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Original Research Paper

Ophthalomology

CLINICAL PROFILE AND VISUAL OUTCOMES OF PATIENTS WITH OPTIC NEUROPATHY AT A TERTIARY HEALTH CARE CENTER IN WESTERN INDIA

Dr Sujit M Murade	Associate Professor, Department Of Ophthalmology, Lokmanya Tilak Municipal Medical College And Sion Hospital, Mumbai, Maharashtra University Of Health Sciences, Maharashtra
Dr Darshana B Rathod	Associate Professor, Department Of Ophthalmology, Topiwala National Medical College And BYL NAIR Hospital, Maharashtra University Of Health Sciences, Maharashtra
Dr Komal Patekar	Resident, TNMC, MUMBAI

ABSTRACT

Purpose: To investigate the demographic characteristics, etiological factors, clinical presentation, and visual outcomes of patients diagnosed with optic neuropathy at a tertiary health care center in western India. **Methods:** In this prospective observational study, 38 eyes of 38 patients with optic neuropathy were included. Diagnosis was based on clinical features, visual field testing, pupillary reactions, and MRI findings. Patients underwent comprehensive ocular examination, relevant investigations, and treatment according to the underlying etiology. The study evaluated demographic profile, etiology, systemic comorbidities, clinical presentation, visual acuity, color vision, fundus findings, visual field defects, and optical coherence tomography (OCT) findings. **Results:** The mean age of the patients was 38.63 ± 12.62 years, with a female preponderance (57.89%). The most common etiology was optic neuritis (71.05%), followed by ischemic optic neuropathy (13.15%). Hypertension and diabetes were significantly associated with ischemic optic neuropathy ($p<0.05$). Diminished vision was present in all patients, and 52.63% experienced pain with eye movements. Disc edema was observed in all patients with optic neuritis. The predominant visual field defect was central scotoma. Patients with optic neuritis had the highest mean RNFL thickness. The majority of patients with optic neuritis showed improvement in visual acuity at 3 months follow-up. **Conclusions:** In this study, optic neuritis was the leading cause of optic neuropathy, with hypertension and diabetes being significant risk factors for ischemic optic neuropathy. OCT and visual field testing are essential for diagnosis and monitoring. Prompt diagnosis and treatment tailored to the specific etiology are crucial for optimal visual recovery.

KEYWORDS :

INTRODUCTION

Optic neuropathy, characterized by damage to the optic nerve, can result from various etiologies, including inflammation, ischemia, infection, trauma, toxicity, and hereditary factors (Behbehani, 2007). The optic nerve, being the second cranial nerve, is responsible for transmitting visual information from the retina to the brain (Sadun, 2004). Damage to the optic nerve fibers leads to impaired signal transmission, resulting in visual dysfunction.

Optic neuritis, an inflammatory demyelinating condition, is frequently associated with multiple sclerosis (MS) (Balcer, 2006; Frohman et al., 2005). It is characterized by acute or subacute visual loss, often accompanied by pain with eye movements, and typically affects young adults (Toosy et al., 2014). The Optic Neuritis Treatment Trial (ONTT) has provided valuable insights into the clinical profile, natural history, and management of optic neuritis (Beck et al., 1992).

Ischemic optic neuropathy, further classified as arteritic (AION) and non-arteritic (NAION), is more prevalent in the elderly population and is associated with vasculopathic risk factors (Hayreh, 2004; Swartz et al., 1995). NAION is the most common form of ischemic optic neuropathy and is characterized by acute, painless, unilateral visual loss with disc edema (Atkins et al., 2010). Arteritic ischemic optic neuropathy, usually caused by giant cell arteritis, requires prompt diagnosis and treatment to prevent irreversible visual loss (Hayreh, 2004).

Other etiologies of optic neuropathy include infectious causes such as syphilis, tuberculosis, and viral infections (Kahloun et al., 2015); compressive lesions such as tumors and aneurysms (Behbehani, 2007); toxic neuropathies due to agents like ethambutol and methanol (Grzybowski et al., 2015); and hereditary optic neuropathies such as Leber's hereditary optic neuropathy (LHON) (Yu-Wai-Man et al., 2011).

The clinical presentation of optic neuropathy varies depending on the underlying etiology and may include acute or chronic vision loss, pain with eye movements, color vision disturbances, and visual field defects (Behbehani, 2007). Fundus findings can range from a normal-appearing optic disc to disc edema, pallor, or atrophy (Behbehani, 2007). Recent advancements in neuro-imaging and diagnostic techniques, such as optical coherence tomography (OCT), have significantly enhanced the understanding and management of optic neuropathies (London et al., 2013).

OCT is a non-invasive imaging modality that provides high-resolution cross-sectional images of the retina and optic nerve head (Huang et al., 1991). It allows quantitative assessment of the retinal nerve fiber layer (RNFL) thickness, which is a sensitive marker of optic nerve damage (Schuman et al., 1995). OCT has been extensively used in the evaluation and monitoring of various optic neuropathies, including optic neuritis, ischemic optic neuropathy, and compressive optic neuropathy (Rebolleda et al., 2015; Contreras et al., 2007; Narayanan et al., 2014).

Visual field testing is another crucial diagnostic tool in the assessment of optic neuropathies. It helps in detecting and characterizing visual field defects, which can provide valuable information about the location and extent of optic nerve damage (Keltner et al., 2014). The Humphrey Visual Field Analyzer, using the 24-2 or 30-2 SITA (Swedish Interactive Threshold Algorithm) protocol, is widely used in clinical practice (Khoury et al., 1999).

The management of optic neuropathy depends on the specific etiology and may include systemic corticosteroids, immunosuppressive agents, antimicrobial therapy, surgical intervention, or supportive care (Toosy et al., 2014; Atkins et al., 2010; Kahloun et al., 2015). In optic neuritis, intravenous methylprednisolone followed by oral prednisolone has been

shown to accelerate visual recovery, although the long-term visual outcome is not significantly affected (Beck et al., 1992).

The primary objectives of this study were to evaluate the demographic profile, etiological factors, clinical presentation, and visual outcomes of patients with optic neuropathy at a tertiary health care center in western India. The findings of this study will contribute to a better understanding of the disease spectrum, risk factors, and prognosis, facilitating early diagnosis and appropriate management strategies.

MATERIALS AND METHODS

Study Design And Participants

This prospective observational study was conducted at a tertiary health care center in western India over a period of one year (November 2017 to November 2018). The study protocol was approved by the institutional ethics committee, and informed consent was obtained from all participants.

The study included 38 eyes of 38 patients who were diagnosed with optic neuropathy and met the inclusion criteria. Patients aged between 18-65 years with decreased visual acuity attributed to optic neuropathy based on clinical features, visual field testing, pupillary reactions, and MRI findings were eligible for inclusion. Exclusion criteria encompassed media opacity hindering fundus examination, uncooperative patients, and those unwilling to provide informed consent.

Data Collection And Investigations

A comprehensive history was obtained, including demographic data, presenting complaints, systemic comorbidities, and treatment history. Patients underwent a thorough ocular examination, which included best-corrected visual acuity (BCVA) assessment using the Snellen chart, color vision testing with Ishihara plates, anterior segment evaluation, pupillary reactions, fundus examination using direct and indirect ophthalmoscopy, and slit-lamp biomicroscopy with a 90D lens. Intraocular pressure was measured using applanation tonometry.

Imaging and diagnostic tests performed included visual field assessment using the Humphrey Visual Field Analyzer (24-2 SITA Standard), OCT for measuring retinal nerve fiber layer (RNFL) thickness, and MRI of the brain and orbit. Blood investigations, such as complete blood count, erythrocyte sedimentation rate, renal function tests, lipid profile, and serological tests for syphilis, HIV, and hepatitis B and C, were carried out.

Treatment

Treatment was tailored according to the specific etiology of optic neuropathy. Patients with optic neuritis received intravenous methylprednisolone (1 g/day for 3 days) followed by oral prednisolone (1 mg/kg/day for 11 days) as per the Optic Neuritis Treatment Trial (ONTT) protocol (Beck et al., 1992). Those with ischemic optic neuropathy were managed with anti-platelet therapy and control of vasculopathic risk factors. In cases of toxic optic neuropathy, the offending agent (e.g., ethambutol) was discontinued. Patients with traumatic optic neuropathy were treated with intravenous methylprednisolone (1 g/day for 3 days) followed by oral prednisolone (1 mg/kg/day for 11 days).

Follow-up

Patients were followed up at 1 week, 2 weeks, 1 month, and 3 months after the initiation of treatment. At each visit, BCVA, color vision, pupillary reactions, fundus examination, and OCT were repeated. Visual field testing was performed at the 3-month follow-up visit.

Statistical Analysis

Data analysis was performed using IBM SPSS Statistics version 20.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The association between categorical variables was assessed using the Chi-square test and Fisher's exact test. A p-value < 0.05 was considered statistically significant.

RESULTS

Demographic Profile

The mean age of the patients in this study was 38.63 ± 12.62 years, with the majority (44.74%) falling into the 31-40 years age group.

Table 1: Age Distribution

Age Range	Frequency	Percentage
18-30 years	9	23.68%
31-40 years	17	44.74%
41-50 years	6	15.79%
51-65 years	6	15.79%
Total	38	100%
Mean ± SD	38.63 ± 12.62 years	

Females constituted 57.89% of the study population (Table 2).

Table 2: Gender Distribution

Gender	Frequency	Percentage
Male	16	42.11%
Female	22	57.89%
Total	38	100%

Right eye involvement (57.89%) was more common than left eye involvement (Table 3).

Table 3: Laterality Distribution

Laterality	Frequency	Percentage
Right	22	57.89%
Left	16	42.11%
Total	38	100%

Etiology And Systemic Comorbidities

The most common etiology of optic neuropathy was optic neuritis (71.05%), followed by ischemic optic neuropathy (13.15%) (Table 4).

Table 4: Etiology Distribution

Etiology	Frequency	Percentage
Optic Neuritis	27	71.05%
- Idiopathic	18	47.36%
- Demyelinating (MS)	1	2.63%
- Retrobulbar	8	21.05%
Traumatic Optic Neuropathy	3	7.89%
Toxic Optic Neuropathy	2	5.26%
Ischemic Optic Neuropathy	5	13.15%
- AION	1	2.63%
- NAION	4	10.52%
Infiltrative Optic Neuropathy	1	2.63%
Total	38	100%

Among the optic neuritis cases, 47.36% were idiopathic, while only one case (2.63%) was associated with multiple sclerosis. The most prevalent systemic comorbidities were hypertension (21.04%) and diabetes mellitus (23.68%).

Table 5: Systemic Comorbidities

Systemic Comorbidities	Frequency	Percentage
Hypertension	8	21.04%
Diabetes Mellitus	9	23.68%
Thyroid Disorder	1	2.63%
Tuberculosis	2	5.26%
Trauma	3	7.89%
Chronic Lymphoid Leukemia	1	2.63%

A significant association was found between ischemic optic

neuropathy and hypertension ($p<0.001$) and diabetes mellitus ($p<0.05$).

Clinical Presentation

All patients presented with diminished vision, and 52.63% experienced pain with eye movements (Table 6).

Table 6: Symptomatology Distribution

Symptoms	Frequency	Percentage
Pain on Eye Movements	20	52.63%
Diminution of Vision	38	100%
Headache and Scalp Tenderness	1	2.63%
Jaw Claudication	1	2.63%
Redness and Swelling of Lids	3	7.89%

The most common fundus finding in optic neuritis was disc edema (70.37%), while disc pallor was observed in toxic optic neuropathy.

Table 7: Distribution of Relative Afferent Pupillary Defect (RAPD)

RAPD	At Presentation	At 3 Months
	Frequency (%)	Frequency (%)
Present	35 (92.11%)	34 (89.47%)
Absent	0 (0%)	1 (2.63%)
Sluggish	3 (7.89%)	3 (7.89%)
Total	38 (100%)	38 (100%)

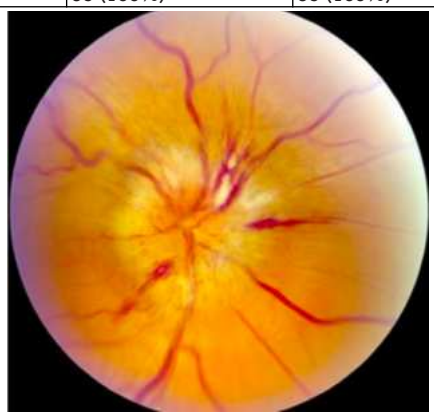

Image 1: Ophthamoscopic appearance of NAION fundus

Patients with NAION exhibited hyperemic discs, and those with retrobulbar neuritis had a normal fundus appearance. At presentation, RAPD was present in 92.11% of patients (Table 8).

Table 8: Distribution Of Color Vision Findings

Color Vision Findings	At Presentation	At 3 Months
	Frequency (%)	Frequency (%)
Normal	0 (0%)	1 (2.63%)
Abnormal	32 (84.21%)	36 (94.73%)
Cannot be Evaluated	6 (15.79%)	1 (2.63%)
Total	38 (100%)	38 (100%)

Visual Function And Imaging

At presentation, 39.47% of patients had a BCVA between 4/60 to 6/60, and 34.21% had vision ranging from hand movements to counting fingers at 3 meters.

Table 9: Distribution of Fundus Findings

Fundus Findings	At Presentation	At 3 Months
	Frequency (%)	Frequency (%)
Chalky White Disc Edema	1 (2.63%)	1 (2.63%)
Disc Edema	23 (60.52%)	18 (47.37%)
Disc Pallor	3 (7.89%)	6 (15.79%)
Hyperemic Disc	3 (7.89%)	3 (7.89%)
Normal	8 (21.05%)	10 (26.32%)
Total	38 (100%)	38 (100%)

Central scotoma was the predominant visual field defect in optic neuritis.

Table 10: Distribution of Treatment Given

Treatment	Frequency	Percentage
Parenteral Steroids	31	81.57%
Ocular Steroids	1	2.63%
Chemotherapy	1	2.63%
Withhold Ethambutol	2	5.26%
Observation, Anti-hypertensives	3	7.89%
Total	38	100%

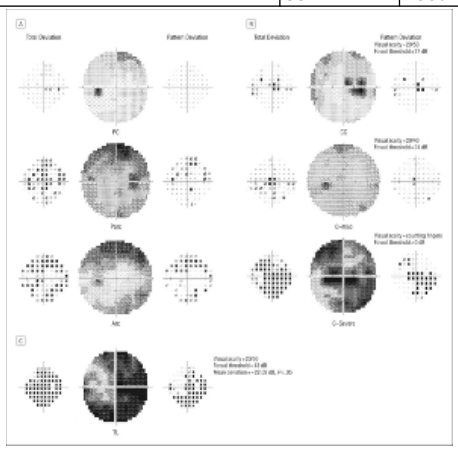

Image 2. Vision field test for optic neuritis

OCT demonstrated increased RNFL thickness in patients with optic neuritis, with six patients having a thickness of 181-190 microns.

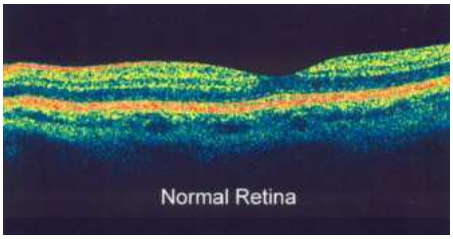

Image 3. OCT of normal retina

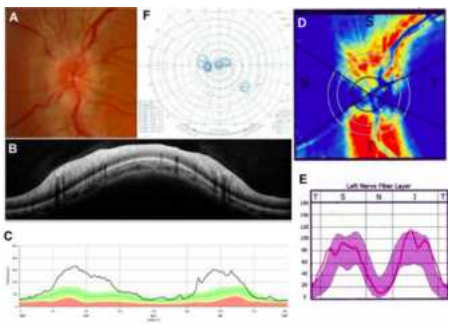

Image 4. Various Positive Findings In Optic Neuritis

Table 11: Treatment Distribution According to Etiology

Etiology	Parenteral Steroids	Ocular Steroids	Chemotherapy	Withhold Ethambutol	Observation, Anti-hypertensives
Optic Neuritis	27	0	0	0	0
Toxic Neuropathy	0	0	0	2	0
Traumatic Neuropathy	3	0	0	0	0
Ischemic Neuropathy	1	1	0	0	3

Infiltrative Neuropathy	0	0	1	0	0

MRI findings were suggestive of optic neuritis in 71.05% of patients, with features such as optic nerve enhancement and hyperintense signals.

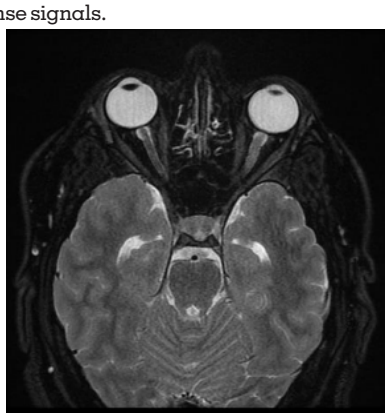

Image 5: MRI showing swollen optic nerve and optic tract in a case of optic neuritis

Table 12: Distribution of Chest X-ray Findings

Chest X-ray Findings	Frequency	Percentage
Normal	36	94.38%
Pulmonary Tuberculosis	2	5.62%
Total	38	100%

Treatment Outcomes

Parenteral corticosteroids were administered in 81.57% of patients.

Table 13: Relation of Random Blood Sugar with Etiology

Random Blood Sugar	Optic Neuritis	Toxic Neuropathy	Traumatic Neuropathy	Ischemic Neuropathy	Infiltrative Neuropathy
<126 mg/dL	24	2	2	4	1
≥126 mg/dL	3	1	1	1	0
p-value	0.267	0.369	0.385	0.781	p-not calculable

The majority of patients with optic neuritis showed improvement in visual acuity at 3 months follow-up. However, color vision abnormalities persisted in 94.73% of patients at 3 months.

Table 14: Relation of Systolic Blood Pressure with Etiology

Systolic Blood Pressure	Optic Neuritis	Toxic Neuropathy	Traumatic Neuropathy	Ischemic Neuropathy	Infiltrative Neuropathy
<130 mmHg	26	2	3	2	1
≥130 mmHg	1	0	0	3	0
p-value	0.031	p-not calculable	p-not calculable	0.000	p-not calculable

In most cases of optic neuritis, disc edema resolved, while other fundus findings remained unaltered.

DISCUSSION

The present study provides valuable insights into the clinical spectrum and visual outcomes of optic neuropathy in a tertiary health care setting in western India. The demographic profile, with a higher prevalence in females and the 31-40 years age group, is in agreement with previous reports (Shams & Plant, 2009; D Pau et al., 2011). Optic neuritis was the leading etiology, with the majority of cases being idiopathic, which is consistent with findings from other Asian studies (Ji et al., 2008; Saxena et al., 2010). The low prevalence of multiple sclerosis-associated optic neuritis in our study is in line with reports from Japan and India (Suehiro et al., 2002; Saxena et al., 2010).

The significant association of ischemic optic neuropathy with hypertension and diabetes highlights the importance of controlling these risk factors to prevent visual morbidity. This finding is consistent with the observations by Pereyra-Munoz et al. (2008) and emphasizes the need for timely management of the metabolic syndrome components. Patients with ischemic optic neuropathy often present with acute, painless visual loss and disc edema, as observed in our study (Atkins et al., 2010).

The clinical presentation of optic neuritis, characterized by diminished vision, pain with eye movements, and disc edema, is in accordance with the descriptions in the literature (Toosy et al., 2014; Saxena et al., 2010). The predominance of central scotoma in optic neuritis is consistent with reports from Nepal and Malaysia (Das et al., 2010; Shatriah et al., 2012). OCT played a crucial role in quantifying RNFL thickness and monitoring the resolution of disc edema in optic neuritis (Rebolleda et al., 2015). The increased RNFL thickness in optic neuritis patients reflects the acute inflammation and edema of the optic nerve (Costello et al., 2006).

MRI is a valuable tool in the diagnosis and evaluation of optic neuropathies, particularly in optic neuritis (Kupersmith et al., 2002). The presence of optic nerve enhancement and hyperintense signals on MRI in the majority of optic neuritis cases in our study is consistent with the literature (Rizzo et al., 2002). MRI also helps in detecting compressive lesions and demyelinating plaques in the brain, which may have prognostic implications (Kupersmith et al., 2002).

The improvement in visual acuity following corticosteroid therapy in patients with optic neuritis reaffirms the findings of the ONTT (Beck et al., 1992). Intravenous methylprednisolone accelerates visual recovery, although the long-term visual outcome is not significantly affected (Beck et al., 1992). However, the persistent color vision abnormalities and unchanged fundus findings at 3 months underscore the need for long-term follow-up and rehabilitation.

The management of other etiologies of optic neuropathy, such as ischemic optic neuropathy, toxic optic neuropathy, and traumatic optic neuropathy, depends on prompt recognition and treatment of the underlying cause (Atkins et al., 2010; Grzybowski et al., 2015; Steinsapir & Goldberg, 2011). In ischemic optic neuropathy, control of vasculopathic risk factors and anti-platelet therapy may help prevent further visual loss (Atkins et al., 2010). In toxic optic neuropathy, discontinuation of the offending agent is crucial for visual recovery (Grzybowski et al., 2015). Traumatic optic neuropathy may benefit from corticosteroid therapy, although the evidence is limited (Steinsapir & Goldberg, 2011).

The strengths of our study include the prospective design, comprehensive clinical evaluation, and the use of advanced diagnostic modalities such as OCT and MRI. However, the study has certain limitations. The sample size was relatively small, and the follow-up duration was limited to 3 months. Longer follow-up periods would provide valuable information on the long-term visual outcomes and the risk of recurrence. Additionally, the study was conducted in a tertiary care setting, which may not be representative of the general population.

In conclusion, this study provides a comprehensive analysis of the clinical profile and visual outcomes of optic neuropathy in a tertiary health care center in western India. Optic neuritis emerged as the leading etiology, with a significant proportion of cases being idiopathic. Hypertension and diabetes were identified as major risk factors for ischemic optic neuropathy,

emphasizing the importance of metabolic control. OCT and visual field testing were valuable tools in diagnosis and monitoring. Early recognition of the underlying etiology and prompt initiation of appropriate treatment are crucial for optimizing visual recovery.

Future Directions and Recommendations

The findings of this study have important implications for clinical practice and future research. The high prevalence of optic neuritis and ischemic optic neuropathy underscores the need for increased awareness among primary care physicians and prompt referral to ophthalmologists for early diagnosis and management. Patient education regarding the importance of regular eye examinations, particularly in the presence of systemic comorbidities such as hypertension and diabetes, is crucial for early detection and prevention of visual morbidity.

Future studies with larger sample sizes and longer follow-up durations are warranted to gain a better understanding of the long-term visual outcomes and the risk of recurrence in various optic neuropathies. Multicenter studies involving both urban and rural populations would provide a more comprehensive picture of the disease burden and regional variations.

The use of advanced imaging techniques, such as OCT angiography and diffusion tensor imaging, may provide additional insights into the pathophysiology and structural alterations in optic neuropathies (Higashiyama et al., 2017; Zhang et al., 2018). These modalities may aid in early diagnosis, monitoring disease progression, and assessing treatment response.

Research into the genetic and molecular mechanisms underlying optic neuropathies, particularly in hereditary and idiopathic cases, may pave the way for targeted therapies and personalized medicine (Yu-Wai-Man et al., 2011). Collaborative efforts between ophthalmologists, neurologists, and basic scientists are essential to advance our understanding of these complex disorders.

The development of standardized diagnostic criteria and treatment protocols for various optic neuropathies would help in ensuring consistent and evidence-based management across different healthcare settings. Regular training and education programs for ophthalmologists and allied healthcare professionals would help in keeping abreast with the latest advances in diagnosis and management.

In addition to medical management, low vision rehabilitation and supportive services should be an integral part of the comprehensive care of patients with optic neuropathies (Gall et al., 2010). Multidisciplinary teams involving ophthalmologists, optometrists, occupational therapists, and social workers can help in improving the quality of life and functional independence of affected individuals.

CONCLUSION

Optic neuropathies are a heterogeneous group of disorders that can lead to significant visual morbidity. This study provides valuable insights into the clinical profile and visual outcomes of optic neuropathy in a tertiary health care center in western India. Optic neuritis and ischemic optic neuropathy were the leading etiologies, with hypertension and diabetes being significant risk factors for the latter. OCT and visual field testing emerged as important diagnostic and monitoring tools.

Early diagnosis and prompt initiation of appropriate treatment, based on the underlying etiology, are crucial for optimizing visual recovery. Increased awareness among primary care physicians, patient education, and regular eye examinations are essential for early detection and prevention of visual impairment.

Future research directions include larger multicenter studies, advanced imaging techniques, genetic and molecular studies, and the development of standardized diagnostic and treatment protocols. A multidisciplinary approach, encompassing medical management, low vision rehabilitation, and supportive services, is necessary to provide comprehensive care for patients with optic neuropathies.

In conclusion, this study contributes to the growing body of knowledge on optic neuropathies in the Indian context and highlights the need for further research and collaborative efforts to improve the diagnosis, management, and outcomes of these challenging disorders. By implementing the recommendations and future directions outlined in this study, we can work towards reducing the burden of visual impairment and improving the quality of life of patients with optic neuropathies.

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