Classic diseases revisited

Neurocysticercosis

Ravindra Kumar Garg

Summary
Neurocysticercosis is the most common parasitic disease of the central nervous system. Varied clinical manifestations occur, due to deposition of larvae of the parasite *Taenia solium* in cerebral parenchyma, meninges, spinal cord, muscles, eyes and skin. The diagnosis of neurocysticercosis can be made with a fairly high degree of accuracy with the help of computed tomography and magnetic resonance imaging. Serological tests and histopathological examination of subcutaneous nodules provide additional support in establishing the diagnosis. The anticysticercal drugs albendazole and praziquantel have been extensively used, and found to be effective for all types of neurocysticercosis. However, recently controversy has been raised about their safety, and long-term clinical usefulness. Preventive health measures, such as provision of safe drinking water and excretion disposal, still offer the best ways to manage this disease.

Keywords: neurocysticercosis; *Taenia solium*

Neurocysticercosis: classification
<ul style="list-style-type: none">● parenchymal● subarachnoidal● ventricular● spinal● muscular● disseminated

Box 1

Division of Neurology, Institute of Medical Sciences, Banaras Hindu University, Varanasi, Pin-221 005, India
R K Garg

Accepted 24 October 1997

Neurocysticercosis is the most common parasitic disease of the central nervous system. The disease in humans is caused by the larval form of the tapeworm *Taenia solium*. Neurocysticercosis has a world-wide distribution and is endemic in most of the developing world, especially in Latin-America, the Indian subcontinent, China, and most of Asia and Africa. It also occurs in industrialised nations, largely as a result of the immigration of infected persons from the endemic areas. In countries where the disease is endemic, cysticercosis may affect 2–4% of general population.¹ Sero-epidemiological studies in India have demonstrated that approximately 2–3% of the general population have anticysticercal antigen antibodies in their serum.² The highest prevalence of cysticercosis is in communities where there is a close contact between humans and pigs, and where hygiene standards are low. It is a disease produced by poor sanitation, lack of a proper water supply and sewage system, and poor personal hygiene.

The parasitic life-cycle and pathogenesis

In the normal life-cycle of *T. solium*, the adult tapeworm, which is 2–8 m in length, resides in the human intestine (definitive host). The worm is attached to the mucosa of the small bowel by several pairs of scolex which are arranged over the head. The terminal egg-bearing segments or proglottids are periodically discharged in the stools. After ingestion by the intermediate host (pig), the eggs are subject to digestion by gastric juices and develop into hexacanth larvae (oncospheres) which penetrate the small bowel and are deposited throughout the body of the pig, especially in the skeletal muscles. The oncosphere then develops into cysticercal or encysted larva. When humans consume raw or ill-cooked pork, the cysticercal larvae develop into adult tapeworms. In cysticercosis, humans acquire infection through the faecal–oral route by consuming contaminated food (eg, raw vegetables) or water. It was previously incorrectly assumed that cysticercosis was acquired mainly through eating raw pork, but it is now clear that any uncooked food may be contaminated. Thus the disease is equally likely for both vegetarians and non-vegetarians.^{1 3 4}

A cysticercus is a fluid-filled bladder that contains the invaginated hood or scolex. The cysts may be located in the brain, subcutaneous tissue, muscles, eyes, and rarely spinal cord and other tissues, in decreasing order of frequency. In the human brain, cysticerci are located mainly in the parenchyma of the cerebral cortex, the subarachnoid space, or in the ventricular system (box 1). Cerebral cysticerci are mainly located in grey matter or at the junction of grey and white matter, areas with a rich blood supply. Cysticerci in brain parenchyma may be single or multiple. Cysts are usually 5–10 mm in diameter in soft tissues but may be larger (up to 5 cm) in the brain.^{1 3}

In the brain parenchyma, cysticerci evolve through different stages. Escobar⁵ has categorised these stages (box 2). In the earliest ‘vesicular’ stage the parasite has a thin, friable translucent membrane. Inside, bathed in transparent fluid, is an invaginated larva, 4–5 mm in length, adopting a curled-up position. When the parasite begins to show degenerative changes due either to aging, or to attack by the host’s immune defence mechanisms, the parasite passes into a ‘colloidal’ stage. At this stage the cyst shows hyaline degeneration and early mineralisation. In a more advanced stage the cyst begins to reduce in size, the walls become thicker, and the cyst contents are transformed into coarse granules, due to mineralization with calcium salt. This is referred to as the ‘granular-nodular’ stage. Ultimately the cyst becomes completely mineralised—the ‘calcified’ stage. Little immunological reaction is provoked by intact cysticercal larvae as they are surrounded by a connective tissue membrane of epithelial cells, collagen fibres and some cellular infiltration in which plasma cells and eosinophils predominate. A more pronounced inflammatory reaction in the host is provoked by dying or degenerating cysticerci and there is a granulomatous response which consists of lymphocytes, plasma cells and eosinophils.^{1 4 6} Subarachnoid cysticerci may also be small and scattered, or may form large clumps of cysts that produce tumour-like effects. The ventricular system of the brain is affected by cysticerci in two

Larval stages and corresponding CT picture

- vesicular stage: hypodense, circumscribed cyst (figure 2)
- colloidal stage: ring enhancing (figure 7)
- nodular-granular stage: disc enhancing (figure 3)
- calcified stage: small, hyperdense (figure 4)

Box 2

Neurocysticercosis: clinical manifestations

Common

- seizure disorder

Uncommon

- stroke
- increased intracranial tension
- vision loss
- cranial nerve palsies

Rare

- neuropsychiatric (dementia, psychosis)
- myelopathy

Box 3

Neurocysticercosis: diagnostic methods

Imaging

- X-ray thigh and calf
- CT scan
- MRI

Immunological tests

- ELISA
- immunoelectrotransfer blot assay

Histopathology

- subcutaneous nodule, rarely brain

Box 4

different ways, by the presence of intraventricular cysts and by the development of granular ependymitis. Intraventricular cysts are usually single and tend to lodge in the fourth ventricle, although they can also be found in the third or lateral ventricle. Granular ependymitis usually occurs at the cerebral aqueduct or posterior foramina as a continuation of arachnoiditis. Spinal cysticerci are of two types, most commonly leptomeningeal but occasionally intramedullary. An unusual lobate or 'racemose' form of cysticercus may be seen in the subarachnoid space or in the ventricles. These grape-like multiloculated cysts are usually sterile (lack scolex), and cause marked adhesive arachnoiditis and often obstructive hydrocephalus.^{3 4 7}

Approximately 20% of human cysticercosis cases show an ocular involvement, ocular cysticerci occurring most commonly in the vitreous humor and subretinal tissues, but also in the extra-ocular muscles, anterior chamber, conjunctiva and other eye tissues. The host reactions to cysticerci in eye vary from slight to severe inflammation, together with sequelae such as retinal detachment, chorioretinitis, and iridocyclitis. Larvae in skeletal muscles go through similar but more rapid morphological changes, such that parasite may be alive in the brain but calcified and dead in the muscles.⁴

Clinical manifestations (box 3)

Epilepsy is the most common presentation of neurocysticercosis. Seizures are the presenting manifestation in 92% of patients with intra-parenchymal lesions and in 74% of patients with mixed intra- and extra-parenchymal neurocysticercosis. Usually patients have partial seizures with or without secondary generalisation, although a few patients may have primary generalised seizures. Partial status epilepticus and post-ictal transient focal neurological deficit can also occur. Simple partial seizures are much more common than complex partial seizures.^{7 8}

An acute encephalitic form of neurocysticercosis occurs mainly in children and adolescents and is the result of widely disseminated small intraparenchymal cysts. These patients have frequent seizures and acutely evolving intracranial hypertension.¹ Systemic disseminated neurocysticercosis is seen in India, with frequent seizures, dementia, muscular pseudohypertrophy, few localising signs and often intracranial hypertension.⁹

Focal neurological deficit of vascular origin is another common manifestation of neurocysticercosis. In Latin American countries this complication is frequently observed, and neurocysticercosis is considered a risk factor for stroke in the young. It is usually caused by inflammatory occlusion of the arteries at the base of brain secondary to cysticercotic arachnoiditis. In most patients small penetrating vessels are involved and the associated neurological picture is like lacunar infarction. Larger infarct is related to the occlusion of middle cerebral artery or internal carotid artery. In patients of the latter group, cysticerci are widely distributed in subarachnoid space and are associated with an intense cerebrospinal fluid (CSF) inflammatory profile.^{10 11}

Involvement of spinal cord has variably been reported at 1–5%. Leptomeningeal cysticercosis is frequently seen in association with cysticerci of the posterior fossa due to downward migration of the cysts from basal cisterns of the brain into spinal cord through CSF pathway. Haematogenous spread is responsible for intramedullary (spinal) cysticercosis. The clinical manifestations are directly related to the location of the parasite. Intramedullary cysts cause symmetric or asymmetric motor weakness and a transverse sensory level below the level of lesion along with sphincteric disturbances. Leptomeningeal cysts produce a syndrome of root pain and asymmetrical weakness of subacute onset and progressive course.^{12 13}

Diagnosis

A definite diagnosis of neurocysticercosis requires histopathological examination of biopsied material obtained from a tissue containing the cysticercus larva. However, a reliable diagnosis can still be made on the basis of findings of imaging of brain and other affected structures (box 4).

IMAGING

Plain radiography of the soft tissues may detect oval as well as linear calcified lesions. These lesions are usually multiple and the long axes of the calcified cysts are arranged in the plain of affected muscle fibres (figure 1). Plain X-ray film of skull may at times show cerebral calcifications.

Figure 1 X-ray of forearm showing cigar-shaped calcified lesions lying parallel to muscle fibres

Figure 2 Cranial CT scan showing multiple hypodense cystic lesions (vesicular stage). Eccentric scolex is also visible in few of the lesions

Figure 3 Contrast-enhanced cranial CT scan showing cysticercotic encephalitis. Numerous enhancing lesions are present

Computed tomography

The most useful procedure for the diagnosis of neurocysticercosis is computed tomographic (CT) imaging. The CT picture depends on the number, location and stage of the lesions. In the vesicular stage (viable cysts) the cysts appear circumscribed and hypodense. These lesions do not enhance after contrast administration. Surrounding oedema may be mild or absent. It is not uncommon to find an eccentric scolex, present in about 44% of cases (figure 2). In the colloidal stage, which represents a dying cyst, there is a ring-enhancing lesion surrounded by white matter oedema (figure 3). In the next 'nodular-granular' stage, the lesions are homogeneously enhancing, and ultimately calcify. The calcified stage, representing a dead parasite, appears as a hyperdense lesion on non-contrast CT scans. Where neurocysticercosis is endemic, patients with epilepsy often have single intracranial calcifications (figure 4) (box 2).¹⁴⁻¹⁶

CT findings in subarachnoid neurocysticercosis are as follows:

- hydrocephalus secondary to the inflammatory occlusion of Luschka's and Magendie's foramina
- abnormal enhancement of tentorium and basal cisterns due to arachnoiditis
- cystic hypodense lesions in sylvian fissure, cerebello-pontine angle or pituitary fossa (some of these lesions represent 'racemose' form of neurocysticercosis)
- brain infarct due to cysticercotic endarteritis.^{17 18}

The intraventricular form of cysticercosis presents as rounded areas of low density in the CT scan that deform the ventricular system and interfere with the CSF circulation, resulting in obstructive hydrocephalus.¹ Ventriculography is needed for the confirmation of diagnosis. In disseminated cysticercosis a 'starry-night' appearance is seen (figure 5). This is produced by the hyperdense scolices of live cysticerci standing out against the lower attenuation density value of the brain, the tightly packed cysts contribute little to the image. These scolices can be mistaken for dead calcified cysticercus lesions.⁷

CT scan of muscles can also be used for the diagnosis of cysticercosis. Wadia *et al*⁸ described a 'honey-comb' appearance produced by a large number of live cysticerci in pseudohypertrophic muscles. The classical appearance is produced by the low attenuation density of the cysts.

Magnetic resonance imaging

Magnetic resonance imaging (MRI) shows living parenchymatous cysts, 5–20 mm in diameter, as round lesions of CSF-equivalent density on both T1- and T2-weighted images (figure 6). An isodense to hyperdense scolex can be identified within most cysts producing a 'pea in the pod' appearance.⁷ In dying cysticerci the difference between scolex and cysts becomes unclear. The fluid shows greater and increasing signal intensity than CSF, in both T1- and T2-weighted images. MRI has been shown to have some superiority over CT as it may show cysticerci missed by CT.¹⁹ Martinez and co-workers²⁰ concluded that MRI is approximately four times more sensitive than CT in the detection of cysts (85% *vs* 21%), in brainstem, subependymal location, cerebellum, subarachnoid space, spinal cord, and inside the ventricles. However, calcified lesions are better seen with CT scan.

SEROLOGICAL TESTS

Several immunological tests in the serum and CSF have been developed over the years. The latest, enzyme-linked immunoelectrotransfer blot assay in serum, is highly sensitive and specific. When Tsang and colleagues²¹ first evaluated this test it proved 98% sensitive and 100% specific. Wilson and colleagues²² reported further experience with the test and showed that in patients with two or more intracranial cysticerci, the test was positive in 94% of patients, but in those who had only a solitary lesion its sensitivity ranged from 28 to 70%. However, more widely available enzyme-linked immunosorbent assay (ELISA) showed 50% sensitivity and 65% specificity in CSF, while older tests (eg, complement fixation) are insufficiently sensitive and specific. Patients presenting only with calcified CT lesions are generally serologically negative. When CSF shows inflammatory changes, serological tests for the detection of anticysticercal antibodies in CSF by ELISA is almost always positive.^{23 24}

STOOLS

Although the patient with neurocysticercosis no longer harbours a tapeworm at the time diagnosis is made, stools from both the patient and family members should be examined (especially if they are pork eaters) over several days for proglottids and eggs.^{4 7}

Figure 4 Cranial CT scan showing a healed calcified lesion in the right frontal lobe

Figure 5 Cranial CT showing disseminated cysticercosis 'starry-night' appearance

Figure 6 MRI of brain showing numerous cystic lesions (T1-weighted image)

Treatment of neuro-cysticercosis

- parenchymal: praziquantel 50 mg/kg/day for 15 days or albendazole 15 mg/kg/day for 30 days
- cysticercotic encephalitis: corticosteroids 1.2 mg/kg/day till symptoms abate; (prednisolone)
- subarachnoidal: anticysticercal drugs, corticosteroids, CSF shunting procedures, surgical removal, or decompression of cyst
- ventricular: surgical or endoscopic removal of the cyst
- spinal: surgical removal

Box 5

Mechanisms of action of anticysticercal drugs

Praziquantel

- spastic paralysis of the parasite musculature
- destroys the scolex
- extensive integumental destruction followed by inflammatory reaction

Albendazole

- inhibits the uptake of glucose by the parasite membranes

Box 6

Figure 7 Contrast-enhanced cranial CT scans showing a single small CT lesion in right parietal lobe. Eccentric scolex is seen

BIOPSY OF SUBCUTANEOUS NODULES

Histopathological examination of a subcutaneous nodule can help to confirm diagnosis. The definitive cytological diagnosis depends on the identification of larval parts of the parasite, eg, tegument, subtegument, parenchyma and/or scolices. Fine-needle aspiration cytology provides a satisfactory alternative to excisional biopsy.²⁵

Treatment

Apart from symptomatic treatment, other options for the treatment of neurocysticercosis include anticysticercal drugs, corticosteroids, CSF shunting, surgical removal or decompression of the cyst (box 5).

ANTICYSTICERCAL DRUGS

Praziquantel, an isoquinoline, and albendazole, an imidazole, are anticysticercal drugs that have been used extensively in the treatment of intracranial cysticercosis. Both agents frequently eliminate all the cysticerci in the brain parenchyma, or at least markedly reduce their number.²⁶⁻²⁸ In several comparative trials albendazole appeared slightly more effective than praziquantel for the treatment of parenchymal neurocysticercosis.²⁹ Albendazole has also been found to be effective against giant parenchymal, subarachnoidal, intraventricular and even spinal forms of cysticercosis, probably because of its unique mechanism of action (box 6).³⁰⁻³¹ Recently, Cruz *et al*³² observed that an 8-day course of albendazole is as effective as 15 or 30 days of treatment. These authors did not find any advantage of continuing treatment for longer periods.

Treatment of cysticercosis with albendazole or praziquantel has been associated with a high frequency of adverse reactions that seem to due to the host's inflammatory reaction to dying parasites. Headache, nausea, vomiting and seizures are common and usually transient.^{1-4,6} Because corticosteroids ameliorate these effects, authors have recommended routine co-administration of corticosteroids along with anticysticercal therapy.⁷ However, there are no controlled trials to support this practice.¹ In one patient, cerebral infarct due to vasculitis which appeared to be precipitated by praziquantel, despite premedication with dexamethasone, has been reported.³³ Other authors^{7,9} have cautioned against careless and unsupervised use of praziquantel in patients with a heavy parasitic load. Such patients may rapidly develop alteration in sensorium after a few doses of anticysticercal drug; a few deaths have also been reported in children following anticysticercal therapy.⁹

Acute development of cerebral oedema secondary to the host's inflammatory reaction to the parasite may markedly elevate the intracranial pressure. Therefore, patients with disseminated lesions should only be treated with corticosteroids. Other forms of cysticercosis (ocular, spinal and subarachnoid) should also be treated with great caution as damage to surrounding tissues is likely (box 7).

SURGICAL TREATMENT

Patients with hydrocephalus due to intraventricular or subarachnoidal cysticercosis usually require CSF shunting. Some authors have recommended that a ventricular shunt be placed in all patients with subarachnoid cysticercosis before medical therapy is attempted. However, the rate of shunt failure is high because of frequent shunt occlusion and infection. In patients with racemose and large

Contraindications of anticysticercal treatment

Should not be treated

- cysticercotic encephalitis

Treat with caution

- subarachnoidal neurocysticercosis
- hydrocephalus due to diffuse arachnoiditis
- ocular
- spinal

Treatment not effective

- parenchymal brain calcification
- colloidal cysts (ring- or disc-enhancing CT lesions)
- giant subarachnoid cysticerci
- intraventricular cysts

Box 7

Case history

An 8-year-old boy of 31 kg presented a week after three episodes of left-sided focal convulsions. All three episodes occurred within a few hours on the same day. In the last episode, the patient became unconscious and had a secondary generalised convulsion. His detailed neurological examination revealed no abnormality. EEG revealed intermittent delta activity in the right parieto-occipital region. CT scan showed a small (10 mm) ring-enhancing lesion with an eccentric dot in the right parietal lobe. He was started on small doses of carbamazepine (100 mg bid). Repeat CT scan was done after 14 weeks, at which time the initial lesion had disappeared. He remained seizure-free during 6 months of follow-up.

Box 8

Causes of single enhancing CT lesions

Common

- cysticercus granuloma
- tuberculoma

Uncommon

- secondaries
- old infarct
- low grade glioma
- lymphoma
- fungal granuloma
- focal encephalitis
- micro-abscesses
- toxoplasmosis

Box 9

cisternal cysts, surgical removal is often recommended. When there is little inflammation the cyst can be removed easily, while it is difficult and hazardous to remove inflamed cysts. Intraventricular cysts also need surgical removal, either with supratentorial or suboccipital approach.^{31 34} Endoscopic removal of intraventricular cysts has also been used with success.

CONTROVERSIES REGARDING EFFICACY AND SAFETY OF ANTICYSTICERCAL TREATMENT

Data on the untreated natural course of neurocysticercosis indicate that all patients with cystic viable lesions, if given time, show spontaneous improvement of the CT picture. The lesions either heal with calcification, or they disappear.³⁵ The response to anticysticercal therapy is also not universal, and several reports indicate treatment failure. Currently, there is an intense debate about the safety and usefulness of anticysticercal therapy. Its opponents argue that, while anticysticercal drugs may hasten larval demise, this may not necessarily lead to an improvement in outcome. It has been suggested that an acute enhanced inflammatory response following anticysticercal therapy may produce profound cerebral cicatrix and could therefore worsen the long-term clinical outcome.^{36–38}

SINGLE ENHANCING CT LESIONS

Small single enhancing (ring/disc) CT lesions (figure 7) are the commonest radiological finding in Indian patients with epilepsy. They are more common in children, being present in 41% of patients below 15 years of age.³⁹ The CT lesions are usually >20 mm in size, well defined, with or without surrounding oedema. The lesions can be seen anywhere in the cerebral cortex but are frequently located in the parieto-occipital region. Patients with these CT lesions usually have focal seizures with or without secondary generalisation (box 8).

Initially, tuberculosis was presumed to account for these intracranial lesions but more recently, serological tests and histopathological examination of tissues obtained from stereotactic biopsy have revealed that the majority of these CT abnormalities are cysticercus granuloma (box 9).⁴⁰ The majority of such lesions spontaneously disappear within 6–12 weeks.⁴¹ Conflicting reports^{42 43} on the efficacy of anticysticercal treatment in patients with single CT lesions, make this form of treatment of questionable value. The patients require only anti-epileptic treatment.

Prognosis

The majority of workers believe that, in patients with neurocysticercosis, the prognosis of associated seizure disorders improves considerably after treatment with anticysticercal drugs.^{44 45} In most patients, seizures are easily controlled with first-line anti-epileptic drugs which can safely be withdrawn. A few patients require prolonged anti-epileptic therapy, especially when parenchymal brain lesions become calcified.⁴⁶

Patients presenting with hydrocephalus, large cysts, multiple granulomas with oedema, chronic meningitis, and vasculitis are often acutely ill, frequently require surgery and do not respond satisfactorily to anticysticercal drugs. A fatal outcome or serious sequelae have been reported in patients with disseminated cysticercosis. Ventriculo-peritoneal shunting in patients with multiple subarachnoidal cysts in the basal cisterns is often unsatisfactory due to blockage of the shunt by the inflammatory debris. The results of excision of large, single, well-circumscribed cysts in brain and spinal cord are excellent.^{4 6 7}

Conclusions

Anticysticercal treatment is considered a significant advancement in the field of neurocysticercosis. This treatment produces excellent radiological improvement. However the prospects for the long-term clinical outcome are far from satisfactory. Health measures should be ensured for the effective prevention of the disease. Indiscriminate human defaecation must be prevented and adequate disposal of excreta ensured. Raw vegetables should be washed thoroughly and cooked adequately. Recent advances in immunology do hold promise for development of a vaccine against *T solium* in the near future.

- 1 Del Brutto OH, Sotelo J. Neurocysticercosis: an update. *Rev Infect Dis* 1988;10:1075–87.
- 2 Mahajan RC. Geographical distribution of human cysticercosis. In: Flisser A, Willms K, Lacleite TP, Larraide C, Ridaura C, Beltram F, eds, *Cysticercosis*. New York: Academic Press, 1982; pp 39–46.

- 3 Brown WJ, Voge M. Cysticercosis: a modern day plague. *Pediatr Clin North Am* 1985;32:953–69.
- 4 Webbe G. Human cysticercosis. Parasitology, pathology, clinical manifestations and available treatment. *Pharmacol Ther* 1994;64:175–200.
- 5 Escobar A. The pathology of neurocysticercosis. In: Placio SE, Rodriguez-Carbajal J, Taveras JN,

eds, *Cysticercosis of the central nervous system*. Springfield, IL: Charles C Thomas, 1983; pp 27–54.

- 6 Cook GC. Neurocysticercosis: parasitology, clinical presentation, diagnosis and recent advances in management. *Q J Med* 1988;256:575–83.

- 7 Wadia NH. Neurocysticercosis. In: Shaker RA, Newman P, Poser CM, eds, *Tropical neurology*. London: WB Saunders, 1995; pp 247-73.
- 8 Commission on Tropical diseases of the International League against Epilepsy. Relationship between epilepsy and tropical diseases. *Epilepsia* 1994;35:89-93.
- 9 Wadia NH, Srinivas D, Bhatt M. Disseminated cysticercosis. New observations, including CT findings and experience with treatment by praziquantel. *Brain* 1988;111:597-614.
- 10 Del Brutto OH. Cysticercosis and cerebrovascular disease: a review. *J Neurol Neurosurg Psychiatry* 1992;55:252-4.
- 11 Alarcon F, Vanormelingen K, Moncayo J, Vinan I. Cerebral cysticercosis as a risk factor for stroke in young and middle aged people. *Stroke* 1992;23:1563-5.
- 12 Isidro-Llorens A, Dachs F, Videll J, Sarrias M. Spinal cysticercosis: case report and review. *Paraplegia* 1993;31:128-30.
- 13 Sharma BS, Banerjee AK, Kak VK. Intramedullary spinal cysticercosis. *Clin Neurol Neurosurg* 1987;82:2111-6.
- 14 Rodriguez-Carbajal J, Boleaga-Duran B, Dorfman J. The role of computed tomography (CT) in diagnosis of neurocysticercosis. *Child Nerv Syst* 1987;3:199-202.
- 15 McCormick GF, Zee CD, Heiden J. Cysticercosis cerebri: review of 127 cases. *Arch Neurol* 1982;39:534-9.
- 16 Kramer LD, Locke GE, Byrd SE, Daryabagi J. Cerebral cysticercosis: documentation of natural history with CT. *Radiology* 1989;170:459-62.
- 17 Carpio A, Placencia M, Santillan F, Escobar A. Proposal for a new classification of neurocysticercosis. *Can J Neurol Sci* 1994;21:43-7.
- 18 Del Brutto OH, Guevara J, Sotelo J. Intracerebral cysticercosis. *J Neurosurg* 1988;69:58-60.
- 19 Desai SB, Shah VC, Tavri DJ, Rao P. Neurocysticercosis: comparison of CT scan and MRI. Study of 35 cases. *Neuroradiology* 1991;33(suppl):578-9.
- 20 Martinez HR, Rangel-Guerra RA, Elizondo G, et al. MR imaging in neurocysticercosis: a study of 56 cases. *Am J Neuroradiol* 1989;10:1011-9.
- 21 Tsang VCW, Brand JA, Boyer AK. An enzyme linked immunoelectrotransfer blot assay and glycoprotein antigens for diagnosing human cysticercosis (*Taenia solium*). *J Infect Dis* 1989;159:50-9.
- 22 Wilson M, Bryan RT, Friend JA, et al. Clinical evaluation of the cysticercosis by enzyme linked immunotransfer blot in patients with neurocysticercosis. *J Infect Dis* 1991;164:1007-9.
- 23 Rosas N, Sotelo J, Neito D. ELISA in the diagnosis of neurocysticercosis. *Arch Neurol* 1986;43:353-6.
- 24 Plancarte A, Espinoza B, Flisser A. Immunodiagnosis of human neurocysticercosis by enzyme linked immunosorbent assay. *Child Nerv Syst* 1987;3:203-5.
- 25 Arora VK, Gupta K, Singh N, Bhatia A. Cytomorphologic panorama of cysticercosis on fine needle aspiration: a review of 298 cases. *Acta Cytol* 1994;38:377-80.
- 26 Sotelo J, Escobedo F, Rodriguez-Carbajal J, Torres B, Rubido-Donnadieu F. Therapy of parenchymal brain cysticercosis with praziquantel. *N Engl J Med* 1984;310:1001-7.
- 27 Del Brutto OH, Sotelo J, Roman GC. Therapy for neurocysticercosis: a reappraisal. *Clin Infect Dis* 1993;17:730-5.
- 28 Escobedo F, Penagos P, Rodriguez J, Sotelo J. Albendazole therapy for neurocysticercosis. *Arch Intern Med* 1987;147:738-41.
- 29 Takayanagui OM, Jardim E. Therapy for neurocysticercosis: comparison between praziquantel and albendazole. *Arch Neurol* 1992;49:535-7.
- 30 Del Brutto OH, Sotelo J, Aguirre R, Diaz-Calderon E, Alarcon A. Albendazole therapy for giant subarachnoid cysticerci. *Arch Neurol* 1992;49:535-7.
- 31 Martinez HR, Rangel-Guerra R, Arredondo-Estrada JH, Marfil A, Onofre J. Medical and surgical treatment in neurocysticercosis - a magnetic resonance study of 161 cases. *J Neurol Sci* 1995;130:25-34.
- 32 Cruz I, Cruz ME, Carrasco F, Horton J. Neurocysticercosis: optimal dose treatment with albendazole. *J Neurol Sci* 1995;133:152-154.
- 33 Woo E, Yu YL, Huang CY. Cerebral infarct precipitated by praziquantel in neurocysticercosis - a cautionary note. *Trop Geogr Med* 1988;40:143-6.
- 34 Coudwell WT, Zee CS, Apuzzo MJB. Definition of the role contemporary surgical management in cysticercosis and parenchymatous cysticercosis cerebri. *Neurosurgery* 1991;28:231-7.
- 35 Mitchell WG, Crawford TO. Intracerebral cysticercosis in children: diagnosis and treatment. *Pediatrics* 1988;82:76-82.
- 36 Padma MV, Behari M, Misra NK, Ahuja GK. Albendazole in neurocysticercosis. *Natl Med J India* 1995;8:255-8.
- 37 Kramer LD. Medical treatment of cysticercosis: ineffective. *Arch Neurol* 1995;52:101-2.
- 38 Carpio A, Santillan F, Leon P, Flores C, Hauser A. Is the course of neurocysticercosis modified by treatment with antihelminthic agents? *Arch Intern Med* 1995;155:1982-8.
- 39 Wadia RS, Makhale CN, Kelkar AV, Grant KB. Focal epilepsy in India with special reference to lesions showing ring or disc like enhancement on contrast computed tomography. *J Neurol Neurosurg Psychiatry* 1987;50:1298-301.
- 40 Rajshekhar V, Haran RP, Shankar Prakash G, Chandy MJ. Differentiating solitary small cysticercus granuloma and tuberculoma in patients with epilepsy. *J Neurosurg* 1993;78:402-7.
- 41 Sethi PK, Kumar BR, Madan VS, Mohan V. Appearing and disappearing CT scan abnormalities and seizures. *J Neurol Neurosurg Psychiatry* 1985;48:466-9.
- 42 Rajshekhar V. Albendazole therapy for persistent solitary cysticercus granuloma in patients with seizures. *Neurology* 1993;43:1238-40.
- 43 Padma MV, Behari M, Misra NK, Ahuja GK. Albendazole in single CT ring lesions in epilepsy. *Neurology* 1994;44:1344-6.
- 44 Del Brutto OH, Santibanez R, Noboa CA, Aguirre R, Diaz-Calderon E, Alarcon TA. Epilepsy due to neurocysticercosis: analysis of 203 patients. *Neurology* 1992;42:389-92.
- 45 Vazquez V, Sotelo J. The course of seizures after treatment for cerebral cysticercosis. *N Engl J Med* 1992;327:696-701.
- 46 Del Brutto OH. Prognostic factors for seizure recurrence after withdrawal of antiepileptic drugs in patients with neurocysticercosis. *Neurology* 1994;44:1706-9.