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Neurocysticercosis masquerading psychotic disorder: A case report

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ABSTRACT

Psychotic manifestations are uncommon in neurocysticercosis. This article describes a ten year girl presented with manic – psychotic manifestation for which she was under treatment with antipsychotics for eight months. Eventually she developed generalized tonic clonic seizure and CT scan of brain revealed small isodense right posterior parietal lesion of 5 mm size with perifocal edema. CECT revealed intense nodular post contrast enhancement. This highlights the possible misdiagnosis of a case of neurocysticercosis as an organic psychotic disorder so it should be considered as a differential diagnosis in patients with neurological as well as psychiatric manifestations in endemic area like India.

1. Introduction

Neurocysticercosis is the most common parasitic infection of central nervous system[1] caused by the larval stage of *Taenia solium* (*T. solium*). The disease has a worldwide distribution but is endemic in rural areas of developing countries, with a prevalence rate varies from 0.1%-4%[2]. Neurocysticercosis can be asymptomatic or having pleomorphic manifestation[3].

Though seizures, raised intracranial pressure, hydrocephalus is the usual presenting features, but various uncommon psychotic symptoms have also been reported in the literature[4]. However, a very few publications are available in pediatric population with these rare manifestations which may be attributed to the long incubation period of the disease, ranging from several months to thirty years. Here we illustrate a case report of a ten year girl with neurocysticercosis masquerading psychotic illness.

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2. Case report

Ten year female child admitted to pediatrics inpatient department with two episodes of generalised tonic clonic seizure in last twelve hours. There was nothing suggestive of prior seizure or family history of convulsion. Since last eight months she was having intermittent euphoria, inappropriate behavior, irrelevant talk, lack of concentration, inability to study and bizarre movements, for which she under treatment by different psychiatrists with olanzapine, haloperidol, promethazine and lorazepam but without any significant improvement.

There was no history of fall, injury, motor weakness and bladder/bowel involvement during the above period. Neuroimaging was not done during the psychiatric treatment. Contact history of tuberculosis was absent. She was delivered at term without any perinatal complications or developmental delay.

On examination there was no physical abnormality.

The child was having at times hyperventilation, euphoria and some bizarre body movements. Neurological examination revealed inability to hold the pen properly and marked in-coordination of the right hand movement which was exacerbated during writing and target reaching, but other limb movements were normal. There was also mild positional sensory loss and limb-kinetic apraxia of the right upper hand on out stretching with closed eyes. Patient's MMSE score was 27 with errors in calculation and naming of the objects but reading being normal. Speech, cranial nerve, muscle tone and power, deep tendon and plantar reflexes were normal.

Cerebellar signs were absent. Ophthalmological examination revealed no papilloedema or Kayser-Fleischer ring. Complete blood counts, blood glucose, serum electrolytes, liver, kidney function test and X-ray chest were within normal limits. Repeated routine and microscopic examination of stool did not show any parasites. EEG findings were sharp and spike waves suggestive of generalized seizure.

CT scan of brain showed small isodense right posterior parietal lesion of 5 mm size with tiny elements of calcific nodule and perifocal edema. CECT revealed intense nodular post contrast enhancement (Figure 1). MRI of brain could not be done due to financial constraint.

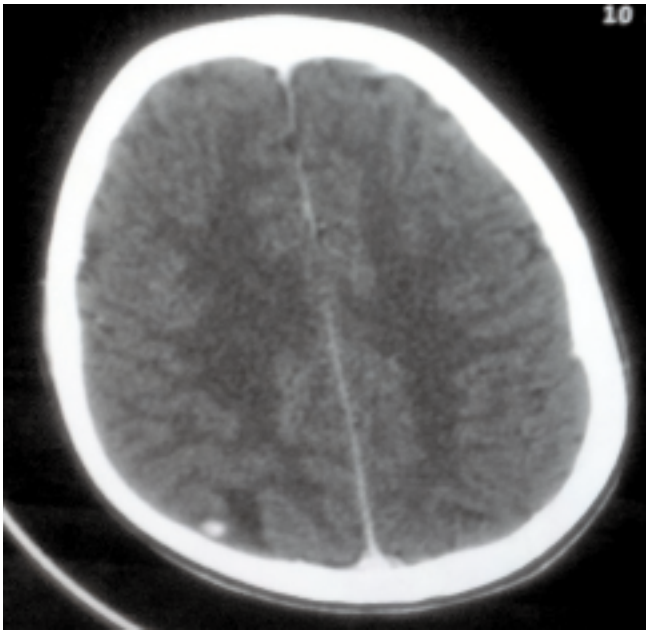

Figure 1. CT scan of brain showed small isodense right posterior parietal lesion of 5 mm size with calcific nodule and perifocal edema.

Basing on the clinical and neuroimaging, the child was diagnosed as a case of neurocysticercosis and treated with albendazole (15 mg/kg/day) in divided doses along with prednisolone (2 mg/kg/day) for 7 days. Valproic acid was selected as the antiepileptic and in view of benefit as a mood stabiliser. The child was discharged after 7 days with valproate and advised to follow up in regular interval. At three months of follow up there was significant decline in psychotic symptoms and complete resolution of lesion in CT scan of brain (Figure 2).

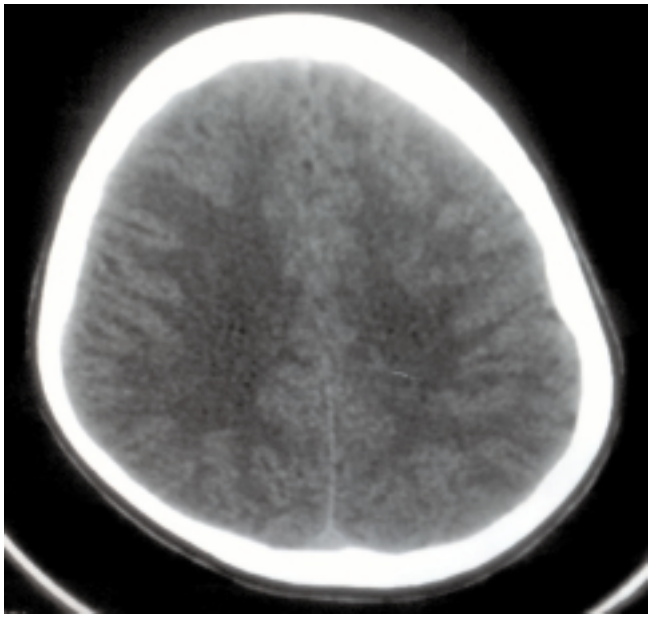

Figure 2. Follow up CT scan shows complete resolution of lesion.

3. Discussion

The clinical presentation of neurocysticercosis varies with the location, size, and number of cysts, their developmental stage and the intensity of immune response of the host to parasite. Psychiatric disorders have been reported in 9% to 65% of neurocysticercosis patients[5,6]. Depression being the most frequent diagnosis (52.6%) followed by psychotic disorder (14.2%), generalised anxiety disorder (8.7%), panic disorder (13%) and phobia (13%).

The study also concluded that active disease and raised intracranial pressure were associated with higher psychiatric morbidity and history of mood disorders was strongly related to current depression. However no correlation was found between mental disturbances and number and type of lesions, severity of neurological deficit, seizures and use of steroids. Our case presented with euphoria, manic episodes and agitation but sensorium and orientation were intact all the time

during the course of illness.

Vijayan *et al* and Venkataraman *et al*[7,8] reported cases with schizophrenia or manic episode with disturbed sleep, hallucinations, paranoid delusions, seizures and intellectual deterioration without any evidence of raised intra-cranial tension. Manic symptoms and intellectual deterioration were also observed in our case from the very beginning without increase in intracranial tension which can be correlated with small size of the lesion and its anatomical localisation in parietal lobe.

In our patient the involuntary movement was dystonic in character but neuroimaging didn't reveal a lesion in putamen, thalamus, globus pallidus or caudate nucleus, which are normally identified as the sites for hemidystonia. We attributed dyskinesia as one of the parietal motor syndromes. The association of focal dystonic posture with lesion in parietal association cortex indicates that it may be feature of damage of brain cortical area far from basal ganglia. In addition it reflects impairment of multiple sensory feedback pathways to motor programs and this plays a role in some hyperkinetic movement disorders[9,10].

The clinical presentation of neurocysticercosis is diverse and perplexing. In developing country, its polymorphous symtomatology is only mimicked by neurotuberculosis which can be differentiated by history, clinical findings, serological and neuroimaging studies. Our case is unique in view of early manic-psychotic symptoms without manifestations of seizure or other neurological abnormality. So it should be considered as a differential diagnosis in all cases with neurological as well as psychiatric symptoms, particularly in endemic areas.

Conflict of interest statement

The authors declare they have no conflict of interests.

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