# Characteristics and Response to Methylprednisolone and Prednisone Treatment of Optic Neuritic Patient at Sanglah General Hospital Denpasar

*[1]Dita-Rinjani, N. P., [1]Mas-Putrawati, A. A., and [2,3]Putra-Manuaba, I. B.*

[1]Department of Ophthalmology, Faculty of Medicine, Udayana University, Sanglah General Hospital, Bali-Indonesia.
[2]Department of Chemistry, Faculty of Math and Science, Udayana University, Bali-Indonesia
[3]Postgraduate School of Biomedicine, Udayana University, Bali-Indonesia

**Objective:** Treatment of optic neuritic as recommended by the Optic Neuritic Treatment Trial (ONTT) was intravenous methylprednisolon followed by oral prednisone. This study aims to describe characteristics and response to intravenous methylprednisolon followed by oral prednisone treatment of optic neuritic patient in Sanglah General Hospital Denpasar.
**Method**: This report is an analytical cross sectional study. Data were collected retrospectively from medical report of optic neuritic patient who came to Sanglah General Hospital during a period of January 1st 2010 until December 31st 2011. Patient characteristics were analyzed with descriptive analyses and presented as frequency, percentage, mean and standar deviation. Visual acuity and contrast sensitivity improvement after intravenous methylprednisolon followed by oral prednisone treatment were statistically analyzed with *Wilcoxon* test
**Results**: Optic neuritic were found in twenty-three patients (33 eyes), majority was in age group of 15-40 years (56.5%) with female predominance (65.2%) and unilateral involvement was 56.3%. Mean onset patient presented to the hospital was 21.7±2.21 days and the most common symptom was decreasing vision (87.9%). The majority of patient presented with papillitis (54.5%), totally color blindness found in 39.4% eyes, and the type of visual field defect at presentation was central scotoma (18.2%). All cases show lesion of optic nerve from visual evoked potential (VEP) examination and magnetic resonance imaging (MRI) shows normal results (39.1% patient). The mean of pretreatment logMAR visual acuity and contrast sensitivity were significant improve after treatment from 1.59±0.47 to 0.59±0.62 ($p$=0.0001) and 0.31±0.56 to 1.25±0.56 ($p$=0.0001), respectively. All cases in this study were idiopathic. Recurrence were seen in 2 eyes and none of patient had clinical features suggestive of multiple sclerosis.
**Conclusions**: Visual acuity and contrast sensitivity improvement after intravenous methylprednisolon followed by oral prednisone treatment in this study is good, with lower rate of recurrence and none of cases associated with multiple sclerosis.
**Keywords: optic neuritic, intravenous methylprednisolon, oral prednisone, visual improvement**

## INTRODUCTION

Optic neuritic is an inflammatory disorder of the optic nerve, classicaly divided into typical and atypical types. Most of cases are idiopathic in nature however it could be associated with demyelinating lesion specifically multiple sclerosis.[1,2]

Optic Neuritic Treatment Trial (ONTT) reported incidence optic neuritic 1-5 cases per 100.000/year and more commonly in Caucasian.[3] There is no study reported incidence of optic neuritic in Indonesia. Individual ages of 20-49 years are most at risk and more common in women (77%). The attack is usually unilateral in 70% of adult.[1,3]

**Correspondence: Dita-Rinjani, N. P.**
**Address: Department of Ophthalmology, Faculty of Medicine, Udayana University, Sanglah General Hospital, Bali-Indonesia.**

The pathogenesis of optic neuritic is not entirely understood. It believed that autoimmun process causing destruction of myelin (demyelinating). Inflammation and infection lead to destruction vascular system and activating the autoimmun process.[4] Optic neuritic clinically present with triad of symptoms, subacute visual loss (days to weeks), periocular pain esspecially with eye movement and dyschromatopsia. Visual field defect, relative afferent pupillary defect (RAPD) and reduced contrast and sensitivity are usually present in the affected eye. Optic disc appearances on optic neuritic can be determined as retrobulbar neuritic when optic disc appearance normal, papillitis characterised by hyperaemia of the disc blurring of the margins and peripapillary hemorrhage and neuroretinitis when papilittis is associated with macular star formation. The diagnosis of optic neuritic is a clinical one, ancillary test such as visual evoked potential

(VEP), magnetic resonance imaging (MRI) and laboratory test are necessary for confirming the diagnosis, to assess the risk of developing multiple sclerosis and to rule out other disorder in atypical cases.[3,5,6]

The treatment of optic neuritic are still controversial. ONTT recomended intravenous methylprednisolon 250 mg every 6 hours for 3 days followed by oral prednisone (1 mg/kg/day) for 11 days and than oral doses taper 20% every 2-3 week depend on visual acuity patient. This treatment showed accelerated visual recovery and reduced risk developing multiple sclerosis in first 2 year especially in patient with MRI scan showing abnormal lesion.[7,8] The long term visual prognosis of optic neuritic is remains good. After 15 years, 72% of the eyes affected with optic neuritic had visual acuity of 20/20. The probability of having recurrent optic neuritic after 10 year of follow up in ONTT was 35% and 48% of those developing multiple sclerosis.[9]

This study aims to describe characteristics and response to intravenous methylprednisolon followed by oral prednisone treatment of optic neuritic patient in Sanglah General Hospital Denpasar.

## MATERIALS AND METHODS

This report is an analytical cross sectional study. Data were collected retrospectively from medical report of optic neuritic patient who came to Sanglah General Hospital during period January 1st 2010 until December 31st 2011. Inclusion criteria were optic neuritic patient which is treated with intravenous methylprednisolon 250 mg every 6 hours for 3 days followed by oral prednisone (1 mg/kg/day) for 11 days and than doses taper depend on visual acuity patient . exclusion criteria is patient with incomplete medical report.

Data were analyzed using SPSS 16.0 computer program. Patient characteristics was analyzed with descriptive analyses and presented as frequency, percentage, mean and standar deviation. Visual acuity and contrast sensitivity improvement after intravenous methylprednisolon followed by oral prednisone treatment were statistically analyzed with *Wilcoxon* test.

## RESULTS

During period January 1st 2010 until December 31st 2011, there were 23 patient (33 eyes) diagnosed as optic neuritis. The majority cases were in age group 15-40 years (56.5%) with female predominance (65.2%) and unilateral involvement was 56.3%. Mean onset patient presented to the hospital was 21.7±2.21 days and the most common symptom was decreasing vision (87.9%). The majority of patient presented with papillitis (54.5%), totally color blindness found in 39.4% eyes, and the type of visual field defect at presentation was central scotoma (18.2%). All cases showed lesion of optic nerve from VEP examination and MRI showed normal result in 39.1% patients. The baseline characteristic and clinical finding of study subjects were seen in Table 1 and 2.

Table 1. Baseline Characteristics of Study Subject

| Variables | n (%) |
|---|---|
| Sex | |
| Male | 8 (34.8%) |
| Female | 15 (65.2%) |
| Age | |
| <15 yo | 2 (8.7%) |
| 15-40 yo | 13 (56.5%) |
| > 40 yo | 8 (34.8%) |
| Eye involvement | |
| Unilateral | 13 (56.5%) |
| Bilateral | 10 (43.5%) |

Table 2. Clinical Sign and Ophthalmology Examination

| Clinical Sign and Examination | Description |
|---|---|
| Onset (days) | 21.7±2.21 |
| Chief complain | |
| Decreasing vision | 29 (87.9%) |
| Pain on eye movement | 4 (12.1%) |
| Optic disc appearance | |
| Retrobulbar neuritis | 15 (45.5%) |
| Papillitis | 18 (54.5%) |
| Neuroretinitis | 0 (0%) |
| Color vision | |
| Can't be evaluated | 15 (45.5%) |
| Total color blindness | 13 (39.4%) |
| Partial color blindness | 5 (15.1%) |
| Visual field | |
| Can't be evaluated | 27 (81.8%) |
| Central scotoma | 6 (18.2%) |
| Ancillary test | |
| VEP | |
| Optic nerve lesion | 33 (100%) |
| MRI | |
| Not evaluated | 12 (52.2%) |
| Normal | 9 (39.1%) |
| Enhancement optic nerve | 2 (8.7%) |
| Recurrence | |
| Recurrence | 2 (6.1%) |
| No recurrence | 31 (93.9%) |

The mean of pretreatment logMAR visual acuity were 1.59±0.47 and significant improve after treatment to 0.59±0.62 ($p$=0.0001). The mean of pretreatment contrast sensitivity were 0.31±0.56 and significant improvement after treatment to 1.25±0.56 ($p$=0.0001). The mean logMAR visual

acuity and contrast sensitivity before and after treatment were seen in Table 3 and 4.

Table 3. Visual Acuity Before and After Treatment

| | Before treatment | After treatment | *p* |
|---|---|---|---|
| LogMAR Visual acuity (Mean±SD) | 1.59±0.47 | 0.59±0.62 | .0001 |

Table 4. Contrast Sensitivity Before and After Treatment

| | Before treatment | After treatment | *p* |
|---|---|---|---|
| Contrast Sensitivity (Mean±SD) | 0.31±0.56 | 1.25±0.57 | .0001 |

## DISCUSSION

The majority of the patients in this study were within 15-40 age group, most common in female and unilateral involvement, which is consistent with other study from optic neuritic study group, Singapore and Taiwan.[10,11] In this study, decrease vision symptom of the patient was the most observed on 21 days of onset to the hospital. This result is similar to the previous study in Nepal and Taiwan.[13] However, in contrast to other study in Singapore and ONTT which were obtain that most patient complain pain was on eye movement.[3,10] Onset patient presented to the hospital is depend on patient symptom. Inflammation of the optic nerve causes decrease of vision because of the swelling and destruction of the protective myelin sheath that covers the optic nerve. Pain is experianced as dull ache with or without tenderness of globe with maximum severity within 24-36 hour and spontaneously resolve 48-72 hour.[4,14]

The majority of the eyes had papillitis (54.5%), retrobulbar neuritis was found in 45.5% cases and no case with neuroretinitis. This was consistent with the study done in India and Singapore.[10,14] In papillitis, inflammation is located anteriorly in the optic nerve.[1] Totaly color blindness and central scotoma visual field defect were most common found in this study. This result is similar with previous study in India and Nepal.[16,17] Dyschromatopsia in optic neuritic typically observed as reduced vividness of saturated color (desaturated) esspecially for red green color. This is because of demyelinating lesion produce loss of electrical conduction in predominant red green opponent fiber.[2] Involvement of papillomacula fiber during attack of optic neuritis produce central or cecocentral scotoma visual field defect.[1]

In our study, All cases showed optic nerve lesion from VEP and MRI showed normal result in most patient. This result consistent with other study in Nepal and India.[12,17] VEP limited usefulness because numerous factors produce abnormal waveform other than damage in visual pathways damage. MRI result on demyelinating lesion demonstrates periventricular white matter with FLAIR (*Fluid-attenuated inversion recovery*) technique or gadolinum contrast. All cases in this study were idiopathic. Recurrence were seen in 2 eyes and none of patient had clinical features suggestive of multiple sclerosis. Recurrent optic neurits is indicative of an underlying disease process especially multiple sclerosis.[1,8]

The mean of pretreatment logMAR visual acuity and contrast sensitivity in this study were improve significantly after treatment with intravenous methylprednisolon followed by oral prednisone as recommended by ONTT. Our result is similar with previous study in India and Nepal.[2,12,15] ONTT showed that intravenous methylprednisolon followed by oral prednisone accelerated visual recovery and reduced risk developing multiple sclerosis in first 2 year especially in patient with MRI scan showing abnormal lesion.[8] Contrast sensitivity is sensitive indicator for visual disorder in ON in acute or recovery phase.[14]

## CONCLUSION

Visual acuity and contrast sensitivity improvement after intravenous methylprednisolon followed by oral prednisone treatment in this study is good, with lower rate of recurrence and none of cases associated with multiple sclerosis.

## REFERENCE

1. American Academy Of Ophthalmology 2010-2011. Neuroophthalmology. United State of America: American Academy of Ophthalmology. p.144-146.
2. Menon V., Saxena R., Misra R., and Phuljhele S. 2011. Management of Optic Neuritic. *Indian J Ophthalmol*, 59: 117-122.
3. Optic Neuritic Study Group. 1991. The Clinical Profile of Optic Neuritic. Experience of The Optic Neuritic Treatment Trial. *Arch Ophthalmol*, 109:1673-1678.
4. Dooley M.C. and Foroozan R. 2010. Optic Neuritic. *J Ophthalmic Vis Res*, 5 (3): 182-187.
5. Murphy M.A. 2008. Clinical Update on Optic Neuritic and Multiple Sclerosis. *Medicine and Health*, 91: 57-59.
6. Shams P.N. and Plant G.T. 2009. Optic Neuritic: A review. *The International MS Journal*, 16: 82-89.
7. Beck R.W. and Gal R.L. 2008. Treatment Of Acute Optic Neuritic A Summary Of Findings

From The Optic Neuritic Treatment Trial. *Arch Ophthalmol*, 126 (7): 994-995.
8. Optic Neuritic Study Group. 2008a. Multiple Sclerosis Risk After Optic Neuritic Final Optic Neuritic Treatment Trial Follow-up. *Arch Neurol*, 65 (6): 727-732.
9. Optic Neuritic Study Group. 2008b. Visual Function 15 Years After Optic Neuritic: A Final Follow-up from Optic Neuritic Treatment Trial. *Ophthalmology*, 115 (6):1079-1082.
10. Lim S.A., Goh K.Y., Tow S., Fu E., Wong T.Y., Seah A., Tan C., and Cullen J.F. 2008. Optic Neuritic in Singapore. *Singapore Med J*, 49 (9): 667-671.
11. Lin C.W., Ching H.L., Ching Y.T., Ming T.T., Jieh R.J., and Pesus C. 2007. Optic Neuritic Among National Health Insurance Enrollees in Taiwan 2000-2004. *Neuroepidemiology*, 29: 250-254.
12. Das H., Gautam M., and Lavaju P. 2010. An Overview Of Idiopathic Optic Neuritic In Easthern Nepal. *Nepal J Ophthalmol*, 2 (3): 10-15.
13. Menon V., Mehrotra A., Saxena R., and Jaffery N.A. 2007. Comparative Evaluation Of Megadose Methylprednisolone With Dexametasone For Treatment Of Primary Typical Optic Neuritic. *Indian J Ophthalmol*, 55:355-359.
14. Wray S.H. 1994. Optic neuritic. In: Albert D.M., Jakobiec F.A., editors. Principle and practice of ophtalmology. Philadelphia: WB Saunder. p. 2539-2550
15. Saxena R., Phuljhele S., Menon V., and Shailesh G.M. 2010. Profile of Optic Neuritic Patients in India. *Indian J Ophthalmol*, 57: 448-450.
16. Shrestha R., Pokharel S., Malla O.K., and Shakya K.N. 2007. Visual Outcome After Intravenous Methylprednisolone for Optic Neuritic. *Nepal Journal of Neuroscience*, 4: 70-74.
17. Sethi H.S., Menon V., Sharma P., Khokhar S., and Tandor R. 2006. Visual Outcome After Intravenous Dexamethasone Therapy for Idiopathic Optic Neuritic in an Indian Population: A Clinical Case Series. *Indian J Ophthalmol*, 54:177-183.