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Adult nonconvulsive status epilepticus in a clinical setting: Semiology, aetiology, treatment and outcome

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ABSTRACT

Purpose: Our objective was to study the semiology, aetiology, treatment and outcome of nonconvulsive status epilepticus (NCSE) in adults.

Methods: All NCSE episodes in an unselected hospital cohort in the period 2004–2009 were identified, and the files reviewed. STESS (Status Epilepticus Severity Scale) was conducted retrospectively and correlated to outcome. Follow-up was undertaken after >2 years.

Results: 48 NCSEs in 39 patients, 22 men and 17 women, were found. Mean age was 63 years. 23/39 (59%) patients had established epilepsy. The underlying cause of NCSE was cerebrovascular disease in 17/39 (44%). 37/48 (77%) NCSEs were complex focal status epilepticus. 3/48 NCSEs (6.3%) lead to death, whereas 8.5% lead to severe sequelae. Cognitive sequelae were found after 14.9% of NCSEs. The outcome was worst in the group with no prior epilepsy ($p = 0.013$). STESS had a negative predictive value of 96% (cut-off value of 3) for severe sequelae and death combined ($p < 0.002$).

Conclusions: NCSE has a potential for severe sequelae and represents an emergency in need of intensive treatment. The major determinant of outcome is the underlying cause. The outcome was worse in patients without epilepsy than in patients with epilepsy. STESS is of value in predicting outcome. Cognitive sequelae following NCSE can occur, but need further investigation with prospective, systematic studies.

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1. Introduction

Status epilepticus (SE) comprises a range of disparate conditions, often categorised into convulsive and nonconvulsive SE. Generalised convulsive or tonic–clonic SE is easily recognised and the severity well established. The numerous other forms of SE are diverse with variable semiology and are thus more difficult to classify and study. Controversies regarding the term “convulsive” is one contributing reason.¹ Some authors define “convulsive” as widespread tonic–clonic activity generalised to the entire body, alternatively hemi-side, as opposed to minor motor activity during a focal seizure.² Motor features are frequent in complex focal seizures (CFS),^{3–5} including complex focal SE (CFSE),⁶ listed as a

subgroup of CFS in the ILAE classification of 1981.⁷ The Epilepsy Research Foundation workshop report from 2005 includes focal seizures with motor elements, e.g. *epilepsia partialis continua*, in NCSE group.⁸ Others define focal seizures with any motor elements as focal convulsive.^{9,10}

NCSE is commonly classified as generalised or focal. Generalised NCSE consists of absence, atypical absence and *de novo* late onset absence.¹¹ Some authors include myoclonic SE.⁸ Focal NCSE can be divided into simple and complex focal SE.^{8,11} Subtle SE is sometimes included in NCSE,^{9,12,13} but as it represents a late or end stage of generalised convulsive SE (GCSE), it is commonly considered separately.^{2,14} NCSE is often characterised by prolonged behavioural and cognitive changes as observed in CFS or absence seizures. Patients may experience subjective, vegetative symptoms or aphasia, etc., as seen in simple focal seizures (SFS). NCSE can occur as persistent unconsciousness with only EEG verification of ongoing epileptic activity, but it is difficult to avoid over-diagnosing NCSE if diagnosis is based mainly on EEG.¹⁵

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By its very nature, NCSE is often overlooked by clinicians, and hence both incidence and true outcome are more uncertain than for GCSE. Limited access to EEG contributes to diagnostic inaccuracy.¹⁶ In European studies, the annual incidence rates of NCSE range from 2.6 to 7.8 per 100,000, depending on the definition of “convulsive”.^{16–18} Lasting sequelae caused by NCSE is considered rare and also difficult to differentiate from the cause of the seizure and complications in the clinical course.^{11,19,20} Prognosis seems to be highly dependent on the underlying cause.²¹ Studies using standardised tests for neurological and neuropsychological sequelae are scarce. Occurrence of less prominent neuropsychological sequelae is not established.

The objective of this study was to examine the semiology, aetiology, treatment and outcome in our unselected patient cohort of NCSE. Much emphasis has been put on the presence or absence of focal motor elements. There is little evidence that this is important for the prognosis. We included NCSE satisfying the conservative definition of “non-convulsive”, excluding all motor elements, as well as seizures with limited motor features. STESS (Status Epilepticus Severity Score)¹³ was evaluated as a predictor of outcome.

2. Materials and methods

2.1. Study design

This is a retrospective study of 39 patients with 48 episodes of NCSE treated consecutively following the established routine at our adult neurological department (≥ 16 years). The department serves as the primary unit for 441,646 inhabitants (per 2003) of Hordaland County. There are no competing neurological departments in the county. The patient population is thus unselected. Status episodes were identified from a clinical database including all patients with an ICD-10 diagnosis of SE. We reviewed the files of all patients admitted 2004–2009, except for those registered with the code G41.0 (generalised tonic-clonic status epilepticus). The ICD-10 codes included were G41.1, G41.2, G41.8 and G41.9. The diagnoses were set at time of care by the treating neurologist. Aetiology of epilepsy, conditions triggering NCSE, duration of seizures, semiology, EEG findings, treatment and outcome measures were recorded. The aetiology of epilepsy was distinguished from the triggers of individual NCSE.

2.2. Definition, classification and duration of NCSE

NCSE was defined as epileptic seizures lasting more than 30 min and without major motor symptoms and/or convulsions. Convulsions were defined as widespread tonic-clonic activity generalised to the entire body or entire hemi-side. Hence, we included focal seizures with motor activity in NCSE. Motor symptoms, defined by simple or complex focal seizures and myoclonic seizures, were included. The length of SE was registered as 30–35 min, 35–60 min, 1–2 h, 2 h–1 day, 1 day–1 week, > 1 week, respectively.

Seizures were classified as “established epilepsy” or “no prior epilepsy”. NCSEs with prior epileptic seizures that occurred solely during the present acute situation and within a week before were classified as “no prior epilepsy”.

2.3. Exclusion criteria

Episodes initiated by a primary or secondary generalised tonic-clonic seizure (GTC) were excluded, hence also subtle SE. Similarly, NCSE episodes with a GTC within 24 h before onset of NCSE were excluded. Moreover, NCSE following anoxic brain damage was

excluded. We did not exclude patients without ictal EEG confirmation, as this would have led to a further selection bias.

2.4. Outcome measures

Outcome was separated into five groups: death, severe sequelae, moderate sequelae, mild sequelae, and full restitution. Neurological, cognitive, and somatic sequelae were considered for all categories. The classification of sequelae was based on clinical evaluation by a neurologist. Severe sequelae were defined as permanent conditions of major significance for daily living (e.g. paresis in a body part, significantly impaired memory or cognitive function, or other somatic disorders (see below)). Moderate sequelae were defined as either severe conditions lasting more than one month, but not being permanent, or permanent conditions with mild or moderate significance for daily living. Mild sequelae implied slight transient conditions or serious conditions lasting less than one month, but no permanent adverse outcome.²²

The sequelae were attributed to the NCSE, to the underlying aetiology and/or trigger, or to secondary consequences of these. Examples of consequences were deep venous thrombosis, pneumonia, and myocardial infarction.

One patient had two NCSEs two days apart, and these were combined for evaluation of sequelae. Minimum observation time was two years after the index NCSE.

STESS (Status Epilepticus Severity Score) was applied retrospectively.¹³ STESS is a prognostic score based on four outcome predictors: age (< 65 years = 0, ≥ 65 years = 1), history of previous seizures (yes = 0, no = 1), worst seizure type (simple focal, complex focal, absence, and myoclonic seizures = 0, generalised convulsive = 1, NCSE in coma/subtle SE = 2), and extent of consciousness impairment (alert or somnolent/confused = 0, stuporous or comatose = 1) as determined before start of treatment. We used a cut off of ≥ 3 and calculated the negative predictive value (NPV) for not dying or suffering severe sequelae in the group with a negative test (i.e. value < 3).

Two-sided Fischer's exact test was used to analyse the dichotomous categorical variables, i.e. “severe sequelae (including fatal outcome)/non-severe sequelae” vs STESS ≥ 3 , “fatal outcome/not fatal outcome” vs STESS ≥ 3 and “severe sequelae (including fatal outcome)/non-severe sequelae” vs “established epilepsy/no prior epilepsy”. In patients with more than one NCSE, only the first NCSE was included in these analyses to avoid dependency between variables.

All the patients were contacted in writing a minimum of two years after the NCSE to investigate their current function. Those capable of completing simple questionnaires were presented with the 5-item (5 level) EuroQol survey (EQ-5D) and the 10-item Quality of Life in Epilepsy survey (QOLIE-10).^{23–25}

3. Results

3.1. Patient characteristics

Forty-eight NCSE episodes in 39 patients were examined: 22 men and 17 women. Two men had three episodes each; two women and three men had two episodes each. Mean age was 63 years, range 18–96 years.

Of the 39 patients, 25 (64%) had suffered epileptic seizures before their index NCSE; however, for two of them (two patients, three seizures) this had happened only two days prior to the NCSE. Hence 23/39 (59%) of the patients (contributing to 31/48 of the NCSEs) qualified for a diagnosis of *established epilepsy*. Of the 16 patients (41%) with *de novo seizures*, seven had acute symptomatic seizures and the remaining had remote underlying aetiology, but

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