Tubercular Abscess: Rare Variant of CNS Tuberculosis.

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ABSTRACT

Tuberculosis is a great burden on global health specially in developing country such as India.This disease has a wide spectrum of presentation most commonly pulmonary. Tuberculosis can involve almost every part of the body but the disease manifestation mainly depends upon the patient health status, bacterial virulence and lastly the environment of the patient. Along with involvement of lungs it can also involve pleura, lymph nodes, skin, uterus, follapian tubes, ovary, meninges, spine, bones, and rarely it can also involve the brain parenchyma leading to brain abscess formation. It's a rare clinical entity only diagnosed on imaging and when diagnosed should be treated accordingly.

Keywords: Abscess, ADA, Seizures, Cystic tuberculoma.

INTRODUCTION

India is a country that shares a greatest disease burden for TB worldwide approximately 40% of Indian population got an infection with tubercle bacilli most of them never develop any active disease. [1]

Tuberculosis when present as active disease can involve any part of the body most commonly pulmonary followed by other extrapulmonary sites (eg pleura, spine meninges, brain, uterus, peritoneum & very rarely it involves the CNS but not meninges but the brain parenchyma leading to formation of tubercular brain abscess .[2]

TBA is a focal collection of pus containing abundant acid fast bacilli (AFB) surrounded by a dense capsule consisting of vascular granulation tissue.[2] TBA always poses a diagnostic dilemma as they are difficult to differentiate from pyogenic brain abscesses, tuberculous meningitis, and tuberculoma on the basis of clinical, laboratory, and roentgenographic information.

It resembles a pyogenic brain abscess clinically and radiologically and poses a problem in diagnosis and treatment. A final diagnosis is established by smear or culture demonstration of acid fast bacilli (AFB) within the abscess.[3] The criteria laid down by Whitenerin 1978 : (i) evidence of a true abscess formation within the brain, as confirmed during surgery or autopsy; (ii) histological proof of presence of inflammatory cells in the abscess wall; and (iii) demonstration of AFB in the pus or abscess wall.[2]

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CASE REPORT

Patient is a young aged male of lower socioeconomic strata also a known case of pulmonary Koch's on irregular ATT [CAT 1] since last 4 months presented to the emergency with the complaints of high grade fever since last one month associated with severe headache and vomiting not preceded by nausea since last 21 days. There is also associated history of progressive deterioration of mental status and alertness along with multiple episodes of GTCS.

On examination his vitals revealed an elevated BP (144/88 mm of Hg) and a temperature of 104^0F along with irregular respiration with a rate of 22/min. There was an associated pallor and a single enlarged post auricular lymph node.

On systemic examination, his respiratory system revealed the deviation of trachea towards the right side along with absent breath sound in right upper lobe (correlating with the post TB collapse of right upper lobe). A normal vesicular sound was present in all other lung fields. CVS & Abdomen were normal. On CNS evaluation there was marked neck rigidity in the patient with positive kernig's and brudzinski's sign. There wasn't any focal deficit. Cranial nerves were normal. Superficial and deep reflexes were intact.But there was a positive babinski sign.

In the presence of evidence of TB in patient,in adherent treatment taken,and involvement of CNS a provisional diagnosis ofTBM was made and patient was kept of revised regimen of ATT along with steroids , osmotic diuretics & anti epileptics .

Investigations revealed the presence of normocytic normochromic anaemia along with depressed reti count of 0.8% with normal KFT & LFT. HIV status was non reactive

CSF report revealed a nearly normal CSF clear fluid with protein 60 mg/dl, sugar 77mg/dl, chloride 710, TLC of 02 cells and a negative ADA value of 6.4 IU/ml.But patient was kept on ATT and imaging (MRI Brain) was done.

Imaging revealed a hypodense lesion in temporal lobe rt sided on T2 imaging [Figure 1] suggestive of shows a well-defined hyperintense lesion in the right temporal lobe with peripheral hypointense rim, perifocal edema, and mass effect on the ventricular system.

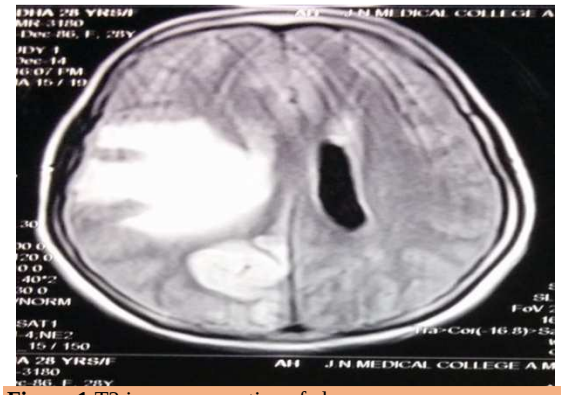

Figure 1:T2 image suggestive of abscess

On T1 [Figure2] imaging there was a hypodense lesion present on temporal lobe along with a definite capsule formation.[8]

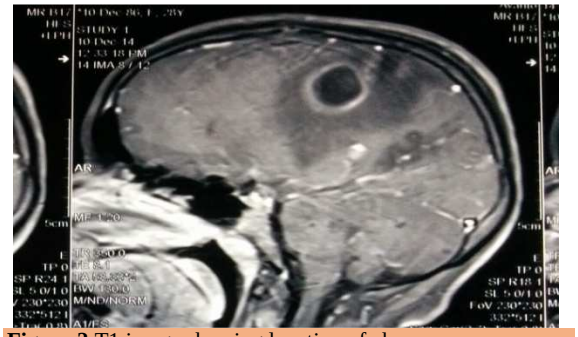

Figure 2:T1 image showing location of abscess

Therefore, the diagnosis of old pulmonary kochs with tubercular brain abscess was made.

DISCUSSION

As the patient was diagnosed as a case of tubercular brain abscess wit secondary seizures. It is one of the most serious complication of tuberculosis involving CNS.[4] The patient was to be kept on ATT for longer duration, but as the patient is immunocompetent the prognosis is better than that of immunocompromised.[5] Their presentation is acute; often in a 3rd or 4th decade.

These are mainly supratentorial and rarely in the cerebellum. These present with focal neurological signs and are associated with histoligical and Laboratory evidence of tuberculosis. Laboratory data shows, high ESR; CSF has pleocytois with increased protein; PCR +ve in a good number of cases. Tubercular abscess should be differentiated from Cystic tuberculoma. In this unlike pus cyst contains yellowish fluid and cyst wall has typical tuberculous pathology.

CT shows hypodense lesion surrounded by enhancing ring. There may be associated surrounding oedema. At times, it becomes difficult to differentiate tubercular abscessfrom pyogenic abscess on the basis of clinical; roentgenologic findings.[6] AFB should be demonstrated on Ziel Nielson stain. Appropriate treatment includes antitubercular treatment with surgical excision / aspiration.[7] At present patient is on ATT & anti epileptics and improving.

CONCLUSION

If the patient of suspected tuberculosis develops GTCS inspite of proper treatment and seizure are refractory in spite of all anti epileptics (metabolic derangements already ruled out) should be screened for involvement of brain parenchyma as a cause of delayed recovery .As involvement of brain parenchyma after involvement of meninges carries a worse prognosis and should be treated immediately.

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