Etiological Spectrum of Status Epilepticus in Ouagadougou, Burkina Faso: A Prospective Cross-sectional Multicenter Hospital Study

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Abstract: *Introduction*: In sub-Saharan Africa, cerebrovascular aetiologies of status epilepticus (SE) are on the rise alongside infectious brain lesions. The aim of our study was to describe the etiologic spectrum of SE in Ouagadougou, Burkina Faso, in a high risk SE setting. *Patients and methods*: This is a prospective, cross-sectional, descriptive, multicenter and hospital study of patients hospitalized consecutively in the university hospitals of the city of Ouagadougou, in Burkina Faso, from 01/01/2015 to 12/31/2019, for SE. The sociodemographic, clinical, paraclinical (biological, critical and / or intercritical EEG, neuroradiological) characteristics and the aetiological data of the patients were analyzed using the Epi-info 7.2.1.0 software: calculations of numbers, frequencies, averages. The significance rate was set at 0.05. *Results*: 91 patients hospitalized for SE were collected, with a male predominance (73.62%). The mean age was 36.6 years +/- 24.5 years (2 days and 86 years); 25 patients (27.5%) already had known epilepsy. Generalized tonic-clonic SE from the start and focal SE with convulsive bilateralization were the most common seizure types with 46 cases (50.5%) and 27 cases (29.7%), respectively. The average duration of an EME episode was 18 hours +/- 31 (5 minutes - 6 days). On admission, a focused motor deficit with 36 cases (46.7%) and fever in 28 patients (30.8%) were the main clinical signs; hyperleukocytosis with 23 cases (25.3) and anemia with 22 cases (24.2%), were the main laboratory abnormalities. On cerebral CT scan, sequelae with 33 cases (51.6%), acute stroke with 14 cases (21.9%) and acute meningoencephalitis with 8 cases (12.5%), were the most frequent. SEs symptomatic of acute brain disease, with 61 cases (67%), were dominated by infectious etiologies with 30 cases (33%) and acute strokes with 16 cases (17.6%). Among the non-acute or sequelae SEs of etiology, the sequelae of traumatic brain injury with 10 cases (11%) and the sequelae of stroke with 8 cases (8.8%) were the most represented. *Conclusion*: The aetiologies of SE are dominated in the Ouagadougou CHUs by CNS infections, acute or sequelae strokes and cranio-encephalic trauma. The fight against infectious diseases and the prevention of vascular risk factors will help reduce the frequency and severity of EMEs.

Keywords: Status Epilepticus, Etiology, CNS Infections, Strokes, Head Trauma, Ouagadougou

1. Introduction

Status epilepticus (SE) is the second most common neurological emergency after stroke, with high mortality and an often poor functional prognosis in survivors [1, 2]. In developed countries, the overall incidence of EME ranges from 10.3 to 41 / 100,000 / year, higher in patients with

epilepsy. In terms of age, its distribution is bimodal with a peak in frequency in childhood and another over 60 years [3, 4]. SEs readily punctuate the course of a known epilepsy and are therefore more frequently caused by underdosing of anti-epileptic drugs (UAEDs). When they are inaugural, they most often complicate an acute stroke, or inaugurate epilepsy secondary to sequelae brain damage [5, 6].

According to studies in sub-Saharan Africa (SSA), the aetiologies appear to be dominated by infectious brain damage, imbalance in anti-epileptic therapy, stroke and metabolic disorders [7, 8]; However, a tendency towards the preeminence of cerebrovascular causes is increasingly observed in recent studies [8-10].

Burkina Faso, like other SSA countries, poor with limited resources, has a high prevalence and incidence of epilepsy, epileptic seizures and etiological factors of epileptic seizures, contrasting with a double gap in diagnosis and treatment, exposing to a high risk of SEs, and high morbidity and mortality [7]. We performed the present hospital and multicenter study on the etiologies of SEs in Ouagadougou, Burkina Faso. Knowing the etiological spectrum of SEs in our context will contribute to better management of this desease and subsequently to improving the prognosis of patients with it.

2. Patients and Methods

This was a prospective, cross-sectional, descriptive, multicenter and hospital study, which took place from January 1, 2015 to December 31, 2019, involving all patients consecutively hospitalized in University Hospital Centers (UHC) in the city of Ouagadougou (Tingandogo, Yalgado Ouédraogo, Bogodogo, Pediatric Charles De Gaulle), for SEs, during the study period.

Were included in our study, patients of all ages consecutively hospitalized in the so-called UHCs (emergency services, neurology, pediatrics, intensive care units, or any other services), for Sus diagnosed clinically and / or confirmed by an electroencephalogram (EEG), and who have given their informed consent or for which the family has given their informed consent. Not included in our study were patients who had a seizure that did not meet diagnostic criteria for SE, those for informed consent to participate in the study had not been obtained. The data was collected directly from patients or their families during hospitalization and recorded in their medical files, then transcribed on a data collection sheet.

Confirmation of the positive diagnosis and the etiology of SEs was made by a neurologist.

The management of SEs at the sites of our study was based on the recommendations of the 2009 SFRLF consensus conference [11].

Information on the history of the disease, history and physical examination were systematically recorded. Complementary, biological and neuroradiological examinations were performed according to the context. In an unknown patient with epilepsy: standard laboratory tests (blood count, C Reactive Protein (CRP), blood ionogram, occasional glycemia, azotemia, creatinine, hepatic transaminases) and cerebral CT. In any febrile patient regardless of the context, a cerebral CT scan and a lumbar puncture with cytological, biochemical and bacteriological study of the CSF were performed. In the epileptic patient known under treatment: standard laboratory assessment, search for imbalance in anti-epileptic therapy factors (discontinuation of treatment, suboptimal dosage, intercurrent events of transit disorders such as diarrhea / vomiting, etc.). Blood or urine assays for toxicants were not performed due to their very high costs. Depending on the clinical course of the SE, after admission to the emergency department, the patient was either transferred to the neurology unit (discontinuation of SE) or to intensive care unit (refractory SEs); patients who were already in refractory SE (larvae or subtle SE) on admission, were quickly transferred to intensive care unit. The patient received clinical and / or biological monitoring depending on the context. The EEG was not essential for the diagnosis of SE except in cases of non-convulsive SE (larvae or subtle SE, non-convulsive SE) where the clinically suspected positive diagnosis had to be confirmed by the demonstration of a prolonged and / or sequential rhythmic paroxysmal epileptic activity.

The following variables were taken into account in our study: sociodemographic characteristics of the patients, clinical data of SE (history of epilepsy, duration of SE, number of recurrent SE episodes, clinical classification of the SE), clinical examination of the patient on admission, laboratory exams on admission, critical and / or intercritical EEG data, results on CT and / or brain MRI of patients, etiological diagnosis of SE (acute structural or functional epileptogenic cerebral disorders / attacks, non-acute or sequelae epileptogenic cerebral lesions, undetermined etiologies). Alertness on admission was assessed using the Glasgow Coma Scale.

The data collected was entered and processed on a microcomputer, then analyzed using the Epi-info 7.2.1.0 software in its French version. The graphs and tables were produced in Excel 2013. The analysis focused on the calculations of the numbers, frequencies and averages of the data collected. The significance rate was set at 0.05.

SE has been defined, in a general sense, by continuous epileptic seizures or by the succession of at least two epileptic seizures without recovery of consciousness in intercritical, over a period of at least 30 minutes. The generalized convulsive SE, because of its severity, was defined by continuous seizures beyond 5 minutes or by the succession of at least two epileptic seizures without complete recovery of consciousness in intercritical over a period of at least less than 5 minutes [1].

The diagnosis of acute symptomatic SE of electrolyte disturbances was made in the event of deep hyponatremia ($\leq$ 120 mmol / l), and / or significant hypocalcemia ($\leq$1.8 mmol / l), or hypercalcemia ($\geq$2, 6 mmoles / l). The blood tests for drugs, alcohol level, the test for toxins in the blood and urine, were not done. The imbalance in AED was indirectly

evaluated by treatment breaks, non-compliance with treatment, suboptimal doses in AED, intercurrent clinical events responsible for treatment imbalance (diarrhea, vomiting, etc.).

We obtained prior authorization from the administrations of the various university hospitals, before starting our study. The confidential and anonymous nature of the information collected from patients or their companions has been guaranteed.

3. Results

During the study period, we consecutively collected 91 patients hospitalized for SU.

The mean age of the patients was 36.6 years +/- 24.5 years (2 days and 86 years). Patients aged 15-45 and 0-14 were the most represented with 34 cases (37.4%) and 29 cases (31.9%), respectively. there was a male predominance, with 67 men (73.62%) and 24 women (26, 37%); the sex ratio M / F was 1.4. The majority of patients, or 43 patients (47%) were married. Unemployed patients with 27 cases (29.7%) and retirees with 14 cases (15.4%) were the most frequent socio-professional categories. Patients residing in urban areas with 63 cases (69.2%) were the most represented (Table 1).

Among our collected patients, 66 patients (72.5%) had no clinical history of epilepsy, while 25 patients (27.5%) were already known to have epilepsy. Among non-epileptic patients, in 55 patients (83.3%), SE occurred early between 1 and 14 days after the onset of an acute brain disease compared to 11 patients (17.5%) in whom SE occurred later (beyond 14 days). For patients with known epilepsy, the mean duration of epilepsy was 39.7 months +/- 54.1 (2 - 252 months); the average seizure frequency was 2.1 seizures +/- 3.2 (1-7 monthly seizures).

Sociodemographic data	Number (n=91)	Percentage (%)
Age groups		
0-5 years	20	22
6-15 years	9	9.9
16-25 years	14	15.4
26-35 years	12	13.2
36-45 years	8	8.8
45-55 years	6	6.6
56-65 years	13	14.3
66 years and over	9	9.9
Marital status		
Married	43	47
Singles	42	45.8
Widowers / Widows	5	6
Divorced / divorced	1	1.2
Socio-professional categories		
Unemployed	27	29.7
Retirees	14	15.4
Housewives	13	14.3
Cultivators / breeders	12	13.2
Employees	9	9.9
Tradespeople	9	9.9
Pupils / students	7	7.7
Residence		
Urban	63	69.2
Rural	28	30.8

Of all the patients collected, 88 patients (96.7%) presented with convulsive SE versus three (3) patients (3.3%) who presented with confusional non-convulsive SE. Generalized tonic-clonic SE (GTC SE) from the start and focal SE with convulsive bilateralization (FBC SE) were the most represented types of seizures with respectively 46 cases (50.5%) and 27 cases (29, 7%) (Table 2).

Table 1. *Distribution of patients according to socio-demographic data.*

Table 2. *Distribution of patients by type of SE seizure (n=91).*

	Frequency	Percentage (%)
Convulsive SEs	88	96.7
GTC straight away SE	46	50.5
Focal SE with convulsive bilateralization	21	23.1
larvae or subtle SE	6	6.6
Focal somatomotor and / or vegetative SE	5	5.5
Kowjewnikow syndrome	5	5.5
Generalized myoclonic SE	3	3.3
Tonic generalized SE	2	2.2
Confusional non-convulsive SE	3	3.3
Total	91	100

The average duration of an SE episode was 18 hours +/- 31 (5 minutes - 6 days). SEs lasting> 2 hours and SEs lasting ≤ 30 minutes were the most represented with 36 cases (39.6%) and 34 cases (37.4%) respectively (Figure 1).
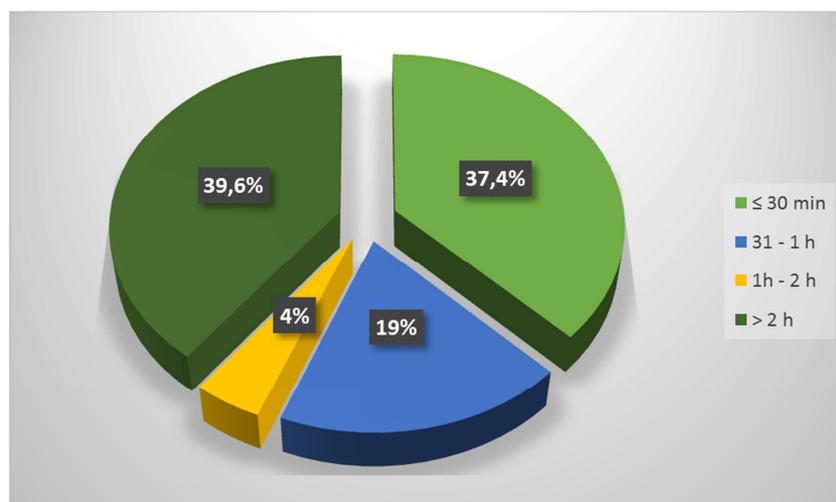
Figure 1. *Distribution of patients according to the duration of the SE episode.*

The mean number of SE episodes during hospitalization was 3.5 +/- 3.1 (1-20 episodes). EEG was performed only in 22 patients (24.2%), after monitoring of vital functions, in 9 patients (9.9%) percritical and 13 patients (14.3%) postcritical. The tracing was normal in 3 patients (13.6%) at post critical and in 19 patients (86.4%), it had found epileptic paroxysms mainly focal or multifocal.

Table 3. Distribution *of patients according to clinical, biological and neuroradiological characteristics on admission.*

	Number (n)	Percentages (%)
Clinical signs on admission		
Vigilance disorder	54	59.3
Focused motor deficit	36	39.6
Fever	28	30.8
HTA	21	23.1
Aphasia	16	17.6
Meningeal syndrome	10	10.9
Confusional Syndrome	9	9.9
Cognitive deterioration	8	8.8
Focused sensory deficit	7	7.7
Laboratory abnormalities on admission		
Hyperleukocytosis	23	25.3
Anemia	22	24.2
Hyponatremia	9	9.9
Renal failure	8	8.8
Hyperglycemia	8	8.8
Hypokalaemia	6	6.6
Hypomagnesemia	5	5.5
Hypermagnesemia	5	5.5
Hypoglycemia	4	4.4
Hypocalcaemia	4	4.4
Hypernatremia	3	3.3
Hypercalcemia	2	2.2
Puncture with CSF study	13	14.3
Normal	7	7.7
Hypercytosis	4	4.4
Hyperproteinorachia	3	3.3
CT results	Number (n=64)	Percentages (%)
Sequelae lesions *	33	51.6
Acute stroke	14	21.9
Acute meningoencephalitis / abscess or cerebral empyema	8	12.5
Brain tumor	3	4.7
Hydrocephalus	3	4.7
Malformation of cortical development	2	3.1
Cerebrovascular malformation: cavernoma	1	1.6

NB: Several lesions were found in the same patient

* Sequelae lesions: cortical atrophy, porencephalic cavity, hypodensity / sequellar gliosis of head trauma, stroke, post-anoxo-ischemic encephalopathy or meningoencephalitis.

On admission, a focused motor deficit with 36 cases (46.7%), fever in 28 patients (30.8%), hypertension in 21 patients (23.1%), aphasia with 16 cases (20.8%), meningeal syndrome with 10 cases (12.9%), were the main clinical signs; hyperleukocytosis with 23 cases (25.3) and anemia with 22 cases (24.2%), were the main laboratory abnormalities. A lumbar puncture (LP) with study of cerebro spinal fluid (CSF) was performed in 10 patients and found hypercytosis and hyperproteinorachia, respectively in 4 patients (4.4%) and 3 patients (3.3%). Brain CT was performed in 64 patients (70.3%), sequelae lesions with 33 cases (51.6%), acute stroke with 14 cases (21.9%) and acute meningoencephalitis with 8 cases (12.5%), were the most frequent (Table 3).

Etiologically, SEs were subdivided into SEs symptomatic of an acute brain disease with 61 cases (67%) and SEs of non-acute or sequelae etiologies with 30 cases (33%) of the type of sequelae brain lesions objectified to CT.

Among SEs symptomatic of acute brain disease, infectious etiologies and acute strokes were the most prevalent. Among SEs of non-acute or sequelae etiology, sequelae of heat trauma with 10 cases (11%) and sequelae of stroke with 8 cases (8.8%) were the most represented (Table 4).

Table 4. *Distribution of patients according to the etiology of the SE.*

EME symptomatic of acute brain disease	Frequency (n=61)	Percentage (%)
Infectious causes	30	33
Acute meningoencephalitis	13	14.3
Systemic febrile infections	8	8.8
Severe malaria neurological form	6	8.8
Brain abscesses / empyemas	3	3.3
Acute stroke	16	17.6
Imbalance of anti-epileptic treatment	9	9.9
Metabolic causes	7	7.7
EME of non-acute or sequelae of etiologies	Frequency (n=30)	Percentage (33)
Sequelae of traumatic brain injury	10	11
Sequelae of stroke	8	8.8
Sequelae of meningoencephalitis	3	3.3
Sequelae of perinatal brain distress	3	3.3
Brain tumor	3	3.3
Cortical development abnormalities	2	2.2
Cerebrovascular malformation: cavernous angioma	1	1.1

4. Limitations of Our Study

Certain important data in this study, in particular the duration of the SE, the semiology of the seizures, obtained solely on the basis of questioning the patients and / or their entourage, may therefore have been biased. Indeed, most SUs started before the patients were admitted.

The relatively low proportion of pediatric cases (31.9%) could have been underestimated given the high frequency of epilepsy and seizures in children.

Nonetheless, the multicenter nature of our study, extended to all 3 university hospitals in the city of Ouagadougou, allowed us to have fairly exhaustive, representative and reliable data.

5. Discussion

The mean age of our patients was 36.6 years, near the mean ages of 36 years and 37.1 years reported respectively in Ethiopia [12] and Côte d'Ivoire [7]; higher mean ages of 44.5 years, 47.7 years, 49 years and 50 years were reported respectively in Madagascar [13], Guinea [9], Cameroon [14] Senegal [8]. Methodological differences with or without inclusion of pediatric cases partly explain these differences.

According to data from the literature in Western countries, people with pre-existing epilepsy account for 30-50% of SEs cases [1]. However, in SSA countries, patients with prior epilepsy are reported in lower proportions, 9.9%, 23.7%, 27.5%, 28.7% and 33%, respectively in Senegal [15], in Nigeria [10], in our study, in Madagascar [13] and in Côte d'Ivoire [7]. This difference could be related to a lack of knowledge of the epileptic history of patients, most often based on the verbal testimony of patients or their entourage, in the absence of documented evidence, in SSA [16].

There seems to be a great disparity in the duration of SEs across studies. Thus in our study, 55% of SEs lasted less than 2 hours, while SEs lasted more than 30 minutes in 20% of cases in a study in Cameroon [14] and more than 30 minutes in the majority of patients, in a Guinean [9] and Senegalese [8] study. A duration of ≤ 1 hour was reported in 70% of patients in a Tunisian study [17], contrasting with an average duration of 20 to 24 hours noted in more than 2 / 3 of the patients in a Swiss study [18]. These disparities in the duration of the SEs according to the different studies could be explained in our opinion, by the extreme difficulty of determining the exact duration, the delays in patient admission (early versus late), the severity and the type of SEs (generalized convulsive SE versus non convulsive SE; rapidly regressive SEs versus refractory SE), the methodology and type of reception structure (emergency service or neurology service versus medical intensive care unit) and the precision of diagnostic means (clinical observation versus prolonged video-EEG monitoring).

In series from developed countries, the semiology of SE is dominated by focal seizures in 2/3 to 3/4 of cases [4, 3, 18],

while in SSA, focal SEs are reported in less than 50% of cases [7, 9, 13], and vary greatly from one study to another, due to methodological differences, some dealing only with convulsive SEs and others only with generalized convulsive SEs, often excluding EME non-convulsive. EME GTCs from the outset represent less than 1/3 of the cases reported in developed countries [18], while in most studies of SSA they represent more than 50 to 90% of cases [7, 9, 13], with the exception of the study by Cissé in Guinea [9], where their frequency was 27.8%. The high rate of generalized convulsive SEs immediately reported in SSA could be explained by the diagnostic difficulties of focal SEs, in particular non-convulsive, which often require the contribution of EEG, unfortunately not always available in the context of work in ASS. Furthermore, focal SEs with convulsive bilateralization are underdiagnosed because the focal onset is sometimes unrecognized or ignored and therefore mistakenly taken for a generalized convulsive SE from the outset, by emergency physicians.

EEG is not essential for the diagnosis of convulsive SE, but it is of great help in the diagnosis of non-convulsive SE, especially confusional [19]. Thus, in our series, EEG was performed only in 24.2% of patients, ie in 9.9% of cases percritical, after control of vital functions, and 14.3% of cases after critical; it enabled the diagnosis of confusional non-convulsive SE in 3% of patients.

The etiological diagnosis of SEs, in particular those occurring in patients not previously epileptic, is based on neuro imaging. While most acute, subacute, or sequelae epileptogenic brain lesions, up to 50% of patients with epileptogenic structural lesions, such as small-volume tumors and vascular malformations, escape CT. Only MRI allows the detection of the majority of subtle lesions such as hippocampal sclerosis, nodular or band heterotopias, focal cortical dysplasias, and tiny tumors such as DNETs, which frequently provide seizures and SE [20, 21], unfortunately still limited availability and accessibility in our context.

In developed countries, AED underdosing, acute stroke, and sequelae brain injury are the three most important causes of SE in adults. Other causes, such as metabolic disorders, complications from alcoholism, CNS infections and brain tumors, come second. In 3% to 10% of cases, no etiology was found [1, 6, 22, 23]. In children, the three most common causes are fever (52%), non-acute brain damage (39%) and underdosing AED (21%). The other aetiologies do not represent more than 10% each [6]. In India, CNS infections, imbalances in anti-epileptic treatment and stroke, are the main aetiologies reported [24], while in Brazil, CNS infections, strokes and metabolic disorders are the predominant aetiologies [25]. In SSA, CNS infections, imbalances in anti-epileptic treatment, metabolic disorders and stroke are the most frequently reported causes, in decreasing order of frequency [8, 12, 15]. However, the most recent studies report a tendency towards the predominance of strokes [8, 9, 14]. In our series, traumatic brain injury is the 3rd most common cause of SE after CNS infections and

stroke. This is due to a sociological particularity in Burkina Faso, where the populations, in particular the young fringe, use in a privileged way individual means of transport on two wheels (bicycles, mopeds, motorcycles), at the expense of the means of public transport, very much. often without a protective helmet, which causes frequent accidents on the public road and head trauma providers of epilepsy and SE.

The predominance of cerebrovascular aetiologies in developed countries could be explained by the high frequency of strokes, due to the aging of the population and the increase in vascular risk factors [22]. The high frequency of underdosing in AED could be linked to the effectiveness of screening for epilepsy patients and the excellent therapeutic coverage of patients, which however contrasts with poor adherence to treatment probably due to the side effects of AEDs [18].

In SSA, the high frequency of infectious diseases favored by ecological conditions, insufficient vaccine coverage, low level of hygiene and weak health systems, could explain the preponderance of the infectious aetiology [26, 27].

6. Conclusion

SEs more often affect young patients with no history of epilepsy. The clinical profile is dominated by generalized convulsive SEs at the outset or secondarily generalized convulsive. The aetiologies are dominated by CNS infections, acute or sequelae strokes and cranio-encephalic trauma. Controlling infectious diseases, primarily malaria and infectious meningoencephalitis, and preventing vascular risk factors, will help reduce the frequency and severity of SEs in our setting.

7. Recommendations

In order to help reduce the frequency and severity of EMEs, we recommend strengthening the fight against infectious diseases in SSA, through vaccination and sanitation of the living environment, as well as the prevention of accidents on public roads, in particular by promoting the use of helmets. Likewise, complementary, multicenter, collaborative studies on EMEs at the national and African level are also needed.

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