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# DIAGNOSTIC DILEMMA IN RING ENHANCING LESION- A CASE REPORT

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## ABSTRACT

Here we report a 22 years old boy with fever, headache and signs of meningitis with CT scan of brain showing multiple small enhancing lesion with minimal perilesional edema seen on both sides of cerebral cortex which was initially thought to be neurocysticercosis (NCC), but diagnosed subsequently to be tuberculomas by Magnetic Resonance (MR) spectroscopy and was subsequently treated with antitubercular drugs. This case report highlights the role of strong clinical suspicion and MR spectroscopy in the conformation of diagnosis of ring enhanced lesions.

**Key Words:** Neurocysticercosis, Tuberculoma, Ring enhancing lesion, MR spectroscopy.

## INTRODUCTION

Neurocysticercosis (NCC) is the most frequent parasitic infestation of CNS in the world. Also cysticercal meningitis is very difficult to distinguish from tuberculomas associated with meningitis, the two most common causes of inflammatory granulomas encountered in clinical practice and differentiation between the two is very difficult and defied conventional investigations<sup>1,2</sup>. We highlight the importance of strong clinical suspicion and MR spectroscopy in differentiating the lesions. This distinction is important because parenchymal cysticercosis is benign and self limiting condition, but tuberculomas are active infection requiring prolonged therapy that involves potentially toxic drugs.

## CASE REPORT

A 21 years old boy presented with 1 month history of low grade continuous fever with frontal headache and vomiting. His symptoms increased over time and gradually became irritable with subsequent drowsiness 4 days prior to hospitalization. He did not have any episode of

convulsion during this 1 month period of illness. History of contact with tuberculosis was absent.

On examination he was drowsy, febrile, apart from tachycardia vital was stable. Neurological examination was positive for meningeal signs without focal neurological deficits. Other systemic examinations were normal.

Chest X-Ray was normal, mantoux test was negative, serology for cysticercal antigen was negative. CT scan revealed presence of multiple small enhancing nodular lesions with minimal perilesional edema seen in both the sides of cerebral cortex, suggestive of neurocysticercosis, there was no evidence of midline shift. On ophthalmological examination there was no papillaedema, or intraocular cysticercal parasite. MRI with contrast was done which showed extensive tiny ring and nodular enhancing focal lesions with mild to moderate perilesional odema scattered in cerebral hemispheres, cerebellum and right thalamic region and mild patchy irregular enhancement in basal cisterns and few cortical sulci of both cerebral hemispheres. Till now diagnostic dilemma persisted between NCC and tuberculoma so CSF was done which showed wbc-120/mm<sup>3</sup> with 60% neutrophil and 40% lymphocyte, protein-151mg/dl, glucose-11mg/dl, cl-

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94m/dl ADA was normal. TBPCR, gram stain, AFB and Indian ink stain was negative. Patient clinical condition deteriorated and he became comatose. ATD (HRZE) with steroid was empirically started and MRI spectroscopy was planned, which subsequently identified lipid lactate peaks within the lesions and diagnosis of tuberculoma was considered. In the meanwhile patient responded to treatment, his conscious level improved and was ultimately discharged with ATD and steroid, and advised to follow-up in OPD. After 6 weeks CT scan was done which showed disappearance of many lesions.

## DISCUSSION

The diagnostic dilemma of inflammatory granuloma and the use of MR spectroscopy to diagnose tuberculoma and importance of starting ATD early on strong clinical suspicion are highlighted from the above case. Common causes of inflammatory granuloma includes neurocysticercosis, followed by tuberculosis, toxoplasmosis, cerebral abscess, fungal lesion.<sup>3</sup> This boy presented with fever, headache, along with meningeal signs with neuroimaging suggestive of neurocysticercosis, all lesions  $\leq 20$  mm in size with regular outline and no midline shift. Yet the patient was actually having tubercular granulomatous lesion where we expect  $\geq 20$ mm lesions with irregular outline and midline shift<sup>4</sup>. The deteriorating clinical condition of our patient prompted us to review our diagnosis on following facts. NCC commonly present with seizures<sup>5, 6</sup>, but there was no seizures in our patient. Fever is also present rarely in NCC. Also in several studies it was shown that cysticercal meningitis lacks nuchal rigidity<sup>5</sup>. Several diagnostic criteria for NCC has been proposed but none of the criteria has addressed the differentiation between cysticercal granuloma and tuberculoma and this is the most difficult clinical situation a clinician face in developing country like India. Also positive serological test may help to confirm the diagnosis of cysticercosis but negative result does not exclude it<sup>7</sup>. Here MR spectroscopy may be helpful to differentiate tuberculoma from NCC. In a study by Pretell et al<sup>8</sup> MR spectroscopy was used to differentiate single enhancing brain lesions as due to tuberculomas or NCC, tuberculomas has  $\uparrow$  peak of lipids, more choline and less NAA.

## CONCLUSION

CNS tuberculomas with tubercular meningitis has a very similar presentation like cysticercal meningitis and differ-

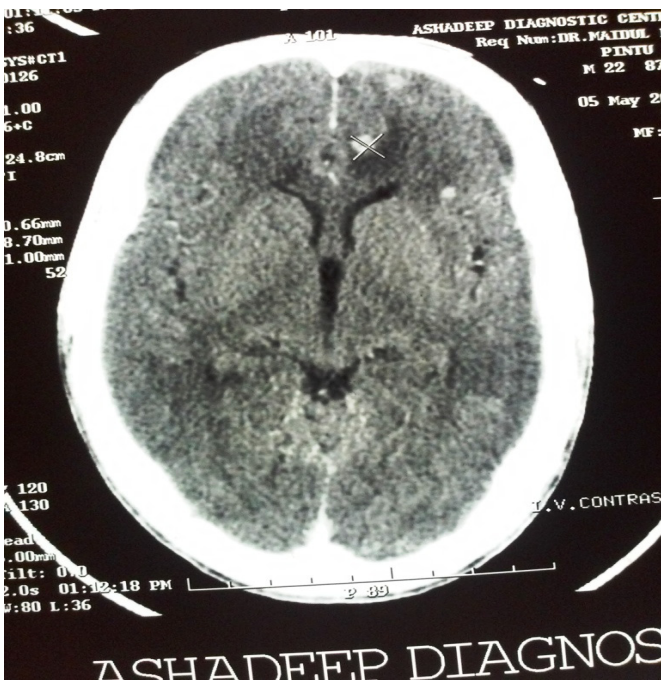
entiating between two is difficult in our country, where both are endemic. With increase in incidence of HIV, co-infection rates have also increased. Clinical examination directed to find evidence of tuberculosis and cysticercosis elsewhere is invaluable, but when absent creates a great diagnostic dilemma. CT scan and MRI can differentiate but again are not confirmatory as in our case. So to conclude, in such case where there is a high index of suspicion, MR spectroscopy may be a non-invasive alternative to determine their etiology.

## ACKNOWLEDGEMENTS

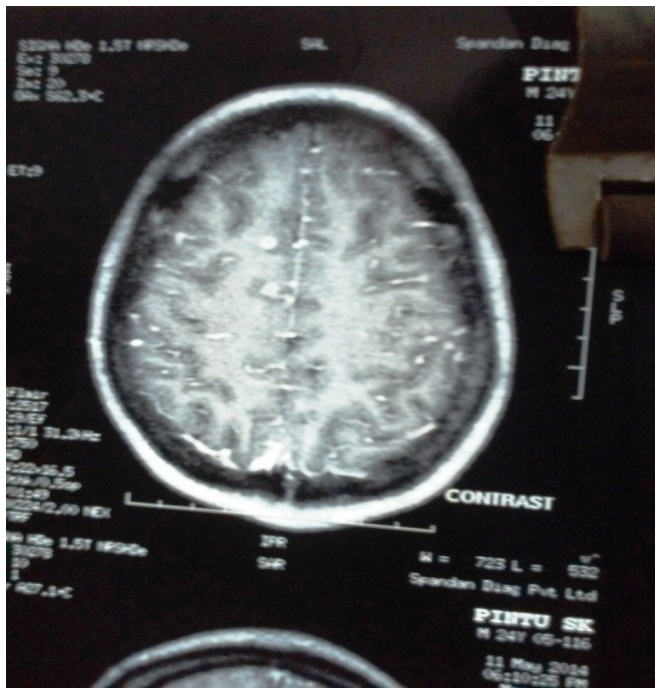
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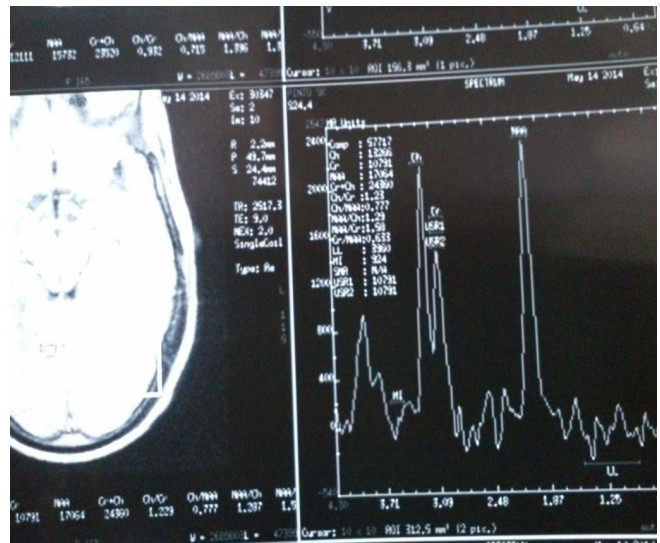
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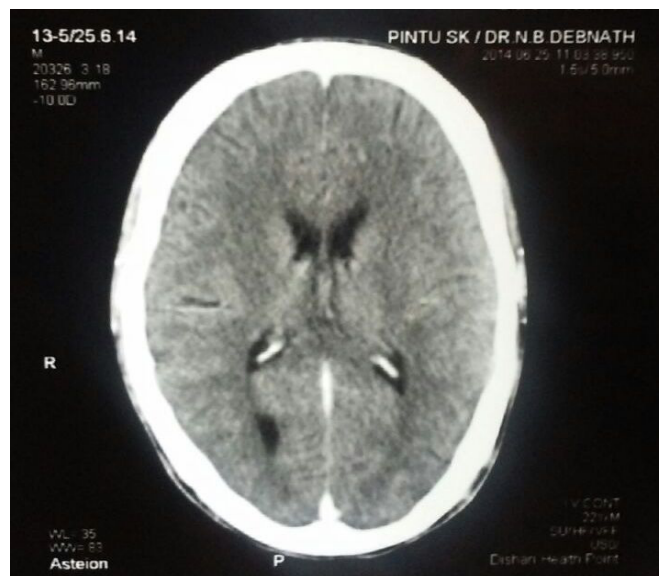
**Figure 1:** CT scan of brain on the day of admission showing ring enhancing lesions



**Figure 2:** MRI brain with contrast showing ring enhancing lesions



**Figure 3:** MR Spectroscopy showing increased peak of lipid and choline



**Figure 4:** Repeat CT scan showing disappearance of ring enhancing lesion after one and half months course of Anti tubercular Drugs