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Case Report

A Case of Neurocysticercosis in a Teenager

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Abstract:

The flatworm Taenia solium, which causes infection in immunocompetent patients in the central nervous system (CNS) as neurocysticercosis (NCC), can cause seizures, behavioral disturbances, obstructive hydrocephalus and can generate other neurological signs. It was considered prevalent in underdeveloped countries as a neglected disease, today due to the ease of transportation and migrant population it can be seen in developed countries, WHO considers cysticercosis as a serious public health problem. Female adolescent patient, 15 years old, with no previous history of importance, who consulted for a clinical picture of 20 minutes of evolution consisting of generalized tonic-clonic movements, sup aversion of the gaze, disconnection with the environment of 5 minutes duration that self-resolved, He referred previous episodes of headache, at admission the patient was in a post-ictal state, magnetic resonance imaging report focal lesions in left frontal subcortical cortical topography and right frontal basal peri ependymal topography that could be related to neurocysticercosis in different evolutionary stages. NCC is an important pathology that affects children in urban and rural areas from an early age. The knowledge of hygiene habits within the home and the keeping of pigs should be increased; knowing the clinical picture in time, epidemiological intervention can be conducted, interrupting the chain of transmission and avoiding complications.

Key words: cysticercosis; neurocysticercosis; headache; epilepsy; attention deficit and hyperactivity disorder

Introduction

The flatworm Taenia solium belongs to the family Taeniidae, includes three parasites that affect humans: Taenia solium (the pork tapeworm), Taenia saginata (the beef tapeworm), and Taenia asiatica, is morphologically similar to T. saginata but biologically similar to Taenia solium, who causes infection in immunocompetent patients in the central nervous system (CNS) as neurocysticercosis (NCC), can cause seizures, alterations in behavior, obstructive hydrocephalus and can generate other neurological signs [1].

In the life cycle of the parasite, humans (definitive host) become infected when they consume undercooked pork containing cysticerci (cysts). The cysts invaginate in the upper part of the small intestine and become adult tapeworms. The eggs of adult worms, shed by a human, are ingested by a pig (intermediate host) and turn into cysticerci. When accidentally ingested by humans, the eggs encyst in many tissues, particularly in the brain, and cause neurocysticercosis [2]. The presentation in the cerebral parenchyma occurs 50% of cases, other less frequent may be intraventricular, subarachnoid, ocular and spinal [3].

It was considered prevalent in underdeveloped countries as a neglected disease but because of the ease of means of transport and migrant population can be seen in developed countries, the WHO considers cysticercosis as a serious public health problem [3]. The spread of the disease is wide in Colombia, as indicated by the study carried out in 23 departments for the period 2008-2010, reporting a general seroprevalence of 8.55% at the national level, the department with the highest frequency, Vaupés (40.19%) and Caldas the lowest (0.53%). Some factors associated with cysticercosis found in the study were: educational level no higher than high school, low socioeconomic status, occupation, gastronomic predilection in the consumption of pork and occasionally or never washing vegetables with drinking water [4].

The objective of this study is to present the case of a patient with calcified nodular stage neurocysticercosis who manifested with headache and seizure.

Case presentation

A 15-year-old female adolescent patient with a socio-economic background, housing in a rural area without drinking water service, without pig ownership, but her eating habits consist of consumption of fruits and vegetables washed with non-potable water and consumption of this, without significant pathological history, who consulted for a clinical picture of 20 minutes of evolution consisting of generalized tonic-clonic movements, supra version of the gaze, disconnection with the medium of 5 minutes of duration that self-resolved, refers previous episodes of

headache, to the admission patient with vital signs within normal limits, without signs of respiratory difficulty, without signs of peritoneal irritation, was considered patient with first convulsive episode, at the time in post-ictal state, extension examinations were requested, which reported (Table 1) and CT scan of skull with retrocerebellar cyst, nonspecific punctate calcification at the level of the right lateral ventricle (Figure 1), given the CT report, it was considered to expand studies with brain magnetic resonance imaging (MRI).

Figure 1: CT scan of the skull. Nonspecific punctate calcification at the level of the right lateral ventricle.

The results of the MRI (Figure 2) reported focal lesions in left frontal subcortical cortical topography and right basal frontal periependymal that, relating to neurocysticercosis in different evolutionary stages, started management with albendazole 400 mg orally every 12 hours for 14 days, dexamethasone 8 mg IV every day for 14 days, praziquantel 1 tablet 600mg every 8 hours for 14 days, Oxcarbazepine suspension 300mg/5ml 9cc orally every 12 hours and omeprazole 20 mg orally daily on an empty stomach.

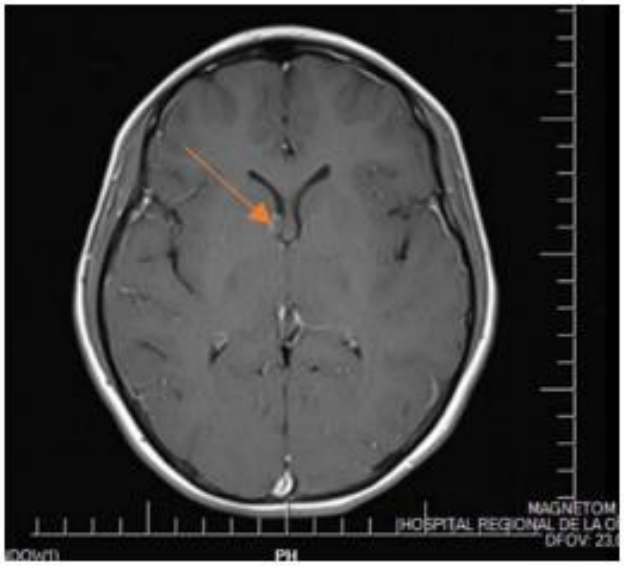

Figure 2: Brain MRI. Focal lesions in left frontal subcortical topography and right basal frontal periependymal in different evolutionary stages.

On the sixth day of hospital stay, studies were requested to cover other etiologies such as cytomegalovirus and toxoplasma in addition to assessment by ophthalmology who considered ametropia, tests reported Toxoplasma IgM and IgG positive, cytomegalovirus IgG positive and IgM negative, rapid HIV test to rule out immunodeficiency for this reason it was decided to suspend dexamethasone which was completed treatment and discharged.

Patient reconsultation 13 days after hospital discharge for clinical picture of 4 hours consisting of intense headache of bitemporal predominance of intensity 7/10, associating dizziness and blurred vision without any other symptoms, tests were requested with results in table 1, brain magnetic resonance without gadolinium details focal calcium lesion right basal periependymal of unchanged sequel appearance is linked to the history of neurocysticercosis, Magnetic resonance imaging with gadolinium reported right basal periependymal focal calcium lesion suggesting

neurocysticercosis in calcified nodular stage in the first instance, arachnoid cyst in posterior fossa, without significant changes; Female adolescent patient with adequate clinical evolution, stable for which exit was indicated with recommendations and control by neuropediatric.

Admission laboratories		Re-entry after 13 days	
Laboratories	**Results**	**Laboratories**	**Results**
Leukocytes	7.61*10^3/mm^3	Leukocytes	8.16*10^3/mm^3
Neutrophils	6.3 %	Neutrophils	72.7%
Lymphocytes	31.4 %	Lymphocytes	20.9%
Monocytes	4.8 %	Monocytes	4.6%
Platelets	296* 10^3/mm^3	Platelets	268* 10^3/mm^3
Hemoglobin	13.2 g/dl	Hemoglobin	11.6 g/dl
C-reactive protein	9.4 mg/l	Creatinine	0.48mg/dl
Urine culture	Negative	ALT	18 U/L
Sodium	140.3 mmol/l	HANDLE	26 U/L
Potassium	3.94mmol/l	Total and direct bilirubin	0.27md/dl; 0.11mg/dl
Colorimetric calcium	9.4 mg/dl	Urea nitrogen	6.1mg/dl
Glucose	141.45 mg/dl	Glucose	84.0 mg/dl

Table 1: Laboratories

Discussion

Neurocysticercosis is due to the location of the larva Taenia solium in the CNS generating a very broad clinical spectrum that also includes asymptomatic infection, forms of mild or moderate clinical manifestations such as headache and sporadic seizures as presented by the patient of the case or severe pictures characterized by clinical signs of intracranial hypertension [5].

Seizures are the most common representation of NCC and usually occur in 50 to 80% of patients with parenchymal brain cysts or calcifications, but are less common in other forms of the disease [6].

These manifestations also depend on a complex range of interconnected factors, including the number and size of cysticerci present, their stage of development, and location within the brain; This contributes to significant difficulties in accurate diagnosis and staging of the disease [7].

The physio pathogenic mechanism of seizures this parasitosis is related to the local inflammatory reaction and perilesional gliosis that is generated around the cyst, as well as some cases of calcified lesions that may represent an epileptogenic focus; in a study conducted in the pediatric population of Colombia the cases described, regardless of the active or inactive state of the lesion, seizures were presented as a clinical manifestation but different from what has been described in most of the pediatric series in none of the histories evaluated was found intracranial hypertension, although it is considered the second most frequent clinical manifestation in children with neurocysticercosis, This variability may be due to the result of pathophysiological mechanisms in the interaction of the host and parasite, since cysticerci generally invade the cerebral parenchyma as in the case of our patient and also evidenced unique injury, other forms of neurocysticercosis, such as arachnoid or ventricular cysts are more common in adults and very rare in children [8].

In another study developed in the general population of Colombia, habits such as the fact of consuming untreated water without boiling and foods such as fruits and vegetables not previously washed with drinking water were identified, relevant factors that intervene in parasitic infection since they enable the ingestion of eggs by humans and reported the seroprevalence of cysticercosis obtained in the general population indicates that our country is part of the group of countries endemic where the parasite is circulating [9]. Due to its epidemiological importance in our environment, it should also be suspected in any patient under 65 years of age, without cardiovascular risk factors, originating or coming from an endemic area and this undergoing a stroke, being a rare but important complication of neurocysticercosis in young patients from endemic countries, in a case of stroke due to NCC antiparasitic treatment is not an emergency due to the risk of worsening cerebral edema [10].

In this case, the definitive diagnosis of NCC with MRI was achieved due to the presence of focal lesions in left frontal subcortical topical topography and right basal frontal peri ependymal in different evolutionary stages, which due to the clinic antiparasitic management was initiated as referred to in the literature with cytocidal drugs currently recommended by experts in parasitology. infectious diseases and neurology [13].

Another important aspect that was obtained in the review, children with structural neurological disorders may also present symptoms of attention deficit hyperactivity disorder (ADHD), among which are severe cranioencephalic trauma with lesions of the frontal lobes, secondary to arachnoid cysts in the temporal lobe, autism, Tourette's syndrome, among others; this relationship would be explained by the theory of front striatal dysfunction, alteration of the executive functions of the frontal lobe secondary to structural damage by NCC lesions. It is necessary to follow these children because it has been shown that ADHD can persist in adult life in 50%, and children can present symptoms such as learning disorders in 24% and behavioral disorders in 12% [11]. However, not all patients with frontal lobe lesions have a poor prognosis, when these occur at an early age it suggests that neuronal plasticity phenomena can sometimes be effective and not as bad prognosis as in other cases [11].

It is important to clarify that there are patients with NCC with its main manifestation of epileptic seizures and in other cases in which they present with attention disorders and poor school performance which are not alarming until the epileptic seizure occurs for which they end up consulting in addition to the complications that may present as intracranial hypertension, arachnoiditis, obstruction of cerebrospinal fluid pathways, hydrocephalus [6, 11].

Due to the mechanism of propagation and maintenance of the taeniasis/cysticercosis, the first related to the fact that taeniasis is an exclusive disease of humans, this is solely responsible for the dispersion of parasite eggs, since open defecation and inadequate excreta disposal are the greatest risk practice. The second is the lack of personal hygiene, especially in habits related to hand washing before eating and after going to the toilet, the consumption of unboiled water and foods such as unwashed vegetables and fruits, as well as their exposure to agents that disperse eggs, practices that favor the ingestion of them by humans. A third aspect is related to the raising of pigs in backyard in conditions that allow or facilitate their contact with human excrement which allows the infection of the pig (12). And finally, the lack of sanitary control of pork, its handling and feeding habits that include the consumption of this undercooked or raw meat, practices that also contribute to infection by this parasite [12], factors evident in our population that indirectly and due

to ignorance of the transmission of taeniasis affected our patient to develop NCC.

This is one of the main risk factors for the acquisition of cysticercosis, since man releases the eggs of the parasite and provides the infective form, in addition a single gravid proglottid can release at least 50,000 eggs, which may be viable under normal environmental conditions [12].

Conclusion

In Latin America, it is necessary to increase knowledge and education on the frequency of neurocysticercosis at the urban and rural levels, as well as on sociodemographic aspects, hygiene habits within the home and pig ownership, in addition to knowing the risk of this pathology in order to initiate an epidemiological intervention to interrupt the chain of transmission and avoid complications.

Ethical considerations:

This research project has the informed consent signed by the patient, where he authorizes the writing and publication of the manuscript. Likewise, the researchers signed the letter of confidentiality, commitment and compliance with research activities.

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Conflicts of interest:

The authors declare that they have no conflicts of interest regarding the research, authorship and publication of this article.

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References

1. Del Brutto OH, Garcia HH. (2015). Taenia solium Cysticercosis — The lessons of history. *Journal of the Neurological Sciences*. 359(1-2): 392–395.
2. Prodjinotho UF, Lema J, Lacorcia M, Schmidt V, Vejzagic N, et al. (2020). Host immune responses during Taenia solium Neurocysticercosis infection and treatment. *PLOS Neglected Tropical Diseases*. 14(4): e0008005.
3. TOZ bastides. (2015). Neurocysticercosis: neglected disease in Colombia. *Morpholia*. 7(3).
4. Giraldo JC, Chala DM, Reinel Vásquez L, Zamora TÓ, Casas JC. (2016). sSymptoms associated with neurocysticercosis in people from the urban and peri-urban areas of the municipality of Coyaima, department of Tolima, and possible associated variables. *Rev Med*. 24(2):10–22.
5. Corti M, Villafañe MF, Yampolsky C, Palmieri O. (2012). Neurocysticercosis with single brain lesions. Presentation of two cases documented by Magnetic Resonance and literature review. *Rev Argentina Radiol*. 76(2):161-166.
6. Garcia HH, Gonzalez AE, Evans CAW, Gilman RH. (2003). Taenia solium cysticercosis. *Lancet*. 362(9383):547–556.
7. Gripper LB, Welburn SC. (2017). The causal relationship between neurocysticercosis infection and the development of epilepsy - a systematic review. *Infect Dis Poverty*. 6(1):31.
8. González-Velásquez T, Barbosa-Ubarnes M. (2012). Neurocysticercosis in the pediatric age: description and five-year experience. *Nova*. 3(2): 217-225.
9. Sánchez ACF, Pastrán SM, Vargas NS, Enriquez Y, Beltrán M, et al. (2013). Cysticercosis in Colombia. Seroprevalence study 2008-2010. *Acta Neurol Colomb*. 2013; 29(2): 73-86.
10. Rueda-Cárdenas LF, Bernal-Cobo RH, Franco-Ruiz CA. (2019). Acute stroke as a presentation of neurocysticercosis: a case report. *Acta Neurol Colomb*. 35(2): 111-115.
11. Barboza M, Sepúlveda S, Montalvo D. (2007). Frontal neurocysticercosis and attention deficit. *Colomb. Med*. 38(1): 21-27.
12. Fica A, Castro M, Soto A, Florez C, Oelker C, et al. (2012). Neurocysticercosis a neglected disease in Chile. *Rev Chilena Infectol*. 29(1): 71-81.
13. López-Valencia D, Medina-Ortega ÁP, Saavedra-Torres JS, Zúñiga-Cerón LF, Zamora-Bastidas TO. (2016). Neurocysticercosis, unusual manifestations. *Rev fac med univ nac colomb*. 64(3):561.

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