

Etiologies of Non-Genetic Epilepsies of Adults Newly Diagnostic in Ouagadougou in Burkina Faso

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Abstract

Introduction: Epilepsy is a common disease in sub-Saharan Africa. The etiological diagnosis of epilepsies is still based on interrogation and clinical examination, due to a lack of means of complementary diagnostic investigations, electroencephalogram (EEG), computed tomography (CT) or magnetic resonance imaging (MRI). The aim of our study was to determine the etiological varieties of non-genetic epilepsies of adults, newly diagnosed in Ouagadougou, according to diagnostic, clinical, EEG and neuroradiological criteria (encephalic CT and/or MRI).

Patients and methods: This was a prospective, cross-sectional, descriptive study that ran from September 1 to August 31, 2017, and included patients diagnosed with non-genetic epilepsy in adults, newly diagnosed in Ouagadougou, Burkina Faso. For each of the patients included in the study, the etiological diagnosis was based on the results of CT and/or brain MRI, in addition to the electroclinical criteria of non-genetic epilepsy.

Results: We collected 137 patients; the average age was 41.8 ± 17.6 years, with 51.8% men (71 patients). The average age of onset of seizures was 34.3 years and the average duration of seizures was 7.3 years. Of all patients, 87.5% had focal seizures, 5.2% generalized seizures and 7.3% non-classifiable seizures. All of our patients had EEG and brain scan, only 11.8% had brain MRI. EEG was normal in 13.1%; there were inter-critical epileptic paroxysms in 86.9%. Localized atrophy associated with underlying parenchymal hypodensity with 48 cases (35%), porencephalic cavities with 16 cases (11.8%), circumscribed cortico-subcortical hypodense without contrast enhancement with 14 cases (10.2%), brain tumors with 12 cases (8.8%), were the most representative neuroradiological abnormalities. The structural causes and unknown causes were found respectively in 54% and in 46% of cases. CNS infections (16.8%), sequelae of stroke (11.7%), sequelae of cranioencephalic trauma (10.9%), brain tumors (8.7%), sequelae of Perinatal encephalopathy (4%) and cerebral vascular malformations (cavernoma) (1.5%) were the epileptogenic structural abnormalities found.

Conclusion: Our results confirm the predominance of infectious and post-traumatic causes and the emergence of cerebrovascular causes in sub-Saharan Africa. Some epileptogenic lesions, such as certain brain tumors, focal cortical dysplasias, hippocampal sclerosis, have been under diagnosed because of the poor availability and accessibility of cerebral MRI.

Keywords: Epilepsies etiologies; Brain; CT scan

Introduction

Epilepsy is a common disease in the tropics and especially in sub-Saharan Africa. Its incidence remains high in sub-Saharan Africa, ranging from 64 to 159/100,000 inhabitants [1,2]. Its prevalence is 2 or 3 times higher than that found in the industrialized countries, in fact, extremely variable depending on the country and the study methodology used, with extremes of 5.2% to 74% and median evaluated at 15% [2]. A terminological and conceptual revision of the organization of epileptic seizures and epilepsies was adopted in 2010 [3]: non-genetic epilepsies (formerly called "non-idiopathic") now include epilepsies from structural, metabolic, infectious, immune (formerly "symptomatic epilepsies") or unknown cause (formerly "cryptogenic or probably symptomatic epilepsies"). In sub-Saharan Africa, there is a probable overestimation of generalized tonic-clonic seizures (59%), due to under-medicalization, the shortage of neurologists and electroencephalograms that could be the cause of 'poor seizure classification [2]; In contrast, there is an underestimation of the proportion of focal and then bilateral tonic-clonic seizures, whose focal point is difficult to recognize by the clinic alone. However for Senanayake and Roman [4], there would be a predominance of focal seizures and even lesional epilepsies, especially in Nigeria.

In developing countries in general and in sub-Saharan Africa in

particular, the etiological diagnosis of epilepsies is most often based on interrogation and clinical examination, the routine complementary diagnostic means that are the electroencephalogram (EEG) and cerebral or specialized CT scans such as magnetic resonance imaging (MRI) with limited availability and accessibility [2]. Thus until 2006, epilepsies of unknown cause accounted for more than 50% of the cases in this part of the world, but this proportion tended to decrease according to the number of paraclinical explorations carried out, in particular neuroradiological. The causes identified are multiple, varying according to age and geographical location, reported in 20 to 50% of patients included in major epidemiological

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investigations [5]. Congenital, developmental and genetic causes are associated with epilepsy in childhood, adolescence and young adults. In the elderly, cerebrovascular causes are the most common. Head trauma, sporadic CNS infections and brain tumors occur at any age, although tumors appear to be more common beyond 40 years of age. The etiology of epilepsy may be multifactorial, and an acquired condition may be more likely to occur if hereditary predisposition is present [6]. It is difficult to determine the relative contributions of each cause of epilepsy in sub-Saharan Africa, even though infectious diseases, especially parasitic diseases such as cysticercosis or malaria, seem to be the most common causes [2,7]. In order to contribute to a better understanding of epilepsies in sub-Saharan Africa in general and in Burkina Faso in particular, we conducted the present descriptive cross-sectional study on etiological varieties of newly diagnosed adult non-genetic epilepsies in Ouagadougou, based on diagnostic, clinical, EEG and neuroradiological specific criteria (brain CT and/or MRI).

Patients and Methods

This was a prospective, cross-sectional, descriptive study that took place from September 1 to August 31, 2017, involving newly diagnosed patients with newly diagnosed non-genetic epilepsy at one of three sites the study in the city of Ouagadougou, with external consultation of neurology with recruitment of epileptic patients, Tingandogo University Hospital, Yalgado Ouedraogo Teaching Hospital and Schiphra Medical Center. Included in the study were all patients, at least 18 years old, who were neurologically outpatient in one of the 3 centers during our study period, in whom non-genetic epilepsy was diagnosed on the clinical, electroencephalographic and neuroradiological arguments (brain CT and/or MRI), having given their consent to their participation in the study.

For each patient collected, the following variables were studied: socio-demographic variables (age, sex, risk factors for epilepsy, level of education); clinical variables (age at onset of seizures, duration of seizures, frequency of seizures, seizure type according to Crisis classification and epilepsy of LICE 2010, neurological abnormalities, cognitive disorders, behavioral disorders), results of the EEG standard, results of encephalic imaging (CT and brain MRI), etiologies of epilepsy according to the 2010 LICE classification. For each patient collected an individual collection card was used. Data entry and analysis was done using the statistical software EPI INFO 7.1.10. The chi-square test and the Fischer test served as statistical tests with a significance level of 5%. Patient confidentiality has been respected. The study was carried out with the authorization of the general direction of the various health structures and the bioethics committee of Burkina Faso.

Operational definitions

The diagnosis of epilepsy of non-genetic causes in adults was based primarily on the absence of arguments in favor of a genetic epileptic syndrome (formerly idiopathic): onset in children between the age of 4-10 years, the possibility of genetic epilepsy in family history but the absence of acquired risk factors for epilepsy in personal history, a neurological, neuroradiological and neuropsychological assessment normal, rare and brief crises often appear in sleep and d other, interictal paroxysmal abnormalities appearing on normal background activity, an increase in these abnormalities in sleep and an evolution most often favorable with healing towards puberty.

The diagnosis of epilepsies of structural cause was based on a bundle of arguments associating:

- The presence of risk factors for epilepsy, except genetic, in the patient's history,
- A beginning of epilepsy at all the ages of the life, a semiology including all the types of possible crises, - The frequent coexistence of neurological signs of focusing, cognitive deterioration, behavioral disorders and sometimes extra neurological signs suggesting an underlying disease responsible for epilepsy,
- Inter-critical EEG patterns suggestive of underlying brain damage: abnormal background activity, slow or asymmetrical; continuous slow waves, associated or not with spikes, spikes waves or polypoints waves; flattening focused along a focal point; independent multifocal abnormalities, diffuse paroxysmal abnormalities with predominantly anterior, predominantly anterior, predominantly anteriorly sloping spike type, showing the appearance of secondary bilateral synchrony [8]; reduction or disappearance of focal critical inter-axial anomalies before the first clinical signs appear, polymorphic critical discharge, the existence of slow rhythmic waves, critical discharges without clinical translation.
- Neuroimaging of a fixed or progressive structural lesion, whose epileptogenicity has already been demonstrated, with a positive anatomo-electro-clinical correlation [8].

The epilepsies of unknown cause (formerly cryptogenic) were based on the same diagnostic criteria, clinical and EEG, as the epilepsies of structural causes, but differed in the negativity of the neuroradiological examinations, notably the absence of causal lesions at the cerebral CT for all our patients and at the encephalic MRI for some. In addition to the diagnostic criteria, epilepsies related to a structural or metabolic cause have been diagnosed by CT or MRI visualization of structural abnormalities for which the existence of an association has been demonstrated with a substantial increase in the risk of developing epilepsy through rigorous studies. These cicatricial lesions, acute or progressive, include stroke, head trauma, infections, brain tumors, pre-perinatal encephalopathy sequelae, certain neuroectodermal diseases, miscellaneous malformations of cortical development, chronic alcoholism. The metabolic causes were not explored in our study, for reasons of financial inaccessibility or unavailability of these tests for our patients.

Results

During our study period we collected a total of 137 patients. The mean age of the patients was 41.8 ± 17.6 years (range 18 to 86 years), there were 71 male patients (51.8%) and 66 female patients (48.2%), which is a sex ratio M/F of 1.07. The age groups between 15 and 45 years were the most represented. One hundred (100) patients (73%) were still in school or had completed at least secondary education, 27 patients (20%) had never been to school and 10 patients (7%) had dropped out of primary school. Of the 110 patients initially enrolled, 34 (30.9%) had a primary level, 43 (39.1%) a secondary level and 33 (30%) a higher level. At least one epileptic FR was identified in the history of 81 patients or 59.1%; the most frequently reported were head trauma in 15 patients (10.9%), stroke and cerebral infectious disease in 22 patients (16.1%) each. The average age of onset of the first seizures was $34.3 \text{ years} \pm 19.1$ (range 1 to 83 years), the age range of onset of seizures between 15 and 45 years was the most representative, or 79% of patients. The average duration of seizures to diagnosis was 7.3 years per patient (range 1 to 49 years). The most frequent periodicity was daily in 48 patients (35%) and monthly in 56 patients (40.5%). Of the 137 patients, 120 (87.50%) had a focal seizure, 7 patients (5.20%) had a generalized crisis and 10 patients (7.30%) had non-classifiable seizures. Among the 120 cases of focal seizures: 100 patients or 83.3% had a focal seizure

without impaired consciousness. Of these 100 cases of CF without impaired consciousness, 95% progressed to convulsive bilateralisation; 20 patients (16.6%) had a focal seizure with impaired consciousness of which 18 cases (15%) with psychomotor automatisms. Cognitive deterioration with 63 cases (46%), focused motor deficit with 34 cases (24.8%), behavioral disorders with 26 cases (19%), focused sensory deficit with 13 cases (9.5%) and the various other neurological disorders with 18 cases (13.1%), were the main neurological and neuropsychic associated signs.

Of the 137 patients, all had an EEG and a brain scan, only 16 patients (11.8%) were able to achieve more encephalic MRI. Standard EEG was normal in 18 patients (13.1%); there were inter-critical epileptic paroxysms in 119 patients (86.9%). Frontal epileptic paroxysms with 42 cases (30.7%), plurifocal or diffuse paroxysms with 40 cases (29.2%) and temporal paroxysms with 13 cases (9.5%) were the most representative. Background activity was abnormal in 121 cases (93.8%); they were slow theta waves (93.4%), slow delta waves (4.1%), and fast rhythms (2.5%). The results of the neuroradiological examinations, CT or brain MRI were abnormal in 74 cases (54%). Non-specific brain atrophy with 61 cases or 44.5%, stroke sequelae with 22 cases (16.1%), and porencephalic cavities with 16 cases (11.8%) were the most frequently encountered neuroradiological abnormalities. At least one etiology of epilepsy was identified in 74 patients, 54%; in 63 patients, 46%, no cause was identified. Central nervous system (CNS) infections with 23 cases (16.8%), stroke sequelae with 22 cases (16%) and sequelae of head trauma with 15 cases (10.9%), were the causes most frequently reported epilepsy. Among CNS infections, there were 10 cases of meningoencephalitis sequelae (7.3%), 6 cases of neurocysticercosis (4.4%), 5 cases of cerebral toxoplasmosis (3.6%) and 2 cases of brain abscess (1.5%). As for the 12 cases of brain tumors they were divided into 7 cases of astrocytoma, 4 cases of meningioma and 1 case of DNET. Regarding cerebral vascular malformations, 2 cases of cavernoma were identified (Table 1).

Variables	Numbers	Frequencies
Risk factors for epilepsy in antecedents		
Head trauma	15	10.9%
AVC	18	13.1%
Infectious encephalic pathology (non-specific meningoencephalitis, toxoplasmosis, neurocysticercosis)	22	16.1%
Perinatal encephalopathy	7	5.1%
Brain tumor already known	7	5.1%
Febrile seizures	2	1.5%
Periodicity of seizures		
Daily seizures	48	35%
Weekly seizures	15	10.9%
Monthly seizures	56	40.5%
Quartely seizures	9	6.6%
Half-yearly seizures	5	3.6%
Annual seizures	4	1.6%
Type of seizures		
Focales seizures	120	87.5%
• Focal motor seizures without impaired consciousness	74	54.0%
• Focal seizures with subjective sensory phenomena	40	29.2%
• Focal seizures with psychic phenomena	25	18.2%
• Focal seizures with dysautonomic signs without impaired consciousness	20	14.6%
Focal seizures with impaired consciousness with or without psychomotor automatisms	20	14.6%

Generalized seizures	7	5.2%
Unclassifiable seizures	10	7.3%
Topography of epileptic paroxysms		
Frontal paroxysms	42	30.7%
Temporal paroxysms	13	9.5%
Central paroxysms	5	3.6%
Pariétal paroxysms	9	6.6%
Occipital paroxysms	3	2.2%
Generalized paroxysms	7	5.1%
Multifocal or diffuse paroxysms	40	29.2%
Normal EEG	18	13.1%
Neuroradiological abnormalities		
Cortico-subcortical atrophy	61	44.5%
• Localized atrophy associated with adjacent cerebral parenchymal hypodensity	48	35%
• Hemi hemispherical atrophy associated with ipsilateral thickening of the cranial vault, ipsilateral dilation of the sinuses of the base. ascent of the rocks	4	2.9%
• Cerebral diffuse atrophy	9	6.7%
Periventricular leuko encephalomalacy	3	2.2%
Porencephalic cavity	16	11.8%
Peripheral cortico-subcortical hypodensities without CT contrast enhancement	14	10.2%
Cerebral tumors	12	8.8%
• Astrocytoma	7	5.1%
• Meningioma	4	2.9%
• DNET	1	0.7%
Multifocal cortico-subcortical nodular brain calcifications	9	6.7%
Inflammatory granulomas or cerebral abscesses	7	5.1%
Neurocysticercosis nodular granular or calcified nodular stages	6	4.4%
Hippocampic sclerosis at RMI	2	1.5%
Cerebral cavernoma at MRI	2	1.5%
Etiologies of epilepsies		
Structural causes identified	74	54%
• Sequelae of cranial trauma	15	10.9%
• Infections of CNS	23	16.8%
• Sequelae of stroke	16	11.7%
• Cerebral tumors	12	8.7%
• Sequelae of perinatal encephalopathy	4	3%
• Hippocampic sclerosis	2	1.5%
• Vascular cerebral malformations: cavernoma	2	1.5%
Causes Unknown causes or undetermined	63	46%

Table 1: The results of the neuroradiological examinations.

Discussion

The mean age of patients at inclusion in this study was 41.8 years, the average age of onset of first seizures of 34.4 years and age groups 15-45 years higher rate of epileptic patients. Our results are similar to those of other studies in sub-Saharan Africa [2,9,10]. However our results differ from those of European and US studies, where epilepsy has a bimodal distribution, marked by a high prevalence at both extremes of life, before the age of 5 years and after the age of 60 [11]. This difference could be explained by the demographic characteristics of the developing countries to which Burkina Faso belongs: high birth rates and low life expectancy, particularly the lower survival of elderly people with head trauma, stroke or neoplasm. The consequences are a significant proportion of people under 15 and a low representation of the over 50s (6.9% to 15.1% of the population) [12].

Almost all epilepsy studies around the world, including in developing countries [2,10,13,14] as in the industrialized countries [11], find a male predominance. The same observation was made in our study. This difference in frequency could be explained in sub-Saharan Africa by the social impact of epilepsy, since girls of marriageable age tend to hide their epilepsy. The hypothesis of an association between the masculine gender and the risk of occurrence of epilepsy also deserves to be explored [2]. A predominance of women has already been reported in Nigerian [2], Egyptian [15] and Ecuadorian studies [16]; the hypothesis of a rural exodus of men or that of a higher male mortality has been advanced to explain this finding. In our series we found a predominance of focal seizures in a proportion of 87.5%, just like the Senanayake et al. series in Nigeria [4], Kuaté et al. [14]. Our results are consistent with European studies of adults and the elderly, who experience focal seizures/epilepsies in 55-83% of cases, initially generalized seizures in 6-32% and seizures not classified in 8 at 20% [11]. These results, however, differ from most studies in sub-Saharan Africa, which report a predominance of generalized tonic-clonic seizures (on average $59\% \pm 21\%$) [2,10,17]. This can be explained by methodological differences such as the non-inclusion of genetic epilepsies in our study, by under-medicalization and more particularly by the insufficient number of neurologists and EEG devices that do not allow a relevant classification of epileptic seizures, in most studies in sub-Saharan Africa.

In our series, head trauma was the third leading cause of non-genetic epilepsy in adults, with a proportion of 10.9%, confirming the high frequency of this etiology in epilepsies in sub-Saharan Africa. Indeed, in most African series, an antecedent of head trauma was found on average in $8\% (\pm 5)$ of all epileptic patients versus 11.4% to 14.5% of epilepsies of structural or metabolic causes in China [18] and 5% of all epilepsies in industrialized countries (2-16% extremes) [11,17,19], an even lower rate of 1.7% reported in India [19]. Road accidents, which are common in Africa due to the lack of traffic regulation, the obsolescence of the road network, the absence of seatbelt use or the helmet for motorcyclists, are the main causes of head trauma. Head injuries can also be caused by workplace accidents, aggression or injuries during wars or violent sports. The high proportion of post-traumatic etiology in our series is due to the specificity of our study population, which focused only on non-genetic epilepsies in adults, whereas in most studies, the study population usually covers all epilepsies in children and adults, including presumed genetic epilepsies. Post-traumatic epilepsy results mainly from sequelae of brain contusions after a free interval usually less than 2 years. The risk of crisis increases with the severity of the trauma and the immediate brain lesions but also with the topography of the initial lesion: the sequelae of parietal contusions are the most epileptogenic [20]. The most severe sequelae of cerebral contusion appear on CT in the form of cerebral atrophy associated with hypodensity of the adjacent cerebral tissue. These lesions predominate in the frontal lobe; the rarer parieto-occipital localizations sometimes result in backache lesions. The use of MRI indicated in CT failures, allows a complete lesion balance thanks to the sequence FLAIR which visualizes the cortical atrophy and the adjacent parenchymatous lesions in the form of a hypersignal which translates a gliosis and a demyelination. The gradient echo T2 sequence identifies the hemorrhagic sequelae as cortical and sub-cortical hypo signal foci in the sub tentorial white matter and the corpus callosum [21]. In our series, all diagnoses of post-traumatic brain injury were confirmed by cerebral CT; no post traumatic cerebral lesion was supported by brain MRI, even with negativity of cerebral CT. This may have contributed to underestimating the proportion of this etiological category in our study.

In our series, brain tumors accounted for 8.7% of non-genetic epilepsy causes in adults, whereas this etiology averaged only $3\% (\pm 3)$ of all epilepsies in all age groups in sub-Saharan African series [2,14,17]. Methodological differences can explain this difference, especially study populations. The respective rates of 3.6% and 6.9% of all structural and metabolic epilepsies have been reported in India [20] and China [18]. In western countries, tumor etiology is found on average in 8% (10-20%) of all epilepsies in children and adults [6,11,22]. The under-medicalization, the low availability and sometimes the financial inaccessibility of cerebral CT exams and especially the unavailability of MRI machines in sub-Saharan Africa, examinations which are nevertheless essential for the diagnosis of brain tumors, can in fact skew the magnitude of brain tumors within epilepsy causes in this part of the world [23]. CT recognizes most epileptogenic tumors; only a few temporal tumors or isodense tumors escape this examination [22]. The sensitivity of MRI is close to 100%, but some glioblastomas may go undetected [24]. In our series, epileptogenic brain tumors were dominated by low grade astrocytomas and meningiomas. Indeed, the epileptogenicity of tumors depending on their natures: tumors of slow evolution, with little or no neurological signs of focusing during a long period of their evolution, such as low-grade gliomas, gangliogliomas, DNETs, meningiomas are epileptogenic in 60 to 85% of cases, compared with 20 to 40% for high-grade brain tumors and 15 to 20% for brain metastases [2,25,26]. Epileptogenicity also depends on the tumor localization: the cortical situations, the temporal cortical topographies or the primary sensorimotor cortex or the additional motor area, seem to be the most likely to give rise to epileptic seizures.

CNS infections were found in 16.8% of our patients and were the leading cause of non-genetic epilepsy in adults. They were divided into sequelae lesions and infectious lesions still evolving. The sequelae lesions of meningoencephalitis (8%) were found on cerebral CT in the form of localized cortico-subcortical atrophy, associated with non-contrasting adjacent parenchymal cerebral hypodensity, indicative of sequelae of gliosis and demyelination. The sequelae lesions of neurocysticercosis (4.4%) were visualized at the calcified and granular nodular stage. The sequelae of toxoplasmic abscess (4.4%) presented on CT as localized hypodensity without enhancement sometimes associated with calcification. With regard to progressive infectious lesions, they consisted mainly of pyogenic, tuberculous and toxoplasmic cerebral abscesses. Infectious aetiologies are suspected on average in $9\% (\pm 7\%)$ of cases in the tropics, possibly as much as 20% to 26% of all epilepsies usually reported in sub-Saharan Africa, from all causes [2,4,10]. The proportion of epilepsies from infectious causes in epilepsies from lesional causes was 25% in adults in a Chinese hospital series [18], whereas it was 17.4% in India [20]. These results are well above the average of 1.25% (0-2%) reported by the European series [11]. These differences could be explained by the fact that infectious diseases that can lead to epileptic manifestations are much more numerous in the tropics, due to the specificities of tropical ecosystems, the insufficiency, or the absence of the campaigns of vaccinations as well as delays or failure to take appropriate care [1,2,4,12]. Among these infectious causes, the most frequent are the meningo-encephalitis virales particularly measles and HIV, the meningo-encephalitis bacterial, in particular with meningococcus, with BK, including the intracranial tuberculomes [2,27]. As for parasitic infections, they seem to be dominated by neurocysticercosis reported in 5 to 50% of the causes of epilepsy in sub-Saharan Africa [1,2]; history of malarial infection with *Plasmodium falciparum* [28,29], toxoplasmosis, schistosomiasis, and toxocariasis [27]. The main predictors of post-infection CNS epileptogenicity are: the occurrence of epileptic status in the acute phase, the infectious agent, the severity of the cerebral parenchymal lesion [30].

In our study, stroke was the second leading cause of non-genetic epilepsy, at 11.7%. This result falls within a wide range of post-stroke epilepsy rates in sub-Saharan Africa, ranging from 1% to 42%, with an average of 7% ($\pm 10\%$). However, our results seem to be lower than those found in industrialized and emerging countries, where an average of 17.5% (14-21%) [11] and 26.4% [20] of epileptic patients, respectively, have a post stroke etiology. The methodological differences in the African series, some with mandatory neuroimaging, the others without neuroimaging based solely on interrogation and clinical examination, and finally the inclusion or not of genetic epilepsies, explain these wide variations observed. However, around the world, post-stroke epilepsy appears to be the preserve of the elderly, and stroke sequelae appear to be the leading cause of epilepsy after age 50 [2,20,31]. Perinatal causes accounted for 3.6% of cases in our study, a proportion lower than the $9\% \pm 8\%$ average reported in the African literature [2]; this difference could be explained by methodological differences, because for most African series, the diagnosis of perinatal causes is often based on the only interrogation of the patient, or possibly of the entourage, and thus subject to memorization biases. But it is also possible that some patients, particularly those with severe disease, did not survive to adulthood. In our series, in addition to a history of perinatal encephalopathy, patients had to have compatible brain imaging. Rates of 8.9% were found in India [20]. Perinatal causes are probably major in Africa because of numerous home births, without skilled help, or difficult deliveries in under-equipped environments [32], causing frequent obstetric trauma, anoxia or cerebral ischemia, aggravated by associated factors such as multiparity, prematurity, anemia, malnutrition, maternal infections [4]. Sequelae of hypoxia-neonatal anoxia are responsible for leukoencephalomalacia and cortical atrophy lesions; clinically mental retardation is associated with epilepsy in 50%. Sequelae of neonatal hypoglycaemia are responsible for bilateral occipital cortical atrophy associated with sub-cortical white matter hypodense and posterior corpus callosal atrophy. Sequelae of stroke or antenatal infection result in porencephalic cavities. Infant cerebral hemiplegia results in a cortico-subcortical cerebral hemisphere associated with ipsilateral thickening of the cranial vault. Such lesions were found in our study.

Epileptogenic cerebral vascular malformations are represented by 1/3 of arteriovenous malformations, 45% of cavernous angiomas and giant arterial aneurysms compressing the cerebral parenchyma [22]. In our series, we identified 2 cases of cavernous angioma, within epileptogenic cerebral vascular malformations. In our study, hippocampal sclerosis accounted for 1.5% of the causes of non-genetic epilepsy in adults, while it accounts for up to 65% of cases in surgical series [22,26]. This is due to the low use of MRI (11%) by our patients, the only exam able to diagnose this lesion. Some causes of epilepsy reported in the literature, especially Western, have not been found in our study; these are: alcoholic epilepsy sometimes representing up to 12 to 25% of the lesional or metabolic causes of epilepsies [33] and found in 1 to 7% (5% on average) of epileptic patients in sub-Saharan Africa [2], neurodegenerative diseases dominated in particular Alzheimer's disease and vascular dementia, described in 1 to 5%, systemic diseases such as systemic lupus erythematosus and vasculitis of the CNS, mitochondrial diseases. This could be explained by differences socio-cultural, low life expectancy in our context, the inaccessibility and/or unavailability of some methods of neuroradiological explorations such as MRI, the exams of autoimmunity. Epilepsies from unknown causes were found in 46% of patients in our series, whereas in most series in sub-Saharan Africa this category accounted for more than 50% of investigations (on average $50\% \pm 26$), especially for series whose etiological diagnosis was based solely on interrogation and clinical examination. This proportion decreased as

a function of the neuroradiological explorations carried out, CT and/or encephalic MRI [2,14,17]. In this type of epilepsy, a cause is presumed, but the current diagnostic means do not make it possible to highlight it [2]. Thus in developed countries where sophisticated neuroradiological explorations (spiral CT, high resolution morphological MRI combined with metabolic and functional techniques, PET) are available and accessible to all epileptics, the rate of unknown causes falls to about 20% of the all epilepsies of the adult and the child [34,35].

Limits and peculiarities of our study

Unlike most studies in sub-Saharan Africa, our study was limited to adult patients over the age of 18 years, with epilepsy of non-genetic cause, excluding the children and adolescent's population and the epilepsy population presumed genetic cause. Some causes of epilepsy, including perinatal events, some CNS infections, some head trauma, found during the interrogation in the antecedents, could not finally be retained, because of a lack of obvious lesion on cerebral CT, helping to overestimate the proportion of unknown causes.

The low achievement rate of brain MRI yet recognized as gold standard for the etiological diagnosis of structural cause epilepsy, probably contributed to overestimate the proportion of unknown causes and underestimated structural causes. The metabolic causes were not sought in our study, because of the additional cost that their explorations would weigh on our epileptic patients, often deprived.

Conclusion

Non-genetic epilepsies in adults in Ouagadougou (Burkina Faso) classified etiologically according to clinical, EEG and neuroradiological criteria. All patients underwent clinical examination, standard EEG, brain CT, but only 11.5% were able to perform brain MRI. No patient was able to perform specialized biological examinations; the metabolic causes could not therefore be sought. The structural causes and unknown causes were found respectively in 54% and in 46% of cases. CNS infections (16.8%), sequelae of stroke (11.7%), sequelae of cranioencephalic trauma (10.9%), brain tumors (8.7%), sequelae of perinatal encephalopathy (4%) and cerebral vascular malformations (cavernoma) (1.5%) were the epileptogenic structural abnormalities found. Our results confirm the predominance of infectious and post-traumatic causes and the emergence of cerebrovascular causes in sub-Saharan Africa. Certain epileptogenic lesions, such as certain brain tumors, focal cortical dysplasias, hippocampal sclerosis, etc., have been underdiagnosed because of the poor availability and accessibility of cerebral MRI. Other causes such as alcoholic epilepsy, neurodegenerative diseases, systemic diseases, mitochondrial diseases, have probably not been diagnosed because of the unavailability of some expensive biological explorations such as the autoimmunity balance. Increased availability and accessibility of neuroimaging examinations, particularly brain MRI and some sophisticated biological investigations, would contribute to a better etiological diagnosis of non-genetic epilepsy in adults.

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