Neurocysticercosis Presenting with Psychosis


Abstract

Cysticercosis consists of infection with the small bladder-like larvae of the pork tapeworm Taenia solium. The life cycle of parasite is maintained between man and pig infected with cysticerci. Epilepsy is the most common presentation of neurocysticercosis; focal signs, headache, involuntary movements and global mental deterioration are other symptoms.1 Psychosis is a rare presentation and may be seen in up to 5% of patients.2 We present a 25 years old male, who had been under treatment from psychiatry OPD for psychosis for one year, developed generalized tonic-clonic seizures. CT scan of brain revealed multiple calcified and hypodense lesions with surrounding oedema. Histopathological examination of subcutaneous nodule confirmed the diagnosis of neurocysticercosis.

INTRODUCTION

Cysticercosis is known since Hippocratic era and is the commonest helminthic infection of human central nervous system.1 It is an infection with larval stage of T. solium, the pork tape worm. The life cycle of this two host zoonotic cestode, human are the only definitive host and harbour the adult tape worm where as pigs are intermediate hosts. The tape worms are acquired by ingesting undercooked pork containing cysticerci. Human cysticerci however is a faecal-oral infection acquired by ingesting eggs excreted in the faeces of a human tape worm carrier. Individual harboring an adult worm are at increased risk of cysticercosis, probably through faecal-oral contamination and also because of internal autoinfection due to reverse peristalsis.

Ingested eggs, activated by gastric and duodenal secretions, develop into invasive larvae (oncosphere) in the small intestines which migrate across intestinal wall and are carried by the bloodstream to the sites where these settle and mature into cysticerci. There process can take place anywhere in the body but there is preference for subcutaneous tissue, muscle and central nervous system. This process takes approximately two months. The ability of parasite to survive in central nervous system more frequently is possibly because of the limited immune response offered by the host in the central nervous system.

CASE REPORT

A 25 years male presented to Psychiatry OPD with symptoms of restlessness, irritability, and episodic anger outburst, delusions of reference and persecution, auditory and visual hallucinations (hearing divine voices and seeing snakes/fires, etc.) and disturbed sleep for a period of about one year. On exploration, he gave history of severe headache off and on, for which he was treated at local hospital with analgesics. His behavioural symptoms aggravated for last two months for which he was brought to Psychiatry OPD and diagnosis of psychosis NOS (ICD-10) was made.

Treatment commenced with a combination of novel antipsychotic risperidone 4 mg/day and clonazepam 2 mg/day. His psychotic symptoms improved partially over a period of four weeks but developed headache followed by generalized tonic-clonic seizures and presented to Medical OPD. On examination he was drowsy and was responding inappropriately to some of the commands and had multiple subcutaneous nodules. Rest of the examination was normal. Haemogram showed slight eosinophilia (5%). X-ray right upper limb showed multiple calcified spots (Fig. 1). CT scan brain revealed multiple calcified and hypodense lesions with surrounding oedema (Fig. 2). He was put on tab phenytoin sodium 300 mg OD, inj mannitol along with prednisolone 40 mg OD. Histopathological examination of biopsy from subcutaneous nodule confirmed the diagnosis of cysticercosis. During hospital stay his psychotic symptoms worsened and he was found to have mixed psychotic and affective (depressive) symptoms along with extrapyramidal symptoms. He was put on olenzapine 10 mg/day with venlafaxine 75 mg/day. He was continued on anticonvulsants, antipsychotic and steroids were tapered off over next six weeks. He was free of seizures and psychiatric symptoms on follow up visits at eight weeks and six months.

DISCUSSION

Neurocysticercosis is a pleomorphic disease whose manifestations vary with the number, size and topography of...
the lesion and the intensity of the immune response of host to parasite. Epilepsy is the most common presentation and occurs in 50-80% of patients. Generalized tonic-clonic seizures are associated with multiple lesions whereas single lesion can present as simple or complex partial seizures. Myoclonic seizures have also been described with this disease. Headache, chronic migraine-like or those associated with intracranial hypertension, both can also occur. Stroke, ischaemic and haemorrhagic both can also occur. Clinical manifestations in order of decreasing frequency are seizures (80%), headache (40%), visual changes (20%), confusion (15%), ataxia (6%), psychosis (5%) and in minority, cranial nerve palsies or other focal neurological manifestations.\(^1,2\)

Attempts have been made to classify various psychiatric manifestations in neurocysticercosis according to site of lesion. A patient can present with psychosis for some time, have seizures before being diagnosed as neurocysticercosis,\(^3,4\) a patient can undergo various neurological manifestations like headache, intracranial hypertension or hydrocephalus before being diagnosed as neurocysticercosis.\(^5\) Similarly our patient presented with psychosis for one year before having seizures and finally diagnosed as neurocysticercosis. As a general rule ventricular cysticercosis and subarachnoid cysticercosis are usually associated with meningitis and/or intracranial hypertension resulting in cognitive dysfunction like attention deficit, impaired consciousness and delirium. Ventricul-subarachnoid forms are prone to present as psychomotor agitation, sleep-wake cycle disturbances and other behavioral symptoms suggestive of cognitive dysfunction. On the other hand, patients with parenchymal cysts and calcifications are prone to experience neuropsychiatric manifestations of epilepsy, intracranial hypertension and space-occupying lesions. Dementia has been associated with massive and scattered infection of brain, parenchyma and subacute form of intraventricular cysticercosis.\(^6\)

Most probably, degenerating cysts and the reactive inflammation with in the adjacent nervous tissue, which are strong determinant of neurocysticercosis induced epilepsy, may well be the trigger of psychiatric symptoms particularly among predisposed individuals. Psychosis possibly correlates with intracranial hypertension not with disease activity. Previous history of depressive disorder may strongly be associated with current depression or psychosis. In one study conducted in Brazil, neuropsychiatric dysfunction was detected in majority (87.5%) although severe cognitive abnormalities were less frequent (15.6%). Memory and language were altered in 78%, paraxis and motor dysfunction (50%), reading (28%), writing (0.6%) abnormalities other deficit present.\(^6\)

CT scan and MRI will provide objective evidence about topography of the lesions and the degree of the host inflammatory response to the parasite. Immunoblot (western blot) is the best available serological test for \textit{T. solium} with 98% sensitivity and 98% specificity in patients with more than one lesion. A set of diagnostic criteria based on neuroimaging studies, neurological tests, clinical and exposure history has been proposed by Del Brutto and colleagues. Major criteria (typical findings on neuroimaging, demonstration of specific anticysticercal antibodies or the presence of typical cigar-shaped calcifications in muscles) are combined with minor criteria along with epidemiological data to suggest a probable or possible diagnosis of neurocysticercosis.\(^1,2\)

Symptomatic treatment is very important. Seizures
secondary to parenchymal neurocysticercosis can be controlled with anticonvulsants, however optimal length of therapy has not been determined and it is difficult to withdraw treatment. Currently albendazole is the drug of choice for antiparasitic therapy (15 mg/kg/day for 7 days with steroids), although a recently described single day praziquantel regimen (75 to 100 mg/kg in three doses at two hour interval followed by steroids 6 hours later). Longer courses may be needed in patients with multiple lesions or subarachnoid cysticercosis. In patients with severe cysticercotic encephalitis, antiparasitic drugs should not be used for the fear of worsening cerebral oedema and fatal herniation. In these cases high dose corticosteroids are mainstay of treatment. Surgery is limited to ventriculo-peritoneal shunt to relieve obstructive hydrocephalus and excision of single cysticercus.1,2 Most patient with neurocysticercosis have chronic depression and mild to moderately severe brain pathology (few cysts and/or calcifications) and in such cases psychiatric treatment is very helpful and should follow guidelines for the treatment of other organic illness. Regarding severe form of neurocysticercosis (massive infection with intracranial hypertension, space-occupying lesion, intraventricular cyst), psychiatric treatment should follow neurological and neurosurgical procedures. Psycho-pharmacological treatment should be complementary to neurological care.6

REFERENCES