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Neurocysticercosis: an unusual presentation of a rare disease

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Sirs: Cysticercosis is a systemic infestation by the larval form of the tape-worm *Taenia solium* and is the most common parasitic disease affecting the central nervous system in the world [9]. Humans acquire intestinal *T. solium* infection by eating undercooked pork containing cysticerci: the larva attaches to the gut wall and grows into the intestinal tape-worm. Cysticercosis can result from ingestion of *T. solium* eggs from human faeces, from contamined food or from faecal-oral autoinfection in patients who harbour adult parasites in their intestinal tract [8]; the egg shell is digested in the stomach, liberating oncospheres that cross the intestinal wall, enter the circulation and are carried to many parts of the body of the host (brain, muscle, eye, subcutaneous tissue). The incidence of cysticercosis is highly variable and is related principally to sociocultural and economic factors; neurocysticercosis is commonly observed in Latin America and developing countries of Asia and Africa where the disease has always been endemic [4]. The frequent world travel today and the increasing immigration of persons from endemic areas spread cysticercosis widely, and clinicians are more often called upon to diagnose and treat patients with conditions that are not familiar in developed countries. We report the case of a patient who had an unusual clinical manifestation of neurocysticercosis.

A 40-year-old Peruvian woman was hospitalized because of 24-h history of agitation, acute confusional state and deteriorating consciousness. The woman had been living in Italy for 2 years, and she did not have a history of fever, seizures, diabetes, asthma, arterial hypertension, drug or alcohol abuse or any other known disease. She had suffered no head injury. At admission to our hospital she was in coma (Glasgow Coma Scale 9). Her temperature was 36.8°C, blood pressure 120/80 mmHg, pulse regular at 70 bpm. No neurological deficits or meningeal signs were present. Routine laboratory tests were normal. Magnetic resonance imaging (MRI) of the brain showed multiple parenchymatous and intraventricular cysts at various stages of development, measuring 5–35 mm in diameter, most of which were located in the left frontal lobe (Fig. 1). The fluid inside the cysts had a MRI signal similar to that of cerebrospinal fluid (CSF), and some cysts were surrounded by a ring enhancement. Inside some cysts could be visualized on T1-weighted images a high-intensity signal. Multiple nodular calcified lesions were also present. Plain radiography of the skull also revealed multiple calcified cysts of the skeletal muscle. The electroencephalography performed at the time of presentation was normal. CSF examination revealed 10 cells/mm^3, 43 mg/dl protein, 69 mg/dl sugar (with a serum glucose of 112 mg/dl). No microorganisms were seen on Gram's stain, and culture for bacteria, fungi and mycobacteria was negative. Serological tests for anticysticercus antibodies by enzyme-linked immunosorbent assay and Immunoblot tests were positive both in the CSF and in the serum. Microscopic search for eggs in stool specimens was negative.

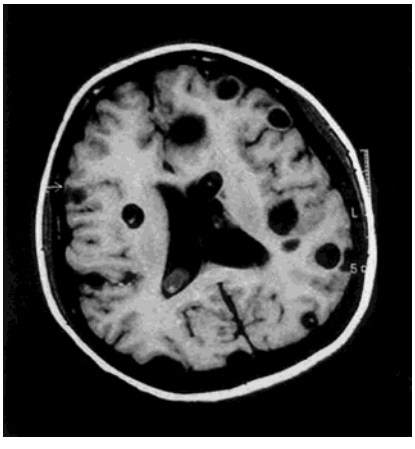

Fig. 1 Axial T1-weighted brain MRI at the time of admission, showing intraventricular and parenchymatous cysts in a 40-year-old woman who presented with coma. The scolex is visible inside the cyst as a hyperintense structure

The patient received intravenous 100 ml 20 % mannitol every 4 h and intravenous dexamethasone 8 mg per day. She showed improvement in consciousness within 36 h. Anthelminthic therapy with albendazole was started orally at a dose of 400 mg twice daily. The patient also received antiepileptic drugs during treatment. Brain MRI after 1 month of albendazole therapy showed no significant changes in number or size of parasitic lesions. Albendazole treatment was continued at the same dose, and the patient was further evaluated. At 3-month follow-up examination the patient was in a good clinical condition with no evidence of neurological abnormalities or clinical manifestation of adverse reactions to the therapy; brain MRI showed a reduction in the size of the cysts.

Cysticercosis is the major cause of late-onset epilepsy in most developing countries [1, 5–7]. Nevertheless, there are no pathognomonic features or typical syndrome of neurocysticercosis because the disease is quite com-

plex in its clinical manifestation, depending on the number, size and location of the parasitic lesions, the host immune response to the parasite and the sequelae of previous infection. Seizures, increased intracranial pressure, focal neurological deficits and intellectual deterioration are the most frequent clinical signs [4]. Cysticercosis today represents a major public health problem not only in developing countries but increasingly in industrialized countries as well, where few clinicians have experience in managing such patients, and most have never even seen a patient with this important neurological condition.

Our patient presented with an unusual clinical manifestation of neurocysticercosis, with a history of agitation, confusion and coma which developed within a few hours, in the absence of seizures and any other neurological symptoms. Neuroimaging is the best procedure for diagnosing neurocysticercosis [2, 3], and in our case the diagnosis was based on the proper integration of the epidemiological data and the MRI findings of cerebral and cerebellar cysts at various stages of development. Immune tests for cysticercosis in serum and CSF and plain radiography of the skull provided additional information upon which to confirm the diagnosis. Diagnosis of neurocysticercosis should be kept in mind and always considered in evaluating patients with coma travelling in or migrating from countries in which the disease is endemic.

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