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DIAGNOSIS AND MANAGEMENT OF RECURRENT OPTIC NEURITIS: A CASE REPORT

Mohammad Haikal Bakry[1], Lukisiari Agustini[1], Gatot Suhartono[1]
[1]Neuro-ophthalmology Division, Department of Ophthalmology, Faculty of Medicine Universitas Airlangga – Dr. Soetomo General Hospital, Surabaya, Indonesia
Correspondence: Lukisiari Agustini, dr.lukisiari@yahoo.com

Background
The phrase "optic neuritis" describes inflammation of the optic nerve caused by diverse factors. This condition can occur by itself or be connected to inflammatory or infectious conditions affecting the nervous system, as well as local or systemic inflammatory diseases.
Case Presentation
A 27-year-old woman came with complaints of blurred vision in both eyes since the last 2 weeks. The patient also feels that his vision is getting narrower. Her visual acuity of right eye is 2/60 PH 5/30, and left eye 5/40 PH 5/10. Color vision examination with Ishihara was 11/14 on both eyes. Anterior segment showed rounded pupils, isochoric, normal light reflex, with no relative afferent pupillary defect. Ocular motility was normal but the patient complains of a little pain within movement. Posterior segment of right eye showed hyperemic and blurred margin in temporal region. Left eye posterior segment was showed hyperemia on the optic disc. Patient is receiving methylprednisolone therapy 1x8mg which has been given for 1 week and has been reduced to 1x4mg, neurotropic 2x1 and folic acid 1x1.
Conclusion
A wide range of inflammatory and viral diseases can cause optic neuritis. Consequently, relevant fundus, neuroimaging, and laboratory evidence supporting different infectious or inflammatory etiologies need to be known by doctors.
Keyword
Demyelinating, Optic nerve-head swelling, Optic Neuritis, Systemic lupus erythematosus

BACKGROUND

Optic neuropathies can be categorized as posterior (beginning with a normal-appearing ONH) or anterior (having ONH edema). It is advisable to take into account potential optic neuropathy mechanisms while assessing a patient who has an optic neuropathy. Finding the underlying cause of the optic neuropathy can be greatly aided by clinical features such as the patient's age, the mode of start, laterality, presence of pain, color vision, type of visual field deficits, appearance of the optic nerve, and the results of orbital magnetic resonance imaging (MRI).[1]

Any cause of optic nerve irritation is referred to as optic neuritis. It could be isolated or connected to a systemic or local inflammatory disease, an infectious or CNS inflammatory disorder. In cases of retrobulbar neuritis, the optic nerve head remains unaffected, so the optic disc initially appears normal. The most prevalent kind in adults, it is often linked to multiple sclerosis (MS). The hallmarks of Papillitis are optic disc oedema and hyperemia, which can be accompanied by peripapillary flame-shaped hemorrhages. It is possible to see cells in the posterior vitreous. The most prevalent kind of optic neuritis in children, while it can also strike

adults, is papillitis. The symptoms of neuroretinitis include papillitis, a macular star figure, and inflammation of the retinal nerve fiber layer. It is the least prevalent kind and only sometimes shows signs of demyelination.[1,2]

Demyelination is the most common cause based on etiology. Following a vaccination or viral infection, an individual can become parainfectious and contagious. This can be due to a sinus infection or associated with conditions such as herpes zoster, syphilis, cryptococcal meningitis, Lyme disease, and cat scratch fever. Non-infectious causes include sarcoidosis and systemic autoimmune disorders like polyarteritis nodosa, systemic lupus erythematosus, and various types of vasculitis.[3]

The majority of patients with isolated optic neuritis are female (77%) and have a mean age of 32 years. The condition usually manifests as subacute monocular vision loss that takes several days to resolve. In 92% of instances, periorbital pain—especially while moving the eyes—occurs and frequently comes on before vision loss. Just one-third of individuals have ONH edema, and 65% of instances are retrobulbar. An RAPD is also present unless the optic neuritis is bilateral and symmetric. While any pattern of visual field loss may manifest, perimetry testing most frequently reveals a central depression or generalized reduction of sensitivity (48%). Dyschromatopsia is almost widespread and frequently disproportionate to the decrease of visual acuity, especially for red-green. Within a month, ocular neuritis improves on its own in the great majority of instances.[1]

Although they don't have to be done on a regular basis, further hematologic, serologic, or other testing can be helpful in unusual situations. These studies could look at the following: Serum and CSF rapid plasma reagen and fluorescent treponemal antibody absorption testing for syphilis; Serum testing for Bartonella infection; Serum testing for Lyme disease (if endemic); Chest x-ray or chest computed tomography (CT); Gallium scan or full-body positron emission tomography (PET); Serum angiotensin-converting enzyme testing for sarcoidosis; Serum antinuclear antibody testing and anti-DNA antibody testing for systemic lupus erythematosus or vasculitis; Antineutrophil cytoplasmic antibodies (ANCA) for granulomatosis with polyangiitis (Wegener granulomatosis); Serum or CSF aquaporin-4 immunoglobulin G (AQP4-IgG) antibody testing and spinal, MRI for neuromyelitis optica spectrum disorder (NMOSD); Genetic testing for Leber hereditary optic neuropathy For compressive or infiltrative illnesses, brain and orbit MRI with gadolinium contrast, and lumbar puncture with cytology for a meningeal process are recommended.[1]

Given their respective roles in the short- and long-term evaluation of patients' neurological and visual health, ophthalmologists and neurologists should be conversant with the acute and long-term management of patients with optic neuritis as well as their prognosis.[4]

CASE PRESENTATION

A 27-year-old woman came with complaints of blurred vision in both eyes, the complaints have been felt since the last 2 weeks. The patient also feels that his vision is getting narrower. There was no complaint of red eye, double vision, headache, vomiting or nausea.

From the general examination, patient revealed normal vital sign. Her visual acuity of right eye is 2/60 PH 5/30, and left eye 5/40 PH 5/10. Color vision examination with Ishihara was 11/14 on both eyes. Intraocular pressure of the right eye was 15 mmHg, left eye 17 mmHg. Anterior segment (Fig.1) showed rounded pupils, isochoric 3 mm diameter, normal light reflex, with no relative afferent pupillary defect. Ocular motility on the both eye was normal but the patient complains of a little pain within movement.

Posterior segment of right eye showed hyperemic and blurred margin in temporal region. Left eye posterior segment was showed hyperemia on the optic disc. (Figure 2)

Optical coherence tomography shown an increase of RNFL thickness in right eyes (Fig. 3). the patient is a patient who routinely controls treatment and is diagnosed with Optic neuritis. The patient's complaint first appeared since 2017 with complaints of sudden blurred vision in both eyes. The patient also complains that he often feels sore in the joints of his body and there are also red rashes on the body. From the results of the laboratory examination, the hemoglobin value was 13.5 g/dl, leukocytes 3300, and protein was found on urine examination. From the results of the ANA test, the value was 13.09, C3 153 mg/L, C4 50.8 mg/dl and normal results were obtained on ANA profile examination (Figure 4).

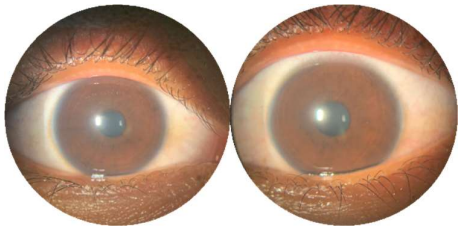
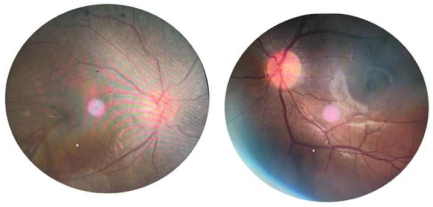


Figure 1. Anterior segment within normal limit



A **B**

Figure 2. (A) Right eye showed hyperemic and blurredmargin in temporal region papil (B) Left eye posterior segment was showed hyperemia on the optic disc

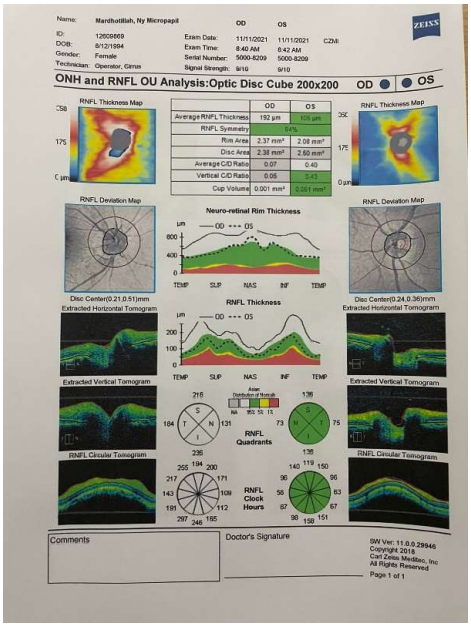


Figure 3. OCT showed increase RNFL thickness of right eye

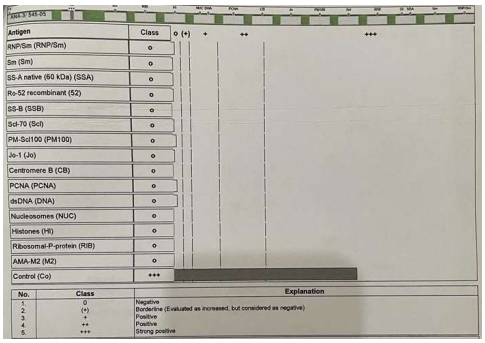


Figure 4. ANA Profile testshowed normal result.

On HFA examination, the visual field is getting narrower than when it first came in 2017 while on examination of the left eye the results were normal. (Figure 5) From the results of the MRI examination, the patient did not find any hypointense/hyperintense lesions in the brain parenchyme, which with contrast administration did not show abnormal contrast enhancement. (Figure 6)

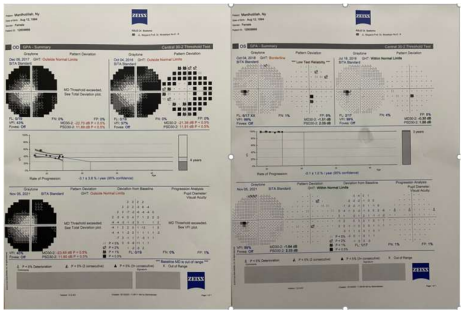


Figure 5. The HFA examination showed a narrowing of the visual field in the right eye and normal results in the left eye

The patient was given methylprednisolone therapy and tapering off was done gradually and patient was also consulted to the internal medicine department to look for possible causes of the underlying systemic disease. From the results of consultations with the department of internal medicine, the patient was diagnosed with SLE and given therapy with Azathioprine 2x 50mg, Bisoprolol 2.5mg, Atorvastatin 20mg, Lisinopril 10mg, Vitamin D, acetylsalicylic acid and Lanzoprazole. Then from the results of a consultation with the neurology department, it was found from the results of the ERG examination that there were demyelinating lesions in the bilateral visual pathway.

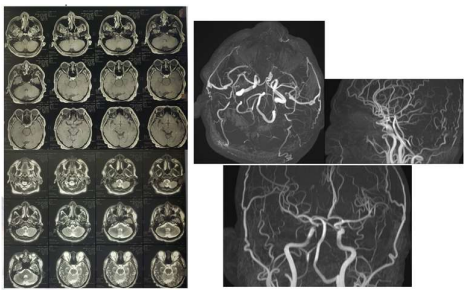


Figure 6. From the MRI examination, there are no hypointense/hyperintense lesions in the brain parenchyme, which with contrast administration did not show abnormal contrast enhancement

In January 2021 the patient again complained of blurred vision and was

again given methylprednisolone 2x32mg and reduced gradually until the last month of July methylprednisolone was no longer used with the last vision in both eyes reaching 5/5. On the funduscopic, there is also a sign of bone spiculae in the peripheral area (Figure 7) and has been consulted to the retinal division and given retivit therapy.

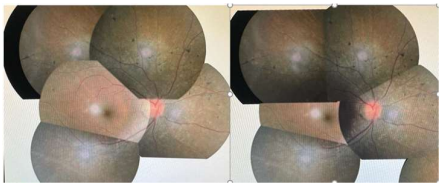


Figure 7. there is a sign of bone spiculae in the peripheral area

The patient is receiving methylprednisolone therapy 1x8mg which has been given for 1 week and has been reduced to 1x4mg, the patient is also receiving neurotropic 2x1 and folic acid 1x1. The patient vision is VOD 3/60 and VOS 5/40 and can reach 5/5 with eyeglass correction and from the results of the funduscopic examination, it was found that the papil in the right eye had firmer borders and normal color in both papil (Figure 8).

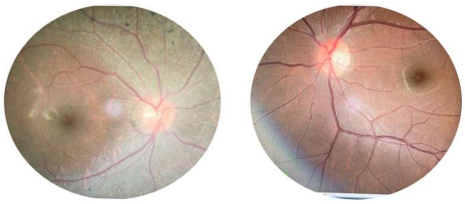


Figure 8. The papil in the right eye had sharp borders and normal color in both papil

DISCUSSION

A 27-year-old woman came with complaints of blurred vision in both eyes, the complaints have been felt since the last 2 weeks. The patient also feels that his vision is getting narrower. There was no complaint of red eye, double vision, headache,vomiting or nausea. Her visual acuity of right eye is 2/60 PH 5/30, and left eye 5/40 PH 5/10. Color vision examination with Ishihara was 11/14 on both eyes. Intraocular pressure of the right eye was 15 mmHg, left eye 17 mmHg. Anterior segment showed rounded pupils, isochoric 3 mm diameter, normal light reflex, with no relative afferent pupillary defect. Ocular motility on the both eye was normal but the patient complains of a little pain within movement.

Patient demographics are consistent with the literature, which states that patients with optic neuritis are usually younger, that the condition peaks in the third and fourth decade of life, and that women are more likely than males to be affected. 77% of the patients in the ONTT were female, 85% were Caucasian, and the average age was 32.[5]

The loss of vision normally occurs quickly, sometimes within hours or even days. For as long as one or two weeks, vision loss may worsen. With optic neuritis, visual loss is always accompanied by reduced colour vision. A common complaint from patients is a darkening or loss of colour vibrancy. Most patients have characteristic pain, which usually appears a few days before visual loss (92% in the ONTT).5. Globe discomfort and pain that gets worse when you move your eyes are common. Although the precise cause of the discomfort is uncertain, it is most likely the result of the eye muscle origin from the annulus of Zinn pulling on the dura, which is in contact with the irritated nerve.5, 6 Pain lasting longer than 7 days should be regarded as atypical and should trigger an investigation for other causes of pain and optic neuropathy, such as a systemic inflammatory condition, scleritis, malignant glioma, orbital inflammatory syndrome, aneurysm, or a sinus mucocele. This is because the pain usually goes away after 3–5 days. 5 Patients with optic neuritis typically describe phosphones, or flashing

lights, in a variety of shapes, such as lights, sparkles, and moving squares. Loud noises or eye movements may make them worse. Thirty percent of the patients in the ONTT had these symptoms. These symptoms may appear concurrently with an abrupt loss of vision and continue for several months following recovery. These are not specific to inflammatory ocular neuropathies; they can also happen when compressive lesions are present. Phosphenes should also be taken into consideration in the event of concurrent retinal illness, such as neuroretinitis. [5]

Patients with optic neuritis typically report phosphones, or flashing lights, as lights, sparkles, or moving squares, among other variations. Loud noises or eye movements may make them worse. Thirty percent of the patients in the ONTT had these symptoms. These symptoms may appear concurrently with an abrupt loss of vision and continue for several months following recovery. These are not specific to inflammatory ocular neuropathies; they can also happen when compressive lesions are present. Phosphenes should also be taken into consideration in the event of concurrent retinal illness, such as neuroretinitis.[5]

The short-term visual impairment linked to an increase in body temperature is known as Uhthoff's symptom. While hot food or cooking might cause the symptom, physical activity or a hot shower are the most common causes. Visibility loss usually manifests as blurring, greying, or diminished colour perception and starts a few minutes after heat exposure. After a few minutes to an hour, vision recovers to normal with no lasting impairments. This symptom has been reported in cases with hereditary, toxic, compressive, 5 and sarcoid7 optic neuropathies and is not specific to people with optic neuritis.[5]

Reduced visual acuity in patients usually ranges from practically normal to NLP. 10% of patients in the ONTT5 had 20/20 vision, 25% had acuities between 20/25 and 20/40, 29% had acuities between 20/50 and 20/190, and 36% had acuities between 20/200 and NLP. When testing with pseudoisochromatic colour plates and observing asymmetry between the eyes, dyschromatopsia is typically easily diagnosed. When compared to age-matched controls, 98% of patients in the ONTT had significantly impaired contrast sensitivity.5. It has been discovered that the most sensitive measure of visual impairment in both acute and recovered cases of optic neuritis is contrast sensitivity.[5]

Patients Posterior segment of right eye showed hyperemic and blurredmargin in temporal region. Left eye posterior segment was showed hyperemia on the optic disc. Optical coherence tomography shown an increase of RNFL thickness in right eyes. On HFA examination, the visual field is getting narrower than when it first came in 2017 while on examination of the left eye the results were normal.

Visual field loss is almost always present in patients with optic neuritis. Central scotomas are the most prevalent visual field abnormalities in optic neuritis on manual kinetic perimetry and tangent screen testing. Using static threshold perimetry as the gold standard, the ONTT discovered that 55% of patients had local abnormalities and 45% had diffuse field loss.5,8 Of these localised visual field impairments, "three-quadrant defects" (14%) and altitudinal defects (20%) were the most prevalent. In sixteen percent of cases, there were combined, central, and centrocecal malformations. The examiner is unable to differentiate optic neuritis from other types of optic nerve dysfunction using these patterns of visual field loss, which collectively cover the entire range of "optic nerve"-type field defects.[5]

Approximately one-third of individuals had modest optic nerve-head enlargement on the fundus examination. Compared to

papilledema, this feature is typically much less pronounced, less sectoral, and unrelated to the capillary dilatation and splinter haemorrhages that are typical of anterior ischemic optic neuropathy.[5] Ocular imaging investigations conducted after the ONTT have shown that optic disc edoema is probably more common than what can be determined only by ophthalmoscopy and clinical examination. OCT has detected a thickening of the peripheral RNFL.OCT can therefore be utilised clinically in acute optic neuritis to evaluate for macular abnormalities that could otherwise account for visual loss or for subclinical optic disc enlargement.[11,12]

Phlebitis, vitreous cells, and retinal exudates occur in less than 5% of patients.[5] Retinal anomalies including periphlebitis and nerve fibre layer atrophy, when present, can indicate the severity of demyelinating illness.[13,14] Since extensive vitritis and retinal vein sheathing are uncommon in optic neuritis, sarcoidosis and syphilis should be explored as alternative diagnosis in this situation. (1) NLP vision; (2) optic disc or retinal haemorrhages; (3) significant optic disc edema; (4) macular exudates; (5) lack of discomfort; (6) uveitis; and (7) bilateral visual loss are among the unusual signs of acute demyelinating optic neuritis. Most adult occurrences of bilateral simultaneous optic neuritis are still demyelinating, although it is uncommon and necessitates a thorough evaluation including testing for neuromyelitis optica (NMO).[15,16]

OCT is now extensively acknowledged for its clinical and structural outcomes documentation in research and trials, as well as its function in treating patients with optic neuritis.[11,17] Acute inflammation of the optic nerve and subsequent axon loss are hallmarks of the pathogenesis of optic neuritis. Thinning of the RNFL occurs over time due to degeneration of retinal ganglion cell axons, which are the axons that will eventually form the optic nerve.[5,18]

OCT investigations have shown a potential threshold of RNFL thinning beyond which visual function may remain considerably compromised, and they have validated reductions in RNFL thickness in eyes with a history of optic neuritis.[19,20] It has also been demonstrated that eyes with a history of optic neuritis in the context of MS had thinner GCLs and RNFLs. The GCL layer experiences the most severe thinning, accounting for most of the RNFL and GCL thinning that occurs in the first two months following the optic neuritis event.[21] The degree of axonal and neuronal loss is highest in MS eyes with a history of acute optic neuritis as compared to disease-free control eyes and eyes without a history of the condition.[11, 17, 22], and [23] These findings highlight the specific unmet need for novel reparative and neuroprotective medicines to lessen the degree of axonal and neuronal damage in the visual pathway in cases with optic neuritis.[11,17,22,23]

From the results of the MRI examination, the patient did not find any hypointense/ hyperintense lesions in the brain parenchyme, which with contrast administration did not show abnormal contrast enhancement. Then from the results of a consultation with the neurology department, it was found from the results of the EMG VEP examination that there were demyelinating lesions in the bilateral visual pathway.

Patients with optic neuritis are known to exhibit well-known MRI abnormalities consistent with demyelinating lesions in the white matter. The blood–brain barrier is compromised, leading to abnormalities and augmentation. In the ONTT, white matter lesions were clinically quiet in 59% of individuals with a prior neurologic history that was normal. Lesions >3 mm in diameter and T2 hyperintense in the periventricular white matter, subcortical white matter, and pons are the most

common findings. These results, which have been observed in "normal" patients as well as those with various systemic inflammatory diseases, may not be specific. Using STIR sequences with fat-suppressed orbital views and an orbital surface coil, over 90% of patients with optic neuritis exhibit visible lesions in the afflicted optic nerve.[24] Low vision could be associated with optic.[5,25]

When there is unexplained vision loss, visual-evoked potentials (VEPs) should not be utilized to diagnose optic neuritis; rather, they should only be used as a continuation of the neuro-ophthalmic evaluation. While variations in the visual equivalent of pain (VEP) can offer impartial data to support the diagnosis of optic neuritis, the diagnosis is always clinical. The degree of acuity loss is typically correlated with the amplitude variations in the occipital pattern VEPs, which exhibit noticeable latency alterations. Optic neuritis is typically characterized by a significantly prolonged latency (p100) with a comparatively preserved amplitude.[5]. In one study, the mVEP was aberrant in 97.3% of eyes with optic neuritis; abnormalities included increased latency in 68.4% of patients and decreased amplitude in 96% of patients.[26]

The patient's complaint first appeared since 2017 with complaints of sudden blurred vision in both eyes. The patient also complains that he often feels sore in the joints of his body and there are also red rashes on the body. From the results of the laboratory examination, the hemoglobin value was 13.5 g/dl, leukocytes 3300, and protein was found on urine examination. From the results of the ANA test, the value was 13.09, C3 153 mg/L, C4 50.8 mg/dl and normal results were obtained on ANA profile examination. HBsAg and Anti-HCV examination showed non-reactive results. The patient was consulted to the internal medicine department to look for possible causes of the underlying systemic disease. From the results of consultations with the department of internal medicine, the patient was diagnosed with SLE and given therapy with Imuran 2x 50mg, Concor 2.5mg, Atorvastatin 20mg, Lisinopril 10mg, Cavid d3, Aptor and Lanzoprazole.

Systemic or neurologic immune diseases may be present in the background when acute optic neuritis occurs. Optic neuritis can occasionally be the initial sign of a systemic immunological process, such as paraneoplastic illness, systemic lupus erythematosus, and Sjögren syndrome..[27]

An uncommon sign of systemic lupus erythematosus (SLE) is isolated optic neuritis. Just 16 patients (4.3%) experienced CNS events in a 3-year prospective study of 370 SLE patients without a history of neurologic involvement; two of the 23 occurrences (8.7%) were optic neuritis. In patients with SLE, optic neuritis is usually severe, and recovery is frequently not complete. In a small group of SLE patients with optic neuritis, over 40% had a recovery acuity of 20/200 or poorer, and 37.5% experienced a repeat episode. High-dose corticosteroids can help some patients with acute SLE optic neuritis; the effects of plasma exchange are unknown. It has been observed that steroid-sparing immunosuppressants, such as cyclophosphamide, azathioprine, methotrexate, and cyclosporine, can effectively control patients with recurrent illness. [27]

Currently the patient is receiving methylprednisolone therapy 1x8mg which had been given for 1 week and has been reduced to 1x4mg, the patient is also receiving neurotropic 2x1 and folic acid 1x1. The patient's current vision is VOD 3/60 and VOS 5/40 and can reach 5/5 with eyeglass correction and from the results of the funduscopic examination, it was found that the papil in the right eye had firmer

borders and normal color in both papil.

The Optic Neuritis Treatment Trial (ONTT) showed that corticosteroid therapy for optic neuritis did not improve vision over the long term; however, recovery could be accelerated by 1-2 weeks with intravenous methylprednisolone (250 mg every 6 hours for 3 days) and oral prednisone (1 mg/kg/day) for 11 days (with a quick taper of 4 days).[1] Although intravenous methylprednisolone in the ONTT accelerated the rate of visual recovery, all treatment groups performed comparably after a year. Oral prednisone was linked to a higher incidence of recurrent ocular neuritis in the ONTT, especially throughout the 2- and 5-year follow-up periods.[5]. Although these trends were not as noticeable as those observed earlier in the trial follow-up, the incidence of optic neuritis recurrence at the 10-year follow-up was nevertheless higher in the oral prednisone group (44%) than in the placebo (31%, p = 0.07) and methylprednisolone (29%, p = 0.03).28 At ten years, a quarter of the ONTT eyes had visual acuity of at least 20/20, and sixty-six percent of patients had both eyes with greater than 20/20 high-contrast visual acuity.

Patients with more severe visual field loss, more substantial losses in contrast sensitivity, and initial poor acuity (20/400 to NLP) have a worse prognosis, according to the ONTT[30] and other reports. Additionally, those who at one month had a visual acuity of 20/50 or below were likely to have moderate to severe persistent visual impairment.[30] Still, there is a lot of variation in the quality of the "20/20" eyesight. The forms of visual impairment and symptomatology that many patients report after recovering from optic neuritis are not fully captured by recovery to 20/20 vision.[31]

CONCLUSION

A case presents a 27-year-old woman came with complaints of blurred vision in both eyes, the complaints have been felt since the last 2 weeks. The patient also feels that his vision is getting narrower. There was no complaint of red eye, double vision, headache,vomiting or nausea.

From the general examination, patient revealed normal vital sign. Her visual acuity of right eye is 2/60 PH 5/30, and left eye 5/40 PH 5/10. Color vision examination with Ishihara was 11/14 on both eyes. Intraocular pressure of the right eye was 15 mmHg, left eye 17 mmHg. Anterior segment showed rounded pupils, isochoric 3 mm diameter, normal light reflex, with no relative afferent pupillary defect. Ocular motility on the both eye was normal but the patient complains of a little pain within movement. Posterior segment of right eye showed hyperemic and blurred margin in temporal region. Left eye posterior segment was showed hyperemia on the optic disc.

Currently the patient is receiving methylprednisolone therapy 1x8mg which has been given for 1 week and has been reduced to 1x4mg, the patient is also receiving neurotropic 2x1 and folic acid 1x1. The patient's current vision is VOD 3/60 and VOS 5/40 and can reach 5/5 with eyeglass correction And from the results of the funduscopic examination, it was found that the papil in the right eye had firmer borders and normal color in both papil.

Treatment choices may need to be made prior to a conclusive serologic, molecular, or histologic diagnosis in order to achieve the best possible visual recovery from acute optic neuritis. Consequently, fundus, neuroimaging, and laboratory evidence supporting different inflammatory or infectious etiologies need to be known by doctors.

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