Role of neuroimaging and electroencephalogram in first unprovoked seizures in children from Cairo

Mohamed S. Elfeshawy[a], Ali A. Afia[b], Ahmed Y. Alsawah[b], Mahmoud I. Mohammed[b], Ann A. Abdelkader[c]

Background First unprovoked seizure (FUS) and complex febrile seizure (CFS) are common pediatric issues of great debate with respect to the role of electroencephalogram (EEG) and neuroimaging in their diagnosis.

Aim To determine the frequency of abnormal EEG and neuroimaging results in children with FUS and CFS and to detect the correlation between EEG and neuroimaging results.

Patients and methods A total of 100 children (6 months to 12 years of age), who presented with first afebrile or CFS underwent EEG and neuroimaging (computed tomography and/or MRI). This was a prospective randomized controlled trial.

Results A total of 100 cases within the age group 6 months to 12 years were recruited. FUS was seen in 63 cases and CFS in 37 cases. Overall, 69.8% cases of FUS were generalized and 30.2% were focal. The prevalence of EEG abnormality was found in 33% of the whole studied population: 44.4% in patients with FUS and 13.5% in patients with CFS. The prevalence of neuroimaging abnormality was found in 15% of the whole studied population: 20.6% in patients with FUS and 5.4% in patients with CFS. Neuroimaging abnormality was seen more commonly in those patients who had an abnormal EEG, with a statistically significant increase in cases with FUS.

Conclusion EEG and neuroimaging abnormalities were more prevalent in children with FUSs than those with CFSs. Abnormal EEG and neuroimaging were more common in children with partial seizures than those with generalized seizures. Neuroimaging was abnormal in a significant number of children having abnormal EEG, so neurologically free patients having normal EEG can be safely discharged without neuroimaging, if follow-up is assured. When EEG is abnormal in FUS, the probability of having abnormal neuroimaging increases as compared with those cases where EEG is normal. In case of generalized seizures, patients with abnormal EEG may have abnormal computed tomography/MRI scans, but there are fewer possibilities of a patient with abnormal EEG to have a normal neuroimaging. In partial seizures, abnormal EEG increases the risk of having abnormal neuroimaging than in generalized seizures, and normal EEG in partial seizures markedly decreases the risk of having an abnormal neuroimaging generalized seizures. CFSs in otherwise neurologically free children rarely indicate the presence of lesion on neuroimaging even if associated with EEG abnormalities. Neuroimaging abnormalities in neurologically free children with FUS and CFSs do not require urgent intervention.

Sci J Al-Azhar Med Fac, Girls 2019 3:503–516

The Scientific Journal of Al-Azhar Medical Faculty, Girls
2019 3:503–516

Keywords: MRI, neurology, seizures

Departments of, [a]Radiodiagnosis, [b]Pediatric, Faculty of Medicine (For Boys), Al Azhar University, Cairo, [c]Department of Clinical Neurophysiology, Faculty of Medicine, Cairo University, Giza, Egypt

Correspondence to Mohamed S. Elfeshawy, MD, Lecturer of Radio Diagnosis, Faculty of Medicine (for boys), Al Azhar university in Cairo, Radiology Department, Al-Hussien University Hospital, 1st Al-Azhar street, Gamaliyya, Cairo, 11311, Egypt. Tel: +201007700167; e-mail: mst122@yahoo.com

Received 20 February 2019 **Accepted** 16 June 2019

Introduction

Seizure is a common neurological problem in the pediatric age group and occurs in 10% of children. Approximately 5% of all medical attendances to accident and emergency department are related to seizures [1].

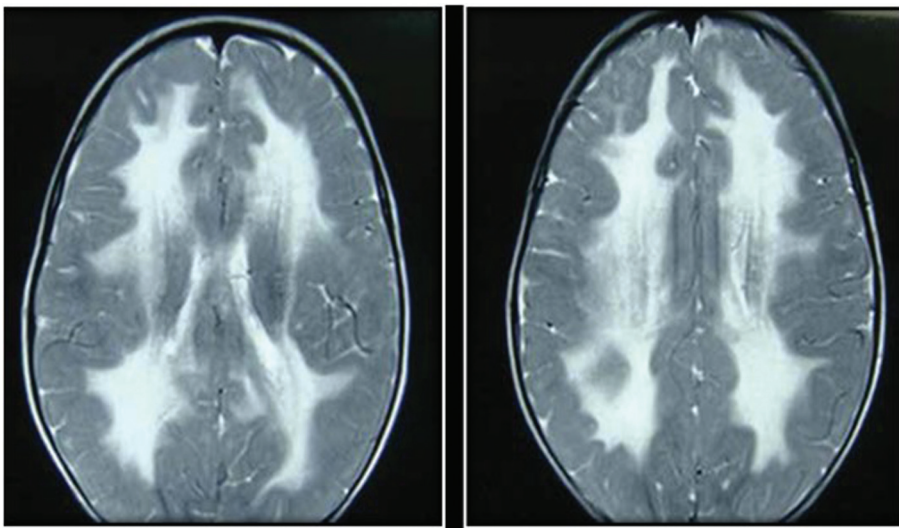
Unprovoked seizures occur in patients older than one month and have no acute precipitant, but improvement of the diagnostic techniques increases the chance of finding the unknown causes [1].

Febrile seizures (FS) are the most common type of childhood seizures. It is also a common cause of pediatric hospital admission and parental concern. The reported incidence of FSs is up to 14%. FS is further classified as simple febrile seizures (SFS) or complex febrile seizures (CFS), with CFSs defined as seizure lasting more than 15 min, repeated seizures occurring within 24 h, and focal seizure activity or focal findings present during the postictal period. Although there are enough investigations and data concerning initial management, there is somewhat less well-developed data in CFSs [2].

Neuroimaging

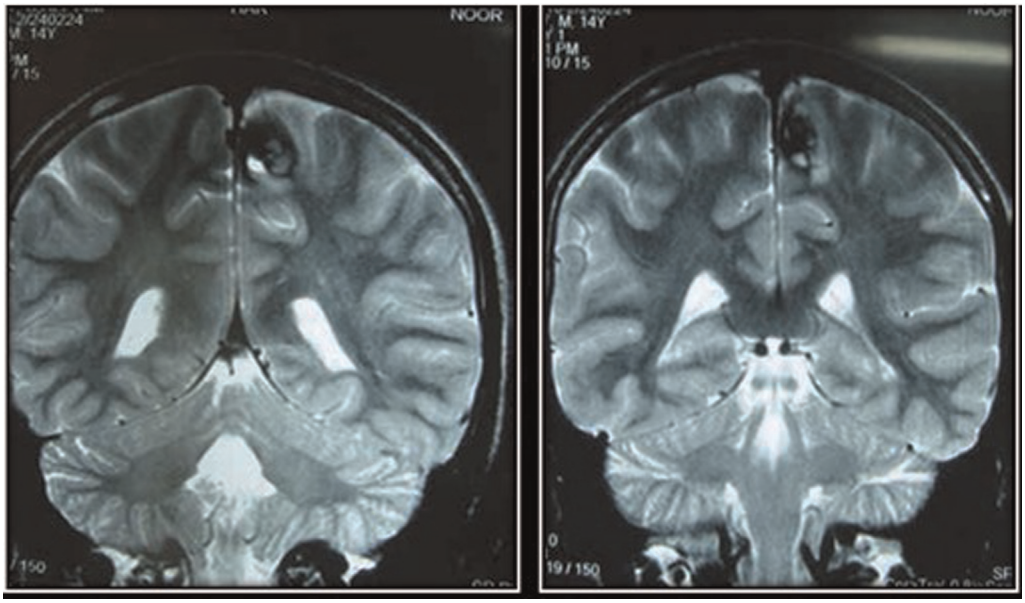
Insufficient evidence is available to make a standard recommendation or guideline for the use of routine neuroimaging in children with first unprovoked seizure

Figure 1



An 18-month-old boy with first unprovoked seizure: axial T2Wimages reveal bilateral butterfly abnormal signal extending from the subcortical U fibers to the periventricular deep white matte changes owing to metachromatic leukodystrophy [9].

Figure 2



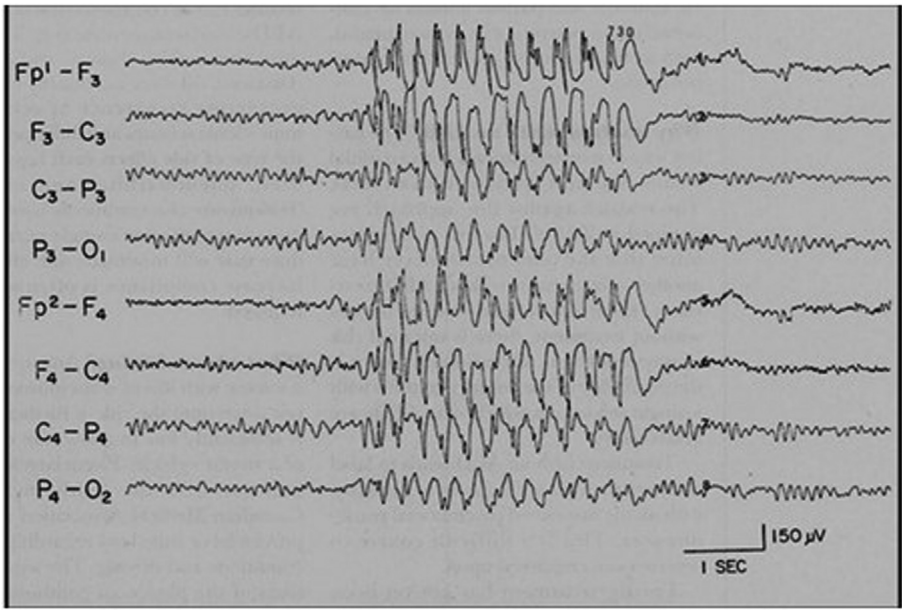
A 13-year-old boy with first unprovoked seizure. Coronal T2W images through occipital horns reveal signal void left parasagittal lesion owing to A-V malformation [9].

(FUS). In contrast, guidelines for obtaining neuroimaging in adult patients presenting with seizure have been published. In a few studies that have reviewed the yield of neuroimaging in children with unprovoked seizure, the prevalence of abnormalities ranged from 0 to 21% [3].

The decision of doing neuroimaging in FUSs should be individualized, and electroencephalogram (EEG) can be helpful. For example, a focal EEG may increase suspicion of structural abnormality. Patients who have clearly defined epileptic syndromes, such as petit mal epilepsy or benign Rolandic epilepsy, do not necessarily require neuroimaging. AAN practice parameters recommend nonurgent imaging after initial seizure if there is associated significant cognitive or motor impairment, unexplained abnormalities on the neurological examination, partial-onset seizures, an EEG inconsistent with benign or primary generalized epilepsy, and in patients younger than 1 year [4].

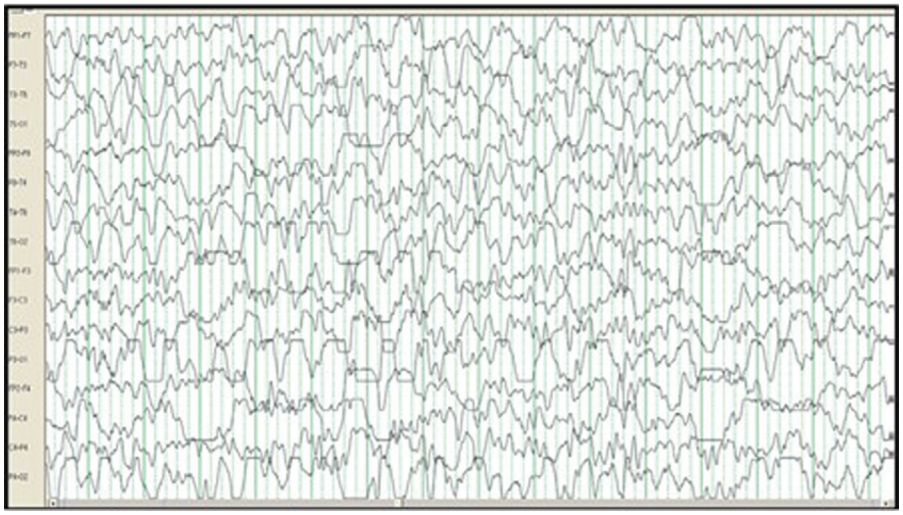
Clinically significant neuroimaging abnormalities have been reported in 2% of children presenting with first afebrile seizure without focal features or predisposing conditions [5].

Figure 3



Bursts of generalized spike waves at 1–4 s in patient with a single generalized tonic-clonic convulsion, indicating a higher probability of seizure recurrence [14].

Figure 4



Generalized slowing recorded a day after a febrile seizure in a 27-month-old girl [14].

The prevalence of abnormal neuroimaging in an adult with a new-onset seizure is 34–45%. However, the role of emergent neuroimaging in children presenting with first afebrile seizure is still not well defined. Based on several studies, the prevalence of abnormal neuroimaging in pediatric patients with a new-onset afebrile seizure is from 0 to 21% [6].

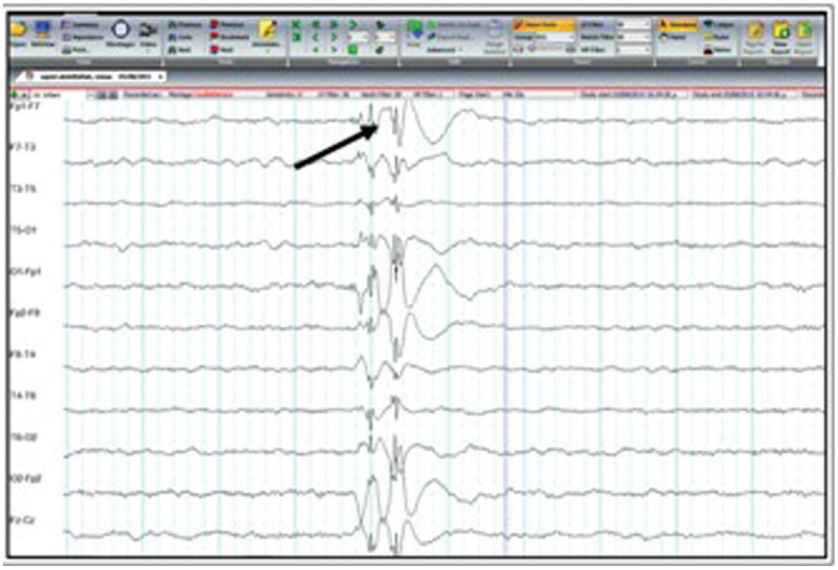
If neuroimaging is obtained, MRI is the preferred method of imaging to avoid radiation exposure while providing more detailed diagnostic information [7].

Identification of lesions altered the acute medical or surgical management in up to 8% of children. Accordingly, AAN recommends considering emergent head computed tomography (CT) for all patients with a first seizure, particularly those who have risk factors for abnormal neuroimaging (Figs 1 and 2) [8,9].

Electroencephalography

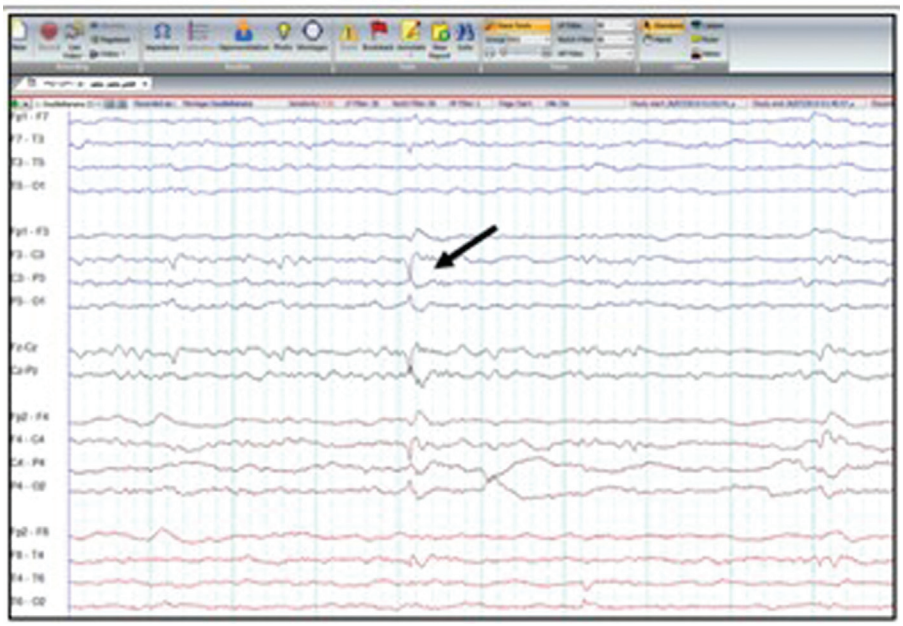
The EEG, which is entirely harmless and relatively inexpensive, is the most important investigative tool in the diagnosis and management of epilepsies. However,

Figure 5



Generalized sharp slow waves in 6-month-old girl with first seizure.

Figure 6



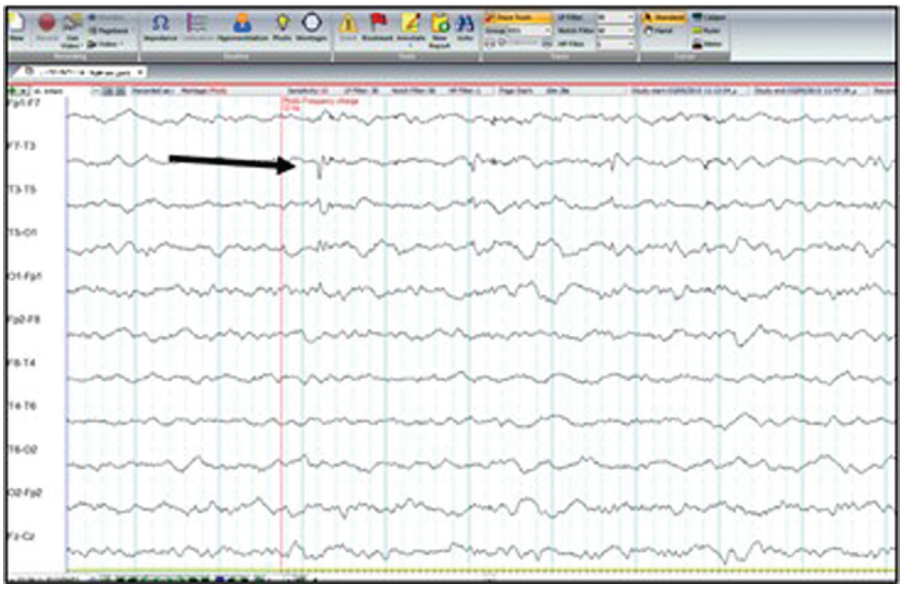
Bilateral centro-temporal spikes waves in a 12-year-old boy with first Rolandic seizure.

for the EEG to provide accurate assessments, it must be properly performed by experienced technologists and carefully studied and interpreted in the context of a well-described clinical setting by experienced physicians [10].

EEG does not generally need to be obtained in the emergency department unless there is concern about nonconvulsive status epilepticus or ongoing seizures in a pharmacologically paralyzed or comatose patient. EEG can detect focal lesions not visible with neuroimaging and show epileptiform findings that allow diagnosis of particular epilepsy syndromes [11].

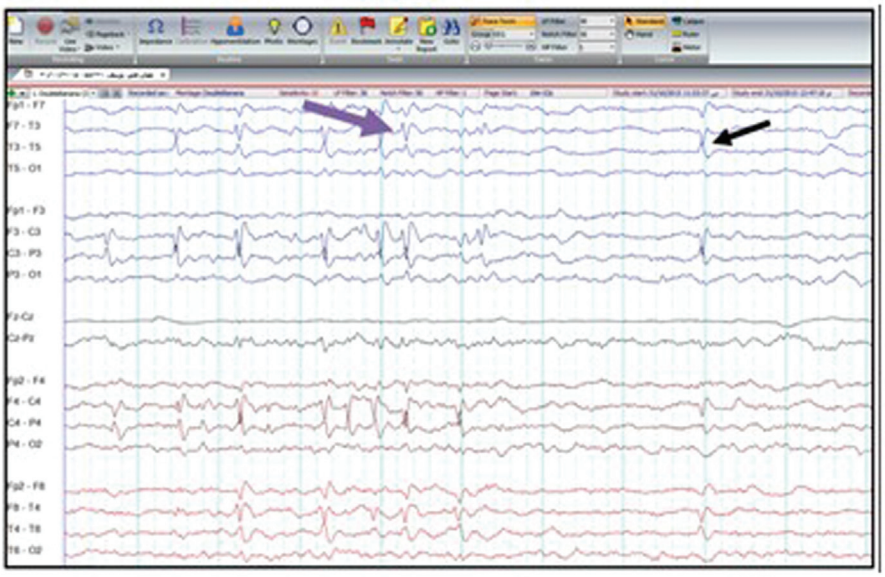
AAN and Child Neurology Society recommend an EEG as a standard of care for a child presenting with a first afebrile seizure. The EEG is useful to evaluate risk of seizure recurrence, to determine whether a seizure is focal or generalized, to screen for focal abnormalities and possible need for MRI, to identify epilepsy syndrome classification, to guide choice of antiepileptics, and to aid in prognosis [12].

Figure 7



Left temporal sharp waves in 1.5-year-old boy with first focal seizure.

Figure 8



Bilateral centro-temporal sharp (black arrow) and sharp slow (move arrow) waves in a 8-year-old boy with first generalized seizure.

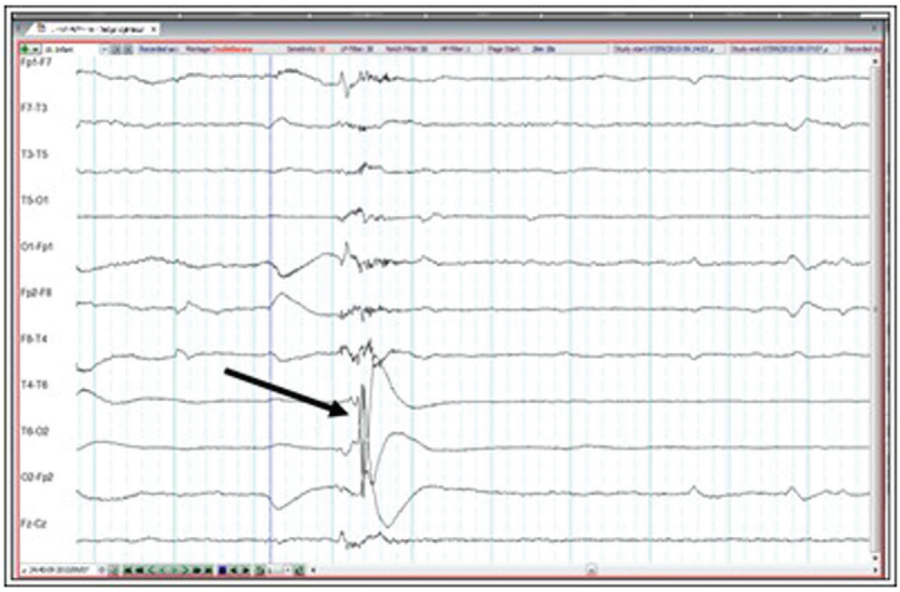
An EEG discharge is of great diagnostic and management significance if it is associated with clinical manifestations. However, these symptoms may be minor and escape recognition without video recordings. Video-EEG recordings are particularly important in the identification of absences, which are easily elicited by hyperventilation, myoclonic jerks, or focal seizures, as well as psychogenic or other nonepileptic seizures, particularly those of the hyperventilation syndrome (Figs 3 and 4) [13,14].

If initial EEG findings are negative and clinical suspicion for epilepsy is high, sleep-deprived EEG is beneficial. This test detects abnormalities in 21–35% of patients with initially normal EEG findings, with the highest yield in the first 3 days after the seizure [15].

Patients and methods

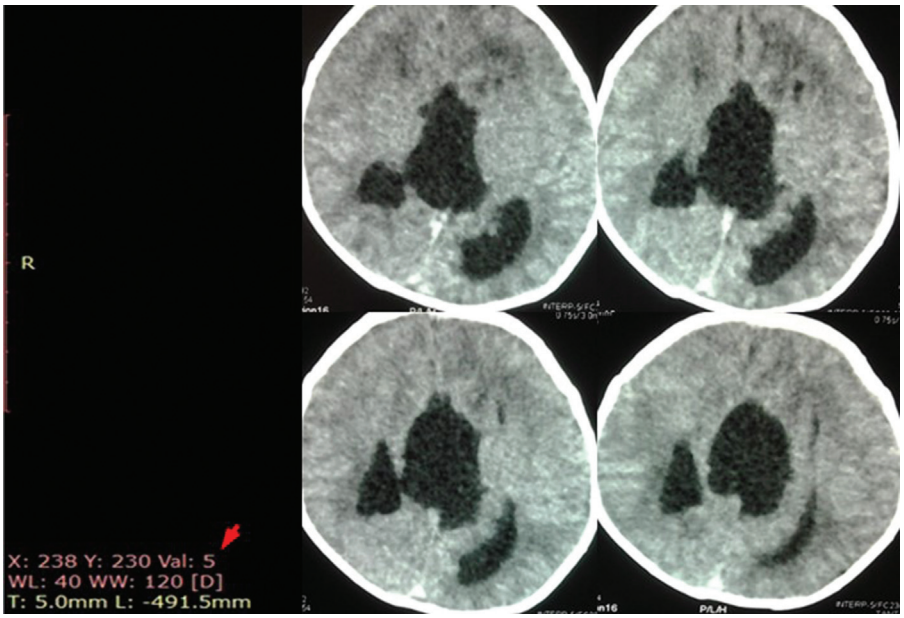
This prospective study was carried out on 100 cases with FUS and CFS in the age group from 6 months to 12 years old attending the Emergency, Inpatient, and

Figure 9



Generalized polyspike activity in a 4-year-old boy with complex febrile seizure.

Figure 10



Case no. 1. computed tomography scan shows interhemispheric well-defined cystic lesion iso-attenuating to the cerebrospinal fluid (CSF) equivalent to 5 HU; such cystic lesions together with coexisting corpus callosum agenesis splay the bodies of both lateral ventricles.

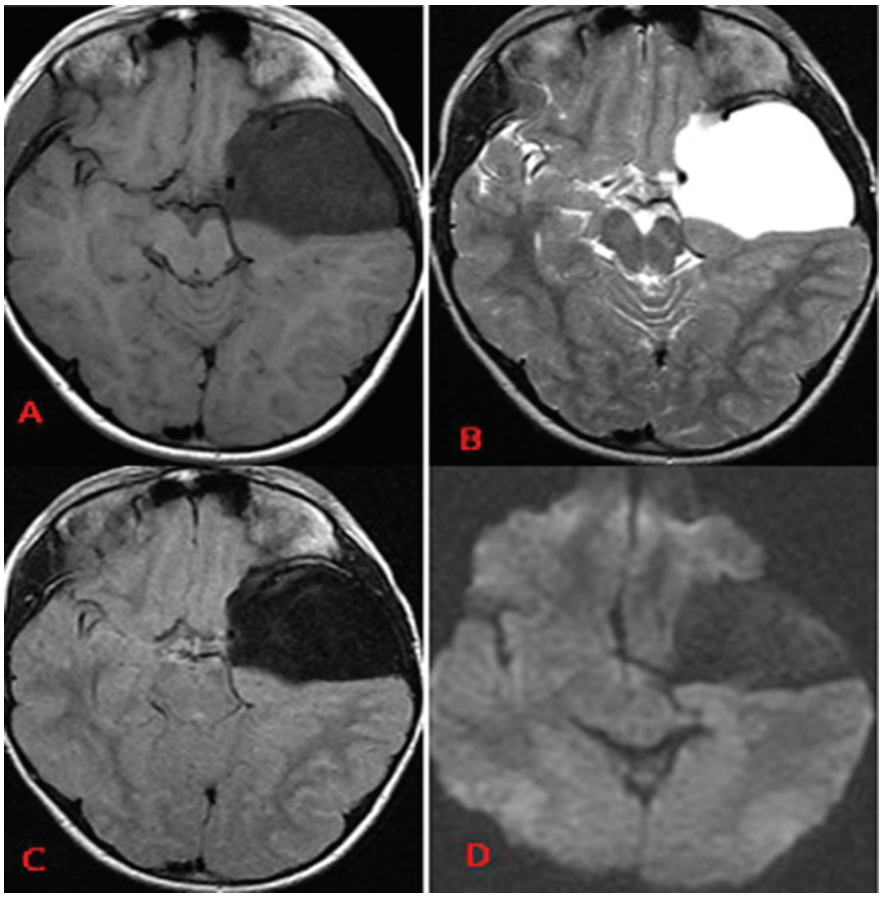
Outpatient Departments of AL Hussein and Sayed Galal University Hospitals during the period from July 2016 to December 2017.

Inclusion criteria

(1) FUS:
FUS includes one or more epileptic seizures occurring within 24 h period with recovery of consciousness between seizures (ILAE Commission Report, 1997). The occurrence of multiple seizures in a 24-h period is considered as one seizure [16].

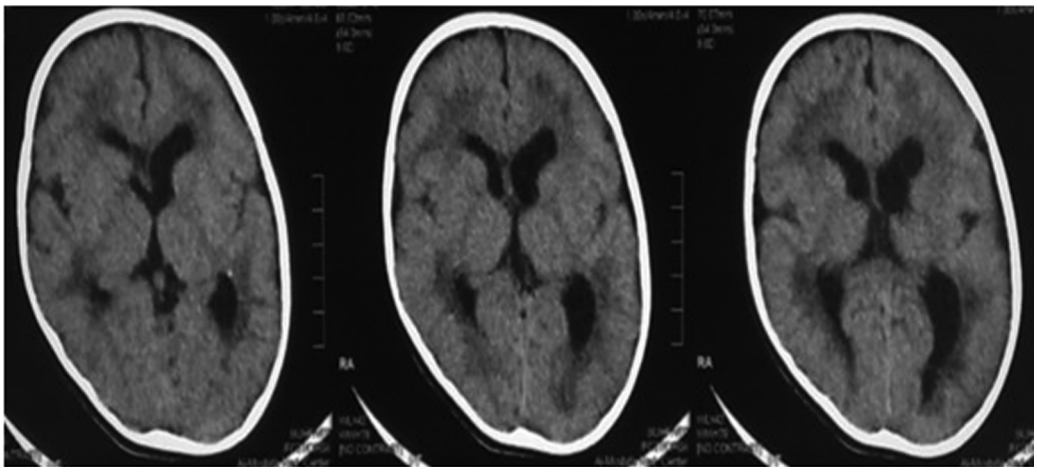
(2) CFSs: patient with FSs having one or more of the following features:
   (a) Longer duration (>15 min).
   (b) Recurs within 24 h.
   (c) Focal seizures or general types of seizures other than generalized tonic-clonic seizure (GTCs).
   (d) No post-ictal neurological abnormalities.

Figure 11



Case no. 2. (a) Axial T1 WI, (b) axial T2 WI, (c) axial FLAIR, and (d) diffusion WI show left basi temporal extra-axial arachnoid cyst that follows the cerebrospinal fluid (CSF) signal at all pulse sequences.

Figure 12



Case no. 3. Selected computed tomography cuts at the level of sylvain fissure show evidence of lissencephaly and mild ventricuolmegaly with periventricular deep white matter volume loss.

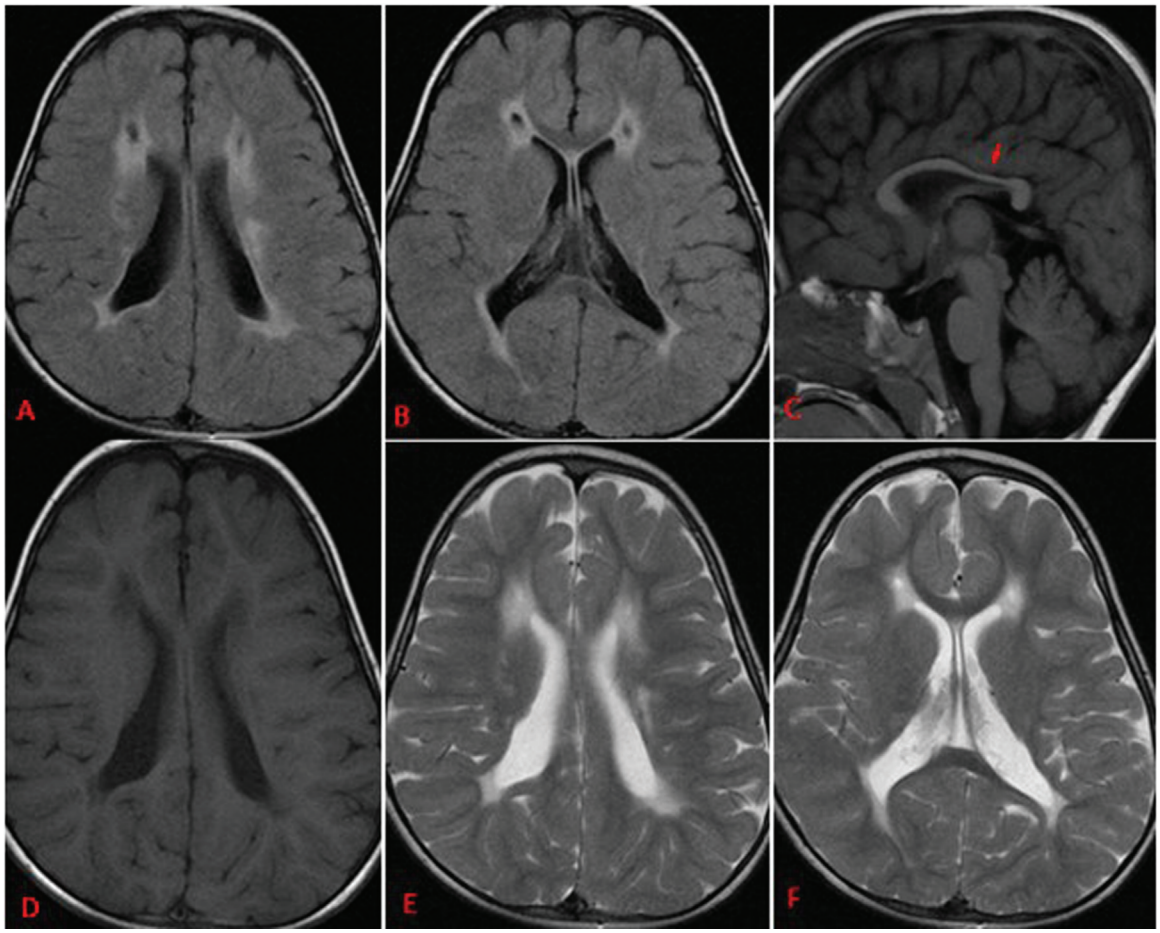
(e) Family history of epilepsy.
(f) Age group from 6 months to 12 years.

Exclusion criteria

The following were the exclusion criteria:

(1) Patients with suspected central nervous system (CNS) infection.
(2) Patients with simple febrile convulsions (because there is no suspected neurological insult).

Figure 13



Case no. 4. Selected axial FLAIR (a, b), sagittal T1 WI (c), axial T1 WI (d), and selected axial T2 WIs (e, f) cuts revealed patchy areas of abnormal high T2 and FLAIR signal are seen in deep white matter periventricular regions of both cerebral hemispheres associated with reduced bilateral peri-trigonal white matter volume and irregular tenting of atria of lateral ventricles. Partial thinning out of the posterior corpus callosum (arrowed), likely representing sequelae of peri-natal hypoxic/ischemic insult (Partial prolonged type).

(3) Patients with acute etiology of seizures such as toxins, trauma, drugs, and metabolic or electrolyte disturbances.
(4) Patients with known chronic neurologic illnesses such as cerebral palsy (CP) or mental retardation.
(5) Patients with neurological abnormalities detected at examination.

All patients were subjected to the following:

(1) Informed consent from a parent or guardian.
(2) Thorough history taking, including developmental, neurological, and family history (of convulsion or neurologic disease), emphasizing on the following:
    (a) Precipitating factor for seizure, such as fever, trauma, drug intake, metabolic abnormality, prior CNS abnormality, and endocrinal or gastrointestinal tract (GIT) disease.
    (b) Complete analysis of the attack, including presence of aura, onset of the seizure (focal or generalized onset), type and duration of convulsion, conscious level during the attack, if associated with cyanosis, incontinence of urine or vocalization, frequency of the attack, and post-ictal state.
(3) Full clinical examination was done focusing on neurological examination, origin of fever (if CFS), and other causes of convulsion.
(4) Laboratory investigations: blood samples are withdrawn according to situation including sepsis workup (according to suspected site of sepsis), random blood sugar (RBS), serum Natrium (Na), serum Ca, and serum Magnesium (Mg).
(5) EEG was done once for all patients using EEG machine (Model: E-series PSG; Compumedics, Australia) with standard 10–20 electrode placement system and recording for at least 40-min duration, in first contact with the child. Young

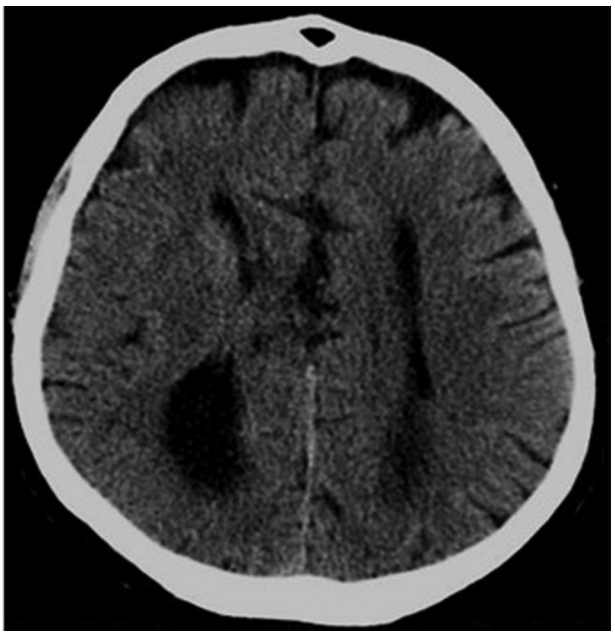
children who are uncooperative are sedated using oral chloral hydrate (30–50 mg/kg/dose; maximum 100 mg/kg/dose).
(6) CT scan of the brain was done for all patients (GE-light-speed-4-slice-spiral-CT-scanner, USA).
(7) MRI of the brain was done only for patients whose CT scan needs further characterization (Philips achieva 1.5T MRI, Netherlands).

.

Statistical methods

(1) Data were analyzed using IBM SPSS statistics, version 22 (IBM Corp., Armonk, New York, USA).
(2) The Shapiro–Wilk test was used to examine the normality of numerical data distribution. Non-normally distributed numerical data were presented as median (interquartile range) and between-group differences were compared using the Mann–Whitney test.
(3) Categorical data were presented as number (%) and between-group differences were compared using Fisher's exact test. The McNemar test was used for comparison of paired categorical data.
(4) Inter-method agreement was examined using Cohen κ coefficient.
(5) The Bonferroni method was used to adjust the level of significance for the number of subgroup comparisons to control the type I error.

Figure 14



Case no 5. Computed tomography brain shows widely separated bodies of both lateral ventricles as a result of corpus callosum agenesis.

Results

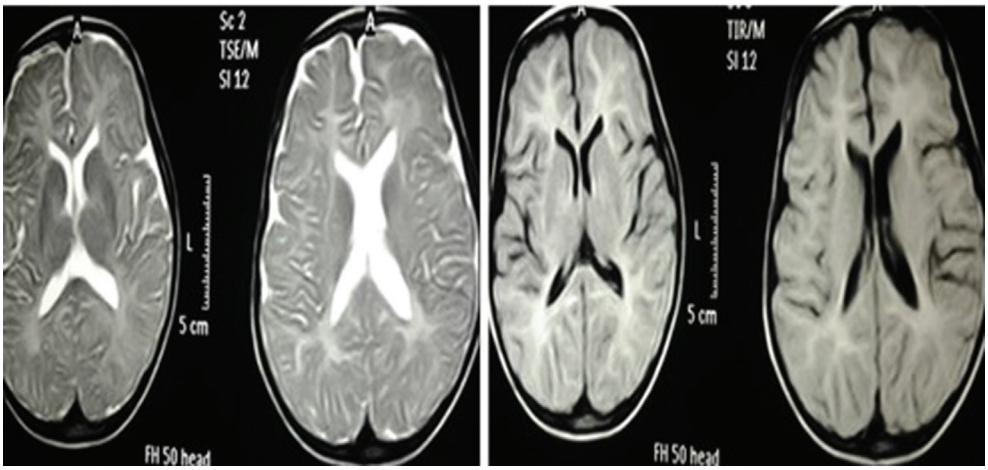
Results of our study are illustrated in Figures 5–16 and Tables 1–11.

Discussion

In this study, EEG abnormality was found in 33% of the whole study cases, including FUSs and CFSs. EEG abnormality was higher in a similar study done by Rasool *et al.* [17], who found that EEG abnormality was detected in 56.2% of all studied population. The difference can be explained by that, in our study, EEG is done on first contact with the complaining child where most cases are examined at third to seventh day of the attack, whereas in the study done by Rasool *et al.* [17]. EEG was done within the first 48 h of the attack.

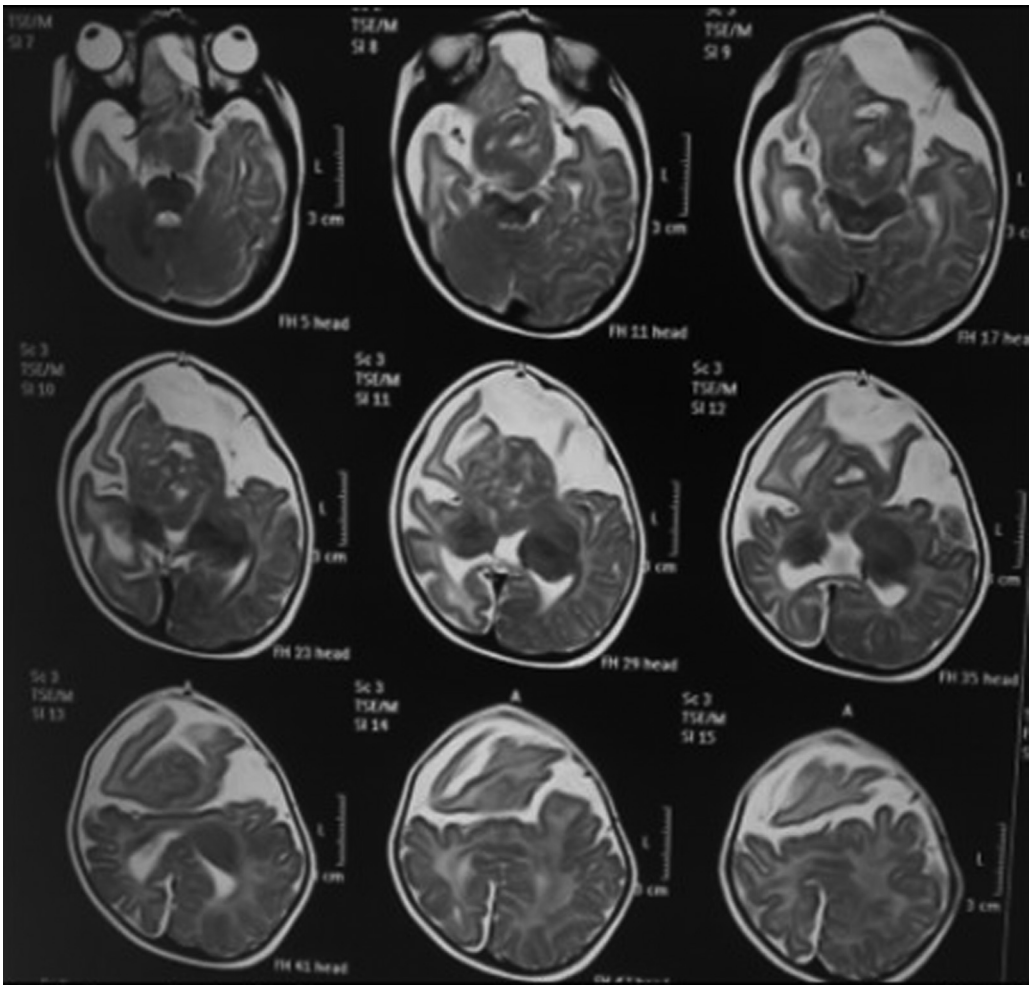
In this study, EEG abnormality was found in 44.4% of cases with FUSs. This is consistent with the study results of Hamiwka *et al.* [18], where EEG was done in

Figure 15



Case no 6. Axial T2 and FLAIR WI of a 3-year-old girl shows bilateral periventricular deep white matter abnormal signal, related to diffuse hypomyelination.

Figure 16



Case no 7. Axial T2 revealed semilobar holoprosencephaly, showing monoventricle with partially developed occipital and temporal horns. Incomplete falx cerebri, incompletely formed interhemispheric fissures are seen, especially anteriorly and high dysplastic corpus callosum.

Table 1 Patient characteristics

Variables	Value
Age (years)	
Median (interquartile range)	3.8 (1–8)
Range	0.5–12
Sex (male/female)	56/44
Family history of seizures	24 (24.0)
Duration of seizures (min)	
Median (interquartile range)	5 (3–10)
Range	0.3–20
Groups of patients	
First unprovoked seizures	63 (63.0)
Generalized unprovoked seizures	22 (34.9)
GTC seizures	10 (15.8)
Clonic seizures	6 (9.6)
Tonic seizures	4 (6.4)
Atonic seizures	3 (4.7)
Focal unprovoked seizures	8 (12.6)
Complex focal seizures	7 (11.3)
Simple focal seizures	3 (4.7)
Complex febrile seizures	37 (37.0)

Data are ratio, or *n* (%).

Table 2 Electroencephalogram findings in the study population

Variables	Value
Abnormal electroencephalogram	33 (33.0)
Abnormal electroencephalogram pattern (Fig. 7)	
Sharp waves	14 (14.0)
Sharp-slow	9 (9.0)
Spike-slow	6 (7.0)
Polyspike	2 (2.0)
Spikes	1 (1.0)
Slowing	1 (1.0)

Data are *n* (%).

127 children with FUSs and was abnormal in 41% of cases.

In this study, EEG abnormalities were significantly low ($P<0.025$) among the patients with CFSs (13.5%) in comparison with FUSs (44.4%). This finding agreed with Rasool *et al.* [17].

Our results disagreed with the study results of Joshi *et al.* [19], who found that 39.43% of children with CFSs had EEG abnormalities. Our results also disagreed with the study results of Jeong *et al.* [16], who detected EEG abnormalities in 43% of children with CFSs. The difference can be explained by early postictal EEG registration (within 24–48 h). Moreover, there is disagreement between our results and another study carried out by Yucel *et al.* [20], who detected abnormal EEGs in 22.5% (16 of 71) of cases with CFS.

Table 3 Neuroimaging findings in the study population

Variables	Value
Abnormal computed tomography	14/100 (14.0)
Abnormal MRI	15/28 (53.6)
Abnormal computed tomography and MRI	15/100 (15.0)
Specific findings by neuroimaging	
Normal findings	85 (85.0)
Ventriculomegaly	1 (1.0)
Atrophic changes	5 (5.0)
Encephalomalacia	2 (2.0)
Arachnoid cyst	2 (2.0)
Lissencephaly	1 (1.0)
Corpus callosum dysgenesis	2 (2.0)
Delayed myelination	1 (1.0)
Encephalomalacia and atrophic changes	1 (1.0)

Data are *n* (%) or ratio (%).

In this study, CT brain were done for all cases, whereas MRI brain were done for cases whose CT of the brain needs further characterization (28/100).

In this study, the prevalence of neuroimaging abnormalities in children with FUSs was 20.6%. This was in agreement with the study results of Shinnar *et al.* [21], who found that 21% of cases with FUSs had neuroimaging abnormalities.

In the study done by Mohammadi *et al.* [22], abnormal neuroimaging result was reported in 27.1% of cases. However, this difference could be owing to the use of MRI in most patients (82/96) in their study compared with 24/63 cases in our study.

In this study, the prevalence of brain CT scan abnormalities in children with FUSs was 20.6%, as presented in Table 4. These finding agreed with the study done by Maytal *et al.* [9], who detected that 21.2% had abnormal CT results, and disagreed with

Table 4 Comparison between patients with first unprovoked and those with complex febrile seizures

Variables	Unprovoked seizures (*n*=63)	Complex febrile seizures (*n*=37)	*P* value[¶]
Age (years)	4.5 (1.2–9.0)	3 (0.9–5.5)	0.057[a]
Sex (male/female)	34/29	22/15	0.678[b]
Family history of seizures	15 (23.8)	9 (24.3)	1.000[b]
Duration of seizures (min)	4 (3–7)	7 (5–10)	0.0006[a]
Abnormal EEG	28 (44.4)	5 (13.5)	0.002[b]
Abnormal EEG pattern			
Sharp waves	12 (19.0)	2 (5.4)	0.075[b]
Sharp-slow	9 (14.3)	0	0.024[b]
Spike-slow	6 (9.5)	1 (2.7)	0.255[b]
Polyspike	1 (1.6)	1 (2.7)	1.000[b]
Spikes	1 (1.6)	0	1.000[b]
Slowing	0	1 (2.7)	0.370[b]
Abnormal CT	13 (20.6)	1 (2.7)	0.015[b]
Abnormal MRI	13 (54.2)	2 (50.0)	1.000[b]
Abnormal CT and MRI	13 (20.6)	2 (5.4)	0.045[b]
Specific findings by neuroimaging			
Normal findings	50 (79.4)	35 (94.6)	0.602[b]
Ventriculomegaly	1 (1.6)	0	
Atrophic changes	4 (6.3)	1 (2.7)	
Encephalomalacia	2 (3.2)	0	
Arachnoid cyst	2 (3.2)	0	
Lissencephaly	1 (1.6)	0	
Corpus callosum dysgenesis	2 (3.2)	0	
Delayed myelination	0	1 (2.7)	
Encephalomalacia and atrophic changes	1 (1.6)	0	

Data are represented as median (interquartile range), ratio, or *n* (%). CT, computed tomography; EEG, electroencephalogram.
[a]Mann–Whitney test. [b]Fisher's exact test. [¶]Significance is set at the $P<0.025$ level using the Bonferroni correction for subgroup comparison.

Table 5 Comparison between patients with generalized and those with focal unprovoked seizures

Variables	Generalized US (*n*=44)	Focal US (*n*=19)	*P* value¶
Age (years)	5.8 (1.8–9.0)	3 (0.7–6)	0.037a
Sex (male/female)	23/21	11/8	0.786b
Family history of seizures	10 (22.7)	5 (26.3)	0.757b
Duration of seizures (min)	5 (2–7)	3 (3–5)	0.970a
Abnormal EEG	15 (34.1)	13 (68.4)	**0.015**b
Abnormal EEG pattern			
Sharp waves	5 (11.4)	7 (36.8)	0.033b
Sharp-slow	5 (11.4)	4 (21.1)	0.434b
Spike-slow	4 (9.1)	2 (10.5)	1.000b
Polyspike	1 (2.3)	0	1.000b
Spikes	1 (2.3)	0	1.000b
Slowing	0	0	NAb
Abnormal CT	7 (15.9)	6 (31.6)	0.186b
Abnormal MRI	7 (63.6)	6 (46.2)	0.444b
Abnormal CT and MRI	7 (15.9)	6 (31.6)	0.186b
Specific findings by neuroimaging			
Normal findings	37 (84.1)	13 (68.4)	0.173b
Ventriculomegaly	0	1 (5.3)	
Atrophic changes	3 (6.8)	1 (5.3)	
Encephalomalacia	1 (2.3)	1 (5.3)	
Arachnoid cyst	1 (2.3)	1 (5.3)	
Lissencephaly	0	1 (5.3)	
Corpus callosum dysgenesis	2 (4.5)	0	
Encephalomalacia and atrophic changes	0	1 (5.3)	

US, ultrasound; Data are represented as median (interquartile range) ratio, or *n* (%). CT, computed tomography; EEG, electroencephalogram; NA, not applicable. [a]Mann–Whitney test. [b]Fisher's exact test. ¶Significance is set at the $P<0.025$ level using the Bonferroni correction for subgroup comparison.

Table 6 Prevalence of neuroimaging abnormalities in patients with normal or abnormal electroencephalogram

Variable	All study population (*n*=100)		*P* value¶
	Normal EEG (*n*=67)	Abnormal EEG (*n*=33)	
Abnormal CT and MRI	3 (4.5)	12 (36.4)	0.0003[a]

Data are *n* (%). CT, computed tomography; EEG, electroencephalogram. [a]McNemar test. ¶Significance is set at the $P<0.008$ level using the Bonferroni correction for multiple subgroup comparisons.

Table 7 Prevalence of neuroimaging abnormalities in patients with complex febrile seizures and normal or abnormal electroencephalogram

Variable	Complex febrile seizures (*n*=37)		*P* value¶
	Normal EEG (*n*=32)	Abnormal EEG (*n*=5)	
Abnormal CT and MRI	2 (6.3)	0	0.453[a]

Data are *n* (%). CT, computed tomography; EEG, electroencephalogram. [a]McNemar test. ¶Significance is set at the $P<0.008$ level using the Bonferroni correction for multiple subgroup comparisons.

Table 8 Prevalence of neuroimaging abnormalities in patients with unprovoked seizures and normal or abnormal electroencephalogram

Variable	Unprovoked seizures (*n*=63)		*P* value¶
	Normal EEG (*n*=35)	Abnormal EEG (*n*=28)	
Abnormal CT and MRI	1 (2.9)	12 (42.9)	0.0003[a]

Data are *n* (%). CT, computed tomography; EEG, electroencephalogram. [a]McNemar test. ¶Significance is set at the $P<0.008$ level using the Bonferroni correction for multiple subgroup comparisons.

the study results of Vieira *et al.* [23], who found abnormalities in 29.44% of cases, which can be explained by larger sample size (387) in their study, and disagreed with the study results of Mathur *et al.* [24], who detected CT brain abnormalities in 32% of all children with a FUS.

Table 9 Prevalence of neuroimaging abnormalities in patients with generalized unprovoked seizures and normal or abnormal electroencephalogram

Variable	Generalized Unprovoked Seizures (n=44)		P value[¶]
	Normal EEG (n=29)	Abnormal EEG (n=15)	
Abnormal CT and MRI	1 (3.4)	6 (40%	0.022[a]

Data are n (%). CT, computed tomography; EEG, electroencephalogram. [a]McNemar test. [¶]Significance is set at the $P<0.008$ level using the Bonferroni correction for multiple subgroup comparisons.

Table 10 Prevalence of neuroimaging abnormalities in patients with focal unprovoked seizures and normal or abnormal electroencephalogram

Variable	Focal unprovoked seizures (n=19)		P value[¶]
	Normal EEG (n=6)	Abnormal EEG (n=13)	
Abnormal CT, MRI, or both	0	6 (46.2)	0.016[a]

Data are n (%). CT, computed tomography; EEG, electroencephalogram. [a]McNemar test. [¶]Significance is set at the $P<0.008$ level using the Bonferroni correction for multiple subgroup comparisons.

Table 11 Prevalence of computed tomography scan abnormalities in patients with normal or abnormal electroencephalogram in the whole study population and in various patient subgroups

Group	Number with abnormal CT	EEG		P value[¶]
		Normal (n=67)	Abnormal (n=33)	
All study population (n=100)	14/100 (14.0)	2 (3.0)	12 (36.4)	0.0001[a]
Complex febrile seizures (n=37)	1/37 (2.7)	1 (3.1)	0	0.219[a]
Unprovoked seizures (n=63)	13/63 (20.6)	1 (2.9)	12 (42.9)	0.0003[a]
Generalized unprovoked seizures (n=44)	7/44 (15.9)	1 (3.4)	6 (40.0)	0.021[a]
Focal unprovoked seizures (n=19)	6/19 (31.6)	0	6 (46.2)	0.016[a]

Data are ratio (%) or n (%). CT, computed tomography; EEG, electroencephalogram. [a]McNemar test. [¶]Significance is set at the $P<0.01$ level using the Bonferroni correction for multiple subgroup comparison.

In this study, the prevalence of neuroimaging (CT/MRI) abnormalities in CFSs was 5.4% (2/33), which agreed with Rasool *et al.* [17] who found that neuroimaging abnormalities was detected in 1.6% of cases, and disagreed with Hesdorffer *et al.* [25], who found that 14.8% of children with complex febrile convulsions had neuroimaging abnormalities. This difference can be explained by that, MRI is the neuroimaging modality used for all patients in their study.

In this study, the prevalence of abnormal CT scan in children with generalized first seizures was 15.9% (7/44) whereas that of focal seizures was 31.6% (6/19). These findings were consistent with that of Maytal *et al.* [9], who reported that in generalized seizures, 82.5% cases had normal CT scan result and 17.5% had abnormal CT scan result, whereas in partial seizures, 70.8% had normal imaging, and abnormal CT scan result was seen in 29.2%, showing overall abnormal CT scan result in 21.2% of the patients, which also agrees with our study.

In this study, brain atrophic changes were the most common neuroimaging abnormality. Other lesions include lissencephaly, ventriculomegaly, encephalomalacia, arachnoid cyst, corpus callosum dysgenesis, delayed maturation and encephalomalacia, and atrophic changes of the total population.

Rasool *et al.* [17] also found that brain atrophic changes were the most common neuroimaging abnormality in their study, and other findings were focal hypodense lesion, white matter hypodensity, and lissencephaly. However, in a study of neuroimaging findings in children with FUSs done by Mohammadi *et al.* [22], the most common imaging findings were gliosis followed by dysmyelination, hemorrhage, brain atrophy, dysgenesis, infarction, and encephalomalacia.

Conclusion

(1) EEG and neuroimaging abnormalities were more prevalent in children with FUSs than those with CFSs.
(2) Abnormal EEG and neuroimaging were more common in children with partial seizures than those with generalized seizures.
(3) Neuroimaging was abnormal in a significant number of children having abnormal EEG, so

neurologically free patients having normal EEG can be safely discharged without neuroimaging, if follow-up is assured.

(4) When EEG is abnormal in FUS, the probability of having abnormal neuroimaging result increases as compared with those cases where EEG result is normal.

(5) In case of generalized seizures, patients with abnormal EEG result may have abnormal CT/MRI scan results, but there are fewer possibilities of a patient with abnormal EEG result to have a normal neuroimaging result.

(6) In partial seizures, abnormal EEG result increases the risk of having abnormal neuroimaging result than in generalized seizures, and normal EEG result in partial seizures markedly decreases the risk of having an abnormal neuroimaging result in generalized seizures.

(7) CFSs in otherwise neurologically free children rarely indicate the presence of lesion on neuroimaging even if associated with EEG abnormalities.

(8) Neuroimaging abnormalities in neurologically free children with FUS and CFSs do not require urgent intervention.

Acknowledgements

This study was supported by Radiology and Pediatric Departments, Al Hussein University Hospital, Faculty of Medicine (for Boys), Al Hussein University Hospital.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1 Beghi E, Carpio A, Forsgren L, Hesdorffer DC, Malmgren K, Sander JW, *et al.* Recommendation for a definition of acute symptomatic seizure. *Epilepsia* 2010; **51**: 671–675.

2 Sharma S, Riviello JJ, Harper MB, Baskin MN. The role of emergent neuroimaging in children with new-onset afebrile seizures. *Pediatrics* 2003; **111**: 1–5.

3 Simone CV, Breno NL, Monica JS, Ortega AB, Olmos AS, Júnior AL. First unprovoked seizure: clinical and electrographic aspects. *J Epilepsy Clin Neurophysiol* 2006; **12**: 69–72.

4 Hirtz D, Ashwal S, Berg A, Bettis D, Camfield C, Camfield P, *et al.* Practice parameter: evaluating a first nonfebrile seizure in children: report of the quality standards subcommittee of the American Academy of Neurology, *The Child Neurology Society, and The American Epilepsy Society. Neurology* 2000; **55**: 616–623.

5 Berg D, Hirtz D, Ashwal S, Bettis D, Camfield C, Camfield P, *et al.* Practice parameter: evaluating a first nonfebrile seizure in children: report of the quality standards subcommittee of the American Academy of Neurology, the Child Neurology Society, and American Epilepsy Society. *Neurology* 2000; **55**: 616–623.

6 Alawaneh H, Bataineh HA. Urgent neuroimaging in children with first nonfebrile seizures. *Middle East J Fam* 2008; **6**: 24–26.

7 Chen DK, So YT, Fisher RS. Use of serum prolactin in diagnosing epileptic seizures: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology* 2005; **65**: 668–675.

8 Harden CL, Huff JS, Schwartz TH, Dubinsky RM, Zimmerman RD, Weinstein S, *et al.* Reassessment: neuroimaging in the emergency patient presenting with seizure (an evidence-based review): report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology* 2007; **69**: 1772–1780.

9 Maytal J, Krauss JM, Novak G, Nagelberg J, Patel M. The role of brain computed tomography in evaluating children with new onset of seizures in the emergency department. *Epilepsia* 2000; **41**: 950–954.

10 Jagoda A, Gupta K. The emergency department evaluation of the adult patient who presents with a first-time seizure. *Emerg Med Clin North Am* 2011; **29**: 41–49.

11 King MA, Newton MR, Jackson GD, Fitt GJ, Mitchell LA, Silvapulle MJ, Berkovic SF. Epileptology of the first-seizure presentation: a clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. *Lancet* 1998; **352**:1007–1011

12 Panayiotopoulos CP. (2010) *Idiopathic generalised epilepsies. In: A Clinical Guide to Epileptic Syndromes and their Treatment*. Second edition. London: Springer; 2010. 978-1-84628-643-8

13 Krumholz A, Wiebe S, Gronseth G, Shinnar S, Levisohn P, Ting T, *et al.* Practice parameter: evaluating an apparent unprovoked first seizure in adults (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology and the American Epilepsy Society. *Neurology* 2007; **69**:1996–2007.

14 Ferrie CD, Agathonikou A, Panayiotopoulos CP. EEG and video-electroencephalography in the classification of childhood epilepsy syndromes. *J R Soc Med* 1998; **91**:251–259.

15 Wiebe S, Téllez-Zenteno JF, Shapiro M. An evidence-based approach to the first seizure. *Epilepsia* 2008; **49**(Suppl 1):50–57.

16 Jeong KA, Han MH, Lee EH, Chung S. Early postictal electroencephalography and correlation with clinical findings in children with febrile seizures. *Korean J Pediatr* 2013; **56**:534–539.

17 Rasool A, ChohS A, Wani NA, Ahmad SM, Iqbal Q. Role of electroencephalogram and neuroimaging in first onset afebrile and complex febrile seizures in children from Kashmir. *J Pediatr Neurosci* 2012; **7**:9–15.

18 Hamiwka LD, Singh N, Niosi J, Wirrell EC. Diagnostic inaccuracy in children referred with 'first seizure': role for a first seizure clinic. *Epilepsia* 2007; **48**:1062–1066.

19 Joshi C, Wawrykow T, Patrick J, Prasad A. Do clinical variables predict an abnormal EEG in patients with complex febrile seizures? *Seizure* 2005; **14**:429–434.

20 Yucel O, Aka S, Yazicioglu L, Ceran O. Role of early EEG and neuroimaging in determination of prognosis in children with complex febrile seizure. *Pediatr Int* 2004; **46**:463–467.

21 Shinnar S, O'Dell C, Mitnick R, Berg AT, Moshe SL. Neuroimaging abnormalities in children with an apparent first unprovoked seizure. *Epilepsy Res* 2001; **43**:261–269.

22 Mohammadi MM, Tonekaboni SH, Khatami AR, Azargashb E, Tavasoli A, Javadzadeh M, Zamani GR. Neuroimaging findings in first unprovoked seizures: a multicentric study in Tehran. *Iran J Child Neurol Autumn* 2013; **7**:24–31.

23 Vieira SC, Liberalesso PB, Spinosa MJ, Ortega AB, Olmos AS, Júnior AL. First unprovoked seizure: clinical and electrographic aspects. *J Epilepsy Clin Neurophysiol* 2006; **12**:69–72.

24 Mathur S, Southern K, Sharma M. Significant findings on cranial CT scan after a first unprovoked seizure in children from North India. *J Trop Pediatr* 2007; **53**:428–430.

25 Hesdorffer DC, Chan S, Tian H, Allen Hauser W, Dayan P, Leary LD, Hinton VJ. Are MRI-detected brain abnormalities associated with febrile seizure type?. *Epilepsia* 2008; **49**:765–771.