De novo Status Epilepticus
What Is It?

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De novo SE: Incidence

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Analysis of clinical characteristics and risk factors for mortality in human status epilepticus

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Purpose: To analyse clinical data including aetiology, age, antecedents, classification and mortality in human status epilepticus (SE), and to assess prognostic factors for mortality.
Methods: A prospective study was performed, including detailed analysis of clinical and laboratorial data of SE in individuals of any age, except neonates.
Results: One hundred and eleven SE were included, with patients' age ranging from 3 months to 98 years. SE incidence peaked in the first year of life, and 59.4% of the individuals had previous epilepsy while 40.6% had not. The main underlying causes were noncompliance to treatment in the first group, and CNS infection, stroke and metabolic disturbances in the second group. Overall mortality was 19.8%, and deaths were correlated to aetiology and patient's age. Refractory SE affected 11.7% of the cases. Clinical types included focal, secondarily generalised and generalised SE. Clinical and clinicoelectrographic classifications were convergent, but EEG was essential for the diagnosis in 4.5% of the cases.
Conclusions: Epileptic patients are at greater risk to develop SE, however, individuals with no prior history of epilepsy and acute neurological problems can also present SE. Aetiology varies with patient's age, and mortality is high and related to age and underlying causes. Clinical and clinicoelectrographic classifications are usually convergent, but in some cases the diagnosis of SE would not be established without the EEG.

De novo SE: Incidence

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FULL-LENGTH ORIGINAL RESEARCH

Status epilepticus: Clinical presentation, cause, outcome, and predictors of death in 119 Ethiopian patients

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SUMMARY
Purpose: Status epilepticus (SE) is a common neurological emergency with high morbidity and mortality. There is no study that has been conducted among Ethiopian patients with SE. The purpose of this study was to analyze clinical presentation, causes, complications, outcomes, and predictors of mortality.
Methods: In this retrospective study, patients aged ≥13 year with SE were included. Medical records were reviewed and demographic and clinical data were collected.
Results: Records of 119 patients were analyzed; preexisting epilepsy was found in 38.7%. Primarily generalized and focal with secondarily generalized (FWSG) seizures were identified in 60.5% and 36%, respectively. Simple partial SE occurred in 3.4%. Central nervous system (CNS) infection was the most common cause of SE in the whole group as well as in those with new onset seizure. Antiepileptic drug withdrawal (AEDW) was the main cause in those with preexisting seizure. One or more complications were detected in 61%. Intravenous diazepam and oral phenytoin were given to 95% and 97.5%, respectively. Case fatality was 20.2%; poor outcome occurred in 24%. Predictors of mortality were FWSG type, acute symptomatic etiology, stroke, systemic infection, and HIV/AIDS and its CNS complications. Idiopathic and SE due to AEDW were associated with good prognosis.
Conclusions: CNS infection was the most common cause of SE in the whole group and AEDW was the major cause in patients with preexisting epilepsy. Parenteral anticonvulsants, emergency measurement of serum AED level, and electroencephalography for urgent diagnosis and monitoring were unavailable. Mortality was related to underlying etiologies especially HIV/AIDS and its CNS complications.
KEY WORDS: Epilepsy, Status epilepticus, Etiology, Complication, Case fatality.

De novo SE: Incidence

Epilepsia, 51(2):251–256, 2010
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FULL-LENGTH ORIGINAL RESEARCH

Refractory status epilepticus: A prospective observational study

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SUMMARY
Purpose: Status epilepticus (SE) that is resistant to two antiepileptic compounds is defined as refractory status epilepticus (RSE). In the few available retrospective studies, estimated RSE frequency is between 31% and 43% of patients presenting an SE episode; almost all seem to require a coma induction for treatment. We prospectively assessed RSE frequency, clinical predictors, and outcome in a tertiary clinical setting.
Methods: Over 2 years we collected 128 consecutives SE episodes (118 patients) in adults. Clinical data and their relationship to outcome (mortality and return to baseline clinical conditions) were analyzed.
Results: Twenty-nine of 128 SE episodes (22.6%) were refractory to first- and second-line antiepileptic treatments. Severity of consciousness impairment and de novo episodes were independent predictors of RSE. RSE showed a worse outcome than non-RSE (39% vs. 11% for mortality; 21% vs. 63% for return to baseline clinical conditions). Only 12 patients with RSE (41%) required coma induction for treatment.
Discussion: This prospective study identifies clinical factors predicting the onset of SE refractoriness. RSE appears to be less frequent than previously reported in retrospective studies; furthermore, most RSE episodes were treated outside the intensive care unit (ICU). Nonetheless, we confirm that RSE is characterized by high mortality and morbidity.
KEY WORDS: Treatment, Predictors, Outcome, Mortality.

De novo SE: Poor Px Predictor?

Table 2. Demographics and clinical features in episodes of refractory and nonrefractory status epilepticus

	Refractory	Nonrefractory	p-value	Test
Patients	29 (23%)	99 (77%)	–	–
Age (years) (mean, SD)	64 (±18)	59 (±19)	0.26	*t*
Female gender	13 (48.1%)	55 (60.4%)	0.256	χ^2
Earlier seizures	9 (31%)	51 (51.5%)	0.052	χ^2
Consciousness				
Alert	1 (3.4%)	24 (24.2%)	**0.018**	Fisher's
Confused/ somnolent	7 (24.1%)	28 (28.3%)		
Stuporous	17 (58.6%)	31 (31.3%)		
Comatose	4 (13.8%)	16 (16.2%)		
Seizure type				
Complex partial	14 (48.3%)	35 (35.4%)	0.186	Fisher's
Generalized convulsive	9 (31.0%)	38 (38.4%)		
Generalized myoclonic	0 (0%)	1 (1%)		
Nonconvulsive SE in coma	2 (6.9%)	1 (1%)		
Simple partial	4 (13.8%)	24 (24.2%)		
Nonconvulsive SE	19 (70.4%)	55 (60.4%)	0.376	χ^2
STESS ≥3	23 (79.3%)	45 (45.5%)	**0.001**	χ^2
Time to treatment >1 h	19 (65.5%)	65 (65.7%)	0.989	χ^2

De novo SE: 43% (58/118)

The multivariate logistic regression analysis
1. Severe C/S impairment
   p=0.043, 95% CI (1.3-6.62)
2. De novo episodes
   p=0.043, 95% CI (1.03-6.62)

De novo SE: Poor Px Predictor?

ORIGINAL CONTRIBUTION

Predictors of Outcome in Refractory Status Epilepticus

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Objective: To further characterize the demographics, outcomes, and prognostic factors for refractory status epilepticus (RSE).

Design: Retrospective analysis of all the episodes of RSE treated between January 1, 1999, and August 30, 2011.

Setting: Neurointensive care unit within a tertiary referral center, Mayo Clinic, Rochester, Minnesota.

Patients: Refractory status epilepticus was defined as generalized convulsive or nonconvulsive status epilepticus (SE) that continued despite initial first- and second-line therapies. Exclusion criteria were aged younger than 18 years, anoxic/myoclonic SE, psychogenic SE, simple partial SE, and absence SE.

Main Outcome Measures: Functional outcome was defined by modified Rankin scale (mRS) dichotomized into good (mRS, 0-3) and poor (mRS, 4-6). Functional decline was defined as a change in mRS greater than 1 from hospital admission to discharge.

Results: We identified 63 consecutive episodes of non–anoxic RSE in 54 patients. Anesthetic agents were used in 55 episodes (87.30%), and duration of drug-induced coma was (mean [SD]) 11.0 (17.9) days. In-hospital mortality was 31.75% (20 of 63 episodes). Poor functional outcome at discharge occurred in 48 of 63 episodes (76.19%). Hospital length of stay was (mean [SD]) 27.7 (37.3) days. Duration of drug-induced coma ($P = .03$), arrhythmias requiring intervention ($P = .01$), and pneumonia ($P = .01$) were associated with poor functional outcome. Prolonged mechanical ventilation was associated with mortality ($P = .04$). Seizure control without suppression-burst or isoelectric electroencephalogram predicted good functional recovery ($P = .01$). Age, history of epilepsy, previous SE, type of SE, and anesthetic drug used were not associated with functional outcome.

Conclusions: Three-quarters of patients with RSE have a poor outcome. Achieving control of the SE without requiring prolonged drug-induced coma or severe electroencephalographic suppression portends better prognosis.

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De novo SE: Poor Px Predictor?

Table. Demographic and Clinical Features in Episodes of Refractory Status Epilepticus

Variable	No. (%)[a]
Age, median interquartile range [range], y	52 [18-93]
Sex	
Male	36 (57)
Ethnicity	
White	52 (82.5)
Black	3 (4.8)
Hispanic	1 (1.6)
Arab	1 (1.6)
Unknown	6 (9.5)
Previous episode of SE[b]	17 (27.9)
History of seizures[c]	37 (59.7)
mRS score at admission[c]	
0	14 (22.6)
1	11 (17.7)
2	10 (16.1)
3	15 (24.2)
4	8 (12.9)
5	4 (6.5)
Classification of SE[b]	
GCSE	20 (31.7)
NCSE partial	32 (52.5)
NCSE generalized	9 (14.8)

De novo SE: 40.3% (25/62)

History of seizures (de novo SE) was not associated with functional outcome

De novo SE: Poor Px Predictor!!

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De novo status epilepticus is associated with adverse outcome: An 11-year retrospective study in Hong Kong☆

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ABSTRACTS

Purpose: To identify predictors of poor clinical outcome in patients presenting to the intensive care units with status epilepticus (SE), in particular for patients presenting with de novo status epilepticus.

Methods: A retrospective review was performed on patients admitted to the intensive care units with status epilepticus in two hospitals in Hong Kong over an 11-year period from 2003 to 2013.

Results: A total of 87 SE cases were analyzed. The mean age of patients was 49.3 years (SD 14.9 years). Eighteen subjects (20.7%) had breakthrough seizure, which was the most common etiology for the status epilepticus episodes. Seventy-eight subjects (89.7%) had convulsive status epilepticus (CSE) and 9 subjects (10.3%) had non-convulsive status epilepticus (NCSE) on presentation. The 30-day mortality rate of all subjects was 18.4%. Non-convulsive status epilepticus was more common in patients with de novo status epilepticus when compared to those with existing history of epilepsy (15.5% Vs. 0%, $p = 0.03$). Patients with de novo status epilepticus were older (52 Vs 43, $p = 0.009$). De novo status epilepticus was associated with longer status duration (median 2.5 days, IQR 5 days), longer ICU stay (median 7.5 days, IQR 9 days) and poorer outcome (OR 4.15, 95% CI 1.53–11.2).

Conclusions: For patients presenting to intensive care units with status epilepticus, those with de novo status epilepticus were older and were more likely to develop non-convulsive status epilepticus. De novo status epilepticus was associated with poorer outcome. Continuous EEG monitoring would help identifying NCSE and potentially help improving clinical outcomes.

De novo SE: Poor Px Predictor!!

Table 1
Clinical characteristics of study subjects ($n = 87$).

Age, mean (SD)	49.3 (14.9)
Male, *n* (%)	48 (56.3%)
Previous history of epilepsy, *n* (%)	29 (33.3%)
Clinical manifestations of status epilepticus, *n* (%)	
Convulsive status epilepticus (CSE)	78 (90.0%)
Non-convulsive status epilepticus (NCSE)	9 (10.0%)
Presumed etiologies of status epilepticus, *n* (%)	
Breakthrough seizure	18 (21.0%)
Encephalitis/meningitis	16 (18.3%)
Cerebrovascular accident	10 (11.5%)
Metabolic causes	9 (10.3%)
Drug overdose/alcohol withdrawal	7 (8.0%)
Hypoxic brain damage	6 (6.9%)
Sepsis	4 (4.6%)
Traumatic subarachnoid hemorrhage/subdural hemorrhage	4 (4.6%)
Idiopathic	13 (14.9%)

De novo SE: 66.7%

De novo SE: Poor Px Predictor!!

	De novo (n=58)	Known Hx (n=29)	
Age, mean (SD)	52 (15)	43 (13)	0.009*
Male, *n* (%)	31 (53.4%)	18 (62.0%)	0.445
Etiology, *n* (%)			
Encephalitis/meningitis	12 (20.7%)	4 (13.8%)	0.562
Cerebrovascular accident	9 (15.5%)	1 (3.4%)	0.155
Hypoxic brain damage	5 (8.6%)	1 (3.4%)	0.659
Drug overdose	5 (8.6%)	2 (6.9%)	1.000
Sepsis	5 (8.6%)	3 (10.3%)	1.000
Traumatic SAH/SDH	4 (6.9%)	0 (0%)	0.296
Breakthrough seizure	0 (0%)	16 (55.0%)	0.000*
Non-convulsive status epilepticus (NCSE)	9 (15.5%)	0 (0%)	0.026*
Status duration, median (IQR), days	2.5 (5.0)	1 (0)	0.002
Length of hospital stay, median (IQR), days	17 (26)	8 (22)	0.055
Length of ICU stay, median (IQR), days	7.5 (9)	4 (4)	0.005*
30-day mortality, *n* (%)	14 (24.1%)	2 (6.9%)	0.077
Poor outcome upon discharge, *n* (%)	33 (56.9%)	7 (24.1%)	0.004*

De novo SE: those are older, and more likely to have longer status & develop NCSE

De novo SE: Predictive factor of Outcome

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Factors predictive of outcome in patients with *de novo* status epilepticus

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Summary

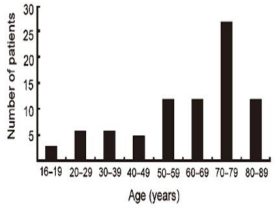
Background: About 50% of status epilepticus (SE) patients have no previous history of epilepsy, but often have worse outcome. The aim of this study was to evaluate potential risk factors that are predictive of poor outcome in non-selected *de novo* status epilepticus patients.

Methods: Eighty-three adult status epilepticus patients without a pre-existing history of epilepsy that were admitted to hospital for treatment were enrolled in this 11-year retrospective study. The baseline prognostic variables were analyzed based on stepwise logistic regression analysis after a minimum of one-and-half years of follow-up.

Results: The overall fatality rate was 55.4% (46/83) during the study period. Poor outcome was associated with older age, presence of refractory status epilepticus, potential fatal etiologies, lower GCS score at presentation and level of consciousness on admission. The results of stepwise logistic regression demonstrated that age on presentation and potential fatal etiologies were independently associated with presence of poor outcome, and any increase in age by 1 year increases poor outcome by 7.5%.

Conclusions: The outcome for those with *de novo* status epilepticus is poor and this poor outcome may be attributed to the older age at onset and the potential fatal underlying conditions such as infection and metabolic derangement.

De novo SE: Predictive factor of Outcome



Etiology	*N* (%)
Acute symptomatic	45 (54.2)
CNS infection	16
Sepsis or metabolic disturbance[a]	11
Stroke[b]	9
Alcohol or drug related	5
Hypoxia	4
Remote symptomatic	29 (34.9)
Past stroke	22
Head injury	6
Past CNS infection	1
Idiopathic/Cryptogenic	6 (7.2)
Progressive symptomatic	3 (3.6)
Brain tumor	2
CNS lupus	1

De novo SE: Predictive factor of Outcome

Table 3 Prognostic factors of patients with *de novo* status epilepticus

	Poor outcome *N* = 58	Good outcome *N* = 25	Total *N* = 83	*P*-value	OR[a]	95% CI
Sex (male/female)				63	0.58	0.22–1.50
Mean age at onset				001	0.93	0.89–0.96
Mean GCS on presentation				04	1.33	1.10–1.61
Seizure type						
Convulsive				93	1.19	0.51–2.77
Non-convulsive						
Simple partial						
Refractory status epilepticus				37	9.14	1.14–73.30
Etiology						
Acute symptomatic				8	1.4	0.54–3.63
Remote symptomatic				12	0.83	0.31–2.25
Progressive symptomatic				0	1.17	0.10–13.49
Idiopathic/cryptogenic	5	1	6	0.47	0.44	0.05–3.99
Potential fatal	25	3	28	0.011	5.06	1.49–20.66
Duration of status epilepticus						
<1 h	11	5	16	0.91	0.94	0.29–3.05
>1 h	47	20	67			
Mean Hospitalization days	35.7 ± 38.3	26.1 ± 34.9	32.8 ± 37.4	0.29	0.99	0.98–1.01

Table 2 Underlying conditions of potential fatal causes

Potential fatal	*N* = 28
Severe infection or metabolic derangement	11
Acute CNS infections	
Bacterial meningitis	8
Viral encephalitis	2
Hypoxia	4
Severe stroke	2
Metastasis	1

De novo SE: Summary

- Incidence: about 40 ~ 60% of SE
- Associated with poor outcome in SE
- The poor outcome may be attributed to the older age at onset, the longer status duration and the potential fatal underlying conditions
- Those with de novo SE were more likely to develop NCSE
  - A delay in diagnosis of NCSE may contribute to drug resistance and bad outcome (Brain Res 1998; Epilepsy Res 2010)

NCSE

Table 1. Operational dimensions with t_1 indicating the time that emergency treatment of SE should be started and t_2 indicating the time at which long-term consequences may be expected

Type of SE	Operational dimension 1 Time (t_1), when a seizure is likely to be prolonged leading to continuous seizure activity	Operational dimension 2 Time (t_2), when a seizure may cause long term consequences (including neuronal injury, neuronal death, alteration of neuronal networks and functional deficits)
Tonic–clonic SE	5 min	30 min
Focal SE with impaired consciousness	10 min	>60 min
Absence status epilepticus	10–15 min[a]	Unknown

[a]Evidence for the time frame is currently limited and future data may lead to modifications.

Table 2. Axis 1: Classification of status epilepticus (SE)

(A) With prominent motor symptoms
A.1 Convulsive SE (CSE, synonym: tonic–clonic SE)
A.1.a. Generalized convulsive
A.1.b. Focal onset evolving into bilateral convulsive SE
A.1.c. Unknown whether focal or generalized
A.2 Myoclonic SE (prominent epileptic myoclonic jerks)
A.2.a. With coma
A.2.b. Without coma
A.3 Focal motor
A.3.a. Repeated focal motor seizures (Jacksonian)
A.3.b. Epilepsia partialis continua (EPC)
A.3.c. Adversive status
A.3.d. Oculoclonic status
A.3.e. Ictal paresis (i.e., focal inhibitory SE)
A.4 Tonic status
A.5 Hyperkinetic SE

Table 2. Axis 1: Classification of status epilepticus (SE)

(B) Without prominent motor symptoms (i.e., nonconvulsive SE, NCSE)
B.1 NCSE with coma (including so-called "subtle" SE)
B.2 NCSE without coma
B.2.a. Generalized
B.2.a.a Typical absence status
B.2.a.b Atypical absence status
B.2.a.c Myoclonic absence status
B.2.b. Focal
B.2.b.a Without impairment of consciousness (aura continua, with autonomic, sensory, visual, olfactory, gustatory, emotional/psychic/experiential, or auditory symptoms)
B.2.b.b Aphasic status
B.2.b.c With impaired consciousness
B.2.c Unknown whether focal or generalized
B.2.c.a Autonomic SE

Epilepsia, 56(10):1515–1523, 2015

Table 2. NCSE etiology or clinical context, forms, and response to treatment

Syndrome	Etiology or clinical context	Clinical form	Response to treatment or prognosis
NCSE in adulthood (and childhood) with epileptic encephalopathy			
NCSE in Lennox-Gastaut syndrome	Various, often cryptogenic	Atypical absence status epilepticus and tonic status epilepticus	Poor
NCSE in other forms of disrupted cerebral development (cryptogenic or symptomatic)	Various, often cryptogenic	Various	Variable
NCSE in adulthood (and childhood) without epileptic encephalopathy			
Typical absence status epilepticus	Idiopathic generalized epilepsy	Generalized absence	Excellent
Complex partial status epilepticus (limbic and nonlimbic origin)	Various - symptomatic or cryptogenic	Complex partial	Good
NCSE in the postictal phase of TCSE	Various	Confusional state with psychiatric features	Good
Subtle status epilepticus	Various	Coma with small irregular myoclonic jerks	Variable
Aura continua	Various - symptomatic or cryptogenic	Simple partial (sensory, special sensory, cognitive)	Good
NCSE in late adulthood			
De novo absence status epilepticus	Psychotropic drug withdrawal or idiopathic generalized epilepsy	Generalized absence	Excellent
Boundary syndromes			
Coma with epileptiform EEG changes			
Epileptic behavioral disturbance or psychosis			
Drug-induced or metabolic confusional state with epileptiform EEG changes			

Adapted from the revised classifications of status epilepticus in children and adults according to Shorvon (1994).
NCSE, nonconvulsive status epilepticus; EEG, electroencephalography.

NCSE in Older People

- Most common in older people
- More common in women
- A recognized cause of delirium
- A high probability of a diagnostic delay
- If untreated, the patient is at higher risk of adverse outcome
- The presenting symptom of an acute brain insult
  - Early diagnosis of NCSE & its cause are important

Diagnostic Criteria for NCSE

1. Clear clinical change in behavior that lasted at least 10 min.
2. Confirmation of a clinical change by clinical or neuropsychological examination
3. Continuous or virtually continuous paroxysmal episodes must have been present on EEG
4. Continuous major seizures either tonic or clonic must have been absent

* Simultaneous improvement in the EEG & clinical symptoms

Epileptic Disord 2005

Clinical Features of NCSE in Older People

Table 2 Clinical features associated with a diagnosis of non-convulsive status epilepticus in confused older people

Sign	% (n=23)
Myoclonus/subtle jerking	30
Aphasia/interrupted speech	26
Automatisms	26
Staring	22
Perseveration/echolalia	17
Increased tone/catalepsy	13
Nystagmus/eye deviation	13
Emotional lability	9
Disinhibition	9
Anosagnosia	9
None of the above	17

Postgrad Med J 2015

Diagnostic Criteria for NCSE

1. Clear clinical change in behavior that lasted at least 10 min.
2. Confirmation of a clinical change by clinical or neuropsychological examination
3. Continuous or virtually continuous paroxysmal episodes must have been present on EEG
4. Continuous major seizures either tonic or clonic must have been absent

* Simultaneous improvement in the EEG & clinical symptoms by anticonvulsant medication

Epileptic Disord 2005

NCSE in adults & late adulthood

- The diagnosis of NCSE was dependent primarily on the presence of electrographic seizure activity.

This allowed the inclusion of a range of "boundary condition" in which such activity occurred but in which there were no obvious clinical "seizures".

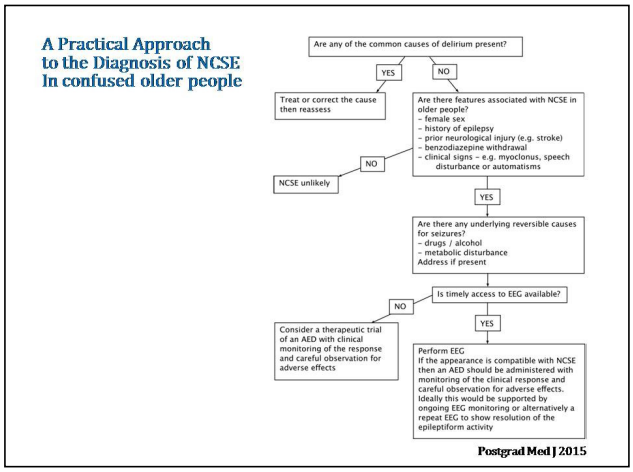
6 Clear-cut Criteria for NCSE in EEG

1. Frequent or continuous focal electrographic seizures, with ictal patterns that wax and wane with change in amplitude, frequency, and/or spatial distribution.
2. Frequent or continuous generalized spike-wave discharges in patients without a previous history of epileptic encephalopathy or epilepsy syndrome.
3. Frequent or continuous generalized spike-wave discharges, which showed significant changes in intensity or frequency (usually a faster frequency) when compared to baseline EEG, in patients with an epileptic encephalopathy or epilepsy syndrome.
4. PLEDs or BIPEDs that occurred in patients in coma in the aftermath of a generalized tonic–clonic status epilepticus (subtle status epilepticus).

6 Clear-cut Criteria for NCSE in EEG

5. EEG patterns that were less easy to interpret included: Frequent or continuous EEG abnormalities (spikes, sharp-waves, rhythmic slow activity, PLEDs, BIPEDs, GPEDs, triphasic waves) in patients whose EEGs showed no previous similar abnormalities, in the context of acute cerebral damage (e.g., anoxic brain damage, infection, trauma).
6. Frequent or continuous generalized EEG abnormalities in patients with epileptic encephalopathies in whom similar interictal EEG patterns were seen, but in whom clinical symptoms were suggestive of NCSE.

Reference for EEG findings of NCSE.
Sutter R and Kaplan PW.EEG criteria for NCSE: synopsis and comprehensive survey. Epilepsia 2012; 53(Suppl. 3):1-51.


A Practical Approach to the Diagnosis of NCSE In confused older people
Are any of the common causes of delirium present?
YES
NO
Treat or correct the cause then reassess
Are there features associated with NCSE in older people?
– female sex
– history of epilepsy
– prior neurological injury (e.g. stroke)
– benzodiazepine withdrawal
– clinical signs – e.g. myoclonus, speech disturbance or automatisms
NO
NCSE unlikely
YES
Are there any underlying reversible causes for seizures?
– drugs / alcohol
– metabolic disturbance
Address if present
Is timely access to EEG available?
NO
YES
Consider a therapeutic trial of an AED with clinical monitoring of the response and careful observation for adverse effects
Perform EEG
If the appearance is compatible with NCSE then an AED should be administered with monitoring of the clinical response and careful observation for adverse effects. Ideally this would be supported by ongoing EEG monitoring or alternatively a repeat EEG to show resolution of the epileptiform activity
Postgrad Med J 2015