Acute Psychosis: An unusual presentation in Disseminated Neurocysticercosis

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ABSTRACT

Neurocysticercosis (NCC) is a common parasitic infestation of the central nervous system worldwide. It is caused by the ingestion of Taenia solium eggs. Its clinical manifestation are very varied ranging from headache, seizure, increased ICT to neuropsychiatric manifestation. The present case highlights the myriad of clinical features of NCC. Patient presented present to us with seizures and acute psychosis. On further evaluation he was diagnosed as a case of disseminated neurocysticercosis. He responded to treatment with anti-helminthic drugs without recurrence of psychosis.

Keywords: Acute psychosis, Disseminated Neurocysticercosis, Neuropsychiatric symptoms

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INTRODUCTION

Disseminated cysticercosis is due to infection by *Cysticercus cellulose*, the larval form of *Taenia solium*. Mode of transmission of Cysticercus is via feco-oral route. Widespread dissemination of the cysticerci can result in the involvement of almost any organ in the body. Neurocysticercosis (NCC) is the common parasitic infestation of the central nervous system worldwide. The clinical manifestations of NCC are varied and depend not only on the site, number and stage of the larval parasite, but also on the status of the host's immune response [1]. While some cases of NCC may remain asymptomatic, most have been reported to present as seizures, headache, raised intra cranial tension (ICT) due to blockage of CSF flow, stroke, dementia, neuropsychiatric symptoms, ophthalmologic and endocrinological manifestations [2]. Disseminated Neurocysticercosis (DNC) is an uncommon manifestation of this common disease [3]. Here, we present a case of Disseminated Neurocysticercosis who presented as acute psychosis.

CASE PRESENTATION

A 15yr old male from a rural background admitted in psychiatry ward with chief complaint of abnormal behaviour and

decreased sleep from last one month. On detailed evaluation, it was found that he was apparently well one month back when he developed an episode of vomiting and headache of moderate intensity which was followed by an episode of generalised tonic clonic seizure. For this he consulted a medical practitioner, where he was prescribed antiepileptics, but even with the treatment there was no improvement in his seizure frequency. Gradually his condition deteriorated and he started talking irrelevantly, his self-care decreased, oral intake decreased, sometimes he used to become aggressive without any reason, started making gestures, tearing his clothes and urinating in front of others. His duration of sleep was also significantly decreased. There is no past or family history of psychiatric illness.

On examination the patient was a young male appropriately dressed but poorly groomed and having poor hygiene. He was not looking in touch with the surrounding, talking irrelevantly, having poor eye contact, uncooperative and rapport could not be established. His overall psychomotor activity was increased. Speech was spontaneous, increased in rate, volume and intensity, content was irrelevant but comprehensible. Other parts of the mental status examination could not be done as patient was uncooperative. Rest of the general physical and neurological examination was normal. He was admitted and routine investigation with MRI-Brain was done considering the possibility of any organic cause. His previous CECT-Head was Normal. **Investigations:** All investigations came out to be normal except the MRI-Brain which showed disseminated ring enhancing lesions in different stages all over the cortex extending into the orbit and neck muscles (Figure-1). He was diagnosed as a case of disseminated neurocysticercosis and transferred to the neurology unit.

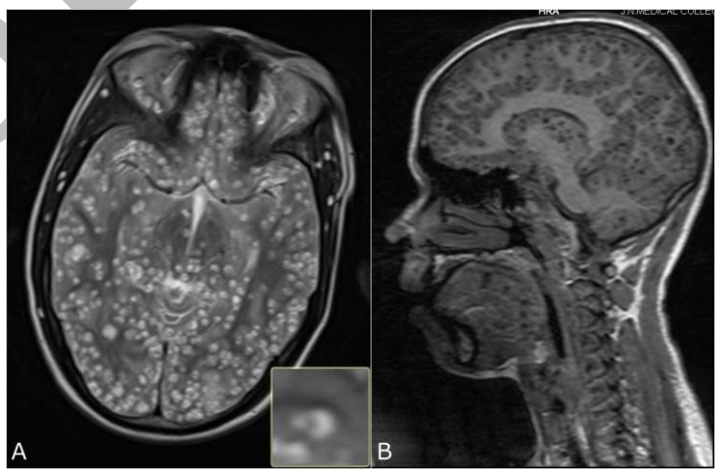

Figure 1 (A and B): MRI Brain showing multiple ring enhancing lesions with eccentric nodules (Inset) suggestive of neurocysticercosis. (A) T-2 weighted sequence showing the presence of cysticerci in the orbit as well as nasal sinuses and also illustrating the conglomeration of lesions in brain typical of NCC. (B) T-1 Weighted sequence showing the dissemination of cysticerci throughout the brain as well as tongue and neck muscles.

Treatment: Patient was started on albendazole (400 mg twice daily), Prednisone (60 mg per day tapering doses) and the antiepileptics were continued. Patient was also given injection haloperidol 5 mg, i.m. on as and when required to control the initial psychosis and the dose was gradually reduced over a period of a week. **Outcome:** Patient improved and the antipsychotics were discontinued. Patient was asymptomatic on antiepileptics on the last follow-up visit.

DISCUSSION

Cysticercosis is caused by the larval form of the cestode *Taenia solium*. Since ages, this pork tapeworm was known as "armed tapeworm" of man but was differentiated from T. saginata only in 1782 by Goez [4]. Neurocysticercosis (NCC) represents the infection of CNS by *Cysticercus cellulose,* the larval form of *Taenia solium.* NCC is the most frequent parasitosis of the CNS [5]. NCC is endemic in many parts of the world, commonly seen in central and South Africa, Mexico, Central and South America, Pakistan, China and India. NCC is linked to poor socioeconomically status. Epidemiological studies have shown association between seropositive people, infected pigs and unsanitary disposal of feces.

The larvae get inside the body by the ingestion of Taenia solium eggs in fecally contaminated food or water. The eggs hatch after reaching the intestine and release the larvae. After penetrating the intestinal mucosa, larvae rich the blood stream and get distributed throughout the body. The skeletal muscles, eyes, or the brain are organs where the larvae can localise as a cyst. [2,6]. Seizure is the most common clinical manifestation in the intraparenchymal NCC. It is estimated that between 5-20% of all cases with epilepsy in India have neurocysticercosis [7]. Extraparenchymal NCC usually refers to the infection of ventricles and subarachnoid spaces [2]. Involvement of the subarachnoid space may lead to the visual and hormonal impairment by direct compression of the hypophyseal stem and the optic nerves, it is reported in 3.5% of the patients. [8]. 4% prevalence of NCC was reported in rural areas of the developing world [9]. Cognitive and behavioural problem in NCC are more commonly associated with hydrocephalus or multiple lesions [2,8]. Neuropsychiatric symptoms have been frequently associated with NCC, however the severity of neuropsychiatric symptoms may correlate with treatment of NCC with anti-parasitic drugs and with an increase in CNS inflammation[9]. The most commonly reported

neuropsychiatric symptoms are: confusion, disorientation, memory loss, hallucinations, psychomotor incoordination, progressive deterioration of language ability, and mental deterioration [10].

38 cases of neurocysticercosis were reviewed by Forlenza et al. and reported 65.8% of having psychiatric illness and 87.5% of having cognitive dysfunction. In psychiatric problems, depressive disorders and psychosis was seen in 52.6% and 14.2% of cases, respectively [9]. 753 cases were reviewed by Sotelo et al. and reported intellectual deterioration in 15.8%, psychosis in 4.7%, and behavioural disturbance in 2.7% [11]. Neuropsychiatric symptoms were more common in cases with hydrocephalus or multiple lesions. 127 cases were reviewed by McCormick et al.[12] and found that 38% had intracranial hypertension, 10% had increased intracranial pressure and dementia. Three cases of disseminated cysticercosis (involving brain and muscle tissue), all exhibited dementia, and two cases experienced psychosis were reported by Wadia et al.[13]. Intracranial hypertension and progression of disease correlated with higher levels of psychiatric comorbidity.

NCC have varied presentation including pure motor hemiparesis, alexia and manic episodes [14-17]. Schizophrenic and manic- like symptoms in the initial phase of neurocysticercosis and 5 cases of true dementia in 450 (1.1%) patients were reported by Dixon and Lipscomb[18]. The neuropsychiatric symptom of NCC can be explained due to mechanical alterations in CSF pressure and inflammatory injury of the brain[19]. It can also be assumed that the parasites may induce expression of several classes of genes in the CNS of the host and these genes encode proteins or neuropeptides involved in the regulation of important physiologic and behavioural processes [20, 21].

CONCLUSION

- CNS symptoms of NCC may be variable.
- In cases of acute psychosis organic causes should always be kept in the differential diagnosis.
- NCC may present with neuropsychiatric symptoms.

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