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Acute Visual Loss in a Child with Tuberculous Meningitis Due To Optic Neuritis

Abstract:
Acute visual loss is a dreaded complication of tuberculous meningitis and it can be due to various causes such as optochiasmatic arachnoiditis, tuberculoma, drug induced optic neuropathy, hydrocephalus. Visual loss due to optic neuritis in tuberculous meningitis is uncommon. We report a child with TBM who developed acute visual loss due to optic neuritis effectively managed with intravenous immunoglobulin.

Key words:
Acute visual loss, tuberculous meningitis, optic neuritis

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I. Introduction:

Acute visual impairment is a devastating complication of tuberculous meningitis(TBM). Vision impairment in TBM can be due to a variety of causes including optochiasmatic arachnoiditis, compression of the optic chiasm or optic nerve by tuberculoma, optic neuropathy caused by inflammation /vascular cause, optic neuritis associated with antitubercular therapy (ethambutol, sometimes isoniazid), secondary to hydrocephalus and raised intracranial tension, bilateral occipital infarcts due to vasculitis and chorioretinitis.[1]. We report a case of tuberculous meningitis presenting with bilateral visual loss due to optic neuritis.

II. Case Report:

A 12-year-old female child presented with fever, headache, and vomiting of one week duration followed by drowsiness for one day. There was no history of cough, loose stools, dysuria, nuchal pain, or photophobia. There was no history of head trauma, ear discharge, recent exanthem/vaccination. No history of contact with an open case of tuberculosis either. On examination, she was drowsy, febrile, resented examination. Doll's eye movements were full and pupils were equal and reacting to light. She could move her limbs against gravity, deep tendon reflexes were normal with flexorplantars. Meningeal signs were positive. General examination was not contributory and other systems were normal.

The child was started on meningitic dose of antibiotics, acyclovir, and antiodema measures. Complete blood count showed neutrophilic predominance. Metabolic parameters, C-reactive protein, blood culture, chest X-ray were normal. CT brain plain and contrast was normal. CSF analysis done on the second day of admission showed 4 lymphocytes, protein - 188mg/dl, sugar- 18mg/dl, and normal gram staining. She was started on antituberculous therapy - isoniazid, rifampicin, pyrazinamide and ethambutol, and oral prednisolone. Sensorium improved on the third day and she complained about the blurring of vision in both eyes. Examination revealed no perception of light with a sluggish pupillary response to light in both eyes. Fundoscopy revealed bilateral hyperemic optic discs and blurred nasal margins. There was partial ptosis with restriction of all extraocular movements in the right eye and right facial lag. Mantoux was positive and CSF CBNAAT was positive with Rif sensitivity.

MRI brain including contrast was normal. Ethambutol was stopped and intravenous methylprednisolone in the dose of 30mg/kg for 5 days was started. CSF virology was negative for Japanese B, herpes, varicella, entero, cytomegalo viruses. CSF fungal staining was negative. As there was no improvement in vision, INH was stopped and levofloxacin was added. The child became conscious, oriented, extraocular movements improving in the right eye and tone, power was normal in all four limbs. However, the vision did not improve. Repeat MRI brain with contrast showed subtle enhancement of left optic nerve. Serum NMO and MOG antibodies were negative. Vasculitic workup and retroviral screening turned out to be negative. There were no waveforms in visual evoked potential. Given no improvement in vision despite high dose steroids and ATT, intravenous immunoglobulin was tried as immunomodulation. There was a dramatic improvement, and visual acuity became 6/12 in both eyes in five days. INH was reinstated. Repeat CT brain was normal. The papillitis had resolved bilaterally and the fundoscopy was normal. After 2 weeks, there was a complete restoration of visual acuity to 6/6 in both eyes.

III. Discussion:

Acute visual loss is a disabling complication of TBM. Sinha et al reported that 27% of TBM patients had decreased vision and the main causes were OCA and optochiasmal tuberculoma.[2]

Optochiasmatic arachnoiditis (OCA) and optochiasmal tuberculoma are severe complications of TBM associated with profound visual impairment.[3]The visual loss is attributed to compression or ischemia or to both mechanisms and sudden visual loss may be due to compromise of blood supply or rarely to a paradoxical reaction during antitubercular treatment (ATT) following the withdrawal of steroids.[4]Magnetic resonance imaging (MRI) with contrast will show features of perichiasmal enhancement and hypertrophy of chiasma and the cisternal segment of optic nerves.[5] MRI brain with contrast did not show any basal exudates or hydrocephalus in our child.

Ethambutol-related optic neuropathy usually presents as bilateral, progressive, painless blurring of vision and decreased color perception which usually develops after several months of exposure but there are reports of severe visual impairment occurring as early as a few days of commencing treatment.[6] Isoniazid-related optic neuropathy generally occurs within 10 days of starting antitubercular therapy and rarely has been reported in children.[7,8] Visual impairment was noticed a day after starting ATT, hence may not be due to ethambutol/isoniazid toxicity. However, both were stopped and levofloxacin was added. Reintroduction of isoniazid is associated with a quicker onset of optic neuritis.[7] Our child did not develop any new visual symptoms after the reintroduction of isoniazid.

The complete ophthalmological examination did not reveal any evidence for uveitis or chorioretinitis. Fundi were hyperemic and no features of papilloedema were evident. Visual evoked potentials were not obtained in both eyes. Hence bilateral optic neuritis was considered and ATT was continued and a course of methylprednisolone followed by oral steroids was given. As there is no improvement in vision, intravenous immunoglobulin 400mg/kg/day for 5 days was given for its immunomodulatory action. There was a dramatic improvement in vision in 5 days.

A variety of modes of action have been attributed to the beneficial effects of IVIg, including its interaction with T-cell function, antigen-presenting cell maturation/presentation, combined with a general "tune down" effect on inflammatory reactions. Neuroprotective effects of IVIG may be due to anti-inflammation including microglial suppression, and direct protection of the optic nerve.[9] A high concentration of exogenous immunoglobulin G molecules may outcompete existing circulating endogenous antibodies and consequently dampen clinically unproductive inflammation.[10]

IV. Conclusion:

To conclude, clinicians should watch for visual symptoms in children with tubercular meningitis to diagnose visual loss, a dreaded complication. Visual loss in tubercular meningitis can occur due to optic neuritis. A trial of intravenous immunoglobulin is worthwhile in visual loss due to tuberculous optic neuritis.

Conflicts of interest: Nil

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