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REELING REELED IT IN - AN UNCOMMON PRESENTATION OF A COMMON DISEASE

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ABSTRACT

This case presents with a unique clinical characteristic of an immunocompetent host with an unusual lower lobe predominant pulmonary tuberculosis infection and multifocal intra-cranial tuberculomas. The prognosis for brain tuberculoma is good, and effective treatment options are available. Enhanced brain and spine MRI should be taken into account for those with neurological sequelae, while those with persistent intracranial hypertension and lesions should only undergo craniotomies.

Keywords: tuberculosis, intra-cranial tuberculomas, immunocompetent, ICT

1. INTRODUCTION

Mycobacterium tuberculosis, the causative agent in tuberculosis, is highly aerobic, and therefore affects primarily the respiratory system.[1] M. tuberculosis can spread up through the circulatory system and seed into the central nervous system (CNS) as tuberculomas. Although rare, cases of immunocompetent patients with less severe lung involvement who developed tuberculomas have been reported in fewer case reports.[2] In this present case, we interestingly found that, an immunocompetent host with an unusual lower lobe pulmonary tuberculosis infection and multifocal cerebral tuberculomas make up a unique clinical triad.

CASE REPORT

History: This patient, aged 45 years female, known case of Hypothyroidism, appeared to be in good health, up until three months prior to admission, when she started coughing up yellowish sputum with minimal expectoration, that was coupled with an unpleasant odour but no H/O haemoptysis; Then began to experience moderate to severe intermittent headaches which started about 10 days prior to admission; unrelated to premonitory symptoms like photophobia, photophobia nausea, or vomiting, and aggravating with cough, alleviating on rest; The patient complained of being unable to stand unaided upon admission, and also experienced generalised fatigue, 1 day before admission.

Clinical Findings: Patient was poorly built. She appeared generally wasted and lethargic upon physical inspection. She was alert, but drowsy. Vital signs were stable. Pallor noted; No lymphadenopathy. At first, the chest revealed only mild Left basal coarse crepitations. The heart and abdomen both showed no evident abnormalities. Neurological evaluation revealed B/L flexor plantar response, however B/L proximal + distal power was rated as 4 out of 5, with diminished DTRs. Examination of the sensory systems was normal. Cerebellar signs could not be assessed precisely due to debility. Her deranged metabolic lab results were initially assumed to be the cause of her frailty (potential D/Ds such as osteomalacia, myopathies, etc.,) and remedial steps were taken; however, she displayed an ataxic gait pattern when she first started walking. Ophthalmological fundus examination was performed in response to intermittent headache episodes, which revealed papilledema. As part of the evaluation for ataxia and due to suspicion of raised ICT, MRI- Brain was ordered, which revealed multi-focal intracranial tuberculomas.

Treatment: Considering the clinical correlate, radiological correlation, and sputum positivity for tuberculosis, anti-TB medications (FDC) were started right away. The recommended dosages of Isoniazid, Rifampin, Ethambutol, and Pyrazinamide were included in the regimen, along with vitamin B6. Steroids were administered and tapered over an 8- 12-week period. OHA had been initiated to treat steroid- induced hyperglycaemia.

Outcome: Lethargy waned after a few days, and her overall health improved within about 10 days, but she continued to experience mild ataxia; discharged, with a near-total recovery reported after routine follow-up and complete resolution of symptoms after 6 months of ATT

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2. DISCUSSION

RISK FACTORS: There are a number of risk factors for this serious phenomenon. These include the rising rate of HIV infection, urban population overcrowding, and abnormal communities (such as prisons, concentration camps, refugee colonies), Nutritional deficiencies, the emergence of drug-resistant tuberculosis strains, and ineffective tuberculosis control programmes

• **PATHOGENESIS:** Tuberculous bacilli reach the central nervous system (CNS) by a route secondary to disease elsewhere in the body. The rupture or growth of one or more of these small tuberculous lesions (Rich Foci) results in various types of CNS tuberculosis, including meningeal irritation. This typically occurs in immunocompromised patients [3], although few immune-competent cases have been documented [2].

• **CLINICAL COURSE:** In most patients with tuberculous meningitis there is a history of vague ill health lasting 2–8 weeks. Cranial nerve palsies occur in 20–30% of patients and may be the presenting manifestation of tuberculous meningitis. Vision loss due to optic nerve involvement may occasionally be a dominant presenting illness.

• **INVESTIGATIONS (MRI):** On T1-weighted images, non-caseating granulomas are iso- or hypointense; after contrast therapy, they become hyperintense. The degree of perilesional oedema in these kinds of granulomas varies. The caseous material in the tuberculoma's centre has liquefied, making it seem hypointense in the centre, with a peripheral hypointense ring which represents the capsule.[4]

3. CONCLUSION

Brain tuberculoma is a non-threatening condition with a good prognosis and effective therapy options. If treatment is started in stage I, mortality and morbidity is very low. About 20% to 30% of survivors manifest a variety of neurological sequelae. These include mental retardation, psychiatric disorders, seizures, blindness, deafness, ophthalmoplegia and hemiparesis. Enhanced brain and spine MRI should be considered to confirm that the diagnosis is not overlooked. Craniotomy is reserved for patients with intracranial hypertension [5], whereas patients with cerebral tuberculomas but no intracranial hypertension should proceed more towards conservative approach.

4. REFERENCES

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IMAGES

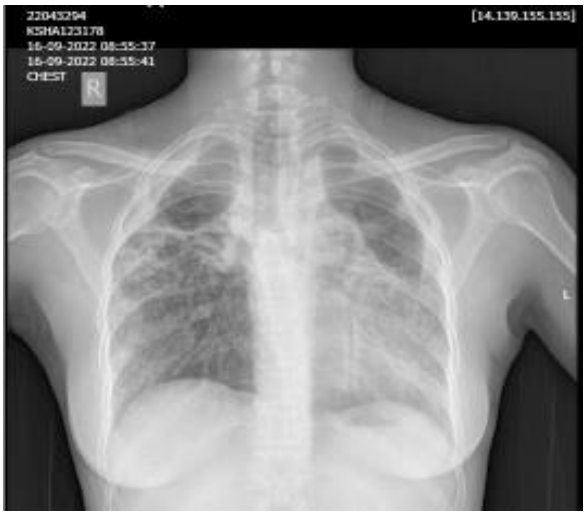

Figure 1: Chest X-ray: Non homogenous infiltrates in Lower lung fields with cavity- Right mid-zone of lung

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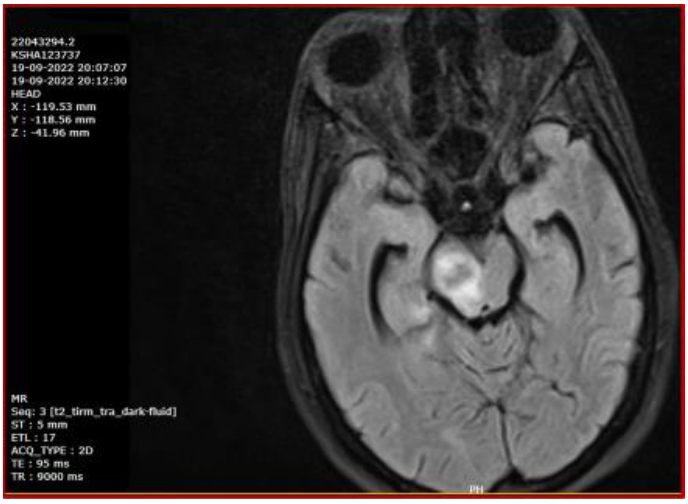
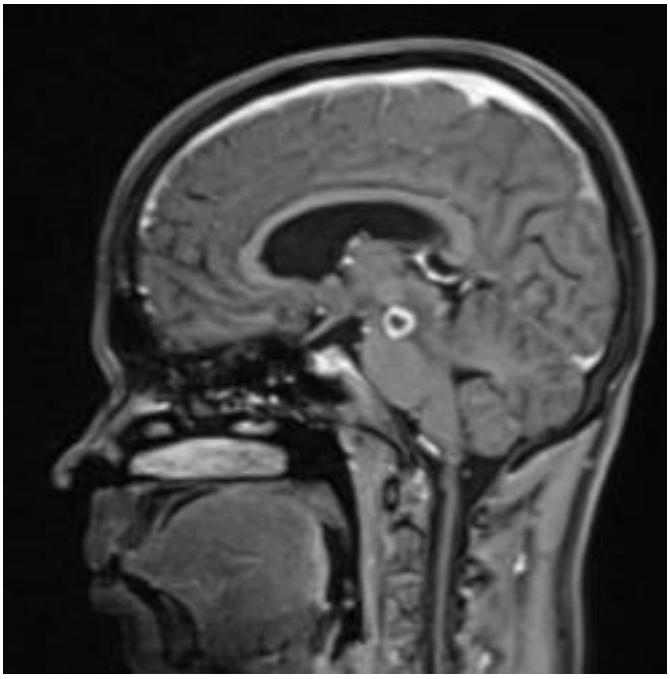


2A



2B

Figure 2a and 2b: MRI T2 image a) Cross section, b) Sagittal section (Post- contrast); Irregular peripherally enhancing lesion noted in right cerebral peduncle measuring 10x14x15 mm.

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3A **3B**

Fig 3: 3A- Patient at the time of admission; 3B- Patient after 6 months post-ATT course completion status