



Case Report:

Racemose Neurocysticercosis: A Rare Cause of Chronic Meningitis

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Abstract: Neurocysticercosis (NCC) is the most common parasitic disease of the nervous system and is the main cause of acquired epilepsy in developing countries. Clinical manifestations result from inflammatory response to cyst degeneration, mass effect, obstruction of CSF pathway or residual scarring but are related to the numbers, size and location of lesions and the severity of host's immune response. The subarachnoid/cisternal form of NCC and majority of the intraventricular NCC are of racemose type, and differ from more common cysticercus cellulose in that they are larger, appear as multiloculated cysts and lack scolex. Racemose NCC is uncommon in India. We are reporting a patient presenting as chronic meningitis due to racemose NCC.

Key Words: Neurocysticercosis; Racemose; Chronic meningitis; Cysticerci

Introduction:

Neurocysticercosis (NCC) is recognized as a common cause of neurologic disease in developing countries, and also seen in developed countries, including the United States. NCC is a chronic disease associated with substantial morbidity and high social and economic costs. The pathogenesis and clinical presentation vary with the site of infection and the host immune response.¹ The cerebral parenchyma is the most frequently affected central nervous system site. However, NCC can also occur in other extraparenchymal locations like intraventricular, subarachnoid/cisternal or mixed.² The subarachnoid/cisternal form of NCC and majority of the intraventricular NCC are of racemose type, and differ from more common cysticercus cellulose in that they are larger, appear as multiloculated cysts and lack scolex.³ Racemose NCC is uncommon in India. We are reporting a patient presenting as chronic meningitis due to racemose NCC.

Case Report

A 23 years old male patient was admitted in the medicine ward in April 2013 with 20 days history of headache which

was diffuse and associated with vomiting. There was no fever, altered consciousness, visual complaints or seizures. He had a past history of doubtful seizure 5 years back. MRI brain at that time was normal. Clinical examination was unremarkable. Fundus revealed bilateral papilloedema. Investigations including blood sugar, renal and liver function tests and hemogram were normal. On MRI brain there was prominent ventricular system lateral and 3rd ventricles and meningeal enhancement along basal cisterns, bilateral sylvian fissure along medial part temporal lobe. On MR venogram, right transverse and sigmoid sinus showed irregular flow and multiple collaterals, suggestive of chronic cortical vein thrombosis. CSF analysis revealed protein 400mg%, glucose 8mg%(concomitant RBS 80 mg%), cytology showed 200 cells, 60% polymorphs, 40% lymphocytes and ADA was 3.2 u/l. In view of basal meningeal enhancement and CSF picture of meningitis, the patient was started on anti tubercular therapy (ATT) with steroids along with anticoagulant (warfarin) for cortical vein thrombosis.

The patient was re-admitted after 2 months with headache and got himself referred to a higher centre the next day, where the intensive phase of ATT was extended for 2 more months and steroids were re-introduced. The patient was admitted to our hospital again in 2nd week of August with severe headache. The NCCT done in emergency revealed hydrocephalus (more than in previous imaging). The patient was started on injection dexamethasone and the ATT was continued. MRI brain was done the next day which showed multiple CSF loculations along basal cisterns and bilateral sylvian fissures with enhancement of basal meninges (Images 1 and 2). CSF analysis revealed proteins 278mg%, glucose 44mg % (concomitant RBS 118mg %), 110 WBC's mostly lymphocytes. CSF ADA was 2.7U/l CSF culture sterile. CSF PCR was negative for mycobacteria. In view of MRI picture suggestive of racemose NCC, CSF and serum serology for cysticerci was done. CSF serology for cysticerci was positive

with titres of 1:160 and serum serology for cysticerci was also positive with titres of 1:1600. Final diagnosis of racemose NCC with chronic meningitis was kept and the patient was started on tab albendazole 400 mg BD along with tab dexamethasone as the patient was not willing for any surgical intervention. ATT was stopped. The patient was observed for two weeks in the hospital. No episode of headache occurred during hospital stay. The patient was followed up in OPD and was asymptomatic one month after discharge.



Image 1: T2 weighted image of MRI brain showing racemose neurocysticerci in perimesencephalic cistern and bilateral sylvian fissures.

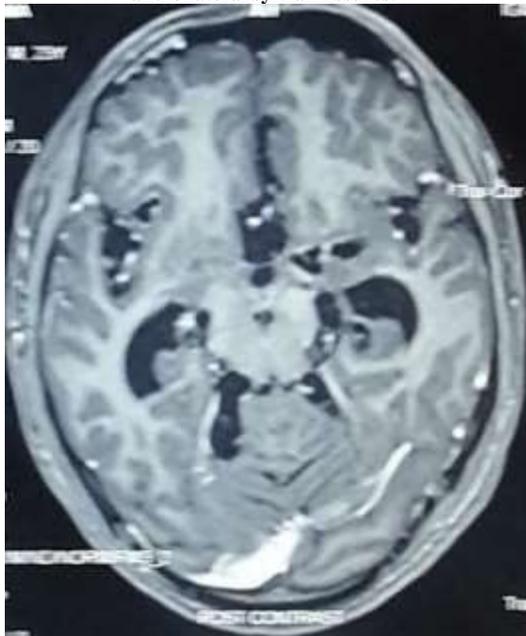


Image 2: Flair image of MRI brain showing racemose neurocysticerci in mesencephalic cistern and bilateral sylvian fissures.

Discussion:

Neurocysticercosis (NCC) is the most common parasitic disease of the nervous system and is the main cause of acquired epilepsy in developing countries. Clinical

manifestations result from inflammatory response to cyst degeneration, mass effect, obstruction of CSF pathway or residual scarring^{4,5}, but are related to the numbers, size and location of lesions and the severity of host's immune response. Many patients are asymptomatic. Possible symptomatic presentations include epilepsy as most common presentation (70%) followed by headache, dizziness, stroke and neuropsychiatric dysfunction. Abnormal physical findings, which occur in 20% or less of patients with neurocysticercosis, depend on where the cyst is located in the nervous system and include cognitive decline, dysarthria, extraocular movement palsy or paresis hemiparesis or hemiplegia, which may be related to stroke, or Todd paralysis hemisensory loss, movement disorders, gait disturbances and meningeal signs.⁶

The multiple cysts of the racemose type occur in nonconfining areas in and around the brain such as the suprasellar, sylvian and quadrigeminal cisterns. These cysts are nonviable, degenerated interconnected bladders of different sizes that often lack scolices, and can reach large sizes producing local mass effect. Arachnoiditis can occur with resulting communicating hydrocephalus secondary to either chronic inflammation or fibrosis of the arachnoid villi causing obstruction to the reabsorption of CSF or extension of the subarachnoid inflammatory reaction to the meninges at the base of the brain occluding the foramina of Luschka and Magendie.⁷ Cysticercotic arachnoiditis can lead to entrapment of cranial nerves in the inflammatory exudates that occur on the ventral aspect of the brain. Racemose neurocysticercosis is characterized by abnormal, large growth of cystic membrane without scolex.^{3,6} Racemose form usually occurs in the ventricles or basal cisterns^{2,6}, and is rare in the brain parenchyma because there is not enough room in the brain parenchyma for growing of large cysts.⁴ This variety differs from the parenchymal lesions by the absence of usual temporal development of degenerative stages and nonvisualization of scolex in majority of the cases.⁹

The specific clinical diagnosis of chronic meningitis caused by cysticercosis is difficult. It is observed that cysticercal meningitis often mimics other forms of chronic meningitis¹⁰ where serial CSF examination shows consistently raised protein, decreased glucose level and pleocytosis. In addition to signs of meningeal irritation, increased intracranial pressure due to inflammation, oedema, or an obstructing cyst may be present.¹¹ Many reports of cysticercal aetiology of chronic meningitis have been documented from various parts of the World. In a recent study, meningitis was found to be a presenting symptom in 4.1% of NCC patients examined in Dehra Dun, India.¹² For any case of chronic meningitis of unknown origin, it would be useful to consider the possibility of cysticercal meningitis. This is important particularly in the area where both tuberculosis and NCC are endemic and make the clinical diagnosis of chronic meningitis most confusing. Cysticercosis may be the underlying aetiology associated with chronic meningitis cases especially when other infectious aetiology is unknown.

References

1. Del Brutto OH. Neurocysticercosis. *Curr Opin Neurol*. Jun 1997;10(3):268-272.
2. Martinez HR, Rangel-Guerra R, Elizondo G, Gonzalez J, Todd LE, Ancer J, et al. MR imaging in neurocysticercosis: A study of 56 cases. *AJNR Am J Neuroradiol*. 1989;10:1011-1019.
3. Bickerstaff ER, Cloake PC, Hughes B, Smith WT. The racemose form of cerebral cysticercosis. *Brain*. 1952;75:1-18.
4. Del Brutto OH, Sotelo J. Neurocysticercosis: an update. *Rev Infect Dis*. 1988;10:1075-1087.

5. Garcia HH, Gonzalez AE, Evans CA, Gilman RH. Cysticercosis Working Group in Peru. *Taenia solium* cysticercosis. *Lancet*. 2003;362:547-556.
6. Chaoshuang L, Zhixin Z, Xiaohong W, Zhanlian H, Zhiliang G. Clinical analysis of 52 cases of neurocysticercosis. *Trop Doct*. Jul 2008;38(3):192-194.
7. Lobato RD, Lamas E, Portillo JM et al. Hydrocephalus in cerebral cysticercosis. Pathogenic and therapeutic considerations. *Journal of Neurosurgery*. 1981;55(5):786-793.
8. Rabiela MT, Rivas A, Flisser A. Morphological types of *Taenia solium* cysticerci. *Parasitol Today*. 1989;5:357-359.
9. Hauptman JS, Hinrichs C, Mele C, Lee HJ. Radiologic manifestations of intraventricular and subarachnoid racemose neurocysticercosis. *Emerg Radiol*. 2005;11:153-157.
10. Torrealba G, Del Villar S, Tagle P, Arriagada P, Kase CS. Cysticercosis of the central nervous system: clinical and therapeutic considerations. *J. Neurol. Neurosurg. Psychiat*. 1984;47:784-790.
11. Shanley JD, Jordan MC. Clinical aspects of CNS cysticercosis. *Arch Intern Med*. 1980;140:1309-1313.
12. Varma A, Gaur KJ. The clinical spectrum of neurocysticercosis in the Uttaranchal region. *J Assoc Phys India*. 2002;50:1398-1400.