Singapore Med J 1997; Vol 38(7):297-299

# Neurocysticercosis – Case Report and Literature Review

A S C Wong, K H Ho


## ABSTRACT

**A 30-year-old Indian migrant worker presented with seizures at the National University Hospital. A CT-scan of the brain showed multiple calcifications and cysts consistent with neurocysticercosis. Plain radiographs of the humeri and femora also revealed multiple soft tissue calcifications. He was given a course of anti-helminthic therapy and started on anti-epileptics. Neurocysticercosis is a common cause of seizures in endemic areas and must be considered in the differential diagnosis of epilepsy involving the members of the large community of migrant workers in Singapore.**


**Keywords: epilepsy, cerebral calcifications, anthelmintic therapy**

## INTRODUCTION

Cysticercosis is the commonest parasitic infection of the central nervous system in patients who are not overtly immunosuppressed. Neurocysticercosis is the most frequent cause of adult seizures in regions where the disease is endemic: this includes the developing countries of Latin America, Asia and Africa[1]. Humans become carriers of *Taenia solium* tapeworm after ingesting undercooked pork containing *T solium* larvae; the disease is acquired by the ingestion of *T solium* eggs through contaminated food, water or by faecal-oral autoinfection. Cysticerci develop in various internal organs, showing predilection of the eye, skeletal muscle and brain.

## CASE HISTORY

Mr M U, a 30-year-old Indian contract worker, was admitted to NUH on 28 May 1996 for 2 episodes of generalised tonic-clonic fits. He gave a history of epilepsy diagnosed in India a year ago. The seizures at that time consisted of clonic movements of the left upper limb. He was initially given anti-epileptic therapy but had defaulted treatment after 2 months. Clinical examination was essentially unremarkable.

Initial blood investigations were normal except for mild eosinophilia. (Absolute eosinophil count was 671/mm$^3$.) The EEG showed bilateral frontal/central epileptogenic activity.

CT scan of the head showed several calcified densities scattered in both hemispheres located at or close to the gray-white junction (Fig 1). Two small non-enhanced cysts were identified – one in the right parietal region and one in the right occipital region. An area of oedema in the left frontal region showed a small nodule of enhancement related to it on the contrast film. Subsequent plain X-rays of the humeri and femora revealed multiple elongated oval soft tissue calcifications (Fig 2).

Blood for cysticercosis IgG antibody was positive at 0.587 (positive range >0.5) using an ELISA test. The stool examination was incidentally positive for hookworm ova.

The patient was diagnosed as a case of neurocysticercosis in view of the typical radiographic features and the positive serology. He was given a 2-week course of praziquantel and was also started on phenytoin. The patient was subsequently discharged on 5 June 1996 and has since been lost to follow-up.

## DISCUSSION

### Clinical presentation

The manifestations of neurocysticercosis are varied. They include seizures, meningeal inflammation, hydrocephalus, 'stroke' syndromes and spinal cord compression. The eye is involved in 20% of cases. Clinical signs and symptoms of illness depend on the number, size and location of the CNS lesions, and may progress as a result of mass effect, inflammation and obstruction of CSF drainage. Patients with

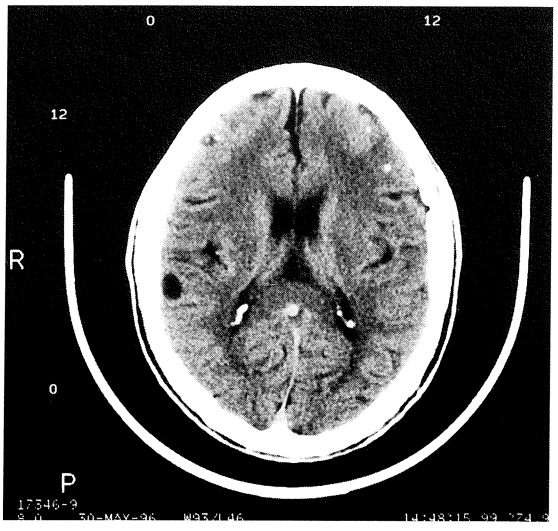


**Fig 1** - CT scan of the patient showing cysts and calcifications.

Department of Medicine
National University Hospital
5 Lower Kent Ridge Road
Singapore 119074

A S C Wong, MBBS
Medical Officer

K H Ho, MBBS, MRCP
Senior Registrar

Correspondence to:
Dr A S C Wong

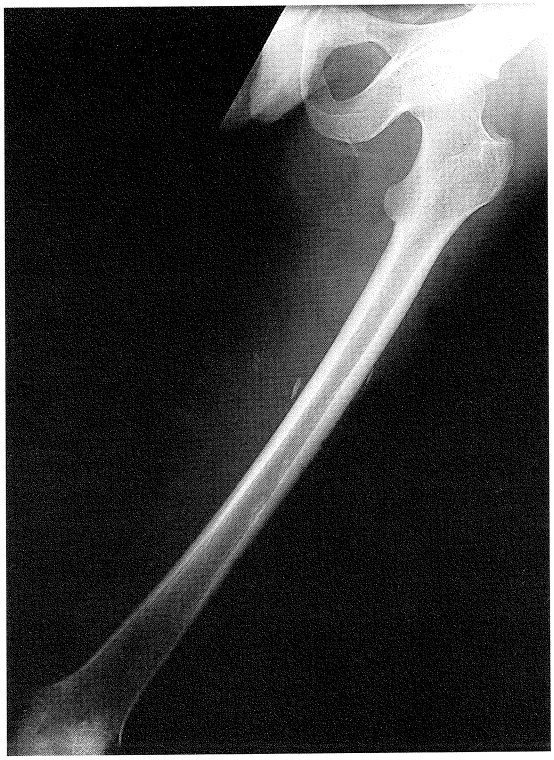

**Fig 2** - Characteristic soft tissue calcifications of cysticercosis.

parenchymal lesions most often present with seizures (as in this case), while 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[2]. Inflammatory reactions are greatest when the larvae die either naturally or as a result of cysticidal drugs. By contrast, living larvae and fully calcified lesions cause very little tissue reaction. A common cause of death is from acute hydrocephalus due to intraventricular cysts.

## Diagnosis

The diagnosis of neurocysticercosis does not depend on finding the ova of *T solium* in stool, since only a minority of adults would harbour the tapeworms in the gut by the time of presentation. Skull X-rays have been largely superseded by more advanced imaging techniques although soft tissue X-rays demonstrate characteristic 'rice-grain' calcifications in up to 50% of patients. CT scanning in adults is more likely to demonstrate older lesions with little contrast enhancement and considerable calcification. Children tend to have more acute lesions with cysts demonstrating contrast enhancement due to surrounding oedema and inflammation. MRI is more sensitive in detecting non-calcific cerebral lesions such as cysts and cerebral oedema, and may show pedunculated larvae within the cysts. CT scanning is however a more sensitive method in detecting parenchymal calcifications[3].

Serological testing for antibodies in serum and CSF is particularly useful in areas of low prevalence, since cross-reactivity between cysticercosis, echinococcus, schistosomiasis and other cestode infections may be less of a problem. Techniques include complement fixation, hemagglutination inhibition and ELISA to detect IgG or IgM antibodies. Serology is more likely to be positive in active disease. The enzyme-linked immunoelectrotransfer blot assay detects antibody to *T solium* in CSF and serum with 95% sensitivity and 100% specificity[4]. In the appropriate clinical setting, the cerebrospinal fluid shows a picture of chronic meningitis (occasionally with eosinophils), myelograms may demonstrate spinal cord compression, while biopsy of an obvious subcutaneous nodule provides accurate histological confirmation of the disease. Histological diagnosis is however, rarely necessary.

Our patient demonstrated classical radiologic features of neurocysticercosis on both CT-scan and soft tissue radiographs.

## Treatment

The use of anthelmintic agents is indicated in cases where there is evidence of disease activity, including presence of parenchymal cysts and meningitis. Praziquantel is the traditional agent of choice, and is active against larval forms as well as any adult worms that may be present in the gut. In a series by Sotelo et al, the administration of praziquantel (50 mg/kg/day for 15 days) saw a reduction in mean cyst number by 67% and reduction in mean cyst diameter by 73% after 1 year of follow-up[5]. Absence of radiographic improvement may warrant a second course of treatment.

Albendazole has been used as an alternative drug to praziquantel since 1987, and recent evidence favours the former as the drug of choice for the active form of disease. In 2 controlled trials, albendazole was significantly superior to praziquantel in reducing the number of cysts detectable by CT (88% vs 50%; $p<0.001$) with one trial also showing a better clinical outcome[6,7]. Cost reduction is an added advantage. An 8-day course of albendazole (15 mg/kg/day) has been shown to be as effective as the previously used 30-day course[8]. Albendazole has also been shown to be effective in eradicating small subarachnoid cysts, which do not generally respond to praziquantel.

During therapy with praziquantel or albendazole, nausea, headache and vomiting are common because of the increased inflammation due to the process of destruction of the parasites. The CSF may show increases in cell counts, protein and a conversion to positive antibody titres while repeat CT scans may show increased ring-enhancement of cysts. Although some authors recommend the simultaneous use of dexamethasone to prevent these reactions, steroids should probably not be used routinely with albendazole as they can lower plasma levels of this drug by up to 50%. Furthermore, most of the reactions are mild and transient and subject to modification by analgesic and anti-emetic drugs. Steroids should probably be reserved for cases where intracranial hypertension develops during anthelmintic therapy. This complication is however, rare and occurs mainly in patients who have multiple cysts or lesions already surrounded by oedema before treatment[9].

Patients with epilepsy due to parenchymal brain cysts should be given anti-epileptics regardless of the specific therapy for neurocysticercosis. A first-line anti-epileptic drug usually results in adequate seizure control, provided that anti-cysticercal drugs have also been given. A recent study of 203 patients showed seizure control in 83% of patients receiving anti-cysticercal drugs versus only 27% in those who did not receive these drugs[10].

Surgery is indicated in cases of ventricular cysts, large mass lesions causing CSF obstruction or simulating tumours, and spinal cord lesions causing compression. Ventriculo-atrial/peritoneal shunting may be required for obstructive hydrocephalus.

In Monteiro et al's retrospective study of 38 cases[11], it was demonstrated that 2 subtypes of active neurocysticercosis (active parenchymal vs extra-parenchymal disease) had strikingly different clinical presentations, responses to medical therapy, complications, morbidity and mortality. In the parenchymal disease group, response to anti-parasitic agents was complete in 81% of the cases. In contrast, all 13 cases of extraparenchymal disease showed poor response to cysticidal drugs. Nine of these 13 patients required ventriculo-peritoneal shunting. Severe complications, including 2 deaths associated with the natural evolution of the disease and with surgery, occurred only in the extra-parenchymal disease group.

Inactive disease (calcifications) generally require only symptomatic treatment eg anti-convulsants for seizures[9].

Prevention of cysticercosis necessitates the screening of family members, especially children, and the proper handling of pork including cooking and freezing.

## CONCLUSION

Although the disease is not endemic in Singapore, the presence of various communities of foreign workers from areas where cysticercosis is prevalent implies the need to have a high index of suspicion in an appropriate clinical context. Radiologic features establish the diagnosis in the majority of cases. The varied manifestations of the disease, different approaches to treatment, and the individual variability in prognosis constitute a therapeutic challenge for physicians.

## REFERENCES


1. Senanayake N, Roman GC. Epidemiology of epilepsy in developing countries. Bull World Health Organ 1993; 71(2): 247-58.
2. Scharf D. Neurocysticercosis: two-hundred thirty-eight cases from a California hospital. Arch Neurol 1988; 45:777-80.
3. Suss RA, Maravilla KR, Thompson J. MR imaging of intracranial cysticercosis: comparison with CT and anatomopathologic features. Am J Neurorad 1986; 7:235-42.
4. Garcia HH, Martinez M, Gilman R, et al. Diagnosis of cysticercosis in endemic regions. Lancet 1991; 338:549-51.
5. Sotelo J, Torres B, Rubio-Donnadieu F, et al. Praziquantel in the treatment of neurocysticercosis: long-term follow-up. Neurology 1985; 35:752-5.
6. Cruz M, Cruz I, Horton J. Albendazole versus praziquantel in the treatment of cerebral cysticercosis: clinical evaluation. Trans R Soc Trop Med Hyg 1991; 85:244-7.
7. Takayanagui OM, Jardim E. Therapy for neurocysticercosis comparison between albendazole and praziquantel. Arch neurol 1992; 49:290-4.
8. Sotelo J, Del Brutto OH, Penagos P, et al. Comparison of therapeutic regimen of anticysticercal drugs for parenchymal brain cysticercosis. J Neurol 1990; 237:69-72.
9. Del Brutto OH, Sotelo J, Roman GC. Therapy for Neurocysticercosis: A Reappraisal. Clin Infect Dis 1993; 17(4): 730-5.
10. Medina MT, Cordova S, Genton P, et al. Prognosis of epilepsy in neurocysticercosis: the effect of anticysticercal treatment. Epilepsia 1991; 32(suppl 1): 110-1.
11. Monteiro L, Almeida-Pinto J, Stocker A, et al. Active neurocysticercosis, parenchymal and extraparenchymal: a study of 38 patients. J Neurol 1993; 241(1):15-21.