Case Report

Atypical Presentation of CNS Tuberculosis

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Abstract : Tuberculosis is an infectious disease which is caused by the pathogenic microorganism Mycobacterium and a highly prevalent disease in developing countries. Approximately at 5-10% of tuberculosis a case central nervous system (CNS) involvement is due to haematogenous spread is not a rare entity. Tuberculoma is one of the manifestations of Central Nervous System (CNS) tuberculosis (TB). A tuberculoma is a tuberculous focus, which enlarges with in brain tissue, firm, avascular, spherical masses, with size varying between 2 cm to 10 cm in diameter and the compressed surrounding tissue shows edema and gliosis. Lesions are most often singular but may be multiple.Our case was a9 years boyhad occasional episodes of severe head ache for 12 months for which he was treated by local doctor. He was admitted at hospital with fever for 1 day with single episode of convulsion. He had also history of undue tiredness during outdoor games with 3 episodes of transient unconsciousness which lasted for 1 to 2 minute and resolved spontaneously. He had no features of signs of raised intracranial pressure or a hemiplegia or cranial nerve palsy and had no history of cough and cold or contact with TB patient.

Key words : Tuberculoma, Computerized Tomography (CT) scan; MRI.

Introduction

Tuberculosis (TB) continues to be an important cause of morbidity and mortality around the world, particularly in the developing as well as in developed countries. Because of the relative ease of travel from endemic areas, the increasing incidence of infections with the human immunodeficiency virus, and the emergence of multiple drug-resistant Mycobacterium strains of tuberculosis[1]are increasing. About 1% of patients with TB develop intracranial tuberculomas, usually as part of miliary TB that arises from extra-cranial spread[2]. In developing countries, the incidence of tuberculoma varies from 5% to 30.5% of all intracranial space occupying lesions.[3,4] Tuberculomas are usually seen in patients with extracranial signs and symptoms tuberculosis. This case is unusual because the patient was young and presented with only with head ache and single episode of convulsion and fever for 1 day before admission to the hospital rather than pulmonary manifestation. The diagnosis was based on clinical and neuroimaging features and response to anti-tuberculous treatment.

Pathophysiology

Most tuberculous infections of the central nervous system are caused by Mycobacterium tuberculosis,as a result of hematogenous spread from a primary location, either the lung or gastrointestinal tract[5]. Initially, small tuberculous lesions (Rich's foci) develop in the CNS, either during the stage of bacteraemia of the primary tuberculous infection or shortly after wards. These initial tuberculous lesions may be inoculated in the meninges; the subpial and subependymal surface of the brain or the spinal cord, and may remain dormant foryears. Later, rupture or growth of one or more of these small tuberculous lesions produces various types of CNS tuberculosis. The type and extent of lesion depend upon the number and virulence of bacilli and the immune response of the host[6]. Pathologically, a tuberculoma is composed of central core of caseous necrosis surrounded by a capsule of collagenous tissues and an outer layer of mononuclear inflammatory cell (including plasma cells & lymphocytes), epitheloid cells and multinucleated Langerhans' giant cells. A tuberculoma harbors few tubercular bacilli within the necrotic center and the capsule. Outside the capsule, there is parenchymal edema and astrocyte proliferation. Unlike caseous tuberculoma, a tubercular abscess has purulent center rich in tubercular bacilli, and lacks epithelioid giant cell granulomatous reaction in its wall.[7]

Clinical presentation

A 9 years boy of a non-consanguineous parent, hailing from a semi-urban area of Dhaka city of low socioeconomic background. He was delivered at term by normal vaginal delivery at a local clinic. Antenatal and natal history was uneventful and was vaccinated against all EPI vaccine. He was admitted at pediatric unit of Ad-din women's medical college hospital with the history fever for 1 day with single episode of convulsion. He had no past history of irregular fever, no history of weight loss or contact with known tuberculous patient. But he had

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history of severe head ache for 12 months for which he was treated by local doctor and also visited to eye specialist. His refraction error was corrected with spectacles and fundoscopy was done for severe head ache which revealed normal. But his headache persisted. He had also history of undue tiredness during outdoor game with 3 episodes of transient unconsciousness which lasted for 1 to 2 minute. On examination the he was conscious, cooperative, well oriented with the surroundings, and normal vital signs. Skin survey revealed BCG mark. Back & spine examination showed no abnormalities. All cranial nerves were intact. On general examination child was mildly pale, not icteric. Admission weight was 21kg, (3th percentiles),height 120cm (5th percentiles). Neurological and all other system including respiratory system reveled normal.

Laboratory investigations

Hb% was 12.8gm/dl, total count were 12,310/cmm and differential count were lymphocyte 10.2%, polymorphs 88.7%, eosinophil 0.1%, monocyte 0.9%, basophil 0%, platelet count was 1,94,000/ cmm. ESR 24 mm in 1st hour.Random blood sugar was 4.4mmol/L, Serum bilirubin was 0.2mg/dl, serum electrolytes and serum calcium were within normal limit. Peripheral blood film showed mature white cells with above distribution. Blood culture showed no growth of bacteria. But the Montous test (MT) was highly positive, induration more than 20 cm.

(Fig. 1 and 2). X ray chest no pulmonary lesion.

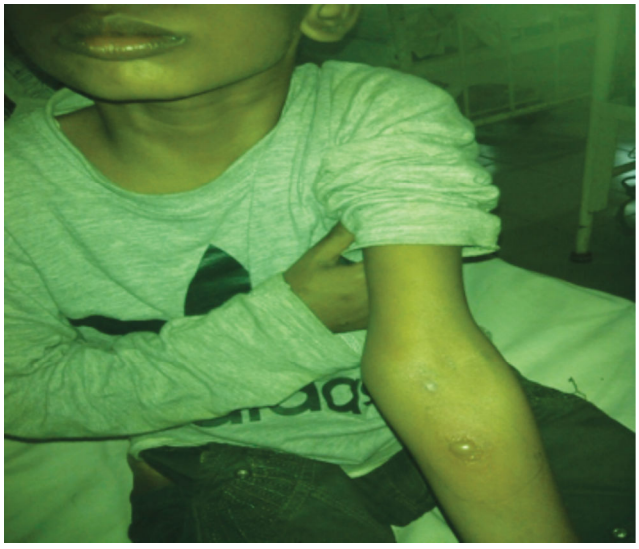

Fig: 1 : Boy with positive MT with blister formation

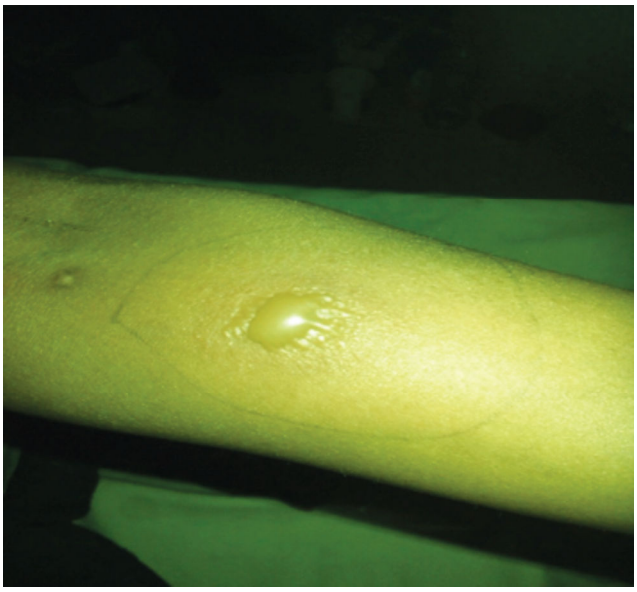

Fig : 2 Large area of indurations with blister formation

CT/MRI scans of the brain

Multiple5 mm axial NECT and CECT scan of the brain was done which shows multiple ring like lesions with rim enhancement, with perilesional hypodense areas, are noted in left frontal, both parieto-occipital region of the brain. Tiny calcification was noted in the hypothalamic area. Frontal horn of the left ventricle was mildly dilated with midline shift to right. Rest of the brain including the scull bone was normal. Report was suggestive of tuberculoma with brain oedema with mild mass effect. MRI shows multi focal T2- FLAIR roundish, conglomerating hypointense mass lesion with huge peri-lesional oedema in left parito-occipital, left frontal and right cerebral hemispheres. After intravenous contrast strong rim as well as nodular enhancement is noted in all the masses. Mass effect is evident by compression on left occipital horn. Evidence of peri-lesional meningeal enhancement is also seen.

Treatment:

On admission child was treated ceftriaxone injection and diazepam for convulsion. Within 48 hours of his admission anti- tubercular drug with RIMCURE 3 FDC (rifampicin 150mg+Isoniazide75 mg+Pyrazinamide 400mg/Tablet) 4 tablets before breakfast, strptomycine with prednisolone injection and pyridoxine. RIMCURE 3 FDC and strptomycine given for 2 months followed by rifampicin and Isoniazide for 10 months. Clinical improvement was observed within48 hours only by remission of headache. Child was vitally stable on regular follow up and there was no complications were observed. After discharge from the hospital progress was assessed regularly in the out-patient clinic and serial CT/MRI examinations were done which showed definite improvement after 12 months treatment and planned to continue for 18 months for complete radiological cure.

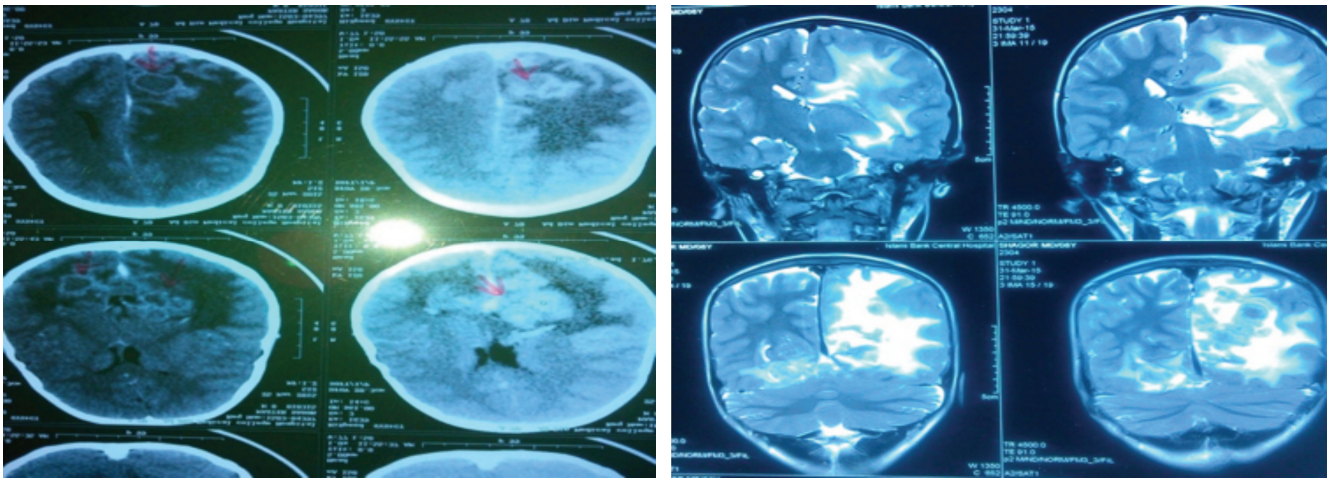

Fig 3 and 4 : CT SCAN Brain -Multiple lesions with peri-lesional hypodense areas with mass effect.

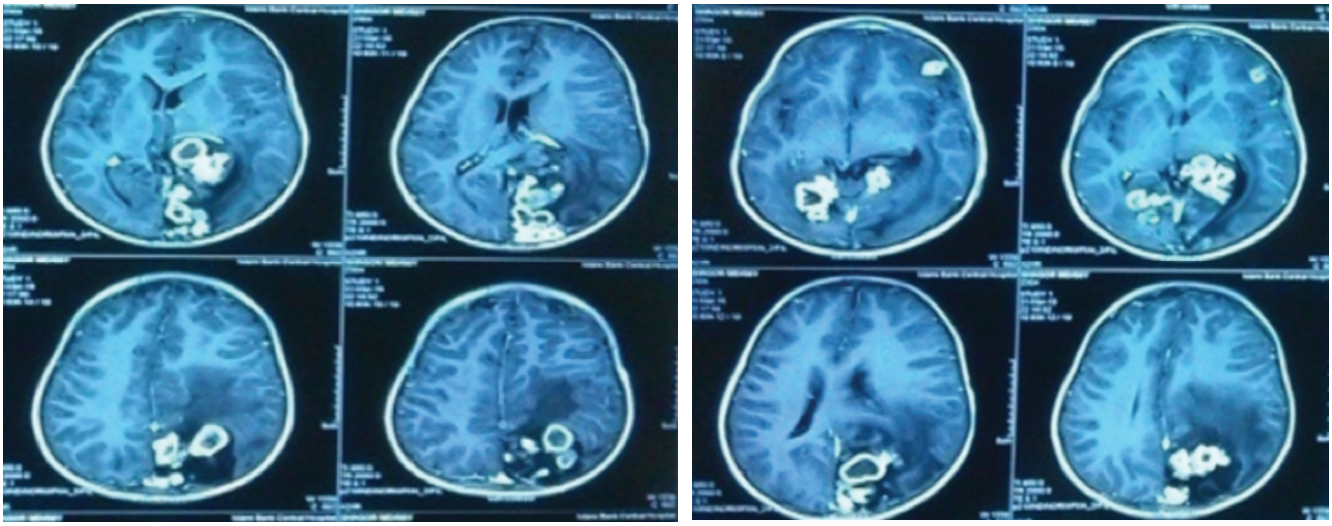

Fig 5: Multiple ring like lesions with rim enhancement after contrast, with perilesional hypodense areas in parieto-occipital region.

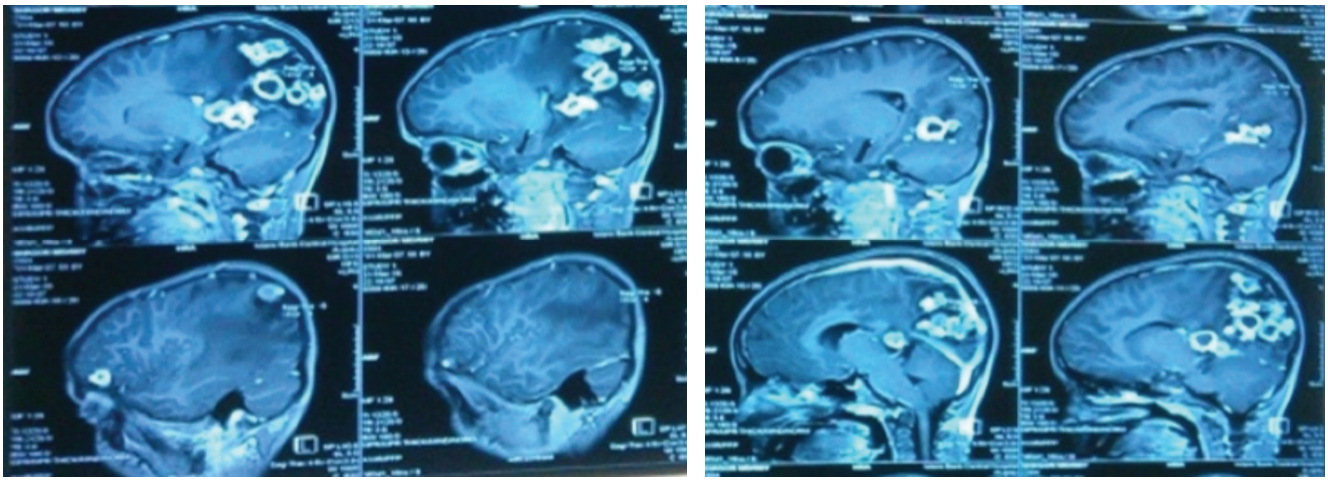

Fig 6: Huge peri-lesional oedema in left parito-occipital, left frontal and right cerebral hemispheres.

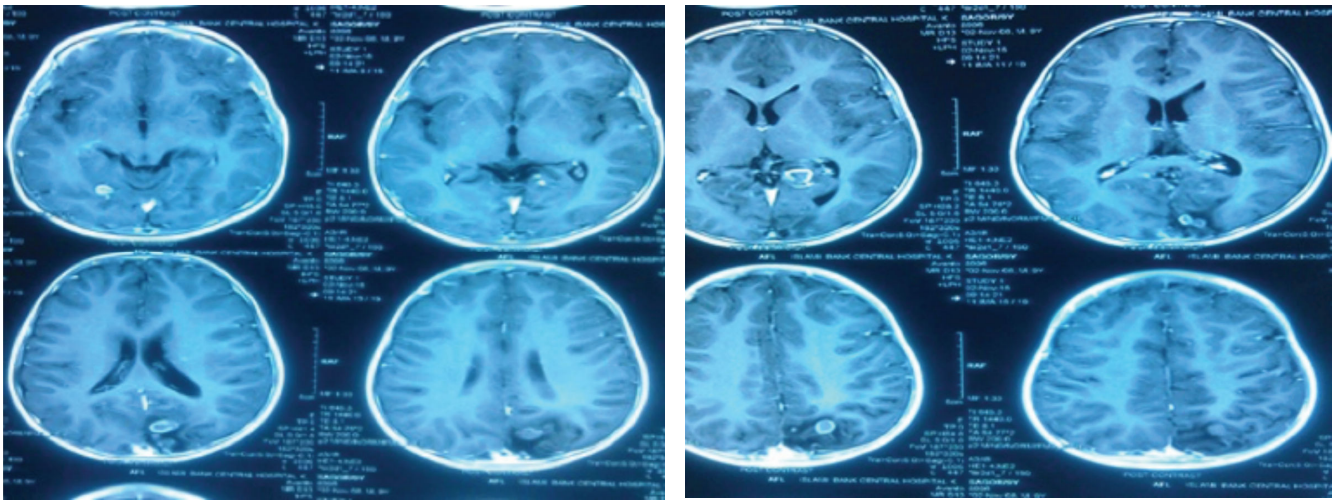

Fig 7: After 9 month treatment with anti TB medications shows improvement on CT SCAN of brain

Discussion

Tuberculomas are firm, avascular, spherical granulomatous masses, measuring about 2–8 cm in diameter. They tend to be located infratentorially in the pediatric population and supra-tentorially in adults. They are well limited from surrounding brain tissuewhich is compressed around the lesion and shows oedema and gliosis. Insideof these masses may contain necrotic areas composed of caseous material, occasionally thick and purulent, in which tubercle bacilli can be demonstrated. Intracranial tuberculomas can occur at any age. In developing countries young adults and children are predominantly affected while in developed countries they are more common in older patients. The symptoms produced by tuberculoma are related to their location. Low-grade fever, headache vomiting, seizures, focal neurological deficit, and papilloedema are characteristic clinical features of supratentorial tuberculomas. Intratentorial tuberculomas are more common in children and may present with brainstem syndromes, cerebellar manifestations and multiple cranial nerve palsies.[8,9,10] Numerous intracranial lesions were found in our patient. However, tuberculomasare usually solitary lesions. Studies

have shown that 15% to 34% of casespresent with multiple lesions.[11] On CT, tuberculomas are characterized as low- or high-density and rounded or lobulated masses and show intense homogenous or ring enhancement aftercontrast administration. They have an irregular wall of varying thickness. Moderateto marked perilesional oedema is frequently present. Tuberculomas may be single or multiple and are more common in frontal and parietal lobes. On CT, the 'target sign', a central calcification or nidus surrounded by a ring that enhances after contrast administration, is considered pathognomonic of tuberculoma.[12] On CT scanning, tuberculoma measure more than 20 mm in diameter, are frequently irregular in outline, and are always associated with marked cerebral oedema with midline shift and progressive focal neurological deficit. The most important factor affecting the prognosis of cerebral TB is early initiation of treatment. In one study 14 patients with tuberculomas of the brain were treated with anti-tuberculous drugs and all of them irrespective of their size, cured by medical treatment.[13] Our case was treated with anti- TB drug only. Surgery is needed with large tuberculoma with space occupy effects even after medical treatment.[14] Another indication of surgery if there is any diagnostic confusion for asrtocytomas. Surgery is also indicated in post tubercular hydrocephalus but surgery can be avoided in 70% cases with conservative management with mannitol, frusemide, acitazolamide and of course dexamethasone.[15]

Conclusion

The diverse clinical manifestations of CNS tuberculosis, a common neurological disorder in developing countries as well as in developed world. Early recognition and timely treatment of CNS tuberculosis is very crucial in order to reduce the mortality and the morbidity of the disease.

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