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TYPICAL OPTIC NEURITIS IN A PATIENT WITH MULTIPLE SCLEROSIS: A CASE REPORT

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ABSTRACT
Background: Optic Neuritis (ON) is often associated with Multiple Sclerosis (MS) and is the first manifestation of MS that causes visual loss in patients. To prevent more severe damage to visual function in MS patients with optic neuritis, it is critical to be aware of the signs and symptoms as well as the appropriate treatment.
Case Presentation: A 25-year-old woman with chief complaint of a sudden decrease in visual acuity in the left eye, accompanied by pain when moving her eyes. Visual acuity is Hand Movement (HM) in the left eye with RAPD grade 2, on funduscopy examination shows hyperemic round optic nerve head and the retina were within normal limits. *Ishihara's* examination obtained demoplate, contrast sensitivity with *Pelli-Robson* obtained 0.75. MRI images showed multiple lesions in the right and left lateral periventricular regions and Dawson's Finger images that support an image of MS.
Conclusion: The patient was given high doses of methylprednisolone (4 x 250 mg IV for 12 cycles), then tapered off with 1 mg/kgBW/day of methylprednisolone. Six weeks after treatment, the visual acuity of the left eye improved, becoming 6/6 with improvements in other visual functions. Typical optic neuritis has a distinctive clinical presentation and is associated with the presence of MS. A correct diagnosis and prompt treatment will improve the visual function.
Keywords: optic neuritis, multiple sclerosis, dawson's finger

BACKGROUND

Optic Neuritis (ON) is an acute inflammatory disorder characterized by demyelination of one or both optic nerves, divided into typical and atypical optic neuritis. The most common form of optic neuritis, typical optic neuritis, is associated with multiple sclerosis (MS), atypical optic neuritis is associated with other conditions.[1]

Optic Neuritis is the initial sign of multiple sclerosis and the common causes of visual impairment in MS patients. It is estimated that 15-20% of all MS cases are affected ON, 75% of these patients experiencing at least one episode in their lifetime.[5–7]

The incidence of ON in Europe is 5 cases per 100,000 people per year.[2] In Asia, optic nerve involvement during the beginning or progression of MS is more prevalent. 56% of MS patients in Taiwan, 38.2% of MS patients in China, and 20% of MS patients in Europe were reported to have ON as their initial symptom.[3] In a cohort study, the Asian Collaborative Longitudinal Optic Neuritis Epidemiology (ACLONE) found that Chinese (46.81%) and Korean (40.4%) suffer from unilateral ON.[4]

Patients with ON and its relationship with MS can be diagnosed using typical clinical symptoms, an ophthalmological examination, and supportive diagnoses. This case report describes the symptoms, diagnosis, and treatment of a patient with typical optic neuritis accompanied with multiple sclerosis at Bali Mandara Eye Hospital.

CASE PRESENTATION

A 25-year-old woman, experienced a sudden loss of vision in her left eye two days before, along with pain while moving the eye. Vision loss was getting worse when patient

doing exercise. Headaches, flu-like symptoms, limb weakness, and other neurological impairments were denied by the patient.

Visual acuity in the left eye was hand movement with Relative Afferent Pupillary Defect (RAPD) on pupillary examination. From fundoscopic examination showed round hyperemic optic nerve head with retina within normal limits. Color function examination and contrast sensitivity test showed demoplate and 0.75% respectively, and examination of the Amsler grid showed a central scotoma without metamorphopsia. The Visual acuity in the right eye 6/9 ph 6/6 with anterior and posterior segments, and other visual functions were within normal limits. Humphrey Visual Field Examination (HVF 30-2) was found general depression in the left eye **(fig.1).** Patient undergone complete blood count test, infection marker and other metabolism test and was found within normal limit.

The patient was diagnosed with typical optic neuritis suspected associated with autoimmune process in the left eye, and was given high doses of methyl prednisolone of 4 x 250 mg IV for 12 cycles and tapering off methyl prednisolone of 1 mg/kgBW/day (48 mg/day) after 12 cycles of high dose methylprednisolone every week. Visual acuity was improved to 1/60 after 12 cycles of IV methyl prednisolone. One week after treatment, there was a significant improvement in visual acuity in the left eye, which was 6/60 ph 6/45f. Six weeks after treatment, the visual acuity of the left eye became 6/6, which was accompanied by improvements in other visual functions. Examination of HVF 30-2 six weeks after treatment showed an improvement in visual field defects when compared to the previous control **(fig. 1).** The patient also undergone Optical Coherence Tomography (OCT) at 1 week and 6 weeks after treatment. It was found an improvement in RNFL thickness from week 1 (144 μm) to week 6 after treatment (86 μm) and accompanied by thinning of the RNFL on the temporal side **(fig. 2).**

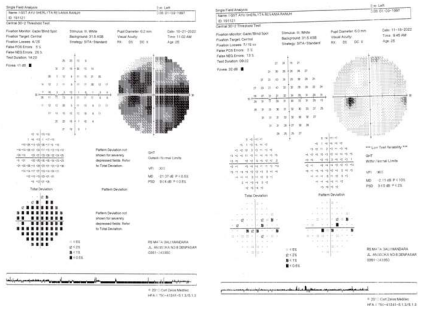

Figure 1. Improved visual field defects on HVF 30-2 examination at 1 week after treatment (A) and 6 weeks after treatment (B)

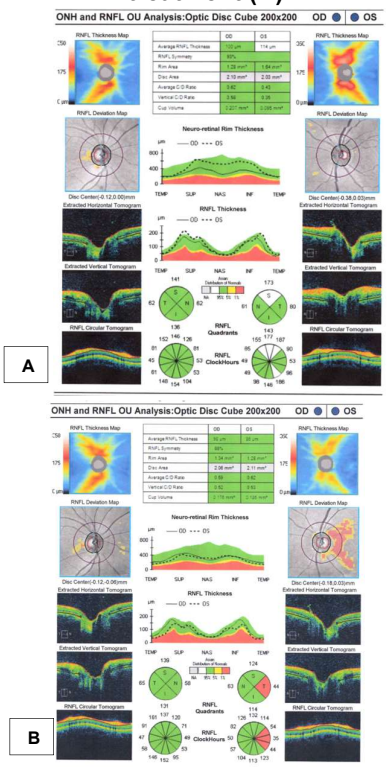

Figure 2. OCT-RNFL A) 1 week after treatment and B) 6 weeks after treatment. There was an improvement in RNFL thickness from week 1 (144 μm) to week 6 after treatment (86 μm) and accompanied by thinning of the RNFL on the temporal side.

Magnetic Resonance Imaging (MRI) examination showed multiple lesions in the right and left lateral periventricular regions and multiple

supratentorial intraaxial lesions, ovoid shape with clear boundaries on the white matter of the right and left frontoparietal lobes, right and left periventricular, right and left pericallosal and forming Dawson's finger images, which support an image of Multiple Sclerosis.

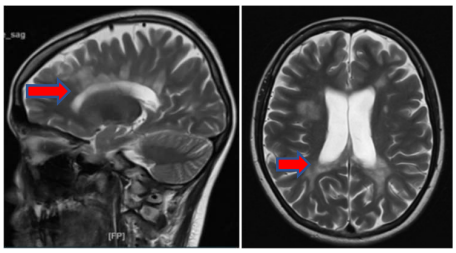

Figure 3. MRI images of the axial and sagittal T2W1 head showed multiple lesions in the left and right lateral periventricular and Dawson's Finger images that supported a Multiple Sclerosis picture

DISCUSSION

Optic Neuritis (ON) is an acute inflammatory disorder characterized by demyelination of one or both optic nerves. The incidence of ON in Europe is 5 cases per 100,000 people per year. ON is associated with MS and could be the first manifestation of MS and the common cause of loss of visual function in patients. It is estimated that 15–20% of total cases of MS suffer from ON and 75% of these patients have one episode of ON in their lifetime.[5–7]

The incidence of ON is more common in women than men and ON associated with demyelinating disease occurs more commonly in women, at 75% compared to 25% in men, which closely matches the epidemiology of MS. ON often occurs in young adults around 20-50 years old, with the average age in Asia is around 36 years.[4]

The most common form of ON is typical optic neuritis, which clinical manifestations of acute or subacute unilateral loss of visual function, ranging from 20/20 to no light perception and accompanied by a Relative Afferent Pupillary Defect (RAPD). Approximately 92%-94% of patients complain of periocular pain, and worsened by movement of the eyeball.[5] These symptoms result from traction of the inflamed optic nerve by the rectus muscles as the eyeball moves, or from inflammation of the meninges or the optic nerve's sheath.[3] In this case, the patient was female, 25 years old and the patient was diagnosed with typical optic neuritis based on the symptoms, which were a sudden decrease in visual acuity that accompanied pain with eye movement. Visual acuity was Hand Movement (HM) with RAPD grade 2.

Additional symptoms may some patients also complain of vertical flashes of light that can be generated by eyeball movement (Moore's lightning streaks), which is the sense of seeing light when no light is entering the eye, Pulfrich effect (a visual stereoillusion that occurs in unilateral ON and is characterized by the erroneous perception of moving objects due to variations in optic nerve conduction), Uhthoff's phenomena (decreased visual function due to increased temperature, hot weather, and exercise).[5] In this case the patient visual acuity was getting worse when the patient doing exersice, might because of the combination of structural and physiological changes within demyelinated axon in central nervous symptoms (CNS) due to elevated body core temperature. [2,3,5,9]

Patients often complain decreased of color vision especially red, and persist for several months after onset. In addition, there are also decreased of contrast sensitivity. In this case, was found decreased of color function and contrast sensitivity. Optic neuritis affects color and contrast vision primarily through inflammation and demyelination of the optic nerve.

Damage to the nerve fibers, particularly in the papillomacular bundle, disrupts the normal transmission of visual signals, including those related to color perception, particulary reduction in their ability to discern red and green colours.[3,4,5,7]

On funduscopic examination found 2/3 cases with normal optic disc, and 1/3 cases with mild-moderate papillitis or swelling of the optic disc. The funduscopic examination of this patient was found round hyperemic optic nerve head. Optic neuritis, characterized by inflammation of the optic nerve, can lead to various changes in the optic nerve head, including hyperemia (increased blood flow and vessel dilation).[2,3,7]

Optic Neuritis does not preferentially affect one visual field location. Instead, the condition is a diffuse process that affects more than one group of axons and therefore causes a diffuse depression of the visual field where central and ceco-central defects are the most common form of visual field defect in ON. The presented patient in this case based on Humphrey Visual Fields, showed cecocentral visual field defect. A cecocentral scotoma is a specific type of visual field defect. It is characterized by a blind spot or area of reduced vision that covers the central and paracentral portions of the visual field. This occurs because optic neuritis often affects the nerve fibers that serve the macular region of the retina, which is responsible for central vision.[2,3,5,10]

The pathogenesis of demyelination in MS results in axonal loss, and thinning of the ganglion cell layer and retinal nerve fiber layer (RNFL) and could be examined by OCT as a non-invasive imaging technique. In this case OCT was used as a quantitative evaluation of RNFL and optic nerve head (ONH) changes in ON after treatment. OCT of the ONH and RNFL was examined 1 week and 6 weeks after treatment, where RNFL thickness reduced after 6 weeks of treatment (114 μm to 86 μm), thinning of RNFL in temporal side, and reducing of ONH swelling. In short term OCT can be used to monitor changes in the RNFL after methylprednisolone treatment, this can be seen as a reduction in swelling of the RNFL. However, over time, particularly in cases of severe optic neuritis or incomplete recovery, thinning of the RNFL might be observed, indicating axonal loss. [2,3,6,9]

Clinically Isolated Syndrome (CIS) refers to a first episode of neurological symptoms caused by inflammation and demyelination in the central nervous system (CNS). Optic neuritis one of the most common form of CIS and might be as first indication of developing MS. The diagnosis of MS following episode of CIS depends on magnetic resonance imaging (MRI) findings. It can detect demyelinating lesions in the brain and spinal cord, which are indicative of MS.[2,5,7,12] In the Optic Neuritis Treatment Trial (ONTT) found patients with the first episode of ON if found without lesions on MRI have a low risk (25%) within 15 years of suffering from MS, whereas if there are one or more lesions on MRI the risk of developing MS becomes 72%.[7,9,10] McDonald's criteria can be used to diagnose MS patients with CIS manifestations, such as optic neuritis. A single MRI examination can diagnose MS, the criteria for Dissemination of lesions in Space (DIS) and in Time (DIT). DIS criteria are defined by one or more lesions in at least 2 of 4 typical areas (periventricular, juxtacortical, infratentorial, and spinal cord). DIT was determined by the presence of asymptomatic gadolinium-enhancing lesions and T2 lesions or new T2 lesions. The presented MRI findings in this case fulfilled DIS criteria, while DIT criteria was not fulfilled because there was not carried follow up MRI examination.

Besides those findings, Dawson's fingers lesion also found in MRI of this patient and associated with MS and result of demyelination process. It appears around the ventricles of the brain, which are detected through MRI.[7,9]

In the Randomized and Controlled Trial (RCT), Optic Neuritis Treatment Trial (ONTT) divided ON treatment into three groups: oral prednisone at a dose of 1 mg/kg BW for 14 days, intravenous Methylprednisolone 1000 mg/day for 3 days followed by prednisolone 1 mg/kg for 11 days, and placebo. Patients treated with intravenous methylprednisolone showed rapid improvement in vision compared to the other groups. A meta-analysis study in also obtained rapid improvement in vision with the administration of steroids in ON patients.[2,14–16]

Patients with typical optic neuritis are thought to recover quickly, with their vision getting better within a few weeks to a few months of the first symptoms. Patients in the ONTT study were followed up for 1 year and found that 95% of patients had visual acuity better than 20/40 and more than 70% with vision 20/20 or better.[14] In 15 years of follow-up only a few experienced recurrent optic neuritis or worsening visual acuity and 90% of patients with vision better than 20/40.[3,14] Patients who received methyl prednisolone therapy recovered faster after 30 days, but the results were not significant after 6 months.[17]

In this case, the initial treatment for this patient was the administration of 4x250 mg methylprednisolone for 3 days, followed by oral methylprednisolone 1 mg/Kg BW In patients. There was an improvement in visual acuity from the first to the sixth week, as well as improvements in visual acuity, color vision, contrast sensitivity, and visual field defect.

CONCLUSION

It is important to know the clinical symptoms, ophthalmological examination, and other ancillary examination such as an MRI examination, which is typical for optic neuritis to carry out the right treatment so as to get maximum therapeutic results. Treatment with intravenous methylprednisolone shows rapid recovery in patients, but further follow-up is required. Patients with optic neuritis can undergo an MRI examination to determine their risk of developing MS, because most of the early symptoms of MS are optic neuritis.

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