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Chapter

Pediatric Epilepsy in West Africa: Prevalence, Causes, and Management

Rhoda Olowe Taiwo and Tawfeeq Shekh-Ahmad

Abstract

Epilepsy is a neurological disorder affecting over 50 million people worldwide. Global epilepsy prevalence has been reported to be the greatest in Africa, prevalent among children living in resource-poor areas compared with all other continents. In West Africa, a meta-analysis of epilepsy prevalence was quoted to be 13–15 per 1000 persons. As a result of the lack of specialists and electroencephalographic facilities, the type of seizures that are more likely reported in rural areas is generalized tonic-clonic seizures. A high prevalence of epilepsy in low- and middle-income countries has been identified with CNS infections due to viral, bacterial, and parasitic infections. Parasitic infections including malaria, onchocerciasis, cysticercosis, and toxocariasis are believed to account for up to 27% of pediatric epilepsy cases reported in Sub-Saharan Africa, of which onchocerciasis has been more documented as a parasitic cause of epilepsy in most of west Africa. The management of epilepsy in West Africa centers around the administration of anti-seizure medications when available, and an onchocerciasis control program that has reduced onchocerciasis-associated epilepsy in these countries. However, several management options put in place still seem insufficient to curb the disease prevalence, hence improved strategy for effective control of parasite-induced epilepsy in West Africa.

Keywords: epilepsy, pediatric, West Africa, prevalence, parasites, general tonic-clonic seizures

1. Introduction

Epilepsy is a neurological condition affecting over 50 million people worldwide. It is characterized by recurrent seizures, following abnormal or excessive neuronal activities in the brain [1], representing a considerable healthcare burden with greater drug resistance and worse clinical outcomes than many other neurological diseases [2, 3]. It remains a global burden that knows no geographic, regional, or racial boundaries, occurring in men and women and affecting people of all ages, though more frequently affecting young people in the first two decades of life [4]. The prevalence of epilepsy has been reported to be greatest in Africa accounting for 37% of the global

epilepsy burden compared to all other continents, as it has been acknowledged that more than 80% of people with epilepsy live in developing countries [5]. Typically, in Africa, the majority of people with epilepsy suffer from the disease from childhood, usually during their first few years of life. Mounting evidence from across five sites in Africa, suggested that over 60% of people with active convulsive seizures experience their first recorded seizure before age 13 [4, 6]. As a result of a lack of specialists, neurologists and electroencephalographic facilities, the type of seizures that are more likely reported in rural areas of Africa is the generalized tonic-clonic seizures (averaging 67% of individuals), because of the conspicuous presentation manifested, while partial seizures (averaging 8%) are most likely underestimated giving that their early stages recognitions are clinically difficult [7]. Although effective antiepileptic drugs are available, however, a substantial treatment gap is more evident in developing countries, mainly as a result of limited human and financial resources for diagnosis and treatment, cumulating in inadequate treatment in many cases [5]. Similarly, about 60% of patients with epilepsy receive no antiepileptic treatment, also for economic and social reasons [8]. Unfortunately, it is common that people with epilepsy left untreated to be faced with devastating social consequences, including stigma, discrimination, premature mortality, and reduced life chances for adults in terms of employment and marriage [9]. Here, we report the possible causes, prevalence, and management of pediatric epilepsy in West Africa.

2. Causes

Numerous causes have been attributed to the development of epilepsy in Africa and therefore it has been perceived that the origin of epilepsy is considered multidimensional in nature [10]. In a couple of decades ago, early knowledge of the etiology of epilepsy in most African countries has long been attributed to beliefs and spiritual reasons including spiritual attack, witchcraft, and other supernatural causes [11]. Moreover, as epidemiological studies in Africa progressed, knowledge of epilepsy prevalence begins to override beliefs, norms, and notions about epilepsy disease, highlighting other causes that might be contributory to the cause of pediatric epilepsy including obstetric injuries, frequent febrile convulsions, head trauma, and meningitis [10]. However, these aforementioned causes are only identified in less than 1% of epilepsy cases. In high-income countries, the main causes of epilepsy have been attributed to traumatic head/brain injuries and strokes [12, 13]. Whereas the high prevalence of epilepsy in low- and middle-income countries has been majorly identified with central nervous system (CNS) infections due to viral, bacterial, and parasitic infections [14].

Moreover, in recent times, the association between several parasitic diseases and epilepsy has gained new insights. Parasitic infection which remains the commonest cause of central nervous system infection and cause of symptomatic epilepsy; is believed to account for up to 27% of pediatric epilepsy cases reported in sub-saharan Africa, inclusive of West Africa [15]. Thus, updating an understanding of the etiology and epidemiology of epilepsy in West Africa. In addition, Africa has the highest burden of parasitic infections, which are associated with the development of epilepsy [16]. Among these parasitic infections are malaria, onchocerciasis, cysticercosis, and toxocariasis, infections with these organisms have been associated with epilepsy and are ubiquitous in Africa [15, 17–19].

2.1 Malaria

Malaria is one of the tropical parasitic diseases commonly thought to have a role in the development of epilepsy [20]. Majorly, most acute seizures are suggested to be caused by the malaria parasite *Plasmodium falciparum* (*P. falciparum*), usually in malaria-endemic areas, and these parasites are common in sub-Saharan Africa among people with epilepsy.

2.1.1 The cell cycle of malaria

Malaria parasites are mainly transmitted by a female anopheles mosquito. The parasite life cycle consists of a vector and human exoerythrocytic (hepatic) and erythrocytic stages [21]. The female mosquito (vector) bites the host during a blood meal, injecting mature sporozoites from its salivary glands into the host's bloodstream. These rapidly enter the liver hepatocytes and begin tissue schizogony-like asexual reproduction and proliferation (exoerythrocytic stage). Thereafter, thousands of merozoites are released into the bloodstream as the tissue schizonts burst the infected hepatocytes. The merozoites invade the erythrocytes, go through several asexual multiplication cycles (the erythrocytic stage), and then create new infectious merozoites that burst the erythrocytes to start a new infective cycle. In the long run, the parasite invades the CNS and causes some disruption of the BBB function, leading to cerebral malaria [22, 23].

Cerebral malaria is the most serious neurological complication of Plasmodium falciparum infection. In most infected children, after 1–3 days of fever, coma develops rapidly and seizures set in [24]. A defining feature of cerebral malaria is the Intravascular sequestration of circulating parasitized erythrocytes in the cerebral microcirculation.

Sequestration is a result of cytoadherence of infected erythrocytes to the vascular endothelial cells through parasite-derived proteins on the surfaces of the infected erythrocytes for example, the Plasmodium falciparum erythrocyte membrane protein-1 (PfEMP1) [25]. These are considered important pathophysiological mechanisms underlining cerebral malaria.

Furthermore, malaria can also cause different forms of seizures in the CNS, and it has already been identified as the most common cause of febrile seizures [26]. Agbéré et al., [27], reported that most febrile seizure cases occur in 55% of patients with malaria fever in Togo. Although the role of malaria in long-term epilepsy is still unclear, Preliminary studies have attributed a high risk of epilepsy to cerebral malaria, as seizures are one of the hallmarks of the clinical presentation of cerebral malaria [28]. An earlier study reporting an association between falciparum malaria and epilepsy found a 9% (4·4, 1·4–13·7) epilepsy occurrence in children exposed to cerebral malaria and 12% (6·1, 2·0–18·3) in those exposed to malaria and complex seizures [19]. However, whether they are febrile or acute symptomatic seizures, remains unclear [25, 29, 30].

2.2 Onchocerciasis

Human Onchocerciasis also referred to as "river blindness" caused by the filarial nematode; *Onchocerca volvulus* (*O. volvulus*), is a chronic parasitic infection transmitted by bites of blackflies [31]. Onchocerciasis is known as a cause of skin and eye

disease infecting a great number of people and has also been implicated in seizure disorders mainly in rural Africa. It is estimated that of the 120 million infected, 99% of them live in Africa [32].

2.2.1 The cell cycle of onchocerciasis

Several mechanisms have been proposed for the pathogenesis of onchocerciasis-related epilepsy. However, one of the most identified mechanisms is the *O. volvulus*-induced immune response via an inflammatory process or autoantibodies against neuron surface proteins such as the voltage-gated potassium channel complex (VGKC), the N-methyl-D-aspartate (NMDA), a-amino-3-hydroxy-5-methyl-4-isox-azole propionic acid (AMPA), gamma-aminobutyric acid (GABA)A, GABAB, and glycine receptors which are recognized causes of severe epileptic disorders [33].

Similarly, *Wolbachia* which are intracytoplasmic symbiotic bacteria found in filarial worms' endosymbiont of *O. volvulus* has also been proposed as a possible cause of onchocerciasis-related epilepsy. *O. volvulus* endemic areas, is a neuroinflammatory disorder caused by antibodies to either *O. volvulus* or its co-symbiotic bacteria, Wolbachia, cross-reacting with host neuron surface proteins, although evidence for neuroinflammation has not been established [34].

Consistent reports from epidemiological studies suggest that *O. volvulus* infection is a trigger and risk factor for epilepsy in Onchocerca endemic areas [35]. In Africa, onchocerciasis-associated epilepsy (OAE) was first documented in a population-based epidemiological study in the Mbam valley in Cameroon between 1991 and 1992, where high epilepsy prevalence was associated with communities near Mbam River, a breeding site for black flies [36].

In addition, several studies have reported an association between the prevalence of onchocerciasis and epilepsy in different areas of east, west, and central Africa [36–38]. In West Africa, epidemiological studies have identified a high prevalence of epilepsy to a high onchocerciasis endemicity, where most infected patients experience generalized tonic-clonic seizures and the risk for children developing epilepsy has been determined by the *O. volvulus* microfilariae load [35, 39]. Although a paucity of information has been reported on *O. volvulus* penetration to the CNS, nonetheless, the presence of microfilariae from various filarial species in the human CNS has been reported. Studies suggest that since antifilariae drugs can provoke the passage of Loa Loa microfilariae (responsible for spontaneous encephalitis) into the cerebrospinal fluid (CSF) [40], therefore it does seem that *O. volvulus* would be able to penetrate the CNS since microfilariae belonging to this species have been found in the CSF of Onchocerciasis patients with or without filaricidal drugs [41, 42]. Furthermore, another explanation that *O. volvulus* could directly or indirectly cause epilepsy could be the occurrence of antibody-mediated autoinflammatory response against *O. volvulus*, cross-reacting with neuronal proteins [43] (**Figure 1**), as antibodies to voltage-gated potassium channels in neurons have recently been observed [44]. However, irrespective of this attribute, the pathophysiological mechanisms involved in onchocerciasis associated with epilepsy for seizure induction still need to be further elucidated.

2.3 Neurocysticercosis

Neurocysticercosis (Cysticercosis), remains the commonest helminthic infection of the CNS, representing an important cause of secondary epilepsy globally
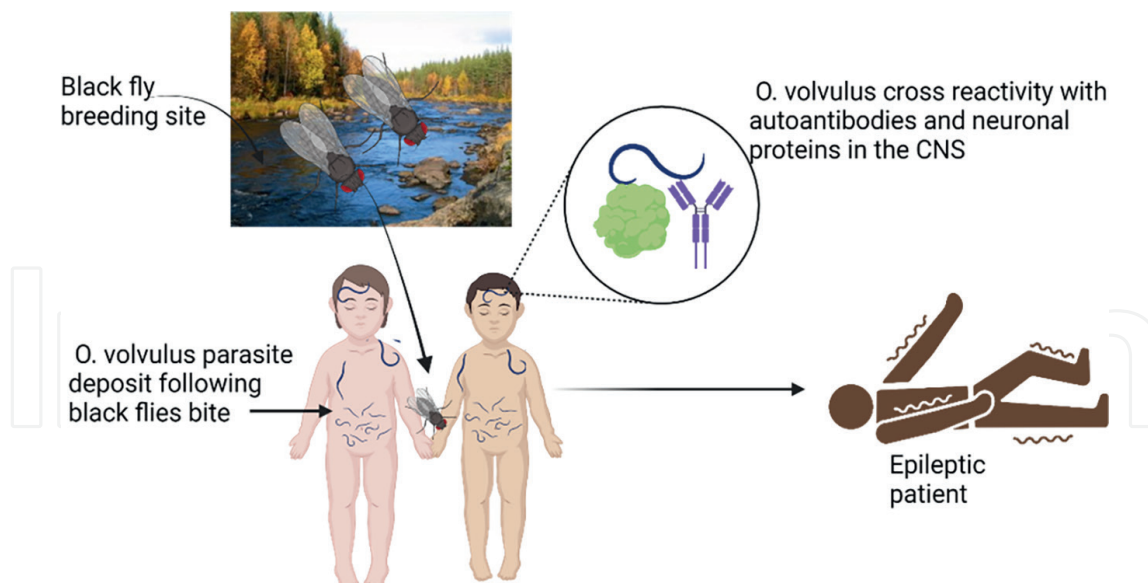
Figure 1.
The postulated scenario of infection or events of onchocerciasis associated epilepsy (AOE).

[45, 46]. It is a cause of epilepsy in some parts of the world, and the introduction of computed tomography (CT) in the 1970s and early 1980s has allowed for its detection [47]. Cysticercosis is regarded as a Zoonotic disease, commonly caused by the larval stage (cysticercus) of the porcine tapeworm *Taenia Solium* (*T. solium*) and is said to be mainly found in the CNS including the Brain and spines. Reports of the parasite development in the brain, suggest an appearance of immature cysticerci in the brain within some weeks after ingestion of *T. Solium* eggs [48, 49].

2.3.1 The cell cycle of neurocysticercosis

In a typical life cycle of *T.* solium, following ingestion of the parasite, the adult worm is found in the small intestine of humans (the sole definite host of the parasite). The worms produce per day tens of thousands of oncospheres (eggs), They are then eliminated via feces, contaminating the food chain and water systems. Pigs (intermediate hosts of parasites) consume these oncospheres, where they are activated by bile salts and digestive enzymes in the small intestine. They subsequently go into the blood supply through the gut wall at blood vessel terminations in a variety of tissues (such as muscle, subcutaneous tissue, or nerve tissue) [50]. When a human consumes improperly cooked pork that contains a cysticercus, the scolex evaginates in the small intestine and clings to the intestinal wall where it develops into an adult worm.

However, when a person accidentally becomes an intermediate host and consumes oncospheres via tainted food or water, the oncospheres are thereafter activated in the human digestive tract just as they are in the pig, allowing them to pass into the bloodstream. They embed themselves in a variety of tissues, particularly the central nervous system. Taenia *solium* cysticerci invade and infect the brain parenchyma, of which seizures are the most common manifestation of parenchyma disease [51]. When cysticerci are present in the nervous system this is referred to as neurocysticercosis (NCC).

Although this parasite is mainly reported to cause 20–50% of all late-onset cases of epilepsy globally. Moreover, in a certain part of the world, it is assumed to be a common cause of juvenile epilepsy, majorly in the southern part of Africa [52, 53].

Nonetheless, this infection has been under-recognized in many developing countries, certainly true for Africa and more specifically West Africa, where this infection has been only reported in a few countries [47]. For instance, it is rarely reported in Nigeria unlike her neighboring countries, Cameroun, Benin, and Togo, where cysticercosis-associated epilepsy showed a significant proportion of epilepsy cases [54]. In an epidemiological study in Togo, cysticercosis prevalence was 135% among people with epilepsy (PWE) compared to 38% of the general population [55]. However, the high prevalence of neurocysticercosis is streamed lined to the rural area where a high consumption of pork infected with *T. Solium* predisposes people to epilepsy, resulting in one of the main causes of acquired epilepsy in middle-income countries around sub-Saharan Africa, inclusive of West Africa [45].

2.4 Toxocariasis

Human toxocariasis is another most prevalent helminthiasis also regarded as a parasitic zoonosis caused by the larval stages of dogs roundworm; *Toxocara canis* (*T. canis*), and by roundworm of cats; *Toxocara cati* (*T. cati*) [56]. Infections are mainly established through direct contact with the animals or via ingestion of contaminated food. Upon ingestion, eggs metamorphose into juvenile larvae capable of crossing the small intestine into the systemic circulation and then migrate to organs particularly muscles, and at times to the CNS where they invoke multisystemic inflammatory reactions [57].

2.4.1 The cell cycle of toxocariasis

The gastrointestinal ascarid nematodes Toxocara spp., including *T.* canis and *T. cati*, are present in canids (definitive hosts), such as dogs, felids (domestic cats), as well as foxes, and jackals. They can also infect people (considered paratenic hosts) through direct contact with diseased cats and dogs as well as contaminated food, soil, and water, of which humans tend to unintentionally consume eggs containing infectious third-stage larvae [58, 59].

Eggs that have been consumed grow and hatch into larvae in the small intestine, pierce the intestinal wall, and move through the circulatory system to various tissues. This causes an immune and inflammatory tissue reaction, which can cause symptoms like fever, headaches, coughing, and abdominal or limb pains [59].

Neurotoxocariasis, also known as cerebral toxocariasis, resulting from toxocara larvae invades the brain. In the brain, the larvae can also induce epileptic convulsions, cerebral vasculitis, and eosinophilic meningitis. Similarly, reports from experimental animal models showed that the presence of Toxocara larvae in the brain leads to neuronal damage via an increase in BBB permeability, expression of proinflammatory cytokines, iNOS, and astrogliosis [60, 61]. In addition, there have also been reports of changes in the profiles of neurotransmitters such as GABA, glutamate, serotonin, dopamine, and noradrenaline [62].

In nutshell, Toxocara larvae can invade the CNS by crossing the blood-brain barrier resulting in neurotoxocariasis [63]. Seroprevalence of the parasite tends to reach up to 80−90% in tropical regions, including West Africa while seroprevalence in western countries ranges from 35 to 42% and 2 to 5% in rural and urban areas, respectively [60]. Early epidemiological studies have provided evidence of a positive association between Toxocara seropositivity and seizure in children. Arpino et al., [64], evaluated the relationship between toxocariasis infection and epilepsy

in children and reported that a significant (p < 0.05) association existed between seropositivity for anti-Toxocara canis and seizures and this correlation was closest in children below age five. Furthermore, an investigation on the possible association between epilepsy and toxocariasis in the kiremba population of Burundi revealed that out of the studied 191 PWE, antibodies of anti-*T. canis* were found in 114 PWE (59.7%) and multivariate analysis showed a significant association between positivity for *T.* canis and epilepsy [65]. Although, some studies have proven an association. However, reports from two other studies revealed a non-significant association between epilepsy and seropositivity (antitoxocaral antibodies) [66, 67], adding up a piece of conflicting evidence that is difficult to interpret. To gain more insights into the association of this parasite with epilepsy, further studies are necessary to clarify its potential role and activity in epilepsy.

3. Prevalence

Reports have shown that the prevalence of epilepsy is higher in less developed than in more developed countries, with prevalence estimated from door-to-door studies almost double that in Asia, Europe, and North America [45]. More precisely, in West Africa, a meta-analysis of epilepsy prevalence was quoted to be between 13.14 and 15 per 1000 persons. However, this incidence has been more prevalent among children living in resource-poor (rural) areas than in urban areas [31]. The region-specific prevalence of epilepsy has yielded a treatment gap, resulting from inadequate access to physicians trained to manage epilepsy, a lack of access to ASM, poor knowledge about epilepsy among the communities, as well as the stigma of epilepsy arising from misconceptions about; epilepsy having a supernatural origin and attributed to possession by ancestral spirits [68]. In addition, the stigma of epilepsy can be profound because it is widely thought to be contagious and associated with witchcraft [69]. This stigma surrounding the disease creates psychosocial impacts on the children, marginalizing their ability to participate in community/societal activities leading to a significant effect on their quality of life [70].

Although a paucity of information has been gathered on epilepsy prevalence in West Africa, nonetheless several studies have reported a higher occurrence of epilepsy in onchocerciasis-endemic sites across countries in West Africa. In Nigeria, epilepsy prevalence based on defined communities varies between 15 and 37 per 1000 [31, 71]. An early epidemiological survey investigating the possible relationship of epilepsy with onchocerciasis in towns and villages in Nigeria observed a 37% epilepsy prevalence in Aiyete; a rural onchocerciasis-endemic village [72] while in Igbo-Ora; a town inhabited by the same ethnic group as Aiyete, situated 20 km away recorded 5.3% epilepsy prevalence, in the age group between 10 and 19 years [72, 73]. The lower prevalence recorded in Igbo-Ora compared to Aiyete may be due to the availability of comprehensive health facilities, effective primary health care with greater emphasis on antenatal care, improved prevention of childhood infectious diseases, and a working health education system [72]. Moreover, Aiyete is said to be located closer to the Ofiki River, a known breeding site for blackflies, causing this village to pose a higher endemicity than Igbo-Ora [74]. Furthermore, in a study carried out on 13 villages in the Imo River basin; a southeastern part of Nigeria that is mesoendemic for onchocerciasis, reports revealed a 12% epilepsy prevalence in these villages with a higher prevalence of onchocerciasis ranging between 8.3 and 36% and higher microfilariae density [39].

Furthermore, epilepsy prevalence reports in Benin; a country bordering Nigeria to the west revealed that 9/13 (69.2%) PWE in the Agbome area were associated with O. volvulus microfilariae which were detected in skin snips while 8 PWE (61.5%) presented with onchocerciasis nodules. In addition, the majority of these patients (76.9%) manifested generalized tonic-clonic seizures [75].

In Ghana, albeit a community-based survey of kintampo village, revealed a lower epilepsy prevalence of as low as 1.92%, the epilepsy cases were attributed to exposure to O. volvulus and were identified as an independent risk factor for epilepsy in children below 18 years [76].

Furthermore, in Mali, epidemiological studies conducted on the endemicity of onchocerciasis about epilepsy prevalence in 18 villages showed an epilepsy prevalence of 16.1 per 1000 in zones of high endemicity of onchocerciasis (23.0%) compared to a prevalence of 10.8 per 1000 in zones of low endemicity (9.3%). Although a non-significant difference was reported between these two zones, however, higher morbidity rates were reported in zones of high endemicity of onchocerciasis [77].

In Cameroon, an outcome of a clinical epilepsy prevalence survey of 156 PWE in selected 5 onchocerciasis-endemic villages, revealed that onchocerciasis-associated epilepsy (OAE) showed a high prevalence in epilepsy patients where 93.2% of PWE met the criteria for OAE, presenting with the most frequent seizure types being generalized tonic-clonic episodes, absences, and nodding seizures. More so, the majority of epilepsies started between the ages of 3 and 18 years [78].

In epilepsy prevalence studies in Liberia, two studies documented a high epilepsy prevalence of 27.73–49.01% in Grad Bassa, which was agreed that the prevalence was in an onchocerciasis endemic area [79, 80].

In Ivory Coast, an epidemiological survey carried out in a village less than 10 km from M'Brou, where a high burden of epilepsy had previously been reported for decades with a microfilariae prevalence of 76%, revealed an epilepsy prevalence of 41% (38 of 920) and 73.7% fulfilled the OAE criteria [81].

In Burkina Faso, a prevalence study of epilepsy in three villages has specifically identified epilepsy prevalence with a positive reaction to cysticercosis Ag-ELISA serology with an association with past pork consumption. The findings reported that 39 of 70 that screened positive for cysticercosis were confirmed to have epilepsy, suggesting the presence of neurocysticercosis as the cause of epilepsy. Collectively, a total of 70 (7.9%) of 888 persons interviewed for epilepsy or seizure screened positive and 29 (74.4%) were reported to have experienced generalized seizures [82].

Epilepsy prevalence in almost all study sites in Togo was reported to be below 20%, however, these epilepsies were said to be associated with cysticercosis, where its prevalence was 135% among PWE compared to 38% of the whole population [83]. In addition, an epilepsy management study in the Batamariba district of Togo revealed an epilepsy prevalence estimation of 15.7% of 98 patients, where the etiologies of 14/98 PWE (14.3%) were confirmed to be associated with neurocysticercosis (subcutaneous cysticercus cyst) [55]. However, in rural Gambia, a lower active epilepsy prevalence of 4.3/1000 was reported which is non-endemic for neither Neurocysticercosis nor onchocerciasis. In patients whose etiology could be suggested, etiology was only attributed to febrile illness in childhood and antenatal or perinatal brain insult [84].

Similarly, in Senegal, in the early 60s, a lower epilepsy prevalence of 1.92% was reported in an area non-endemic for onchocerciasis located in the Moyenne Vallée of Senegal. However, a more recent study from Pikine Health District of Senegal reported an epilepsy prevalence of 14.22% also in a non-endemic area [85]. The

Country	Towns/Villages	Causative parasite	Prevalence	Reference
Nigeria	Ayete	Onchocerciasis	37%	[72]
Nigeria	Igbo ora	Onchocerciasis	5.9%	[72, 73]
Nigeria	Imo	Onchocerciasis	12%	[39]
Benin	Agbome	Onchocerciasis	69.2%	[75]
Ghana	Kintampo	Onchocerciasis	1.92%	[76]
Mali	18 pooled villages	Onchocerciasis	16.1%	[77]
Liberia	Grad Bassa	Onchocerciasis	27.73–49.01%	[79, 80]
Cameroon	5 pooled villages	Onchocerciasis	93.2%	[78]
Ivory Coast	M'brou	Onchocerciasis	41%	[81]
Burkinafaso	3 pooled villages	Neurocysticercosis	7.9%	[82]
Togo	Batamariba	Neurocycticercosis	15.7%	[55]
The Gambia	Farafenni	Febrile Seizure	<1%	[84]
Senegal	Moyenne valle	NA	1.92%	[85]
Senegal	Pikine	NA	14.2%	[85]
NA-Not applicable.				

Table 1.
Summary of identified prevalence and cause of epilepsy in west African countries.

majority of these findings strongly incriminate onchocerciasis as an important yet neglected contributor to the epilepsy burden in West Africa (**Table 1**).

4. Management

Management of Epilepsy in West Africa centers around the administration of ASM to control seizures as epilepsy surgery is rarely available in most parts of Africa [86]. However, in rural areas, a wide treatment gap is usually reported, and a systemic review has identified several reasons for this treatment gap including scarcity of knowledgeable staff, lack of investigative resources to ensure a diagnosis, cost of treatment, far-flung distance to health care facilities, cultural beliefs and inadequate availability of anti-seizure medications [87]. The presence of treatment gaps in West Africa has accounted for a high mortality rate related to poorly controlled epilepsy, with a greater proportion of children dying from status epilepticus compared to other continents [88]. In addition, in a systematic review, the World Health Organization (WHO) reported a greater treatment gap of 95% in some African countries including Ethiopia, Nigeria, Togo, Uganda, Tanzania, Gambia, and Zambia [89]. In most developing countries, available medications in the treatment and management of partial seizures include but are not limited to Carbamazepine, Phenobarbital, and Phenytoin, while drugs of choice for patients with tonic-clonic seizures are Valproic acid and Phenobarbital [90, 91]. However, Phenobarbital is the single most commonly prescribed anti-seizure medication for reasons due to availability, economic considerations, and its effectiveness in reducing the frequency of partial and generalized tonic-clonic seizures [92]. Moreover, the WHO has recommended Phenobarbital as the first-line therapy for convulsive epilepsy in Africa since it's the most readily

available and cost-effective ASM for seizure management [93]. Furthermore, another management practice particularly influenced by the introduction of the vector control program, that has been deployed in West Africa is the OCP (1974–2002), executed through a largescale onchocerciasis elimination control measure, followed by annual microfilaricidal treatment with Ivermectin distribution [94]. These control measures have significantly reduced OAE and drastically decrease the incidence of epilepsy in 11 countries in West Africa [95, 96]. However, despite the documented success, several challenges remain unmet, including banishing the stigma associated with epilepsy and dealing with cultural and social beliefs culminating in poor help-seeking behaviors among family and community of epilepsy patients. Ensuring easy access to anti-seizure medications at a cheaper or subsidized cost; Improving sensitization on relying on the primary health care system for treatments in rural areas.

5. Discussion

In most developing countries like West Africa, epilepsy disease remains prevalent in the younger population of resource-deprived communities. The majority of epidemiological studies indicate that the age of epilepsy onset is younger in under-developed countries than in areas with greater access to resources [97]. The survey methodology commonly employed in population studies of epilepsy is the door-to-door epidemiological survey, where a questionnaire is provided to screen members of a household with epilepsy [98].

However, this survey method poses a likely problem of underestimation as the screening tends to identify majorly individuals with generalized tonic-clonic seizure, and under-recognition is given to individuals with partial complex, dyscognitive, or myoclonic seizures, thereby underestimating the prevalence of the disease [99]. Additionally, people who have children with epilepsy tend not to declare the condition in a bid to avoid epilepsy-associated stigmas. The stigma attached to epilepsy may make people reluctant to acknowledge that someone in their home has the illness, thus resulting in the unavailability of epidemiological data [45].

Although, about three decades ago, epilepsy patients are rarely stigmatized because the condition is believed to be of demonic origin, attributed as being possessed and it is only combated with sorcery and traditional medicines with no seeming solutions [72, 73]. Just in recent times, following community sensitization about the disease condition and communities were enlightened on the disease being of medical origin, identified as epilepsy, bringing about the stigmatization of the disease. It is commonly established that epilepsy stigma has an impact on many aspects of its victim's life, such as social isolation, low self-esteem, worse psychological function, and increased future uncertainty [100]. Nonetheless, a well establishes effective epilepsy enlightenment campaign, as well as epilepsy education, could help patients and their families have comprehensive information on epilepsy disorder, avoid misconceptions and overcome the challenge of stigma in the resource-poor areas where epilepsy prevalence is pronounced [101].

Moreover, it is known that 80% of people with epilepsy reside in resource-poor, developing countries, however, a huge treatment gap exists, where epilepsy care in these regions remains limited and a majority of epilepsy patients go untreated [8]. Treatment gaps resulting from weak healthcare structures, inadequate financial resources, unreliable supply and quality of pharmaceuticals, the high rate of inappropriate self-medication, unavailability of ASM, inadequate ASM storage conditions are

likely to lead to ineffective and possibly dangerous ASMs [102], and even when good-quality ASMs are initially imported. Highlighting the difficulties with antiepileptics and, indeed, all drug use in developing countries [103].

To overcome these challenges, sustainable epilepsy care services that can deliver first-line antiepileptic drugs through established primary healthcare facilities, are needed to reduce these treatment gaps. Furthermore, Neurologists with knowledge of the culture and local experiences, who are willing to serve as advocates, educators, and policy advisors, can help make a difference. Nonetheless, a more sophisticated approach to overcoming the hurdles of epilepsy treatment in resource-poor nations is the availability of expertise for reliable EEG video interpretation of different seizure types for effective treatment and the establishment of a sustainable epilepsy monitor program.

6. Conclusions

Epilepsy in West Africa remains an undebatable challenge majorly in children in their first few years of life. Knowing fully that prevalence studies are a prerequisite for successful intervention. The primary purpose of this report was to assess the prevalence, causes, and management of pediatric epilepsy in West African countries at a community level and to bring to the limelight the available knowledge with previously existing data on this subject that dates way back to over 3 decades ago. We have recalled a parasitic cause of epilepsy that has been the main concern in tropical countries like West Africa. Parasitic infections including malaria, onchocerciasis, cysticercosis, and toxocariasis remain a common cause of CNS infection and cause of symptomatic epilepsy; where onchocerciasis is believed to account for up to 27% of pediatric epilepsy cases. These findings strongly incriminate onchocerciasis as an important yet neglected contributor to epilepsy in West Africa. Parasite-induced epilepsy remains a burden in many parts of West Africa, where generalized tonic-clonic and partial seizures were the predominant seizure types that have been reported. So far, the main achievements in dealing with epilepsy in West Africa involve the administration of available ASM such as Carbamazepine, Phenobarbital, Valproic acid, and Phenytoin, albeit a wide treatment gap is usually reported. Currently, resource-poor communities in low-income nations are unable to fund desirable but expensive initiatives like specialized epilepsy centers, which would appear to be the best method to assist epileptic patients in poor nations. Nonetheless, subsidized antiseizure medications could be provided to community health care centers so as to be at the disposal of the patients. More so, a national organization that is associated with international organizations for epilepsy could solicit and receive support and encouragement from international bodies like the International Bureau of Epilepsy. In addition, the introduction of the onchocerciasis control program has been deployed in West Africa as a parasitic control measure for onchocerciasis-induced seizures.

However, pediatric epilepsy in West Africa has long been underestimated as a paucity of information has only been gathered so far on its prevalence, causes, and management. It could be a result of the lower number of epidemiological studies carried out, arising from the stigma attached to epilepsy, as some people still hide the diagnosis; hence contributing to the underestimated prevalence of the disease. To overcome this challenge, education relevant to epilepsy health education such as patient-centered care allows individuals with epilepsy and their families to access usable information including Knowledge of epilepsy in general, an explanation of what happens during a seizure, and the importance of getting medical help, the significance of trigger factors that may

induce a seizure, a guide to ASMs; how they work, their side effects and the importance of compliance, orientations on improved quality of life, managing lifestyle and wellness should be encouraged at all levels to debunk wrong notions and myths about epilepsy as well to control the stigmatization of affected people. Furthermore, more epidemiological studies for improved interventions are required.

Acknowledgements

Conflict of interest

The authors declare no conflict of interest.

Acronyms and abbreviations

ASM	Anti-seizure medication
CNS	Central nervous system
CSF	Cerebrospinal fluid
CT	Computed tomography
OAE	Onchocerciasis associated epilepsy
OCP	Onchocerciasis control program
BBB	Blood-Brain-Barrier
PWE	People with epilepsy
P. falciparum	Plasmodium falciparum
T. canis	Toxocara *Canis*
T. cati	Toxocara *cati*
O. volvulus	Onchocerca *volvulus*
T. solium	Taenia solium
WHO	World Health Organization

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