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Etiological analysis of pale optic disc and its association with six-month visual outcome in patients attending a tertiary care hospital: a prospective observational cohort study.

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Abstract

Background

Pale optic disc indicates established optic nerve damage caused by inflammatory, ischemic, traumatic, compressive, infectious, toxic, or metabolic disorders. Etiological identification is important because visual recovery varies substantially across causes.

Objectives

To determine the etiological spectrum of pale optic disc and assess its association with six-month best-corrected visual acuity outcome.

Methods

This prospective, hospital-based observational cohort study included 50 adults with optic disc pallor attending a tertiary-care ophthalmology department. Participants underwent standardized ocular examination, colour vision and visual field assessment, selected electrophysiology, laboratory investigations, and neuroimaging when indicated. Visual improvement was defined as a gain of at least one Snellen line at six months. Etiology and visual outcome were compared using Pearson's chi-square test and the Fisher-Freeman-Halton exact test.

Results

The mean age was 38.68 ± 12.09 years, and 56.0% were male. Traumatic optic neuropathy was the leading etiology (28.0%), followed by optic neuritis and tumor-related optic neuropathy (22.0% each) and primary optic atrophy (16.0%). Overall, 17 of 50 patients improved (34.0%; 95% CI, 22.4%-47.8%). Improvement occurred in 72.7% of optic neuritis cases (95% CI, 43.4%-90.3%), 54.5% of tumor-related cases (95% CI, 28.0%-78.7%), and 21.4% of traumatic optic neuropathy cases (95% CI, 7.6%-47.6%). Visual improvement differed significantly by etiology (Pearson $\chi^2(9)=17.62$, $p=0.040$; Fisher-Freeman-Halton exact $p=0.011$; Cramér's $V=0.59$).

Conclusion

Pale optic disc had heterogeneous causes and generally guarded visual outcomes. Optic neuritis showed the most favourable recovery, whereas established primary optic atrophy showed no improvement.

Recommendations

Early neuro-ophthalmic assessment, directed imaging, cause-specific treatment, and structured follow-up should be prioritized.

Keywords: *best-corrected visual acuity; optic atrophy; optic neuritis; pale optic disc; prospective cohort; traumatic optic neuropathy; visual outcome.*

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Introduction

Optic disc pallor is a clinically important fundus sign that reflects loss or dysfunction of retinal ganglion cell axons within the anterior visual pathway. It is not a single disease entity; rather, it represents the end stage of diverse optic nerve insults involving the retina-to-lateral geniculate pathway. Degeneration of optic nerve fibres reduces the normal vascular and neural appearance of the disc and produces the pale optic nerve head observed during ophthalmoscopy. In clinical practice, optic atrophy is usually accompanied by variable reduction in visual acuity, colour desaturation, relative afferent pupillary defect, contrast sensitivity loss, and characteristic visual field abnormalities [1,2].

The differential diagnosis of pale optic disc is broad. Common etiological groups include demyelinating optic neuritis, non-arteritic and arteritic ischemic optic neuropathy, compressive lesions, traumatic optic neuropathy, toxic and nutritional optic neuropathies, hereditary optic neuropathies, infectious or inflammatory disorders, post-papilloedema atrophy, and optic nerve involvement secondary to intracranial disease [2,3]. The clinical tempo is often helpful: acute painful visual loss suggests optic neuritis, sudden painless loss in an older patient suggests ischemic optic neuropathy, progressive painless loss suggests compressive or toxic causes, and a history of cranio-orbital trauma supports traumatic optic neuropathy. However, overlapping presentations are frequent, and a pale disc alone rarely defines the diagnosis.

A methodical neuro-ophthalmic evaluation is therefore essential. Best-corrected visual acuity, pupil examination, colour vision, visual fields, slit-lamp biomicroscopy, fundus photography, and retinal nerve fibre layer assessment help to characterize the functional and structural burden of optic nerve damage. Systemic evaluation and neuroimaging are particularly important when compressive, demyelinating, infectious, inflammatory, or post-traumatic causes are suspected [2,4]. This is clinically relevant because the underlying cause has direct implications for both treatment and prognosis. Optic neuritis often shows favourable recovery, while traumatic and established atrophic optic neuropathies frequently have limited improvement. Ethambutol-related or other toxic neuropathies require early withdrawal of the offending agent, whereas compressive lesions need timely medical, surgical, or radiotherapeutic intervention [4-8].

Regional hospital-based evidence on the etiological distribution of pale optic disc remains limited, particularly in tertiary care settings where trauma, tuberculosis, demyelinating disease, tumors, and systemic vascular risk factors coexist. Generating local data assists clinicians in prioritizing investigations, counselling patients regarding expected recovery, and planning follow-up. The present study was conducted with the objectives of studying the etiological pattern of pale optic disc among patients attending a tertiary care hospital and correlating the etiological cause with visual outcome over six months of follow-up.

Methodology

Study design and setting

This prospective, hospital-based observational cohort study was conducted in the Department of Ophthalmology, SVS Medical College and Hospital, Yenugonda, Mahabubnagar, Telangana, India, from 06 th June 2023 to 05 th November 2023, including six months of follow-up for each participant. SVS Medical College and Hospital is a multispecialty tertiary-care teaching institution with approximately 750 teaching beds and provides outpatient, inpatient, emergency, operative, critical-care, laboratory, pharmacy, specialty-clinic, and advanced radiology services. The Ophthalmology Department provides comprehensive ocular assessment and coordinates neuroimaging and multidisciplinary referral when compressive, traumatic, inflammatory, infectious, or neurological disease is suspected. The cohort comprised adult patients attending the ophthalmology outpatient department with optic disc pallor confirmed on fundus examination.

Study participants

Patients aged more than 20 years with pale optic disc on fundus examination were eligible. Unconscious or terminally ill patients, known cases of glaucoma, patients with no perception of light, and patients whose disc pallor was primarily attributable to retinal disease were excluded. Consecutive eligible patients were approached until the required sample size was achieved. Written informed consent was obtained before enrolment. Demographic information, duration of symptoms, laterality, ocular history, trauma, systemic illness, drug exposure, alcohol intake, and previous neurological or neurosurgical events were recorded using a structured proforma.

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Sample size

The sample size was calculated for estimating a single proportion. Because no dependable local estimate of visual improvement among patients with pale optic disc was available, an expected proportion of 50% was used to provide the maximum variance. At 95% confidence and 14% absolute precision, the formula $n = Z^2p(1-p)/d^2$ yielded 49 patients ($1.96^2 \times 0.50 \times 0.50 / 0.14^2 = 49.0$). The target was rounded to 50 participants, all of whom completed follow-up and were included in the analysis.

Bias control

Potential sources of selection bias were reduced through consecutive enrolment of all eligible patients. Information bias was limited by using a structured proforma and a uniform examination sequence. Etiological classification was based on predefined clinical criteria supported by laboratory testing and CT or MRI when indicated, thereby reducing diagnostic misclassification. The same operational definition of visual improvement was applied at baseline and six months. Follow-up reminders and scheduled review visits were used to limit attrition; all enrolled participants completed outcome assessment. Because the sample was small and several causes were rare, multivariable adjustment was not attempted, and this limitation was considered during interpretation.

Clinical evaluation and investigations

All participants underwent uncorrected and best-corrected visual acuity assessment using Snellen chart, refraction, intraocular pressure measurement by non-contact tonometry, slit-lamp anterior segment examination, fundus biomicroscopy, and fundus photography. Colour vision was assessed with Ishihara charts. Humphrey automated perimetry was performed where feasible, and contrast sensitivity was evaluated with Pelli-Robson chart in selected cases. Visual evoked potential testing was used in selected patients. Blood investigations included complete blood count, erythrocyte sedimentation rate, liver function tests, lipid profile, blood sugar testing, rheumatoid factor, C-reactive protein, Mantoux test, sputum acid-fast bacilli test, thyroid function tests, VCTC, and VDRL where indicated. CT brain or MRI brain/orbit was obtained based on clinical suspicion.

Outcome measures

Etiology was assigned after integrating history, ocular examination, systemic findings, laboratory results, and neuroimaging. Disc pallor was categorized as temporal, segmental, diffuse, or total pallor. The primary outcome variables were initial best-corrected visual acuity and best-corrected visual acuity at six months. Visual outcome was classified as improved when best-corrected visual acuity increased by at least one Snellen line. Etiology and type of disc pallor were considered secondary outcome variables.

Statistical analysis

Quantitative variables were summarized using mean, standard deviation, median, range, and 95% confidence intervals, whereas categorical variables were reported as frequencies and percentages. Normality was assessed using visual plots and the Shapiro-Wilk test. Categorical comparisons were performed using Pearson's chi-square test and two-sided Fisher's exact test. For the 10 × 2 comparison of etiology with visual improvement, the Pearson chi-square statistic was reported; because several expected cell frequencies were below five, the Fisher-Freeman-Halton exact test was treated as the primary significance test. Pairwise comparisons of major etiologies versus all remaining etiologies used two-sided Fisher's exact tests. Wilson 95% confidence intervals were calculated for improvement proportions, and Cramér's V quantified association strength. A two-sided p-value <0.05 was considered statistically significant. Analyses were performed using IBM SPSS Statistics version 22; the exact test was verified by conditional enumeration.

Ethical Considerations

The study was conducted after obtaining approval from the Institutional Ethics Committee of SVS Medical College and Hospital, Mahabubnagar, Telangana, India. Written informed consent was obtained from all participants before enrolment in the study. The study procedures were performed in accordance with ethical principles and the Declaration of Helsinki. Confidentiality of patient identity and clinical information was strictly maintained throughout the study. Participation was voluntary, and patients were allowed to withdraw from the study at any stage without affecting their treatment.

Results

During the study period, 50 potentially eligible patients were assessed. All 50 satisfied the eligibility criteria and consented to participate; no patient was excluded or declined enrolment. All enrolled participants completed the six-month follow-up, and complete outcome data were available for all 50 patients included in the final analysis (Figure 1).

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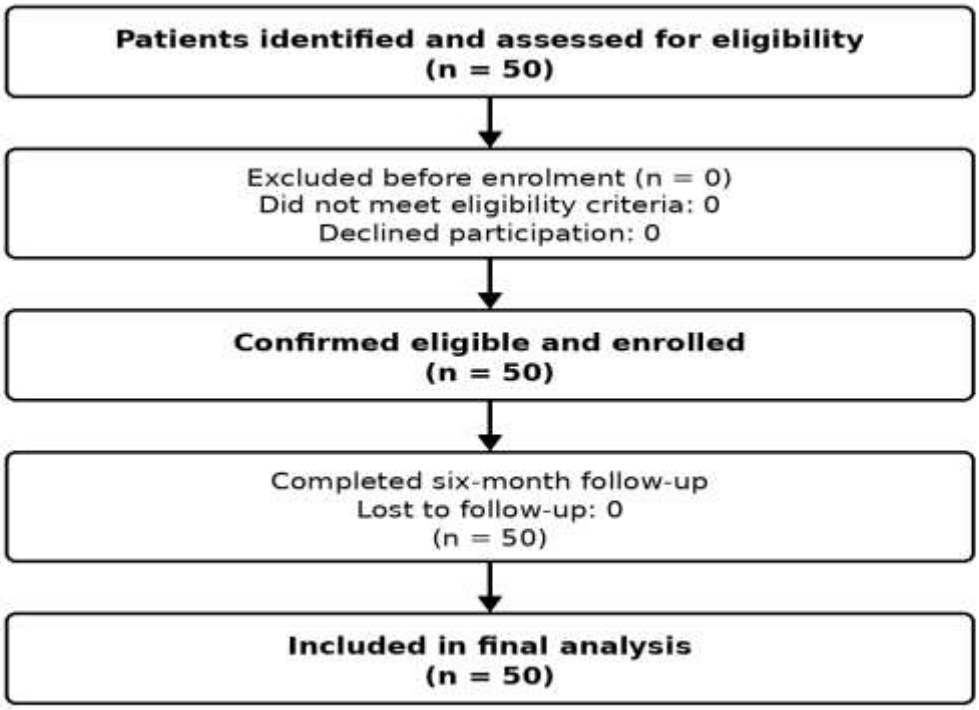

Figure 1. Participant flow through the study

The mean age was 38.68 ± 12.09 years (median, 35 years; range, 21-66 years). The mean symptom duration was 5.4 ± 9.3 years. Males constituted 56.0% of the cohort. Right-eye involvement was observed in 38.0%, left-eye involvement in 32.0%, and bilateral involvement in 30.0%. Baseline characteristics are presented in Table 1.

Table 1: *Baseline demographic and clinical characteristics of the study population (N = 50)*

Variable	Category / Statistic	Frequency / Value	Percentage / Measure
Age, years	Mean ± SD	38.68 ± 12.09	95% CI: 35.25-42.11
Age, years	Median [range]	35 [21-66]	-
Duration, years	Mean ± SD	5.4 ± 9.3	95% CI: 2.75-8.04
Duration, years	Median [range]	1.50 [0.10-35.00]	-
Sex	Male	28	56.0
Sex	Female	22	44.0
Eye involved	Right eye	19	38.0
Eye involved	Left eye	16	32.0

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Variable	Category / Statistic	Frequency / Value	Percentage / Measure
Eye involved	Both eyes	15	30.0

Past clinical history revealed trauma/head injury in 28.0%, optic neuritis in 14.0%, systemic hypertension or diabetes mellitus in 12.0%, and cerebellopontine angle tumor in 4.0%. Other relevant histories, including alcohol use, lacrimal gland tumor, neurosurgery, tuberculosis meningitis, hyperlipidemia, ethambutol intake, and thyroid orbitopathy, were each documented in 2.0% of patients. Details are provided in Table 2.

Table 2: *Distribution of positive past clinical history (N = 50)*

Past clinical history	Frequency	Percentage
Trauma/head injury	14	28.0
Optic neuritis	7	14.0
Cerebellopontine angle tumor	2	4.0
Systemic hypertension/diabetes mellitus	6	12.0
Alcohol use	1	2.0
Lacrimal gland tumor	1	2.0
Post-neurosurgery status	1	2.0
Post-tuberculosis meningitis	1	2.0
Hyperlipidemia	1	2.0
Ethambutol intake	1	2.0
Thyroid orbitopathy	1	2.0

Etiological analysis showed that traumatic optic neuropathy was the most frequent cause of pale optic disc, accounting for 28.0% of cases. Optic neuritis and tumor-related optic neuropathy each accounted for 22.0%, followed by primary optic atrophy in 16.0%. Rare causes included toxic optic neuropathy, inflammatory thyroid-related optic neuropathy, tuberculosis-related infection, post-papilloedema optic atrophy, ethambutol-induced optic neuropathy, and anterior ischemic optic neuropathy. The pattern of disc pallor differed by etiology, as summarized in Table 3.

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Table 3: *Etiology and pattern of optic disc pallor (N = 50)*

Etiology	Disc pallor pattern	Frequency	Percentage
Optic neuritis	Segmental disc pallor	11	22.0
Tumor-related optic neuropathy	Diffuse disc pallor	11	22.0
Primary optic atrophy	Total disc pallor	8	16.0
Toxic optic neuropathy	Temporal pallor	1	2.0
Inflammatory thyroid-related optic neuropathy	Diffuse pallor	1	2.0
Tuberculosis-related infection	Diffuse pallor	1	2.0
Post-papilloedema optic atrophy	Diffuse pallor	1	2.0
Traumatic optic neuropathy	Temporal pallor	14	28.0
Ethambutol-induced optic neuropathy	Temporal pallor	1	2.0
Anterior ischemic optic neuropathy	Segmental pallor	1	2.0

Most blood investigations were within normal limits. Abnormal or contributory laboratory/systemic findings were detected in a small proportion of cases. CT brain revealed fracture of frontal bone in 10.0%, parietal space-occupying lesion in 8.0%, and fracture of optic canal in 6.0%. MRI showed demyelinating plaques in 22.0% and tumor or shunting-related abnormalities in selected patients. Investigation findings are presented in Table 4.

Table 4: *Laboratory and neuroimaging findings (N = 50)*

Investigation	Finding	Frequency	Percentage
Blood investigations	Within normal limits	45	90.0
Blood investigations	Chest X-ray cavity in right lung base	1	2.0
Blood investigations	Hypertensive cardiomyopathy	1	2.0
Blood investigations	Increased lipid profile	1	2.0
Blood investigations	Sputum AFB positive	1	2.0
Blood investigations	Thyroid function tests within control	1	2.0
CT brain	Nil	29	58.0
CT brain	Fracture of frontal bone	5	10.0
CT brain	Parietal SOL	4	8.0

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Investigation	Finding	Frequency	Percentage
CT brain	Fracture of optic canal	3	6.0
CT brain	Fracture in temporal bone	1	2.0
CT brain	Lacrimal gland tumor involving orbital apex	1	2.0
CT brain	Pituitary tumor	1	2.0
CT brain	Shunting	1	2.0
MRI brain/orbit	Nil	31	62.0
MRI brain/orbit	Demyelinating plaques	11	22.0
MRI brain/orbit	Parietal SOL	2	4.0
MRI brain/orbit	Shunting seen	2	4.0
MRI brain/orbit	Increased extraocular muscle thickness	1	2.0
MRI brain/orbit	Lacrimal gland tumor involving orbital apex	1	2.0
MRI brain/orbit	Lesion in pituitary region	1	2.0
MRI brain/orbit	Pineal astrocytoma	1	2.0

Treatment was individualized according to etiology. Post-intravenous steroid treatment was documented in 28.0%, ONTT-based management in 22.0%, and observation in 18.0%. Excision, tumor excision status, decompression status, alcohol control, systemic risk factor control, stopping antitubercular therapy/ethambutol, and radiation were used in selected patients. Treatment distribution is shown in Table 5.

Table 5: *Treatment modalities used in the study population (N = 50)*

Treatment modality	Frequency	Percentage
Post-intravenous steroids	14	28.0
ONTT-based regimen	11	22.0
Observation	9	18.0
Excision	6	12.0
Post-tumor excision status	3	6.0
Post-decompression status	2	4.0
Control of alcohol use	1	2.0
Control of systemic hypertension/hyperlipidemia	2	4.0

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Treatment modality	Frequency	Percentage
Observation and stoppage of ATT/ethambutol	1	2.0
Radiation	1	2.0

At six months, 17 of 50 patients improved by at least one Snellen line (34.0%; 95% CI, 22.4%-47.8%), whereas 33 (66.0%) did not improve. Improvement was observed in 8 of 11 patients with optic neuritis (72.7%; 95% CI, 43.4%-90.3%), 6 of 11 with tumor-related optic neuropathy (54.5%; 95% CI, 28.0%-78.7%), and 3 of 14 with traumatic optic neuropathy (21.4%; 95% CI, 7.6%-47.6%). None of the eight patients with primary optic atrophy improved. Etiology was significantly associated with visual improvement (Pearson $\chi^2(9)=17.62$, p=0.040; Fisher-Freeman-Halton exact p=0.011), with a large effect size (Cramér's V=0.59). Pairwise Fisher's exact testing showed a significantly higher likelihood of improvement in optic neuritis than in all other etiologies combined (p=0.004), whereas tumor-related (p=0.151) and traumatic cases (p=0.327) did not reach statistical significance.

Table 6: *Association between etiology and six-month visual outcome (N = 50)*

Etiology	Improved, n (%)	Not improved, n (%)	95% CI for improvement	Pairwise p
Overall	17 (34.0)	33 (66.0)	22.4%-47.8%	0.011*
Optic neuritis	8 (72.7)	3 (27.3)	43.4%-90.3%	0.004
Tumor-related optic neuropathy	6 (54.5)	5 (45.5)	28.0%-78.7%	0.151
Primary optic atrophy	0 (0.0)	8 (100.0)	0.0%-32.4%	0.039
Traumatic optic neuropathy	3 (21.4)	11 (78.6)	7.6%-47.6%	0.327
Toxic optic neuropathy	0 (0.0)	1 (100.0)	0.0%-79.3%	—
Inflammatory thyroid-related optic neuropathy	0 (0.0)	1 (100.0)	0.0%-79.3%	—
Tuberculosis-related infection	0 (0.0)	1 (100.0)	0.0%-79.3%	—
Post-papilloedema optic atrophy	0 (0.0)	1 (100.0)	0.0%-79.3%	—
Ethambutol-induced optic neuropathy	0 (0.0)	1 (100.0)	0.0%-79.3%	—
Anterior ischemic optic neuropathy	0 (0.0)	1 (100.0)	0.0%-79.3%	—

Note. CI = Wilson confidence interval. The overall Pearson chi-square result was $\chi^2(9)=17.62$, p=0.040; because sparse expected frequencies were present, the Fisher-Freeman-Halton exact p=0.011 (*) was treated as the primary test. Cramér's V=0.59. Pairwise p-values compare each major etiology with all remaining etiologies using two-sided Fisher's exact tests.

Pairwise analyses for single-patient categories were not interpreted.

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The BCVA distribution among major etiologies showed that optic neuritis had the most favourable shift, with several patients improving to 6/18 or 6/12 at six months. Tumor-related optic neuropathy also showed improvement in selected cases, whereas primary optic atrophy remained unchanged across all documented categories. Traumatic optic neuropathy showed limited improvement, with most cases remaining at 6/60 or worse. Detailed BCVA distribution is shown in Table 7.

Table 7: *Initial and six-month best-corrected visual acuity distribution for major etiologies*

Etiology	Initial BCVA distribution	Six-month BCVA distribution	Interpretation
Optic neuritis [n=11]	6/60: 9; 6/36: 2	6/60: 1; 6/36: 3; 6/24: 1; 6/18: 3; 6/12: 3	Favourable visual shift
Traumatic optic neuropathy [n=14]	4/60: 2; 5/60: 2; 6/60: 7; 6/36: 3	4/60: 1; 5/60: 1; 6/60: 8; 6/36: 3; 6/24: 1	Limited improvement
Primary optic atrophy [n=8]	2/60: 2; 3/60: 1; 4/60: 1; 5/60: 2; 6/60: 2	2/60: 2; 3/60: 1; 4/60: 1; 5/60: 2; 6/60: 2	No documented improvement
Tumor-related optic neuropathy [n=11]	2/60: 2; 4/60: 1; 6/60: 3; 6/36: 2; 6/18: 2; 6/12: 1	2/60: 2; 6/60: 2; 6/36: 2; 6/18: 3; 6/12: 2	Improvement in selected cases

Among rare etiologies, the single cases of toxic optic neuropathy, inflammatory thyroid-related optic neuropathy, tuberculosis-related optic neuropathy, post-papilloedema optic atrophy, anterior ischemic optic neuropathy, and ethambutol-induced optic neuropathy retained the same BCVA category at six months. Thus, the observed visual recovery in this study was concentrated mainly among optic neuritis, tumor-related optic neuropathy, and a smaller subset of traumatic optic neuropathy cases.

Discussion

This prospective observational study evaluated the etiological spectrum of pale optic disc and its relationship with six-month visual outcome in a tertiary care hospital population. The cohort was relatively young, with a mean age of 38.68 years, and showed a male predominance. The most frequent etiology was traumatic optic neuropathy, followed by optic neuritis and tumor-related optic neuropathy. This distribution reflects the broad differential diagnosis of optic neuropathy described in neuro-ophthalmic literature, where demyelinating, traumatic, compressive, ischemic, inflammatory, infectious, toxic, and nutritional disorders can culminate in optic disc pallor [1-3].

The predominance of traumatic optic neuropathy in the present series is clinically important. Trauma accounted for 28.0% of cases and was associated with temporal pallor, optic canal or cranial fractures in selected patients, and poor visual recovery in most cases. Only 3 of 14 traumatic cases showed one-line BCVA improvement. This finding is consistent with the uncertain therapeutic benefit and variable prognosis reported in traumatic optic neuropathy studies, where initial severity, associated optic canal injury, and timing of intervention strongly influence recovery [7,8]. The high proportion of trauma also explains the young adult profile and male excess observed in this cohort.

Optic neuritis accounted for 22.0% of cases and demonstrated the best functional recovery, with 8 of 11 patients improving by at least one Snellen line (72.7%; 95% CI, 43.4%-90.3%). The pairwise association was statistically significant (Fisher's exact p=0.004). This favourable pattern agrees with the Optic Neuritis Treatment Trial and subsequent evidence showing that corticosteroid therapy accelerates recovery in acute optic neuritis, although long-term final visual acuity is often less dependent on steroid exposure [4-6]. MRI evidence of demyelinating plaques in 22.0% also supports neuroimaging in suspected inflammatory or demyelinating optic neuropathy. In contrast, primary optic atrophy showed no improvement, consistent with the limited reversibility of established axonal loss.

Tumor-related optic neuropathy contributed 22.0% of cases, and 6 of 11 patients improved (54.5%; 95% CI, 28.0%-78.7%). Although the pairwise comparison did not reach statistical

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significance (Fisher's exact p=0.151), the direction of effect supports early detection of compressive lesions, because recovery depends on the duration of compression, pretreatment optic nerve reserve, and adequacy of decompression or tumor-directed therapy. Population-based evidence similarly indicates that earlier treatment of compressive optic neuropathy is associated with better visual outcomes [9].

Overall, 34.0% of patients improved (95% CI, 22.4%-47.8%). The significant overall exact association between etiology and outcome (Fisher-Freeman-Halton p=0.011; Cramér's V=0.59) indicates that prognosis was strongly cause-dependent, although sparse subgroups limit precision. The single case of anterior ischemic optic neuropathy showed no measurable improvement; ischemic optic neuropathies often produce persistent visual impairment, and prognosis varies with the ischemic mechanism and baseline damage [13,14]. The modest overall recovery rate suggests that optic disc pallor is frequently recognized after irreversible injury has occurred. Consequently, disc pallor should prompt structured etiological evaluation rather than observation alone, using history, fundus examination, colour vision, visual fields, laboratory screening, and CT or MRI according to clinical suspicion [2,3,12].

Generalizability

The findings are most applicable to tertiary ophthalmology centres in similar Indian settings where young trauma patients, optic neuritis, intracranial tumors, tuberculosis, toxic exposure, and systemic vascular risk factors are encountered together. The hospital-based design, 50-patient sample, and six-month follow-up restrict population-level extrapolation and reduce certainty for rare etiologies. Even so, the etiological pattern and outcome gradient offer practical guidance for comparable outpatient and neuro-ophthalmology services managing pale optic disc.

Conclusion

Pale optic disc in this tertiary care cohort represented a heterogeneous group of optic neuropathies rather than a single diagnosis. Traumatic optic neuropathy was the most common etiology, followed by optic neuritis, tumor-related optic neuropathy, and primary optic atrophy. Visual outcome was guarded, with only one-third of patients gaining at least one Snellen line at six months. Optic neuritis showed the most favourable recovery, while primary optic atrophy and most rare etiologies remained unchanged. These findings support early etiological identification, prompt neuroimaging when indicated, systemic evaluation, and close follow-up. A cause-directed approach is essential to preserve residual visual function and prevent avoidable progression in patients with pale optic disc.

Limitations

The study was limited by small sample size, single-centre hospital-based recruitment, and absence of a control group. Several etiological subgroups contained only one patient, restricting comparative interpretation. Follow-up was limited to six months, which reduced assessment of delayed recovery. Advanced imaging markers such as OCT retinal nerve fibre layer thickness and visual field indices were not uniformly analysed across all participants.

Recommendations

Every patient with pale optic disc should undergo structured neuro-ophthalmic evaluation, including detailed history, visual acuity, pupil assessment, colour vision, visual field testing, fundus documentation, and targeted systemic assessment. CT or MRI should be performed early when trauma, demyelination, tumor, orbital apex disease, raised intracranial pressure, or atypical features are suspected. Patients receiving ethambutol or exposed to toxins require baseline and follow-up ocular monitoring. Trauma prevention, timely referral after head injury, and multidisciplinary care involving ophthalmology, neurology, neurosurgery, medicine, and radiology can improve diagnostic accuracy. Patient counselling should include realistic recovery expectations and warning signs requiring urgent review.

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Abbreviations

AFB – Acid-fast bacilli;
ATT – Antitubercular therapy;
BCVA – Best-corrected visual acuity;
CI – Confidence interval;
CT – Computed tomography;
MRI – Magnetic resonance imaging;
ONTT – Optic Neuritis Treatment Trial;
OCT – Optical coherence tomography;

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SD – Standard deviation;
SOL – Space-occupying lesion;
VDRL – Venereal Disease Research Laboratory.

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Conflict of interest

The authors declare no conflict of interest.

Author contributions

YB-Concept and design of the study, clinical evaluation, data collection, results interpretation, review of literature and preparation of the first draft of the manuscript. **BS**-Concept and design of the study, clinical evaluation, data collection, statistical interpretation, manuscript revision and corresponding author responsibilities

BC-Review of literature, supervision of clinical assessment, results interpretation, critical revision of the manuscript and final approval of the manuscript

Data availability

Data available on request

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