A Dissertation on

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

Dissertation submitted for M.S.Degree in Ophthalmology

May 2020

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

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DECLARATION

I hereby declare that this dissertation entitled "**AN ETIOLOGICAL ANALYSIS OF PALE OPTIC DISC AND ITS CORRELATION WITH VISUAL OUTCOME IN PATIENTS ATTENDING A TERTIARY CARE HOSPITAL**'' is a bonafide and genuine research work carried out by me under the guidance of **Dr.C JEEVAKALA M.S.,D.O** Assistant Professor, Department of Ophthalmology, Coimbatore Medical College & Hospital, Coimbatore.

This is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of regulations required for the M.S Ophthalmology, Branch III Degree Examination to be held in May 2020.

Date:

Place: **DR SATHYA PRIYA P**

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"AN ETIOLOGICAL ANALYSIS OF PALE OPTIC DISC AND ITS CORRELATION WITH VISUAL OUTCOME IN PATIENTS ATTENDING A TERTIARY CARE HOSPITAL"** is a bonafide and research work done by Dr SATHYA PRIYA P Post Graduate in M.S. Ophthalmology under my direct guidance and supervision to my satisfaction in partial fulfillment of the requirement for the degree of Master of Surgery in Ophthalmology, Branch III .

Date: **Guide,**

Department of ophthalmology

Date: **HOD & Professor,**

Department of Ophthalmology

Date: **Dean,**

Coimbatore Medical College & Hospital,

Coimbatore

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CERTIFICATE OF APPROVAL

To
Dr.Sathya Priya P
Post Graduate,
Department of Ophthalmology,
Coimbatore Medical College & Hospital,
Coimbatore -18.

Dear Dr.Sathya Priya P

The Institutional Ethics Committee of Coimbatore Medical College, reviewed and discussed your application for approval of the proposal entitled **"An Etiological Analysis of Pale Optic disc and its Correlation with Visual Outcome in Patients Attending a Tertiary Care Hospital."**No.044/2017.

The following members of Ethics Committee were present in the meeting held on 23.11.2017.conducted at MM - II Seminar Hall, Coimbatore Medical College Hospital Coimbatore-18.

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We approve the Proposal to be conducted in its presented form.

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The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

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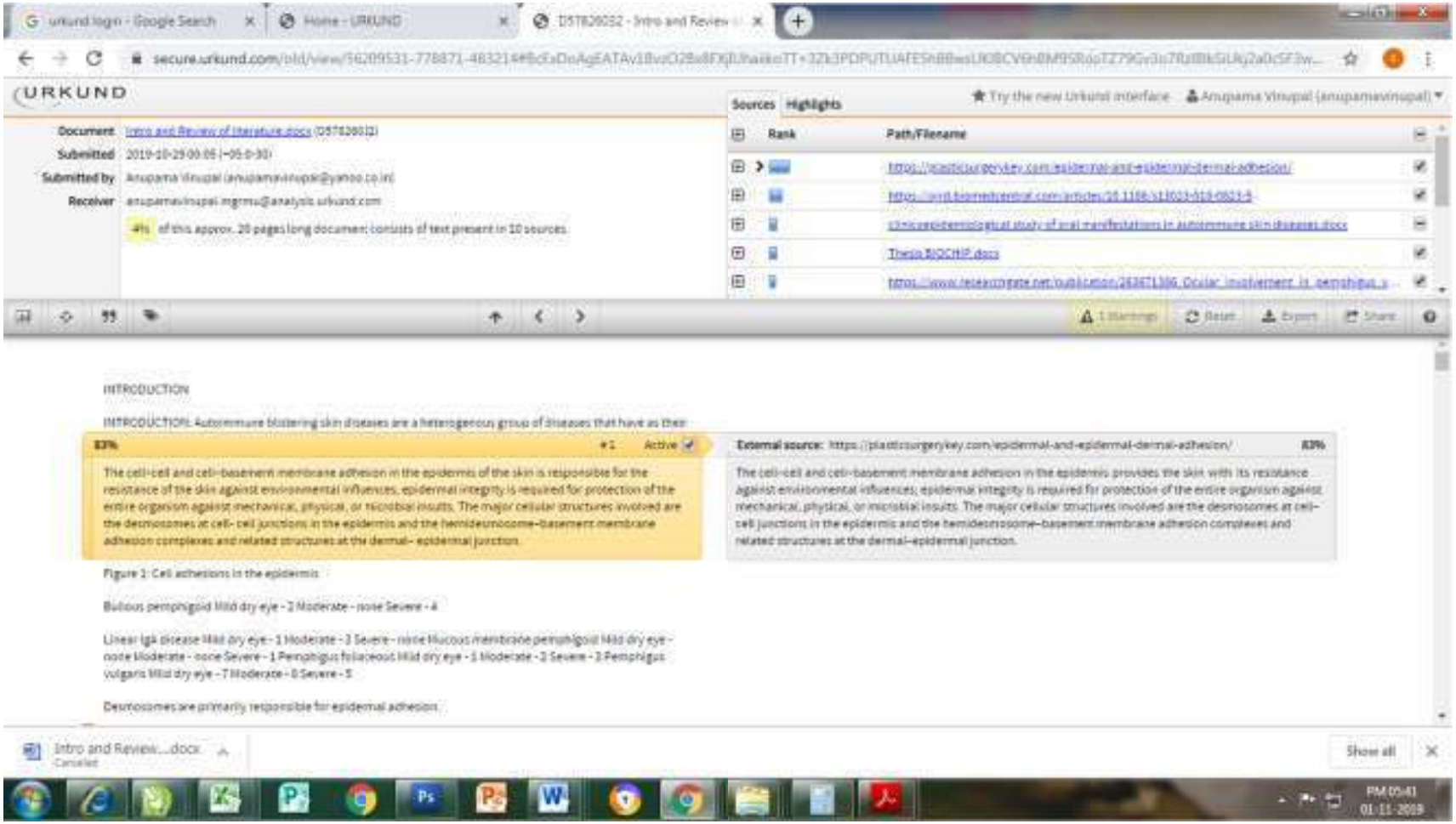
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INTRODUCTION
INTRODUCTION: Autoimmune blistering skin diseases are a heterogenous group of diseases that have as their
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Active
The cell–cell and cell–basement membrane adhesion in the epidermis of the skin is responsible for the resistance of the skin against environmental influences, epidermal integrity is required for protection of the entire organism against mechanical, physical, or microbial insults. The major cellular structures involved are the desmosomes at cell- cell junctions in the epidermis and the hemidesmosome-basement membrane adhesion complexes and related structures at the dermal– epidermal junction
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The cell–cell and cell–basement membrane adhesion in the epidermis provides the skin with its resistance against environmental influences; epidermal integrity is required for protection of the entire organism against mechanical, physical, or microbial insults. The major cellular structures involved are the desmosomes at cell–cell junctions in the epidermis and the hemidesmosome–basement membrane adhesion complexes and related structures at the dermal–epidermal junction.
Figure 1: Cell adhesions in the epidermis
Bullous pemphigoid Mild dry eye - 2 Moderate - none Severe - 4
Linear IgA disease Mild dry eye - 1 Moderate - 3 Severe - none Mucous membrane pemphigoid Mild dry eye - none Moderate - none Severe - 1 Pemphigus foliaceus Mild dry eye - 1 Moderate - 3 Severe - 3 Pemphigus vulgaris Mild dry eye - 7 Moderate - 0 Severe - 5
Desmosomes are primarily responsible for epidermal adhesion
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https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2594524/
https://www.ncbi.nlm.nih.gov/pubmed/19597111
https://www.researchgate.net/publication/51792070_Clinical_characteristics_of_optic_neuritis_in_Taiwanese_children
https://www.ncbi.nlm.nih.gov/pubmed/16908813
https://www.researchgate.net/publication/6878358_Detection_and_quantification_of_retinal_nerve_fiber_layer_thickness_in_optic_disc_edema_using_stratus_OCT
https://www.researchgate.net/publication/8185570_Image_analysis_of_optic_nerve_disease
https://www.ncbi.nlm.nih.gov/pubmed/8573019
https://www.cehjournal.org/article/differential-diagnoses-of-the-pale-white-atrophic-disc/
https://www.researchgate.net/publication/12355051_Use_of_sequential_Heidelberg_retina_tomograph_images_to_identify_changes_at_the_optic_disc_in_ocular_hypertensive_patients_at_risk_of_developing_glaucoma
https://newgradoptometry.com/optic-nerve-pallor-what-to-do/

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Date:

Place: Dr .SATHYA PRIYA P

ABBREVATIONS

AION	-	Anterior Ischemic Optic Neuropathy
AD	-	Autosomal Dominant
AR	-	Autosomal Recessive
XL	-	X Linked
CNS	-	Central Nervous System
ICT	-	Intracranial Hypertension
SSPE	-	Subacute Sclerosing Pan Enencephalitis
NAAION	–	Non Arteritic Anterior Ischemic Optic Neuropathy
AAION	-	Arteritic Anterior Ischemic Optic Neuropathy
PION	-	Posterior Ischemic Optic Neuropathy
LHON	-	Leber Hereditary Optic Neuropathy
ODP	-	Optic Disc Pallor
CRA	-	Central Retinal Artery
MNF	-	Myelinated Nerve Fibres
MCI	-	Multicolour Imaging
SD OCT	-	Spectral Domain Optical Coherence Tomography

ON	-	Optic Neuritis
BCVA	-	Best Corrected Visual Acuity
RAPD	-	Relative Afferent Pupillary Defect
CAR	-	Cancer Associated Retinopathy
RNFL	-	Retinal Nerve Fibre Layer
PVL	-	Periventricular Leucomalacia
PVR	-	Periventricular Haemorrhage
OHT	-	Ocular Hypertension
HRT	-	High resolution Tonography
RE	-	Right Eye
LE	-	Left Eye
BE	-	Both Eye
TB	-	Tuberculosis
ONTT	-	Optic Neuritis Treatment Trial
ON	-	Optic Neuritis
TO	-	Tumour
TON	-	Traumatic Optic Neuropathy

SOL	-	Space Occupying Lesion
I.V.	–	Intravenous
CP	-	CerebroPontine
SHT	-	Systemic Hypertension
DM	-	Diabetes Mellitus
CVA	-	Cerebro Vascular Accident
TFT	-	Thyroid Function Tests
AFB	-	Acid Fast Bacilli
RT	-	Right
CT	-	Computed tomography
MRI	-	Magnetic Resonance Imaging

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INTRODUCTION

The death of the retinal ganglion cell axons that comprise the optic nerve leads to optic atrophy and giving the resultant picture of a pale optic nerve. The term optic atrophy describes a group of clinical conditions which have an abnormal pallor of the disc as a common physical sign. Optic atrophy is not a disease; it is the end result any pathological process that damages the retinal ganglion cells and axons of 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 the 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 defect.[2] There are numerous causes of optic nerve damage anywhere along the path from the retina to the lateral geniculate. The etiological factors like intracranial tumors, meningitis, optic neuritis and toxic atrophy could lead to optical atrophy.[1]

Any insult occurring primarily to the anterior visual pathway results in optic atrophy through retinal ganglion cell loss. Posterior visual

pathway involvement may also cause atrophy due to transynaptic 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 etiology behind this clinical presentation. Disc pallor is the manifestation of partial or total optic atrophy and is a consequence of loss of nerve fibers. Optic atrophy has classically been described 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. The disc in such cases 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 etiology of unexplained disc pallor can be revealed by appropriate investigations in a large majority of cases. This was demonstrated in a multicenter study where of all cases of optic atrophy only 8% remained unexplained. Further directed investigations including neuroimaging led to an etiological diagnosis in another 20% of these cases. The study supports that the neuro imaging can be prescribed for the diagnosis of all the 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 very varied manner while carry different treatments and outcomes. Some disorders such as optic neuritis are self-limiting but may be recurrent whereas others like toxic neuropathies are partially reversible. Hereditary optic atrophies may be progressive and with rare exception, do not show improvement. An Ischemic optic neuropathy such as arteritic AION can rapidly involve the fellow eye if not treated on time. Damage to nerve in toxic optic neuropathy can be halted by removing the offending agent.[4]

The age, gender and race of a patient is often the first clue to the diagnosis, but has to be interpreted with caution.

The age is probably the most important of the demographic parameters while short listing possible etiologies of disc pallor. In children, causes for disc pallor could be hereditary optic neuropathies (AD/AR/XL), nutritional deficiency neuropathy, atrophy associated with CNS disorders, atypical optic neuritis, Schilder's disease, hypoxic ischemic syndrome (antenatal, perinatal and postnatal), optic nerve glioma, secondary to papilloedema (hydrocephalus, osteopetrosis) and metabolic disorders (methylglutaconic aciduria, ceroid lipofucinosis). In adolescents, the causes for disc pallor could be Leber's hereditary optic neuropathy, atypical optic neuritis, multiple sclerosis, neuromyelitis

optica, toxic optic neuropathy, pituitary adenoma, tepetoretinal degeneration and associated with systemic/CNS disorders. In young adults, the causes could be optic neuritis and multiple sclerosis, toxic optic neuropathy, traumatic neuropathy, meningioma associated with systemic disorders (neurosyphilis, tuberculosis, toxoplasmosis, diabetes mellitus, HIV) and associated with CNS disorders (raised ICT, spinocerebellar ataxia, Friedreich ataxia, encephalitis, encephalopathy, meningitis, SSPE, Gullian barre syndrome, etc). In older adults, etiology of optic disc pallor could be ischemic optic neuropathy (AAION, NAAION, PION), toxic optic neuropathy, nutritional deficiency, optic neuropathy, associated with systemic disease (hypertension, diabetes mellitus, paraneoplastic), associated with CNS disorders (ICSOL, neurodegenerative disorders) and central retinal artery obstruction.

One has to be aware that there is no clear segregation of optic nerve afflictions on gender. The causes of optic disc pallor in males could be LHON, Traumatic neuropathy, some tapeto-retinal degenerations, toxic neuropathy (lead, arsenic heavy metal occupational exposure, methyl alcohol), nutritional deficiency (chronic alcoholism). In females, reasons could be multiple sclerosis, meningioma, autoimmune/collagen vascular diseases, Sheehan syndrome, and ecclampsia.

The type of optic neuropathy and severity may demonstrate a ethnic variation. For example, blacks were found to have lower incidence of ischemic optic neuropathy than whites and had lower incidence of severe visual loss secondary to idiopathic intracranial hypertension than whites.[6] Caucasians are more likely to be afflicted with multiple sclerosis and optic neuritis than Asians or Hispanics. Overall optic atrophy is more prevalent in African-Americans (0.3%) than whites (0.05%).

NEED FOR THE STUDY

Disc pallor is the manifestation of partial or total optic atrophy and is a consequence of loss of nerve fibers. Optic atrophy is not a disease but a clinical sign, it refers to pallor of the optic disc which results from irreversible damage to fibers of the anterior visual pathway. The causes of optic atrophy are numerous, some of which may be life or sight threatening. A detailed clinical evaluation is helpful in the differential diagnosis and management of optic atrophy. There is no specific treatment for optic atrophy itself. The underlying cause whether inflammatory, ischaemic, compressive or metabolic should be treated if known.[2]

AIM OF THE STUDY

The aim of study is to analyse the etiology of pale optic disc and its correlation with visual outcome over a period of 6 months follow up .

OBJECTIVES OF THE STUDY

1. To study the etiological pattern of pale optic disc

2. To correlate the etiological cause of pale optic disc with visual outcome

REVIEW OF LITERATURE

Anatomy of optic disc

Chatziralli et al.[7] in 2019 investigated the coexistence of cilioretinal arteries (CRAs) with optic disc pit (ODP), and delineated the characteristics of CRAs related to their number, location of their emergence and their association with the size of ODP. 47 patients (49 eyes) with ODP were diagnosed and followed up between 1997 and 2017, using slit-lamp biomicroscopy, color fundus photographs, fluorescein angiography and indocyanine green angiography. The presence of CRAs was recorded in association with the size of the ODP, along with their number and location of emergence. The fellow normal eyes of patients were also analyzed. Results: 42 out of 49 eyes with ODP (85.7%) presented CRAs, in 35 out of 42 eyes (83.3%) CRAs emerged from the pit, either from bottom or from its margin. In 7.1% of cases, CRAs were emerged outside the ODP, while in 9.6% of cases, the type of CRA emergence could be characterized as mixed. The number of CRAs that ranged from 1 to 4, were positively associated with ODP size. In the fellow normal eyes, CRAs were found in 22.2% of cases, difference which was significant compared to patients with ODP. It is concluded that based on the high percentage of CRAs coexistence with ODP and the excessive frequency of their emergence from ODP (83.3%), it is

supported that ODP as a developmental disorder could go along with further anatomic peculiarities, that also include the presence of multiple CRAs.

Bhattacharya et al.[8] in 2019 did a study utilizing multicolor imaging (MCI) identifying optic disc anatomy in a case of myelinated nerve fibers (MNF) to the disc. MNF are characterized as whitish, relatively sharply demarcated, feather-like structures located in the retinal nerve fiber layer. MNF are located quite frequently in contiguity with the optic nerve head. This may lead to a diagnostic dilemma by preventing clear visualization of the optic disc margins. Here they described the utility of multicolor imaging (MCI) in identifying optic disc anatomy in a case of MNF contiguous to the disc. MCI and infrared reflectance were superior to color fundus photography in delineating disc margins. The pilot study describes the efficacy of MCI in delineating optic disc anatomy in a case of MNF.

Gopi et al.[9] in 2017 proposed an efficient method for optic disc segmentation and detection for the diagnosis of retinal diseases. The optic disc is the origin of the optic nerve, where the axons of retinal ganglion cells join together. The size, shape and contour of optic disc are used for classification and identification of retinal diseases. Automatic detection of eye disease requires development of an efficient algorithm.

The methodology involved optic disc localization, blood vessel inpainting and optic disc segmentation. Localization is based on principal component analysis, and segmentation is based on Markov random field segmentation.In order to get reasonable background images, blood vessel inpainting is done before segmentation.The proposed method tested with two standard databases MESSIDOR and DRIVE, and achieved an average overlapping score of 92.41, 92.17%, respectively; also validation experiments were done with one local database from Venu Eye Hospital, New Delhi, and obtained an average overlapping score of 91%. They concluded that an efficient algorithm is developed for detecting optic disc using principal component analysis-based localization and Markov random field segmentation. The comparison with alternative method yielded results that demonstrate the superiority of the proposed algorithm for optic disc detection.

Sedai, S. et al.[10] in 2016 did a segmentation of optic disc and optic cup in retinal fundus images using shape regression. Glaucoma is one of the leading cause of blindness. The manual examination of optic cup and disc is a standard procedure used for detecting glaucoma. This paper presents a fully automatic regression based method which accurately segments optic cup and disc in retinal colour fundus image. First, they roughly segment optic disc using circular hough transform. The

approximated optic disc is then used to compute the initial optic disc and cup shapes. They proposed a robust and efficient cascaded shape regression method which iteratively learns the final shape of the optic cup and disc from a given initial shape. Gradient boosted regression trees are employed to learn each regressor in the cascade. A novel data augmentation approach is proposed to improve the regressors performance by generating synthetic training data. The proposed optic cup and disc segmentation method is applied on an image set of 50 patients and demonstrated high segmentation accuracy for optic cup and disc with dice metric of 0.95 and 0.85 respectively. Comparative study showed that this proposed method outperforms state of the art optic cup and disc segmentation methods.

Reis et al.[11] in 2012 did a study to characterize optic nerve head (ONH) anatomy related to the clinical optic disc margin with spectral domain optical coherence tomography (SD-OCT).Design is cross-sectional study.Participants were open-angle glaucoma patients with focal, diffuse and sclerotic optic disc damage, and age-matched normal controls.Methods used were high-resolution radial SD-OCT B-scans centered on the ONH were analyzed at each clock hour. For each scan, the border tissue of Elschnig was classified for obliqueness (internally oblique, externally oblique, or non-oblique), and presence of Bruch's

membrane overhang over border tissue. Optic disc stereo-photographs were co-localized to SD-OCT data with customized software. The frequency with which the disc margin identified in stereo-photographs coincided with (1) Bruch's membrane opening, defined as the innermost edge of Bruch's membrane; (2) Bruch's membrane/border tissue, defined as any aspect of either, outside Bruch's membrane opening or border tissue; or (3) border tissue, defined as any aspect of border tissue alone, in the B-scans was computed at each clock hour. Main Outcome Measures—SD-OCT structures coinciding with the disc margin in stereophotographs.Results—There were 30 patients (10 with each type of disc damage) and 10 controls, with median (range) age 68.1 (42–86) and 63.5 (42–77) years respectively. Although 28 (93%) patients had 2 or more border tissue configurations, the most predominant one was internally oblique, primarily superiorly and nasally, frequently with Bruch's membrane overhang. Externally oblique border tissue was less frequent, observed mostly inferiorly and temporally.In controls, there was predominantly internally oblique configuration around the disc. While the configurations were not statistically different between patients and controls, they were among the 3 glaucoma groups. At most locations the SD-OCT structure most frequently identified as the disc margin was some aspect of Bruch's membrane and border tissue, outside Bruch's membrane opening. Bruch's membrane overhang was regionally present

in the majority of glaucoma patients and controls, however, in most cases not visible as the disc margin. It is concluded that the clinically perceived disc margin is most likely not the SD-OCT detected innermost edge of Bruch's membrane. These findings have important implications for the automated detection of the disc margin and estimates of the neuroretinal rim.

Mistlberger et al.[12] in 2002 did an assessment of optic disc anatomy and nerve fiber layer thickness in ocular hypertensive subjects with normal short-wavelength automated perimetry. The objective was to compare optic disc topography and nerve fiber layer thickness in ocular hypertensive eyes and normal subjects. Design was a prospective, case-controlled study. Participants and controls were one eye in each of 20 normal and 27 ocular hypertensive patients were enrolled. Consecutive normal and ocular hypertensive patients were enrolled. Each patient underwent complete ophthalmic examination, achromatic automated perimetry, short-wavelength automated perimetry, confocal scanning laser ophthalmoscopy, confocal scanning laser polarimetry, and optical coherence tomography. The intraocular pressure was 21 mmHg or less for normal subjects and at least 25 mmHg on two separate occasions in ocular hypertensive eyes. Structural parameters were compared between the two groups. Eyes with evidence of glaucomatous optic neuropathy,

achromatic visual field loss, or evidence of focal visual field injury during short-wavelength automated perimetry were excluded. Main outcome measures optic nerve head topography and nerve fiber layer thickness. Results, the three imaging technologies could not detect differences in optic disc or nerve fiber layer anatomy between the two groups. Ocular hypertensive eyes had a greater corrected pattern standard deviation than normal eyes during short-wavelength automated perimetry (P = 0.04). Conclusions, ocular hypertensive eyes with normal achromatic automated perimetry and short-wavelength automated perimetry could not be distinguished from normal subjects with confocal scanning laser ophthalmoscopy, confocal scanning laser polarimetry, and optical coherence tomography.

Krivoy et al.[13] in 1996 did imaging of congenital optic disc pits and associated maculopathy using optical coherence tomography. The objective was to elucidate the anatomy of congenital optic disc pits with and without maculopathy using optical coherence tomography.All patients were examined, photographed, and scanned at the New York Eye and Ear Infirmary.Ten eyes of eight consecutive patients with congenital optic disc pits were studied. Three eyes had associated serous macular detachment (group 1), four had evidence of resolved detachment (group 2), and three had no clinical macular pathologic lesion (group 3).

Methods, optical coherence tomography, a new, noninvasive, noncontact, imaging technology capable of producing cross-sectional images of the retina in vivo with high resolution (<17 μm) was used to obtain multiple cross-sectional images of the pit, peripapillary retina, and macula. Ophthalmologic examination and standard fundus photography were performed on all eyes. Fluorescein angiography was performed in eyes that had associated macular detachment.Results: Communication between a schisis cavity or subretinal space and the optic nerve pit was imaged in all eyes in group 1. No such communication could be identified in groups 2 and 3. Cystic degeneration and schisis were imaged in the peripapillary retina, macula, or both in all eyes of groups 1 and 2 and in one patient in group 3. A direct communication between the subretinal space and vitreous cavity could not be identified in any eye.Conclusions: Schisis formation plays an integral role in the development of serous retinal detachment in the presence of congenital optic disc pits. Findings are consistent with the theory that the optic disc pit acts as a conduit for fluid flow between the schisis cavity or subretinal space and the subarachnoid space.

Common diseases affecting optic disc

Chaddah M R et al [14] observed that Out of 100 cases *66* were males and *34* females. In both the sexes the incidence of the disease was

more in the first four decades of life . The disease was bilateral in 72 patients whereas *28* patients presented with uni lateral manifestations. In unilateral cases, right and left eyes were involved in an equal number, The disease manifested as primary optic atrophy in 48 patients, as secondary optic atrophy due to papilloedema in 15 cases and due to papillitis in *26* cases. 11 cases had consecutive optic atrophy.In *27* cases no cause could be detected. Of the established causes, meningitis topped the list involving *16* cases. Other common pathology detected was syphilis and intra-cranial space occupying lesions 10 cases each, demyelinat ing process 7 cases, trauma 7 cases, choroidal sclerosis 5 cases, pigmentary degeneration of retina 4 cases.

Jung et al.[15] in 2011 investigated the clinical manifestations and diagnoses of optic disc swelling. Methods used were the medical records of 49 patients who experienced optic disc swelling between March 2008 and June 2009 were retrospectively reviewed. The characteristics of non-arteritic anterior ischemic optic neuropathy (NA-AION) and optic neuritis (ON), which showed optic disc swelling most commonly, were compared. Results, NA-AION was the most common disorder (34.7%) that presented with optic disc swelling. ON was identified in 15 patients (30.6%). Seven out of 49 patients (14.3%) had intracranially associated diseases, such as papilledema and compressive optic neuropathy.

Pseudopapilledema was noted in four patients (8.2%). Other diseases (e.g., papillophlebitis, neuroretinitis, and diabetic papillopathy) were seen in six patients (12.2%). Ocular pain was observed more commonly in patients with ON ($p = 0.001$). Patients with ON expected a better visual prognosis than patients with NA-AION (0.12 ± 0.32 vs. 0.49 ± 0.35, $p = 0.001$). Conclusions, NA-AION and ON should be considered in the differential diagnosis when patients with optic disc swelling present to the neuro-ophthalmology clinic. Detailed history taking and supportive examinations, such as visual field, color-vision and imaging tests, should also be performed as indicated. Regular follow-up of such exams is necessary for the differential diagnosis of these diseases.

Eckert et al.[16] in 2007 did a study to determine the prevalence and features of the different types of involvement of the optic nerve in ocular toxoplasmosis. Methods used were retrospective cross-sectional study. All patients with active ocular toxoplasmosis, consulting in the Uveitis Section of the Ophthalmology Department were selected. The involvement of the optic nerve was classified in the following categories: Juxtapapillaryretinochoroiditis, pure papillitis, neuroretinitis, distant lesion, and mixed lesion. Results: The prevalence of involvement of the optic nerve found was 5.3%. The optic nerve involvement with the presence of a concurrent active distant lesion occurred in 22 eyes

(43.1%). A juxtapapillary lesion was found in 18 eyes (35.3%). Eight eyes (15.7%) presentedlesions characterised as mixed.Isolatedpapillitis occurred in 3 eyes (5.9%). Fortysevenlesions (95.9%) were unilateral and two(4.1%) were bilateral. Twenty-eight eyes(54.9%) had pre-existing lesions and 23 (45%)were primary lesions. Visual acuity improvedin 35 eyes (71.4%) and remained unchanged in14 eyes (28.5%).It is concluded that the involvement of the opticnerve most frequently found in ocular toxoplasmosis was optic nerve oedema with aconcurrent distant active lesion. The second type of lesion most often found was juxtapapillary retinochoroiditis. Involvement was monocular in most cases and the visualprognosis was favourable. Toxoplasma gondii may cause a lesion in the optic disc because of contiguousness;[17],[18] by direct involvement or even become involved when a retinochoroiditis lesion is located far from the optic nerve.[19]. Lesions had granulomatous character and marked necrosis in many instances led to a pathologic diagnosis of tuberculosis. Currently it is accepted that most cases of Jensen's choroiditis are of toxoplasmic aetiology. This type of lesion consists of a typical area of retinochoroiditis in contact with a swollen optic disc and accompanied by a typical sectorial deficit in the visual field. In pure papillitis the parasite affects the optic disc directly, causing a swollen papilla with sheathing of the peripapillary veins and there may be no concurrent active retinochoroiditis lesion.8 However, in the majority of

these cases a peripheral scar lesion and vitritis is always present over the optic disc.[20],[21] They have observed another form of involvement of the optic disc due to toxoplasmosis that would be caused secondarily by an active distant retinal lesion. In these cases, there is an active focal necrotising retinochoroiditis lesion located at variable distances from the optic disc that presents changes that resembles papillitis.[19]

Hayreh, et al.[22] in 2001 evaluated the appearance of the nerve head in patients after giant cell arteritis–induced arteritic anterior ischemic optic neuropathy (A-AION). Design used was noncomparative clinical case series. The study comprised 29 patients who presented with unilateral A-AION and temporal artery biopsy–proven giant cell arteritis. Stereoscopic optic disc photographs, taken of both the affected and unaffected eyes at the onset of the disease and after a follow-up period of 20.10 ± 25.36 months (median, 11 months; range, 2–102 months), were morphometrically evaluated. Main outcome measures size and shape of the optic disc, neuroretinal rim, optic cup, and alpha and beta zones of parapapillary atrophy. Results, in the eyes after A-AION, at the end of the study, the neuroretinal rim was significantly ($P = 0.002$) smaller, and the optic disc cup area was significantly ($P = 0.001$) larger than those of the contralateral unaffected eyes. Alpha zone and beta zone of parapapillary atrophy did not vary significantly ($P > 0.50$). Conclusions,

A-AION, like glaucomatous optic neuropathy, results in neuroretinal rim loss and optic disc cupping. However, in contrast to glaucoma, A-AION is not associated with an enlargement of parapapillary atrophy. The reasons and mechanisms responsible for these similarities and dissimilarities are discussed. Marked clinical, morphologic, and histopathologic similarities in optic disc cupping and loss of neuroretinal rim between A-AION and glaucomatous optic neuropathy are highly suggestive of a common mechanism for the development of the two diseases (i.e., ischemia of the optic nerve head).

Pale optic disc: definition

Optic disc swelling is a pathological condition with a variety of causes. The clinical features associated with unilateral optic disc swelling are demyelinating optic neuritis (ON), non-arteritic anterior ischemic optic neuropathy (NA-AION), compressive optic neuropathy, retinal-vein occlusion, and diabetic papillopathy. Cases with bilateral optic disc swelling are often associated with papilledema, infiltrative optic neuropathy, toxic optic neuropathy, and malignant hypertension[23].

Evaluation of a patient with optic disc, role of various diagnostic modalities in diagnosing the cause of pale disc. A brief summary of management of patients with pale optic disc.

The optic nerve head (a synonym for the optic disc) is a sort of keyhole through which one has a direct view of the cells that make up the afferent visual pathway – including its intracranial segment as far as the lateral geniculate body. There are numerous indirect, in part slit-lamp–supported, methods of fundoscopy that also give a stereoscopic view that improves the examiner's understanding of the in vivo anatomy of the optic disc. They provide an inverted virtual image of the living fundus. These methods, however, have not replaced the direct ophthalmoscope, which affords an erect image of the fundus that is 12 to 15 times magnified, allowing close inspection of the subtlest details. It requires mydriasis for its maximal benefit, but can be done at the bedside with no special requirements.

Ideally, direct ophthalmoscopy should include use of a red-free light (i.e., with the green filter included in the light path). This method enhances the view of detailed features such as arteriolar, venular, and even capillary structures and the retinal nerve fiber layer. Pathological signs such as hemorrhages, traumatic folds, defects in the retinal nerve fiber layer, and the depth and location of optic atrophy are easily seen.

Photographic documentation of the findings (also including stereoscopic pairs and/or color filters, where applicable) is particularly useful for closer inspection of uncertain findings or for the monitoring of the patient's clinical course.

The following criteria should be included in the documentation of the optic disc appearance: The color of the optic disc, the sharpness of the optic disc margin, any elevation of the optic disc, the margin of the neuroretinal rim, cupping, the apparent size of the disc, the vessels on and near the disc, the peripapillary region, any other distinctive features.

Color of the Optic Disc: There is no single feature that determines a healthy optic disc appearance. A comparison to the contralateral disc and of the various segments within the disc is helpful. When judging the color of the optic disc, a number of considerations should play a role, including ametropia, papillary size, and hair and skin color. Fair-skinned, blond, myopic people with large optic discs have a lighter color to their discs, and vice versa.

Retrogeniculate damage in the human is not manifested by any change in fundus appearance; the only exception to this rule is in infants and very young children, in whom damage to the retrogeniculate pathway can produce a trans-synaptic degeneration with resultant disc pallor. The capillary bed of the surface of the optic disc is especially easy to see in

red-free light, which causes the tiny vessels to contrast more sharply with the surrounding tissue elements.

Sharpness of the Optic Disc Margin: The nasal margin of the optic disc is commonly blurred or difficult to see. In addition, the disc margin at the superior and inferior poles is commonly obscured in part by the thick layer of healthy nerve fiber bundles arriving from the temporal retina and crowding into the narrow space at the vertical extremes of the disc margin. Only the temporal quadrant of the disc margin should always be expected to appear sharp. It is also generally true that small optic discs and/or the discs of strongly hyperopic eyes are usually not sharp. This is again due to the crowding of many healthy nerve fiber bundles into a tiny space. If the color is healthy, surface striations are visible, and there are no signs of hemorrhage, exudate formation or edema, blurred vision, or invisible disc margins need not be a cause for concern.

Optic Disc Elevation: An estimate of the prominence of the optic disc is easily made from the appearance in a stereo pair of photographs – quantification is possible with the direct ophthalmoscope with its high magnification and comparative shallow depth of field. If one at first focuses on the center of the macula and then on the papillary surface, the difference can be expressed in the dioptric power shift necessary to achieve focus. This is a rather accurate comparison of the relative focal

distances to the macula and the optic disc surface. The reverse approach offers the risk of error, especially for younger observers with their ample range of accommodation. It is all too easy to exaggerate the difference when dialing in an increase in minus power to focus on the macula. Starting at the macula and adding positive dioptric power to focus on the disc minimizes the chance for error.

Pearl Rule of thumb: For any eye that is emmetropic, or nearly so, 1 mm of elevation corresponds to +3 diopters of change in focal length. It should be noted whether the papillary elevation is segmental (as is seen in acute anterior ischemic optic neuropathy) or uniform in distribution. Optic Disc Cupping and the Neuroretinal Rim Conventional ophthalmoscopy allows only a qualitative estimate of the surface of the neuroretinal rim. The vertical cup-to-disc ratio (the ratio of the cup size to the diameter of the optic disc) allows only a semiquantitative assessment of the degree of cupping. Generally, this measure is usually higher in eyes with large optic discs, which are more likely to have a physiologic form of cupping. It has been noted that this is particularly common in African-Americans, who with larger optic discs and large physiological cups, are at greater risk for visual loss to primary open angle glaucoma than do those with small cups and small optic discs. Small discs with small or no cups are also at risk, but can withstand

higher pressures for longer periods without suffering glaucomatous loss of vision. Changes in cupping are indicative of damage, and even small increases in the cupping of small discs are cause for concern. Physiologic cupping is more likely to be horizontally oval and deeper in the nasal quadrant than in the temporal quadrant. More important than the size of the cup – which should be proportional to the size of the disc – is the appearance of the neuroretinal rim. Is it healthy in color? Are there notches in the rim, i.e., narrow zones in which the nerve fiber layer at the disc margin disappears? Locally circumscribed cupping, i.e., notches in the inner margin of the neuroretinal rim, are closely associated with glaucomatous optic nerve damage.[24]

A complete and thorough ophthalmic examination is mandatory. The neuro-ophthalmology specific ocular examination includes:

Visual acuity: Typically disc pallor secondary to optic neuritis, LHON, Nutritional deficiency optic neuropathy, NAAION and inflammatory neuropathies presents with a visual acuity of around 20/200. Hereditary optic neuropathies (AD/AR), AAION, post papilledema and traumatic optic neuropathies on the other hand present with a poor visual acuity and even absence of light perception. Toxic optic neuropathies have a variable and unpredictable visual acuity.

Visual fields: Confrontation visual fields can give a quick assessment of any large scotomas or hemi field or altitudinal defects. A carefully done confrontation field test and a tangent screen test with a red target provide a fairly accurate result.

Color desaturation test: The patient may be shown a red capped bottle and asked to compare the red color separately with both eyes. The eye with disc pallor would give a washed out red or pink appearance in contrast to the bright red color seen by the fellow eye.

Pupils: A relative afferent papillary defect is characteristically found in cases of disc pallor though it is absent in bilateral symmetrically affected cases. RAPD can be quantitatively assessed using neutral density filters for comparison of any future worsening of neuropathy.

Macula/Posterior pole: One needs to look for presence of exudates in the form of a star or fan or sequel thereof. Occasionally an optic disc pit may be associated with central serous choroidoretinopathy or a choroidal neovascular membrane.

Disc Size This may be measured by various techniques using a direct ophthalmoscope (use 50 cone of Welch Allen ophthalmoscope), indirect ophthalmoscope or on slit lamp biomicroscopy (When using a 90D lens multiply the height of the slit measured in mm by 1.3 when it is focused

and just equal to the disc to get disc size in mm). The importance of disc size comes when a case of optic disc hypoplasia is confused with a disc pallor post neuropathy.

Shape: The disc normally appears round or oval. Any variation from this should alert towards a congenital anomaly or traumatic avulsion.

Color: The disc is normally salmon pink in color though the actual color varies from race to race. A disc is described as pale if it loses the pink hue to turn towards a whitish yellow color or a lemon tint. It is described as hyperemic if it becomes reddish pink (a sign of increased vascularity). There are methods described in literature to objectively or subjectively assess and document the color of the disc. These involve recording ophthalmoscopic appearances and digital stereoscopic disc images followed by analyzing them. One should establish their own protocol and document progression of disc pallor during follow up visits. A word of precaution which merits mention is that the 90D lens may make the disc look falsely healthier(less pale) due to its the yellow tint.

Cup/Disc Ratio: This shows great variability and can range from no visible cupping to 0.6 and beyond in the normal subjects. A deep excavated cup is of more significance compared to a large shallow cup. The size of a cup can also be measured similar to that described for the

disc above. Both direct and indirect ophthalmoscopy underestimates the cupping in comparison to slit lamp biomicroscopy.

Neuroretinal rim: This is a congregation of nerve fibers from the retina converging upon the disc to form the optic nerve. A pale disc secondary to a neuro-ophthalmic disease often has a uniform thin neuroretinal rim but there is no focal notching or loss in contrast to glaucoma.

Disc Margins: These should be well defined. In disc edema they are usually blurred in the INST sequence. Often one may notice a pallid disc edema in circulatory compromise of the disc. LHON may present with pseudoedema.

Disc Vascularity: Kestenbaum count is the number of capillaries observed on the disc. The normal number is 10 while optic atrophy will have a count of less than 6.

Peripapilla: Presence of peripapillary atrophy (alpha zone and beta zone) needs to be documented.

Retinal nerve fibre layer: The presence of any RNFL defects should be noted. This is best examined with a red free green filter.[4]

The appearance of the disc can give a clue about the possible etiology.[25]

MOST RELEVANT GLOBAL AND INDIAN STUDIES

How et al[26] in 2009 did a study to determine the prevalence of tilted and torted optic discs and associated risk factors among Chinese adults in Singapore. The methods used, as part of a population-based survey, optic disc stereo photographs of both eyes were obtained, and left eyes were analyzed using imaging software. A tilted optic disc was defined as an index of tilt (ratio of minimum to maximum optic disc diameter) less than 0.75.The angle of tilt was defined as the angle between the maximum and vertical optic disc diameter, and optic discs were graded as torted if the angle of tilt exceeded 15°. Results, 26 of 739 subjects (3.5%) had tilted optic discs, and 478 (64.7%) had torted optic discs. Myopia was present in 23 of 26 eyes (88.5% [95% confidence interval, 69.9%-97.6%]) with tilted optic discs and in 211 of 661 eyes (31.9% [28.4%-35.6%]) without tilted optic discs (*P*_.001). On multivariate analysis, myopia(spherical equivalent) was a significant risk factor for tilted optic discs (*P*_.001). Index of tilt was not associated with corneal astigmatism or with cylindrical refractive error. Seventeen eyes (65.4%) with tilted optic discs had an optic disc morphologic abnormality, but none were glaucomatous. It is concluded that the prevalence of tilted optic discs among this Chinese population was 3.5%. Tilted optic discs were associated with myopia but not with glaucoma.

Naidu, A. et al[1] One hundred clinically established cases of optic atrophy who attended to the department of ophthalmology, government general hospital, Kakinada during 2008- 09 were taken into the study. Authors have determined the etiology of optic atrophy and to study its clinical manifestations and visual prognosis. Age and Gender distribution of optic atrophy–the gender distribution of the cases shows a male preponderance with 57% of cases belonging to male gender, Optic atrophy and visual acuity – Out of 180 eyes of 100 patients included in their study, 66 eyes have no perception of light and 65 eyes have visual acuity of <CF 1mt. belong to pressure and traction atrophy of which glaucoma is the major cause, next most common causes being intracranial tumors, papilledema and basal arachnoiditis. Optic atrophy and visual field defects–25 eyes having visual acuity of 6/60 or better are subjected for visual field examination. In 10 eyes with glaucoma, the following field defects were noted: arcuate scotomas in 6 eyes, tubular vision in 2 eyes, concentric constriction in 1 eye and 1 patient was not cooperative. Out of 9 eyes of RP having visual acuity 6/60 or better, 5 eyes have peripheral constriction of fields and in 2 eyes the visual fields were not recorded as the patients were not cooperative. Centrocaecal scotoma was found in 5 eyes in patients of toxic atrophy, metabolic atrophy and in multiple sclerosis. Concentric constriction of visual fields was noted in a patient with papilledema. Study findings have conclude that Early

diagnosis and treatment of the etiological factors like intracranial tumors, meningitis, optic neuritis and toxic atrophy can prevent or limit visual loss from optic atrophy.

Chinta, S., et al. [27] have analyzed the clinical features and etiology of diagnosed cases of optic nerve atrophy in children <16 years of age. A total of 324 children (583 eyes) were identified. Among these 160 (49%) presented with defective vision, 71 (22%) with strabismus, 18 (6%) with only nystagmus. Rest had a combination of two or three of the above symptoms. Sixty‐five patients (20%) had a unilateral affection. Hypoxic ischemic encephalopathy seen in 133 patients (41%) was the most frequent cause of childhood optic atrophy, followed by idiopathic in 98 (30%), hydrocephalus in 24 (7%), compressive etiology in 18 (5%), infective in 19 (6%), congenital in 6 (2%), inflammatory in 5 (2%) patients, respectively. Hypoxic ischemic encephalopathy appears to be the most common cause of optic atrophy in children in this series. The most common presenting complaint was defective vision.

Jacobson et al.[28] evaluated the relation between optic disc morphology and timing of periventricular white matter damage, defined as either periventricular leucomalacia (PVL) or periventricular haemorrhage (PVH). 35 children with periventricular white matter damage who had neuroradiology performed and ocular fundus

photographs were compared with a control group of 100 healthy full term children. Timing of brain lesion was estimated by analysis of the brain lesion pattern on neuroradiological examinations. 4 of 35 children had a small optic disc area; these four children had a brain lesion estimated to have occurred before 28 weeks of gestation. Nine of 11 children with a large cup area had a PVL/PVH estimated to have occurred after 28 weeks of gestation. The children with PVL/PVH had a significantly larger cup area (median 0.75 mm2) than the control group (median 0.33 mm2) (p=0.001) and a significantly smaller neuroretinal rim area (median 1.58 mm2) than the controls (median 2.07 mm2) (p=0.001). They concluded that in a child with PVL/PVH and abnormal optic disc morphology, the possibilities of timing of the lesion should be considered.

Kamal et al.[29] determined if global and segmental changes in optic disc parameters of sequential Heidelberg retina tomograph (HRT) images developed in individual ocular hypertensive (OHT) patients without white on white visual field defects. The subject groups consisted of 21 OHT patients who had converted to early glaucoma on the basis of visual field criteria (24-2 program on the Humphrey perimeter), 164 OHT subjects with normal visual fields, and 21 normal controls. Sequential HRT images 16–21 months apart were obtained for each subject and segmental optic disc parameters were measured to determine if any

change had occurred. Individual discs in each group showing changes above the 95% limit of normal variability were then sought. Several segmental and global optic disc parameters were found to show significant change in the converter group before confirmed visual field change. Individual optic disc analysis demonstrated glaucomatous change in 13 out of 21 converter eyes. 47 of the 164 OHT eyes with normal visual fields showed change in global and segmental parameters in a "glaucomatous" direction above the level expected for normal variability. The parameters which changed most frequently in the OHT eyes were: global cup volume (6.7% of discs), inferonasal cup volume (11%), inferotemporal cup volume (8.5%), and superotemporal cup area (7.3%). They concluded that the HRT could be of value in the sequential followup of those suspected of having glaucoma by identifying eyes at risk of developing glaucoma.

Quigley et al.[30] did a study to obtain quantitative measurements of capillary numbers, areas, and diameters in atrophic (pale) and normal primate optic nerve heads. The number of capillaries per square millimeter in pale optic disks was not significantly different from that in normal optic disks. Because the loss of all nerve fibers leads to a 50% decrease in nerve head substance, capillaries must atrophy to maintain a constant relationship between capillary number and tissue volume. The

mean size of individual capillaries in atrophic nerve heads was smaller than normal, leading to a decrease of more than 27% in the percentage of tissue volume occupied by capillaries. When this decrease in capillary volume was mimicked in the normal optic disk by reducing the hematocrit value, optic disk pallor did not result. Hence, the development of optic disk pallor appears to be the result of thinning of the neural tissue of the rim of the optic disk and the consequent change in tissue composition and optical transparency, rather than of a loss of optic disk capillaries.

Savini et al.[31] investigated the ability of optical coherence tomography (OCT) to assess changes in retinal nerve fiber layer (RNFL) thickness in optic disc edema. Prospective observational case series were done in a private eye clinic (Centro Salus). Twelve consecutive eyes (9 patients) with optic disc edema were analyzed, including 6 patients with anterior ischemic optic neuropathy, 1 patient with multiple sclerosis–associated papillitis, and 2 patients with bilateral papilledema. Repeated measurements were performed with follow-up ranging from 8 to 30 weeks. Optical coherence tomography detected and quantified diffuse thickening of the RNFL. Compared with eyes in a control group of 75 healthy subjects, eyes with optic disc edema showed a significant increase in the mean RNFL thickness in all quadrants (temporal, *P*=.002;

superior, *P*_.001; nasal, *P*_.001; and inferior, *P*_.001). In patients who were followed up, progressive thinning was observed as the disease evolved toward optic atrophy or clinical resolution. They concluded that the optical coherence tomography can identify and measure RNFL edema. This ability of OCT may help elucidate pathophysiological mechanisms in optic disc edema and provide a valuable aid to clinicians.

Ambika et al.[32] did an institutional study on visual outcomes and clinical manifestations of pediatric optic neuritis in Indian Population. They reviewed the medical case records of patients with optic neuritis who were younger than 18 years, from 1999 to 2016. 117 eyes of 78 children with mean age of 11.84 (±4.58) years were identified. Forty-two (53.8%) were females and 36 (46.2%) were males. Thirty-nine patients (50%) had bilateral involvement and a similar number had unilateral involvement. Fifty-nine eyes (50.4%) had optic disc edema, 20 eyes (17.1%) had disc pallor, and 38 eyes (32.4%) had normal discs. Of 63 patients who had neuroimaging, 36 had MRI, and 27 underwent computed tomography. Eighty-four eyes (of 59 patients) received steroid therapy according to the protocol of the Optic Neuritis Treatment Trial (ONTT). Sixty of the 84 eyes (72.3%) recovered visual acuity of 20/40 or better. Visual acuity improvement was statistically significant between initial and final visual acuity (logMAR) in our patients treated with the ONTT protocol ($P \leq 0.001$).

They concluded that Indian pediatric population had good visual recovery after steroid treatment for optic neuritis. Profound loss of visual acuity on presentation and bilateral involvement were significantly associated with poor visual outcome.

Kang et al.[33] evaluated optic disc pallor using ImageJ in traumatic optic neuropathy (TON). This study examined unilateral TON patients. The optic disc was divided into 4 quadrants (temporal, superior, nasal, and inferior), consistent with the quadrants on optical coherence tomography (OCT) retinal nerve fiber layer (RNFL) thickness maps. The correlation between optic disc pallor and RNFL thickness was examined in each quadrant. A total of 35 patients (31 male, 4 female) were enrolled in the study. The mean participant age was 34.8 ± 15.0 years (range, 5 to 63 years). Overall RNFL thickness decreased in 6 patients, with thinning most often occurring in the inferior quadrant (28 of 35 eyes). There was a significant correlation between optic disc pallor and RNFL thickness (superior, rho = -0.358, p = 0.04; inferior, rho = -0.345, p = 0.04; nasal, rho = -0.417, p = 0.01; temporal, rho = -0.390, p = 0.02). The highest level of correspondence between disc pallor and RNFL thickness values outside of the normative 95th percentiles was 39.3% and occurred in the inferior quadrant. They concluded that Optic disc pallor in TON was quantified with ImageJ and was significantly correlated with RNFL thickness

abnormalities. Thus, ImageJ evaluations of disc pallor may be useful for evaluating RNFL thinning, as verified by OCT RNFL analyses.

Hoogewoud et al[34] aimed to show ophthalmic findings are consistent enough with the diagnosis of CAR to trigger investigations aimed at detecting a previously unknown malignancy. Patients with a diagnosis of CAR were included. Diagnosis was based on the clinical presentation, the visual field and electroretinogram alterations. The clinical presentation, visual field testing and electroretinographic results were analyzed as well as the malignancies identified following the diagnosis of CAR. Four patients (two men, two women, median age 65.5 years) were included. All patients presented with posterior segment inflammation at initial presentation as well as advanced visual field loss and an extinguished electroretinogram. The best corrected decimal visual acuity was 0.8 or better in both eyes of three patients and decreased to 0.3 OD and O.2 OS in one patient due to a bilateral macular edema. No patient had a previously known history of cancer. Once the diagnosis of CAR was made, investigations aimed at identifying a malignant tumors subsequently led to the diagnosis of two cases of small cell lung tumors, of one prostate carcinoma and of a uterine sarcoma. They concluded that findings suggestive of CAR can be useful for the early detection of a cancer.

LACUNAE IN LITERATURE

Detailed history taking and supportive examinations, such as visual field, color-vision and imaging tests should also be performed. Regular follow-up of such exams is necessary for the differential diagnosis of ophthalmological diseases. The appearance of the disc can give a clue about the possible etiology. The picture of a pale optic nerve on fundoscopy can be due to numerous reasons which result in visual outcomes. It is important to find out the etiology that causes optic disc pallor, which can save patient's vision with appropriate and timely intervention. There are lesser studies conducted in particular with pale optic disc significance. There is a need to conduct more definite studies to find out the causes of pale optic disc and hence making an impact on visual prognosis.

MATERIALS AND METHODS

Study design ; This is a prospective observational study

Setting : Study conducted at the Department of Ophthalmology, Coimbatore Medical College Hospital, Coimbatore.

Duration of the study

One year period – from January 2018 to December 2018

Study population

Patients attending the Ophthalmology OPD in Coimbatore medical college hospital will be included in the study based on selection criteria. A minimum of 50 patients will be included in the study.

Inclusion criteria

Patients >20 years of age with pale optic disc on fundus examination

Exclusion criteria

Unconscious patients

Terminally ill patients

Known case of glaucoma

Patients with visual acuity of no light perception

Pale optic disc due to retinal condition

STUDY METHODS

Informed Consent is obtained from the patients selected for study.

Data are collected using structured questionnaire which comprises socio-demographic characteristics like age, gender and detailed history.

Biochemical investigations and neuroimaging are ordered when indicated to identify etiology .Clinical ocular examination done and patients are followed up for period of 6 months for visual outcome.

Clinical Examination includes

Uncorrected and best corrected Visual Acuity by Snellen's chart

IOP measurement by NCT

Refraction

Anterior segment examination

Slit lamp biomicroscopy

Fundus examination using ophthalmoscope

Fundus photograph 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)

ANALYSIS RESULTS OF PATIENTS WITH PALE OPTIC DISC

Initial BCVA, BCVA at 6 months Were considered as primary outcome variables. Etiology, Disc pallor Were considered as Secondary outcome variables.

Descriptive analysis: Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram and box plots.

All Quantitative variables were checked for normal distribution within each category of explanatory variable by using visual inspection of histograms and normality Q-Q plots. Shapiro- wilk test was also conducted to assess normal distribution. Shapiro wilk test p value of >0.05 was considered as normal distribution.

Categorical outcomes were compared between study groups using Chi square test /Fisher's Exact test (If the overall sample size was < 20 or if the expected number in any one of the cells is < 5, Fisher's exact test was used.)

P value < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.

Results:

A total of 50 subjects were included in the analysis.

Table 1: Descriptive analysis of age in study population (N=50)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
Age	38.68 ± 12.09	35.00	21.00	66.00	35.25	42.11

The mean age was 38.68 ± 12.09 in the study population, minimum was 21 and maximum was 66 in the study population (95% CI 35.35 to 42.11). (Table 1)

Table 2: Descriptive analysis of gender in the study population (N=50)

Gender	Frequency	Percentages
Male	28	56.0%
Female	22	44.0%

Among the study population, 28 (56.0%) were male and remaining 22 (44.0%) were female. (Table 2 & Figure 1)

Figure 1: Pie chart of gender in the study population (N=50)

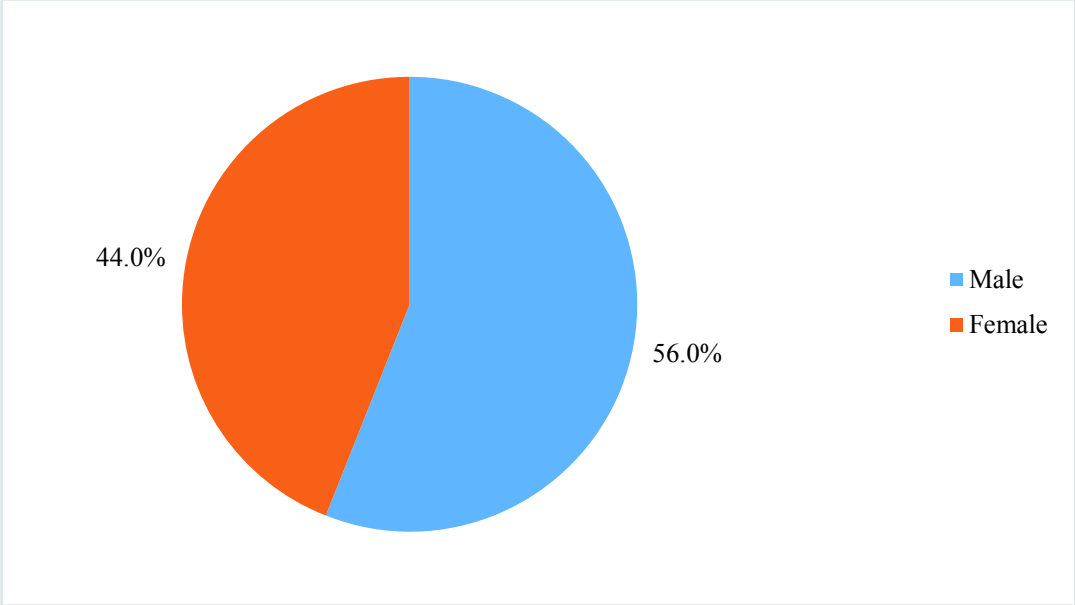


Table 3: Descriptive analysis of duration in years in study population (N=50)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
Duration in years	5.4 ± 9.3	1.50	0.10	35.00	2.75	8.04

The mean duration in years was 5.4 ± 9.3 in the study population, minimum was 0.10 and maximum was 35 in the study population (95% CI 2.75 to 8.04). (Table 3)

Table 4: Descriptive analysis of eye in the study population (N=50)

Eye	Frequency	Percentages
Right Eye	19	38.0%
Left Eye	16	32.0%
Both Eye	15	30.0%

Among the study population, 19 (38.0%) were with right eye, 16 (32.0%) were with left eye and 15 (30.0%) were with both eyes. (Table 4 & Figure 2)

Figure 2: Bar chart of eye in the study population (N=50)

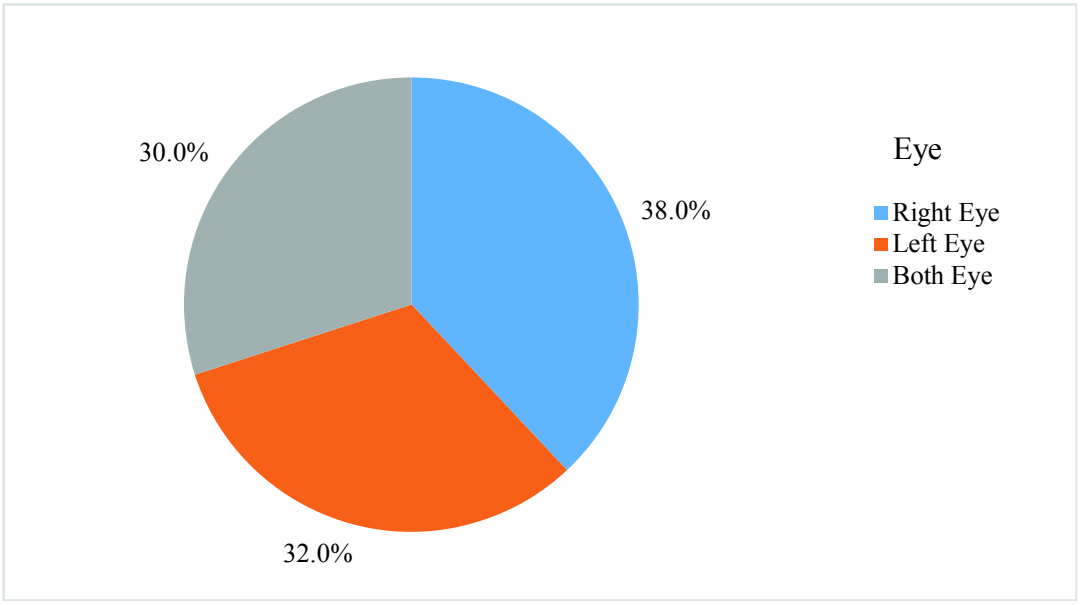


Table 5: Descriptive analysis of positive past history in the study population (N=50)

Positive Past History	Frequency	Percentages
Trauma Head Injury	14	28.0%
Optic neuritis	7	14.0%
CP angle tumor	2	4.0%
SHT/DM	6	12.0%
Alcoholic	1	2.0%
Lacrimal gland tumor	1	2.0%
Post neurosurgery	1	2.0%
Post TB meningitis	1	2.0%
Hyperlipidemia	1	2.0%
Ethambutol Intake	1	2.0%
Thyroid orbitopathy	1	2.0%

Among the study population, 14 (28%) had trauma head injury, 7 (14%) had optic neuritis, 2 (4.0%) had CP angle tumor, 6 (12%) had SHT/DM, 1 (2%) were alcoholic, 1 (2%) had lacrimal gland tumor, 1 (2%) were with post neurosurgery, 1 (2%) were with post TB meningitis, 1 (2%) had hyperlipidemia, 1 (2%) were with ethambutol intake and 1 (2%) had thyroid orbitopathy. (Table 5 & Figure 3)

Figure 3: Pie chart of positive past history in the study population (N=50)

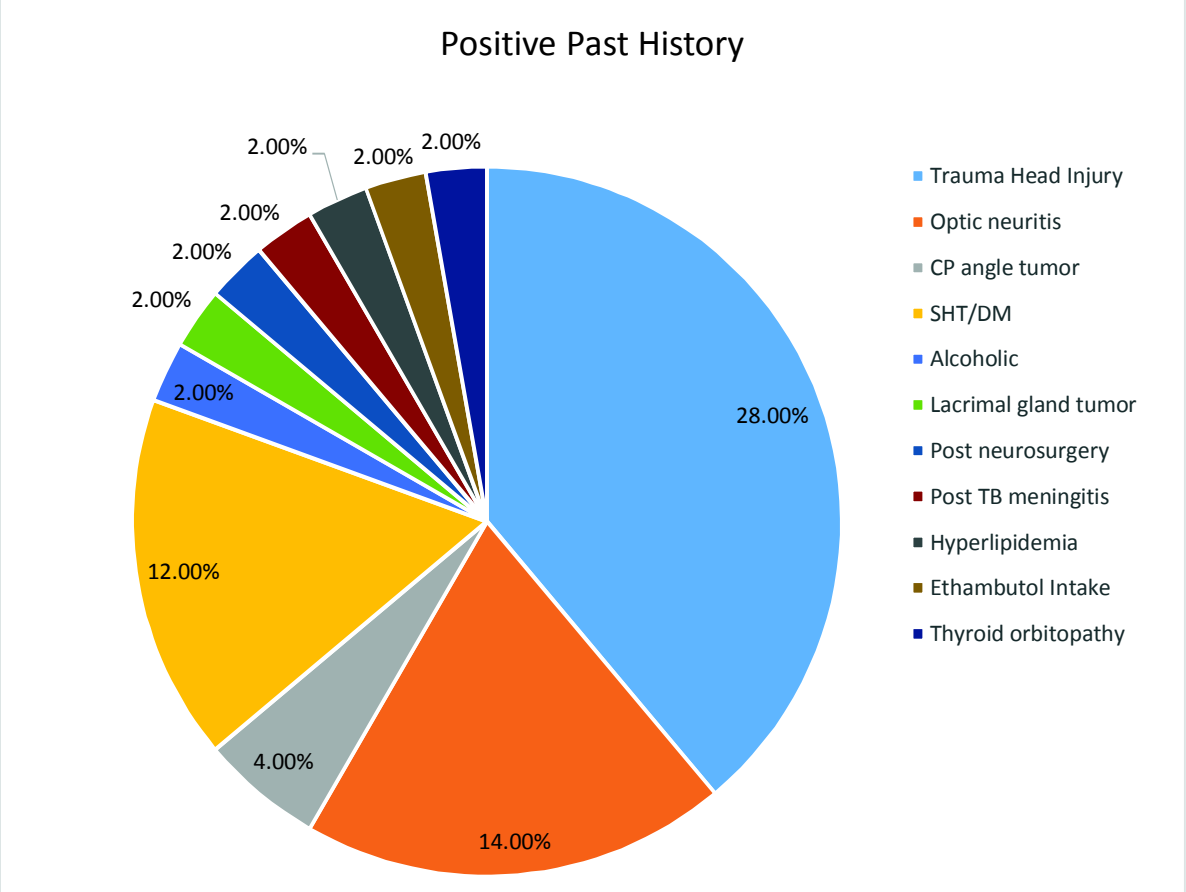


Table 6: Descriptive analysis of disc pallor types based on etiology

(N=50)

S.NO	ETIOLOGY	DISC PALLOR	FREQUENCY	PERCENTAGE
1.	Optic Neuritis	Segmental Disc Pallor	11	22%
2.	Tumor	Diffuse Disc Pallor	11	22%
3.	Primar Optic Atrophy	Total Disc Pallor	8	16%
4.	Toxic	Temporal Pallor	1	2%
5.	Inflammatory Thyroid	Diffuse Pallor	1	2%
6.	Infection Tb	Diffuse Pallor	1	2%
7.	Post Papilloedema	Diffuse Pallor	1	2%
8.	Traumatic Optic Neuropathy	Temporal Pallor	14	28%
9.	Ethambutol Induced	Temporal Pallor	1	2%
10.	Anterior Ischemic Optic Neuropathy	Segmental Pallor	1	2%

In optic neuritis etiology 11(22%) were with SEGMENTAL DISC PALLOR, in tumor etiology 11(22%) were with DIFFUSE DISC PALLOR, in primary optic atrophy 8 (16%) were with TOTAL DISC PALLOR and in traumatic optic neuropathy 14 (28%) were with TEMPORAL PALLOR. (Table 6)

Figure 4: Pie chart of etiology wise disc pallor types in the study population (N=50)

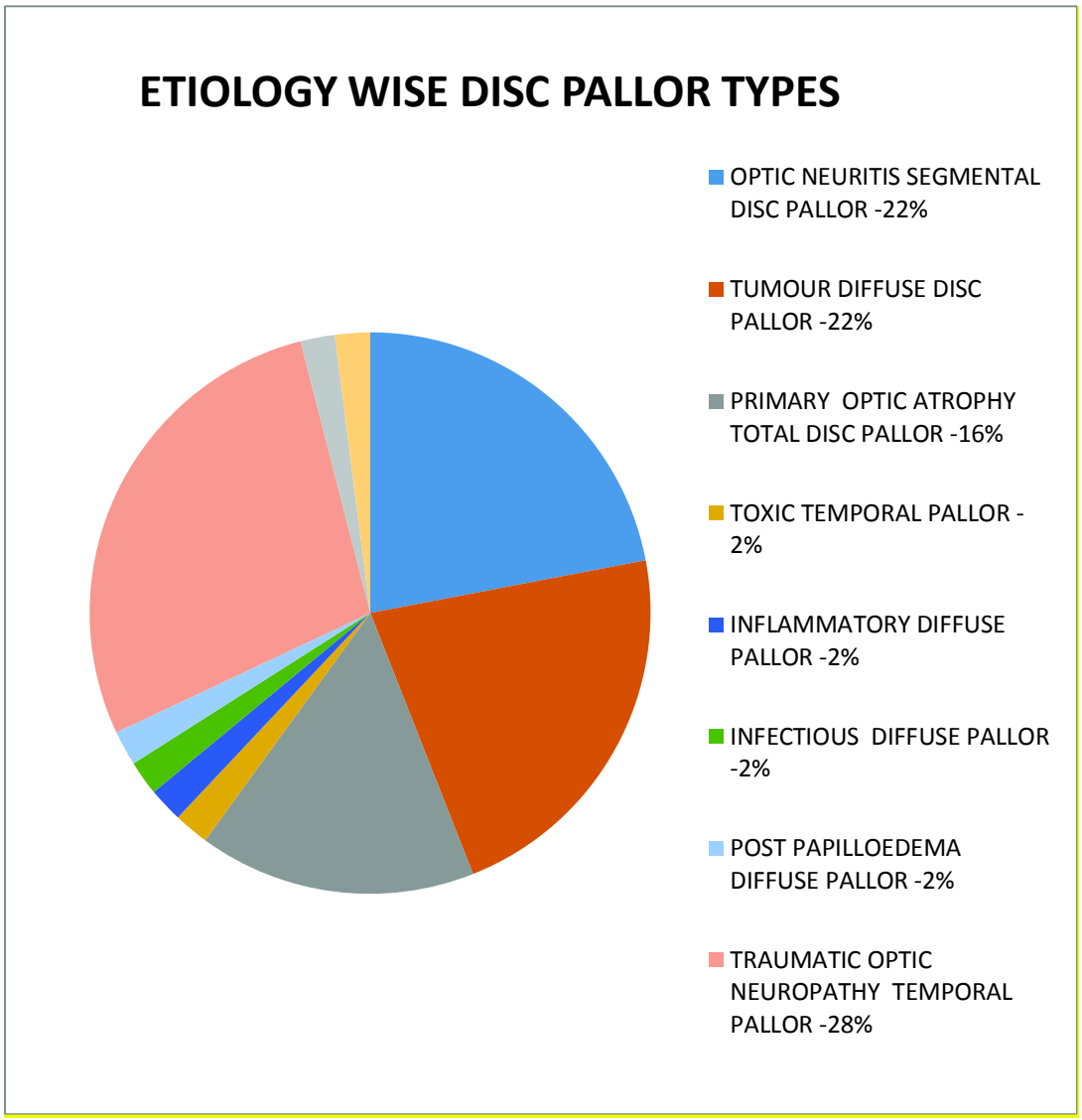


Table 7: Descriptive analysis of blood investigations in the study population (N=50)

Blood Investigations	Frequency	Percentages
Chest x-ray : cavity in RT lung base	1	2.0%
Hypertensive cardiomyopathy	1	2.0%
Increased lipid profile	1	2.0%
Sputum AFB positive	1	2.0%
TFT within control	1	2.0%
Within normal	45	90%

Among the study population, 1 (2%) were with chest x-ray: cavity in RT lung bose, 1 (2%) were with hypertensive cardiomyopathy, 1 (2%) were with increased lipid profile, 1 (2%) were with sputum AFB positive, 1 (2%) were with TFT within control and 45 (90%) had with in normal. (Table 7 & Figure 5)

Figure 5: Pie chart of blood investigations in the study population (N=50)

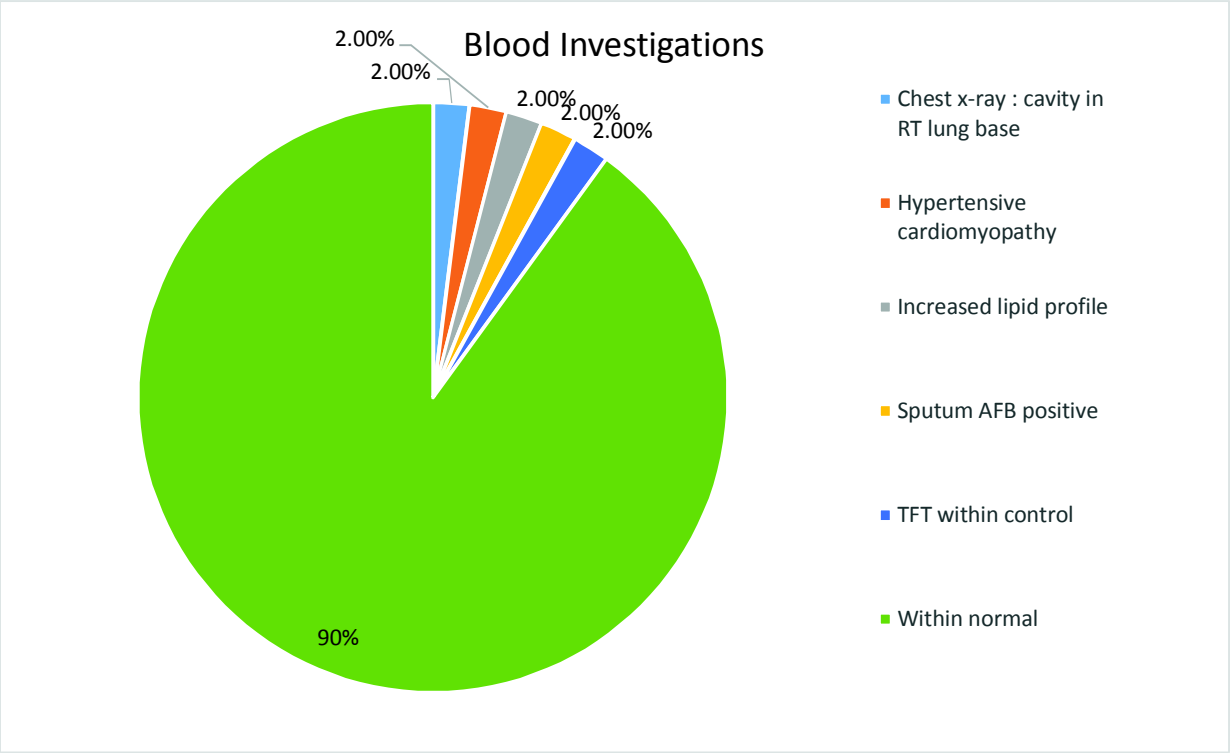


Table 8: Descriptive analysis of CT brain in the study population (N=50)

CT Brain	Frequency	Percentages
Fracture of frontal bone	5	10.0%
Parietal sol	4	8.0%
Fracture of optic canal	3	6.0%
Fracture in temporal bone	1	2.0%
Lacrimal gland tumor involving orbital apex	1	2.0%
Pituitary tumor	1	2.0%
Shunting	1	2.0%
Nil	29	58.0%

Among the study population, 5 (10%) had fracture in frontal bone, 4 (8%) were with Parietal sol, 3 (6%) had fracture of optic canal, 1 (2%) had fracture in temporal bone, 1 (2%) were with lacrimal gland tumor involving orbital apex, 1 (2%) were with pituitary tumor and 1 (2%) were with shunting. (Table 8 & Figure 6)

Figure 6: Pie chart of CT brain in the study population (N=50)

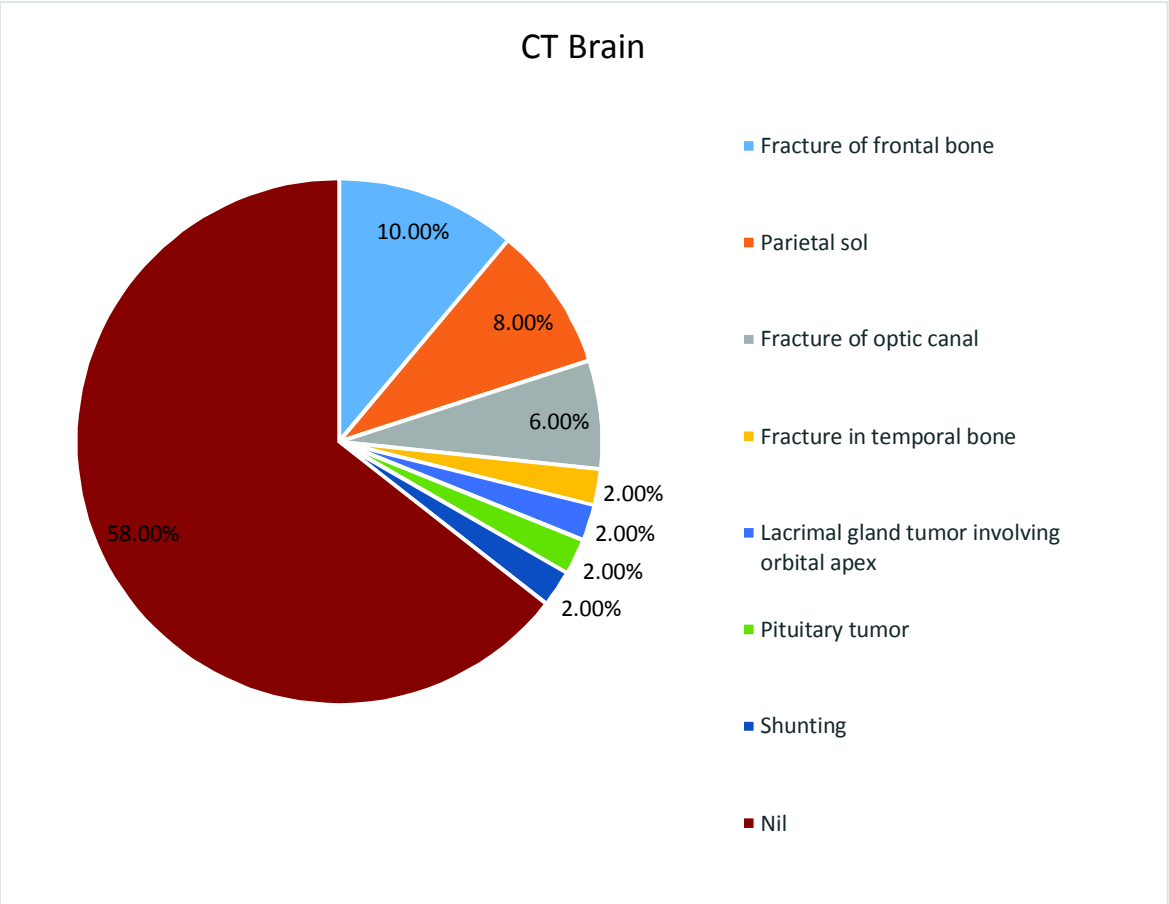


Table 9: Descriptive analysis of MRI brain etiology in the study population (N=50)

MRI Brain etiology follow Up	Frequency	Percentages
Demyelinating plaques	11	22.0%
Parietal SOL	2	4.0%
Shunting seen	2	4.0%
Increased EOM thickness	1	2.0%
Lacrimal gland tumor involving orbital apex	1	2.0%
Lesion in pituitary region	1	2.0%
Pineal astrocytoma	1	2.0%
Nil	31	62.0%

Among the study population, 11 (22%) were with demyelinating plaques, 2 (4%) were with parietal SOL, 2 (4%) were with shunting seen, 1 (2%) was increased EOM thickness, 1 (2%) was Lacrimal gland tumor involving orbital apex, 1 (2%) was lesion in pituitary region and 1 (2%) were with pineal astrocytoma. (Table 9 & Figure 7)

Figure 7: Pie chart of MRI brain etiology in the study population (N=50)

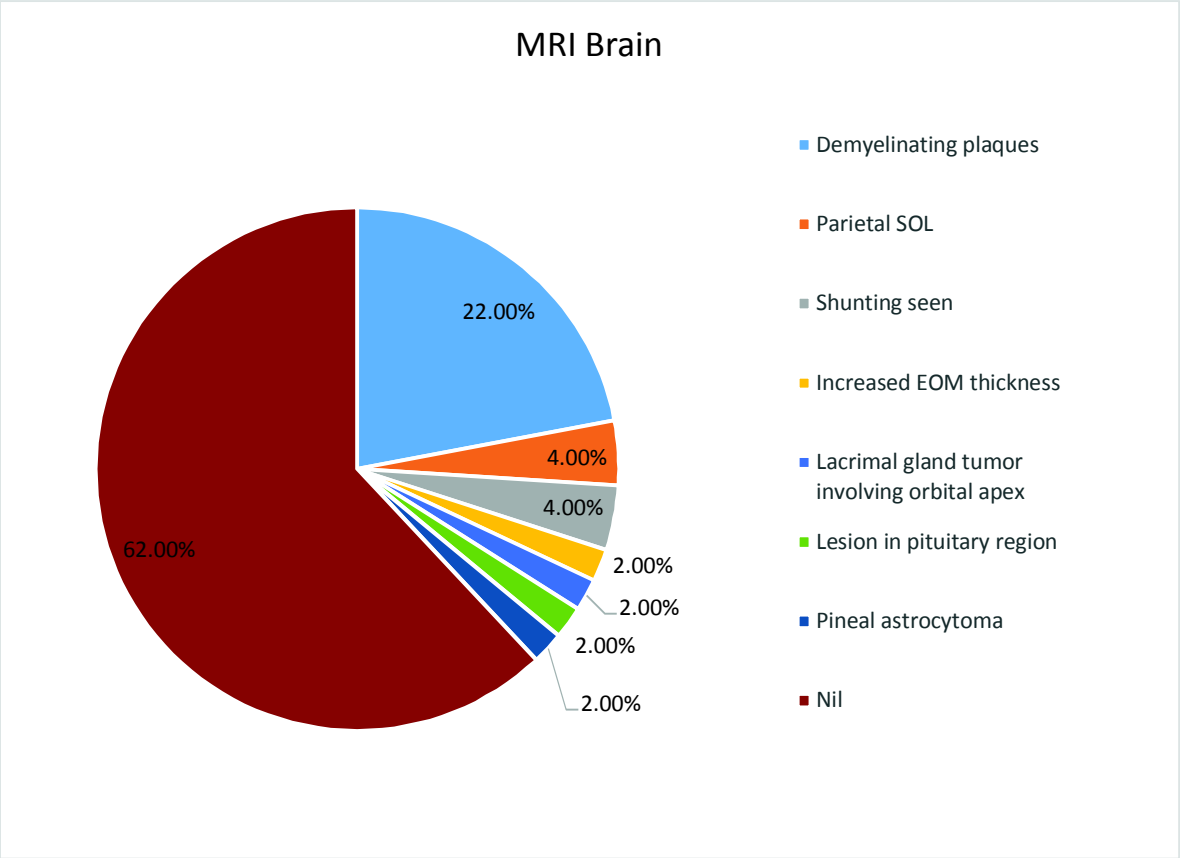


Table 10: Descriptive analysis of treatment given in the study population (N=50)

Treatment Given	Frequency	Percentages
Post i.v. steroids	14	28.0%
ONTT	11	22.0%
Observation	9	18.0%
Excision	6	12.0%
Post tumour excision	3	6.0%
Post decompression status	2	4.0%
Control of alcohol	1	2.0%
Control of SHT/hyperlipidemia	2	4.0%
Observation and stop of ATT	1	2.0%
Radiation	1	2.0%

Among the study population, 14 (28%) were with post i.v. steroids, 11 (22%) were with ONTT, 9 (18%) were in observation, 6 (12%) were with

excision, 3 (6%) were with post tumor excision, 2 (4%) were with post decompression status, 1 (2%) had control of alcohol, 2 (4%) were with control of SHT/hyperlipidemia, 1 (2%) had observation and stop of ATT, and 1 (2%) taken radiation. (Table 10 & Figure 8)

Figure 8: Pie chart of treatment given in the study population (N=50)

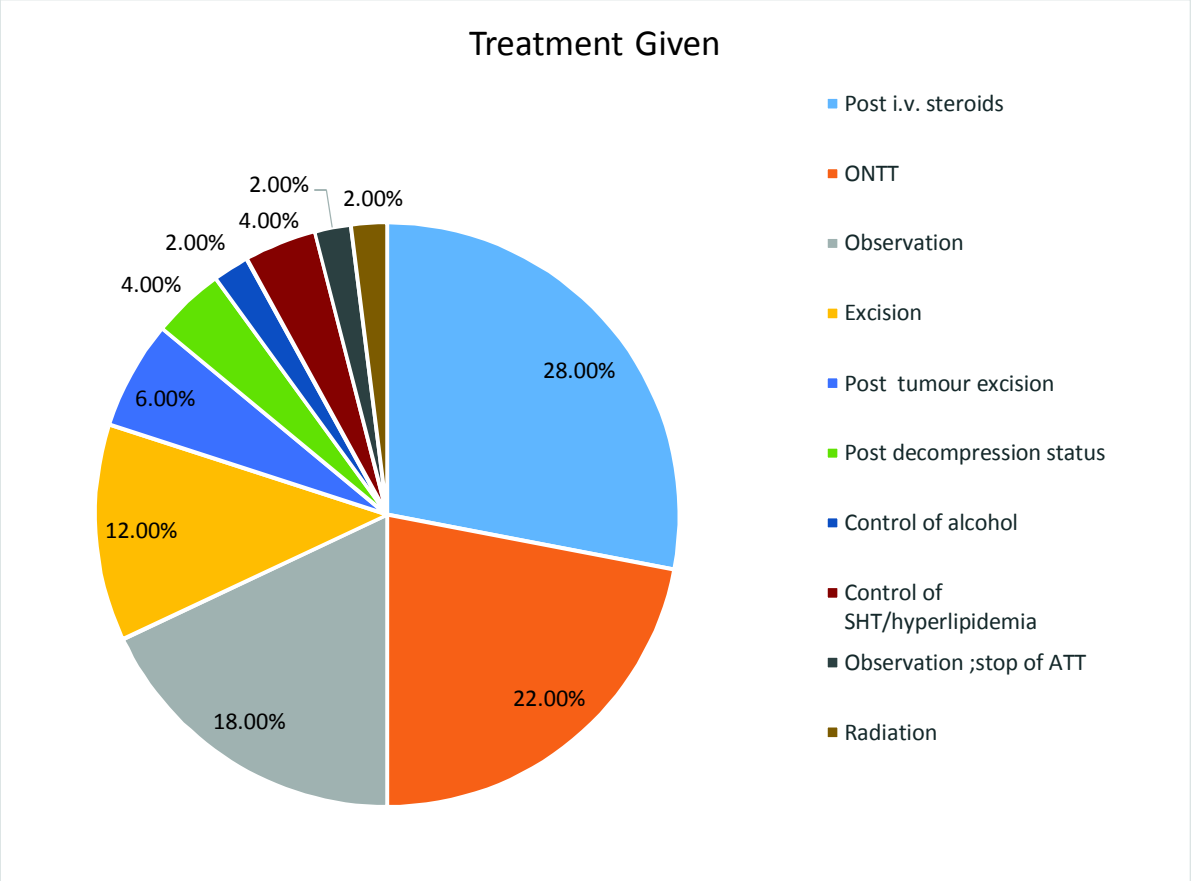


Table 11: Descriptive analysis of Best Corrected visual acuity improved or not atleast one line of Snellen's chart in the study population (N=50)

Best Corrected visual acuity improved Or Not At least One Line of Snellen's chart	Frequency	Percentages
Improved	17	34.0%
Not improved	33	66.0%

Among the study population, 17 (34%) had improved Best Corrected visual acuity improved Or Not At least One Line of Snellen's chart (Table 11 & Figure 9)

Figure 9: Bar chart of Best Corrected visual acuity improved or not atleast one line of Snellen's chart in the study population (N=50)

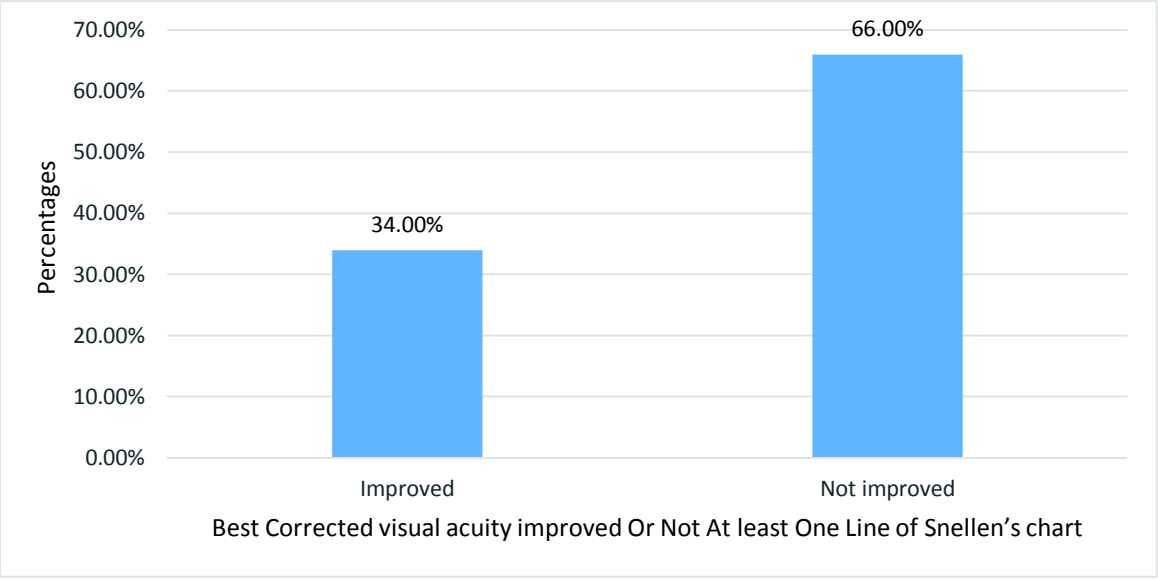


Table 12: Descriptive analysis of visual acuity improved or not based on etiology (N=50)

S.NO	ETIOLOGY	BCVA IMPROVED		BCVA NOT IMPROVED	
		FREQUENCY	PERCENTAGE	FREQUENCY	PERCENTAGE
1.	Optic Neuritis	8	16%	3	6%
2.	Tumour	6	12%	5	10%
3.	Primary Optic Atrophy	-	-	8	16%
4.	Toxic	-	-	1	2%
5.	Inflammatory Thyroid	-	-	1	2%
6.	Infectious Tuberculosis	-	-	1	2%
7.	Post Papilloedema	-	-	1	2%
8.	Traumatic Optic Neuropathy	3	6%	11	22%
9.	Ethambutol Induced	-	-	1	2%
10.	Anterior Ischemic Optic Neuropathy	-	-	1	2%

Among optic neuritis etiology people 8(16%) had improved BCVA. Among tumor etiology people 6 (12%) had improved BCVA. Among traumatic optic neuropathy 3 (6%) had improved BCVA. (Table 12 & Figure 10 & 11)

Figure 10: Pie chart of visual acuity improved or not based on etiology (N=50)

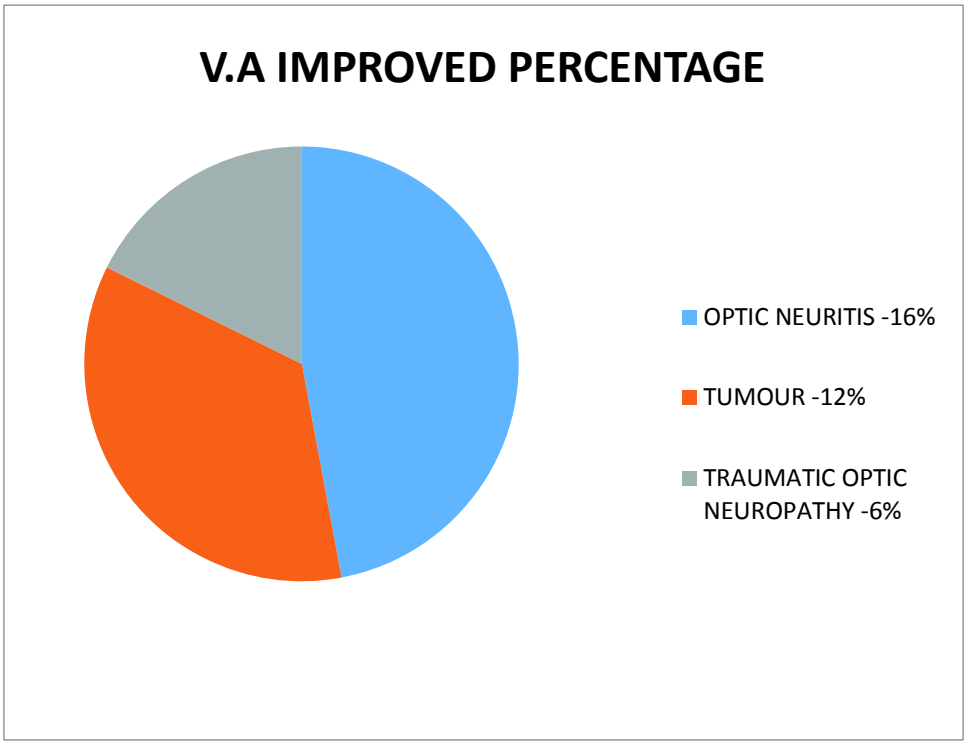


Figure 11: Pie chart of visual acuity not improved based on etiology (N=50)

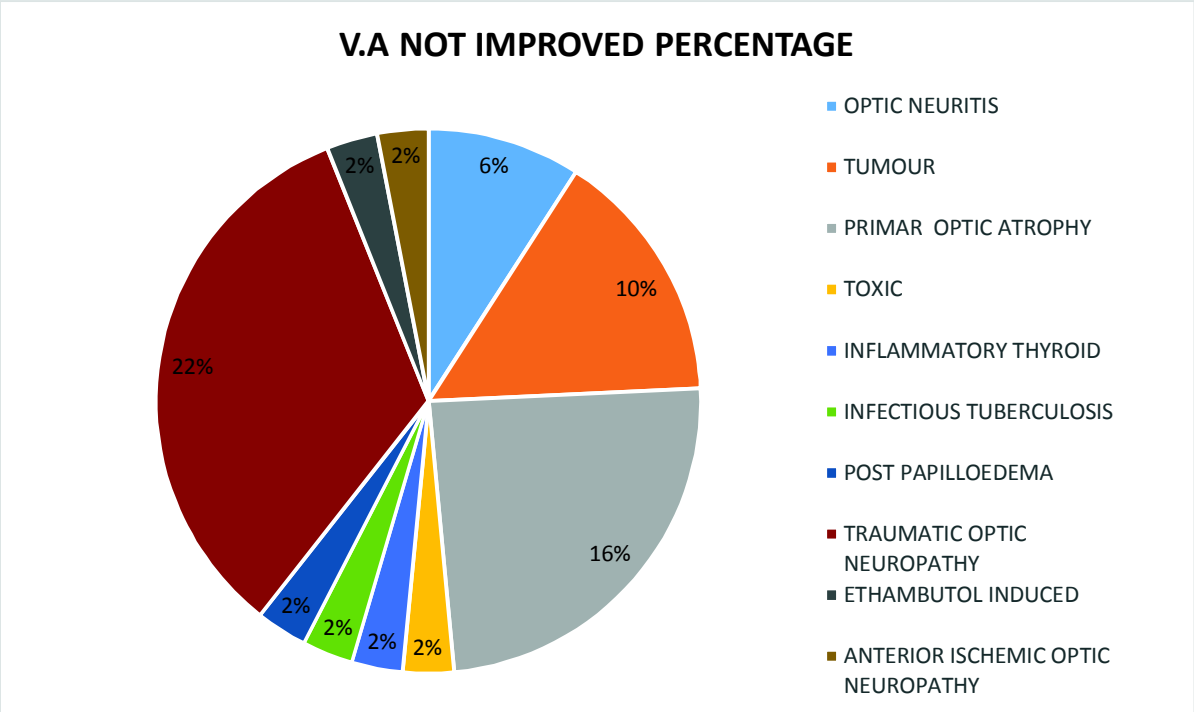


Table 13: Descriptive analysis of best corrected visual acuity of optic neuritis etiology (N= 50)

Optic neuritis	Initial BCVA	Frequency	Percentage	BCVA at 6 months	Frequency	Percentage
	6/60	9	18%	6/60	1	2%
	6/36	2	4%	6/36	3	6%
				6/24	1	2%
				6/18	3	6%
				6/12	3	6%

Among optic neuritis etiology, 9(18%) had initial BCVA as 6/60, 1(2%) had BCVA at 6 months as 6/60, 2 (4%) had initial BCVA as 6/36, 3 (6%) had BCVA at 6 months as 6/36, 1 (2%) had BCVA at 6 months as 6/24, 3 (6%) had BCVA at 6 months as 6/18 and 3 (6%) had BCVA at 6 months as 6/12. (Table 13)

Table 14: Descriptive analysis of visual acuity of trauma etiology (N=50)

TRAUMA	INITIAL BCVA	FREQUENCY	PERCENTAGE	BCVA AT 6 MONTHS	FREQUENCY	PERCENTAGE
	4/60	2	4%	4/60	1	2%
	5/60	2	4%	5/60	1	2%
	6/60	7	14%	6/60	8	16%
	6/36	3	6%	6/36	3	6%
				6/24	1	2%

Among trauma, 2 (4%) had initial BCVA as 4/60, 1 (2%) had BCVA at 6 months as 4/60, 2 (4%) had initial BCVA as 5/60, 1 (2%) had BCVA at 6 months as 5/60, 7 (14%) had initial BCVA as 6/60, 8 (16%) had BCVA at 6 months as 6/60, 3 (6%) had initial BCVA as 6/36, 3 (6%) had BCVA at 6 months as 6/36 and 1 (2%) had BCVA at 6 months as 6/24. (Table 14 & Figure 12)

Figure 12: Cluster bar chart of visual acuity of trauma etiology (N=50)

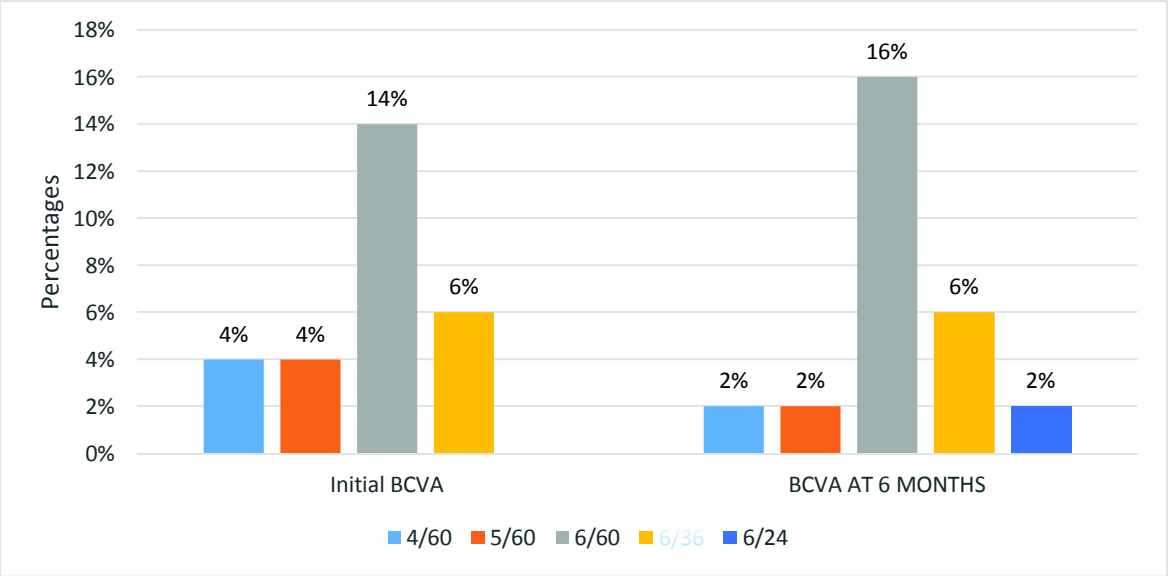


Table 15: Descriptive analysis of visual acuity of primary optic atrophy of etiology (N=50)

PRIMARY OPTIC ATROPHY	INITIAL BCVA	FREQUENCY	PERCENTAGE	BCVA AT 6 MONTHS	FREQUENCY	PERCENTAGE
	2/60	2	4%	2/60	2	4%
	3/60	1	2%	3/60	1	2%
	4/60	1	4%	4/60	1	2%
	5/60	2	4%	5/60	2	4%
	6/60	2	4%	6/60	2	4%

Among primary optic atrophy, 2 (4%) had initial BCVA as 2/60, 2 (4%) had BCVA at 6 months as 2/60, 1 (2%) had initial BCVA as 3/60, 1 (2%) had BCVA at 6 months as 3/60, 1 (4%) had initial BCVA as 4/60, 1 (2%) had BCVA at 6 months as 4/60, 2 (4%) had initial BCVA as 5/60, 2 (4%) had BCVA at 6 months as 5/60, 2 (4%) had initial BCVA as 6/60 and 2 (4%) had BCVA at 6 months as 6/60. (Table 15 & Figure 13)

Figure 13: Cluster bar chart of visual acuity of primary optic atrophy of etiology (N=50)

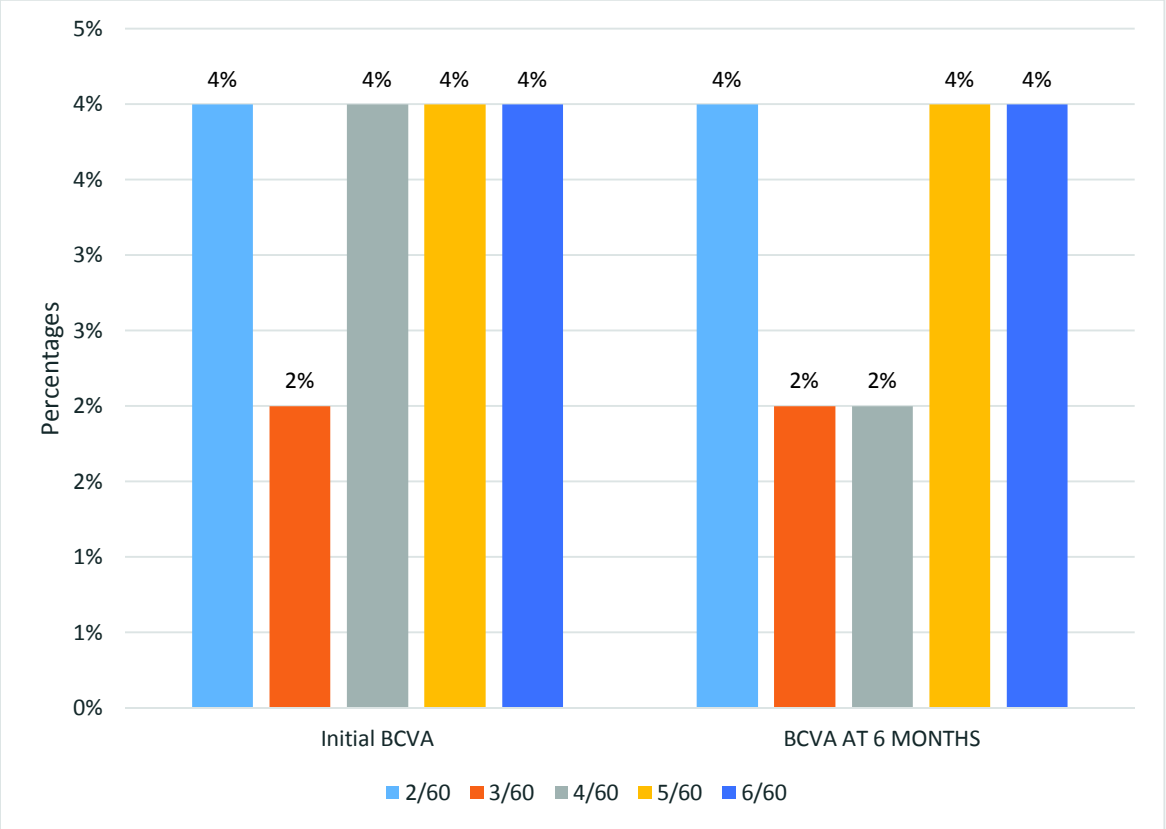


Table 16: Descriptive analysis of visual acuity of tumour etiology

(N=50)

TUMOUR	INITIAL BCVA	FREQUENCY	PERCETAGE	BCVA AT 6 MONTHS	FREQUENCY	PERCENTAGE
	2/60	2	4%	2/60	2	4%
	4/60	1	2%	6/60	2	4%
	6/60	3	6%	6/36	2	4%
	6/36	2	4%	6/18	3	6%
	6/18	2	4%	6/12	2	4%
	6/12	1	2%			

Among tumour, 2 (4%) had initial BCVA as 2/60, 2 (4%) had BCVA at 6 months as 2/60, 1 (2%) had initial BCVA as 4/60, 2 (4%) had BCVA at 6 months as 6/60, 3 (6%) had initial BCVA as 6/60, 2 (4%) had BCVA at 6 months as 6/36, 2 (4%) had initial BCVA as 6/36, 3 (6%) had BCVA at 6 months as 6/18, 2 (4%) had initial BCVA as 6/18, 3 (6%) had BCVA at 6 months as 6/12 and 1 (2%) had initial BCVA as 6/12. (Table 16 & Figure 14)

Figure 14: Cluster bar chart of visual acuity of tumour etiology (N=50)

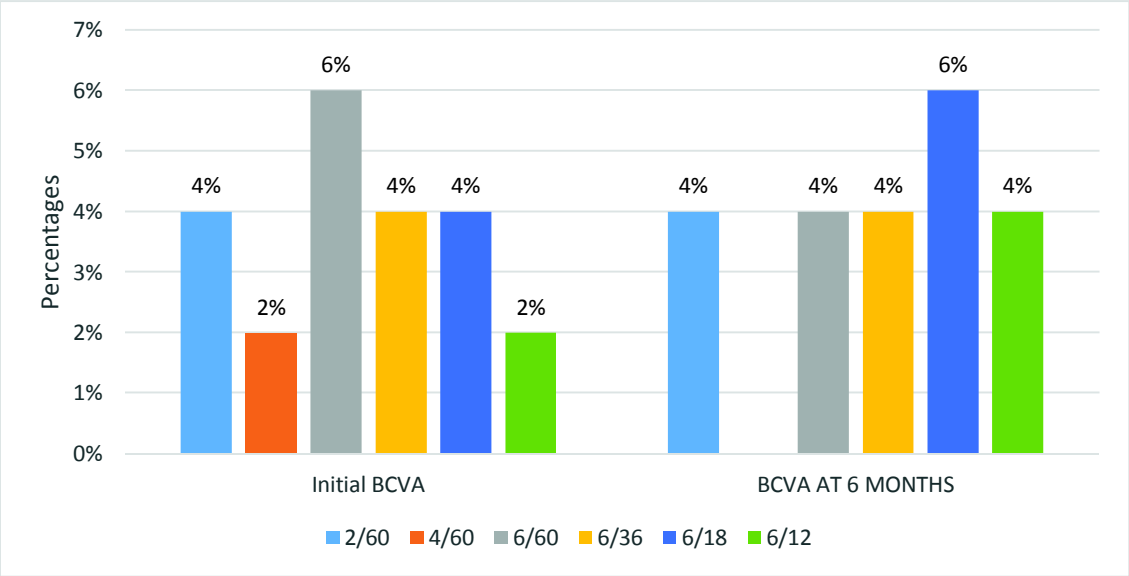


Table 17: Descriptive analysis of visual acuity of other etiologies (N= 50)

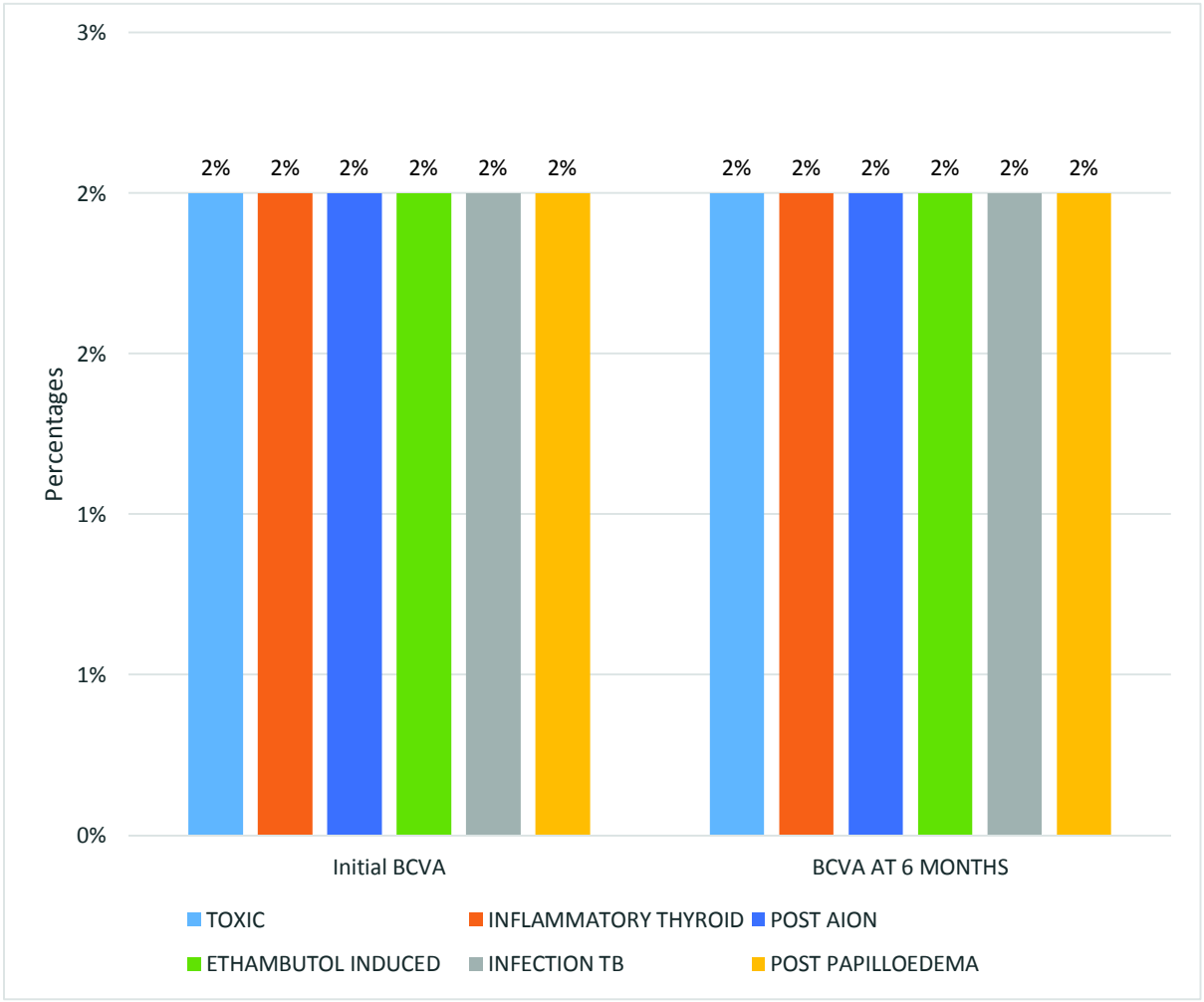
ETIOLOGY	INITIAL BCVA	FREQUENCY	PERCENTAGE	BCVA AT 6 MONTHS	FREQUENCY	PERCENTAGE
TOXIC	6/36	1	2%	6/36	1	2%
INFLAMMATORY THYROID	6/60	1	2%	6/60	1	2%

INFECTION TB	4/60	1	2%	4/60	1	2%
POST PAPILLOEDEMA	6/36	1	2%	6/36	1	2%
POST AION	5/60	1	2%	5/60	1	2%
ETHAMBUTOL INDUCED	6/36	1	2%	6/36	1	2%/

Among toxic etiology, 1 (2%) had initial BCVA as 6/36, 1 (2%) had BCVA at 6 months as 6/36. Among inflammatory thyroid, 1 (2%) had initial BCVA as 6/60, 1 (2%) had BCVA at 6 months as 6/60. Among infection TB, 1 (2%) had initial BCVA as 4/60, 1 (2%) had BCVA at 6 months as 4/60. Among post papilloedema, 1 (2%) had initial BCVA as 6/36, 1 (2%) had BCVA at 6 months as 6/36. Among post aion, 1 (2%) had initial BCVA as 5/60, 1 (2%) had BCVA at 6 months as 5/60. Among ethambutol induced, 1 (2%) had initial BCVA as 6/36, 1 (2%) had BCVA at 6 months as 6/36. (Table 17 & Figure 15)

Figure 15: Cluster bar chart of visual acuity of other etiologies

(N= 50)



DISCUSSION

Optic atrophy is one the common findings observed in ophthalmic out patient departments and it results in permanent visual disability depending on the etiology. Identifying the actual cause of optic atrophy helps in deciding its treatment course. Disc pallor is the main manifestation of optic atrophy. A detailed clinical evaluation is helpful in the differential diagnosis and management of optic atrophy. There is no specific treatment for optic atrophy itself. The underlying cause whether inflammatory, ischaemic, compressive or metabolic should be treated if known.[2]

The main objective of this study was to enumerate etiology of pale optic disc in patients attending Ophthalmology opd and correlate it with visual outcome in a tertiary care hospital. Initial BCVA, BCVA at 6 months were considered as primary outcome variables. Etiology, Disc pallor were considered as secondary outcome variables. A total of 50 subjects were included in the analysis with a mean age of 38.68 ± 12.09. The age of subjects was widely variable ranging from as low as 21 years to as high as 66 years. This variation is useful for the study because age is considered as an important demographic parameter while short listing possible etiologies of disc pallor. Studies have established possible causes of disc pallor depending on presenting age. [4] As the average age of

subjects in this study is above 35 majority of subjects may be considered as belonging to middle age group. The most common etiology observed in the study is traumatic optic neuropathy in 28% of subjects. This can be correlated with the observation mentioned in the study by **Singh D et al.** [4] that traumatic optic neuropathy is the most common etiology for disc pallor in young adults. Among the study population, 28 (56.0%) were male and remaining 22 (44.0%) were female. Among the study population, 28% had past trauma head injury and this can be related to traumatic optic neuropathy observed in 28% of subjects. Among the study population 22% of subjects were found to have tumor etiology which is similar to that found in a study by Menon et al [36] where tumor etiology was found in 24.4% of patients. However this percentage was lesser than that found in the study by Mbekeani JN et al. [37] in which 62.2% had tumor etiology for optic atrophy.

Among study population optic neuritis etiology was found in 22% patients. With demyelinating plaques in MRI which can be correlated with optic neuritis etiology found in the study.

Among optic neuritis etiology, 18% had initial BCVA as 6/60, 2% had BCVA at 6 months as 6/60, 4% had initial BCVA as 6/36, 3 6% had BCVA at 6 months as 6/36, 2% had BCVA at 6 months as 6/24, 6% had BCVA at 6 months as 6/18 and 6% had BCVA at 6 months as 6/12. In a

study by **Wang I-H, et al.** [38] 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.

The following table gives values of visual acuity in meters and its corresponding value in feet which is the unit used for measurement in US:

Table : 18 Visual Acuity

Visual acuity **in meters**	**Visual** acuity **in feet**
6/60	20/200
6/36	20/120
6/24	20/80
6/18	20/60
6/12	20/40
6/9	20/30
6/6	20/20
6/5	20/16

In the current study, the study population median age was 35 with minimum being 21 and maximum 66. **Ambika et al.**[32] did a study on visual outcomes and clinical manifestations of pediatric optic neuritis in Indian Population and enrolled 42 (53.8%) females and 36 (46.2%) who are all younger than 18 years and found out that 60 out of the 84 eyes (72.3%) recovered visual acuity of 20/40 or better whereas in the current study with median age of 35 (ranging 21 to 66) improved visual outcome was only 28%. It implies that success rate of visual outcome is more in younger population than in older. The proportion of male population is more than female (28 Male (56.0%) 22 Female (44.0%) as compared to the current study that has 42 (53.8%) females and 36 (46.2%) males.

Kang et al.[33] studied 35 patients, age ranging from 5 to 63 years. The etiology of these patients is traumatic optic neuropathy. They found out that overall RNFL thickness decreased in 6 patients. In our current study, the study population with etiology of traumatic optic neuropathy is 28% (14 out of 50 subjects). Only 6% had BCVA improved and 22% did not improved. 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.

The present study showed that the study population with positive past history of tumors such as CP angle tumor and lacrimal gland tumor

(22%) had diffuse disc pallor and among tumor etiology people, 12% had improved BCVA and 10% had not improved BCVA.

Optic atrophy signifies a potentially more serious clinical sign of an underlying condition. In our present study, among subjects with etiology of primary optic atrophy, 16% were with total disc pallor and visual outcome was poor in entire subjects. BCVA had not improved in 16%. Dewitt et al. did a detailed study on visual function in patients with optic nerve pallor (optic atrophy) and found out that good visual acuity was found in 55/86 (64.0%) mild, 54/119 (45.4%) moderate, and 21/65 (32.3%) marked optic atrophy eyes. As the graded severity of optic atrophy increases, the proportion of eyes with good visual function decreases. Both this study and our present study have similar findings that marked optic atrophy leads to poor visual outcome

SUMMARY

1. A total of 50 subjects were included in the analysis. The mean age was 38.68 ± 12.09 years. The proportion of males were 56.0% and remaining 44.0% were female.

2. The disc pallor is prominent only in population with past history of optic neuritis etiology which consists of 22% of the study participants were with segmental disc pallor. In tumor etiology 22% were with diffuse disc pallor, in primary optic atrophy 16% were with total disc pallor . In traumatic optic neuropathy, 28% were with temporal pallor.

3. The disc pallor is diffuse in population with past history of 2 (4.0%) CP angle tumor, 6 (12%) SHT/DM, 1 (2%) alcoholic, 1 (2%) lacrimal gland tumor, 1 (2%) post neurosurgery, 1 (2%) post TB meningitis, 1 (2%) hyperlipidemia, 1 (2% and 1 (2%) thyroid orbitopathy. 1 (2%) ethambutol intake case presented with temporal disc pallor and 1(2%) AION case presented with segmental disc pallor.

4. 34% of the participants had improved Best Corrected visual acuity atleast one line in Snellen's chart and 33 (66%) not improved.

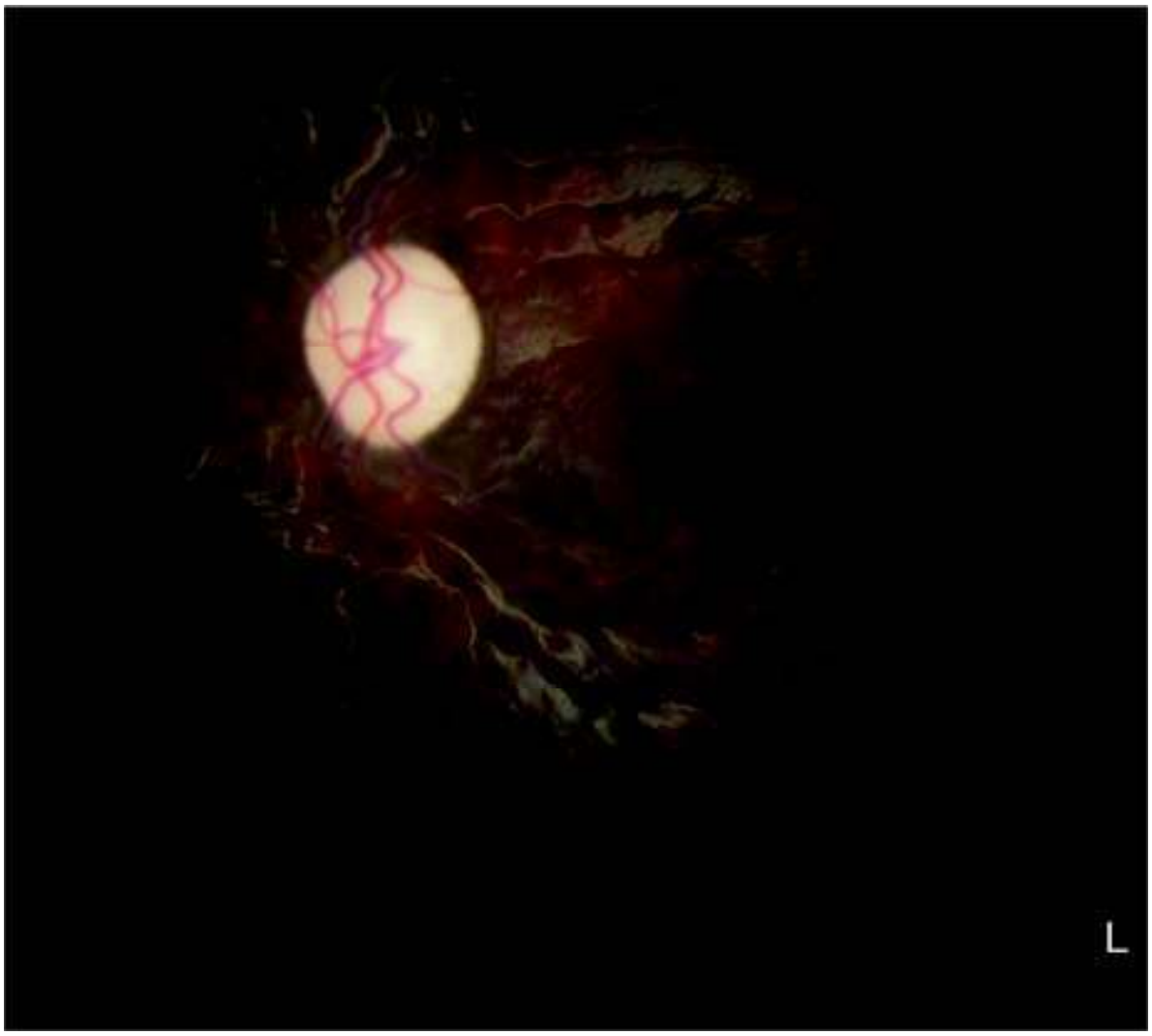
5. Visual acuity improved in population with optic neuritis etiology people 8(16%) had improved BCVA. Among tumor etiology people 6 (12%) had improved BCVA. Among traumatic optic neuropathy 3 (6%) had improved BCVA. The main etiology observed for optic atrophy among the study population was traumatic optic neuropathy. Majority of study population were young adults and observation of this etiology may be attributed to their age group. Visual activity outcome measurements showed Best Corrected visual acuity improved only in 34% of study population. Hence visual activity outcome can be concluded to be poor among study population after treatment.

CONCLUSION

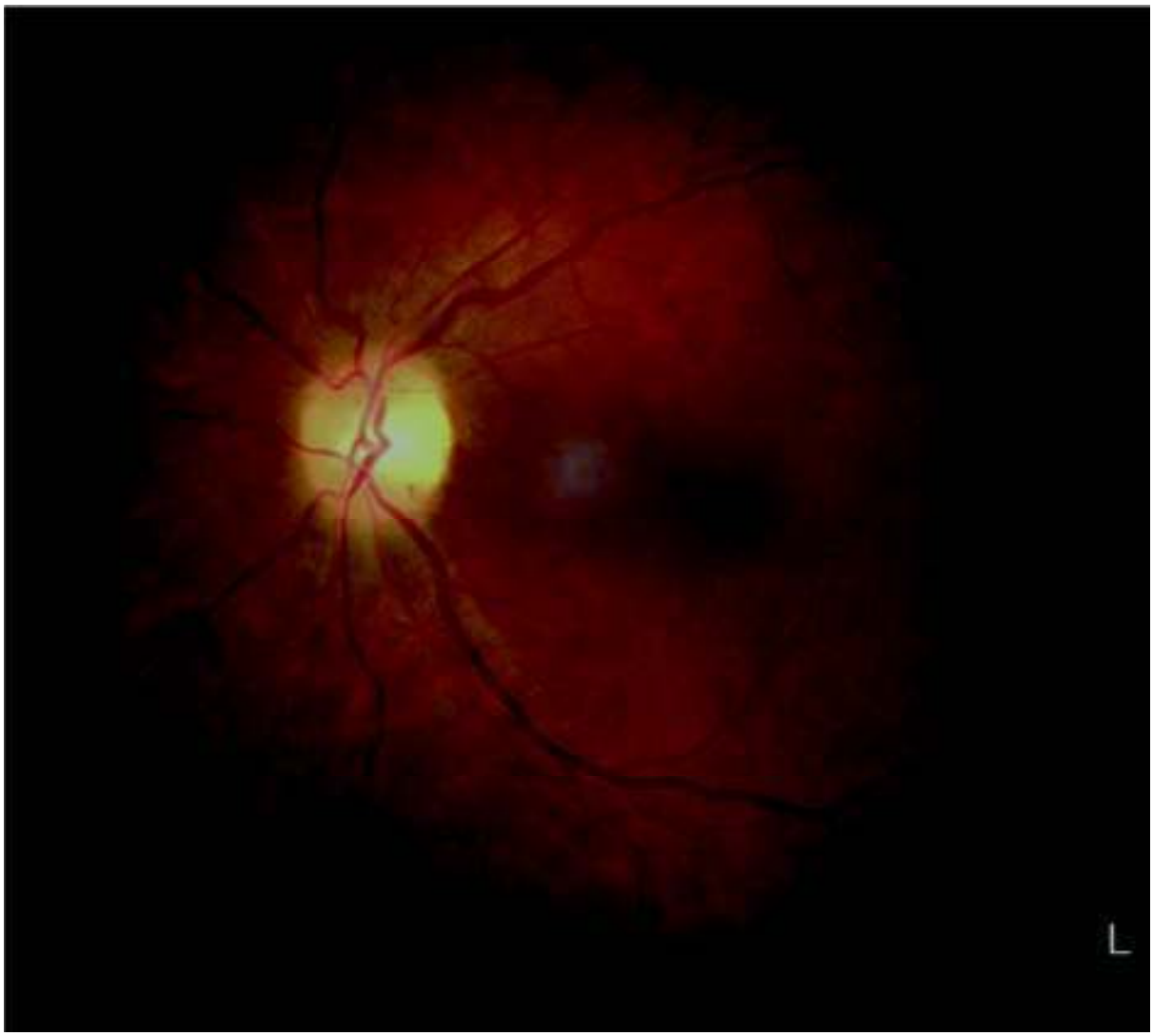
Patients presenting with pale optic disc with optic neuritis, tumour, trauma etiology showed visual acuity of one line improvement in Snellen's chart , whereas pale optic disc with other etiology showed no improvement in visual acuity over a period of 6 months.

Pale optic disc may be threat to patient's vision and identifying the etiology can save patient's vision and also associated underlying systemic causes by timely intervention. Moreover further optic nerve damage can be halted by identifying the etiology and treating the cause.

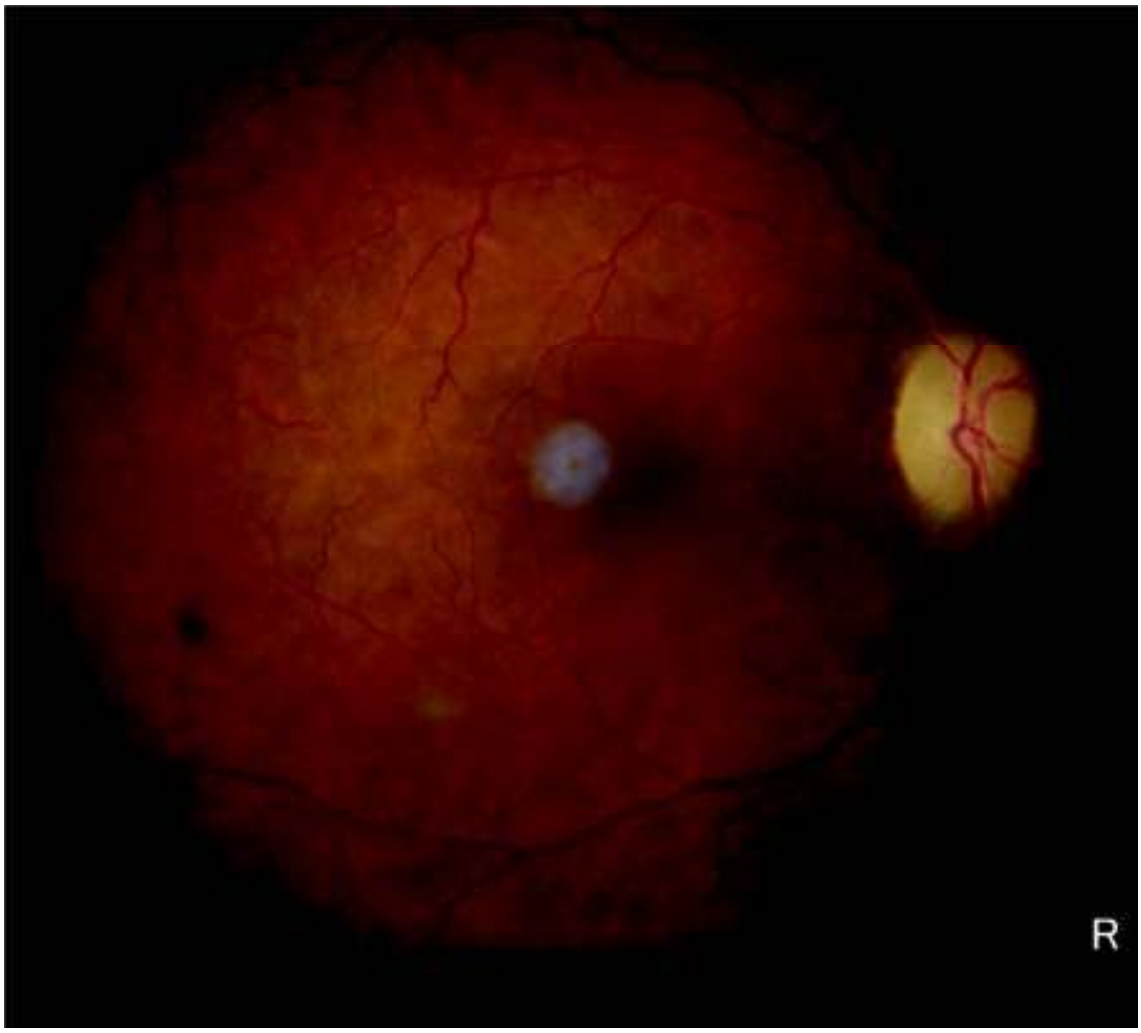
Picture 1: Fundus photo showing LE disc pallor due to primary optic atrophy



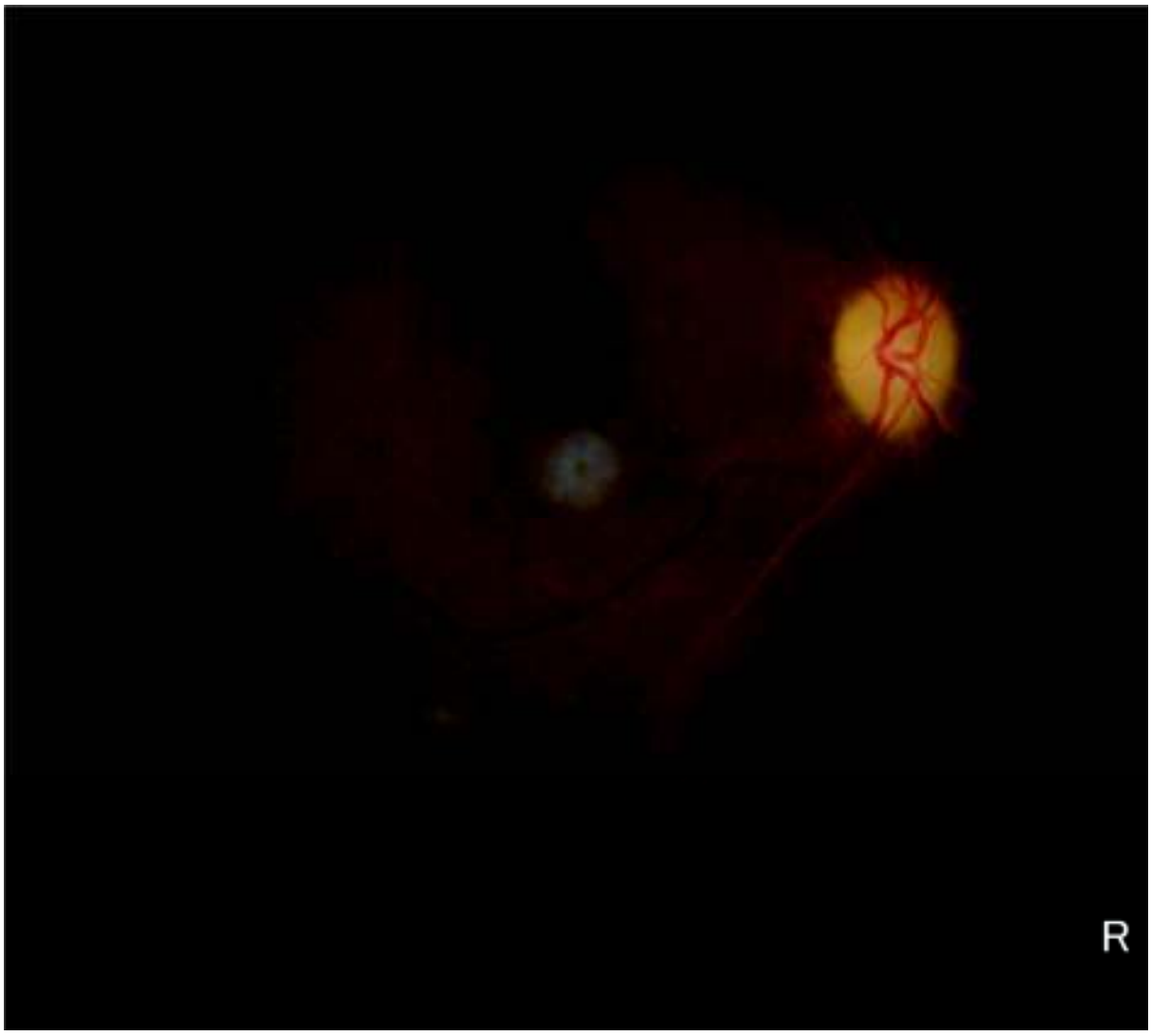
Picture 2:Fundus photo showing LE Temporal disc pallor due to trauma



Picture 3 : Fundus photo showing RE disc pallor due to tumour

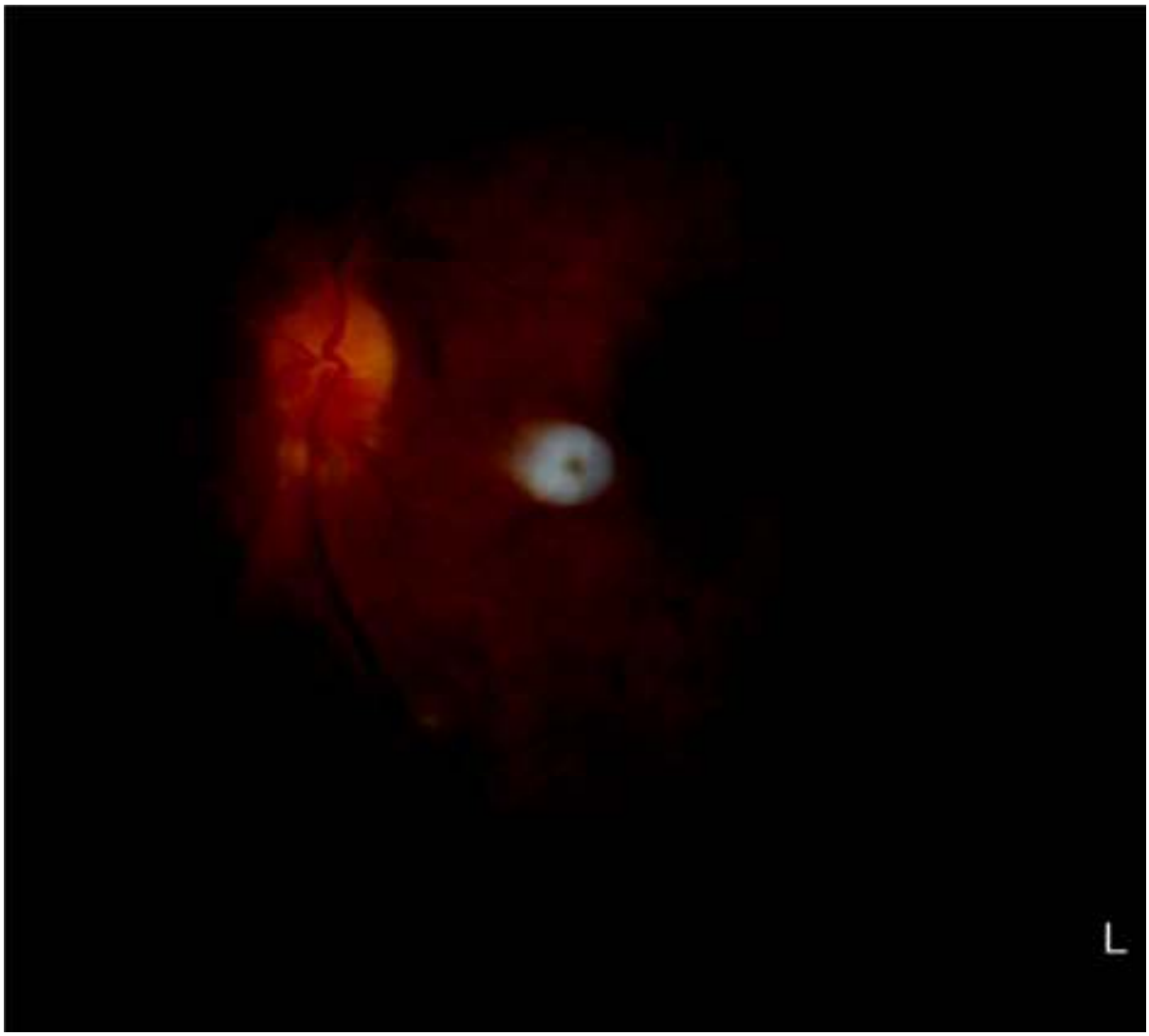


Picture 4: Fundus photo showing RE disc pallor due to post papilloedema

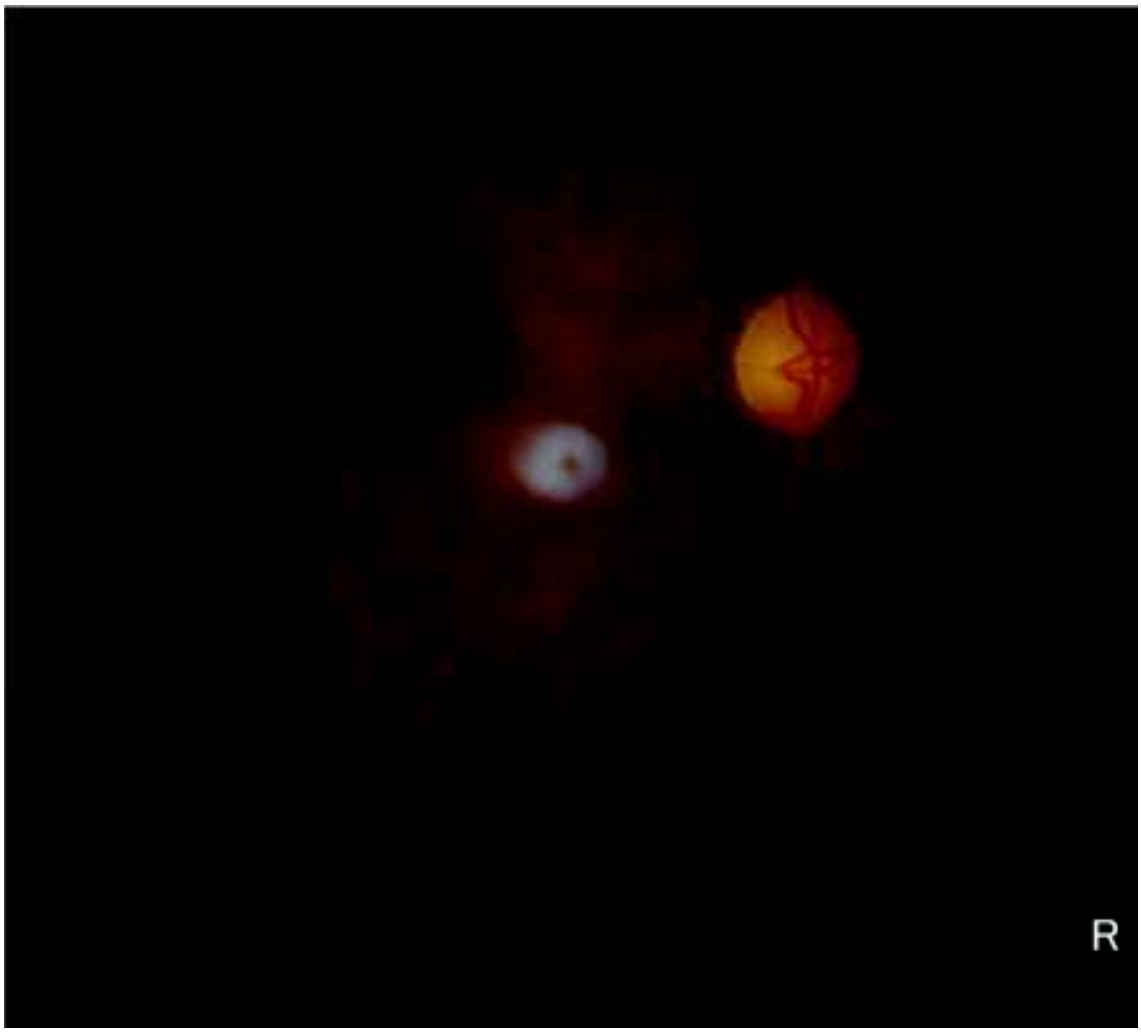


Picture 5:Fundus photo showing RE disc pallor due to toxin (alcohol)

Picture 6:Fundus photo showing LE segmental disc pallor due to post AION



Picture 7 :Fundus photo showing RE disc pallor due to optic neuritis



BIBILOGRAPHY

1. Naidu A, Srinivas VS. A CLINICAL STUDY OF OPTIC ATROPHY-ITS AETIOLOGY, PATHOGENESIS, CLINICAL MANIFESTATIONS, MANAGEMENT AND VISUAL OUTCOME.

2. Osaguona VB. Differential diagnoses of the pale/white/atrophic disc. Community Eye Health. 2016;29(96):71-4.

3. Jacobson LK, Dutton GN. Periventricular leukomalacia: an important cause of visual and ocular motility dysfunction in children. Survey of ophthalmology. 2000;45(1):1-13.P

4. Singh D, Saxena R, Sharma P, Menon V. Systematic approach to a case of disc pallor. The Official Scientific Journal of Delhi Ophthalmological Society. 2011;21(3):28-32.

5. Lee AG, Chau FY, Golnik KC, Kardon RH, Wall M. The diagnostic yield of the evaluation for isolated unexplained optic atrophy. Ophthalmology. 2005;112(5):757-9.

6. Mansour AM, Hamed LM. Racial variation of optic nerve diseases. Neuro-ophthalmology. 1991;11(6):319-23.

7. Chatziralli I, Theodossiadis P, Theodossiadis G. New Observations regarding the Assessment of Cilioretinal Arteries Coexistence with Optic Disc Pit. Semin Ophthalmol. 2019;34(5):375-9.

8. Bhattacharya S, Goel S, Saurabh K, Roy R. Multicolor Imaging of Myelinated Nerve Fibers Contiguous to the Optic Disc. J Neuroophthalmol. 2019.

9. Gopi VP, Anjali MS, Niwas SI. PCA-based localization approach for segmentation of optic disc. Int J Comput Assist Radiol Surg. 2017;12(12):2195-204.

10. Sedai S, Roy PK, Mahapatra D, Garnavi R. Segmentation of optic disc and optic cup in retinal fundus images using shape regression. Conf Proc IEEE Eng Med Biol Soc. 2016;2016:3260-4.

11. Reis AS, Sharpe GP, Yang H, Nicolela MT, Burgoyne CF, Chauhan BC. Optic disc margin anatomy in patients with glaucoma and normal controls with spectral domain optical coherence tomography. Ophthalmology. 2012;119(4):738-47.

12. Mistlberger A, Liebmann JM, Greenfield DS, Hoh S-T, Ishikawa H, Marmor M, et al. Assessment of optic disc anatomy and nerve fiber layer thickness in ocular hypertensive subjects with normal short-

wavelength automated perimetry. Ophthalmology. 2002;109(7):1362-6.

13. Krivoy D, Gentile R, Liebmann JM, Stegman Z, Walsh JB, Ritch R. Imaging congenital optic disc pits and associated maculopathy using optical coherence tomography. Archives of Ophthalmology. 1996;114(2):165-70.

14. Chaddah MR, Khanna KK, Chawla GD. Optic atrophy (review of 100 cases). Indian J Ophthalmol. 1971;19(4):172-6.

15. Jung JJ, Baek S-H, Kim US. Analysis of the causes of optic disc swelling. Korean Journal of Ophthalmology. 2011;25(1):33-6.

16. Eckert G, Melamed J, Menegaz B. Optic nerve changes in ocular toxoplasmosis. Eye. 2007;21(6):746.

17. Uchida Y, Kakehashi Y, Kameyama K. Juxtapapillary retinochoroiditis with a psychiatric disorder possibly caused by toxoplasma. American journal of ophthalmology. 1978;86(6):791-3.

18. Banta JT, Davis JL, Lam BL. Presumed toxoplasmosic anterior optic neuropathy. Ocular immunology and inflammation. 2002;10(3):201-11.

19. Gonçalves EC, Oréfice F, Mendes AG, Pedroso EP. Toxoplasmose ocular adquirida tardia: Relato de três casos simultâneos em membros da mesma família. Rev Bras Oftalmol. 1995;54(5):57-60.

20. Folk JC, Lobes LA. Presumed toxoplasmic papillitis. Ophthalmology. 1984;91(1):64-7.

21. Song A, Scott IU, Davis JL, Lam BL. Atypical anterior optic neuropathy caused by toxoplasmosis. American journal of ophthalmology. 2002;133(1):162-4.

22. Hayreh SS, Jonas JB. Optic disc morphology after arteritic anterior ischemic optic neuropathy. Ophthalmology. 2001;108(9):1586-94.

23. Van Stavern GP. Optic disc edema. Semin Neurol. 2007;27(3):233-43.

24. Wilhelm H, Schiefer U. Optic disc signs and optic neuropathies. Clinical Neuro-Ophthalmology: Springer; 2007. p. 101-25.

25. O'neill EC, Danesh-Meyer HV, Connell PP, Trounce IA, Coote MA, Mackey DA, et al. The optic nerve head in acquired optic neuropathies. Nature Reviews Neurology. 2010;6(4):221.

26. How AC, Tan GS, Chan Y-H, Wong TT, Seah SK, Foster PJ, et al. Population prevalence of tilted and torted optic discs among an adult

Chinese population in Singapore: the Tanjong Pagar Study. Archives of ophthalmology. 2009;127(7):894-9.

27. Chinta S, Wallang BS, Sachdeva V, Gupta A, Patil-Chhablani P, Kekunnaya R. Etiology and clinical profile of childhood optic nerve atrophy at a tertiary eye care center in South India. Indian J Ophthalmol. 2014;62(10):1003-7.

28. Jacobson L, Hård A, Svensson E, Flodmark O, Hellström A. Optic disc morphology may reveal timing of insult in children with periventricular leucomalacia and/or periventricular haemorrhage. British Journal of Ophthalmology. 2003;87(11):1345-9.

29. Kamal D, Garway-Heath D, Hitchings R, Fitzke F. Use of sequential Heidelberg retina tomograph images to identify changes at the optic disc in ocular hypertensive patients at risk of developing glaucoma. British Journal of Ophthalmology. 2000;84(9):993-8.

30. Quigley HA, Hohman RM, Addicks EM. Quantitative study of optic nerve head capillaries in experimental optic disk pallor. American journal of ophthalmology. 1982;93(6):689-99.

31. Savini G, Bellusci C, Carbonelli M, Zanini M, Carelli V, Sadun AA, et al. Detection and quantification of retinal nerve fiber layer

thickness in optic disc edema using stratus OCT. Archives of Ophthalmology. 2006;124(8):1111-7.

32. 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):462-5.

33. Kang S, Kim US. Using ImageJ to evaluate optic disc pallor in traumatic optic neuropathy. Korean J Ophthalmol. 2014;28(2):164-9.

34. Hoogewoud F, Butori P, Blanche P, Brezin AP. Cancer-associated retinopathy preceding the diagnosis of cancer. BMC Ophthalmol. 2018;18(1):285.

35. IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.

36. Menon V, Arya AV, Sharma P, Chhabra V. An aetiological profile of optic atrophy. Acta ophthalmologica. 1992;70(6):725-9.

37. Mbekeani JN, Fattah MA, Poulsen DM, Al Hazzaa S, Dababo MA, Eldali A, et al. Etiology of optic atrophy: a prospective observational study from Saudi Arabia. Annals of Saudi Medicine. 2017;37(3):232-9.

38. Wang I-H, Lin S-Y, Woung L-C, Shih Y-F, Jou J-R. Clinical prospective study of visual function in patients with acute optic neuritis. Journal of the Formosan Medical Association. 2013;112(2):87-92.

DATA SHEET

Name : Op/Pin no:

Age /sex: Date:

Address:

History :

Presenting Complaints: Duration :Laterality:

Associated symptoms: Duration:

H/O episodic blurring of vision

H/O Pain on eyeball movement

H/O Double vision, protrusion of eye ball

H/O Fever,jaw claudication

H/O Head ache

H/o motor weakness /T IA

H/O Paresthesia of face and body

H/O Vertigo

H/O Tremors

H/O Ataxia

H/O Weight loss/Malaise

H/O Neck pain/ Joint pain

H/O palpitation or breathlessness

H/o Headtrauma

H/O ear discharge

H/O bowel and bladder disturbances

Past history: DM/ SHT/ Dyslipidemia/ hyperthyroidism/CAD/ CVA/ trauma/ TB/ Similar episodes/syphilis

H/O Smoking or alcohol intake or drug intake

Occupational history

H/O similar illness in family

H/O previous medication intake

Ocular Examination (At 0,1,3 and 6 months)

Anterior segment examination: RE LE

Lids:

Conjunctiva :

Cornea:

Anterior Chamber :

Iris:

Pupil:

Lens:

EOM:

Ophthalmoscopic fundus examination :

Fundus photograph:

Visual acuity (uncorrected and best corrected):

IOP measurement:

Refraction:

Visual fields:

Colour vision:

Contrast sensitivity (in selected cases):

VEP (in selected cases) :

Systemic examination:

Pulse/BP/Pallor/icterus/lymphadenopathy

CVS/RS/CNS/ABDOMEN examination

Biochemical investigations

CBC With PS -

ESR -

LFT -

Lipid profile -

Random blood sugar -

FBS/PPBS -

RF,CRP -

MANTOUX ,SPUTUM AFB -

Serum T3,T4,TSH -

VCTC,VDRL -

Neuroimaging

CT/MRI Brain (if indicated) -

OTHERS (IF INDICATED) -

CHEST X-RAY,ECG -

ECHO -

CAROTID DOPPLER -

IMPRESSION:

CONSENT FORM

Hereby I volunteer and consent to participate in this study "**AN ETIOLOGICAL ANALYSIS OF PALE OPTIC DISC AND ITS CORRELATION WITH VISUAL OUTCOME IN PATIENTS ATTENDING A TERTIARY CARE HOSPITAL**".I was fully explained about the nature of this study by the doctor; knowing which I Mr/Ms/Mrs……. Fully consent to volunteer in this study.

Date: Signature of the volunteer

Place:

Signature of the witness

CONSENT FORM

I Dr. SATHYA PRIYA P is carrying out a study on the topic "**AN ETIOLOGICAL ANALYSIS OF PALE OPTIC DISC AND ITS CORRELATION WITH VISUAL OUTCOME IN PATIENTS ATTENDING A TERTIARY CARE HOSPITAL**".

My research project guide is **Dr.C.JEEVAKALA M.S.,D.O.,**

My research project is being carried out in the Department of Ophthalmology, Coimbatore Medical College Hospital, Coimbatore.

RESEARCH BEING DONE:

AN ANALYTICAL STUDY OF ETIOLOGICAL CAUSES OF PALE OPTIC DISC AND ITS VARIOUS VISUAL OUTCOME PRESENTATIONS.

PURPOSE OF RESEARCH:

To find out the cause of pale optic disc and prompt treatment of underlying disease prevents further vision loss, prevents systemic morbitidity and improves patient's quality of life.

PROCEDURE INVOLVED:

Detailed history including the past medical history is recorded. A comprehensive Opthalmological examination is done including visual acuity, colour vision, visual fields, slitlamp biomicroscopy, ophthalmoscopic examination, fundus photograph, Contrast sensitivity done. Biochemical investigations and neuroimaging are ordered when indicated to identify etiology and patients are followed up over a period of 6 months for visual outcome.

You, Shri./Smt./Kum. _____, aged_____ years

S/o/ W/o/D/o_____, residing at_____

_____ are requested to be a participant in the research study titled

“AN ETIOLOGICAL ANALYSIS OF PALE OPTIC DISC AND ITS CORRELATION WITH VISUAL OUTCOME IN PATIENTS ATTENDING A TERTIARY CARE HOSPITAL’in Government Coimbatore Medical College, Coimbatore. You satisfy eligibility criteria as per the inclusion criteria. You can ask any questions or seek any clarifications on the study that you may have before agreeing to participate.

DECLINE FROM PARTICIPATION

You are hereby made aware that participation in this study is purely voluntary and honorary and that you have the option and the right to decline from participation in the study.

PRIVACY AND CONFIDENTIALITY

You are hereby assured about your privacy. Privacy of subject will be respected and any information about you or provided by you during the study will be kept strictly confidential.

AUTHORIZATION TO PUBLISH RESULTS

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified; neither will your privacy be breached.

STATEMENT OF CONSENT

I, _____, do hereby volunteer and consent to participate in this study being conducted by Dr SATHYA PRIYA P I have read and understood the consent from/or it has been read and explained to me in my own language. The study has been fully explained to me, and whenever I ask questions at any time.

Date: Signature/Left Thumb Impression of the Volunteer

Date: Signature and Name of witness

ஒப்புதல் படிவம்

பெயர் : வயது :

பாலினம் :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் கண் மருத்துவத் துறையில் பட்டமேற்படிப்பு பயிலும் மாணவி **Dr. ப. சத்தியபிரியா** அவர்கள மேற்கொள்ளும் **"கண்பார்வை வட்டு நிறமிழப்புக்கான காரணங்களும் அதனுடன் தொடர்புடைய பார்வை விளைவுகளும்"** என்ற ஆய்வின செய்முறை தொடர்பான அனைத்து விபரங்களையும் கேட்டு எனது சந்தேகங்களைத் தெளிவுபடுத்திக் கொண்டேன்.

நான் இந்த ஆய்வில் என்னை பரிசோதனை செய்ய முழு மனதுடன் சுய சிந்தனையுடனும் சம்மதிக்கிறேன்.

எனது நோய் பற்றிய இந்த ஆய்வில் எங்களது அனைத்து விபரங்களும் பாதுகாக்கப்படுவதுடன் நோய் பகுதியின் புப்பைடம் மற்றும் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு.

நோயாளியின் கையொப்பம்

இடம் :

நாள் :

KEY TO MASTER CHART

RE	:	Right Eye
LE	:	Left Eye
BE	:	Both Eyes
SHT	:	Systemic Hypertension
CVA	:	Cerebrovascular Accident
DM	:	Diabetes Mellitus
TB	:	Tuberculosis
CP	:	Cerebropontine
M	:	Male
F	:	Female
+	:	Present
-	:	Absent
TFT	:	Thyroid Function Test
SOL	:	Space Occupying Lesion
EOM	:	Extraocular Muscle

DM	:	Demyelinating Plaques
ON	:	Optic Neuritis
TON	:	Traumatic Optic Neuropathy
TO	:	Tumour
DI	:	Drug Induced (Ethambutol)
POA	:	Primary Optic Atrophy
AION	:	Anterior Ischemic Optic Neuropathy
ONTT	:	Optic Neuritis Treatment Trial
ATT	:	Antitubercular Therapy
V.A	:	Visual Acuity

S.NO	NAME	AGE	SEX	DEFECTIVE VISION DURATION	EYE	RAPD	POSITIVE PAST HISTORY	V.A. RE	V.A.LE	C.V. RE	C.V. LE	FIELDS RE	FIELDS LE	C.S RE	C.S LE	DISC PALLOR RE	DISC PALLOR LE	BLOOD INVESTIGATIONS	CT BRAIN	MRI BRAINETIOLOGYFOLLOW UP	ETIOLOGY	TREATMENT GIVEN	FOLLOW UP AT 1 MONTH V.A	FOLLOW UP AT 3 MONTHS V.A	FOLLOW UP AT 6 MONTHS V.A.	VISUAL ACUITY IMPROVED OR NOT ATLEAST ONE LINE
1	BHAVANI	26	F	2 MONTHS	RE	+	OPTIC NEURITIS	RE 6/60	LE 6/6	-	+	-	+	-	+	RE:DIFFUSE DISC PALLOR	-	WNL	-	DP	RE:ON	ONTT	RE:6/12	RE: 6/12	RE: 6/12	IMPROVED
2	CHITRA	29	F	6 MONTHS	RE	+	OPTIC NEURITIS	RE 6/60	LE 6/6	-	+	-	+	-	+	+	-	WNL	-	DP	RE:ON	ONTT	RE V.A: 6/12	RE: 6/12	RE: 6/12	IMPROVED
3	DEVAKI	29	F	6 MONTHS	RE	+	OPTIC NEURITIS	RE 6/60	LE 6/6	-	+	-	+	-	+	+	-	WNL	-	DP	RE:ON	ONTT	RE V.A: 6/12	RE: 6/12	RE: 6/12	IMPROVED
4	ESWARI	32	F	2 YEARS	RE	+	OPTIC NEURITIS	RE:6/60	LE:6/9	-	+	-	+	-	+	+	-	WNL	-	DP	RE:ON	ONTT	RE:6/24	RE:6/24	RE:6/24	IMPROVED
5	DHIVYA	36	F	2 MONTHS	LE	+	OPTIC NEURITIS	RE:6/6	LE 6/60	+	-	+	-	+	-	-	+	WNL	-	DP	LE:ON	ONTT	LE V.A: 6/18	LE: 6/18	LE: 6/18	IMPROVED
6	DHARSHINI	35	F	2 MONTHS	RE	+	NIL	RE:6/60	LE:6/6	-	+	-	+	-	+	+	-	WNL	-	DP	RE:ON	ONTT	RE:6/60	RE:6/60	RE:6/60	NOT IMPROVED
7	GOPIKA	28	F	3 MONTHS	LE	+	NIL	RE:6/6	LE: 6/60	+	-	+	-	+	-	-	+	WNL	-	DP	LE:ON	ONTT	LE:6/18	LE:6/18	LE:6/18	IMPROVED
8	DHARSHINI	37	F	3 MONTHS	RE	+	NIL	RE:6/60	LE:6/6	-	+	-	+	-	+	+	-	WNL	-	DP	LE:ON	ONTT	RE:6/36	RE:6/36	RE:6/36	IMPROVED
9	SOWMIYA	39	F	6 MONTHS	RE	+	OPTIC NEURITIS	RE 6/60	LE 6/6	-	+	+	+	-	+	+	-	WNL	-	DP	RE:ON	ONTT	RE V.A: 6/18	RE: 6/18	RE: 6/18	IMPROVED
10	PAVITHRA	31	F	2 YEARS	BE	-	OPTIC NEURITIS	RE:6/60	LE:6/36	-	-	-	-	-	-	+	+	WNL	-	DP	BE:ON	ONTT	RE:6/60;LE: 6/36	RE:6/60;LE: 6/36	RE:6/60;LE: 6/36	NOT IMPROVED
11	GUNAVATHI	38	F	4 YEARS	BE	-	NIL	RE:6/36	LE:6/60	-	-	-	-	-	-	+	+	WNL	-	DP	BE:ON	ONTT	RE:6/36;LE:6/60	RE:6/36:LE:6/60	RE:6/36:LE:6/60	NOT IMPROVED
12	MANIKANDAN	28	M	ONE YEAR	LE	+	TRAUMA	RE:6/6	LE:6/60	+	-	+	-	+	-	-	+	WNL	FRACTURE OF FRONTAL BONE	-	LE:TON	POST I.V.STEROIDS	LE : 6/36	LE: 6/36	LE: 6/36	IMPROVED
13	SHANKAR	32	M	6 MONTHS	RE	+	TRAUMA	RE:4/60	LE:6/6	-	+	-	+	-	+	+	-	WNL	FRACTURE OF OPTIC CANAL	-	RE:TON	POST I.V.STEROIDS	RE V.A: 4/60	RE: 4/60	RE: 4/60	NOT IMPROVED
14	RAJENDRAN	28	M	ONE YEAR	LE	+	TRAUMA	RE:6/6	LE:6/60	+	-	+	-	+	-	-	+	WNL	FRACTURE OF FRONTAL BONE	-	LE:TON	POST I.V.STEROIDS	LE : 6/60	LE: 6/60	LE: 6/60	NOT IMPROVED
15	PERUMAL	32	M	6 MONTHS	RE	+	TRAUMA	RE:5/60	LE:6/6	-	+	-	+	-	+	+	-	WNL	FRACTURE OF OPTIC CANAL	-	RE:TON	POST I.V.STEROIDS	RE V.A: 5/60	RE: 5/60	RE: 5/60	NOT IMPROVED
16	ASHOK	28	M	2 YEARS	RE	+	TRAUMA	RE:6/60	LE: 6/6	+	+	-	+	-	+	+	-	WNL	FRACTURE OF FRONTAL BONE	-	RE:TON	POST I.V.STEROIDS	RE:6/60	RLE:6/60	RE:6/60	NOT IMPROVED
17	DAVID	28	M	ONE YEAR	LE	+	TRAUMA	RE:6/6	LE:6/60	+	-	+	-	+	-	-	+	WNL	FRACTURE OF FRONTAL BONE	-	LE:TON	POSTI.V.STEROIDS	LE : 6/60	LE: 6/60	LE: 6/60	NOT IMPROVED
18	SARAVANAN	32	M	6 MONTHS	RE	+	TRAUMA	RE:4/60	LE:6/6	-	+	-	+	-	+	+	-	WNL	FRACTURE OF OPTIC CANAL	-	RE:TON	POST I.V.STEROIDS	RE V.A: 6/60	RE: 6/60	RE: 6/60	IMPROVED
19	RAJESH	30	M	6 MONTHS	LE	+	TRAUMA	RE:6/6	LE: 6/60	+	-	+	-	+	-	-	+	WNL	FRACTURE IN TEMPORAL BONE	-	LE:TON	POST I.V.STEROIDS	LE:6/60	LE:6/60	LE:6/60	NOT IMPROVED
20	GANDHIMATHI	50	F	10 YEARS	RE	+	TRAUMA	RE:6/60	LE:6/9	-	+	-	+	-	+	+	-	WNL	FRACTURE IN FRONTAL BONE	-	RE:TON	POST I.V.STEROIDS	RE:6/60	RE:6/60	RE:6/60	NOT IMPROVED
21	MUTHUSAMY	30	M	3 MONTHS	LE	+	TRAUMA	RE:6/6	LE:6/60	+	-	+	-	+	-	-	+	WNL	FRACTURE IN FRONTAL BONE	-	LE:TON	POST I.V.STEROIDS	LE:6/60	LE:6/60	LE:6/60	NOT IMPROVED
22	MAHESHWARAN	22	M	2 MONTHS	LE	+	TRAUMA	RE:6/6	LE:6/36	+	-	+	-	+	-	-	+	WNL	FRACTURE IN FRONTAL BONE	-	LE:TON	POST I.V.STEROIDS	LE:6/24	LE:6/24	LE:6/24	NOT IMPROVED
23	KANDHASAMY	53	M	2 YEARS	LE	+	TRAUMA	RE:6/36	LE:5/60	+	-	+	-	+	+	-	+	WNL	FRACTURE IN FRONTAL BONE	-	LE:TON	POST I.V.STEROIDS	LE:6/60	LE:6/60	LE:6/60	IMPROVED
24	ARAVIND	35	M	ONE YEAR	LE	+	TRAUMA	RE:6/6	LE:6/36	+	-	+	-	+	-	-	+	WNL	FRACTURE OF FRONTAL BONE	-	LE:TON	POST I.V.STEROIDS	LE:6/36	LE:6/36	LE:6/36	NOT IMPROVED
25	GOKILA	45	F	1 MONTH	RE	+	TRAUMA	RE:6/36	LE:6/6	-	+	-	+	-	+	+	-	WNL	FRACTURE IN FRONTAL BONE	-	RE:TON	POST I.V.STEROIDS	RE: 6/36	RE: 6/36	RE: 6/36	NOT IMPROVED
26	KUMAR	30	M	25 YEARS	BE	-	SINCE CHILDHOOD	RE 2/60	LE 3/60	-	-	-	-	-	-	+	+	WNL	-	-	BE:POA	OBSERVATION	RE:2/60,le2/60	RE:2/50;LE:2/60	RE:2/60;LE:2/60	NOT IMPROVED

27	PRIYA	35	F	30 YEARS	BE	-	NIL	RE:4/60	LE: 3/60	-	-	-	-	-	-	+	+	WNL	-	-	BE:POA	OBSERVATION	RE:4/60;LE:3/60	RE:4/60;LE:3/60	RE:4/60;LE:3/60	NOT IMPROVED
28	DEV	40	M	25 YEARS	BE	-	NIL	RE:6/60	LE:5/60	-	-	-	-	-		+	+	WNL	-	-	BE:POA	OBSERVATION	RE: 6/60;LE:5/60	RE:6/60;LE:5/60	RE:6/60;LE:5/60	NOT IMPROVED
29	SURYA	30	M	20 YEARS	BE	-	SINCE CHILDHOOD	RE 2/60	LE 3/60	-	-	-	-	-	-	+	+	WNL	-	-	BE:POA	OBSERVATION	RE:2/60;LE:3/60	RE:2/60;LE:3/60	RE:2/60;LE:3/60	NOT IMPROVED
30	SIVAKUMAR	48	M	30 YEARS	BE	-	SHT/CVA	RE:6/60	LE:6/60	-	-	-	-	-	-	+	+	HT CARDIOMYOPATHY	-	-	BE:POA	OBSERVATION	RE:6/60;LE:6/60	RE:6/60;LE:6/60	RE:6/60;LE:6/60	NOT IMPROVED
31	MANI	35	M	20 YEARS	BE	-	NIL	RE:6/60	LE: 5/60	-	-	-	-	-	-	+	+	WNL	-	-	BE:POA	OBSERVATION	RE:6/60;LE:5/60	RE:6/60;LE:5/60	RE:6/60;LE:5/60	NOT IMPROVED
32	BASKAR	35	M	20 YEARS	BE	-	NIL	RE:6/60	LE:6/60	-	-	-	-	-	-	+	+	WNL	-	-	BE:POA	OBSERVATION	RE:6/60;LE:6/60	RE:6/60;LE:6/60	RE:6/60;LE:6/60	NOT IMPROVED
33	ANANDBABU	40	M	30 YEARS	BE	-	NIL	RE:5/60	LE:5/60	-	-	-	-	-	-	+	+	WNL	-	-	BE:POA	OBSERVATION	RE:5/60;LE:5/60	RE:5/60;LE:5/60	RE:5/60;LE:5/60	NOT IMPROVED
34	MANIKKAM	49	M	ONE YEAR	LE	+	NIL	RE 6/9	LE 6/60	+	-	-	-	+	-	-	+	WNL	PARIETAL SOL	-	LE:TO	EXCISION	LE V.A : 6/36	LE: 6/36	LE: 6/36	IMPROVED
35	SAKAYAM	55	M	1 YEAR	RE	+	SHT/DM	RE: 6/60	LE:6/18	-	+	+	-	-	+	+	-	WNL	PARIETAL SOL	-	RE:TO	EXCISION	RE:6/36	RE:6/36	RE:6/36	IMPROVED
36	PALANISAMY	66	M	1 YEAR	RE	+	SHT/DM	RE:6/60	LE:6/12	-	+	-	+	-	+	+	-	WNL	PARIETAL SOL	PARIETAL SOL	RE:TO	EXCISION	RE:6/36	RE:6/36	RE:6/36	IMPROVED
37	SARASAMMAL	65	F	6 MONTHS	LE	+	NIL	RE:6/36	LE:6/60	+	-	+	-	+	-	-	+	WNL	-	PINEAL ASTROCYTOMA	LE:TO	EXCISION	LE:6/60	LE:6/60	LE:6/60	NOT IMPROVED
38	JHANSIRANI	32	F	2 YEARS	RE	+	CP ANGLE TUMOUR	RE:6/36	LE:6/6	-	+	-	+	-	+	+	-	WNL	-	SHUNTING SEEN	RE:TO	POST EXCISION	RE:6/18:	RE:6/18	RE:6/18	IMPROVED
39	UMAMAHESHWARI	38	F	1 YEAR	BE	-	HEADACHE	RE:6/12	LE:6/12	+	-	+	-	+	-	-	+	WNL	-	LESION IN PITUITARY REGION	BE:TO	RADIATION	LE:6/12	LE:6/12	LE:6/12	NOT IMPROVED
40	ARUMUGAM	65	M	2 YEARS	LE	+	SHT	RE:6/36	LE:2/60	+	-	+	-	+	-	-	+	WNL	PITUITARY TUMOUR	-	RE:TO	EXCISION	RE:6/36;LE:2/60	RE:6/36;LE:2/60	RE:6/36;LE:2/60	NOT IMPROVED
41	SARASWATHI	33	F	2 YEARS	RE	+	CP ANGLE TUMOUR	RE:6/18	LE:6/9	-	+	-	+	-	+	+	-	WNL	-	SHUNTING SEEN	LE:TO	POST EXCISION	RE:6/18	RE:6/18	RE:6/18	NOT IMPROVED
42	GIRIJA	35	F	2 YEARS	RE	+	POST SURGERY	RE:6/36	LE:6/6	-	+	-	+	-	+	+	-	WNL	SHUNTING	-	RE: TO	POST EXCISION	RE:6/18;	RE:6/18	RE:6/18	IMPROVED
43	SOUNDAPPAN	65	M	2 YEARS	RE	+	SHT/CVA/DM	RE:4/60	LE:6/36	-	+	-	+	-	+	+	-	NIL	PARIETAL SOL	PARIETAL SOL	RE:TO	EXCISION	RE:6/60	RE:6/60	RE:6/60	IMPROVED
44	MANIKANDAN	21	M	4 YEARS	LE	+	LACRIMAL GLAND TUMOUR	RE:6/6	LE:2/60	+	-	+	-	+	-	-	+	NIL	LACRIMAL GLAND TUMOR INVILVING ORBITAL APEX	LACRIMAL GLAND TUMOR INVILVING ORBITAL APEX	LE:TO	POST DECOMPRESSION STATUS	LE:2/60	LE:2/60	LE:2/60	NOT IMPROVED
45	ANN GEORGE	45	F	3 MONTHS WITH PROPTOSIS	LE	+	THYROID ORBITOPATHY	RE:6/6	LE:6/60	+	-	+	-	+	-	-	+	TFT WITHIN CONTROL	-	INCREASED EOM THICKNESS	LE:INFLAMMATORY OPTIC ATROPHY	POST DECOMPRESSION STATUS	LE:6/60	LE:6/60	LE:6/60	NOT IMPROVED
46	MUTHUPANDI	28	M	3 YEARS	BE	-	ALCOHOLIC	RE:6/36	LE:6/36	-	-	-	-	-	-	+	+	WNL	-	-	BE:TOXIC	CONTROL OF ALCOHOL	RE:6/36;LE:6/36	RE:6/36;LE:6/36	RE:6/36;LE:6/36	NOT IMPROVED
47	PETCHIAMMAL	55	F	2 YEARS	BE	+	TB BEFORE 2 YEARS	RE:6/36	LE:6/36	-	-	-	-	-	-	+	+	CHEST XRAY : CAVITY IN RT LUNG BASE	-	-	BE:DI	OBSERVATION;STOP OF ATT	RE:6/36;LE:6/36	RE:6/36;LE:6/36	RE:6/36;LE:6/36	NOT IMPROVED
48	VIJAYALAKSHMI	38	F	3 YEARS	BE	-	POST TB MENINGITIS	RE:4/60	LE:6/60	-	-	-	-	-	-	+	+	SPUTOM AFB POSITIVE	-	-	BE:POST TB MENINGITIS;DI	OBSERVATION	RE:4/60;LE:6/60	RE:4/60;LE:6/60	RE:4/60;LE:6/60	NOT IMPROVED
49	SHIVA	63	M	4 YEARS	BE	-	SHT	RE:6/18	LE:6/36	-	-	-	-	-	-	+	+	WNL	-	-	BE:POST PAPILLOEDEMA	CONTROL OF SHT	RE:6/18;LE:6/36	RE:6/18:LE:6/36	RE:6/18:LE:6/36	NOT IMPROVED
50	NAVEEN	55	M	2 YEARS	LE	+	SHT/HYPERLIPIDEMIA	RE:6/18	LE: 5/60	+	-	+	-	+	-	-	+	INCREASED LIPID PROFILE	-	-	LE:POST AION	CONTROL OF SHT/HYPERLIPIDEMIA	LE:5/60	LE:5/60	LE:5/60	NOT IMPROVED