

Parenchymal neurocysticercosis

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A 36-year-old man presented with headache, confusion, and seizure. The patient was brought by his co-workers to the emergency department (ED) because he was noted to be “acting drunk” at work. He did not have a past medical and surgical history of note. He emigrated from Mexico and had lived in the USA for 10 years. His vital signs were normal. Physical examination showed normal findings without neurological deficits. He could not recall the events that happened at work. Laboratory findings were within the normal range. Toxicological screen and blood alcohol level were undetectable. Computed tomography (CT scan) of the head demonstrated bilateral multiple cerebral parenchymal cystic lesions (Fig. 1a–d). Some cystic lesions contained a bright spot, depicting the characteristic “hole-with-dot” appearance (*white arrows*, Fig. 1b–d). Given the typical clinical presentation, CT scan findings and epidemiological history, a diagnosis of parenchymal neurocysticercosis was made. He was treated with a combined albendazole (400 mg every 12 h) and prednisone (60 mg every 24 h) therapy for 14 days, together with phenytoin to prevent further seizure episodes. He had an uneventful recovery and did not have breakthrough seizure in the 3- and 6-month follow-ups.

Taenia solium infestation is caused by the pork tapeworm and is endemic in developing countries, mainly in Central and South America, Africa, and Asia, where sanitation conditions and personal hygiene are suboptimal. Its prevalence is higher in rural areas in those countries in

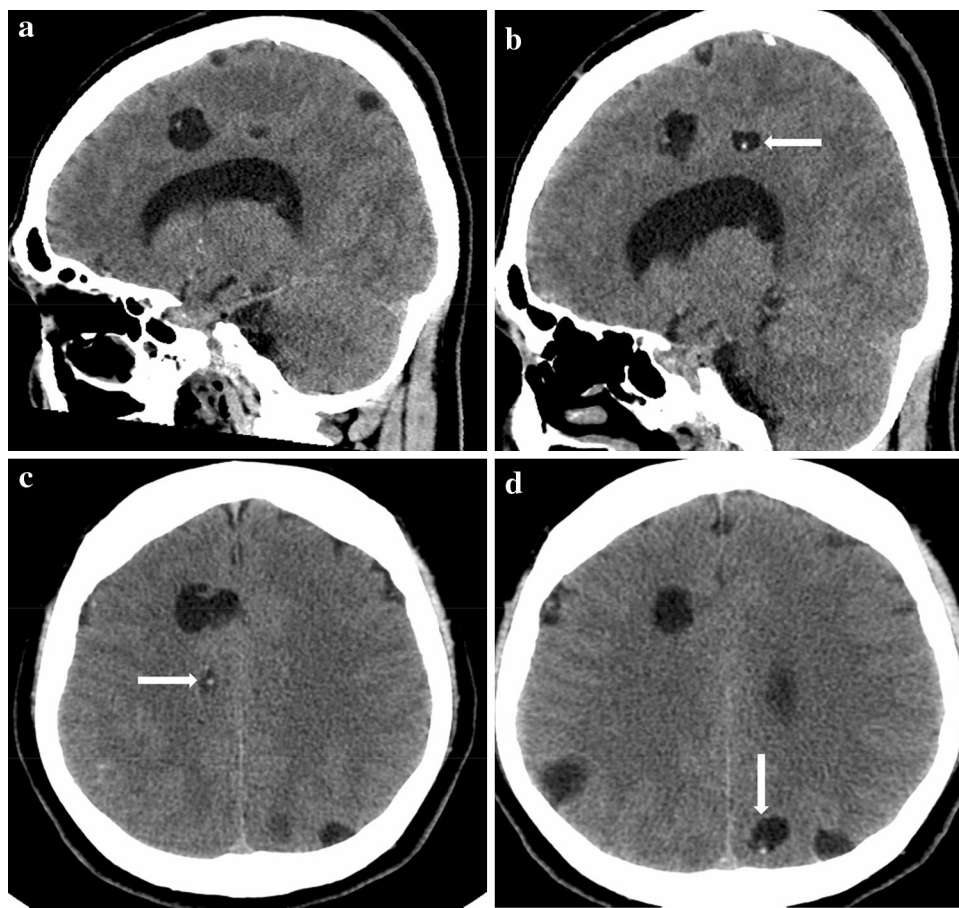
which pigs are raised in free-roaming fields where human fecal contamination of soil is a common practice [1, 2]. *T. solium* causes two distinct forms of parasitic infection in humans, known as taeniasis and cysticercosis [2–4]. Taeniasis is a localized human intestinal infection, transmitted by eating raw or undercooked pork muscle tissue containing *T. solium* cysts. Taeniasis usually results in asymptomatic human tapeworm carriers with shedding *T. solium* eggs in stool. On the other hand, cysticercosis is a systemic tissue infection, acquired by consumption of food contaminated with *T. solium* eggs, derived from another person or self. Then the embryos hatch in the intestine into the larvae, which penetrate the bowel wall and finally migrate to different parts of the body, becoming encysted in end organs. Cysticercosis can involve the nervous system and the extra-neural system (subcutaneous tissues, striated muscle, liver, or lung) [1, 3].

Neurocysticercosis (NCC) is a clinical syndrome due to cysticercosis that involves the nervous system. It, in turn, is subdivided into parenchymal and extra-parenchymal NCC. Extra-parenchymal forms of NCC affect the CSF space, eyes, and spinal cord [1, 4, 5]. In endemic countries, parenchymal NCC is regarded as the most common preventable cause of acquired seizure [3]. It is recognized by the World Health Organization as one of the major neglected tropical diseases [2]. NCC is also observed in parts of the industrialized countries, particularly where there are significant numbers of immigrant communities from *T. solium* endemic countries [2, 4].

Clinical manifestations of NCC depend on the number, location, size, stages of cysticercal cysts in the brain, and the degree of the host immune response [1, 3, 5]. Parenchymal NCC is the second most common form of NCC after subarachnoid NCC. The cystic lesions, usually less than 1 cm, of parenchymal NCC are mostly located in the

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Fig. 1 Brain CT, in sagittal (a, b) and axial views (c, d), showed bilateral multiple cerebral cystic lesions in varying sizes. White arrows (b–d) demonstrate the diagnostic “hole-with-dot” appearance of parenchymal neurocysticercosis (NCC)



cerebral cortex and basal ganglia [5]. Patients with parenchymal NCC may be asymptomatic for many years. When the host immune system induces inflammatory response around the cysts, headaches and seizures are the most commonly presented features, followed by focal neurologic deficits or altered mental status. Fever is typically absent [3, 5]. Both the CT scan and MR imaging have increased the possibility of diagnostic accuracy for NCC. A typical cranial imaging finding of parenchymal NCC is multiple cerebral cystic lesions, within which *T. solium* scolices appear as eccentric bright hyperintense nodules, giving rise to the pathognomonic “hole-with-dot” pattern (Fig. 1b–d) [4, 5]. Serologically, the recommended test is enzyme-linked immunoelectrotransfer blot assay (EITB) for detection of anti-cysticercal antibodies, with both sensitivity and specificity >98 % in patients with more than 2 CNS lesions [4]. A brain biopsy is rarely required for the diagnosis of parenchymal NCC. A set of the diagnostic criteria of NCC has been developed, and it is based on clinical features, imaging, serological test, and epidemiological data [4].

Symptomatic parenchymal NCC is an indication for anti-parasitic treatment in order to hasten resolution of

parenchymal cysts and prevention of additional seizure episodes [1, 3]. Albendazole (15 mg/kg/day, maximum 800 mg/day) is the preferred cysticidal drug given for a length of 3–14 days, depending on the number of parenchymal NCC lesions. Praziquantel (50–100 mg/kg/day) is an alternative therapy. An anti-epileptic drug, preferably phenytoin, should be administered to patients presented with seizures from parenchymal NCC. Anti-inflammatory therapy, either dexamethasone (0.1 mg/kg/day) or prednisone (1 mg/kg/day), is also recommended in conjunction with anti-parasitic treatment to prevent worsening neurological symptoms from inflammation and cerebral edema around the dying or degenerating cysts [1, 3].

In summary, NCC is no longer considered as a disease only of developing countries given advances in globalization and ease of intercontinental travel. In both North America and Western Europe, there are increasing numbers of reported cases of NCC in returned travelers or immigrants from endemic regions. Thus, practicing physicians and interpreting radiologists should be familiar with the typical clinical features, diagnostic radiological findings, and standard treatment of different forms of NCC.

Conflict of interest None.

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