A Rare Case of Acute B/L Optic Neuritis with B/L Papilloedema with Anti Mog Antibodies Positive

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Abstract

A 46 years old ladycame to ED with c/o B/L progressive loss of vision since 6 days associated with intermittent headache. With Left eye perception of hand movements at 10 inches. With Right eye she can see objects about 5 meters but unable to identify color and face MRI orbit contrast/MRI brain contrast s/o B/L optic neuritis with subtle T2/flair hyperintense signal is seen in the periaqueductal region, ? artefactual, ? changes of NMO/NMOSD Hematology report revealed anti MOG antibodies:Positive Visual Evoked Potential(VEP) suggestive of B/L demyelinating anterior visual pathway defect ANA and vasculitic panel negative. Ophthalmo opinion revealed B/Lpapilloedema.

Keywords: B/L optic neuritis; B/L papilloedema; Anti mog antibodies; Neuromyelitis Optica (NMO); AQP4 India.

Introduction

MOG antibody demyelination and Neuromyelitis Optica (NMO): are autoimmune neurological conditions which cause attacks of inflammation in the optic nerves and/or the spinal cord.¹⁻⁵

MOG stands for myelin oligodendrocyte glycoprotein and is found in myelin sheath of nerve cells. Myelin sheath insulates and protects nerves and helps them to work effectively. It is thought that MOG helps to repair myelin sheath when it gets damaged. In MOG antibody demyelination, MOG antibodies attack the myelin oligodendrocyte glycoprotein and causing damage to myelin sheath

Most people with NMO have proteins in their blood called anti-aquaporin 4 antibodies (AQP4), which are cause of the disease.⁷⁻⁹

When AQP4 or MOG antibodies attack optic nerve and/or the spinal cord, they also damage the myelin sheath simultaneously. This is a layer that covers and protects the nerves. Demyelination is defined as damage to myelin sheath.¹⁰⁻¹⁴

Optic Neuritis

It is described as any condition that causes inflammation of optic nerves. It may be associated with demyelinating diseases, or infections, or inflammatory processes.¹⁵ It is often associated with multiple sclerosis and it may lead to complete or partial loss of vision in one or both eyes. Symptoms include sudden loss of vision, sudden blurred or foggy vision, and pain on movement of affected eye.other early symptoms are decreased night vision, photophobia and red eyes. Many patients

with optic neuritis may present with loss of color visionin the affected eye.¹⁶

Papilloedema

It is optic disc swelling that is caused by increased intracranial pressuredue to any cause. The swelling is usually B/L and can occur over a period of hours to weeks. U/L presentation is extremely rare.In early stages it may be asymptomatic or present with a headache. It can progress to enlargement to blind spot,blurring of vision,visual obscurations and ultimately total loss of vision may occur

Case Study

A 46 years old female patient was admitted with c/o B/L acute onset of progressive vision loss, left more than right since 6 days associated with intermittent headache.as per patient, she was alright before event when she went to bed after dinner, but when she woke up next day in the morning she had blurred vision. Patient felt like fogging in front of both eyes. She was noticed pain in both eye movements with progressive loss of vision. On detailed examination, in left eye perception of hand movements was at 10 inches, but from right eye she can see objects about 5 meters but unable to identify colors and face.

Physical examination revealed the patient stable, but moderateheadache, conscious, oriented, vitally stable: Pulse: 84/min,BP: 140/80 mmhg,RR:18/min,Temperature: afebrile,Spo2:99% on room air, No Pallor seen. GCS: 15/15

P/A - Soft, non tender; No organomegaly/guarding/rigidity.

LMP: menopause achieved 4 years back as informed by patient herself Neurological, Cardiovascular, Respiratory examinations were insignificant.

MRI brain contrast/MRI orbit contrast advised. Lumbar puncture done on L3-L4. CSF sample collected. Ophthalmology reference taken. Relevant blood samples sent for further management.

She was admitted and discharged in a stable condition after 13 days.

Course in the Hospital and Outcome

After initial pain management, patient was immediately taken for MRI brain contrast and MRI orbit contrast which revealedsign of B/L optic neuritis with subtle T2/flair hyperintense signal is seenin periaqueductal region? Artefactual,? changes of NMO/NMOSD. Hematology report revealed

anti MOG antibodies present.VEP suggestive of B/L demyelinating anterior visual pathway defect. ANA and vasculitic panel negative. Ophthalmology opinion taken for c/o sudden painless diminished vision. Under aseptic conditions lumbar puncture done on L3-L4. CSF samples collected. CSF pressure 10 cm H2O.CSF reports s/o cells-20 (lymphocytes-95%, neutrophils-5%), sugar-112, protein-46.06, opening pressure-10.

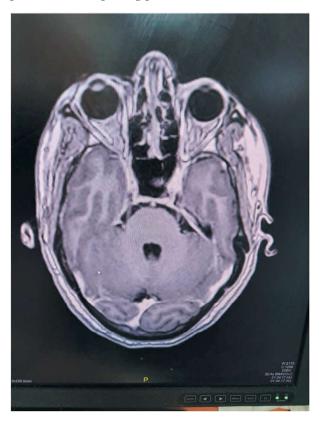


Fig 1: CT Scan Shows B/L Optic Neuritis.

Patient was taken on diuretics, steroids, antibiotics, benzodiazepines, NSAIDS, calcium channel blockers, multivitamins and symptomatic supportive treatment. She was discharged in a stable condition after 13 days.

Discussion and Therapeutic considerations

In MOG antibody demyelination, MOG antibodies attack the myelin oligodendrocyte glycoprotein, causing damage to myelin sheath. AQP4 antibody NMO may cause repeated attacks which can lead to severe disability if not treated. MOG antibody demyelination is less likely to cause further attacks and appears to cause less severe disability in most people. B

MOG antibody have been recently found in

people who have the following:

- 1. Optic neuritis: most oftenly associated with multiple sclerosis. Other causes may include-hereditary optic neuritis, autoimmune ds i.e sarcoidosis, SLE, PAN, granulomatosis with polyangiitis, infections liketuberculosis, lyme disease, cryptococcal meningitis in AIDS patients.²⁰
- 2. Transverse myelitis:It is inflammation of spinal cord and can have different effect on each person,depending on which area of spinal cord has been affected. The main symptoms are: muscle weakness in the legs and/or arms,altered sensations(pins and needles,numbness),bladder and bowel problems and pain.²¹
- 3. ADEM: i.e acute disseminated encephalomyelitis This is an inflammatory condition affecting the brain often after an infection and is more common in children.²²

Papilloedema: causes may include raised intracranial pressure due to brain tumor, intracerebral hemorrhage or idiopathic, medicines like isotretinoin, tetracycline, hypervitaminosis A, hyperammonemia, GBS, malignant hypertension, medulloblastoma.¹⁹

Conclusion

Why should an emergency physician be aware of headache with diminished/blurring/fogging of vision with progressive painless loss of vision! This is because in emergency department headache with diminished vision is a common presentation, and if the patient have MOG antibodies positive, then the patient have a "on-off"attack and will usually recover well. Some peoples do experience further attacks, but the risk of this may be reduced if the patient is prescribed for corticosteroid treatment. ²³⁻²⁵ so making the diagnosis accurately and initiation of treatment can lead to favorable outcome

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