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NEUROCYSTICERCOSIS PRESENTING AS SCHIZOPHRENIA: A CASE REPORT

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Psychiatric manifestations of neurocysticercosis presenting mainly as organic psychosis are well documented. A patient with neurocysticercosis who presented with a schizophrenia like functional psychiatric illness is hereby reported.

Key words: neurocysticercosis, seizure, schizophrenia, haloperidol, albendazole, symptomatic psychosis.

INTRODUCTION

Neurocysticercosis is the most common parasitic disease in the world which affects the central nervous system (Grisolia & Wiederhold, 1982). Although it has been reported globally (Feng et al, 1979; McCormick et al, 1982; Grisolia & Wiederhold, 1982), important foci exist in China, India, Pakistan, Indonesia, Philippines and Latin America (Muller et al, 1987). Neurocysticercosis produces a variable clinical picture (Sotelo et al, 1985). Although seizures, intracranial hypertension, hydrocephalus and stroke are the most frequent clinical presentations (McCormick et al, 1982; Tandon, 1983; Venkataraman, 1989), a few reports of psychiatric manifestations have been published from time to time. Psychotic episodes occur in about 4.7% of patients with neurocysticercosis (Sotelo et al, 1985), who usually present with an organic psychosis; rarely however, is this the presenting symptom. Our case merits attention as the presenting symptoms were those of a functional psychosis.

CASE REPORT

A 25 year old, illiterate married Hindu male, an occasional non- vegetarian from a lower socio-economic class and a hawker by profession presented with a three month history of gradual change in behavior in the form of irrelevant talk, laughing and weeping without reason, fearfulness, suspiciousness and sleep disturbance. The patient was initially able to take care of his personal upkeep and hygiene but after two months of illness these functions deteriorated. Occasional forgetfulness was also reported. He had one attack of a generalized tonic-clonic seizure one week prior to attending the psychiatric out patient department. No past or family history of mental illness was present.

On mental status examination, he was well oriented to time, place and person, cooperative,communicative and responded well to questions asked. No evidence of memory impairment was found. Delusions of persecution and reference were present. No perceptual anomaly was detected. Insight was partial; he accepted the illness but attributed the cause to evil spirits. Psychological testing on the Bender Gestalt Test and Post Graduate Institute (PGI) Memory Scales did not reveal any evidence of organicity. A provisional diagnosis of schizophrenia (ICD-9) with primary generalized tonic-clonic seizure was made. The patient was treated with haloperidol 20 mg/day and advised follow up after three weeks. He was referred to the Division of Neurology for opinion and management of seizure.

Examination by the neurologists revealed subcutaneous nodules over the trunk, both thighs and calves. These nodules were soft, mobile and nontender. Examination of cardiovascular system, chest and abdomen revealed no abnormality. Detailed central nervous system examination was normal. He was treated with phenytoin sodium, 300 mg/day in divided doses. Investigations were carried out and he was asked to return after three weeks.

After three weeks of follow up, there was no improvement in his behavior. However, he did not have any seizure during this period. Investigations were as follows: Leukocytosis (total leucocyte count 12,000/mm^3) with evidence of eosinophilia (eosinophil count 14%) was present in peripheral blood smear examination. Erythrocyte sedimentation rate was normal. General blood picture, blood urea, blood sugar, serum creatinine, electrolytes and liver function tests were within normal limits. Serum VDRL was non reactive. Stool examination was negative for Taenia solium in three consecutive early morning stool samples. Examination of fundus oculi did not reveal any sign of raised intracranial tension or deposits of cysticerci. Roentgenograms of skull, thighs and calves did not show any calcification. An electroencephalogram (EEG) showed generalized inter-ictal discharge in the form of intermittent high amplitude sharp and slow waves, lasting for 1-2 seconds. There were no spikes, phase

reversal or voltage asymmetry. Cerebrospinal fluid examination revealed lymphocytic pleocytosis, raised protein and normal glucose level. No eosinophils were seen in the sediments of centrifuged CSF. Cranial computerized tomography scan showed multiple, high attenuating (less than 5 mm size) pin head lesions with perifocal oedema in fronto-parieto-occipital regions. A few calcified lesions were also present. Ventricle size was normal. There was no evidence of generalized oedema.

A provisional diagnosis of neurocysticercosis was made; histopathology report of subcutaneous nodule confirmed the diagnosis of cysticercosis cellulosae. The patient was admitted in the neurology ward and was treated with albendazole, at a dose of 15 mg/Kg/day in divided doses for one month. The dose of haloperidol was also increased from 20 mg to 30 mg per day. A cranial CT scan was repeated three months after albendazole therapy, which showed a decrease in the number of pin head lesions; however, calcified lesions persisted as such. There was no evident perifocal oedema. Subcutaneous nodules started regressing after albendazole therapy and completely disappeared in four months. Significant improvement in psychiatric symptoms was also observed following albendazole therapy. Delusions of persecution and delusions of reference were not found on mental status examination. Insight also improved; instead of attributing the illness to evil spirits, the patient accepted having a physical illness. Haloperidol was gradually tapered and stopped after three months. However, considering the presence of persistent calcified lesions in the brain, phenytoin sodium was continued.

DISCUSSION

Psychiatric manifestations of neurocysticercosis have been reported mainly in the form of organic psychosis. Stepien and Chorobski (1949) and Stepien (1962) observed mental changes in 28% of cases and recorded a loss of orientation in space and time as well as visual and auditory hallucinations as the most frequent symptoms. Apathy or euphoria, confusion or agitation, impairment of memory and slowing in mental process were seen less frequently. Dixon and Lipscomb (1961) observed mental changes in 8.7% of cases. Sotelo et al (1985) reported occurrence of intellectual deterioration, psychotic episodes and disturbances of behavior in one out of every five patients, especially in those with hydrocephalus or in patients with parenchymal lesions. From India, Armstrong in 1888 first reported a case of extensive cysticercosis of the brain in a lunatic who died in the Madras asylum. Kala and Wig (1977) reported two cases of organic psychosis with marked disorientation, excitement and irrelevant talk with raised intracranial tension. They also reported a case of slowly progressing dementia with loss of memory and impairment of judgement. Trivedi et al (1983) reported a case of neurocysticercosis in an alcoholic who presented with a history of recurrent generalized tonic-clonic seizures followed by organic psychosis.

Our patient presented mainly with a functional psychiatric illness. History of a recent seizure and the presence of subcutaneous nodules were the only pointers to organic illness. Antipsychotic therapy was of no help initially but when albendazole therapy was added, significant improvement in behavior was reported. Although the possibility of purely functional psychiatric illness (schizophrenia) cannot be ruled out, the definite evidence of neurocysticercosis, clinical response to haloperidol only after the addition of specific albendazole therapy and previously reported psychiatric abnormalities in patients with neurocysticercosis do suggest the possibility of a symptomatic psychosis as a result of neurocysticercosis. To the best of our knowledge, this is the first report where organic psychiatric findings are not part of the presenting features.

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