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FULL-LENGTH ORIGINAL RESEARCH

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Estimating the period prevalence of non-convulsive status epilepticus among comatose adults at the University Teaching Hospital in Lusaka, Zambia

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Abstract

Objective: In Western settings, non-convulsive status epilepticus (NCSE) and non-convulsive seizures (NCSz) are associated with high mortality. In comatose patients, interictal epileptiform discharges (IEDs) identified on routine electroencephalogram (EEG) are predictive of NCSE/NCS. Little is known regarding the prevalence, causes, or outcomes of NCSE/NCSz in sub-Saharan Africa (SSA). We sought to investigate the prevalence of IEDs and NCSE/NCSz at a single teaching institution in SSA.

Methods: From October 3, 2017, to May 21, 2018, adult inpatients on the internal medicine service at Zambia's University Teaching Hospital (UTH) with a Glasgow Coma Score (GCS) of ≤10 were identified, excluding patients with mechanical ventilation or open head wounds. Signed consent by a proxy was required for enrollment and 30-minute EEG. Chart abstractions provided coma duration, presence/absence of clinical seizures during/prior to admission, history of epilepsy, and presumed coma etiology. A structured neurological examination was completed. Patients were followed to discharge or death. Risk factors for IEDs were evaluated.

Results: Of 392 eligible patients, 250 had EEGs. EEGs were not completed on eligible patients due to death (74), improved GCS (37), transfer within UTH (25), or lack of proxy (6). NCSE occurred in 22 of 250 (8.8%), NCSz in 3 of 250 (1.2%), and IEDs in 46 of 250 (18.4%) patients. Of the 250, 197 (78.8%) died. No specific risk factors for IEDs were identified.

Significance: If the association between IEDs and NCSE among monitored populations in developed settings holds true for SSA, a projected 17%-21% of comatose African adults have NCSE. No clinical characteristics identified those at risk.

KEYWORDS
electroencephalograph, epilepsy, Global health, non-convulsive status epilepticus, Sub-Saharan Africa

1 | INTRODUCTION

In resource-limited tropical settings, common causes of coma include head injury, cerebral malaria, and central nervous system infections including HIV-associated opportunistic infections and strokes, tumors, and metabolic disturbances.[1] Seizures and status epilepticus are known secondary complications of the acute brain insults and chronic epilepsy, and may present as coma. Untreated status epilepticus is associated with high morbidity and mortality, especially in cases caused by an acute brain insults.[2] Non-convulsive status epilepticus (NCSE) is a condition in which electrographic seizures are not accompanied by motor activity and requires a high index of suspicion for diagnosis.[3] Where available, an electroencephalograph (EEG) provides identification of non-convulsive seizures (NCSz), identification of NCSE, and assessment of electrographic background to direct treatment, and provides insights into prognosis. NCSE is present in around eight percent of comatose patients in high-income settings with no immediate history of seizure, and among the US patients admitted to Neurointensive Care Units (NeuroICU), 10%-43% have NCSE.[4–6]. In Qatar, a study of an Emergency Department reported an NCSE prevalence of 26% among adults with altered mental status. A Belgian Emergency Department found NCSE in 28% of elder adults with delirium. A single ICU in Japan found an NCSE prevalence of 30% among adults with altered mental status.[7–9] No epidemiological studies have been published regarding NCSE/NCSz in comatose or obtunded non-intubated adults in sub-Saharan Africa (SSA). Given that the etiologies of coma in SSA differ significantly from those in high-income, non-tropical areas, data from more developed countries may not be applicable in this setting. Furthermore, the recognition of NCSE/NCSz and the extent of medical care provided to the patients with this condition in resource-limited countries may be limited due to the lack of awareness among healthcare workers about the possibility of NCSE/NCSz as well as the absence of EEG capacity for making the diagnosis. Thus, the prevalence, presumed etiology, and outcomes of NCSE/NCSz in SSA are unknown.

Reported clinical risk factors for NCSE in comatose adults include acute brain insults, an immediate history of seizure prior to coma onset, and preexisting epilepsy.[5,10–12] Of the 50 million people worldwide that have epilepsy, 80% live in developing countries with the majority untreated.[13] Additionally, the rate of acute symptomatic seizures and NCSE/NCSz following an insult to the brain has been shown even in high-income countries to vary significantly based on the nature of the insult, with seizure rates over fifty percent for hypoxic-ischemic injury vs less than twenty percent for aneurismal subarachnoid hemorrhage.[12] With suboptimal epilepsy treatment and different etiology profiles of cerebral injuries, the prevalence of NCSE in SSA may be higher compared to developed countries.[14]

Key Points

- In our study, 71 of 250 (28.4%) of patients had epileptiform activity on 30-minute EEG including 22 cases of NCSE, three cases of NCSz, and 46 cases of IEDs
- Lack of clinical predictive factors for epileptiform activity makes it difficult to direct inpatient EEG capacity toward those at risk
- Mortality among enrolled comatose patients was very high and was not associated with epileptiform activity

NCSE is associated with lifelong disability and high mortality and can be adequately treated with medications that are effective, affordable, and readily available, such as phenobarbital.[15] The gold-standard test for identifying NCSE in high-income tertiary care settings is continuous video EEG monitoring (cEEG) recorded for at least 24 and longer if patient is comatose.[16] Interictal epileptiform discharges (IEDs) identified on the EEGs have been found to herald seizures.[17–20] The Neurocritical Care Society recommends that any comatose patient found to have IEDs on a routine EEG should have cEEG because the risk of NCSz is 40%-60%.[17–19] In Zambia, cEEG is not available. To estimate the prevalence of NCSE/NCSz among comatose adults, we assessed the presence of NCSE, NCSz, and IEDs on routine 30-minute EEG and estimated NCSE/NCSz prevalence based upon observation of NCSE/NCSz on the EEG and estimates of NCSE/NCSz incidence given the presence of IEDs. We also evaluated risk factors for epileptiform activity and tracked patient outcomes.

2 | MATERIALS AND METHODS

To estimate the prevalence of NCSE in comatose adults, we screened every adult patient admitted to the internal medicine (IM) service at Zambia's University Teaching Hospital (UTH) from October 3, 2017, to May 21, 2018, for a Glasgow Coma Score (GCS) of ≤10 and sought to complete routine EEGs on these patients. The inclusion criteria for the study were age ≥18 years, GCS ≤ 10, and patients currently admitted to the IM service at UTH. Patients with open scalp wounds precluding EEG lead placement were excluded. Additionally, patients in the ICU and on ventilators were not enrolled due to electrical interference of the EEG acquisition station with the ventilator. These patients were subsequently eligible to enroll if

transferred out of the ICU. Proxy consent from relatives of qualifying patients was required for participation in the study. The GCS score of patients on the IM service was assessed by a medical student who had completed three years of medical school (CB), and identified qualifying patients' names were passed along to the interviewer (IT) who obtained consent. Patients with subtle signs of seizure (finger twitching, blinking, nystagmus, etc) were prioritized first, followed by patients with the lowest GCS scores. Enrolled patients underwent a 30-minute bedside 21-lead EEG using either a Natus Xltek Trex or Natus EEG32U device and utilizing the International 10-20 system of electrode placement. Gold-plated electrodes were used. Demographic and clinical characteristics were extracted from charts and/or from interviewing the patient's proxy. Presumed etiology was that assigned prior to the EEG completion by the internal medicine clinical team caring for the patient. If they listed more than one potential etiology, the first etiology on their differential diagnoses was used. Structured neurological examination findings were captured (CB). EEGs were considered part of patient care and were interpreted by one of the neurologists with subspecialty training in epilepsy (OKS, GLB) who provided test interpretation to internal medicine team taking care of the patient. For the purpose of the research study, the EEG reader further categorized each EEG record according to the Modified Synek Coma Classification Scale, a previously validated scale providing prognostic value in patients with traumatic brain injury or anoxic coma.[21,22] Seizure was defined as per published criteria, and status epilepticus was defined as if there were 3 or more seizures over the period of 30-minute recording or if continuous $\geq$1 Hz spike-wave patterns lasted for >50% of the EEG record.[23] Any interventions made by the medical teams in response to the EEG findings were also recorded. Patients were followed to discharge or death in the hospital. The patients' functional status at discharge was captured using the Karnofsky scale, subdivided into >80 if patient was able to perform all activities of daily living, <80 if they needed assistance, and zero if the patient died in-hospital.[24]

To assess for risk factors for IEDs, odds ratios with 95% confidence intervals and *P*-values were calculated using a chi-square test for the categorical values, while mean differences with 95% confidence intervals and *P*-values were calculated using the Student's *t* test for continuous variables. A $P < .05$ was considered significant. The association between IEDs and outcome (death, functional status) as well as the Synek score's value in predicting mortality was also assessed.

3 | RESULTS

Among 392 eligible patients, informed consent was obtained in 273 (69.6%). Of the remainder that was not in the ICU, lack of participation was due to death (56), improvement of mental status to GCS >10 (21), transfer off of the IM service (20), or lack of proxy (6). There were 17 patients admitted to ICU that were followed but were not approached for consent due to inability to conduct the EEG in the ICU setting secondary to electrical interference. One ICU patient was transferred out of the ICU and was subsequently enrolled, while six died, five were discharged home, and five were transferred out of the IM service. Of consenting patients, EEG was conducted on 250 (91.6%) patients. Twenty-three consenting patients did not have EEG completed either because of death (12) or coma recovery to GCS > 10 (11) prior to start of the test. Figure 1 shows the patient enrollment flowchart. The total screening events reflect daily screening procedures and include duplicate screening events of the same excluded patients. Table 1 depicts baseline demographic characteristics and their association with epileptiform discharges (EPD) (which includes NCSE, NCE, and IEDs) as well as with NCSE specifically. No significant demographic or clinical risk factors for having EPD or NCSE were identified, including cause of coma. Table 2 shows the Synek score definitions, and Table 3 shows the distribution of the Synek scores. Seventy-one patients (28.4%) were found to have epileptiform activity: 22 were in NCSE, three were noted to have NCSz, and 46 had either generalized or focal IEDs without ictal patterns. Fifty-one (71.8%) of these received additional treatment with anti-epileptic medications based upon EEG findings. Table 4 shows the outcomes of patients with and without various epileptiform activities. The presence of epileptiform activity was not associated with worse functional status at discharge or mortality in the overall study population (OR 1.62, 95% CI [0.71-3.75], $P = .25$), nor was IEDs, NCS, or NCSE specifically. This was also true for patients with preexisting epilepsy (OR 1.64, 95% CI [0.68-3.97], $P = .27$).

4 | DISCUSSION

We estimated the prevalence of NCSE based on routine EEG. In our study population, 71 of 250 (28.4%) had epileptiform activity on a 30-minute EEG recording including 22 cases of NCSE, three cases of NCSz, and 46 cases of IEDs. Assuming that 40%-60% of patients with IEDs would go on to have NCSz on cEEG, our estimated minimum prevalence of NCSE/NCSz among comatose/obtunded adults is 11%-13% with the estimated study prevalence being 17%-21%.[17–19] The actual rates of NCSE/NCSz in this Zambia hospital are likely to be higher since ICU patients were excluded from this study and because not all eligible patients were able to undergo EEG evaluation. This estimate also assumes that none of the 179 patients who had EEGs without evidence of epileptiform activity went on to develop NCSE/NCSz at any point in their hospitalization.

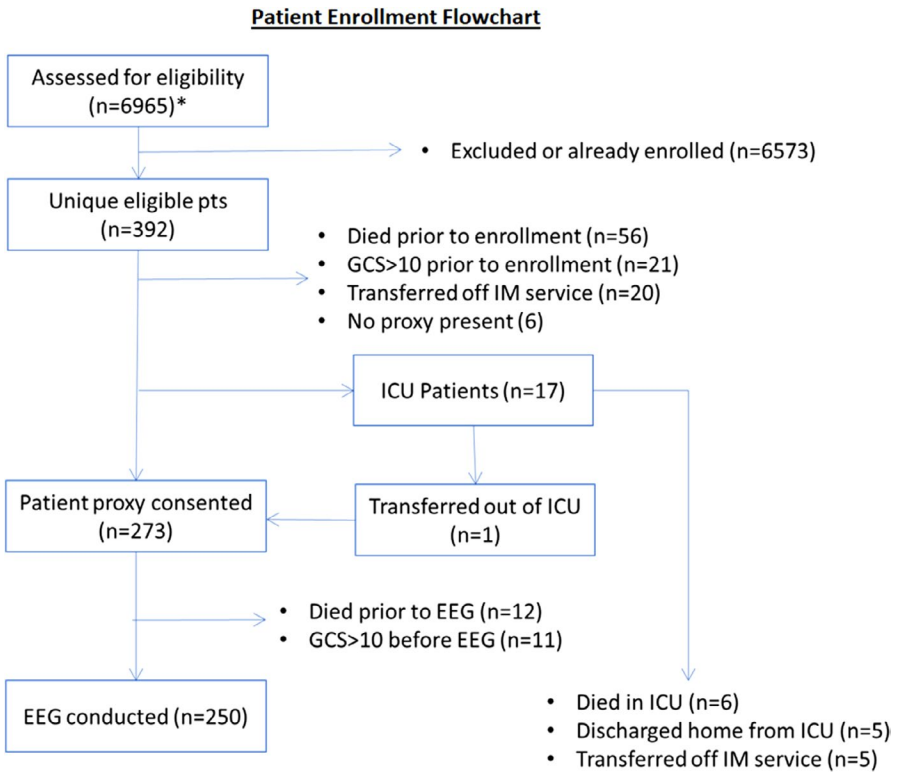

FIGURE 1 Patient enrollment flowchart. *The 6965 assessment events include duplicate assessments of the same patients, as many patients' GCS scores changed with time. Abbreviations: EEG, electroencephalogram; GCS, Glasgow Coma Scale; ICU, intensive care unit; IM, internal medicine

The 2HELPS2B study has shown predictive value of specific cEEG features in accurately stratifying 72-hour NCSz risk (though the studies used in formulating the scoring system were all greater than six hours long) and has shown that depending on the features of the IEDs and background, the risk of NCSz could be anywhere from 12% to >95%.[20] Thus, more detailed characterization of the EEG information could provide more insight into the risk of NCSz and this is being pursued by our research team.

Mortality among enrolled comatose patients was high and was not associated with epileptiform activity. The high mortality rate is likely due to patients' underlying primary etiology for an admission as well as restricted ability to provide respiratory support. Additionally, the average length of time between the coma onset and EEG acquisition was four days due to delayed presentation and limited EEG availability. This delay limits our ability to compare our results to similar studies done in Western settings, and may limit the sensitivity of EEG—however, this wide distribution of presentation times and delays in diagnostic assessments is quite typical of care in the region. EEG findings were provided in real time to clinicians caring for patients resulting in most patients with epileptiform activity receiving anti-seizure medications for acute treatment or seizure prevention. Contrary to prior publications, none of the evaluated demographic or clinical factors in our cohort of patients were predictive of epileptiform activity, including a history of epilepsy or reports of clinical seizures prior to or during the admission. This may be explained by the limited treatment of epilepsy prior to the admission, or the possibility of co-existing acute and chronic neurological problems with high co-existing HIV infection coupled with AIDS-defining illness. The lack of clinical predictive factors for the presence of epileptiform activity makes it difficult to direct inpatient EEG capacity toward those at risk. Future studies evaluating the impact on clinical outcomes made by the availability of EEG would be valuable for determining whether developing EEG capacity should be a high priority in resource-limited hospitals.

The EEG findings in this study did impact care. Of the 71 patients with epileptiform activity, 51 (71.8%) received treatment with carbamazepine, diazepam, and/or phenobarbital and/or an ICU transfer. While the rationale of medical decision-making was not formally assessed, the most common reason cited by providers for not treating patients who had epileptiform activity was the concern for respiratory instability as available agents for treatment are often limited to benzodiazepines and barbiturates and there is a lack of ventilatory rescue support should a patient develop respiratory insufficiency as a result of treatment. Of note, two patients were found to have normal EEG despite clinical appearance of coma (Table 3), further supporting diagnostic utility of the test in identification of presumed conversion disorder and preventing unnecessary escalation of the medical treatment.

As mentioned in Results, 273 of the 392 eligible patients were consented for study participation (69.6%). This low rate

TABLE 1 Patient demographics and OR/t test difference of having EPD or NCSE, n = 250

Characteristic	Study Population	OR/ Difference in *T*-Test Mean of having EPD	95% Confidence Interval	*P*-value	OR/ Difference in T-Test Mean of having NCSE	95% Confidence Interval	*P*-value
Female gender, # (%)	124/250 (49.6)	1.06	0.61-1.84	.82	0.83	0.35-2.01	.68
Age, median years (IQR)	44 (33-63)	4.79	−0.29-9.87	.65	5.31	−2.81-13.43	.20
HIV positive, # (%)	112/242 (46.3)	0.72	0.41-1.27	.26	0.96	0.40-2.32	.94
GCS, median (IQR)	7 (5-9)	−0.44	−1.09-0.21	.18	−0.97	−2.00-0.06	.06
Coma duration, median days (IQR)	4 (2-7)	−1.54	−3.24-0.16	.08	−1.91	−4.63-0.80	.17
Past medical history of epilepsy, # (%)	14/247 (5.7)	1.44	0.46-4.44	.63	0.78	0.10-6.23	.81
Suspected coma etiology of infection, # (%)	109/250 (43.6)	0.66	0.38-1.18	.16	0.72	0.29-1.78	.48
Suspected coma etiology of stroke, # (%)	67/250 (26.8)	1.21	0.66-2.24	.53	1.64	0.65-4.10	.29
Reported seizure activity prior to/since admission, # (%)	96/250 (38.4)	1.15	0.66-2.02	.62	1.68	0.70-4.05	.25
Presence of subtle signs of seizure, # (%)	68/250 (27.2)	1.43	0.78-2.60	.25	1.98	0.81-4.88	.14
Presence of temperature > 38°C, # (%)	39/250 (15.6)	0.72	0.32-1.61	.42	0.52	0.12 - 2.30	.39
Presence of SBP > 180 mm Hg or DBP > 120 mm Hg, # (%)	10/250 (4.0)	0.27	0.03-2.17	.22	0.46	0.03-8.16	.60
Presence of heart rate > 100 bpm, # (%)	126/250 (50.4)	0.63	0.36-1.10	.11	0.66	0.27-1.60	.35
Pre-admit Karnofsky score, median (IQR)	100 (70 - 100)	−4.72	−11.22-1.78	.15	0.70	−9.20-10.59	.89
Discharge Karnofsky score, median (IQR)	0 (0 - 0)	−0.56	−7.62-6.50	.88	−0.32	−11.64-11.01	.96
Presence of EPD (Synek of Va, Vb, or both), # (%)	71/250 (28.4)	N/A	N/A	N/A	N/A	N/A	N/A

Abbreviations: EPD, epileptiform discharges; GCS, Glasgow Coma Scale; IQR, interquartile range; NCSE, non-convulsive status epilepticus; OR, odds ratio.

of patient capture is a limitation to the study. However, this is reflective of the reality of clinical medicine in low-resource settings with logistic constraints and very limited capacity for respiratory support and EEG.

This study had several other limitations. Routine 30-minute EEG was exclusively used because the resources for cEEG were not available. There were also delays between identification and enrollment of patients due to the logistical limitations

TABLE 2 Modified synek coma classification scale

Category	Subcategory
I. Delta/theta > 50% of record (not theta coma)	A. Reactivity B. No reactivity
II. Triphasic waves	
III. Burst-suppression	A. With epileptiform activity B. Without epileptiform activity
IV. Alpha/theta/spindle coma (not in burst-suppression)	
V. Epileptiform activity	A. Generalized B. Focal or multifocal
VI. Suppression	A. <20 μV but > 10 μV B. ≤ 10 μV

TABLE 3 Patient synek scores

Synek score	Number of subjects (n = 250)
Ia	139
Ib	31
II	3
IIIa	1
IIIb	1
IV	0
Va	20
Vb	51
Via	0
VIb	1
Normal EEG	2
Unable to assess	1

of having one EEG machine and a solo technologist, reducing the recruitment rate. Given limited access to advanced diagnostic assessments including neuroimaging and CSF analyses, definitive diagnoses were not available in the vast majority of patients. A period prevalence study conducted in this same facility has previously established that the most common inpatient neurological diagnoses in this clinical setting are CNS infections (especially among HIV infected individuals), strokes, and seizures and the etiologic assignments in this EEG study were congruent with this past period prevalence data.[25] While this is a limitation of this work, inpatient studies in Nigeria and Tanzania show very similar causes for neurological admissions as is seen in Zambia and suggest that within the diagnostic capacities available in these African settings, the findings here have some generalizability.[26,27] Another limitation of this study is that patients with GCS scores of 3-10 were enrolled, while many Western studies focusing on comatose patients use a GCS cutoff of 8. Because this study occurred in a resource-limited setting where most comatose patients were not ventilated (a situation associated with high mortality), the higher GCS ceiling was selected to ensure that an adequate number of patients could be enrolled. Additionally, patients in the ICU were excluded from our study due to severe electrical interference. This is another factor that makes it difficult to directly compare our study population to that in Western studies that enroll mainly ventilated patients.

NCSz and NCSE can be a reversible cause of coma and is often more difficult to diagnose than to treat, remaining largely under-recognized due to limited resources in SSA. The prevalence of epileptiform activity including NCSE in this patient population is high. Further investment in EEG resources is needed to improve the diagnosis of NCSE/NCSz in comatose patients in resource-limited settings similar to UTH. However, future studies evaluating the utility of EEG in changing patient outcomes in this setting are needed since

TABLE 4 Patient outcomes[a]

Outcome	Patients without EPD (n = 179)	Patients with EPD (n = 71)	*P*-value	Patients with NCSE (n = 22)	*P*-value	Patients with NCSz (n = 3)	*P*-value	Patients with IEDs (n = 46)	*P*-value
Discharge Karnofsky ≥ 80, number (%)	7 (4)	4 (6)	.555	2 (9)	.267	0 (0)	.728	2 (4)	.902
Discharge Karnofsky < 80, number (%)	25 (14)	4 (6)	.062	0 (0)	.061	1 (33)	.344	3 (7)	.171
Discharge Karnofsky, median (IQR)	0 (0-0)	0 (0-0)	.56	0 (0-0)	.56	0 (0-25)	-	0 (0-0)	.56
Death, number (%)	140 (78)	57 (80)	.715	18 (82)	.164	2 (67)	.630	37 (80)	.746
Transferred or lost to follow-up, number (%)	7 (4)	6 (8)	.141	2 (9)	.347	0 (0)	.728	4 (9)	.179

Abbreviations: EPD, epileptiform discharge; IEDs, interictal epileptiform discharges; IQR, interquartile range; NCSE, non-convulsive status epilepticus; NCSz, non-convulsive seizures.

[a]Karnofsky group median analyses are limited to survivors.

this unblinded study showed similarly poor outcomes for all comatose adults regardless of EEG findings. More detailed evaluation of patient outcomes for neuropsychiatric sequelae would be of value in this setting.

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CONFLICTS OF INTEREST

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