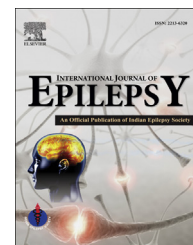


Available online at www.sciencedirect.com

ScienceDirect

journal homepage: <http://www.journals.elsevier.com/international-journal-of-epilepsy>

Original Article

Status epilepticus in adults: A study from Nigeria

Lukman Femi Owolabi ^{a,*}, Aliyu Ibrahim ^a, Alhassan Datti Mohammed ^b,
Shakirah D. Owolabi ^c^a Department of Medicine, Aminu Kano Teaching Hospital, Bayero University, Kano PMB 3452, Nigeria^b Department of Anesthesiology and Intensive Care, Aminu Kano Teaching Hospital, Bayero University, Kano PMB 3452, Nigeria^c Department of Psychiatry, Aminu Kano Teaching Hospital, Bayero University, Kano PMB 3452, Nigeria

ARTICLE INFO

Article history:

Received 24 May 2014

Accepted 4 November 2014

Available online 26 December 2014

Keywords:

Etiology

Predictor

Outcome

Status epilepticus

Nigeria

ABSTRACT

Background: Status epilepticus (SE) is a common neurologic emergency. Immediate treatment to stop seizure activity and prompt diagnostic evaluation to recognize potentially treatable causes are paramount in the management of SE. Thus, increased awareness of presentation, etiologies, and treatment of status epilepticus SE is central in the practice of critical care medicine. However, there is a paucity of information on SE from Nigeria.

Objective: We evaluated the clinical profile and predictors of one-month outcome in a group of Nigerian patients with SE.

Methodology: Patients with SE were recruited from the medical, high dependency unit, intensive care unit and accident and emergency departments of a tertiary hospital from 2008 to 2013. The outcome was assessed using Glasgow Outcome Score (GOS). The outcome, which was categorized into dead (GOS = 1) or alive was analyzed in a multivariate logistic regression model.

Result: A total of 76 patients was studied. The four most common underlying etiologies were stroke, antiepileptic drug (AED) non-compliance, CNS infections and metabolic derangement. Fifty-nine (77.6%) patients survived. Duration of seizure, delay in initiation of treatment (Odd ratio (OR) = 4.4, 95% CI = 1.17–16.56), refractory status epilepticus (OR = 87.1, 95% CI = 12.94–781.1) were significantly associated with death. On multivariate analysis, however, refractory status epilepticus remained an independent predictor of death.

Conclusion: Our study showed that the most common underlying etiologies in SE were stroke, antiepileptic drug non-compliance CNS infections and metabolic derangement. Duration of seizure, delay in treatment and refractory the SE were significantly associated with death, but refractory seizure was an independent predictor of death in SE.

Copyright © 2014, Indian Epilepsy Society. Published by Reed Elsevier India Pvt. Ltd. All rights reserved.

* Corresponding author.

E-mail address: drlukmanowolabi@yahoo.com (L.F. Owolabi).<http://dx.doi.org/10.1016/j.ijep.2014.11.001>

2213-6320/Copyright © 2014, Indian Epilepsy Society. Published by Reed Elsevier India Pvt. Ltd. All rights reserved.

1. Introduction

Status epilepticus (SE) is a potentially life-threatening medical and neurological emergency. It can occur from a variety of insults to the central nervous system.¹ In the United States of America, status epilepticus is a major neurologic emergency that affects 50,000–60,000 people annually² and in Nigeria it is a common cause of neurologic admission.^{3,4}

Generally, certain etiologies of status epilepticus have been found to be associated with a worse outcome than others.^{5,6} Some studies have suggested that the etiology of SE may affect the response of a patient to antiepileptic drugs⁷ while others reported that status epilepticus of longer duration carries a less favorable outcome.⁵

In addition, the age of the patient, female gender and time elapsed from onset to treatment have been reported as prognostic factors.^{8,9} Information about clinical characteristics and etiology as well as drawing the attention of the clinicians to whether early symptoms and signs correlate with outcome of SE is desirable. Such information is essential if clinicians are to identify those SE patients that are more likely to recover with proper therapy.

However, to the best of our knowledge, such clinical study highlighting the etiology and predictors of outcome of status epilepticus in adult Nigerians has not been conducted.

Against this background, we investigated the demographic data, clinical features and etiology as well as independent predictors of death among patients who were admitted with SE in a tertiary hospital in North western Nigeria.

2. Methods

In this prospective observational study, adults with a diagnosis of SE, admitted to the emergency unit, medical wards and intensive care unit of a tertiary hospital in North western Nigeria were recruited. The study was conducted over a period of 5 years (2008–2013).

SE was defined as continuous seizure activity lasting 30 min or more or recurrent seizure activity lasting 30 min or more without a full return of consciousness. Electroencephalography (EEG) was not a prerequisite for diagnosis.

Seizure types were categorized according to the International Classification of Epileptic Seizures, based on information from eyewitnesses, nursing and physician staff.¹⁰ The seizure episodes were classified as overt generalized convulsive, non-convulsive, partial motor SE.

Onset of SE was based upon observations of eyewitnesses or physician and nurses' observations for in-hospital status epilepticus. Patients who did not meet the definition of SE were excluded.

The etiology was defined as the presence of a remote or acute symptomatic injury that was assumed to have caused the episode. In line with previous studies, etiologic causes were categorized as drug non-compliance, cerebrovascular disease, central nervous system infection, toxic-metabolic disorders, trauma, tumors and idiopathic.^{2,11} The etiology was considered idiopathic if there was no clinical, laboratory or radiological evidence sufficient to support a specific cause.

All the patients were treated in accordance with the hospital management guidelines on SE¹² which was based on published recommendations^{13,14} with intravenous diazepam followed by phenytoin loading of 15–20 mg/kg as first line anti-convulsive therapy and phenobarbitone as the second line AEDs. No response to first and second line AEDs was considered refractory SE.

In this study, the clinical end point was defined as return of consciousness in the case of non-convulsive status epilepticus (NCSE) and return of consciousness as well as the cessation of convulsions in the case of convulsive status epilepticus (CSE).

Response to antiepileptic drugs were defined as the cessation of status epilepticus for at least 12 h after completion of drug administration. Delay in treatment was defined here as initiation of AED 30 min after the onset of SE as reported by eyewitnesses or medical personnel.¹⁵ Patient outcomes, recorded in all patients at the time of discharge, were defined in accordance with GOS.¹⁶ Outcome was categorized into dead (GOS = 1) or alive.¹⁵ All patients, including those patients with idiopathic cause, were discharged on maintenance AED. The patients were followed-up on a daily basis.

Analysis of data was carried out using the "Statistical Package for Social Sciences" (SPSS) program for Windows version 16.0 (SPSS Inc., Chicago, IL). The numerical variables (age and duration of seizure) did not pass normality tests (Shapiro–Wilk and Kolmogorov Smirnov), and hence, were described using range and median. Categorical variables were compared using Chi-square or Fisher exact test. Because the numerical data (duration of seizures) were not normally distributed, the Man Whitney *U* test was used to compare their median in the survivor and dead patients while Kruskal–Wallis test was used in the case of comparison of greater than two groups. Post hoc analysis was conducted using Dunn's test. The predictive factors for poor outcome were determined using the multivariate logistic regression model adjusting for age, gender, pre-existing epilepsy, seizure type, delay in treatment and etiology. $P < 0.05$ was considered a statistically significant level.

3. Result

A total of 76 patients was recruited for the study. Their age ranged between 16 and 85 with a median age of 50 years. They comprised 45 (59.2%) males and 31 (40.8%) females. Eighteen (23.7%) patients had pre-existing epilepsy. Delay in initiation of treatment was present in 18 (23.7%) patients. Status epilepticus was refractory in 17 (22.4%). The most common etiology was stroke (28.9%), followed by AED noncompliance, CNS infections, metabolic causes, CNS tumor and idiopathic accounting for 21.1%, 19.8%, 15.7%, 9.2 and 5.3% respectively (Table 1). Seizure types recorded in the patients were generalized tonic-clonic in 54 (71.1%) patients, simple partial in 13 (17.1%), non-convulsive (complex partial seizure) in 6 (7.9%) and secondarily generalized 3 (3.9%).

The median duration of SE was 3.7 h (range; 1–72 h). All the patients had intravenous diazepam, 67% of the patients had phenytoin, and 56% of the patients had other AEDs.

Eight (10.5%) patients were admitted into the Intensive Care Unit (ICU). Fifty-nine (77.6%) patients survived. When the

Table 1 – Etiology of status epilepticus (SE).

Etiology	Frequency	Percentage (%)
Stroke	22	28.9
Metabolic	12	15.8
Hypoglycemia	5	6.6
Hyperglycemia	3	3.9
Uremia	2	2.6
Hyponatremia	2	2.8
CNS Infection	15	19.7
Pyogenic meningitis	8	10.5
Tuberculous meningitis	3	3.9
Encephalitis	4	5.3
Idiopathic	4	5.3
AED noncompliance	16	21.1
CNS tumor	7	9.2
Total	76	100

median duration of seizure was compared across GOS score, overall, the difference was statistically significant ($P < 0.0001$), however, post hoc test (Dunn's test) showed that the patients with GOS 1, 3, 4 against GOS 5 accounted for the difference (Fig. 1).

On bivariate analysis, age of the patients, gender, pre-existing epilepsy, seizure type and etiology of the seizure were found not to be associated with death while duration of seizure, delay in treatment, refractory status epilepticus were significantly associated with death (Table 2). On multivariate analysis, only refractory status epilepticus remained an independent predictor of death (Table 3).

4. Discussion

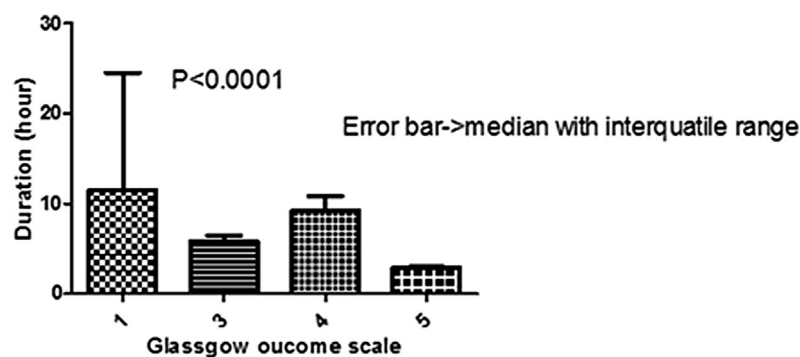
Status epilepticus is a neurologic and medical emergency. Considering the significant risk of mortality, particularly in

the developing world, and the possibility of successful therapeutic intervention, prompt identification and treatment of patients in SE by the physician, is of the utmost importance. This study, to the best of our knowledge, is the first prospective clinical study emanating from Nigeria.

In our study, delay in receiving treatment for SE was seen in 23.7% of the patients. In developing countries, poor health infrastructure, poor road connectivity and delays in transportation, lower socioeconomic and educational status of the local population and poor access to the hospital and specialist care are some of the factors militating against prompt initiation of treatment for SE. Similar finding was reported from other places in developing countries.^{15,17} Mhodj et al, in a similar study in Senegal, reported mean latency of 16.6 h with only 4.6% of the patients arriving at the hospital within 6 h of onset of SE.¹⁷

In conformity to reports from elsewhere,^{15,18–20} generalized convulsive SE (GCSE) was the most commonly identified form of SE in our study. It is worthy of note that this description referred to GCSE in which overt motor movement was seen. Nonetheless, subtle GCSE in which minimal motor movement is restricted to continuous, rhythmic motor movements or twitches that can be seen to involve the eyes, eyelids, face, jaw has been described.^{20,21} Because patients with NCSE can exhibit a wide variety of clinical features ranging from coma, confusion, speech disturbance, aphasia, autonomic manifestation, delusions, to behavioral manifestations, including hallucinations, and paranoia.²² a high index of suspicion is often required and the distinction often rests on electroencephalography (EEG) finding.²³ Though the absence of EEG for diagnosis in this study raised questions about the possibility of missing some NCSE, however, all the patients with NCSE in the study were those with pre-existing complex partial seizure.

The etiological spectrum of SE in the current study was in agreement with what was reported from other developing



Dunn's Multiple Comparison Test	Difference in rank sum	$P < 0.05?$
1 vs 3	8.278	No
1 vs 4	-2.772	No
1 vs 5	32.39	Yes
3 vs 4	-11.05	No
3 vs 5	24.11	Yes
4 vs 5	35.16	Yes

Fig. 1 – Relationship between duration of seizure and GOS in SE.

Table 2 – Relationship between demographic and clinical variables of SE patients.

Variables	Proportion of patients who died	Unadjusted OR (CI)	Unadjusted P value
Age	17/76	-*	0.770
Duration of seizure episode	17/76	-*	<0.001**
Sex			
Male	9/45	0.7 (0.21–2.43)	0.551
Female	8/31		
Convulsive			
Yes	16/70	1.5 (0.15–36)	0.999
No	1/6		
Generalized/Partial			
Generalized	13/54	1.4 (0.36–6.05)	0.764
Partial	4/22		
Pre-existing epilepsy			
Yes	1/18	0.2 (0.01–1.27)	0.058
No	16/58		
Refractory SE			
Yes	14/17	87.1 (12.94–781.1)	<0.001**
No	3/59		
Delay in treatment			
Yes	8/10	4.4 (1.17–16.56)	0.020**
No	9/58		
Etiology Stroke			
Yes	5/22	1.03 (0.27–3.84)	0.999
No	12/54		
Metabolic			
Yes	2/12	0.65 (0.09–3.78)	0.995
No	15/64		
CNS infection			
Yes	5/15	2.04 (0.49–8.28)	0.304
No	12/61		
AED noncompliance			
Yes	1/16	0.18 (0.01–1.53)	0.101
No	16/60		
Tumor			
Yes	3/7	2.95 (0.45–18.42)	0.182
No	14/69		
Idiopathic			
Yes	1/4	1.17 (0.20–6.48)	0.997
No	16/72		

* No OR because unpaired t-test was conducted.
** Statistically significant.

countries.^{11,15,24} Acute or remote stroke was the most common etiology of SE in our study. This finding is compatible with reports from elsewhere.^{11–17,24,25} The finding, however, differ from a report of a neuropathologic study from Ibadan, Southwestern Nigeria. In the Ibadan study 41 cases of SE were recruited over a 10-year period in a tertiary hospital. In that study the most common etiology was an infection of the central nervous system (17 cases).⁴ In that study, however, the

Table 3 – Independent predictor of death from status epilepticus.

Variable	Odds ratio	95% CI	P value
Duration of SE	0.8	0.650–1.021	0.074
Delay in treatment	1.4	0.170–10.818	0.775
Refractory SE	24.2	3.590–163.560	0.001

most frequent cause in subjects above 12 years of age was stroke. Given that our data sets included only adult population, the most common etiology found in the Ibadan study is indirectly in conformity with the finding in our study. In children, up to 51% of SE are secondary to infectious etiologies.² Stroke is increasingly becoming the leading etiologic factor of SE owing to the changing incidence of the disease with age. Besides, stroke, like in many other parts of Nigeria and elsewhere, is the most prevalent disorder involving the central nervous system in adult in the community where the study was conducted.^{26–28}

Poor compliance with antiepileptic drug resulting in exacerbation of a pre-existing seizure disorder appeared the second most common cause in the study community. This finding, which is in keeping with reports from elsewhere, deserves further elaboration as it is a preventable precipitant of SE. Previous studies have shown that poor adherence to AED is associated with a three-fold higher risk of dying.^{29,30} In another prospective population-based study, low serum level of antiepileptic drug, in patients with epilepsy, was reported the most common etiology of SE.³¹

Reasons for the poor drug compliance with AEDs are many, they include high costs of medication for which the patients lower the dose of the AED, intentionally missing of AEDs with the aim of delaying running out of the medication, forgetting treatment dose, presence of comorbid mood disorders which could make the patients feel hopeless about the treatment efficacy and toxicity to AED.³²

Like in studies elsewhere,^{2,18–26} other common etiologies of SE in our study included CNS infections and metabolic abnormalities. Some studies in developing countries showed that CNS infections accounted for 28–67% of etiological spectrum,^{17,33} and that this was much more prevalent among child population.^{17,34,35} The CNS infections, including pyogenic meningitis, tuberculous meningitis and encephalitis, observed in the current study, were similar to reports from elsewhere.^{4,36,37}

Nevertheless, compared with previous reports,^{38–40} alcohol-related SE was not seen in our study. This finding, may be attributed to the lower prevalence of alcohol use and dependence in Muslim Hausa-Fulani communities among whom the study was conducted. Similarly, the absence of neurocysticercosis as a cause of SE in the study patients may also be ascribed to religion factor.

One-month mortality in the current study was 22.4%. This figure compared well with the short term mortality in patients with SE reported in other developing countries with figures ranging from 16% to 19.8%.^{15,17,33} However, this rate markedly contrasted those reported in the developed countries. For instance, a large-sample study conducted in the United States of America obtained short term mortality as low as 3.45%.³⁶ Availability and affordability of drugs, health care infrastructure, and transportation with attendant impact on the outcome¹⁵ may account for the marked regional disparity in the mortality figures. Furthermore, very few of our patients had ICU care; the ICU of the tertiary care hospital where the study was conducted is a 4-bedded facility serving as a general ICU for a number of states in Northwestern Nigeria. Consequently, access to this vital care is often very competitive and bed occupancy is often between 75 and 100%.⁴¹

It is also worthy of note that intravenous diazepam and/or intravenous phenytoin and/or phenobarbitone intramuscular diazepam, for breakthrough seizure, were used in the majority of the patients that were recruited in the study. Effective first line AEDs such as, midazolam, and propofol are readily available in our setting, but their use is limited to the Intensive Care Unit where adequate continuous monitoring of cardio-respiratory vitals could be rendered. Access to the ICU is often impossible in our setting, consequently, the range of AED for use in SE patients is significantly limited.

In conformity with previous reports,^{11,15,24} this study also showed that the duration of seizure, delay in initiation of treatment and refractory status epilepticus were significantly associated with death. Delay in initiation of treatment seen in this study could be attributed to such factors as those that can add to the time that is critical for survival of patients with SE and such factors include delay in transportation and undue logistic at the medical emergency unit. Given that factors such as delay in treatment and duration of SE are potentially modifiable, efforts focused on reducing critical time may translate into an appreciable reduction in mortality from SE.

Out of all the variables considered in this study, refractory status epilepticus emerged an independent predictor of death from SE in the study. Nevertheless, duration of seizure differ significantly across GOS score in the patients.

Absence of ictal EEG for diagnosis of NCSE, which might have resulted in under diagnosis of SE, poor access of SE patients to care in the ICU, lack of serum AED assay to ascertain poor drug compliance as well as the unavailability of neuro-imaging in all the patients were some of the shortcomings of the current study. Despite these limitations, the results from our study, which are comparable with reports from elsewhere in the developing countries, highlights the importance of early diagnosis and early treatment in the management of SE.

5. Conclusion

Our study showed that the most common underlying etiologies in SE were stroke, antiepileptic drug non-compliance, CNS infections and metabolic derangement. Duration of seizure, delay in treatment and refractory status epilepticus were significantly associated with death, but refractory seizure was an independent predictor of death in SE.

Conflicts of interest

All authors have none to declare.

REFERENCES

- Lowenstein DH. Status epilepticus: an overview of the clinical problem. *Epilepsia*. 1999;40:3–8.
- Lowenstein DH, Alldredge BK. Status epilepticus at an urban public hospital in the 1980s. *Neurology*. 1993;43:483–488.
- Owolabi LF, Shehu MY, Shehu MN, Fadare J. Pattern of neurological admissions in the tropics: experience in Kano, northwestern Nigeria. *Ann Indian Acad Neurol*. 2010;13:167–170.
- Ogunniyi A, Ogunniyi JO, Bademosi O, Osuntokun BO, Adeuja AO. Etiology of status epilepticus in Ibadan: a neuropathologic study. *West Afr J Med*. 1992;11:263–267.
- Aminoff MJ, Simon RP. Status epilepticus: causes, clinical features and consequences in 98 patients. *Am J Med*. 1980;69:657–666.
- Hauser WA. Status epilepticus: frequency, etiology, and neurological sequelae. In: Delgado-Escueta AV, Wasterlain CG, Treiman DM, Porter RJ, eds. *Advances in Neurology*. vol. 34. New York: Raven Press; 1983:3–14. Status Epilepticus.
- Cranford RE, Leppik IE, Patrick B, Anderson CB, Kostick B. Intravenous phenytoin in acute treatment of seizures. *Neurology*. 1979;29:1474–1479.
- Claassen J, Lokin JK, Fitzsimmons BFM, Mandelsohn FA, Mayer SA. Predictors of functional disability and mortality after status epilepticus. *Neurology*. 2002;58:139–142.
- Sagduyu A, Tarlacci S, Sirin H. Generalized tonic-clonic status epilepticus: causes, treatment, complications and predictors of case fatality. *J Neurol*. 1998;245:640–646.
- Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for revised clinical and electroencephalographic classification of epileptic seizures. *Epilepsia*. 1981;22:489–501.
- Hui ACF, Joynt GM, Li H, Wong KS. Status epilepticus in Hong Kong Chinese: etiology, outcome and predictors of death and morbidity. *Seizure*. 2003;12:478–482.
- Committee on Protocol for the Management of Medical Emergencies in Aminu Kano Teaching Hospital. In: *Protocol for the Management of Status Epilepticus*. vol. 1. Endem Press; 2009:102–105.
- Brainin M, Barnes M, Baron JC, et al. Guidance for the preparation of neurological management guidelines by EFNS scientific task forces – revised recommendations 2004. *Eur J Neurol*. 2004;11:577–581.
- Claassen J, Hirsch LJ, Mayer SA. Treatment of status epilepticus: a survey of neurologists. *J Neurol Sci*. 2003;211:37–41.
- Murthy JMK, Jayalaxmi SS, Kanikannan MA. Convulsive status epilepticus: clinical profile in a developing country. *Epilepsia*. 2007;48:2217–2223.
- Jennett B, Bond M. Assessment of outcome after severe brain damage. A practical scale. *Lancet*. 1975;1:480–484.
- Mhodji I, Nadiaye M, Sene F, et al. Treatment of status epilepticus in a developing country. *Neurophysiol Clin*. 2000;30:165–169.
- Towne AR, Pellock JM, Ko D, DeLorenzo RJ. Determinants of mortality in status epilepticus. *Epilepsia*. 1994;35:27–34.
- Ozdilek B, Midi I, Agan K, Bingol CA. Episodes of status epilepticus in young adults: etiologic factors, subtypes, and outcomes. *Epilepsy Behav*. 2013;27:351–354.
- Tatum WO, French JA, Benbadis SR, Kaplan PW. The etiology and diagnosis of status epilepticus. *Epilepsy Behav*. 2001;2:311–317.
- Treiman DM, DeGiorgio CM, Salisbury SM, Wickboldt CL. Subtle generalized convulsive status epilepticus. *Epilepsia*. 1984;25:653.
- Kaplan PW. Nonconvulsive status epilepticus. *Sem Neurol*. 1996;16:33–40.
- Treiman DM. Effective treatment for status epilepticus. In: Schmidt D, Schachter SC, eds. *Epilepsy Problem Solving in Clinical Practice*. United Kingdom: Martin Dunitz Ltd; 2000:253–265.
- Li JM, Chen L, Zhou B, Zhu Y, Zhou D. Convulsive status epilepticus in adults and adolescents of southwest China: mortality, etiology, and predictors of death. *Epilepsy Behav*. 2009;14:146–149.
- Celesia GC, Messert B, Murphy J. Status epilepticus of late adult onset. *Neurology*. 1972;22:1045–1055.
- Owolabi LF, Nagoda M. Stroke in developing countries: experience at Kano, Northwestern Nigeria. *Sudan JMS*. 2012;7:9–14.

27. Massaro AR. Stroke in Brazil: a South America perspective. *Int J Stroke*. 2006;1:113–115.
28. Owolabi LF, Akinyemi RO, Owolabi MO, Sani MU, Ogunniyi A. Profile of stroke-related late onset epilepsy among Nigerians. *J Med Trop*. 2013;15:29–32.
29. Jones JE, Hermann BP, Barry JJ, Gilliam FG, Kanner AM, Meador KJ. Rates and risk factors for suicide, suicidal ideation, and suicide attempts in chronic epilepsy. *Epilepsy Behav*. 2003;4:S31–S38.
30. Faught E, Duh MS, Weiner JR, Gu'erin A, Cunnington MC. Non-adherence to anti-epileptic drugs and increased mortality: findings from the RANSOM study. *Neurology*. 2008;70:1572–1578.
31. DeLorenzo RJ, Hauser WA, Towne AR, et al. A prospective, population-based epidemiologic study of status epilepticus in Richmond, Virginia. *Neurology*. 1996;46:1029–1035.
32. Owolabi LF. Precipitants of seizure among patients with epilepsy: experience at Kano, Northwestern Nigeria. *Sahel Med J*. 2012;15:24–29.
33. Garzon E, Fernandes RM, Sakamoto AC. Analysis of clinical characteristic and risk factors for mortality in human status epilepticus. *Seizure*. 2003;12:237–245.
34. Maharaj M, Henry D, Alik K, Mohammed PD. Status epilepticus: recent experience at the Port-of-Spain General Hospital, Trinidad. *West Indian Med J*. 1992;41:19–22.
35. Kwong KL, Lee SL, Yung A, Wong VC. Status epilepticus in 37 Chinese children: etiology and outcome. *J Paediatr Child Health*. 1995;31:395–398.
36. Koubeissi M, Alshekhlee A. In-hospital mortality of generalized convulsive status epilepticus: a large US sample. *Neurology*. 2007;69:886–893.
37. Legriél S, Mourvillier B, Bele N, et al. Outcomes in 140 critically ill patients with status epilepticus. *Intensive Care Med*. 2008;34:476–480.
38. Hesdorfer DC, Logroscino G, Cascino G, Annegers JF, Hauser WA. Incidence of status epilepticus in Rochester, Minnesota, 1965–1986. *Neurology*. 1998;50:735–741.
39. Waterhouse EJ, Garnett LK, Towne AR, et al. Prospective population-based study of intermittent and continuous convulsive status epilepticus in Richmond, Virginia. *Epilepsia*. 1999;40:752–758.
40. Coeytaux A, Jallon P, Galobardes B, et al. Incidence of status epilepticus in French speaking Switzerland (EPISTAR). *Neurology*. 1998;50:735–741.
41. Owolabi LF, Mohammed AD, Dalhat MM, Ibrahim A, Aliyu S, Owolabi DS. Factors associated with death and predictors of 1-month mortality in nontraumatic coma in a tertiary hospital in Northwestern Nigeria. *Indian J Crit Care Med*. 2013;17:219–223.