A Case of Bilateral Optic Neuritis

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Abstract

A 22 year male patient presented with headache, fever, and vomiting since 3 days duration and was initially managed as a case of meningitis or suspected brain abscess. He developed painful blurred vision in the right eye 2 days after his admission.

On ocular examination the vision in right eye was reduced to counting fingers close to face with presence of colour de-saturation in right eye. The vision in the left eye was 6/6. There was a presence of relative afferent pupillary defect in the right eye. Examination of the fundus showed presence of disc oedema with surrounding parapapillary oedema in the right eye. A provisional diagnosis of optic neuritis secondary to demyelination was made as MRI showed demyelinating plaques. Patient was started on IV Methylprednisolone and oral steroids as per ONTT (Optic Neuritis treatment trial) guidelines. Patient vision improved to 6/15 in the right eye and the oral steroids were tapered.

After 2 weeks of stopping steroid therapy patient visited the outpatient clinic with painful blurring of the vision of the left eye. On examination the visual acuity in the left eye was hand movements close to face with colour desaturation and presence of relative afferent pupillary defect in the left eye. Patient was put on IV methylprednisolone and oral steroids. Visual acuity at the end of treatment was 6/9p in right eye and 6/12 in left eye. On follow up MRI at 3 months there was resolution of the demyelinating changes on MRI and the final visual acuity was 6/9 in right eye and 6/9p in left eye. This patient needs to be followed up to detect development of demyelinating diseases later in life.

Introduction

The causes of acute Vision Loss in white eye include Optic Neuritis, Anterior Ischemic Optic Neuropathy, Severe Vitreous Hemorrhage, and Retinal Detachment with macular involvement. This is a case of bilateral optic neuritis secondary to demyelination.

Case Report(s)

A 22 year male patient presented to the Medicine outpatient department with headache, fever, vomiting since 20 days and blurred vision since 3 days duration. Patient was treated for upper respiratory tract infection and was prescribed antibiotics by previous doctors. There were no co-morbid conditions and past medical history was not significant. He was a non smoker and non alcoholic.

General examination revealed an alert and conscious patient. Temperature was 38°C, BP 96/60 mm Hg. Cardiovascular System was normal. Respiratory system was normal. Abdomen examination was normal. Neurological Examination revealed no neurological deficit and there were no meningeal signs. He was initially managed as a case of meningitis or suspected brain abscess. He was referred to the Ophthalmologist for fundus examination.

He gave history of blurred vision in the right eye since three days. There was a mild pain in eye on ocular movement. There was no history of redness, discharge, flashes of light. There was no history of alcohol abuse.

On ocular examination the vision in right eye was reduced to counting fingers close to face with presence of colour desaturation in right eye. The vision in the left eye was 6/6. Ocular movements were normal in both eyes. Anterior segment of the right eye revealed presence of relative afferent pupillary defect. Rest of the anterior segment was normal. Anterior segment examination of left eye was normal.

Examination of the fundus showed presence of disc oedema with surrounding parapapillary oedema in the right eye. (Figure 1). Patient was advised to get MRI Brain and Orbit (with Gadolinium enhancement and fat suppression). On Day 3: Fundus showed the following picture (Figure 2).

MRI showed multiple demyelinating plaques present in the brain (Figure 3).

A provisional diagnosis of optic neuritis secondary to demyelination was made. Due to severe visual loss and confirmed findings of demyelination in the brain, the patient was started on intravenous methylprednisolone 250mg 6 hourly in 250 ml of 5% dextrose for 3 days followed by oral prednisolone 1mg/Kg for 11 days as per ONTT (Optic Neuritis treatment trial) guidelines.

Patient vision improved to 6/15 in the right eye and color contrast improved. Patient was started on oral steroids. The fundus findings on day six (three days...
post IV steroids) (Figure 4).

At the end of Day 17 (14 day of steroids) the vision in Right eye improved to 6/9 P and the fundus showed the following picture (Figure 5).

The oral steroids were tapered and stopped. After 2 weeks of stopping steroid therapy patient visited the outpatient clinic with painful blurring of the vision of the left eye. On ocular examination the visual acuity in the Left eye was hand movements close to face with colour desaturation and in the Right eye 6/9 P. Anterior segment examination of the left eye revealed presence of relative afferent pupillary defect in the left eye. Fundus examination revealed the following picture (Figure 6).

Patient was put on IV methyl prednisolone 1gm daily x 3 days followed by oral prednisolone 1mg/ Kg x 11 days. Patient was advised neurological consultation and was followed up on daily basis in outpatient clinic (Figure 7).

Patient tolerated steroids well and his visual acuity at the end of treatment was 6/9p in Right eye and 6/12 in the left eye. His fundus findings at the end of day 14 is shown in Figure 8.

At the last follow up the visual acuity of RE was 6 / 12 and in the LE 6 /15. Pupils are back to normal. Patient developed steroid induced acne. There was no neurological deficit. Patient was last reviewed on December 2009. He underwent a repeat MRI scan on which was reported as normal

**DISCUSSION**

The causes of acute Vision Loss in white eye include Optic Neuritis, Anterior Ischemic Optic Neuropathy, Severe Vitreous Hemorrhage, and Retinal Detachment with macular involvement. It is important to recognize optic neuritis which is a Demyelinating inflammatory condition involving primary inflammation of optic nerves. It causes acute visual loss and is associated with Multiple sclerosis.

Optic neuritis is best known for its association with Multiple Sclerosis (MS). Acute demyelinating optic neuritis is the presenting feature in 15-20% of patients with MS. It occurs at some time during the course of disease in 50% of patients with MS. In a 10 year follow up 38 % of patients with first episode of Optic neuritis developed MS.

**Epidemiology:**

- Most Common in females
- Age 20-30s
- Annual US prevalence of 5.1 to 6.4 per 100,000 population

**Symptoms (Optic Neuritis Treatment Trial):**

1. **Vision loss:** Acute visual loss typically over several hours to days and reaches nadir 1-2 weeks and may reach as low as No PL. It is generally associated with pain on ocular movement. Typically the vision starts to recover 2-4 weeks and it may take 6-12 months to recover completely. Majority of patients recover the visual acuity even without treatment however they may have subtle defects and may say the vision has “washed out appearance” or “Not Right”

2. **Pain:** It is a very common symptom. 87 % of patients affected with pain in ONTT. It starts almost simultaneously or little before than visual symptoms and worsens on ocular movement

3. **Loss of Color Vision:** Loss of color vision out of proportion to loss of visual acuity is characteristic of optic neuritis. 88 % of involved eyes had abnormal color vision in ONTT

4. **Temporary obscurations of vision also known as Uthhoff phenomenon**

**Signs:**

1. Impaired Visual Acuity (20/20 to NO PL)
2. Impaired Color Vision / Color Contrast (Tested with Mydriacyl bottle)
3. Presence of Relative afferent pupillary defect
4. Visual Field Defects consists of classic central scotoma and generalized depression of visual field
5. Optic Disc Changes include retro bulbar neuritis (Most Common), Papilitis and Neuro retinitis

**Differential Diagnosis of acute demyelinating optic neuritis include:**

- Ischemic Optic Neuropathy
- Neuromyelitis Optica
- Inflammatory Optic Neuropathy secondary to Infectious – Syphilis, Non infectious – Sarcoidosis, Lyme’s disease, SLE
- Compressive optic neuropathies due to mass lesion and tumors
- Toxic and Metabolic causes including methanol poisoning

**Investigations:** The Diagnosis of Multiple sclerosis is basically clinical however gadolinium enhanced fat saturated T1 weighted sequences of brain and orbit is cornerstone of evaluation. Is not mandatory to get the MRI done.

T1 weighted gadolinium enhanced axial image with fat suppression shows diffuse enlargement of optic nerves however MRI findings may be normal in many cases. The typical lesions are 3mm or larger demyelinating lesions which are ovoid and located in the periventricular areas of white matter.

Statistics show that patients who develop ON but normal MRI has 16 % probability of MS in 5 years. At
the first episode of Optic neuritis approximately 50% of patients with no signs of MS will show demyelinating lesions on MRI. Evidence of optic neuritis may be found in 70% of the established cases of MS.

Other methods of diagnosis include CSF analysis and VEP. CSF analysis can show leucocytosis, IgG level more than 15% and oligoclonal banding of proteins in CSF. Multifocal VEP can be done which shows decreased amplitude and increased latency.

**Clinical Course of the disease:** Visual Acuity impairment reaches maximum after 1-2 weeks and may fall to NO PL. Recovery takes 2-6 weeks may be delayed in some cases.

**Treatment:** The mainstay of treatment Guidelines comes from Optic Neuritis Treatment Trial. In cases of mild visual loss treatment is unnecessary. Indications of Treatment include severe visual loss, bilateral involvement, poor vision other eye. The treatment may speed up the recovery of vision but not the long term visual outcome and may reduce the rate of developing MS.

Treatment regimen recommendations come from Optic Neuritis Treatment Trial which recommended use of intravenous methylprednisolone sodium succinate 1 gram daily for 3 days followed by 1mg/kg of oral prednisolone for 11 days followed by tapering of steroids. The treatment hastens visual recovery but no long term benefit on visual acuity. It delays the neurologic events consistent with MS by two years however at three years there is no additional benefit of treatment

Oral Steroids alone are contraindicated as they offer no benefit and may increase the recurrence rate of Optic Neuritis.

Recently Interferon beta-1a and interferon beta 1b has shown to reduce the development of MS in patients with optic neuritis. Some clinicians suggest an early role for this drug at the first demyelinating episode.

**Conclusion**

Our case presented with unilateral severe optic neuritis which relapsed in the other eye within 3 weeks in spite of treatment with methylprednisolone. He was confirmed as having demyelinating areas in the brain. He should be followed up with a neurologist and to rule out neuromyelitis optica and consideration of treatment with interferons.

**Reference**

1. Osborne BJ, Volpe NJ. Optic neuritis and risk of MS:
Illustrations

Illustration 1

Figure: Presenting optic discs Day 1

Illustration 2

Figure 2. Optic discs Day 3 (Vision RE)
Illustration 3

Figure 3. MRI of Brain

Illustration 4

Figure 4. Optic disc Day 6
Illustration 5

Figure 5. Optic discs Day 14

Illustration 6

Figure 6. Episode 2: Day 1
Illustration 7

Figure 7. Episode 2: Day 3: Steroids started

Illustration 8

Figure 8. Episode 2: Day 16 (14 days of steroid)
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