# SOLITARY CYSTICERCAL GRANULOMA COEXISTING WITH CYSTICERCAL MENINGITIS

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#### ABSTRACT

Neurocysticercosis is commonest parasitic infection of brain but cysticercal meningitis is uncommon. Multiple cysticercal granulomas occurring with eosinophilic meningitis are known but coexistence of solitary granuloma with meningitis is rare. We report a 14 years boy with signs and symptoms of chronic meningitis with bilateral papilloedema whose CSF showed eosinophilic meningitis with positive ELISA for cysticercosis. His MRI brain showed small cystic lesion with eccentric mural nodule in right thalamic region with mild perilesional edema. CSF eosinophilia was confirmed by Wright Geimsa stain. The importance of having high degree of suspicion is highlighted along with need of examining CSF with Wright Geimsa stain so as not to miss CSF eosinophilia.

Key Words: Neurocysticercosis, CSF Eosinophils, Eosinophilic Meningitis, Cycticercal Meningitis, Chronic Meningitis, SSECTL (Single Small Enhancing CT Lesion)

#### CASES

A 14-year-old, right-handed boy was admitted to Neurology ward with complaints of headache and recurrent projectile vomiting of 3 months duration. He complained of decreased and double vision on looking distant objects for 2 weeks prior to presentation. The headache was non-throbbing starting from frontal area and spreading all over the head, was present throughout the day. There were no flashes of light, floaters, lacrimation or any weakness of limbs or paraesthesias. There was no history of alteration in sensorium or convulsions. There was no history of fever, trauma, rash and urinary or bowel complaints. He was developmentally normal and had sibling with epilepsy. The family had a non vegetarian diet pattern but there was no history of eating pork. There was no history of contact with tubercular patient and no history of foreign travel was noted.

On physical examination, the patient was afebrile with stable vital signs. His mental status, motor and sensory examination including deep tendon reflexes and plantar response were normal. He had bilateral papilloedema on fundus examination with normal extra ocular movements. There was mild neck stiffness and a positive Kernig's sign.

The child was admitted with a provisional diagnosis of chronic meningitis. Investigations revealed Hemoglobin 12.0 g/dl, total leukocyte count of 10,360/cumm with 42% polymorphonuclear cells; 35.3% lymphocytes; 9% monocyte; and 13.4% eosinophils with normal platelet counts (179,000 per cumm). His Erythrocyte sedimentation rate (ESR) was 26 mm in 1<sup>st</sup> hour. His routine biochemistry, Xray chest, blood & urine culture were normal. Lumbar puncture revealed clear cerebrospinal fluid (CSF) with 210 cells (33% polymorphonuclear cells, 55% lymphocytes, and12% eosinophils,), protein-55 mg/dL, sugar 28.8mg/dL (concurrent blood sugar, 104 mg/dL) and Adenosine deaminase activity (ADA) levels 2U/L. CSF gram stain and culture revealed no bacterial presence or growth. India ink preparation for cysticercosis was negative. Wright Geimsa stain was done which confirmed raised eosinophilic counts in CSF. Mantoux test was negative. CSF ELISA for cysticercal antigen was negative. Electro immuno transfer blot (EITB) could not be done in our set up. A magnetic resonance imaging (MRI) of the brain

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revealed a small cystic lesion with eccentric mural nodule in right thalamic region with mild perilesional edema (Figure 1, 2). On the basis of the radiological picture the lesion was considered to be neurocysticercosis.

On the basis of compatible clinical and CSF profile, a diagnosis of chronic eosinophilic meningitis was made. Patient was treated with steroids and glycerol. Cysticidal therapy with Albendazole was started (15mg/kg in two divided doses) and patient was discharged after 4 days. His hospital course was uneventful. His headache and vomiting subsided within 48 hours. During 3 weeks of follow up, patient remained asymptomatic with occasional insignificant headache. Albendazole was stopped after 3 weeks.

## DISCUSSION

The initial clinical presentation was consistent with the diagnosis of chronic meningitis. The presence of CSF eosinophilia and the coexistent inflammatory granuloma suggested the possibility of cysticercal meningitis. The cyst could have been coincidental, as viable cysts are known to remain asymptomatic for long periods and reported to be present in up to 1.3% of normal persons (autopsy study) (Mahajan, 1982). Although described in 42-48% of patients in Latin American series, cysticercal meningoencephalitis has been reported in less than 10% adult patients with NC in India (Singh, 1997; Venkataraman *et al.*, 1990). Of the two large series of Indian children with NC, only one reported meningoencephalitis in 0.3% of the subjects (Singhi *et al.*, 2000; Kalra *et al.*, 1998). Slightly higher proportions have been reported in a small case-series (Puri *et al.*, 1991). Around 60% cysticercal meningoencephalitis cases have been reported to have associated parenchymal lesions (Kalra *et al.*, 1998).

Cysticercal meningitis may present with increased intracranial pressure, cerebellar ataxia, dementia, and internuclear ophthalmoplegia (Joubert, 1990; Venkataraman *et al*, 1990). Our patient had increased intracanial tension without seizures.



Figure 1: T2 weighted MRI brain showing a small cystic lesion with eccentric mural nodule in right thalamic region with mild perilesional edema

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Figure 2: T1 weighted MRI showing small cystic lesion with eccentric mural nodule

Most cases of meningitis described are chronic in evolution and isolated acute meningitis has rarely been described. A case report from Thailand describes two children presenting as pyogenic meningitis, with normal cranial CT but not responding to antibiotic therapy (Visudhipan *et al.*, 1997). A South African study showed that all diagnosed cases of chronic cysticercal meningitis had hydrocephalus, either obstructive or communicating and severe clinical sequelae, such as dementia, blindness and gait ataxia, were common despite protracted medical treatment and ventriculoperitoneal shunting (Joubert, 1990). Though co-occurrence of multiple granuloma with cysticercal meningitis is known (Bonametti *et al.*, 1994; Monteiro *et al.*, 1992), to the best of our knowledge there is no report of chronic cysticercal meningitis coexisting with solitary cysticercal granuloma.

Cysticercosis is by far the commonest cause of CSF eosinophilia in endemic areas (Joubert, 1990) but cysticercal meningitis does not have well-defined diagnostic criteria. The other CSF findings in cysticercal meningitis consist of pleocytosis (usually lymphocytic but frequently polymorphonuclear), and may show an elevated protein level, low glucose level, with up to 70% patients with cysticercal meningitis having CSF eosinophilia between 2-40% (Wang *et al.*, 1993; Wilber *et al.*, 1980; Visudhipan *et al.*, 1997). The presence of eosinophils more than 4% of leukocytes in the CSF is of diagnostic significance and is seen in the initial phases of the illness only (Wang *et al.*, 1993). For visualizing eosinophils in the CSF, Wright-Giemsa staining of the CSF is required as in the absence of this staining eosinophils can be mistaken for polymorphonuclear cells (Singh, 1997). The most common misdiagnosis is tuberculous meningitis, followed by malignancy.

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The current serological assay of choice for the diagnosis of neurocysticercosis is EITB; this assay has a specificity approaching 100 % and a sensitivity of 94 to 98 % for patients with two or more cystic or enhancing lesions (Tsang *et al.*, 1989; Garg, 2004). It has been found that the EITB assay is more sensitive than the ELISA, especially when serum is being tested. Both techniques are more sensitive in cases with multiple living cysts than in cases with single cysts or calcified lesions. In the patients with cysts within the parenchyma, the sensitivity of the EITB assay was higher with serum than with cerebrospinal fluid (Katti, 2002; Katti, 2001).

The optimal treatment for cysticercal meningitis is unknown and there are no treatment guidelines.

For medical therapy, albendazole combined with a corticosteroid is the treatment of choice. Dexamethasone increases albendazole blood levels and may attenuate treatment-associated inflammatory reactions, which can be severe (Jung *et al.*, 1990). Praziquantel may not be as effective in intraventricular and meningeal forms of NCC (Joubert, 1990; Monterio *et al.*, 1992).

#### Conclusion

Cysticercal meningitis is probably underdiagnosed and underreported; coexistence of solitary cysticercal granuloma with eosinophilic meningitis is even rarer. It needs to be considered in differential diagnosis of all patients of infectious chronic meningitis living in endemic areas. The compatible clinical data, imaging, CSF inflammatory profile including eosinophilia and negative bacterial and fungal cultures helps in arriving in diagnosis. CSF should be examined by Wright Geimsa stain to confirm eosinophilia whenever indicated.

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