Neurocysticercosis in a 14-year-old boy in Italy: An unexpected case

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1. Introduction

Neurocysticercosis (NCC) is a parasitic infection of the central nervous system caused by larvae of *Taenia solium*. It represents the most common cause of neurological disease in children living in developing countries. In recent years, NCC is increasingly being diagnosed also in high-socioeconomic countries, mainly due to the high rate of immigration. We describe a case of a 14-year-old Ecuadorian boy living in Italy, who experienced a generalized tonic-clonic seizure and was diagnosed with NCC. The boy was successfully managed with anticonvulsant, anticysticercal and anti-inflammatory treatment. With the present case we would like to emphasize the importance of considering NCC as a possible cause of non-febrile seizures in children living in developed countries, particularly in those immigrated from an endemic region or had a long-term stay in an area of high prevalence.

2. Case report

A 14-year-old Ecuadorian boy was admitted to the Emergency Department for generalized tonic-clonic seizures. He had experienced loss of consciousness, generalized hypertonia with “morsus linguae” followed by clonic contractions of upper and inferior limbs for almost five minutes. Subsequently he awakened spontaneously complaining drowsiness. At arrival to the Emergency Department the boy was conscious and afebrile. Neurologic examination and blood tests were normal except for mild eosinophilia (630 cells/µL, 8.3%). He was admitted to our Pediatric Department for further evaluation. Family history was silent for epilepsy; the boy’s history revealed he was born and had lived in Ecuador until he was six years old and after immigration to Italy he never returned to his native country. Further blood tests confirmed mild eosinophilia and demonstrated elevation of IgE levels (3611 kU/L). Postictal electroencephalogram recorded slow
spike waves in right temporal–occipital leads with normal background activity. Brain magnetic resonance imaging (MRI) documented a single 11 mm ring-enhancing cyst with a scolex in the cortical–subcortical junction of the right occipital lobe, surrounded by intense perilesional oedema (Figures 1 and 2).

The imaging was highly suggestive for neurocysticercosis. Serologic tests for toxoplasmosis, cytomegalovirus, echinococcus and Western Blot assay for cysticercus were negative, as well as tuberculin skin test. Antiepileptic therapy with carbamazepine chrono (100 mg/d) was started with successful seizure control. Therapy for cyst destruction was initiated with albendazole (15 mg/kg/d) in two doses, in conjunction with prednisone (50 mg/d), both drugs for 14 days. While on antiepileptic and anticysterceral therapy the patient remained symptom free. A month after discontinuation of definitive treatment eosinophil count, IgE levels and electroencephalogram recording were normal, brain MRI demonstrated complete resolution of perilesional oedema but persistence of a 6 mm cyst; therefore anticonvulsant therapy was continued. Six months later, brain MRI showed complete resolution of the parenchymal cyst (Figure 3), thus antiepileptic therapy was interrupted. During the following 6 months the patient remained seizure-free and to date, the boy is healthy and presents good general conditions.

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3. Discussion

Cysticercosis is acquired by ingesting food or water contaminated with eggs of T. solium. Rarely infection may occur after autoingestion of eggs in patients who have intestinal tapeworms. As in pigs, in the human intestine the eggs hatch to release larvae that penetrate the intestinal mucosa and migrate throughout the body to produce human cysticercosis. Although cysts may be found in any human tissue, most mature cysts are found in the central nervous system, skeletal muscle, subcutaneous tissue and eyes. When the parasite infects the central nervous system, neurocysticercosis develops: encysted larvae preferably locate in areas with high blood flow such as the grey–white matter junction and subsequently a progressive decay occurs.

As described for our patient, cysts may remain viable and asymptomatic for up to 5–10 years and subsequently degenerate causing symptoms[6]. In childhood the most common neuroradiologic finding is a single degenerating
parenchymal cyst, presenting with partial seizures in more than 80% of cases[7]. Our patient harboured a single lesion and presented with a generalized tonic–clonic seizure. In our patient Western Blot assay for identification of specific antibodies against cisticercus was negative, confirming low sensitivity of this immunological test, especially for a single lesion[8–10]. Neuroimaging remains the main instrument for diagnosis[7]. With regards to cisticidal treatment, albendazole is considered superior to praziquantel, due to its greater penetration ability into the cerebral spinal fluid, increased bioavailability when co-administered with steroids and because plasma levels aren’t affected by phenytoin or carbamazepine[11]. The main controversies revolve around the effectiveness of antiparasitic drugs for treatment of degenerating lesions and duration of therapy. Studies have evidenced that cisticidal drugs are effective in destroying both viable and degenerating cysts. Treatment with albendazole, 15 mg/kg/d in 2–3 doses for 28 d, is considered to be effective for cyst destruction. Nevertheless, shorter durations of 14 and 8 d have also been used and demonstrated to be equally effective[12–14]. Our case is of epidemiological and clinical relevance. Although NCC is a typical infection of developing countries, sporadic cases have also been reported in Europe (Portugal, Spain, Poland and Romania), mainly due to the high rate of immigration from endemic areas. In Italy the disease is very rare. Recently just one case has been described in a six year old boy[5]. In conclusion, the present report reminds us to consider neurocysticercosis as a possible cause of non febrile seizures in children living in developed countries, especially in those who have immigrated from an endemic region or have had a long-term stay in an area of high prevalence.

**Conflict of interest statement**

We declare that we have no conflict of interest.

**Comments**

**Background**

Neurocysticercosis is the most common cause of neurological diseases in children in developing countries, but is often not considered in developed countries. The authors present an interesting case of a young boy in Italy who is diagnosed with neurocysticercosis after emigrating from Ecuador.

**Research frontiers**

The authors report the successful diagnosis and treatment with albendazole of neurocysticercosis of a young boy in Italy.

**Related reports**

There is a similar report of a young boy in Italy being diagnosed with neurocysticercosis (Raffaldi et al. – cited by the authors). The current report confirms that, although rare, neurocysticercosis may present in patients in developed countries, despite no exposure to *T. solium* for a period of almost 10 years.

**Applications**

The applications are clear, this will be of value to practitioners in many countries when faced with patients undergoing seizures and who may not be familiar with the symptoms/epidemiology of *T. solium* cysticercosis.

**Peer review**

This short report will mainly be of value to medical practitioners, mainly those who are not experienced with diagnosing this disease. It may also be of interest to scientists interested in the epidemiology and distribution of *T. solium*. As such, it is a useful addition to the literature.

**References**

[1] Singhi P, Singhi S. Neurocysticercosis in children. Indian J Pediatr 2009; 76: 537–545.

[2] Nash T. Edema surrounding calcified intracranial cysticerci: clinical manifestations, natural history, and treatment. Pathog Glob Health 2012; 106: 275–279.

[3] Gaffo AL, Guillen–Pinto D, Campos–Olazábal P, Burneo JG. Cysticercosis as the main cause of partial seizures in children in Peru. Rev Neurol 2004; 39: 924–926.

[4] Adhikari S, Sathian B, Koirala DP, Rao KS. Profile of children admitted with seizures in a tertiary care hospital of Western Nepal. BMC Pediatr 2013; 13: 43.

[5] Raffaldi I, Scolfaro C, Mignone F, Aguzzi S, Denegri F, Tovo PA. An uncommon cause of seizures in children living in developed countries: neurocysticercosis—a case report. Ital J Pediatr 2011; 37: 9.

[6] San–Juan Orta D. Clinical manifestations of neurocysticercosis. Neurologia 2009; 24: 331–335.

[7] Coyle CM, Tanowitz HB. Diagnosis and treatment of neurocysticercosis. Interdiscip Perspect Infect Dis 2009; doi: 10.1155/2009/180742.

[8] Foyaca–Sibat H, Cowan LD, Carabin H, Targonska I, Anwary MA, Serrano–Ocana G, et al. Accuracy of serological testing for the diagnosis of prevalent neurocysticercosis in outpatients with epilepsy, Eastern Cape Province, South Africa. PLoS Negl Trop Dis 2009; 3: e562.

[9] Del Brutto OH. Diagnostic criteria for neurocysticercosis, revisited. Pathog Glob Health 2012; 106: 299–304.

[10] Rodriguez S, Wilkins P, Dorny P. Immunological and molecular diagnosis of cysticercosis. Pathog Glob Health 2012; 106: 286–298.

[11] Cruz M, Cruz L, Horton J. Albendazole vs praziquantel in the treatment of cerebral cisticercosis: clinical evaluation. Trans R Soc Trop Med Hyg 1991; 85: 244–247.

[12] Singh P, Dayal D, Khandelwal N. One week versus four week of albendazole therapy for neurocysticercosis in children: a randomized placebo controlled double blind trial. Pediatr Infect Dis J 2003; 22: 268–272.

[13] Sinha S, Sharma BS. Neurocysticercosis: a review of current status and management. J Clin Neurosci 2009; 16: 867–876.

[14] Baird RA, Wiebe S, Zunt JR, Halperin JJ, Gronseth G, Roos KL. Evidence-based guideline: Treatment of parenchymal neurocysticercosis: Report of the guideline development subcommittee of the american academy of neurology. Neurol 2013; 80: 1424–1429.