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Original Research Article

AN ETIOLOGICAL ANALYSIS OF PALE OPTIC DISC AND ITS CORRELATION WITH VISUAL OUTCOME IN PATIENTS ATTENDING A TERTIARY CARE HOSPITAL

P Sathya Priya[1], N.M. Tharani[2], P. Sumathi[3], V. Karthikeyan[4]

[1]Assistant Professor, Department of Ophthalmology, Government Medical College and Hospital, Nilgiris, Tamilnadu, India.
[2]Assistant Professor, Department of Ophthalmology, Government Medical College and Hospital, Nilgiris, Tamilnadu, India.
[3]Associate Professor, Department of Ophthalmology, Government Erode Medical College, Tamilnadu, India.
[4]Assistant Professor, Department of Ophthalmology, Government Medical College and ESI Hospital, Coimbatore, Tamilnadu, India.

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Corresponding Author:
Dr. V. Karthikeyan,
Email: drcartik@yahoo.in.

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Abstract

Background: Pale optic disc is mainly caused by damage to the optic nerve from the retinal ganglion cells to the lateral geniculate body. **Aim:** This study aimed to analyse the aetiology of pale optic disc and its visual outcomes. **Material and Methods:** This prospective observational study included 50 patients with non-glaucomatous pale optic discs at Coimbatore Medical College Hospital. Detailed history including the past medical history was recorded. A comprehensive ophthalmological examination, including visual acuity, colour vision, visual fields, slit-lamp biomicroscopy, ophthalmoscopic examination, fundus photography, and contrast sensitivity, was performed. Biochemical investigations and neuroimaging were ordered when indicated to identify aetiology, and patients were followed up for 6 months for visual outcomes. **Results:** The main aetiology of the pale optic disc in the study population was traumatic optic neuropathy. The majority of the study population included young adults, and the observation of traumatic optic neuropathy may be attributed to their age group. The improvement in visual outcome was less in the study population than in other similar studies. Best-corrected visual acuity of at least one line of improvement in Snellen's chart after 6 months was observed in only 34% of the study population which was contributed by 16% optic neuritis, 12% tumour, and 3% trauma aetiology. **Conclusion:** A definitive diagnosis of optic disc pallor which is a sign of an underlying disease, is very important. Identifying the actual cause of optic disc pallor will help in appropriate management.

INTRODUCTION

Death of retinal ganglion cell axons that comprise the optic nerve leads to optic atrophy, resulting in a pale optic nerve. The term optic atrophy describes a group of clinical conditions that have an abnormal pallor of the disc as a common physical sign. Optic atrophy is not a disease; it is the result of any pathological process that damages the retinal ganglion cells and axons of the reticulogeniculate pathway.[1] The axons of the retinal ganglion cells make up the optic nerve and continue onto the optic chiasm, optic tract, and up to the lateral geniculate body where they synapse. Injury to retinal ganglion cells and axons anywhere along their course from the retina to the lateral geniculate body may result in optic atrophy. Clinically, optic atrophy is associated with a decrease in visual acuity and visual field defects.2 There are numerous causes of optic nerve damage along the path from the retina to the lateral geniculate retina. The etiological factors like intracranial tumours, meningitis, optic neuritis, and toxic atrophy could lead to optical atrophy.[1]

Any insult occurring primarily in the anterior visual pathway results in optic atrophy through retinal ganglion cell loss. Posterior visual pathway involvement may also cause atrophy due to transsynaptic degeneration.[3] An ophthalmologist is frequently faced with optic disc pallor on fundoscopy and may be perplexed regarding how to approach the case and identify the aetiology behind this clinical presentation. Disc pallor is a manifestation of partial or total optic atrophy and a consequence of the loss of nerve fibres. Optic

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atrophy has been classically classified into primary and secondary types. Primary optic atrophy is secondary to a lesion affecting the visual pathway from the optic nerve head to the lateral geniculate body. In such cases, the disc is flat and pale with clearly demarcated margins. Disc edema precedes secondary optic atrophy which presents with a dirty white to grey looking disc with poorly delineated margins.[4]

The aetiology of unexplained disc pallor can be revealed by appropriate investigation in a large majority of cases. This was demonstrated in a multicentre study, in which only 8% of all cases of optic atrophy remained unexplained. Further direct investigations, including neuroimaging, led to an etiological diagnosis in another 20% of these cases. This study supports that neuroimaging can be prescribed for the diagnosis of all cases of unexplained optic atrophy.[5] The need for a definitive diagnosis in any case of disc pallor stems from the fact that optic nerve diseases behave in a varied manner while carrying out different treatments and outcomes. Some disorders, such as optic neuritis, are self-limiting but may be recurrent, whereas others, such as toxic neuropathies, are partially reversible. Hereditary optic atrophies may be progressive and with rare exceptions, do not show improvement. Ischaemic optic neuropathy, such as arteritic AION, can rapidly involve the fellow eye if not treated promptly. Damage to a nerve in toxic optic neuropathy can be halted by removing the offending agent.[4]

The diagnosis of optic disc pallor involves consideration of demographic factors such as age, sex, and race, although these must be interpreted cautiously. In children, the potential causes include hereditary optic neuropathies, nutritional deficiencies, CNS disorders, optic nerve glioma, and metabolic disorders. Adolescents may experience disc pallor due to conditions such as Leber's hereditary optic neuropathy, multiple sclerosis, or toxic optic neuropathy. Young adults may be affected by optic neuritis, multiple sclerosis, trauma, and systemic or central nervous system disorders. Older adults may experience ischaemic optic neuropathy, toxic neuropathy, nutritional deficiencies, and systemic or central nervous system disorders. Gender-specific causes include LHON, traumatic neuropathy, and tapetoretinal degeneration in males, while females may experience optic disc pallor due to multiple sclerosis, meningioma, autoimmune diseases, Sheehan syndrome, and eclampsia. It is important to note that there was no clear gender-based segregation of optic nerve afflictions. The type and severity of optic neuropathy may demonstrate ethnic variation. For example, blacks were found to have a lower incidence of ischemic optic neuropathy than whites and had a lower incidence of severe visual loss secondary to idiopathic intracranial hypertension than whites.[6]

Aim

This study aimed to analyse the aetiology of a pale optic disc and its correlation with visual outcomes over a 6-month follow-up period.

MATERIALS AND METHODS

This prospective observational study included 50 patients at the Department of Ophthalmology, Coimbatore Medical College Hospital, Coimbatore, between January 2018 and December 2018. The study was approved by the institutional ethics committee before initiation, and informed consent was obtained from all patients.

Inclusion Criteria

Patients aged > 20 years with a pale optic disc on fundus examination were included.

Exclusion Criteria

Unconscious patients terminally ill with known glaucoma with visual acuity of no light perception and pale optic disc due to retinal condition were excluded.

Data were collected using a structured questionnaire comprising sociodemographic characteristics such as age, sex, and detailed history. Biochemical investigations and neuroimaging are ordered when indicated to identify aetiology and clinical ocular examination is performed, and patients are followed up for 6 months for visual outcomes.

Clinical Examination included uncorrected and best-corrected visual acuity using Snellen’s chart, IOP measurement by NCT, refraction anterior segment examination, slit-lamp biomicroscopy 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 (in selected cases).

Statistical Analysis

The Shapiro-Wilk test was also conducted to assess the normal distribution. A Shapiro-Wilk test with a p-value >0.05 was considered as a normal distribution. Categorical outcomes were compared between the study groups using the chi-square test or Fisher's exact test (if the overall sample size was <20 or if the expected number in any one of the cells was <5, Fisher's exact test was used). Statistical significance was set at $p < 0.05$. IBM SPSS version 22 was used for the statistical analysis.

RESULTS

The mean age was 38.68 ± 12.09 in the study population, the minimum was 21, and the maximum was 66 years in the study population (95% CI 35.35 to 42.11). The mean duration in years was 5.4 ± 9.3 in the study population, with a minimum of 0.10 and a maximum of 35 years (95% CI 2.75 to 8.04). Among the study population, 28 (56%) were male and the remaining 22 (44%) were female. [Table 1]

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In the study population, 19 (38%) patients had the right eye, 16 (32%) had the left eye, and 15 (30%) had both eyes. Among the study population, 14 (28%) had traumatic head injury, seven (14%) had optic neuritis, two (4%) had CP angle tumours, six (12%) had SHT/DM, one (2%) had alcohol, one (2%) had lacrimal gland tumour, one (2%) had post-neurosurgery, one (2%) had post-TB meningitis, one (2%) had hyperlipidaemia, one (2%) had ethambutol intake, and one (2%) had thyroid orbitopathy.

Among the study population, 1 (2%) had chest X-ray: cavity in RT lung bose, 1 (2%) with hypertensive cardiomyopathy, 1 (2%) with increased lipid profile, 1 (2%) with sputum AFB positive, 1 (2%) with TFT within control, and 45 (90%) with normal. Among the study population, 5 (10%) had fractures in the frontal bone, 4 (8%) had parietal sol, 3 (6%) had fractures of the optic canal, 1 (2%) had fractures in the temporal bone, 1 (2%) had a lacrimal gland tumour involving the orbital apex, 1 (2%) had a pituitary tumour, and 1 (2%) had shunting.

Among the study population, 11 (22%) had demyelinating plaques, 2 (4%) had parietal SOL, 2 (4%) had shunting, 1 (2%) had increased EOM thickness, 1 (2%) had lacrimal gland tumour involving the orbital apex, 1 (2%) had a lesion in the pituitary region, and 1 (2%) had pineal astrocytoma. Among the study population, 14 (28%) were treated with post intravenous steroids, 11 (22%) with ONTT, 9 (18%) with observation, 6 (12%) with excision, 3 (6%) with post tumour excision, 2 (4%) with post-decompression status, 1 (2%) with control of alcohol, 2 (4%) with control of SHT/hyperlipidemia, 1 (2%) with observation and cessation of ATT, and 1 (2%) received radiation. Among the study population, 17 (34%) had improved best corrected visual acuity improved. [Table 2]

Regarding optic neuritis aetiology, 11(22%) patients had segmental disc pallor, 11(22%) had diffuse disc pallor, in primary optic atrophy 8 (16%) had total disc pallor, and in traumatic optic neuropathy 14 (28%) had temporal pallor. [Table 3]

Among patients with optic neuritis aetiology, eight (16%) had improved BCVA. Among the patients with tumour aetiology, 6 (12%) had improved BCVA. Among the traumatic optic neuropathies, three (6%) had improved BCVA. [Table 4]

Among the optic neuritis aetiologies, 9(18%) had an initial BCVA of 6/60, 1(2%) had a BCVA at 6 months as 6/60, 2 (4%) had an initial BCVA of 6/36, 3 (6%) had a BCVA at 6 months as 6/36, 1 (2%) had a BCVA at 6 months as 6/24, 3 (6%) had a BCVA at 6 months as 6/18 and 3 (6%) had a BCVA at 6 months as 6/12. Among the trauma patients, 2 (4%) had an initial BCVA of 4/60, 1 (2%) had a BCVA at 6 months as 4/60, 2 (4%) had an initial BCVA of 5/60, 1 (2%) had a BCVA at 6 months as 5/60, 7 (14%) had an initial BCVA of 6/60, 8 (16%) had a BCVA at 6 months as 6/60, 3 (6%) had an initial BCVA of 6/36, 3 (6%) had a BCVA at 6 months as 6/36 and 1 (2%) had a BCVA at 6 months as 6/24.

Among patients with primary optic atrophy, 2 (4%) had an initial BCVA of 2/60, 2 (4%) had a BCVA at 6 months as 2/60, 1 (2%) had an initial BCVA of 3/60, 1 (2%) had a BCVA at 6 months as 3/60, 1 (4%) had an initial BCVA of 4/60, 1 (2%) had a BCVA at 6 months as 4/60, 2 (4%) had an initial BCVA of 5/60, 2 (4%) had a BCVA at 6 months as 5/60, 2 (4%) had an initial BCVA of 6/60 and 2 (4%) had a BCVA at 6 months as 6/60. Among the tumours, 2 (4%) had an initial BCVA of 2/60, 2 (4%) had a BCVA at 6 months of 2/60, 1 (2%) had an initial BCVA of 4/60, 2 (4%) had a BCVA at 6 months of 6/60, 3 (6%) had an initial BCVA of 6/60, 2 (4%) had a BCVA at 6 months of 6/36, 2 (4%) had an initial BCVA of 6/36, 3 (6%) had a BCVA at 6 months of 6/18, 2 (4%) had an initial BCVA of 6/18, 3 (6%) had a BCVA at 6 months of 6/12, and 1 (2%) had an initial BCVA of 6/12. [Table 5]

Among the toxic aetiologies, 1 (2%) had an initial BCVA of 6/36 and 1 (2%) had a BCVA at 6 months at 6/36. Among the inflammatory thyroid nodules, 1 (2%) had an initial BCVA of 6/60, and 1 (2%) had a BCVA at 6 months (6/60). Among the TB infections, 1 (2%) had an initial BCVA of 4/60, and 1 (2%) had a BCVA at 6 months (4/60). Among the post-papilledema cases, 1 (2%) had an initial BCVA of 6/36 and 1 (2%) had BCVA at 6 months (6/36). Among the post-treatment patients, 1 (2%) had an initial BCVA of 5/60 and 1 (2%) had a BCVA at 6 months (5/60). Among the ethambutol-induced cases, 1 (2%) had an initial BCVA of 6/36 and 1 (2%) had BCVA at 6 months (6/36). [Table 6]

Table 1: Demographic data of the study

		Mean ± SD/ (%)	95% C. I	
			Lower	Upper
Age		38.68±12.09	35.25	42.11
Duration in years		5.4±9.3	2.75	8.04
Gender	Male	28(56%)	-	
	Female	22(44%)		

Table 2: Descriptive analysis of the eye, positive history, blood investigations, CT brain, MRI brain aetiology, treatment was given, and BCAV improved

		Frequency (%)
Eye	Right eye	19(38%)
	Left eye	16(32%)

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	Both eye	15(30%)
Positive history	Trauma head injury	14(28%)
	Optic neuritis	7(14%)
	CP angle tumour	2(4%)
	SHT/DM	6(12%)
	Alcoholic	1(2%)
	Lacrimal gland tumor	1(2%)
	Post neurosurgery	1(2%)
	Post tb meningitis	1(2%)
	Hyperlipidemia	1(2%)
	Ethambutol intake	1(2%)
	Thyroid orbitopathy	1(2%)
Blood investigations	Chest x-ray: cavity in the RT lung base	1(2%)
	Hypertensive cardiomyopathy	1(2%)
	Increased lipid profile	1(2%)
	Sputum AFB positive	1(2%)
	TFT within control	1(2%)
	Within normal	45(90%)
CT brain	Fracture of the frontal bone	5(10%)
	Parietal sol	4(8%)
	Fracture of the optic canal	3(6%)
	Fracture in the temporal bone	1(2%)
	Lacrimal gland tumour involving orbital apex	1(2%)
	Pituitary tumour	1(2%)
	Shunting	1(2%)
	Nil	29(58%)
MRI brain aetiology	Demyelinating plaques	11(22%)
	Parietal sol	2(4%)
	Shunting seen	1(2%)
	Increased EOM thickness	1(2%)
	Lacrimal gland tumour involving orbital apex	1(2%)
	A lesion in the pituitary region	1(2%)
	Pineal astrocytoma	1(2%)
	nil	31(62%)
Treatment given	Post i.v. steroids	14(28%)
	ONTT	11(22%)
	Observation	9(18%)
	Excision	6(12%)
	Post tumour excision	3(6%)
	Post decompression status	2(4%)
	Control of alcohol	1(2%)
	Control of SHT/hyperlipidemia	2(4%)
	Observation and stop of ATT	1(2%)
	Radiation	1(2%)
BCVA improved	Improved	17(34%)
	Not improved	33(66%)

Table 3: Descriptive analysis of disc pallor types based on aetiology

Aetiology	Disc pallor	Frequency (%)
Optic Neuritis	Segmental Disc Pallor	11(22%)
Tumour	Diffuse Disc Pallor	11(22%)
Primary Optic Atrophy	Total Disc Pallor	8(16%)
Toxic	Temporal Pallor	1(2%)
Inflammatory Thyroid	Diffuse Pallor	1(2%)
Infectious Tuberculosis	Diffuse Pallor	1(2%)
Post Papilledema	Diffuse Pallor	1(2%)
Traumatic Optic Neuropathy	Temporal Pallor	14(28%)
Ethambutol Induced	Temporal Pallor	1(2%)
Anterior Ischemic Optic Neuropathy	Segmental Pallor	1(2%)

Table 4: Descriptive analysis of visual acuity improved or not based on aetiology

Aetiology	BCVA improved	BCVA not improved
Optic neuritis	8(16%)	3(6%)
Tumour	6(12%)	5(10%)
Primary optic atrophy	-	8(16%)
Toxic	-	1(2%)
Inflammatory thyroid	-	1(2%)
Infectious tuberculosis	-	1(2%)
Post papilledema	-	1(2%)
Traumatic optic neuropathy	3(6%)	11(22%)
Ethambutol induced	-	1(2%)
Anterior ischemic optic neuropathy	-	1(2%)

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Table 5: Descriptive analysis of visual acuity of optic neuritis, trauma, primary optic atrophy, and tumour

			Frequency (%)
Optic neuritis	Initial BCVA	6/60	9(18%)
		6/36	2(2%)
	BCVA at 6 months	6/60	1(2%)
		6/36	3(6%)
		6/24	1(2%)
		6/18	3(%6)
		6/12	3(6%)
Trauma	Initial BCVA	4/60	2(4%)
		5/60	2(4%)
		6/60	7(14%)
		6/36	3(6%)
	BCVA at 6 months	4/60	1(2%)
		5/60	1(2%)
		6/60	8(18%)
		6/36	3(6%)
		6/24	1(2%)
Primary optic atrophy	Initial BCVA	2/60	2(4%)
		3/60	1(2%)
		4/60	1(4%)
		5/60	2(4%)
		6/60	2(4%)
	BCVA at 6 months	2/60	2(4%)
		3/60	1(2%)
		4/60	1(2%)
		5/60	2(4%)
		6/60	2(4%)
Tumour	Initial BCVA	2/60	2(4%)
		4/60	1(2%)
		6/60	3(6%)
		3/36	2(4%)
		6/18	2(4%)
		6/12	1(2%)
	BCVA at 6 months	2/60	2(4%)
		6/60	2(4%)
		6/36	2(4%)
		6/18	3(6%)
		6/12	2(4%)

Table 6: Descriptive analysis of visual acuity of other aetiologies (N= 50)

			Frequency (%)
Toxic	6/36	Initial BCVA	1(2%)
		BCVA at 6 months	1(2%)
Inflammatory thyroid	6/60	Initial BCVA	1(2%)
		BCVA at 6 months	1(2%)
Infection TB	4/60	Initial BCVA	1(2%)
		BCVA at 6 months	1(2%)
Post papilledema	6/36	Initial BCVA	1(2%)
		BCVA at 6 months	1(2%)
Post AION	5/60	Initial BCVA	1(2%)
		BCVA at 6 months	1(2%)
Ethambutol induced	6/36	Initial BCVA	1(2%)
		BCVA at 6 months	1(2%)

DISCUSSION

Detailed clinical evaluation is helpful for the differential diagnosis and management of optic atrophy. There are currently no specific treatments for optic atrophy. The underlying cause whether inflammatory, ischaemic, compressive, or metabolic should be treated if known.[2] Fifty subjects with a mean age of 38.68 ± 12.09. The age of subjects varied widely, ranging from 21 years to 66 years. This variation is useful for the study because age is considered an important demographic parameter, while short listing the possible aetiologies of disc pallor. Studies have established possible causes of disc pallor depending on presenting age.[4]

As the average age of the subjects in this study was above 35 years, the majority of subjects may belong to the middle age group. The most common aetiology observed in the study was traumatic optic neuropathy (28% of the subjects). This can be correlated with the observation mentioned in the study by Singh et al. that traumatic optic neuropathy is the most common aetiology of disc pallor in young adults.[4] of the study population, 28 (56%) were male and the remaining 22 (44%) were female. Among the study population, 28% had a past traumatic head injury, which can be related to

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traumatic optic neuropathy observed in 28% of the subjects. Among the study population, 22% of subjects were found to have tumour aetiology which is like that found in a study by Menon et al where tumour aetiology was found in 24.4% of patients.[7] However, this percentage was lesser than that found in the study by Mbekeani et al. in which 62.2% had tumor aetiology for optic atrophy.[8]

Among the study population, optic neuritis aetiology was found in 22% of patients. Demyelinating plaques on MRI can be correlated with the aetiology of optic neuritis. Among the patients with optic neuritis aetiology, 18% had an initial BCVA of 6/60, 2% had a BCVA at 6 months as 6/60, 4% had an initial BCVA of 6/36, 3 6% had a BCVA at 6 months as 6/36, 2% had a BCVA at 6 months as 6/24, 6% had a BCVA at 6 months as 6/18 and 6% had a BCVA at 6 months as 6/12. In a study by Wang et al. visual outcomes of acute optic neuritis in adult patients were investigated the results showed 72.7% had good visual recovery better than 20/40 which was much higher than that found in the present study where only vision improvement of 6/12 which equals 20/40 was noticed only in 6% of patients with optic neuritis etiology.[9]

The median age of the study population was 35 years, with a minimum of 21 years and a maximum of 66. Ambika et al. studied the visual outcomes and clinical manifestations of paediatric optic neuritis in the Indian Population and enrolled 42 (53.8%) females and 36 (46.2%) who were all younger than 18 years and found that 60 out of the 84 eyes (72.3%) recovered visual acuity of 20/40 or better, whereas in the current study with a median age of 35 (range, 21-66), improved visual outcome was only 28%. This implies that the success rate of visual outcomes was higher in the younger population than in the older population. The proportion of the male population is higher than females (28 males (56%) and 22 females [44%]) than that in the current study (42 women (53.8%) and 36 men [46.2%]).[10]

Kang et al. studied 35 patients aged 5–63 years. The aetiology in these patients was traumatic optic neuropathy. They found that the overall RNFL thickness decreased in six patients. In the current study, the study population with aetiology of traumatic optic neuropathy was 28% (14 out of 50 subjects). Only 6% had improved BCVA and 22% did not improve. Both the current study and the study performed by Kang et al. have similar findings that the improvement rate is low with traumatic optic neuropathy.[11]

The present study showed that the study population with a positive history of tumours such as CP angle tumour and lacrimal gland tumour (22%) had diffuse disc pallor, 12% had improved BCVA, and 10% had not improved BCVA. Optic atrophy is a potentially more serious clinical sign of an underlying condition. Among subjects with aetiology of primary optic atrophy, 16% had total disc pallor, and the visual outcome was poor in all subjects. BCVA did not improve in 16% of patients. Dewitt et al. conducted a detailed study on visual function in patients with optic nerve pallor (optic atrophy) and found good visual acuity in of 55/86 (64%) mild, of 54/119 (45.4%) moderate, and 21/65 of (32.3%) eyes with optic atrophy. As the graded severity of optic atrophy increased, the proportion of eyes with good visual function decreased.[12] Both this study and our present study have similar findings in that marked optic atrophy leads to poor visual outcomes.

CONCLUSION

Patients presenting with a pale optic disc with optic neuritis, tumour, or trauma aetiology showed a one-line improvement in visual acuity in the Snellen chart, whereas a pale optic disc with other aetiologies showed no improvement in visual acuity for 6 months. A pale optic disc may be a threat to a patient's vision, and identifying the aetiology can save the patient's vision and associated underlying systemic causes by timely intervention. Moreover, further optic nerve damage can be halted by identifying and treating the aetiology.

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