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Case Report

CEREBRAL CYSTICERCOSIS PRESENTING AS PROGRESSIVE DEMENTIA

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Cysticercosis is a parasitic infestation that commonly affects the central nervous system. This is a report of an elderly man who presented with progressive dementia, labelled as Alzheimer's disease, in whom diffuse cerebral cysticercosis was subsequently demonstrated. The dementia responded favorably to treatment with steroids and mebendazole. This report demonstrates the importance of excluding cerebral cysticercosis as a treatable cause of progressive dementia.

Keywords: cysticercosis, dementia, Alzheimer's disease, mebendazole, steroids, seizures

Alzheimer's disease is the most common cause of dementia in the elderly (Terry, 1976). While this condition is irreversible, recognition and prompt therapy of the so-called "reversible" condition of dementia is of utmost importance

We recently cared for a patient whose history and examination led to an admission diagnosis of Alzheimer's disease, but who ultimately proved to have cerebral cysticercosis, a distinctly uncommon and potentially treatable cause of progressive dementia.

CASE REPORT

A 71-year-old man was well until six months prior to admission when he became increasingly forgetful, with periods of confusion and disorientation, "unintelligible" speech and aimless wandering. Admission blood pressure was 150/90 mmHg, pulse rate 70/min and regular, and a temperature of 37°C. He was alert, but disoriented to place and time, showed inappropriate abnormal responses such as giggling, and was unable to recall any of three unrelated objects after three minutes. He could repeat five digits forward and three in reverse. There was no obvious language deficit. He was pleasant and cooperative and seemed unaware that his memory was impaired. Frontal lobe release signs (snout, palmomental, glabellar tap) were present bilaterally. The rest of the examination was normal, except for absent ankle jerks bilaterally and a slow hesitant gait.

Laboratory investigations including full blood count, serum electrolyte estimations, urea and glucose, coagulation profile, liver and thyroid function tests, serum vitamin B_{12} and folate, syphilis serology were normal or negative. Electrocardiogram and radiograms of the skull, chest and thighs were normal. A purified protein derivative (intermediate strength) skin test was negative. Lumbar puncture yielded clear fluid under normal opening pressure (150 mm H_2O);

analysis showed protein 0.88 g/l, glucose 3.1 mmol/l, chloride 123 mmol/l, no cells or bacteria. An initial diagnosis of senile dementia, probably of the Alzheimer's type, was made.

Three days later he was observed to have a grand mal seizure and developed pyrexia (38°C). The right pupil was 5 mm in diameter and sluggishly reactive to light, the left was 3 mm and normally reactive. There were no other localizing signs,

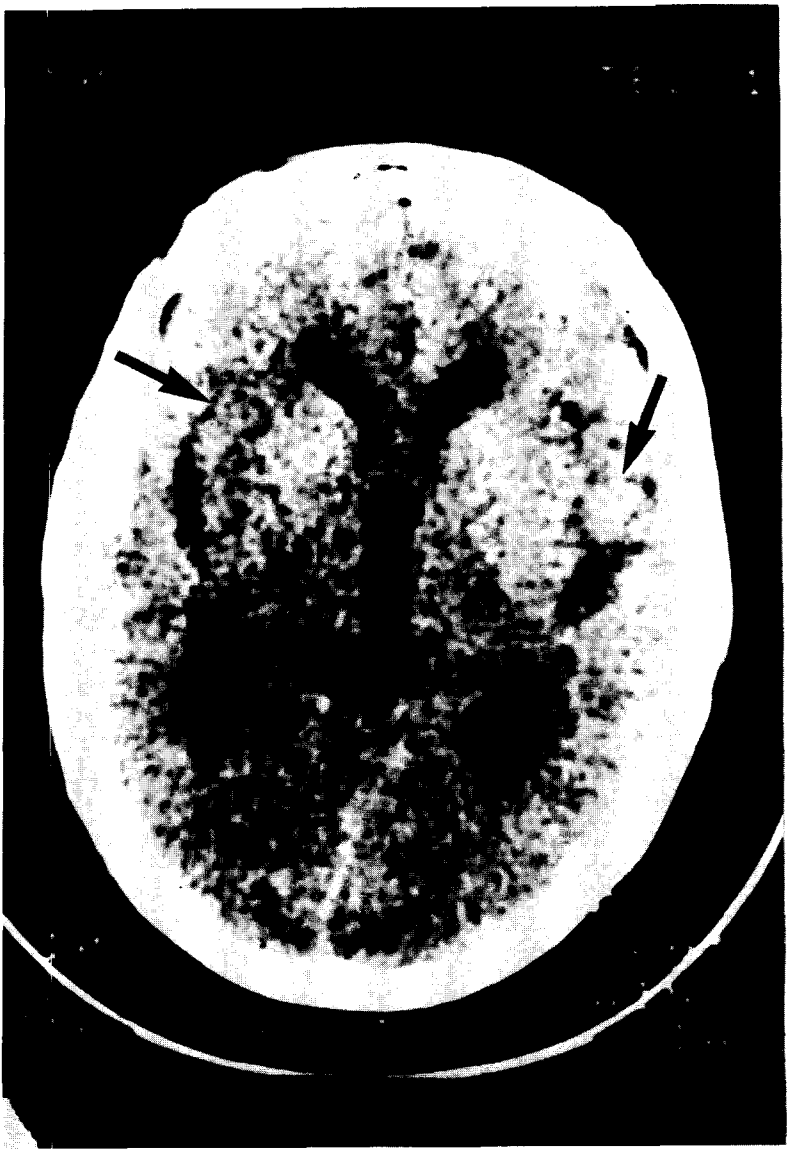

FIGURE 1 CT scan (without contrast administration) reveals enlargement of the lateral ventricles without midline shift, and the presence of multiple areas of increased attentuation surrounded by ringlike areas of increased density; the lesions were most concentrated in the left temporal and occipital lobes and right temporal lobe.

papilledema or nuchal rigidity. A CT scan of the head without contrast disclosed enlargement of the lateral ventricles without midline shift, and the presence of multiple areas of increase density; the lesions were most concentrated in the left temporal and occipital lobes and right temporal lobe (Figure 1). Infusion of a test dose of contrast medium produced vomiting and brochospasm; the procedure was abandoned. A ^{99m}Tc diphosphonate bone scan and a ^{99m}Tc sulfur-colloid liver–spleen scan were normal. A second specimen of CSF was negative for syphilis serology, cryptococcal antigen, cultures for bacteria, fungi and tubercle bacilli, but strongly positive for antigens associated with *T. solium* cysts. Microscopic examination of a stool specimen disclosed no ova or parasites.

Treatment was started with mebendazole (Vermox®) 200 mg twice daily for two weeks, prednisone 40 mg per day for seven days reducing the dosage to 10 mg per day over three weeks and phenytoin (300 mg daily). Surgical intervention was not advised.

The patient was seen one month later. He had not had any further seizures, and had no focal neurological deficit. He was oriented to place and time and fairly normal emotionally. His memory was still poor, but his daughter reported that he functioned considerably better, since initiation of the therapy.

DISCUSSION

Cerebrospinal cysticercosis produces a wide variety of neurological signs and symptoms (Bickerstaff, 1955; Trelles, 1961; Obrador, 1982; Stepien & Chorobski, 1949; Pawlowski & Schultz, 1972). Cysts can be meningobasal (39%), parenchymal (20%), intraventricular (17%), mixed (23%) or intraspinal (1%) in location (Carbajal et al., 1977). Symptoms usually develop within four years of the parasitic infestation, with ranges varying from a few months to many years. Symptoms and signs produced by intracranial cysticercosis are dependent on the site, number, and location of the lesions. The clinical manifestations may be quite complex, because obstruction from posterior fossa lesions may occur in concert with supratentorial signs, owing to multiple cysts in the cerebral hemispheres (Bickerstaff et al., 1956). In addition, brainstem dysfunction can coexist with obstructive signs or with signs of recurrent and chronic meningitis. Characteristic features of multiple cysticercosis within the cerebrum include recurrent convulsions, either focal or generalized and variable, other localizing signs depending on the site of the lesions. In some patients, the outstanding features are those of a posterior fossa syndrome caused by a racemose cysticercus within the fourth ventricle. Headache, vomiting, papilledema, and ataxia are the cardinal symptoms, but many patients have had a prior history of seizures or other cerebral signs before the posterior fossa localization becomes evident (Bell & McCormick, 1975).

In our case, cerebral cysticercosis presented with a picture resembling progressive senile dementia. There has been only one previous report of diffuse parenchymal involvement in cerebral cysticercosis producing progressive dementia (Case Records, 1977). In our case mental deterioration was mainly caused by an encephalitic form of the disease. This is supported by the very strong positive CSF serological test.

Treatment of cerebral cysticercosis has been generally unsatisfactory. Antiparasitic agents, except praziquantel, have proven to be of little value (Bell & McCormick, 1975). The role mebendazole played in the management of this particular case may have been beneficial and needs to be evaluated further. In

addition, administration of sterioids also has been shown to be useful in the management of cerebral cysticercosis (Case Records, 1977).

The present case illustrates the importance of excluding secondary conditions including cerebral cysticercosis in the differential diagnosis of progressive dementia.

REFERENCES

Bell, W. E., & McCormick, W. F. (1975). *Neurologic infections in children.* Philadelphia: W. B. Saunders Company, Ch. 19, pp. 351–381.

Bickerstaff, E. R. (1955). Cerebral cysticercosis: Common but unfamiliar manifestations. *British medical Journal, 1,* 1055–1058.

Bickerstaff, E. R., Small, J. M., & Woolf, A. L. (1956). Cysticercosis of the posterior fossa. *Brain, 79,* 622–634.

Carbajal, J. S., Palacios, E., Azar-kia, B., & Churchill, R. (1977). Radiology of the central nervous system including computed tomography. *Radiology, 125,* 127–131.

Case Records of the Massachusetts General Hospital (1977). Case 40. *New England Journal of Medicine, 297,* 773–780.

Obrador, S. (1962). Cysticercosis cerebri. *Acta Neurochirurgie, 10,* 320–364.

Pawlowski, Z., & Schultz, M. G. (1972). Taeniasis and cysticercosis (Taenia saginata). *Advances in Parasitology, 10,* 269–343.

Stepien, L., & Chorobski, J. (1949). Cysticercosis cerebri and its operative treatment. *Archives of Neurology and Psychiatry, 61,* 499–527.

Terry, R. D. (1976). Dementia. *Archives of Neurology, 33,* 1–3.

Trelles, J. O. (1961). Cerebral cysticercosis. *World Neurology, 2,* 488–497.