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Visual function after treatment for optic neuritis

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Abstract
Background and objectives: For the purpose of investigating the visual function, prognosis, and therapeutic response to treatment in patients with optic neuritis who attend the ophthalmology clinic.
Methods: This prospective study was carried out at Department of Ophthalmology, Sambhram Institute of Medical Sciences and Research, Kolar, Karnataka, India over a period from April 2016 to March 2017, during which 94 eyes were examined. Following treatment, visual function was assessed.
Results: Seventy-four percent of the eyes had a reduction in median log MAR visual acuity from 1.49 at baseline to 0.8 at the one-month follow-up. Additionally, statistically significant improvements were observed in color vision, central fields, brightness sensitivity, and red desaturation. The average age was 40.0 years, and 55.6% of the participants were.
Conclusion: After treatment, there was a noticeable improvement in visual function. Concomitant demyelination was observed in our investigation in relation to this study.

Keywords: Optic neuritis, visual function, visual acuity, colour vision, MAR visual acuity

Introduction
An inflammatory, infectious, or demyelinating disorder that affects the optic nerve is called optic neuritis. It is characterized by a sudden loss of vision that is progressively returned, usually with accompanying pain that lasts for several hours or days. Impacts on women occur more often than on men. The majority of cases are idiopathic, with the possibility of multiple sclerosis playing a role. The most common cause of optic neuritis is a demyelinating disease. Other typical causes include viral, parainfectious, inflammatory, paravaccinative, and immunological reactions [1, 2].

Sudden unicular vision loss is a common symptom of optic neuritis, which is often accompanied by pain that worsens with eye movement and is linked to multiple sclerosis. RAPD is almost always present when there is a unilateral circumstance. Dyschromatopsia is one of the apparent field abnormalities in injured eyes. The first month is usually when eyesight starts to improve. Without the usage of steroids, it improves. Atypical optic neuritis is characterized by a noticeable swelling of the optic nerve, retinal exudates, hemorrhages, and lack of pain. Multiple sclerosis is less common in patients who have unusual neuritis symptoms [2, 3].

After an incident of optic neuritis, the risk of developing multiple sclerosis increases. Optic neuritis is referred to as monosymptomatic, idiopathic, or a clinically isolated condition when there are no signs of multiple sclerosis. Optic neuritis initially manifests as dyschromatopsia, ocular pain, and vision loss. The most common cause of both impaired vision and pain during eye movements is retrobulbar optic neuritis. Pain may arise from the roots of the superior and medial recti pressing against the optic nerve sheath at the orbital apex. (Whitnall's hypothesis) [3, 4].

Other aspects of vision that are affected include color perception, contrast sensitivity, and visual field. Many visual field abnormalities, such as quadrantic, altitudinal, diffuse depressed, and centrocaecal scotomas, can be seen in patients with optic neuritis. The optic neuritis is still not entirely healed. The cornerstone of treatment, corticosteroids, expedites eyesight recovery. Even six to twelve months later, several studies show little to no improvement in the visual outcome. 500 mg of methylprednisolone were injected intravenously every day for three days. Following that, patients received oral steroid therapy for 11 days at a dose of 1 mg per kilogram of body weight [4, 5].

The gold standard treatment for optic neuritis is based on the Optic Neuritis Treatment Trial. Between 1988 and 1991, 455 people were recruited to investigate the efficacy of corticosteroids.

ONTT looked at the relationship between optic neuritis and multiple sclerosis as well as the efficacy of corticosteroid therapy for acute optic neuritis [5].

Materials and Methods

To assess the visual function, treatment response, and visual outcome in patients with optic neuritis, a prospective observational case study was conducted. 88 patients and 94 eyes were subjected to the investigation from April 2016 to March 2017, at Department of Ophthalmology, Sambhram Institute of Medical Sciences and Research, Kolar, Karnataka, India**.**

Inclusion criteria

1. Age range: 16 to 65
2. Visual field or visual acuity loss, with or without pain, lasting less than the one month duration.
3. Bidirectional versus unilateral
4. Fundus change
5. Field defects

Exclusion criteria

1. Age 16 or < 16
2. Visual loss due to compressive, toxic, metabolic, vascular, inherited neuropathies.
3. An optic neuritis episode that occurred in the affected eye previously.
4. Informed consent was taken from all the patients.

Results

The study included 94 eyes from 88 patients who had optic neuritis. Our patient's visual function metrics returned to pre-treatment levels after receiving steroid treatment quite soon.

Table 1: Visual acuity

Visual acuity	Baseline	1month
6/6 – 6/18	18(19.1)	44(60.3)
6/24 – 6/60	12(12.7)	14(19.2)
6/60 – 3/60	35(37.2)	10(13.7)
<3/60	29(30.8)	4(7)
Total	94	72

Table 2: Log mar vision

Log mar vision	N	Median (Snellen's Equivalent)	Mean (SD)	Min-Max	P-Value
Baseline	94	1.49(4/55)	1.34(0.84)	0 – 3.2	-
2 weeks	90	0.36(4/12)	0.65(0.65)	0 – 2.9	<0.001
1 month	73	0.2(8/10)	0.52(0.78)	0 – 2.9	<0.001

Table 3: Fundus parameters

Fundus at baseline	Fundus at 1month		Total	P-value
	Normal	Abnormal		
Normal	6(8.3)	3(4.16)	10	0.180 (Using McNemar's test)
Abnormal	11 (15.2)	52(72.2)	62	
Total	17	55	72	

Table 4: Colour vision

Colour vision at baseline	Colour vision at 1month		Total	P-value
	Normal	Abnormal		
Normal	10 (83)	2 (16)	12	0.0035 (Using McNemar's test)
Dyschromatopsia	54 (90)	6 (10)	60	
Total	64(88.8)	8 (11.12)	72	

Table 5: Central field

Central fields at baseline	Central fields at 1month		Total	P-value
	Normal	Abnormal		
Normal	5(100.0)	-	5	<0.001 (Using McNemar's test)
Abnormal	29(43)	38(56)	67	
Total	31 (43.1)	41(56.9)	72	

Discussion

Optic neuritis, or inflammation of the optic nerve, usually has an idiopathic cause. A week is usually all that is needed for spontaneous recovery from optic neuritis, however it might sometimes take longer. The usual occurrences of optic neuritis associated with multiple sclerosis were not common in India. Several studies have demonstrated the connection between optic neuritis and multiple sclerosis. Prior to ONTT, our work aims to understand visual function after therapy for optic neuritis in South India. The first study to look into the use of oral corticosteroids in treating optic neuritis was the Optic Neuritis Treatment Trial, which contributed to our current understanding of the condition. Following intravenous methyl prednisolone injections (1 gramme once day) for the first three days, all trial participants received oral steroids (1 mg/kg body weight) at progressively lower doses for a duration of 2–6 weeks. There were 47 men and 41 women among the 88 patients in the study, ages ranging from 20 to 64 (mean 40.0). Ten participants had bilateral involvement and 78 instances had unilateral involvement [6, 7].

Similar to this study, Rohit Saxena *et al*. included 83 participants between the ages of 16 and 58. Sixteen of these cases featured bilateral involvement, and the remaining 67 involved unilateral participation. ONTT comprised 457 patients, ages ranging from 18 to 46, with a mean age of 31.8 years. Males were affected in our study more often than females (53.4%). According to Jain *et al*., 67% of participants in a related study were men. Women made about 77% of the affected patients in ONTT. 91.8% of subjects in our study (90 out of 98 eyes) had impaired vision, 12.5% had headaches (11 out of 98 eyes), 20.4% had dyschromatopsia (20 out of 98 eyes), and 29.6% experienced pain when moving their eyes. Parallel to this, Jain *et al*. found that 33.3% of their patients (7 out of 42) had pain during eye movements. In ONTT, 93% of 295 eyes with retrobulbar neuritis and 162 of 295 eyes with papillitis showed signs of pain [8, 9, 10].

During their study, Majid *et al*. noted that 12 people had uncomfortable eye movements. In the majority of the eyes, the baseline visual acuity was lower. At the 1-month follow-up, the visual acuity was 6/24 or better in 15 (19.5%) of the eyes. 45 eyes (58.4%) had visual acuity ranging from 6/18 to 6/6. Pedro *et al*. discovered a 52.4% increase in visual acuity in 18 out of 35 eyes at a later date. Out of the 98 eyes, 56.86 (87.8%) showed RAPD. Twelve (12.2%) of the eyes were normal at first. Fifteen eyes initially had a normal optic disc; forty-four eyes exhibited disc edoema; twenty eyes showed temporal pallor; and twenty eyes showed hyperaemia. There were also splinter hemorrhages in two of the eyes. 35% of individuals have enlarged optic discs, according to ONTT. In 46% of instances, the optic disc was normal, 20% were hyperaemic/blurred, 23% had disc edoema, and 11% had temporal pallor, according to Singhal *et al*. (1985). Retrobulbar neuritis affected 34.7% of patients in our study, while papillitis affected 65.3% of them. In a similar vein, Saxena *et al*. observed that 53.5% of eyes had papillitis and 46.5% of individuals had retrobulbar neuritis. Just 48 eyes

were able to have baseline visual fields completed since the remaining 50 had poor vision. With 22 (22.7) cases, centrocaecal scotoma was the most common field defect. 7.8% of participants in our study had superior field loss, generalized visual field constriction, and inferior and superior field abnormalities. A baseline observation in our investigation revealed that one patient had an elevation field defect, which is common in NAION but can also happen in ON. Consistent with the findings of ONTT, altitudinal field defects accounted for 23.4% of all baseline field defects, followed by central or centrocaecal scotomas (8.3%) and widespread loss (48.2%). 19.1% of eyes had central scotoma, while 25% of eyes had concentric contraction, according to Jain *et al*. [11,12,13].

Dyschromatopsia instances were recorded using Ishihara charts. Results for 86 (87.8%) of the baseline eyes were abnormal. It was average for twelve eyes, or 12.2%. After a month, 53 (68.8%) of the eyes showed improvement. Of the individuals, 24 (31.2%) showed no improvement. Jain *et al*.'s study found that in addition to eyesight improvement, dyschromatopsia had also improved. Saxena *et al*. 48 reported that 60.6% of subjects in their study had better color vision. Vimala *et al*. observed that the dyschromatopsia had improved at a follow-up after one month. (The baseline Mean Log Mar increased from 9.14 to 18.57.) Of the 98 eyes, 83 exhibited abnormal brightness sensitivity at baseline; most of these people could not perform the test due to vision impairments. 43 (55.8%) of the 77 eyes showed improvement after a month-long follow-up. Compared to other ways, intelligence testing proved to be a more accurate diagnostic tool. Red desaturation was observed in 86 out of the 98 baseline abnormal eyes. Forty-three of the 77 eyes that were checked again a month later showed improvement. Two essential components that we employed in our investigation to assess visual function were brightness sensitivity and red desaturation. Almong *et al*.'s investigation found that ON had more desaturation, which is comparable. Only 3 cases of demyelinating lesions were found utilizing neuroimaging in our analysis, suggesting that MS is not very common in South India [14, 15, 16].

In contrast, 48.7% of patients (203 out of 417) had MS-related demyelinating changes, whereas they were present in 37.5% of cases (8 out of 32), according to ONTT. It also stated that there was a 25% risk of MS if the initial MRI came back negative and a 50% likelihood if there was an optic neuritis episode. Saxena *et al*. found that 12 cases of demyelinating lesion and 4 cases of MS in their examination. Southern India has a lower incidence of MS than northern India, according to Jain *et al*. [17].

Conclusion

A range of 20 to 64 years old makes up the median age of 40.0 (12.9) years. Unanimity was shown in 79.6% of situations. Of those, 20.4% had bilateral presentations. Males are somewhat more common than females in our analysis. In our research, impaired vision and eye pain were the most often reported symptoms. There had been notable improvement in optical acuity. One of the most common presentations was retrobulbar neuritis (34.7%), followed by papillitis (65.3%). Skills related to color vision, brightness sensitivity, red desaturation, and central fields all showed notable improvements after treatment. A neuroimaging test for demyelination identified three patients. It was determined that 3.9% of the patients were abnormal and required more observation and study. Our data showed a lower frequency of demyelinating disease linked to ON. Visual function improved significantly after receiving treatment for optic neuritis. This study found a lower prevalence of concurrent demyelination.

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Reference

1. Lepore FE. The origin of pain in optic neuritis. Determinants of pain in 101 eyes with optic neuritis. Arch Neurol. 1991 Jul;48(7):748–9.
2. Glaser JS. Neuro-ophthalmology. 3rd edition. Philadelphia, Pa: Lippincott, Williams & Willkins 1999; 118-98.
3. Miller NR, Newman NJ. Walsh and Hyot's Clinical Neuro- opthalmology. Williams & Willkins, Vol 1; 1998 : 599-648
4. Liu GT, Volpe NJ, Galetta SL, 1st edition. Neuro-ophthalmology Philadelphia Pennsylvania: WB Saunders company 2001: 103-87.
5. Perkin GD, Rose FC. Optic neuritis and its differential diagnosis. Oxford, UK, Oxford medical publications 1979.
6. Rodriguez M, Siva A, Cross SA, O'Brien PC, Kurland LT. Optic neuritis: a population-based study in Olmsted County, Minnesota. Neurology. 1995 Feb;45(2):244–50.
7. Wakakura M, Minei-Higa R, Oono S, Matsui Y, Tabuchi A, Kani K, *et al*. Baseline features of idiopathic optic neuritis as determined by a multicenter treatment trial in Japan. Optic Neuritis Treatment Trial Multicenter Cooperative Research Group (ONMRG). Jpn J Ophthalmol. 1999 Apr;43(2):127–32.
8. Ebers GC. Optic neuritis and multiple sclerosis. Arch Neurol. 1985 Jul;42(7):702–4.
9. The clinical profile of optic neuritis. Experience of the Optic Neuritis Treatment Trial. Optic Neuritis Study Group. Arch Ophthalmol. 1991 Dec;109(12):1673–1678.
10. Singhal BS. Multiple sclerosis--Indian experience. Ann Acad Med Singap. 1985 Jan;14(1):32–6.
11. Das A, Puvanendran K. A retrospective review of patients with clinically definite multiple sclerosis. Ann Acad Med Singap. 1998 Mar;27(2):204–209.
12. Lin YC, Yen MY, Hsu WM, Lee HC, Wang AG. Low conversion rate to multiple sclerosis in idiopathic optic neuritis patients in Taiwan. Japanese journal of ophthalmology. 2006;50(2):170–175.
13. Beck RW, Trobe JD, Moke PS, Gal RL, Xing D, Bhatti MT, *et al*. High- and low-risk profiles for the development of multiple sclerosis within 10 years after optic neuritis: experience of the optic neuritis treatment trial. Arch Ophthalmol. 2003 Jul;121(7):944-949.
14. Beck RW. The optic neuritis treatment trial. Archives of ophthalmology. 1988;106(8):1051–3.
15. Poser CM, Paty DW, Scheinberg L, McDonald WI, Davis FA, Ebers GC, *et al*. New diagnostic criteria for multiple sclerosis: guidelines for research protocols. Ann Neurol. 1983 Mar;13(3):227–231.