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Original Research Article

Evaluation of the etiological factors in acute optic neuropathy

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Abstract

Objectives: To evaluate the etiological factors in patients of acute optic neuropathy reporting to tertiary care centre and to assess the sensitivity of various tests for optic neuropathy.
Materials and Methods: All the patients selected were subjected to detailed ocular examination which included best corrected visual acuity, pupillary reaction, colour vision, visual evoke potential, Visual field assessment and CT & MRI. After ocular and systemic examination and relevant investigations the underlying etiological diagnosis of acute optic neuropathy was established.
Results: A total of 30 patients with acute optic neuropathy presenting in the tertiary care centre during the study period out of which majority of the patients with acute optic neuropathy, that is, 43.3% had optic neuritis as their etiology, followed by 36.7% cases who had traumatic optic neuropathy. Only 20% cases had AION as a cause for acute optic neuropathy.
Conclusion: The present study concludes that optic neuritis is most important cause for acute optic neuropathy. VEP is most important test to detect and confirm optic neuropathy. NAION is associated with a poor visual outcome in spite of timely intervention with corticosteroids.

Keywords: Acute optic neuropathy, Traumatic optic neuropathy, Acute ischemic optic neuropathy, Optic neuritis.

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1. Introduction

The term “Optic neuropathy” describes optic nerve damage due to various causes. The damage to the optic nerve leads to characteristic features of optic neuropathy which include diminution of vision, afferent pupillary defect, dyschromatopsia, change in optic disc and visual field defect (unilateral).[1] Optic neuropathy may results from various etiologies including congenital or acquired causes. Congenital causes include mitochondrial disorders or hereditary illness whereas acquired causes may result in acute or chronic optic neuropathy. Causes underlying acute optic neuropathy include inflammation, ischemia, demyelinating disorders, and trauma. Acquired chronic optic neuropathy may results from associated compression of optic nerve, infiltration, nutritional deficiencies or toxic substances.[1]

The exact incidence of optic neuropathies is not known. Optic neuritis is one of the most common causes of optic neuropathy.[2]

Laboratory investigations such as complete blood count, ESR, CRP, virological assays (HIV/HSV) or bacterial assays (VDRL) are helpful in cases with inflammatory etiology. In cases with systemic or ischemic optic neuropathy, RBS, lipid profile may be raised.

Visually evoked potential (VEP), Visual field examination (HVF 30-2) along with changes in optic disc, neuroimaging (CT/MRI), Optical coherence tomography

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(OCT) may be helpful in establishing the definitive etiological diagnosis.[3]

Previous studies have focused on individual etiologies of optic neuropathy. Data evaluating the etiological factors and their outcome following acute optic neuropathy are lacking especially in Indian scenario. With the above background, the present study was conducted at tertiary care centre to evaluate the etiological factors, the role of investigations in cases with acute optic neuropathy.

2. Materials and Methods

2.1. Study design

This was a prospective observational study conducted in the department of ophthalmology Gandhi medical college, Bhopal and Kamla Nehru Hospital Bhopal.(M.P.).This study was done from October 2019 to May 2021 after obtaining approval from the institutional ethics committee.

2.2. Sample size

All the patients diagnosed with acute optic neuropathy with recent onset of impaired vision were included using purposive sampling

2.3. Inclusion criteria

1. Patients of optic neuropathy with recent onset (within hours to days) of impaired vision
2. No H/O previous treatment with steroid for optic neuritis in other eye.

2.4. Exclusion criteria

1. Preexisting/coexisting ocular diseases
   a. Old and treated case.
   b. Traumatic optic neuropathy associated with severe head injury
   c. Traumatic optic neuropathy associated with berlin edema.

All the patients were then subjected to detailed ocular examination which included

1. Best corrected visual acuity on Snellen's E-chart.
2. Bright light used in dark room to detected pupillary reaction was tested for relative afferent pupillary defect by performing the swinging light-pupil test.
3. Colour vision was tested using Ishihara pseudoisochromatic chart 38 plates edition.
4. Posterior segment examination was done with indirect ophthalmoscope.
5. Contrast enhance MRI (brain+ orbit) was done in acute optic neuropathy patient to rule out space occupying lesions and demyelinating diseases.
6. Visual field assessment was done with the help of Humphrey visual field analyser using 30-2 program
7. VEP done in all patients.

After ocular and systemic examination and relevant investigations the underlying etiological diagnosis of acute optic neuropathy was established.

2.5. Stastistical analysis

Data was compiled using MsExcel and analysed using IBM SPSS software version 20. All the variables were grouped as per mathematic transformation into nominal/ Ordinal/interval and ratio. Extent of type one error was measured with parametric analysis. Z test was applied for proportion and t-test was used to find out any significant difference among detected proportion and mean. Chi square test was applied at appropriate places. P value less than 0.05 was considered statistically significant.

3. Observation Results

The study was conducted on a total of 30 patients with acute optic neuropathy presenting in the tertiary care center during the study period.

In the present study, majority of the patients with acute optic neuropathy, that is, 43.3% had optic neuritis as their etiology, followed by 36.7% cases who had traumatic optic neuropathy. Only 20% cases had AION as a cause for acute optic neuropathy. (**Table 1**)

Table 1: Etiology of acute optic neuropathy

Diagnosis	Frequency (n = 30)	Percentage
AION	6	20
Traumatic optic neuropathy	11	36.7
Optic neuritis	13	43.3

AION- Acute ischemic optic neuropathy

The mean age of patients with acute optic neuropathy was found to be 38.57±16.59 years in our study. Majority of the patients, 66.6% (n=20) belonged to 19 to 45 year age group. The mean age of presentation of AION was 59.2 years, the same for optic neuritis was 36.6 years and that in traumatic optic neuropathy was found to be 38.5 years. Thus, AION presented in majority of the older subjects (50–70 years), whereas optic neuritis and traumatic optic neuropathy were more common in younger subjects (10–50 years). (**Table 2**)

Table 2: Age distribution in cases of acute optic neuropathy

Diagnosis	Frequency (n= 30)	Mean age	Age (Max)	Age (Min)
AION	6	59.2	68	50
Optic neuritis	13	28.6	45	15
Traumatic optic neuropathy	11	38.5	50	12

AION: Acute ischemic optic neuropathy

In the present study, females (76.92%) were more commonly affected compared to males in cases with optic neuritis. Traumatic optic neuropathy was found to be predominant in males (81.81%), while in cases with AION, there was no gender predilection. (**Figure 1**)

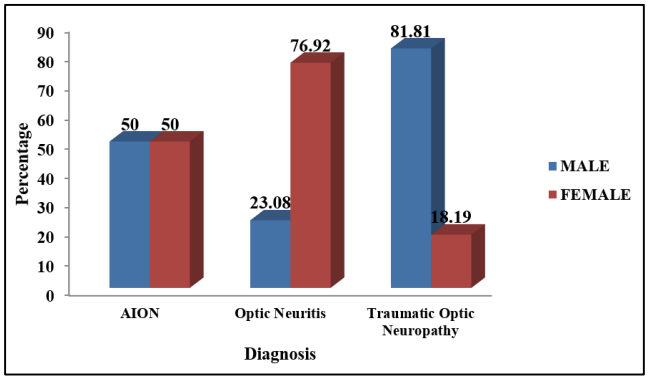

Figure 1: Distribution of patients according to gender in acute optic neuropathy
(AION: Acute ischemic optic neuropathy, TON: Traumatic optic neuropathy)

In the present study, better vision was found to be in cases with Optic Neuritis compared to the cases with AION and traumatic optic neuropathy where majority of the cases had poor vision (<1/60).

All the cases, that is, 6 (100%) cases with AION had poor vision (<1/60).

In cases with Optic Neuritis, 3 (23.07%) cases had 6/6 – 6/12 vision, 1 (7.69%) case had 6/18 – 6/36 vision, 4 (30.76%) cases had 6/60 – 2/60, whereas majority, 5 (38.46%) cases of this group also had poor vision (<1/60).

In cases with traumatic optic neuropathy, a maximum of 8 (72.72%) cases had poor vision (<1/60) whereas 1 (9.09%) case each had a vision of 6/6 - 6/12, 6/18 – 6/36 and 6/60 – 2/60.(**Table 3**)

Table 3: Distribution of patients according to BCVA in affected eye

Visual acuity	Etiology		
	AION (6)	OPTIC Neuritis (13)	Traumatic optic neuropathy (11)
6/6 – 6/12	0	3(23.07%)	1(9.09%)
6/18 – 6/36	0	1(7.69%)	1(9.09%)
6/60 – 2/60	0	4(30.76%)	1(9.09%)
<1/60	6(100%)	5(38.46%)	8(72.72%)
Total	30		

*BCVA: Best corrected visual acuity, AION: Acute ischemic optic neuropathy

In the present study, all the cases had abnormal pupillary response, where in, RAPD was found in all the cases 100% (n=13) of optic neuritis, 83.3% (n=5) cases of AION and 81.8% (n=9) cases of traumatic optic neuropathy. Sluggish pupillary Reaction to light was found in a single (16.7%) case of AION, while 2 (18.18%) cases of Traumatic optic neuropathy had afferent pupillary defect. (**Figure 2**)

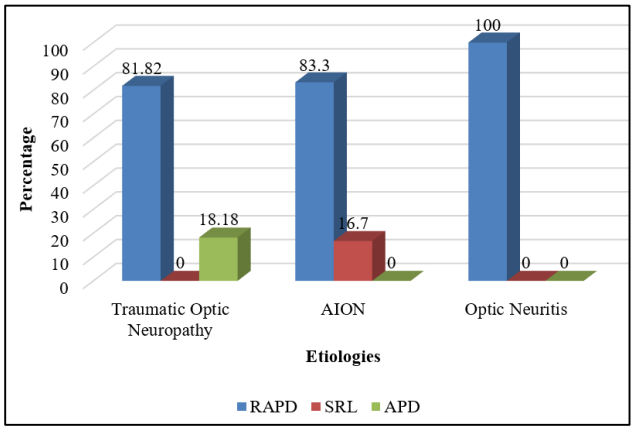

Figure 2: Distribution of patients according to pupillary reaction in affected eye
(RAPD: Relative afferent pupillary defect, SRL: Sluggish reactive to light, APD: Afferent pupillary defect)

In the present study, colour vision could not be recorded in 17(56.7%) cases of acute optic neuropathy with AION, optic neuritis and traumatic optic neuropathy etiologies due to poor vision (<1/60). In those subjects in whom colour vision could be recorded, Red green deficit was the most common colour vision defect seen among 8(61.54%) cases of optic neuritis, where as it was found in a single (16.67%) case of AION and 2(18.18%) cases of traumatic optic neuropathy. No colour vision defect was seen in a single case each of Optic Neuritis (7.69%) and traumatic optic neuropathy (9.09%).(**Table 4**)

Table 4: Distribution of patients according to colour vision in affected eye

Colour vision	Diagnosis		
	AION (6)	Optic Neuritis (13)	Traumatic Optic Neuropathy (11)
Could not be recordable	5(83.33%)	4(30.76%)	8(72.72%)
Red and green deficit	1(16.67%)	8(61.54%)	2(18.18%)
WNL	0(0)	1(7.69%)	1(9.09%)
Total	6	13	11

AION: Acute ischemic optic neuropathy, WNL: Within normal limit

In the present study, the most common optic disc findings in AION were disc pallor with blurred disc margins seen in 100% (n=6) cases, with disc haemorrhages seen in the peripapillary region in 83.33% (n=5), which was absent in a single case.

Table 5: Distribution of patients according to fundus examination in affected eye in cases of AON

Diagnosis	Fundus finding					
	Margin		Hyperemia		Disc Hemorrhage	
	Blurred	Normal	Present	Absent	Present	Absent
AION (6)	6	0	0	6	5(83.33%)	1
Optic neuritis (13)	5 (38.46%)	8 (61.53%)	5 (38.46%)	8 (61.53%)	0	13 (100%)
Traumatic optic neuropathy (11)	0	11(100%)	0	11 (100%)	0	11(100%)

AON: Acute optic neuropathy, AION: Acute ischemic optic neuropathy

Most of the cases, 61.53% (n=8) of optic neuritis had normal disc findings with no hyperemia or blurring of disc margins as they had retrobulbar neuritis. Rest 5 (38.46%) cases of optic neuritis had hyperemic discs with blurred disc margins. Normal disc findings were seen in 100% (n = 13) cases of Traumatic Optic Neuropathy. (**Table 5**)

In the current study, decreased amplitude with increased latency was observed in VEP of all 13(100%) cases of Optic Neuritis, 5 (83.3%) cases of AION and 7 (63.6%) cases of traumatic optic neuropathy. VEP was non recordable in a single (16.6%) case of AION and 3 (27.3%) cases of traumatic optic neuropathy, while only a single (9.1%) case of traumatic optic neuropathy had a normal VEP.(**Figure 3**)

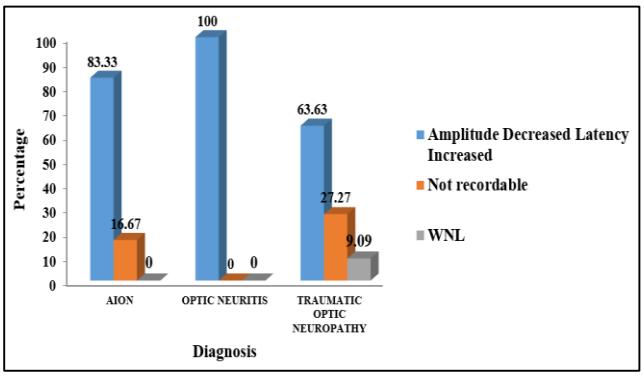

Figure 3: Distribution of patients according to visual evoked potentials in affected eye
(WNL: Within normal limit)

In the present study, field charting could not be done in 83.3% (n=5) cases of AION, 30.7% (n=4) cases of optic neuritis and 72.7% (n=8) cases of traumatic optic neuropathy, due to poor vision (<1/60). In those subjects in whom perimetry could be recorded, only a single (16.7%) case of AION showed Altitudinal field defect with 0 cases of both optic neuritis and traumatic optic neuropathy. While, 69.2% (n=9) cases of Optic neuritis and 18.2% (n=2) cases of Traumatic Optic Neuropathy had centrocaecal scotoma. Of all the cases, only a single (9.1%) case of traumatic optic neuropathy had no field defects. (**Figure 4**)

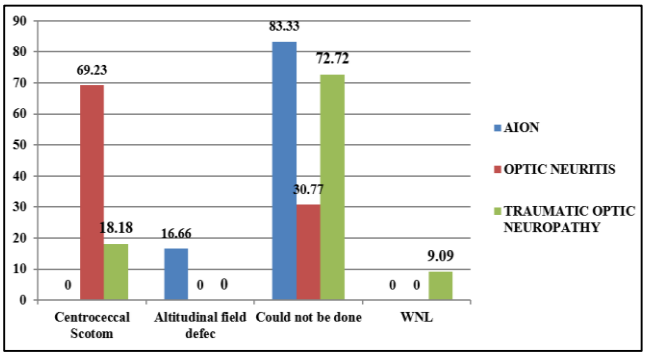

Figure 4: Distribution of patients according to perimetry testing affected eye
AION: Acute ischemic optic neuropathy, WNL: Within normal limit

4. Discussion

In the present study, majority of the patients with acute optic neuropathy, that is, 43.3%, had optic neuritis as their etiology, followed by 36.7% cases who had traumatic optic neuropathy. Only 20% cases had AION as a cause for acute optic neuropathy. This is similar to the cross-sectional observational study done by Pandey R et al[4] on etiological profile of optic neuropathy in which majority of the cases had idiopathic optic neuritis (35%) which was found to be the most common cause of optic neuropathy whereas anterior ischemic optic neuropathy was present in (7.5%) cases. The results of the present study are comparable to the study done by Pandey R et al as both are observational studies done in a tertiary eye care centre with a limited sample size.

4.1. Visual acuity

In the present study, better vision was found to be in cases with optic neuritis compared to the cases with AION and traumatic optic neuropathy where majority of the cases had poor vision (<1/60).

All the cases, that is, 6 (100%) cases with AION had poor vision (<1/60). This was comparable to other studies conducted by Atkins et al[5] which reported similar visual acuity at the time of presentation in 35 to 53% of the patients included.

In cases with optic neuritis, 3 (23.07%) cases had 6/6 – 6/12 vision, 1 (7.69%) case had 6/18–6/36 vision, 4 (30.76%) cases had 6/60–2/60, whereas majority, 5 (38.46%) cases of this group also had poor vision (<1/60). This was comparable

to the optic neuritis treatment trial where 11% of the patients had visual acuity of 6/6 or better.

4.2. Pupillary reaction

In the present study, all the cases had abnormal pupillary response, where in, RAPD was found in all the cases 100% (n=13) of optic neuritis, 83.3% (n=5) cases of AION and 81.8% (n=9) cases of traumatic optic neuropathy. Sluggish pupillary reaction to light was found in a single (16.7%) case of AION, while 2 (18.18%) cases of traumatic optic neuropathy had afferent pupillary defect.

This is similar to study done by T A Cox, H S Thompson[6] measured relative afferent pupillary defects in 105 patients and detected pupillary defects in 96% of acute unilateral cases.

4.3. Colour vision

In the present study, colour vision could not be recorded in 17(56.7%) cases of acute optic neuropathy with AION, optic neuritis and traumatic optic neuropathy etiologies due to poor vision (<1/60). In those subjects in whom colour vision could be recorded, Red green deficit was the most common colour vision defect seen among 8(61.54%) cases of optic neuritis, where as it was found in a single (16.67%) case of AION and 2(18.18%) cases of traumatic optic neuropathy. No colour vision defect was seen in a single case each of optic neuritis (7.69%) and traumatic optic neuropathy (9.09%).

The results are correspondent to the study conducted by Stephen C. Pollock et al[7] on Colour vision in anterior ischemic optic neuropathy where records of forty-five patients with non-arteritic AION who had been evaluated at Duke University over a consecutive four-year period were reviewed retrospectively.

4.4. Ophthalmoscopic findings at the time of presentation

In the present study, the most common optic disc findings in AION were disc pallor with blurred disc margins seen in 100% (n=6) cases, with disc haemorrhages seen in the peripapillary region in 83.33% (n=5), which was absent in a single case.

Most of the cases, 61.53% (n=8) of optic neuritis had normal disc findings with no hyperemia or blurring of disc margins as they had retrobulbar neuritis. Rest 5 (38.46%) cases of optic neuritis had hyperemic discs with blurred disc margins.

Normal disc findings were seen in 100% (n = 13) cases of traumatic optic neuropathy.

The results are also comparable to the study done by Beck RW, et al[256] in which 66% cases had optic disc normal but edematous in 33% cases.

In the study conducted by T.N. Dubey et al[9] on Clinical and neuroradiological profile on patients of optic neuritis in Central India, 75 eyes were examined for fundus. 44 eyes (58.6%) showed optic disc hyperemia or blurring of disc margin (Papillitis) while 31(41.4%) eyes were normal on fundus examination.

In the study conducted by SS Hayreh et al[10] on Ischaemic optic neuropathy they observed that, initially, the optic disc is oedematous, which may be more marked in one part of the disc than the other. Frequently there are splinter haemorrhages at the disc margin. Gradually the optic disc develops pallor and the oedema starts to resolve.

In the study conducted by MIM Kyung Kim[11] on analysis of fundus photography and fluorescein angiography in nonarteritic anterior ischemic optic neuropathy and optic neuritis, 23 patients with NAION had disc hypermia absent in 100% cases, diffuse swelling present in 70% cases, sphincter haemorrhage present in 56.5% cases.

In the prospective observational study conducted by Jainy Joseph Emmatty et al[12] on optic neuritis treatment trial protocol in patients with traumatic optic neuropathy, 26 patients were included out of which all the patients had normal fundus findings.

The results are also similar to the study done by M. G. Rajiniganth et al[13] on 44 patients with posttraumatic indirect optic nerve injury, where on initial fundus evaluation, a normal optic disc was seen in 40/44 patients (91%).

4.5. VEP finding at time of presentation

In the current study, decreased amplitude with increased latency was observed in VEP of all 13(100%) cases of optic neuritis, 5 (83.3%) cases of AION and 7 (63.6%) cases of traumatic optic neuropathy. VEP was non recordable in a single (16.6%) case of AION and 3 (27.3%) cases of traumatic optic neuropathy, while only a single (9.1%) case of traumatic optic neuropathy had a normal VEP.

The results are comparable to the study conducted by Clare L Fraser et al,[14] where no abnormality was recorded on VEP in the control group. Of all the eyes with ON, 74 (97.3%) were abnormal on VEP testing. Amplitude values were abnormal in 92.6% cases.

4.6. Visual field defects on automated perimetry at the time of presentation

In the present study, field charting could not be done in 83.3% (n=5) cases of AION, 30.7% (n=4) cases of Optic Neuritis and 72.7% (n=8) cases of Traumatic Optic Neuropathy, due to poor vision (<1/60). In those subjects in whom perimetry could be recorded, only a single (16.7%) case of AION showed altitudinal field defect with 0 cases of both optic neuritis and traumatic optic neuropathy. While, 69.2% (n=9) cases of Optic neuritis and 18.2% (n=2) cases of traumatic optic neuropathy had centrocaecal scotoma. Of all the cases, only a single (9.1%) case of traumatic optic neuropathy had no field defects.

The results are also similar to a study done by Gerling J, et al,[15] in which most common visual field defect central scotoma in 86% cases of acute optic neuropathy.

5. Conclusion

The present study concludes that optic neuritis is most important cause for acute optic neuropathy. RAPD is most sensitive indicator to indicate optic nerve involvement.

Colour vision and visual fields unable to performed in which patients present with poor vision.

6. Authors Contribution

All authors have contributed to study design, manuscript writing, and review, data analysis, and article finalization.

7. Conflict of Interest

None.

8. Author Funding or Author Sponsorship

None.

9. Acknowledgement

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References

1. Rath EZ. Visual Loss in Neuro-Ophthalmology [Internet]. Causes and Coping with Visual Impairment and Blindness. In Tech; 2018. Available from: http://dx.doi.org/10.5772/intechopen.80682
2. Sanz-Morello B, Ahmadi H, Vohra R, Saruhanian S, Freude KK, Hamann S, et al. Oxidative Stress in Optic Neuropathies. Antioxidants (Basel). 2021;10(10):1538.
3. Mustafa S, Pandit L. Approach to diagnosis and management of optic neuropathy. *Neurol India*. 2014;62(6):599–605.
4. Pandey R, Bihari R. Etiological profile of optic neuropathy in tertiary hospital in eastern India- A cross sectional observational study. *Int J Sci Eng Res*. 2017;8(4):1144–55.
5. Atkins EJ, Bruce BB, Newman NJ, Biousse V. Treatment of nonarteritic anterior ischemic optic neuropathy. *Surv Ophthalmol*. 2010;55(1):47–63.
6. Corbett JJ, Thompson HS, Musser C, Kardon RH, Farrell T, Graff-Redford NR, et al. The clinical profile of optic neuritis. Experience of the optic neuritis treatment trial. Optic Neuritis Study Group. *Arch Ophthalmol.* 1991;109(12):1673–8.
7. Pollock SC, Hubbe RE. Color vision in anterior ischemic optic neuropathy. *Am J Ophthalmol Case Rep*. 2020;19:100823.
8. Beck RW, Cleary PA, Backlund JC. The course of visual recovery after optic neuritis. Experience of the Optic Neuritis Treatment Trial. *Ophthalmology.* 1994;101(11):1771–8.
9. Dubey TN, Gupta A. Study on Clinical and Neuroradiological Profile on Patients of Optic Neuritis in Central India. *J Med Sci Clin Res.* 2017;6(5):23430–5.
10. Hayreh SS, Zimmerman MB. Non-arteritic anterior ischemic optic neuropathy: role of systemic corticosteroid therapy. *Graefes Arch Clin Exp Ophthalmol.* 2008;246(7):1029–46.
11. Kim MK, Kim US. Analysis of Fundus Photography and Fluorescein Angiography in Nonarteritic Anterior Ischemic Optic Neuropathy and Optic Neuritis. *Korean J Ophthalmol*. 2016;30(4):289–94.
12. Emmatty JJ, Narayan S, Cherayath RK. Trial for a trial is there hope? – Optic Neuritis Treatment Trial protocol in patients with traumatic optic neuropathy. *Kerala J Ophthalmol*. 2020;32(3):258–62.
13. Rajiniganth MG, Gupta AK, Gupta A, Bapuraj JR. Traumatic optic neuropathy: visual outcome following combined therapy protocol. *Arch Otolaryngol Head Neck Surg.* 2003;129(11):1203–6.
14. Fraser CL, Klistorner A, Graham SL, Garrick R, Billson FA, Grigg JR. Multifocal visual evoked potential analysis of inflammatory or demyelinating optic neuritis. *Ophthalmology*. 2006;113(2):323.
15. Gerling J, Meyer JH, Kommerell G. Visual field defects in optic neuritis and anterior ischemic optic neuropathy: distinctive features. *Graefes Arch Clin Exp Ophthalmol.* 1998;236(3):188–92.

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