Brief Reports

Neurocysticercosis: An Old Disease with New Questions

Michael A. Cook, DO; Octavio Cosme, MD; Deeptha Nedunchezian, MD; and
Matthew Stern, MD
Staten Island, New York

Cysticercosis is a parasitic infection of the central nervous system that occurs in some geographic regions and is transmitted through contaminated food or by the fecal–oral route. Treatment depends on the degree of central nervous system involvement. Ventricular cysts require surgery. Parenchymal involvement requires systemic antiparasitic therapy. Computed tomography and magnetic resonance imaging are used to image patients with neurocysticercosis. Magnetic resonance imaging is more sensitive for cerebral involvement than is computed tomography. Computed tomography is more sensitive to calcifications than is magnetic resonance imaging.

Key words. Cysticercosis; tomography, X-ray computed; magnetic resonance imaging; parasitic diseases.
(*J Fam Pract* 1994; 39:583-587)

Cysticercosis is the most common parasitic infection of the central nervous system and the most frequent cause of adult seizures in regions where the disease is endemic.[1] It is acquired by the ingestion of *Taenia solium* eggs through contaminated food or by the fecal–oral route.[2] The clinical manifestations are caused by the migration of the larval form to multiple internal organs, such as the brain.

T solium is endemic in Latin America, Africa, Indonesia, China, and eastern Europe. In the United States, neurocysticercosis is not uncommon in communities that have a large number of immigrants from those regions where the the disease is prevalent. It has recently been documented in indigent US populations through local transmission by the fecal–oral route.[2-4] In this report, we present our experience with a case of neurocysticercosis and discuss the diagnosis and treatment.

Case Report

A 49-year-old Peruvian woman, who immigrated to the United States 18 years ago, presented to the hospital with new-onset generalized seizures. Five years earlier, the patient had received a diagnosis of hydrocephalus secondary to ventricular cysticercosis by means of ventricular biopsy during placement of a ventriculoatrial shunt. At the time of the original diagnosis, there was no computerized tomography (CT) evidence of a ventricular cyst or parenchymal lesions. Since cranial CT 5 years ago demonstrated no parenchymal lesions, no systemic antiparasitic agents were administered at that time. During the current presentation, the patient reported urinary incontinence, ataxia, headache, nausea, vomiting, and emotional lability.

The physical examination revealed nystagmus and diminished muscle tone in the lower extremities. There was no papilledema and the pupils were normal. There were no meningeal signs, rashes, or skin lesions. Babinski's reflex, complete blood count, and electrolytes were normal. An electroencephalogram demonstrated diffuse cerebral dysfunction. Cranial CT (Figure 1) showed diffuse ventricular dilatation with marked dilatation of the right frontal horn and multiple intracranial calcifications measuring less than 10 mm each. Cranial magnetic resonance imaging (MRI) revealed lateral and fourth-ventricular dilatation consistent with communicating hydrocephalus (Figure 2). The right frontal horn dilatation proved to be a cyst within the frontal horn rather than frontal horn expansion (Figures 3 and 4). Multiple ring and nodular enhancing intracerebral lesions accompanied by cerebral edema were present.

Examination of the cerebral spinal fluid (CSF) re-
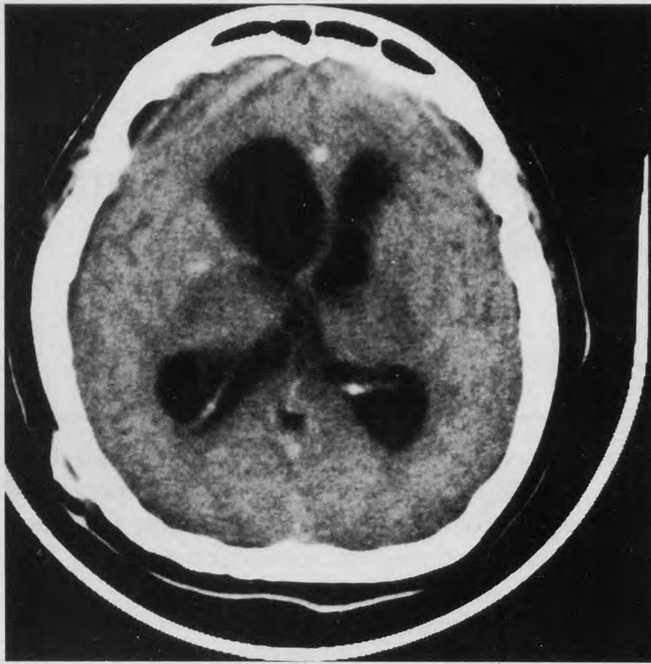
ISSN 0094-3509

Figure 1. A cranial axial computerized tomographic image shows multiple cerebral calcifications and dilatation of the lateral ventricles, particularly of the right frontal horn.

vealed glucose 29 mg/dL (1.6 mmol/L) and protein 80 mg/dL (0.80 g/L). The CSF cell count was 78 white cells (95% lymphocytes, 5% granulocytes). The CSF cysticercosis titer by enzyme-linked immunosorbent assay was 1:256 (normal 1:8). The stool did not contain ova or
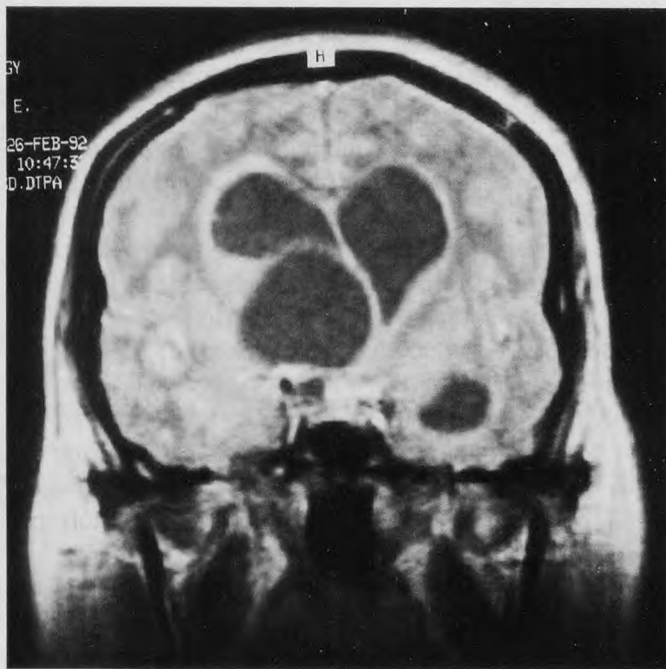
Figure 2. Coronal magnetic resonance imaging with gadolinium reveals a nonenhancing right frontal horn cyst with mass effect.

parasites. The seizures were controlled with phenytoin (100 mg q8h po). She received intravenous dexamethasone (4 mg q6h) to treat cerebral edema and a 15-day course of praziquantel (50 mg/kg/d). Her clinical picture slowly improved and she was discharged. Two months later she was asymptomatic. A follow-up cranial CT scan (Figure 5) demonstrated resolution of the hydrocephalus, frontal horn cyst, and intracerebral calcifications.

Discussion

The diagnosis of neurocysticercosis should be considered in patients who present with seizures, obstructive hydrocephalus or meningitis, and cystic brain lesions.[2-4] The patients need not be from an area where the infection is endemic[2,3] Recently, four New York City residents developed neurocysticercosis thought to have been acquired through fecal–oral transmission from infected Latin American housekeepers.[2]

The signs and clinical manifestations depend on the area of the central nervous system that is affected. Patients with parenchymal lesions most often present with seizures, whereas those with intraventricular cysts usually present with signs and symptoms attributable to obstructive hydrocephalus. The most common presentations are new-onset seizures, altered mental status, and headache.[4] Symptoms develop as a result of mass effect, inflammation, and obstruction of the foramina and ventricular system of the brain.[5]

When neurocysticercosis is suspected, patients should have MRI of the brain, a spinal tap, and CSF cysticercosis titer. Computed tomography alone may underestimate the extent of the disease. A presumptive clinical diagnosis can be based on history and physical examination and supported by radiographic findings and CSF analysis.[6,7] Although a definitive diagnosis is made by surgical excision of a lesion, it is rarely necessary.

Computed tomography is able to demonstrate parenchymal lesions clearly but is less effective at imaging intraventricular cysts. Magnetic resonance imaging is superior to CT in the detection of intraventricular, parenchymal, and subarachnoid cysts and cerebral edema but is not as sensitive to parenchymal calcifications as is CT.[8]

The CT and MRI scans in our patient were classic representations of neurocysticercosis. The intracerebral ring and nodular enhancing lesions were most likely the source of the seizures. The large ventricular cyst in the frontal horn probably was responsible for the patient's emotional lability. Obstructive hydrocephalus caused the headaches.

Examination of the CSF revealed elevated protein, pleocytosis, low glucose level, and an elevated opening

Figure 3. Coronal magnetic resonance imaging reveals a right frontal horn cyst with associated frontal horn expansion.

pressure consistent with chronic meningitis.[9] The pleocytosis was composed primarily of lymphocytes. The CSF findings in our patient were also classic.

Inactive disease can be differentiated from active disease by obtaining a CSF enzyme-linked immunosorbent assay, a test that measures IgM antibodies to cysticercus antigens.[10] Elevated IgM titers are 87% sensitive and 95% specific for active disease. Serum titers are not clinically useful because they have only 50% sensitivity and 70% specificity.

Inactive forms, such as granulomas and calcifications, do not induce IgM production. False-positive reactions occur because of cross-reactivity with antibodies to other parasitic antigens, such as *Echinococcus*. The enzyme-linked immunoelectrotransfer blot assay detects antibody to *T solium* in CSF and serum with 95% sensitivity and 100% specificity.[11]

Treatment options include anticonvulsants, steroids, and surgery. Our patient remained seizure-free with one anticonvulsant. One study found, however, that optimal seizure control may require two anticonvulsants (phenytoin 300 to 400 mg/d, and carbamazepine 600 to 800 mg/d). Withdrawal of the anticonvulsants may cause the seizures to recur.[12]

The treatment of intraventricular and meningeal cysts is limited to surgery since the antiparasitic agents do not achieve an adequate level in cysts. Ventriculoatrial and ventriculoperitoneal shunts can be placed to lower intracranial pressure. Before the advent of MRI, patients who

Figure 4. Saggital magnetic resonance imaging reveals a large right frontal horn cyst.

did not have evidence of parenchymal involvement on CT did not necessarily receive systemic antiparasitic therapy.[13]

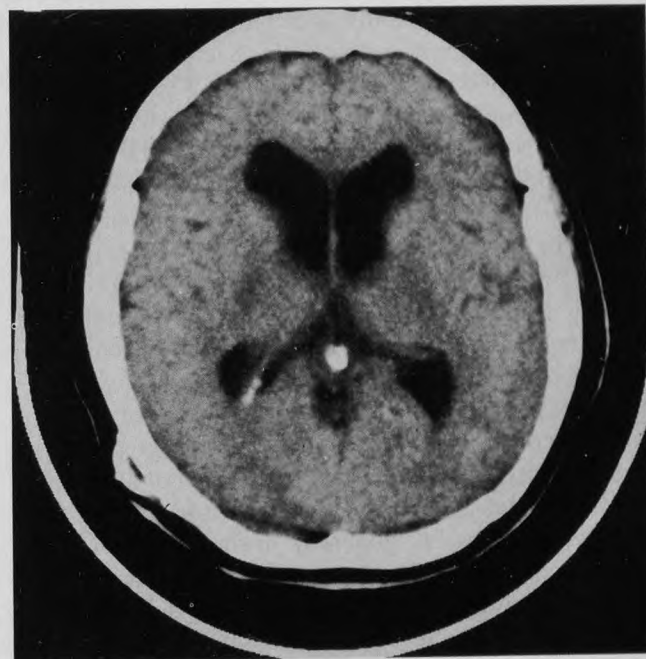Parenchymal involvement requires systemic antipar-

Figure 5. Follow-up cranial axial computerized tomographic image shows resolution of the hydrocephalus, ventricular cyst, and cerebral calcifications.

asitic treatment with either praziquantel (50 mg/kg/d) or albendazole (15 mg/kg/d).[14–20] Most patients require a 15-day course of praziquantel. Those with large or more numerous cysts require a 30-day course.[21] The length of treatment for albendazole is controversial. Studies comparing 30-, 14-, 8-, and 3-day regimens have found them equally efficacious.[17–20] The advantages of the shorter regimens are reduced expense, better compliance, and fewer adverse reactions.

Symptoms usually worsen initially as a result of cerebral edema, which develops secondary to a superimposed inflammatory reaction surrounding the dying cysts. One week after initiation of treatment, worsening cerebral edema and accentuated ring enhancement can be expected.[16] If there is no radiographic evidence of improvement after 15 days of therapy, the patient should be given a second course of treatment. Follow-up scans should be obtained as clinically indicated.

The most common symptom during treatment is headache.[14,19,20] Other adverse reactions include nausea, vomiting, hyperthermia, seizures, somnolence, diplopia, and transient hemiparesis.[19] Steroids can alleviate the symptoms; however, dexamethasone lowers the plasma level of praziquantel by 50% and should not be used routinely.[22] Patients should be treated symptomatically with aspirin, reserving steroids for severe reactions. Our patient received steroids to treat cerebral edema and severe, progressive neurologic symptoms.

When our patient was diagnosed with neurocysticercosis 5 years ago, there was no clinical or CT evidence of parenchymal involvement. Therefore, she did not receive systemic antiparasitic therapy. That the patient developed cerebral involvement 5 years later and in the interim had not traveled to any areas where the infection is endemic suggests that either she was reinfected while in the United States or the parenchymal infection was occult. Since no close contacts were known to have cysticercosis, we favor the latter hypothesis.[2,3] In retrospect, if our patient had occult parenchymal cysticercosis, she might have benefited from an antiparasitic agent when the diagnosis of ventricular cysticercosis was made.

The prognosis is best if the diagnosis is made early, when there are minimal clinical and radiographic findings. Once ventricular cysts develop and obstructive hydrocephalus ensues, the prognosis is less favorable.[22] Parenchymal disease generally has a good prognosis. Extraparenchymal disease, such as ventricular, spinal, and subarachnoid involvement, has a poorer prognosis.[23] Hydrocephalus carries a 50% mortality.[24] In one study, CSF glucose levels lower than 10 mg/dL (0.6 mmol/L) were associated with chronic intracranial hypertension and death within 6 months of presentation.[9]

In areas where cysticercosis is endemic, prevention should focus on educating people about the natural history of the disease, the improvement of personal hygiene, and proper food preparation. Physicians should consider neurocysticercosis in immigrants from those areas who present with neurologic complaints, and in all patients who present with seizures and brain cysts.[2] A high index of suspicion must be maintained in order to make an early diagnosis. Close contacts of infected persons should have their stools screened for evidence of cysticercosis.

References

1. Medine MT, Rosas E, Rubio-Donnadieu F, Sotelo J. Neurocysticercosis as the main cause of late-onset epilepsy in Mexico. Arch Intern Med 1990; 150:325–7.
2. Schantz PM, Moore AC, Munoz JL, Hartman BJ, Schaefer JA, Aron AM, et al. Neurocysticercosis in an orthodox Jewish community in New York City. N Engl J Med 1992; 327:692–5.
3. Centers for Disease Control. Locally acquired neurocysticercosis: North Carolina, Massachusetts, and South Carolina, 1989–1991. MMWR 1992; 41(1):1–4.
4. Scharf D. Neurocysticercosis: two hundred thirty-eight cases from a California hospital. Arch Neurol 1988; 45:777–80.
5. Krammer LD, Locke GE, Byrd SE, Daryabagi J. Cerebral cysticercosis: documentation of natural history with CT. Radiology 1989; 171:459–62.
6. Sotelo J, Guerrero V, Rubio F. Neurocysticercosis: a new classification based on active and inactive forms. Arch Intern Med 1985; 145:442–5.
7. Teitelbaum GP, Otto RJ, Lin M, Watanabe AT, Stull MA, Munz HJ, Bradley WG Jr. MR imaging of neurocysticercosis. Am J Roentgen 1989; 153:857–66.
8. Suss RA, Maravilla KR, Thompson J. MR imaging of intracranial cysticercosis: comparison with CT and anatomopathologic features. Am J Neurorad 1986; 7:235–42.
9. McMormick GF, Chi-shing Z, Heiden J. Cysticercosis cerebri: review of 127 cases. Arch Neurol 1982; 39:534–9.
10. Ross N, Sotelo J, Nieto D. ELISA in the diagnosis of neurocysticercosis. Arch Neurol 1986; 43:353–6.
11. Garcia HH, Martinez M, Gilman R, Herrera G, Tsang VCW, Diaz F, et al, and The Neurocysticercosis Working Group in Peru. Diagnosis of cysticercosis in endemic regions. Lancet 1991; 338:549–51.
13. Vasconcelos D, Cruz-Segure H, Mateos-Gomez H, Alanis GZ. Selective indications for the use of praziquantel in the treatment of brain cysticercosis. J Neurol Neurosurg Psychiatry 1987; 50:383–8.
12. Del Brutto OH, Santibanez R, Noboa CA, Aguirre R, Diaz E, Alaron TA. Epilepsy due to neurocysticercosis. Analysis of 203 patients. Neurology 1992; 42:389–92.
14. Sotelo J, Escobedo F, Rodriguez-Carbajal J, et al. Therapy of parenchymal brain cysticosis with praziquantel. N Engl J Med 1984; 310:1001–7.
15. Sotelo J, Torres B, Rubio-Donnadieu F, Escobedo F, Rodriguez-Carbajal J. Praziquantel in the treatment of neurocysticercosis: long-term follow-up. Neurology 1985; 35:752–5.
16. Rawlings D, Ferriero DM, Messing RO. Early CT re-evaluation after empiric praziquantel therapy in neurocysticercosis. Neurology 1989; 39:739–41.
17. Escobedo F, Penagos P, Rodriguez J, Sotelo J. Albendazole therapy for neurocysticercosis. Arch Intern Med 1987; 147:738–41.
18. Sotelo J, Esconedo F, Penagos P. Albendazole vs praziquantel for therapy of neurocysticercosis. Arch Neurol 1988; 45:532–4.

19. Sotelo J, Pemagos P, Escobado F, Del Brutto OH. Short course of albendazole therapy for neurocysticercosis. Arch Neurol 1988;45: 1130–3.

20. Alarcon F, Escalante L, Duenas G, Montalvo M, Roman M. Neurocysticercosis: short course of treatment with albendazole. Arch Neurol 1989; 46:1231–6.

21. Estanol B, Corona T, Abad P. A prognostic classification of cerebral cysticercosis: therapeutic implications. J Neurol Neurosurg Psychiatry 1986; 49:1131–4.

22. Vasquez JL, Jung H, Sotelo J. Plasma levels of praziquantel decrease when dexamethasone is given simultaneously. Neurology 1987; 37:1561–2.

23. Bandres JC, White AC Jr, Samo T, Murphy EC, Harris RL. Extraparenchymal neurocysticercosis: report of five cases and review of management. Clin Infect Dis 1992; 15:799–811.

24. Sotelo J, Marin C. Hydrocephalus secondary to cysticercotic arachnoiditis: a long-term follow-up review of 92 cases. J Neurosurg 1987; 66:686–9.