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Case Report

Neurocysticercosis and Status Epilepticus: About A Clinical Case in the Neurology Department in Dr Congo and Review of the Literature

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Abstract

Neurocysticercosis is a parasitosis of the central nervous system, caused by the cysticerci of a flatworm: Taenia solium. Humans contract it by consuming food or water contaminated with eggs of t. Solium, which is excreted in the stools of individuals with intestinal tapeworm. We report the case of a 45-year-old man living since the age of 32 in Central Africa in the Democratic Republic of Congo, with no particular history, regularly consuming pork meat, having been admitted to the neurology department for the malaise. epileptic. The presence of cystic lesions showing the scolex on imaging highlighted a suspicion of cerebral cysticercosis.

Neurocysticercosis is endemic in all latitudes where pig farming is common practice, and where the level of hygiene and sanitary control of livestock are insufficient. The DRC is surrounded by cysticercal endemic countries where it is responsible for 50 to 70% of epilepsies. Thus, it seemed relevant to us to publish this case to draw attention to this curable pathology in most cases, but it can be fatal or leave disabling sequelae. Improving sanitary and hygiene conditions should allow to eradicate it on the condition that neighbouring countries take the same measures and, if not pool their resources, at least coordinate them.

Keywords: Neurocysticercosis; Epilepsy and Status Epilepticus; Scolex; Taenia Solium

Introduction

Neurocysticercosis or cerebral cysticercosis is a parasitosis of the central nervous system (CNS), due to the cysticerci of a flatworm: Taenia solium. Recognized as the most common CNS parasitosis, it infects approximately 50 million people worldwide. On the European continent, cysticerci parasitosis is considered a controlled or even eradicated infection, particularly through strict quality control of pork and more efficient treatment of wastewater [1].

In addition, the development of stables has also improved health conditions. Sub-Saharan Africa is one of the endemic areas

of T. solium, along with South Asia, India and South America [2]. An increase in the incidence of parasitosis is observed in territories where pig farming is an important economic activity. Hygiene and health conditions are often insufficient [1,2].

Neurocysticercosis can present a clinical polymorphism which largely depends on the number of lesions, the type, the topography, the evolutionary stage of the cysticerci, as well as the immune response of the individual against the parasite. There are no recognized pathognomonic features of a typical neurocysticercosis syndrome [3,4]. Although current practice requires that neurocysticercosis be evoked in an endemic region in the presence of any neurological symptom (unusual headaches, intracranial hypertension, neurological deficits, epilepsy, intellectual deterioration, etc.) for which the etiological diagnosis has not been established [5].

Neurocysticercosis is one of the twenty neglected tropical diseases [6]. And this, not only by the population and the political authorities, but also by health professionals [7]. In the DRC, as in all endemic countries, the scarcity of epidemiological data on neurocysticercosis should not be an excuse for continuing to neglect this parasitosis. The Democratic Republic of Congo is surrounded by human cysticercal endemic countries [7]. The vigilance of these countries vis-à-vis this parasitosis would help our country to control it.

It is in this context that this case report fits, the specific objective of which is to recall the presence of neurocysticercosis and its great contribution to the occurrence of epileptic seizures in our environments.

Case Report

We report the case of a 45-year-old man living since the age of 32 in Central Africa in the Democratic Republic of Congo (DRC), an endemic area for cysticercosis, with only a history of cerebral malaria successfully treated with injectable quinine. A few months later he was admitted to the emergency room for generalized epileptic seizure with tonic-clonic movements without regaining consciousness with loss of urine for more than 30 minutes (status epilepticus). On regaining consciousness, the neurological examination reveals a Glasgow (GCS 15/15) disorientation in time and space, apraxia, fluctuating agnosia. The rest of the neurological examination was normal. General examination and other systems were unremarkable. Brain imaging performed, Cerebral CT scan without and with injection showed multiple, nodular cystic lesions with a small central hyperdense focus (scolex) scattered in the cerebral and cerebellar hemispheres, basal ganglia, thalamus and brainstem. Some of them present with peri-lesional edema suggesting neurocysticercosis with multiple localizations, but without signs of intracranial hypertension.

EEG showed asymmetric rhythmic polyspike and slow waves with flattening of background activity, as well as generalized bifrontotemporal spikes. ECG and chest X-ray were unremarkable.

Figure 1: Cerebral CT scan without injection of contrast product Axial section A, coronal and sagital reconstruction B and C, shows a small central hyperdense focus scattered in the cerebral and cerebellar hemispheres, the basal ganglia, the thalamus and the brainstem within certain places peri-lesional edema.

The biological assessment showed a CRP at 5.2 mg/dl and CPK at 1329 IU/l, without renal insufficiency. The complete blood count was normal, as well as the lumbar puncture. The ELISA test showed the presence of IGM and the absence of IgG, which testifies to a recent infection of cysticercosis in indirect immunofluorescence.

Therapy began with maintaining hemodynamic balance and controlling status epilepticus in intensive care: benzodiazepine 20mg/day, sodium valproate 40mg/kg IVDL, corticosteroid therapy (Dexamethasone 8mg/day for 21 digressively), then by antiparasitics: albendazol 800mg/day for 21 days, metronidazole 1g/day for 10 days.

Under this treatment, the evolution of the patient was favorable with cessation of seizures, and improvement of orientation in time and space. The normalized in 72 hours. A follow-up imaging is planned in 3 months. Hygieno-dietetic and prophylactic advice was given to him on discharge.

Discussion

Epilepsy occurs in almost 50-80% of cases of neurocysticercosis. As most authors point out [8].

This is the second case report of neurocysticercosis revealed by a seizure admitted to the Dr. Joseph Guislain Neuropsychiatric Center, after the one published by BUGEME M. in 2015 [9]. Indeed, the data in the medical literature recognizes epilepsy as the most common clinical presentation of neurocysticercosis, making this parasitosis the leading cause of epilepsy in endemic areas [8]. This is about 30% of cases of epilepsy that are attributable to Taenia solium in many endemic areas [8,10], and this figure can be as high as 70% in high-risk populations [10].

In their studies on the Neurocysticercosis-Epilepsy relationship, on the one hand Ruth Rottbeck., *et al*. reported a high prevalence of cysticercosis in people with epilepsy in southern Rwanda [11]. On the other hand, KABEMBA E., *et al.* reported a figure of 57.1% neurocysticercosis in people with active epilepsy in eastern Zambia [12]. Similarly in Tanzania Dominik., *et al*. reported that more than 30% of all people with epileptic seizures had brain damage from neurocysticercosis [13].

Cysticerci can be located in all parts of the central nervous system [8]. An intraparenchymal localization, a form found in more than 60% of cases, causes epileptic seizures in 75% of people who present with it [14]. The multiplicity of cerebral lesions could account for the severity of the seizures in our patient, especially since they involve both hemispheres and affect the cerebral parenchyma. Moreover, the presence of the scolex in the cerebellar regions and/or the side effects of the treatment as well as the recent nature of the convulsions account for the dizziness and confusion, while the fronto-temporo-parietal lesions would be responsible for the praxic and gnostics and the lack of sphincter control at a distance from the status mal.

One can be surprised by the absence of clinical signs linked to the involvement of the brainstem and the central gray nuclei can be masked by the sedative effect of the treatment and the apraxo-agnosic disorders.

The ELISA test, which revealed positive IgM and negative IgG, indicates a recent inflammatory process, which explains the intensity of the clinical signs. Moreover, the lysis of the larvae increases the inflammation and consequently the symptoms. The master examinations are on the one hand the Western blot and on the other hand the biopsy which makes it possible to highlight the taenia solium [5,8] but these examinations are not available in our technical environment.

Conclusion

Neurocysticercosis is an infectious, parasitic pathology evoked which must be before any inaugural epileptic seizure or unexplained neurological deficit, particularly in endemic areas. In this context, a thorough anamnesis, neuroimaging, and biology help establish the diagnosis with certainty.

Neurocysticercosis is a very preventable cause of epilepsy, caregivers living in endemic areas must be aware of this reality so that early treatment is instituted and prophylactic measures are reinforced. Indeed this parasitosis is a public health problem due to its neurological and psychiatric complications which handicap our communities.

Improving health and hygiene conditions should make it possible to eradicate it provided that neighboring countries take the same measures and, if they do not pool their resources, at least coordinate them.

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