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Original Research Article

Study of Pale Optic Disc and Its Correlation with Visual Outcome

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Abstract

Background: Pale optic disc is due to damage to optic nerve. It could have many etiologies. Proper diagnose and treatments is very important because fundus signs that reflects loss or dysfunction of retinal neurons.

Method: Every patients undergone biochemical investigations and neuro imaging studies. The patient were followed up for 6 (Six) months. Clinical examinations included visual acuity by using snellen's charts, IOP measurement by NCT, refraction anterior sequent examination, slit-lamp biomicroscopy fundus. Photography by fundus camera colour vision by Inshihara's chart, visual fields by Humphreys automated perimeter contrast sensitivity by Pelli Robson's chart.

Results: Out of 48 cases 18(37.5%) had right eye, 15 (31.5%) left eye 15 (31.5%) both eyes were involved. Major history was 13 (27%) trauma (Head injury) followed by 6 (12.5%) optic neuritis and 5 (10.4%) SHT/DM. Radiological investigation had 4 (8.3%) fracture of frontal bone 3 (6.25%) parietal SOL, 3 (6.25%) fracture of optic canal MRI study had 10 (2.8%) demyelinating plaques. BCVA repon revealed 16 (33.3%) improved, 32 (66.6%) not improved.

Conclusion: Pale optic disc has heterogeneous causes a proper diagnose of optic disc pallor will help the ophthalmologist to treat actual cause of disease, so that patients can lead a normal quality of life.

Keywords: Snellen's Chart, IOP, Slit-lamp, biomicroscopy fundus, Ophthalmoscope, BCVA.

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Introduction

Pale optic disc is mainly caused by damage to the optic nerve which is developmentally part and parcel of the brain. Optic disc is clinically important, fundus sign that reflects loss or dysfunction of retinal ganglion. Cell axons within the anterior visual pathway. Optic disc pallor is not a single disease entity rather it represents the end stage of diverse optic nerve involving the retina to lateral geniculate pathway [1].

Degeneration of optic nerve fibers reduces the normal vascular and neural appearance of the disc and produces the pale optic nerve head observed during ophthalmoscopy. The patients with pale optic disc usually accompanied in the reduction in visual acuity colour desaturation, relative afferent pupillary defect contrast sensitivity loss and characteristic visual abnormalities [2]. Proper neuro-ophthalmic assessment is necessary to rule out. Best corrected visual acuity pupil examination, colour vision, visual fields, slit-lamp, biomicroscopy, fundus photography and retinal nerve fiber layer assessment help to characterize the functional and structural burden of optic nerve damage [4]. Hence an attempt is made to evaluate the etiology of pale optic disc.

Material and Method

48 adult patients age between 30 to 45 years regularly visited to adichunchungiri institute of medical sciences, Nagamangala, Mandya Karnataka were studied.

Inclusive Criteria: The patients clinically diagnosed as pale optic disc on fundus examination. The patients who gave their consent in writing for study were selected.

Exclusion Criteria: The patients below 18 years. Unconscious patients terminally ill with known glaucoma. The visual acuity of no light perception and pale optic disc due to retinal condition were excluded.

Method: Past history related to ophthalmology, socio-economic status, sex, age, habits were recorded. The biochemical investigations and neuro-imaging studies were carried to identify the etiology and clinical ocular examination was performed. The patients were followed up for 6 (Six) months for visual outcomes. Clinical examination include uncorrected and best corrected visual acuity using snellen's chart, IOP measurement by NCT, refraction anterior segment examination, slit lamp biomecroscoy, fundus examination using ophthalmoscope fundus photography by fundus camera, colour vision by Ishihara's chart, visual fields by Humphrey's automated perimetry, contrast sensitivity by Pelli Robson's chart visual evoked potential (if necessary in certain cases). Duration of study was Feb 2025 to march 2026

Statistical Analysis: The pale optic disc manifestations, types of disc pallor descriptive study of visual acuity. Analysis of visual acuity with other antilogies were classified with percentage. The statistical analysis was carried out in SPSS soft were the ratio of male and female was 2:1.

Observation and Results

Table 1: Involvement of affected eyes: 18 (37.5%) right eye, 15 (31.5%) left eye and 15 (31.5%) both eyes.

History of patients: 13(27%) Trauma (Head injury), 6 (12.5%) optic neuritis, 2 (4.16%) CP angle tumour, 5 (10.4%) SHT/DM, 1 (2.08%) alcoholic, 1(2.08%) lacrimal gland tumour, 1 (2.08%) hyperlipidemia, 1(2.08%) Ethambutol intake, 1 (2.08%) thyroid orbitopathy.

Investigations: 1 (2.08%) chest X-ray (in right lung base), 1 (2.08%)) hypertensive cardiomyopathy, 1 (2.08%) elevated lipid profile, 1 (2.08%) sputum AFB positive, 44 (91.6%) thyroid function test within control.

CT Brain: 4 (4.3%) fracture of frontal bone, 3 (6.25%) parietal SOL, 3 (6.25%) fracture of optic canal, 1 (2.08%) fracture of temporal bone, 1 (2.08%) lacrimal gland tumour, 1 (2.08%) pituitary tumour, 1 (2.08%) shunting, 28 (58.3%) nil (normal).

MRI study: 10 (20.8%) demyelinated plaques, 3 (4.16%) parietal SOL, 1 (2.08%) lacrimal gland tumour involving orbital apex 1 (2.08%) lesion in pituitary region, 1 (2.08%) pineal astrocytoma, 30 (62.5%) nil (normal).

Treatment details: 13 (27.08%) post I.V. steroids, ONTT 10 (20.8%), 8 (16.6%) observation, 5 (10.4%) excision, 3 (6.25%) post tumour excision, 2 (4.16%) post-decompression status, 1 (2.08%) control of alcohol, 2 (4.16%) control of STH/ hyperlipidemia, 1 (2.08%) observation of ATT, 1 (2.08%) radiation, BCVA 16 (33.3%) improved, 32 (66.6%) not improved.

Table 2: Analysis of disc pallor types 10 (20.8%) optic neuritis, 10 (20.8%) diffuse disc pallor, 7 (14.5%) primary optic atrophy, 1 (2.08%) toxic, 1 (2.08%) inflammatory thyroid, 1 (2.08%) diffuse pallor in infections tuberculosis, 1 (2.08%) diffuse pallor in post papilledema, 13 (27.1%) temporal pallor in traumatic optic neuropathy, 1 (2.08%) ethambutol induced temporal pallor, 1 (2.08%) AION segmental pallor.

Table 3: Descriptive study of visual acuity;

Optic neuritis:

- Initial BCVA - 8 (16.6%) 6/60, 2 (4.16%) 6/36.
- BCVA at 6 months – 1 (2.08%) 6/60, 3 (6.25%) 6/36, 1 (2.08%) 6/24, 3 (6.25%) 6/18, 3 (6.25%) 6/12.

Trauma:

- Initial BCVA – 2 (4.16%) 4/60, 2 (4.16%) 5/60, 6 (12.5%) 6/60, 3 (6.25%) 6/36.
- BCVA after 6 months – 1 (2.08%) 4/60, 1 (2.08%) 5/60, 7 (145. %) 6/60, 3 (6.25%) 6/36, 1 (2.08%) 6/24.

Primary optic atrophy:

- Initial BCVA – 2 (4.16%) 2/60, 1 (2.08%) 3/60, 1 (2.08%) 4/60, 2 (4.16%) 5/60, 2 (4.16%) 6/60.
- BCVA at 6 months – 2 (4.06%) 2/60, 1 (2.08%) 3/60, 1 (2.08%) 4/60, 2 (4.16%) 5/60, 2 (4.16%) 6/60.

Tumour:

- Initial BCVA – 2 (4.16%) 2/60, 1 (2.08%) 4/60, 3 (6.25%) 6/60, 2 (4.16%) 3/36, 2 (4.16%) 3/36, 2 (4.16%) 6/18, 1 (2.08%) 6/12.
- BCVA at 6 months – 2 (4.16%) 2/60, 2 (4.16%) 6/60, 2 (4.16%) 6/36, 3 (6.25%) 6/18, 2 (4.16%) 6/12.

Table 4: Analysis of visual acuity etiologies

- **Toxic: 6/36 –** 1 (2.08%) at initial BCVA, 1 (2.08%) at 6 month of BCVA
- Inflammatory – 6/60 – 1 (2.08%) at initial BCVA, 1 (2.08%) after 6 month of BCVA.
- Infection – TB – 4/60 – 1 (2.08%) initial BCVA, 1 (2.08%) after 6 month of BCVA.
- Post- papillodema – 6/36 – 1 (2.08%) at initial, 1 (2.08%) at 6 month of BCVA.
- Post AION – 5/60 – 1 (2.08%) at initial BCVA, 1 (2.08%) at 6 month of BCVA.
- Ethambutol induced – 6/36 – 1 (2.08%) at initial BCVA, 1 (2.08%) at 6 month of BCVA.

Table 1: Analysis of optic disc manifestations

		Frequency
Eye	Right Eye	18 (7.5%)
	Left Eye	15 (31.5%)
	Both Eye	15 (31.5%)
History	Trauma (Head injury)	13 (27%)
	Optic Neuritis	6 (12.5%)
	Cpangle Tumour	2 (4.16%)
	SHT /DM	5 (10.08%)
	Alcoholic	1 (2.08%)
	Lacrimal gland tumour	1 (2.08%)
	Post neurosurgery	1 (2.08%)
	Post meningitis	1 (2.08%)
	Hyperlipidemia	1 (2.08%)
	Ethambutol Intake	1 (2.08%)
	Thyroid orbitopathy	1 (2.08%)
Investigation	Chest X-ray cavity in right lungbase	1 (2.08%)
	Hypertensive cardiornyopathy	1 (2.08%)
	Elevated lipid profile	1 (2.08%)
	Sputum AFB positive	1 (2.08%)
	Thyroid Function test within control	44 (91.6%)
CT Brain	Fracture of Frontal bone	4 (8.3%)
	Parietal SOL	3 (6.25%)
	Fracture of optic canal	3 (6.25%)
	Fracture of temporal bone	1 (2.08%)
	Lacrimal gland tumour	1 (2.08%)
	Pituilary tumour	1 (2.08%)
	Shunting	1 (2.08%)
	Nil (Normal)	28 (58.3%)
MRI Brain study	Demyelinating plagues	10 (20.8%)
	Parietal SOL	2 (4.16%)
	Shunting seen	1 (2.8%)
	Increased EOM thickness	1 (2.8%)
	Lacrimal gland tumour involving orbital apex	1 (2.8%)
	A lesion in pituitary region	1 (2.8%)
	Pineal astrocytoma	1 (2.8%)
	Nil (normal)	30 (62.5%)
Treatment details	Post iv steroids	13 (27.08%)
	ONTT	10 (20.8%)
	Observation	8 (16.6%)
	Excision	5 (10.4%)
	Post tumour excision	3 (6.25%)
	Post decompression status	2 (4.16%)
	Control of alcohol	1 (2.08%)
	Control of SHT / hyper lipidemia	2 (4.16%)
	Observation and stop of ATT	1 (2.08%)
	Radiation	1 (2.08%)
BCVA	Improved	16 (33.3%)
	Not improved	32 (66.6%)

ONTT = Optic Neuritis Treatment Trial, ATT = Antitubercular therapy, BCVA = Best Corrected Visual Acuity

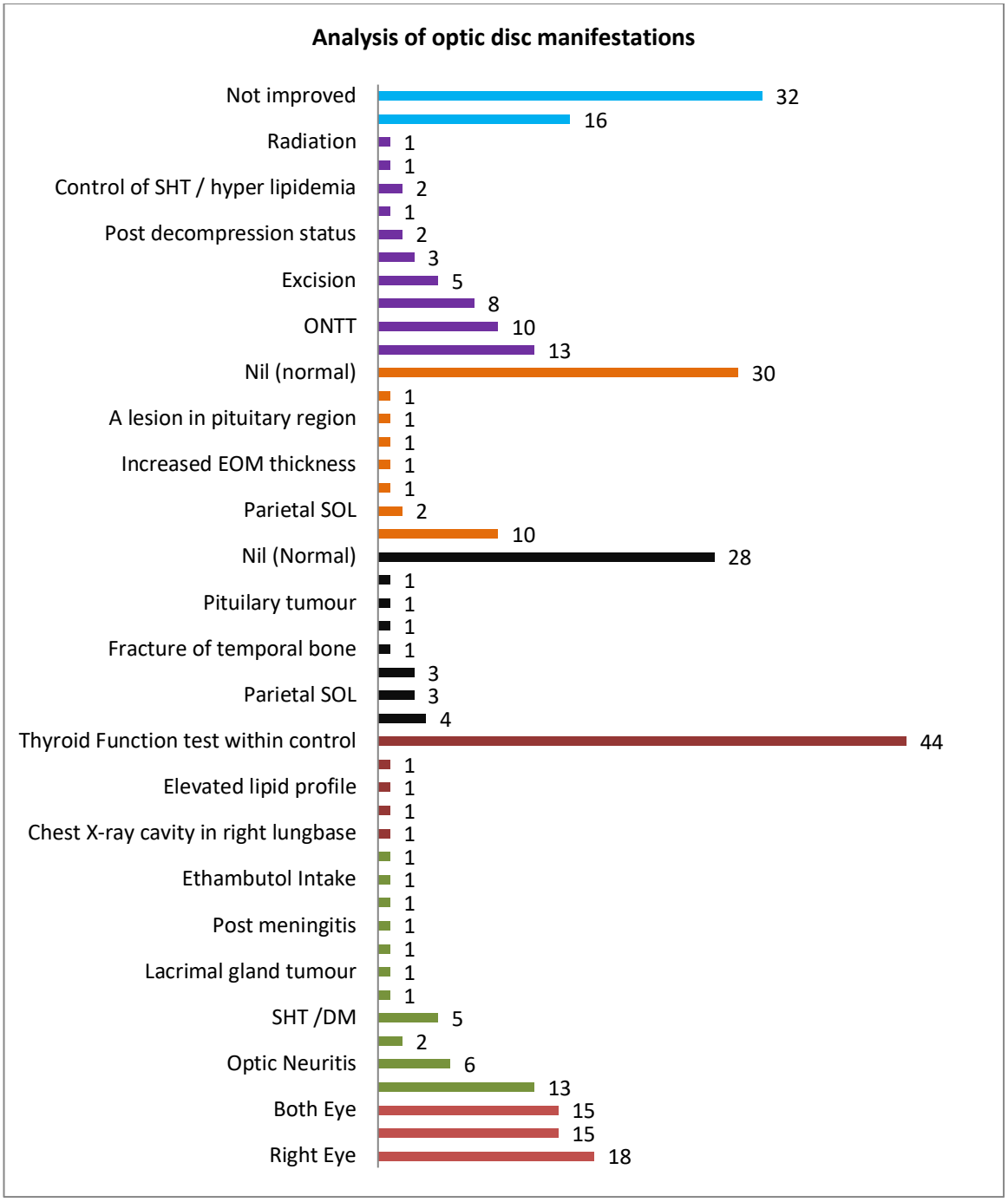

Figure 1: Analysis of optic disc manifestations

Table 2: Analysis of Disc pallor types

Details	Disc pallor	Frequency
Optic Neuritis	Segmental disc pallor	10 (20.8%)
Tumour	Diffuse disc pallor	10 (20.8%)
Primary optic atrophy	Total disc pallor	7 (14.5%)
Toxic	Temporal pallor	1 (2.08%)
Inflammatory Thyroid	Diffuse pallor	1 (2.08%)
Infectious tuberculosis	Diffuse pallor	1 (2.08%)
Post papilledema	Diffuse pallor	1 (2.08%)
Traumatic optic neuropathy	Temporal pallor	13 (27%)
Ethambutol induced	Temporal pallor	1 (2.08%)
Anterior Ischemic optic Neuropathy	Segmental pallor	1 (2.08%)

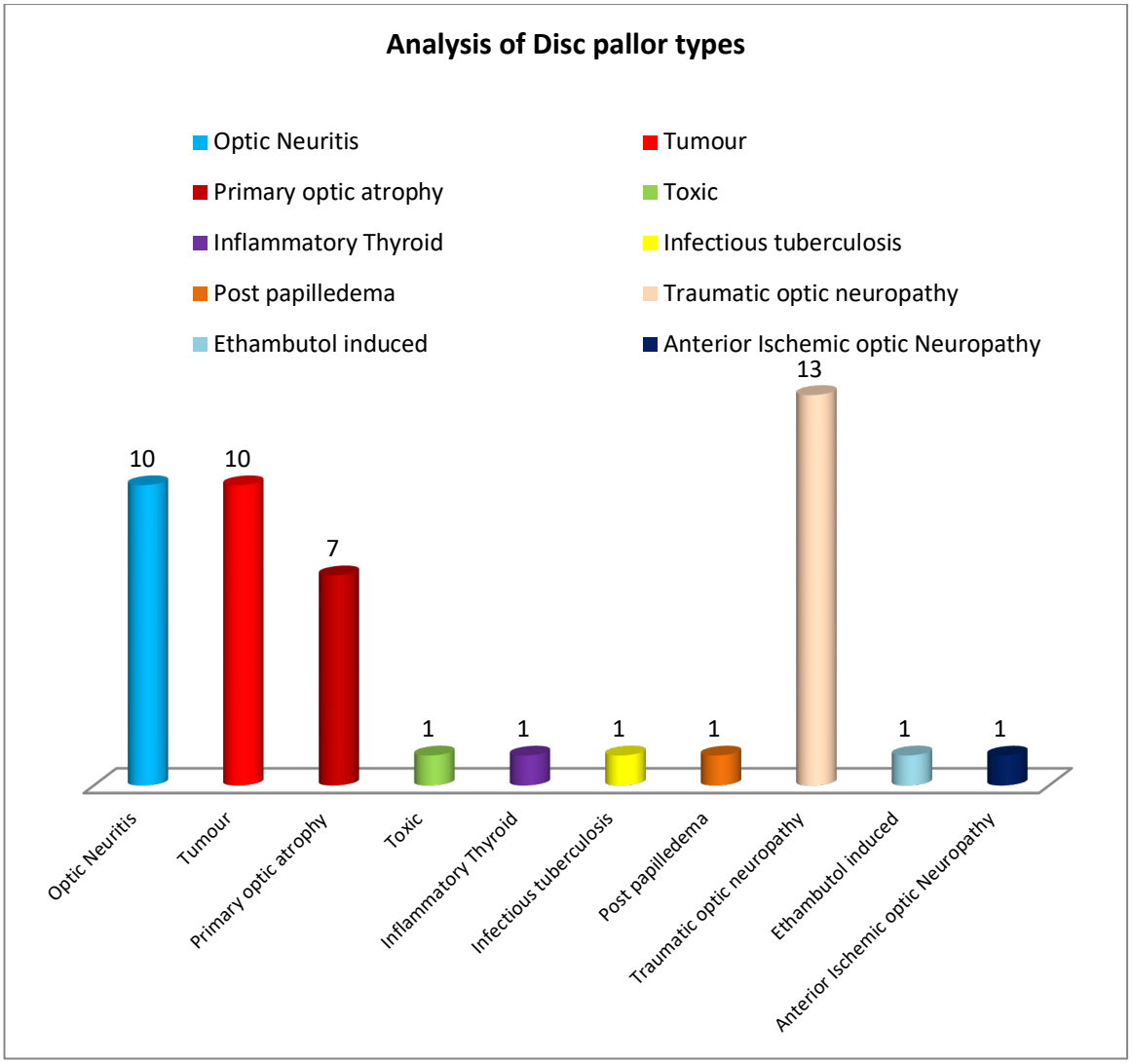

Figure 2: Analysis of Disc pallor types

Table 3: Descriptive study of visual acuity

			Frequency
Optic Neuritis	Initial BCVA	6/60	8 (16.6%)
		6/36	2 (4.16%)
	BCVA at 6 months	6/60	1 (2.08%)
		6/36	3 (6.25%)
		6/24	1 (2.08%)
		6/18	3 (6.25%)
		6/12	3 (6.25%)
Trauma	Initial BCVA	4/60	2 (4.16%)
		5/60	2 (4.16%)
		6/60	6 (12.5%)
		6/36	3 (6.25%)
	BCVA at 6 months	4/60	1 (2.08%)
		5/60	1 (2.08%)
		6/60	7 (14.5%)
		6/36	3 (6.25%)
		6/24	1 (2.08%)
Primary optic atrophy	Initial BCVA	2/60	2 (4.16%)
		3/60	1 (2.08%)
		4/60	1 (2.08%)
		5/60	2 (4.16%)

		6/60	2 (4.16%)
	BCVA at 6 months	2/60	2 (4.16%)
		3/60	1 (2.08%)
		4/60	1 (2.08%)
		5/60	2 (4.16%)
		6/60	2 (4.16%)
Tumour	Initial BCVA	2/60	2 (4.16%)
		4/60	1 (2.08%)
		6/60	3 (6.25%)
		3/36	2 (4.16%)
		6/18	2 (4.16%)
		6/12	1 (2.08%)
	BCVA at 6 months	2/60	2 (4.16%)
		6/60	2 (4.16%)
		6/36	2 (4.16%)
		6/18	3 (6.25%)
		6/12	2 (4.16%)

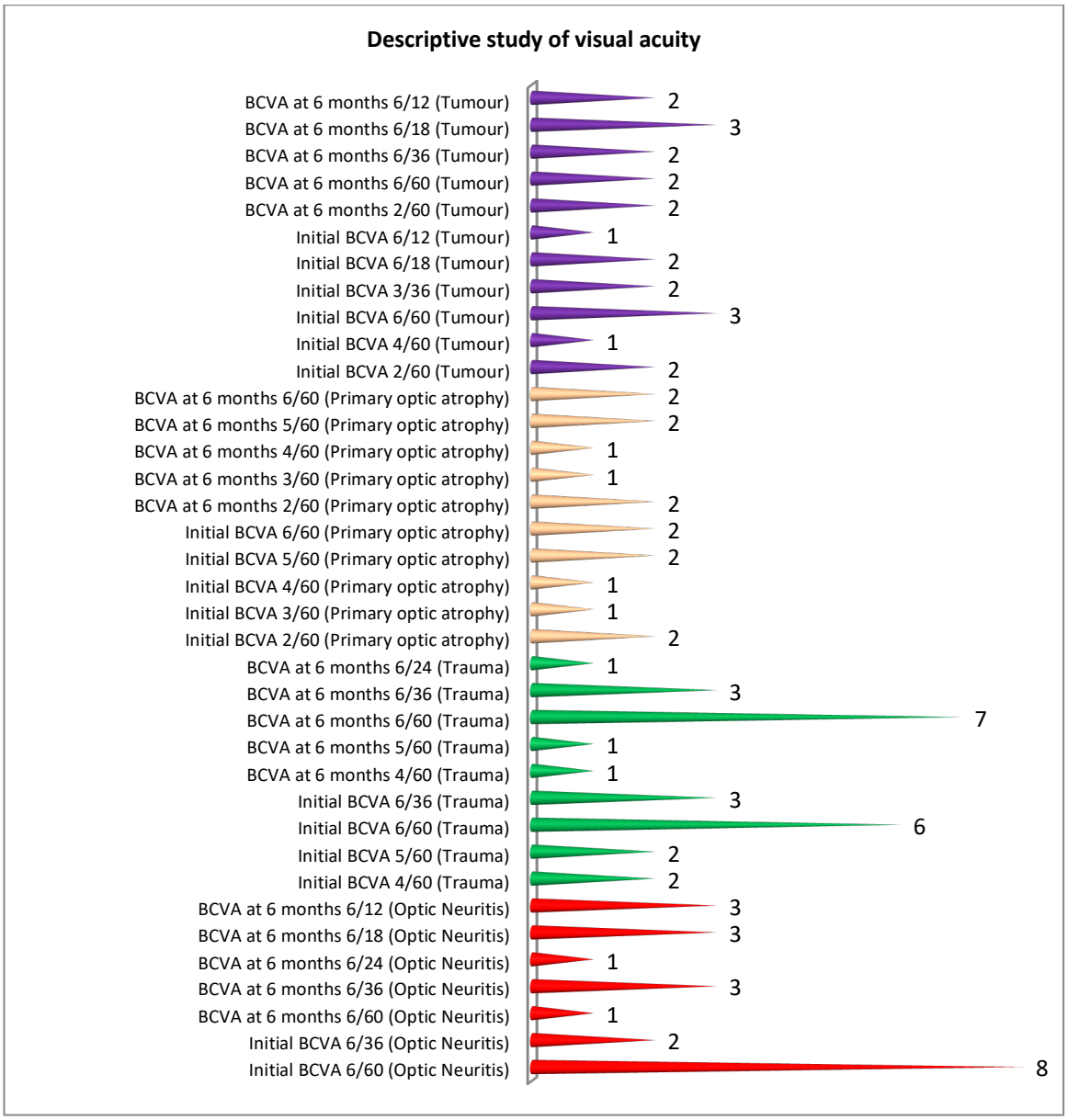

Figure 3: Descriptive study of visual acuity

Table 4: Analysis of visual acuity of other aetiologies

			Frequency
Toxic	6/36	Initial BCVA	1 (2.08%)
		BCVA at 6 months	1 (2.08%)
Inflammatory	6/60	Initial BCVA	1 (2.08%)
		BCVA at 6 months	1 (2.08%)
Infection TB	4/60	Initial BCVA	1 (2.08%)
		BCVA at 6 months	1 (2.08%)
Post papillodema	6/36	Initial BCVA	1 (2.08%)
		BCVA at 6 months	1 (2.08%)
Post AION	5/60	Initial BCVA	1 (2.08%)
		BCVA at 6 months	1 (2.08%)
Ethambutol induced	6/36	Initial BCVA	1 (2.08%)
		BCVA at 6 months	1 (2.08%)

AION: - Anterior Ischemic Optic Neuropathy

Discussion

Present study of pale optic disc and its correlation with visual out come in the analysis pale optic disc manifestations out of 48 cases 18 (37.5%) right eye, 15 (31.5%) left eye, 15 (31.5%) both eyes were involved. In the history of trouma 13 (27.1%) head injury, followed by 6 (12.5%) optic neuritis, 5 (10.4%) SHT/DM in radiological study, 4 (8.3%) had fracture of frontal bone followed by 3 (6.25%) parietal SOL, 3 (6.25%) fracture of optic canal. MRI study revealed 10 (20.8%) demyelinating plaques, BCVA outcome was 16 (33.3%) cases were improved, 32 (66.6%) not improved (Table-1). Types of disc pallor were segmental disc pallor had 10 (20.8%) optic neuritis, diffuse disc pallor had 10 (20.8%) tumour, toal disc pallor had 7 (14.5%) primary optic atrophy temporal pallor had 1 (2.08%) toxic and major temporal pallor had 13 (27%) had traumatic optic neuropathy (Table-2).

In the study of visual acuity optic neuritis had 6/60, 6/36 in 8 (16.6%) and 2 (4.16%) cases at initial BCVA, but after six month it was 6/12 In trauma cases, 4/60 to 6/36 BCVA but at 6 month 6/24 In primary optic atrophy initial BCVA was 2/60 to 6/60 but at 6 month 6/60, In tumour cases initial BCVA was 2/60 to 6/12 but at 6 month it was 6/12 (Table-3).

In the analysis of visual acuity 6/36 in toxic, 6/60 in inflammatory cases, 4/60 in infection TB, 6/36 papillodema 5/60 post AION, 6/36 in ehtambutol induced cases (Table-4). These findings are more or less in agreement with previous studies [5,6,7].

The clinical diagnosis is often helpful that is acute painful visual loss suggests optic neuritis. Sudden painless loss in an older patients suggest compressive or toxic causes and a history of Cranio – orbital trauma supports traumatic optic neuropathy. However overlapping presentation are frequent and pale disc alone rarely defines diagnosis. Hence systemic evaluation and neuro imaging are particularly important when compressive, demyelinating, infectious, inflammatory or post – traumatic cases are suspected [8] because it is clinically relevant as under lying cause has direct implications for both treatment and prognosis. Optic neuritis often shows favourable recovery while traumatic and established atrophic optic neuropathies have limited improvement [9]. Ehtambutol related or other toxic neuropathies require early withdrawal of offending agent, whereas compressive lesions need timely medical, surgical or radiopathic intervention [10].

The favourable pattern of optic neuritis is treated with corticosteroids therapy. It accelerates recovery in acute optic neuritis, although long-term final visual acuity is less dependent on steroid therapy. Primary optic atrophy showed no improvements, consistent with the limited reversibility of established axonal loss [11].

Summary and Conclusion

The pale optic disc has multiple causes. It is a heterogeneous group of optic neuropathies rather than single diagnosis. Traumatic optic neuropathy was the most conman etiology followed by optic neuritis tumour 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 most favourable recovery while optic atrophy remained unchanged. These findings supports early etiological identification and neuro imaging findings. Such studies must be conducted in large number of patients to validate present results and findings.

Limitation of study: Owing to remote location of study centre, small number of patients and lack of latest techniques we have limited findings and results.

This research work is approved by the ethical committee of Adichunchungiri institute of medical

sciences, Nagamangala, Bellur, Mandya, and Karnataka.

References

1. Liu A, Craver EC, Bhatti MT, Chen JJ. Population-Based Incidence and Outcomes of Compressive Optic Neuropathy. Am J Ophthalmol. 2022; 236:130-135.
2. Chuenkongkaew W, Samsen P, Thanasombatsakul N. Ethambutol and optic neuropathy. J Med Assoc Thai. 2003; 86(7):622-5.
3. Grzybowski A, Zülsdorff M, Wilhelm H, Tonagel F. Toxic optic neuropathies: an updated review. Acta Ophthalmol. 2015; 93(5):402-410.
4. Jung JJ, Baek SH, Kim US. Analysis of the causes of optic disc swelling. Korean J Ophthalmol. 2011; 25(1):33-36.
5. Biousse V, Newman NJ. Ischemic Optic Neuropathies. N Engl J Med. 2015; 372(25):2428-2436.
6. Bassi ST, George R, Sen S, Asokan R, Lingam V. Prevalence of the optic disc anomalies in the adult South Indian population. Br J Ophthalmol. 2019; 103(1):94-98.
7. Ambika S, Padmalakshmi K, Venkatraman V, Noronha OV. Visual Outcomes and Clinical Manifestations of Pediatric Optic Neuritis in Indian Population: An Institutional Study. J Neuroophthalmol. 2018; 38(4):462-465.
8. Mbekeani JN, Fattah MA, Poulsen DM, Hazzaa SA, Dababo MA, Eldali A, Ahmed M. Etiology of optic atrophy: a prospective observational study from Saudi Arabia. Ann Saudi Med. 2017; 37(3):232-239.
9. Singh D, Saxena R, Sharma P, Menon V. Systematic Approach to a Case of Disc Pallor. Delhi Journal of Ophthalmology. 2023; 21(3):28-32.
10. Osaguona VB. Differential diagnoses of the pale/white/atrophic disc. Community Eye Health. 2016; 29(96):71-74.
11. Naidu, Srinivas S. A clinical study of optic atrophy, its aetiology, pathogenesis, clinical manifestations, management and visual outcome. J. Evid. Based Med. Health. 2015; 2:83-87.