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Wisdom of the Land

Challenges In Treatment of NCSE

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NCSE

- Definition & Classification
- Diagnosis
- Issues in specific age groups
- Treatment
- Prognosis & Outcome

Definition

- A state of ongoing or non-recovery between seizures without convulsion including altered cognitive or behavioral change, usually for more than 30 minutes along with evidence of seizures on EEG recording.

Kaplan PW. Epileptic Disord 2000;2:185-193.
Walker MC. CNS Drug 2001;15:931-939.
Kaplan PW. Neurologist 2005;11:348-360.
Wlaker M, et al. Epileptic Disord 2005;7:253-296.

Definition

- A term used to denote range of condition in which electrographic seizure activities is prolonged and results in nonconvulsive clinical symptoms.
  - Clinical symptoms are dependently largely on the level of cerebral development, epileptic syndrome, and location of epileptic activities
  - Electrographic activity can take various forms

Shorvon S. Epilepsia 2007;48Suppl 8:35-8.

Classification

- Various types of classifications according to age-groups, clinical presentations, and EEG
- Practical classification
  - Absence NCSE
  - Simple NCSE
  - Complex partial NCSE
  - Subtle NCSE

Pang T, Drislane FW. Curr Treat Op Neurol. 2012:14:307-31.
Chang AK, Shinnar S. Emerg Med Clin N Am 2011;29:65-72.

Classification of NCSE

1. NCSE occurring in the neonatal and infantile epilepsy syndromes
- 1a. *West syndrome*
- 1b. *Ohtahara syndrome*
- 1c. *Severe myoclonic encephalopathy of infancy (SMEI; Dravet syndrome)*
- 1d. NCSE in other forms of neonatal or infantile epilepsy

2. NCSE occurring only in childhood
- 2a. NCSE in *Early-onset benign childhood occipital epilepsy (Panayiotopoulos syndrome)*
- 2b. NCSE in other forms of childhood epileptic encephalopathies, syndromes and etiologies, e.g., *Ring chromosome X and other karyotype abnormalities*, Angelman syndrome, *Rett syndrome*, myoclonic-astatic epilepsy, other childhood myoclonic encephalopathies.
- 2c. *Electrical status epilepticus in slow wave sleep* (ESES)
- 2d. *Landau-Kleffner syndrome*

3. NCSE occurring in both childhood and adult life

With epileptic encephalopathy
- 3a. NCSE in the *Lennox-Gastaut syndrome*
  - i. Atypical absence status epilepticus
  - ii. Tonic-status epilepticus
- 3b. Other forms of NCSE in patients with learning disability or disturbed cerebral development (cryptogenic or symptomatic)

Without epileptic encephalopathy
- 3c. Typical absence status epilepticus in *idiopathic generalized epilepsy*
- 3d. Complex partial status epilepticus:
  - i. Limbic
  - ii. Nonlimbic
- 3e. NCSE in the postictal phase of tonic–clonic seizures
- 3f. Subtle Status epilepticus (myoclonic SE occurring in the late stage of convulsive SE)
- 3g. Aura continua (with: i. sensory, ii. special sensory, iii. autonomic, iv. cognitive symptoms)

4. NCSE occurring in late adult life
- 4a. De novo absence status epilepticus of late onset

5. Boundary syndromes*
- 5a. Some cases of epileptic encephalopathy.
- 5b. Some cases of coma due to acute brain injury with epileptiform EEG changes.
- 5c. Some cases of epileptic behavioral disturbance or psychosis.
- 5d. Some cases of drug induced or metabolic confusional state with epileptiform EEG changes.

*Boundary syndromes = cases in which it is not clear to what extent the continuous epileptiform electrographic abnormalities are contributing to the clinical impairment

Shorvon S. Epilepsia 2007;48Suppl 8:35-8.

Epidemiology

- Incidence: 5.6 – 18.3 per 100,000 individuals per year
- 4 – 25% of convulsive SE
- Up to 58% of patients did not have history of epilepsy
- 8 – 40% of comatose patients in ICU
  - High percentage in patient with infection, CVD, acute traumatic brain injury
  - 32% in children with critically ill
  - Subtle NCSE: not well documented

Towne AR, et al. Neurology 2000;54:340-5.
Saengpattrachai M, et al. Epilepsia 2006;47:1510-8.
Narayanan JT, Murthy JMK. Epilepsia 2007;48:900-6.
Abend NS, et al. Neurology 2011;76:1071-7.
Chang AK, Shinnar S. Emerg Med N Am 2011;29:65-72.

NCSE in Infants & Children

- Determination of significant changes in cognitive function or behavior is not straight forward as in adults
  - Children with preexisting delayed development
  - Children with intractable epilepsy and specific syndrome
- EEG issue
  - Chaotic base-line EEG

NCSE in Infants & Children

Suggested Criteria for Diagnosis of NCSE in Children

Clear and persistent clinical changes in behavior (including cognition, memory, arousal or motor behavior), confirmed by comparisons with previous functioning observation and/or by neuropsychological examination, in the presence of continuous paroxysmal electrographic activity, and in the absence of clonic, tonic or tonic-clonic seizures"

Walker M, et al. Epileptic Disord 2005;7:253-296.

Diagnosis

- Diagnosis is based on awareness of this condition, clinical suspicion
- Clinical presentation
- EEG monitoring
- Exclusion of other causes

Clinical Features of NCSE

- Functional changes
  - Slow mentation /responses
  - Disorientation
  - Confusion
  - Psychosis
  - Unresponsiveness
  - Altered behavior
- Motor manifestations
  - Gross movement ie. positioning, raising, limb flexion or extension, head deviation
  - Rhythmic myoclonia
  - Twitches
- Automatism
  - Gestural
  - Verbal
  - Mimicry

Chang AK, Shinnar S. Emerg Med Clin N AM 2011;29:65-72.

Clinical Features of NCSE in Infants & Children

- Apathy
- Absentmindedness
- Aggressiveness
- Decreased alertness
- Restlessness
- Pseudodementia
- Mutism
- Inappropriate verbal outbursts
- Oral automatism
- Perioral or facial twitching
- Regression
- Infantile behavior
- Head nodding
- Increased salivation
- Decreased eye contact
- Eye blinking, eye staring

Kaplan PW. Epilepsy Beh 2002;3:122-139.

Missed or Delayed Diagnosis of NCSE: Examples of Clinical Presentations

- Lethargy and confusion attributed to post-ictal state
- Ictal confusion mistaken of metabolic encephalopathy
- Unresponsiveness & catalepsy presumed to be psychogenic
- Hallucination and agitation secondary to hyperglycemia
- Mutism attributed to aphasia
- Laughing & crying ascribed to emotional liability

Kaplan PW. Epilepsy Beh 2002;3:122-139.

Red-flag for Recognition of NCSE

- Patient with generalized tonic-clonic seizure with prolonged postictal state
- Altered sensorium with subtle signs or fluctuation of mental status
- Stroke patients with clinical worsening than expected
- Elderly with unexplained stuporous or confusion
- Infant and child with intermittent altered vital signs and deterioration of consciousness

Korff CM, Nordli DR. Nature Clin Pract 2007;3:505-516.
Chang AK, Shinnar S. Emerg Med Clin N AM 2011;29:65-72.

Etiology

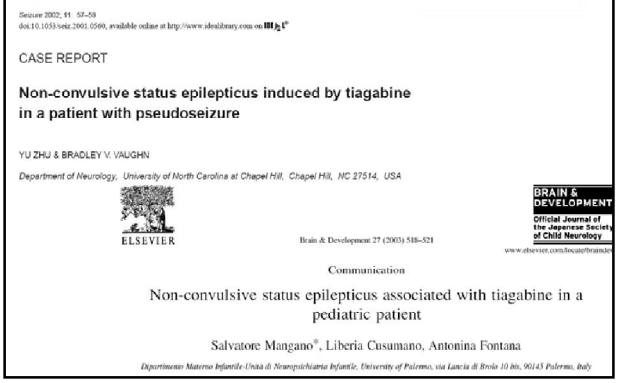
- Primary CNS pathology: remote & acute
  - Infection
  - Cerebrovascular diseases
  - Trauma
  - Ischemia
  - Pre-existing epilepsy
- Metabolic derangement
- Intoxication & drug induced
- Hormonal disturbance

Differential Diagnosis of NCSE

- Neurologic
  - Mitochondrial encephalopathies
  - Transient global amnesia
  - Posttraumatic amnesia
  - Confusional migraine
  - Vascular compromise
- Toxic/metabolic
  - Toxic encephalopathy
  - Drugs
  - Intoxications
  - Metabolic derangement
  - Withdrawal syndromes
- Epilepsy/seizure-related
  - Typical absence epilepsy
  - Atypical absence epilepsy
  - LGS with encephalopathy
  - Altered mental status with PLEDs/BPEDs/BiPLEDs
  - Prolonged postictal confusion
  - Postictal psychosis
- Psychiatric
  - Acute psychotic reaction
  - Dissociative conversion reaction
  - Malingering

Kaplan PW. Epilepsy Beh 2002;3:122-139.

Drug-induced NCSE: Tiagabine



Seizure 2002; 11: 57–59
doi:10.1053/seiz.2001.0560, available online at http://www.idealibrary.com on

CASE REPORT

Non-convulsive status epilepticus induced by tiagabine in a patient with pseudoseizure

YU ZHU & BRADLEY V. VAUGHN

Department of Neurology, University of North Carolina at Chapel Hill, Chapel Hill, NC 27514, USA

ELSEVIER

Brain & Development 27 (2003) 518–521

BRAIN & DEVELOPMENT
Official Journal of the Japanese Society of Child Neurology

www.elsevier.com/locate/braindev

Communication

Non-convulsive status epilepticus associated with tiagabine in a pediatric patient

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Diagnosis: Electrographic Criteria

Clear-cut criteria

- Frequent or continuous focal electrographic seizures with ital patterns that change in amplitude frequency or localization
- Frequent or continuous generalized spikes-and-waves in patients without prior history of epilepsy
- Frequent continuous generalized spikes-and-waves, significantly different in amplitude or frequency as compared to previous finding, in patients with a history of epileptic encephalopathy
- Periodic lateralized epileptiform discharges (PLEDs) in comatose patients after convulsive status epilepticus

Walker M, et al. Epileptic Disord 2005;7:253-296.

Diagnosis: Electrographic Criteria

Equivocal patterns

- Frequent or continuous electroencephalographic abnormalities in patients with acute cerebral injuries whose EEG showed no previous similar findings
- Frequent or continuous generalized spike-and-waves, not significantly different in amplitude or frequency as compared to previous findings, in patients with a history of epilepticencephalopathy whose clinical symptoms suggest NCSE

Walker M, et al. Epileptic Disord 2005;7:253-296.

NCSE in Elderly

- Risk from multiple co-morbidities and baseline cognitive impairment in elderly
- Unusual causes of NCSE: antibiotics
- Frequent subtle signs
  - Slight motor manifestation: eyelid myoclonia, automatism, nystagmus, dystonia, interrupted speech
  - Psychological manifestation: depression
- Risk of delayed treatment
- High morbidity & mortality: up to 57%

Bottoro FJ, et al. Epilepsia 2007;48:966-972.
Veran O, et al. Epilepsia 2009;5:1030-35.
Shavit L, et al. Euro J Intern Med 2012;23:701-4.
Chang YM. J Formosa Med Assoc 2013/dx.doi.org/10.1016/j.jfma.2013.05.002.

Treatment of NCSE

NCSE type	Treatment	Prognosis
Absence status epilepticus	Oral clobazam, intravenous lorazepam or intravenous valproic acid	Good
Simple partial status epilepticus	Oral clobazam, intravenous lorazepam or intravenous valproic acid	Good
Complex partial status epilepticus	Treatment of the underlying etiology. Oral clobazam, intravenous lorazepam, intravenous fosphenytoin or intravenous valrpoic acid	Uncertain; depends on etiology
NCSE in patients with learning difficulties	Oral clobazam, oral steroids. Surgery (multiple subpial transections)	Uncertain; good for seizures and electrographic abnormalities
NCSE in coma	Intravenous lorazepam, intravenous fosphenytoin, general anesthesia	Poor

Korff CM, Nordli DR Jr. Nature Clin Pract 2007;3:505-516.

Treatment

- Initial therapy: Benzodiazepines
- Second line
  - ASE: VPA
  - SPSE: less aggressive than generalized form
  - CPSE: similar to convulsive SE
  - Subtle NCSE: PHT, VPA and other AEDs
- New AEDs: levetiracetam, lacosamide
- Others: ketamine, topiramate, KD

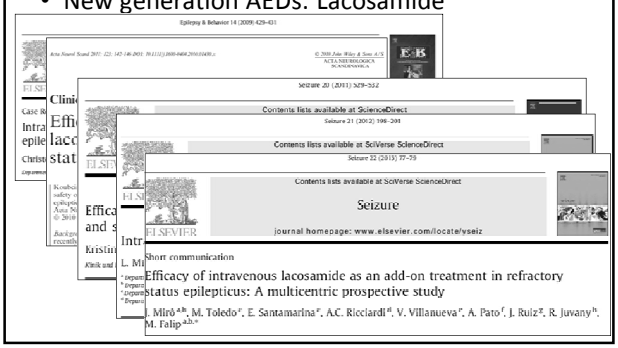
Pang T, Drislane FW. Curr Treat Op Neurol 2012;14:307-312.

Treatment of NCSE

- New generation AED: Levetiracetam

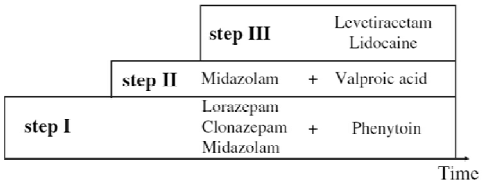
Treatment of NCSE

- New generation AEDs: Lacosamide



Treatment of NCSE

- Stepwise treatment 24 – 48 hrs in each step



Lorenzl S, et al. J Pain Sympt Manage 2008;36:200-205.

Types	Response to AEDs	Recurrence
I. Generalized nonconvulsive status epilepticus (GNSE)		
Ia. Absence status epilepticus (ASE)		
i. Typical absence status epilepticus (TAS)	Excellent	Frequently
ii. "de novo"absence status in the elderly	Good, but sometimes delayed	Occasionally (situation rated; triggers can be removed)
iii. Absence status with degenerative generalized epilepsies, progressive myoclonic epilepsies	Variable	Frequent
Ib. Atypical absence status epilepsy (AASE)	Relatively refractory (when seen in the setting of epileptic encephalopathy/ mental retardation)	Frequent
IIa. Simple Partial nonconvulsive status epilepticus (SPSE)	Excellent	Frequent
IIb. Complex partial status epilepticus (CPSE)		
i. Complex partial status epilepticus of frontal lobe origin (FCPSE)	Good to very good, but often delayed	Frequent
ii. Complex partial status epilepticus of temporal lobe origin (TCPSE)	Good to very good, but often delayed	Frequent

Kaplan PW. Neurologist 2005;11:348-361.

Types	Response to AEDs	Recurrence
III. NCSE Presentation by Age		
Electrical status epilepticus during slow sleep (ESES)		
Landau–Kleffner (acquired epileptic aphasia)		
Minor epileptic status of Brett	Relatively refractory (when seen in the setting of epileptic encephalopathy/ mental retardation)	Frequent
IV. Electrographic Seizures and Coma		
i. Subtle status usually postconvulsive status epilepticus (CSE)	Poor	Few
ii. With major CNS damage, often with multiorgan failure, (with facial, perioral, and/or limb myoclonias), but without apparent preceding CSE	Poor	Few

Kaplan PW. Neurologist 2005;11:348-361.

Ketamine & NCSE

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Age	4 years	7 years	4 years	6 years	4 years
Gender	M	F	F	M	F
Seizure disorder	LGS	PME	MAE	LGS	ABPE
Maintenance antiepileptic drugs	Sodium valproate, clonazepam	Sodium valproate, lamotrigine, ethosuximide	Lamotrigine, clonazepam, felbamate	Sodium valproate, clobazam, lamotrigine, topiramate	Sodium valproate, lamotrigine
Previous NCSE	++	++	++	++	+
Mean duration of previous NCSE	2–3 weeks	2–6 weeks	1–3 weeks	2–3 weeks	3 weeks
Interictal EEG findings	Diffuse slow background activity; bursts of generalized SW of maximal amp. Anteriorly (2–3/second, 300 μV)	Poor background activity; right parietal discharges	Slow background activity with occasional SW (2–3/second), more in left fronto-temporal regions	Disorganized background activity with frequent right hemisphere SW	Excess rhythmic theta activity with centro-temporal emphasis, runs of delta activity in frontal regions, few frontal SW
Clinical presentation	Slow mentation, ataxia, bradykinesia, drooling, myoclonic jerks, hypotonia	Slow mentation, ataxia, bradykinesia, dysarthria, myoclonic jerks	Slow mentation, ataxia, bradykinesia, palpebral myoclonias, drooling, hypotonia	Slow mentation, ataxia, bradykinesia	Slow mentation, ataxia, bradykinesia
Duration of NCSE prior to presentation	2 weeks	4 weeks	4 weeks	2 weeks	10 weeks
Treatment prior to ketamine	Lorazepam, prednisolone	Midazolam, lorazepam, prednisolone	–	–	–
EEG during NCSE	Continuous generalized SW (1.5–2.5/second, 500–800 μV)	Continuous generalized SW (2–3/second, 200–300 μV)	Continuous generalized spikes, SW and complexes of maximal amp. anteriorly (1.5–2.5/second, 200–300 μV)	Continuous generalized spikes, SW and complexes (~3/second, 100–200 μV)	Continuous generalized SW (1–2/second, 200–800 μV)
Response to ketamine[a]	Within 24 hours	Within 48 hours	Within 48 hours	Within 48 hours	Within 24 hours

Mewasingh LD, et al. Seizure 2003;12:483-489.

Ketamine in Refractory SE

Epilepsia, *(*):1–6, 2013
doi: 10.1111/epi.12247

FULL-LENGTH ORIGINAL RESEARCH

Intravenous ketamine for the treatment of refractory status epilepticus: A retrospective multicenter study

*Nicolas Gaspard, †Brandon Foreman, ‡Lilith M. Judd, §James N. Brenton, §Barnett R. Nathan,

SUMMARY

Purpose: To examine patterns of use, efficacy, and safety of intravenous ketamine for the treatment of refractory status epilepticus (RSE).

Methods: Multicenter retrospective review of medical records and electroencephalography (EEG) reports in 10 academic medical centers in North America and Europe, including 58 subjects, representing 69 episodes of RSE that were identified between 1999 and 2012. Seven episodes occurred after anoxic brain injury.

Key Findings: Permanent control of RSE was achieved in 57% (34 of 60) of episodes. Ketamine was felt to have contributed to permanent control ("possible" or "likely" responses) in 32% (19 of 60) including seven (12%) in which ketamine was the last drug added (likely responses). Four of the seven likely responses, but none of the 12 possible ones, occurred in patients with postanoxic brain injury. No likely responses were observed when infusion rates were lower than 0.9 mg/kg/h, when ketamine was introduced at least 8 days after SE onset, or after failure of seven or more drugs. Ketamine was discontinued due to possible adverse events in five patients. Complications were mostly attributed to concurrent drugs, especially other anesthetics. Mortality rate was 43% (26 of 60), but was lower when SE was controlled within 24 h of ketamine initiation (16% vs. 56%, p = 0.0047).

Significance: Ketamine appears to be a relatively effective and safe drug for the treatment of RSE. This retrospective series provides preliminary data on effective dose and appropriate time of intervention to aid in the design of a prospective trial to further define the role of ketamine in the treatment of RSE.

KEY WORDS: Ketamine, Treatment, Refractory status epilepticus, Anesthetics, Antiepileptic drugs.

Prognosis

- Various outcomes due to multiple confounding factors
  - Identifications of patients
  - Unavailable diagnostic tool (EEG) and proper interpretation
  - Correct classification of NCSE types
  - Lack of baseline data
  - Differentiating morbid effects of treatment from the morbidity of underlying cause of NCSE

Kaplan PW. Neurologist 2005;11:348-361.

Prognosis

- Poor outcomes in those with multiple medical problems, traumatic head injury, anoxia, refractory status epilepticus
- Morbidity varies with
  - Concomitant brain injury 27%
  - NCSE due to epilepsy 3%
  - Initial level consciousness (7% in obtundation VS 9% in deep lethargy)
- Types of NCSE
- Response to Treatment

Drislane FW, Schomer DL. Epilepsy Res 1994;19:111-121.
Shneker BF, Fountain NB. Neuology 2003;61:1066-1073.
Kaplan PW. Neurologist 2005;11:348-361.

Types	Prognosis	Outcome
I. Generalized nonconvulsive status epilepticus (GNSE)		
Ia. Absence status epilepticus (ASE)		
i. Typical absence status epilepticus (TAS)	Excellent	No morbidity or mortality
ii. "de novo"absence status in the elderly	Excellent	Excellent
iii. Absence status with degenerative generalized epilepsies, progressive myoclonic epilepsies	Guarded to fair	Variable, some with cognitive decline (difficulty in determining whether this is due to disease or to episodes of AASE)
Ib. Atypical absence status epilepsy (AASE)	Fair to poor	Frequent cognitive morbidity, but it is difficult to differentiate this from the effects of disease progression and consequences of comorbid seizure types
IIa. Simple Partial nonconvulsive status epilepticus (SPSE)	Usually good to excellent, occasionally poor	Morbidity and mortality negligible to absent
IIb. Complex partial status epilepticus (CPSE)		
i. Complex partial status epilepticus of frontal lobe origin (FCPSE)	When not associated with comorbid insults, good to excellent	Only very rare cognitive sequelae (<1% of patients)
ii. Complex partial status epilepticus of temporal lobe origin (TCPSE)	When not associated with comorbid insults, good to excellent	Only very rare cognitive sequelae (<1% of patients)

Kaplan PW. Neurologist 2005;11:348-361.

Types	Prognosis	Outcome
III. NCSE Presentation by Age		
Electrical status epilepticus during slow sleep (ESES)		
Landau–Kleffner (acquired epileptic aphasia)		
Minor epileptic status of Brett	Fair to poor	Frequent cognitive comorbidity, but it is difficult to differentiate this from the effects of disease progression and consequences of comorbid seizure types
IV. Electrographic Seizures and Coma		
i. Subtle status usually postconvulsive status epilepticus (CSE)	Poor	Particularly difficult to separate comorbidity from consequent morbidity, but overall high morbidity and mortality
ii. With major CNS damage, often with multiorgan failure, (with facial, perioral, and/or limb myoclonias), but without apparent preceding CSE	Poor	Particularly difficult to separate comorbidity from consequent morbidity, but overall high morbidity and mortality

Kaplan PW. Neurologist 2005;11:348-361.

Prognosis

Types	Prognosis	Diagnosis	Response to AEDs	Recurrence	Outcome
I. Generalized nonconvulsive status epilepticus (GNSE)					
Ia. Absence status epilepticus (ASE)					
i. Typical absence status epilepticus (TAS)	Excellent	Frequently missed	Excellent	Frequently	No morbidity or mortality
ii. 'de novo' absence status in the elderly	Excellent	Frequently missed	Good, but sometimes delayed	Occasionally (situation raised, triggers can be removed)	Excellent
iii. Absence status with degenerative generalized epilepsies, progressive myoclonic epilepsies	Guarded to fair	Less frequently missed	Variable	Frequent	Variable, some with cognitive decline (difficulty in determining whether this is due to disease or to episodes of ASE)
Ib. Atypical absence status epilepsy (ASSE)	Fair to poor	Less frequently missed	Relatively refractory (when seen in the setting of epileptic encephalopathy/ mental retardation)	Frequent	Frequent cognitive morbidity, but it is difficult to differentiate this from the effects of disease progression and consequences of comorbid/seizure types
IIa. Simple Partial nonconvulsive status epilepticus (SPSE)	Usually good to excellent, occasionally poor	Frequently missed	Excellent	Frequent	Morbidity and mortality negligible to absent
IIb. Complex partial status epilepticus (CPSE)					
i. Complex partial status epilepticus of frontal lobe origin (FCPSE)	When not associated with comorbid insults, good to excellent	Frequently missed	Good to very good, but often delayed	Frequent	Only very rare cognitive sequelae (< 1% of patients)
ii. Complex partial status epilepticus of temporal lobe origin (TCPSE)	When not associated with comorbid insults, good to excellent	Less frequently missed	Good to very good, but often delayed	Frequent	Only very rare cognitive sequelae (< 1% of patients)
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Electrical status epilepticus during slow sleep (ESES)					
Landau–Kleffner (acquired epileptic aphasia)					
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IV. Electrographic Seizures and Coma					
i. Subtle status usually postconvulsive status epilepticus (CSE)	Poor	Frequently missed	Poor	Few	Particularly difficult to separate comorbidity from consequent morbidity, but overall high morbidity and mortality
ii. With major CNS damage, often with multiorgan failure, (with facial, perioral, and/or limb myoclonus), but without apparent preceding CSE	Poor	Frequently missed	Poor	Few	Particularly difficult to separate comorbidity from consequent morbidity, but overall high morbidity and mortality

Kaplan PW. Neurologist 2005;11:348-361.

Markers for NSE

Epilepsia. 1995 May;36(5):475-9.

Neuron-specific enolase is increased after nonconvulsive status epilepticus.

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Abstract

Serum **neuron-specific enolase** (s-NSE), a marker of brain injury and acute seizures, was increased in 2 patients with **nonconvulsive** SE. Neither patient had an acute neurologic insult other than **nonconvulsive** SE (NCSE) accounting for s-NSE changes. Increase in s-NSE provides further in vivo evidence of transient brain injury after NCSE.

NCSE

- Often overlooked and frequently can be mistaken for other medical condition
- Diagnosis is based on awareness of this condition, clinical suspicion, EEG monitoring, and response to AED
- AED is effective at the right time
- NCSE must be included in D/Dx of coma
- High concern should be exercised in elderly, infant and young children especially in those with unexplained acute confusional state