

# Clinical Profile and Etiological Patterns of Optic Neuropathy at a Tertiary Healthcare Center

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## ABSTRACT

**Background:** Optic neuropathy encompasses a spectrum of conditions affecting the optic nerve with various etiologies and clinical presentations. This study aimed to characterize the demographic profile, etiological patterns, and HBdclinical characteristics of patients with optic neuropathy presenting to a tertiary healthcare center in Western India.

**Methods:** This prospective observational study included 38 eyes of 38 patients diagnosed with optic neuropathy between November 2017 and November 2018. Comprehensive ophthalmic examination including visual acuity, color vision, pupillary reactions, fundoscopy, visual field testing, and optical coherence tomography (OCT) was performed. Demographic characteristics, etiological factors, clinical presentation, and systemic comorbidities were analyzed.

**Results:** The mean age was  $38.63 \pm 12.62$  years with a female preponderance (57.89%). Idiopathic optic neuritis (47.36%) was the most common etiology, followed by retrobulbar neuritis (21.05%), non-arteritic ischemic optic neuropathy (10.53%), traumatic optic neuropathy (7.89%), and toxic optic neuropathy (5.26%). There was a significant association between age below 40 years and optic neuritis ( $p < 0.0001$ ), while ischemic optic neuropathy predominated in patients above 50 years. Diabetes mellitus (23.68%) and hypertension (21.04%) were the most common systemic comorbidities, with a significant association between ischemic neuropathy and hypertension. All patients presented with diminution of vision, and 52.63% reported pain on eye movements. Relative afferent pupillary defect was observed in 92.11% of cases, and 84.21% demonstrated abnormal color vision at presentation.

**Conclusion:** Optic neuritis was the predominant cause of optic neuropathy in this Western Indian population, particularly in younger patients, while ischemic optic neuropathy was more common in older patients with vascular risk factors. Understanding these demographic and etiological patterns aids in early recognition and appropriate management of optic neuropathy in similar clinical settings.

**Keywords:** Optic neuropathy, optic neuritis, ischemic optic neuropathy, Western India, clinical profile

## INTRODUCTION

Optic neuropathy refers to damage to the optic nerve due to various etiologies, resulting in characteristic features of visual dysfunction (Behbehani, 2007). The optic nerve, as the second cranial nerve, is responsible for transmitting visual information from the retina to the brain. Damage to this nerve can lead to significant visual impairment and, if left untreated, permanent vision loss (Liu et al., 2001). The

pathology causing optic neuropathy can arise from a diverse range of conditions including inflammatory, infectious, ischemic, traumatic, toxic, and genetic factors (Vimla et al., 2011).

The clinical presentation of optic neuropathy typically includes decreased visual acuity, visual field defects, color vision abnormalities, and the presence of a relative afferent pupillary defect (RAPD) (Behbehani, 2007). The severity and speed of onset often provide diagnostic clues, while ophthalmoscopic findings vary depending on the cause and stage of the disease. Acute inflammatory optic neuropathy, known as optic neuritis, typically affects younger populations and has a better prognosis compared to ischemic optic neuropathies, which are more common in elderly individuals (Balcer, 2006; Arnold, 2001). Global epidemiological studies suggest significant geographical and ethnic variations in the prevalence and etiology of optic neuropathy (Shams & Plant, 2009; Kurtzke, 1985). While studies from Western countries have extensively documented the clinical profile and natural history of conditions like optic neuritis and ischemic optic neuropathy, there is limited data from the Indian subcontinent (Phillips et al., 1998; Bhigjee et al., 2007). The demographic characteristics, etiological factors, and clinical presentation patterns may differ in the Indian population compared to Western populations due to variations in genetic factors, environmental exposures, and prevalence of systemic comorbidities.

Understanding the clinical profile and etiological patterns of optic neuropathy in specific populations is crucial for early recognition, appropriate management, and prognostication. This becomes particularly important in developing countries where limited resources necessitate targeted diagnostic approaches and treatment strategies. Previous studies from India have reported varying patterns of optic neuropathy, with both similarities and differences compared to Western data (Saxena et al., 2010; Jain et al., 1980).

The present study was undertaken to characterize the demographic profile, etiological patterns, clinical presentation, and associated systemic conditions of patients with optic neuropathy presenting to a tertiary healthcare center in Western India. By documenting these characteristics, we aim to contribute to the existing knowledge about optic neuropathy in the Indian context and provide valuable information for clinicians managing this condition in similar settings.

## **MATERIALS AND METHODS**

### **Study Design and Population**

This prospective observational study was conducted at the Department of Ophthalmology of a tertiary healthcare center in Western India over a period of one year (November 2017 to November 2018). The study included 38 eyes of 38 patients diagnosed with optic neuropathy based on clinical features, visual field testing, pupillary reactions, and neuroimaging. Patients between 18-65 years of age who could cooperate for visual field and OCT testing were included. Exclusion criteria encompassed patients unwilling to provide written informed consent, those unable to follow up for the study duration, patients with media opacity precluding clinical examination or investigations, and those with debilitating illness precluding detailed ophthalmic examination.

### **Clinical Evaluation**

All patients underwent a comprehensive evaluation including detailed history-taking focusing on ocular symptoms, systemic illnesses, previous ocular conditions, family history, and medication use. Best-corrected visual acuity was assessed using Snellen's chart. For visual acuity less than 6/60, assessment was performed using counting fingers, hand movements, or perception of light. Color vision was tested using Ishihara plates.

Anterior segment examination was performed using slit-lamp biomicroscopy, and pupillary reactions were evaluated in dim illumination to detect RAPD. Intraocular pressure was measured using Schiottz or applanation tonometry. Fundus examination was conducted with direct and indirect ophthalmoscopy, and slit-lamp biomicroscopy using 90D or 78D lens to evaluate the optic disc and other posterior segment structures.

### **Investigations**

Visual field testing was performed using Humphrey Field Analyzer with the Swedish interactive threshold algorithm (SITA) standard 24-2 program. Reliable visual field tests (with <33% false positives and <20% fixation losses) were included in the analysis.

Optical coherence tomography (OCT) was performed through dilated pupils using Zeiss Cirrus HD (spectral domain) machine. Optic disc cube of 200×200 was obtained to assess retinal nerve fiber layer (RNFL) thickness and cup-to-disc ratio.

Laboratory investigations included complete blood count, erythrocyte sedimentation rate, renal function tests, lipid profile, blood glucose, VDRL, HIV, and hepatitis B and C serology. Chest X-ray was obtained for all patients. Neuroimaging with magnetic resonance imaging (MRI) of the brain and orbits was performed to differentiate between multiple sclerosis, tuberculosis, or other etiologies before initiating treatment.

### **Diagnostic Criteria and Classification**

Diagnosis of optic neuropathy was established based on decreased visual acuity, color vision abnormalities, visual field defects, RAPD, and optic disc changes (normal, swollen, pale, or anomalous). Specific etiological diagnoses were made based on clinical features, laboratory findings, and neuroimaging results.

Patients were classified into different etiological categories:

1. Optic neuritis (idiopathic, demyelinating, or retrobulbar)
2. Ischemic optic neuropathy (arteritic or non-arteritic)
3. Traumatic optic neuropathy
4. Toxic optic neuropathy
5. Infiltrative optic neuropathy

### **Statistical Analysis**

Data were analyzed using descriptive statistics. Frequencies and percentages were calculated for categorical variables, while means and standard deviations were computed for continuous variables. Associations between variables were assessed using Chi-square test, with  $p < 0.05$  considered statistically significant.

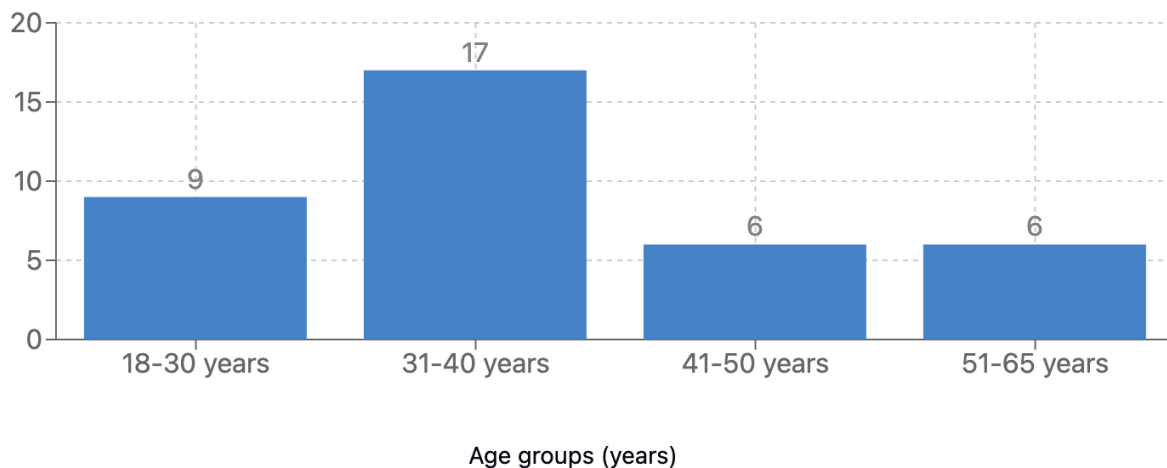
## **RESULTS**

### **Demographic Characteristics**

The study included 38 patients diagnosed with optic neuropathy. The mean age of patients was  $38.63 \pm 12.62$  years (range: 18-65 years). The majority of patients (44.74%) were in the age group of 31-40 years, followed by 18-30 years (23.68%), with equal distribution (15.79% each) in the 41-50 years and 51-65 years age groups (Table 1).

**Table 1: Demographic Characteristics of Patients with Optic Neuropathy**

Characteristic	Number (n=38)	Percentage (%)
<b>Age (years)</b>		
18-30	9	23.68
31-40	17	44.74
41-50	6	15.79
51-65	6	15.79
Mean ± SD	38.63 ± 12.62	-
<b>Gender</b>		
Male	16	42.11
Female	22	57.89
<b>Laterality</b>		
Right eye	22	57.89
Left eye	16	42.11
Bilateral	0	0



**Figure 1: Age Distribution of Patients with Optic Neuropathy (n=38)**

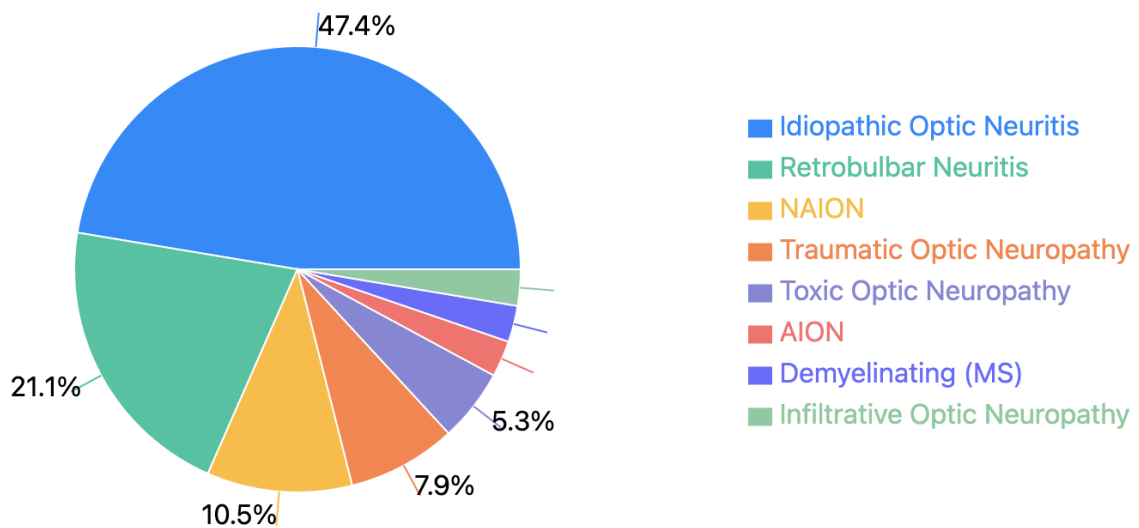
Female patients constituted 57.89% (n=22) of the study population, while 42.11% (n=16) were male. Right eye involvement (57.89%) was slightly more common than left eye involvement (42.11%), and no patient presented with bilateral disease during the study period.

**Etiological Profile**

Optic neuritis was the most common etiology, diagnosed in 27 patients (71.05%), of which 18 cases (47.36%) were idiopathic, 8 cases (21.05%) were retrobulbar neuritis, and 1 case (2.63%) was associated with multiple sclerosis. Ischemic optic neuropathy was diagnosed in 5 patients (13.15%), with non-arteritic anterior ischemic optic neuropathy (NAION) accounting for 4 cases (10.53%) and arteritic anterior ischemic optic neuropathy (AION) for 1 case (2.63%). Other etiologies included traumatic optic neuropathy in 3 patients (7.89%), toxic optic neuropathy in 2 patients (5.26%), and infiltrative optic neuropathy in 1 patient (2.63%) (Table 2).

**Table 2: Etiological Distribution of Optic Neuropathy**

Etiology	Number	Percentage (%)
Optic neuritis	27	71.05
Idiopathic	18	47.36
Retrobulbar	8	21.05
Demyelinating (Multiple Sclerosis)	1	2.63
Ischemic optic neuropathy	5	13.15
Non-arteritic (NAION)	4	10.53
Arteritic (AION)	1	2.63
Traumatic optic neuropathy	3	7.89
Toxic optic neuropathy	2	5.26
Infiltrative optic neuropathy	1	2.63
<b>Total</b>	<b>38</b>	<b>100</b>



**Figure 2: Etiological Distribution of Optic Neuropathy (n=38)**

### Association Between Age and Etiology

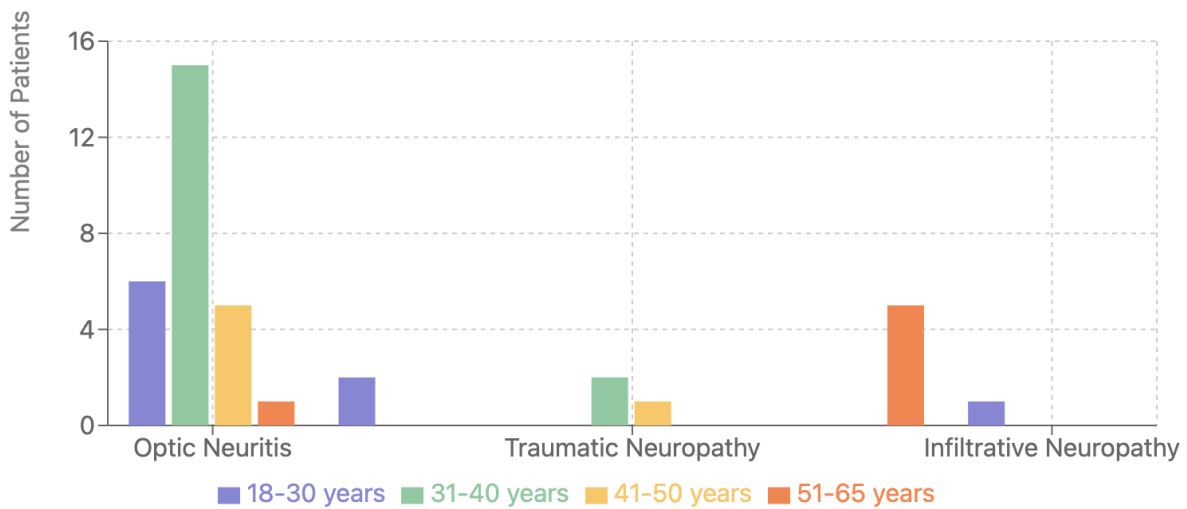
A significant association was observed between age and etiology of optic neuropathy ( $p < 0.0001$ ). Optic neuritis predominated in younger age groups, with 15 patients (39.45%) in the 31-40 years category and 6 patients (15.78%) in the 18-30 years category. In contrast, all 5 patients (13.15%) with ischemic optic neuropathy were in the 51-65 years age group. Both patients with toxic optic neuropathy were in the youngest age group (18-30 years), while traumatic optic neuropathy showed no clear age predilection (Table 3).

**Table 3: Association Between Age and Etiology of Optic Neuropathy**

Etiology	Age Group (years)				Total (n=38)
	18-30	31-40	41-50	51-65	
	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)

<b>Optic neuritis</b>	6 (15.78)	15 (39.45)	5 (13.15)	1 (2.63)	27 (71.01)
<b>Toxic neuropathy</b>	2 (5.26)	0 (0)	0 (0)	0 (0)	2 (5.26)
<b>Traumatic neuropathy</b>	0 (0)	2 (5.26)	1 (2.63)	0 (0)	3 (7.89)
<b>Ischemic neuropathy</b>	0 (0)	0 (0)	0 (0)	5 (13.15)	5 (13.15)
<b>Infiltrative neuropathy</b>	1 (2.63)	0 (0)	0 (0)	0 (0)	1 (2.63)
<b>Total</b>	<b>9 (23.67)</b>	<b>17 (44.71)</b>	<b>6 (15.78)</b>	<b>6 (15.78)</b>	<b>38 (100)</b>

Chi-square p-value <0.0001



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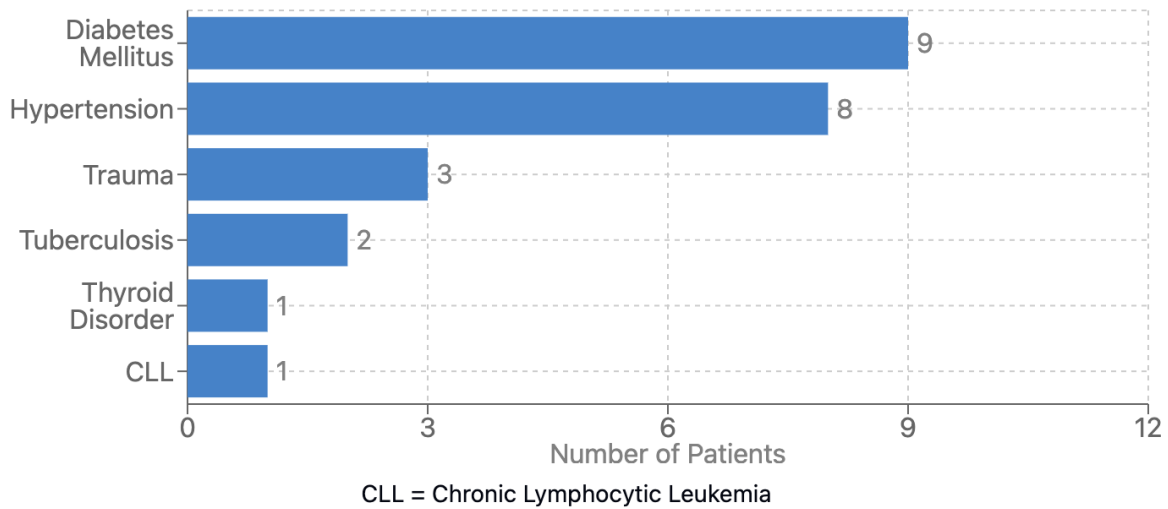
**Figure 3: Association Between Age and Etiology of Optic Neuropathy (n=38)**

**Systemic Comorbidities**

Among the study population, diabetes mellitus was the most common systemic comorbidity, present in 9 patients (23.68%), followed by hypertension in 8 patients (21.04%). Other conditions included tuberculosis in 2 patients (5.26%), thyroid disorders in 1 patient (2.63%), and chronic lymphocytic leukemia in 1 patient (2.63%).

**Table 4: Systemic Comorbidities in Patients with Optic Neuropathy**

<b>Comorbidity</b>	<b>Number</b>	<b>Percentage (%)</b>
<b>Diabetes mellitus</b>	9	23.68
<b>Hypertension</b>	8	21.04
<b>Tuberculosis</b>	2	5.26
<b>Trauma</b>	3	7.89
<b>Thyroid disorder</b>	1	2.63
<b>Chronic lymphocytic leukemia</b>	1	2.63



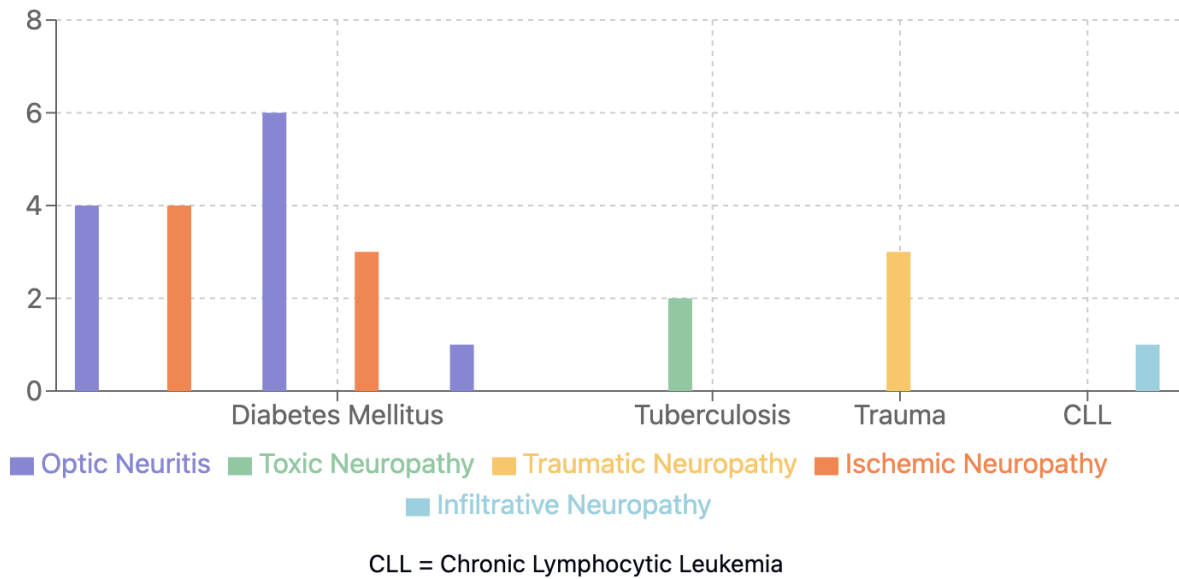
**Figure 4: Systemic Comorbidities in Patients with Optic Neuropathy (n=38)**

Analysis of comorbidities in relation to etiology revealed a significant association between ischemic optic neuropathy and hypertension, with 4 out of 5 patients with ischemic neuropathy having hypertension. Similarly, 3 out of 5 patients with ischemic neuropathy had diabetes mellitus. Both patients with toxic optic neuropathy had pulmonary tuberculosis and were on anti-tubercular therapy including ethambutol at the time of presentation.

**Table 5: Association Between Etiology and Systemic Comorbidities**

Etiology	Hypertension	Diabetes Mellitus	Thyroid Disorder	Tuberculosis	Trauma	CLL
<b>Optic neuritis (n=27)</b>	4	6	1	0	0	0
<b>Toxic neuropathy (n=2)</b>	0	0	0	2	0	0
<b>Traumatic neuropathy (n=3)</b>	0	0	0	0	3	0
<b>Ischemic neuropathy (n=5)</b>	4	3	0	0	0	0
<b>Infiltrative neuropathy (n=1)</b>	0	0	0	0	0	1

CLL = Chronic Lymphocytic Leukemia



**Figure 5: Association Between Etiology and Comorbidities (n=38)**

### Clinical Presentation

All patients (100%) presented with diminution of vision. Pain on eye movements was reported by 20 patients (52.63%), primarily those with optic neuritis (17 patients) and traumatic optic neuropathy (3 patients). One patient with arteritic ischemic optic neuropathy reported headache, scalp tenderness, and jaw claudication. The three patients with traumatic optic neuropathy presented with additional symptoms of redness and swelling of eyelids following trauma.

**Table 6: Clinical Presentation of Patients with Optic Neuropathy**

Symptoms	Number	Percentage (%)
Diminution of vision	38	100.00
Pain on eye movements	20	52.63
Headache and scalp tenderness	1	2.63
Jaw claudication	1	2.63
Redness and swelling of eyelids	3	7.89

Evaluation of visual acuity at presentation revealed that 15 patients (39.47%) had vision in the range of finger counting at 4 meters to 6/60, while 13 patients (34.21%) had vision between hand movements and finger counting at 3 meters. Seven patients (18.42%) had better visual acuity between 6/36 and 6/18, and 3 patients (7.89%) could only perceive light.

**Table 7: Visual Acuity at Presentation**

Best Corrected Visual Acuity	Number	Percentage (%)
6/36 to 6/18	7	18.42
FC 4 meters to 6/60	15	39.47
HM+ to FC 3 meters	13	34.21
PL+	3	7.89
<b>Total</b>	<b>38</b>	<b>100</b>



FC = Finger Counting; HM = Hand Movements; PL = Perception of Light

RAPD was present in 35 patients (92.11%) and sluggish in the remaining 3 patients (7.89%). Color vision assessment showed abnormalities in 32 patients (84.21%), while in 6 patients (15.79%), color vision could not be evaluated due to poor visual acuity.

**Fundoscopy Findings**

Fundoscopy revealed disc edema in 23 patients (60.52%), normal fundus in 8 patients (21.05%), disc pallor in 3 patients (7.89%), and hyperemic disc in 3 patients (7.89%). One patient (2.63%) presented with chalky white disc edema.

**Table 8: Fundoscopic Findings in Patients with Optic Neuropathy**

Fundus Finding	Number	Percentage (%)
Disc edema	23	60.52
Normal fundus	8	21.05
Disc pallor	3	7.89
Hyperemic disc	3	7.89
Chalky white disc edema	1	2.63
<b>Total</b>	<b>38</b>	<b>100</b>

Analysis of fundoscopic findings in relation to etiology showed that among patients with optic neuritis, 19 had disc edema while 8 (with retrobulbar neuritis) had a normal fundus appearance. Both patients with toxic optic neuropathy demonstrated disc pallor. Among patients with ischemic optic neuropathy, 3 had hyperemic discs, 1 had chalky white disc edema, and 1 had disc edema. All 3 patients with traumatic optic neuropathy showed fundoscopic abnormalities, with 2 having disc edema and 1 showing disc pallor.

**Visual Field Defects**

Visual field testing could not be performed in 13 patients due to poor visual acuity. Among the remaining 25 patients, central scotoma was the most common visual field defect, observed in 11 patients (including 8 with optic neuritis, 1 with toxic neuropathy, 1 with ischemic neuropathy, and 1 with infiltrative neuropathy). Six patients with optic neuritis demonstrated centrocecal scotoma, while 3 had enlarged blind spots. Inferior altitudinal field defects were observed in 2 patients with ischemic optic neuropathy. Three patients had unreliable visual field test results.

**OCT Findings**

OCT examination was performed to evaluate RNFL thickness and cup-to-disc ratio. At presentation, 11 patients could not undergo OCT examination due to poor fixation associated with visual acuity less than finger counting at 3 meters. Among patients who underwent OCT, those with disc edema due to optic neuritis demonstrated the highest RNFL thickness, with 6 patients showing values between 181-190 microns. The cup-to-disc ratio was small (0.00-0.10) in 23 patients at presentation.

**Neuroimaging Findings**

MRI of the brain and orbits revealed features consistent with optic neuritis in 27 patients, including one with multiple sclerosis. Ischemic changes of the optic nerve were observed in 3 patients with ischemic optic neuropathy. Among patients with traumatic optic neuropathy, 2 showed fracture of the lateral wall of orbit, and 1 demonstrated swelling of the optic nerve in the optic canal. Enhancement of the optic nerve was noted in 4 patients, while 2 patients had normal neuroimaging findings.

**Laboratory Investigations**

Patients with ischemic optic neuropathy had a mean serum cholesterol level of 194 mg/dL, which was

higher compared to other etiological groups, though the difference was not statistically significant. Chest X-ray revealed evidence of pulmonary tuberculosis in 2 patients (5.26%), corresponding to those diagnosed with toxic optic neuropathy secondary to ethambutol therapy.

Analysis of random blood sugar (RBS) levels in relation to etiology showed that although not statistically significant, patients with optic neuritis had lower RBS levels compared to other etiologies. Systolic blood pressure was significantly higher in patients with ischemic neuropathy compared to other groups ( $p < 0.05$ ).

## DISCUSSION

This prospective observational study provides insights into the demographic profile, etiological patterns, and clinical characteristics of patients with optic neuropathy in Western India. Our findings demonstrate that optic neuritis was the predominant cause of optic neuropathy in this population, particularly affecting younger individuals, while ischemic optic neuropathy was more common in older patients with vascular risk factors.

### Demographic Characteristics

The mean age of patients in our study was  $38.63 \pm 12.62$  years, with the majority in the 31-40 years age group. This finding aligns with previous reports suggesting that optic neuropathy, particularly optic neuritis, predominantly affects young to middle-aged adults (Shams & Plant, 2009; Kurtzke, 1985; Pau et al., 2011). The female preponderance (57.89%) observed in our study is consistent with findings from Japan (Suehiro et al., 2002) and China (Zhang et al., 2008), but differs from studies in Nepal that reported male predominance (Das et al., 2010). This gender distribution likely reflects the higher incidence of inflammatory optic neuropathies in females, as documented in various epidemiological studies (Shams & Plant, 2009).

Unilateral presentation was universal in our cohort, with no cases of bilateral simultaneous involvement. This pattern is similar to observations from other Indian studies (Saxena et al., 2010) and Asian reports (Zhang et al., 2008; Suehiro et al., 2002). The right eye was affected slightly more frequently than the left, though this difference lacks clear pathophysiological significance.

### Etiological Profile

Optic neuritis emerged as the most common etiology in our study, accounting for 71.05% of cases, with idiopathic optic neuritis predominating (47.36%). This finding corresponds with other Asian studies that have reported optic neuritis as the leading cause of optic neuropathy (Suehiro et al., 2002; Zhang et al., 2008). However, the proportion of demyelinating optic neuritis associated with multiple sclerosis was notably lower in our study (2.63%) compared to reports from Japan, where 22.8% of optic neuritis cases were associated with multiple sclerosis (Suehiro et al., 2002). This discrepancy reflects the known geographical variation in the prevalence of multiple sclerosis, which is less common in tropical and subtropical regions including India (Bhigjee et al., 2007; Mbonda et al., 1990).

Ischemic optic neuropathy constituted the second most common etiology (13.15%), predominantly affecting older individuals. This aligns with established understanding that ischemic optic neuropathy, particularly the non-arteritic type, typically affects patients over 50 years of age (Arnold, 2001). The proportion of ischemic optic neuropathy in our study is comparable to reports from other centers in Asia and India (Saxena et al., 2010; Zhang et al., 2008).

Toxic optic neuropathy represented 5.26% of cases in our study, with both cases associated with ethambutol therapy for tuberculosis. This reflects the continuing challenge of tuberculosis and its treatment complications in India. Traumatic and infiltrative optic neuropathies accounted for smaller

proportions, consistent with their generally lower prevalence in most epidemiological studies (Saxena et al., 2010; Das et al., 2010).

### **Age-Etiology Relationship**

Our study demonstrated a significant association between age and etiology of optic neuropathy ( $p < 0.0001$ ). Optic neuritis predominantly affected younger patients, with 84.6% of cases occurring in individuals below 40 years. This pattern is consistent with the established epidemiology of optic neuritis, which typically affects young adults (Balcer, 2006). Conversely, all cases of ischemic optic neuropathy occurred in patients above 50 years, corresponding with the understanding that vascular risk factors accumulate with age, predisposing older individuals to ischemic events (Pereyra-Munoz et al., 2008; Arnold, 2001).

Both cases of toxic optic neuropathy occurred in younger patients (below 30 years), which may reflect the demographic profile of tuberculosis patients in India rather than any specific age predilection for toxic neuropathy itself. The relationship between age and traumatic or infiltrative neuropathies was less distinct due to the small number of cases in these categories.

### **Systemic Comorbidities and Risk Factors**

Diabetes mellitus (23.68%) and hypertension (21.04%) were the most prevalent systemic comorbidities in our cohort. Notably, there was a significant association between ischemic optic neuropathy and these vascular risk factors, with 80% of ischemic optic neuropathy patients having hypertension and 60% having diabetes. This association reinforces the vascular etiopathogenesis of ischemic optic neuropathy and aligns with findings from previous studies (Jacobson et al., 1997; Salomon et al., 1999).

The mean cholesterol level in patients with ischemic optic neuropathy (194 mg/dL) was higher than in other groups, though below the threshold of 200 mg/dL recommended by the American Heart Association (Stone et al., 2014). This observation adds to the evidence supporting the role of the metabolic syndrome triad (hypertension, diabetes, and dyslipidemia) in the pathogenesis of ischemic optic neuropathy, as previously reported by Pereyra-Munoz et al. (2008).

The association between tuberculosis and toxic optic neuropathy in our study highlights the importance of vigilant monitoring for ocular complications in patients receiving ethambutol therapy. This is particularly relevant in India, where tuberculosis remains endemic and ethambutol continues to be a cornerstone of anti-tubercular regimens (Sharma & Sharma, 2011).

### **Clinical Presentation and Ophthalmological Findings**

Universal presentation with diminution of vision in our cohort underscores the central role of the optic nerve in visual function. The high prevalence of pain on eye movements (52.63%), particularly in patients with optic neuritis, is consistent with the inflammatory nature of this condition. Our finding exceeds the rates reported in studies from China (42.9%, Zhang et al., 2008), Nepal (33.33%, Das et al., 2010), and Japan (17.6%, Suehiro et al., 2002), but aligns with another Indian study (66%, Saxena et al., 2010), suggesting possible regional variations in presentation.

The high prevalence of RAPD (92.11%) and color vision abnormalities (84.21%) in our study confirms the sensitivity of these parameters in detecting optic nerve dysfunction, as emphasized by Behbehani (2007). The pattern of visual field defects, with central scotoma predominating, corresponds to typical findings in optic neuropathy, particularly of inflammatory etiology (Keltner et al., 2010).

Fundoscopy findings varied with etiology, with disc edema predominating in inflammatory optic neuropathy, disc pallor in toxic neuropathy, and hyperemic discs in ischemic neuropathy. The presence of normal fundus in 29.6% of optic neuritis cases represents the retrobulbar variant, which is typically

characterized by normal ophthalmoscopic appearance despite significant visual dysfunction. This proportion is lower than the approximately 65% reported in the Optic Neuritis Treatment Trial (Beck et al., 1992), suggesting potential geographical or ethnic variations in presentation.

OCT findings correlated with clinical observations, demonstrating increased RNFL thickness in patients with disc edema due to optic neuritis. This technology provides objective quantification of structural changes in optic neuropathy and aids in monitoring disease progression and response to treatment (Yuksel et al., 2018).

### **Strengths and Limitations**

The strength of this study lies in its prospective design and comprehensive evaluation of patients using a standardized protocol including clinical examination, visual field testing, OCT, and neuroimaging. The detailed characterization of demographic profiles, clinical presentations, and etiological patterns provides valuable information about optic neuropathy in the Western Indian context.

However, several limitations should be acknowledged. The relatively small sample size limits the statistical power for subgroup analyses and may not fully represent the entire spectrum of optic neuropathy in the region. The single-center design potentially introduces selection bias, as patients presenting to a tertiary care center may represent more severe or complicated cases. The follow-up period of three months may not capture the long-term course and outcomes of chronic optic neuropathies. Additionally, the lack of a control group limits comparative analyses of risk factors.

### **CONCLUSION**

This study provides insights into the demographic profile, etiological patterns, and clinical characteristics of optic neuropathy in a tertiary healthcare center in Western India. Optic neuritis emerged as the predominant cause, particularly affecting younger patients, while ischemic optic neuropathy was more common in older individuals with vascular risk factors. The significant association between age and specific etiologies, as well as between systemic comorbidities and ischemic optic neuropathy, underscores the importance of considering these factors in the diagnostic approach.

The findings highlight the need for comprehensive evaluation of patients presenting with visual symptoms suggestive of optic nerve dysfunction, including detailed history-taking, clinical examination, and appropriate investigations tailored to the suspected etiology. Early recognition and appropriate management based on the underlying cause are crucial for optimizing visual outcomes.

Future research with larger cohorts, longer follow-up, and multicenter designs would further enhance our understanding of optic neuropathy in the Indian population. Additionally, studies investigating genetic, environmental, and socioeconomic factors that may influence the presentation and outcomes of optic neuropathy would provide valuable insights for developing targeted prevention and management strategies.

### **REFERENCES**

1. Arnold, A. C. (2001). Ischemic optic neuropathies. *Ophthalmology Clinics of North America*, 14(1), 83-98.
2. Balcer, L. J. (2006). Clinical practice. Optic neuritis. *New England Journal of Medicine*, 354(12), 1273-1280.

3. Beck, R. W., Cleary, P. A., Anderson, M. M., Keltner, J. L., Shults, W. T., Kaufman, D. I., ... & Optic Neuritis Study Group. (1992). A randomized, controlled trial of corticosteroids in the treatment of acute optic neuritis. *New England Journal of Medicine*, 326(9), 581-588.
4. Behbehani, R. (2007). Clinical approach to optic neuropathies. *Clinical Ophthalmology*, 1(3), 233-246.
5. Bhigjee, A. I., Moodley, K., & Ramkisson, K. (2007). Multiple sclerosis in KwaZulu Natal, South Africa: an epidemiological and clinical study. *Multiple Sclerosis*, 13(9), 1095-1099.
6. Das, H., Gautam, M., & Lavaju, P. (2010). An overview of idiopathic optic neuritis in eastern Nepal. *Nepalese Journal of Ophthalmology*, 2(1), 10-15.
7. Jacobson, D. M., Vierkant, R. A., & Belongia, E. A. (1997). Nonarteritic anterior ischemic optic neuropathy: a case-control study of potential risk factors. *Archives of Ophthalmology*, 115(11), 1403-1407.
8. Jain, I. S., Munjal, V. P., Dhir, S. P., & Gangwar, D. N. (1980). Profile of optic neuritis in Chandigarh and surrounding areas. *Indian Journal of Ophthalmology*, 28(4), 195-200.
9. Keltner, J. L., Johnson, C. A., Cello, K. E., Dontchev, M., Gal, R. L., & Beck, R. W. (2010). Visual field profile of optic neuritis: a final follow-up report from the optic neuritis treatment trial from baseline through 15 years. *Archives of Ophthalmology*, 128(3), 330-337.
10. Kurtzke, J. F. (1985). Optic neuritis or multiple sclerosis. *Archives of Neurology*, 42(7), 704-710.
11. Liu, G. T., Volpe, N. J., & Galetta, S. L. (2001). Visual loss: optic neuropathies. In *Neuro-Ophthalmology: Diagnosis and Management* (pp. 103-187). Philadelphia: WB Saunders.
12. Mbonda, E., Larnaout, A., Maertens, A., Appel, B., Lowenthal, A., Mbede, J., & Gabre-Selassie, D. (1990). Multiple sclerosis in a black Cameroonian woman. *Acta Neurologica Belgica*, 90(4), 218-222.
13. Pau, D., Al Zubidi, N., Yalamanchili, S., Plant, G. T., & Lee, A. G. (2011). Optic neuritis. *Eye*, 25(7), 833-842.
14. Pereyra-Munoz, N., Camargo-Suárez, M., & Nuñez-Gómez, R. (2008). Epidemiology of Non-Arteritic Anterior Ischemic Optic Neuropathy in a Referral Center in Mexico-City. *Investigative Ophthalmology & Visual Science*, 49(13), 6007.
15. Phillips, P. H., Newman, N. J., & Lynn, M. J. (1998). Optic neuritis in African Americans. *Archives of Neurology*, 55(2), 186-192.
16. Salomon, O., Huna-Baron, R., Kurtz, S., Steinberg, D. M., Moisseiev, J., Rosenberg, N., ... & Seligsohn, U. (1999). Analysis of prothrombotic and vascular risk factors in patients with nonarteritic anterior ischemic optic neuropathy. *Ophthalmology*, 106(4), 739-742.
17. Saxena, R., Phuljhele, S., Menon, V., & Shailesh, G. M. (2010). Profile of optic neuritis patients in India. *Indian Journal of Ophthalmology*, 57(6), 448-450.
18. Shams, P. N., & Plant, G. T. (2009). Optic neuritis: a review. *International MS Journal*, 16(3), 82-89.
19. Sharma, P., & Sharma, R. (2011). Toxic optic neuropathy. *Indian Journal of Ophthalmology*, 59(2), 137-141.
20. Stone, N. J., Robinson, J. G., Lichtenstein, A. H., Merz, C. N. B., Blum, C. B., Eckel, R. H., ... & Smith, S. C. (2014). 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*, 63(25 Part B), 2889-2934.

21. Suehiro, S., Adachi-Usami, E., Miyauchi, O., Mizota, A., Tsuyama, Y., Fujimoto, N., ... & Igarashi, Y. (2002). Clinical profiles of patients with optic neuritis at the ophthalmological department of Chiba University. *Neuro-ophthalmology*, 27(1-3), 153-162.
22. Vimla, M., Rohit, S., Ruby, M., & Swati, P. (2011). Management of optic neuritis. *Indian Journal of Ophthalmology*, 59(2), 117-122.
23. Yuksel, B., Dogan, B., Koctekin, B., Atis, N., Erdal, A., Kurtulus, F., ... & Gomceli, Y. B. (2018). Color vision testing versus pattern visual evoked potentials and optical coherence tomography parameters in subclinical optic nerve involvement in multiple sclerosis. *Journal of Clinical Neuroscience*, 57, 173-178.
24. Zhang, X., Wang, W., Wei, W., Wang, Q., Wei, Y., Kermode, A. G., ... & Qiu, W. (2008). Etiological profile of presumptive optic neuritis in China. *Journal of Clinical Neuroscience*, 15(12), 1346-1349.