

## Original Article      Clinical characteristics and outcome in children with convulsive status epilepticus

Pediatrics

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### ABSTRACT

**Background:** Status epilepticus (SE) is a serious neurological emergency that is associated with increased morbidity and mortality. There is emerging interest in understanding the clinical characteristics and identification of the prognosis of children with SE.

**Objective:** To identify the clinical profile and prognosis of children presenting by status epilepticus.

**Methodology:** This is a descriptive analytical study carried out on 31 children who presented by convulsive SE and were admitted to the pediatric intensive care unit. Clinical data, Complete blood count, C-reactive protein, erythrocyte sedimentation rate, serum glucose & electrolytes and cerebrospinal fluid analysis were estimated. Magnetic resonance imaging was done to all included children.

**Results:** Most of the children who presented with SE were younger than 5 years of age (45.2% were <2 years and 32.2% were 2-5 years). 74.2% were males and 83.9% had generalized seizures. 29% had SE as a first presentation. 77.4% were known cases of epilepsy. Non epileptic causes included encephalitis (12.9%), febrile convulsion (a typical) (6.5%) and acute hypoxia (shocking) (3.2%). The main precipitating factors for SE were missing doses in 29%, acute febrile illness in 12.9%, acute hypoxia in 6.5%, while precipitating factors could not be identified in 41.9% of children. Most children had normal MRI (64.5%). The commonest abnormalities were diffuse hypoxia and encephalitis (12.9% for each). 70.9% of children survived without neurological sequelae, 19.4% developed neurological sequelae while 9.7% died. Non-epileptic etiology, febrile illness, hypoxia, elevated acute phase reactant (erythrocytes sedimentation rate and C-reactive protein) was significantly associated with poor outcome.

**Conclusion:** Despite that epilepsy is the main cause of SE, non-epileptic causes are more serious and are associated with a worse outcome. Male gender, poor compliance to antiepileptic medications and febrile illness are linked to the development of SE in children. SE induced by hypoxia and encephalitis had the highest morbidity and mortality.

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### INTRODUCTION

Status Epilepticus (SE) is a major acute, life-threatening neurological emergency. It may be an initial presentation of many neurological disorders in children. The overall incidence of pediatric SE is roughly 20 per 100,000 children per year, with overall mortality of 3% [1]. The International League Against Epilepsy (ILAE) defines SE as "a seizure caused by failure of the mechanisms that terminate seizure activity or development of mechanisms that induce abnormally prolonged seizure activity that shows no clinical signs of arresting after a duration encompassing the great majority of seizures of that type in most patients or recurrent seizures without

interictal resumption of baseline central nervous system function causing a risk of long-term consequences (this time point is defined as 30 minutes after initiation of seizure activity)" [2].

Status Epilepticus represent a heterogeneous rather than a single entity disorder that include many electro-clinical syndromes with different etiologies and several triggering factors. Despite advance in treatment, it is still associated with significant neuro-morbidity and mortality. Neuronal damage is a serious sequel if seizures are not promptly controlled [3]. Mechanisms of neuronal injury includes combination of direct damage

from seizure-related activity (excitotoxicity) and secondary effects of the associated metabolic cascade, hypoxia, oxidative stress and activation of inflammatory signals. Seizures exceeding 30 minutes duration are associated with long-term consequences including neuronal death, neuronal injury, and altered neuronal networks<sup>[4]</sup>.

Children are vulnerable group as human brain undergoes rapid growth and maturation in early years of life which continue throughout adolescence. Prolonged seizure activity adversely affects the brain structure and function. Early initiation of treatment is a major step in management aiming to abort seizures activity<sup>[5]</sup>.

Prognosis of children who develop SE it is significantly affected by duration of seizures, seizure types and underlying etiology, in addition to the child age and associated comorbidities. Identifying children at greater risk for development of neurological sequels is an emerging area of research with conflicting findings<sup>[6]</sup>. The current study aimed to identify the clinical profile and prognosis of children presented by status epilepticus.

**PATIENTS AND METHOD**

The present study is a cross sectional descriptive analytical study. It was carried out on 31 children presented by status epilepticus who were admitted to pediatric intensive care unit (PICU). Children were recruited consecutively from the PICU of Al-Galaa and Ghamra military hospital during the period from November 2018 to July 2020. A written informed consent was received from parents of included children after explaining the aim and outcome of the study. The protocol of the study was approved by local ethics committee at faculty of medicine for girls, Al-Azhar University (IRB 202103714).

**Inclusion criteria:**

1. Age group from 1 month to 18 years. 2- Both sexes were included
2. Patients presented by convulsive status epilepticus according to definition of status epilepticus from the International League Against Epilepsy (ILAE)<sup>[2]</sup>

**Exclusion criteria:**

1. Children presented with short duration seizure that did not fulfill the criteria for status epilepticus.
2. Seizures caused by drug or chemical poisoning.
3. Seizures caused by inborn errors of metabolism (based on history, examination, and MRI findings)
4. Seizures caused by non-neurological disorders (e.g., hepatic, renal, hypoglycemia, electrolytes disturbances, or psychological diseases)
5. Post-concussion cases or traumatic brain insults

**All patients were subjected to:**

**I. Detailed history taking including:**

- History suggestive of neurological disorders e.g., convulsions, headache, photophobia or behavioral changes, age of first seizures, precipitating factors, duration of previous seizures, and any previous episodes of status epilepticus.
- History suggestive of developmental delay or psychiatric disorders (e.g., ADHD, anxiety, depression disorders)
- History suggests inborn error of metabolism (developmental delay, failure to thrive, organomegally, intellectual disability)
- History of drug intake, antiepileptic therapy (dose, duration, and compliance)
- Family history including consanguinity, similar family condition or family history of neurological disease.

**II. Clinical assessment:**

- Thorough clinical examination was including all body systems with special emphasis on neurological system.
- Vital signs on admission and on regular basis during hospital stay.
- Full neurological examination focused on signs of lateralization, brain herniation, size and reactivity of the pupils, pattern of respiration and abnormal motor activity (flaccidity, decorticate or decerebrate rigidity) and Glasgow Coma Scale (GCS) was performed after cessation of convulsion attacks to identify those who did not regain consciousness after cessation of seizures.

NB: seizures were considered of unknown etiology (idiopathic epilepsy): if previously healthy, no abnormal neurological examination and normal neuroimaging)<sup>[2]</sup>.

**II. Electroencephalographic (EEG) monitoring using digital EEG; NicoletOne, Natus Medical Incorporated (USA; 2014) was performed to all included children at admission to PICU to detect the abnormal EEG discharge and identify the type of seizures (generalized or focal).**

**III. Neuroimaging study:** Magnetic resonance imaging after stabilization of the condition (using Philips Healthcare device, Holland, 2010).

**IV. Laboratory investigations including:**

- **Complete blood count (CBC):** using Siemens SYSMEX XS-500 hematology analyzer.
- **Erythrocyte sedimentation rate (ESR) & C-reactive protein (CRP):** ESR: Using SD-1000 automated ESR analyzer. CRP: using Micro Analysis II multiple - channel dry- type fluoroimmunoassay quantitative analyzer.
- **Kidney function test:** serum urea, serum creatinine using the Automated Chemistry

Analyzer.

- **Liver function test:** serum alanine aminotransferase (ALT), serum aspartate aminotransferase (AST). SGM Italia: Kit for measurement of alanine – aminotransferase in serum or plasma- Kinetic UV optimized method IFCC (international Federation of clinical chemistry and laboratory medicine).
- **Serum glucose and electrolytes** (sodium, potassium, calcium, and magnesium). **Serum glucose** was measured using Ion selective electrode (ISE) Parnassus: Nova 8 CRT Mt. Zion: Gem Premier 3500
- **Arterial blood gases** to determine PH, partial pressure of carbon dioxide and partial pressure of oxygen in the blood.
- **CSF analysis for suspected cases of CNS infection:** Usually three tubes are collected. The first is used for chemical and serological analysis and the last two tubes are used for hematology and microbiology tests.

All laboratory tests were done at the central laboratory of pediatric hospital, Galaa and Ghamra military hospitals.

#### Statistical Analysis

Data were tabulated and analyzed using statistical package for social science (SPSS Inc., Chicago, Illinois, USA) version 22. Numerical data were expressed as mean and standard deviation (SD) and non-numerical data were expressed as number and percentage. Comparison between groups was done using student t-test if normal distribution data and Mann Whitney U test if non normal distribution of numerical data and chi square test for non-numerical data. Also, Pearson correlation coefficient (r) was used to measure the association between two quantitative variables. The level of significance was set as P-value <0.05 (95 % confidence interval).

#### RESULTS

Among our included children, the age at presentation by SE ranged from 6 months to 11 years with mean age of  $5.36 \pm 3.72$  years. Most of children presented by SE were younger than age of 5 years (45.2% were <2 years and 32.2% were 2-5 years). Regarding the gender, 74.2% were males. 29% had SE as 1st presentation while the remaining 77.4% were known cases of epilepsy. 45% developed 1<sup>st</sup> seizure at age younger than 2 years. The cause of seizures could not

be detected in 51.6% of cases (idiopathic epilepsy), while secondary epilepsy represents 25.8% of cases. Non epileptic causes included encephalitis (12.9%), febrile convulsion (atypical type) (6.5%) and acute hypoxia (3.2%). CSF analysis revealed that none of the studied children have meningitis. Based on electroencephalography, seizures were generalized in 83.9% of children and focal in 16.1% of them. Most of children were receiving AEDs (29% on monotherapy and 41.9% on polytherapy). The main precipitating factor for SE were missing doses in 29% of children followed by acute febrile illness in 12.9%, inappropriate AEDs in 9.7% and acute hypoxia in 6.5% of them, while precipitating factor could not be identified in 41.9% of children. Regarding the clinical characteristics of seizures, 77.4% of children had continuous seizures for > 30 minutes while 22.6% had recurrent seizures with impaired consciousness in between attacks for total duration exceed 30 minutes. In most cases (54.8%), seizures were terminated by using three AEDs while 2 cases needed to add general anesthesia (table 1 and figures 1-3).

Regarding routine laboratory investigations among our studied children, the mean hemoglobin level was  $10.90 \pm 1.86$  mg/dl, arterial blood gas revealed average PH of  $7.36 \pm 0.10$ , acute phase reactant was elevated in some cases: CRP ranged between 0.4-148 mg/dl and ESR ranged between 4-120 (table 2).

Regarding the MRI findings of children presented by SE, most of the children had normal MRI (64.5%). The commonest abnormalities were diffuse hypoxia and encephalitis (12.9% for each) followed by delayed myelination, focal cortical dysplasia, and mesial temporal sclerosis (3.2% for each) (table 3).

Regarding the outcome of studied children. 70.9% survives without neurological sequelae, 19.4% developed neurological sequelae (in the form of neurological motor defect, psychobehavioural impairment detected by clinical follow up for 3 months after SE) while 9.7% was died (table 4).

Comparison of clinical, laboratory, neuroimaging findings in relation to the outcome showed significant association between non epileptic etiology, febrile illness, hypoxia, elevated acute phase reactant (ESR, CRP) and poor outcome (table 5).

**Table (1): Clinical data of the studied children presented by status epilepticus (n=31)**

Clinical data	(n=31)
<b>Age (years)</b>	
Range	2 -11
Mean ±SD	5.36±3.72
<b>Sex</b>	
Female	8 (25.8%)
Male	23 (74.2%)
<b>Age at 1<sup>st</sup> seizure (years)</b>	
Range	6 months-7years
Mean ±SD	2.45±2.59
<b>Age distribution at 1<sup>st</sup> seizure</b>	
<2 years	14 (45.2%)
2-5years	10 (32.2%)
>5 years	7 (22.6%)
<b>Age distribution at SE</b>	
<2 years	9 (29.1%)
2-5years	7 (22.6%)
>5 years	15 (48.3%)
<b>Etiology of seizures</b>	
Idiopathic epilepsy (genetic, unknown etiology)	16 (51.6%)
Secondary epilepsy (symptomatic)	8 (25.8%)
Encephalitis	4 (12.9%)
Febrile convulsion	2 (6.5%)
Acute hypoxia	1(3.2%)
<b>Clinical characteristics of seizures</b>	
Continuous seizures for > 30 minutes	24 (77.4%)
Recurrent seizures with impaired consciousness	7 (22.6%)
<b>Type of seizures (based on EEG)</b>	
Focal seizures	5 (16.1%)
Generalized seizures	26 (83.9%)
<b>Previous antiepileptic drugs</b>	
Mono therapy	9 (29.0%)
Poly therapy	13 (41.9%)
Not on AEDs	9 (29.0%)
<b>Precipitating factors for SE:</b>	
Hypoxia	2 (6.5%)
Inappropriate AED type	3 (9.7%)
Febrile Illness	4 (12.9%)
Missing Doses	9 (29.0%)
Unknown	13(42%)
<b>Duration of convulsive seizures (hours)</b>	
Range	30 minutes –5 days
Mean ±SD	8.19±6.65
<b>Antiepileptic drugs used to control seizures</b>	
Two AEDs	12 (38.7%)
Three AEDs	17 (54.8%)
>3 AEDS + general anesthesia	2 (6.5%)

SE: status epilepticus; AEDs: antiepileptic drugs; EEG: electroencephalography

**Table (2): Laboratory data of the studied children presented by status epilepticus**

Laboratory data	Range	Mean ±SD
WBCs (x10 <sup>3</sup> /mm <sup>3</sup> )	3.8-14	7.92±2.90
Hemoglobin (gm/dl)	7.7-14.8	10.90±1.86
Platelets (x10 <sup>3</sup> /mm <sup>3</sup> )	118-460	285.84±98.49
Total Calcium (mEq/dl)	6.8-10.2	8.65±0.88
Serum glucose (mg/dl)	78-256	152.06±49.98
Urea (mg/dl)	10-32	19.55±4.78
Creatinine (mg/dl)	0.2-0.9	0.45±0.19
Serum sodium(mEq/dl)	113-155	134.65±9.31
Serum potassium (mEq/dl)	2.3-6.4	3.62±0.88
Serum magnesium (mEq/dl)	1.8-3.3	2.51±0.45
AST (mg/dl)	10-60	29.39±12.75
ALT (mg/dl)	7-50	21.00±10.87
C-RP (mg/dl)	0.4-148	18.90±31.18
ESR	4-120	24.42±33.09
pH	7.02-7.48	7.36±0.10
HCO <sub>3</sub>	10-33	24.92±5.13
PaCO <sub>2</sub>	25-71	38.84±10.56

ALT: alanine aminotransferase, AST: aspartate aminotransferase

**Table (3): Magnetic resonance imaging findings of children presented by status epilepticus**

MRI	Total (no=31)
Diffuse hypoxia	4(12.9%)
Encephalitis	4(12.9%)
Delayed myelination	1(3.2%)
Focal cortical dysplasia	1(3.2%)
Mesial temporal sclerosis	1(3.2%)
Normal	20 (64.5%)

**Table (4): The outcome of the studied children presented by status epilepticus**

Outcome	Total (no=31)
Survivors without neurological squeals	22 (70.9%)
Survivors with neurological squeals	6 (19.4%)
Died	3 (9.7%)

**Table (5): The relation of clinical, laboratory and magnetic resonant imaging findings to the outcome of status epilepticus**

Item	Favorable outcome (n=22)	Unfavorable outcome (n=9)	Chi square test/ independent student t test/ Mann Whitney U test	
			$\chi^2/t$	p-value
<b>Precipitating factors</b>				
Unknown	12 (54.5%)	1 (11.1%)	12.094	0.017*
Missing Doses	7 (31.8%)	2 (22.2%)		
Febrile Illness	1 (4.5%)	3 (33.4%)		
Hypoxia	0 (0%)	2 (22.2%)		
Inappropriate AED	2 (9%)	1 (11.1%)		
<b>MRI findings</b>				
Normal	16 (72.7%)	4 (44.4%)	2.232	0.135
abnormal	6 (27.3%)	5 (55.6%)		
<b>Type of seizures</b>				
Generalized	17 (77.3%)	9 (100%)	2.439	0.118
Focal	5 (22.7%)	0 (0%)		
<b>Cause of SE</b>				
Epilepsy	20 (90.1%)	4 (44.4%)	0.788	0.005**
Not epilepsy	2 (9.9%)	5 (55.6%)		
<b>Sex</b>				
Male	16 (72.7%)	7 (77.8%)	0.085	0.771
Female	6 (27.3%)	2 (22.2%)		
<b>Age (years)</b>	5.491 ± 3.501	5.122 ± 4.338	0.227	0.824
<b>SE Duration (hour)</b>	10.257 ± 26.057	9.580 ± 23.504	0.070	0.945
<b>WBCs (x103/mm<sup>3</sup>)</b>	7.486 ± 2.863	8.967 ± 2.873	-1.303	0.212
<b>Hemoglobin gm/dl</b>	11.091 ± 2.010	10.433 ± 1.432	1.025	0.317
<b>Platelets (x103/mm<sup>3</sup>)</b>	271.000 ± 93.735	322.111 ± 105.940	-1.260	0.229
<b>Calcium (mEq/dl)</b>	8.573 ± 0.948	8.822 ± 0.721	-0.795	0.436
<b>Serum glucose (mg/dl)</b>	142.455 ± 49.835	175.556 ± 44.444	-1.816	0.087
<b>Urea (mg/dl)</b>	18.818 ± 4.625	21.333 ± 4.950	-1.309	0.212
<b>Creatinine (mg/dl)</b>	0.477 ± 0.207	0.400 ± 0.112	1.338	0.192
<b>Serum sodium (mEq/dl)</b>	133.000 ± 9.376	138.667 ± 8.276	-1.663	0.115
<b>Serum potassium (mEq/dl)</b>	3.755 ± 0.886	3.278 ± 0.803	1.456	0.164
<b>Serum magnesium (mEq/dl)</b>	2.546 ± 0.413	2.422 ± 0.561	0.597	0.562
<b>AST (mg/dl)</b>	29.455 ± 12.850	172.556 ± 441.590	-0.972	0.359
<b>ALT (mg/dl)</b>	20.273 ± 11.059	139.444 ± 360.226	-0.992	0.350
<b>CRP (mg/dl)</b>	9.182 ± 9.978	42.667 ± 49.900	-2.924	0.019*
<b>ESR</b>	14.818 ± 24.272	47.889 ± 41.074	-2.347	0.047*
<b>pH</b>	7.351 ± 0.114	7.374 ± 0.049	-0.803	0.428
<b>HCO<sub>3</sub></b>	25.159 ± 5.755	24.344 ± 3.326	0.493	0.627
<b>PaCO<sub>2</sub></b>	38.773 ± 10.619	39.000 ± 11.068	-0.053	0.959

SE: status epilepticus; AEDs: antiepileptic drugs, ESR: erythrocyte sedimentation rate; un-favorable outcome: Survivors with neurological sequelae (6 cases) and died (3 cases), \*: p-value<0.05.



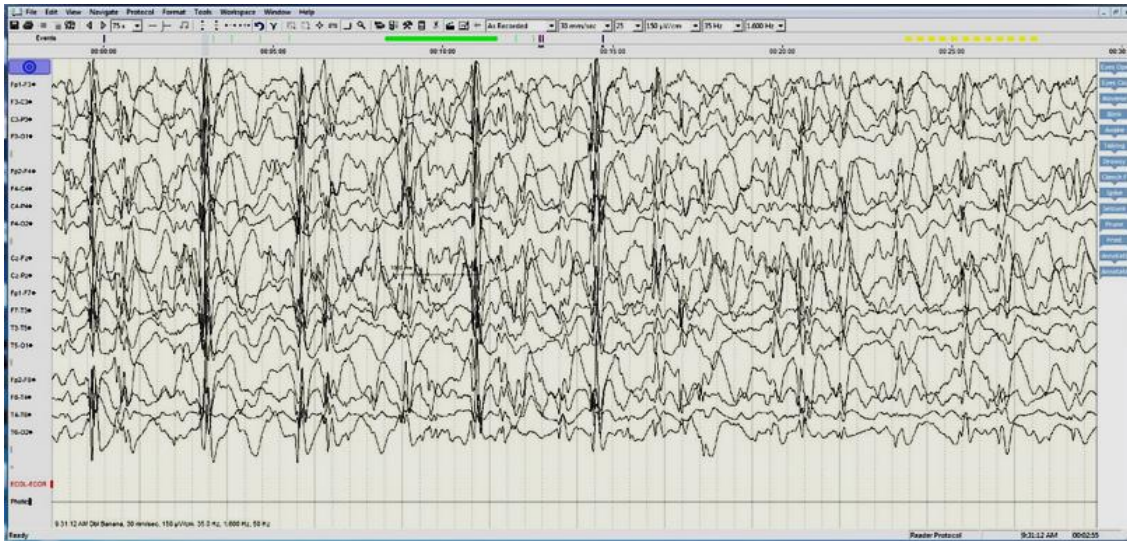


Figure 1: EEG monitors of 5-year male child with convulsive status epilepticus

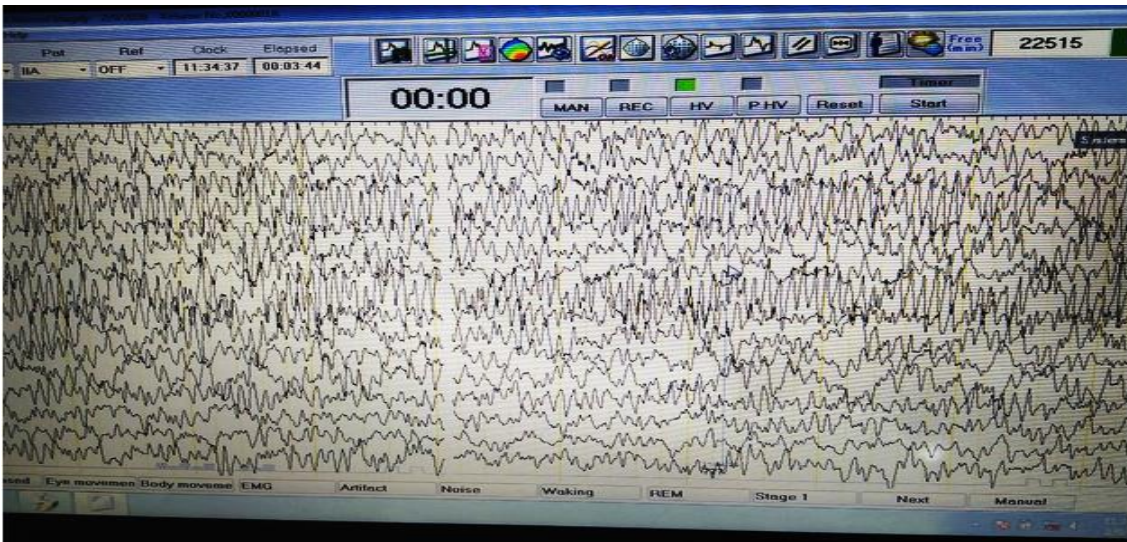


Figure (2): EEG monitor of 8-year-old female with non-convulsive status epilepticus after cessation of convulsive status epilepticus



Figure (3): EEG monitor show termination of non-convulsive status epilepticus after midazolam injection (7-year-old male child known case of landau kleffner epileptic syndrome)

## DISCUSSION

Despite advance in treatment of SE, it is still associated with mortality and morbidity. Identifying the clinical characteristics, pathophysiology and risk factors for poor prognosis is an area of great interest with conflicting results due to the heterogeneity of this disorder<sup>[7]</sup>.

Our study revealed higher incidence of SE is higher in children younger than age of 5 years (45.2% were <2 years and 32.2% were 2-5 years). The male predominance among children with SE in our study agrees with Tiamkao et al.<sup>[8]</sup> and Dham et al.<sup>9</sup> who reported that males are more likely to suffer from SE than females. Ong et al.<sup>[10]</sup> demonstrated that the age distribution in children with SE showed a higher incidence at under 5 years of age (10.18/100,000 person-years) and the incidence of SE was higher in males than females with an overall male-to-female rate ratio of 1.57. The cause for male predominance is unclear. Some studies suggested that hormonal factors may account for the observed gender difference during adolescence. Sex may influence the pathogenesis of developmental brain injuries because immature brain appears to be strongly influenced by the intrinsic difference between male and female brain cells due to their distinct chromosomal complements.<sup>[11]</sup> The cause for the higher incidence in younger children may be attributed to immature mechanism for control of seizures activity in younger age<sup>[12]</sup>.

Our study revealed high incidence of SE among children with epilepsy (77.4%); 29% had SE as 1st presentation (22.6% were due to non-epileptic causes while 6.4% progressed to epilepsy). Similar findings were reported by Bergin et al.<sup>[11]</sup> who found that about 70% of children with SE were known cases of epilepsy (29% were on monotherapy and 42% were on polytherapy) while 29% had no previous seizure attacks and SE was an initial presentation. The frequency of epilepsy in our study is much more than Nishiyama et al.<sup>[13]</sup> who found that 34 (28.3%) of the 120 patients had been diagnosed with epilepsy. Das et al.<sup>[14]</sup> reported that 63.82% of children who developed SE were younger than five years of age and 64.9% of children presented by SE as the first attack of seizure and 35.1% had previously repeated seizures; drug default was the main predisposing factor for SE in 42.4% of children. This contradiction could be explained by differences in definitions, classification of seizures, and etiologic criteria in epidemiological studies on epilepsy have often yielded discordant results between studies leading to controversial results that reflect differences in genetically determined susceptibility<sup>[12]</sup>.

Regarding the characteristics of seizures in children

presented by SE, generalized seizures were the predominant form of convulsion among our studied children with no significant association between the type of seizures and the outcome. In accordance with our findings, Kumar et al.<sup>[15]</sup> demonstrated that generalized seizures are the predominant type of convulsion in children presented by SE (91%). Das et al.<sup>[14]</sup> found no significant difference between the mortality and the type of seizures.

Our study showed that missed dose of antiepileptic medications or using inappropriate type of antiepileptic drugs (AEDs) were the commonest precipitating factor for SE in children with epilepsy while hypoxia and febrile illness were more common in non-epileptic children. Prognosis was significantly worse in non-epileptic children; hypoxia and encephalitis have the highest morbidity and mortality. As acute phase reactants level increased during infection, so it was not a surprise that elevated ESR, and CRP are associated with worse outcome. Supporting our findings Sculier et al.<sup>[16]</sup> concluded that etiology of SE is the main determinant of outcome, and the effect of age or SE duration is often difficult to distinguish from the underlying cause. Kravljanc et al.<sup>[17]</sup> found that neurologic sequels occurred in 12.9% of children with SE and mortality rate was 9.3%. Etiology and prior neurologic abnormalities were the major predictors of poor outcome. Sutter et al.<sup>[18]</sup> demonstrated that increased C- reactive protein levels at status epilepticus onset were associated with higher rates of refractory SEs and death (with every 1mg/ç: odds ratio 1.01, 95% confidence interval 1.00- 1.02, p = 0.021; odds ratio 1.01, 95% confidence interval 1.00-1.02, p < 0.007, respectively).

As this study was single center study, so a major limitation of the current study is the small number of included children. Further large-scale studies are required to identify children at higher risk to develop SE and those with poor outcome to allow early optimal intervention.

## CONCLUSION

Despite that epilepsy is the main cause of SE, non-epileptic causes are more serious and are associated with worse outcome. Male gender, poor compliance to antiepileptic medications and febrile illness are linked to development to SE in children. SE induced by hypoxia and encephalitis has the highest morbidity and mortality.

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## الملخص العربي

### الخصائص السريرية و نتائج الاطفال المصابين بحالة الصرع المتشنجه

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#### ملخص البحث

**الخلفية:** تمثل التشنجات المستمرة حاله طارئه و خطيرة في طب الاعصاب حيث ترتبط بزيادة حدوث المضاعفات والوفيات و لهذا فهناك اهتمام شديد بفهم الخصائص السريرية و طرق تشخيص الأطفال المصابين بنوبات تشنجات مستمرة

**الهدف:** التعرف على الاعراض السريرية و المظاهر التشخيصي للأطفال الذين يعانون من تشنجات مستمرة.

**الطرق:** اجريت هذه الدراسة التحليلية الوصفية على 31 طفلاً يعانون من تشنجات مستمرة استوجبت إدخالهم إلى وحدة العناية المركزة للأطفال. تم رصد الاعراض السريرية و عمل صورته دم كامله و قياس البروتين التفاعلي C و معدل ترسيب كرات الدم الحمراء و تحليل نسبة الجلوكوز و الاملاح في الدم وتحليل السائل النخاعي. كما تم إجراء التصوير بالرنين المغناطيسي لجميع الأطفال المشمولين في الدراسة.

**النتائج:** كان عمر معظم الأطفال الذين يعانون من نوبات تشنجات مستمرة أصغر من 5 سنوات (45.2 % كانوا أقل من سنتين و 32.2 % من 2-5 سنوات). 74.2 % كانوا ذكور و 83.9 % لديهم نوبات تشمل الجسم بالكامل. وجد أن 29 % كانت هذه هي المرة الأولى لحدوث تشنجات بينما 77.4 % قد سبق تشخيص اصابتهم بمرض الصرع. بالإضافة لمرض الصرع اظهرت الدراسة وجود اسباب اخري للتشنجات المستمرة ممثله في التهاب المخ (12.9 %) و التشنج الحراري (6.5 %) و نقص الأوكسجين الحاد (الاختناق) (3.2 %). كان العامل المحفز الرئيسي لحدوث التشنجات المستمرة هو عدم تناول جرعات الأدوية المضادة للتشنجات في 29 % من الحالات، والإصابة بالحمى في 12.9 % ، و نقص الأوكسجين الحاد في 6.5 % ، بينما تعذر تحديد السبب في 41.9 % من الأطفال. كان التصوير بالرنين المغناطيسي طبيعياً في 64.5 % من الاطفال بينما اظهر الرنين المغناطيسي للمخ تغيرات ناتجه عن نقص الأوكسجين و التهاب المخ في نسبه 12.9 % من الاطفال لكل منهما. تبين أن 70.9 % من الأطفال قد تعافوا دون حدوث إعاقة عصبية ، و 19.4 % أصيبوا بإعاقة عصبية بينما توفي 9.7 % من الاطفال. اظهرت الدراسة و جود ارتباط كبير بين المسببات غير الصرعية و الحمى و نقص الأوكسجين و ارتفاع دلالات الالتهاب في الدم (معدل ترسيب كريات الدم الحمراء و البروتين التفاعلي C) و حدوث المضاعفات والوفيات.

**الاستنتاجات:** على الرغم من أن الصرع هو السبب الرئيسي لنوبات التشنجات المستمرة ، إلا أن المسببات غير الصرعية هيا الأكثر خطورة و ترتبط بنتائج أسوأ. تزداد معدلات حدوث نوبات التشنجات المستمرة لدي الذكور و الأطفال غير الملتزمين بتناول الأدوية المضادة للصرع و عند الإصابة بالحمى لدي الأطفال. تزداد معدلات المضاعفات والوفيات في حالات التشنجات المستمرة الناتجة عن نقص الأوكسجين و التهاب المخ في الاطفال.

**الكلمات المفتاحية:** تشنجات مستمرة، التشنجات، النتيجة، الأطفال.

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