A DISSERTATION ON

"TO STUDY THE EFFICACY OF GRID LASER PHOTOCOAGULATION ON CONTRAST SENSITIVITY, VISUAL FIELDS AND OVERALL VISUAL OUTCOME IN PATIENTS WITH DIABETIC MACULAR EDEMA - INTERVENTIONAL STUDY"

Submitted to

THE TAMIL NADU DR. M. G. R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements

For the award of degree of

M.S. (Branch III) --- OPHTHALMOLOGY

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

MAY 2022

CERTIFICATE

This is to certify that the study entitled **"TO STUDY THE EFFICACY OF GRID LASER PHOTOCOAGULATION ON CONTRAST SENSITIVITY, VISUAL FIELDS AND OVERALL VISUAL OUTCOME IN PATIENTS WITH DIABETIC MACULAR EDEMA - INTERVENTIONAL STUDY"** is the result of original work carried out by **DR. ANU. V**, under my supervision and guidance at **GOVERNMENT STANLEY MEDICAL COLLEGE, CHENNAI.** The thesis is submitted by the candidate in partial fulfilment of the requirements for the award of M.S Degree in Ophthalmology, course from 2019 to 2022 at Government Stanley Medical College, Chennai.

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CERTIFICATE FROM GUIDE

This is to certify that the dissertation entitled **"TO STUDY THE EFFICACY OF GRID LASER PHOTOCOAGULATION ON CONTRAST SENSITIVITY, VISUAL FIELDS AND OVERALL VISUAL OUTCOME IN PATIENTS WITH DIABETIC MACULAR EDEMA-INTERVENTIONAL STUDY**" is a bonafide record of research work done by **Dr. ANU. V,** Post graduate at Dept of ophthalmology, Government Stanley Medical College, Chennai

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DECLARATION

I hereby declare that this dissertation entitled **"TO STUDY THE EFFICACY OF GRID LASER PHOTOCOAGULATION ON CONTRAST SENSITIVITY, VISUAL FIELDS AND OVERALL VISUAL OUTCOME IN PATIENTS WITH DIABETIC MACULAR EDEMA - INTERVENTIONAL STUDY"** is a bonafide and genuine research work carried out by me under the guidance of **Prof. Dr. THANGERANI RAAJASEHARAN, M.S., D.O.,** Unit chief and Head of the Department, Department of Ophthalmology, Government Stanley Medical college and Hospital, Chennai – 600001.

Date : Signature

Place: Chennai **Dr ANU V**

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I express my immense gratitude to The **Dean, Prof. Dr. BALAJI, M.S, FRCS , PhD ,** Government Stanley Medical College for giving me the opportunity to work on this study.

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GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01

INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK	:"TO STUDY THE EFFICACY OF GRID LASER PHOTOCOAGULATION ON CONTRAST SENSITIVITY, VISUAL FIELDS AND OVERALL VISUAL OUTCOME IN PATIENTS WITH DIABETIC MACULAR EDEMA" - INTERVENTIONAL STUDY
PRINCIPAL INVESTIGATOR	: DR V. ANU,
DESIGNATION	: PG IN OPHTHALMOLOGY,
DEPARTMENT	: DEPARTMENT OF OPHTHALMOLOGY, GOVT. STANLEY MEDICAL COLLEGE.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 29.01.2020 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

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3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
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CERTIFICATE - II

This is to certify that this dissertation work titled **"TO STUDY THE EFFICACY OF GRID LASER PHOTOCOAGULATION ON CONTRAST SENSITIVITY,VISUAL FIELDS AND OVERALL VISUAL OUTCOME IN PATIENTS WITH DIABETIC MACULAR EDEMA - INTERVENTIONAL STUDY"** of the candidate **Dr. ANU. V.** with Registration number **221913051** for the award of **M.S DEGREE** in the branch of **OPHTHALMOLOGY**. I personally verified the original.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and the result shows 7 % percentage of plagiarism in the dissertation.

Guide and supervisor sign with seal

TABLE OF CONTENTS

LIST OF ABBREVIATIONS

DME	Diabetic macular edema
CSME	Clinically significant macular edema
NPDR	Non proliferative diabetic retinopathy
BCVA	Best corrected visual acuity
ETDRS	Early treatment diabetic retinopathy study
DD	Disc dioptres
mm	Millimetre
mmhg	Millimetres of mercury
LDL	Low density lipoprotein
VEGF	Vascular endothelial growth factor
IRMA	Intraretinal micro vascular anomalies
RPE	Retinal pigment epithelium
µM	Micrometre
Nd YAG	Neodymium Yttrium-Aluminium-Garnet

PROFORMA

Serial no. :

Name :

Age :

Sex :

Occupation :

Address :

Ocular complaints :

History of disease :

Treatment history :

Duration of treatment :

Drug history :

Ass. Systemic illness :

Family history :

BASELINE CLINICAL EXAMINATION:

Blood Pressure :

BASELINE OCULAR EXAMINATION

	RE	LE
UCVA		
BCVA		
Eyelids and lashes		
Extraocular movements		
Slit lamp examination		
Conjunctiva		
Cornea		
Anterior chamber		
Iris		
Pupil		
Lens		
EOM		
Fields		
Colour vision		
Contrast sensitivity		

Fundus Examination :

DIAGNOSIS:

Investigations

FBS, PPBS, HbA1c,

Diagnosis

Treatment: grid laser photocoagulation

- Power
- Spot size
- Duration
- Follow up and outcome of the treatment

PATIENT INFORMATION SHEET

TITLE: To study the efficacy of grid laser photocoagulation on contrast Sensitivity, visual fields and overall visual outcome in patients with diabetic macular edema- interventional study

I , **Dr. ANU V.** , post graduate at dept of Ophthalmology, Government Stanley Medical College is going to undertake the study on the above mentioned topic. If you are willing to participate in this study you will be asked some questions regarding duration of your illness, treatment history ,family history and regarding the illness. You may need to undergo grid laser photocoagulation as a part of treatment for diabetic macular edema The study may be of direct benefit for you in knowing your visual outcome and contrast sensitivity following grid laser photocoagulation. The study may be of great help in planning treatment strategies for you and other people in future.

I assure that all the information provided by you will be kept highly confidential and the privacy is assured. Your identity won't be revealed to anyone. The study may be published in scientific journal, but your identity will not be revealed. Your participation in this study is voluntary and you can withdraw from this at any point of time

Signature/left thumb impression of the participant

INFORMED CONSENT

TITLE: To study the efficacy of grid laser photocoagulation on contrast Sensitivity, visual fields and overall visual outcome in patients with diabetic macular edema-interventional study

The content of the information sheet dated _____ that was provided have been read carefully by me/explained in detail to me, in a language that I comprehend and fully understood the contents. I confirm that I have had the opportunity to ask questions. The nature and purpose of the study and its potential risks/benefits and expected duration of the study and other relevant details of the study have been explained to me in detail. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal right being affected.

I agree to take part in the above study

_____

(Signature/Left thumb impression)

Name of the Participant: _____

Son/Daughter/Spouse of _____

Complete postal address: _____

This is to certify that the above consent has been obtained in my presence.

_____

Date: Signature of the principal investigator

Place:

1) Witness – 1

2) Witness – 2 _____

_____

Signature:

Name:

Address:

Signature:

Name:

Address:

INFORMATION SHEET

தகவல் நகல்

TITLE: To study the efficacy of grid laser photocoagulation on contrast Sensitivity, visual fields and overall visual outcome in patients with diabetic macular edema-interventional study

இந்த ஆய்வில் உங்களிடம் கேட்கும் கேள்விகளுக்கு முழு மனதுடன் பதில் அளிக்க வேண்டும். இந்த ஆய்வில் உங்கள் நாள்பட்ட நோய்களின் விபரம் , உடல்நலம் மற்றும் நடத்தை தொடர்பான விபரங்கள் கேட்கப்படும். இந்த ஆய்வினால் உங்களுக்கு எந்த பின்விளைவுகளும் ஏற்படாது என்று உறுதி அளிக்கிறேன். உங்களிடம் உங்களின் சுய விபரம் , குடும்ப விபரம், தொழில் விபரம் மற்றும் இதர விபரங்கள் பற்றி கேட்கப்படும் . உங்களுக்கு பணம் எதுவும் அளிக்க படாது என்பதை தெரிவித்துக் கொள்கிறேன்.

இந்த ஆய்வில் நீரழிவு நோயினால் விழித்திரையில் ஏற்படும் நோய்க்கு லேசர் சிகச்சை அளிக்கப்பட்டு , அதற்கு பின்பு பார்வையில் ஏற்படும் மாற்றங்கள் அனைத்தும் குறித்து பதிவு செய்யப்படும். இந்த ஆய்வில் மூலம் உங்களின் பார்வையில் ஏற்படும் மாற்றங்களை பற்றி நீங்கள் அறியலாம். மேலும் இதன் மூலம் வருங்காலத்தில் உங்களை போன்ற மக்களுக்கு சிகிச்சை அளிக்க தேவைப்படும் கோட்பாடுகளை வகுக்கலாம்.உங்களின் விபரங்கள் எதுவும் மற்றவர்களுக்கு தெரிவிக்க படாது என்று உறுதி அளிக்கிறேன்.இந்த ஆய்வின் முடிவுகள் பத்திரிக்கைகளில் பிரசுரிக்க படலாம். ஆனால் உங்களின் விபரங்கள் எதுவும் வெளியிட படாது. உங்களுக்கு விருப்பம் இல்லையெனில் எப்போது வேண்டுமானாலும் இந்த ஆய்வில் இருந்து விலகி கொள்ளலாம். இதனால் உங்களுக்கு எந்த பாதிப்பும் ஏற்படாது.

கையொப்பம்/ இடதுகை பெருவிரல் ரேகை

INFORMED CONSENT

ஒப்புதல் படிவம்

TITLE: To study the efficacy of grid laser photocoagulation on contrast Sensitivity, visual fields and overall visual outcome in patients with diabetic macular edema-interventional study

நான் தகவல் நகளில் கொடுக்கப்பட்டுள்ள முழு விபரங்களையும் கவனமாக படித்தேன். ஆய்வின் முழு விபங்களும் எனக்கு தமிழில் விளக்கமாக எடுத்துரைக்கப்பட்டது.நான் இந்த ஆய்வின் விபரங்களை முழுமையாக புரிந்துகொண்டேன். இந்த ஆய்வில் பங்கெடுக்கும் போது ஏற்படும் சாத்தியமான அபாயங்கள் மற்றும் பயன்களை அறிந்து கொண்டேன். மேலும் நான் எந்த ஒரு வேளையிலும் ஆய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அதன் பின்னர் நான் வழக்கம் போல் மருத்துவ சிகிச்சை பெற முடியும் என்றும் புரிந்து கொண்டேன். நான் ஆய்வில் பங்கு கொள்ள பணம் எதுவும் கொடுக்க படாது என்றும் அறிந்து கொண்டேன்.நான் இந்த ஆய்வின் போது முழு ஒத்துழைப்பு கொடுப்பேன் என்று உறுதி அளிக்கிறேன்.

ஆய்வில் பங்கேற்பவர் பெயர் : கையொப்பம்/ இடதுகை பெருவிரல் ரேகை

சாட்சி

பெயர் மற்றும் முகவரி பெயர் மற்றும் முகவரி

ஆராய்ச்சியாளர் கையொப்பம் மற்றும் தேதி

KEY TO MASTER CHART

M	Male
F	Female
RE	Right eye
LE	Left eye
NPDR	Non proliferative diabetic retinopathy
CSME	Clinically significant macular edema
BCVA	Best corrected visual acuity
PRE GRID VA	Pre grid laser visual acuity
POST GRID VA 1 W	Post grid laser visual acuity at 1 week
POST GRID VA 1 M	Post grid laser visual acuity at 1 month
POST GRID VA 3 M	Post grid laser visual acuity at 3 months
PRE GRID CS	Pre grid laser contrast sensitivity
POST GRID CS 1 W	Post grid laser contrast sensitivity at 1 week
POST GRID CS 1 M	Post grid laser contrast sensitivity at 1 month
POST GRID CS 3 M	Post grid laser contrast sensitivity at 3 months
PRE GRID CV	Pre grid laser colour vision
POST GRID CV 1 W	Post grid laser colour vision at 1 week
POST GRID CV 1 M	Post grid laser colour vision at 1 month
POST GRID CV 3 M	Post grid laser colour vision at 3 months
PRE GRID VF	Pre grid laser visual field
POST GRID VF	Post grid laser visual field

INTRODUCTION

Diabetes mellitus is a metabolic disease which is characterised by hyperglycaemia resulting from impaired secretion of insulin or impaired action of insulin or both. Chronic hyperglycaemia associated with uncontrolled diabetes leads to multi organ damage especially to eyes and blood vessels. Diabetic retinopathy is a major preventable cause of blindness all over the world. Diabetic macular edema that occurs due to diabetic retinopathy leads to moderate vision loss in patients with diabetic retinopathy and it is the commonest cause of vision loss in patients with diabetic retinopathy.

The ETDRS coined the term “clinically significant macular edema” (CSME) to define the threshold severity level of edema. Grid laser photocoagulation is the mode of treatment for CSME

As the macula is the area concerned with visual acuity, contrast sensitivity, colour vision and form vision, the diseases of macula leads to defective form vision, colour vision and contrast sensitivity. Visual acuity measured with snellen visual acuity chart which is a high contrast visual acuity chart doesn’t encounter the difficulties faced by the patient in a normal surrounding of varying contrast. Hence measurement of contrast sensitivity plays a pivotal role in assessment of patients with diabetic macular edema

EPIDEMIOLOGY

The prevalence of diabetic retinopathy among the patients with diabetes is estimated to be 35.4% globally(1). The prevalence was found to vary between various parts of the world and this broad range of variation may be due to socioeconomic factors and health facilities. The prevalence of diabetic retinopathy in India based on a study at Chennai was found to be 18% in urban population and 10.8% in rural population(1) which may be due to difference in the diet pattern between the two population and poor access to health care in case of rural population. The prevalence of Diabetic macular edema was between 1.4 to 12.8% among the patients with type 2 diabetes mellitus. (1)

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ANATOMY OF RETINA AND MACULA(2)

Retina is the most highly developed innermost layer of the eyeball and it extends from the optic disc to oraserrata.

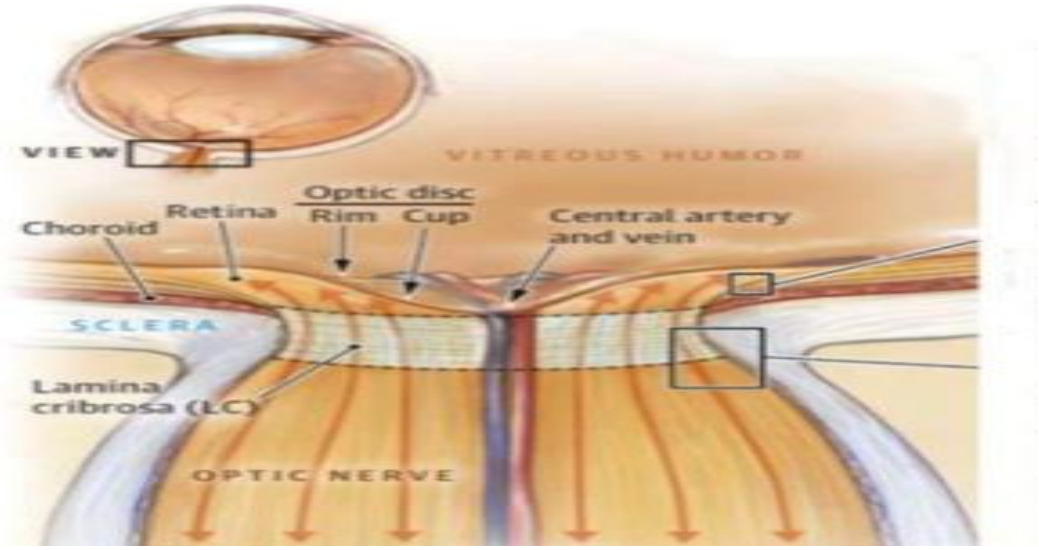
Retina can be grossly divided into following regions

- Optic disc
- Macula
- Peripheral retina

Optic disc:

Optic disc is a well-defined vertically oval structure of about 1.5 mm (1DD) where all the layers of the retina terminate except the nerve fibre layer. The axons from the nerve fibre layer pierce the lamina cribrosa and form the optic nerve. The optic disc consists of a depression known as physiological cup from where the central retina artery and vein emerge into the retina and they branch dichotomously to supply the entire retina.

FIGURE 1 : ANATOMY OF OPTIC DISC



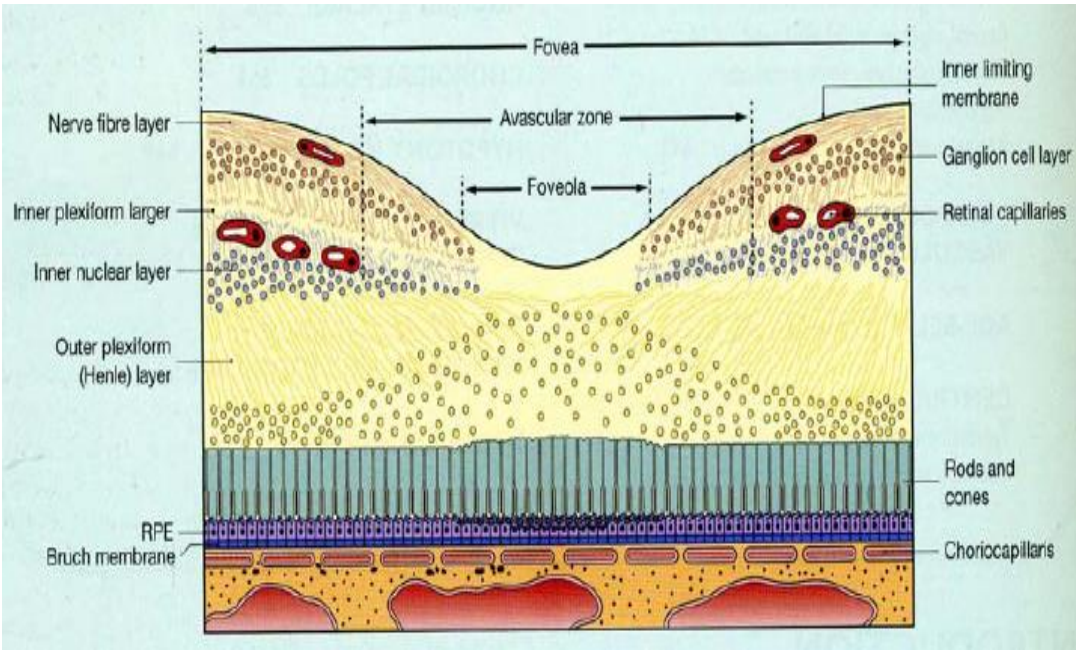
Macula lutea

The central 5.5 mm of retina is known as macula or area centralis(3). It lies temporal to the optic disc. It appears yellow in colour due to accumulation of lutein and zeaxanthine.The macula contain two or more ganglion cell layer and its periphery is where the ganglion cell layer is reduced to a single layer. The macula corresponds to central 15º of the visual field.

Fovea centralis

A concave depression in the central 1.5 mm of the macula is known as fovea. The photoreceptor layer in this area consists entirely of cones. Fovea being the area of highest visual acuity corresponds to central 5 º of visual field

FIGURE 2 : LAYERS OF MACULA



Foveola

Ill-defined area of 0.35 mm diameter of the centre of the fovea in which the inner nuclear layer and the ganglion cell layer are absent.

Umbo

The central of foveola has a tiny depression known as umbo which corresponds to the normal foveal reflex.

Foveal avascular zone

The central 500 microns of the fovea is devoid of retinal blood vessels known as foveal avascular zone

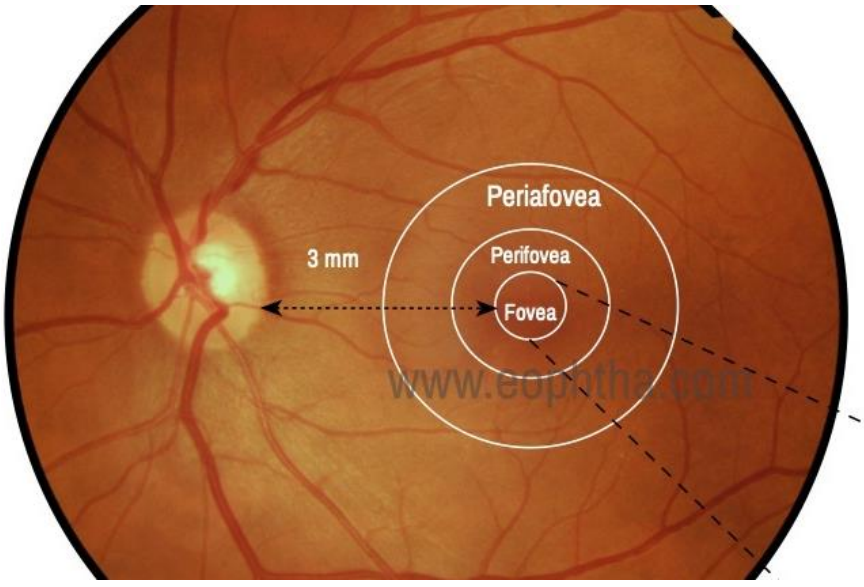
Para foveal zone

The area surrounding the fovea is known as para fovea which is 0.5 mm. The ganglion cell layer, inner nuclear layer, and outer plexiform layer are thickest in the parafoveal region

Perifoveal zone

The area surrounding the para foveal region is perifovea which is 1.5mm wide.

FIGURE 3 : ANATOMY OF MACULA



Peripheral retina

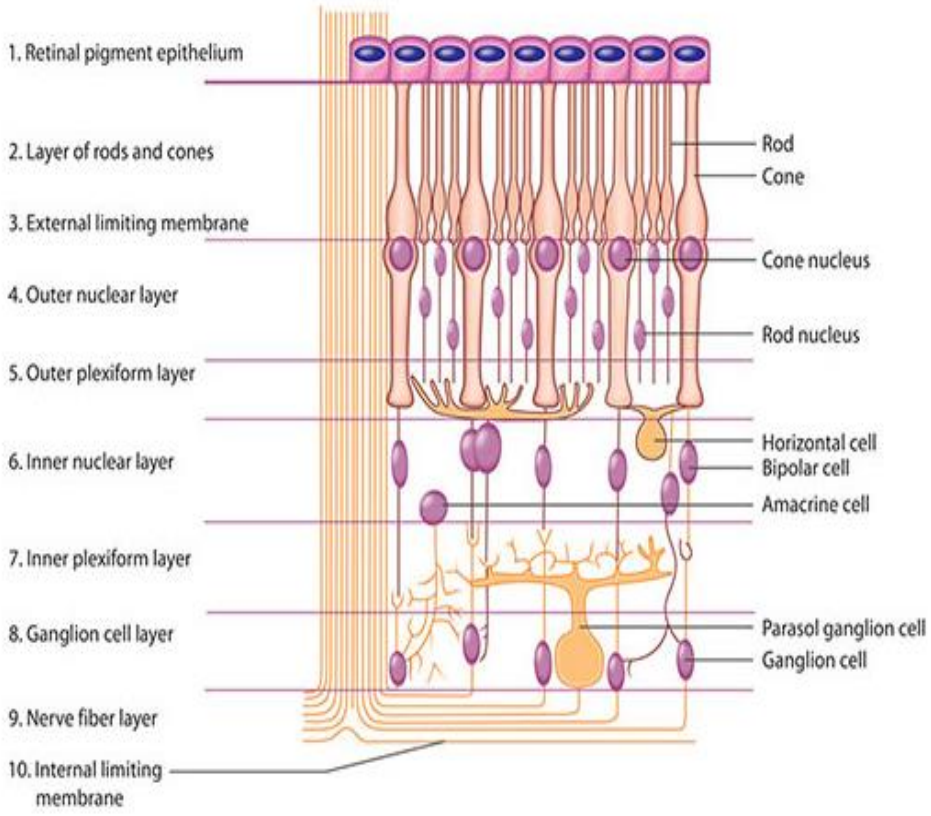
- Near periphery is 1.5 mm around the macula
- Mid periphery is 3 mm wide zone around the near periphery which corresponds to the equator.
- Far periphery extends from equator to ora serrata
- Extreme periphery is area of ora serrata and pars plana.
- Ora serrata is the region where the retina ends and ciliary body begins

Layers of retina

The layer of retina from inner to outer are

1. Internal limiting membrane
2. Nerve fibre layer
3. Ganglion cell layer
4. Inner plexiform layer
5. Inner nuclear layer
6. Outer plexiform layer
7. Outer nuclear layer
8. External limiting membrane
9. Layers of rods and cones
10. Retinal pigment epithelium

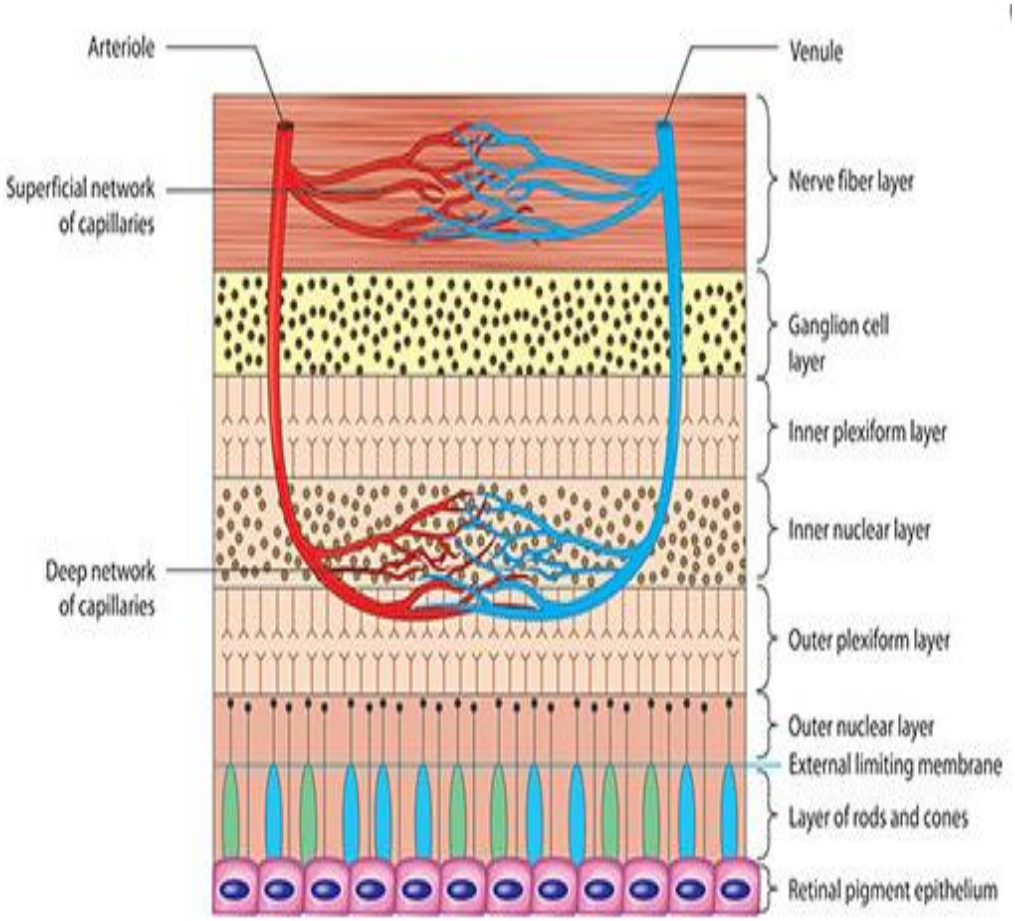
FIGURE 4: Layers of retina



Blood supply of retina

- Outer 4 layers – chorio capillaries
- Inner 6 layers – central retinal artery
- Outer plexiform layer – partly from central retinal artery and partly from choriocapillaries
- Fovea – avascular zone supplied by choriocapillaries
- Macula – small twigs from supero temporal and inferotemporal branches of central retinal artery

FIGURE 5 : BLOOD SUPPLY OF RETINA



RISK FACTORS OF DIABETIC RETINOPATHY (1)

Non modifiable risk factors

- Duration of diabetes

Longer duration is found to be significantly associated with diabetic retinopathy and it is independent of glycaemic control.

- Pregnancy

DR and DME progresses rapidly during pregnancy which is transient and may be undergo rapid regression following delivery and the possible mechanism may be both humoral and immune theories.

Modifiable risk factors

- Hyperglycaemia

A graded relationship exists between the glycaemic levels and frequency of diabetic retinopathy. The DCCT study showed that strict glycaemic control decreased the occurrence of DR by 76% and DME by 58%.

- Hyperlipidaemia

A relationship exists between the levels of LDL cholesterol and diabetic retinopathy. Fenofibrate was found to have moderate action in reducing macular volume in patients with diabetic macular edema.

- Hypertension

The risk of micro vascular disease is reduced by 37% and the rate of progression of DR by 34% when the target blood pressure is less than 150/85 mmhg

- Obesity

Obstructive sleep apnea which is closely related to obesity is found to be a potential risk factor for DME.

Novel risk factors

- Oxidative hormones

Adipocytes secrete leptin and adiponectin which were found to a risk factor. Leptin has a role in upregulation of VEGF which in turn stimulates neovascularisation and ischemia. Adiponectin counters ischemia by stimulating perfusion of ischaemic retina.

- Metabolic stress

Oxidative stress causes various histopathological changes associated with diabetic retinopathy. Diabetic patients were found to have increased reactive oxygen species and decreased antioxidant potential

- Vitamin D

The anti angiogenic and anti-inflammatory properties of vitamin D has a protective effect on diabetic retinopathy and DME.

PATHOGENESIS OF DIABETIC RETINOPATHY(4)

Hyperglycaemia

Hyperglycaemia plays an important role in pathogenesis of retinal micro vascular damage. It leads to dilatation of blood vessels and blood flow changes which occurs due to metabolic auto regulation in order to compensate for the increased retinal metabolism. Hyperglycaemia triggers apoptosis of pericytes which leads to loss of pericytes resulting in loss of structural support and localized outpouching of capillary wall. Microaneurysm is the earliest sign of diabetic retinopathy. The loss of pericytes, endothelial cells and thickening of basement membrane leads to breakdown of blood retinal barrier and occlusion of the capillaries. This leads on to retinal ischemia which causes up regulation of VEGF through activation of hypoxia inducible factor. VEGF increases the vascular permeability and promotes endothelial proliferation.

Inflammation

Chronic low grade inflammation along with leucostasis has been found to be the reason for the occlusion of microvasculature. Increased leucostasis leads to endothelial damage and breakdown of BRB.

ETDRS CLASSIFICATION OF NON PROLIFERATIVE DIABETIC RETINOPATHY(5)

TABLE 1 : ETDRS CLASSIFICATION OF NPDR

No DR	
Very mild NPDR	Microaneurysms only
Mild NPDR	Any or all of: microaneurysms, retinal haemorrhages, Exudates, cotton-wool spots, up to the level of moderate NPDR. No intraretinal microvascular anomalies (IRMA) or Significant beading
Moderate NPDR	Severe retinal haemorrhages (more than ETDRS Standard photograph 2A: about 20 medium–large per Quadrant) in 1–3 quadrants or mild IRMA • Significant venous beading can be present in no more than 1 quadrant • Cotton-wool spots commonly present
Severe NPDR	The 4–2–1 rule; one or more of • Severe haemorrhages in all 4 quadrants • Significant venous beading in 2 or more quadrants • Moderate IRMA in 1 or more quadrants
Very severe NPDR	Two or more of the criteria for severe NPDR

FIGURE 6 : MILD NPDR WITH CSME

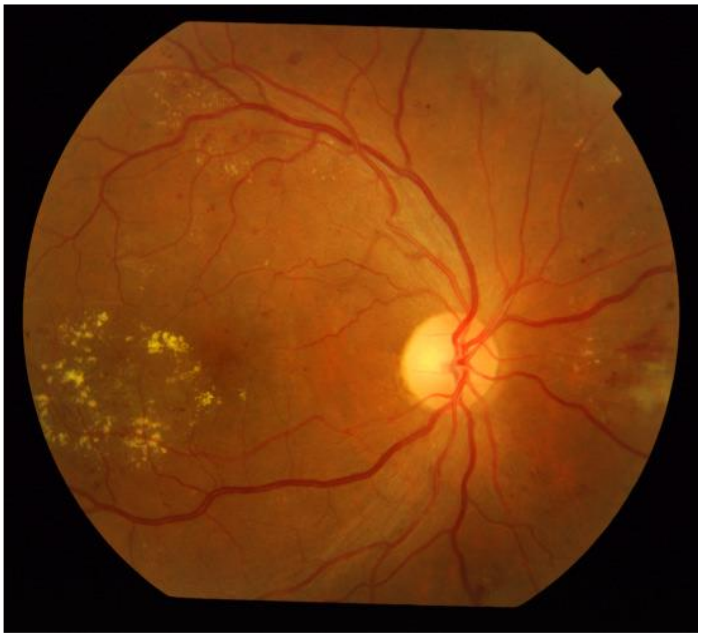


FIGURE 7: MODERATE NPDR WITH CSME

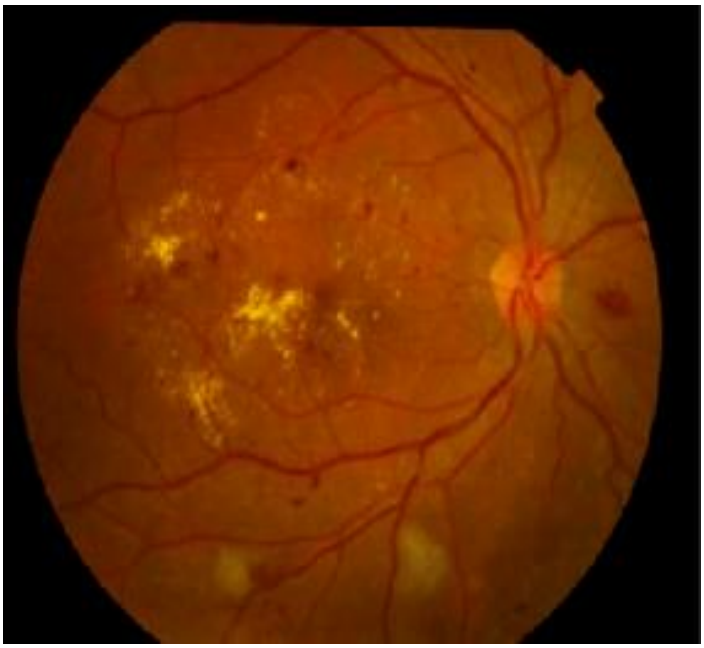


FIGURE 8 : SEVERE NPDR WITH CSME

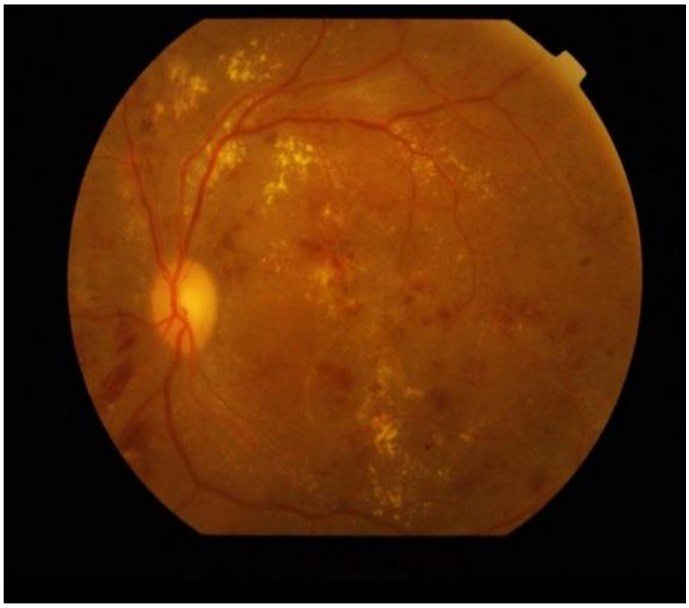
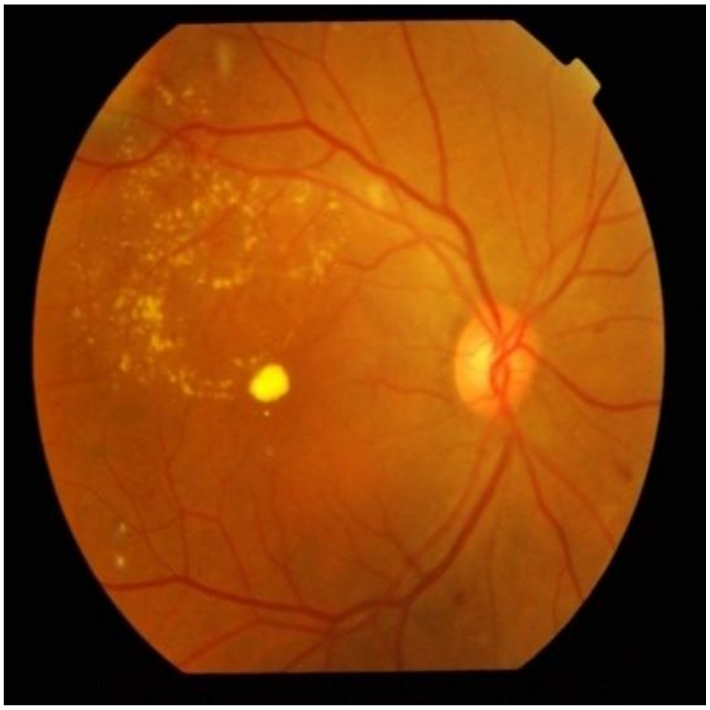


FIGURE 9 : CSME



DIABETIC MACULAR EDEMA

DME is defined as hard exudate and retinal thickening involving the macular area(6). It occurs commonly in patients with type 2 diabetes mellitus. The edema occurs due to leakage from the retinal vessels or deep leakage through the RPE.

Risk factors for diabetic macular edema

- Duration of diabetes
- Poor control of diabetes
- Associated systemic hypertension
- Hyperlipidaemia
- Diabetic nephropathy

Pathophysiology of diabetic macular edema(7)

Longer duration of hyperglycaemia leads to hypoxia and reduced perfusion which activates the auto regulatory mechanism leading to vasodilatation , elevated hydrostatic pressure and hyperpermeablity resulting in flow of water, ion and macromolecules from intravascular space into the extravascular space.Hypoxia leads to increase in vascular permeability mediated by VEGF and retinal vascular auto regulation which leads to increased accumulation of fluid in the foveal avascular zone as RPE pump is only mechanism of extracellular fluid resorption(7).The vitereoretinal interface abnormalities in diabetes may also cause diabetic macular edema. The macula and the disc are strongly adherent to the posterior hyaloid. The traction exerted during vitreous separation may lead to break down of blood retinal barrier which contributes to cellular proliferation which in turn further aggravates the macular traction(7)

High glycated haemoglobin (HbA1C) levels indicative of longer duration of hyperglycaemia causes increased production of VEGF. Increased accumulation of inflammatory mediators is associated with capillary nonperfusion and breakdown of blood retinal barrier.Type 2 diabetic patients may have a greater reduction in the subfoveal choroidal blood flow leading to relative hypoxia of outer retina and RPE resulting in increased permeability of outer blood retinal barrier. Cross linking and glycation is increased in the vitreous of diabetic which may cause a tangential macular traction leading to diabetic macular edema.

Classification of diabetic maculopathy and macular edema

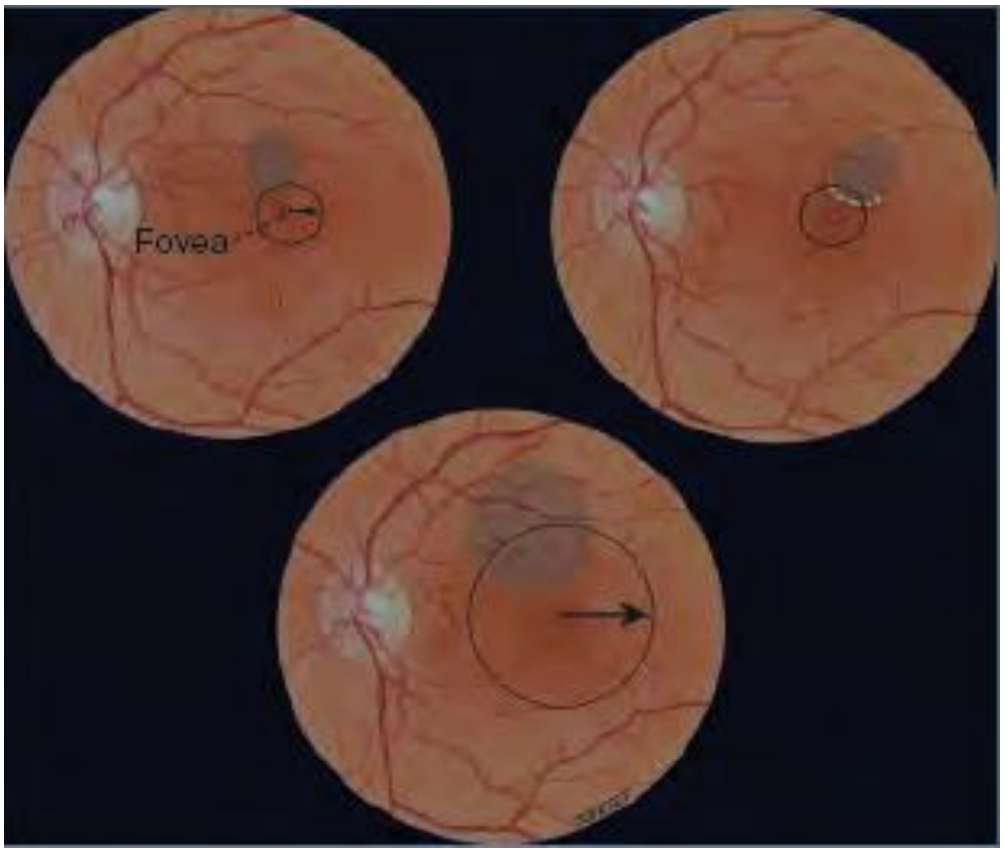
- Focal exudative macular edema
- Diffuse exudative macular edema
- Ischaemic maculopathy
- Mixed forms

Clinically significant macular edema

Clinically significant macular edema (CSME) is detected on clinical examination as defined in the ETDRS(6)

- Retinal thickening within 500 μm of the centre of the macula
- Exudates within 500 μm of the centre of the macula, if associated with retinal thickening. The thickening itself may be outside the 500 μm
- Retinal thickening one-disc area (1500 μm) or larger, any part of which is within one disc diameter of the centre of the macula

FIGURE 10 : ETDRS DEFINITION OF CSME



Diffuse diabetic macular edema

This occurs due to diffuse leakage from the retinal capillaries. The patients have a sudden loss of visual acuity which may be due to sudden decompensation of the retinal capillaries. Hard exudates may be seen at the junction of normal and abnormal retina .In central diffuse diabetic macular edema leakage occurs adjacent to foveal arcade along with cystoid macular edema. In generalised diffuse diabetic macular edema, retinal thickening is seen in the entire posterior pole .Grid laser photocoagulation is found to be effective in controlling diffuse diabetic macular edema.

MODALITIES OF TREATMENT OF DIABETIC MACULAR EDEMA(7)

The various modalities available for treatment of diabetic macular edema are

- Focal / grid laser photocoagulation
- Sub threshold laser photocoagulation
- Intra vireal injection of corticosteroids
- Intravitreal injection of anti VEGF
- Vitrectomy

Focal / grid laser photocoagulation

The ETDRS study has demonstrated a superior visual outcome with grid laser when compared with natural history. The grid laser will produce an approximately 10 µm of macular thinning for every additional baseline macular thickening of about 100 µm at 3 month follow up

Side effects of grid laser are

- Paracentral scotomas
- Subretinal fibrosis
- Secondary choroidal neovascularization

Sub threshold laser photocoagulation

Reduction in macular edema occurs slowly

Advantages

- Absence of RPE scarring
- No subsequent choroidal neovascularization,
- Elimination of paracentral visual field scotomas

Disadvantages

- No visible endpoint for treatment

Intra vireal injection of corticosteroids

- Triamcinolone
- Dexamethasone
- Fluocinolone

Side effects:

- Cataract in phakic eyes
- Intraocular pressure elevation

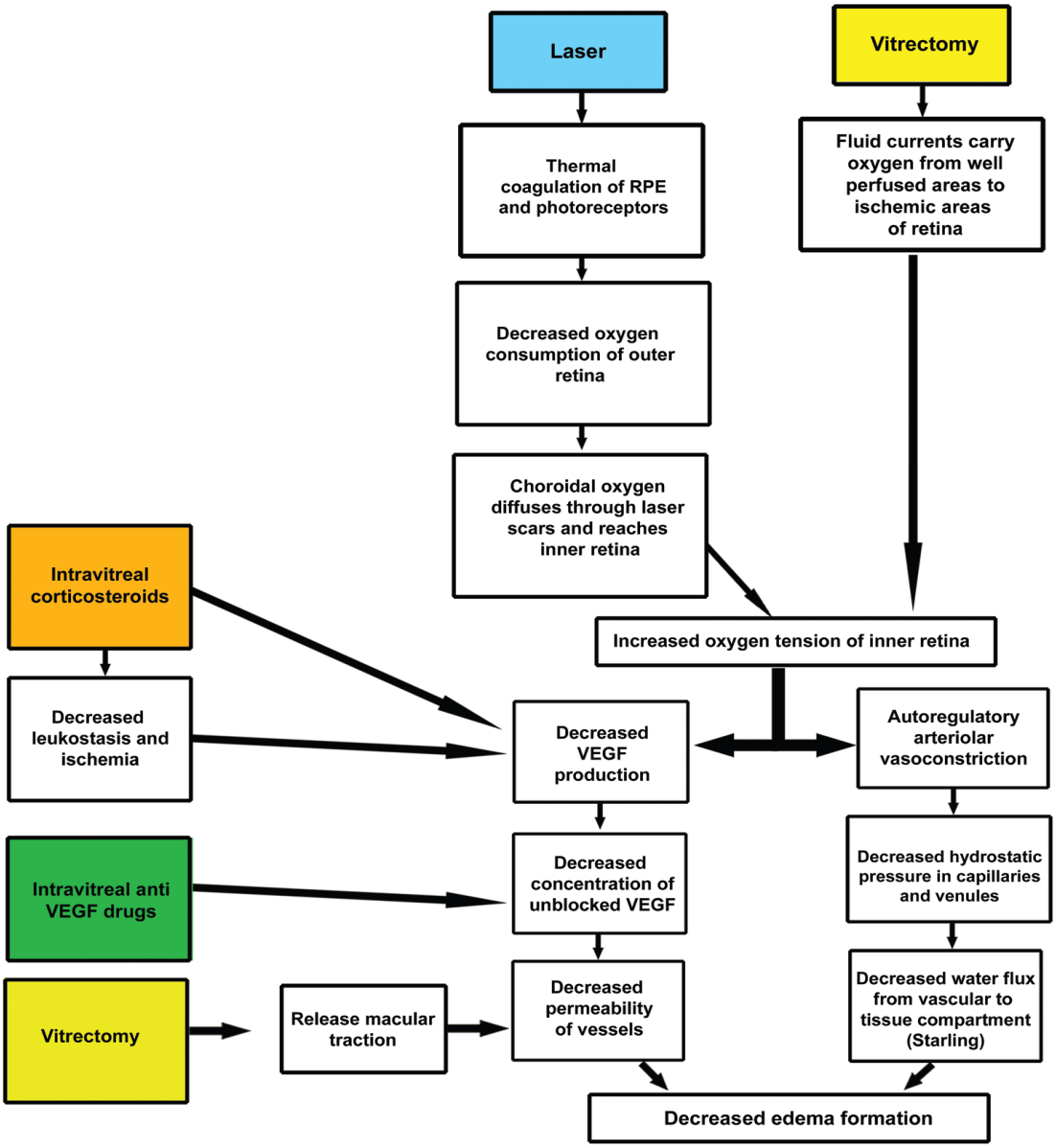
Intravitreal injection of anti VEGF

- Anti-VEGF drugs
- Aptamers (pegaptanib)
- Antibodies to VEGF (bevacizumab)
- Antibody fragments to VEGF(ranibizumab)
- Fusion proteins, which combine a receptor for VEGF with the Fc fragment of an immunoglobulin (aflibercept and conbercept).

Vitrectomy

DME usually resolves after posterior vitreous detachment which strengthens the fact that vitreomacular separation may reduce the DME. Vitrectomy was done for eyes with non-resolving DME despite other modes of treatment and irrespective of the status of posterior hyaloid. Recently vitrectomy has been found to be the potential therapy for eyes presenting with severe macular edema and greater loss of visual acuity.

FIGURE 11 : MODALITIES OF TREATMENT OF DME



VISUAL ACUITY

Visual acuity is the spatial resolving capacity of the visual system. It expresses the angular size of detail that can just be resolved by the observer(8).Moderate visual loss is defined as a doubling of the visual angle or a drop of three or more lines of Snellen equivalent.(9).Severe visual loss is defined as visual acuity < 5/200 at two consecutive follow-up visits scheduled at 4-month intervals(10)

Tests of visual resolution

- **Minimum detectable resolution**

The minimum detectable resolution is the threshold size of a spot or a line required to detect its presence against its background.(8)

- **Minimum separable resolution**

The minimum separable resolution is the least separation between two adjacent points or adjacent lines that allows the two to be seen as separate(8)

- **Recognition resolution**

Most clinical tests of visual acuity are recognition tests that determine the smallest symbols, letters, or words that can be identified correctly. Test targets used for these tests are often called optotypes. The Snellen chart uses letters as the optotypes(8)

SNELLEN CHART

The snellen chart expresses the values in fraction by specifying the testing distance and the height of the letters with the denominator indicating the height of the letter is also the distance at which the letter subtend at an angle of 5 min of arc(8)

Visual acuity = (test distance/distance at which letters subtend 5 minarc)

The snellen fraction can be converted into decimal notation. So 6/6 becomes 1.0 and 6/60 becomes 0.1 and so on and it also gives a single number to quantify an angle.

A specific testing distance of 6 m is used to facilate scoring by snellen notation. The patients are tested monoocularly while an occluder is placed in the other eye followed by testing the other eye. The patient is asked to start reading the chart from the top most line down the chart as much as they could read. An occasional error of one letter in between could be ignored. The appropriate refractive correction is also made and best corrected visual acuity is obtained.

The snellen visual acuity chart consist of series of letters of diminishing size with each letter in such a shape that it can be enclosed in a square of size which is 5 times the thickness of lines. The breadth of the line is such that their edges subtend at an angle of 1 minute at a specified distance so that the whole letter will subtend at an angle of 5 minutes at a particular distance(11)

FIGURE 12 : SNELLEN CHART

FIGURE 13 : SNELLEN LETTERS ARE CONSTRUCTED SUCH THAT THEY SUBTEND AN ANGLE OF 5 ARCMIN

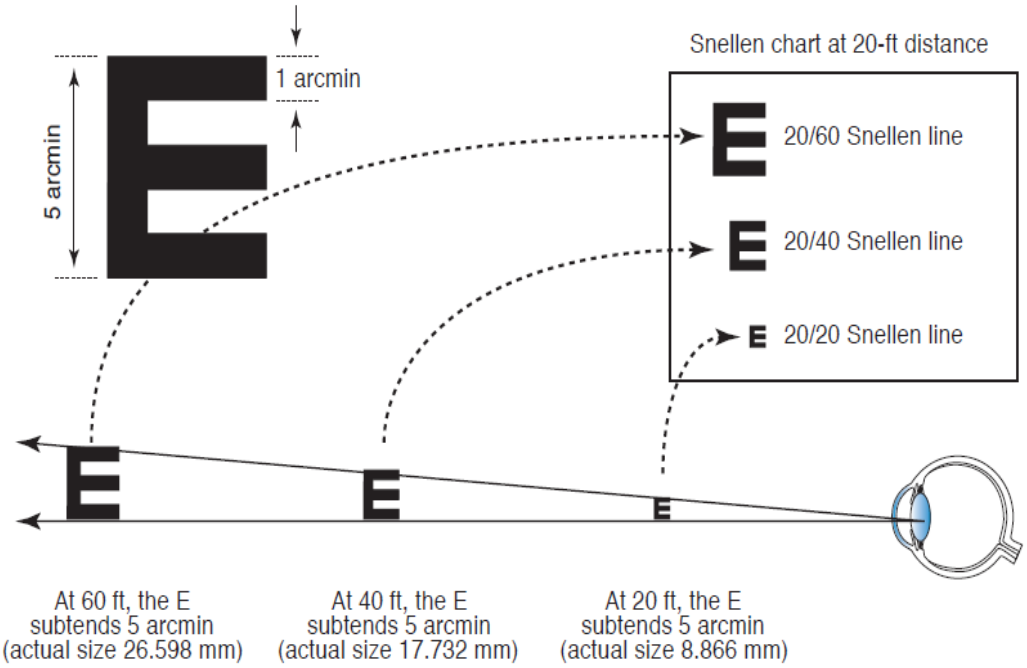


FIGURE 14 : VISUAL ACUITY CONVERSION CHART

Snellen Fraction					
Feet	Meters	4-Meter Standard	Decimal Notation (Visus)	Visual Angle Minute of Arc	LogMAR (Minimum Angle of Resolution)
20/10	6/3	4/2	2.00	0.50	–0.30
20/15	6/4.5	4/3	1.33	0.75	–0.12
20/20	**6/6**	**4/4**	**1.00**	**1.00**	**0.00**
20/25	6/7.5	4/5	0.80	1.25	0.10
20/30	6/9	4/6	0.67	1.50	0.18
20/40	6/12	4/8	0.50	2.00	0.30
20/50	6/15	4/10	0.40	2.50	0.40
20/60	6/18	4/12	0.33	3.00	0.48
20/80	6/24	4/16	0.25	4.00	0.60
20/100	6/30	4/20	0.20	5.00	0.70
20/120	6/36	4/24	0.17	6.00	0.78
20/150	6/45	4/30	0.13	7.50	0.88
20/200	6/60	4/40	0.10	10.00	1.00
20/400	6/120	4/80	0.05	20.00	1.30

CONTRAST SENSITIVITY

A contrast threshold is the smallest amount of contrast required to be able to see a target. CS is the reciprocal value of the contrast threshold(8). One will require a high amount of contrast to view a target when they have low contrast sensitivity.

A contrast sensitivity function (CSF) is derived by measuring the lowest detectable contrast across a range of spatial frequencies(3).Visual acuity is the ability of the eye to see fine details but does not illustrate the ability of the person to see objects of low contrast. So one might have a low contrast sensitivity with normal visual acuity.Contrast sensitivity is associated with decreased quality of life. The symptoms of decreased contrast sensitivity becomes more evident in low light and fog conditions

The lowest contrast level that can be recognized by a person for a fixed size target is known as contrast sensitivity. It measures the size and the contrast. The pathogenesis behind the loss of contrast sensitivity is thought to be due to hyperglycaemia leading to accumulation of fluid in the macula and enlargement of foveal avascular zone. [1]

Contrast sensitivity test detects vision loss in the presence of normal visual acuity.

Types of contrast sensitivity tests

- Grating tests
- Letter tests.

Contrast sensitivity reduction will be a limitation in certain occupations like driving heavy vehicles. Recognizing the difficulties and complaints of these patients is important as they may have a good snellen visual acuity with high contrast eye charts. Visual acuity measure only the high end spectrum of vision under high contrast whereas it fails to evaluate the ability to see low contrast objects which is also important in daily activities. So contrast sensitivity plays a complementary role (9)

PELLI ROBSON CHART

The Pelli- Robson chart is commonly used for clinical testing. It consists of letters of a fixed size which vary in contrast. The minimum level of contrast at which the letters can be seen is recorded. The pelli-Robson chart is an 86 x 63 cm wall mounted chart placed at a distance of 1 metre from the patient. The Pelli-Robson chart consists of letters of the same size which were grouped into triplets and two triplet groups appear on a single line of the chart. The chart is externally illuminated which may lead to a little change in scores over a broad range of luminance levels. This has a good reliability when compared to grating contrast sensitivity chart

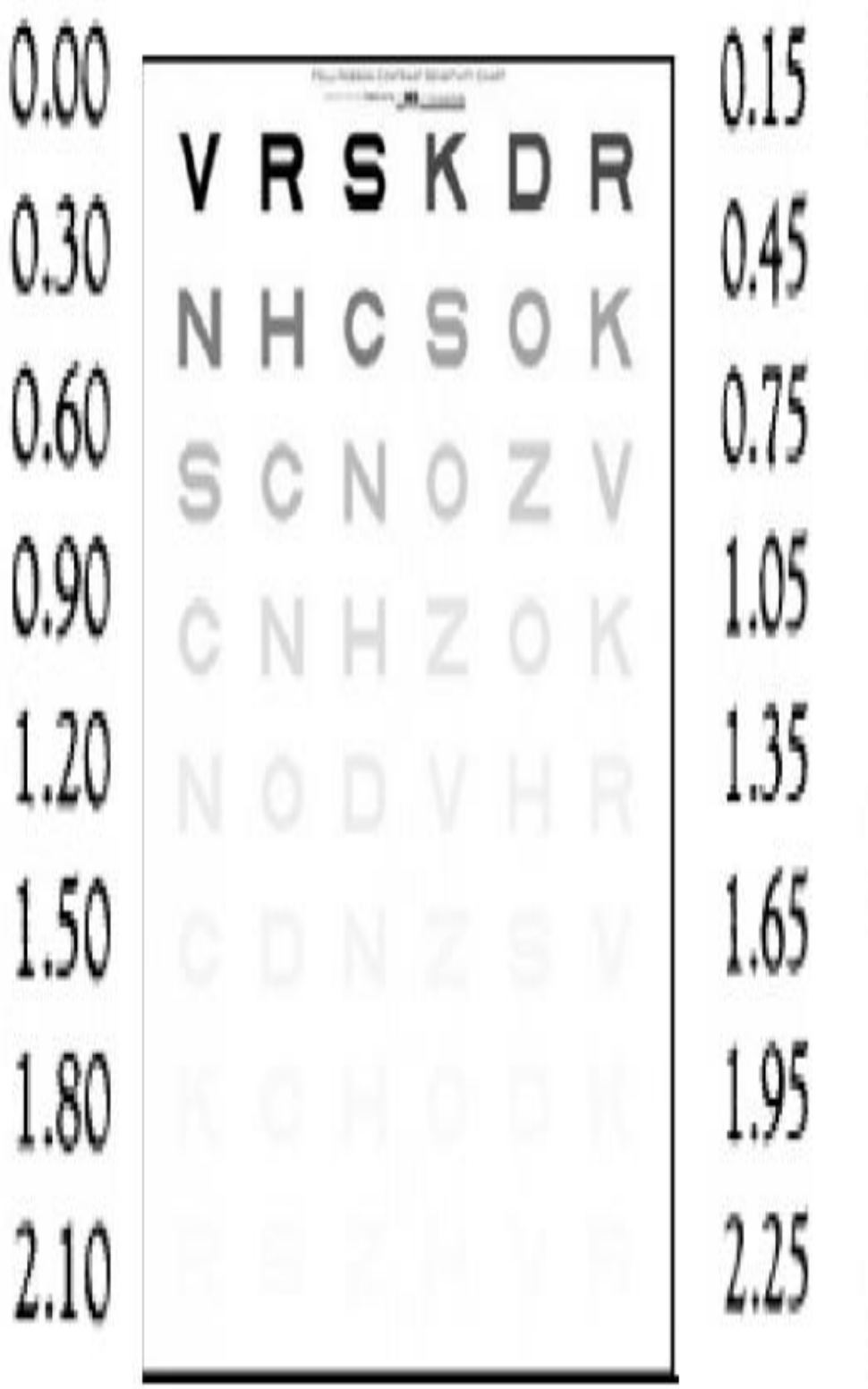
The study participant is seated at one metre from the chart and asked to start at the top left-hand to read the letters from the high contrast and till they are unable to read 2 or 3 letters in a group. The participants was encouraged to guess the letter and if he reads correctly a score of 0.05 log units was given.(12) . The score is recorded based on the last group in which he read 2 or 3 letters. It is a logarithmic measure of subjects contrast sensitivity.

2.0 - normal contrast sensitivity of 100

Less than 1.5 - visual impairment

Less than 1.0 - visual disability

FIGURE 15 : PELLI ROBSON CHART



DME AND CONTRAST SENSITIVITY

Contrast sensitivity was found to be reduced in diabetic maculopathy and it is a useful tool for monitoring. The subtle defects or improvements in the retinal ganglion cell activity can be detected by testing contrast sensitivity(13).

The bipolar cells in the retina receive inputs from the cone cells. A specific type of bipolar cell is activated when the light falls on the centre of the receptive field and it is further activated when the surrounding areas are dark which shows that bipolar cells are activated when contrast is present(13). The horizontal cells interact with the cones and they increase the action potential of the light stimulated cones on light side and inhibit the cones on the dark side thereby enhancing the contrast at light dark borders(13). The information gets transmitted from the bipolar cells via the synapse to the ganglion cells and gets transmitted to lateral geniculate body and occipital cortex. The difference in the luminance between the adjacent receptive fields are utilized to form our recognition of objects(13). These anatomical and physiological factors of the retina play a role in contrast sensitivity.

The macula which is responsible for central vision when affected by various diseases leads to defective perception of contrast. A slower retinal ganglion cell functional loss may occur in diabetic macular edema which is a chronic micro vascular condition.(13). The retinal ganglion cell layer and nerve fibre layer are totally dependent on the ocular vasculature for oxygen,, nutrients and removal of waste products which is ensured by the positive perfusion pressure provided by the healthy vascular system(13). The alteration of the above homeostasis will lead to change in oxygenation and micro vascular perfusion adversely affecting the retina(13). The macula is the area of high density of cones and highest oxygen

demand but has a lesser blood flow anatomically and it complies with this early in development. However the neurons in this region may need a little more of their metabolic demand which makes them prone to be affected in ischemic events caused by micro vascular changes in diabetic retinopathy(13). The retinal capillary dropout in diabetics is associated with loss of contrast sensitivity.

The pathogenesis of loss of contrast sensitivity in diabetic macular edema is still not well known. Hyperglycaemia -related changes of the retina causes accumulation of abnormal fluid. The loss of contrast sensitivity was found to be related to with enlargement of the foveal avascular zone.(9) . It might also be due to macular edema causing thickening of the macular region leading to break down of inner retinal barrier at endothelial level(14)

Changes in contrast sensitivity occurs after diabetic macular edema and grid laser therapy however it improved at the last follow-up(9)The contrast sensitivity was found to improve by three months after grid laser which indicate that contrast sensitivity is a better parameter than visual acuity to monitor diabetic macular edema post grid laser patients(9)

VISUAL FIELD

The concept of understanding the visual field is depicted by the island of traquair. The island of Traquair is defined as a “hill of vision” surrounded by a “sea of blindness. The normal shape of the visual field is oval. The sensitivity of the retina decreases with increasing eccentricity in the visual field. The visual field extends 60 ºsuperiorly and nasally, 70 – 75 ºinferiorly and 100 - 110º temporally from the point of fixation with the blind spot represented as a hole in the hill lying 15º temporal to the foveal peak.

Visual field assessment by perimetry is used to measure the sensitivity of light at various locations on the retina. The automated perimetry assess the visual function by static threshold test which applies stimulus at predetermined locations on the visual field based on various programs. The threshold at a specific location is determined by the dimmest stimulus that is visible.

The perimetric sensitivity can be used to measure the paracentral visual function in evaluating the severity of macular edema. A standard central 10 degree programme on Humphrey field analyser can be done and assessed in terms of pattern deviation probability maps(15). The macular edema affects the central 5 to 10 degree of the visual field and also causes depression of the visual field corresponding to the area of macular edema. The 10 degree programme includes 22 test points covering central 10 degree and the test points that was significantly depressed at $p<0.5$ % level were counted and marked as abnormal(16) Visual fields can provide a relationships between the functional loss and structural defect by measuring the sensitivities at points over the visual field. The mean defect and corrected loss of variance can be

used to evaluate the changes in the retinal sensitivity. The goal of our study is to find the effect of grid laser for diabetic macular edema and to compare the before and after visual field changes.

The eyes are tested before grid laser and after 3 months of grid laser so as to minimize the influence of reactive edema and other sequelae of laser on visual fields.

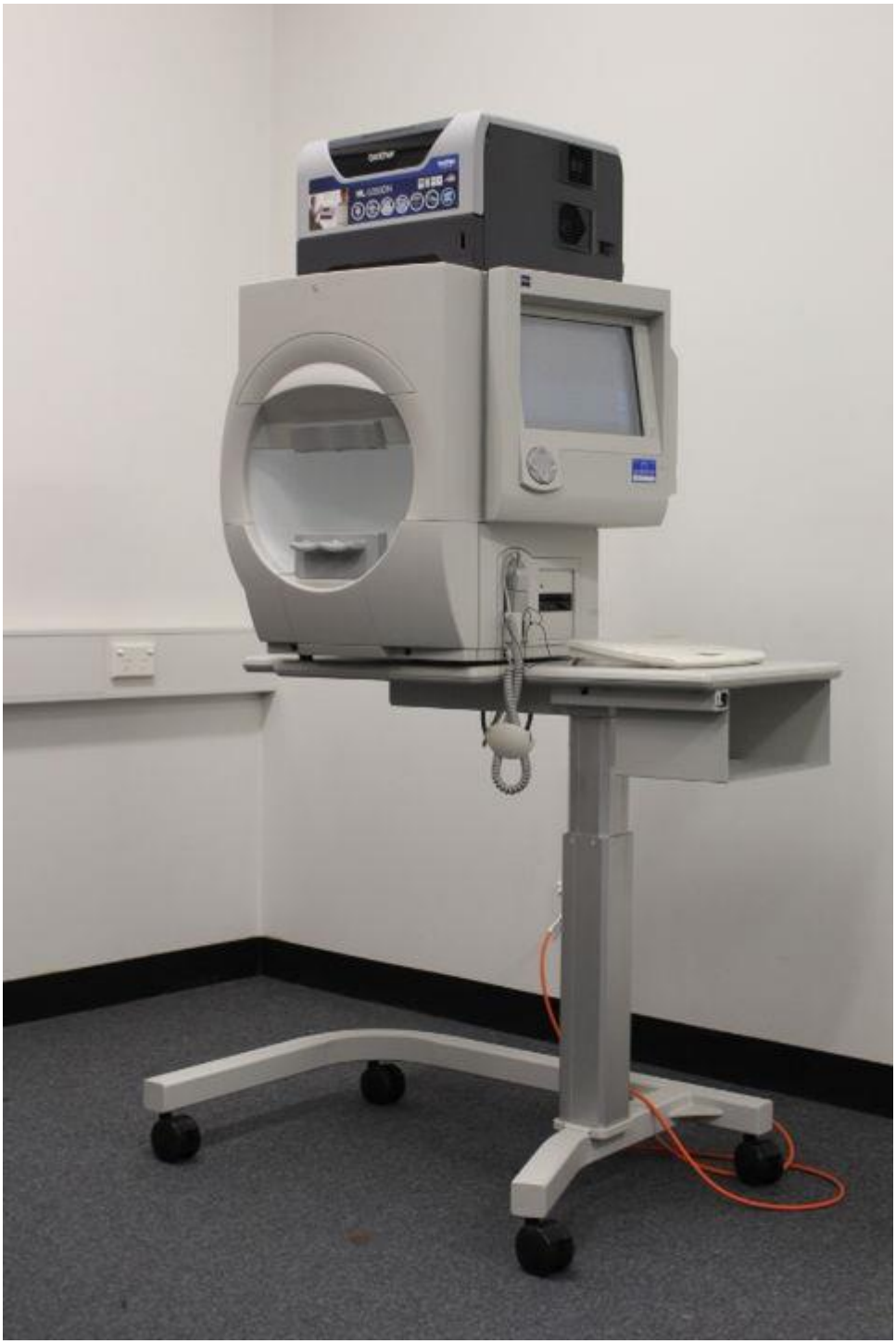
Perimetry can be used to as a functional test in patients with diabetes.

AUTOMATED PERIMETRY

Automated static perimetry was performed with Humphrey field analyser full threshold central 10 program which tests the threshold sensitivity at 22 points within the central 10 degree using Stimulus size 3. The retinal threshold sensitivities are determined at pre determined locations within the central 10 º on a background illumination of 31.5 apostilbs. Goldmann size 3 stimulus is used. The threshold values are corrected for the deviations from age. P values are displayed on the maps marking the location of the defect.

Visual function in diabetes can be assessed with Humphrey field analysis which can be correlated with diabetic retinopathy.

FIGURE 16 : AUTOMATED PERIMETRY



COLOUR VISION

Colour vision is a feature of visual perception, is an ability to perceive differences between light composed of different wavelengths independent of light intensity. Acquired colour vision defects may be an earliest sign of certain diseases and it can also be used to assess the effect of treatment(17)

Theory of colour vision

The perception of colour has individual variations which depend on the wavelength composition of light entering the eye. The mixture of various wavelength of light appears as various colours of different gradations. An apparent change in the colour of the object may occur when the brightness of the surrounding light changes or when the person looks into a brighter light(17).

Based on this

- Short wavelength – blue
- Middle wavelength – green
- Long wavelength – red

Levels of processing of colour

Pre receptoral factors

- Pupil controls the amount of light entering the retina and retinal illuminance which has a small effect on distribution of wavelength. The discrimination of a colour is decreased at low retinal illuminance
- Crystalline lens : The density of crystalline lens varies with age and also there occurs accelerated changes in the lens of diabetic patients

which leads to yellowing of lens and increased absorption of shorter wavelengths

- Macular pigment. The density of macular pigments varies between individuals and with diet and age. Macular pigments are transparent when wavelength is above 540 nm and it drops for wavelength between 420 to 500 nm.

Photoreceptors

Both the rods and cones play an important role in colour vision.

Cones are classified based on their absorption spectra

Short-wavelength–sensitive (S-)
Middle-wavelength–sensitive (M-)
Long-wavelength–sensitive (L-) cones

Post receptoral factors

Post receptoral processes – channels

A red-green channel,
An s-cone channel, and

A luminance channel

Acquired colour vision defects

The changes in the pre receptoral filters such as changes in the pupil size and crystalline lens can decrease the sensitivity of blue light due to increased absorption by lens leading to impaired colour vision.(17) . The changes in RPE can result in red green colour loss known as macular colour defect which may be due to photoreceptor misalignment. This decreases the length the light that has to pass through the photoreceptor resulting in reduced effectiveness of the cone photo pigment and narrowing of absorption spectrum(17).

The loss of colour vision may also be due to damage to a specific cone type or a disruption in the post receptoral processing where there is a damage at the level of inner retina as the vascular diseases that produces hypoxia may produce blue defects(17)

Acquired colour vision defects are classified under 4 categories

Two types of red green defect

- Type 1 red-green defect is caused by macular cone damage
- Type 2 red-green defect is caused by optic nerve involvement
- Blue yellow defect
- Nonspecific defect

Colour vision impairment is about 83.3% in patients with diabetic maculopathy. Impairment of colour vision specifically red green and blue yellow defects occurs in diabetic retinopathy. The red green defects may be due to damage to inner retina whereas blue yellow defects are attributed to photoreceptor damage. The significant association between diabetic macular edema and colour vision impairment is attributable to edema which causes a reduced transmission of light to photoreceptors. A large number of blue coloured defect may be due to lower number of cones in fovea or oblique orientation of cones due to macular edema(18)

The presence of diabetic macular edema is a significant risk factor for impaired colour vision which may be due to inadequate supply of oxygen to photoreceptors leading to death of photoreceptors

Test for colour vision:

- Pseudo isochromatic plates
- Lantern tests
- Arrangement tests
- FM 100-hue test
- D-15 test
- Lanthony tests
- Automated/computerised hue discrimination tests
- Anomaloscopes.
    - Type 3 tritan (often referred to as ‘blue-yellow’) defects congenital tritan defects

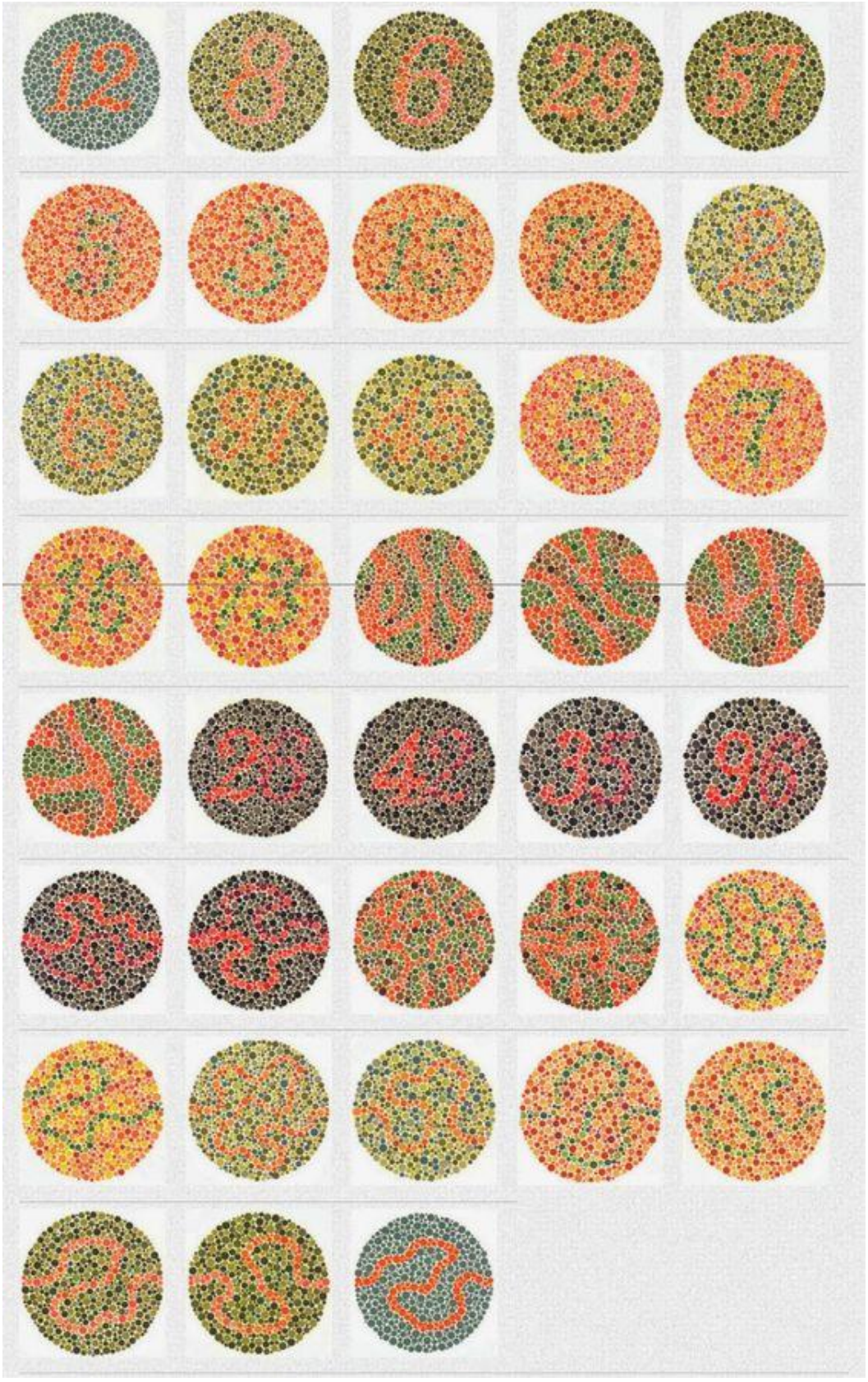
ISHIHARA COLOUR VISION CHART

Ishihara Pseudo-iso-chromatic plate quantitative test

The Ishihara pseudo isochromatic test is the most commonly used test for testing colour vision. It consist of a series of plates where the patient has to name the coloured number from a coloured background(19). The colours in the plate are such that the symbol and the background colour are distinct. the person with normal colour vision will readily identify the numbers whereas a person with defective colour vision will not see any pattern(17). The 25 plates are made in such a way that it can be appreciated adequately in day light. It should be kept at a distance of 75 cm and the plate should be adjusted such that it is at right angle to the line of vision. The patient has to identify the numbers within 3 seconds and traced within 10 seconds(20)

Colour vision in each eye was assessed by using ishihara 24 plates test in which reading from 1- 15 plates determine the normality or defectiveness of colour vision. If the patients read 13 or more than 13 plates the colour vision is recorded as normal. If the 9 or less than 9 plates were read normally, the colour vision is considered as deficient. If 6 or less than 6 plates read normally the colour vision is considered as severely impaired

FIGURE 17 : ISHIHARA COLOUR VISION CHART



LASER

LASER – Light amplification by stimulated emission of radiation

When an electron returns from a high energy level to lower energy level the radiation is emitted as photons.

The characteristics of laser(6)

1. Monochromatic light

2. Spatial coherence

3. High density of electrons

Laser effects(21)

- Photocoagulation- thermal effect.

  This leads to a raise in temperature within the tissues causing denaturalization of proteins and vapourizing of fluid within the tissue leading to cell death.

  - Argon (514.4 green and 488 blue-green nanometres)
  - Krypton (647.1 red nanometres
  - Double-diode (532 nanometres)
  - Double-YAG (532 nanometres)

- Photo disruption- electromechanical effect.

  This type of laser produces a very high power and short duration burst of optical pulse leading to formation of plasma which expands at high temperature and causes acoustic shock wave that disrupts or breaks the target tissue.

- Neodymium YAG (Yttrium-Aluminium-Garnet) - 1064 nanometres- capsulotomy after opacification following cataract surgery, and peripheral iridotomy to prevent risks of acute angle-closure glaucoma.

- Photo chemical

These lasers change the chemical composition and molecular structure of the target tissue before photosensitization which is known as photodynamic therapy. Photosensitization is done with agents like verteporfin which binds to lipoprotein LDL cholesterol and activated by a nonthermal diode laser of 689 nm for 83 seconds, giving a dose of 50 joules/cm2 luminous light intensity of 600mw/cm3. Verteporfin radicals release oxygen on activation and alter the endothelial cells of the ocular blood vessels during this process which leads to platelet aggregation and thrombus formation resulting in occlusion of the vessel.

- Photodecomposition

They emit ultraviolet radiation of 193 nm nanometres in pulses of 10 nanoseconds which interact with the target tissue and destroy the molecular union.

- The Excimer lasers (Argon - Fluoride) used in refractive surgery sculpt the corneal stroma using the photo ablation

Diabetic macular edema is treated by using laser photocoagulation. Initially argon and krypton laser were used but these types of lasers have been changed to solid laser now a days. The Nd YAG laser is located at green 532 nm which is more efficient and effective than the conventional lasers. When compared with conventional laser the YAG laser have higher absorption for

haemoglobin and oxyhaemoglobin and lower absorption by xanthophyll pigment and lesser dispersion.

Grading of laser burns

Grade 1(light) - barely visible, blanching of pigment epithelium

Grade 2(mild) -faint blanching

Grade 3(moderate) - grey, dirty white

Grade 4(severe) - dense chalky white

Two different treatment techniques was defined by ETDRS for treatment of DME

1. Focal laser: focal lesions located between 500 and 3000 µm from the centre of the macula.it consist of 50 to 100 µm burns of moderate intensity and 0.05 to 0.1 second duration and the end point being whitening of the focal lesion.
2. Grid laser

Grid laser for DME

According to the Early Treatment of Diabetic Retinopathy Study (ETDRS), macular photocoagulation (MPC) of the leaking micro-aneurysms and diffuse leaking areas responsible for the development of clinically significant macular edema (CSME) reduces the risk of moderate visual loss.(9). GRID LASER as a treatment for diabetic macular edema results in resolution of the macular edema and reduces the occurrence of moderate visual loss and it also increases the chance of visual improvement in certain circumstances(9). The ETDRS study has showed that grid laser photocoagulation for DME was found to decrease the occurrence of vision loss by 50% at 3 year follow up. The various parameters for laser settings has evolved in

order to reduce the adverse effects resulting from photocoagulation which includes lower laser energy settings and shorter pulse durations which will produce paler laser spots(22)

Mechanism of action of laser in DME

1. LASER destroys the photoreceptors which consumes oxygen so that the oxygen that diffuses from the choriocapillaries reaches the inner retina and relieves the hypoxia without being utilized by the photoreceptors(23)
2. Laser acts on RPE thereby reducing the production of cytokines and vasoactive factors that antagonize the effects of VEGF and also increases the permeability of RPE.(22)
3. Auto regulatory vasoconstriction occurs following grid laser as the laser relieves hypoxia decreases edema(21)
4. RPE responds to laser injury by proliferation and spreading to fill up the RPE defect.(21)
5. RPE repair promotes the metabolism and movement of fluids from the retina to the choroid (22)

Grid laser

a. Spot size- 50 to 200 µm,
b. Duration of 0.05 to 0.5 sec
c. Mild retinal whitening
d. Power adjustment to prevent the burns from spreading to more than 200 µm in diameter
e. The grid laser spot is not applied within 500 µm of the centre of macula and disc margin

FIGURE 18 : POST GRID LASER

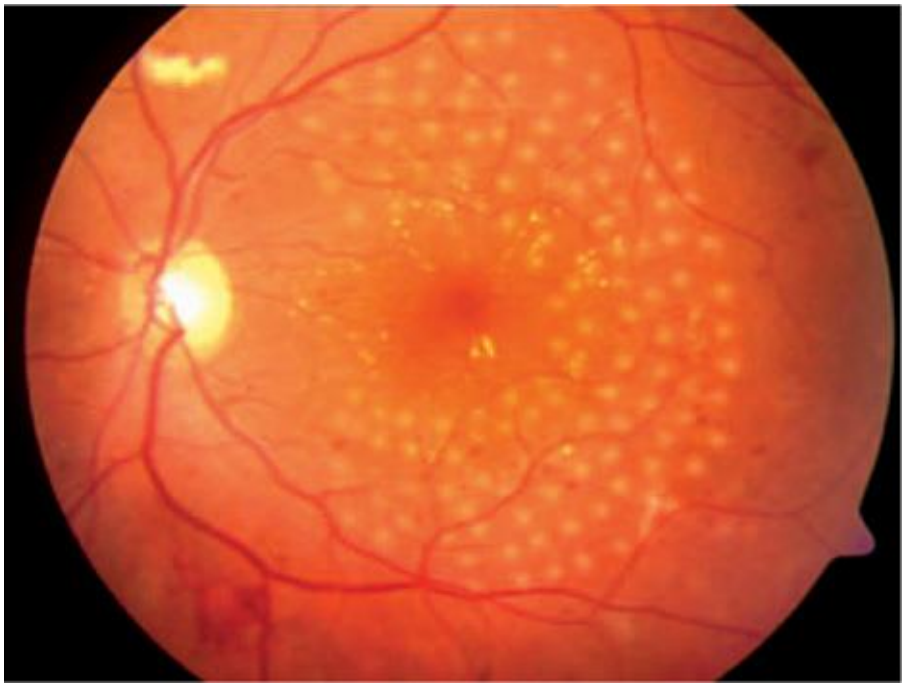


FIGURE 19 : AREA CENTRALIS LENS FOR GRID LASER

Complications of laser photocoagulation

- Atrophic creep

  The laser scar can increase in size and pigmentation and coalesce with each other which might threaten the visual prognosis.

- Choroidal neovascular membrane
- Photophobia
- Appearance of scotomas in the visual field

REVIEW OF LITERATURE

Contrast sensitivity

- Sheldon et al found that Visual acuity and contrast sensitivity showed statistically significant improvement after 4 months following focal/ grid pattern of macular photocoagulation in CSME. The BCVA improved by a line or more in 55.37% of the eyes which was a statistically significant improvement and BCVA remained stable in 31.81% of the eyes and there was a worsening in 12.71% of the eyes. The contrast sensitivity function improved in 10.74% which was a statistically significant improvement and it remained stable in 85.53 % and worsened in 3.71% (24)

- Jahan et al reported that the contrast sensitivity function improved in 24 eyes (68%), worsened in 2 eyes (6%) while it was stable in 9 eyes (26%). The mean contrast sensitivity showed a statistically significant improvement after a period of 6 weeks of laser in patients of diabetic retinopathy(25)

- Shah et al in their study concluded that there was a mean improvement in BCVA and CS by 0.07 log MAR and 0.11 logCS units respectively. The study found that the BCVA improved by 38% and remained stable in 40% and dropped in 23% of the eyes following laser photocoagulation. Contrast sensitivity improved in 61.5 % of the eyes that had undergone focal laser for DME(26)

- Midena et al in his study reported that the Contrast sensitivity improved with a significant difference after 3 months but did not reach normal values after

laser and the visual acuity remained stable in all subjects during follow up visit.(27)

- Talwar et al demonstrated that the argon laser photocoagulation in patients with clinically significant macular oedema shows improvement in contrast sensitivity and stabilization or improvement in visual acuity. The visual acuity remained stationary but did not worsen in any patients.(28)

Visual acuity

- Aiello et al reported that visual acuity improved by >10 letters in 32% of eyes and worsened in 19% of the eyes by 2 years. The eyes that worsened during initial follow up at 4 months also showed improvement subsequently (15)

- Scott et al found that 18% of eyes had a visual acuity improvement of ≥5 letters and 32% had a decrease of ≥5 letters(29)

- Ladas et al found that at the end of one year the visual acuity improved in the treatment group and worsened in the control group. There was a statistically significant difference between the two groups at the end of first and second year and no statistical significant difference at the end of 3 year (30)

- Pei – pei et al demonstrated an improvement in the BCVA at 6 months following grid laser. The BCVA improved in 47.62 % and it remained stable in 42.86 % (22)

Visual fields

- Striph et al studied and demonstrated a slight improvement in visual acuity and no change in colour vision following grid laser. There was no apparent alteration of colour vision after grid treatment. There was no statistically significant change in the foveal threshold following grid laser (31)

- Tababat-khani et al found a few significantly depressed points on visual fields associated with clinically significant macular edema and there was no significant change observed after grid laser. The central retinal sensitivity was not affected by grid laser (16)

- Agardh et al SWAP fields showed a large number of depressed retinal sensitivity points in eyes with macular than in eyes without oedema(15)

- Sims et al in his study reported small scotomas in the visual field in treated regions following focal photocoagulation based on the mean deviation as compared to untreated region which was statistically significant(32)

Colour vision

- Fong et al concluded that the impaired colour vision in diabetic patients were strongly associated with macular edema and the presence of new vessels. The tritan defect was most commonly observed and the severity was associated with severity of macular edema(33)

- Dr Parul M. Danayak et al found that tritanopic defect was found in all diabetic patients which was found to be a statistically significant association (34)

- Green et al; in their study established that the colour vision which is a function of cones is impaired in patients with diabetic retinopathy is more commonly associated with exudative maculopathy (35)

AIM OF THE STUDY

To study the efficacy of grid laser photocoagulation on contrast sensitivity, visual fields and overall visual outcome in patients with diabetic macular edema

OBJECTIVES

- To evaluate contrast sensitivity with pelli robson chart before and after grid laser
- To assess visual fields with humphrey field analyser before and after grid laser
- To evaluate visual acuity after grid laser.
- To assess the changes in colour vision after grid laser

MATERIALS AND METHODS

STUDY POPULATION:

Patients attending ophthalmology OPD at Government Stanley Medical College and Hospital and diagnosed with diabetic macular edema.

STUDY DESIGN: Interventional study

DURATION OF STUDY: March 2020 to November 2021

SAMPLE SIZE:

Based on pre and post-test contrast sensitivity score which has mean difference of (-5.380) +- 19.756(SD). (24)

Sample size is calculated using the formula

$n = € ^2 (Z_{1-\beta} + Z_{1-\alpha/2})^2 / (\text{difference})^2$

$=19.76*19.76*7.84 / 5.4*5.4$

$=105$

Rounded of to 110

INCLUSION CRITERIA

- Patients with non proliferative diabetic retinopathy with clinically significant macular edema
- Visual acuity equal to or better than 6/24

EXCLUSION CRITERIA

- Cataract surgery with complications
- Age related macular degeneration
- Glaucoma
- Proliferative diabetic retinopathy
- Post pan retinal photocoagulation
- Ischemic maculopathy
- Preretinal or vitreous haemorrhage at the time of evaluation
- History of retinal detachment or retinoschisis
- Significant media opacity
- Retinal or other intraocular surgery that could interfere with adequate treatment
- Patients with renal failure

DATA COLLECTION:

A study was conducted at department of ophthalmology, Government Stanley medical college Chennai in patients diagnosed with diabetic macular edema and treated with grid laser during the year 2020 to 2021. The study population included 110 patients with diabetic macular edema posted for grid laser. All the patients were tested for visual acuity, contrast sensitivity, field of vision and colour vision before giving grid laser and post grid laser. The pre and post grid laser parameters will be compared to find the impact of grid laser as treatment modality in patients with diabetic macular edema. The study results will demonstrate the impact of grid laser on visual acuity, contrast sensitivity, visual fields and colour vision in patients with diabetic macular edema.

Materials

Baseline examination includes

- Best-corrected visual acuity with snellen chart
- Slit-lamp examination
- Direct and indirect ophthalmoscopy
- Contrast sensitivity with pelli robson chart
- Humphrey static visual fields
- Ishihara colour vision chart

All patients were followed up post grid laser at 1 week, 1 month and 6 months.

TESTING OF VISUAL ACUITY

Best corrected visual acuity was recorded using self- illuminated snellen visual acuity chart placed at 6 metres from the patient and expressed in snellen fraction and subjective refraction was done simultaneously for each eye and best corrected visual acuity is recorded separately before gird laser and after grid laser at 1 week,1 month and 3 months. The BCVA snellen fraction values were converted to log MAR values for statistical analysis. The gain in visual acuity by one or more line on Snellen chart is considered as improvement and loss of visual acuity by 1 or more line is defined as worsening of visual acuity

TESTING OF CONTRAST SENSITIVITY

The contrast sensitivity was measured with pelli robson contrast sensitivity chart under full refractive correction. The chart is wall mounted under adequate lighting conditions and the patient is placed at 1 metre distance from the chart. The

patient is encouraged to read the letters from above downwards with the patient wearing their best spectacle correction and the outcome is recorded as log values.

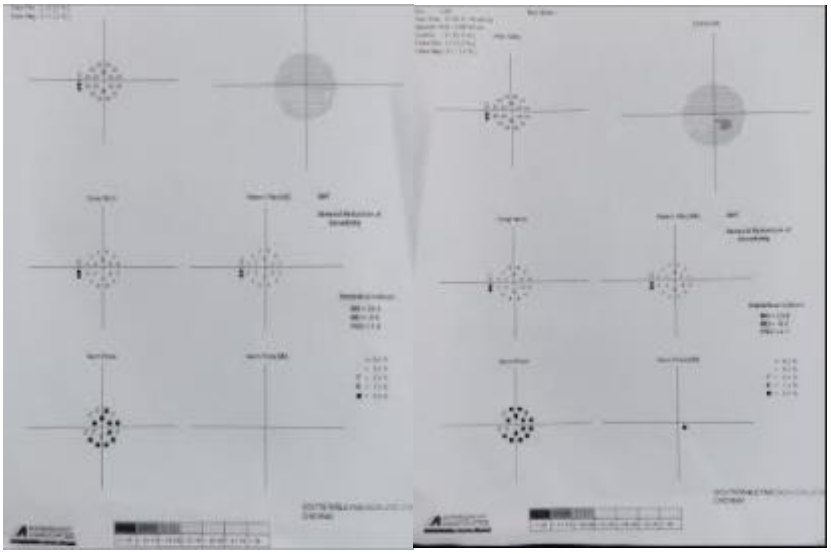
FIGURE 20 : TESTING OF CONTRAST SENSITIVITY

TESTING OF VISUAL FIELDS WITH HUMPHREY FIELD ANALYZER

The visual field was examined by full threshold central 10 program of Humphrey field analyser with full refractive correction. A training session was conducted and patients were thought about the responses they have to record while performing the test in order to minimize the learning effects on the outcome. The parameters recorded are the number of significantly depressed points at $p < 0.5$ % flagged in total deviation probability maps were assessed before grid laser and after grid laser at 3 months. The visual field reports with reliable parameters were taken into account.

FIGURE 21 : EXAMINATION OF VISUAL FILEDS

Figure 22 : PRE AND POST GRID LASER VISUAL FIELDS



TESTING OF COLOUR VISION

The colour vision was tested with Ishihara pseudo isochromatic plates- 38 plate edition. Under good illumination the plates are held at 75 cm from the patient and the patient is asked to read the plates. Based on the number of plates read, the inference is recorded as normal, deficient or severely impaired.

FIGURE 23 : TESTING OF COLOUR VISION

EVALUATION OF THE PATIENT

All the 110 patients included in the study after a clinical diagnosis of clinically significant macular edema were included in the study and a detailed history and ocular examination was done. The complete examination as per the proforma attached is done and recorded.

General management:

- The patient is educated regarding the treatment schedules in order to improve the visual outcomes.
- The patient is also referred to diabetologist to achieve good glycaemic control
- If the patient has other risk factors especially systemic hypertension and hyperlipidaemia, they were adviced to keep it under control.
- The patient is counselled to discontinue smoking.

Grid laser photocoagulation

Preparation of the patient

Written informed consent

Topical anaesthesia

Pupillary dilatation

Procedure

The laser burns are applied to the macular areas of diffuse retinal thickening 500µm away from the foveola superiorly temporally and inferiorly and 500µm from the optic disc.

Duration – 0.05 – 0.1 seconds

Spot size – 50-100 am

Power adjusted to give a mild reaction.

Post procedure

Antibiotic steroid eye drops 4times per day

Timolol eye drops stat

Lubricating eye drops 6 times per day

DATA ANALYSIS

The collected data were entered in Microsoft excel and was analysed using statistical package for social science software (SSPS) version 16 . Data are presented as percentages and the number of cases. Continuous variables were compared using paired sample t test. Categorical data were analysed with Pearson chi-square tests. Significance was defined by P values less than 0.05 using a two-tailed test.

RESULTS

In our study we included 110 eyes of the patients of DME fulfilling the inclusion criteria of which 8 eyes were lost follow up. So 102 eyes out of 110 eyes were included in final analysis. All the patients were given grid laser treatment and followed up at 1 week, 1 month and 3 months. Visual acuity, contrast sensitivity and colour vision were recorded before giving grid laser and after laser at 1 week,1 month and 3 month whereas visual fields were recorded prior to grid laser and after grid laser at 3 months.

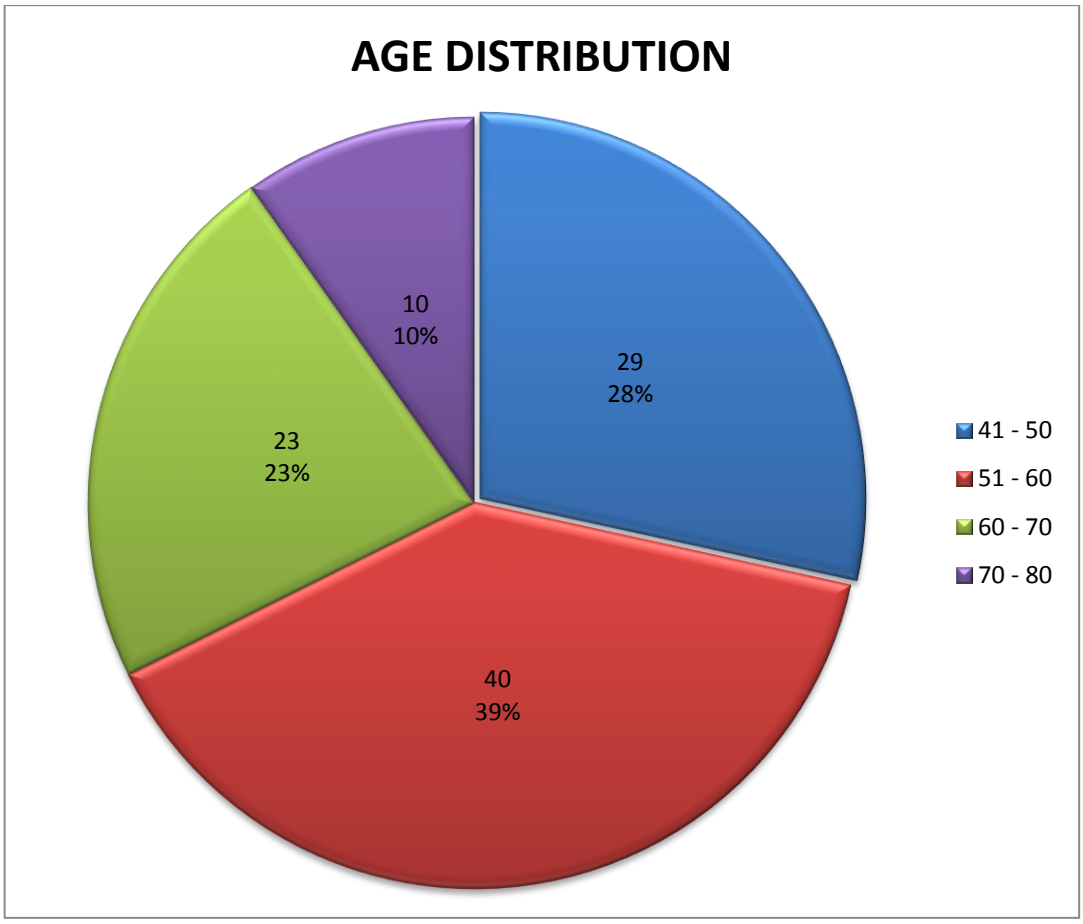
AGE DISTRIBUTION

The following table shows the age distribution in our study

TABLE 2 : DISTRIBUTION OF AGE

AGE	FREQUENCY	PERCENTAGE
41 - 50	29	28
51 - 60	40	39
61 – 70	23	23
71 - 80	10	10
TOTAL	102	100

CHART 1 : AGE DISTRIBUTION

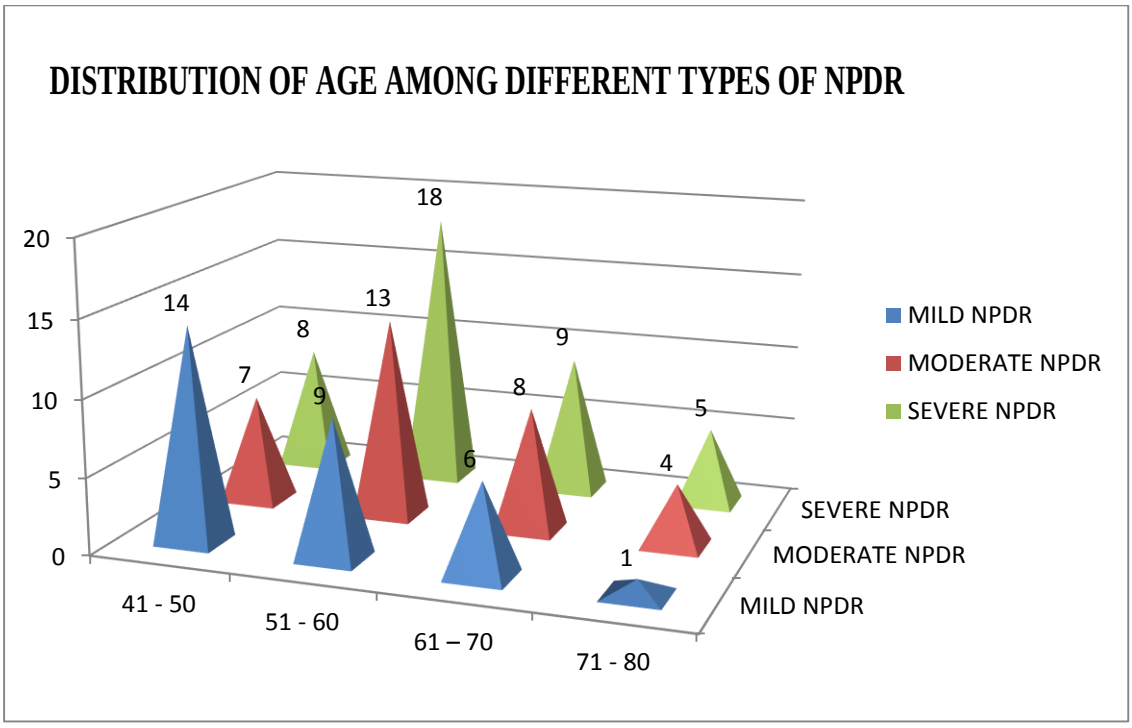


Majority of the patients in the study were in the age group of 51 to 60 years. 29 patients were in the age group 41 to 50 years, 23 patients were in the age group of 60 to 70 years and 10 patients were in the age group of 70 to 80 years. The youngest patient in the study was 41 years old and the oldest patient was 73 years old.

TABLE 3 : DISTRIBUTION OF AGE AMONG DIFFERENT TYPES OF NPDR

AGE	MILD NPDR	MODERATE NPDR	SEVERE NPDR	TOTAL
41 - 50	14	7	8	29
51 - 60	9	13	18	40
61 – 70	6	8	9	23
71 - 80	1	4	5	10
TOTAL	30	32	40	102

CHART 2 : DISTRIBUTION OF AGE AMONG DIFFERENT TYPES OF NPDR



Among the 102 cases of NPDR with CSME 40 cases were in the age group of 51 – 60 years followed by 29 cases in the age group of 41 – 50 years and 23 cases in the age group of 61 – 70 years and 10 cases belong to the age group 71 – 80 years .

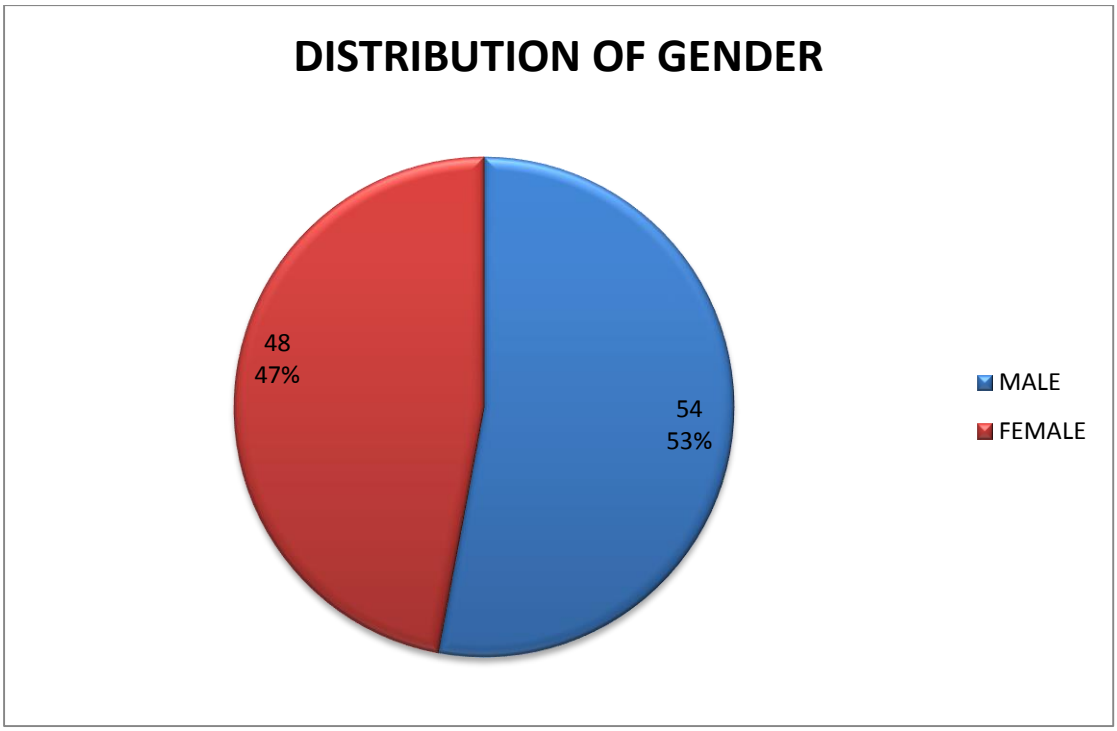
GENDER DISTRIBUTION

The following table shows the gender distribution in this study

TABLE 4 : DISTRIBUTION OF GENDER

SEX	No of patients	Percentage
Male	54	53%
Female	48	47%
TOTAL	102	100

CHART 3: DISTRIBUTION OF GENDER



Majority of the patients were males. 53 % were males and 47% were females in our study

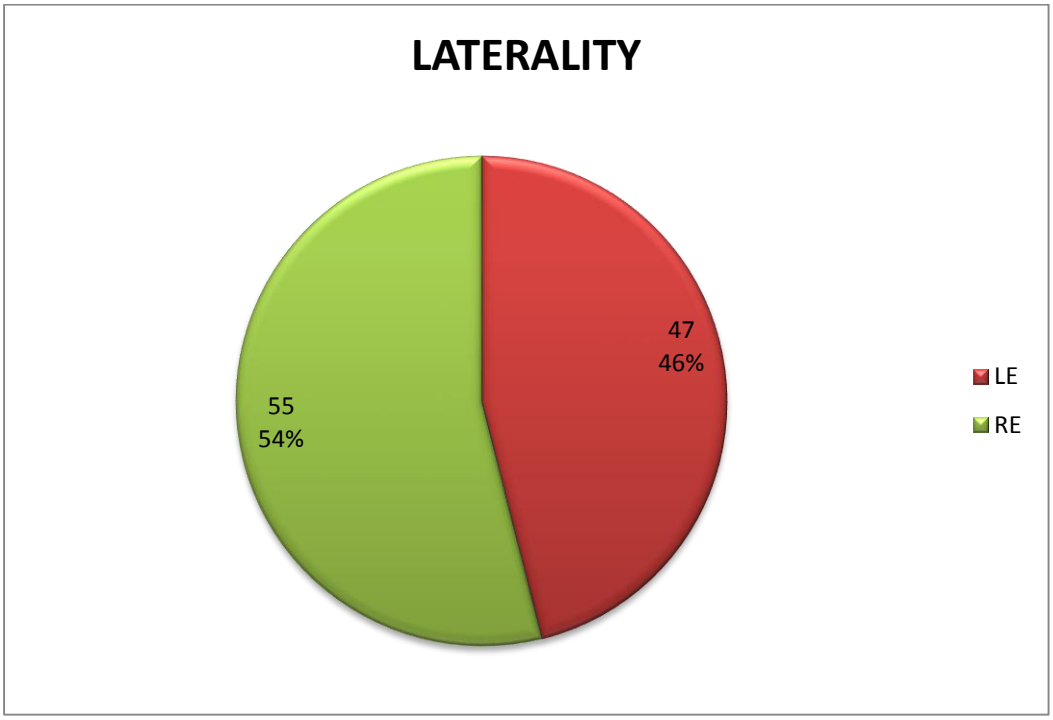
LATERALITY

TABLE 5 : LATERALITY OF PRESENTATION

EYE	Frequency	Percentage
RE	55	46
LE	47	54
TOTAL	102	100

.

CHART 4 : LATERALITY OF PRESENTATION



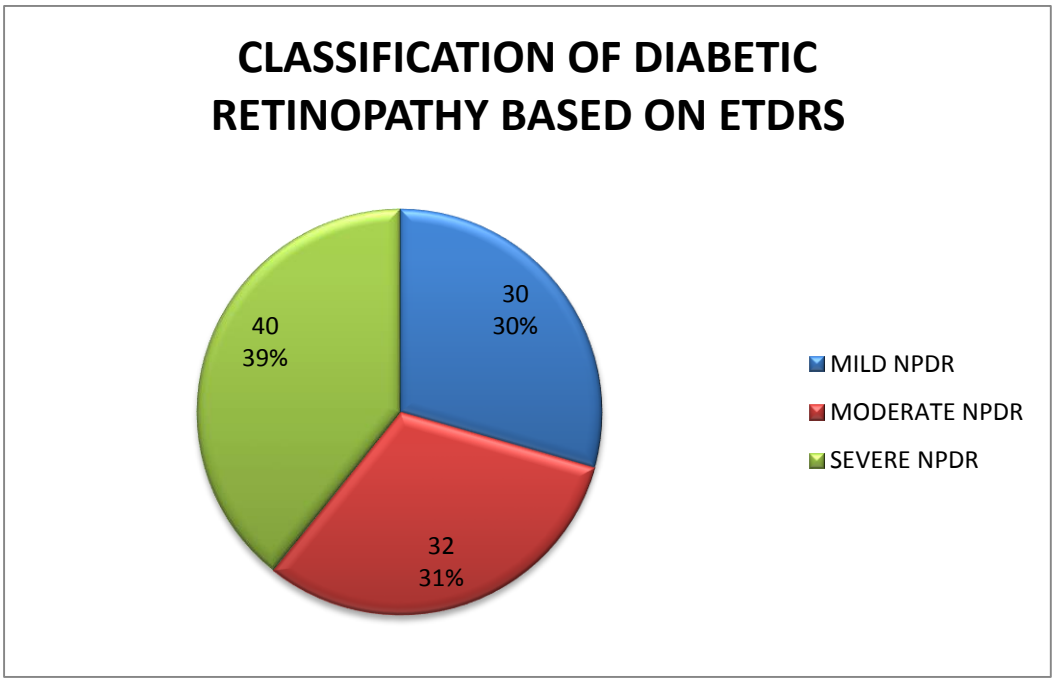
Right eye was affected in 55 cases while in 47 cases left eye was affected.

CLASSIFICATION OF DIABETIC RETINOPATHY BASED ON ETDRS

TABLE 6 : DISTRIBUTION OF VARIOUS TYPES OF NPDR

TYPE	No of patients	Percentage
MILD NPDR	30	29.41
MODERATE NPDR	32	31.37
SEVERE NPDR	40	39.21
TOTAL	102	100

CHART 5: DISTRIBUTION OF VARIOUS TYPES OF NPDR



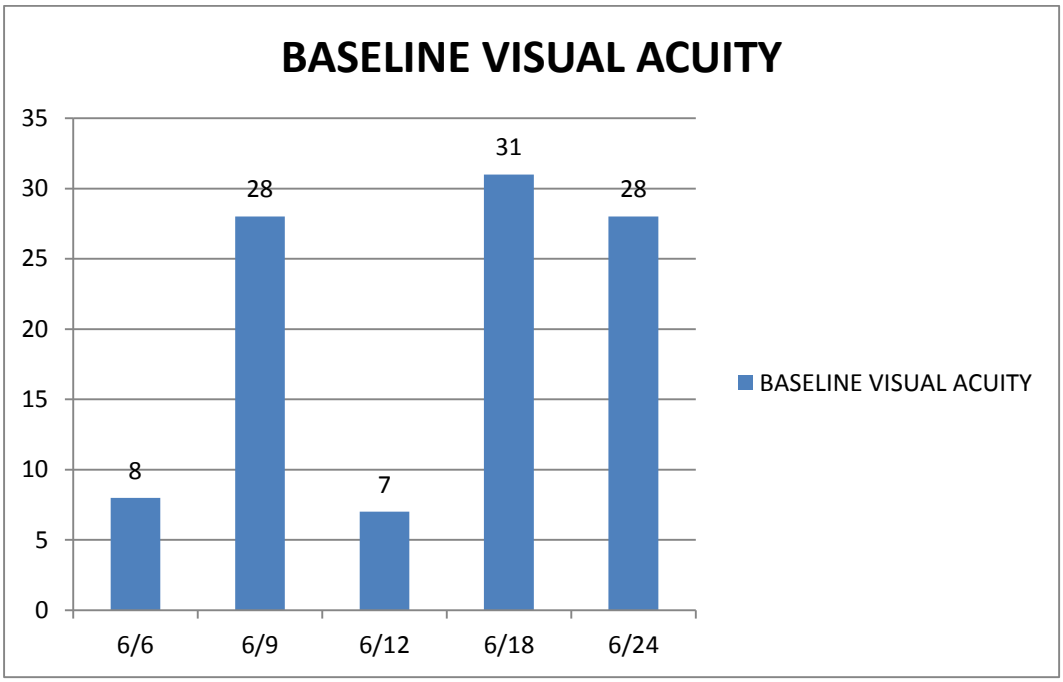
Of the total 102 eyes with CSME included in the final analysis, 30 eyes had mild NPDR, 32 eyes had moderate NPDR and 40 eyes had severe NPDR.

VISUAL ACUITY AT BASELINE

TABLE 7: BASELINE VISUAL ACUITY

VISUAL ACUITY	NO OF PATIENTS	PERCENTAGE
6/6	8	8%
6/9	28	27%
6/12	7	7%
6/18	31	30%
6/24	28	27%
TOTAL	102	100

CHART 6: BASELINE VISUAL ACUITY



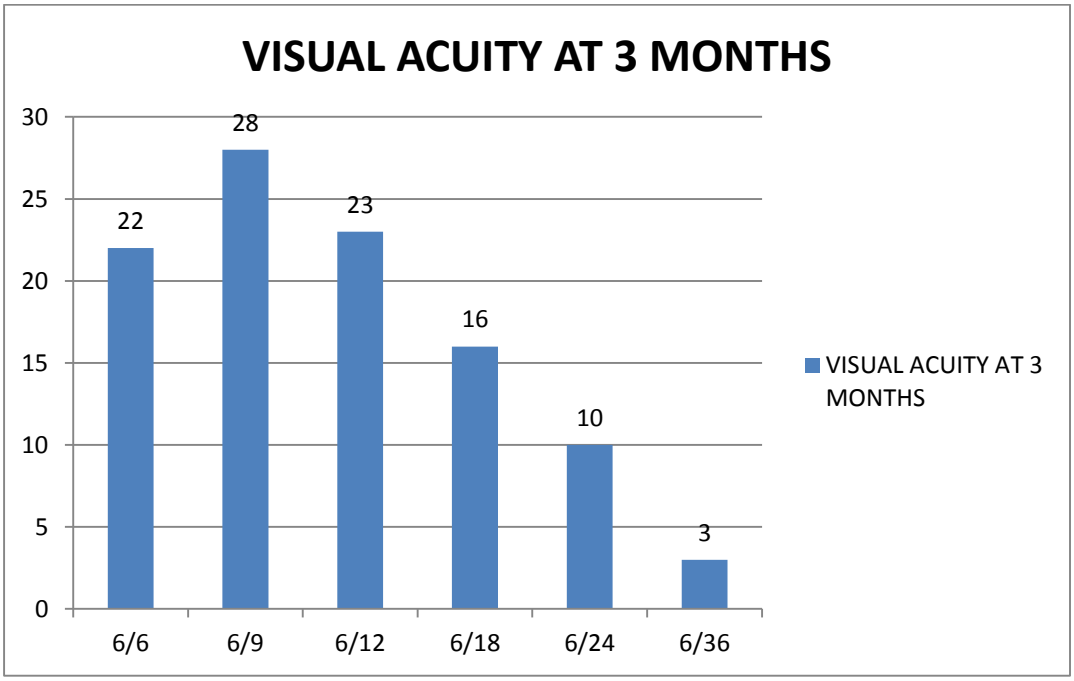
28 out of 102 cases (27 %) had a visual acuity of 6/24 at presentation. 31 cases had visual acuity of 6/18 (30%) , 7 cases had visual acuity of 6/12 (7 %) , 28 cases had visual acuity of 6/9 (27%) and 8 cases had visual acuity of 6/6 (8%).

VISUAL ACUITY AT 3 MONTHS

TABLE 8 : VISUAL ACUITY AT 3 MONTHS

VISUAL ACUITY	NO OF PATIENTS	PERCENTAGE
6/6	22	22%
6/9	28	27%
6/12	23	23%
6/18	16	16%
6/24	10	10%
6/36	3	3%
Total	102	100

CHART 7 : VISUAL ACUITY AT 3 MONTHS



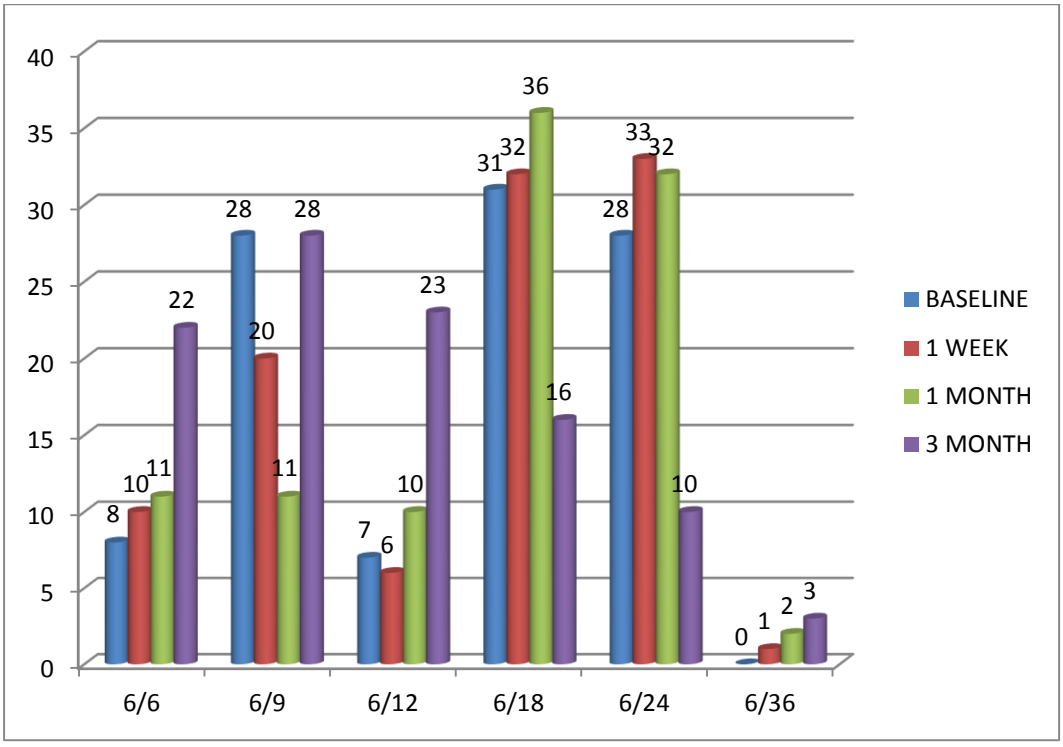
The visual acuity improved to 6/6 in 22 patients out of 102 cases (22%), 6/9 in 28 patients (27%) , 23 patients in 6/12 (23%) , 6/18 in 16 patients (16%) at 3 months after grid laser. The visual acuity remained 6/24 in 10 patients (10%) and worsened to 6/36 in 3 patients (3%)

VISUAL ACUITY BEFORE AND AFTER GRID LASER

TABLE 9 : DISTRIBUTION OF EYES BASED ON VISUAL ACUITY BEFORE AND AFTER GRID LASER

Visual acuity	Base Line	%	1 week	%	1 month	%	3 months	%
6/6	8	8%	10	10%	11	11%	22	22%
6/9	28	27%	20	20%	11	11%	28	27%
6/12	7	7%	6	6%	10	10%	23	23%
6/18	31	30%	32	31%	36	35%	16	16%
6/24	28	27%	33	32%	32	31%	10	10%
6/36	0	0	1	1%	2	2%	3	3%
TOTAL	102	100	102	100	102	100	102	100

CHART 8 : DISTRIBUTION OF EYES BASED ON VISUAL ACUITY BEFORE AND AFTER GRID LASER



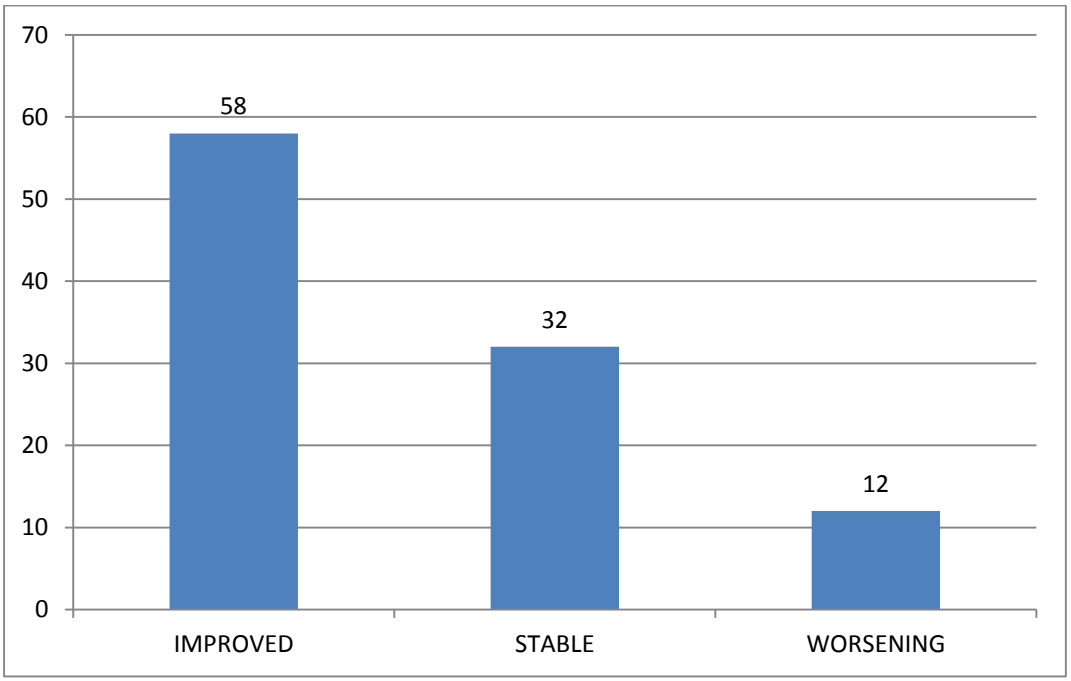
The status of the vision as per snellen visual acuity chart at baseline and after grid laser at 1 week, 1 month and 3 months are given in the table 8

EFFECT OF GRID LASER ON VISUAL ACUITY AT FOLLOW UP VISITS

TABLE 10 : VISUAL OUTCOME AT FOLLOW UP VISITS

BCVA	1 WEEK	1 MONTH	3 MONTH
IMPROVED	4	20	58
STABLE	79	50	32
WORSENING	19	32	12
TOTAL	102	102	102

CHART 9 : VISUAL OUTCOME AT FOLLOW UP VISITS



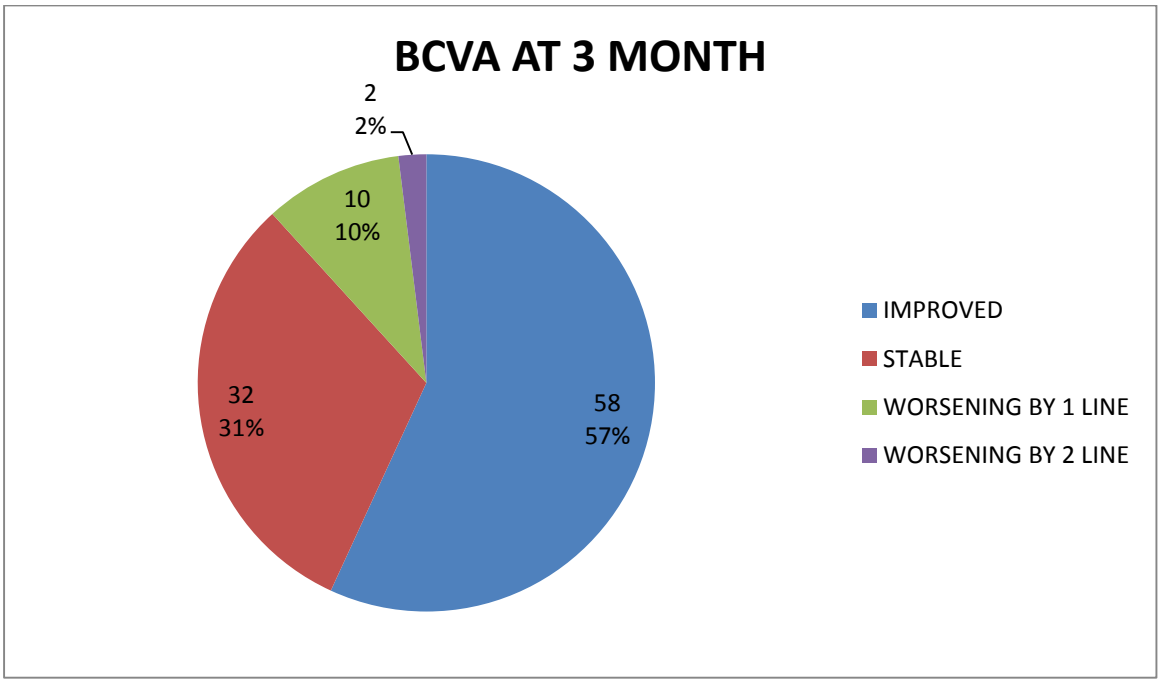
Of the 102 eyes treated with grid laser, 32 eyes had stable vision, 58 eyes had improvement in BCVA by atleast one line and 12 eyes showed worsening by atleast 1 line.

EFFECT OF GRID LASER ON VISUAL ACUITY AT 3 MONTHS

TABLE 11 : BCVA AT 3 MONTHS

BCVA	3 MONTH
IMPROVED	58
STABLE	32
WORSENING BY 1 LINE	10
WORSENING BY 2 LINE	2
Total	102

CHART 10 : BCVA AT 3 MONTHS



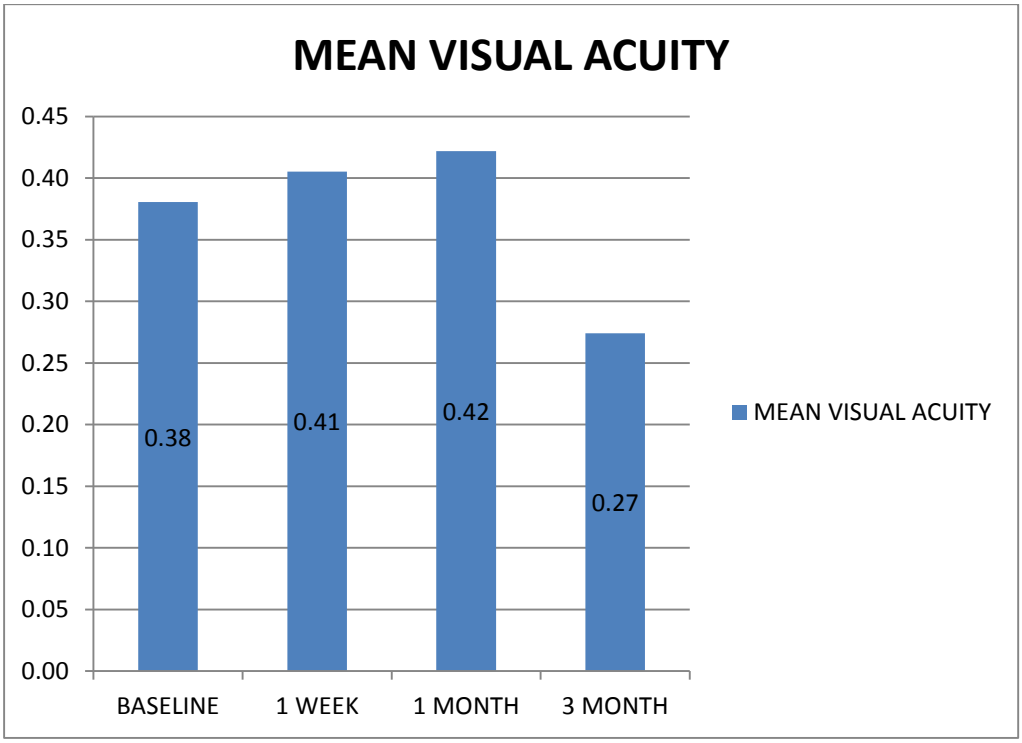
The BCVA worsened by 1 line in 10 eyes and 2 lines in 2 eyes at 3 months of follow up.

STATISTICAL ANALYSIS OF VISUAL ACUITY OUTCOME

TABLE 12 : RESULTS OF PAIRED T-TEST FOR PRE AND POST LASER BCVA

Pair	Mean difference after laser	SD	95% confidence interval		P value
Pre and Post Laser BCVA	0.11	0.17	0.08	0.14	<0.0001 STATISTICALLY SIGNIFICANT

CHART 11 : MEAN VISUAL ACUITY BEFORE AND AFTER GRID LASER



The mean BCVA was 0.38 at baseline and the mean BCVA at 3 months after the grid laser was 0.27. A paired t-test was done to compare the BCVA before and after the grid laser and it was found to be a statistically significant improvement with p value <0.001

CONTRAST SENSITIVITY

TABLE 13 : CONTRAST SENSITIVITY OUTCOME AT FOLLOW UP VISITS

CONTRAST SENSITIVITY	1 WEEK	1 MONTH	3 MONTH
IMPROVED	2	5	46
STABLE	97	93	40
WORSENING	3	4	16
TOTAL	102	102	102

CHART 12 : CONTRAST SENSITIVITY AT 1 WEEK

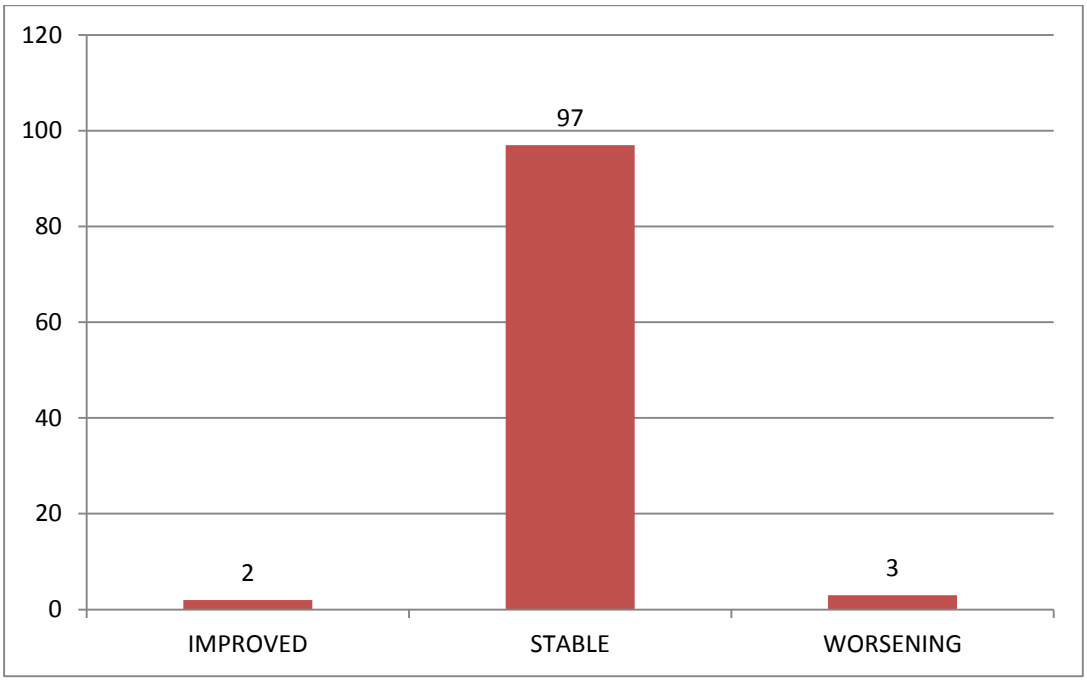
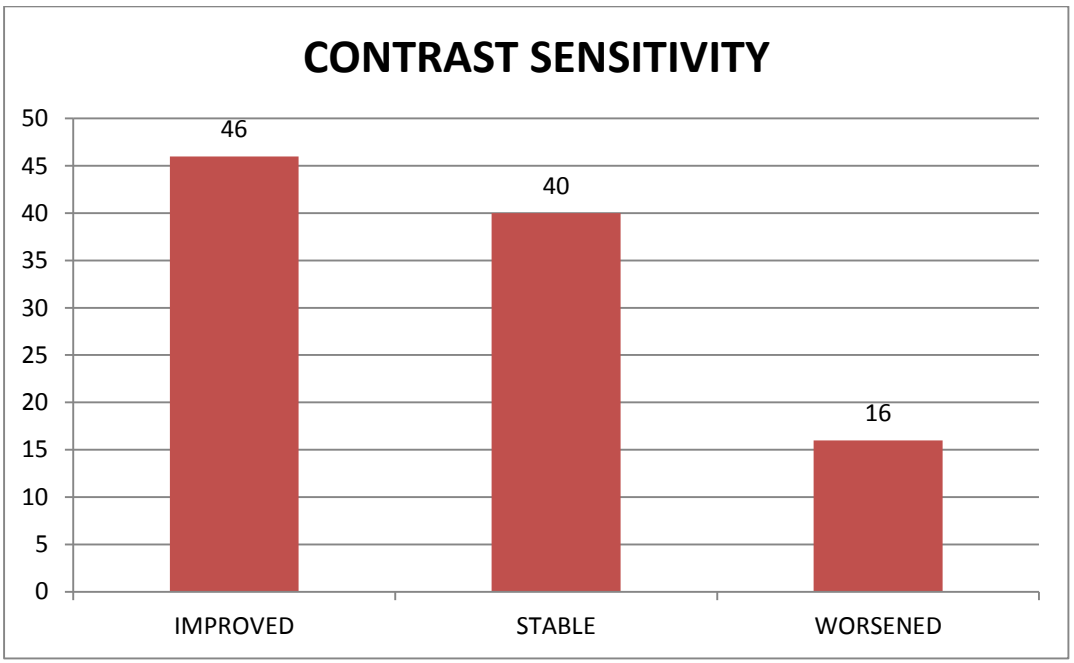


CHART 13 : CONTRAST SENSITIVITY AT 1 MONTH

CHART 14 : CONTRAST SENSITIVITY AT 3 MONTHS



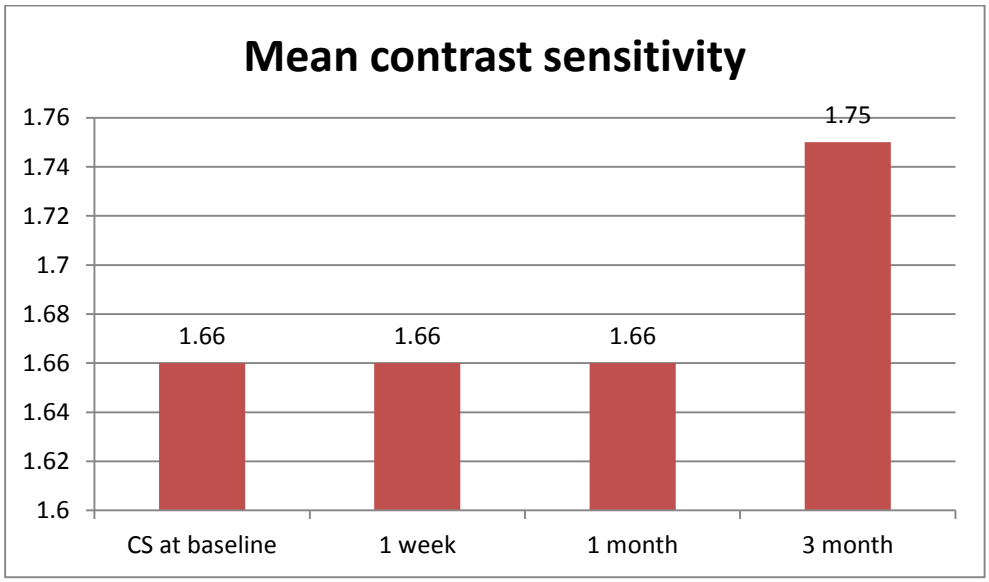
At the end of 3 months, 46 eyes showed an improvement in contrast sensitivity, 40 eyes maintained stable contrast sensitivity and 16 eyes showed worsening of contrast sensitivity.

STATISTICAL ANALYSIS OF CONTRAST SENSITIVITY FUNCTION

TABLE 14 : RESULTS OF PAIRED T-TEST FOR PRE AND POST LASER CONTRAST SENSITIVITY

Pair	Mean difference after laser	SD	95% confidence interval		P value
Pre and Post Laser CS	0.09	0.22	0.05	0.13	< 0.0001 STATISTICALLY SIGNIFICANT

CHART 15 : RESULTS OF PAIRED T-TEST FOR PRE AND POST LASER CONTRAST SENSITIVITY



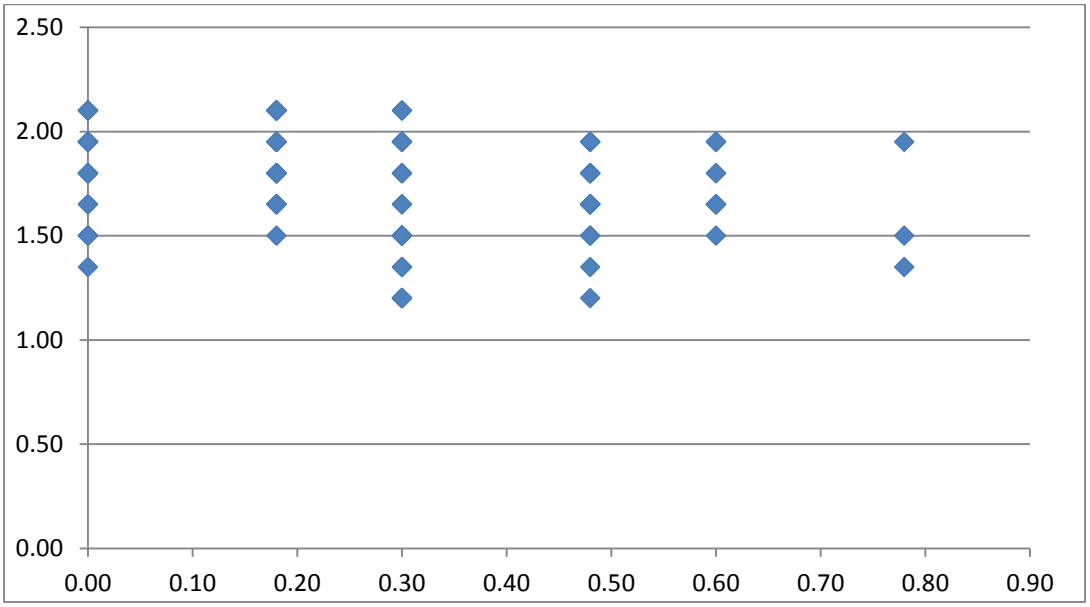
Contrast sensitivity was recorded at baseline and 3 months after grid laser as log MAR units. The mean contrast sensitivity score at baseline was 1.66 and at 3 months was 1.75. A paired T-test was performed to compare the pre laser and post laser values and found to be a statistically significant improvement with $p<0.001$.

CORRELATION BETWEEN VISUAL ACUITY AND CONTRAST SENSITIVITY AT 3 MONTHS AFTER GRID LASER

TABLE 15 : CORRELATION BETWEEN VISUAL ACUITY AND CONTRAST SENSITIVITY

	Mean visual acuity at 3 months	Mean contrast sensitivity at 3 months	Correlation co-efficient	P value
Correlation between visual acuity and contrast sensitivity	0.27	1.75	-0.24416	0.013395 **Statistically insignificant**

CHART 16 : CORRELATION BETWEEN VISUAL ACUITY AND CONTRAST SENSITIVITY



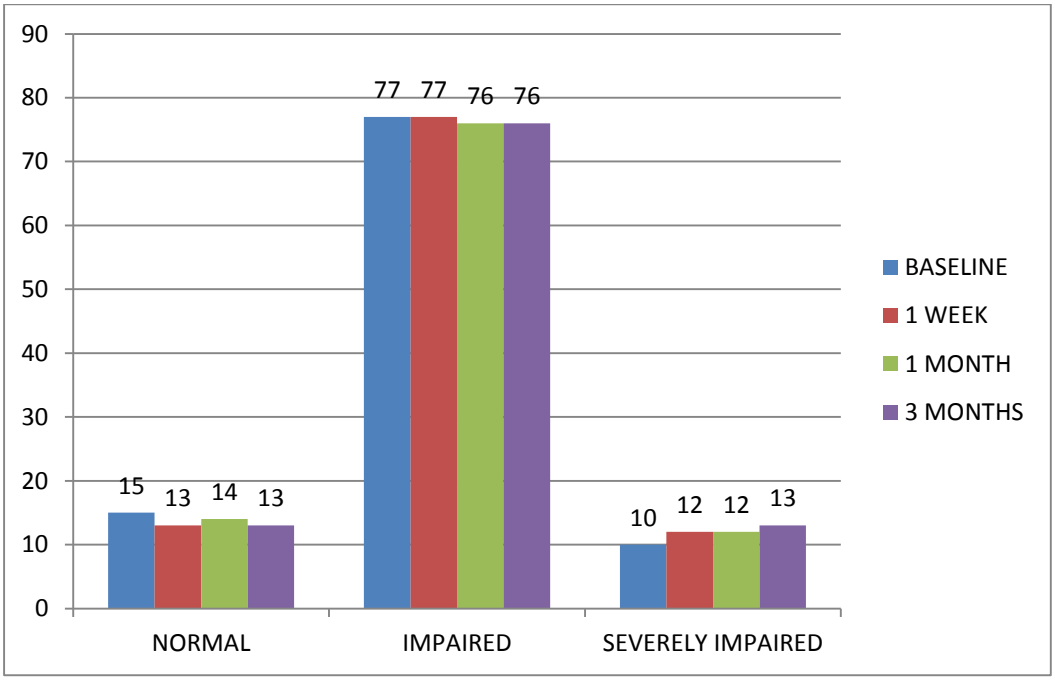
The scatter diagram shows no correlation between the visual acuity and contrast sensitivity at 3 months. The correlation co-efficient was found to be -0.24416 which shows that there is no positive correlation between the visual acuity and contrast sensitivity at 3 months after grid laser. The p value was calculated to be 0.013395 which indicate that there is no statistically significant association between the visual acuity and contrast sensitivity at 3 months after grid laser

COLOUR VISION

TABLE 16 : DISTRIBUTION BASED ON COLOUR VISION

	NORMAL		DEFICIENT		SEVERELY IMPAIRED	
BASELINE	15	15%	77	75%	10	10%
1 WEEK	13	13%	77	75%	12	12%
1 MONTH	14	14%	76	75%	12	12%
3 MONTH	13	13%	76	75%	13	13%

CHART 17 : DISTRIBUTION OF PATIENTS BASED ON THE COLOUR VISION



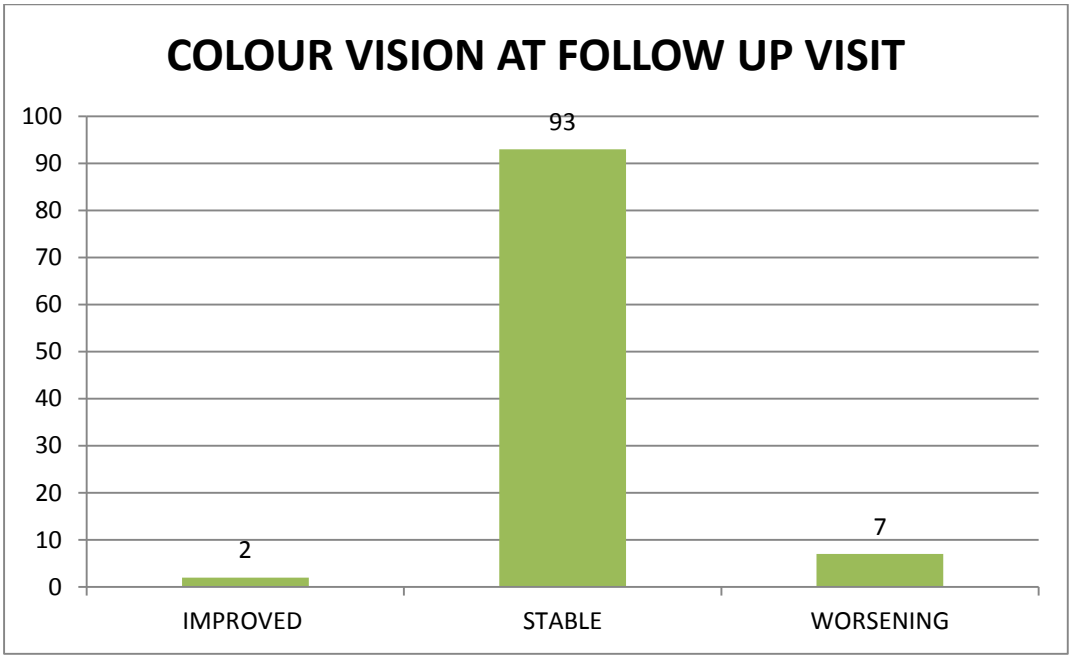
The colour vision was impaired and severely impaired in most of the patients at the baseline and at 3 months but there was no statistically significant change in the colour vision before and after grid laser.

EFFECT OF GRID LASER ON COLOUR VISION

TABLE 17 : COLOUR VISION AT 3 MONTHS

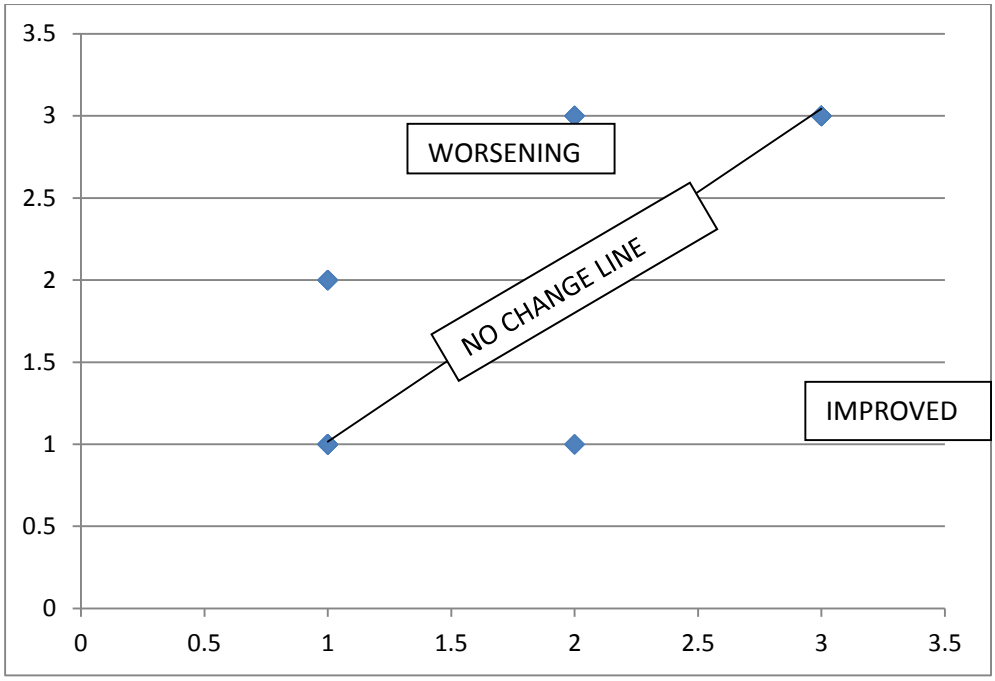
COLOUR VISION	1 WEEK	1 MONTH	3 MONTH
IMPROVED	0	0	2
STABLE	98	99	93
WORSENING	4	3	7
TOTAL	102	102	102

CHART 18 : COLOUR VISION AT 3 MONTHS



The colour vision improved from baseline in 2 patients, remained stable in 93 patients and worsened in 7 patients at 3 months following grid laser.

CHART 19 : CHANGES IN THE COLOUR VISION BEFORE AND AFTER GRID LASER AT 3 MONTHS



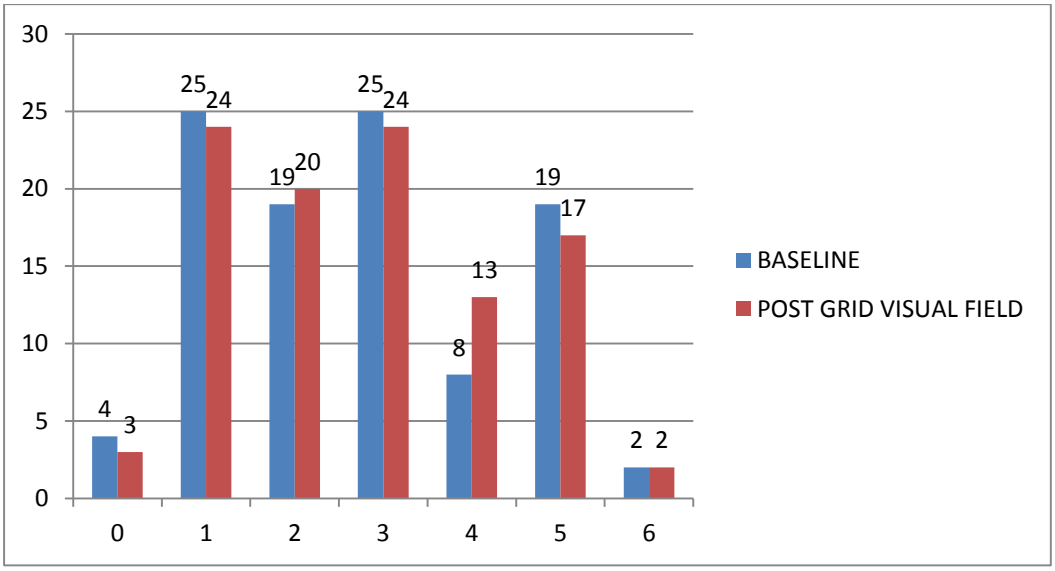
The majority of points fall on the “no change “line which indicate that the colour vision remains stable in most of the patients.

VISUAL FIELDS

TABLE 18 : VISUAL FIELD CHANGES BEFORE AND AFTER GRID LASER

No of depressed points on visual field	Baseline	3 months
0	4	3
1	25	24
2	19	19
3	25	24
4	8	13
5	19	17
6	2	2

CHART 20 : VISUAL FIELD CHANGES BEFORE AND AFTER GRID LASER



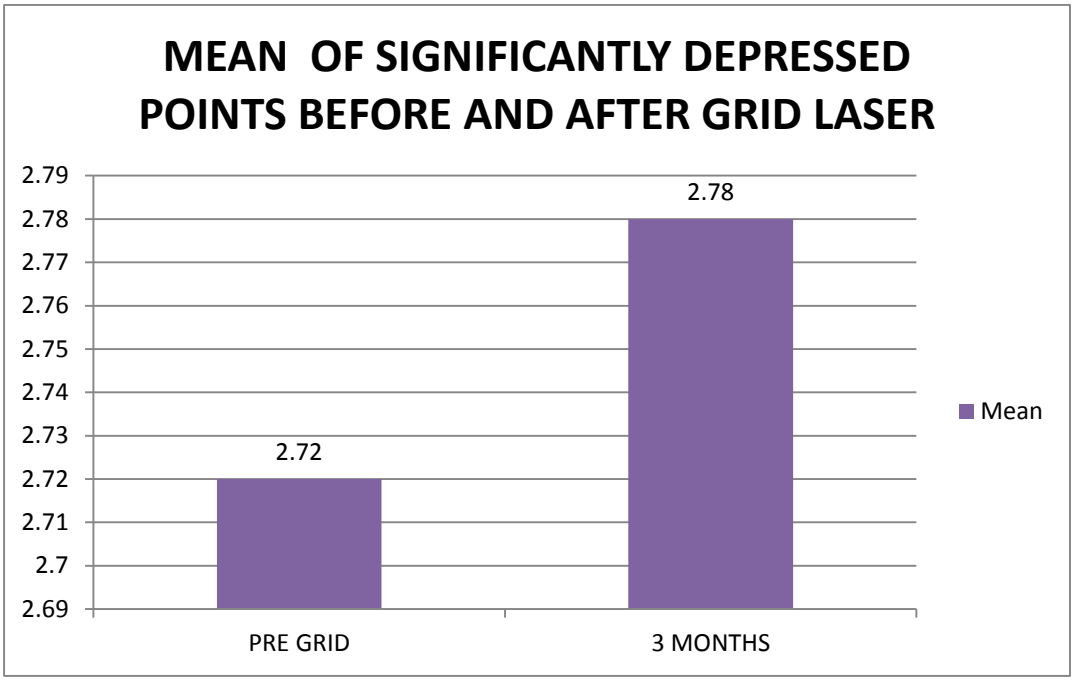
There were 4 patients with no significantly depressed points and 2 patients with a maximum of 6 significantly depressed points at baseline. After grid laser there was 3 patients with no significantly depressed points and 2 patients with 6 significantly depressed points. The number of significantly depressed points varies from 0 to 6 depressed points.

STATISTICAL ANALYSIS OF VISUAL FIELD CHANGES BEFORE AND AFTER GRID LASER

TABLE 19 : RESULTS OF PAIRED T-TEST FOR PRE AND POST LASER VISUAL FIELD CHANGES

VISUAL FIELD	Mean	Std. Deviation	P value
PRE GRID	2.72	1.57	0.195
3 MONTHS	2.78	1.52	

CHART 21 : RESULTS OF PAIRED T-TEST FOR PRE AND POST LASER VISUAL FIELD CHANGES



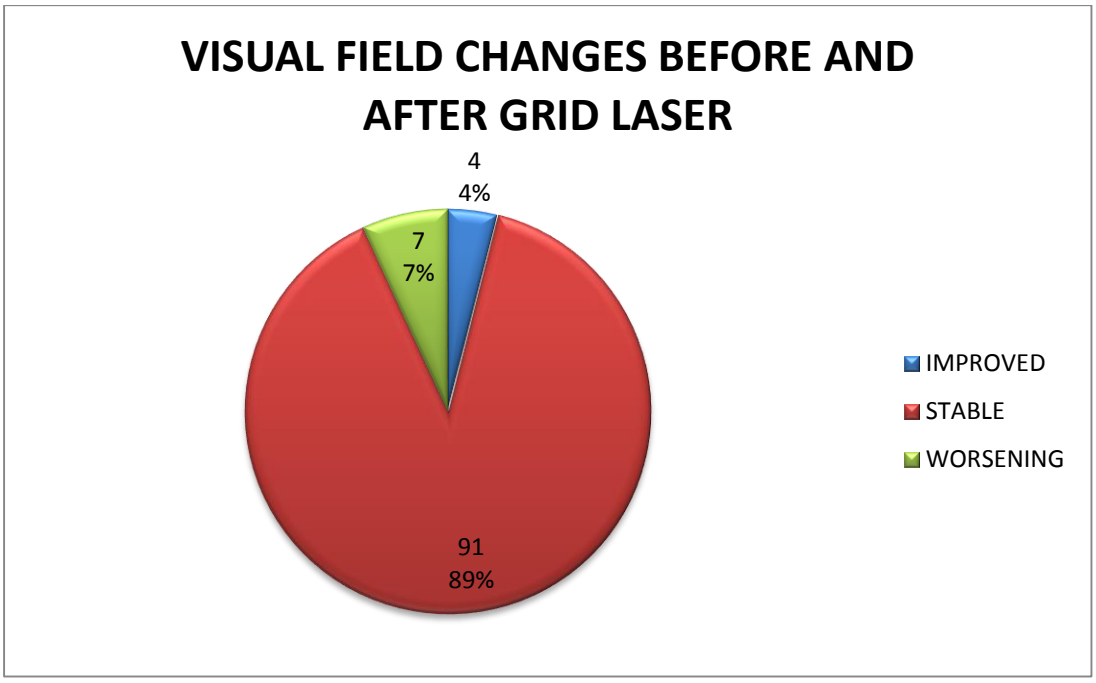
The mean depressed points on the visual field at baseline were 2.72 and at 3 months were 2.78. The mean difference was 0.06 between the pre grid laser and post grid laser values. There was no statistically significant improvement after the grid laser in visual fields.

VISUAL FIELD OUTCOME AFTER GRID LASER

TABLE 20 : VISUAL FIELD CHANGES AFTER GRID LASER

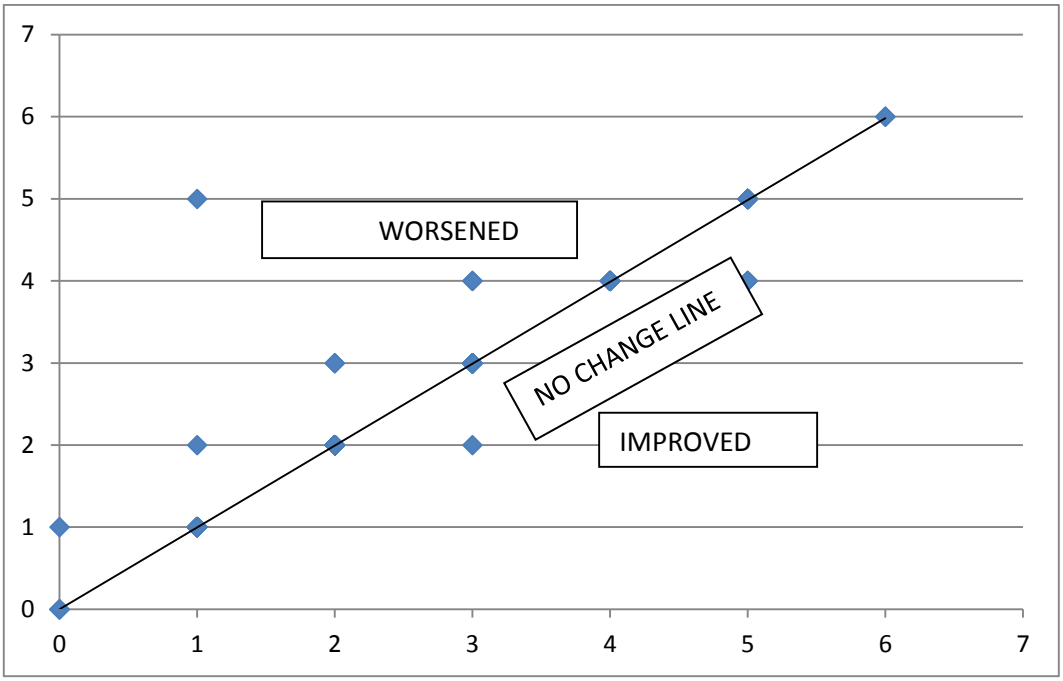
VISUAL FIELD	3 MONTH
Improved	4
Stable	91
Worsening	7

CHART 22 : VISUAL FIELD CHANGES AFTER GRID LASER



The number of significantly depressed points decreased in 4 patients, remained stable in 91 patients and increased in 7 patients.

CHART 23 : DEPRESSED POINTS ON VISUAL FIELD BEFORE AND AFTER GRID LASER



Scatter diagram shows the depressed points on the visual field before and after grid laser. Majority of points fall on the “no change line”.Most of the patients had no change in the number of depressed points before and after grid laser

DISCUSSION

Diabetic macular edema is the most common cause of loss of central vision in patients with diabetic retinopathy. Laser photocoagulation and intensive glycaemic control are the demonstrated means of reducing the risk of visual loss in patients with DME as reported by various studies.

The study was conducted in 110 eyes of 110 patients with Non proliferative diabetic retinopathy with CSME of which 102 eyes were included in the final analysis. All the patients were treated with grid laser and followed up at 1 week, 1 month and 3 months after the grid laser.

In our study the age group of the patients included in the study ranged from 41 to 73 years. The majority of the patients were in the age group of 51 to 60 years. The mean age of presentation was 57.58 years. This is similar to Seo and Park et al study which reported the mean age of 57.9±13.8 years(36) . In jahan et al study, the mean age of patients was reported as 58.11 ± 4.96 years. 28 % of the patients were in the age group of 41 – 50 years, 39 % of the patients were in the age group 0f 51 – 60 years, 23 % of the patients were in the age group of 61 to 70 years and 10 % of the patients were in the age group of 71 to 80 years. None of the patients in our study were in the age group of 1 to 40 years

In our study the males represented 53% with a peak incidence at 51 to 60 years and females represented 47 % which showed a mild male preponderance. This is in concordance with Jahan et al study which showed that 56% patients were male and 44% of the patients were female(25)

In 46 % of the cases right eye was affected and 54 % of the cases left eye was affected. In our study the left eye was affected in more number of patients.

Severe NPDR was predominantly present in 39 % of the patients whereas mild and moderate NPDR was present in 29% and 31% of the patients respectively.

In the study we found that around 58 eyes treated with grid laser had an improvement in BCVA by atleast 1 line when compared with the baseline at the end of 3 months. About 32 eyes had stable vision whereas there was a drop in vision in 12 eyes of which 10 eyes had worsening by 1 line and 2 eyes by 2 lines. The mean difference between the BCVA at baseline and 1 week and 1 month is -0.3 and -0.4 log MAR units respectively which show that there was no statistically significant change in the BCVA at 1 week and 1 month. However the mean BCVA at baseline was 0.38 at baseline and 0.27 log MAR units at 3 months after grid laser. The mean difference was 0.11 log MAR units which was found to be statistically significant by applying a paired T-test with a P value <0.0001. This is similar to shah et al study which reported a net improvement in the BCVA of about 0.07 log MAR units at 3 months following retinal laser for diabetic macular edema(26)

In our study we found that the contrast sensitivity function improved in 46 eyes, remained stable in 40 eyes and worsened in 16 eyes. The mean contrast sensitivity at baseline was 1.66 and at the end of 3 months it was 1.75. The mean difference between the pre laser and post laser contrast sensitivity at 3 months was 0.09 log units. The difference was found to be a statistically significant improvement by applying a paired T test with P value <0.0001. There was no statistically significant change in the contrast sensitivity function at 1 week and 1 month. Jahan et al in his study reported a statistically significant improvement in the contrast

sensitivity function in 68 % of the eyes which was similar to our study(25). Shah et al reported a significant improvement in the contrast sensitivity function in 49.1 % of the eyes who have undergone laser(26)

In our study it was found that there was no significant positive correlation between the visual acuity and colour vision at 3 months after grid laser with P value 0.0133 which indicate a that there is no statistically significant correlation.

At baseline the colour vision was normal in 15 %, where as it was deficient in 75% and severely impaired in 10 %. At 3 months after grid laser, the colour vision was normal in 13 % whereas it was deficient in 75% and severely impaired in 13 %. The colour vision remained stable in 93 patients, improved in 2 patients and worsened in 7 patients .There was no apparent change in the colour vision in patients before and after grid laser. Most of the patients began with a colour vision defect and ended up in the same. The change between the pre and post grid laser values was found to be statistically insignificant. Striph et al in his study confirmed the presence of tritan defect in patients with diabetic macular edema and also reported that the grid laser therapy doesn't alter the colour vision defect(31)

The mean difference between the number of significantly depressed points on the visual field in the central 10 degrees at the base line and at 3 months was 0.06 which was found to be statistically insignificant which indicate that there was no significant improvement or worsening of the depressed points on the visual fields before and after grid laser . At the baseline, 4 patients had no depressed points and 2 patients had a maximum of 6 depressed points. At the end of 3 months, 3 patients still had no depressed points. The number of significantly depressed points decreased in 4 patients, remained stable in 91 patients and increased in 7 patients after grid laser.

Tababat – khani et al concluded that grid laser photocoagulation for CSME did not affect the retinal sensitivity after the treatment which is similar to our study. Most of the points on the scatter diagram chart 23 lie on the “no change line” which indicate that majority of the patients did not have any change in the number of depressed points on the visual field. The P value between the pre and post visual field changes was found to be 0.195 which indicate that there is no statistically significant change.

SUMMARY

A study was conducted at department of ophthalmology, Government Stanley medical college, Chennai – 01 in 102 patients diagnosed with diabetic macular edema. The patients were treated with grid laser and the pre and post grid visual parameters were recorded and compared to test the effect of grid laser on the overall visual outcome.

- In our study, the age of the patients presenting with diabetic macular edema ranged between 41 to 73 years with a mean age being 57.58 years.
- There is a male preponderance of 53 %
- The most common type of NPDR in our study is severe NPDR which accounts to 39.21 % followed by moderate NPDR that accounts to 31.37 % and mild NPDR which accounts for 29.41 %
- Out of 102 eyes treated with grid laser, 58 eyes had improvement in BCVA by atleast 1 line, 32 eyes had stable vision and 10 eyes showed worsening by 1 line 2 eyes had worsening by 2 lines
- The mean difference between the BCVA at baseline and after 3 months of grid laser is 0.11 log MAR units which was found to be a statistically significant improvement.
- A correlation co-efficient of - 0.244 with a P value of 0.133 indicate that there is no significant positive correlation between the visual acuity and contrast sensitivity at 3 months after grid laser

- Contrast sensitivity function improved in 46 eyes , remained stable in 40 eyes and worsened in 16 eyes at 3 months after grid laser when compared with baseline

- The mean difference between the contrast sensitivity at baseline and 3 months is 0.09 log units with a P value of <0.0001 which was found to be a statistically significant improvement.

- The colour vision was deficient and severely impaired in most of the patients at baseline and there was no significant change after grid laser

- The effect of grid laser for CSME on the central 10 º of visual fields appears to be a statistically insignificant change.

CONCLUSION

Our study showed that grid laser for CSME plays a definitive role in improving visual acuity as well as contrast sensitivity function. There was a statistically significant improvement in contrast sensitivity and visual acuity at 3 months which are independent of each other. Contrast sensitivity and visual acuity assessment can be used as a sensitive tool to pick up subtle changes in visual outcome and it may also serve as a sensitive indicator to assess the effect of grid laser on diabetic maculopathy. Contrast sensitivity measured with pelli robson chart can also be used widely for routine examination in patients with diabetic macular edema as it can offer important information about the effect of grid laser on the visual outcome and it also measures the visual outcome better than the uniformly high contrast snellen visual acuity chart. The colour vision defect was found in 85 % of the patients with diabetic macular edema before the grid laser and there was no statistically significant change in the colour vision after 3 months of grid laser. We found that the grid laser does not significantly affect the retinal sensitivity. The tools used in this study to assess the effect of grid laser may be used to assess and modify the intensity and duration of laser burn in grid as well as focal retinal or macular lasers so that the optimum intensity and optimum duration can be ascertained. The main limitation of our study is a small sample size and limited study duration.

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PROFORMA

Serial no. :

Name :

Age :

Sex :

Occupation :

Address :

Ocular complaints :

History of disease :

Treatment history :

Duration of treatment :

Drug history :

Ass. Systemic illness :

Family history :

BASELINE CLINICAL EXAMINATION:

Blood Pressure :

BASELINE OCULAR EXAMINATION

	RE	LE
UCVA		
BCVA		
Eyelids and lashes		
Extraocular movements		
Slit lamp examination		
Conjunctiva		
Cornea		
Anterior chamber		
Iris		
Pupil		
Lens		
EOM		
Fields		
Colour vision		
Contrast sensitivity		

Fundus Examination :

DIAGNOSIS:

Investigations

FBS, PPBS, HbA1c,

Diagnosis

Treatment: grid laser photocoagulation

- Power
- Spot size
- Duration
- Follow up and outcome of the treatment

PATIENT INFORMATION SHEET

TITLE: To study the efficacy of grid laser photocoagulation on contrast Sensitivity, visual fields and overall visual outcome in patients with diabetic macular edema-interventional study

I , Dr. ANU , post graduate at dept of Ophthalmology, Government Stanley Medical College is going to undertake the study on the above mentioned topic. If you are willing to participate in this study you will be asked some questions regarding duration of your illness, treatment history ,family history and regarding the illness. You may need to undergo grid laser photocoagulation as a part of treatment for diabetic macular edema The study may be of direct benefit for you in knowing your visual outcome and contrast sensitivity following grid laser photocoagulation. The study may be of great help in planning treatment strategies for you and other people in future.

I assure that all the information provided by you will be kept highly confidential and the privacy is assured. Your identity won't be revealed to anyone. The study may be published in scientific journal, but your identity will not be revealed. Your participation in this study is voluntary and you can withdraw from this at any point of time

Signature/left thumb impression of the participant

INFORMED CONSENT

TITLE: To study the efficacy of grid laser photocoagulation on contrast Sensitivity, visual fields and overall visual outcome in patients with diabetic macular edema-interventional study

The content of the information sheet dated _____ that was provided have been read carefully by me/explained in detail to me, in a language that I comprehend and fully understood the contents. I confirm that I have had the opportunity to ask questions. The nature and purpose of the study and its potential risks/benefits and expected duration of the study and other relevant details of the study have been explained to me in detail. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal right being affected.

I agree to take part in the above study

_____

(Signature/Left thumb impression)

Name of the Participant: _____

Son/Daughter/Spouse of _____

Complete postal address: _____

This is to certify that the above consent has been obtained in my presence.

_____ Date:

Signature of the principal investigator Place:

1) Witness – 1 2) Witness – 2

_____

_____

Signature: Signature:

Name: Name:

Address: Address:

INFORMATION SHEET

தகவல் நகல்

TITLE: To study the efficacy of grid laser photocoagulation on contrast Sensitivity, visual fields and overall visual outcome in patients with diabetic macular edema-interventional study

இந்த ஆய்வில் உங்களிடம் கேட்கும் கேள்விகளுக்கு முழு மனதுடன் பதில் அளிக்க வேண்டும். இந்த ஆய்வில் உங்கள் நாள்பட்ட நோய்களின் விபரம் , உடல்நலம் மற்றும் நடத்தை தொடர்பான விபரங்கள் கேட்கப்படும். இந்த ஆய்வினால் உங்களுக்கு எந்த பின்விளைவுகளும் ஏற்படாது என்று உறுதி அளிக்கிறேன். உங்களிடம் உங்களின் சுய விபரம் , குடும்ப விபரம், தொழில் விபரம் மற்றும் இதர விபரங்கள் பற்றி கேட்கப்படும் . உங்களுக்கு பணம் எதுவும் அளிக்க படாது என்பதை தெரிவித்துக் கொள்கிறேன்.

இந்த ஆய்வில் நீரழிவு நோயினால் விழித்திரையில் ஏற்படும் நோய்க்கு லேசர் சிகச்சை அளிக்கப்பட்டு , அதற்கு பின்பு பார்வையில் ஏற்படும் மாற்றங்கள் அனைத்தும் குறித்து பதிவு செய்யப்படும். இந்த ஆய்வில் மூலம் உங்களின் பார்வையில் ஏற்படும் மாற்றங்களை பற்றி நீங்கள் அறியலாம். மேலும் இதன் மூலம் வருங்காலத்தில் உங்களை போன்ற மக்களுக்கு சிகிச்சை அளிக்க தேவைப்படும் கோட்பாடுகளை வகுக்கலாம்.உங்களின் விபரங்கள் எதுவும் மற்றவர்களுக்கு தெரிவிக்க படாது என்று உறுதி அளிக்கிறேன்.இந்த ஆய்வின் முடிவுகள் பத்திரிக்கைகளில் பிரசுரிக்க படலாம். ஆனால் உங்களின் விபரங்கள் எதுவும் வெளியிட படாது. உங்களுக்கு விருப்பம் இல்லையெனில் எப்போது வேண்டுமானாலும் இந்த ஆய்வில் இருந்து விலகி கொள்ளலாம். இதனால் உங்களுக்கு எந்த பாதிப்பும் ஏற்படாது.

கையொப்பம்/ இடதுகை பெருவிரல் ரேகை

INFORMED CONSENT

ஒப்புதல் படிவம்

TITLE: To study the efficacy of grid laser photocoagulation on contrast Sensitivity, visual fields and overall visual outcome in patients with diabetic macular edema-interventional study

நான் தகவல் நகளில் கொடுக்கப்பட்டுள்ள முழு விபரங்களையும் கவனமாக படித்தேன். ஆய்வின் முழு விபங்களும் எனக்கு தமிழில் விளக்கமாக எடுத்துரைக்கப்பட்டது.நான் இந்த ஆய்வின் விபரங்களை முழுமையாக புரிந்துகொண்டேன். இந்த ஆய்வில் பங்கெடுக்கும் போது ஏற்படும் சாத்தியமான அபாயங்கள் மற்றும் பயன்களை அறிந்து கொண்டேன். மேலும் நான் எந்த ஒரு வேளையிலும் ஆய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அதன் பின்னர் நான் வழக்கம் போல் மருத்துவ சிகிச்சை பெற முடியும் என்றும் புரிந்து கொண்டேன். நான் ஆய்வில் பங்கு கொள்ள பணம் எதுவும் கொடுக்க படாது என்றும் அறிந்து கொண்டேன்.நான் இந்த ஆய்வின் போது முழு ஒத்துழைப்பு கொடுப்பேன் என்று உறுதி அளிக்கிறேன்.

ஆய்வில் பங்கேற்பவர் பெயர் : கையொப்பம்/ இடதுகை பெருவிரல் ரேகை

சாட்சி

பெயர் மற்றும் முகவரி

பெயர் மற்றும் முகவரி

ஆராய்ச்சியாளர் கையொப்பம் மற்றும் தேதி

KEY TO MASTER CHART

M	Male
F	Female
RE	Right eye
LE	Left eye
NPDR	Non proliferative diabetic retinopathy
CSME	Clinically significant macular edema
BCVA	Best corrected visual acuity
PRE GRID VA	Pre grid laser visual acuity
POST GRID VA 1 W	Post grid laser visual acuity at 1 week
POST GRID VA 1 M	Post grid laser visual acuity at 1 month
POST GRID VA 3 M	Post grid laser visual acuity at 3 months
PRE GRID CS	Pre grid laser contrast sensitivity
POST GRID CS 1 W	Post grid laser contrast sensitivity at 1 week
POST GRID CS 1 M	Post grid laser contrast sensitivity at 1 month
POST GRID CS 3 M	Post grid laser contrast sensitivity at 3 months
PRE GRID CV	Pre grid laser colour vision
POST GRID CV 1 W	Post grid laser colour vision at 1 week
POST GRID CV 1 M	Post grid laser colour vision at 1 month
POST GRID CV 3 M	Post grid laser colour vision at 3 months
PRE GRID VF	Pre grid laser visual field

POST GRID VF	Post grid laser visual field

S.NO	NAME	AGE	SEX	EYE	DIAGNOSIS	PRE GRID BCVA	POST GRID BCVA 1 W	POST GRID BCVA 1 M	POST GRID BCVA 3 M	PRE GRID CS	POST GRID CS 1 W	POST GRID CS 1 M	POST GRID CS 3 M	PRE GRID CV	POST GRID CV 1 W	POST GRID CV 1 M	POST GRID CV 3 M	PRE GRID VF	POST GRID VF
1	LAKSHMI	41	F	RE	MILD NPDR WITH CSME	0.48	0.48	0.60	0.48	1.35	1.20	1.20	1.20	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	5
2	SUGUMAR	45	M	RE	MILD NPDR WITH CSME	0.18	0.60	0.60	0.00	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2
3	SAKUNTHALA	45	F	RE	MILD NPDR WITH CSME	0.18	0.18	0.60	0.00	1.95	1.95	1.95	1.95	NORMAL	NORMAL	NORMAL	NORMAL	1	1
4	CHITHRA	45	F	RE	MODERATE NPDR WITH CSME	0.60	0.78	0.78	0.60	1.95	1.95	1.95	1.95	NORMAL	NORMAL	NORMAL	NORMAL	2	2
5	RAJASEKARAN	45	M	RE	SEVERE NPDR WITH CSME	0.48	0.48	0.48	0.30	1.95	1.95	1.95	1.65	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
6	REVATHI	45	F	RE	MILD NPDR WITH CSME	0.60	0.60	0.48	0.30	1.35	1.35	1.35	1.20	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
7	VIJAYALAKSHMI	45	F	LE	MODERATE NPDR WITH CSME	0.60	0.60	0.60	0.78	1.80	1.80	1.80	1.35	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
8	JAYANTHI	45	F	RE	MILD NPDR WITH CSME	0.18	0.48	0.48	0.00	1.80	1.80	1.80	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
9	RAVIKUMAR	45	M	LE	MODERATE NPDR WITH CSME	0.48	0.48	0.48	0.18	1.95	1.95	1.95	1.95	NORMAL	NORMAL	NORMAL	IMPAIRED	1	1
10	BHARATHI	46	F	LE	SEVERE NPDR WITH CSME	0.60	0.60	0.60	0.48	1.20	1.20	1.20	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2
11	KARUPAIAH	46	M	LE	SEVERE NPDR WITH CSME	0.00	0.00	0.00	0.30	1.65	1.65	1.65	1.20	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
12	RANJANI	47	F	RE	SEVERE NPDR WITH CSME	0.60	0.60	0.60	0.30	1.65	1.65	1.65	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	5
13	KAVITHA	47	F	RE	MODERATE NPDR WITH CSME	0.48	0.48	0.48	0.30	1.65	1.65	1.65	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	4	4
14	JAMUNA	47	F	LE	MILD NPDR WITH CSME	0.48	0.60	0.48	0.30	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2
15	JAYALAKSHMI	48	F	LE	SEVERE NPDR WITH CSME	0.60	0.48	0.48	0.30	1.20	1.20	1.20	1.35	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	5
16	SARATHI	48	M	RE	MODERATE NPDR WITH CSME	0.18	0.18	0.48	0.18	1.35	1.35	1.35	1.65	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2
17	SATHYAVATHI	48	F	RE	MODERATE NPDR WITH CSME	0.18	0.60	0.60	0.00	1.35	1.35	1.35	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2
18	BALAMURUGAN	48	M	LE	MILD NPDR WITH CSME	0.60	0.60	0.60	0.60	1.35	1.35	1.35	1.65	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	5
19	GOWRI	48	F	RE	MILD NPDR WITH CSME	0.48	0.48	0.48	0.18	1.80	1.80	1.80	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	5
20	KALAIMATHI	48	F	LE	MILD NPDR WITH CSME	0.18	0.60	0.60	0.00	1.95	1.95	1.95	1.95	NORMAL	NORMAL	NORMAL	NORMAL	1	1
21	DHANASEKAR	49	M	LE	SEVERE NPDR WITH CSME	0.18	0.18	0.18	0.18	1.95	1.95	1.95	2.10	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
22	RAJA	49	M	LE	MILD NPDR WITH CSME	0.60	0.60	0.48	0.30	1.20	1.35	1.35	1.50	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	4	4
23	ARUN	49	M	RE	MILD NPDR WITH CSME	0.18	0.18	0.60	0.00	1.95	1.95	1.95	1.80	NORMAL	NORMAL	NORMAL	NORMAL	2	2
24	MOHAMMED SHAH	49	M	RE	MODERATE NPDR WITH CSME	0.30	0.30	0.30	0.30	1.65	1.65	1.65	1.50	IMPAIRED	IMPAIRED	IMPAIRED	NORMAL	3	3
25	AMEENABEE	49	F	RE	SEVERE NPDR WITH CSME	0.48	0.48	0.48	0.18	1.35	1.35	1.35	1.65	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
26	SEKAR	49	M	RE	MILD NPDR WITH CSME	0.30	0.30	0.30	0.18	1.80	1.50	1.50	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	5
27	DEEPAK	50	M	RE	SEVERE NPDR WITH CSME	0.60	0.60	0.60	0.60	1.35	1.35	1.35	1.65	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
28	SAMUEL RAJA	50	M	LE	MILD NPDR WITH CSME	0.18	0.18	0.48	0.00	1.95	1.95	1.95	1.65	NORMAL	NORMAL	NORMAL	NORMAL	2	2
29	VASANTHA	50	F	LE	MILD NPDR WITH CSME	0.60	0.60	0.60	0.18	1.95	1.95	1.95	1.65	NORMAL	NORMAL	NORMAL	IMPAIRED	0	0
30	BALAJI	51	M	RE	SEVERE NPDR WITH CSME	0.48	0.48	0.00	0.48	1.50	1.50	1.50	1.65	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	5

31	MUMTAZ	52	F	RE	MILD NPDR WITH CSME	0.18	0.18	0.18	0.00	1.95	1.95	1.95	1.65	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
32	GOPALAN	52	M	RE	SEVERE NPDR WITH CSME	0.18	0.18	0.48	0.00	1.35	1.35	1.35	1.50	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
33	SHIVARAMAN	53	M	LE	SEVERE NPDR WITH CSME	0.00	0.00	0.00	0.00	1.95	1.95	1.95	2.10	IMPAIRED	IMPAIRED	SEVERELY IMPAIRED	IMPAIRED	0	0
34	LAKSHMANAN	53	M	LE	MODERATE NPDR WITH CSME	0.18	0.00	0.00	0.00	2.10	2.10	2.10	2.10	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
35	SARITHA	54	F	RE	SEVERE NPDR WITH CSME	0.48	0.48	0.60	0.60	1.20	1.20	1.50	1.65	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	5	5
36	VETRIMURUGAN	54	M	RE	MODERATE NPDR WITH CSME	0.60	0.60	0.30	0.30	1.35	1.35	1.35	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
37	DEVAKI	54	F	RE	SEVERE NPDR WITH CSME	0.00	0.00	0.18	0.18	1.35	1.35	1.35	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
38	MALIKHABEE	54	F	RE	MODERATE NPDR WITH CSME	0.60	0.60	0.60	0.18	1.80	1.80	1.80	1.50	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
39	BHAVANI	54	F	LE	SEVERE NPDR WITH CSME	0.48	0.48	0.48	0.18	1.95	1.95	1.95	1.95	NORMAL	NORMAL	NORMAL	NORMAL	0	0
40	ELAVAZHAGAN	54	M	RE	MILD NPDR WITH CSME	0.30	0.60	0.60	0.18	1.95	1.95	1.95	1.95	NORMAL	IMPAIRED	NORMAL	NORMAL	1	1
41	GODHANDARAMAN	55	M	LE	SEVERE NPDR WITH CSME	0.60	0.60	0.48	0.48	1.20	1.20	1.20	1.50	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	5
42	RAMANATHAN	55	M	LE	MILD NPDR WITH CSME	0.48	0.48	0.48	0.30	1.35	1.35	1.35	1.50	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
43	PERIAMMAL	55	F	RE	SEVERE NPDR WITH CSME	0.18	0.60	0.60	0.00	1.35	1.35	1.35	1.50	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	5
44	PADMAVATHI	55	F	RE	MILD NPDR WITH CSME	0.18	0.48	0.48	0.00	1.80	1.80	1.80	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
45	AMBIKA	55	F	RE	SEVERE NPDR WITH CSME	0.60	0.60	0.60	0.78	1.35	1.35	1.35	1.50	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2
46	ARULDAS	56	M	LE	MODERATE NPDR WITH CSME	0.18	0.18	0.18	0.18	2.10	2.10	2.10	2.10	IMPAIRED	IMPAIRED	SEVERELY IMPAIRED	IMPAIRED	3	3
47	MEENA	56	F	RE	MODERATE NPDR WITH CSME	0.60	0.60	0.60	0.60	1.65	1.65	1.65	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	4	4
48	VETTIYAN	57	M	RE	MODERATE NPDR WITH CSME	0.60	0.18	0.18	0.30	1.05	1.05	1.05	1.20	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	6	6
49	SAMUEL	57	M	LE	SEVERE NPDR WITH CSME	0.48	0.48	0.48	0.48	1.50	1.50	1.65	1.65	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	4
50	NOORISHAM	57	F	LE	MILD NPDR WITH CSME	0.48	0.48	0.30	0.18	1.50	1.50	1.50	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2
51	MUNUSAMY	58	M	LE	MILD NPDR WITH CSME	0.60	0.60	0.60	0.60	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2
52	VEERAMMAL	58	F	RE	MODERATE NPDR WITH CSME	0.30	0.30	0.30	0.18	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	5
53	SHANMUGAM	58	M	LE	MODERATE NPDR WITH CSME	0.60	0.60	0.78	0.78	1.95	1.95	1.95	1.95	NORMAL	NORMAL	NORMAL	NORMAL	1	1
54	VARADHARAJAN	59	M	RE	SEVERE NPDR WITH CSME	0.18	0.18	0.18	0.18	1.95	1.95	1.95	2.10	IMPAIRED	IMPAIRED	IMPAIRED	SEVERELY IMPAIRED	3	4
55	MANIVANNAN	59	M	RE	SEVERE NPDR WITH CSME	0.48	0.48	0.00	0.60	1.35	1.35	1.50	1.80	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	5	4
56	GAJENDRAN	59	M	LE	MILD NPDR WITH CSME	0.48	0.48	0.00	0.48	1.35	1.35	1.35	1.65	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	4	4
57	MUNUSAMY	59	M	RE	SEVERE NPDR WITH CSME	0.48	0.48	0.48	0.18	1.80	1.80	1.80	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
58	GANESAN	59	M	RE	MODERATE NPDR WITH CSME	0.48	0.48	0.60	0.18	1.80	1.80	1.95	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
59	IBRAHIMA	60	M	LE	SEVERE NPDR WITH CSME	0.48	0.48	0.48	0.60	1.20	1.20	1.05	1.65	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	5	5
60	RAJAN	60	M	LE	MODERATE NPDR WITH CSME	0.30	0.30	0.48	0.30	1.95	1.95	1.95	2.10	IMPAIRED	SEVERELY IMPAIRED	IMPAIRED	IMPAIRED	2	3
61	MUHAMMAD HUSSAIN	60	M	RE	SEVERE NPDR WITH CSME	0.60	0.60	0.30	0.30	1.05	1.05	1.05	1.35	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	6	6
62	SELVI	60	F	RE	MODERATE NPDR WITH CSME	0.60	0.60	0.18	0.48	1.05	1.20	1.20	1.35	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	5	5
63	MUHAMMAD HUSSAIN	60	M	LE	MODERATE NPDR WITH CSME	0.00	0.00	0.00	0.00	2.10	2.10	2.10	2.10	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
64	PICHAIAMMAL	60	M	LE	SEVERE NPDR WITH CSME	0.60	0.00	0.30	0.30	1.20	1.20	1.20	1.50	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	4	4
65	GUNASEKAR	60	M	LE	MILD NPDR WITH CSME	0.48	0.48	0.48	0.48	1.50	1.50	1.50	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	2
66	KAMALAKANNAN	60	M	LE	MODERATE NPDR WITH CSME	0.18	0.00	0.48	0.18	1.50	1.50	1.50	1.65	IMPAIRED	IMPAIRED	IMPAIRED	SEVERELY IMPAIRED	5	5
67	SRIDHAR	60	M	LE	MILD NPDR WITH CSME	0.00	0.18	0.00	0.18	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2

68	CHINNASAMY	60	M	RE	SEVERE NPDR WITH CSME	0.48	0.48	0.30	0.30	1.65	1.65	1.65	1.50	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
69	LAKSHMIAMMAL	60	F	LE	SEVERE NPDR WITH CSME	0.18	0.18	0.60	0.30	1.80	1.50	1.50	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	4	4
70	PARVATHY	62	F	LE	MODERATE NPDR WITH CSME	0.18	0.48	0.48	0.18	2.10	2.10	2.10	2.10	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
71	MARIAPPAN	62	M	RE	MILD NPDR WITH CSME	0.60	0.18	0.18	0.18	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2
72	TAMILARASAN	63	M	RE	SEVERE NPDR WITH CSME	0.60	0.60	0.60	0.48	1.05	1.05	1.05	1.50	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	5	4
73	CHINNAIAYA	63	M	LE	MODERATE NPDR WITH CSME	0.48	0.48	0.48	0.48	1.35	1.35	1.35	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	5	4
74	MARIMUTHU	63	M	RE	SEVERE NPDR WITH CSME	0.30	0.30	0.18	0.18	1.80	1.80	1.80	1.80	IMPAIRED	SEVERELY IMPAIRED	IMPAIRED	IMPAIRED	4	4
75	RAMABHAI	64	F	LE	MODERATE NPDR WITH CSME	0.00	0.00	0.00	0.00	1.95	1.95	1.95	2.10	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
76	JAYAPAL	64	M	LE	MODERATE NPDR WITH CSME	0.18	0.18	0.00	0.18	1.95	1.95	1.95	2.10	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
77	SELVARAJ	64	M	RE	MODERATE NPDR WITH CSME	0.60	0.60	0.60	0.00	1.95	1.95	1.95	1.95	NORMAL	NORMAL	NORMAL	NORMAL	1	1
78	RAGAVAN	65	M	LE	SEVERE NPDR WITH CSME	0.48	0.48	0.60	0.60	1.20	1.20	1.20	1.50	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	5	5
79	JAGANATHAN	65	M	RE	SEVERE NPDR WITH CSME	0.18	0.18	0.00	0.00	1.35	1.35	1.35	1.50	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
80	SIVAGAMI	65	F	RE	MILD NPDR WITH CSME	0.18	0.18	0.18	0.00	1.35	1.35	1.35	1.35	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	2
81	LALITHA	66	F	RE	SEVERE NPDR WITH CSME	0.18	0.18	0.18	0.18	1.95	1.95	1.95	2.10	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	0	1
82	TAMILARASI	66	F	LE	SEVERE NPDR WITH CSME	0.48	0.48	0.48	0.48	1.35	1.35	1.35	1.65	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	3	3
83	DIWAKARAN	66	M	LE	MODERATE NPDR WITH CSME	0.18	0.48	0.48	0.00	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	NORMAL	2	2
84	BALAMBIGAI	66	F	RE	SEVERE NPDR WITH CSME	0.60	0.60	0.60	0.60	1.80	1.80	1.80	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
85	KADHIR BASHA	67	M	LE	MODERATE NPDR WITH CSME	0.48	0.48	0.48	0.48	1.95	1.95	1.95	1.95	NORMAL	NORMAL	NORMAL	NORMAL	1	1
86	MARIYAMMAL	67	F	LE	MODERATE NPDR WITH CSME	0.48	0.60	0.60	0.18	1.80	1.80	1.80	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
87	SARASWATHI	68	F	RE	MILD NPDR WITH CSME	0.60	0.60	0.60	0.48	1.50	1.50	1.50	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
88	MURUGESAN	69	M	RE	MILD NPDR WITH CSME	0.48	0.48	0.48	0.30	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	2	3
89	RAJAGOPAL	69	M	RE	SEVERE NPDR WITH CSME	0.60	0.60	0.60	0.30	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
90	ANTHONY	70	M	LE	SEVERE NPDR WITH CSME	0.60	0.60	0.60	0.18	1.20	1.20	1.20	1.65	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	SEVERELY IMPAIRED	5	5
91	VARATHAMBAL	70	F	LE	MILD NPDR WITH CSME	0.48	0.48	0.48	0.30	1.95	1.95	1.95	1.65	NORMAL	NORMAL	NORMAL	IMPAIRED	2	2
92	KALA	70	F	LE	MILD NPDR WITH CSME	0.30	0.60	0.60	0.48	1.80	1.80	1.80	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
93	RANIYAMMAL	71	F	RE	SEVERE NPDR WITH CSME	0.48	0.48	0.48	0.30	1.80	1.80	1.80	1.50	IMPAIRED	IMPAIRED	IMPAIRED	SEVERELY IMPAIRED	3	3
94	PALANISWAMY	73	M	RE	MODERATE NPDR WITH CSME	0.48	0.00	0.48	0.48	1.50	1.50	1.50	1.65	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	5
95	MEENAKSHI	73	F	RE	SEVERE NPDR WITH CSME	0.48	0.30	0.30	0.48	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	2
96	RANI	73	F	RE	MODERATE NPDR WITH CSME	0.18	0.18	0.48	0.00	1.80	1.80	1.80	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
97	NAGAMMA	73	F	LE	SEVERE NPDR WITH CSME	0.18	0.60	0.60	0.18	1.80	1.80	1.80	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
98	SUBBULAKSHMI	71	F	LE	MODERATE NPDR WITH CSME	0.18	0.18	0.48	0.00	1.35	1.35	1.35	1.80	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	4	4
99	KOSALAI	73	F	LE	MODERATE NPDR WITH CSME	0.00	0.48	0.30	0.30	2.10	2.10	2.10	2.10	NORMAL	NORMAL	IMPAIRED	IMPAIRED	3	3
100	KAMALA	72	F	LE	SEVERE NPDR WITH CSME	0.00	0.00	0.48	0.00	1.95	1.95	1.95	1.95	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	1	1
101	KANNIYAMMAL	72	F	RE	SEVERE NPDR WITH CSME	0.48	0.60	0.48	0.30	1.35	1.35	1.35	1.20	IMPAIRED	IMPAIRED	IMPAIRED	IMPAIRED	3	3
102	DURAIAMMAL	71	F	RE	MILD NPDR WITH CSME	0.18	0.18	0.60	0.18	1.95	1.95	1.95	1.80	NORMAL	IMPAIRED	NORMAL	NORMAL	2	2