

The Value of MRI in the Diagnosis and Management of Neurocysticercosis

S H Ng, T Y Tan, K M Fock

ABSTRACT

The clinical manifestation of neurocysticercosis is quite protean and variable making it the 'great imitator' of almost any neurological disorder. In the last decade, developments in diagnostic imaging and effective anticysticercus drug therapy have changed the outlook of the disease. Two cases investigated with CT scan and MRI are reported here. One case was treated effectively with antihelminthic therapy. In both cases MRI was found to be much more sensitive than CT scan in picking up the multiple cystic lesions. In addition, 'protoscoleces' and 'differential ring enhancements' not apparent in CT scan were well shown in MRI hence enabling the demonstration of different activity stages. In the treated patient, repeat MRI showed degeneration of the cyst. These illustrate that MRI is superior to CT scan in both the diagnosis and management of the illness.

Keywords: neurocysticercosis, epilepsy, MRI, CT scan, antihelminthic therapy

INTRODUCTION

Cysticercus is the encysted larva of the cestoda *Taenia Solium*. The life cycle of *Taenia Solium* starts in the intestine of taenia carriers where several mature proglotides are released daily, each containing thousands of fertilized ova which in turn will contaminate food. Ingestion of contaminated food will induce cysticercosis in humans or pigs. By far the most common form of cysticercosis in man is neurocysticercosis which is probably the most common parasitic disease of the central nervous system and is endemic in Mexico, Central and South America, India and China⁽¹⁾. Here in Singapore, a place with frequent visitors and expatriates from neighbouring countries, neurocysticercosis is likely to be encountered by us occasionally. In the last decade, developments in diagnostic imaging and effective anticysticercus drug therapy have changed the outlook for neurocysticercosis⁽²⁾. These are well illustrated by the 2 cases reported here recently encountered by the authors with particular emphasis on the value of magnetic resonance imaging (MRI).

CASE REPORTS

Case 1

A 32-year-old Ghurka gentleman who had been residing in Singapore for the past 15 years presented with acute generalised tonic-clonic seizures in March 1996. Screening tests including chest X-ray, ECG, routine urine analysis, blood for full blood count, ESR, renal function tests and liver function tests and computerized tomography (CT) brain scan (including intravenous contrast) were all normal. The EEG study showed occasional slow and sharp waves over the left hemisphere. He was started on Phenytoin. However, in the subsequent months, he still experienced headaches on and off and repeated attacks of epileptic seizures resulting in several hospital admissions. Hence he was assessed by a neurologist in August 1997. Brain MRI was done. It showed multiple small cystic lesions of different signal characteristics scattered in both cerebral hemispheres, particularly the grey-white matter junction showing differential enhancement highly suggestive of neurocysticercosis of different stages of activity including 'protoscolex' in some cysts (Fig 1). X-ray of his lower limbs showed 'cigar-shaped' calcifications (Fig 2). Cerebrospinal fluid (CSF) study was normal apart from mildly raised protein level of 0.64 g/Litre. Anticysticercal antibodies in serum and CSF by the enzyme-linked immunosorbent assay (ELISA) method were negative.

He was treated with Praziquantel at dose of 50 mg/Kg/day in 3 divided doses for 15 days together with Prednisolone 30 mg a day. Brain MRI repeated 3 months after treatment showed transformation of the active 'vesicular' stage of cysts to the degenerating stages with evidence of cyst retraction (Fig 3). Meanwhile his headache was less and epilepsy under full control with Phenytoin at the dose of 200 mg/day with serum level within the therapeutic range.

Case 2

A 46-year-old Thai expatriate who had been working in Singapore for the past 3 years presented acutely with generalized tonic-clonic seizures in October 1997. Screening tests including chest X-ray, ECG, routine urine analysis, blood for full blood count, ESR, renal function tests and liver function tests were all normal. EEG showed paroxysmal sharp waves over both frontal regions with occasional spikes. CT brain

Department of Medicine
Changi General Hospital
2 Simei St 3
Singapore 529889

S H Ng, FRCP (Edin)
Consultant Neurologist

K M Fock, FRCP (Edin)
Chief of Medicine

Department of Radiology
Changi General Hospital

T Y Tan, FRCP
Consultant

Correspondence to:
Dr S H Ng

Dept of Medicine
Tuen Mun Hospital
Tsing Chung Koon Road
Tuen Mun
New Territories
Hong Kong

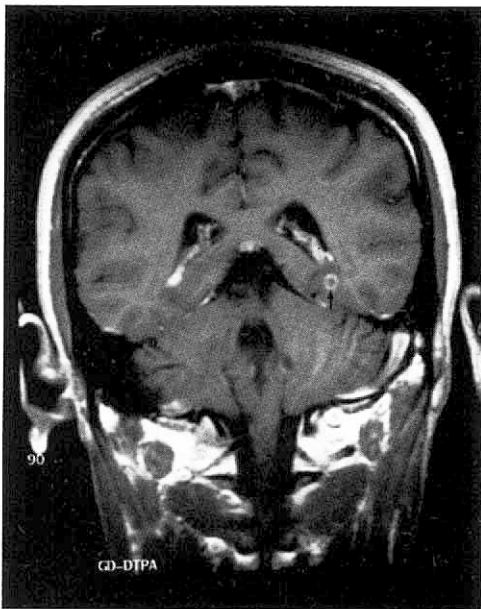


Fig 1 – A cystic lesion with minimally enhancing wall, representing the active vesicular stage of neurocysticercosis, is seen in the medial aspect of the left temporal lobe (arrowhead). There is no significant adjacent oedema. The protoscolex is seen as a nodule within the cyst.



Fig 2 – Cigar-shaped calcifications representing *Taenia Solium* infection are seen behind the right upper tibia. The white arrow points to one of the calcifications.

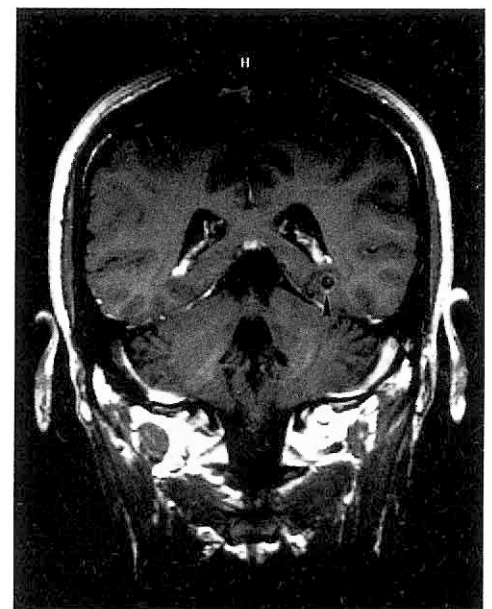


Fig 3 – Follow-up MRI after treatment of the same patient in Fig 1 shows that the lesion has entered the granular nodular stage where the cyst has become smaller in size, the wall is now thicker and more intensely enhancing, and the mural nodule has disappeared (arrow).

scan showed a focal area of unenhanced low density in the white matter of the left occipital lobe. X-ray of his left arm showed 'cigar-shaped' calcifications. Subsequently brain MRI showed a ring enhanced cystic lesion in the left occipital lobe with surrounding oedema (Fig 4). In addition, a small unenhanced cystic lesion with 'protoscolex' was noted at the left frontal lobe, suggestive of neurocysticercosis with both active and degenerating cysts. CSF study was normal and anticysticercal antibodies in serum and CSF by the ELISA method were negative. Antihelminthic therapy was advised but his employer did not support this and he was repatriate to Thailand for further medical treatment.

DISCUSSION

Seizures, pyramidal tract dysfunction, increased intracranial pressure and intellectual deterioration are the most frequently recognised clinical manifestations of neurocysticercosis⁽³⁾. In fact, our 2 cases both presented with seizures. The latter, being the most common clinical manifestation, is present only in 50% of cases and about 25% of all patients have a normal neurological examination⁽⁴⁾. Thus, a high index of suspicion for this condition is important for diagnosis. Del Brutto has recently proposed diagnostic criteria for human cysticercosis⁽⁵⁾. The absolute criteria include (a) histologic demonstration of the parasite, (b) direct visualisation of the parasite by fundoscopic examination, (c) evidence of cystic lesions showing the scolex on CT or MRI. Major criteria include (a) evidence of lesions suggestive of neurocysticercosis on neuroimaging studies, (b) positive immunologic tests for the detection of anticysticercal antibodies, (c) plain X-ray films showing 'cigar-shaped' calcifications in

thigh and calf muscles. Presence of one absolute or two major criteria will give a definite diagnosis. Hence, our 2 cases both fit into the criteria for definite diagnosis.

Serological tests have been developed for the detection of anticysticercal antibodies in serum and CSF⁽⁶⁾. However, most of these tests lack both sensitivity and specificity. They were negative in our 2 cases using the ELISA method although current data indicate that the most effective immunologic diagnostic test is the serum immunoblot method⁽⁵⁾.

Recent studies showed the neuroimaging findings in CT scan or MRI in neurocysticercosis are quite often characteristic⁽⁷⁻¹¹⁾. The initial infection results in a small minimally enhanced thin walled cystic lesion, the active 'vesicular stage', with little if any surrounding oedema (Fig 1). The protoscolex may be identified as a mural nodule within the cyst. As the larva dies the host inflammatory response results in the formation of a fibrous capsule that often demonstrates ring enhancement with proteinaceous fluid and debris accumulated within the cyst and surrounding oedema, the 'colloidal vesicular stage' (Fig 4). It then goes to the 'granular nodular stage' where the cyst retracts and forms a granulomatous nodule, which may show ring or solid enhancement (Fig 3). In the final 'nodular calcified stage', the lesion becomes mineralised. The granulomatous nodule is replaced by gliosis and eventually calcification. Thus neuroimaging is not only useful for initial diagnosis but also for showing the different activity stages. The latter not only helps in monitoring the response to antihelminthic therapy as illustrated in Case 1 here but also in selecting patients for such therapy as some suggest that it may be appropriate to treat patients with the active 'vesicular stage' only⁽¹²⁾.



Fig 4 – Colloidal vesicular stage of neurocysticercosis of another patient showing a ring lesion (arrowhead) in the left occipital lobe which has an intensely enhancing wall associated with adjacent low signal changes representing oedema. A faint mural nodule can still be seen.

In case one, the multiple cystic lesions were completely missed by CT scan but picked up in MRI. In the second case, the CT scan only showed a focal area of low density oedema in the left occipital lobe but not the ring enhanced cystic lesion at the centre of the oedema which was well demonstrated in MRI. In addition 'protoscoleces' were shown only on MRI in both cases. Although recent studies suggest that neurocysticercal cysts are usually readily apparent on CT scan and MRI, measuring 5 mm to 20 mm in diameter⁽⁸⁾, our two cases illustrate that MRI is superior to CT scan when the cyst sizes are small.

Albendazole and Praziquantel have potent cestocidal properties^(2,13,14) Albendazole was found to have better penetrance into the subarachnoid space⁽¹⁵⁾. In fact, we planned to treat Case 2 with Albendazole. Unfortunately, he had to be repatriated to Thailand. Case 1 was treated with Praziquantel because we are more experienced in using the latter drug. Because the efficacy of anticysticercal drugs was initially evaluated by counting the percentage of cyst destruction on imaging, some authors have argued that these drugs only improve the imaging without modifying the clinical course of the disease^(12,16). However, recently it has been noted that the control of seizures in patients with neurocysticercosis is better after a course of anticysticercal drug than when the disease is left untreated⁽¹⁷⁻¹⁹⁾. Moreover the chance of remaining seizure-free after the withdrawal of anti-epileptic drugs seems to be greater in those patients who were previously treated with anticysticercal drugs⁽²⁰⁾.

However, it is to be noted that two forms of active cysticercosis, intra-ocular and intra-ventricular cysts, do not respond to medical therapy because drug levels are extremely low in ventricular and ocular fluids. Surgical extirpation in both forms is mandatory.

CONCLUSION

The two cases reported here well illustrate that neuroimaging in the form of MRI plays a vital role in the diagnosis and management of neurocysticercosis.

ACKNOWLEDGEMENTS

The authors wish to thank Ms Maudrene Lee for the preparation of the manuscript.

REFERENCES

1. Davis LE, Kornfeld M. Neurocysticercosis: neurologic, pathogenic, diagnostic and therapeutic aspects. *Eur Neurol* 1991; 31:229-40.
2. Sotelo J, Del Brutto OH, Roman GC. Cysticercosis. Current clinical topics in infectious diseases, Vol 16. Edited by Remington JS, Swartz MN. Cambridge: Blackwell Science; 1996:240-59.
3. Wadia NH. Neurocysticercosis. Tropical Neurology. Edited by Shakir RA, Newman PK, Poser CM. London: WB Saunders Co; 1996:247-73.
4. McCormick G, Zee C, Heiden J. Cysticercosis Cerebri. *Arch Neurol* 1978; 39:534-9.
5. Del Brutto OH. Neurocysticercosis. *Curr Opin Neurol* 1997; 10:268-72.
6. Richards F Jr, Schantz PM. Laboratory diagnosis of cysticercosis. *Clin Lab Med* 1991; 11:1011-28.
7. Kramer LD, Locke GE, Byrd SE, Daryabagi J. Cerebral Cysticercosis: Documentation of Natural History with CT. *Radiology* 1989; 171:459-62.
8. Lotz J, Hewlett R, Albeit B, Bowen R. Neurocysticercosis: correlative pathomorphology and MR imaging. *Neuroradiology* 1988; 30:35-41.
9. Suss RA, Maravilla KR, Thompson J. MR imaging of intracranial cysticercosis: comparison with CT and anatomopathologic features. *AJNR* 1986; 7:235-42.
10. Spickler EM, Lufkin RB, Teresi L, Lanman T, Levegue M, Benston JR. High signal intraventricular cysticercosis on T1 weighted MR imaging. *AJNR* 1989; 10:S64.
11. Chang K-H, Lee JH, Han MH, Han MC. The role of contrast-enhanced MR imaging in the diagnosis of neurocysticercosis. *AJNR* 1991; 128:509-12.
12. Kramer LD. Medical treatment of Cysticercosis - Ineffective. *Arch Neurol* 1995; 52:101-2.
13. Sotelo J, Del Brutto OH, Penagos P, Escobado F, Torres B, Rodriguez, Carbajal J, et al. Comparison of therapeutic regimen of anticysticercal drugs for parenchymal brain cysticercosis. *J Neurol* 1990; 237:69-72.
14. Corona T, Lugo R, Medina R, Sotelo J. Single-day praziquantel therapy for neurocysticercosis [letter]. *N Engl J Med* 1996; 334:125.
15. Jung H, Hurtado M, Sanchez M, Medina MT, Sotelo J. Plasma and CSF levels of albendazole and praziquantel in patients with neurocysticercosis. *Clin Neuropharmacol* 1990; 13:559-64.
16. Garg RK, Agrawal A. Medical treatment for cysticercosis [letter]. *Arch Neurol* 1996; 53:295.
17. Del Brutto OH, Santibanez R, Noboa CA, Aguirre R, Diaz E, Alarcon TA. Epilepsy due to neurocysticercosis: analysis of 203 patients. *Neurology* 1992; 42:389-92.
18. Vazquez V, Sotelo J. The course of seizures after treatment for cerebral cysticercosis. *N Engl J Med* 1992; 327:696-701.
19. Cruz I, Cruz ME, Carrasco F, Horton J. Neurocysticercosis: optimal dose treatment with albendazole. *J Neurol Sci* 1995; 133:152-4.
20. Del Brutto OH. Prognostic factors for seizure recurrence after withdrawal of antiepileptic drugs in patients with neurocysticercosis. *Neurology* 1994; 44:1706-9.