

Atypical clinical and imaging manifestation in neurocysticercosis

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Abstract

A 20-year-old man presented with left-sided headache and seizures of three years duration. Conglomerate ring-enhancing lesions were seen in the first magnetic resonance imaging study. He was initially treated with anticonvulsants for two years. Because the symptoms and the lesions were persisting, antitubercular treatment was added. He was asymptomatic after antitubercular treatment despite persisting lesion. Lesion showed exuberant ring enhancement with increased perfusion. Because the lesion was persisting even after 24 months of antitubercular treatment, excision was considered. Lesionectomy was done and histopathology reported meningoencephalitis secondary to neurocysticercosis. The case report highlights the difficulty in differentiating cysticercosis from tuberculoma in patients from countries where both the conditions are endemic.

Key Words

Chronic meningoencephalitis, magnetic resonance imaging, neurocysticercosis, perfusion magnetic resonance imaging

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Introduction

Neurocysticercosis is one of the common intracranial parasitic infections caused by larval stage of *Taenia solium*.^[1] It is sometimes difficult to differentiate it from tuberculosis (TB), especially when both the diseases are endemic. Both diseases can co-exist.^[2] It is very important to differentiate these two entities, considering the wide difference in management of these two entities.^[3] Furthermore, many patients in developing countries are put on anti-TB chemotherapy for long periods based on computed tomography/magnetic resonance imaging (MRI) findings. In most patients, there is no pathological proof. Some patients could be harboring neurocysticercosis but may be receiving anti-TB drugs for long period of time. This case report highlights the difficulty in differentiating cysticercosis from tuberculoma in patients from countries where both the conditions are endemic. It is essential that we have better imaging tools for differentiating these conditions. We report a case of neurocysticercosis presenting as chronic

focal encephalitis with overlapping imaging features of granulomatous lesion.

Case Report

A 20-year-old man presented with left side intermittent headache of three years duration. He also gives history of seizures with the frequency of 1 to 2 episodes/month. Seizures were without aura and were associated with tonic-clonic movements, up rolling of eyeball, deviation of angle of mouth, urinary incontinence followed by loss of consciousness. He was seen by the local physician and imaging was performed. MRI done (March 21, 2006) outside our institute showed conglomerate ring-enhancing lesions in right frontal lobe [Figure 1a and b]. The lesions showed a hypointense peripheral ring and a central hyperintensity in the T2 weighted image. Smooth complete ring enhancement was seen following contrast administration. Mild-to-moderate perilesional edema was also seen. Initially, he was managed with anticonvulsant for a period of 1 year. In view of the persisting symptoms and lesion, he was subsequently started on anti-TB treatment. After antitubercular treatment, he was symptom free. Repeat MRI was done at the end of the antitubercular treatment on December 22, 2008 to see the status of the lesion. It showed a persisting lesion of almost the same size but with some change in the morphology [Figure 1c and d].

He was referred to our institute for further evaluation. No neurological deficits were identified on clinical examination.

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His total white blood cell count was 8 900 cells with 63% polymorphs. ESR (*erythrocyte sedimentation rate*) was within normal range (5 mm at end of 1 hour). MRI showed [Figure 2] conglomerate lesion (2.9 × 1.7 cm) in frontal lobe without significant decrease in size. Central cystic region of the larger lesion had decreased in size with exuberant ring enhancement in periphery. Spectroscopy showed drop in NAA (N-acetylaspartate) levels and increased choline as compared with contralateral side. Lipid peak was also seen. There was a two-fold increase in rCBV (relative cerebral blood volume) in perfusion MRI as compared with contralateral side. Considering the morphology of the lesion on MRI which was persisting over a period of 4 years, the possibility of granulomatous (resistant tubercular or fungal) was raised.

Despite extended course of anti-TB therapy (24 months), lesion was persisting without significant change in size [Figure 3a], and hence it was decided to do lesionectomy. Histopathology sections showed dense collections of inflammatory cells, mainly lymphocytes, plasma cells, eosinophils, neutrophils, sheets of foamy macrophages, fibrosis, and necrosis [Figure 3b]. In none of the sections studied, there was any evidence of granulomas. Sections from the dura studied showed dural tissue with the same histological findings, along with degenerated cysticercosis in the centre. All these features suggested the possibility of neurocysticercosis, morphologically manifesting as localized meningoencephalitis.

Discussion

Cysticercosis is a common parasitic infection of the central nervous system caused by ingestion of *T. solium* eggs. Approximately 60 to 90% of patients with cysticercosis develop neurocysticercosis.^[4] The vesicular stage does not produce any immunological response and is characterized by nonenhancing cystic lesions in superficial neuroparenchymal or intraventricular region. Diagnosis can be made by identification of the scolex, which appears as eccentric nodule within the cyst. For unknown reasons, the parasites in the next stage start degenerating, inducing host response. Stage 2 and 3 are characterized by ring-enhancing lesions with surrounding edema. In our case, the lesion was most likely imaged in second and third stages. Enhancement suggestive of inflammation is rarely described in a stage 4 cyst.^[5,6]

The unusual finding in our case is the persistence of stage two or three neurocysticercosis, causing chronic meningoencephalitis for 4 years without involution or calcification. The duration of each of these stages of infection has not been established in literature. There is considerable difference in the duration of the disease existing among individuals and this mainly depends on host immune response.^[7] When there is no host immune response (that is vesicular stage), parasite may persist for many years. Usually the second and third stage parasites have a duration of about 4 to 6 months.^[7,8] Conglomerate lesions are unusual in neurocysticercosis and are commonly seen in TB.^[9,10] Peripheral T2 hypointense ring with increased perfusion is also an unusual MRI finding in neurocysticercosis.^[11,12] Increased perfusion generally favors diagnosis of neoplastic lesion rather than neurocysticercosis.^[11,12] Overlap between CBV values in infective and neoplastic lesions are reported in

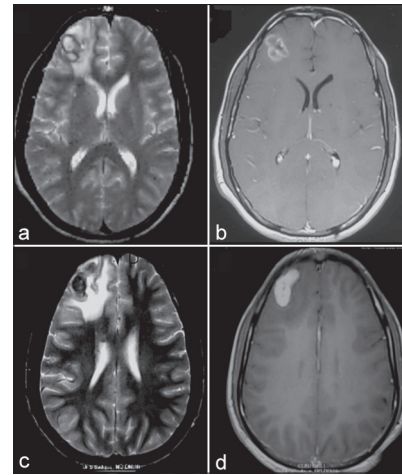


Figure 1: T2 weighted axial (a) and postcontrast T1 (b) MRI images (dated March 2006) showing conglomerate lesions in right frontal lobe, which shows ring enhancement following gadolinium injection. Corresponding MRI images (c and d) showing images done on December 2008 reveals persisting lesion with decreased center cystic area

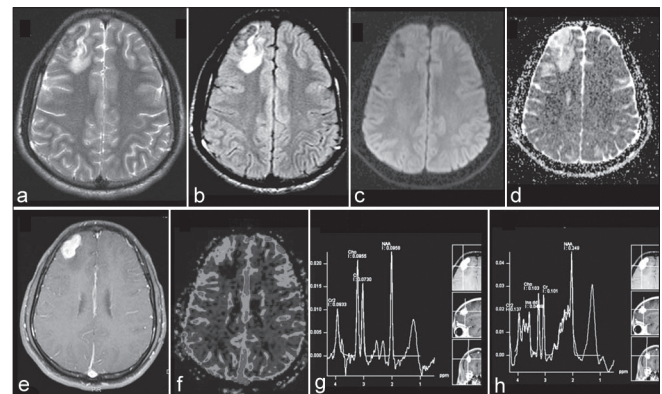


Figure 2: Axial T2 and FLAIR images of MRI images taken on April 2009, showing right frontal lobe lesion which is predominately hypointense (a and b). No diffusion restriction is seen (c and d). Exuberant contrast enhancement is seen (e) along with increased rCBV (f). Spectroscopy at TE 135 and TE 30 (g and h) showing decreased NAA peak and increased choline. Lipid peak is also seen

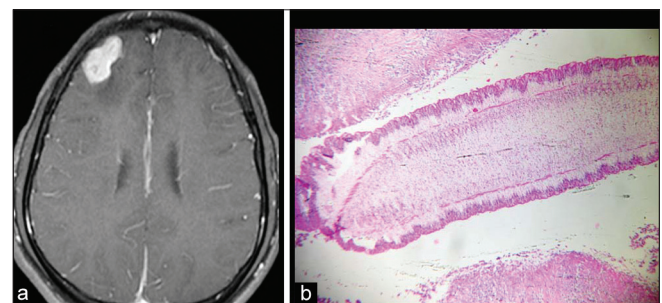


Figure 3: Axial postcontrast T1 weighted MRI image (September 2009) showing persisting lesions (a). Histopathology sections showing degenerated cysticercosis cyst (b) (H&E, ×200)

literature due to disrupted blood brain barriers. A number of cytokines are secreted in infective brain lesions as response

to the primary immunity against pathogens. The cytokinin upregulates the endothelial gaps, resulting in extravasation of inflammatory molecules and leukocytes through the blood brain barrier opening. The transfer coefficient (k^{trans}) values may be of help in such cases to differentiate neoplastic and inflammatory lesions.^[11]

This report highlights the difficulty in differentiating cysticercosis and TB only on the basis of neuroimaging. Many patients are put on anticonvulsants and antituberculous treatments empirically without proving the diagnosis. This method of management may not be correct in many patients as was seen in our patient. It is also difficult to understand why a young man with a mass lesion in the right frontal lobe with a suspicious diagnosis of cysticercosis was exceptionally set under anticonvulsant therapy for one year. Then, without clarifying of the diagnosis, antituberculous therapy was applied. Such management decisions are probably taken due to confusion in diagnosis by neuroimaging.

Though there are diagnostic criteria for neurocysticercosis based on objective clinical, imaging, immunologic, and epidemiologic data, in our case the final diagnosis could be made only with the help of the one absolute criterion, which is histopathology.

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