e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(11); 873-877

Original Research Article

Clinical and Etiological Profile of Optic Atrophy in Patients Attending a Tertiary Eye Care Centre

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Received: 13-09-2025 / Revised: 12-10-2025 / Accepted: 13-11-2025

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Conflict of interest: Nil

Abstract:

Background: Optic atrophy represents the final common pathway of various optic nerve insults leading to irreversible visual impairment. Identifying its etiological spectrum is vital for targeted diagnosis and management.

Aim: To study the clinical and demographic profile of patients presenting with optic atrophy at a tertiary eye care centre.

Methods: This prospective observational study was conducted at the Lions Club of Hyderabad Sadhuram Eye Hospital and Post Graduate Institute of Ophthalmology, Hyderabad, from October 2018 to March 2020. A total of 106 patients with clinically diagnosed optic atrophy were included. Detailed demographic, clinical, and etiological evaluations were performed, including ocular and systemic assessments. Data were analyzed using SPSS version 19.0, with qualitative variables expressed as percentages and quantitative variables as mean ± SD.

Results: Most patients belonged to the 41–50 years age group (29.2%), with male predominance (74.5%). The leading causes were glaucomatous optic atrophy (19.8%), NAION and TRON (14.2% each), nutritional (10.4%), and intracranial lesions (10.4%). Unilateral cases were mainly traumatic or ischemic, while bilateral cases were largely nutritional or toxic in origin.

Conclusion: Glaucoma, ischemic, and nutritional etiologies are predominant causes of optic atrophy. Early identification of systemic and toxic risk factors can help prevent progression and bilateral involvement.

Keywords: Optic Atrophy; Glaucoma; NAION; Toxic Neuropathy; Nutritional Deficiency

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Introduction

Optic atrophy is the terminal, non-specific manifestation of damage to the retinogeniculate pathway, characterized by pallor of the optic disc due to loss of retinal ganglion cell axons and supporting glial remodeling [1]. Though the term atrophy is a misnomer (since it suggests disuse, whereas the underlying mechanism is degeneration), optic atrophy essentially reflects a final common pathway of many different insults to the optic nerve or its upstream connections [2]. Because optic atrophy is a sign not a diagnosis uncovering the etiology is critical: the cause may range from compressive lesions, ischemic insults, hereditary or mitochondrial optic neuropathies, inflammation, trauma or toxicity, to nutritional or metabolic disorders [2, 3]. In clinical practice, patients present with diminution of vision, visual field defects, color vision impairment, and a relative afferent pupillary defect, and the pattern of disc pallor (diffuse, temporal, sectoral) may offer clues to the underlying cause [2].

In view of this, the aim of the present study is to evaluate the clinical and demographic profile of patients presenting with a diagnosis of optic atrophy at a tertiary eye care center, with a particular focus on delineating the etiological spectrum. The objectives are: (i) to characterise the demographic features (age, sex, laterality) of patients with optic atrophy; and (ii) to systematically categorise the etiologies responsible.

Methods

This prospective observational study was conducted at the Lions Club of Hyderabad Sadhuram Eye Hospital and Post Graduate Institute of Ophthalmology, Hyderabad, a tertiary care eye centre catering to a large population. The study period extended from October 2018 to March 2020.

Approval for the study protocol was obtained from the Institutional Ethics Committee prior to initiation. The study population comprised all patients presenting with a diagnosis of optic atrophy, either as a primary diagnosis or referred from other centres. Optic atrophy was diagnosed based on characteristic clinical features observed on ocular examination, corroborated by history, visual acuity testing, and supporting ancillary findings.

The inclusion criteria comprised male and female patients diagnosed with optic atrophy, presenting with decreased visual acuity, dyschromatopsia, visual field defects such as central, arcuate or altitudinal scotomas, diminished contrast sensitivity, and demonstrable absolute or relative afferent pupillary defect. Patients with media opacities precluding optic disc evaluation and single-eyed individuals were excluded.

All patients underwent a structured evaluation protocol designed for the study. A detailed demographic profile including age, sex, occupation, and socioeconomic status was recorded, along with presenting complaints and duration of symptoms. Comprehensive ocular examination consisted of visual acuity assessment using LogMAR charts for distance and appropriate English reading or numerical charts for near, colour vision testing with the Ishihara pseudoisochromatic chart, and contrast sensitivity assessment with digital low contrast charts. Extraocular motility was tested, followed by cover tests to identify ocular misalignment. Pupillary reactions were evaluated for anisocoria and relative afferent pupillary defect (RAPD), graded systematically from Grade 1 to Grade 5 depending on the degree of pupillary constriction and redilatation.

Anterior segment evaluation was performed using slit lamp biomicroscopy, intraocular pressure was measured with Goldmann applanation tonometry, and gonioscopy was carried out with a four-mirror goniolens. Fundus evaluation included direct and indirect ophthalmoscopy, supplemented by 90D slit lamp biomicroscopy. Fundus photography was performed to document optic disc changes. Patients were categorised clinically into primary, secondary, consecutive, or glaucomatous optic atrophy, and further subclassified according to etiology, which included hereditary, ischemic, compressive, inflammatory, toxic, traumatic, or nutritional causes. In selected patients, additional investigations such as neuroimaging, visual evoked potentials, and systemic work-up were carried out to confirm the etiology.

For statistical analysis, qualitative variables were presented in frequencies and percentages, while quantitative variables were expressed using mean and standard deviation. Data were entered in Microsoft Excel and analysed using SPSS version 19.0. Subgroup comparisons were carried by using chi-square test for categorical variables and t-test or ANOVA for continuous variables, with a p-value of <0.05 considered statistically significant.

Results:

The present study included 106 patients with optic atrophy, majority (29.2%) belonging to the 41–50 years group, followed by 51–60 years (20.8%) and 61–70 years (19.8%). Males predominated (74.5%). Optic atrophy due to retinitis pigmentosa was noted in six cases, predominantly during the 4th–5th decades. Overall, glaucomatous optic atrophy was the most frequent cause (19.8%), followed by NAION and TRON (14.2% each), nutritional neuropathy (10.4%), and intracranial lesions (10.4%).

Unilateral optic atrophy was seen in 48 patients, with TRON (29.2%) and glaucomatous optic atrophy (27.1%) being the leading causes, followed by NAION (20.8%) and CRAO (16.7%). Among 58 bilateral cases, nutritional optic neuropathy (19%) and toxic optic neuropathy (15.5%) were predominant, with additional contributions from intracranial lesions (15.5%), glaucoma (13.8%), and retinitis pigmentosa (10.3%). Fifteen NAION patients had multiple risk factors, with hypertension and diabetes (20%) being most common, along with metabolic derangements including dyslipidemia, hyperhomocysteinemia, and CAD. In NAION, 70% of patients had the fellow eye disc at risk.

Toxic optic neuropathy risk factors included tobacco smoking (60%), ATT-related ethambutol toxicity (30%), and combined ATT with B12 deficiency (10%). Nutritional optic neuropathy was mainly due to microcytic hypochromic anaemia with B12 deficiency (66.7%). CRAO patients showed vascular comorbidities, most frequently hypertension, diabetes, and CAD combined (25%). Visual acuity assessment revealed profound impairment (<CF1m to PL) in many unilateral cases, especially glaucoma and CRAO, while bilateral cases commonly showed moderate to severe impairment. Among 21 glaucomatous optic atrophy patients, primary open-angle glaucoma was most common (47.6%), followed by primary angle closure (33.3%) and secondary subtypes.

Discussion

The demographic and etiologic profile in this cohort largely mirrors patterns described in the literature, but also reveals some distinctive local trends. The concentration of cases in the 41–50 year age bracket (29.2%) with a gradual decline into older decades suggests that optic atrophy in this setting is often a consequence of middle-aged onset pathologies, rather than solely senescent degeneration. The male predominance (74.5%) is consistent with earlier series, such as the Indian review of 100 cases where

males constituted 66% of subjects, though that series showed younger age predilection as well (66 males out of 100; 73% with identifiable cause) [4]. That earlier study also noted that 27% of cases remained idiopathic, emphasizing that even in well-characterized cohorts, a proportion lacks a definable cause.

In our series, glaucomatous optic atrophy emerged as the leading cause (19.8 %), followed by NAION and TRON (14.2 % each), and nutritional optic neuropathy (10.4 %) and intracranial lesions (10.4 %) equally contributing. That glaucoma is the top contributor aligns with the global burden of glaucomatous optic neuropathy—affecting over 60 million individuals worldwide and causing irreversible optic nerve damage via retinal ganglion cell degeneration and cup-to-disc changes [5]. The significant share of ischemic etiologies (NAION, TRON) is also in keeping with the fact that nonarteritic anterior ischemic optic neuropathy is the most common acute optic neuropathy in adults over 50, with an estimated incidence of 2.3 to 10.3 per 100,000 per year in U.S. populations [6]. The representation of nutritional optic neuropathy underscores the continuing significance of metabolic and micronutrient deficiencies in optic nerve injury, particularly in tertiary hospital catchment areas where systemic comorbidities are common.

Thus, while our findings substantiate global trends favoring glaucoma and ischemic causes, the proportionate contribution of nutritional and intracranial etiologies highlights regional health determinants. The relatively smaller yet non-negligible fraction due to intracranial lesions underscores the importance of neuroimaging and neurologic workup in unexplained optic atrophy, echoing findings from other tertiary-centre case series [3].

The distinction between unilateral and bilateral optic atrophy in your series highlights divergent etiological spectra and possibly distinct pathogenetic mechanisms. In the 48 unilateral cases, traumatic optic neuropathy (TRON) comprised 29.2 %, and glaucomatous atrophy 27.1 %, followed by NAION at 20.8 % and central retinal artery occlusion (CRAO) at 16.7 %. The prominence of TRON in unilateral presentations is biologically plausible: traumatic optic neuropathy is classically unilateral, often resulting from indirect head or orbital injury transmitting forces to the optic canal, and leading to axonal shearing, ischemia, and secondary optic atrophy in a delayed fashion [7, 8]. The relatively high proportion of glaucomatous atrophy in unilateral eyes may reflect asymmetric disease progression or earlier manifestation in one eye, though glaucoma typically is bilateral but asymmetric. In contrast, bilateral optic atrophy (n = 58) showed predominance of nutritional (19 %) and toxic (15.5 %) optic neuropathy, along with contributions from intracranial lesions (15.5 %), glaucoma (13.8 %) and retinitis pigmentosa (10.3 %). This pattern is consistent with systemic exposures or metabolic insults that tend to affect both optic nerves symmetrically, e.g. toxins, vitamin deficiencies, or diffuse central nervous system processes. The bilateral involvement of intracranial lesions also underscores how compressive or infiltrative pathology along the chiasm or optic pathways may affect both sides.

Focusing on the NAION subset (n = 15), the observation of multiple concurrent risk factors hypertension with diabetes (20 %) and other metabolic derangements such as dyslipidemia, hyperhomocysteinemia, and coronary artery disease (CAD) emphasizes the multifactorial vascular burden in ischemic optic neuropathy. NAION is understood as a small-vessel ischemic event in the anterior optic nerve head, occurring in susceptible crowded discs (disc-at-risk) in the presence of systemic vascular risk factors [9]. The finding that 70 % of NAION patients had the fellow eye disc at risk (crowded disc anatomy) aligns with previous literature that disc morphology (small cup-to-disc ratio) is a significant predisposing feature [10]. In large series, new NAION in the fellow eye occurs in about 10–15 % over years, although rates vary by cohort and follow-up (e.g. 14.7 % in one follow-up over 5 years) [10]. The relatively high proportion (70 %) with fellow eye disc-at-risk in your series suggests many of your NAION eyes emerged in anatomically susceptible discs—possibly predicting future bilateral involvement. Together, your findings support that unilateral optic atrophy is more likely related to localized injury (trauma, ischemia, vascular occlusion), while bilateral optic atrophy favors systemic, metabolic, toxic or neurologic etiologies; and in NAION patients, vascular comorbidities and anatomical predisposition warrant close monitoring of the fellow eye.

The predominance of toxic optic neuropathy risk factors in your series—tobacco smoking in 60%, ethambutol-related toxicity in 30%, and combined ethambutol with B12 deficiency in 10%—underscores the multifactorial vulnerability of the optic nerve to both exogenous and endogenous insults. Ethambutol-induced optic neuropathy is a well-recognized, though dose- and duration-dependent, ocular toxicity associated with antitubercular therapy; early detection and discontinuation of the drug are critical, though recovery may be incomplete, particularly in older patients [11]. The drug's chelating properties interfere with mitochondrial function in retinal ganglion cell axons, leading to dysfunction and eventual apoptosis [12]. In many reports, color vision deficits, central scotomata, and visual acuity loss precede optic disc pallor, which may only

appear later in the disease course [13]. The additive role of B12 deficiency likely compounds mitochondrial injury and oxidative stress in the optic nerve, an association supported by published cases of optic neuropathy in B12-deficient individuals [14].

Your series also highlights nutritional optic neuropathy (B12-associated microcytic hypochromic anemia in 66.7% of cases) as a significant cause of bilateral optic atrophy. This is consistent with reviews that emphasize vitamin B12 among the key micronutrient deficiencies implicated in nutritional optic neuropathy, often alongside folate, thiamine, and copper deficits [15]. Chronic B12 deficiency has been mechanistically linked to retinal ganglion cell loss via mitochondrial dysfunction and superoxide-mediated apoptosis [16]. Meanwhile, in your CRAO subset, vascular comorbidities—particularly hypertension, diabetes, and coronary artery disease (25%)—predominated. This mirrors the known risk profile for CRAO, whose pathophysiology parallels that of ischemic stroke, with hypertension, diabetes, hyperlipidemia, and vascular disease being established risk factors [17]. Visual acuity outcomes in your cohort were poor: unilateral cases, particularly from glaucoma and CRAO, commonly had profound impairment (<CF1 m to perception of light), while bilateral cases more often had moderate to severe loss. Among 21 glaucomatous optic atrophy cases, primary open-angle glaucoma was most frequent (47.6%), followed by primary angle closure (33.3%) and secondary variants. This predominance of open-angle glaucoma aligns with global epidemiology POAG constitutes around three-quarters of all glaucomas in many cohorts.

Overall, your data reinforce that toxic and nutritional etiologies remain important, and often modifiable, contributors to optic atrophy in your population, particularly in bilateral presentations. Meanwhile, vascular occlusive events like CRAO confer severe and often irreversible vision loss, and glaucomatous damage remains a significant chronic burden. A strong implication is the need for vigilant screening—especially among patients on ethambutol therapy, those with nutritional deficiencies, and those with vascular comorbidity—to identify optic nerve injury early and possibly preserve residual vision.

In conclusion, toxic and nutritional optic neuropathies significantly contribute to bilateral optic atrophy, often linked to smoking, ethambutol toxicity, and vitamin B12 deficiency. CRAO cases demonstrated strong vascular associations such as hypertension, diabetes, and CAD, emphasizing systemic disease control. Visual acuity loss was severe in glaucoma and CRAO, highlighting irreversible neuronal damage. Primary open-angle glaucoma remained the leading subtype, reaffirming the need for early screening, metabolic management, and patient education to prevent optic nerve damage progression.

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