

PSYCHIATRIC MANIFESTATIONS OF CYSTECERCOSIS : REVIEW OF LITERATURE AND CASE REPORT

J. K. TRIVEDI¹, M.D. (Psychiat.)

R. K. SINGH², M.D. (Psychiat.)

P. K. DALAL³, M.B.B.S.

SUMMARY

This article describes the report of a case of cerebral cystecercosis, diagnosed only after C. T. head scan, who presented primarily with a picture of an organic psychotic condition. The literature in relation to psychiatric aspects of cystecercosis is reviewed.

REVIEW OF LITERATURE

Cystecercosis is a common parasitic disease affecting the C. N. S. It has been reported from all parts of the world (Lombardo and Mateos, 1961; Olive and Angulo-Revero, 1962; Franco-Ponce, 1974; Gonzalez-Cruchaga, 1974; McCornick *et al.*, 1982; Grisolia and Wiederholt, 1982; Arseni and Samiter, 1957; Arseni and Cristescu, 1972; Stepien, 1962; Dixon and Lipscomb, 1961 and Feng *et al.*, 1979). Indian reports over the past 3 decades (Singh *et al.*, 1963; Chandy and Isiah, 1952; Jacob and Mathew, 1968; Dinakar *et al.*, 1970; Vijayan *et al.*, 1976, 1977; Venkataraman *et al.*, 1977, 1979, 1982, Ahuja *et al.*, 1978 and Wadia, N. H., 1973) suggest higher incidence of cystecercosis in North-Western States.

The earliest documentation of cerebral cystecercosis was made by pernoli in 1550. It was first reported from India by Surgeon H. Armstrong, who, in 1888, detected extensive cystecercosis of the brain in a hantic who died in the Madras asylum.

Man is the definitive host of *Taenia Solium*, the pork tapeworm. The usual route of infestation is through ingestion of inadequately cooked pork containing embryos. Cystecercosis is a systemic infection that occurs when a human

being becomes the intermediate host. This results from ingestion of food contaminated by human faeces containing eggs, from faecal-oral autoinfection or from autoinfection caused by reverse peristalsis. The eggshell is digested in the stomach and releases onco-spheres that then penetrate the intestinal mucosa, enter the blood stream and lodge in brain and spinal cord parenchyma, ventricles and subarachnoid space, meninges, eyes, skeletal muscles and, rarely, the heart. The incidence of invasion of brain may be as high as 60%. The clinical presentation of neurocystecercosis is diverse and perplexing and the polymorphous symptomatology seen in neurocystecercosis is only mimicked by neurotuberculosis (Venkataraman *et al.*, 1977) and neuroyphillia (Srinivasan *et al.*, 1977) in developing countries like India and multiple sclerosis in Western countries (Chordenas, 1962).

Stepien and Chorobski (1949) and Stepien (1962) observed mental changes in 28 percent cases and recorded loss of orientation in space and time, visual and auditory hallucinations as the most frequent symptom, and apathy or euphoria, confusion or agitation, impairment of memory and slowing in mental process less frequently. Lombardo and Mateos

1. Lecturer
2. Research Associate
3. Jr. Resident.

Department of Psychiatry K.G.'s Medical College, Lucknow.

(1961) noted mental deterioration in about a sixth of their cases whereas Dixon and Lipscomb (1961) observed mental changes in only 8.7 percent of cases. Vijayan *et al.* (1977 and 1979) and Venkataraman *et al.* (1982) stressed upon the primarily psychiatric presentation of neurocysticercosis occurring without any sign of intracranial hypertension, which had been an important feature in cases with mental changes described by others. They reported cases of neurocysticercosis with schizophrenic or manic behaviour with disturbed sleep, hallucinations and paranoid delusions, seizures and intellectual deterioration, without any evidence of raised intracranial tension. Kala and Wig (1977) reported two cases of acute organic psychosis with marked disorientation, excitement and irrelevant talk with raised intracranial tension whereas Venkataraman and Vijayan (1979) reported a similar case without raised intracranial tension. Kala and Wig (1977) also reported a case of a slowly oncoming dementia with loss of memory and impairment of judgement, without raised intracranial tension.

CASE REPORT :

The patient, a 42 years old, non-vegetarian, married, Hindu, just-literate male, an unskilled labourer, migrated to Lucknow about 16 years ago from Punjab. He presented to our out patient department for the first time with the primary complaints of excessive alcohol intake for last 15 years. 15 days prior to consultation he suddenly developed abnormal behaviour characterised by irritability, annoyance, stubbornness, abusiveness, assaultiveness, absconding tendencies, illogical talking, forgetfulness, sleep disturbance and neglect of personal hygiene. He was disoriented to time, place and person and was manifesting ataxia and tremors of hands and feet. He was markedly anxious, apprehensive and his

speech was slurred. A provisional diagnosis of transient organic psychotic condition with a possibility of delirium tremens was entertained. He was advised oral chlordiazepoxide and injectible thiamine and to come within a week for hospitalization. However, it was only 3 months later that the patient was brought again with similar complaints and was then admitted. Detailed enquiries from his brother-in-law and colleagues revealed the history of seizures of grand mal type thrice in the previous one year and nodular swellings over the trunk and thighs for the last 5-6 years. His physical examination confirmed the presence of soft, nodular, subcutaneous swellings trunk. Central nervous system examination revealed the presence of generalized hyperreflexia with bilateral planter flexor and bilateral positive wartenburg's sign, coarse tremors over fingers and tongue and mild ataxia. There were no signs suggestive of focal neurological deficit or of an intracranial space occupying lesion. His mental status this time revealed catastrophic reaction, disorientation for time and place, global impairment of memory and intelligence with loss of insight and judgement.

Biopsy of the nodule confirmed the diagnosis of cystecercosis cellulosa. There was no pleocytosis or eosinophilia. ESR, PCV, GBP, Blood Urea, Blood sugar and liver function tests were within normal limits. Serum WR/VDRL was negative. 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 cysteceri. Roentgenograms of skull, cervical spine, chest, abdomen, forearms, upperarms, thighs and calves did not show any calcification. Electroencephalogram and carotidangiogram were within normal limits. Nerve conduction velocity showed no evidence of peri-

pheral neuropathy. Electrocardiogram and cerebrospinal fluid examination were normal. Psychological testings viz. Rorschach testing and B. G. T. revealed evidences of organic involvement.

The patient was then subjected to C. T. head scan which revealed two definite calcified areas surrounded by small oedematous zones. The diagnosis of cerebral cysticercosis was established. Absence, in the head scan, of a racemose pattern of cysticerci, throttled appearance of ventricles, or any sign of raised intracranial tension negated any surgical intervention and the patient was put on conventional treatment of antiepileptics and steroids. As expected the patient had no further seizures and has not shown any further deterioration although any improvement was also lacking.

There is, as yet, no drug which can eradicate the cysticercus stage in man though newer drugs are available to treat tapeworm infestation. Of late, radio-immunotreatment with Iodine¹³¹ labelled anticysticercosis antibodies had been reported to be of value in the treatment of generalised cysticercosis of nervous system (Venkataraman *et al.*, 1982).

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