

# Choroidal Tuberculosis, Multiple Intracranial Tuberculomas and Spinal Cord Tuberculoma in a Patient with Choroidal Coloboma and Corectopia of Left Eye: A Case Report

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## Abstract

Tuberculosis (TB) of the central nervous system (CNS) can present in varied fashions both clinically as well as radiologically with disseminated tuberculomas, and those involving eye is extremely rare. The most common forms of ocular TB include choroidal TB and tubercular uveitis. Early diagnosis and prompt treatment may be sight saving so screening for ocular involvement in every case of CNS TB. In comparison to ocular TB, spinal TB is a frequently observed extrapulmonary manifestation, in the form Pott's spine, or can it exclusively involve spinal cord disseminated tuberculomas are a common in immunocompromised patient. We report an immunocompetent patient with no past h/o TB who presented with concurrent choroidal tubercles with multiple intracranial tuberculomas and intramedullary tuberculoma.

**Key words:** Choroidal tubercles, Tuberculoma, Eye

## INTRODUCTION

The central nervous system (CNS) manifestation of tuberculosis (TB) is a rare but fatal without appropriate therapy. It accounts for 1% of TB cases with high morbidity and mortality across all age groups. Ocular disease is considered as a rare manifestation and has the wide prevalence across the patient populations and geography.<sup>1</sup> It may be because of direct mycobacterial infection or hypersensitivity reaction. The most common forms of ocular TB include choroidal TB and tubercular uveitis. Early diagnosis and prompt treatment may be sight saving so screening for ocular involvement in every case of CNS TB. In comparison to ocular TB, spinal TB is a frequently observed extrapulmonary manifestation, in the form Pott's spine, or can it exclusively involve spinal cord as seen in our case.

## CASE REPORT

A 15-year-old boy visited our hospital with the chief complaints of fever of 10 days duration, weakness of both lower limbs for 4 days, double vision since 2 days, and headache and vomiting for 2 days before the hospitalization. A fever was low grade, intermittent associated with chills and the weakness of both lower limbs manifested in the form of inability to get up and walk was sudden in onset, non-progressive. He also had c/o blurring of vision and double vision on leftward gaze. The following day, he developed a headache associated with recurrent vomiting.

On examination, he was a febrile with a regular pulse of 108/min, a supine blood pressure of 100/70 mmHg, a RR of 20/min, and SpO<sub>2</sub> 95% with room air. On the nervous system examination, he was conscious, oriented with normal higher mental functions, pupil on the right side was 2-3 mm reacting to light and in left eye pupil was eccentric reacting sluggishly. He had signs of meningeal irritation like neck rigidity and Kernig's sign. Cranial nerve examination showed left lateral rectus palsy with restriction of movement of the left eye in lateral gaze. On diplopia charting, his c/o diplopia increased in the left lateral gaze. Rest of the cranial nerves was normal. On

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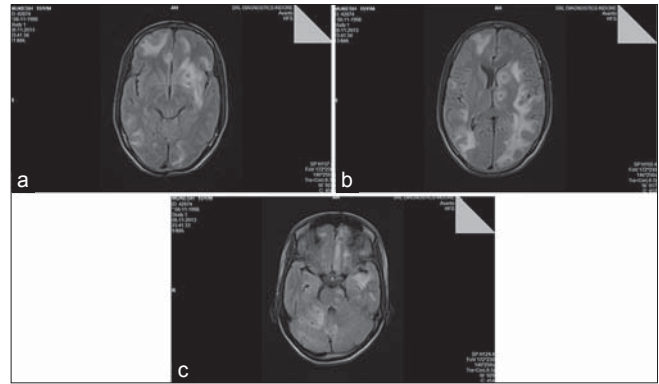
motor system examination, he had spastic paraparesis of both lower limbs with exaggerated reflexes. Upper limbs were normal and bilateral planters were extensor. Sensory system examination was essentially within normal limits with no cerebellar signs. On the basis of history and clinical examination, a possibility of meningitis was kept and in view of his paraparesis, raised ICT, headache and vomiting a possibility of an intracranial space occupying lesion was also considered. All his routine investigations including electrocardiogram and chest X-Ray were normal. His magnetic resonance imaging (MRI) brain revealed multiple ring-enhancing lesions distributed throughout the brain and screening through the spinal cord showed a single hypointense lesion in the cord opposite D5-D6 level.

In view of his abnormal pupil on the left side and c/o blurring of vision, we did a detailed ophthalmological examination which revealed the presence of an eccentric pupil (Corectopia) and choroidal coloboma of the left eye. Right eye showed the presence of choroidal tubercle along superotemporal quadrant with papilledema in both eyes. To rule out meningitis cerebrospinal fluid (CSF) was done, which showed a mild reduction of protein with normal cell count and glucose.

Our final diagnosis was multiple tuberculomas in brain, spinal cord, and choroid with congenital anomalies in the form of left eye choroidal coloboma and corectopia. He was started on anti-TB therapy daily regimen. He was improving during his course of hospitalization. His signs and symptom of intracranial tension were reduced. His left lateral rectus palsy improved over the period of 1-week. Power in lower limbs also improved and started walking. After the total hospital stay of 15 days at the time of discharge, he had no complaints of a headache and diplopia, his weakness in both lower limbs was recovered completely. A follow-up MRI is planned after a 1 month to look for resolution of tuberculomas in the brain, spinal cord as well as choroid (Figures 1-6).

## DISCUSSION

Intracranial tuberculomas are a rare, the incidence is variably quoted between 2.3% and 18%.<sup>2</sup> A major health hazard in developing countries usually involve cerebral and cerebellum due to rich blood supply and only 4% are located in brainstem.<sup>3</sup> Our patient had tuberculomas in all these locations. CNS tuberculoma usually confused with intracranial neoplasm whenever present without signs and symptoms of TB. Depending on their size and location, intracranial tuberculomas can have many signs mimicking primary CNS tumors. Although intracranial tuberculomas generally exist as single lesions, 15-34% of them may be



**Figure 1: (a-c) Multiple randomly distributed nodular lesions 5-10 mms in size, in both cerebral hemispheres, Lt basal ganglia, thalami, brain stem and cerebellar hemisphere**



**Figure 2: (a-b) On screening of whole spine during magnetic resonance imaging there was a solitary nodular T2 hypointense intramedullary lesion in cord opposite D5/6 disc level**



**Figure 3: Fundus picture showing hyperemic disc with blurred margins suggestive of papilloedema**

multiple. They have been found in all parts of the CNS and associated with focal neurological signs, seizure or increase in the intracranial pressure, they can also be asymptomatic.<sup>4</sup> Miliary TB was found the most important independent factor linked to the development of ocular TB. Neuro-ophthalmic involvement, a part of its presentation spectrum, is important, and numerous reported neuro-ophthalmologic complications are combinations of oculomotor palsies, pupil abnormalities, disc changes suggestive of papillitis, papilledema, or optic atrophy and



**Figure 4: Fundus picture showing presence of choroidal tuberculoma in the right superotemporal quadrant**



**Figure 5: Eccentrically placed pupil on the left side (Corectopia)**



**Figure 6: Fundus of left eye showing coloboma of left eye**

choroidal tubercles.<sup>5</sup> Choroidal tuberculoma is the most common form of ocular TB and it is suggested that this is related to high blood supply of choroid.<sup>6-10</sup> Only a few

cases of choroidal tuberculomas have been reported and it has been considered uncommon (1%). However, Bouza *et al.* reported a high incidence of ocular TB (18%), and they suggested that these results might be related to the increase in the incidence of ocular TB and/or to detailed ocular examination of all TB patients.<sup>11-13</sup>

Choroidal tuberculoma may present with or without active TB. Some cases with ocular TB may be asymptomatic and may be detected during routine ocular examinations. Choroidal and intracranial tuberculomas are formed by hematogenous spread from other active infected foci.<sup>8</sup> Increasing the use of neuroimaging techniques has greatly helped in the early diagnosis of intracranial tuberculomas. It may be difficult to differentiate the MRI findings of tuberculomas from these lesions. Noncaseous tuberculomas usually have a hypointense signal on T1WI and a hyperintense signal on T2WI, with homogeneous enhancement after gadolinium administration.<sup>6,7</sup> Solid caseous tuberculomas have iso- or hypo-intense signal on both T1WI and T2WI sequences. Diffusion-weighted MRI and magnetic resonance spectroscopy characteristics of TB lesions are neither sensitive nor specific.<sup>11</sup> CSF analysis in intracranial tuberculomas without meningitis shows a mild lymphocytic pleocytosis with a nonspecific increase in protein content, and CSF bacteriology is usually negative.<sup>9</sup>

Choroidal tuberculomas are frequently unilateral and appear predominantly in the posterior pole as solitary or multiple lesions. They are gray, grayish-white or yellowish and has indefinite borders. Although retinal vessels overlying these lesions appear normal, sometimes hemorrhage and/or exudation may be found. In the literature some authors reported late hyperfluorescence, others reported minimal early fluorescence with late staining by fundus fluorescein angiography (FFA). Since there are no typical FFA findings for choroid tuberculoma, FFA can contribute to exclusion of other causes. Histopathologic confirmation, which is needed for definite diagnosis of choroidal tuberculoma is not practical, therefore the diagnosis of choroidal tuberculoma is usually presumptive and is based on clinical and laboratory findings. Response to the treatment may be helpful to confirm the diagnosis of choroidal tuberculoma. Our patient also had congenital ocular anomalies in the form of choroidal coloboma and corectopia of pupil which may be a chance detection and no reported association was found between these and tuberculomas.

## CONCLUSION

Although CNS tuberculoma is a rare entity and usually presents with typical manifestation like headache, seizure, focal neurological deficit, and signs of raised intracranial

pressure. It can present with atypical manifestation like diplopia and nystagmus as in our case. In developing countries where TB is endemic, CNS tuberculoma can present with atypical manifestations.

Since the delay in diagnosis and treatment results in poor prognosis and severe sequel, the effective therapy should be initiated as early as possible. In conclusion, early diagnosis, adequate treatment and follow-up for the response to the treatment of choroidal tuberculoma can prevent serious complications. Detailed ocular examinations should be performed in all cases with TB, for possible presence of early asymptomatic choroidal tuberculoma.

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