



Contents lists available at ScienceDirect

Seizure: European Journal of Epilepsy

journal homepage: www.elsevier.com/locate/seizure

Elderly patients with nonconvulsive status epilepticus: Clinical-EEG data, hospital mortality, STESS and EMSE

Gloria MAS Tedrus^{a,*}, Elizardo Nogueira Junior^b, Mariana Almeida Vidal^b

^a Postgraduate Program in Health Sciences, PUC-Campinas

^b Clinical Neurology Service - University Hospital, PUC-Campinas

ARTICLE INFO

Keywords:

Elderly
EEG
Mortality
Non-convulsive status epilepticus

ABSTRACT

Purpose To assess the clinical-EEG aspects, characterization of subtypes, relationships with prognostic scales and the occurrence of death in elderly patients in the acute phase of nonconvulsive status epilepticus (NCSE).

Methodology Clinical variables, EEG data, Status epilepticus severity score (STESS), and the Epidemiology-based mortality score in status epilepticus (EMSE) were related to the death of 96 patients who were over 60 years old, with NCSE.

Results NCSE with coma was observed in 31 patients (19 non-subtle and 12 “subtle” SE) and focal NCSE with impairment of consciousness in 65 cases. There were no significant EEG differences according to the type of NCSE. Higher STESS scores occurred in the comatose NCSE patients when compared to those with focal NCSE and impairment of consciousness (4.8 ± 1.2 vs 3.7 ± 1.2 ; T-Test; $p < 0.001$). It was observed that 25 (26%) elderly died, with a mean survival time of 19.3 days. Elderly people with a higher risk of death are those diagnosed with NCSE with coma (HR 2.76; 95% CI 1.15–6.65; $p = 0.023$), with $STESS \geq 3$ (HR 16.0; CI 1.77–45.08; $p = 0.014$), with $EMSE \geq 64$ (HR 3.67; CI 1.54–8.72; $p = 0.003$), and those with no history of recurrent SE (HR 6.80; CI 1.42–32.64; $p = 0.017$), in Cox regression.

Conclusion The ictal EEG patterns did not distinguish the subtypes of NCSE. Thirty-day mortality rate was high in elderly patients with NCSE. The clinical variables are related to prognosis. Mortality in the elderly was associated with comatose NCSE patients, with $STESS \geq 3$, with $EMSE \geq 64$, and no history of recurrent SE.

1. Introduction

Mortality in elderly people with status epilepticus (SE) is high, almost 3 times higher than in young adults, and is associated with the type, duration, severity, the presence of comorbidities, and *de novo* SE [1–3]. Clinical-EEG characteristics with the prognostic values of SE have been investigated and, recently, clinical scales to predict mortality in SE have been created [4–6].

Nonconvulsive status epilepticus (NCSE) contemplates several types of clinical conditions, nonspecific and pleomorphic clinical manifestations, and represents a prolonged state of seizures, without convulsions, without interictal full recovery, and it often goes unrecognized, leading to delays in diagnosis and treatment [7,8].

In the elderly, NCSE is the most common status epilepticus [9], with a prevalence of 26.6% [10,11] and an incidence of 55–86 cases per 100,000 individuals [12]. There are still gaps in the identification of variables associated with the prognosis for NCSE in the elderly. The potential of

NCSE to cause brain damage is controversial, so the rapid identification of the factors that predict the clinical outcome and survival condition is important and can be useful for the therapeutic decision, in this age group.

The EEG data in NCSE patients have been debated for the last 20 years and only recently there was a consensus on the EEG criteria for the diagnosis of NCSE [8,13]. In recent years, some studies have assessed the accuracy and adequacy of these criteria [14,15] and, in 2021, the American Clinical Neurophysiology Society included new EEG terms and concepts for critically ill patients [16].

The aim of this study was to assess the clinical-EEG aspects, its subtypes, and the relationships with the prognostic scales and with the occurrence of death in elderly patients in the acute phase of the NCSE.

2. Methods

This was a prospective study that assessed 96 consecutive patients

* Corresponding author at: Av. John Boyd Dunlop s/n - Jardim Ipaussurama, Campinas - SP, Brazil.

E-mail address: gmtedrus@uol.com.br (G.M. Tedrus).

<https://doi.org/10.1016/j.seizure.2021.11.004>

Received 10 September 2021; Received in revised form 18 October 2021; Accepted 8 November 2021

Available online 11 November 2021

1059-1311/© 2021 The Authors. Published by Elsevier Ltd on behalf of British Epilepsy Association. This is an open access article under the CC BY-NC-ND license

(<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

over 60 years old, who met the NCSE diagnostic criteria of the International League Against Epilepsy (ILAE) Task Force on Classification of SE and Salzburg criteria[8,13]. All patients presented, at the initial assessment, a clinical picture characterized by decreased cognitive performance or level of consciousness, mental confusion, or behavioral change (continuous or with fluctuation). On the EEG, they presented alterations that may suggest NCSE. Cases diagnosed with hypoxic-ischemic encephalopathy secondary to cardiopulmonary arrest were excluded.

Patients seen at the clinical neurology service, in the emergency or in the intensive care unit of the University Hospital of the PUC—Campinas, SP, Brazil. The Human Research Ethics Committee of Pontifical Catholic University of Campinas approved the study.

2.1. Clinical assessment

The presence, degree, and occurrence of fluctuations in the level of consciousness, the presence of minor motor manifestations, and the time of onset of symptoms were assessed. This information was collected from patients or family members, or by the Intensive Care Unit's medical team. Data on age, sex, medical history and history of epilepsy or SE, the results of imaging (computerized tomography and/or magnetic resonance) and screening tests for metabolic and infectious disorders were collected. NCSE etiology was determined according to ILAE criteria and categorized into: 1) Known - with acute or remote injury, 2) Unknown [8].

2.2. EEG data

The EEG was performed with a duration of 30–60 min as soon as possible after patient admission, using international 10–20 system, with 21 fixed gel electrodes and auricular electrodes as a reference in a Braintech 3.0 equipment (EMSA Equipamentos Médicos). The EEG was repeated in a short period, until the resolution of the clinical picture, as recommended in the literature[13]. Neurologists trained in EEG and epilepsy blindly interpreted the EEGs.

The EEGs parameters used for the definitive diagnosis of NCSE were: the presence of epileptiform discharges (ED) [spikes, sharp waves, polyspikes] >2.5 Hz, or ED ≤ 2.5 Hz or rhythmic delta/theta activity (RDA) (>0.5 Hz); and one of the following criteria: 1) EEG and clinical improvements after intravenous antiseizure medications (ASM), 2) subtle clinical ictal phenomena in the EEG patterns, 3) typical ictal-EEG spatiotemporal evolution (frequency, amplitude, and location). The presence of superimposed (modifiers - plus) fast activity (+F), or RDA (+R), or spikes and sharp waves (+S)[13] was assessed.

2.3. Scales used to estimate in-hospital mortality at the time of diagnosis

- Status Epilepticus Severity Score (STESS)⁵: it is a prognostic score based on four clinical criteria on admission - a) age (< 65 years old; ≥ 65 years); b) level of consciousness (awake or somnolent; comatose); c) worst clinical seizure type (focal; generalized-convulsive; NCSE with coma); d) history of previous seizures (yes/ no).

The score ≥ 3 was used as a negative predictive cutoff score, as initially proposed by Rossetti et al. (2008)[4]. The score ≥ 4 , as proposed by Sutter et al. (2013)[5], was also used as a negative predictive cutoff score.

- Epidemiology-based Mortality Score in Status Epilepticus (EMSE)⁶: This scale includes clinical factors - a) age (age group); b) etiology (withdrawal from ASM; metabolic disorder or brain injury); c) comorbidities (yes/ no); d) EEG-pattern (periodic discharges, burst suppression). An EMSE score ≥ 64 was the cutoff score for a high risk of mortality.

After confirming the diagnosis of NCSE, the protocols for SE were applied and intravenous benzodiazepines and ASM were used. Cases

refractory to the initial treatment were treated with anesthetic doses of midazolam and/or propofol.

These cases were classified into NCSE subtypes, according to the ILAE[8] criteria. Clinical-EEG data, STESS and EMSE scores, and the occurrence of mortality were related to the types of NCSE. For this study, mortality data during hospitalization were considered within the 30-day period after the diagnosis of NCSE.

2.4. Statistical analysis

The categorical variables were described using absolute values and percentages, and continuous variables, such as mean and standard deviations. The student's *t*-test, analysis of variance (ANOVA), Mann-Whitney U test and Pearson chi-squared test were used to compare the continuous and categorical variables. The Pearson correlation coefficient was used to assess the degree of associations among the quantitative variables.

Cox univariate regression with multiple analysis was used to model the death rate in relation to clinical variables, EEG data, and STESS and EMSE scores. A univariate Cox regression analysis was performed to assess the factors associated with death. The variables (recurrent SE, type of NCSE, STESS, and ENSE) were used. These, together with the univariate regression analysis, were good factors to explain the risk of death. For the multiple regression analysis, the backward process was used to select the variables.

The study data were analyzed using the Statistical Packages for Social Sciences software, version 23, and a *p*-value of <0.05 was considered statistically significant.

3. Results

3.1. NCSE: clinical aspects and subtypes

A total of 96 elderly patients with a mean age of 72.3 (± 8.3) years were included. Of these patients, 31 cases were classified as NCSE with coma and 65 as focal NCSE with impairment of consciousness, according to the ILAE clinical-EEG criteria[8]. There was no significant difference in age, sex, and etiology according to the type of NCSE (Table 1).

In the 65 elderly patients with focal NCSE with impairment of consciousness, the clinical manifestation was a decreased cognitive performance or level of consciousness, mental confusion or continuous or fluctuating behavioral changes, with an average duration of 10 h.

Elderly individuals classified as NCSE with coma presented a clinical picture of persistent coma without apparent cause and were divided into 2 groups: 1) "subtle" SE[17] consisting of 12 cases that presented lowered level of consciousness after a sequence of generalized convulsive seizures or SE convulsive. 2) non-subtle SE composed of 19 comatose patients, without clinical signs of seizure activity.

There were subtle motor manifestations, twitching of mouth or periorbital region, sustained eye deviation or ocular movements in 34 (35.4%) individuals. There was a higher occurrence of automatisms in focal NCSE with impairment of consciousness than in NCSE with coma (Table 1). Stereotyped automatism and previous history of epilepsy were observed respectively in 5 (41.6%) cases and in 4 (33%) of elderly with "subtle" SE. In the elderly with non-subtle SE, the presence of automatisms was not observed and there was no previous history of seizures or epilepsy.

Structural cerebral alteration was observed in 64 (66.6%) cases and in 32 (33.3%) cases the cause could not be identified (cryptogenic). The brain injury was remote in 40 (62.5%) cases and acute in 24 (37.5%) cases. There was no association between structural changes and the type of NCSE (Table 1).

3.2. EEG

There was a record of focal electroclinical SE (ECSE-focal) in 64

Table 1
Demographic aspects, clinical-EEG data, and STESS and EMSE scores according to the type of NCSE.

	Focal with impairment of consciousness (n = 65)	NCSE with coma (n = 31)	p
Age (years old)	72.3 (±8.3)	72.1 (±8.6)	0.994 ^a
Women	39 (60%)	18 (58%)	0.857 ^b
Death	16 (24.6%)	9 (29%)	0.630 ^c
Etiology: Structural/ cryptogenic	39/26	25/6	0.463 ^c
Structural			
Cerebrovascular disease (acute or remote)	23 (35.3%)	9 (29%)	
Dementias	1 (1.5%)	1 (3.2%)	
Brain tumor	2 (3.0%)	2 (6.4%)	
Subarachnoid or subdural hemorrhage	5 (7.6%)	4 (12.9%)	
Head trauma	3 (4.6%)	4 (12.9%)	
Others	5 (7.6%)	5 (16.1%)	
Initial clinical manifestation			
Coma	–	31 (100%)	
Decreased level of consciousness	65 (100%)	–	
Stereotyped automatisations EEG	29 (44.6%)	5 (16.1%)	0.007 ^{*,*}
ECSE-focal (n = 64)	46 (70.7%)	18 (58%)	0.217 ^{hi}
Presence of LPDs/RDA	15/4	12/q	0.625 ^c
STESS (total score)	3.7 (±1.2)	4.8 (±1.2)	<0.001 ^d
			*
Level of consciousness at onset:			
Alert or somnolent; confused/ Stuporous or comatose	65/0	0/31	<0.001 ^{*,*}
Seizure type at onset:			
Generalized convulsive/ Nonconvulsive	0/65	12/19	<0.001 ^{*,*}
Age at onset: (65; > 65)	12/53	6/25	1.0 [†]
History of seizure at onset: yes; no	16/49	26/5	0.434 ^c
EMSE (total score)	55.3 (±34.4)	64.8 (±34.1)	0.158 ^d
Etiology	13.1 (±8.7)	15.2 (±8.0)	0.252 ^a
Comorbidity	17.6 (±18.4)	15.7 (±18.1)	0.635 ^a
Age	7.9 (±2.9)	9.9 (±6.3)	0.094 ^a
EEG	16.8 (±20.4)	23.8 (±21.0)	0.128 ^a

NCSE: Nonconvulsive status epilepticus; STESS: Status epilepticus severity score; EMSE: Epidemiology-based mortality score in status epilepticus; RDA: Rhythmic delta activity; LPDs: lateralized periodic discharges; Plus: superimposed activity; ECSE-focal: focal electroclinical status epilepticus;

^a : T Test.
^b : chi-square test.
^c : Fisher's exact test.
^d Mann-Whitney U test.
* p<0.05.

(66.6%) cases. The ED were regional and located in the temporal and posterior regions in 49 (76%) cases, and in the anterior regions in 15 cases. The ictal pattern was characterized by a high frequency of ED, and the discharges were continuous, with a duration of ≥20% of the EEG, with typical ictal spatiotemporal evolution on the EEG and association (or not) with subtle clinical findings. Lateralized periodic discharges (LPDs) were observed in 6 cases, and RDA em 9 cases. During the EEG, intravenous ASM were used in 49 cases and the ED disappeared in 30 (61.2%) of these cases, being associated with clinical improvement in only 17 (34.6%) cases.

In 32 cases, ECSE-focal could not be identified. LPDs were observed in 27 cases, being very frequent in 14 cases and continuous in 13 cases. LPDs involved the whole hemisphere in 5 cases or were regional or focal in 21 cases, and, in one case, the LPD were bilateral, markedly asymmetric. Superimposed (modifiers - plus) was observed in 18 cases. In 5

cases there was a quasi-continuous lateralized RDA during the EEG. In all cases, a superimposed (modifiers - plus) fast activity was observed (+F in 4 cases, +FS in 1 case).

There was no significant difference in EEG aspects according to the type of NCSE (Table 1).

3.3. NCSE: outcome, STESS, EMSE

STESS scores were higher in NCSE with coma when compared to those observed in focal NCSE with impairment of consciousness. There was no significant difference in the total EMSE scores when comparing focal NCSE with impairment of consciousness and NCSE with coma (Table 1).

A total of 25 elderly people with NCSE did not recover and died within 30 days of the onset of the condition, while in hospital. Demographic aspects, clinical-EEG variables, and STESS and EMSE scores according to the occurrence of death are shown in Table 2.

In the Cox multivariate regression analysis to assess factors associated with death, it was observed that no variable, analyzed individually, significantly changes the risk of death. In the multivariate analysis, in Cox regression, a higher risk of death was observed for the elderly who do not have recurrent SE and who were diagnosed with NCSE with coma, and with STESS≥3 and EMSE≥64 (table 3).

4. Discussion

This study assessed the clinical aspects and EEG data of elderly patients in the acute phase of NCSE with coma and with focal NCSE with impairment of consciousness according to the ILAE[8] criteria and related them with outcome scale data and with the occurrence of death within up to 30 days from diagnosis. Studies assessing prognosis of NCSE subtypes in the elderly are rare.

When comparing the NCSE subtypes, no differences in sex and age were observed. NCSE was associated with different etiologies, acute or chronic, with a predominance of vascular cause, with no differences according to the type of NCSE. Similar data have been described in population and clinical studies in elderly people with NCSE[11,18].

Table 2
Demographic aspects, clinical-EEG data, and STESS and EMSE scores, according to the occurrence of death in thirty-day in 96 elderly patients with NCSE.

	Deceased (n = 25)	Not deceased (n = 71)	p
Women	14 (56%)	43 (60.5%)	0.813 ^a
Age (years old)	71.7 (±7.2)	72.5 (±8.7)	0.628 ^b
STESS (mean score)	4.4 (±1.1)	4.0 (±1.3)	0.169 ^b
STESS≥3	24 (96%)	61 (85.9%)	0.278 ^c
STESS≥4	21 (84%)	51 (71.8%)	0.289 ^c
EMSE (mean score)	75.2 (±36.9)	52.5 (±31.7)	0.010 ^{h,*}
EMSE ≥64	15 (60%)	19 (26.7%)	0.004 ^{*,*}
Previous epilepsy	6 (24%)	15 (21.1%)	0.783 ^c
Recurrent SE	2 (8%)	12 (16.9%)	0.344 ^c
Etiology: Structural/ cryptogenic EEG	15/10	49/22	0.463 ^c
Presence of RDA	9 (36%)	12 (16.9%)	0.047 ^{*,*}
Presence of LPDs	16 (64%)	27 (38%)	0.025 ^{*,*}
ECSE-focal	13 (52%)	51 (71.8%)	0.070 ^c
NCSE with coma/ focal with impairment of consciousness	9/16	22/49	0.630 ^c
NCSE with coma: Non-subtle/ Subtle	6/3	13/9	1.0 [†]

NCSE: Non-convulsive status epilepticus; SE: status epilepticus; STESS: Status epilepticus severity score; EMSE: Epidemiology-based mortality score in status epilepticus; RDA: Rhythmic delta activity; LPDs: Lateralized periodic discharges; Plus: Superimposed activity; ECSE-focal: focal electroclinical status epilepticus.

^a : chi-square test.
^b : T Test.
^c : Fisher's exact test. *p<0.05.

Table 3
Cox multivariate regression analysis to assess factors associated with death.

Variable	Effect vs Reference	p-value	HR	CI _{95%HR}
Recurrent SE	No vs Yes	0.017*	6.80	1.42 - 32.64
NCSE	NCSE-coma vs focal with impairment of consciousness	0.023*	2.76	1.15 - 6.65
STESS _{≥3}	Yes, vs No	0.014*	16.0	1.77 - 45.08
EMSE _{≥64}	Yes, vs No	0.003*	3.67	1.54 - 8.72

HR: hazard ratio; SE: status epilepticus; EMSE: Epidemiology-based mortality score in status epilepticus; STESS: Status Epilepticus Severity Score.

* : $p < 0.05$.

Clinical history of epilepsy occurred in 22% of the elderly, with a higher occurrence in patients with “subtle” SE when compared to those with non-subtle SE. Some studies describe that the history of seizures, epilepsy or SE in elderly people with NCSE is low[1,11].

A greater occurrence of automatisms was observed in the elderly with focal NCSE with impairment of consciousness. Among the elderly with NCSE with coma, stereotyped automatisms occurred, as expected, only in the elderly with “subtle” SE. No similar studies were found in the literature.

There was no difference in the ictal EEG patterns according to the NCSE type and thus the EEG data did little to distinguish the NCSE subtypes assessed. Similar data have already been described in other studies[19]. Despite advances in the area, there are few studies that assess EEG patterns in NCSE subtypes. EEG findings in the NCSE, especially in patients with NCSE with coma remain under debate even among experts. Some studies assess the various EEG aspects, including borderline patterns, which allowed for the categorization of NCSE, in different age groups, into specific syndromes[20].

4.1. Thirty-day mortality rate, STESS, EMSE

Mortality in the acute phase was high and occurred in 26% of the elderly people with NCSE. The data found in this study are similar to those described in other studies[21,22]. Elderly people with NCSE have a less favorable prognosis, higher mortality rates and longer periods of hospitalization when compared to younger patients[1,11,21,23,24]. Studies assessing the prognosis in elderly people with NCSE are rare.

There was no relationship between the favorable or unfavorable prognosis and the type of etiological factor, whether acute or remote. Studies with different samples associate greater severity and mortality to the underlying cause of NCSE in the elderly[9,10,11,23], similarly to what has been found in adults[7,25-27].

It was observed that elderly people with repetitive NCSE have a better outcome. Similar data have been described in elderly people with a positive history of epilepsy, which may suggest an association between *de novo* SE and severe acute brain structural injury that results in poor outcome^{9,11,21,25}.

In the univariate analysis, it was observed that higher mortality rates were associated with the presence of RDA and LPD on the EEG, as described in some studies[22,24,29]. In the Cox regression multivariate analysis, the EEG data did not remain in the equation to predict the risk of death. Some studies describe that the presence of LPD has a worse outcome, regardless of etiology, in the elderly[24] and in adults with NCSE[27]. Despite the importance of the EEG in the diagnosis and follow-up of patients with NCSE, the predictive significance of the EEG aspects is still not fully known[19,20,24,25,28,29].

These data confirm that the occurrence of NCSE with coma is a robust predictor for the poor outcome. Literature data describe a higher risk of death in comatose patients with NCSE among the various types of NCSE in the elderly[24,25,30]. There were no differences in mortality

rates according to the subtype of NCSE with coma (“subtle” SE and non-subtle SE). There is a scarcity in the literature of studies comparing the prognosis in NCSE subtypes in the elderly.

In this study, it was observed that STESS_{≥3} and EMSE_{≥64} were associated with an increased risk of death. Higher STESS scores associated mortality with NCSE[22]. Other studies describe, in different types of SE, that the EMSE showed a better positive predictive value in the elderly when compared to younger adults[11].

In conclusion, ictal EEG patterns did not distinguish NCSE subtypes. Mortality was high in elderly people with NCSE. Clinical variables are related to the unfavorable prognosis. In elderly with NCSE with coma and no history of recurrent SE, EMSE_{≥64} and STESS_{≥3} scores were associated with higher risk of death. The data indicate that the EMSE and STESS scales and clinical variables are useful factors to differentiate the possibility of death and survival in elderly patients with NCSE.

5. Limitations

This study has several limitations. Data were obtained from a single healthcare center, which makes it difficult to generalize the results. While 96 is a significant sample size for any institution, due to the patients’ subcategorization into either different etiologies or EEG findings or outcomes, the actual sample size to calculate individual predictors becomes small. Another limitation of the study was the lack of systematic screening for autoimmune encephalitis, which may have contributed to the high number of cases classified as cryptogenic. The data in this study need to be confirmed with a different sample.

Author statement

ENJ, MAV contributed to study conception and design, data acquisition and interpretation, critical revision of the manuscript, and final approval.

GMAST contributed to study conception and design, data acquisition, analysis, and interpretation, drafting and critical revision of the manuscript, final approval, and agrees to be accountable for all aspects of this study, ensuring integrity and accuracy.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Declaration of Competing Interest

The authors have no financial or nonfinancial relationships that could impact the content of this article.

References

- [1] DeLorenzo RJ, Towne AR, Pellock JM, Ko D. Status epilepticus in children, adults, and the elderly. *Epilepsia* 1992;33:S15–S85. <https://doi.org/10.1111/j.1528-1157.1992.tb06223.x>.
- [2] Towne AR. Epidemiology and outcome of status epilepticus in elderly. In *Rev Neurobiol* 2007;81:111–27. [https://doi.org/10.1016/S0074-7742\(06\)81007-X](https://doi.org/10.1016/S0074-7742(06)81007-X). PMID: 17433920.
- [3] Ong CT, Sheu SM, Tsai CF, Wong YS, Chen SC. Age-dependent sex difference of the incidence and mortality of status epilepticus: a twelve-year nationwide population-based cohort study in Taiwan. *PLoS ONE* 2015;10:53. <https://doi.org/10.1371/journal.pone.0122350>.
- [4] Rossetti AO, Logroscino G, Milligan TA, Michaelides C, Ruffieux C, Bromfield EB. Status epilepticus severity score (STESS): a tool to orient early treatment strategy. *J Neurol* 2008 Oct;255(10):1561–6. <https://doi.org/10.1007/s00415-008-0989-1>. Epub 2008 Sep 3. PMID: 18769858.
- [5] Sutter R, Kaplan PW, Ruegg S. Independent external validation of the status epilepticus severity score. *Crit Care Med* 2013 Dec;41(12):e475–9. <https://doi.org/10.1097/CCM.0b013e31829eca06>. PMID: 23982031.
- [6] Leitinger M, Holler Y, Kalss G, Rohrer A, Novak HF, Hofler J, et al. Epidemiology-based mortality score in status epilepticus (EMSE). *Neurocrit Care* 2015 Apr;22(2):273–82. <https://doi.org/10.1007/s12028-014-0080-y>. PMID: 25412806.

- [7] Kaplan PW. Assessing the outcomes in patients with nonconvulsive status epilepticus: nonconvulsive status epilepticus is underdiagnosed, potentially overtreated, and confounded by comorbidity. *J Clin Neurophysiol* 1999 Jul;16(4): 341–52. <https://doi.org/10.1097/00004691-199907000-00006>. discussion 353 PMID: 10478707.
- [8] Trinka E, Cock H, Hesdorffer D, Rossetti AO, Scheffer IE, Shinnar S, et al. A definition and classification of status epilepticus – report of the ILAE task force on classification of status epilepticus. *Epilepsia* 2015 Oct;56(10):1515–23. <https://doi.org/10.1111/epi.13121>. Epub 2015 Sep 4 PMID: 26336950.
- [9] Canoui-Poitrine F, Bastuji-Garin S, Alonso E, Darcel G, Verstichel P, Caillet P, et al. Risk and prognostic factors of status epilepticus in the elderly: A case-control study. *Epilepsia* 2011;52:1849–56. <https://doi.org/10.1111/j.1528-1167.2011.03168.x>.
- [10] Vilela L, Gonzales-Cuevas M, Luque MQ, Toledo M, Gil MS, Salas-Puig J, et al. Prognosis of status epilepticus in elderly patients. *Acta Neurol Scand* 2018 Mar;137(3):321–8. <https://doi.org/10.1111/ane.12867>. Epub 2017 Nov 22 PMID: 29168175.
- [11] Rohrer A, Reiter DP, Brigo F, Kalss G, Thomschewski A, Novak H, et al. Status epilepticus in the elderly – a retrospective study on 120 patients. *Epilepsy Res* 2016 Nov;127:317–23. <https://doi.org/10.1016/j.eplepsyres.2016.08.016>. Epub 2016 Aug 22 PMID: 27694014.
- [12] Meerkord Boon P, Engelsen B, Gocke K, Shorvon S, Tinuper P, et al. European Federation of Neurology Societies. EFNS guideline on the management of status epilepticus in adults. *Eur J Neurol* 2010 Mar;17(3):348–55. <https://doi.org/10.1111/j.1468-1331.2009.02917.x>. Epub 2009 Dec 30 PMID: 20050893.
- [13] Hirsch LJ, LaRoche SM, Gaspard N, Gerard E, Svoronos A, Herman ST, et al. American Clinical Neurophysiology Society's Standardized Critical Care EEG Terminology: 2012 version. *J Clin Neurophysiol* 2013;30:1–12. <https://doi.org/10.1097/WNP.0b013e3182784729>. PMID: 23377439.
- [14] Leitinger M, Beniczky S, Rohrer A, Gardella E, Kalss G, Qerama E, et al. Salzburg consensus criteria for non-convulsive status epilepticus – approach to clinical application. *Epilepsy Behav* 2015 Aug;49:158–63. <https://doi.org/10.1016/j.yebeh.2015.05.007>. Epub 2015 Jun 17 PMID: 26092326.
- [15] Leitinger M, Trinka E, Gardella E, Rohrer A, Kalss G, Qerama E, et al. Diagnostic accuracy of the Salzburg EEG criteria for non-convulsive status epilepticus: a retrospective study. *Lancet Neurol* 2016;15(10):1054–62. [https://doi.org/10.1016/S1474-4422\(16\)30137-5](https://doi.org/10.1016/S1474-4422(16)30137-5). PMID: 27571157.
- [16] Hirsch LJ, Fong MWK, Leitinger M, LaRoche SM, Berniczky S, Abend NS, et al. American Clinical Neurophysiology Society's Standardized Critical Care EEG Terminology: 2021 version. *J Clin Neurophysiol* 2021;38:1–29. <https://doi.org/10.1097/WNP.0000000000000806>.
- [17] Treiman DM, DeGiorgio CMA, Salisbury SM, Wickboldt CL. Subtle generalized convulsive status epilepticus. *Epilepsia* 1984;25:653. <https://doi.org/10.1111/j.1528-1157.1993.tb05902.x>.
- [18] Kanake S, Rosenow F, Vescovi M, Oertel WH, Mueller HH, Wirbatz A, et al. Incidence of status epilepticus in adults in Germany: a prospective, population-based study. *Epilepsia* 2001 Jun;42(6):714–8. <https://doi.org/10.1046/j.1528-1157.2001.01101.x>. PMID: 11422324.
- [19] Gosavi TD, See SJ, Lim SH. Ictal and interictal EEG patterns in patients with nonconvulsive and subtle convulsive status epilepticus. *Epilepsy Behav* 2015 Aug; 49:263–7. <https://doi.org/10.1016/j.yebeh.2015.05.011>. Epub 2015 Jun 14 PMID: 26081674.
- [20] Sutter R, Semmlack S, Kaplan PW. Nonconvulsive status epilepticus in adults – insights into the invisible. *Nat Rev* 2016 May;12(5):281–93. <https://doi.org/10.1038/nrneuro.2016.45>. Epub 2016 Apr 11. PMID: 27063108.
- [21] Botarro FJ, Martinez OA, Pardo MMF, Bruetman JE, Reisin RC. Nonconvulsive status epilepticus in the elderly: a case-control study. *Epilepsia* 2007;48:966–72. <https://doi.org/10.1111/j.1528-1167.2007.01033.x>.
- [22] Canas N, Delgado H, Silva V, Pinto AR, Sousa S, Simões R, et al. The electroclinical spectrum, etiologies, treatment and outcome of nonconvulsive status epilepticus in the elderly. *Epilepsy Behav* 2018 Feb;79:53–7. <https://doi.org/10.1016/j.yebeh.2017.10.034>. Epub 2017 Dec 15 PMID: 29253676.
- [23] Labar D, Barrera J, Salomon G, Harden C. Nonconvulsive status epilepticus in elderly: a case series and a review of the literature. *J Epilepsy* 1998;11:74–8.
- [24] Litt B, Wityk RJ, Hertz SH, Mullen PD, Weiss H, Ryan DD, Henry TR. Nonconvulsive status epilepticus in critically ill elderly. *Epilepsia* 1998 Nov;39(11):1194–202. <https://doi.org/10.1111/j.1528-1157.1998.tb01311.x>. PMID: 9821984.
- [25] Sheneker BF, Fountain NB. Assessment of acute morbidity and mortality in nonconvulsive status epilepticus. *Neurology* 2003 Oct 28;61(8):1066–73. <https://doi.org/10.1212/01.wnl.0000082653.40257.0b>. PMID: 14581666.
- [26] Kang BS, Jhang Y, Kim Y, Moon J, Shin J, Moon HJ, et al. Etiology, and prognosis of non-convulsive status epilepticus. *J Clin Neurosci* 2014 Nov;21(11):1915–9. <https://doi.org/10.1016/j.jocn.2014.03.018>. Epub 2014 Jul 3 PMID: 24998856.
- [27] Al-Said YA, Baeesa SS, Shivji Z, Kayyali H, Alqadi K, Kadi G, Cupler EJ, Abuzinadah AR. Non-convulsive seizures and electroencephalography findings as predictors of clinical outcomes at a tertiary intensive care unit in Saudi Arabia. *Clin Neurol Neurosurg* 2018 Aug;171:95–9. <https://doi.org/10.1016/j.clineuro.2018.06.002>. Epub 2018 Jun 5 PMID: 29890460.
- [28] Vespa PM, Nuwer MR, Nenov V, Ronne-Engstrom E, Hovda DA, Bergsneider M, et al. Increased incidence and impact of nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous electroencephalographic monitoring. *J Neurosurg* 1999 Nov;91(5):750–60. <https://doi.org/10.3171/jns.1999.91.5.0750>. PMID: 10541231; PMID: PMC4347935.
- [29] Nei M, Lee JM, Shanker VL, Sperling MR. The EEG and prognosis in status epilepticus 1999 Feb;40(2):157–63. <https://doi.org/10.1111/j.1528-1157.1999.tb02069.x>. PMID: 9952261.
- [30] Yuan F, Yang F, Li W, Yang X, Gao Q, Bi L, et al. Nonconvulsive status epilepticus after convulsive status epilepticus: Clinical features, outcomes, and prognostic factors. *Epilepsy Res* 2018 May;142:53–7. <https://doi.org/10.1016/j.eplepsyres.2018.03.012>. Epub 2018 Mar 12 PMID: 29555354.