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Case Report

Giant Tuberculoma Masquedering as Brain Tumor

Prasad R, Rai A, Singh A, Mishra OP and Singh UK*

Department of Pediatrics, Institute of Medical Sciences, Banaras Hindu University, India

Abstract

Tuberculosis of Central Nervous System (CNS) has wide range of presentation among which tuberculoma is quite common, but giant tuberculoma mimicking brain tumour is very rare. We are reporting a seven year female, who had presented with altered sensorium and upper motor neuron type of weakness of all four limbs. Neuroimaging revealed intracranial space occupying lesion with mass effect suggestive of tuberculoma by MR spectroscopy. She was treated with four drugs antitubercular drug (2HRZE/10HRE), steroid, 20% mannitol and V-P shunt. Cerebrospinal fluid CBNAAT was positive for Mycobacterium tuberculosis and rifampicin sensitive. Patient improved completely at 12 months. In tuberculosis endemic areas, tuberculoma should be considered in differential diagnosis of CNS space occupying lesion

Keywords: Giant tuberculoma; Brain tumor; MR spectroscopy

Introduction

Tuberculosis is common in South East Asia including India. Tuberculosis of Central Nervous System (CNS) accounts for about 1% of total and 5% to 10% of total extrapulmonary tuberculosis cases [1]. CNS tuberculosis occurs as a result of secondary hematogenous spread from extracranial tuberculous lesion and has wide range of presentations i.e. tubercular meningitis, tuberculoma, tubercular abscess, spinal tuberculosis and tuberculous encephalopathy. Recently, due to high incidence of HIV, there is increase in incidence all over the world. Obstructive hydrocephalus secondary to intraparenchymal cerebral tuberculoma is very rare as observed in our case. Rarely, multiple tuberculoma coalesces together to form giant mass, and producing severe mass effect and focal neurological deficit. Here we are reporting a female child with giant tuberculoma with midline shift and obstructive hydrocephalus masquedering as brain tumor. Only a few case reports have been reported [2-4].

Case Presentation

A seven years female presented in pediatric emergency with complaints of fever for six months and abnormal body movements and headache for eight days. After care of airways, breathing and circulation, Seizure was controlled with intravenous lorazepam (0.1 mg/kg) and phenytoin sodium (loading dose: 15 mg/kg I.V. over 15 minutes followed by 5 mg kg/day b.i.d.). On neurological examination, child had GCS (10/15), decerebrate posturing, areflexia, power grade I/V bilaterally with bilateral extensor plantar reflex. There was no sign of meningeal irritation. Higher function and cranial nerve examinations were normal. Examination of chest, cardiovascular and abdomen were normal.

In laboratory investigations, blood counts showed neutrophilic leukocytosis (TLC-13,800/cubic mm with 95% neutrophils) and CSF analysis showed lymphocytic leukocytosis (Total count: 112 cells/mm^3, lymphocyte: 94%) with raised protein (286 mg/dL) and glucose; 45 mg/dL. Cerebrospinal fluid CBNAAT was positive for Myocobacterium tuberculosis and rifampicin sensitive. Tuberculin test was positive (25 mm) and ELISA for HIV was negative. CECT brain showed space occupying lesion in left basal ganglia and left medial temporal lobe with surrounding edema, dilated lateral ventricles and meningeal enhancement suggestive of probably tuberculoma with obstructive hydrocephalus (Figure 1A). MRI brain with MR Spectroscopy revealed a mass lesion measuring 3 cm × 2.5 cm × 3 cm in left insular cortex with subtle post contrast enhancement and with non-communicating hydrocephalus and surrounding parenchymal edema and an acute infarct in right caudate nucleus (Figure 1B). MR Spectroscopy revealed lipid peak. The overall feature was suggestive of giant tuberculoma (Figure 1C).

Patient was treated with antitubercular drug (2HRZE/10HRE), steroid (Initially dexamethasone followed by prednisolone for 6 weeks), and 20% mannitol in first 48 hours and phenytoin sodium. There was no improvement with decongestive measures; therefore, ventriculo-peritoneal shunt was put on day 3. Patient was discharged on day 14 of admission with GCS of 15/15. In follow-up, patient improved completely at 12 months without any residual neurological deficit. MRI of brain showed complete resolution of lesion.

Discussion

Tuberculosis of the Central Nervous System (CNS) is life threatening in children and fatal without appropriate treatment. Tuberculoma results from aggregation of caseous tubercles that presents as space occupying lesion of brain. It accounts for about 30% of intracranial space occupying lesion. In children they are single, often infratentorial, located at base of brain near cerebellum. Its clinical manifestations depend on their location and include headache, fever, focal neurological deficits, signs of raised intracranial pressure and convulsions. Generally, tuberculomas are small but could be giant causing pressure symptoms, which mimics tumor like lesions [1,2,4].

The basic principles in reducing morbidity and mortality are early diagnosis and correct treatment [5]. Brain MRI and histopathological examination are useful for diagnosis. The gold standard for diagnosis is histopathological examination, which was not done in our patient but CSF CBNAAT was positive for Mycobacterium tuberculosis. The differential diagnosis are sarcoidosis, neurocysticercosis, toxoplasma,

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***Corresponding author:** Rajniti Prasad, Department of Pediatrics, Institute of Medical Sciences, Banaras Hindu University, Varanasi, India, E-mail: rajnitip@gmail.com

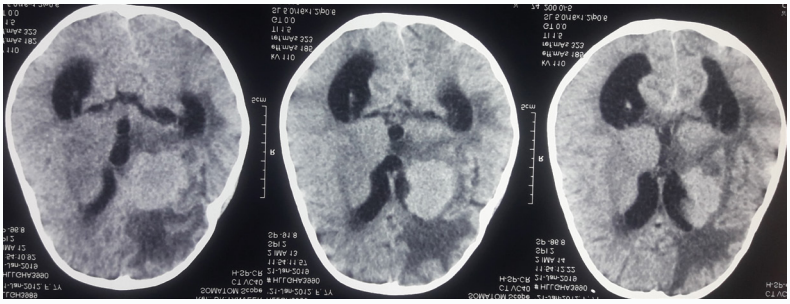

Figure 1A: CT scan showing space occupying lesion in left basal ganglia and left medial temporal lobe with surrounding edema, dilated lateral ventricles and meningeal enhancement

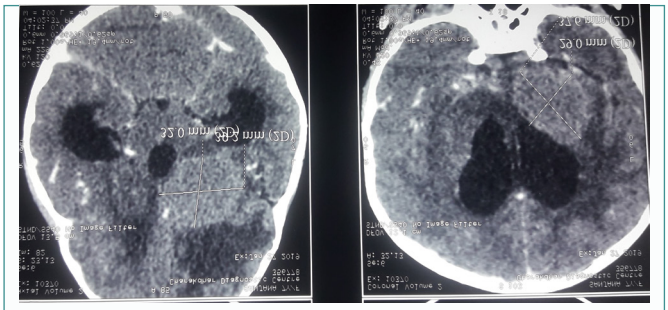

Figure 1B: MRI brain showing mass lesion measuring 3 X 2.5 X 3 cm in left insular cortex with subtle post contrast enhancement and with non-communicating hydrocephalus

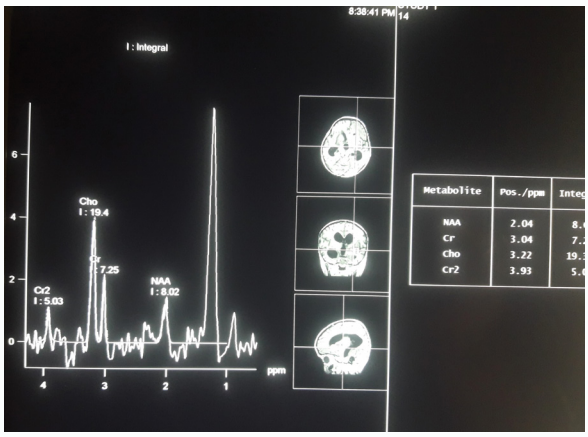

Figure 1C: MR Spectroscopy revealed lipid peak.

fungal infections and neoplasms [6,7]. CT findings are a low or high density rounded or lobulated mass with ring enhancement on contrast as in our case. On MRI, a non caseating granuloma is hypointense on T1, hyper intense on T2 weighted image and homogeneously enhancing on contrast administration whereas, and caseating granuloma is hypointense or isointense on both T1 and T2 weighted images with ring enhancement on contrast administration. Although MRS is useful for diagnosing tuberculoma, histological examination is gold standard [2,6].

Treatment is primarily medical as surgical removal is not needed because most tuberculomas resolve with medical management, but giant tuberculoma with mass effect usually needs surgical intervention as in our case. Surgical excision is also necessary to distinguish tuberculoma from other causes of brain tumor. Antituberculous treatment (regimen: 2HRZE/10HRE) with steroid (4 weeks followed by tapering over next 2 weeks) are given along with phenytoin sodium [8]. Prognosis depends on site, association with tubercular meningitis, response to therapy.

Conclusion

In tuberculosis endemic South East Asia including India, tuberculoma should be considered in differential diagnosis of CNS space occupying lesion.

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