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Case Report

Gait and cognitive disorders revealing massive neurocysticercosis: a case report

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Abstract

Neurocysticercosis is frequent in Madagascar. Its clinical presentations depends on the topography of the lesions. We report a case with gait and cognitive disorders. A 49-year-old man, right-handed, with progressive gait disorders (small steps with magnetization). On examination, he had gait disorders, associated with frontal syndrome with Frontal Assessment Battery (FAB) scale of 10/18, a Mini-Mental Status Examination (MMSE) scale of 24/30, a constructive apraxia and a clock test disturbance. The brain CT scan showed massive lesions of fronto-parietal and temporal neurocysticercosis, meningeal neurocysticercosis with racemose forms. Lesions had different ages associated with a communicating hydrocephalus. Gait disturbance and cognitive function were improved after an evacuating lumbar puncture and cysticidal treatments. This case illustrate the importance of neurological examination in a patient with neurocysticercosis. Neurocysticercosis could be a curable cause of dementia.

Keywords: cognitive disorders, gait disorders, neurocysticercosis

INTRODUCTION

Neurocysticercosis is one of the most frequent neuroparasitosis and endemic in Madagascar [1]. Its manifestations depend on the topography of the lesion, its size and its evolutionary stage [2]. It is often diagnosed by the occurrence of epileptic seizures but a careful neurological examination may reveal other neurological abnormalities that may not be reported spontaneously by the patient and family. That may include cognitive disorders. Cognitive assessment also are not a routine examination in a patient with neurocysticercosis. This is a case seen in a Neurology reference center at Befelatanana university Teaching hospital, Antananarivo, Madagascar. It is a didactic case of a cognitive manifestations of neurocysticercosis.

CASE REPORT

It is a case of a 59 years old, farmer admitted to our department for a first episode of generalized tonic-clonic in epileptic seizures less than 02 min. No fever or headache were reported. He had also a progressive walking difficulty evolving for more than 5 months, aggravated since 3 months. He walks with small steps, hesitating, as if he didn't know how to move forward by taking one step after the other. He had a sensation that his feet were stuck to the ground while walking. He needed to widen the distance between his feet to balance walk. However, no episodes of falling have been reported.

Clinical examination showed walking apraxia, with an enlargement of the sustentation polygon and a magnetization when walking, even an impossibility to walk with an attitude of anteflexion as the walking went on. We found discrete kinetic cerebellar syndrome with hypermetria on the finger-nose test in the two upper limbs. No extrapyramidal rigidity or rest tremors were noted. He had a frontal syndrome with dysexecutive syndrome, decrease in verbal fluency, programming dysfunction, disinhibition, grasping, urinary incontinence, his Frontal Assessment Battery (FAB) was 10/17. He also had a constructive apraxia (Fig. 1) and a defect in visuo-spatial representation on the clock test with a score of 7/10 according to Rouleau's rating (Fig. 2). Language, gnosia, memory and temporo-spatial orientation were preserved and the MMSE score was 24/30. In general examination, we found a subcutaneous nodule in the left temporal muscle, about 0.5 cm, not painful, firm to palpation, mobile and the aspect of the overlying skin is healthy.

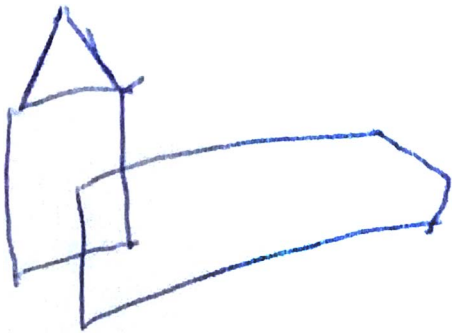

Figure 1. Constructive apraxia.

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Figure 2. Defect in visuo-spatial representation on the clock test.

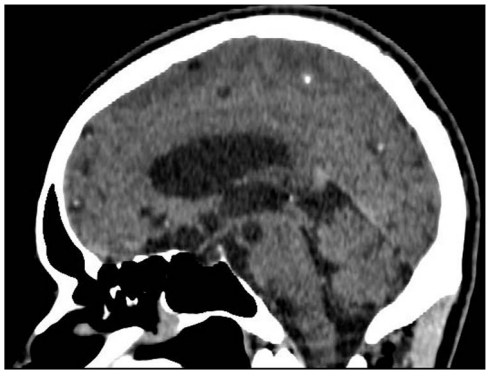

Figure 3. Multiple cystic hypodensities with eccentric, centimetric calcium hyperdensities in the parietal lobe, frontal lobe and cerebellar hemisphere.

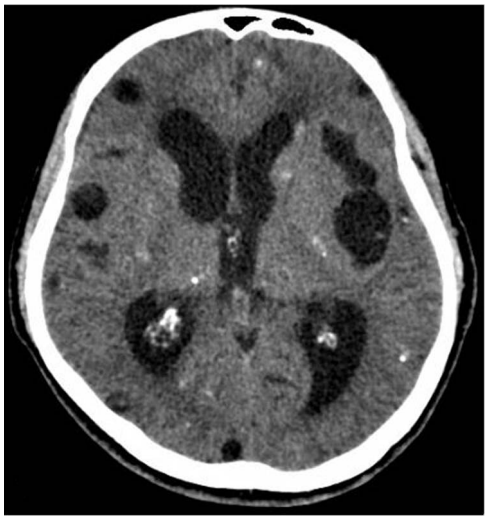

Figure 4. Meningeal bulky cystic hypodensities with ventricular communicating ectasias and left temporal muscle cysticercosis.

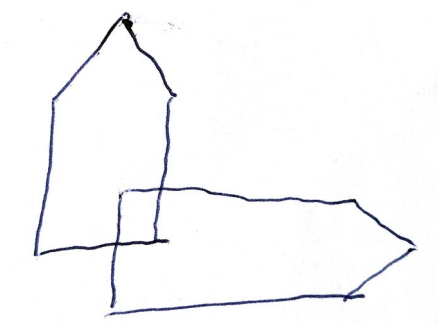

Figure 5. Disappearance of constructive apraxia.

Figure 6. Clock test increase at 10/10.

The patient's brain CT scan with contrast showed multiple cystic hypodensities with eccentric, centimetric calcium hyperdensities in the parietal lobes, frontal lobes and cerebellar hemispheres (Fig. 3); meningeal bulky cystic hypodensities and ventricular communicating ectasias (Fig. 4). Those suggested a massive neurocysticercosis lesions with intra parenchymatous vesicular cysts in supra and sub-tentorial and meningeal localization including racemose forms with communicating hydrocephalus. A round cystic hypodensity is also visible in the left temporal muscle, suggestive of muscle cysticercosis (Fig. 4). The fundus did not show any intraocular localization of cysticercosis. Biological assessment did not reveal any inflammatory or infectious syndrome. Cell count, protein, glucose level in CSF were normal. Immunological test for cysticercosis of the CSF was not performed due to the presence of Del Bruto absolute criteria for neurocysticercosis.

The patient received, CARBAMAZEPINE at 10 mg/kg/day for his seizures, corticosteroid therapy at 1 mg/kg and PRAZIQUANTEL 50 mg/kg per day for 15 days. He had lumbar evacuation puncture, without incident and physical therapy. After the 1st and 2nd day of larvicide, the patient was discharge home.

One month later, improvement in cognitive scores were noted (FAB at 15/18 improvement on the conflict instructions, the go no go test and the Luria sequence; the clock test to 10/10; the MMSE at 27/30 and disappearance of constructive apraxias (Figs. 5, 6). Patient's gait was back to normal and no longer dysmetry on the finger-nose test. The patient was lost to follow-up so a control CT scan was not performed.

DISCUSSION

We retained as diagnosis a massive neurocysticercosis, of different stages, of parenchymatous and meningeal localizations, including racy forms with a communicating hydrocephalus and a left temporal muscle localization.

According to the Del Bruto criteria, the diagnosis of neurocysticercosis is based on clinical, paraclinical and epidemiological arguments [3]. For our patient, for the clinical criteria, an epileptic seizure is a symptom compatible with neurocysticercosis since 80% of neurocysticercosis is manifested by epileptic seizures [4].

Moreover, cognitive disorders are rarely described in the literature. For our patient, these disorders could be related to the presence of major quadriventricular hydrocephalus. For the paraclinical criteria, the patient presents lesions of parenchymatous neurocysticercosis at the multiple vesicular stage and meningeal localization, the presence of which constitutes an absolute criterion according to Del Bruto [3]. In Brazil, apart from epileptic seizures, gait disorders and cognitive disorders were associated and the evolution of the disease can vary from a few months to years. Neurological examination is dominated by imbalanced walk, enlargement of the sustentation polygon with frontal syndrome [5]. Other clinical presentations are headaches, acalculia, forgetfulness with behavioral disorders but always with a progressive evolution as for the case of Verma in India [6].

Our patient received antiepileptic drug, lumbar evacuation puncture, corticosteroid therapy, antiparasitics and physical therapy with a great improvement. The most commonly used antiparasitics are ALBENDAZOLE at 15 mg/kg/day for 8 to 30 days or PRAZIQUANTEL at 50 mg/kg/day for 2 weeks [2]. The combination of these two molecules and the duration of treatment depend on the location and number of lesions found. Perilesional oedema of neurocysticercosis required the use of corticosteroids. First generation antiepileptic drugs are prescribed during the active phase of the infection and should be continued for at least 2 years even in calcified or inactive lesions [2]. For extraparenchymatal location, the treatment of neurocysticercosis with racemose forms and hydrocephalus is not yet well codified. However, for hydrocephalus, two mechanisms can explain it, either an obstruction of the circulation of the CSF or an inflammatory reaction within the subarachnoid space (archnoiditis). If obstructive, an endoscopic extraction of the lesion would be adopted after a brain MRI with a FLAIR sequence. If inflammatory, corticosteroids would reduce the inflammatory process associated with larvicides [7]. For Verma, corticosteroids followed by Acetazolamide resulted in improvement of cognitive symptoms in two weeks [6]. For the Brazilian team, gait disorders with frontal syndrome improved after ventriculoperitoneal shunting associated with an antihistamine, Dexchlorpheniramine [5]. These two observations highlights the involvement of the inflammatory mechanism in cognitive disorders.

This case showed that clinical manifestations of neurocysticercosis are polymorphic and can involve cognitive functions. However, this is a single observation that does not reflect all neurocognitive manifestations of neurocysticercosis. The cognitive disorders in neurocysticercosis can be explained by the existence of meningeal localizations with the presence of communicating hydrocephalus. The diagnostic criteria of Del and Bruto, including absolute criteria such as histological evidence or the presence of cystic lesions showing the larva form (scolex) on the CT scan or brain MRI, were found in our case [2, 3].

About neurocysticercosis, seizures remain the most frequent reason for consultation, although patients may already have underlying cognitive disorders. Apart from normal pressure hydrocephalus and lacunar syndromes, neurocysticercosis with racemose form and hydrocephalus can be an etiology of non-degenerative dementia syndrome which can be improved with treatment.

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None.

CONFLICT OF INTEREST STATEMENT

There are no known conflicts of interest associated with this publication.

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ETHICAL APPROVAL

Not applicable.

CONSENT

A written consent of participation and publishing was obtained from the patient.

GUARANTOR

Dr. Rabearisoa Parany Haja had full access in the data and is the guarantor of this work.

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