OPTICAL COHERENCE TOMOGRAPHIC FEATURES OF MACULAR PATHOLOGIES IN DIABETIC RETINOPATHY

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This is to certify that the dissertation entitled **OPTICAL COHERENCE TOMOGRAPHIC FEATURES OF MACULAR PATHOLOGIES IN DIABETIC RETINOPATHY** is a Bonafide work done by **Dr.M.MURALI , MS**, student during May 2005 to March 2008 under our direct supervision and guidance at our institute in partial fulfillment of regulations governing the award of Master of Surgery Branch III ophthalmology Tamilnadu **Dr.MGR MEDICAL UNIVERSITY CHENNAI MARCH 2008.**

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CONTENTS

INTRODUCTION

INTRODUCTION

High resolution cross sectional imaging of retina is useful for identifying ,monitoring and quantitatively assessing macular disease . Optical Coherence Tomography (OCT) Is a new medical diagnostic imaging technology which can perform high resolutional cross sectional tomographic imaging in biological tissues. Cross sectional images of retina are obtained at the resolution 10 microns. OCT uses low coherence or white light interferometry to perform high resolutional measurements and imaging. The infra red light beam has a wave length of 820nm

Macular pathologies in diabetic retinopathy consists of 1)Macular edema which may be localized , diffuse , CSME and ischemic which may show on OCT as (a) Sponge like thickening (b) Cystoid macular edema (c) Subfoveal serous detachment (d) Taut posterior hyaloid membrane (e) Foveal tractional retinal detachment

OCT offers an objective method for qualitative identification and quantitatively monitoring and evaluation of patients with diabetic maculopathy which are not seen clinically and by FFA

AIMS AND OBJECTIVES

AIMS OF THE STUDY

- To study the OCT features of various macular pathologies in diabetic retinopathy.
- To compare these features with clinical features and fundus fluorescein angiographic features.
- To compare foveal thickness by OCT with visual acuity.
- To quantitatively monitoring the response by measuring the foveal thickness by OCT in various types of diabetic macular pathologies after Intra vitreal triamcinolone acetonide [IVTA].

REVIEW OF LITERATURE

REVIEW OF LITERATURE

ANATOMY OF MACULA [1]

Macula refers to that part of central retina having a diameter of 5.5mm and limited by superior and inferior temporal vascular arcades, including optic disk on nasal side and histologically by outer boundary of perifovea where the ganglion cells are reduced to a single layer

PARTS OF THE MACULA

- UMBO it is a tiny depression in the very center of the foveola which corresponds to the ophthalmoscopically visible foveal reflux

- FOVEOLA (350microns-0.35mm) it is a small central region in which the thickness of retina is reduced so as to contain only photoreceptors glialcells and mullercells

- FOVEAL AVASCULAR ZONE(FAZ-800microns-0.8mm)it is located inside fovea but outside foveola

- FOVEA (1500microns-1.5mm)it is a small depression where the retina is reduced to half its normal thickness. Moving towards the centre of retina the inner nuclear layer is reduced to a double row of cells at edge of fovea

- PARA FOVEAL AREA (2500microns-2.5mm) it is characterized by densest accumulation of nerve cells in the entire retina especially ganglion cells and inner nuclear layer. The outer boundary is the point where the ganglion cell layer has four rows of nuclei

- PERI FOVEAL AREA(5500 microns-5.5mm) it ends where ganglion cells are reduced to a single layer

- HISTOLOGY OF MACULA: The center of macula is the fovea containing the following layers; internal limiting membrane, outer plexiform layer, outer nuclear layer, layer of cones, and retinal pigment epithelium.

Fig. 1. Topography of Macula

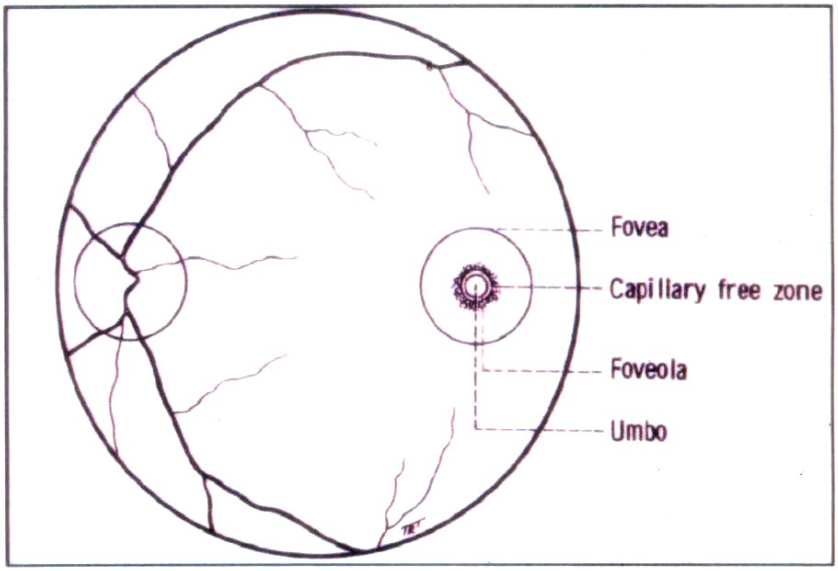
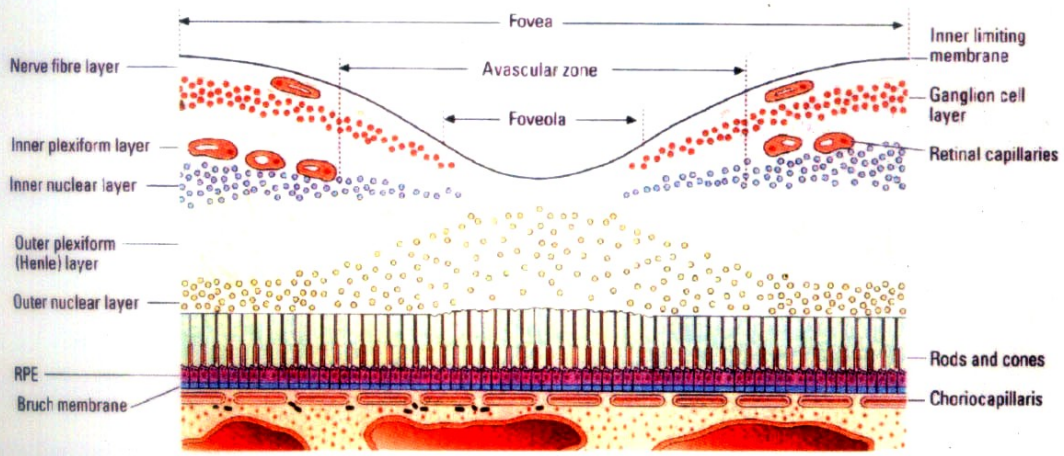


Fig. 2. Histology of Macula



DIABETIC RETINOPATHY

Diabetic retinopathy is a leading cause of severe loss of visual acuity (3rd common cause) in the world. About 25% of diabetic patients have sight threatening levels of retinopathy with legal blindness. DR being 25 times more common in diabetics when compared to the non diabetic population (2)

ETIOPATHOGENESIS

The exact cause of diabetic microvascular disease is unknown. It is believed that exposure to chronic hyperglycemia results in a number of biochemical and physiological changes that ultimately causes vascular endothelial damage.

There is an association between higher levels of glycosylated hemoglobin (Hb A1c) and diabetic retinopathy and CSME HLA B 8, B15, DR3, DR4, is more commonly found more in patients with IDDM than NIDDM.

PATHOGENESIS OF MICROVASCULAR OCCLUSION (AAO-2000-2001)

1) Basement membrane thickening,2) Damage and proliferation of endothelial cells. 3) Erythrocyte aggregation leading to defective O2 transport. 4) Increased platelet adhesiveness 5] Abnormal levels of growth hormones like VEGF. 6) Defective fibrinolysis. 7) Abnormal serum lipids. 8) Increased blood viscosity .All these leading to

A) Breakdown of inner blood retinal barrier (tight junctions of capillary endothelium)

B) Loss of pericytes leading to physical weakening of capillary wall resulting in localized vascular out pouching termed micro aneurysms which may leak or become thrombosed

FEATURES OF DIABETIC RETINOPATHY

1) **VENOUS CHANGES:** Earliest, consists of dilatation of veins and later looping, beading and sausage like segmentation, FFA shows these findings

2)**MICROANEURYSMS:** Earliest due to weakening of capillary walls, results in localized secular out pouching of vessel wall and appears as tiny round red dots .FFA shows hyper fluorescent dots

3) **HARD EXUDATES:** Due to capillary leakage and appears as waxy yellow lesions with relatively distinct margins often arranged in clumps or rings around blood vessels at posterior pole. FFA shows hypo fluorescence due to blockade of background choroidal fluorescence. OCT shows hyper reflective lesions

4**) HEMORRHAGE:** Intra retinal dot and blot; Arises from venous end of capillaries located in compact middle layers of retina .Blot hemorrhages due to hemorrhagic infarcts ,Nerve fiber layer flame shaped Hemorrhage arises from superficial pre capillary arterioles FFA shows blocked fluorescence (18,19).OCT shows in thin hemorrhages increased scattering and hyper reflective lesions and thick hemorrhages blocks the reflections from under lying structures and appears hypo reflective

5) **COTTON WOOL SPOTS:** They represent focal infarcts of retinal nerve fiber layer due to occlusion of pre capillary arterioles which in turn leads to interruption of

axoplasmic transport with subsequent build up of transported materials within axons (axoplasmic stasis) and appears as white fluffy superficial lesions with ill defined borders and obscures underlying blood vessels and seen mostly in post equatorial retina. FFA shows hypo florescence due to capillary non perfusion. OCT shows hyper reflective lesions

6)**INTRA RETINAL MICROVASCULAR ABNORMALATIES(IRMA**): They represent shunts that run from retinal arterioles to venules bypassing capillary bed and are therefore often seen adjacent to areas of capillary closure. They are seen as fine red lines running from arterioles to venules thus resembling focal areas of flat retinal new vessels but they are intra retinal location and fail to cross major blood vessels. FFA shows hyper fluorescence with absence of leakage adjacent to areas of capillary closure

7**) ARTERIAL CHANGES:** Narrowing, obliteration and silver wiring.

8) NEOVASCULARISATION: They are fresh endothelial channels arising due to severe ischemia and appear frond like rising above retina may be associated with fibrous tissue which may appear over the disc or within 1disk diameter around the disk(NVD) or they may appear in other areas elsewhere(NVE) which may be high risk or low risk. FFA shows hyper fluorescence with leakage

NVD Low risk < 1/3DD, NVD High risk >1/3DD, associated with hemorrhage

NVE Low risk <1/2 DD, NVE High risk >1/2DD, associated with hemorrhage

9**) INVOLUTIONAL, END STAGE, BURNT OUT RETINOPATHY**: Attenuated vessels pale disk, neovascularisation replaced by glial scar tissue, opaque membranes and tractional retinal retinal detachment

ETDRS CLASSIFICATION OF DIABETIC RETINOPATHY (4,5)

1) NON PROLIFERATIVE DIABETIC RETINOPATHY.

A) MILD: Few micro aneurysms in less than 4 quadrants
Few dot hemorrhages in less than 4 quadrants
Hard exudates and macular edema may or may not be present

B**) MODERATE:** Micro aneurysms or dot haemmorages present in 4 Quadrants
Hard exudates, Macular edema, cotton wool spots
Venous beading and looping in 1 quadrant

C**) SEVERE (4:2:1rule**): Severe intra retinal hemorrhages (dot and blot) in 4 Quadrant
Venous beading in 2 quadrants
IRMA in 1 quadrant
Along with microaneurysms ,hardexudates, macular edema, cws

D) VERY SEVERE: Any 2 of the features of NPDR

2) **PROLIFERATIVE DIABETIC RETINOPATHY:** All of the above features with neovascularisation

DIABETIC MACULOPATHY(6,7,8,9)

Due to involvement of macula by edema and hard exudates due to capillary leakage or ischemia due to capillary closure and it is the most common cause for visual impairment in diabetic patients mostly type 2 diabetics which may be

- Focal exudative
- Diffuse exudative
- CSME
- Ischemic
- Mixed

FOCAL EXUDATIVE:

Well circumscribed retinal thickening due to edema associated with complete or incomplete rings of perifoveal hard exudates. FFA shows late focal hyper fluorescence due to focal leakage with good macular perfusion .OCT shows focal sponge like thickening

DIFFUSE EXUDATIVE:

Diffuse retinal edema and thickening which may be associated with cystoid changes with obliteration of land marks which may render localization of the fovea impossible. FFA shows widespread late diffuse hyperfluorescence with flower petal pattern if cystoid macular edema is present, OCT shows diffuse sponge like thickening and fluid filled cyst like spaces if cystoid macular edema is present

CLINICALLY SIGNIFICANT MACULAR EDEMA; (CSME);

1) Retinal edema within 500 microns of centre of fovea

2) Retinal edema of one disk diameter(1500 microns) or larger any part of which is within one disk diameter of centre of fovea

3) Hard exudates within 500 microns of centre of fovea with adjacent retinal edema which may be outside 500 microns

FFA shows focal hyper fluorescence or blocked fluorescence within 500 microns of centre of fovea .OCT shows sponge like or cyst like spaces within 500 microns

ISCHEMIC MACULOPATHY: Due to macular non perfusion with decreased visual acuity with relatively normal appearance of fovea . Dark blot haemmorages may be seen .FFA shows capillary non perfusion areas at the fovea and enlargement of foveal avascular zone (FAZ)

MIXED EXUDATIVE AND ISCHEMIC: Signs characterized by features of both ischemic and exudative maculopathy

Optical Coherence Tomography

Optical Coherence Tomography is a new diagnostic tool that can perform tomography / cross – sectional imaging of biological tissues with ≤10 microns axial resolution using light waves.

Principle:

It uses infrared light. The speed of light is almost a million times faster than sound and this difference allows the measurement of structures with resolution of ≤ 10 microns compared to 100 micron scale of ultrasound. Ultrasound needs contact with the tissue under study, whereas OCT does not, require any contact.

It is a non contact, non – invasive device where a broad band width of near infra – red light beam (820nm) is projected on to the retina. The light gets reflected from the boundaries between the microstructure and also gets scattered differently from tissues with different optical properties. It then compares the echo time delay of the same wavelength that is reflected from a reference mirror at a known distance.

Optical coherence tomography uses, low coherence or white light interferometry to perform high resolution measurements and imaging.

An optical beam from a laser or light source which emits short optical pulses or short coherence length light, is directed onto a partially reflecting mirror (optical beam splitter). The partially reflecting mirror splits the light into two beams, one beam is reflected and the other is transmitted. One light beam is directed on to the patients eye and is reflected from intraocular structures at different distances.

The reflected light beam from the patient's eye consists of multiple echoes which give information about the range or distance and thickness of different intra-ocular structures. The second beam is reflected from a reference mirror at a known spatial position. This retro-reflected reference optical beam travels, back to the partial mirror (beam splitter) where it combines into the optical beam reflected from the patient's eye.

When the two light pulses co inside they produce a phenomenon known as interference which is measured by a light sensitive detector (photodetector). Thus the interferometer can precisely measure the echo structure of reflected light and perform high resolution measurements of the distance and thickness of different tissue structures.

The key feature of interferometer is that it can measure the time delay of optical echoes by comparing the reflected light beam with a reference beam. While the explanation presented here assumes that the light is composed of short optical pulses, the measurement may also be performed using non-pulsed or continuous light with a short coherence length. For this reason, the measurement techniques has been termed 'low coherence interferometry'.

The light source for the interferometer is a compact super luminescent diode, which is coupled directly into an optical fiber. This light source is similar to laser diode used in optical compact disc players, except in OCT, the diode source is designed to emit short coherence length light. The interferometer is constructed using a fiber optic coupler which functions, analogous to a beam splitter. The arm of the interferometer which consists of reference mirror is located within the instrument, while the optical fiber in the second arm of the interferometer is connected to the OCT ophthalmic instrument resembling a slit lamp biomicroscope or fundus camera.

Image resolution

The image resolution of OCT in the axial (or longitudinal) verses transverse directions is determined by different mechanisms. The resolution of the image in the axial (longitudinal) direction is determined by the resolution of the optical ranging measurement. This is determined by the physical properties of light source which is used for the measurement. If a short pulse laser source is used, the axial resolution is determined by the pulse duration. Conversely, if a continuous, low-coherence light source is used, the axial resolution is determined by the 'coherence length' of the light source. It is important to note that the measurement of distance or tissue thickness can, in practice, be performed with significantly higher resolution than this limit.

The transverse resolution of the image is determined by the size of the focused optical beam. This is a function of the optics used to project the beam onto the eye and this is determined by factors such as whether imaging is performed over a large

depth, such as in the anterior eye, or whether the focusing angle is restricted, as in imaging the retina. The image resolution is also a function of the size of the tomogram that is desired.

OCT Scan Protocols in Macula

The protocols that are helpful in macular diseases are the following.;

(i) Line Scan

The line scan gives an option of acquiring multiple line scan without returning to main window. The length of the line scan and the angle can be altered, though one has to keep in mind that as the scan length increases the resolution decreases.

(ii) Radial Lines

The scan protocol consists of 6 -24 equally spaced line scans that can be varied in size and parameters. All the lines pass through a central common axis. The radial lines are useful for acquiring macular scan and retinal thickness / volume analysis.

(iii) Macular thickness map

This is the same as radial lines except that the aiming circle has a fixed, diameter of 6mm. This helps in measuring the retinal thickness.

(iv) Fast macular thickness map

It is designed for use with retinal thickness analysis. When done in both the eyes, it can be used for comparative retinal thickness / volume analysis. It is a quick protocol that takes only 1.95 sec to acquire six scans of 6mm length each

(v) Raster Line

This provides an option of acquiring series of line scans that are parallel, equally spaced and are 6 – 24 in number. These multiple lines scans are placed over rectangular regions, the area of which can be adjusted so as to cover the entire area of pathology. This is especially useful in conditions like choroidal neovascular membrane one wishes to obtain scans at multiple levels.

(vi) Repeat:

Repeat protocol enables one to repeat any of the previously saved protocols using same set of parameters, that includes scan size, angle, placement of fixation, light emitting diode (LED) and landmark.

Normal macular scan

On a 10mm horizontal line scan passing through the foveal centre, one can clearly demarcate two major landmarks namely optic disc and fovea.

The optic disc is seen towards the right of the tomogram and is easily identifiable by its contour. The central depression represents the optic head cup and the stalk

continuing behind is the anterior part of the optic nerve. The fovea is seen to the left and is easily identifiable by the characteristic thinning of retinal layers. The vitreous anterior to the retina is non-reflective and is seen as a dark space. The interface between the non – reflective vitreous and back scattering retinal layers is the vitreoretinal interface. The retinal nerve fiber layer (NFL) is highly reflective and increases in thickness towards the optic nerve. The posterior boundary of the retina is marked by a hyper – reflective layer that represents retinal pigment epithelium (RPF) and chorio capillaries.

Just anterior to RPE – choriocapillaries complex, is a minimally reflective layer that represent photoreceptors. Above this layer of photoreceptors are alternating layers of moderate and low reflectivity that represents different layers of neurosensory retina. The retinal blood vessels within the neurosensory retina shows back scatter and also cast a shadow behind.

Image Interpretation:

a) Objective
b) Subjective

1) Objective

Hyper – reflective lesions are:

Hard exudates: are seen as hyper – reflective shadows in the neurosensory retina that completely blocks the reflections from the underlying retina.

Blood: Blood causes increasing scattering. Small to thin hemorrhages are seen as hyper reflective lesions .Thick hemorrhages blocks the reflections from the underlying structure.

Scars : All fibrotic lesions including disciform scars, choroidal rupture scars, healed choroiditis etc are hyper – reflective.

Hypo – reflective lesions are:

Serous fluid: Retinal edema is the commonest cause of reduced back scattering and one can actually point out the site of fluid accumulation. The serous fluid that is devoid of any particular matter, produces an optically empty space with no back scattering.

Hypo – pigmented lesions of RPE.

Subjective analysis.

Qualitative,

Protocols are
a) Normalize
b) Align
c) Median Smoothing
d) Gaussian Smoothing

Quantitative:

a) Retinal thickness / volume
b) Retinal thickness or volume tabular
c) Retinal thickness / volume change

Advantages of OCT over FFA

- Non contract, non invasive
- Time saving technique
- Totally avoids mild complications like nausea to life threatening hypersensitivity reactions seen in FFA.
- Measurement of retinal thickness by OCT correlate more strongly with visual acuity than the presence of leakage on angiography.
- OCT is effective and superior to FFA in demonstrating axial distribution of fluid.
- Can be repeated as many times needed.
- Can quantitatively assess retinal thickness and demonstrate any associated RPE structural anomalies.

OCT : Features in Diabetic maculopathy (10)

OCT almost gives the in vivo histopathology of retinal layers that help in the better disease understanding and pathogenesis. Oct is a useful tool in monitoring response to an intervention (IVTA) in maculopathy. There are five patterns of diabetic maculopathy. They are.

1. Sponge like thickening of retinal layers : This is mostly confined to outer retinal layers and reduced back scattering from intra retinal fluid accumulation.
2. Cystoid macular edema : Large cystoid spaces involving variable depth of retina with intervening septae. The cystoid spaces are initially confined to outer retina mostly but in long standing cases these cysts fuse to involve almost entire thickness of retina.
3. Subfoveal serous detachment; This is shown as hypo reflective area in subfoveal region
4. Taut posterior hyaloid membrane; This may result in recalcitrant macular edema with foveal detachment that can be diagnosed easily on OCT even when sub clinical. In advanced cases it can be diagnosed as a taut shiny glistening membrane with retinal striae on bio microscopic examination. CSME with TPHM is generally non responsive to laser and is an indication for Pars plana vitrectomy
5. Tractional detachment of fovea; Foveovitreal traction may result in detachment of fovea, This can be diagnosed easily in OCT. This is an indication for pars plana vitrectomy to release the traction. Laser photo coagulation may only worsen macular edema.

MATERIALS & METHODS

MATERIALS AND METHODS

Patients with diabetic maculopathy who presented to the Retina Clinic of Institute of Ophthalmology Joseph Eye Hospital Trichy between July 1st 2006 to June 30th 2007 are included in this study

PATIENTS: Sixty three eyes of forty patients with diabetic retinopathy

INCLUSION CRITERIA:

All patients with clinical diagnosis of diabetic maculopathy whether NPDR or PDR

EXCLUSIONCRITERIA:

Patients with diabetic retinopathy NPDR or PDR with out maculopathy

Patients with other causes of maculopathy

METHODS:

A standard protocol was used to collect and document all details regarding the cases included in this study

A detailed information about the **history of diabetes,** with duration and associated risk factors like Hypertension, Nephropathy, and Cardiovascular disease was taken

The **history of treatment of diabetes,** whether oral hypoglycemic drugs or insulin was taken

Visual acuity, Distance vision, Near vision, Best corrected visual acuity was taken initially and after intra vitreal triamcinolone acetonide at 1, 2, 3, 6 months follow up

A complete **ocular examination** was done for each patient which included slit lamp examination of anterior segment, 90D slit lamp indirect ophthalmosocpic examination, indirect ophthalmoscopic examination, and following were noted **clinically**

1) Type of diabetic retinopathy; NPDR or PDR

2) Type of maculopathy; Focal, Diffuse, CSME, CME , foveal tract ional RD were noted

A **fundus photograph** was taken to document clinical findings

A digital **fundus fluorescein angiography** was done using Carl Zeiss ff450 plus IR digital camera. 3ml of 20% sodium fluorescein is injected into anterior cubital vein. A series of photographs are taken. Normally fluorescein begins to show in the choroid in10 to12 seconds after injection. The type of leaks and area of leak (hyper fluorescence) whether focal, or diffuse or specific pettaloid pattern is taken. A note was made on macular perfusion , late staining and blocked fluorescence due to hard exudates

Optical coherence tomography was done using Carl Zeiss stratus OCT 3. Fast macular scan was done at the time of presentation .This protocol is designed for use with retinal thickness analysis in all cases. It is a quick protocol that takes only 1.92 seconds to acquire 6 scans of 6mm length each and various parameters noted for presence of maculopathy, The type of maculopathy, CME, Sponge like thickening, Subfoveal serous detachment, Taut posterior hyaloid membrane and Vitreo macular tract ional RD was noted

TREATMENT

Patients who had CME, Sponge like thickening and subfoveal serous detachment was given intra vitreal triamcinolone acetonide injection of 0.1ml (4mg) and immediate post intervention the patients was given tab Diamox 250mg tds for 2 days, Timolol eye drops 0.5% bd for 2 weeks, ciprofloxacin eye drops 0.03% for 2 days

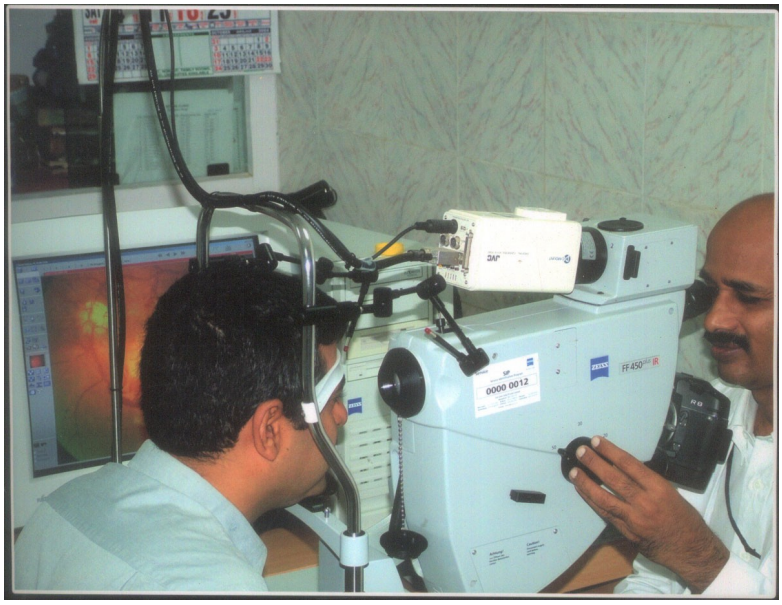
FOLLOW UP

The patients were followed up at 1 week, 1 month, 3months,and 6 months and all patients who under went IVTA underwent OCT examination and foveal thickness measurement at 6 months follow up and IOP recorded at each visit.

STATISTICAL ANALYSIS

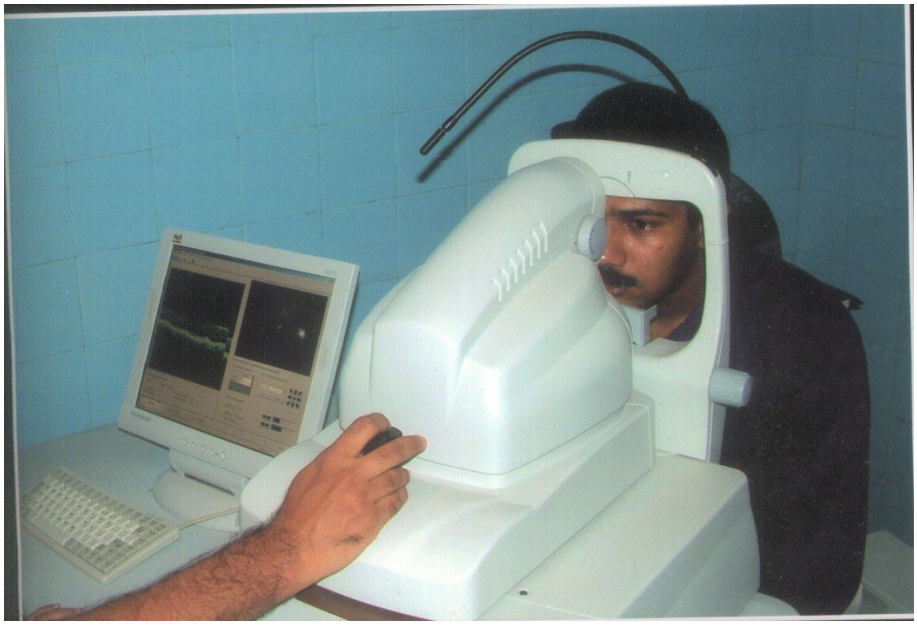
Statistical analysis was done by student t test, regression analysis, chi square test and p value < 0.05 was considered significant

Fig 2



Carl Zeiss Digital Digital Camera with Fundus Fluorescein Angiogram

Fig 3



Carl Zeiss Stratus Optical Coherence Tomogram Model 3000

RESULTS

RESULTS

In this prospective comparative case series study 63 eyes of 40 patients with diabetic maculopathy who presented at retina clinic of Institute of Ophthalmology Joseph Eye Hospital Trichy between July 1st 2006 to June 30th 2007 was included in this study

GENDER : In this study there were 28 male (70%) and 12 female (30%)

AGE : In this study the mean age was 57.75 years(range 35 to 70 years) and Maximum number of patients were in 51 to 60 yrs range 21 patients (52.7%), and 61 to 70 yrs 14 patients (35%) , and 41 to 50 yrs 2 patients (5%) ,and 31 to 40 yrs 3 patients (7.5%)

TYPE OF DIABETES MELLITUS; In this study there were 37 patients with NIDDM (92.5%),and 3 patients with IDDM (7.5%).

DURATION OF DIABETES MELLITUS: In this study duration of diabetes was 10 to 15 years in 28 patients (70%), and 5 to 10 yrs in 6 patients (15%), and less than 5 years in 6 patients (15%).

ASSOCIATION WITH SYSTEMIC DISEASES: In this study Hypertension was associated with diabetes in 5 patients, and Nephropathy in 1 patient

TYPE OF DIABETIC RETINOPATHY; In this study NPDR was present in 26 patients (65%) ,PDR in 14 patients (35%).

TYPE OF DIABETIC MACULOPATHY CLINICALLY; In this study Clinically Significant Macular Edema was seen In 58 eyes (92%) , Cystoid Macular edema in 2 eyes3.(2%),Diffuse Edema in 2 eyes(3.2%), Fibrous traction in1eye(1.6%)

TYPE OF DIABETIC MACULOPATHY WITH FFA: In this study With FFA, Diffuse leak was seen in 49 eyes (78%), Focal leaks in 7 eyes (11%), Pettaloid pattern in 3 eyes (5%), Ischemic pattern in 3 eyes (5%), Blocked fluorescence in 1eye (2%).

TYPE OF DIABETIC MACULOPATHY WITH OCT: In this study with OCT, Cystoid macular edema was seen in 32eyes (51%), Sponge like thickening in21eyes (33%), Subfoveal serous detachment in 9 eyes (14%) ,Foveal tract ional retinal detachment in 1 eye (2%).

CORRELATION BETWEEN FOVEAL THICKNESS AND VISUAL ACUITY IN DIABETIC MACULOPATHY; In this study there was no correlation between visual acuity and foveal thickness measured by OCT

In GRADE 1 visual acuity 6/6 to 6/18 There were 6 eyes (10%) in 400 to 500 microns thickness

In GRADE 2 VA 6/24 to 6/60 There were 15 eyes (24%) are in 300 to 400 microns thickness

In GRADE 3 VA < 6/60 There were 8 eyes(13%) in 300 to 400 micron thickness

IMPROVEMENT OF VISUAL ACUITY AFTER IVTA IN DIABETIC MACULOPATHY; In this study there was a definite increase in visual acuity after intravitreal triamcinolone acetonide after three months.

CME group showed more definite increase in visual acuity than other two groups.
The Pretreatment mean visual acuity in decimals was 0.2 (standard deviation=+/-0.16 and Post treatment mean visual acuity in decimals was 0.25(std dev=+/-0.16). and Visual acuity improved in 27 eyes and did not improve in 5 eyes paired “t” test(p value=0.009) which is statistically significant.

SPONGE LIKE THICKENING group showed increase in visual acuity than Subfoveal group but less than CME group

The Pretreatment mean visual acuity in decimals was 0.16(standard deviation=+/-0.14) and Post treatment mean visual acuity in decimals was 0.18(std dev=+/-0.16) and Visual acuity improved in 14 eyes and did not improve in 7 eyes paired “t” test(p value=0.25) statistically not significant

SUBFOVEAL SEROUS DETACHMENT group showed insignificant increase in visual acuity than above two groups

The Pretreatment mean visual acuity in decimals was 0.20(std dev=+/-0.14) and Post treatment mean visual acuity in decimals was 0.23(std dev=+/-0.16) and Visual acuity improved only in 5 eyes and did not improve in 4 eyes paired “t” test(p value=0.57) statistically very insignificant

IMPROVEMENT (DECREASE) OF FOVEAL THICKNESS AFTER IVTA IN DIABETIC MACULOPATHY: In this study there was a definite decrease in foveal thickness after intra vitreal triamcinolone acetonide after three months.

CME group showed more definite decrease in foveal thickness

The Pretreatment mean foveal thickness in microns was 452(std dev=+/-173) and Post treatment mean foveal thickness in microns was 273(std dev=+/-76) and the Thickness reduced in 31 eyes and was not reduced in 1 eye paired “t” test(p value=<0.001) statistically very significant

SPONGE LIKE THICKENING group also showed more definite decrease in foveal thickness

The Pretreatment mean foveal thickness in microns was 464(std dev=+/-102) and Post treatment mean foveal thickness in microns was 282(std dev=+/-50.55)
and the Thickness reduced in all 21 eyes paired “t” test (p value=<0.001) statistically very significant

SUBFOVEAL SEROUS DETACHMENT group showed only a slight reduction in foveal thickness

The Pretreatment mean foveal thickness in microns was 404(std dev=+/-96 and
Post treatment mean foveal thickness in microns was 344(std dev=+/-192) and the Thickness reduced in 6 eyes and was not reduced in 3 eyes paired “t” test (p value=0.43) statistically very insignificant

COMPARISION OF VISUAL ACUITY IMPROVEMENT AFTER IVTA BETWEEN THREE GROUPS OF DIABETIC MACULOPATHY: (Chi square test)

In this study CME group showed more improvement in visual acuity than Sponge like thickening group (p=0.13) and Sub foveal group (p=0.06).And Sponge like thickening showed more improvement than Sub foveal group(p=0.55)

COMPARISION OF FOVEAL THICKNESS REDUCTION AFTER IVTA BETWEEN THREE GROUPS OF DIABETIC MACULOPATHY (Chi square test)

In this study CME group and Sponge like thickening both showed more reduction in foveal thickness (p=0.55)

The CME group showed more significant reduction in foveal thickness than Sub foveal group (p=0.007) and Sponge like thickening group more significant reduction in foveal thickness than Sub foveal group (p=0.006).

RESULTS

GENDER

SEX	MALE	FEMALE
NUMBER OF EYES	28	12
PERCENTAGE	70%	30%

AGE DISTRIBUTION

AGE	31 TO40	41 TO50	51 TO 60	61 TO 70
NO PATIENTS	3	2	21	14
PERCENTAGE	7.5%	5%	52.5%	35%

TYPE OF DIABETES

TYPE OF DM	IDDM	NIDDM
NO OF PATIENTS	3	37
PERCENTAGE	7.5%	92.5%

DURATION OF DIABETES

DURATION	< 5 YEARS	5 TO 10 YEARS	10 TO 15 YEARS
NO OF PATIENTS	6	6	28
PERCANTAGE	15%	15%	70%

TYPE OF DIABETIC RETINOPATHY

TYPE OF DR	PDR	NPDR
NUMBER OF EYES	14	26
PERCENTAGE	35%	65%

TYPE OF DIABETIC MACULOPATHY CLINICALLY

TYPE OF MACULOPATHY	CSME	CME	DIFFUSE EDEMA	FIBROUS PROLIFERATION
NO OF EYES	58	2	2	1
PERCENTAGE	92%	3.2%	3.2%	1.6%

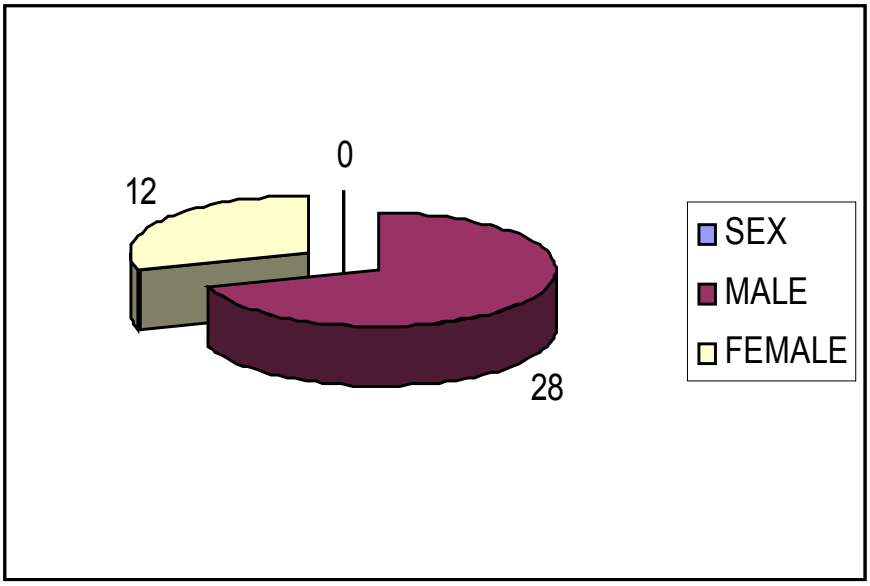
TYPE OF DIABETIC MACULOPATHY WITH FFA

TYPE	DIFFUSE LEAK	PETTALOID PATTERN	FOCAL LEAKS	NON PERFUSION	BLOCKED FLUORESCENCE
NO OF EYES	49	3	7	3	1
PERCENT	77.8%	4.8%	11.2%	4.8%	1.6%

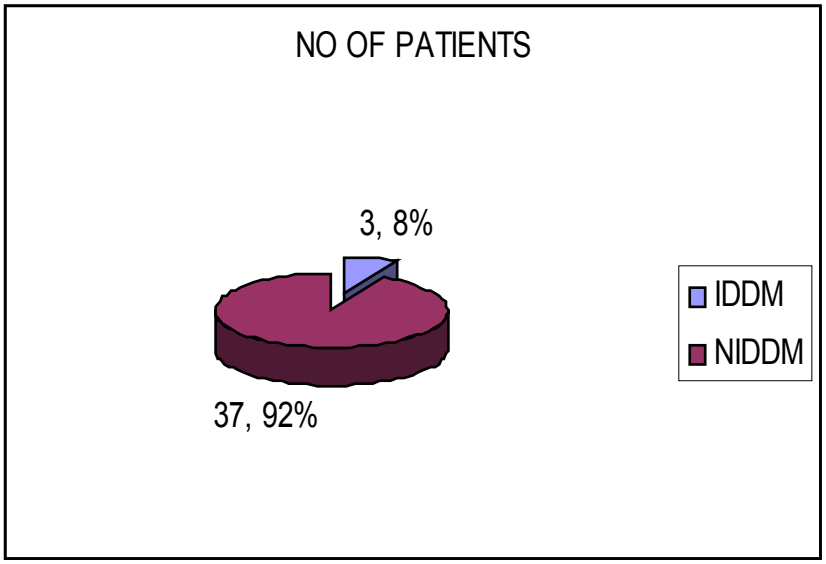
TYPE OF DIABETIC MACULOPATHY BY OCT

TYPE	CYSTOID MACULAR EDEMA	SPONGE LIKE THICKENING	SUBFOVEAL SEROUS DETACHMENT	FOVEAL TRACTIONAL DETACHMENT
NO OF EYES	32	21	9	1
PERCENTAGE	50.8%	33.3%	13.8%	1.6 %

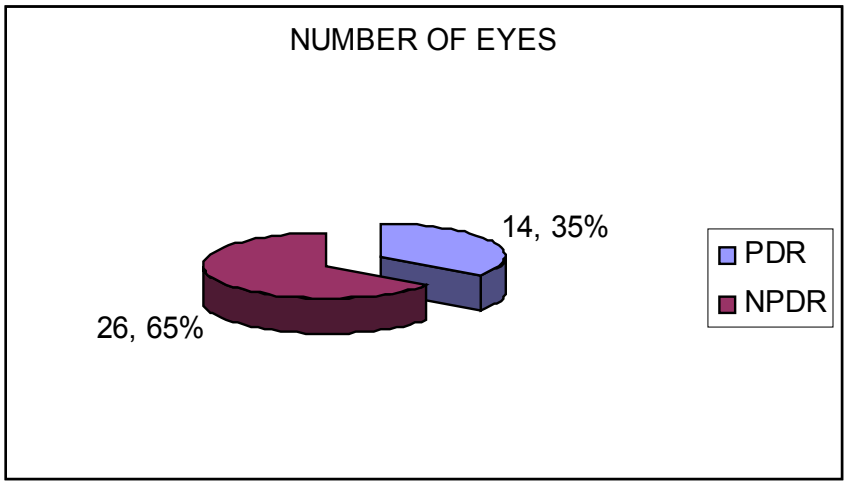
GENDER DISTRIBUTION

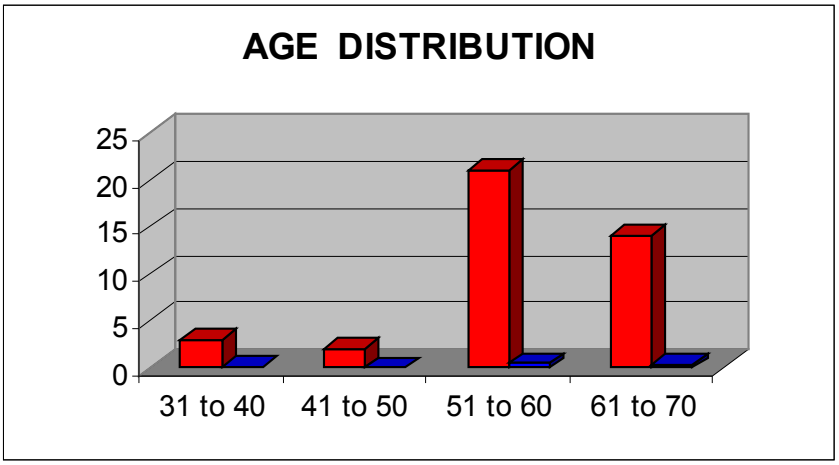


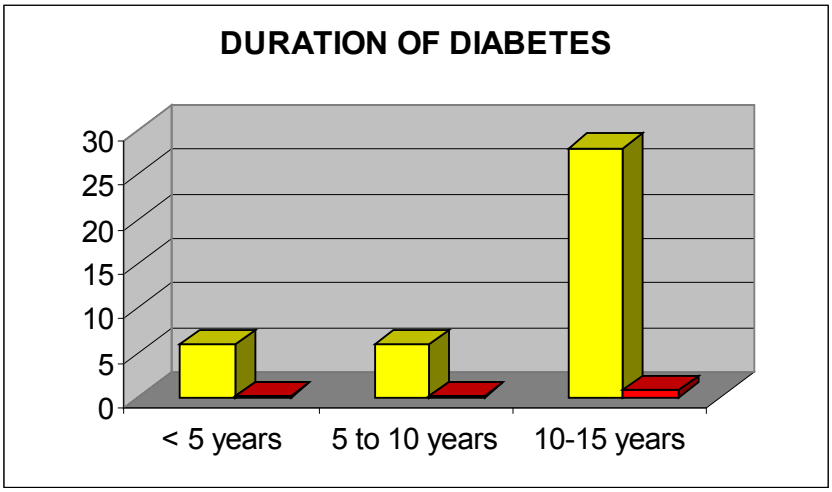
TYPE OF DIABETES

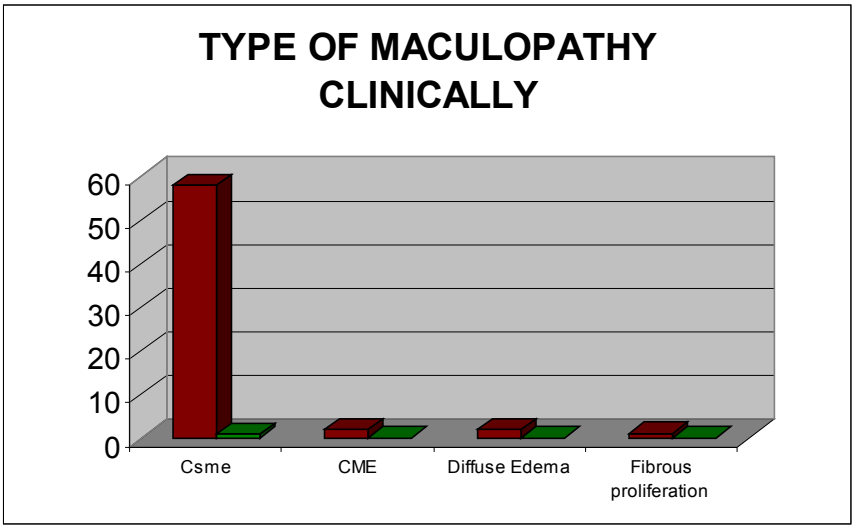


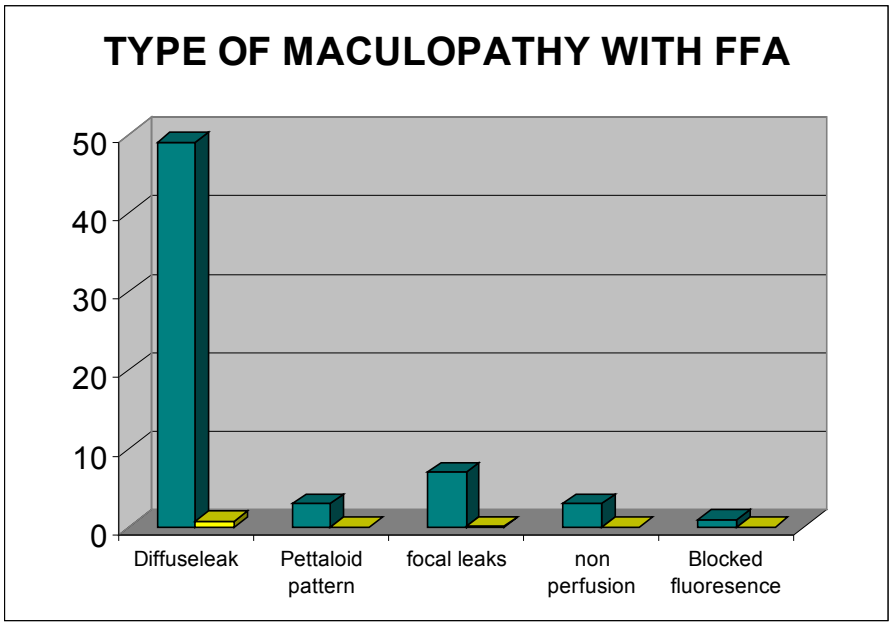
TYPE OF DIABETIC RETINOPATHY


NUMBER OF EYES
14, 35%
26, 65%
PDR
NPDR


AGE DISTRIBUTION
25
20
15
10
5
0
31 to 40
41 to 50
51 to 60
61 to 70


DURATION OF DIABETES
30
25
20
15
10
5
0
< 5 years
5 to 10 years
10-15 years


TYPE OF MACULOPATHY CLINICALLY
60
50
40
30
20
10
0
Csme
CME
Diffuse Edema
Fibrous proliferation


TYPE OF MACULOPATHY WITH FFA
50
40
30
20
10
0
Diffuseleak
Pettaloid pattern
focal leaks
non perfusion
Blocked fluoresence

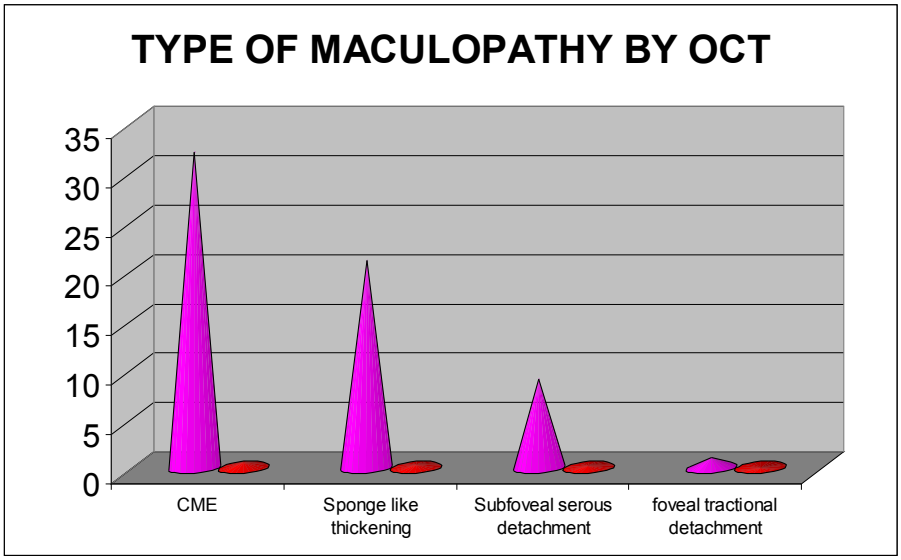

TYPE OF MACULOPATHY BY OCT
35
30
25
20
15
10
5
0
CME
Sponge like thickening
Subfoveal serous detachment
foveal tractional detachment

Fig-1 OCT Picture shows Sponge like thickening of macula

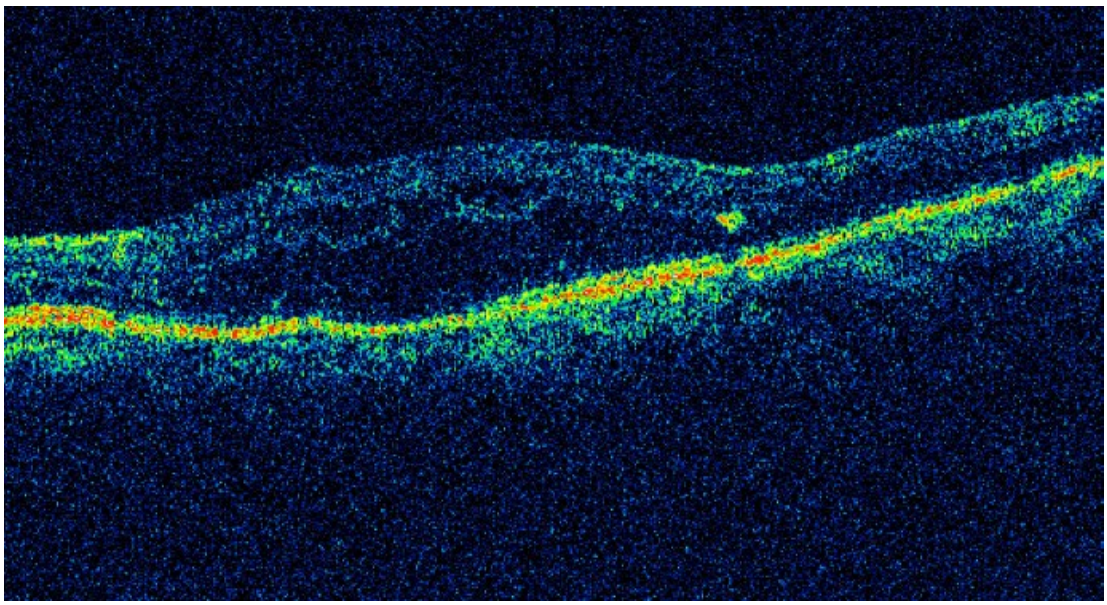


fig-2 OCT Picture shows cystoid macular edema

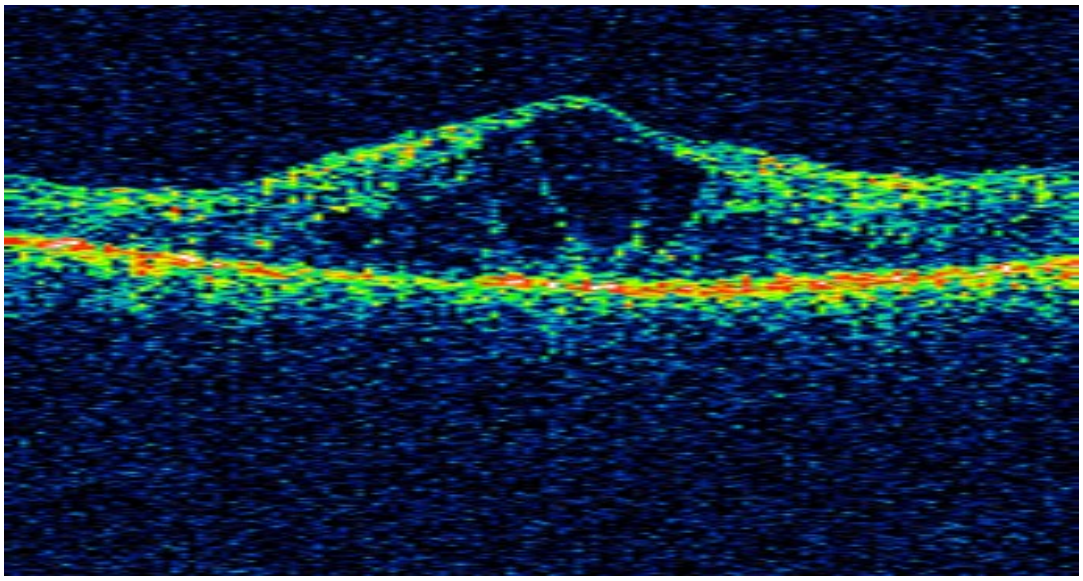


fig -3 Sub foveal serous detachment

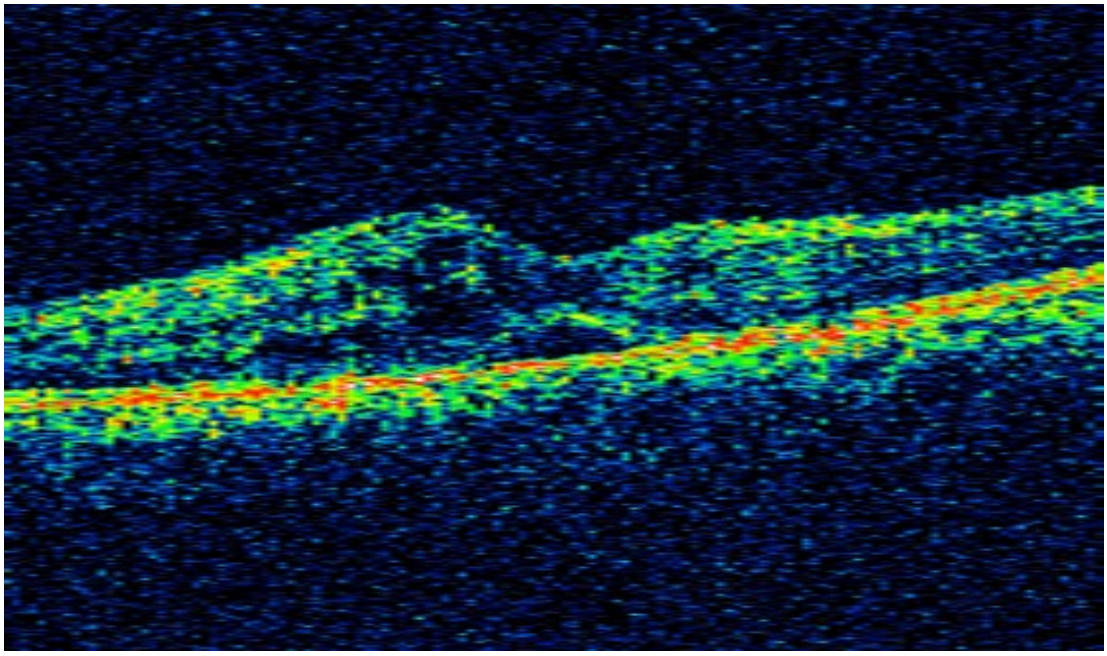
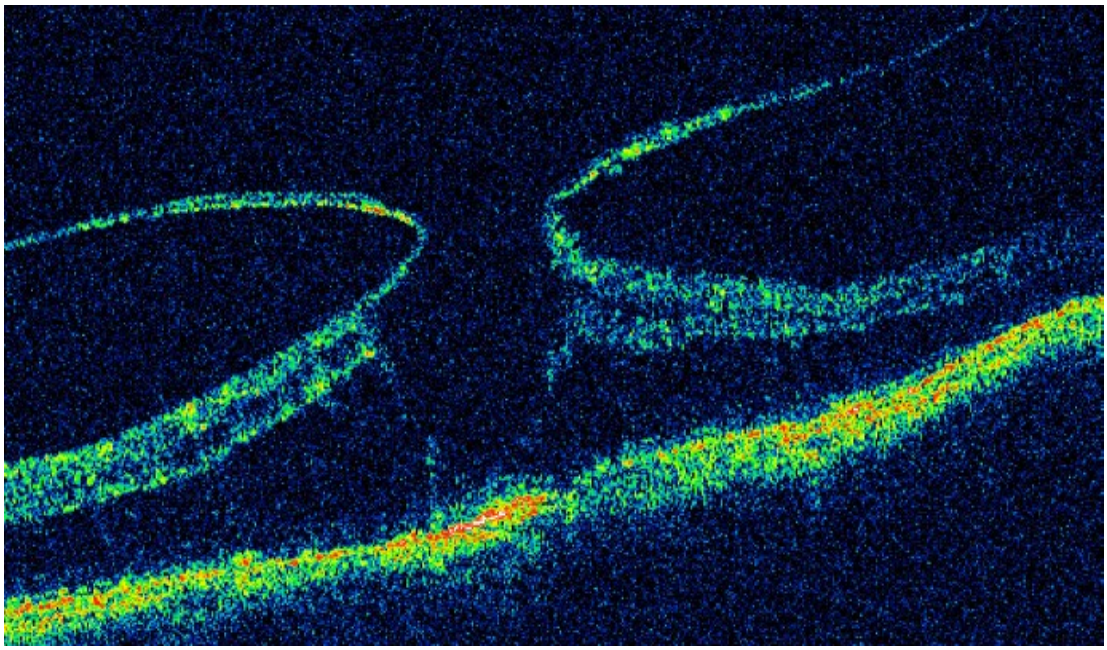
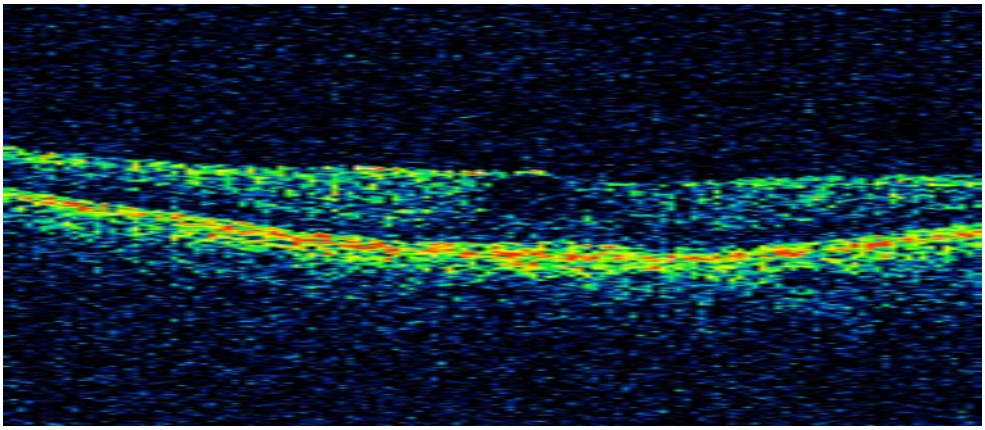


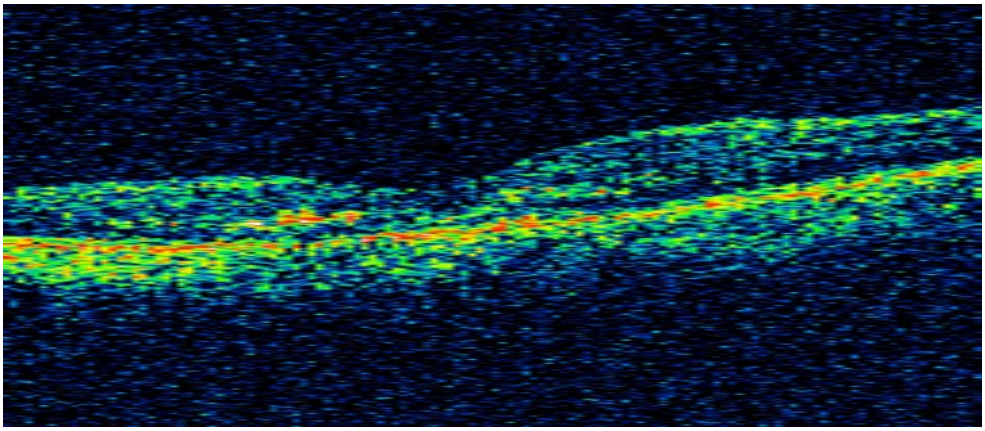
fig -4 Foveal tractional retinal detachment



OCT Picture shows cystoid macular edema
after 6 Months post IVTA



OCT Picture shows Sponge like thickening of macula
after 6 Months post IVTA



Sub foveal serous detachment after 6 Months post IVTA

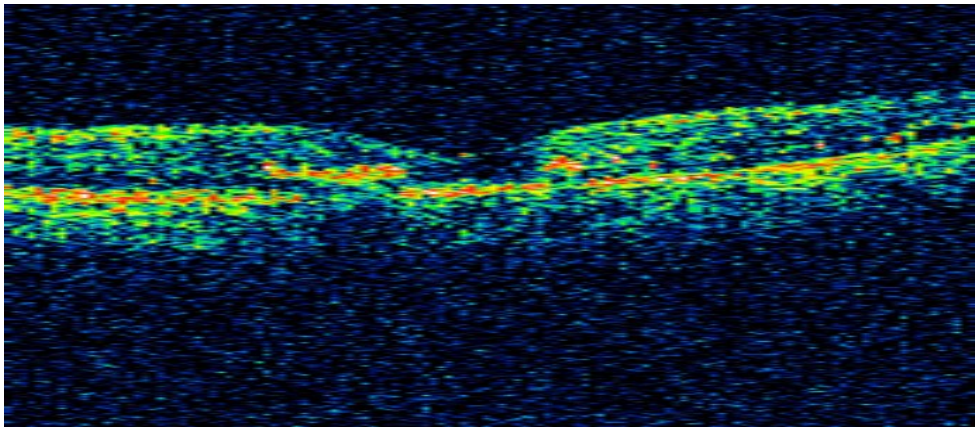


fig-8 Fundus picture shows NPDR

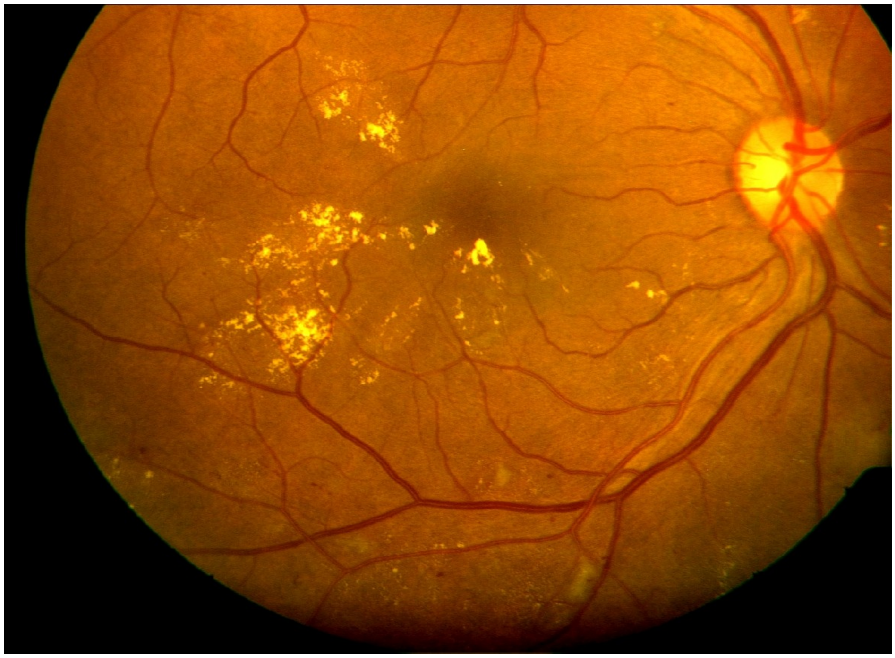


fig -9 fundus picture shows PDR with Fibrous traction

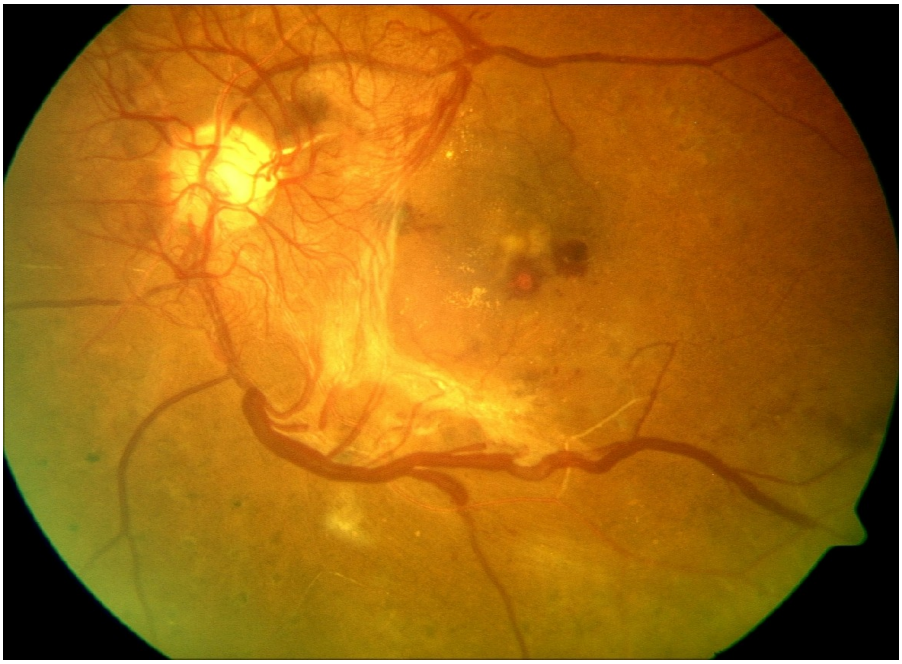


fig-10 PDR with Foveal tract ional retinal detachment

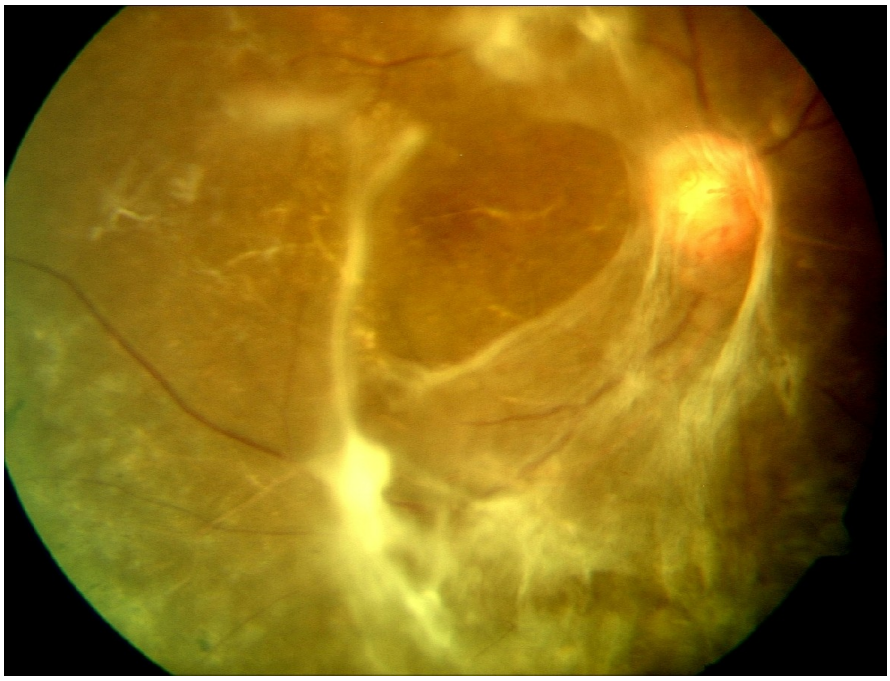


fig-11 NPDR with CSME

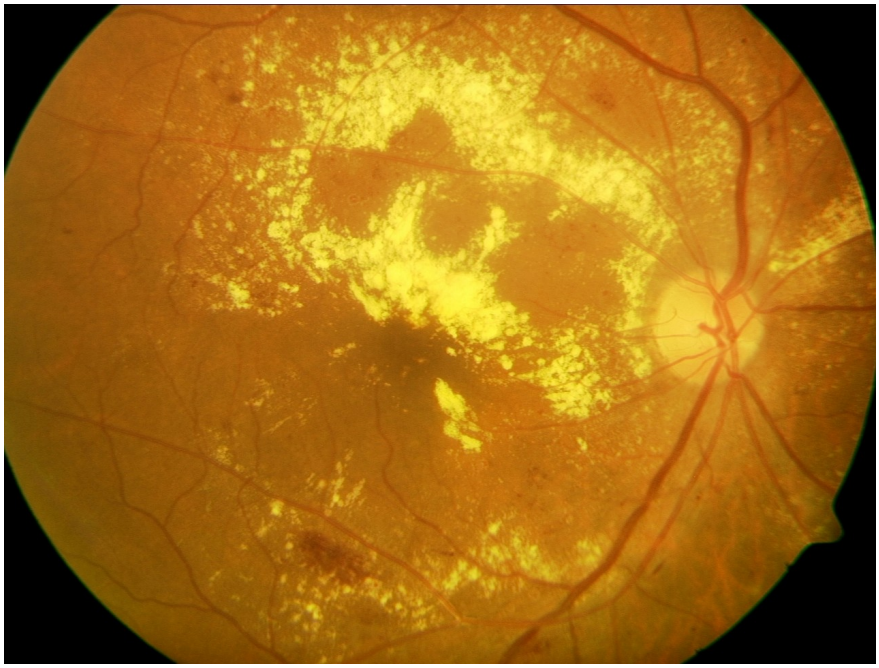


fig –12 PDR with CSME

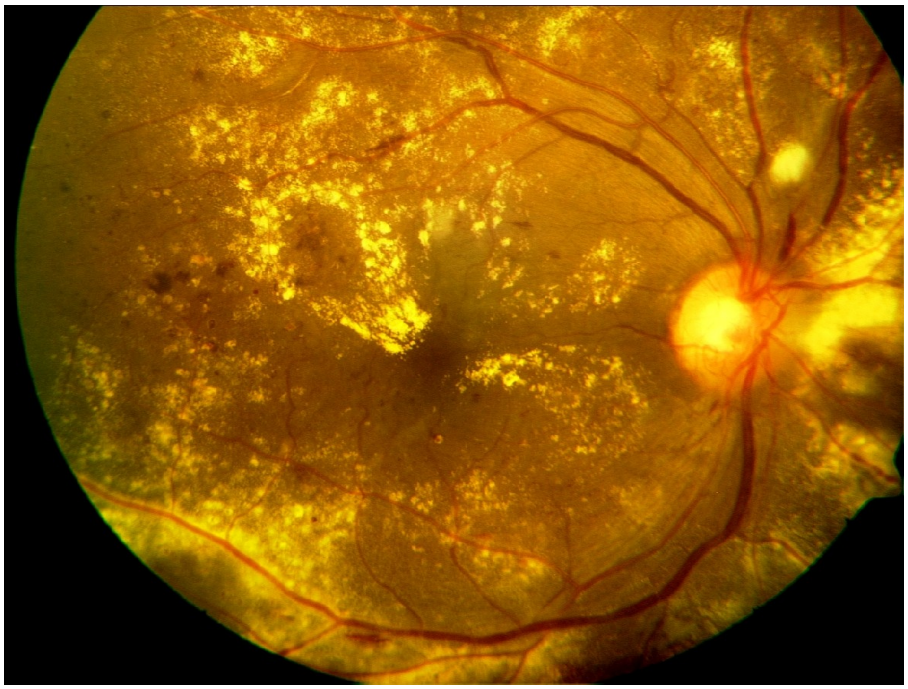


fig-13 FFA shows cystoid macular edema(flower petal pattern)

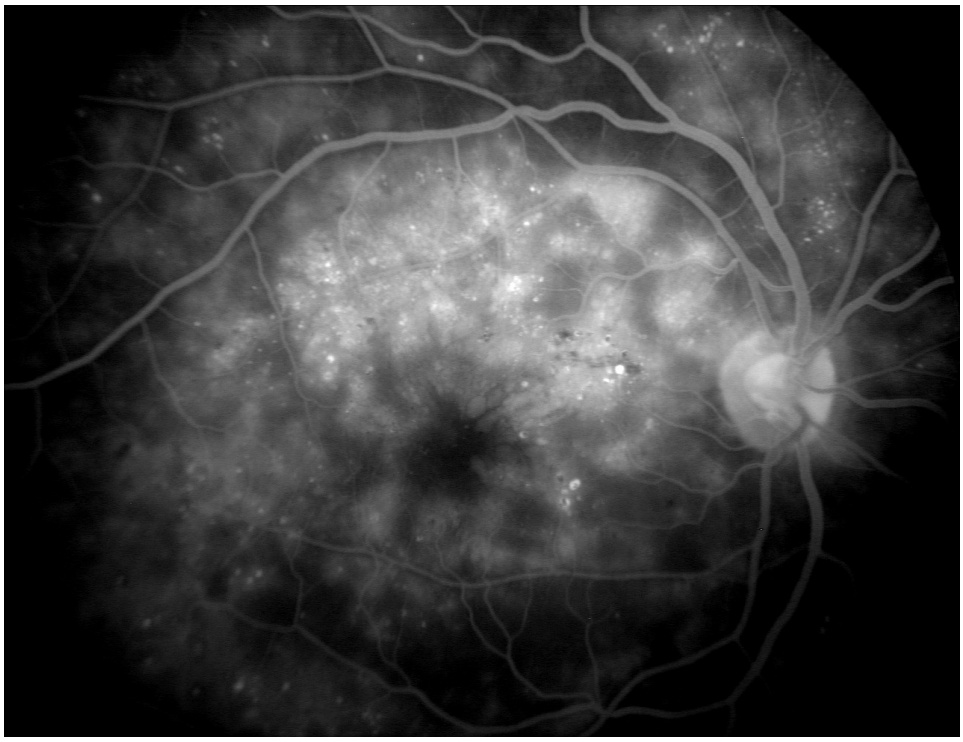


fig-14 FFA shows PDR with ischemic areas

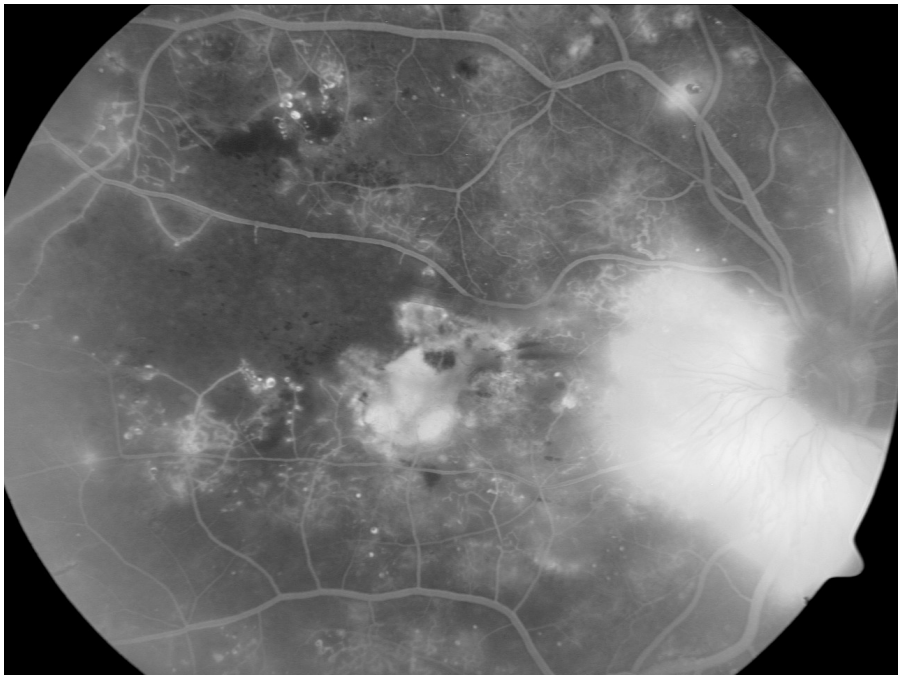


fig-15 FFA shows PDR with diffuse leak.

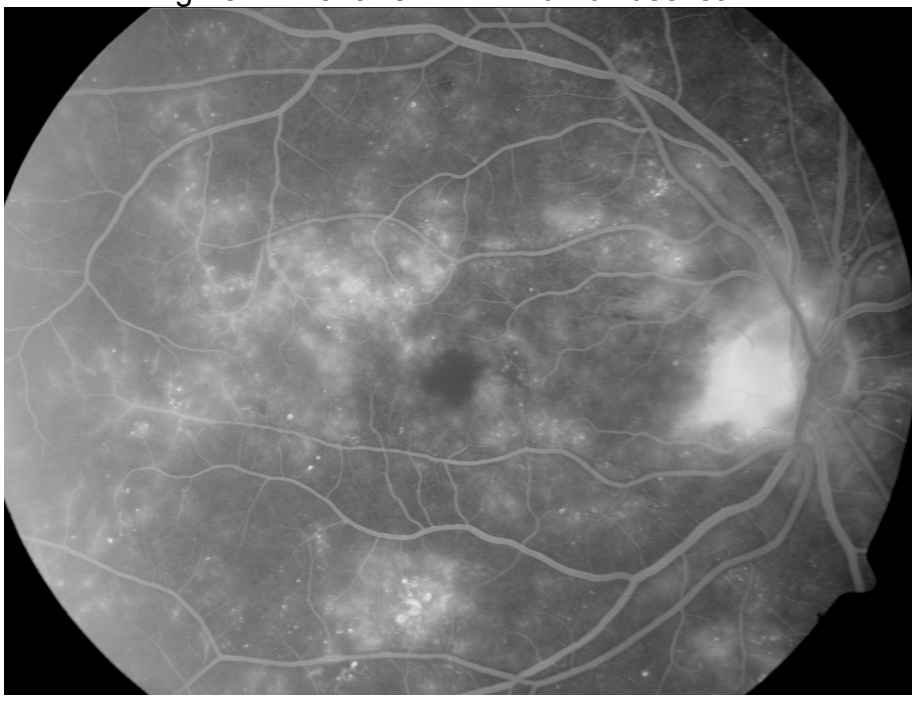
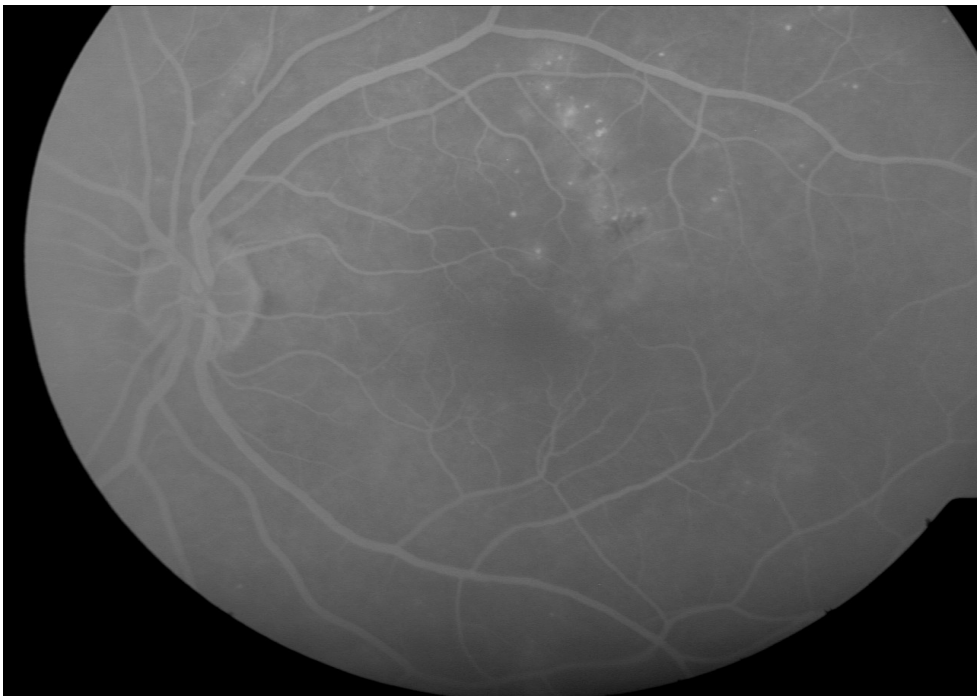


fig-16 FFA shows NPDR with focal leaks



DISCUSSION

DISCUSSION

Optical coherence tomography is a novel, objective test for the qualitative and quantitative evaluation of patients with diabetic maculopathy who may not be so precisely evaluated clinically and by fundus fluorescein angiographically

In this study 63 eyes of 40 patients were examined clinically, by FFA and by OCT . On comparison between them ,OCT precisely delineated more types of maculopathy than clinically and by FFA and so OCT is a more sensitive method of diagnosis in diabetic maculopathy.

This is similar to the study conducted by David J Browing et al (11,12) (Ophthalmology,2004. AJO 2005) in which they Showed 4 abnormalities (1) increased total macular volume (2) increased foveal zone thickness (3) increased inner parafoveal zone thickness and (4) increased outer parafoveal zone thickness. And so the diagnosis of diabetic macular edema by stereoscopic slit lamp biomicroscopic examination of fundus with diagnosis by OCT showed slit lamp diagnosis of DME is less sensitive than OCT.

In this study on comparison between FFA findings and OCT findings, OCT showed more types of maculopathy and so OCT is a useful tool to diagnose the type of maculopathy and as an adjuvant to FFA.

This is similar to the study conducted by SE WOONG KANG et al (13)(AJO FEB 2004) in which 145 eyes of 91 patients were categorized by FFA into 3 types ,focal leakage type ,diffuse leakage type, cystoid leakage type and by OCT into 4 types, Type 1-thickening with homogenous optical reflectivity, Type 2-thickening with markedly decreased optical reflectivity in outer retinal layer, Type 3a-foveolar detachment with out traction, Type 3b- foveolar detachment with traction and showed (a) The prevalence of OCT type 1 was higher in focal leakage type and diffuse leakage type than in cystoid leakage type (b) The prevalence of OCT type 2 and 3a was higher in cystoid leakage type than in focal and diffuse leakage type and they concluded that there was a correlation between features of FFA and OCT

In this study there is no correlation between foveal thickness by OCT and visual acuity.

In this study there is a definite increase in visual acuity after IVTA in all three groups but was statistically more significant in eyes with CME group.

In this study there is a significant reduction in foveal thickness after treatment with IVTA for diabetic macular edema.

The above three results are similar to the study conducted by JORGEN LARSSON et al (14) AJO APRIL 2005 in which they showed that 24 eyes with diabetic macular edema treated by IVTA had reduction in foveal thickness from mean of 462 +/- 154 microns to 257 +/- 114 microns (p value < 0.0001)after 3 months and the log MAR average visual acuity increased from 60.5+/-10.5 to 65.5 +/-11.1 (p value =0.0001) 3 months after IVTA treatment.

In this study eyes with CME group showed more improvement in visual acuity after IVTA than Sponge like thickening group and Sub foveal serous detachment group showed very little improvement

In this study eyes with both CME group and Sponge like thickening group showed significant reduction in foveal thickness after IVTA. Sub foveal serous detachment group showed very little reduction in foveal thickness after IVTA.

SUMMARY

SUMMARY

In this prospective comparative study 63 eyes of 40 patients with Diabetic maculopathy were included. All underwent examination clinically, by FFA, and by OCT. Optical coherence tomography precisely showed four types of diabetic maculopathy (a) Cystoid macular edema 32 eyes (b) Sponge like thickening 21 eyes (c) Subfoveal serous detachment 9 eyes (d) Foveal tract ional retinal detachment 1 eye, which were not seen clinically and by FFA.

The foveal thickness was measured by OCT in all patients at presentation and intravitreal triamcinolone acetonide is given to 62 eyes (except one eye with vitreo macular traction for which the treatment is mainly surgical) and after 6 months foveal thickness measured by OCT. The foveal thickness in diabetic macular edema reduced after intravitreal triamcinolone acetonide. More significantly in Cystoid macular edema group and Sponge like thickening group than in subfoveal serous detachment group.

There is no correlation between foveal thickness and visual acuity. But there is a increase in visual acuity after treatment with intravitreal triamcinolone acetonide. More significantly in Cystoid macular edema group.

OCT with its high resolution imaging of retina has a role in diagnosis of different types of diabetic maculopathy and to monitor the thickness of fovea before and after treatment with intra vitreal triamcinolone acetonide.

CONCLUSION

CONCLUSION

Optical Coherence Tomography a non contact, non invasive investigation is slowly emerging as an important diagnostic tool in differentiating various types of diabetic maculopathy. In comparison with clinical assessment and FFA it is definitely turning out to be a superior and more useful method of evaluating and monitoring Diabetic Maculopathy either as a single procedure and may be more useful when used in conjunction with fundus fluorescein angiography.

OCT is a useful tool to quantitatively monitor the response of different diabetic macular pathologies to intra vitreal triamcinolone acetonide by measuring the pre treatment foveal thickness and post treatment foveal thickness and there is a significant reduction in foveal thickness after intra vitreal triamcinolone acetonide.

There is no correlation between thickness of fovea measured by OCT and visual acuity.

Option for surgical intervention in cases of Diabetic Maculopathy with macular traction and taut posterior hyaloid membrane depends mainly on the OCT images which will clearly delineate accurately.

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APPENDIX

PROFORMA

Institute of Ophthalmology, Joseph Eye Hospital, Trichy.

OCT IN DIABETIC MACULOPATHY

Study No	**MRD No**	**Retina No**
Name	AGE	Sex
Systemic diseases	IHD/HT/Nephropathy/Others	
Diabetic Type 1 IDDM	Type 2 NIDDM	Duration
Treatment undergone	Diet and Exercise/Drugs	Insulin

VISUAL ACUITY

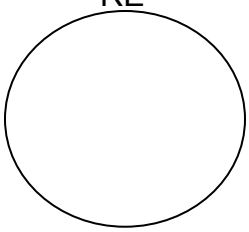
	Distant vision unaided	**With correction**	**BC DVA**
Right eye			
Left eye			
	Near vision unaided	With correction	BC NVA
RE			
LE			

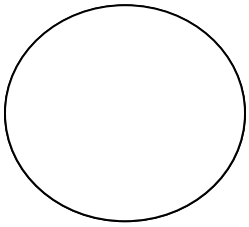
SLIT LAMP EXAMINATION

Anterior segment	**Right eye**	**Left eye**
Cornea		
Iris		
Pupil		
Lens		

FUNDUS EXAMINATION

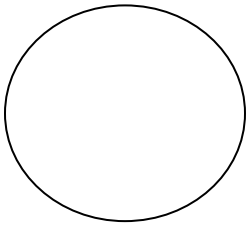
(Indirect/90D) RE LE

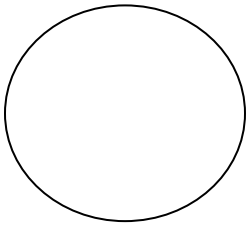




Diagnosis ; NPDR ;Mild/Moderate/Severe ;PDR ;MACULOPATHY ;Focal/Diffuse

FFA RE LE





OCT

SET UP

Size	Site	Length	Width	Height

MACULOPATHY(OCT)

1)SPONGE LIKE THICKENING

2)CYSTOID MACULAR EDEMA

3)SUBFOVEAL SEROUS DETACHMENT

4)TAUT POSTERIOR HYALOIDAL MEMBRANE

5)FOVEAL TRACTIONAL RETINAL DETACHMENT(Vitreo Retinal Traction)

MASTER CHART

MASTER CHART

MRD NO	AGE	SEX	DM TYPE	DURATION	RISK FACTOR	DR TYPE	EYE	CLINICAL	other findir	FFA	OCT	INTERVEN	PRE THIC	POSTTHIC	PRE V/A	DECIMAL	POST V/A	DECIMAL
485328	56	M	2	10 YEARS	NIL	PDR	LE	CSME	Nvd,HE	Diffuseleal	CME	IVTCA	314 micro	265	6/24.	0.25	6/12.	0.5
544776	64	M	2	12	NIL	NPDR	LE	CSME	HE at fov	Diffuseleal	CME	IVTCA	411	308	6/36.	0.17	6/24.	0.25
515998	57	F	2	25	HT	NPDR	RE	CSME	HE HGE	Diffuseleak	CME	IVTCA	336	313	6/60.	0.1	6/60.	0.1
515998	57	F	2	25	HT	NPDR	LE	CSME	HE HGE	Diffuseleal	CME	IVTCA	314	293	6/24.	0.25	6/18.	0.33
543060	65	F	2	10	NIL	NPDR	RE	CME	MA HeHge	pett diffuse	CME	IVTCA	276	216	6/36 IOL	0.17	6/24.	0.25
543060	65	F	2	10	NIL	NPDR	LE	CME	MA HeHge	Pettpattern	CME	IVTCA	269	246	6/36 iol	0.17	6/24.	0.25
522184	65	M	2	14	NIL	NPDR	RE	CSME	Nve,He	Diffuseleal	CME	IVTCA	503	212	5/60.	0.083	6/36.	0.17
522184	65	M	2	14	NIL	NPDR	LE	CSME	HE HGE	Diffuseleal	CME	IVTCA	660	260	6/36.	0.17	6/9.	0.67
521378	50	F	2	5	Renal	NPDR	RE	Diff edema	Nvd,HESE	Diffuseleal	CME	IVTCA	1162	254	3/60	0.05	6/36.	0.17
521378	50	F	2	5	Renal	NPDR	LE	Diff edema	MA HeHge	Diffuseleal	CME	IVTCA	670	253	6/36.	0.17	6/18.	0.33
528565	70	M	2	10	NIL	NPDR	RE	CSME	HE HGE	Diffuseleal	CME	IVTCA	531	348	6/18.	0.33	6/12.	0.5
527841	35	F	1	10	NIL	NPDR	LE	CSME	HE HGE	Diffuseleal	CME	IVTCA	619	276	6/60	0.1	6/36.	0.17
541412	65	M	2	10	NIL	NPDR	LE	CSME	SEHEHge	Diffuseleal	CME	IVTCA	303	212	6/24.	0.25	6/18.	0.33
539021	44	M	2	2	NIL	NPDR	RE	CSME	Edema	Diffuseleal	CME	IVTCA	698	209	6/24.	0.25	6/18.	0.33
494123	60	M	2	10	NIL	NPDR	LE	CSME	HE HGE	Diffuseleal	CME	IVTCA	306	214	6/36.	0.17	6/18.	0.33
481189	53	M	2	7	HT	PDR	LE	CSME	HE at fov	Diffuseleal	CME	IVTCA	439	218	6/9.	0.67	6/9.	0.67
553058	65	M	2	15	NIL	NPDR	RE	CSME	HE HGE	focal nve	CME	IVTCA	389	230	5/60.	0.083	6/60.	0.1
553058	65	M	2	15	NIL	NPDR	LE	CSME	HE HGE	focal nve	CME	IVTCA	319	240	5/60.	0.083	6/60.	0.1
549317	53	M	2	10	NIL	NPDR	RE	CSME	HE at fov	Diffuseleal	CME	IVTCA	391	256	6/24.	0.25	6/18.	0.33
555431	62	M	2	20	NIL	NPDR	LE	CSME	HE at fov	Diffuseleal	CME	IVTCA	590	313	3/60.	0.05	6/36.	0.17
569210	55	M	2	11	NIL	NPDR	RE	CSME	HE at fov	Diffuseleal	CME	IVTCA	289	212	6/18.	0.33	6/9.	0.67
569210	55	M	2	11	NIL	NPDR	LE	CSME	HE at fov	Diffuseleal	CME	IVTCA	313	240	6/60.	0.1	6/12.	0.5
589759	60	M	2	10	NIL	PDR	RE	CSME	HE HGE	Diffuseleal	CME	IVTCA	481	244	6/36.	0.17	6/12.	0.5
589759	60	M	2	10	NIL	NPDR	LE	CSME	Nve,He	Diffuseleal	CME	IVTCA	390	243	6/36	0.17	6/24.	0.25
580641	60	M	2	15	NIL	NPDR	RE	CSME	HE HGE	Diffuseleal	CME	IVTCA	315	260	6/24.	0.25	6/18.	0.33
495478	56	M	2	15	NIL	NPDR	RE	CSME	HE HGE	Diffuseleal	CME	IVTCA	497	437	6/36.	0.17	6/36.	0.17
495478	58	M	2	15	NIL	NPDR	LE	CSME	HE HGE	Diffuseleal	CME	IVTCA	549	272	6/36.	0.17	6/24.	0.25
489323	52	M	2	12	NIL	PDR	RE	CSME	Nve,ede hg	Diffuseleal	CME	IVTCA	469	300	6/18.	0.33	6/18.	0.33
489323	52	M	2	12	NIL	PDR	LE	CSME	Nve edehe	Diffuseleal	CME	IVTCA	433	589	6/12.	0.5	6/36.	0.17
480150	57	F	2	12	NIL	PDR	LE	CSME	Nvd vit he	Diffuseleal	CME	IVTCA	500	342	6/60.	0.1	6/36.	0.17
554361	65	F	2	15	NIL	NPDR	LE	CSME	cws hehge	Nvenonpel	CME	IVTCA	374	242	6/60.	0.1.	6/36.	0.17
582540	65	F	2	10	NIL	NPDR	RE	CSME	HE HGE	irmadiffeal	CME	IVTCA	353	214	4/60iol	0.067	6/60.	0.1
569772	61	M	2	15	NIL	PDR	LE	CSME	Nve hehge	Diffuseleal	sponge	IVTCA	580	355	6/60.	0.1	6/60.	0.1
567295	52	F	2	7	NIL	NPDR	RE	CSME	HE	Diffuseleal	sponge	IVTCA	320	269	1/60.	0.016	6/60.	0.1
567295	52	F	2	7	NIL	NPDR	LE	CSME	HE	Diffuseleal	sponge	IVTCA	367	270	6/60.	0.1	6/60.	0.1
545773	63	M	2	5	NIL	PDR	RE	CSME	NVD	Diffuseleal	sponge	IVTCA	456	290	6/24.	0.25	6/18.	0.33
545773	63	M	2	5	NIL	PDR	LE	CSME	NVEHGE	Diffuseleal	sponge	IVTCA	531	313	2/60iol	0.033	4/60iol	0.067
513574	54	M	2	10	HT	NPDR	LE	CSME	HE HGE	Diffuseleal	sponge	IVTCA	420	270	6/18.	0.33	6/18.	0.33
516281	65	F	2	20	NIL	PDR	RE	CSME	HE HGE	Diffuseleal	sponge	IVTCA	534	204	5/60.	0.083	6/60	0.1
525204	55	M	2	5	NIL	NPDR	RE	CSME	HE HGE	CME	sponge	IVTCA	631	392	6/60IOL	0.1	6/36IOL	0.17
543052	56	M	2	10	HT	NPDR	RE	CSME	HE HGE	focal nve	sponge	IVTCA	500	318	6/36.	0.17	6/36	0.17
582732	59	F	2	20	NIL	PDR	RE	CSME	vithgeHE	Diffuseleal	sponge	IVTCA	554	314	3/60.	0.05	3/60.	0.05
582732	59	F	2	20	NIL	PDR	LE	CSME	Nvehe	Diffuseleal	sponge	IVTCA	661	354	5/60.	0.083	6/60.	0.1
593942	56	M	2	4	NIL	PDR	LE	CSME	HE HGE	NveDiffeal	sponge	IVTCA	445	280	2/60.	0.033	5/60.	0.083
502214	53	M	2	10	NIL	NPDR	RE	CSME	HE HGE	focal leaks	sponge	IVTCA	416	229	6/12.	0.5	6/9.	0.67
502214	53	M	2	10	NIL	NPDR	LE	CSME	HE HGE	focal leaks	sponge	IVTCA	402	237	6/12.	0.5	6/9.	0.67
501697	51	F	2	6M	NIL	NPDR	RE	CSME	HE HGE	Diffuseleal	sponge	IVTCA	558	292	6/60.	0.1	6/36.	0.17
481744	63	M	2	10	NIL	NPDR	RE	CSME	HE HGE	Nve Diffea	sponge	IVTCA	370	220	6/36.	0.17	6/18.	0.33
481744	63	M	2	10	NIL	NPDR	LE	CSME	HE HGE	Nve Diffeal	sponge	IVTCA	328	216	6/36.	0.17	6/18.	0.33
514512	53	M	2	15	NIL	PDR	RE	CSME	Nvd he	focal nve	sponge	IVTCA	325	254	5/60	0.083	6/36	0.17
514512	53	M	2	15	NIL	PDR	LE	CSME	Nvd he	focal nve	sponge	IVTCA	489	315	6/60.	0.1	6/60.	0.1
554361	61	F	2	15	NIL	NPDR	RE	CSME	cws hehge	Nve Nonpr	sponge	IVTCA	497	294	6/24.	0.25	6/24.	0.25
582540	65	F	2	10	NIL	NPDR	LE	CSME	HE HGE	irmadiffeal	sponge	IVTCA	353	229	4/60iol	0.067	6/60.iol	0.1
481189	53	M	2	7	HT	PDR	RE	CSME	HE at fov	Diffuseleal	Subfovsert	IVTCA	323	130	6/12.	0.5	6/12.	0.5
580641	56	M	2	15	NIL	NPDR	LE	CSME	HE HGE	Diffuseleal	Subfovsert	IVTCA	289	287	6/18.	0.33	6/18.	0.33
589390	35	M	1	1	NIL	NPDR	LE	CSME	HE Edema	MA Nonpe	Subfovsert	IVTCA	400	800	6/36.	0.17	6/60.	0.1
545934	50	F	2	7	HT-	NPDR	RE	CSME	HE HGE	Diffuseleal	Subfovsert	IVTCA	506	266	6/36.	0.17	6/12.	0.5
513574	54	M	2	10	HT	NPDR	RE	CSME	HE HGE	Diffuseleal	Subfovsert	IVTCA	312	312	5/60.	0.083	5/60.	0.083
516281	65	F	2	20	NIL	PDR	LE	CSME	HE HGE	Diffuseleal	Subfovsert	IVTCA	375	176	4/60.	0.067	6/60.	0.1
501697	51	F	2	6M	NIL	NPDR	LE	CSME	HE HGE	Diffuseleal	Subfovsert	IVTCA	581	351	6/24.	0.25	6/18.	0.33
521036	65	M	2	2	NIL	NPDR	RE	CSME	HE HGE	Diffuseleal	Subfovsert	IVTCA	465	412	3/60.	0.05	4/60.	0.067
521036	65	M	2	2	NIL	NPDR	LE	CSME	HE HGE	Diffuseleal	Subfovsert	IVTCA	382	362	6/24iol	0.25	6/24iol	0.25
549905	60	M	2	14	NIL	PDR	RE	fibrprolifer		vitrettractio					3/60.	0.05		