Visual Function following Treatment of Optic Neuritis

Majid Abrishami, MD[1]; Mir-Naghi Moosavi, MD[1]; Abbas Azimi-Khorasani, PhD[1]
Azardokht Vazifeshenas, MD[1]

ABSTRACT

Purpose: To evaluate changes in different aspects of visual function including visual acuity, visual field, contrast sensitivity, colour vision and stereopsis in patients with optic neuritis before and after medical intervention.
Methods: In a noncomparative interventional case series on 31 eyes of 30 patients with optic neuritis, the following aspects of visual function were compared before and after treatment. Medical intervention was conducted following the Optic Neuritis Treatment Trial (ONTT) guidelines. Visual function was assessed by evaluating changes in visual acuity, visual field (Goldmann in all patients and automated in some patients), contrast sensitivity using Cambridge low contrast grating, colour vision using Ishihara plates and stereopsis using Titmus stereoacuity test.
Results: Visual acuity was significantly lower (3/10) in the affected eyes than unaffected eyes (8/10) [P<0.001]. Contrast sensitivity was also significantly better in the unaffected eyes. Mean stereoacuity was 310 sec/arc. The visual field impairment was also significantly higher than that of the unaffected eye and also normal population sample. Weak deutan defects were present in 60% of the patients. After medical treatment, visual acuity, visual field defects, contrast sensitivity, colour and stereopsis were significantly improved.
Conclusion: Different aspects of visual function including visual acuity, visual field, contrast sensitivity, colour vision, and stereopsis are impaired in optic neuritis. Medical treatment with intravenous methylprednisolone followed by oral steroids is effective in improving these parameters. However, some deficits may persist after therapy. Since spontaneous recovery after optic neuritis is common, clinical trials are needed to determine the true effect of treatment versus follow-up.

Introduction

Optic neuritis, a common inflammatory disorder of the optic nerve, is often associated with demyelinating diseases. Optic neuritis is more prevalent in women aged 15-45 years. Patients most often experience sudden onset of decreased vision, which is aggravated during the first week and followed by gradual recovery. In most cases, patients recover completely. In addition to decreased visual acuity, other aspects of visual function such as contrast sensitivity, colour vision, stereopsis (especially in moving objects) and visual field are affected but do not recover fully.[1,2] Some studies indicate no correlation between initial visual acuity or treatment and residual visual function disturbances.[3] From a histopathologic standpoint, optic neuritis is an acute demyelinating lesion of the optic nerve.[4]

[1] Mashad University of Medical Sciences, Iran

The present study was performed to evaluate changes in different aspects of visual function including visual acuity, visual field, contrast sensitivity, colour vision and stereopsis in patients with optic neuritis before and one week after medical treatment at Imam Reza Hospital, Mashad, Iran.

Methods

This non-comparative interventional case series was conducted at Imam Reza Hospital, Mashad, Iran from March 2001 to March 2002. Patients were referred with a diagnosis of optic neuritis based on acute decrease in visual acuity, painful eye movements, relative afferent pupillary defect (RAPD), colour desaturation and funduscopic findings. Ischemic optic neuropathy was ruled out based on typical clinical course of the disease, lack of predisposing systemic factors such as hypertension or diabetes mellitus and lack of suggestive funduscopic findings such as pale swelling of the optic disc. In the presence of neurologic symptoms other than those related to typical optic neuritis such as paresthesia and paralysis, neurologic consultation was performed and MRI was obtained to detect multiple sclerosis (MS) plaques. However, patients with isolated optic neuritis did not undergo neuroimaging. MS was considered in patients with recurrent attacks of optic neuritis. The rest of the patients were diagnosed as idiopathic optic neuritis. Only patients with typical symptoms of optic neuritis were enrolled in the study; cases who did not have typical signs and symptoms of optic neuritis or developed other signs such as retinitis were excluded from the study.

All patients underwent the following work-up before and after intervention: snellen visual acuity, visual field evaluation using Goldmann perimetry in all subjects and Humphrey automated perimetry in selected cases, contrast sensitivity using Cambridge low contrast grating test, stereopsis using the Titmus stereoacuity test and colour vision using Ishihara color plates. The time interval between onset of symptoms and referral was also documented.

After conducting the basic workup, patients were hospitalised and treated with intravenous methylprednisone 250mg every 6 hours for 3 days followed by oral prednisone 1mg/kg for 11 days based on the Optic Neuritis Treatment Trial (ONTT) reccomendations. Tests were conducted before initiation and repeated one week after completion of treatment (three weeks after admission). Mean test values before and after treatment were compared using paired t-test and P values less than 0.05 were considered significant.

Results

This study included 31 eyes of 30 patients with optic neuritis including 17 female and 13 male subjects aged 31.3±8.9 years. Overall, 60% of the patients were 20-40 years of age and of the 31 enrolled eyes 15 were left, 14 were right and involvement was bilateral in one patient. All 30 patients had decreased visual acuity, 12 patients had painful eye movements, 13 patients had painful eye movements and headaches and 5 patients had headaches. Mean visual acuity was 3/10 in the affected eyes and 8/10 in the unaffected eyes ($P<0.0001$). Fundoscopy was unremarkable in 22 patients. Optic nerve head swelling with peripapillary hemorrhage or disc hyperemia was present in 5 and 3 patients respectively.

Titmus stereotest showed normal stereopsis (stereoacuity better than 80 sec/arc) in 4 patients; 100-180 sec/arc in 11; and more than 200 sec/arc in 15. Mean stereoacuity was 310 sec/arc which was different from a normal value of 40 sec/arc ($P<0.001$).

Mean contrast sensitivity was 260±145 cycle/degree in unaffected eyes and 50±45 cycle/degree in affected eyes. Mean normal contrast sensitivity based on age was 188±66 cycle/degree. Contrast sensitivity in affected eyes was significantly worse than normal ($P<0.0001$). Mean contrast sensitivity

in unaffected eyes was better than normal (P<0.01). Mean contrast sensitivity in eyes with optic neuritis was significantly different from the unaffected eyes (P<0.0001). (Figures 1-3)

III-4e and V-4e targets were used to detect scotomata and generalized constriction respectively. The V-4e isopter was not significantly different between affected and unaffected eyes. Humphrey automated perimetry (30-2 program, SITA standard strategy) was performed in 10 patients which was similar to the central field obtained by Goldmann perimetry. Perimetry revealed central scotoma in 21 patients (65.2%), paracentral scotoma in two (8.7%), arcuate scotoma in two (8.7%), superior constriction in one (4.3%) and enlargement of the blind spot in one (4.3%). The horizontal and vertical diameters of the blind spot were measured in all patients (figure 4). The normal horizontal and vertical diameters are 10° and 12° respectively.

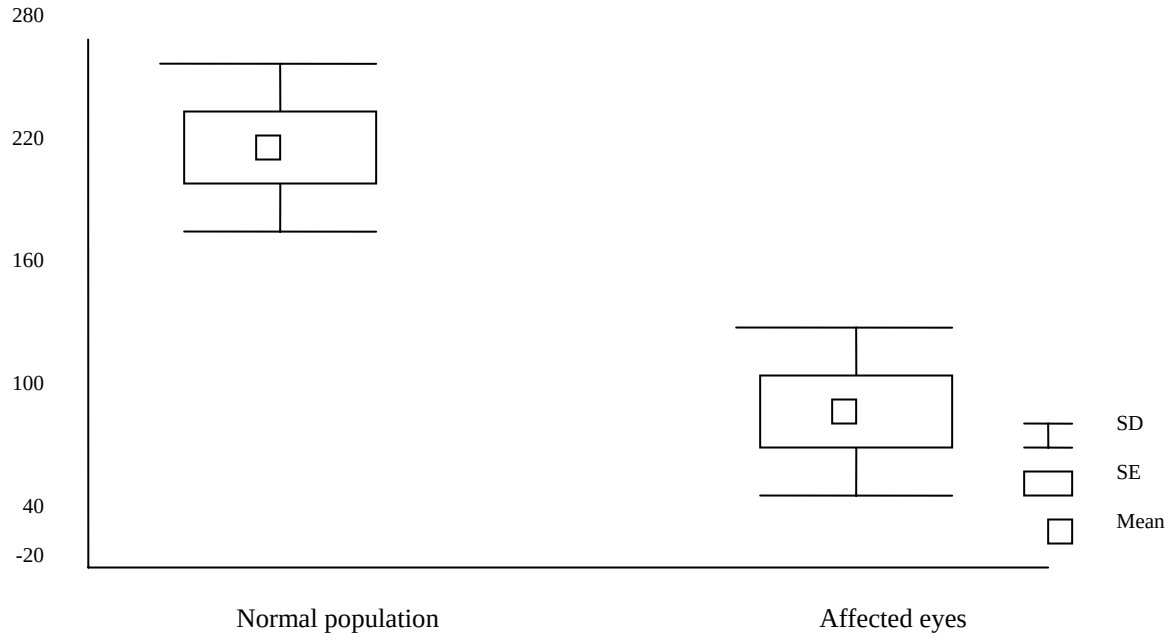

Figure 1. Comparison of contrast sensitivity in affected eyes with that of normal population before intervention

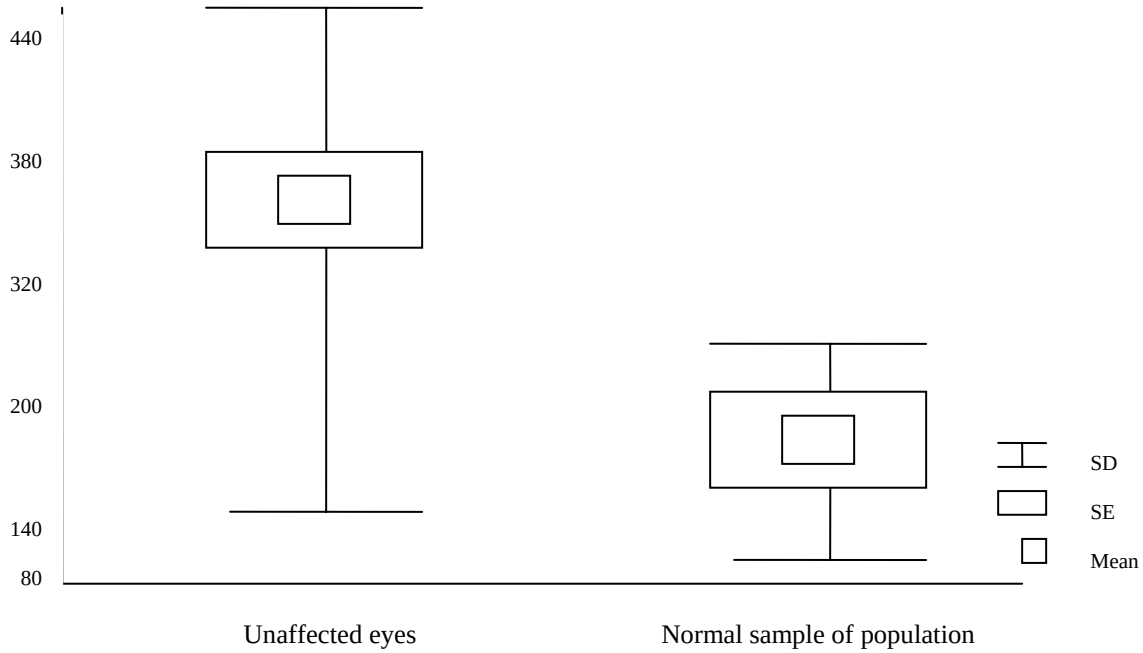

Figure 2. Contrast sensitivity in unaffected eyes versus normal population before intervention.

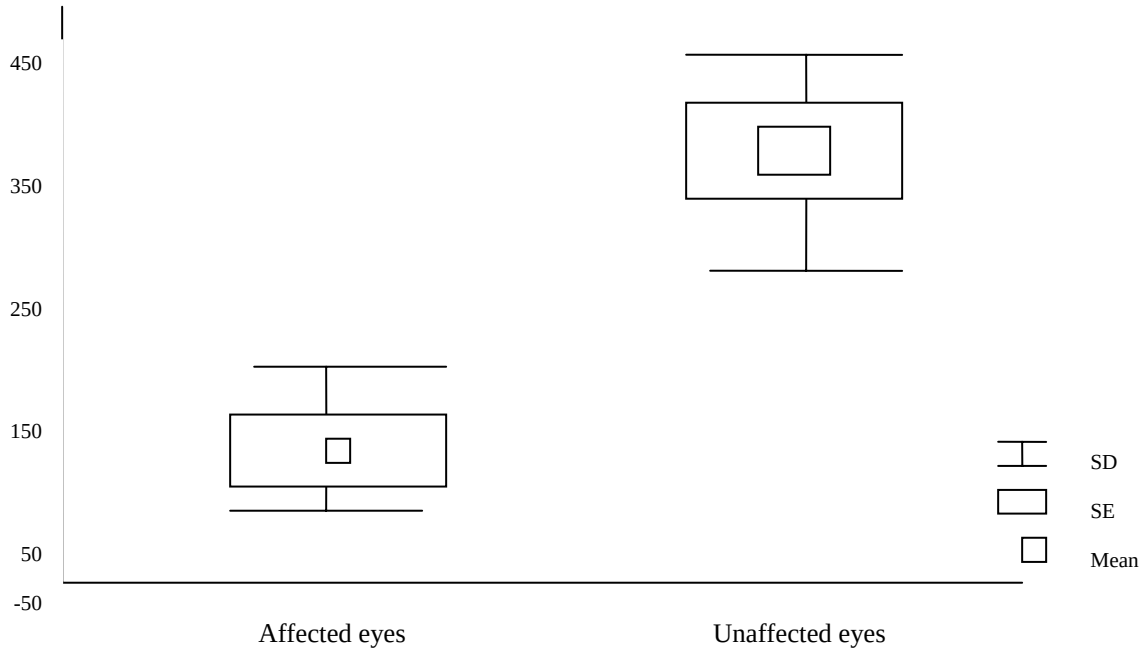

Figure 3. Comparison of contrast sensitivity in affected and unaffected eyes before intervention.

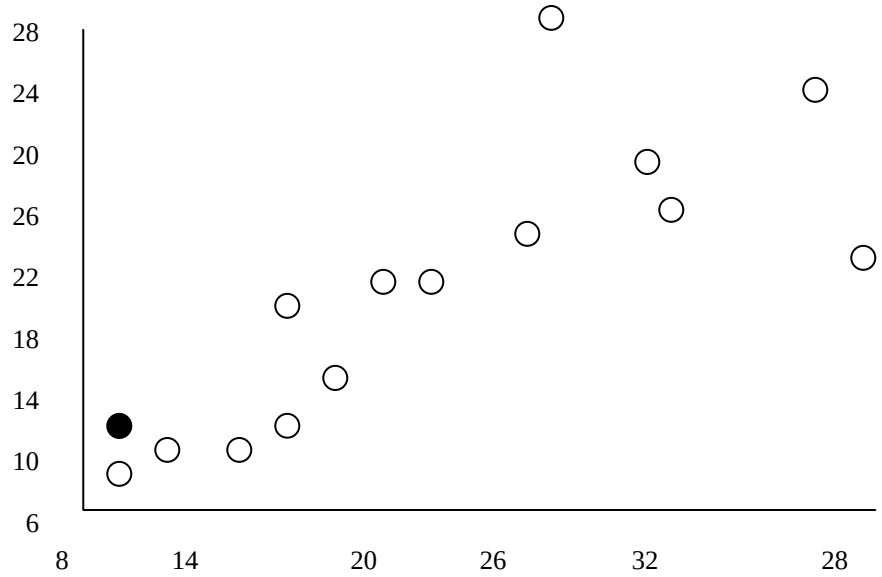

Figure 4. Frequency of the affected eyes according to different blind spot sizes before intervention. (black circle is normal size)

Colour vision was examined using Ishihara colour plates; 60% of the patients showed weak deutan disturbances manifesting as inability to read two of the plates numbered 8 to13 accompanied by inability to clearly see six pages, or inability to read four pages accompanied by inability to clearly see two pages as compared to the unaffected eye. Inability to see more Ishihara plates is regarded as intensive deutan defect, which was not observed in any patient.

One week after medical intervention, the same tests and examinations were conducted and compared. Visual acuity, contrast sensitivity and stereopsis were significantly improved after intervention ($p<0.01$) (Table 1). As shown in figure 5, after treatment, blind spot size became closer to normal in the affected eyes.

Table1. Contrast sensitivity, colour vision and stereopsis before and after treatment

Intervention	visual acuity	contrast sensitivity	stereopsis
Before	3/10	50±45	310
After	7/10	209±114	60
P value	<0.001	<0.0001	<0.01

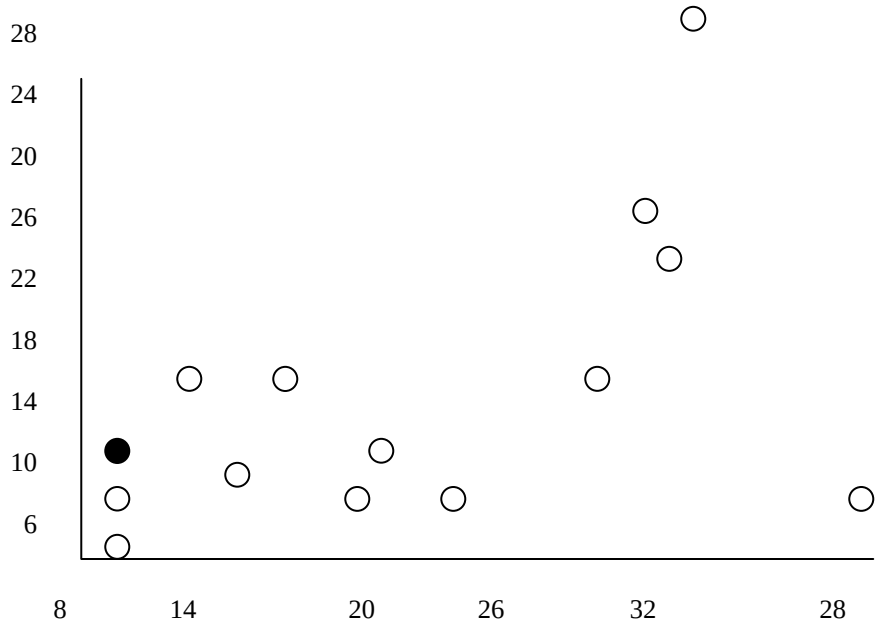

Figure 5. Distribution of affected eyes according to different blind spot sizes after treatment. (black circle is normal size)

Discussion

According to the present study, visual acuity, contrast sensitivity and stereopsis in eyes with optic neuritis were significantly worse than those in unaffected fellow eyes and the normal population. However after treatment, contrast sensitivity, visual acuity and stereopsis improved significantly and blind spot size also decreased. Medical intervention for optic neuritis led to less dramatic improvement in contrast sensitivity which complies with studies demonstrating that contrast sensitivity is more severely affected than other aspects of visual function.[5]

Decreased colour vision was observed in 60% of our patients which was lower compared to other studies. One possible explanation for this discrepancy may be the low sensitivity of Ishihara colour plates compared to other color vision tests such as Munsell 100 hue test. In one study using Munsell 100 hue test certain color combinations such as blue-yellow and also red-yellow were difficult for patients to perceive, however the differences were not significant.[6]

One interesting finding was that contrast sensitivity in unaffected fellow eyes was better than that of the normal population in other studies.[7,8] One possible explanation for this discrepancy is that contrast sensitivity in the Iranian population has not been standardized yet.

Magnetic resonance neuroimaging was not performed in patients with isolated and typical optic neuritis because of the general belief that such patients do not need neuroimaging. Other studies support the role of MRI in the determining disease prognosis. One study indicated that optic nerve involvement of less than 17.5 mm has better prognosis than greater than 17.5 mm and/or with intracanalicular involvement.[9] The findings of such studies suggest MRI for prognostic purposes, however the purpose of our study was to examine the therapeutic efficacy of the clinical rather than determining the prognosis of the disease.

Based on the results of our study there were significant improvements in signs and symptoms of optic neuritis after treatment, however certain deficits persisted. This provides support for other studies which reported visual acuity, contrast sensitivity and visual field disturbances to be present six months after treatment.[10] Another study stated that colour vision deficits (57%), contrast

sensitivity impairment (77%), visual field disturbances (26%) and stereopsis abnormalities (80%) may persist after recovery of optic neuritis.[11] One study demonstrated persistent visual evoked potential abnormalities after treatment of optic neuritis and improvement in signs and symptoms.[12]

The results of the current study indicate that visual function recovers after treatment, which is in accordance with other studies.[13] However other reports indicate that patients treated with pulse therapy with methylprednisone showed a faster improvement in the early stages but no significant difference was observed between the intervention group and control group during follow-up.[14]

It may be concluded that different aspects of visual function are impaired in optic neuritis, however, medical treatment using intravenous methylprednisone followed by oral prednisone improves these deficits. We recommend further studies comparing the efficacy of medical treatment with a control group receiving placebo.

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