Idiopathic Bilateral Optic Neuritis

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ABSTRACT

Idiopathic bilateral optic neuritis in adult has been reported very rarely. The objective of this report is to present a case of idiopathic bilateral optic neuritis in adult and treatment responses. A nineteen year old female presented with bilateral optic neuritis. It was characterized by decreased visual acuity, painful ocular motility and sluggish pupillary reaction with Relative Afferent Pupillary Defect (RAPD) in left eye, hyperemic and generalized optic disc swelling and central scotoma in Humphrey visual field of both eyes. MRI showed diffuse thickening and irregularly outlined optic nerves of both eyes. Idiopathic bilateral optic neuritis in adults is a rare presentation. Prompt treatment with optic neuritis treatment trial (ONTT) improved the visual outcome.

KEY WORDS

Bilateral optic neuritis, Optic neuritis treatment trial, Visual field

INTRODUCTION

Optic neuritis (ON) is an inflammation of the optic nerve and one of the most common manifestations of central nervous system involvement caused by various etiologies. Most common causes of optic neuritis include demyelinating diseases, autoimmune diseases, inflammatory diseases, infections and vaccinations.[1-5] The cardinal signs of ON are decreased visual acuity, a central visual field defect, dyschromatopsia, and a relative afferent pupillary defect.[2-5]

Bilateral optic neuritis is usually thought to affect children, following viral syndrome, and is not typically associated with multiple sclerosis. In contrast, in adults simultaneous bilateral acute optic neuritis has been considered rare particularly in individuals without known systemic inflammatory or autoimmune disorders. Adult onset optic neuritis is typically unilateral and is commonly linked to multiple sclerosis.[6]

Here we present a rare case of idiopathic bilateral optic neuritis in adult with pre and post treatment findings.

CASE REPORT

A nineteen year old female presented to the emergency department of Nepal Medical College Teaching Hospital with a sudden loss of vision in both eyes of 10 days' duration that was associated with pain on ocular movement especially on dextroversion and levoversion. She also had severe headache on the frontal and parietal regions with 2-3 episodes of vomiting. But there was no history of loss of consciousness and rise in temperature. There was no similar episode in the past. There was no history of any drug use for systemic diseases.

On examination, the best corrected visual acuity (BCVA) was finger counting at 1 ft. in the right eye and hand movement in the left eye. Extraocular eye movements were normal but were painful on dextroversion and levoversion. The pupillary reaction was sluggish in both eyes, with RAPD grade II in the left eye. The cranial nerve examination was normal. A slit-lamp examination of the anterior segment was also normal in both eyes. The posterior segment examination showed bilateral hyperemic and generalized optic disc swelling without hemorrhages and exudates. Both eyes had a normal macula. Intraocular pressure was 10mm Hg in both eyes. Lhertmitte sign was also negative.

A magnetic resonance imaging (MRI) of the brain and orbit revealed, bilateral optic nerves were thickened and irregularly outlined and showed diffuse hyper intensity (Left > Right). Maximum diameter of right optic nerve head was 5.6x5.3 mm and that of left optic nerve head was 6.5x5.6 mm (fig. 1). No intracranial evidence of demyelination was detected (fig. 2). The erythrocyte sedimentation rate (ESR) was mildly elevated to 25 mm/h. Other blood investigations, including complete blood count (CBC), Random blood sugar (RBS), antinuclear antibody (ANA) test, hepatitis B surface antigen (HBsAg), Anti Hepatitis C Virus (HCV), C-reactive protein (CRP) were normal. Baseline chest X ray (CXR) was also normal (fig. 3).

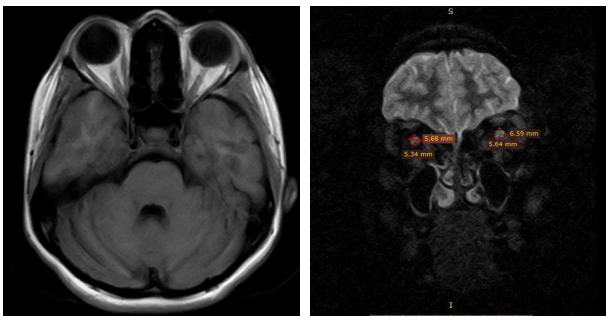

Figure 1. MRI of the orbits showing bilateral optic nerves thickened and irregularly outlined.

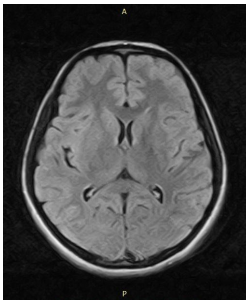

Figure 2. Showing normal MRI of brain with no intracranial demyelination.

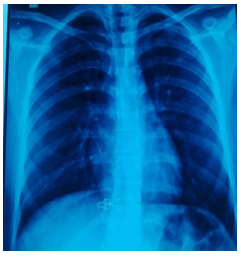

Figure 3. Normal chest X-ray.

A diagnosis of idiopathic bilateral optic neuritis, more severe in the left eye, was made. After the patient was treated with intravenous methylprednisolone 1 gm daily for 3 days, minimal improvement in visual acuity was detected. The BCVA had improved to 3/60 in the right eye and 2/60 in the left eye. Only the nasal disc margins were blurred in both eyes. Visual field test was done by Humphrey Visual Filed Analyzer (HFA) 24-2 which showed central scotoma in both eyes (fig. 4). Color vision with Ishihara plates was normal in both eyes. Then the patient was discharged on oral steroid 50 mg once a day along with oral antacid for 11 days and followed by gradual dose reduction. On the seventh day, the BCVA had further improved to 6/6 in right eye and 6/9 in left eye with a completely normal ophthalmic examination. Fundus photograph showed normal optic disc with intact margins (fig. 5), pupillary reaction was normal and no pain on ocular movements. The patient was seen at 6 months follow up and her VA had improved to 6/6 in both eyes. All ophthalmic examinations were normal. Repeat HFA 24-2 was done which was normal in both eyes (fig. 6).

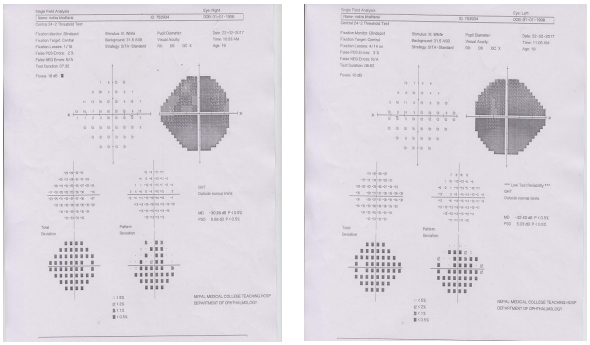

Figure 4. Showing both eyes central scotoma in HFA 24-2.

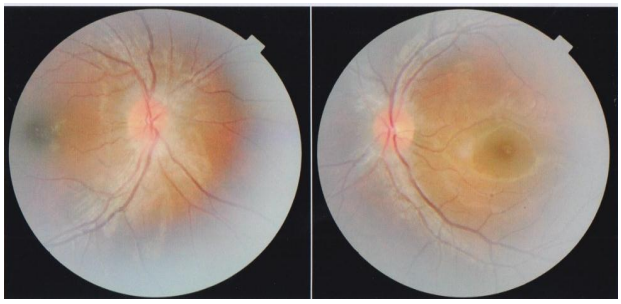

Figure 5. Fundus photograph showing normal BE fundus after treatment.

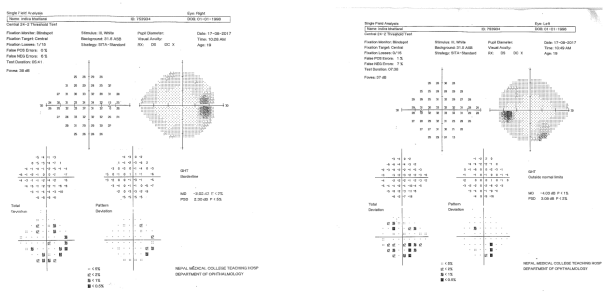

Figure 6. Showing normal HFA 24-2 on follow up.

DISCUSSION

Optic neuritis is an inflammatory, demyelinating condition that causes acute, usually monocular, visual loss. It is highly associated with multiple sclerosis (MS). Optic neuritis is the presenting feature of MS in 15 to 20 percent of patients and occurs in 50 percent at some time during the course of their illness.[7-10]

It may also be caused by other autoimmune diseases such as sarcoidosis, systemic lupus erythematosus, Sjogren's syndrome or Behçet's disease. Infectious ON may be caused by herpes Zoster, Lyme disease, syphilis, tuberculosis or toxoplasmosis. Many other conditions such as tumors or ischemic diseases can also cause an optic neuropathy and clinically mimic idiopathic inflammatory ON.[11]

Optic neuritis is usually monocular in its clinical presentation. In about 10 percent of cases, symptoms occur in both eyes, either simultaneously or in rapid succession.[12] Bilateral optic neuritis is more common in children younger than 12 to 15 years old but is uncommon in adults.[12]

But here we present a case of bilateral optic neuritis in 19 years old female without any features of demyelination or autoimmune or infectious diseases. In our case, she presented with sudden loss of vision in both eyes of 10 days' duration. No systemic diseases were present. Her MRI of orbit and brain revealed diffuse and irregularly thickening of both optic nerves without evidence of intracranial demyelination. Her blood investigations including CBC, RBS, ANA, CRP, HbsAg, anti HCV, CXR were within normal limit.

The pathogenesis of optic neuritis is not well understood. It is likely due to some inflammatory process which leads to delayed type IV hypersensitivity reaction induced by released cytokines and other inflammatory mediators from activated peripheral T-cells which can cross the blood brain barrier and cause destruction of myelin, neural cell death and axonal degeneration. Latest technologies such as optical coherence tomography (OCT) suggest involvement of axons (gray matter) in addition to myelin sheath (white matter) in this process.[13-16]

The classic triad for diagnosis of ON is visual loss, periocular pain and dyschromatopsia.[13,14, 17]

Eye pain occurred in 92 percent of patients in the ONTT and often worsened with eye movement.[18] Abnormal color vision by Ishihara plates was found in 88 percent of involved eyes in the ONTT, this increased to 94 percent with the more sensitive Farnsworth-Munsell 100 hue test.[18]

Our patient also presented to emergency with sudden vision loss in both eyes. Her VA was finger counting at 1 ft in the right eye and hand movement in the left eye. She also had painful ocular movements. Pain was more on dextroversion and levoversion. Since the visual acuity was very poor, colour vision test was not possible to perform at the time of presentation. But it was normal after treated with IV methylprednisolone.

A relative afferent pupillary defect remains in approximately one-fourth of patients two years after presentation.[19] According to ONTT, RAPD may disappear when visual recovery is full.[13] Similar to ONTT, our patient had grade II RAPD in her left eye which disappeared after visual recovery.

ON is usually accompanied by impaired central or paracentral vision.[17] Visual field defects usually resolve, about 56 percent had normalized at one year and 73 percent had normalized at 10 years in the ONTT.[20,21] In our case also, HFA 24-2 showed central scotoma in both eyes which was normalized at 6 months follow up.

About seventy two percent of affected eyes recover central visual acuity of 20/20 or better, and 92 % recover to 20/40 or better.[22,23] Eye pain usually resolves within a week.[24,25] Our patient also recovered visual acuity of 20/20 after treatment and painful ocular movement was resolved on seventh day.

Idiopathic bilateral optic neuritis in adult is a rare presentation and it responded well to ONTT in our case.

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