

Neurocysticercosis in a young Indian male

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A 33-year-old Indian male was hospitalised for a 3-day history of headache and left lower limb weakness in December 2014. He had experienced no fever or seizures. He had visited New Delhi, India, the year before. Physical examination revealed the patient to be fully conscious with left lower limb monoparesis. There was no sensory deficit. Computed tomography (CT) revealed a superior parietal intra-axial lesion with a calcified focus (Fig 1a). Magnetic resonance imaging (MRI) delineated a 2 x 1.5 x 1.5 cm circumscribed hypointense cystic lesion with a contrast-enhancing wall and an eccentric intracystic signal with perilesional oedema (Figs 1b to 1d). Differential diagnoses included neurocysticercosis, brain abscess, brain metastasis, and malignant glioma. Craniotomy for excision was performed

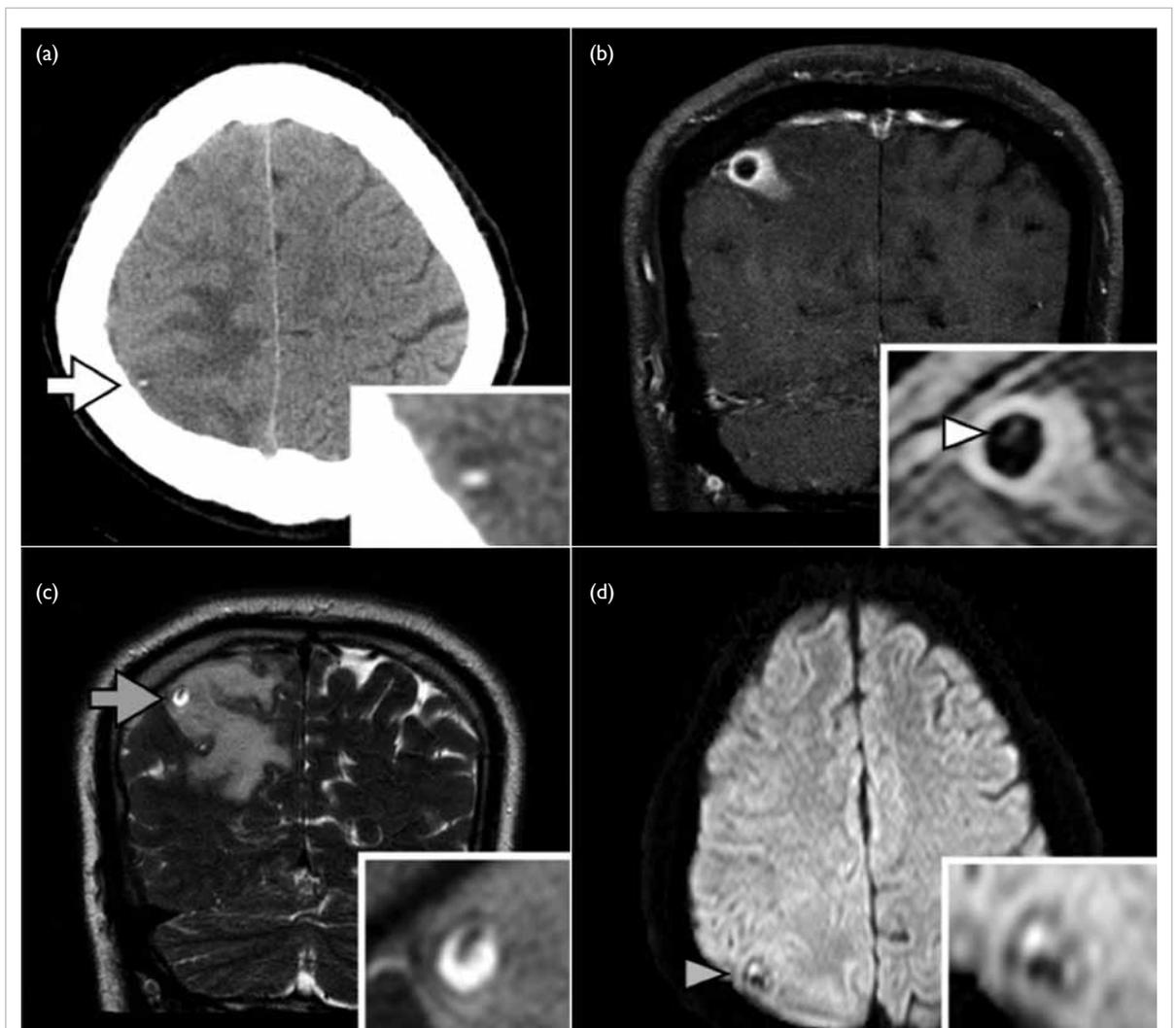


FIG 1. Images of the patient

(a) Computed tomography demonstrating an intra-axial right parietal lesion with a calcific focus (axial, white arrow). (b) Hole-with-dot sign: gadolinium contrast T1-weighted coronal magnetic resonance imaging demonstrating a ring-enhancing hypointense cystic lesion with an eccentric intracystic enhancing signal suggestive of a scolex at the colloidal vesicular stage of infection (white arrowhead, inset). (c) T3-weighted coronal image showing a hyperintense cyst (grey arrow) with a hypointense scolex (inset) and marked perilesional oedema. (d) Fluid-attenuated inversion recovery image depicting a hyperintense lesion (axial, grey arrowhead) with corresponding hypointense scolex (inset)

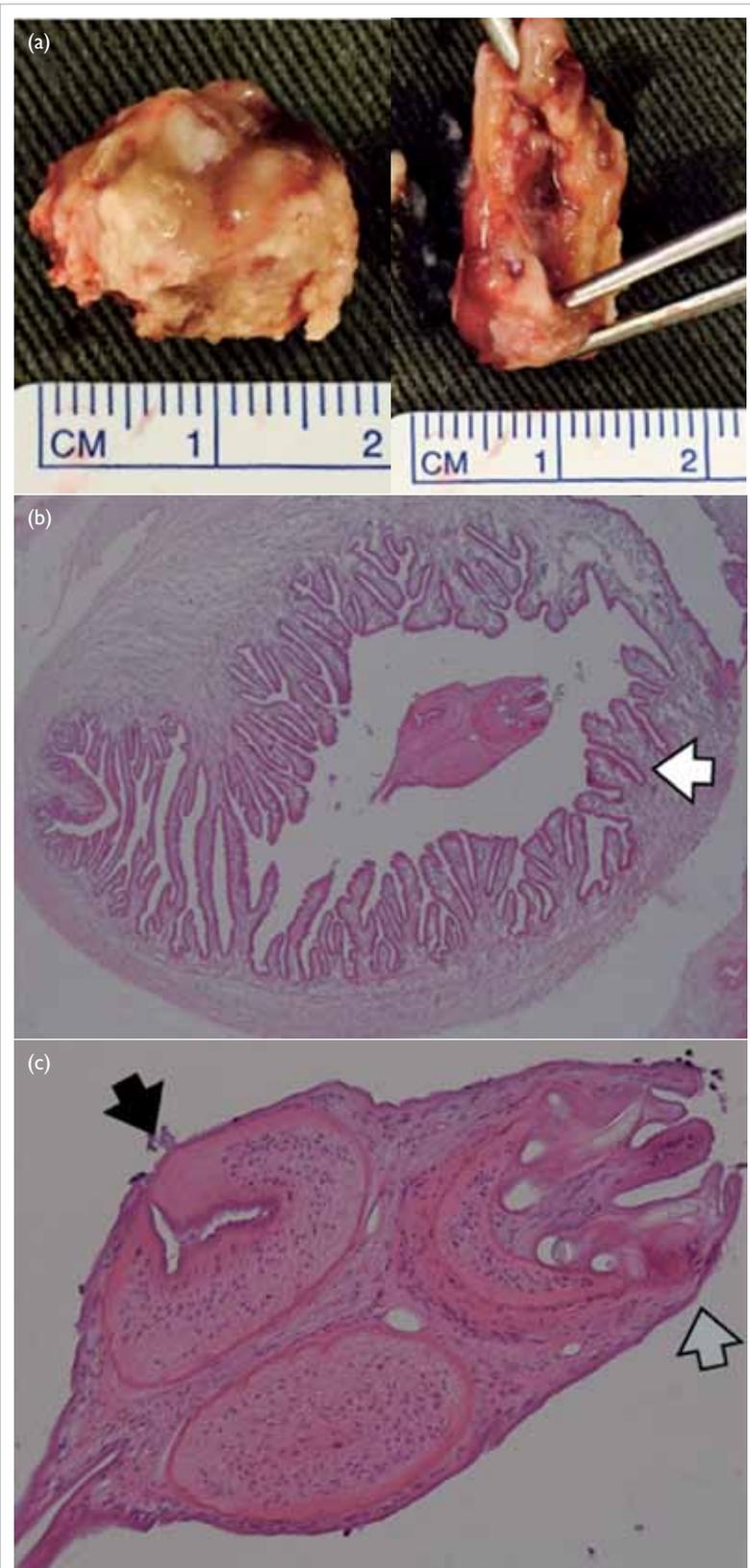


FIG 2. Intra-operative photographs and photomicrographs of the patient (a) Intra-operative photographs of resected neurocysticercus with a fibrous wall containing thick opaque material. (b) The typical colloid cyst membrane (white arrow; H&E; original magnification, x 40). (c) *Taenia solium* scolex comprises four suckers (black arrow) and a double row of hooks (grey arrow) for host intestinal wall attachment (H&E; original magnification, x 100)

in view of the possibility of malignancy and the surgically accessible superficial location of the lesion. Intra-operatively, a firm capsular mass containing thick opaque material was seen (Fig 2a). Histology revealed a cysticercus within a fibrous capsule, compatible with the diagnosis of neurocysticercosis (Figs 2b and 2c). A 2-week course of oral albendazole was commenced and the patient was discharged with full recovery.

Neurocysticercosis is the most common parasitic infection of the central nervous system (CNS) caused by the larval form of *Taenia solium*. The peak age of incidence is between 25 and 35 years¹ and the condition is endemic to the Indian subcontinent, coastal North Africa, sub-Saharan Africa, Latin America, and China. The main mode of transmission is by faecal-oral ingestion of tapeworm embryos.^{1,2} Consumption of contaminated poorly cooked pork is a less-frequent alternative source of infection since pigs are intermediate hosts.^{2,3} Within 72 hours of ingestion, larvae known as oncospheres are released and pass through the intestinal wall into the circulation, subsequently depositing in the CNS, retina, and skeletal muscle as cysticerci.^{1,2} The parasite can remain viable in the brain for several years after which it undergoes calcific degeneration.³ In endemic areas, the most common presentation is epilepsy, responsible for 30% of cases.¹ Focal neurological deficits may occur including cranial nerve palsy due to basal meningitis. Obstructive hydrocephalus develops when lesions occupy the fourth ventricle.^{1,2}

Characteristic radiological features include dystrophic calcification on CT imaging, cyst wall contrast enhancement on T1-weighted MRI and identification of the pathognomonic scolex, an eccentric focus of enhancement representing the tapeworm's head, best delineated with fluid-attenuated inversion recovery sequences (hole-with-dot sign).^{3,4} Brain abscess or metastases are important differential diagnoses as they are also similarly located at the grey-white matter junction of the middle cerebral artery distribution, associated with disproportionately significant perilesional cerebral oedema and classically exhibit heterogeneous contrast enhancement. Malignant glioma was less likely in our patient since they are morphologically more infiltrative, and the present lesion was well-circumscribed. For this patient, the major distinguishing feature that supported a diagnosis of neurocysticercosis was the presence of dystrophic calcification on CT and, in retrospect, the presence of a scolex on MRI. Absolute criteria for a definitive diagnosis are either histological parasitic proof, imaging demonstration of a scolex, or subretinal parasites on fundoscopy (Table⁵). Serological enzyme-linked immunoelectrotransfer blot detection of anti-cysticercus antibodies or cerebrospinal fluid enzyme-linked immunosorbent

TABLE. Diagnostic criteria of neurocysticercosis⁵

Criteria	
Categories of criteria	
Absolute	<ol style="list-style-type: none"> 1. Histological demonstration of the parasite from biopsy of a brain or spinal cord lesion 2. Cystic lesions showing the scolex on CT or MRI 3. Direct visualisation of subretinal parasites on fundoscopic examination
Major	<ol style="list-style-type: none"> 1. Lesions highly suggestive of neurocysticercosis on neuroimaging studies 2. Positive serum EITB for the detection of anti-cysticercus antibodies 3. Resolution of intracranial cystic lesions after therapy with albendazole or praziquantel 4. Spontaneous resolution of small single enhancing lesions
Minor	<ol style="list-style-type: none"> 1. Lesions compatible with neurocysticercosis on neuroimaging studies 2. Clinical manifestations suggestive of neurocysticercosis 3. Positive CSF ELISA for detection of anti-cysticercus antibodies or cysticercus antigens 4. Cysticercosis outside the central nervous system
Epidemiological	<ol style="list-style-type: none"> 1. Evidence of household contact with <i>Taenia solium</i> infection 2. Individuals coming from or living in an area endemic for cysticercosis 3. History of frequent travel to disease-endemic areas
Diagnostic certainty	
Definitive	<ol style="list-style-type: none"> 1. Presence of one absolute criterion 2. Presence of two major criteria plus one minor and one epidemiological criterion
Probable	<ol style="list-style-type: none"> 1. Presence of one major criterion plus two minor criteria 2. Presence of one major criterion plus one minor and one epidemiological criterion 3. Presence of three minor criteria plus one epidemiological criterion

Abbreviations: CSF = cerebrospinal fluid; CT = computed tomography; EITB = enzyme-linked immunoelectrotransfer blot; ELISA = enzyme-linked immunosorbent assays; MRI = magnetic resonance imaging

assays are adjunctive investigations.^{1,6} Management of active neurocysticercosis includes antiepileptic drug administration, anti-inflammatory glucocorticoid therapy, and definitive antiparasitic therapy with albendazole (15 mg/kg per day) or praziquantel (50 mg/kg per day) for 2 to 4 weeks.¹ Surgical excision is reserved for cysts that cause mass effect, hydrocephalus, or if the diagnosis is unclear.²

In an era of increasing migration and international travel, patients from developing countries who present with seizures, raised intracranial pressure symptoms, or focal neurological deficit should be suspected of having neurocysticercosis when characteristic imaging findings are identified. In probable cases, a trial of antiparasitic therapy is recommended with serial scans arranged to monitor treatment response.

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