.00)	205(90.31)
Treatment			
emergency treatment			
Without AEDs	9(4.14)	1(10.00)	10(4.41)
One kind of AEDs	44(20.28)	3(30.00)	47(20.70)
Two kind of AEDs	55(25.35)	3(30.00)	58(25.55)
≥ Three kind of AEDs	109(50.23)	3(30.00)	112(49.34)
Sequential therapy			
Without AEDs	41(17.37)	-	41(17.37)
One kind of AEDs	72(30.51)	-	72(30.51)
Two kind of AEDs	47(19.92)	-	47(19.92)
≥ Three kind of AEDs	76(32.20)	-	76(32.20)
Mechanical ventilation	82(34.7)	-	82(34.7)
Mild hypothermia therapy	17(7.20)	-	17(7.20)

Table 2: Detailed information of SE.

Demography

There were 247 SE patients including in this study, including 236 inpatients and 11 outpatients. 132 of 236 inpatients came to paediatric intensive care unit (PICU). The ratio of males to females was 1.25:1. The mean age was 2.4 years, with 58.30% less than 3 years of age.

Etiology

245 of 247 subjects can clear the etiology. Within our patients, most of them had acute symptomatic SE (130, 53.06%) including viral encephalitis (48.46%), followed by acute-on-remote-symptomatic SE in 54(22.04%) cases. 28(11.43%) cases were remote symptomatic SE 28(11.43%). Febrile SE accounted for 16(6.12%) cases. Others were unclassified cases (3, 1.22%).

Clinical features

Within our patients, most of them had CSE (206/247, 83.4%). Others had NCSE (39/247, 16.6%). Most of CSE were generalized seizure (144/202, 71.3%) including tonic-clonic seizure (65.3%), tonic seizure (31.2%), clonic seizure (2.8%), myoclonic seizure (0.7%). 56.2% CSE were intermittent seizure. 47.3% CSE lasted 30 min-1 h. Most of NCSE were partial seizure (73.2%). 85.4% NCSE were intermittent seizure. 85.4% NCSE lasted longer than 3 h. Most of NCSE were diagnosed by VEEG (95.12%).

Auxiliary examination

In this study, abnormal cranial imaging results mainly include signal changes (such as cortex, thalamus, basal), brain dysplasia, intracranial haemorrhage, vascular disease (vascular malformations, cerebral infarction), intracranial placeholder, cerebral edema and hypoxia ischemic injury, etc. Statistical analysis showed that cerebral dysgenesis ($\chi2=15.801$, $P=0.003$), cerebral edema ($\chi2=15.520$, $P=0.002$), ventricular expansion ($\chi2=8.276$, $P=0.036$) and brain atrophy ($\chi2=10.780$, $P=0.012$) with bad prognosis.

184 of 227 EEG cases had video electroencephalogram (VEEG). Statistical analysis showed that bad outcome were related to frequent discharge ($\chi2=17.87$, $P=0.001$), the PLEDs ($\chi2=8.60$, $P=0.037$), three phase wave ($\chi2=9.913$, $P=0.005$).

Treatment

AEDs include clonazepam (vein), phenobarbital (intramuscular injection, intravenous and nasogastric), chloral hydrate (oral and enema), midazolam (vein), diazepam (vein), valproate (intravenous and oral), levetiracetam (oral), topiramate (oral), etc. The most of them was clonazepam (209 cases).

Within our patients, 4.41% without AEDs can relieve themselves for failing to see a doctor. Most of them had combination therapy (74.89%). 40.0% (99cases) were refractory status epilepticus (RSE). All of RSE were combination therapy. 14.14% RSE used anaesthetics. 34.75% inpatients used mechanical ventilation. The mean ventilation time was 8 days.

Hospital charge and duration of hospitalization

Mean ± SD duration of hospitalization was 17.81 ± 17.35 days (range, 1–114 days), and median (P25, P75) direct hospital charge was equal to 20165.51(9178.33, 43325.81) RMB. Direct hospital charge was significantly longer for patients with RSE than for those with SE ($P=0.004$). Duration of hospitalization was significantly shorter for patients with good outcome (GOS=5) than for those with bad outcome (GOS=1-4) ($P=0.005$).

Outcome

Lengths of follow-up ranged from 1 month to 3 years after onset of SE. Most of cases were bad outcome (73.80%). During the follow-up, outpatients were excepted. Data after onset of SE are shown in Table 3.

Item	Number	Percentage
Auxiliary examination		
Cranial imaging (n=48)		
normal	16	33.33%
abnormal	32	66.67%
Intellectual assessment (n=23)		
normal	7	30.42%
abnormal	16	69.58%
EEG(n=75)		
normal	40	53.33%

abnormal	35	46.67%
Treatment		
AEDs (n=159)		
Without AEDs	61	38.34%
One kind of AEDs	49	30.82%
Combination therapy	49	30.82%
Rehabilitation therapy (n=187)	41	21.93%
Outcome		
GOS(n=187)		
Good(GOS=5)	49	26.20%
Bad(GOS=1-4)	138	73.80%

Table 3: Data after onset of SE.

Single factor Logistic analysis of P<0.05 items include CNS diseases history, etiology, SE types, SE forms (partial/generalized seizures), duration of SE, neurological image, the kind of AEDs, recurrence. With multiple logistic regression analysis, causes, neurological image were selected as independence predictors of SE risk (Table 4).

Item	OR	95%CI	χ^2	P
Etiology				
Febrile SE	-	-	-	-
Acute-symptomatic	0.074	0.190, 9.013	0.012	0.914
Remote-symptomatic	0.607	0.249, 25.073	0.038	0.846
Acute-on-remote-symptomatic	6.147	1.805, 156.072	16.786	0.013
Cryptogenic/idiopathic epilepsy-related	2.229	0.570, 63.603	6.023	0.135
Abnormal neurological image	3.299	1.157, 9.405	4.988	0.026

Table 4: Comparison of time intervals.

Death

Mortality rate was 16.95% (40/236). Most of cases died within 1 month after onset of SE (65.0%). According to single-factor logistic regression analysis, CNS disease history, seizure character (continuous/intermittent), duration of SE, combined anesthesia, mechanical ventilation, recurrence were related to Death. To further analysis, the death rate was higher in cases with duration of SE ≥ 3 hours and mechanical ventilation by multiple logistic regression analysis (Table 5).

Item	OR	95%CI	χ^2	P
CNS disease history	0.440	0.140, 1.381	1.980	0.159
Continuous seizure	1.487	0.418, 5.291	0.375	0.540
Duration of SE				
<1 h	-	-	-	-
1-3 h	1.063	0.213, 5.303	0.003	0.094
≥ 3 h	4.171	1.213, 14.339	5.138	0.023
Combined anesthesia	1.795	0.328, 9.817	0.455	0.500
Mechanical ventilation	4.385	1.696, 11.200	9.349	0.002
Recurrence	1.565	0.512, 4.781	0.618	0.432

Table 5: Multiple logistic regression analysis for SE.

Recurrence

Item	HR	95%CI	χ^2	P
Seizure features(mode 1)				
CSE	15.175	2.030, 113.440	7.022	0.008
Duration of SE				
<1 h	-	-	-	-
1-3 h	1.767	0.694, 4.497	1.427	0.232
≥ 3 h	3.521	1.471, 8.428	7.988	0.005
Continuous seizure	0.609	0.265, 1.402	1.385	0.244
Treatment(Mode 2)				
Sequential therapy				
Without AEDs	-	-	-	-
Single AEDs	0.434	0.084, 2.238	0.993	0.319
Two kind of AEDs	1.541	0.372, 6.377	0.356	0.551
≥three kind of AEDs	3.462	0.994, 12.060	3.802	0.051
Combined anesthesia	3.722	1.512, 9.160	8.178	0.004
mechanical ventilation	1.648	0.760, 3.570	1.601	0.206
Mode 3				
CSE	10.901	1.446, 82.169	5.373	0.020
Duration of SE				
<1 h	-	-	-	-
1-3 h	1.898	0.770, 4.678	1.937	0.164
≥ 3 h	2.058	0.915, 4.629	3.049	0.081
Sequential therapy				
Without AEDs	-	-	-	-

Single AEDs	0.508	0.102, 2.535	0.682	0.409
Two kind of AEDs	2.001	0.491, 8.159	0.935	0.334
≥ three kind of AEDs	3.760	1.066, 13.256	4.243	0.039
Combined anesthesia	3.600	1.632, 7.942	10.074	0.002

Table 6: Multiple cox survival analysis for recurrence.

Recurrence rate was 20.76% (49/236). Most of cases also recurred within 1 month (81.63%). According to single-factor Cox survival analysis, SE types, duration of SE, seizure character (continuous/intermittent), the kind of AEDs, combined anesthesia and mechanical ventilation were related to recurrence. Finally, sequential therapy ≥ 3 AEDs and anesthetic treatment were related to higher recurrence rate by multiple Cox survival analysis (Table 6). Kaplan-Meier curves of recurrence showed in Figure 1.

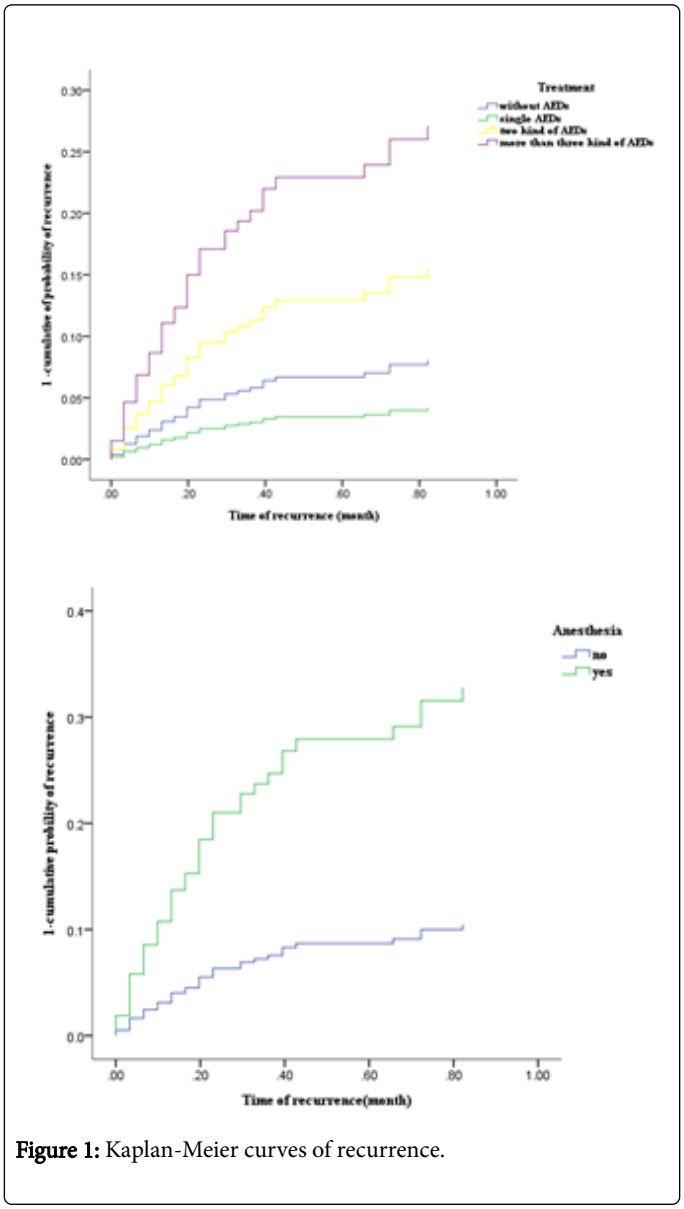

Figure 1: Kaplan-Meier curves of recurrence.

Discussion

Status epilepticus is the most common childhood medical neurological emergency. Most data for this disorder are from mainly adult populations and might not be relevant to childhood. At present, domestic related research is incomplete. We required data from Children's Hospital of Fudan University. We hope it will provide evidences for the current clinical practice and further research.

Clinical characteristics

Within our patients, the ratio of males to females was 1.25:1. This showed that bays ratio is higher than girls. De Lorenzo et al. [8] reported the same results. However, the conclusion is debatable. The most patients were aged younger 1years following cases aged from 1 to 3years. Chin et al. [7] showed the incidence of SE was highest in children younger than 1 year. Nishiyama et al. [6] developed multicenter epidemiologic study. The results showed that the highest incidence was seen in patients aged <2 years, especially in the second year of life. Lin et al. [9] reported the highest incidence of SE was in the 1-5 year age group. Approximately two thirds of the patients were less than 5 years old when they manifested SE.

In this study, most of them had acute symptomatic SE including viral encephalitis, followed by acute-on-remote-symptomatic SE. Febrile SE accounted for only 6.12%. A number of studies show that febrile SE is the most common cause in developed country [6,10]. However, a study in India showed their conclusion is the same with us. The causes of children SE may be different. Between developed countries and developing countries. In addition to, the differences may be the reason for this study is given priority to with hospitalized children, and most of children were from PICU.

Within our patients, most of them had CSE (83.4%). Others had NCSE (16.6%). Most of NCSE were diagnosed by VEEG. The clinical manifestations of NCSE in children typically include cognitive or behavioral impairments. The availability of VEEG monitoring has allowed for increased recognition of this condition [11-13]. Seizures in the acute setting, ocular movements and ongoing CNS infection are associated with NCSE [14]. In China, children NCSE Still need further study.

Understand disease burden is conducive to improve medical quality, and rational distribute medical resources. Children SE related researches are rare [15,16].

Outcome

With multiple logistic regression analysis, causes, neurological image were selected as independence predictors of SE risk. Asadi-Pooya et al. [16] reported outcome was significantly correlated with etiology of SE. This result is same with us. Lin et al. [9] reported that only the etiology and age were significant predictors of outcomes. SE with overall prognosis is poor [17], but significant predictors vary in different research. In a word, it remains to be further discussed.

Death of SE

In our study, mortality rate was 16.95%. The death rate was higher in cases with duration of SE≥3 hours and mechanical ventilation by multiple logistic regression analysis. Some studies showed the significant predictors included age, etiology, duration of SE and effective treatment [18-21]. Within our study, most of cases died within

1 month after onset of SE. Ostrowsky et al. [18] showed short-term (during the first 30-60 days after SE) mortality rates of 3% to 5%. It was different from our conclusion. Domestic related data is rare. And it needs to be further discussed.

Recurrence of SE

Recurrence rate was 20.76%. Sequential therapy ≥3 AEDs and anesthetic treatment were related to higher recurrence rate by multiple Cox survival analysis. Multivariate analyses showed that progressive encephalopathy and preexisting neurologic deficit were independent predictors for SE recurrence [22]. Pediatric related data is rare. An adult study showed recurrence risk is independently associated with chronic etiology and to a lesser extent with female gender [23]. Follow-up time, etiology classification, object of study, resulted in different research results. It is necessary to further study.

Conclusion

The proportion of SE was highest in children younger less than 1 year. The most common etiology of SE in children was acute symptomatic, especially viral encephalitis. Duration of SE, continuous and intermittent seizure were related to the etiology.

The clinical features between CSE and NCSE were different. The characteristics of NCSE were variable. We can use VEEG to improve the rate of diagnosis.

Risk factors between recurrence and death were different. The death rate was higher in cases with duration of SE ≥3 hours and mechanical ventilation. Maintenance treatment ≥ 3 AEDs and anesthetic treatment were related to higher recurrence rate. These results suggested we can improve the prognosis by terminating seizure in time.

References

1. Dham BS, Hunter K, Rincon F (2014) The epidemiology of status epilepticus in the United States. Neurocrit Care 20: 476-483.
2. Ng YT, Maganti R (2013) Status epilepticus in childhood. J Paediatr Child Health 49: 432-437.
3. Sánchez Fernández I, Abend NS, Agadi S, An S, Arya R, et al. (2014) Gaps and opportunities in refractory status epilepticus research in children: A multi-center approach by the Pediatric Status Epilepticus Research Group (pSERG). Seizure 23: 87-97.
4. Sánchez Fernández I, Abend NS, Arndt DH, Carpenter JL, Chapman KE, et al. (2014) Electrographic seizures after convulsive status epilepticus in children and young adults: A retrospective multicenter study. J Pediatr 164: 339-346.
5. Capovilla G, Beccaria F, Beghi E, Minicucci F, Sartori S, et al. (2013) Treatment of convulsive status epilepticus in childhood: Recommendations of the Italian League Against Epilepsy. Epilepsia 7: 23-34.
6. Nishiyama I, Ohtsuka Y, Tsuda T, Kobayashi K, Inoue H, et al. (2011) An epidemiological study of children with status epilepticus in Okayama, Japan: Incidence, etiologies, and outcomes. Epilepsy Res 96: 89-95.
7. Chin RF, Neville BG, Peckham C, Bedford H, Wade A, et al. (2006) Incidence, cause, and short-term outcome of convulsive status epilepticus in childhood: prospective population-based study. Lancet 368: 222-229.
8. DeLorenzo RJ (2006) Epidemiology and clinical presentation of status epilepticus. Adv Neurol 97: 199-215.
9. Lin KL, Lin JJ, Hsia SH, Wu CT, Wang HS (2009) Analysis of convulsive status epilepticus in children of Taiwan. Pediatr Neurol 41: 413-418.
10. Seinfeld S, Shinnar S, Sun S, Hesdorffer DC, Deng X, et al. (2014) Emergency management of febrile status epilepticus: Results of the FEBSTAT study. Epilepsia 55: 388-395.
11. Galimi R (2012) Nonconvulsive status epilepticus in pediatric populations: Diagnosis and management. Minerva Pediatr 64: 347-355.
12. Greiner HM, Holland K, Leach JL, Horn PS, Hershey AD, et al. (2012) Nonconvulsive status epilepticus: The encephalopathic pediatric patient. Pediatrics 129: e748-755.
13. Freilich ER, Schreiber JM, Zelleke T, Gaillard WD (2014) Pediatric status epilepticus: Identification and evaluation. Curr Opin Pediatr 26: 655-661.
14. Tu TM, Loh NK, Tan NC (2013) Clinical risk factors for non-convulsive status epilepticus during emergent electroencephalogram. Seizure 22: 794-797.
15. Kortland LM, Knake S, Rosenow F, Strzelczyk A (2015) Cost of status epilepticus: A systematic review. Seizure 24: 17-20.
16. Asadi-Pooya AA, Poordast A (2005) Etiologies and outcomes of status epilepticus in children. Epilepsy Behav 7: 502-505.
17. Prins A, Chengo E, Mung'Ala Odera V, Sadarangani M, Seaton C, et al. (2014) Long-term survival and outcome in children admitted to kilifi district hospital with convulsive status epilepticus. Epilepsy Research and Treatment 2014: 1-7.
18. Ostrowsky K, Arzimanoglou A (2010) Outcome and prognosis of status epilepticus in children. Semin Pediatr Neurol 17: 195-200.
19. Maegaki Y, Kurozawa Y, Tamasaki A, Togawa M, Tamura A, et al. (2015) Early predictors of status epilepticus-associated mortality and morbidity in children. Brain Dev 37: 478-486.
20. Pujar S, Neville BG, Scott RC, Chin RF, North London Epilepsy Research Network (2011) Death within 8 years after childhood convulsive status epilepticus: A population-based study. Brain 134: 2819-2827.
21. Koubeissi M, Alshekhlee A (2007) In-hospital mortality of generalized convulsive status epilepticus: A large US sample. Neurology 69: 886-893.
22. Kravljanac R, Jovic N, Djuric M, Jankovic B, Pekmezovic T (2011) Outcome of status epilepticus in children treated in the intensive care unit: A study of 302 cases. Epilepsia 52: 358-363.
23. Tsetsou S, Novy J, Rossetti AO (2015) Recurrence of status epilepticus: prognostic role and outcome predictors. Epilepsia 56: 473-478.