# Mycobacterium Tuberculosis Meningoencephalomyelitis Induced Optic Nerve Atrophy and Permanent Blindness

Capt Matthew Rendo, MD, MC, USAF[1], Adrianna Vasquez, MD[2]

Brooke Army Medical Center, Fort Sam Houston, TX, Department of Internal Medicine[1], Texas Center for Infectious Disease[2]

## 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.

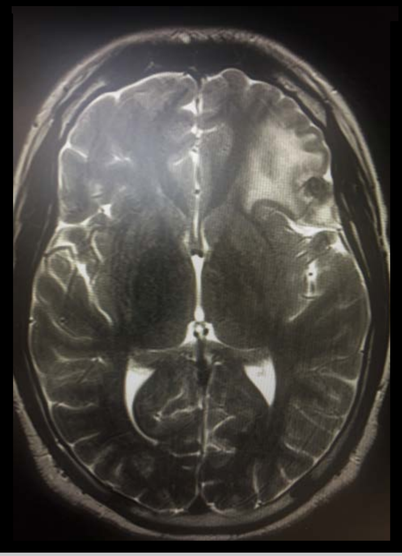

**Exhibit 1.** Axial MRI of the brain illustrating an ill-defined mass in the left frontal lobe without localized inflammatory changes.

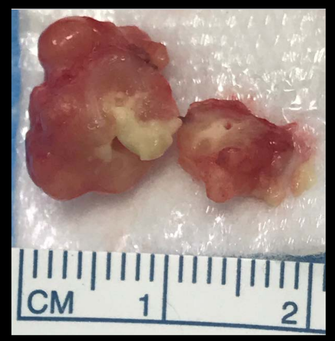


**Exhibit 2.** Surgical specimen removed from left frontal lobe showing gross caseous necrosis. Pathology found caseating granulomatous inflammation with positive acid-fast smears. Negative cultures indicated dead, trapped MTB.



## 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 meningoecephalomyelitis 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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