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Case Report

An Atypical Presentation of Tuberculous Meningitis with complete blindness secondary to Retrobulbar neuritis

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ABSTRACT:
Blindness is an uncommon but devastating complication of tuberculosis meningitis. The main causes are chronically raised intracranial pressure (hydrocephalus and/or tuberculomas) or direct involvement of the optic chiasm or optic nerves by the basal arachnoiditis (inflammation and/or compression).Some patients develop blindness, mainly as a result of progressive optochiasmatic arachnoiditis. The major side effect of Ethambutol is retrobulbar optic neuritis of two types: axial and periaxial. The most common form is associated with macular degeneration, decreased visual acuity, and decreased colour perception.The recommended dose for adults, irrespective of the stage of treatment, is 15 mg/kg/day or 30 mg/kg three times a week.Early reports of ethambutol in adults found toxicity to be a dose related phenomenon. A single daily dose of between 60 and 100 mg/kg caused optical toxicity in eight (44%) of 18 patients. Hence, we present a case here with an atypical presentation of tuberculous meningitis with complete blindness secondary to Retrobulbar neuritis.
Key words: Retrobulbar neuritis, Tuberculous Meningitis.

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INTRODUCTION:

Blindness is an uncommon but devastating complication of tuberculosis meningitis. The main causes are chronically raised intracranial pressure (hydrocephalus and/or tuberculomas) or direct involvement of the optic chiasm or optic nerves by the basal arachnoiditis (inflammation and/or compression.[1] Anti-tuberculosis therapy combined with corticosteroids and control of intracranial pressure constitutes the mainstay of therapy for tuberculous meningitis. Despite these treatment measures, some patients develop blindness, mainly as a result of progressive optochiasmatic arachnoiditis. The major side effect of Ethambutol is retrobulbar optic neuritis of two types: axial and periaxial. The most common form is associated with macular degeneration, decreased visual acuity, and decreased colour perception.[2] The periaxial type is associated with visual field defects. The mechanism is unclear. Toxicity is generally dose related, becomes evident three to six months after starting the drug, and is often reversible on stopping treatment with the drug. If the diagnosis is delayed, however, as it may be if symptoms are not reported, visual damage may be permanent.[3,4] The recommended dose for adults, irrespective of the stage of treatment, is 15 mg/kg/day or 30 mg/kg three times a week.[5] Early reports of ethambutol in adults found toxicity to be a dose related phenomenon. A single daily dose of between 60 and 100 mg/kg caused optical toxicity in eight (44%) of 18 patients.[6] Liebold reported a 19% incidence of ocular toxicity among 59 patients receiving dosages of over 35 mg/kg compared with 3% (two patients) among 59 receiving doses of less than 30 mg/kg. Studies using doses ranging from 15 to 25 mg/kg have reported complications in 1–3% of adults.[7-10] Hence, we present a case here with an

atypical presentation of tuberculous meningitis with complete blindness secondary to Retrobulbar neuritis.

CASE REPORT

A 40 years old female reported to the casualty with chief complaints of fever with chills, vomiting and loose stools from the last 4 days. Patient was apparently pleasing 4 days back when she started experiencing the symptoms. She had high grade fever associated with chills, continuous and not relieved on taking medications from an outside doctor. Patient also complained of headache associated with fever. She had vomiting from past 4 days with 1-2 episodes per day, containing food particles, non-blood, and non-bile stained. Patient complained of loose stools from last 4 days with 2-3 episodes per day, watery in consistency, foul smelling, and non-blood stained.

She had no medical history of Koch's disease, diabetes mellitus, bronchial asthma, hypertension or epilepsy. Family history of Koch's disease contact through son was reported.

Vitals noted on admission:

GC: Fair; Pulse- 88/min; BP- 110/60 mmhg ; Spo2- 99% on RA.

Working Diagnosis was established as Acute Gasteroenteritis:

Three days later, the patient suddenly became drowsy and disoriented; however, responded to verbal commands (neck stiffness was present). CT Brain was done which was suggestive of infective calcified granuloma. Lumbar Puncture was also done the same day and CSF was sent for MGIT and GENE Xpert. CSF labs revealed proteins at 78 mg/dl, sugar at 44.8 mg/dl, total WBC at 160 cells/cu.mm, lymphocytes at 95%, and neutrophils at 5%. The reports where suggestive of Tuberculous Meningitis and the patient was shifted to ICU and started on anti tuberculous treatment, (tablet Akurit 4 : 3-0-0)

At that time, all the routine reports where normal except serum-Na level which had dropped from 130 to 121.

After two days of starting Tablet Akurit 4, patient started complaining of diplopia, blurred vision followed by complete loss of vision in the next two days. (Ethambutol was stopped considering ETB induced loss of vision and calculated doses of individual drugs were given; Rcinex (450/300) , Pyrizina (1000))

MRI Brain / Optic Nerve / MRI Angio was done which did not reveal significant changes to suggest sudden blindness. The patient was referred to Ophthalmology who reported that fundus is normal and there is no cause for blindness. Considering retrobulbar neuritis, VEP was planned; and injection Methyl Prednisolone was started.

Patient was planned for VEP; however, the patient's condition suddenly became disoriented; patient became irritable and drowsy with presence of severe neck rigidity and stiffness. Repeat CT Brain was done, which was suggestive of acute onset obstructive hydrocephalus. Neurosurgery reference was taken and was advised for VP shunt.Patient was started on T. Ethambutol 250 (3-0-0). (There was history of KOCHS to Patients mother as well as son, history of similar complaints to brother 4-5 years back for which patient was started on ATT and the patient did not respond and expired after 2 years).

DISCUSSION:

Khanna SR et al described a rare case of tuberculous meningitis in an immunocompetent host, questioning the conventional view that tuberculous meningitis is a disease of immunocompromised individuals. It emphasizes the importance of maintaining a strong clinical suspicion of tuberculous meningitis even in an immunocompetent patient in a geographical area with low prevalence if the patient has risk factors. Missed or delayed diagnosis is commonly fatal. AHMETGJEKAJ I et al described a 5-year-old child with documented TBM being treated with first line antituberculous drugs which developed visual impairment 3 months after starting the treatment. MRI after gadolinium administration revealed multiple perichiasmatic ring enhancing lesions due to tuberculomas. Visual impairment developing in a patient on treatment with antituberculous drugs should give rise to a suspicion of rare optochiasmatic tuberculomas; this necessitates urgent contrast-enhanced MRI of the brain and prompt treatment with steroids.[11, 12]

Jawad N et al described a case of TBM in a 19-year-old Asian female. She had a progressive motor weakness with no sensory findings. She was started on antituberculous therapy. Her magnetic resonance imaging (MRI) contrast of dorsolumbar spine showed syringomyelia. Her culture and sensitivity for Mycobacterium tuberculosis (MTB) came negative. She was given a therapeutic trial of quinolones and Steroids. She had an uneventful recovery and was followed up for the past one year.[13]

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