



ARKA JAIN
University
Jharkhand (Jamshedpur)



PROJECT REPORT

**“ COLOUR VISION DEFICIENCY AND ITS ASSOCIATION
WITH SEVERITY OF GLAUCOMA”**

**DISSERTATION SUBMITTED TO ARKA JAIN
UNIVERSITY, JAMSHEDPUR IN PARTIAL FULFILLMENT
OF THE REQUIREMENT FOR THE AWARD OF THE
DEGREE OF BACHELOR OF CLINICAL OPTOMETRY**

SUBMITTED BY

SHABISTAN AFSHAN

BACHELOR OF OPTOMETRY 4th YEAR

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UNDERTAKING

I confirm that this record drawn up by me is an accurate record of the work I have undertaken.

Student: SHABISTAN AFSHAN

Date: 16.04.22

I confirmed that I have examined the data and approved it as being an accurate record to the best of my knowledge.



MR.APOORV SHARMA.

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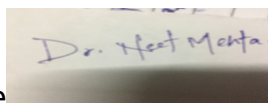
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Co-Guide

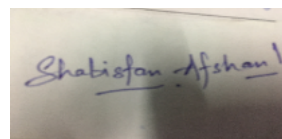
DECLARATION

I hereby declare that the thesis entitled "**COLOUR VISION DEFICIENCY AND ITS ASSOCIATION WITH SEVERITY OF GLAUCOMA**" submitted by me ,for the award of the degree of Bachelor in optometry to Arka Jain University is a recorded of bonafide work carried out by me at LV prasad eye institute,Hyderabad .The project work was carried out ,under the supervision of **MR. Apoorv sharma** (Consultant optometrist) as the external supervisor and Internal guide **MR.Sarbojeet goswami** (Programme coordinator school of allied and health science.

I further declare that the work reported in this thesis has not been submitted and will not be submitted either in part or in full for the award of any other degree or diploma in this institute or any other institute or university.

Place: HYDERABAD

Date: 16.04.22

A photograph of a handwritten signature in blue ink on a white surface. The signature reads "Shabistan Afshan" with a horizontal line underneath the name.

Signature of the Candidate

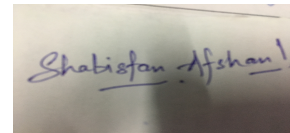
CERTIFICATE

This is to certify that that the thesis entitled “**COLOUR VISION DEFICIENCY AND ITS ASSOCIATION WITH SEVERITY OF GLAUCOMA**” submitted by **Shabistan Afshan**, for the award of the degree of Bachelor in optometry, is a record of *bonafide* work carried out by the student under my supervision, at LV prasad eye institute, Hyderabad as per the academic code of the University.

The contents of this report have not been submitted and will not be submitted either in part or in full, for the award of any other degree or diploma in this institute or any other Institute or University. The thesis fulfills the requirements and regulations of the University and in my opinion meets the necessary standards for submission

Date: 16.04.22
Candidate

Signature of the

A rectangular box containing a handwritten signature in blue ink that reads "Shabistan Afshan".

External Guide

A handwritten signature in blue ink, appearing to read "Aparna Sharma".

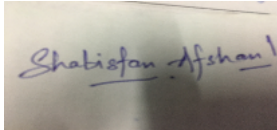
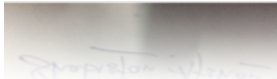
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This is to certify that the thesis entitled "**COLOUR VISION DEFICIENCY AND ITS ASSOCIATION WITH SEVERITY OF GLAUCOMA**" submitted by **Shabistan Afshan** or the award of the degree of **Bachelor in optometry** is a record of *bonafide* work carried out by the student under my supervision, at LV prasad eye institute, Hyderabad as per the academic code of the University.

The contents of this report have not been submitted and will not be submitted either in part or in full, for the award of any other degree or diploma in this institute or any other Institute or University. The thesis fulfills the requirements and regulations of the University and in my opinion meets the necessary standards for submission.

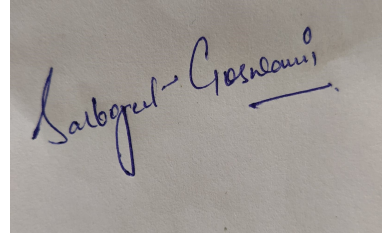
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(Internal Guide)



ACKNOWLEDGEMENTS

I would like to express my sincere thanks to my project guide MR. APOORV SHARMA for his vital support, invaluable guidance, constant encouragement, constructive comments, sympathetic attitude and immense motivation, which has sustained my efforts at all stages of this project work.

I am also thankful to DR..Neet mehta and all the people who help me to carry out this project work successfully and for their valuable advice and support, which I received from time to time.

Let me not forget to give a special thanks to my friends, colleagues and library staff for their helpful attitude, and most important, for all the patients for their kind cooperation.

Thanking You

MS. SHABISTAN AFSHAN

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ABSTRACT

Purpose: To study colour vision deficiency in patient which is having glaucomatous damage and severity of glaucoma.

Methods: In this prospective study we evaluated 102 patient having a Glaucoma and Visual field defect is occur and distinguish the colour vision defect were studied by using a HRR (Hardy Rand and Rittler) pseudo isochromatic test plates with visual field analysis/defect detect by Humphrey visual field 30-2 programme.

Results: In 404 eyes of 102 patient having observed a colour vision deficiency and severity of glaucoma in different age group and analyse that the mostly the patients is having a advanced glaucomatous damage occur red-green or blue-yellow color vision deficiency.

Conclusion: In this study we conclude that the glaucoma is also having a cause of color vision changes in patients. At the age of 38-50 having a early damage and almost normal colour vision age group 51-65 having a moderate damage and occur blue yellow deficiency age group 66-85 having a advanced damage occur blue-yellow, red-green deficiency.

Data analysis of colour vision defects provides the possibility of colour vision changes and can prove a useful mean for detecting the severity of Glaucoma.

Keyword: Colour vision defect, Glaucoma, deficiency.

INTRODUCTION

Glaucoma is a progressive optic neuropathy with raised with Intraocular pressure associated with optic nerve change and visual field loss In worldwide glaucoma is postulated to the 3rd leading cause of blindness .If the damage is worsened glaucoma can cause permanent vision loss or even total blindness within a few years. Risk factor included

- Having a family history of glaucoma.
- Having any optic nerve disease.
- Having high myopic.
- Having high blood pressure and Diabetes.
- Cornea is thinner than usual.
- Use of topical or systemic corticosteroid.

Mostly in glaucoma the nerve connecting the eye to the brain is damaged, usually

due to high pressure in eye

Glaucoma is often called the silent thief of sight because most patients are unaware that they have the disease until they have experienced visual changes and vision loss. The patient may not seek health care until he or she experienced blurred vision, halos around light, difficulty in focusing, loss of peripheral vision, colour vision deficiency and headache

Colour vision deficiency in glaucoma was first described in 1883. Any ocular condition that damage the retina or effect particular areas of the eyes can lead to colour blindness. Colour vision is possible due to photoreceptor in retina of the eyes known as cones. Cones have light sensitive pigments found in macula. Cones cells contains three form of opsin resulting in trichromatic colour vision. Each cone is sensitive to red, green or blue light. The all three types of cone cell have a different sensitivity to light wavelength. One type of cone perceived blue light. Second perceived green and third perceived red light. The red green and blue all work together and allowing you to see the whole spectrum of colour⁵ Cones have high density at the fovea and low density in the rest of the retina. The colour information is mostly taken as fovea. Visual information is then sent to the brain from retinal ganglion cell via the optic nerve to the optic chiasma.

After the optic chiasma the visual tract are referred to as optic tract which enter the lateral geniculate body. The lateral geniculate body divided into 2 types Magnocellular (M) cells and parvocellular (P) cells. The magnocellular cells carries information about large, fast things and his colour blind. The parvocellular pathway carries information about small, slow and colourful things. M and P cells received balanced input from both large and medium cones throughout most of the retina. The koniocellular retina received Axon from the small bistratified ganglion cell. After synapsing at the later geniculate body, The visual tract continues on back to the primary visual cortex located at the back of the brain within the occipital lobe.

REVIEW OF LITERATURE

•The first study in colour vision deficiency in glaucoma was first described by 1883 and although many early investigation indicated that red green defects accompanied glaucomatous optic neuropathy, later studies suggested that Tristram defect is predominant.

•The another study done by DF Edger in 1999 colour vision defect associated with ocular disease have been reported since 17th century. Kollner in 1912 wrote an acute description in the progressive nature of colour vision loss secondary to ocular disease. They dividing defect into "Blue yellow" and progressive "Red green" blindness. This classification has become kollner's rule include some optic nerve disease. Notably glaucoma which are primarily associated with blue yellow defect

and also some retinal disorder such as central cone degeneration which may result in red-green defect.

•This study done by P.papconstantinor at 2009 in Acquired color vision defect and visual field loss in a patient with early glaucoma stage. He concluded that the color vision deficiency seems to precedes glaucomatous defect in HVF test that play an important role as a predictive factor for developing glaucomatous changes in future. Glaucoma related color vision defect affect mainly blue-yellow stimuli.

AIM AND OBJECTIVES

AIM OF STUDY

- To study colour vision deficiency in recent and advanced Glaucoma patients and its visual field damage.
- To understand the type of colour vision defect associated with Glaucomatous changes.

- The aim of study was to determine whether there is correlation between the manifestation of colour vision defect and glaucomatous visual field loss.

OBJECTIVE OF STUDY

- * To assess the colour vision defect in early and advanced Glaucoma patients and its visual fields defect.
- * To assess color vision deficiency in a patient which is having glaucomatous damage and severity of glaucoma.

MATERIALS

Place of study: L V Prasad eye Institute, Hyderabad

Study design: Database cross sectional study

Study duration: 2nd November, 2021 to 18th April, 2022

Sample size: 102 patients (male & female)

Inclusion criteria:

- 1) Age is greater than 38.
- 2) Intraocular pressure is greater than 08.
- 3) Doing HVF on same day.

METHODOLOGY

This prospective study was conducted on the subject with age group 38 to 85 Years old patients.

The prospective study involved Glaucoma suspects and Glaucoma patients at the age group 38 to 85 years old male and female, all of these patients had used Anti-glaucoma medication.

The exclusive criteria include –

- Congenital colour vision defect
- Lack of ability for scheduled follow up

The aim of study was to determine whether there is correlation between the manifestation of colour vision defect and glaucomatous visual field loss.

All patients were examined and follow up account to protocol involved –

- Taking medical history
- Measurement of visual acuity
- Taking colour vision
- Slit lamp examination
- IOP measurement
- Gonioscopy
- Fundoscopy
- HVF test

Taking medical history: A throughout past medical and ophthalmic history included any systemic disease, any kind of head or ocular injury, recently diagnosed any disease, any family history or previous ophthalmic surgery or any eye disorder.

Measurement of visual acuity : Visual acuity was tested by Snellen chart at the distance of 6 meters binocularly and near visual acuity was tested by jaeger chart at the distance of 25cm binocularly.

Taking color vision: Colour vision testing is done by HRR plates binocularly.

Slit lamp examination: Slit lamp examination was performed with a Haag-Streit 900 slit-lamp.

The cornea was examined for any anomalies (pterygium, corneal scarring, etc).the anterior chamber depth was evaluated ,iris was examined for local atrophies, colobomas, neovascularization, presence of pseudo exfoliation material, irregularities in the pupil and the transparency of crystalline lens.

Intraocular pressure measurement: Intraocular pressure was measured by GAT tonometry .

Gonioscopy: Gonioscopy was performed on the slit lamp using the indirect Goldmann gonioscope. Patient with narrow angle, open angle or congenital dysgenesias of the anterior chamber's angle .

Fundoscopy: The cup disc ratio of the optic nerve head was studied thoroughly and all patients included in the study had horizontal and vertical cup disc ratio greater than 0.2

HVF: We used automated perimetry with a 30-2 programme of the Humphrey perimeter .It was necessary there was good patient reliability in order to evaluate the HVF test and in case of low patient reliability, the test had to be repeated.

DATA ANALYSIS

TABLE 1:

Pt age group	38-50 Years	51-65 Years	66-85 Years
No. of patient	29	38	35
Color vision plates (HRR)	Pass in all plates (1-10)	Failed in B-Y plates (5-6)	Failed in R-G & B-Y plates (1-10)
% of damage	20%	55%	90%
Visual field damage	Normal/Early damage	Moderate damage	Advanced damage
Types of color vision defect	Trichromats (Normal)	Tritanopia	Achromatopsia

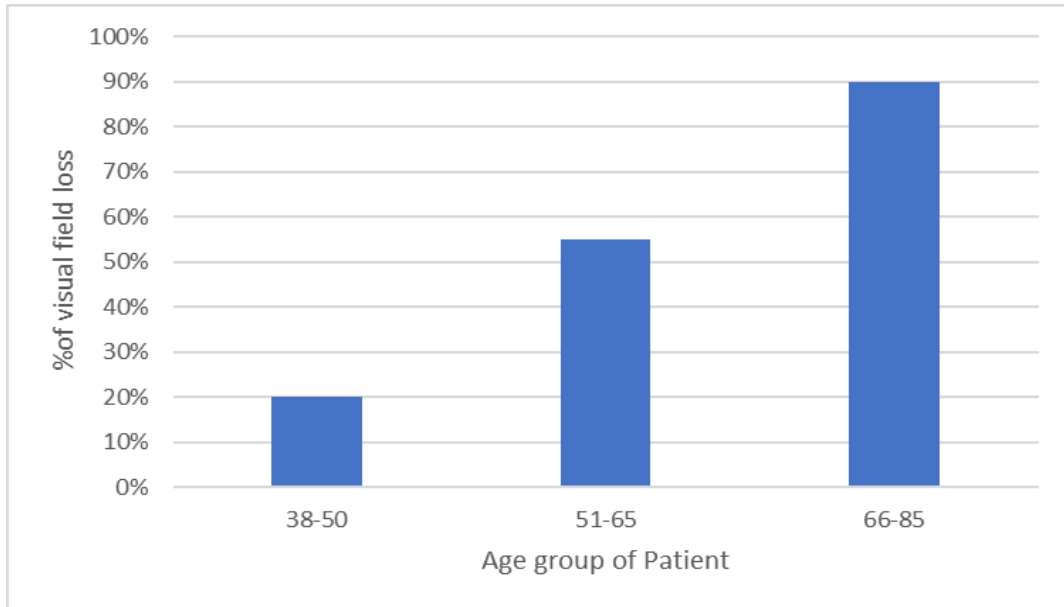
We have differentiate age into 3 groups i.e, 38-50, 51-65 & 66-85 and in that

- 1st age group patient read all 1-10 HRR color test plates properly and they have only 20% damage in visual field and these patients are normal in color vision and noted normal/early damage in visual field
- 2nd age group patient read all plates properly but somewhere confused in 5-6 plates(B-Y defect) and they have 55% damage in visual field and these patients are Tritanopia and noted moderate damage in visual field.
- 3rd age group patients read only demo plates these patients are failed in color plates and these patient have 90% or more than 90% are damaged in visual field and these patients are Achromatopsia (B-Y & R-G defect).

RESULT

TABLE 1:

Pt age group	38-50 Years	51-65 Years	66-85 Years
% of damage	20%	55%	90%
Visual field damage	Early field damage	Moderate damage	Advanced damage



Here we have shown that how much visual field loss in different age groups-

- In 38-50 age group of patient have damage 20% of visual field.
- In 51-65 age group of patient have damage 55% of visual field.
- In 66-85 age group of patient have damage 90% of visual field.

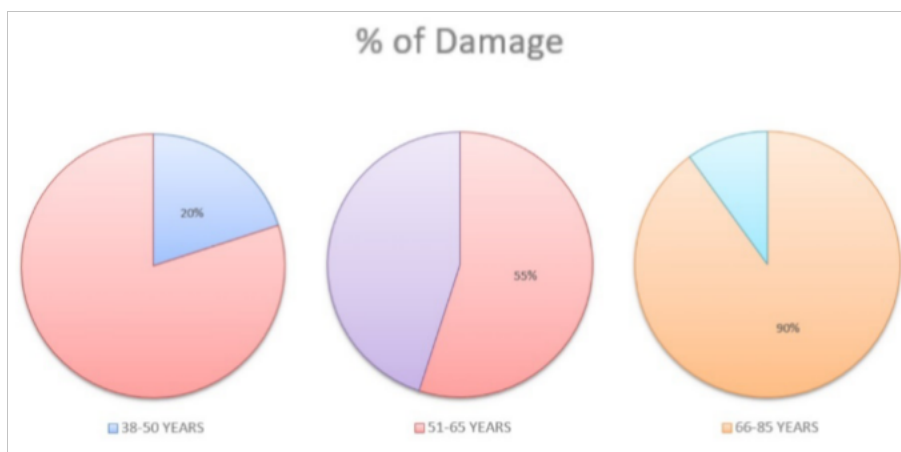
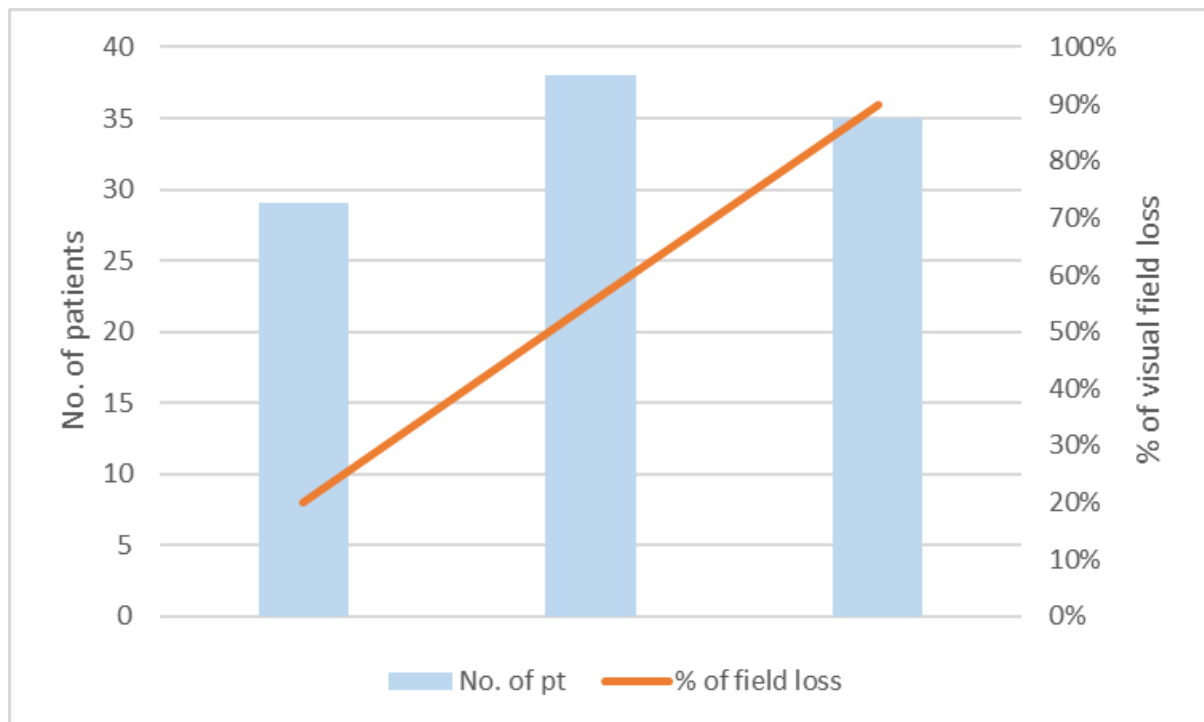


TABLE 2:

Pt age group	38-50 Years	51-65 Years	66-85 Years
No. of patient	29	38	35

% of damage	20%	55%	90%
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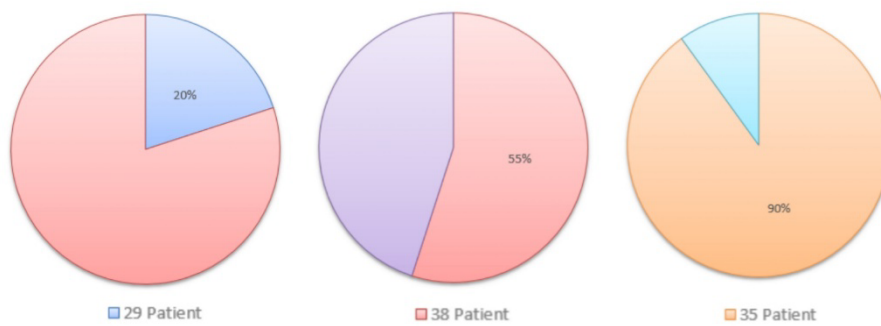
Here we have shown the number of patients in different age group and how much percentage of field loss or visual field damage-

- In 1st column 1st age group of patients are 29 in numbers and they have 20% of visual field damage.
- In 2nd column 2nd age group of patients are 38 in numbers and they have 55% of visual field damage.
- In 3rd column 3rd age group of patients are 35 in numbers and they have 90% of visual field damage.

The graphical representation of the patient in colour vision illustrate that the fact of predominant colour vision deficiency and severity of glaucoma in certain age of the

patients. The (90%) patients having age group 66-85 is having a (Red-Green, Blue-Yellow) defect and noted advanced glaucomatous changes while (55%) patients having age group 51-65 is having a (Blue-Yellow) defect and noted moderate glaucomatous changes and only (20%) of the having age group 38-50 is having normal colour vision and noted early glaucomatous changes.

% of Damage



DISCUSSION

This result can be attributed that the prevalence of color vision defect is reported to be approx 90% of that patient who is having a advanced glaucomatous changes seen in HVF.

In our study we took into consideration the known influence of age and existence of glaucoma in elderly patient.

One of the limitation of our study is that we used HRR plates as a screening tool and HRR cannot distinguish between anomalous Trichromats and dichromats. All the previous result confirm that the fact that colour vision deficiency precede the glaucomatous changes and obviously are a strong predictive factor for the future development of glaucoma.

The defect in other psychophysical function, including color vision deficiet, reduced contrast sensitivity, loss of pattern discrimination, and reduction of movement discrimination which appeared prior to HVF defect ,strengthened the suggestion that these general damages occur before the onset of glaucomatous changes in visual field testing.

Thus, we can conclude that the functional deficit of color perception in glaucoma patients can be traced and developed in time, underlying a progressive qualitative or quantitative damage of retinal ganglion cells, which can be quantitatively evaluated by testing color vision perception.

CONCLUSION

In this study we conclude that the colour vision deficiency are mostly present on that patient who is having a advanced field damage. At the age of 38-50 most of the patient having early glaucomatous changes are noted a normal color vision at the age of 51-65 age group of the patient having a moderate field damage noted a blue-yellow deficiency and the age group 66-85 having a advanced field damage noted a red-green or blue-yellow deficiency.

- 1) Color vision deficiency seems to precede glaucomatous defect in HVF test, a fact that can play an important role as a predictive factor for developing glaucomatous changes in the future.
- 2) Glaucoma related color vision defect affect mainly the blue-yellow stimuli.
- 3) Blue yellow deficiency generally are associated with early glaucoma and red green deficiency are associated with advanced glaucoma.

Keyword: HVF, Glaucoma, Colour Vision Deficiency, Age Group, red green-blue yellow deficiency.

FOOTNOTES

Disclosure

The authors (Shabistan Afshan) report no conflicts of interest in this work.

APPENDIX

Sl. No.	MR.no	AGE	DIAGNOSIS	VISUAL ACUITY		INTRAOCULAR PRESSURE		5-6 Plates	7-10 Plates	5-6 plates	7-10 Plates	HVf damage
				OD-20/30	OS-20/60	OD-14	OS-17	OD-Pass	Pass	OS-Pass	Fail	
1	P1382354	62	RE-PAC, LE-PACG	OD-20/30	OS-20/60	OD-14	OS-17	OD-Pass	Pass	OS-Pass	Fail	BE-WNL
			BE.Nuclear cataract									
2	P929474	84	BE-PAC,PACG,PXF.	OD-20/80	OS-20/800	OD-15	OS-17	OD-Fail	Fail	OS-Fail	Fail	RE-Advanced disc damage.
			RE-Trab done									
3	P1409563	78	BE-Glaucoma in	OD-20/25	OS-20/25	OD-10	OS-10	OD-Fail	Fail	OS-Pass	Fail	BE Advanced disc damage, field loss
			pseudophakia.									
4	P1297043	74	RE-POAG,LE-PXF	OD-20/60	OS-20/160	OD-12	OS-14	OD-Fail	Fail	OS-Fail	Fail	BE -Advanced disc damage, field loss
			BE-Total glaucomatous atrophy.									
5	P1417964	46	RE-PACG, LE-PAC	OD-20/30	OS-20/20	OD-14	OS-18	OD-Pass	Fail	OS-Pass	Pass	BE-WNL
			BE-Yag PI done									
6	P1415782	60	RE-Disc suspect,	OD-20/50	OS-20/30	OD-16	OS-16	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
			RE-Senile cataract									
7	P1226534	63	RE-Angle closure	OD-20/250	OS-20/25	OD-11	OS-10	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
			RE-DSEAK done									
8	P1418928	59	BE-Disc suspect	OD-20/30	OS-20/25	OD-09	OS-10	OD-Pass	Pass	OS-Pass	Pass	BE-WNL.
9	P1416801	65	BE-PAC.Cataract	OD-20/25	OS-20/20	OD-24	OS-27	OD-Pass	Pass	OS-Pass	Pass	BE-WNL.
			BE-Yag PI done.									
10	P1376434	61	BE-PACG,Cataract	OD-20/40	OS-20/40	OD-15	OS-15	OD-Pass	Pass	OS-Pass	Pass	BE-WNL

			BE-Yag PI done									
11	P1404807	72	BE-PACG,Cataract	OD-20/320	OS-20/40	OD-10	OS-10	OD-Fail	Fail	OS-Pass	Fail	RE-Advanced disc damage, field damage
			BE-Yag PI done.									LE-Early damage
12	N216384	46	BE-PCG,Nystagmus	OD-20/600	OS-20/800	OD-05	OS-18	OD-Fail	Fail	OS-fail	Fail	BE-Advanced field loss
			Megalocornea									
			RE-Trab done									
13	P1403798	65	BE-POAG,NTG	OD-20/30	OS-20/30	OD-14	OS-14	OD-Pass	pass	OS-Pass	Pass	BE-WNL.
			Early cataract									
14	P795576	56	BE-Disc suspect	OD-20/20	OS-20/25	OD-16	OS-18	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
15	P669122	65	BE-POAG,RE-PXF	OD-20/25	OS-HM+	OD-12	OS-46	OD-Pass	Fail	OS-Fail	fail	BE-Advance damage
			RE-Trab done									
16	P1410504	65	BE-PXF,Cataract	OD-20/60	OS-20/60	OD-12	OS-12	OD-Pass	Fail	OS-Pass	Fail	BE-Moderate field loss
17	P1418834	45	BE-PACS,PI done	OD-20/30	OS-20/30	OD-15	OS-17	OD-Pass	Pass	OS-Pass	Fail	BE-WNL
			Plateau iris syndrome									
18	P1420658	46	BE-Primary develop									
			mentol glaucoma,	OD-20/160	OS-20/30	OD-13	OS-12	OD-Fail	fail	OS-Pass	Pass	RE-Total field damage.
			Nystagmus,RE-Optic									
			atrophy.									
19	P1030072	63	BE-Disc suspect	OD-20/30	OS-20/30	OD-21	OS-22	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
20	P979833	54	RE-PACG,LE-PAC	OD-NPL	OS-20/40	OD-44	OS-12	OD-Fail	Fail	OS-Pass	Pass	LE-WNL
			BE-Yag PI done									
21	P1396431	42	BE-PACG,PI done	OD-20/50	OS-PL+PR	OD-18	OS-22	OD-Pass	Fail	OS-Fail	Fail	BE-Moderate field damage
22	P758602	46	BE-POAG,Pseudo	OD-20/30	OS-20/25	OD-12	OS-09	OD-Pass	Fail	OS-Fail	Fail	BE-Moderate field damage
			phakia									
23	N161957	45	BE-PACG,PI done	OD-20/125	OS-20/40	OD-10	OS-07	OD-Pass	Pass	OS-Pass	Pass	BE-Low reliability indices
24	P1395227	42	BE-PAC,PI done	OD-20/25	OS-20/25	OD-18	OS-15	OD-Pass	Pass	OS-Pass	Pass	BE-WNL.
25	PI395259	55	BE-PAC,PI done	OD-20/40	OS-20/30	OD-14	OS-16	OD-Fail	Fail	OS-Pass	Pass	RE-Very advanced disc damage.
26	P1213978	42	LE-Glaucomatoud	OD-20/20	OS-20/20	OD-12	OS-12	OD-Pass	Pass	OS-Fail	Fail	RE-Early damage,LE-Advanced damage
			matous Damage									
27	P1381747	43	RE-PAC,LE-PACG	OD-20/20	OS-20/20	OD-26	OS-29	OD-Pass	Fail	OS-Pass	Pass	BE-WNL

			BE-PI done.									
28	P915013	45	BE-JOAG,High	OD-20/60	OS-20/250	OD-16	OS-15	OD-Pass	Pass	OS-Pass	Fail	RE-WNL, LE- Early damage
			Myopia									
29	P773891	48	BE-PACG,PI done	OD-20/125	OS-20/80	OD-17	OS-16	OD-Pass	Fail	OS-Pass	Fail	BE-Moderate field damage
30	P039104	57	BE-POAG,Trab done	OD-20/50	OS-NPL	OD-17		OD-Fail	Fail	OS-Fail	Fail	BE-Advance field damage
31	P084167	79	BE-POAG,Pseudo	OD-20/20	OS-20/25	OD-10	OS-10	OD-Pass	Pass	OS-Pass	Fail	BE- WNL
			phakia									
32	N309461	43	BE-Surgical aphakia	OD-20/50	OS-20/600	OD-14	OS-16	OD-Fail	Fail	OS-Fail	Fail	BE-Advance damage
			RE-Secondary glauc									
33	P1396792	58	BE-Nanophthalmos	OD-20/125	OS-20/100	OD-18	OS-18	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
			Yag PI done									
34	N253405	40	BE-PCG	OD-20/40	OS-20/40	OD-16	OS-11	OD-Pass	Fail	OS-Pass	Pass	BE-WNL
35	P659406	59	BE-PACG,PI done	OD-20/50	OS-20/40	OD-18	OS-18	OD-Pass	Pass	OS-Pass	Pass	BE-Early damage
36	P538462	48	RE-POAG,Trab done	OD-20/20	OS-NPL	OD-19	OS-29	OD-Pass	Pass	OS-Fail	Fail	RE-WNL
37	P087662	75	BE-PACG,Trab done	OD-20/30	OS-20/20	OD-18	OS-18	OD-Fail	Fail	OS-Fail	Fail	BE-Advance damage
38	P1026004	65	LE-PACG,PI done	OD-20/25	NPL	OD-19	OS-49	OD-Fail	Fail	OS-Fail	Fail	BE-Advance damage
39	P781496	67	BE-PACG,PI done	OD-20/20	PL+PR	OD-12	OS-14	OD-Pass	Fail	OS-Fail	Fail	RE-WNL
40	P222548	68	BE-PACG,Trab done	OD-20/50	OS-20/25	OD-13	OS-15	OD-Fail	Fail	OS-Pass	Pass	RE-Advance damage, LE-Wnl
41	P1283745	71	BE-PACG,PI done	OD-20/30	OS-20/200	OD-16	OS-20	OD-Fail	Fail	OS-Fail	Fail	BE-Advance damage
42	P1398069	48	RE-PACG,LE-PAC	OD-PL	OS-20/20	OD-40	OS-11	OD-Fail	Fail	OS-Pass	Pass	RE-Moderate field loss, LE-WNL
			BE-PI done.									
43	N296833	60	BE-Ocular hyperten	OD-CF CF	OS-20/30	OD-22	OS-20	OD-Fail	Fail	OS-Pass	Pass	RE-Moderate damage, LE-WNL
			sion									
44	P1397962	40	RE-Secondary glauc	OD-PL+PR	20/25	OD-23	OS-35	OD-Fail	Fail	OS-Pass	Fail	LE-Moderate damage
			LE-JOAG									
45	P1418960	62	BE-POAG,Pseudo	OD-20/60	OS-20/50	OD-24	OS-27	OD-Pass	Fail	OS-Fail	Fail	BE-Moderate field damage
			Phakia									
46	P001905	72	BE-Glaucoma in	OD-20/250	OS-20/80	OD-19	OS-15	OD-Pass	Fail	OS-Pass	Pass	BE-WNL
			Aphakia									
47	P575237	47	BE-Disc suspect	OD-20/30	OS-20/25	OD-16	OS-14	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
48	P866378	79	BE-POAG	OD-NPL	OS-20/20	OD-27	OS-20	OD-Fail	Fail	OS-Pass	Fail	LE-Peripheral damage

49	P1072710	54	RE-PACG, LE-PAC	OD-20/20	OS-20/20	OD-11	OS-14	OD-Pass	Fail	OS-Pass	Pass	RE-Advance field damage, LE-WNL
50	P493533	62	BE-PACG, Trab done	OD-20/125	OS-20/30	OD-17	OS-13	OD-Pass	Fail	OS-Pass	Fail	BE-Advanced field loss
51	P141983	70	BE-POAG, Cataract	OD-NPL	OS-20/160	OD-13	OS-15	OD-Fail	Fail	OS-Fail	Fail	LE-Advanced damage
52	P190365	65	BE-POAG, PI done	OD-20/800	OS-20/200	OD-12	OS-17	OD-Fail	Fail	OS-Fail	Fail	BE-Advanced field loss
53	N453621	71	BE-PXF Glaucoma	OD-20/30	OS-20/60	OD-14	OS-18	OD-Pass	Fail	OS-Pass	Pass	RE-Superior defect LE-WNL
			Senile cataract									
54	P1419222	59	RE-Disc suspect	OD-20/30	OS-HM+	OD-34	OS-25	OD-Pass	Fail	OS-Fail	Fail	RE-Moderate field damage
			LE-PDR, s/p CRVO									LE-Advanced damage
			BE-Yag PI done									
55	N179967	33	BE-PRK	OD-20/20	OS-20/25	OD-14	OS-17	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
			LE-Glaucoma with									
			angle recession									
56	P843249	57	BE-PACG, s/p YAG PI	OD-20/20p	OS-20/20	OD-20	OS-18	OD-Fail	Fail	OS-Pass	Fail	BE-Moderate field damage
57	P1412532	69	BE-POAG	OD-NPL	OS-20/50	OD-28	OS-14	OD-Fail	Fail	OS-Pass	Pass	LE-WNL
58	P1330046	58	LE-Disc suspect	OD-CFCF	OS-20/60p	OD-12	OS-16	OD-Fail	Fail	OS-Pass	Fail	LE-Advanced damage
			BE-Pseudophakia									
59	P161643	53	BE-PAC, S/p YAG PI	OD-20/20	OS-20/20	OD-22	OS-21	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
60	P1050035	72	BE-PAC, S/p YAG PI	OD-20/25p	OS-20/40	OD-14	OS-16	OD-Pass	Fail	OS-Pass	Pass	RE-Moderate damage
												LE-WNL
61	P857448	65	BE-PACG	OD-20/20p	OS-HM+	OD-12	OS-18	OD-Pass	Pass	OS-Fail	Fail	RE-WNL LE-Advanced damage
62	P1282825	79	BE-PACG, s/p YAGPI	OD-20/30p	OS-20/30p	OD-11	OS-16	OD-Fail	Fail	OS-Fail	Fail	BE-Advanced damage
63	P1408387	68	BE-PAC, s/p YAGPI	OD-20/30	OS-20/30	OD-14	OS-14	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
64	P1121889	71	BE-PAC, s/p YAGPI	OD-20/25	OS-20/30	OD-16	OS-26	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
65	P859725	68	RE-POAG, S/p BARRAGE LASER	OD-20/25p	OS-20/30	OD-17	OS-18	OD-Fail	Fail	OS-Pass	Fail	BE-Advanced damage
			LE-BRVO									
66	P954455	67	BE-Glaucoma suspect, Early cataract	OD-20/40	OD-20/50	OD-15	OS-18	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
67	P1376526	70	BE-PACG, s/p YAGPI	OD-20/25	OS-20/25	OD-12	OS-10	OD-Pass	Fail	OS-Pass	Fail	BE-Advanced damage
68	P1416889	65	BE-Phy. Large cupping	OD-20/125	OS-20/80	OD-16	OS-16	OD-Fail	Fail	OS-Fail	Fail	BE-Advanced damage
69	P1416823	47	BE-PAC	OD-20/40p	OS-20/20p	OD-12	OS-10	OD-Pass	Pass	OS-Pass	Pass	BE-WNL

70	P1416801	65	BE-PAC, s/p YAGPI	OD-20/25	OS-20/25p	OD-24	OS-27	OD-Pass	Pass	OS-Pass	Pass	BE- WNL
71	P1416726	73	BE-POAG, Senile cataract	OD-20/80p	OS-20/50	OD-16	OS-18	OD-Fail	Fail	OS-Pass	Fail	RE-Advanced damage LE-Moderate damage
72	P1416705	57	RE-Secondary glaucoma, s/p Blunt trauma	OD-20/400	OS-20/25p	OD-47	OS-10	OD-Fail	Fail	OS-Pass	Pass	RE-Moderate damage LE-WNL
73	P150724	44	BE-POAG, s/p TRAB+MMC	OD-CF CF	OS-20/20p	OD-10	OS-09	OD-Fail	Fail	OS-Fail	Fail	BE-Advanced damage
74	P118134	61	BE-Phy. Large cupping, F/h/o Glaucoma	OD-20/25p	OS-20/20p	OD-11	OS-11	OD-Pass	Pass	OS-Pass	Pass	BE- WNL
75	P526082	54	BE-POAG	OD-NPL	OS-20/160	OD-37	OS-11	OD-Fail	Fail	OS-Fail	Fail	BE-Advanced damage
			LE-TRAB+MMC, Pseudophakia									
76	P568570	66	BE-PAC, s/p YAGPI	OD-20/30	OS-20/20	OD-16	OS-15	OD-Pass	Pass	OS-Pass	Pass	BE- WNL
77	P981804	68	BE-PACG, s/p YAGPI	OD-20/20p	OS-20/40	OD-13	OS-12	OD-Pass	Fail	OS-Pass	Fail	BE-Moderate damage
78	P918601	33	BE-JOAG	OD-20/20p	OS20/20p	OD-12	OS-14	OD-Pass	Fail	OS-Pass	Pass	RE-Moderate damage LE-WNL
79	P607000	65	BE-NTG, Senile cataract	OD-20/30	OS-20/30	OD-14	OS-14	OD-Pass	Pass	OS-Pass	Pass	BE-Moderate damage
80	P402134	62	RE-Angle recession	OD-PL+PR Inacc	OS-20/20p	OD-12	OS-14	OD-Fail	Fail	OS-Pass	Pass	LE-WNL
			LE-Ocular hypertension									
81	P1144956	58	RE-PACG, LE-PAC	OD-20/20p	OS-20/20p	OD-19	OS-14	OD-Fail	Pass	OS-Pass	Pass	RE-Moderate damage, LE-WNL
			BE-s/p YAG PI									
82	P077108	53	BE-Phy. Large cup	OD-20/20	OS-20/20	OD-15	OS-12	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
83	P1416869	22	BE-Glaucoma suspect, optic atrophy	OD-20/320	OS-20/100	OD-14	OS-14	OD-Fail	Fail	OS-Fail	Fail	BE-Advanced damage
84	P1416877	74	BE-PAC, s/p YAGPI, Cataract	OD-20/30	OS-20/100	OD-18	OS-22	OD-Pass	Fail	OS-Pass	Fail	BE-Moderate damage
85	P1416937	45	RE-PACG, LE-PAC	OD-20/30	OS-20/20	OD-42	OS-18	OD-Pass	Fail	OS-Pass	Pass	RE-Moderate damage LE-WNL
86	PN1264823	35	BE-Secondary Glaucoma	OD-20/200	OS-NPL	OD-54	OS-58	OD-Fail	Fail	OS-Fail	Fail	BE-Advanced damage
87	N463745	54	BE-Glaucoma in Pseudophakia	OD-PL	OS-20/600	OD-21	OS-24	OD-Fail	Fail	OS-Fail	Fail	BE-Advanced damage
88	P1416759	84	BE-Glaucoma suspect, Senile cataract	OD-20/80	OS-20/60p	OD-14	OS-14	OD-Pass	Pass	OS-Pass	Pass	BE- WNL
89	P1416739	63	BE-Glaucoma suspect	OD-20/60	OS-20/100	OD-15	OS-15	OD-Pass	Pass	OS-Pass	Pass	BE- WNL
90	P716760	65	RE-PACS, LE-PACG	OD-20/60	OS-20/20	OD-18	OS-16	OD-Pass	Pass	OS-Pass	Fail	RE-WNL, LE-Moderatedamage
			BE-s/p YAG PI, LE-TRAB									
91	P850604	44	RE-Ocular hypertension	OD-20/20	OS-20/20	OD-11	OS-13	OD-Pass	Pass	OS-Pass	Pass	BE- WNL

			LE-POAG									
92	P1412366	48	BE-PACS, Early cataract	OD-20/100	OS-20/50	OD-20	OS-31	OD-Pass	Pass	OS-Pass	Pass	BE- WNL
93	P124043	67	BE-Disc suspect, cataract	OD-20/25	OS-20/20	OD-19	OS-20	OD-Pass	Pass	OS-Pass	Pass	BE- WNL
94	P1416496	46	RE-NVG, Secondary glaucoma	OD-20/200	OS-20/80	OD-30	OS-10	OD-Fail	Fail	OS-Pass	Pass	RE-Advanced damaged, LE-WNL
95	P1416510	41	BE-Steroid induced Glaucoma	OD-20/20	OS-20/20	OD-28	OS-24	OD-Pass	Pass	OS-Pass	Pass	BE- WNL
96	N463674	58	LE-Lens induced glaucoma	OD-20/30p	OS-PL+PR Acc	OD-13	OS-45	OD-Pass	Pass	OS-Fail	Fail	RE-WNL LE-Moderate damage
97	P1268730	49	BE-Glaucoma suspect	OD-20/20	OS-20/20p	OD-20	OS-20	OD-Pass	Pass	OS-Pass	Pass	BE-WNL
98	P1407433	63	RE-PAC,LE-PACG	OD-20/40	OS-20/100	OD-16	OS-45	OD-Pass	Pass	OS-Pass	Fail	RE- WNL LE-Moderate damage
			BE-s/p YAG PI									
99	P1019837	75	BE-POAG, s/p TRAB+MMC	OD-NPL	OS-20/20p	OD-48	OS-16	OD-Fail	Fail	OS-Pass	Fail	LE-Moderate damage
100	P964805	67	BE-PACG, s/p YAGPI	OD-20/20p	OS-20/30	OD-14	OS-16	OD-Pass	Pass	OS-Pass	Fail	BE- WNL
101	P1416587	54	BE-POAG	OD-20/320	OS-20/400	OD-15	OS-16	OD-Pass	Fail	OS-Pass	Fail	BE-Moderate damage
102	P1007522	68	BE-Steroid induced Glaucoma	OD-20/40p	OS-20/60	OD-22	OS-23	OD-Pass	Fail	OS-Pass	Fail	BE-Advance damage
			BE-Pseudophakia									

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