

## Case Report

# Neurocysticercosis in an aircrew: A case report

Wg Cdr S Damodaran\*

Neurocysticercosis is known to present with varied clinical manifestations. Epilepsy with variable neurological deficits is one of the common mode of presentation. Psychiatric symptoms with behavioural abnormalities is an unusual clinical presentation. A case of neurocysticercosis in an aircrew who initially presented with unspecified psychosis and later developed epilepsy is reported.

**Keywords:** Aircrew evaluation, epilepsy, behavioural disorder.

The most common parasitic disease of the nervous system is neurocysticercosis [1], caused by the larval form of the pork tapeworm *Taenia solium*. It is widely distributed and is common in many of the developing countries. In India, it forms 1.2-2.5 percent of all intracranial space occupying lesions seen in specialized centres [2,3]. The condition continues to baffle the neurologist because of varied clinical manifestation. Nearly 5-10% of patients can be asymptomatic [2]. It can present with epilepsy, hydrocephalus, paresis, chronic meningitis and psychiatric illness. The most common clinical presentation is epilepsy [4, 5]. Presentation with psychiatric symptoms is unusual [5].

Cerebral cysticercosis often poses a diagnostic challenge to the clinician because of its protean manifestation mimicking many neurological and psychiatric disorders. This is mainly related to the topographic location, the numerical density and the host immune reaction to the cestode larval parasite. The host response and the resultant brain parenchymal changes, in turn are dependent on the morphological viability of the cyst. The larval cyst remains viable in the intermediate host

for many years (approximately 5-10 years in human) [6].

The immunological reaction to cysticercosis is a complex one [7]. Young and viable cyst incites little or no inflammatory reactions. There may be liquefaction in the centre of cyst with pus formation (granulomatous abscess). The surrounding tissues may show variable degree of edema and vasculitis. Well preserved cysts may show a fibrous capsule separating them from brain parenchyma and may be devoid of inflammation. Finally there is complete resolution, fibrosis, gliosis or calcification. When the parasite enters the brain in multiple batches one may come across the temporal evolution of the pathological lesions in the same brain in response to that host reaction [5]. Most symptoms due to parenchymal neurocysticercosis reflect relatively recent host immune response with inflammation or epileptiform foci formed due to healed/healing lesions with prior inflammation.

\* Graded Specialist (Aviation Medicine), 2 AMTC, AF Academy, Hyderabad

A case of neurocysticercosis in an aircrew who initially presented with symptoms of unspecified psychosis and after 5 years developed epilepsy is presented.

### **Case report**

A 37 years old aircrew with 2081 hours of flying while on a ground tenure was admitted on 07 Dec 92 with a h/o shouting at a junior officer after an altercation. APMSF-10 revealed erratic behaviour, social withdrawal, suspiciousness, restlessness and persecutory delusions. There was no family h/o mental illness. Personal history showed stressors in 1991 when he failed in a missile test course after which he became suspicious and occasionally agitated and earlier had an aborted love affair. He was a vegetarian, married and had two children. Past history revealed a compression fracture of D12 and L1 in 1980 after an ejection. He regained his flying status. During 1986-87 while on a QFI tenure, he used to suffer sensory blunting in the form of "as if left arm and left leg are at times flying off and some times being drained of its blood".

Physical examination revealed an average built individual. Height 167 cms, weight 60 kg, pulse 80/min, BP 130/80 mm of Hg. There was no subcutaneous nodule or lymphadenopathy. Initial mental state examination (MSE) by psychiatrist showed a hostile, suspicious, agitated, withdrawn individual who on serial MSE and ward observation showed persecutory delusions, ideas of reference, feeling of rejection, reduced biological drives and impaired insight and judgement. He also had strained interpersonal relationship with his family members and wife. Psychometric tests showed paranoid traits and psychotic tendencies. Neurological examination did not reveal any focal neurological deficit. Fundi were normal. Blood DLC P62 L31 B2 E5; Hb 13 gm%, sugar fasting 78mg% and post

prandial 110 mg%; Blood urea 28 mg%, creatinine 1mg%. EEG normal. CT scan head showed a pin head calcified lesion in right parietal lobe, reported as healed granuloma and taken as non-explanatory for the psychiatric symptoms. He was diagnosed as a case of unspecified psychosis. He was treated with antipsychotic drugs, showed improvement and was followed up in low medical category with medication. In the follow up period of the next 5 years he continued to exhibit some residual features like reduced socialization, guardedness and paranoid ideas.

In Oct 97, while he was still under follow up for unspecified psychosis he had left lower limb onset partial sensory motor seizure with generalization. He had two recurrences one in Nov 97 and another in Dec 97.

CT scan done now showed multiple cystic lesions in right cerebral hemisphere with one of them showing an eccentrically placed mural nodules. MRI showed two cystic lesions in Rt parietal lobe (one in centrum semi ovale and another in medial surface para sagittally). Repeat CT scan after 2 months showed another cystic lesion in Rt frontal lobe with slight perifocal edema. There was no evidence of ocular cyst. EEG showed epileptic discharges. Serum Na 138 mEq/l, serum K 4.0 mEq/l and serum chloride 102 mEq/l. Blood sugar, urea, creatinine and cholesterol were within normal limits. Initially he was treated with dilantin sodium for epilepsy and albendazole for cysticercosis. Subsequently he was put on mazelol considering its double benefit for psychosis and seizure. Anti-psychotic drugs were continued.

### **Discussion**

The chronological events in this patient started in 1986-87 when the patient used to feel "as if

left arm and left leg are at times flying off and some times being drained of its blood". Subsequently psychiatric symptoms in the form of agitation, delusional ideas, strained inter personal relations were noted by family members and colleagues, which finally culminated in his admission for psychiatric evaluation in late 1992. CT scan at that time showed pin head calcified lesion in R1 parietal lobe which was considered non-contributory. While under psychiatric care he developed epilepsy 5 years later and the CT scan and MRI now showed multiple cystic lesions in the parietal and frontal lobes of the right hemisphere. In the absence of demonstrable cyst in the temporal lobe or limbic area, the contribution of neurocysticercosis to the psychiatric symptoms in this patient may be questionable. However, the fact that the psychiatric symptoms did not fit in to any of the specified psychosis and considering the complex host-parasite immune response with temporal variation of pathological changes which may occur in the same brain if the larval parasite enters it in batches does not rule out the possibility of

neurocysticercosis as the cause for both the symptoms in this patient.

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