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SHORT COMMUNICATION

Visual Function following Treatment of Optic Neuritis

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Abstract

Background: Optic neuritis is a common cause of visual loss in young adults and often the first manifestation of multiple sclerosis**.** Recent studies have shown that treatment with intravenous methylprednisolone results in more rapid recovery of vision, but without any long term difference in visual acuity**.** This study was carried out to evaluate clinical characteristics of patients with optic neuritis and visual outcome after intravenous methylprednisolone treatment.

Methods: In a case series study, 40 cases with optic neuritis were evaluated. Before and after treatment with methylprednisolone according to optic neuritis treatment trial, visual acuity, contrast sensitivity, color vision, streopsis and visual field were analyzed.

Results: 67.5% of the patients were females. The most common age group was between 20 and 40 (60%). Blind spot enlargement and other visual field defects were also returned to relatively normal value after the treatment. Central scotoma was the most common field defect (70%) and mild Dutan defect was the most common color vision (60%) defect in this study. Visual acuity, contrast sensitivity, color vision, streopsis and visual field were significantly reduced in optic neuritis, relatively returning to the normal level after treatment.

Conclusion: It seems that the assessment of other visual functions, besides visual acuity, is important in a patient with optic neuritis, because patients usually remain aware of visual deficits other than decreased visual acuity.

Keywords: Optic neuritis; Visual function; Treatment

Introduction

Optic neuritis (ON) is a common cause of visual loss in young adults and is often the first manifestation of multiple sclerosis.[1] Most of the patients are women (77%), between 15 to 45 years old.[1] Recent studies, particularly from the Optic Neuritis Study Group, have helped to clarify the natural history and treatment of optic neuritis.[2] These studies have shown that, as compared with oral prednisolone or placebo, treatment with intravenous methylprednisolone (IVMP) results in a more rapid recovery of vision, but without any long term difference in visual acuity. Moreover, there was a higher rate of recurrence of optic neuritis in the oral prednisolone treated group.[3] The subsequent development of clinically definite multiple sclerosis was delayed for up to 2 years in patients treated with IVMP.[1] Although the most common visual presenting symptom is deterioration of visual acuity, contrast sensitivity, color vision, streopsis (especially in moving targets), and visual field will also be involved in this process. Patients usually remain aware of visual deficits in the affected eye after recovery.[1,4]

Despite widespread publication of the ONTT results, few publications exist in this field in our country. We aimed to clarify whether optic neuritis treatment according to ONTT will change contrast sensitivity, visual field, color vision and streopsis, besides the visual acuity.

Materials and Methods

In a non-comparative interventional case series study, 40 patients with optic neuritis were investigated in

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Ophthalmology Department of Mashhad University of Medical Sciences. The diagnosis of optic neuritis was based on clinical examination. Besides funduscopic examination, the patients with acute decrease in visual acuity were examined to detect relative afferent pupillary defect (RAPD) and dyschromatopsia. The cases without the typical symptoms of optic neuritis or those with other ophthalmologic and systemic problems, such as retinitis, vitritis or papilophlebitis, diabetes and hypertension were excluded from our study and only patients with typical optic neuritis were investigated.

In four patients over 50 years of age, the diagnosis was made on the basis of typical clinical presentation and negative funduscopic findings for ischemic optic neuropathy. They did not have systemic disorders such as diabetes mellitus and hypertension and had normal ESR, so they were included in our study. Visual acuity was examined by Snellen chart, contrast sensitivity by Cambridge low contrast grating test, color vision by Ishihara test, visual field by Goldman and Humphrey automated perimetry, and streoacuity by Titmus test. Those patients who sought medication up to 11 days from the beginning of the symptoms (Mean: 3.5 days) were included in our study.

After the primary assessment by an ophthalmologist and an optometrist, the patients underwent treatment. The therapeutic protocol, on the basis of ONTT protocol,[5] was as follows: Intravenous infusions of methyl prednisolone (250 mg every six hours, for three days) and subsequent oral prednisolone (1 mg/kg) for 11 days. The tests were conducted before the initiation and one week after the completion of treatment (three weeks after admission). Mean test values before and after treatment were compared by paired T test, using SPSS version 11 software (Chicago, IL, USA) and P value less than 0.05 was considered significant.

Results

Demographic findings showed that 27 (67.5%) patients were female and 13 (32.5%) were male. Out of 40 patients, 20 had right eye involvement and 19 had left eye involvement and only one patient had bilateral involvements. Twenty four (60%) cases were between 20 and 40 years; their age distribution is summarized in Figure.1. Only two patients had other neurological symptoms such as paraesthesia and paresis. According to clinical manifestations, MRI and recurrent attack of optic neuritis, and also consultation with a neurologist, only one patient turned out to have multiple sclerosis.

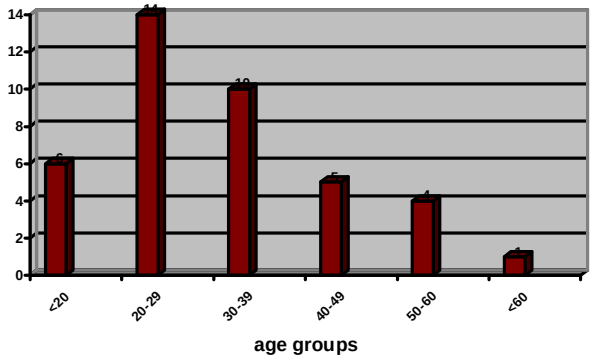

Fig 1: Age distribution.

Deterioration of visual acuity was the main complaint in all patients. Other symptoms were painful eye movement (40%), painful eye movement and headache (42.5%) and headache (17.5%). The mean visual acuity in the involved eye was 0.3±0.11 and in the uninvolved eye 0.8±0.26, (p<0.0001). Visual acuity (16.5%) and contrast sensitivity (6.6%) were affected in the uninvolved eye. In fundoscopic examination, 29 patients did not have any findings. In four cases, optic nerve head elevation plus hyperemia and in 7 cases optic disc elevation plus hemorrhage were seen. The median of contrast sensitivity in the involved eye was 250 cycles per degree and in the uninvolved one 49 cycles per degree. Most patients had steroacuity above 200 seconds of arc.

Goldman perimetry was done for all patients. Central, paracentral, temporal and inferior scotoma were observed in 70%, 7.5%, 5% and 5% of cases respectively. Nasal and superior constriction field defects were visible in 2.5% of patients. Blind spot enlargement was noticed in 2,5% of subjects. In these patients, III4e target was used to find scotoma. V4e target was used to find generalized constriction; the isopter of this target did not show any statistically-significant difference between the two eyes. Automated perimetry in 13 patients with 30-2 strategy had been done and the calculated central field was similar to that of Goldman perimetry.

The size of the blind spot in the involved eye was measured in all patients. The enlargement of this spot in these patients was greater than normal distribution, as shown in vertical and horizontal diameters in Figure 2.

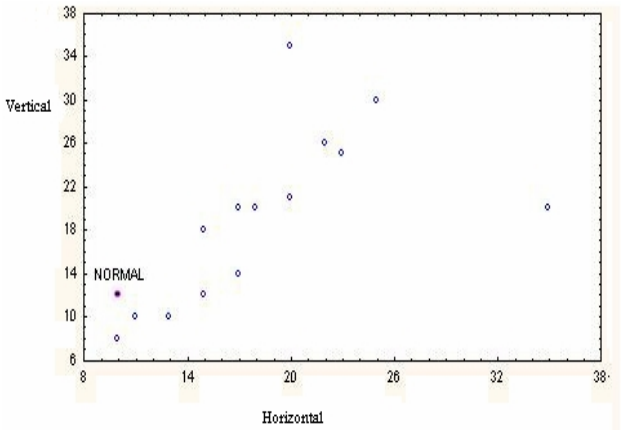

Fig 2: Blind spot enlargement before intervention- Vertical and Horizontal size (degree)

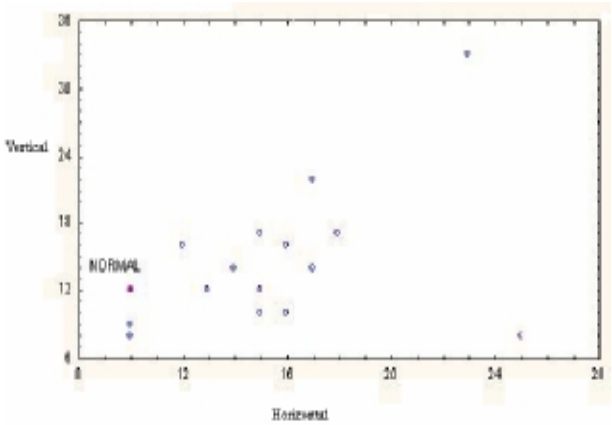

Fig 3: Blind spot enlargement after treatment- Vertical and Horizontal size (degree)

The Ishihara test results showed that 65% of patients had mild Dutan defect. This defect is defined as inability to read two pages from page 8 to 13 of Ishihara book, or blurredness of 6 pages relative to the uninvolved eye, or inability to see 4 pages relative to the uninvolved eye and blurredness of 2 pages. Inability to read more than those defined previously is categorized as severe Dutan defect, but we did not have any cases in our study. Post-therapeutic results of the same data are shown in Table 1.

In perimetry, the size of the blind spot returned to normal values and had a decrease in size, as shown in vertical and horizontal diameters (Figure 3).

Discussion

We had funduscopic findings similar to those described by Bee *et al.*[6] It was found that optic neuritis affects contrast sensitivity and streoacuity and these two strongly get better after appropriate treatment. In Trobe *et al.*'s study, contrast sensitivity was affected more than other functions and it was the most abnormal function after treatment.[7] Decreased color vision was observed in 60% of our patients, being lower than that in other studies. This difference seems to be due to different measuring techniques. The other studies have used Munsell 100 hue test, which is more sensitive than Ishihara test.[8]

In our findings, visual acuity and contrast sensitivity were also affected in the clinically uninvolved eye but not color vision and visual field. Beck *et al.* showed that color vision (21.7%) and visual field (48%) were also affected in the clinically uninvolved eye, as well as visual acuity (13.8%) and contrast sensitivity (15.4%).[9] Contrast sensitivity, color vision, visual field, streopsis disorders seem to remain partly in a 6 month follow up period.[4,10,11] Similar to our results, Hickman *et al.* also show the effectiveness of steroid therapy.[12] In Jiraskova *et al.*'s study, complete recovery of visual function was seen in almost all cases.[13] In Chirapapaisan *et al.*'s study, the final visual outcome in patients with isolated optic neuritis who received earlier treatment was better than those who received treatment later.[14] It has been indicated that corticosteroids do not prevent optic nerve atrophy in follow up studies.[15] MRI is a diagnostic and prognostic factor in optic neuritis.[16] VEP latency is influenced by optic neuritis, indicating an improved optic neuritis.[4] In an interesting study by Gosh *et al.*, more neurologists (55%) than

Table 1: Visual acuity, contrast sensitivity and stereopsis before and after treatment.

Intervention	Visual Acuity	Contrast Sensitivity (cycle/degree)	Stereopsis (second of arc)
Before	0.3 ± 0.11	49	320
After	0.7 ± 0.20	200	60
p value	0.0006	0.0001	0.004

ophthalmologists (9%) chose to treat the patients with intravenous methylprednisolone (p<0.005). Significantly, more ophthalmologists (64%) than neurologists (32%) chose not to give steroids (p<0.025). Oral prednisolone alone was rarely selected for treatment.[17]

It is concluded that optic neuritis more affects other visual functions than visual acuity. Therapeutic regimens have partial effect on primary lesions, but careful assessment of the effect of the treatment, in contrast to spontaneous improvement of disease, needs further investigations.

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Conflict of interest: None declared.

References

1 American Academy of Ophthalmology. Basic and Clinical Science Course Neuro-Ophthalmology. San Francisco: American Academy of Ophthalmology; 2008; Vol. 5, pp 146-149.

2 Celesia GG, Kaufman DI, Brigell M, Toleikis S, Kokinakis D, Lorance R, Lizano B. Optic neuritis: a prospective study. *Neurology* 1990;**40**:919-23. [2345615]

3 Jiraskova N, Rozsíval P. Visual functions in optic neuritis. *Cesk Slov Oftolmol* 2004;**60**:394-9. [15745407]

4 Beck RW, Gal RL, Bhatti MT, Brodsky MC, Buckley EG, Chrousos GA, Corbett J, Eggenberger E, Goodwin JA, Katz B, Kaufman DI, Keltner JL, Kupersmith MJ, Miller NR, Moke PS, Nazarian S, Orengo-Nania S, Savino PJ, Shults WT, Smith CH, Trobe JD, Wall M, Xing D; Optic Neuritis Study Group. Visual functions more than 10 years after optic neuritis: experience of optic neuritis treatment trial. *Am J Ophthalmol* 2004;**137**:77-83. [14700647] [doi:10.1016/S0002-9394(03)00862-6]

5 Glaser JS. Duane's Clinical Ophthalmology. Philadelphia: Lippincott Raven; Vol 2. Chap 5, 1998;pp.31-50.

6 Schneck ME, Haegerstrom-Portnoy G. Color vision defect type and spatial vision in the optic neuritis treatment trial. *Invest Ophthalmol Vis Sci* 1997;**38**:2278-89. [9344351]

7 Midgard R, Seland JH, Hovdal H, Celius EG, Eriksen K, Jensen D, Heger H, Mellgren SI, Wexler A, Beiske AG, Myhr KM. Optic neuritis diagnosis, treatment and follow up. *Tidsskr Nor Laegeforen* 2005;**125**:425-8. [15742012]

8 van der Worp HB, van Gijn J, Plant GT. Treatment of optic neuritis. *Ned Tijdschr Geneeskd* 2004;**148**:61-65. [14753126]

9 Wakakura M, Mashimo K, Oono S, Matsui Y, Tabuchi A, Kani K, Shikishima K, Kawai K, Nakao Y, Tazawa Y, Kiyosawa M, Abe H, Ohba N, Yago K, Maeda S, Sugita M, Ishikawa S. Multicenter clinical trial for evaluating methylprednisolone pulse treatment of idiopathic optic neuritis in Japan. Optic Neuritis Treatment Trial Multicenter Cooperative Research Group (ONMRG). *Jpn J Ophthalmol* 1999;**43**:133-8. [10340796] [doi:10.1016/S0021-5155(98)00071-9]

10 Moro SI, Rodriguez-Carmona ML, Frost EC, Plant GT, Barbur JL. Recovery of vision and pupil responses in optic neuritis and multiple sclerosis. *Ophthalmic Physiol Opt* 2007;**27**:451-60. [17718884] [doi:10.1111/j.1475-1313.2007.00501.x]

11 Chuenkongkaew W, Chirapapaisan N. Optic neuritis: characteristics and visual outcome. *J Med Assoc Thai* 2003;**86**:238-43. [12757063]

12 Hickman SJ, Kapoor R, Jones SJ, Altmann DR, Plant GT, Miller DH. Corticosteroids do not prevent optic nerve atrophy following optic neuritis. *J Neurol Neurosurg Psychiatry* 2003;**74**:1139-41. [12876255] [doi:10.1136/jnnp.74.8.1139]

13 Ghosh A, Kelly SP, Mathews J, Cooper PN, Macdermott N. Evaluation of the management of optic neuritis: audit on the neurological and ophthalmological practice in the north west of England. *J Neurol Neurosurg Psychiatry* 2002;**72**:119-21. [11784839] [doi:10.1136/jnnp.72.1.119]

14 Trobe JD, Beck RW, Moke PS, Cleary PA. Contrast sensitivity and other vision tests in the optic neuritis treatment trial. *Am J Ophthalmol* 1996;**12**:547-53. [8610798]

15 Optic Neuritis Study Group. Visual Function 15 Years after OpticNeuritis;A Final Follow-up Report from the Optic Neuritis Treatment Trial. *Ophthalmology* 2008;**115**:1079-1082. [17976727] [doi:10.1016/j.ophtha.2007.08.004]

16 Bee YS, Lin MC, Wang CC, Sheu SJ. Optic neuritis: clinical analysis of 27 cases. *Kaohsiung J Med Sci* 2003;**19**:105-12. [12751870]

17 Beck RW, Kupersmith MJ, Cleary PA, Katz B. Fellow eye abnormalities in acute unilateral optic neuritis. Experience of the optic neuritis treatment trial. *Ophthalmology* 1993;**100**:691-7. [8493012]