



Mycobacterium Tuberculosis Meningoencephalomyelitis Induced Optic Nerve Atrophy and Permanent Blindness



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Introduction

- One third of the world's population is infected with *Mycobacterium tuberculosis* (MTB)
- Central nervous system (CNS) infections account for less than 1% of cases
- Rarely does it result in irreversible neurologic deficits

Case Presentation

- 21 year old male with no past medical history presented with 2 week history of leg weakness and head and neck stiffness
- Developed somnolence and mental incoherence with reported fevers, headaches, nausea and vomiting

Clinical Course

- MRI showed multiple enhancing cortical and subcortical lesions without mass effect
- CT Chest showed bilateral upper lobe reticular infiltrates
- Lumbar puncture revealed pleocytosis with negative Gram stain. Polymerase chain reaction (PCR) on cerebrospinal fluid (CSF) and sputum positive for MTB
- Started on rifampin, isoniazide, pyrazinamide and ethambutol (RIPE) for MTB meningoencephalomyelitis and discharged in a stable condition after 3 weeks
- Re-admitted two weeks later with fevers, bilateral cranial nerve VI and left-sided cranial nerve VII palsies, hyponatremia and urinary retention
- Given IV antibiotics, steroids, and underwent lumbar puncture with unchanged findings
- Repeat head CT: obstructive hydrocephalus and unchanged cerebral lesions
- Underwent external ventricular drain placement with marked neurologic improvement albeit continued debilitating blurry vision.
- Discharged to the Texas Center for Infectious Disease (TCID) with the addition of levofloxacin to his RIPE therapy while he received high dose Rifampin 1500mg daily.
- Gained 90 pounds over the next 10 months of prolonged steroid taper
- Subsequent MRI brain showed resolution of cerebral lesions, new bilateral optic nerve atrophy and growth of a left frontal lobe mass (**Exhibit 1**)
- Underwent surgical resection with specimen showing caseating granulomas with positive acid-fast smear and negative cultures consistent with non-viable MTB (**Exhibit 2**).
- Completed his treatment, discharged near his presenting weight, legally blind, without evidence of residual infection.

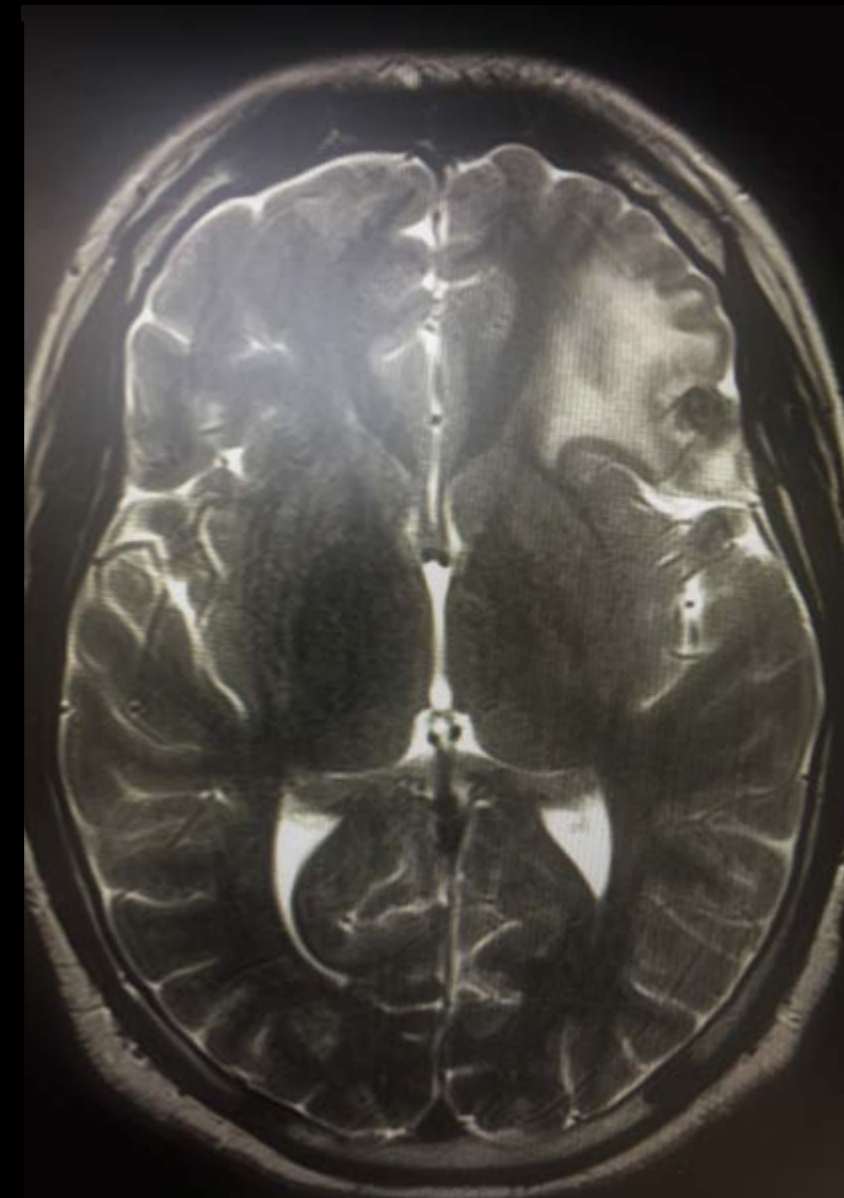


Exhibit 1. Axial MRI of the brain illustrating an ill-defined mass in the left frontal lobe without localized inflammatory changes.



Exhibit 2. Surgical specimen removed from left frontal lobe showing gross caseous necrosis. Pathology found caseating granulomatous inflammation with positive acid-fast smears. Negative cultures indicated dead, trapped MTB.

Discussion

- MTB is an acid fast bacillus infecting more than 2 billion people world wide
- Common manifestations include pulmonary disease with cough, sputum and hemoptysis with concomitant constitutional symptoms of fever, night sweats, and weight loss, though any organ system may be affected
- MTB meningitis is more typically seen in infants or as reactivation in immunocompromised adults
- MTB meningoencephalomyelitis leading to bilateral optic nerve atrophy and blindness has seldom before been documented
- Tubercular proteins entering the subarachnoid space cause a hypersensitivity reaction that may contribute to CN palsies, delirium and hydrocephalus
- Diagnosis through early lumbar puncture is imperative
- Steroids are vital for treatment of meningoencephalomyelitis, though steroid-related complications can be encountered such as extreme weight gain and unresolved tuberculoma as seen in this patient
- Cerebral tuberculomas require surgical removal as they may remain a reservoir of persistent infection
- Treatment for MTB is largely unchanged over last few decades and consists primarily of RIPE therapy lasting 6-24 months depending on site of infection
- CNS infection requires high dose rifampin and treatment up to 2 years
- Afflicted with delirium, blindness, cranial nerve palsies, urinary retention, and paraparesis, the patient underwent 24 months of therapy with good clinical and radiographic response though he was left permanently blind

Conclusion

- MTB infection may manifest in any organ system, with CNS involvement occurring in 1% of patients
- Mainstay of treatment includes initial RIPE therapy with regimen modifications based on MTB susceptibilities, clinical response, tolerance and requires a prolonged course up to 2 years for CNS involvement
- Meningoencephalomyelitis from pan-susceptible MTB is uncommon, particularly in the immunocompetent host
- Seldom before has MTB been documented to induce bilateral optic nerve destruction and permanent blindness

References

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