**Case Report**

# BILATERAL OPTIC NEURITIS IN A CHILD ASSOCIATED MULTIPLE SCLEROSIS

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## Introduction

Optic neuritis is an acute inflammatory demyelinating disorder of the optic nerve. The general characteristics of optic neuritis include unilateral, subacute, and painful visual loss without systemic or other neurological symptoms and optic neuritis is mostly seen in young females.[1]The etiology for optic neuritis varies including infections, inflammation, exposure to toxins, and genetic disorders. In most cases, the etiology may not be known for optic neuritis. In some cases, optic neuritis can be associated with demyelinating disorders of the central nervous system (CNS)including multiple sclerosis (MS) or neuromyelitis optic (NMO). Therefore, accurate diagnosis, risk assessment and management of patients with optic neuritis are warranted, and possibility of conversion to MS should be assessed [1,2] .

Common characteristic symptoms of optic neuritis include visual problems, periorbital pain, and color vision deficits. Vision loss is usually unilateral and deficits can range in severity from mild (20/20) to severe (no light perception). Patients usually complain about periocular, retro-ocular pain prior to vision loss that commonly occurs with eye movements. Visual field defects are also common in acute demyelinating optic neuritis. Different visual field patterns are seen in optic neuritis and include diffuse visual field defects or focal defects with centrocecalscotoma being the most common visual field defect. The presence of afferent pupillary defect (APD) would suggest unilateral optic neuropathy. Vision defectsusually progress for the first 1–2 weeks and then visualrecovery usually begins within the first 4 weeks. Opticatrophy and disc pallor develops in 4–6 weeks [3,4]. Color vision is commonly affected and is evaluated by usingvarious color plates (Ishihara color plates, American Optical Hardy-Rand-Rittler Color Vision Plates [HRR]).Optic neuritis is one of the most common initial clinical presentationsof MS without any prior history of a demyelinating event [1,4].

MSis an inflammatory autoimmune of the central nervous system that is characterized by pathologic changes, including demyelination and axonal injury.Magnetic Resonance Imaging (MRI) has become a routine clinical examination in MS. MRI is the most important paraclinical tool for MS, and derived measures have been established as standard outcome markers to monitor the treatment response in various MS clinical trials [5].

MRI findings seem to predict strongly the risk of developing MS. Studies have shown that spinal cord lesions might be strong indicators for predicting MS risk. Usually the involved optic nerve would be hyperintense with contrast enhancement. If there are brain and spinal cord lesions compatible with MS, the characteristics of the demyelinating lesions would include 3 mm ovoid lesions that are mostly located in periventricular areas of the white matter and radiate toward the ventricular spaces. The chance of developing MS increases for patients with one or more lesions. In recent practice, McDonald's criteria are the most utilized diagnostic criteria to assess the MS conversion risk in patients with a single demyelinating episode by providing MRI evidence for dissemination in space (DIS) and dissemination in time (DIT). While evaluating patients with a first episode of optic neuritis, in addition to MRI, additional tests including cerebrospinal fluid (CSF) analysis, evoked potentials (visual, motor, somatosensory, brain auditory) might also be helpful. Visual Evoked Potentials (VEPs) reflect demyelination in the afferent visual pathways. VEPs seem to be sensitive and specific for detecting optic neuritis with no apparent clinical presentation and abnormal findings even in silent cases.Abnormal VEP findings include increased latencies and reduced amplitudes and abnormal waveforms [6,7].

Corticosteroids have been recommended for treating acute optic neuritis. There is still no consensus about the application of corticosteroid (intravenous or oral forms), dosage, and treatment duration. Often, clinical presentation of optic neuritis is the main factor for the decision maker for initiating and duration of acute therapy. Management of optic neuritis could be with oral prednisone (1 mg/kg/d for 14 days) or intravenous methylprednisolone (250 mg every 6 hours for 3 days) followed by an oral prednisone taper (1 mg/kg/d for 11 days). The recommendation of Optic Neuritis Treatment Trial (ONTT) is to treat acute optic neuritis with iv high dose corticosteroid [8,9].

## Case report

A 5 years old girl suddenly blurred vision in both eyes and headache within 4 days before admitted in outpatient clinic. Patient had no fever and neurologic deficit. Visual acuity in first day were hand movement, color vision difficult to be evaluated in both eyes and right eye positive relative afferent pupillary defect. Posterior segment evaluation from fundus photograph showed bilateral edema optic nerve (Figure 1).

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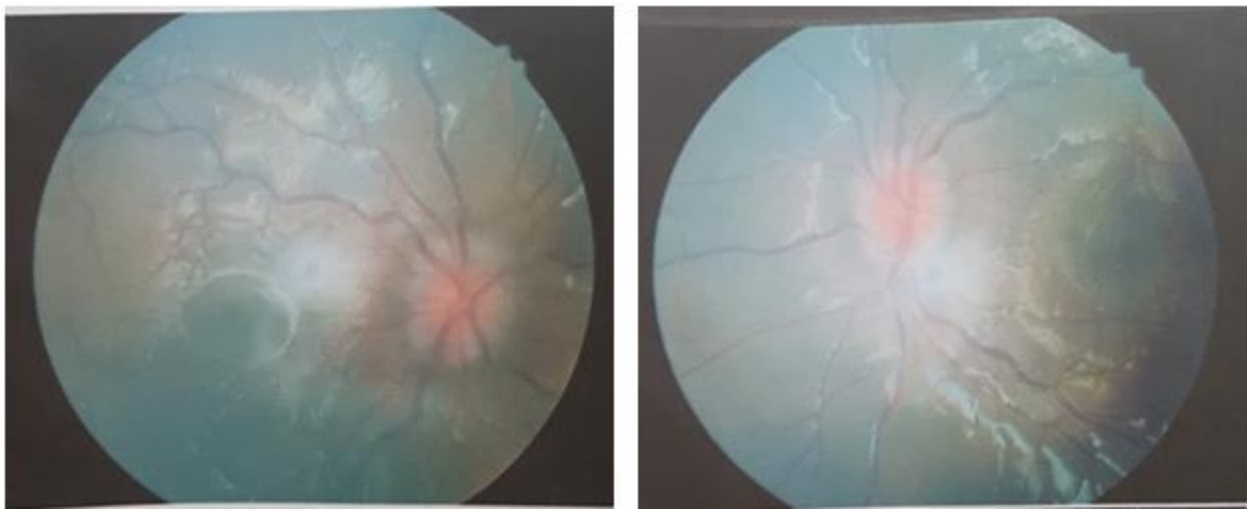

**Figure 1.** Fundus photograph before intravenous methylprednisolone. Both eyes showed optic disc edema

MRI showed bilateral optic neuritis, chronic plaque in right parietal lobe, right and left centrum semiovale (Figure 2). VEP showed demyelinating lesion in bilateral visual pathway.

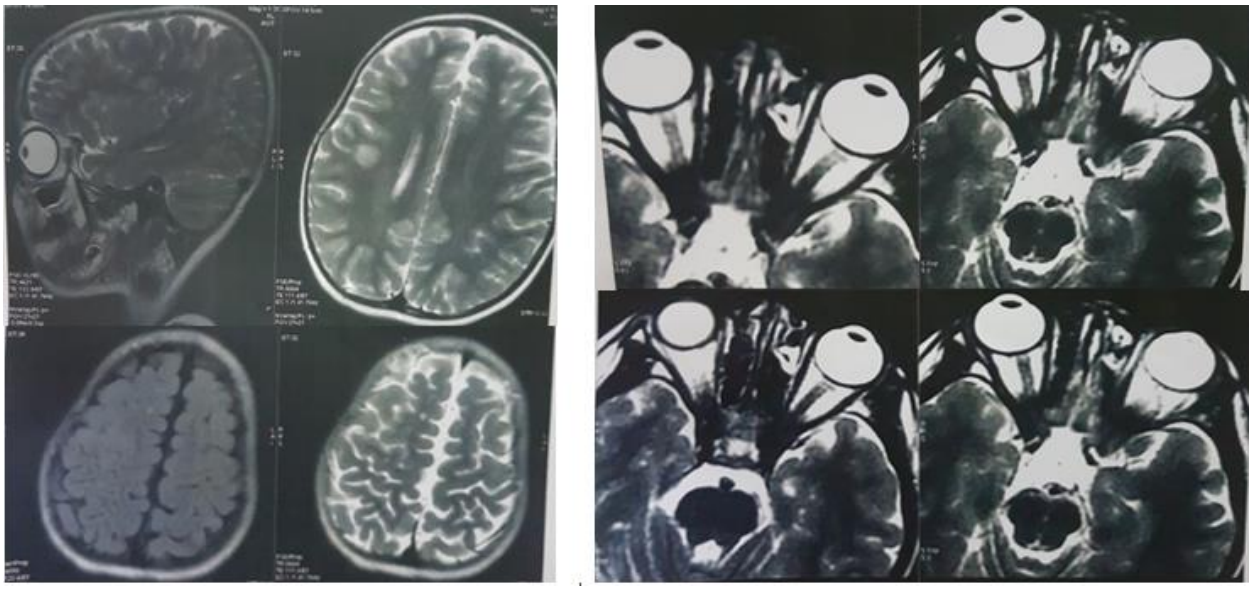

**Figure 2.** MRI showed bilateral optic neuritis, chronic plaque in right parietal lobe, right and left centrum semiovale according to multiple sclerosis.

The patient was diagnosed with bilateral optic neuritis associated multiple sclerosis and hospitalized with treatment of intravenous methylprednisolone 500 mg divided 4 times a day and continued with oral prednisone start on the sixth days. After treatment with intravenous corticosteroid until fifth days follow up, BCVA and color vision improve gradually. At fifth day, the visual acuity on right eye was 3/60 and left eye 1/60 with presence of APD. Funduscopy on both eye were improved with optic nerve head sharp margin but still hyperemia condition (Figure 3).

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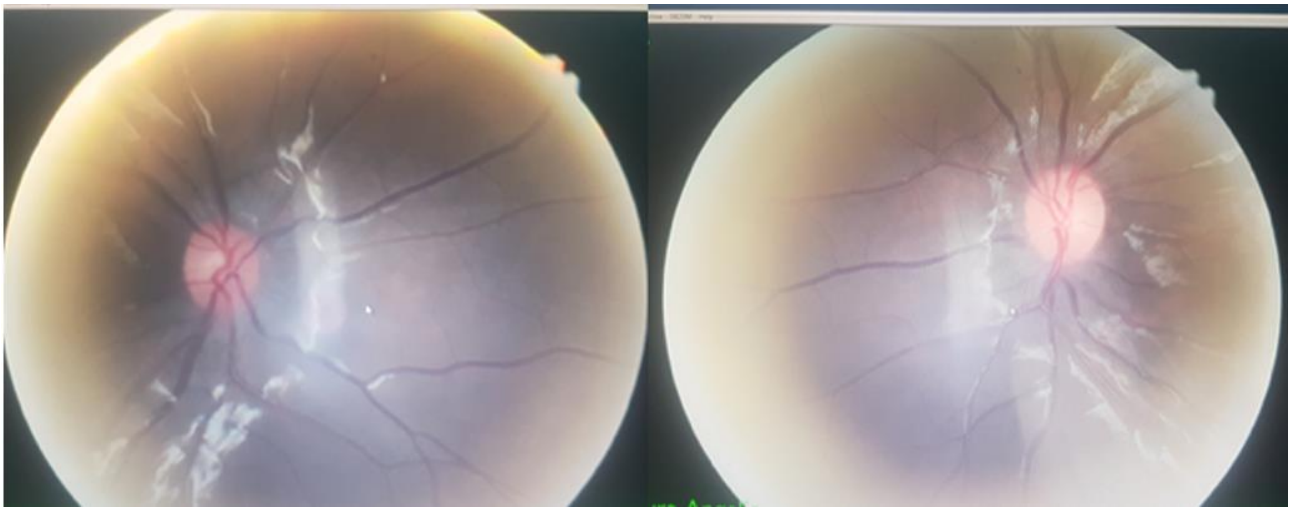

**Figure 3.**Fundus photograph after intravenous methylprednisolone on 5th days follow up

At sixth day follow up showed the improvement of visual acuity with 5/60 on the right eye and 1/60 on the left eye. At the ninth day follow up, the visual acuity significantly improved become 5/5 on right eye and 5/8.5 on left eye. It had show that corticosteroid became successful therapy for this patient. Fundus photograph both eyes at ninth days follow up after oral prednisone therapy showed a disc edema subsided (Figure 4). A month follow up, BCVA on right and left eye were 5/5 and Ishihara test on both eyes were 14/14. Fundus photograph showed normal optic nerve head (Figure 5).

**Figure 4.** At 9th days follow up. Fundus photograph after 3 days of oral prednisone treatment

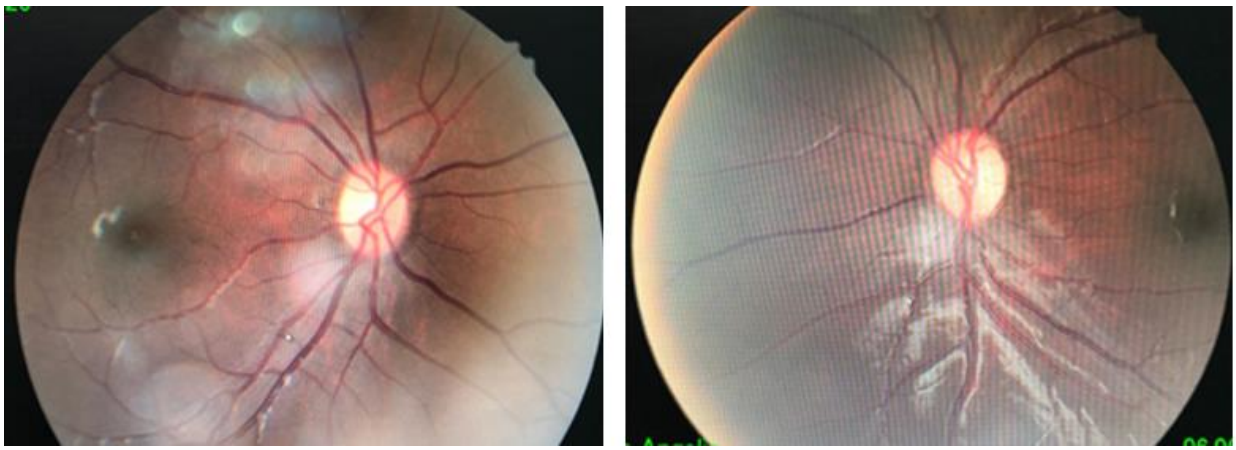

**Figure 5.** Fundus photograph after oral prednisone treatment on a month follow up. It showed a normal fundus

**Discussion**

Optic neuritis is an acute inflammatory disorder of the optic nerve, typically presents with sudden monocular visual loss and eye pain in young adults, without systemic or other neurological symptoms and also more commonly in women. It is a common initial manifestation of multiple sclerosis (MS) [10,11]. In this

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case, patient suddenly blurred vision in both eyes and got headache but patient had no fever and neurologic deficit [10].

In most cases, the etiology may not be known for optic neuritis that can be associated with multiple sclerosis (MS). A relationship between optic neuritis and MS has been well recognized for many years. The risk was strongly related to MRI evidence of prior demyelination in the white matter ofthe brain at the time of optic neuritis onset.

Multiple sclerosis (MS) is an inflammatory autoimmune of the central nervous system
that is characterized by pathologic changes, including demyelination and axonal injury. MR imaging is the most important paraclinical tool for MS. Magnetic Resonance Imaging (MRI) findings seem to predict strongly the risk of developing MS. The characteristics of the demyelinating lesions would include 3 mm ovoid lesions that are mostly located in periventricular areas of the white matter and radiate toward the ventricular spaces [5].

In this case, MRI showed bilateral optic neuritis, chronic plaque in right parietal lobe, right and left centrum semiovale. When at least one lesion was present, the risk was fairly consistent throughout the 15 years and did not substantially increase when additional lesions were present. Regardless of whether brain MRI lesions were present at the time of optic neuritis, neurologic disability was mild in most patients who developed MS [1].

Vision loss is usually unilateral and deficits can range in severity from mild (20/20) to severe (no light perception). Vision defects usually progress for the first 1–2 weeks and then visual recovery usually begins within the first 4 weeks. From patient condition, the visual acuity on both eyes were a hand movement that showed severe vision loss, but after follow up until $9^{th}$ days, had showed significantly improvement of visual acuity. At a month follow up, this patient had no complained about blurred vision. From the visual acuity examination on both eye showed normally with normal fundus from fundus photograph.

VEPs seem to be sensitive and specific for detecting optic neuritis even in silent cases with no apparent clinical presentation and abnormal findings are often observed in silent cases. Abnormal VEP findings include increased latencies and reduced amplitudes and abnormal waveforms. In this case, VEP showed demyelinating lesion in bilateral visual pathway [1,7].

Management of optic neuritis could be with oral prednisone (1 mg/kg/d for 14 days) or intravenous methylprednisolone (250 mg every 6 hours for 3 days) followed by an oral prednisone taper (1 mg/kg/d for 11 days). The recommendation of ONTT is to treat acute optic neuritis with intravenous high dose corticosteroid. Other study showed that a 3-day course of methylprednisolone given intravenously in a dose of 250 mg every 6 hours followed by 2 weeks of daily oral prednisone in a dose of 1 mg/kg/day accelerated visual recovery but did not improve the eventual visual outcome.In this patient condition, initial treatment with intravenous methylprednisolone 500 mg divided 4 times daily for 5 days then followed by prednisone 1 mg/kg/day for 2 weeks. The result was patient had better visual outcome and had significantly improvement of visual acuity and fundus photograph [1,3].

**Conclusion**

Optic neuritis can be associated withmultiple sclerosis. Eye examination include BCVA, color vision test, fundus photograph, and radiology imaging with MRI, also additional test include visual evoked potential might be helpful to diagnosis optic neuritis associated multiple sclerosis. Management therapy of optic neuritis with high dose corticosteroid followed by oral prednisone give better visual outcome.

**Bilateral Optic Neuritis in a Child Associated Multiple Sclerosis**

**Dwita Permatasari, Lukisiari Agustini, Gatot Suhartono**

**Introduction :**Optic neuritis can be associated with multiple sclerosis (MS). Therefore, accurate diagnosis, risk assessment and management of patients with optic neuritis associated multiple sclerosis should be assessed.
**Method:** A 5 years old girl suddenly blurred vision in both eyes and headache within 4 days before admitted in outpatient clinic. Patient had no fever and neurologic deficit. Visual acuity in first day were hand movement, color vision difficult to be evaluated in both eyes and right eye positive relative afferent pupillary defect. Posterior segment evaluation showed bilateral edema optic nerve. MRI showed bilateral optic neuritis, chronic plaque in right parietal lobe, right and left centrum semiovale. VEP showed demyelinating lesion in bilateral visual pathway. Patient was given intravenous methylprednisolone 500 mg divided 4 times a day and continued with oral prednisone start on the sixth days.
**Result:** After treatment with intravenous corticosteroid until fifth days follow up, BCVA and color vision improve gradually. At the ninth days follow up, BCVA on right eye (RE) 5/5 and left eye (LE) 5/8.5 pinhole not improve. Ishihara on RE 12/14 and LE 10/14. Ophthalmoscopy examination showed disc edema subsided in the follow up. A month follow up, BCVA and Ishihara on both eye were5/5 and 14/14. Opthalmoscopy examination showed normal optic nerve head.
**Conclusion:** Bilateral optic neuritis in a child associated multiple sclerosis is a challenging case. Intravenous methylprednisolone is the first line drug therapy give better visual outcome.
**Keywords:** bilateral optic neuritis, multiple sclerosis, methylprednisolone

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