" HOSPITAL BASED STUDY ON THE ROLE OF FUNDUS FLUORESCEIN ANGIOGRAPHY IN CLASSIFICATION AND DIAGNOSIS OF MACULAR DISEASES AT KLE'S DR.PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE BELAGAVI"

By

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Dissertation

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Endorsement by the Head Of Department, Principal/ Head of the Institution

This is to certify that the dissertation entitled **"HOSPITAL BASED STUDY ON THE ROLE OF FUNDUS FLUORESCEIN ANGIOGRAPHY IN CLASSIFICATION AND DIAGNOSIS OF MACULAR DISEASES AT KLE'S DR.PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE BELAGAVI"** is a bonafide research work done by **REG. NO.BK0115003.**

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LIST OF ABBREVIATIONS USED

AJO	-	American Journal of Ophthalmology
ARMD	-	Age Related Macular Degeneration
BRVO	-	Branch Retinal Vein Occlusion
CME	-	Cystoid Macular Edema
CNV	-	Choroidal Neovascularisation
CNVM	-	Choroidal Neovascular Membrane
CO	-	Clinical ophthalmoscopy
CRAO	-	Central Retinal Artery Occlusion
CRVO	-	Central Retinal Vein Occlusion
CSCR	-	Central Serous Chorio Retinopathy
CSME	-	Clinically significant macular edema
CV	-	Colour Vision
DM	-	Diabetic Maculopathy
DME	-	Diabetic Macular Edema
DR	-	Diabetic Retinopathy
FAZ	-	Foveal Avascular Zone

FFA	-	Fundus Fluorescein Angiography
GA	-	Geographic atrophy
ICGA	-	Indocyanine green angiography
IV	-	Intravenous
MD	-	Macular disorders
MVL	-	Moderate Visual Loss
NPDR	-	Non Proliferative Diabetic Retinopathy
NV	-	Near Vision
NVE	-	Neovascularisation of the disc
NVE	-	Neovascularisation Elsewhere
OPD	-	Outpatient department
PED	-	Pigment Epithelial Detachment
RPE	-	Retinal Pigment Epithelium
SRNVM	-	Subretinal Neovascular Membrane
VA	-	Visual Acuity

ABSTRACT

Background and objectives

Retinal disease is the primary cause of 12.7% of blindness in a population based surveys in India. Macular diseases are the most frequent constituiting 35.6% of all posterior segment disease. Macular diseases like Age Related Macular Degeneration, Central Serous Chorio Retinopathy, Diabetic Macular Edema, Vascular occlusive diseases can cause irreversible blindness and thus requires detailed evaluation and management. Fundus Fluorescein Angiography (FFA) acts as an important diagnostic modality in the evaluation of retinal disorders. It is very useful for tracing retinal lesions and it is conclusive in almost 80% of cases. It helps us to examine structures in macular region which are beyond the reach of direct ophthalmoscopy and fundus photography

The objective of our study is

1. To assess the role of Fundus Fluorescein Angiography (FFA) in classification and diagnosis of macular diseases

Methodology

The present study was a hospital based one year study conducted in the Department of Ophthalmology, KLES Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi to study the role of Fundus Fluorescein Angiography (FFA) in classification and diagnosis of macular diseases during the period of 1stJanuary 2016 to 31st December 2016. The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belagavi. Detailed history was

taken from the patient and a thorough ocular and systemic examination was done. All patients were examined by conventional methods of ophthalmoscopy (direct, indirect and slit lamp examination with +90 D lens) followed by a Fundus Fluorescein angiography. Ophthalmoscopic and fluorescein angiography findings were analyzed and categorized.

Results

In present study majority of the participants were males (63.6%).The mean age of the study population was 58.95 $\pm$ 19.70 years.

66 participants were analyzed and sub-divided into categories of ARMD, Diabetic Maculopathy, vascular occlusive disorders, macular dystrophy, CSCR, inflammatory causes and macular hole. Majority of the participants were having ARMD (30.30%) and Diabetic Maculopathy (31.81%) where as 15.15% participants were having CSCR and 12.12% were having vascular occlusions. The least participants were having inflammatory causes followed by macular dystrophy and macular hole.

FFA confirmed the diagnosis in 62.12. % of cases and altered the diagnosis in 37.87% cases. FFA has classified the lesions in 64% of cases. On statistical analysis there was 90.91% agreement between clinical ophthalmoscopy and FFA with $P<0.001$ which is found to be statistically significant.4.68% of the patients experienced only nausea as adverse reaction, no anaphylactic reaction was observed in our study.

CONCLUSION

FFA has played a major role in diagnosing wet ARMD, especially in diagnosing CNVM. It is a superior diagnostic modality in differentiating macular edema from macular ischaemia in vascular disorders .It is of immense value in

confirming and classifying the lesions in diabetic maculopathy. FFA provides definitive diagnosis in CSCR by detecting the exact leakage points.FFA played a major role in diagnosing new vessels and planning for further treatment. FFA is a superior diagnostic tool and is a necessity for evaluating, localizing and classifying of lesions in macular diseases

Keywords: Fundus Fluorescein angiography, Macular diseases, Age Related Macular Degeneration, Diabetic Maculopathy, Central Serous Chorio Retinopathy, Vascular diseases

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

LIST OF PHOTOGRAPHS

INTRODUCTION

The trend of retinal blindness has changed its pattern over the years in developing countries. Diabetic retinopathy, age-related macular degeneration (ARMD) and retinopathy of prematurity (ROP) have become the important and increasing causes of retinal blindness. It is expected that there would be 57 million diabetics in India by 2025 and 137 million people older than 65 years of age by 2021. By 2030, it is estimated that the number of diabetics greater than 64 years of age will be greater than 82 million in developing countries and greater than 42 million in developed countries. The prevalence of ARMD ranges from 0.6 to 1.1% in developing countries.[1]

Diabetic Retinopathy is a well-known complication of diabetes mellitus . Diabetic macular edema is a common complication of diabetic retinopathy and is one of the leading causes of loss of visual acuity and blindness worldwide. The prevalence of DME varies with the type, duration and stage of diabetic retinopathy and found to be 3% in mild non proliferative diabetic retinopathy (NPDR), 38% in moderate to-severe NPDR and 71% with proliferative diabetic retinopathy (PDR) [2]

ARMD is the leading cause of vision loss and blindness in people over the age of 50 years in the developed world. ARMD leads to blindness in 18% of the population in the age group of 65-75 years and in 30% of persons aged above 75 years.[3]

Macula lutea also known as clinical posterior pole which contains central 1.5mm depression the fovea centralis, where only cones are present in the neuro epithelial layers.

Macula is concerned with precise visual function of acuity, form sense, colour differentiation and stereopsis.

Macular diseases like Age Related Macular Degeneration (ARMD), Central Serous Retinopathy (CSCR),Diabetic Macular Edema(DME), Vascular occlusive diseases can cause irreversible blindness and thus requires detailed evaluation and management.[4]

Fundus Fluorescein Angiography(FFA) involves photographic surveillance of the passage of fluorescein through the retinal and choroidal circulations following intravenous injection. A modern angiogram consists of a series of high contrast black and white transparencies taken at a speed of 0.6 seconds interval [2]

FFA was introduced in clinical use in 1961 by Novotny and Alvis who perfect the photographic study of the human retinal circulation. For over few decades fundus photography and fluorescein angiography have been extremely valuable for expanding our knowledge of anatomy and pathophysiology of the retina and choroid. FFA helps in the diagnosis and monitoring of the treatment of various retinal and choroidal diseases.[5]

FFA is relatively a safe procedure with no life threatening complications. In a recent survey of 11,898 instances of FFA in Australia, only 132 adverse reactions (1.1%) were recorded, mostly of nausea and vomiting, and no serious adverse reactions or deaths occurred.[6]

Fundus fluorescein angiography acts as an important diagnostic modality in the evaluation of retinal disorders. It is very useful for tracing retinal lesions and it is conclusive in almost 80% of cases. It helps us to examine structures in macular region which are beyond the reach of direct ophthalmoscopy and fundus photography.

AIMS AND OBJECTIVES

- To assess the role of Fundus Fluorescein Angiography (FFA) in classification and diagnosis of macular diseases.

REVIEW OF LITERATURE

HISTORY

Adolf Baeyer in 1871 described the methods of producing new organic dyes including sodium fluorescein.[7] Ehrlich used fluorescein dye to examine flow of aqueous humor. Maumene and Mclean published first article on fluorescein angiography in 1960 in the diagnosis of choroidal hemangioma in a 30 year old patient.[8]

In 1959, Harold Novotny and David Alvas the two medical students of Indiana University began working under John Hickam, The Chairman of Medicine on a research project funded by the United States Air Force. They searched for a photographic technique to estimate blood oxygen concentrations in retinal vessels. They thought whether it would be possible to photograph fluorescence in the retinal vessels using a fluorescent dye such as fluorescein. They got approval from and sent a blood sample mixed with fluorescein to James Hartigan at the Eli Lily lab to determine the excitation and emission wavelengths of fluorescein. The peak excitation of fluorescein was found around 480nm and peak emission to be 520nm with spectrofluorometer.[9]

Donald Gass began publishing his experience with FFA in 1967 and his efforts led to the wider acceptance of the technique in the evaluation of retinal disease.[10]

The Zeiss fundus camera was used with an electric flash which took one photograph every 12 seconds. The blue and green filters were used in the pathway of incident and emitted light respectively .A control picture was taken before injecting

fluorescein followed by 5ml of Fluorescein injection in the anticubital vein. The first photograph was taken at arterial fluorescence followed by serial photographs at 12 seconds interval.[11]

Properties of fluorescein:[12]

Fluorescein is a manufactured organic compound and dye. It is available as a dark orange/red powder slightly soluble in water and alcohol. It is widely used as a fluorescent tracer for many applications.

Fluorescein is a fluorophore commonly used in microscopy, in laser as the gain medium, in forensics and serology to detect latent blood stains, and in dye tracing. Fluorescein has an absorption maximum at 494 nm and emission maximum of 512 nm (in water). The major derivatives are fluoresce in isothiocyanate (FITC) and 6-FAM phosphoramidite.[12]

It is on the World Health Organization's List of Essential Medicines, the most important medications needed in a basic health system.[13]

It has the following properties :[14]

- ✓ Low Molecular weight - 367.27 daltons.
- ✓ Freely soluble in water.
- ✓ Heat stable.
- ✓ Yellowish red colour and shows yellowish green fluorescence when the solution is neutral or alkaline.
- ✓ The yellowish green fluorescence is visible in aqueous solutions of sodium fluorescein in concentration as low as 1 : 1000000.

Medical use of fluorescein:[15]

Fluorescein sodium, the sodium salt of fluorescein, is used extensively as a diagnostic tool in the field of ophthalmology and optometry, where

1. Topical fluorescein is used in the diagnosis of corneal abrasions, corneal ulcers and herpetic corneal infections.

2. It is also used in rigid gas permeable contact lens fitting to evaluate the tear layer under the lens.

3. It is available as sterile single-use sachets containing lint-free paper applicators soaked in fluorescein sodium.

4. Intravenous or oral fluorescein is used in fluorescein angiography in research and to diagnose and categorize vascular disorders including retinal macular degeneration, diabetic retinopathy, inflammatory intraocular conditions, and intraocular tumors.

5. It is also being used increasingly during surgery for brain tumors.

6. Diluted fluorescein dye has been used to localise multiple muscular ventricular septal defects(VSD) during open heart surgery and confirm the presence of any residual defects.

Chemistry:[16]

The fluorescence of this molecule is very intense.The peak excitation occurs at 494 nm and peak emission at 521 nm.

Fluorescein has a pK_a of 6.4, and its ionization equilibrium leads to pH-dependent absorption and emission over the range of 5 to 9. Also, the fluorescence lifetimes of the protonated and deprotonated forms of fluorescein are approximately 3 and 4 ns, which allows for pH determination from non intensity based measurements. The lifetimes can be recovered using time-correlated single photon counting or phase-modulation fluorimetry.

Fluorescein has an isosbestic point (equal absorption for all pH values) at 460 nm.

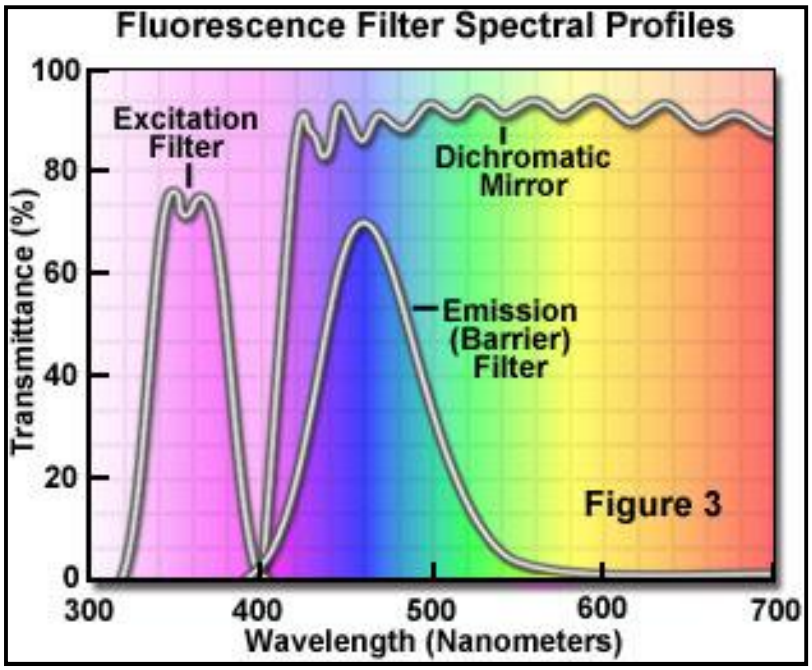


Figure 1- Fluorescence filter spectral profile

Synthesis:[17]

Fluorescein was first synthesized by Adolf von Baeyer in 1871.[20] It can be prepared from phthalic anhydride and resorcinol in the presence of zinc chloride via the Friedel-Crafts reaction.

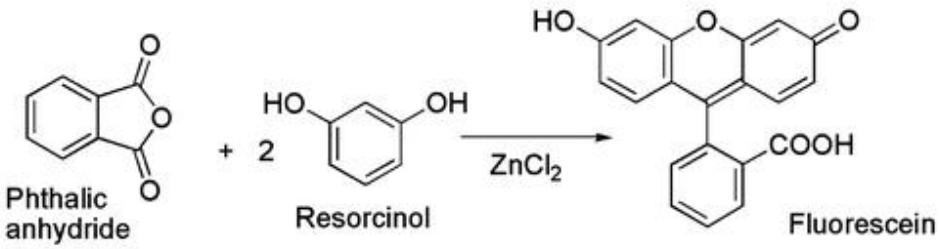


Figure no.2- synthesis of fluorescein

Fluorescein angiography principle: [18,19,20]

- Angiography uses the physical phenomenon of luminescence
- Luminescence: results when there is molecular absorption of electromagnetic radiation.

Luminescence : Whenever light is absorbed by certain substances, the photic energy may be converted into heat or chemical energy. This is seen when energy in the form of electromagnetic radiation is absorbed and then remitted at another frequency. This phenomenon is called as luminescence. There is always a shift from a shorter wave length to longer wave length of lower energy.

Fluorescence : Luminescence that stops as soon as the exciting stimulus is withdrawn is called fluorescence.It is the luminance which is maintained by continuous emission..

If there is a persisting after glow (after cessation of the exciting stimulus) it is called phosphorescence

- Electrons within the fluorescein molecule, on absorbing blue light (465 nm-490 nm) rise to higher energy orbital level .Because the molecules have become unstable after absorbing extraneous energythis state is transient. To

revert to a more stable state, the electrons give up the excess energy (as a longer wavelength green light, 520-530 nm) and fall back to their native orbital. This change in energy levels occurs over a brief period of time (less than 10-8 seconds) and the phenomenon so occurring is known as fluorescence

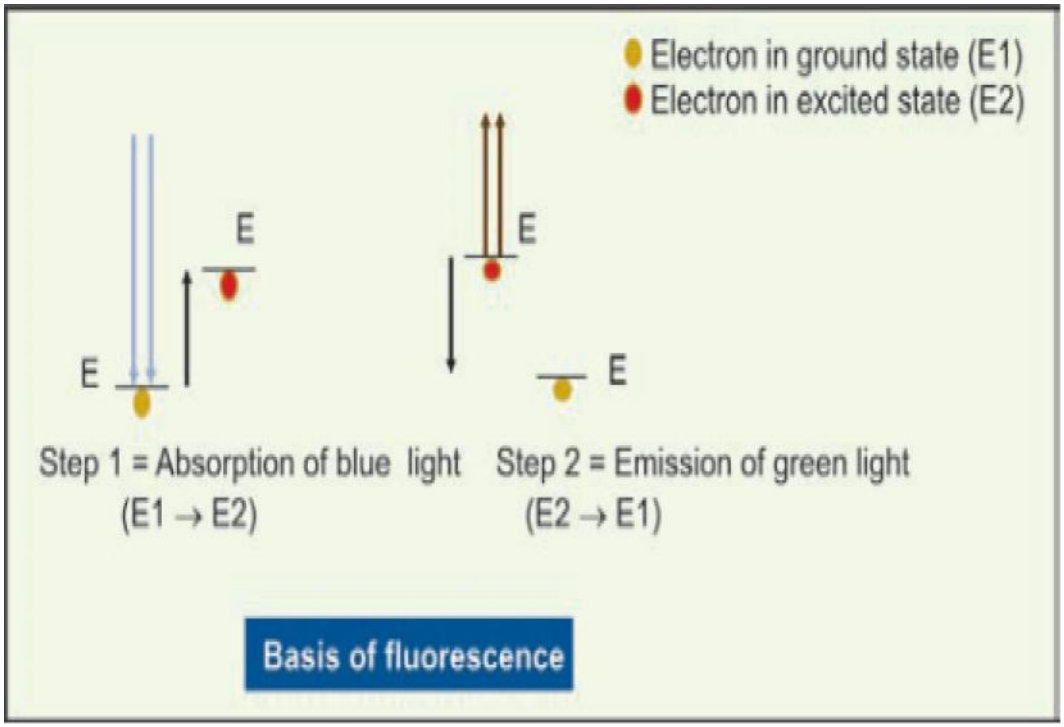


Figure 3-Principle of fluorescence

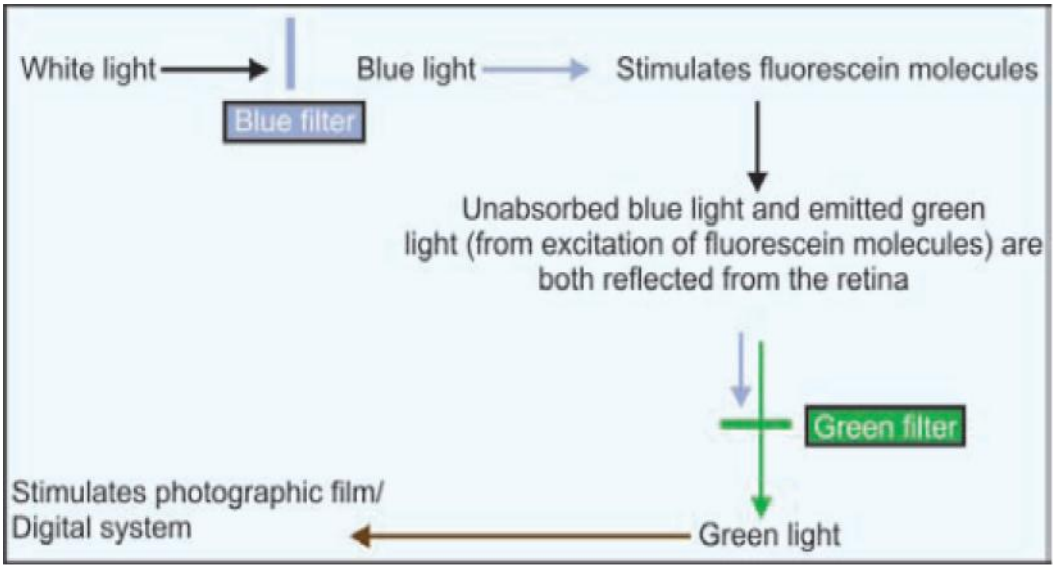


Figure 4- Principle Of Fluorescein Angiography

Optical principle-Excitation and emission of fluorescence

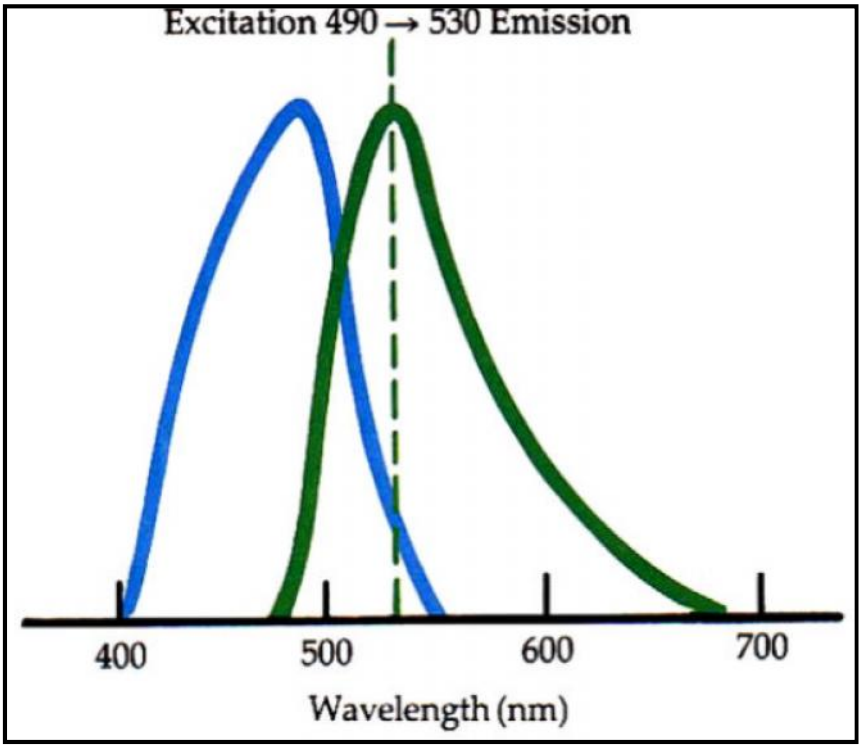


Figure 5- Optical principle of fluorescein angiography

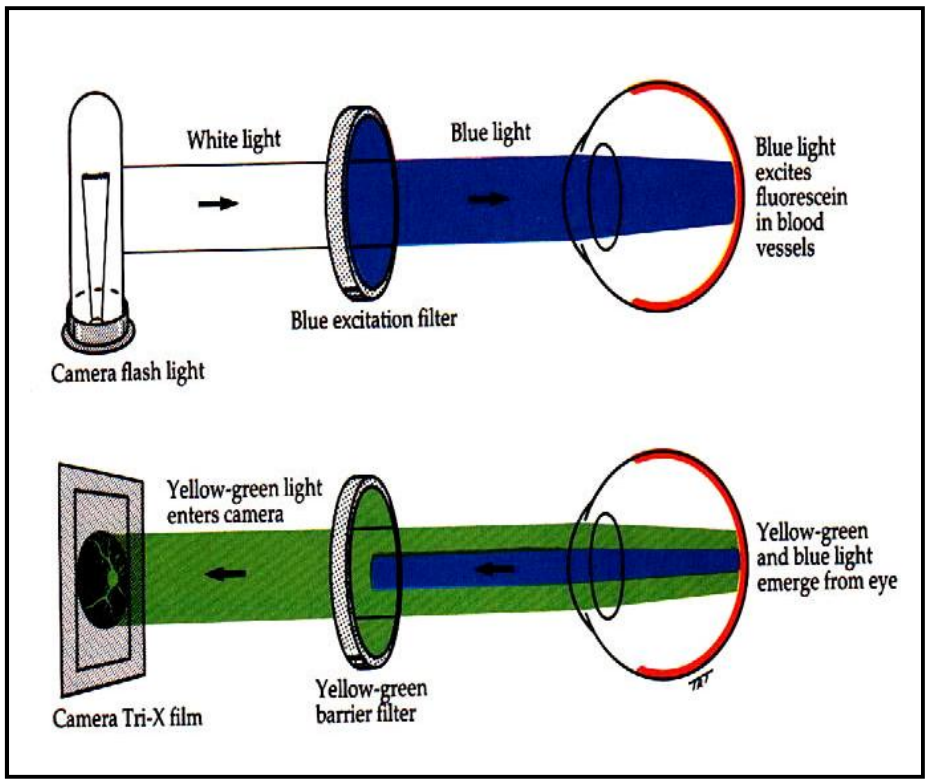


Figure 6- Filters In Fluorescein Angiography

FLUORESCEIN SOLUTION[21]

Solutions containing 500 mg of fluorescein are available in vials of :

1. 10 ml of 5%.
2. 5 ml of 10%.
3. 3 ml of 25% (750 mg).

The greater the volume, the longer the injection time will be; the smaller the volume, the more likely a significant percentage of fluorescein will remain in the venous dead space between the arm and the heart. For this reason we prefer 5 mL of 10% solution (500 mg fluorescein).

FLUORESCEIN ANGIOGRAPHY: THE TECHNIQUE PROCEDURE:[19,22]

PATIENT PREPARATION:

1. **INFORMED CONSENT**: This involves informing the patient about why and how (briefly) the procedure is being performed, the side-effects and also eliciting history that would be a contraindication for the procedure. He is emphatically told about the essence of time during the angiographic procedure and that a great deal would depend on his co-operation during the actual procedure.
2. **PUPILLARY DILATATION:** All patients pupils are dilated with a short acting mydriatic-cycloplegic combination (tropic amide 1% and phenylephrine 10%).
3. **INJECTION OF THE DYE** :

a) The patient is seated comfortably in front of the fundus camera, and an intravenous cannula is inserted.

b) A standard venous cannula should be used rather than a less secure 'butterfly' winged infusion set. After cannulation the line should be flushed with normal saline to check patency and exclude extravasation

c) Fluorescein, usually 5 mL of a 10% solution, is drawn up into a syringe. In eyes with opaque media, 3 mL of a 25% solution may afford better results

d) A 'red-free' image is captured first .

e) If indicated, a pre-injection study is performed to detect autofluorescence , with both the excitation and barrier filters in place

f) Fluorescein is injected over the course of a few seconds.

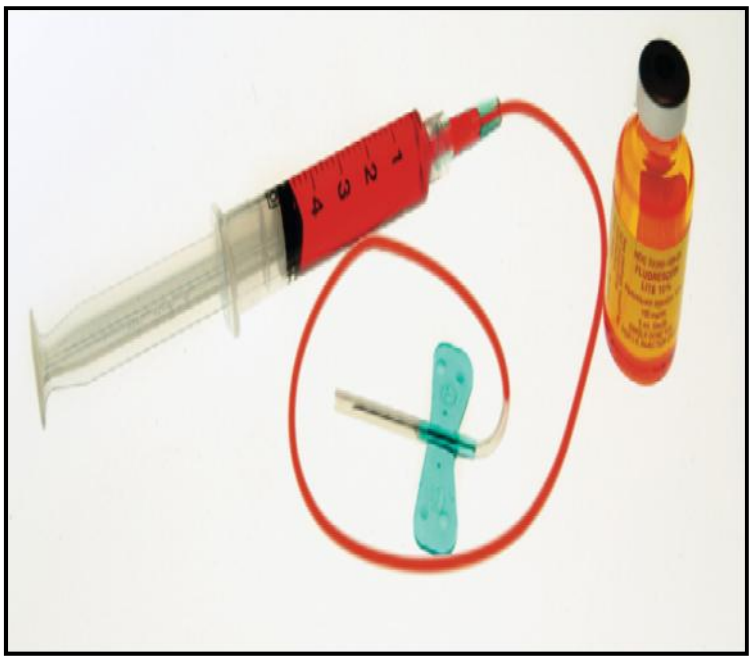


Figure 7-Fluorescein Dye With Cannula

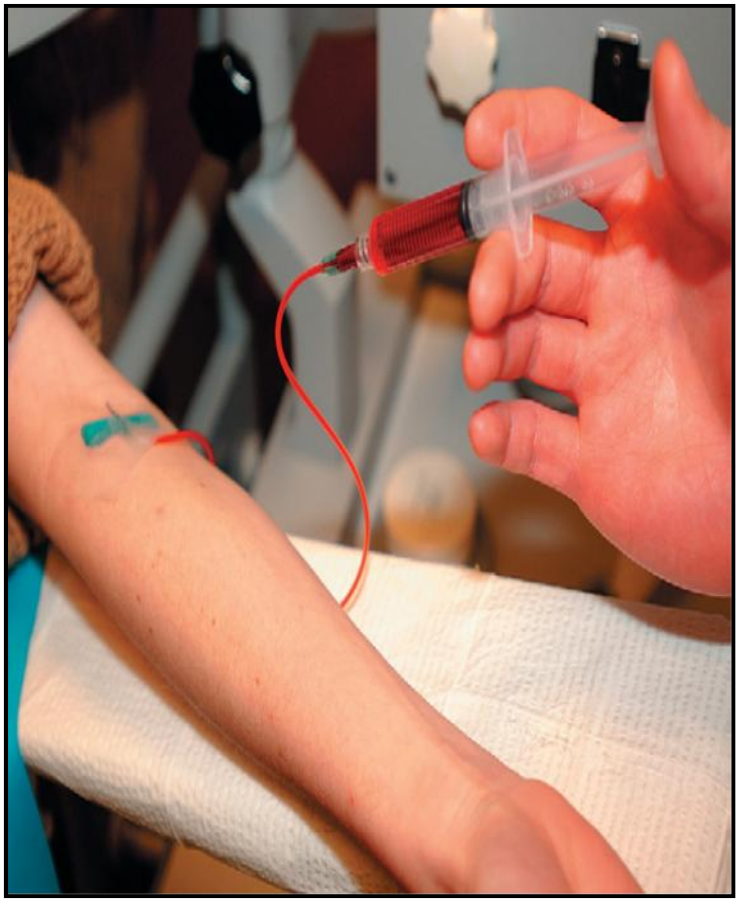


Figure 8-Injection of fluorescein dye

4. **COLOUR FUNDUS PHOTOGRAPHS:** With the patient seated comfortably and the camera parameters doubly checked, The red free photographs are taken first of the fellow eye followed by diseased eye

5. **FLUORESCEIN ANGIOGRAPHY SEQUENCE:**[19]

a) Images are taken at approximately 1 second intervals, beginning 5–10 seconds after injection of the dye and continuing through the desired phases

b) If the pathology is monocular, control pictures of the opposite eye should still be taken, usually after the transit phase has been photographed in one eye

c) If appropriate, late photographs may be taken after 10 minutes to show leakage, and occasionally after 20 minutes.

d) Stereo images may be helpful to demonstrate elevation and are usually taken by manually repositioning the camera sideways or by using a special device (a stereo separator) to adjust the image.

ANAPHYLAXIS DURING ANGIOGRAPHY [23,24]

- Anaphylaxis is a multisystem allergic reaction. The severity of the reaction is difficult to predict at its outset.The internal medicine literature advocates treating it early with subcutaneous or intramuscular epinephrine (1:1000).
- Anaphylaxis can involve four major organ systems: respiratory, cardiovascular, gastrointestinal and cutaneous. Involvement of any two of these organ systems meets the definition of anaphylaxis. So if a patient has itching and shortness of breath, the internal medicine literature advocates treating with epinephrine.

EMERGENCY EQUIPMENT NEEDED DURING ANGIOGRAPHY [24]

The things that must be available prior to injecting:

- Personnel —which is an MD in the office
- A cardiac monitor and bag and mask
- Supplies — such as IV fluid, needle, tape and gauze;
- Paperwork — the consent form, the symptoms checklist, and treatment flowsheets.

Drugs —

- IV diphenhydramine, and
- IM/SQ epinephrine (1:1000)

ADVERSE REACTIONS TO FFA: [23,25,26,27]

The study conducted by Fayyaz Musa on adverse reactions of fluorescein angiography in hypertensive and elderly males with mean age of 66.1 years. The most common adverse reaction seen was nausea .There were no cases of death, anaphy-laxis or serious cardiovascular complications.

A study by Anthony S L et al on Fluorescein angiography and adverse reactions on 11898 FFAs were performed over the study period. There were 132 (1.1%) adverse reactions. Nausea and vomiting were the two main adverse reactions and were found in 87 (0.7%) and 47 (0.4%) of the subjects, respectively. There were no severe adverse reactions such as seizure, myocardial infarction, anaphylactic type attack or death.

A prospective study carried out by Lira et al on Adverse reactions of fluorescein angiography concluded that cumulative incidence of 9.72% adverse reactions was observed in patients who had undergone this test for the first time. The

presence of the allergy history, diabetes or systemic arterial hypertension increased the incidence of adverse reactions to the dye.[27]

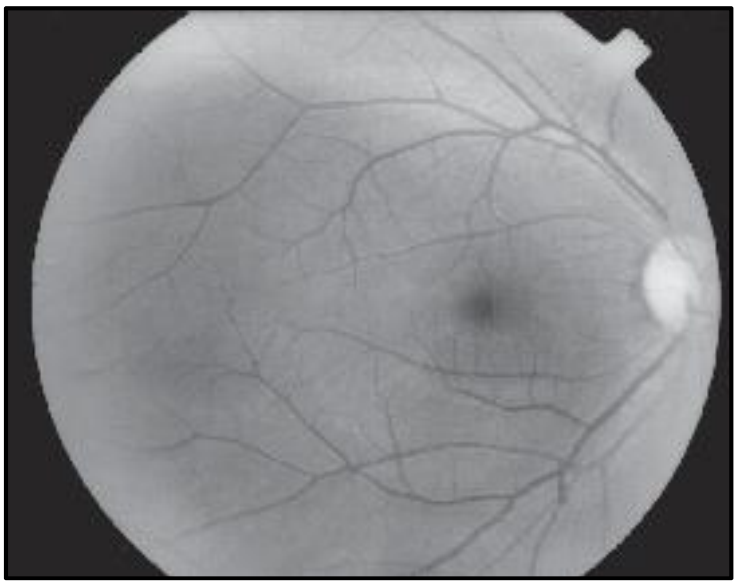
Adverse Reaction What to do immediately? [28]

Nausea / Vomiting	Reassure the patient that it is transient and would pass off with in some seconds (30-90) • Ask patient to breathe deeply and slowly • Provide emesis basin if vomiting sets in • For prophylaxis: Oral or Intramuscular injection of promethazine(Phenergan) 45 minutes before the procedure • For prophylaxis: rinse mouth with cool water before the procedure
Vasovagal Response	• Deep breathing if only lightheadedness (usually within ~ 30-90 seconds) • Protect from injury by preventing fall • Place patient supine and elevate foot end • Reassure patient as he recovers • Monitor blood pressure and keep under observation until he becomes stable • For prophylaxis: Small dose of diazepam 30 minutes before the procedure
Urticaria	• Intramuscular promethazine if mild (may be delayed for~30 minutes) • Intravenous corticosteroids if moderate to severe • Seek medical consultation • For prophylaxis: Oral or Intramuscular injection of promethazine (Phenergan) 45 minutes before the procedure can be given
Phlebitis	• Seek medical consultation if severe (usually diagnosed ~ 24 hours)
Generalized convulsions	• Protect from injury (prevent fall) • Prevent tongue bite (mouth gag) • Intravenous diazepam 5-10 mg • Seek medical consultation
Bronchospasm	• Intravenous bronchodilators, promethazine and corticosteroids • Administer oxygen using ambu bag • Monitor blood pressure and pulse • Seek medical consultation
Cardiac Arrest	• CPR • Assisted ventilation • Monitor pulse and blood pressure • Seek medical attention
Myocardial Infarction	• Reassure patient • Intravenous morphine / pethidine • Assisted ventilation with vitals monitoring • Seek medical attention

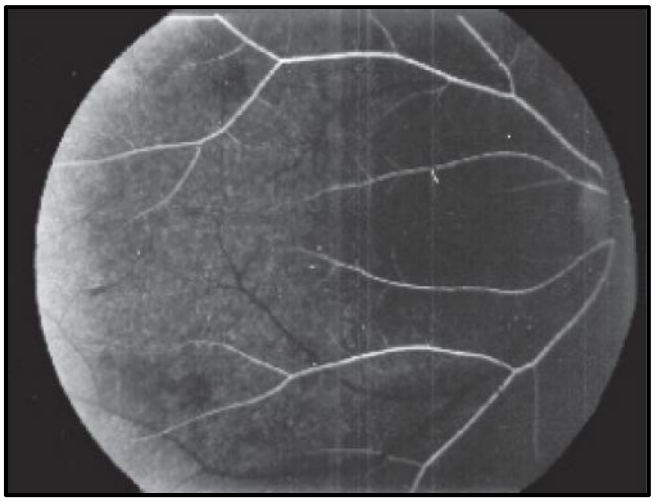
PHASES OF FLUORESCEIN ANGIOGRAPHY [29]

Fluorescein enters the eye through the ophthalmic artery, passing into the choroidal circulation through the short posterior ciliary arteries and into the retinal circulation through the central retinal artery. Because the route to the retinal circulation is slightly longer than that to the choroidal, the latter is filled about 1 second before the former . In the choroidal circulation, precise details are often not discernible mainly because of rapid leakage of free fluorescein from the choriocapillaris and also because the melanin in the RPE cells blocks choroidal fluorescence. The angiogram consists of the following overlapping phases.

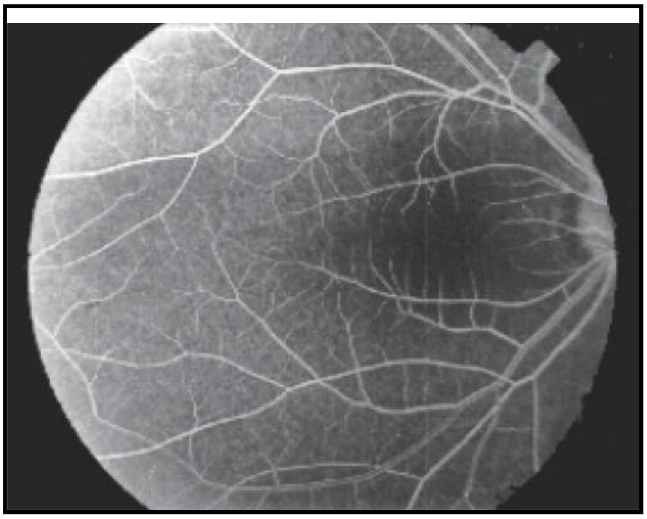
1. **The choroidal** (pre-arterial) phase typically occurs 9–15 seconds after dye injection (longer in patients with poor general circulation) and is characterized by patchy lobular filling of the choroid due to leakage of free fluorescein from the fenestrated choriocapillaris. A cilioretinal artery, if present, will fill at this time because it is derived from the posterior ciliary circulation .



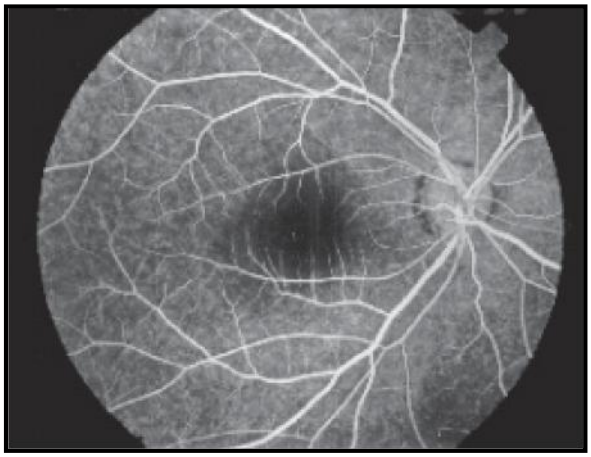
2. **The arterial phase** starts about a second after the onset of choroidal fluorescence, and shows retinal arteriolar filling and the continuation of choroidal filling .



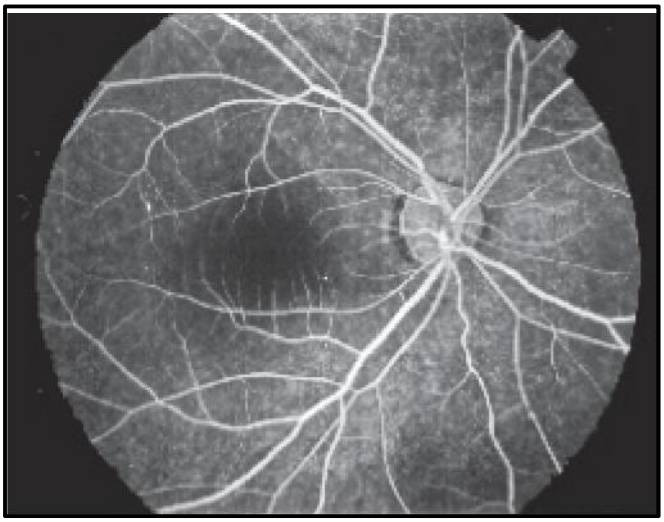
3. **The arteriovenous (capillary) phase** shows complete filling of the arteries and capillaries with early laminar flow in the veins in which the dye appears to line the venous wall leaving an axial hypofluorescent strip . This phenomenon reflects initial drainage from posterior pole capillaries filling the venous margins, as well as the small-vessel velocity profile, with faster plasma flow adjacent to vessel walls where cellular concentration is lower.



4. **The venous phase.** Laminar venous flow progresses to complete filling, with late venous phase featuring reducing arterial fluorescence. Maximal perifoveal capillary filling is reached at around 20–25 seconds in patients with normal cardiovascular function, and the first pass of fluorescein circulation is generally completed by approximately 30 seconds.



5. **The late (recirculation) phase** demonstrates the effects of continuous recirculation, dilution and elimination of the dye. With each succeeding wave, the intensity of fluorescence becomes weaker although the disc shows staining. Fluorescein is absent from the retinal vasculature after about 10 minutes.



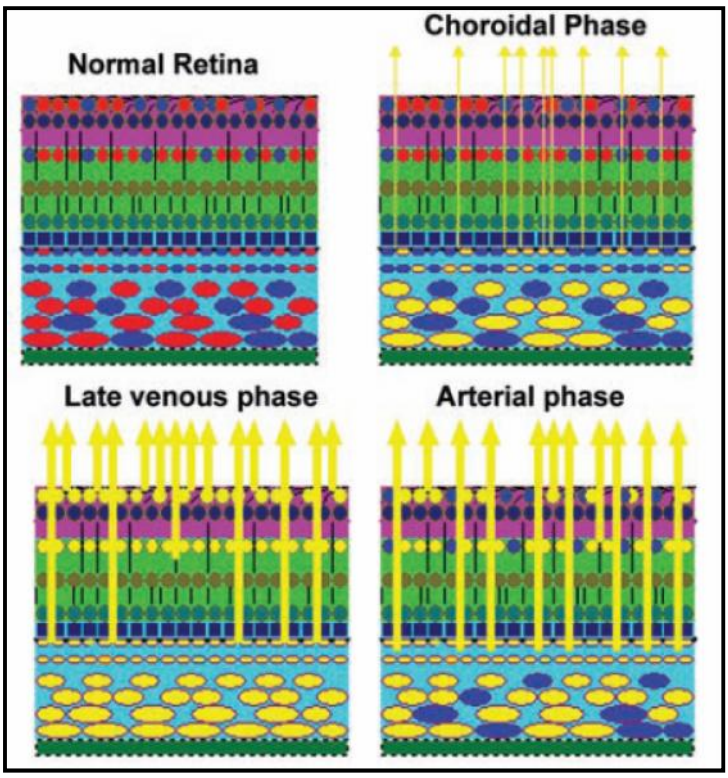


Figure 9- Phases of fluorescein angiogram

ANATOMY OF THE MACULA[30,31,32]

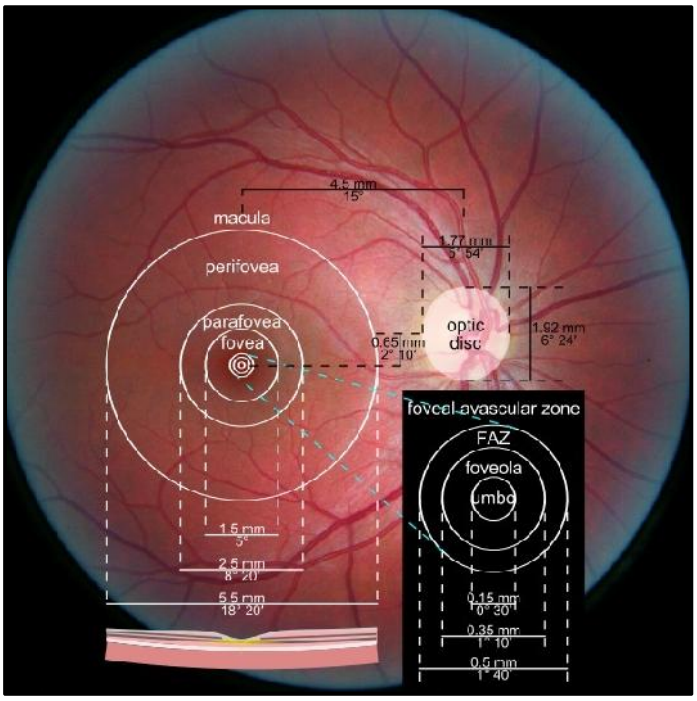


Figure 10-Anatomy of the Macula

Macula can be subdivided in to four zones [31]

1. Fovea
2. Foveola
3. Parafovea
4. Perifovea

1. Fovea (Latin: fovea means small pit or depression) is 1.5mm in diameter. Fovea is fully developed at the age of 4 years. It is located at the posterior pole of the globe, 4mm temporal to the optic disc & about 0.8 mm below the horizontal meridian. It represents 50^0 of the visual field. It has an average thickness of 0.25mm. At the centre of the fovea, the layers of the retina are thinner so that central concave indentation Foveola is produced. The downward slopping border of fovea which meets the floor of the foveal pit is known as clivus.

2. Foveola which measures 0.35mm in diameter represents the area of the highest visual acuity in the retina. This is partly due to the sole presence of cone receptors & partly due to its avascular structure. Tiny Depression in the very centre of foveola is known as Umbo, which corresponds to the ophthalmoscopically visible foveolar reflex which is seen in most of the normal eyes.

In the fovea, there is a densely packed arrangement of red & green cones with a density approaching 2, 00,000 cones per square millimeter. The central fovea has no rods. The density of cones falls off rapidly outside the fovea. This structural variation results from the centripetal migration of the first order & centrifugal lateral displacement of second & third order neuron during foetal maturation which occurs 3 months before & 3 months after term.

The Foveola largely consists of cones & their nuclei covered by a thin internal limiting membrane. All other retinal layers are absent in this region.

In the foveal region surrounding Foveola, cone axons are arranged obliquely (Henle's layer) to reach the margin of the fovea. The nerve fibres course obliquely & actually run almost parallel to the retinal surface in contrast to the vertical orientation (perpendicular to the retinal surface) Of the nerve fibres in extramacular retina.

Macula[32]

Fovea:

Diameter: 1.5mm

Thickness: 0.25mm

Represents $\mathbf{5^0}$ of the visual field

Foveola:

Diameter: 0.35 mm

Thickness: 0.13 mm

Represents $\mathbf{1^0}$ of the visual field

Macula:

Diameter: 5.5mm

Represents $\mathbf{15^0}$ of the visual field

The outer nuclear layer composed of the cell bodies of the rods and cones is about the same thickness in central and peripheral retina. In central retina the cones have oblique axons displacing their cell bodies from their synaptic pedicles in the outer plexiform layer (OPL). These oblique axons with accompanying Muller cell processes form a pale-staining fibrous-looking area known as the Henle fibre layer.

Within the central retina two other regions are distinguished outside the fovea.The Parafovea (0.5mm in width) & Perifovea (1.5mm in width).

Neuronal arrangement of central Retina:

- In central retina every cone is interconnected with a single bipolar cell, which in turn synapses with a single ganglion cell. This provides direct one-to-one relationship between the first three visual neurons.
- In extra macular region, At least 1/3rd of all the nerve fibres which enter the optic nerve originates in macular region and also the macular nerve fibres enter the optic nerve at its temporal edge but immediately course into the central region of the nerve

The dark appearance of the fovea: [33]

It is caused by three factors

- Absence of blood vessels in the FAZ.
- Blockage of background choroidal fluorescence due to the high density of xanthophyll at the fovea.
- Blockage of background choroidal fluorescence by the RPE cells at the fovea, which are larger and contain more melanin and lipofuscin than else where in the retina.

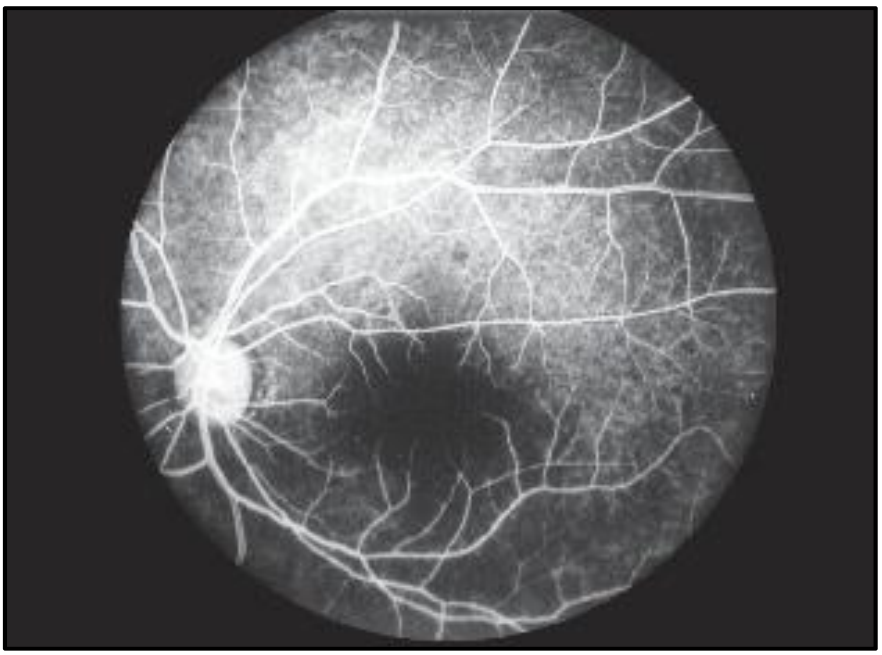


Figure 11-Foveal Avascular Zone(FAZ)

ALGORITHM FOR FFA INTERPRETATION :[19]

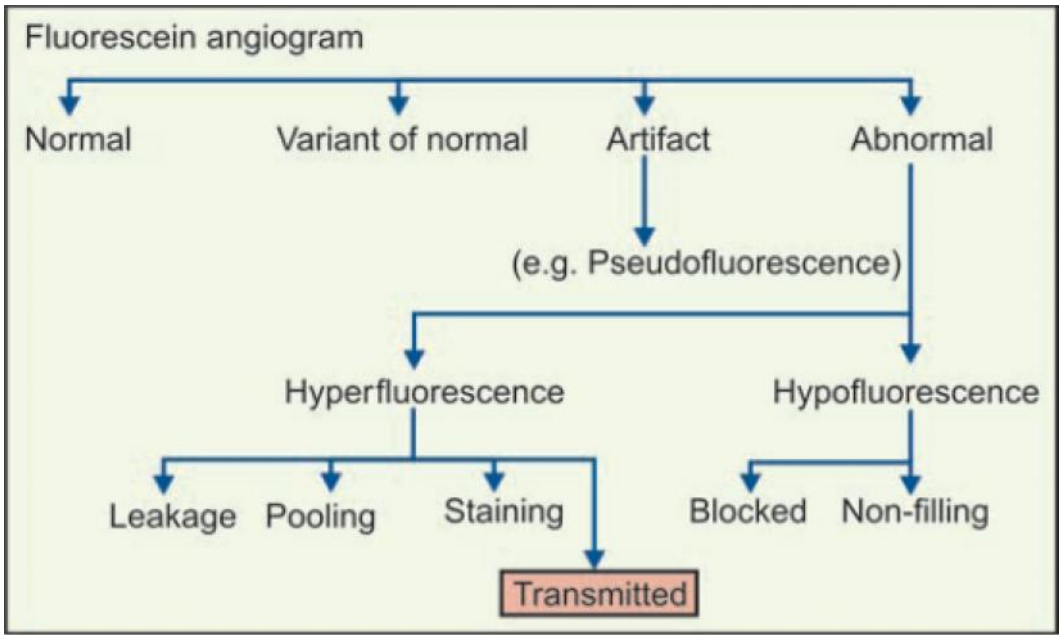


Figure 12-Interpretation of FFA

ABNORMAL FLUORESCEIN ANGIOGRAPHIC PATTERNS [34,35,36]

Abnormalities seen with FFA can be grouped into 3 categories, associated with one of the following:

1. Autofluorescence
2. Hypofluorescence
3. Hyperfluorescence

1. **Autofluorescence** : is fluorescence that can be seen before the fluorescein dye is injected. This is caused by naturally highly reflective substances such as optic disc drusen.

2. **Hypofluorescence** occurs when normal fluorescence is reduced or absent; it is present in 2 major patterns:

A. Vascular filling defect

B. Blocked fluorescence

A. Vascular filling defects occur where the retinal or choroidal vessels do not fill properly as in nonperfusion of an artery, vein, or capillary in the retina or choroid. These defects produce either a delay or a complete absence in filling of the involved vessels.

B. Blocked fluorescence occurs when the stimulation or visualization of the fluorescein is blocked by fibrous tissue or other barriers such as pigment or blood, producing an absence of normal retinal or choroidal fluorescence in the area.

Blocked fluorescence is most easily differentiated from hypo fluorescence due to hypoperfusion by evaluating the ophthalmoscope view, where a lesion is usually visible that corresponds to the area of blocked fluorescence. If no corresponding area is visible clinically, then it is likely an area of vascular filling defect and not blocked fluorescence. By evaluating the level of the blocked fluorescence in relation to the retinal circulation one can determine how deep the lesion resides.

1) **Hyperfluorescence** occurs when there is an excess of normal fluorescence.It is seen in following major patterns:

A. Leakage

B. Staining

C. Pooling

D. Transmission, or window defect

A. Leakage: It is the gradual, marked increase in fluorescence throughout the angiogram when fluorescein molecules seep through the pigment epithelium into the subretinal space or neurosensory retina, out of retinal blood vessels into the retinal interstitium, or from retinal neovascularization into the vitreous. The borders of hyperfluorescence become increasingly blurred, and the greatest intensity of hyperfluorescence is found in the late phases of the study, when the only significant fluorescein dye remaining in the eye is extravascular . Leakage occurs in CNV, in microaneurysms in telangiectatic capillaries, in diabetic macular edema, and in neovascularization of the disc.

B. Staining refers to a pattern of hyperfluorescence where the fluorescence gradually increases in intensity through transit views and persists in late views, but its borders remain fixed throughout the angiogram. Staining results from

fluorescein entry into a solid tissue or similar material that retains the fluorescein, such as a scar, drusen, optic nerve tissue or sclera .

C. **Pooling** refers to the accumulation of fluorescein in a fluid-filled space in the retina or choroid. At the beginning of the angiogram, the fluid in the space contains no fluorescein and is not visible.Pooling is seen in RPE detachment in central serous chorioretinopathy.

D. **A transmission defect or window defect** refers to a view of the normal choroidal fluorescence through a defect in the pigment or loss of pigment in the RPE. In a transmission defect, the hyperfluorescence occurs early, corresponding to filling of the choroidal circulation and reaches its greatest intensity with the peak of choroidal filling. The fluorescence does not increase in size or shape and usually fades in the late phases of the angiogram, as the choroidal fluorescence becomes diluted by blood that does not contain fluorescein. The fluorescein remains in the choroid and does not enter the retina.

3. HYPOFLUORESCENCE

This is decreased fluorescence in comparison to the normal expected fluorescence.

This occurs by 2 mechanisms :

A. Obstruction to the visualisation of normal intravascular fluorescein by haemorrhage, pigment accumulation abnormal tissue proliferation or deposition of abnormal material

B. Decrease in vascularity of the retina or choroid or both with consequent reduction in the amount of fluorescein present as in retinal vascular occlusion or choriocapillary atrophy.

MACULAR DISORDERS: [37,38,39]

AGE RELATED MACULAR DEGENERATION [37]

Age-related macular degeneration (ARMD) is the most common cause of irreversible visual impairment in older populations in industrialized nations. ARMD is a late-onset deterioration of photoreceptors and retinal pigment epithelium in the central retina caused by various environmental and genetic factors. ARMD is the most common cause of visual impairment and blindness in the elderly in industrialized nations. In 2000, more than nine million individuals were estimated to have ARMD in the United States . Its prevalence is predicted to double by 2020.

AMD is classified into two main forms:

1. Non-neovascular (also known as “dry” or “nonexudative”) or
2. Neovascular (also known as “wet” or “exudative”).

1. Non-neovascular (Dry ARMD): [40,41]

- The clinical hallmark of non-neovascular AMD is drusen, which are yellowish deposits at the level of the retinal pigment epithelium (RPE) which lies just under the neurosensory retina.
- Numerous intermediate-large soft drusen, focal hyper and/or hypopigmentation of RPE

- Focal RPE hyperpigmentation and atrophy can also be seen. "Geographic atrophy" is the advanced stage of non-neovascular AMD, where areas of atrophy become confluent and cause visual loss.

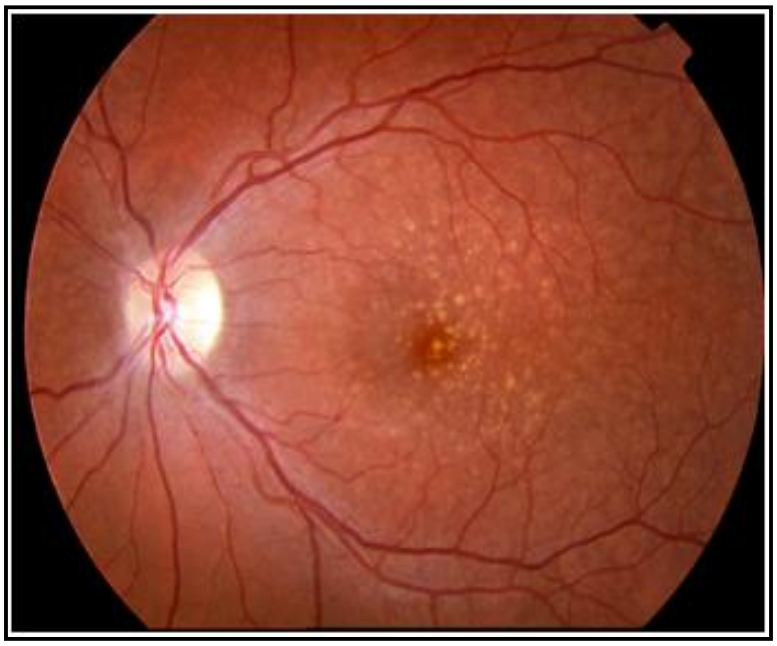


Figure no.13-Geographic Atrophy

GEOGRAPHIC ATROPHY

2. **NEOVASCULAR AMD:** [42, 43 ,44] Is also an advanced manifestation of ARMD, characterized by **choroidal neovascularization (CNV)—**abnormal blood vessels typically arising in the choriocapillaris and often invading the subretinal space.

- CNV membranes that remain sub RPE are termed type1 and when growth extends subretinally are known as type2

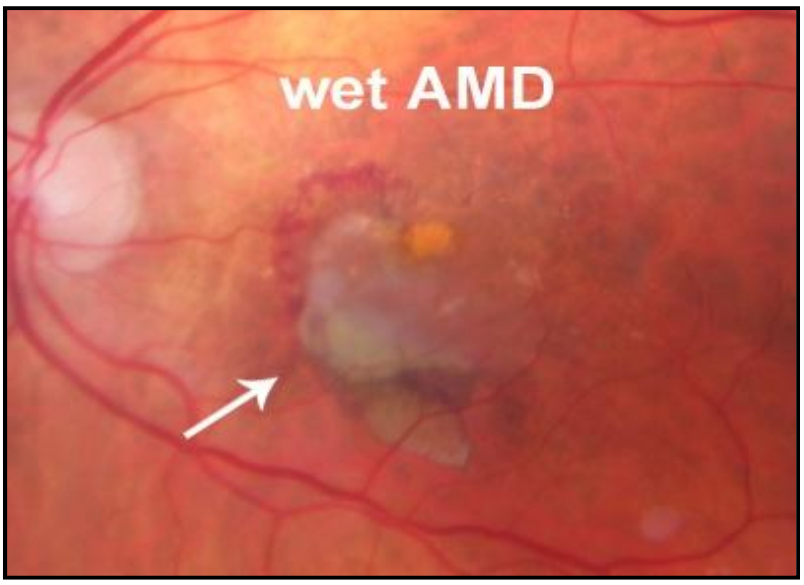


Figure 14-Wet ARMD

COMPLICATIONS OF CNV: [45, 46, 47]

1. Localised sub retinal fluid
2. Intra and subretinal lipid deposition
3. Subretinal, Preretinal and vitreous haemorrhage associated with PED
4. Disciform scar
5. Exudative Retinal detachment

A retrospective study carried out by Aditya S et al on retrospective hospital‐based analysis of age‐related macular degeneration patterns in India concluded that Occult CNVM was the most common form of wet AMD with more asymmetrical presentation at baseline.[20]

A study conducted by Olsen TW et al on fluorescein angiographic lesion in neovascular age-related macular degeneration concluded that most angiographic lesions of patients who undergo FFA for ARMD are subfoveal and occult. [21]

FFA IN ARMD [48,49,50,51]

Classic CNV : Typical Classic CNV is characterized by well-demarcated areas of intense hyperfluorescence appearing early and showing progressive leakage. The fluorescein tends to be most intense at the perimeter of the CNV; the center may show hypofluorescence. Leakage progresses into the late phase of the angiogram and usually obscures the boundaries of the CNV

Occult CNV: The appearance of occult CNV varies widely and can be difficult to identify. The two subtypes of the occult category: fibrovascular pigment epithelial detachment (FV-PED) and late leakage of undetermined source (LLUS).

FV-PED is identified by ill-defined areas of irregular elevation of the staining/leaking RPE with "stipples" of hyperfluorescence often intermixed on the surface. Good stereoscopic effect is essential to determine the presence of FV-PED. The lesion size is usually measured in early-mid phase.

LLUS is a rare type of occult CNV in which poorly demarcated areas of leakage appear at the level of the RPE in the late phase of the angiogram. There are no abnormalities discernible in either the early or mid phase that account for the leakage. Lesion size and leakage area are the same and are both measured from the late phase.

A third type of occult CNV, called "occult other" is not identified as part of the MPS protocol and includes CNV that has an appearance similar to that of FV-PED, but without elevation.

- Most angiographic lesions of patients who undergo FFA for neovascular ARMD are subfoveal and occult. In which 20% of subfoveal lesions are predominantly classic. Approximately half of the juxtafoveal and extrafoveal lesions are predominantly classic. Nearly 30% of all neovascular ARMD lesions have both small occult lesions and a visual acuity less than 20/50.

FFA IN DRY ARMD [29,52,]

- On FA, they appear hyperfluorescent transmission defects due to overlying RPE thinning. On occasion there may be a myriad of small drusen, termed cuticular or basal laminar drusen, which appear as a "starry sky" on FA .
- Soft drusen are larger (>63 μm) with poorly defined borders and they tend to coalesce and become confluent. They are hyperfluorescent with phospholipid accumulation and in younger patients.
- The confluence of soft drusen can produce a drusenoid pigment epithelial detachment (PED), which shows hyperfluorescence and dye pooling without leakage beyond its margin with typical areas of focal hyperpigmentation. Focal hyperpigmentation is a risk factor for the development of choroidal neovascularization (CNV) and angiographically appears as a blocked fluorescence .

A study conducted by Gatut Suhendro et al on fundal fluorescein angiographic lesion in ARMD found that the frequency of ARMD was 4.5% in retinal consultation patient. This study concluded that 47.3% wet AMD and 52.7% dry AMD. Of the wet AMD 5.4% was classic CNV, 17.57% predominantly classic CNV, 17.57% minimally classic CNV and 6.76% occult CNV. [53]

A study conducted bt Nils F. Mokwa et al on Grading of Age-Related Macular Degeneration Comparison between Color Fundus Photography, Fluorescein Angiography and Spectral Domain Optical Coherence Tomography showed that SDOCT is highly sensitive for the detection of AMD, CNV, and CNV activity; however it cannot fully replace FA.[54]

A study conducted by Arvind R et al on Role of Fluorescein angiography in evaluation of posterior segment disorders analyzed 15 cases of ARMD FFA confirmed the diagnosis in 33% of cases, altered the diagnosis in 20 % of cases and categorized the lesion in 47% of cases. [55]

A study conducted by Nandini et al on role of fundus fluorescein angiography in macular disorders found that FFA confirmed diagnosis in 66.66% cases of ARMD and altered diagnosis in 33.3% cases of ARMD and played an important tool in diagnosing wet AMD.[56]

DIABETIC RETINOPATHY [35,57,58]

Category	*Description*
Non-proliferative diabetic retinopathy (NPDR)	
No DR	
Very mild	Microaneurysms only
Mild	Any or all of: microaneurysms, retinal haemorrhages, exudates, cotton wool spots, up to the level of moderate NPDR. No IRMA or significant beading
Moderate	Severe retinal haemorrhages (more than ETDRS standard photograph 2A: about 20 medium-large per quadrant) in 1–3 quadrants *or* mild intraretinal microvascular abnormalities (IRMA) Significant venous beading can be present in no more than 1 quadrant
Severe	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
Proliferative diabetic retinopathy (PDR)	
Mild-moderate	New vessels on the disc (NVD) or new vessels elsewhere (NVE), but extent insufficient to meet the high-risk criteria
High-risk	New vessels on the disc (NVD) greater than ETDRS standard photograph 10A (about 1/3 disc area) Any NVD with vitreous or preretinal haemorrhage NVE greater than ½ disc area with vitreous or preretinal haemorrhage (or haemorrhage with presumed obscured NVD/E)

DIABETIC MACULOPATHY [59, 60]

Diabetic Maculopathy is the most common cause of visual impairment in diabetic patients, particularly type 2. Diffuse retinal oedema is caused by extensive capillary leakage, and localized oedema by focal leakage from microaneurysms and dilated capillary segments. The fluid is initially located between the outer plexiform and inner nuclear layers; later it may also involve the inner plexiform and nerve fibre layers, until eventually the entire thickness of the retina becomes oedematous. With

further accumulation of fluid the fovea assumes a cystoid appearance leading to cystoid macular oedema.

FOCAL MACULOPATHY

1. **Signs**. Well-circumscribed retinal thickening associated with complete or incomplete rings of exudates .
2. **FA** shows late, focal hyperfluorescence due to leakage, and good macular perfusion

DIFFUSE MACULOPATHY

1. **Signs**. Diffuse retinal thickening, which may be associated with cystoid changes. Landmarks are obliterated by severe oedema which may render localization of the fovea impossible .
2. **FA** shows late diffuse hyperfluorescence which may assume a central flower-petal pattern if CMO is present.

ISCHAEMIC MACULOPATHY

1. **Signs** are variable and the macula may look relatively normal despite reduced visual acuity. In other cases PPDR may be present .
2. **FA** shows capillary non-perfusion at the fovea (an enlarged FAZ) and frequently other areas of capillary non-perfusion at the posterior pole and periphery .

CLINICALLY SIGNIFICANT MACULAR OEDEMA(CSME)

Clinically significant macular oedema (CSME) was defined in the ETDRS :

- 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 (which 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 .

A study conducted by Qamar Mehaboob on diagnosis of diabetic macular edema (DME) based on fundus fluorescein angiography findings classified the diabetic macular edema into diffuse type, focal type and ischaemic type [61]

A study conducted by Wykes et al on retinal diseases concluded that Fluorescein angiography was useful in differentiating between cystoid macular oedema and ischaemic maculopathy, and therefore whether laser therapy was indicated. [62]

A study conducted by Mihaela et al on the role of fluorescein angiography in diagnosis and treatment of diabetic retinopathy concluded that the fluorescein angiography is an essential examination for the proper monitoring of the diabetic patients and particularly useful in the diagnosis and classification of diabetic macular edema, with the different therapeutic approach depending on the type of macular edema. [63]

Study conducted by Sarfaraz et al on the Incidence of Angiographic Patterns of Diabetic Maculopathy concluded that the treatment modalities of three types of

diabetic maculopathy are different. For proper diagnosis and management only clinical examination can not be relied upon. FFA must be performed before deciding on the appropriate mode of treatment .[64]

A study conducted by Arthi Rasquinha et al on correlation of Angiographic Findings to the Clinical Maculopathy concluded that the FFA is useful in the early detection and treatment of subclinical maculopathy. Guidelines can be framed based on the FFA findings for the early treatment of subclinical Maculopathy. [65]

CENTRAL SEROUS RETINOPATHY [66, 67]

Central serous chorioretinopathy (CSCR) is an idiopathic disorder characterized by a localized serous detachment of the sensory retina at the macula secondary to leakage from the choriocapillaris through focal , or less commonly diffuse, hyperpermeable RPE defects. Imperfectly defined additional risk factors include psychological stress, type A personality, steroid administration, Cushing syndrome, systemic lupus erythematosus and pregnancy.

A study conducted by Liew G et al on Central serous chorioretinopathy showed that the main risk factors for CSCR are systemic corticosteroid use, type A personality, pregnancy and endogenous Cushing's syndrome. The pathophysiology of CSCR remains obscure, although disorders in both the choroidal circulation and retinal pigment epithelium are implicated.[68]

FFA IN CSR:

Fundus fluorescein angiography (FA) of CSCR typically shows a focal area of leakage of the dye from the RPE into the subretinal space with two main patterns:

1. **Ink blot**
2. **Smoke-stack.**

Ink-blot leakage pattern appeared more frequently than smoke-stack pattern on Fundus fluorescein angiography (FFA) in patients of CSCR. Single leak was seen in most of the cases and peri- foveal area was the site of predilection of leakage in majority of cases.

A study conducted by Alicia CSW on Angiographic Characteristics of Acute Central Serous Chorioretinopathy in an Asian Population on 128 patients concluded that the inkblot pattern of leakage was the most common pattern seen on angiography. There were a significant number of cases with bilateral and multifocal involvement, exceeding those reported in non-Asian populations.[69]

A study conducted by Shahin M M et al on Angiographic characteristics of central serous chorioretinopathy in an Egyptian population found that CSCR is seen at younger age with higher male to female ratio and more frequent smokestack leaks than other populations.[70]

A study conducted by Shahid J et al on Pattern of Central Serous Chorioretinopathy (CSCR) on Fundus Fluorescein Angiography showed that the ink-blot appearance is more frequent than smoke-stack appearance. [71]

MACULAR HOLE [29, 72]

Idiopathic age-related macular hole is a relatively common cause of central visual loss, with a prevalence of approximately 3 : 1000 individual.The peak incidence of onset in females in the 7th decade. Presentation may be with impairment of central vision in one eye, or as a relatively asymptomatic deterioration, first noticed when the fellow eye is closed or at a routine sight test. The risk of involvement of the fellow eye at 5 years is around 10%

STAGES

Stage 1a: 'Impending' macular hole

Flattening of the foveal depression with an underlying yellow spot

Stage 1b: Occult macular hole

A yellow ring that may be associated with metamorphopsia or a mild decrease in visual acuity.

Stage 2: Small full-thickness hole

Full-thickness hole less than 400 µm in diameter. The defect may be central, slightly eccentric or crescent-shaped

Stage 3: Full-size macular hole

Full-thickness hole greater than 400 µm in diameter with a red base in which yellow-white dots may be seen. A surrounding grey cuff of subretinal fluid is usually present, and an overlying operculum may be visible.

Stage 4: Full-size macular hole with complete PVD

FFA CHARACTERISTICS OF MACULAR HOLE [29, 73]

In stage I : Faint hyper Fluorescence or more typically no abnomlality at all is seen on fluoresce in angiography

In stage 2 : Fluorescein angiography may reveal a round area of window defect or may remain normal.

Stage 3 and 4 holes typically produce a window defect with early transmission of fluorescence in phase with choroidal filling through the central retinal defect. No late leakage or accumulation of dye is seen.

FFA IN RETINAL VEIN OCCLUSIONS [74, 75]

- Arteriolar filling is usually normal but venous filling in the affected vessel is usually delayed in the acute phase. Hypofluorescence caused by hemorrhage and capillary nonperfusion are common findings and dilated, tortuous veins are seen . The retinal vessels, particularly the vein walls, may stain with fluorescein, especially at the site of the occlusion . The very important distinction should be made between neovascular fronds, which may show profuse leakage of dye vs. collateral vessels, which do not leak fluorescein.
- Cystoid macular edema appears in the late stage of the angoigram shows typically petaloid pattern and may involve the entire fovea or just several clock hours, depending on the distribution of the obstruction.

FFA IN HEREDITARY MACULAR DYSTROPHIES [29, 76]

1. **STARGARDTS:** On fluorescein angiography decreased choroidal fluorescence. dark "silent" choroid in at least 80% of cases due to masking of choroidal fluorescence by accumulation of lipofuscin in RPE
2. **Best Vitelliform Dystrophy:** FFA shows variable blockage, staining, window defects depending on stage
3. **Pattern Dystrophy:** FFA shows hypofluorescence from blocking by lipofuscin/pigment window defects from atrophy
4. **Adult-onset Foveomacular Vitelliform Dystrophy:** FFA shows hypofluorescence in the lesion and ring of hyperfluorescence surrounding it.

METHODOLOGY

The present study was conducted in the Department of Ophthalmology, KLES Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi on patients presenting to the Ophthalmology OPD .

Study design

One year cross sectional study

Study period

The present study was conducted from 1st January 2016 to 31st December 2016

METHOD OF COLLECTION OF DATA:

Source of Data

All the patients attending the Ophthalmology OPD and presenting with macular diseases at KLES Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Sample size

Sample size for the study is calculated by following formula:

$$N = \frac{(Za)^2[p \times q]}{d^2}$$

P=true positivity rate of FFA in detection of macular diseases=80%, so p=80

q=100-p=20

Taking 10% as error

d=10

If the values are normally distributed, then 95% of the values will fall within 2 standard errors of the mean. The value of z corresponding to this is 1.96 (from the standard normal variate tables)

$$N = \frac{(1.96)^2(80\times20)}{10^2}$$

$$N = \frac{4[80\times20]}{100}$$

N=64

Sampling method: Purposive sampling

Total 66 patients were included in our study

SELECTIN CRITERIA:

Inclusion criteria:

All patients with retinal disorders with suspected macular pathologies were included in the study.

EXCLUSION CRITERIA:

- Pregnant women.
- Immunocompromised patients.
- Those with hypersensitivity to fluorescein dye.
- Patients with renal insufficiency, cardiovascular diseases.
- Patients whose ocular fundus can not be made out due to very hazy media.

The ethical clearance for our study was given by the ethical review committee of the institute

PROCEDURE:

After finding the suitability as per the inclusion and exclusion criteria patients were selected for the study and briefed about the nature of the study and written informed consent was obtained.

Patient history and general examination

- A thorough and detailed history was taken from the selected patients. Complete ophthalmic and medical history regarding the duration and symptoms of the disease was noted with special emphasis on history of risk factors and associated any systemic illness if present.

Ocular examination

- A thorough and detailed ocular examination of the anterior segment was performed on the slit – lamp biomicroscopy.

Vision and refraction

- Visual acuity was recorded and best corrected visual acuity was done for both eyes .

Intra-ocular pressure

- IOP was measured using a Schiotz or Goldmann's applanation tonometer.

Ophthalmoscopy

- A thorough careful and detailed examination of the fundus was done firstly by a direct ophthalmoscope and subsequently with an indirect ophthalmoscope giving special attention to macula
- Slit-lamp biomicroscopy with +90D lens was performed to look for pathologies in the posterior pole and documented.

Fluorescein angiography

Consent

- Patients were explained in detail in their own vernacular language about the condition of the eye. The procedure and purpose of fundus fluorescein angiography and possible side effects.
- Informed consent was taken.

Investigations

The following investigations were performed :

- Complete haemogram
- Urine - albumin, sugar, microscopy
- Random blood sugar
- Glycosylated Hb, if required
- ECG, if required
- Renal function tests - Blood urea and Serum creatinine
- Lipid profile
- Evaluation by a physician was done for all cases to note the presence of any systemic diseases and fitness for the above procedure was taken.
- All cases were examined by an anaesthetist and his presence was compulsory during the procedure.

  Emergency medications were kept ready for use if any side effects occurs

Procedure of FFA

- Patient's pupils were dilated with a combination of 5% phenylephrine and 1% tropicamide 20-30 minutes prior to the procedure.
- An intravenous or intradermal test dose of the dye was given 10 minutes prior to the procedure.
- A 21 gauge scalp vein set was put in the anticubital vein.
- Patient was seated comfortabaly in front of the fundus camera and the dye was injected.
- Patient's pulse and general condition were monitored all throughout the procedure and any adverse reaction was noted.
- Serial photographs were taken through the desired phases of FFA

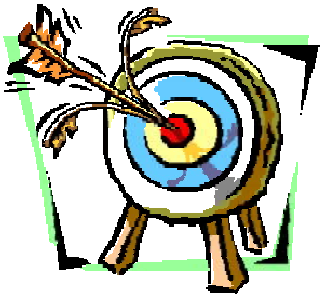
After the procedure

- Patient was made to lie down and rest for 30 minutes.
- Patient was explained about the side effects of dye that is change in colour of urine and skin.
- The findings were recorded in fundus drawings in the patient's case-sheet and analysed.

Advise

- The patient was advised about the diagnosis and further plan of treatment

Introduction



Objectives

Review of Literature

Methodology

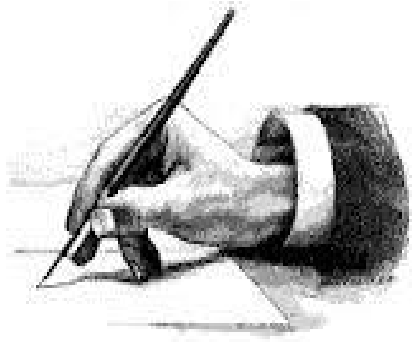
Results

Discussion

Conclusion

Summary

Bibliography



Annexure-I

Annexure-II

Annexure-III

Annexure-IV

Annexure-V

RESULTS

The present study was conducted at Department of Ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi during the study period of 1 year from 1st January 2016 to 31st December 2016.

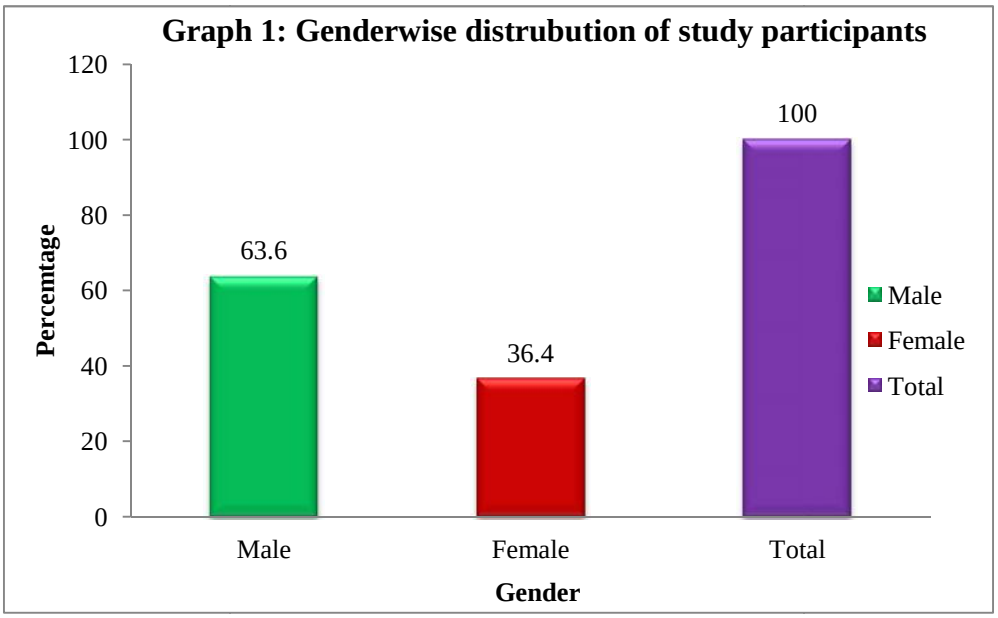
Total 66 participants were included in our study. These participants with macular diseases were evaluated in detail by using clinical ophthalmoscopy and FFA.

Table 1 and Graph1 represent the demographic profile of study participants. In present study majority of the participants were males(63.6%).The mean age of the study population was 58.95 $\pm$ 19.70 years.

1. DEMOGRAPHIC DATA

Table 1: Gender-wise distribution and Mean age of Study participants

Sex	Number of Participants in the study (N=66)	Percentage (%)	Mean Age
Male	42	63.6	58.95±19.70
Female	24	36.4	
Total	**66**	**100**	



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2. DISTRIBUTION OF MACULAR DISEASES

Table 2: Distribution of Macular diseases by FFA diagnosis

Diagnosis	Number of Participants (n=66)	Prevalence (%)
AMD	20	30.30
Diabetic Maculopathy	21	31.81
Vascular Occlusions	08	12.12
CSCR	10	15.15
Macular Dystrophy	02	03.03
Inflammatory causes	03	04.54
Macular hole	02	03.03

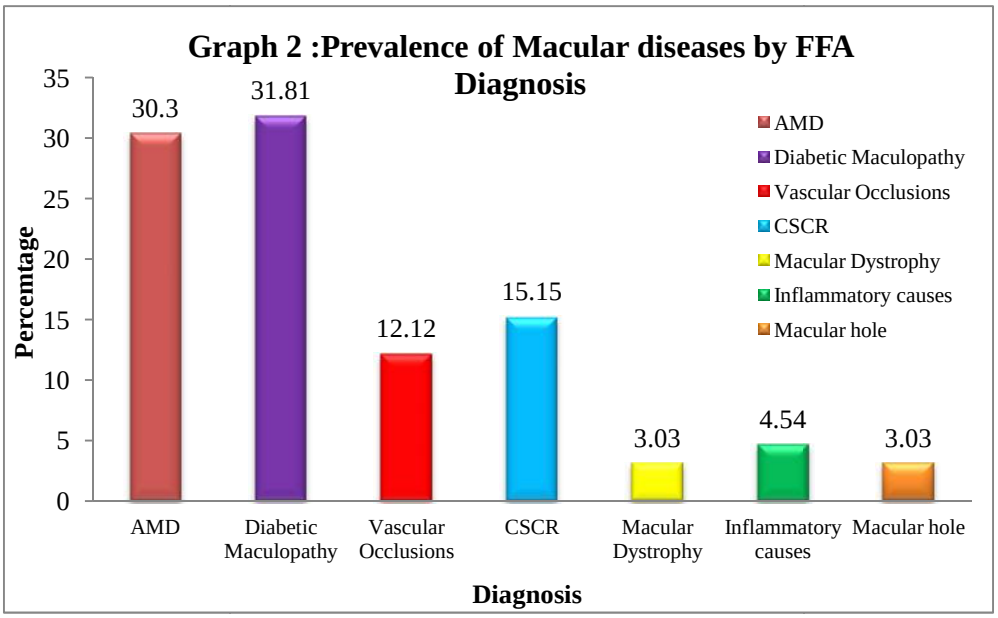

Table 2 and graph 2 represents the distribution of macular diseases. Majority of the participants were having ARMD (30.30%) and Diabetic Maculopathy (31.81%) where as 15.15% participants were having CSCR and 12.12% were having vascular occlusions. The least participants were having inflammatory causes followed by macular dystrophy and macular hole.

3. AGE RELATED MACULAR DEGENERATION

Table 3: Distribution of ARMD

Diagnosis	Number of Participants (n=66)	Prevalence (%)
ARMD(Total)	20	30.30
Types of ARMD		
Dry ARMD	10	50%
Wet ARMD	10	50%

Graph 3: Distribution of (Dry and Wet) ARMD by FFA

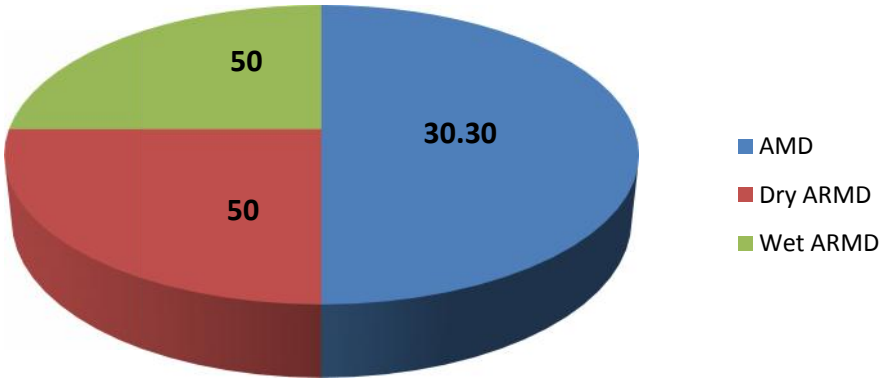

Table 3 and Graph 3 represents the distribution of ARMD. In present study 30.30% patients were diagnosed as ARMD. Out of which 50% were of Dry ARMD and 50% were of Wet ARMD.

4. WET ARMD

Table 4: Distribution of Wet ARMD by FFA

Disease	FFA Confirmed diagnosis	FFA Altered diagnosis
Wet ARMD(Total)	2	8
Types of Wet ARMD		
Classic CNVM	1	2
Occult CNVM	1	6

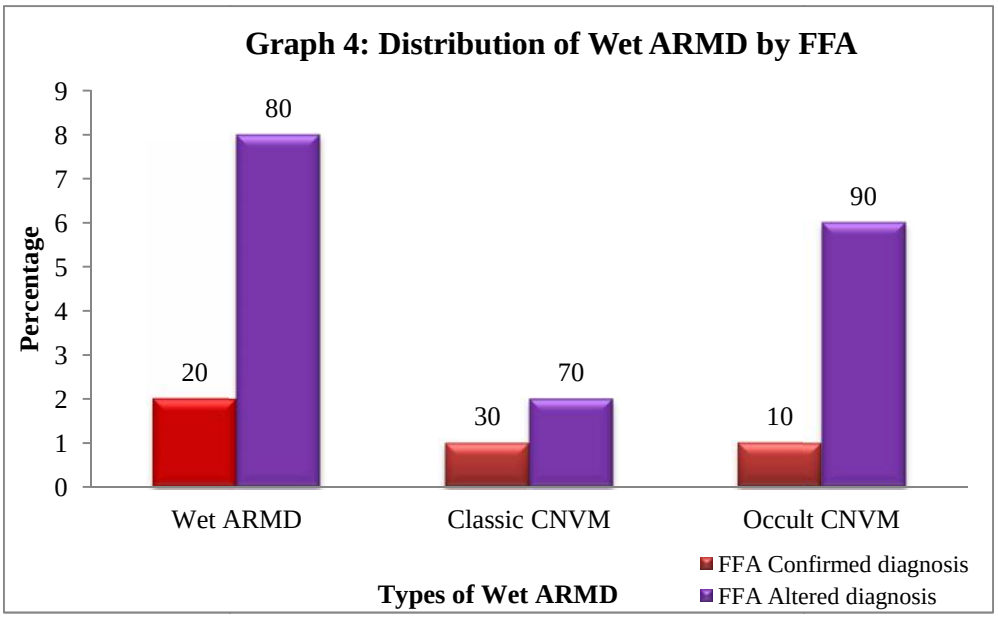

Table 4 and Graph 4 represents the distribution of Wet ARMD by FFA. Majority (80%) of Wet ARMD cases were altered in diagnosis by FFA, where as FFA confirmed the diagnosis in 20% cases which were diagnosed by ophthalmoscopy.

5. ROLE OF FFA IN ARMD

Table 5: Role of FFA in ARMD

Disease	Number	Percentage (%)
ARMD (Total)	20	30.30
Diagnosis by FFA		
FFA Confirmed	12	60%
FFA Altered	08	40%
FFA Classified the lesion in (out of 20)	10	50%

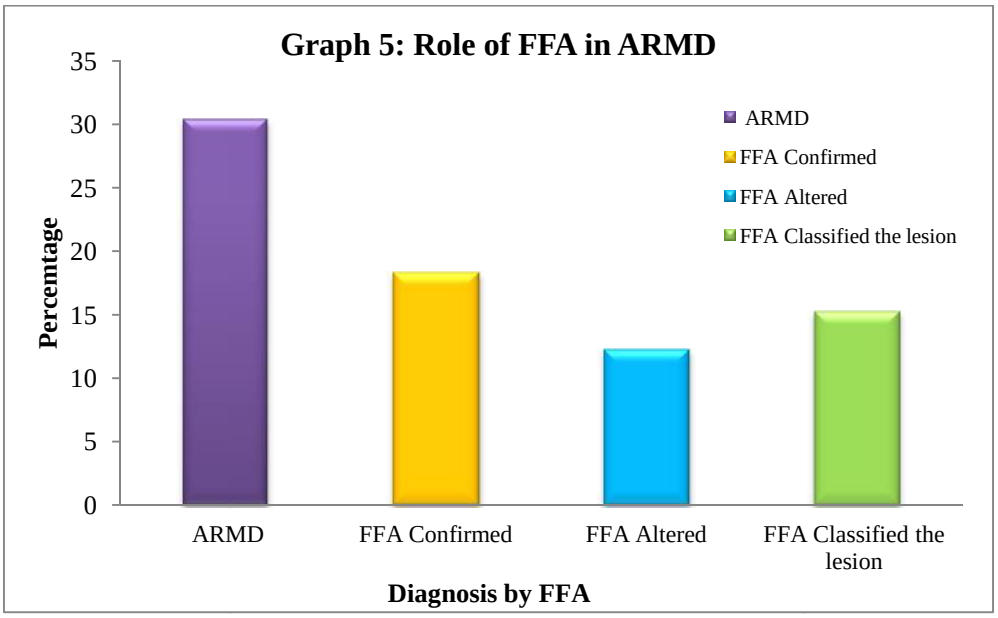

Table 5 and Graph 5 reprents the Role of FFA in ARMD. On analysis of 20 cases of ARMD, FFA confirmed the diagnosis in 60% of cases and altered the diagnosis in 40% of cases. FFA classified the lesions in 50% of cases of ARMD.

Thus FFA played a major role in the diagnosis and classification of ARMD cases which could not be made by clinical ophthalmoscopy alone.

6. DIABETIC MACULOPATHY

Table 6: Distribution of diabetic Maculopathy

Disease	Number of patient	Prevalence (%)
Diabetic Maculopathy(Total)	21	31.81
Types of Diabetic Maculopathy		
CSME	07	33.33
Focal Maculopathy	06	28.57
Diffuse Maculopathy	05	23.80
Ischemic Maculopathy	03	14.28

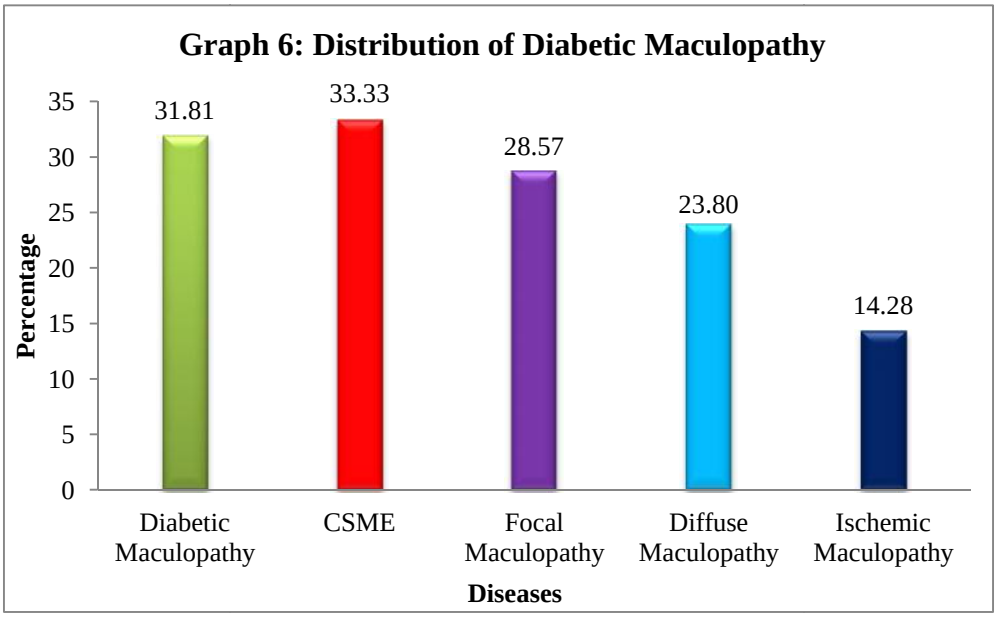

Table 6 and Graph 6 represents the distribution of Diabetic Maculopathy by FFA. Out of 21 cases, majority of participants were having CSME (33.33%) and Focal Diabetic Maculopathy (28.57%) followed by Diffuse Maculopathy seen in 23.80% cases. The least participants (14.28%) were having Ischaemic Maculopathy.

7. ROLE OF FFA IN DIABETIC MACULOPATHY

Table 7 : Role of FFA in Diabetic Maculopathy

Disease	Number of patient	Percentage(%)
Diabetic Maculopathy(Total)	21	31.81
Diagnosis by FFA		
FFA Confirmed diagnosis	07	33.33
FFA altered diagnosis	14	66.66
Classification of lesion by FFA		
OUT OF 21	14	66.66

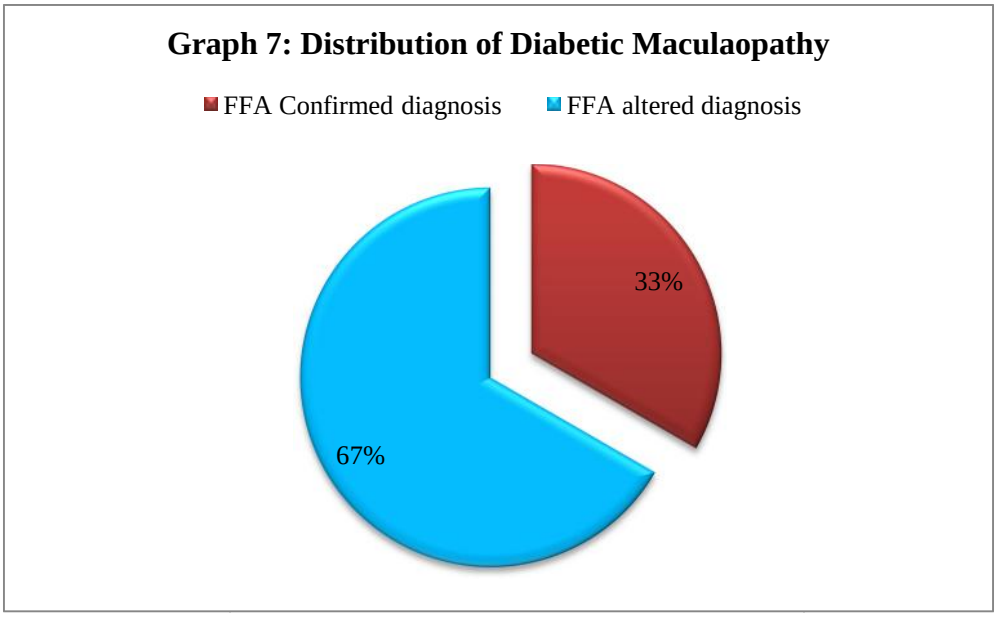

Table 7 and Graph 7 represents the role of FFA in Diabetic Maculopathy. Among 21 Diabetic Maculopathy cases, FFA confirmed the diagnosis only in 33.33% cases and altered the diagnosis in 66.66% cases. In our study Ischaemic Maculopathy cases were only confirmed by FFA. FFA is gold standard in the diagnosis of Ischaemic Maculopathy.

Out of 21 cases, FFA classified the Diabetic Maculopathy into CSME,Focal Maculopathy , Diffuse Maculopathy and Ischaemic maculopathy in 66.66% cases. Thus FFA played major role in confirmation and classification of Diabetic Maculopathy cases.

8. DISTRIBUTION OF VASCULAR OCCLUSIONS

Table 8: Distribution of Vascular Occlusions

Disease	Number of patient	Prevalence(%)
Vascular Occlusions(Total)	08	12.12
Types of Vascular Occlusions		
BRVO	06	75%
CRVO	01	12.5%
Cilio retinal artery occlusion	01	12.5%

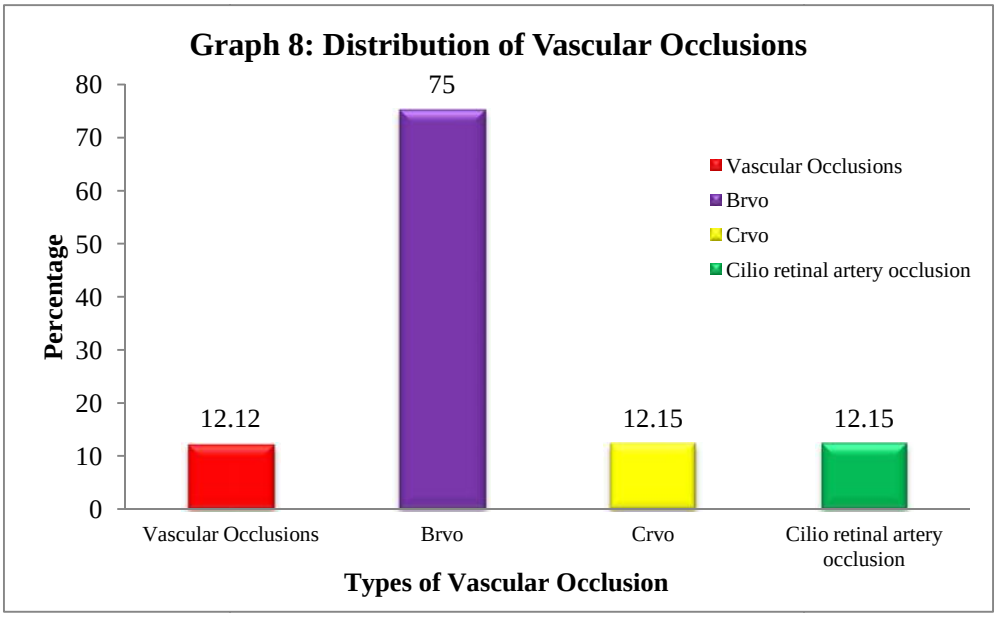

Table 8 and Graph 8 represents the distribution of vascular occlusions. Majority (75%) of the cases were having BRVO.CRVO and cilioretinal occlusion was seen in least cases.

9. DISTRIBUTION OF MACULAR LESIONS IN VASCULAR OCCLUSION BY FFA

Table 9: Distribution of Macular lesions in Vascular Occlusions by FFA

Disease	Number of patient	Prevalence(%)
Vascular Occlusions(Total)	08	12.12
Types of macular lesion		
Macular edema	05	62.50
Macular Ischemia	03	37.50

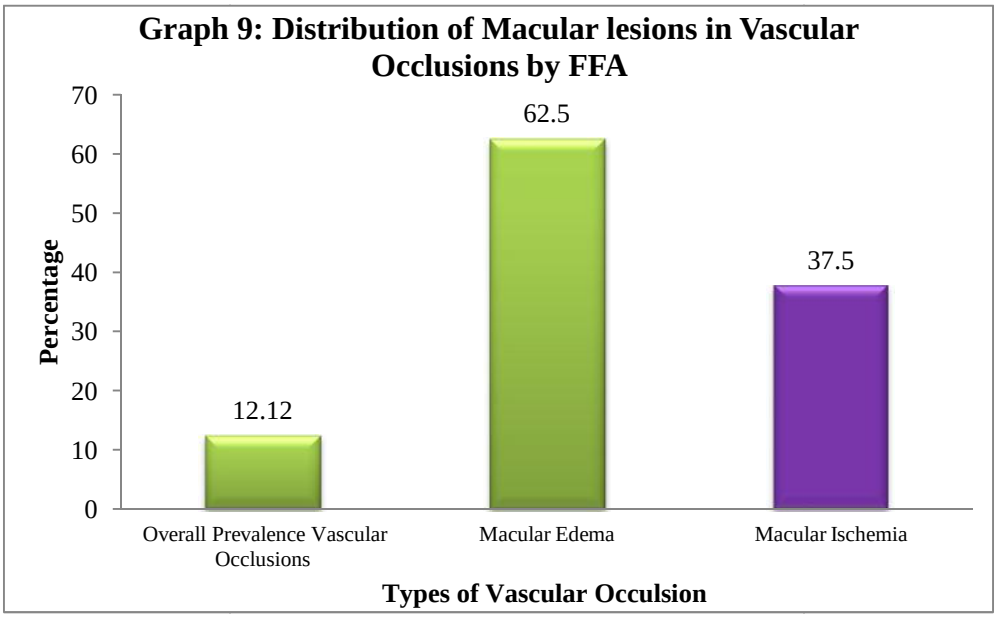

Table 9 and Graph 9 represents the distribution of macular lesions in vascular occlusions by FFA. Majority (62.50%) of the cases were having Macular edema followed by Macular ischaemia seen in less (37.5%) cases.

10. ROLE OF FFA IN VASCULAR OCCLUSIONS

Table 10: Role of FFA in Vascular Occlusion

Disease	Number of patient	Prevalence(%)
Vascular Occlusions(Total)	08	12.12
Diagnosis by FFA		
FFA Confirmed diagnosis	05	62.50
FFA altered diagnosis	03	37.50
Classification by FFA		
Out of 08	08	100

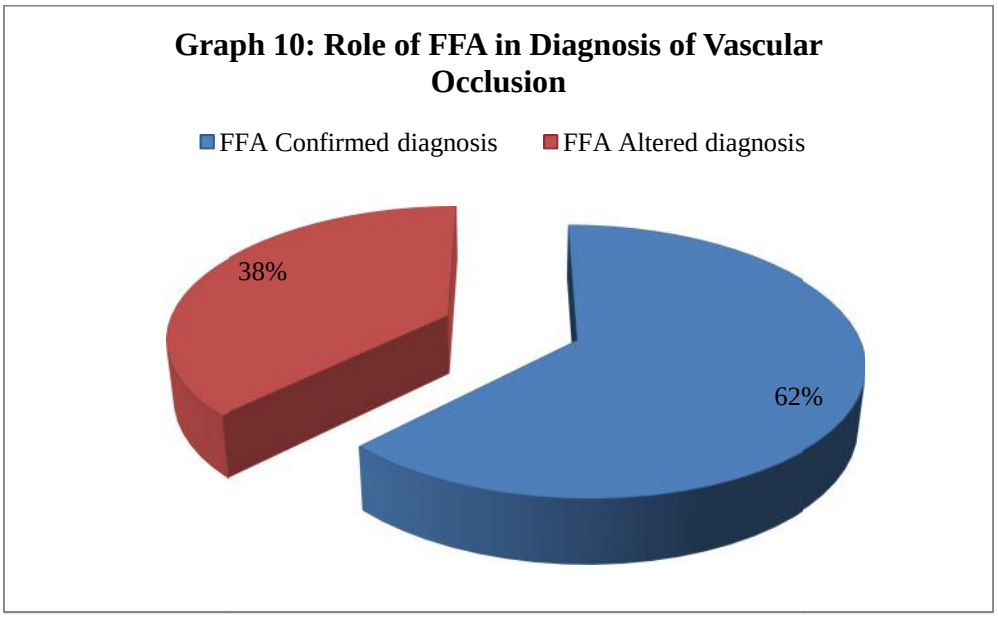

Table 10 and Graph 10 reprents the role of FFA in vascular occlusions. Out of 8 cases, FFA has confirmed the diagnosis in 62.50% cases and altered the diagnosis in 37.50% cases. Macular ischaemia due to vascular occlusion was confirmed only by FFA.

Thus FFA remains a valuable tool in differentiating the macular edema by macular ischemia in vascular occlusions. FFA is a gold standard in the diagnosis of macular ischaemia.

11. DISTRIBUTION OF MACULAR DYSTROPHY

Table 11: Distribution of Macular dystrophy by FFA

Disease	Number of patient	Prevalence(%)
Macular Dystrophy(Total)	02	03.03
Types of Macular Dystrophy		
Stargardt's Dystrophy	01	50
Adult Onset Vitelliform Dystrophy	01	50

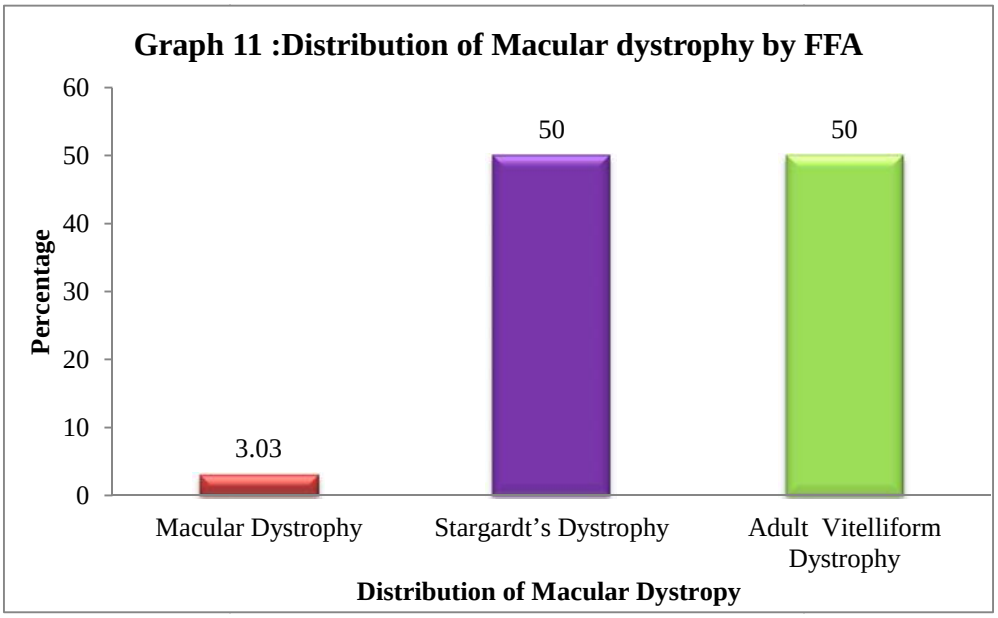

Table 11 and Graph 11 represents the distribution of Macular dystrophy by FFA .Out of 03.03% participants,50% had Stargadt's disease and 50% had Adult Vitelliform dystrophy.

12. ROLE OF FFA IN MACULAR DYSTROPHY

Table 12: Role of FFA in Macular Dystrophy

Disease	Number of patient	Prevalence(%)
Macular Dystrophy	02	03.03
FFA Confirmed diagnosis	02	100
FFA altered diagnosis	00	00

Table 12 and Graph 12 represents the role of FFA in Macular Dystrophy. In present study all the cases of Macular Dystrophy were confirmed by FFA.

13. CENTRAL SEROUS RETINOPATHY (CSCR)

Table 13: Distribution of leakage pattern on FFA in CSCR

Disease	Number of patient	Prevalence (%)
CSCR(Total)	10	15.15
Classification		
Smoke Stack	04	40
Ink Blot	06	60

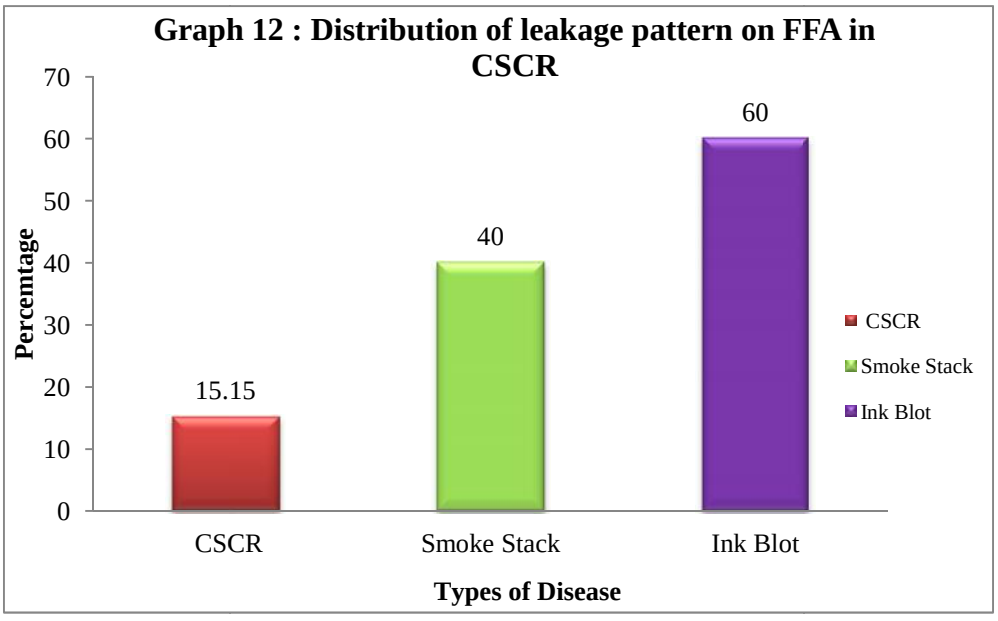

Table 13 and Graph 12 represents the distribution of leakage pattern on FFA. The present study showed that 15.15% participants were diagnosed as CSCR. Out of which 60% were diagnosed as Ink Blot pattern and 40% diagnosed as smoke stack pattern by FFA.

14. ROLE OF FFA IN CSCR

Table 14: Role FFA in CSCR

Disease	Number of patient	Prevalence(%)
FFA Confirmed diagnosis	10	100
FFA altered diagnosis	00	00
Classification of lesion by FFA		
Out of 10	10	100

Table 14 and Graph 14 represents the role of FFA in CSCR. FFA has confirmed the diagnosis in all the cases. FFA classified the CSCR according to pattern of leakage into inkblot type and smoke stack type. Out of 10 cases majority were of ink blot type constituiting 60% of cases and 40% were of smoke stack pattern.

15. ROLE OF FFA IN MACULAR HOLE

Table No 15: Role of FFA in Macular hole

Disease	Number of patient	Prevalence(%)
Macular hole(Total)	02	03.03
FFA Confirmed diagnosis	02	100
FFA altered diagnosis	00	00

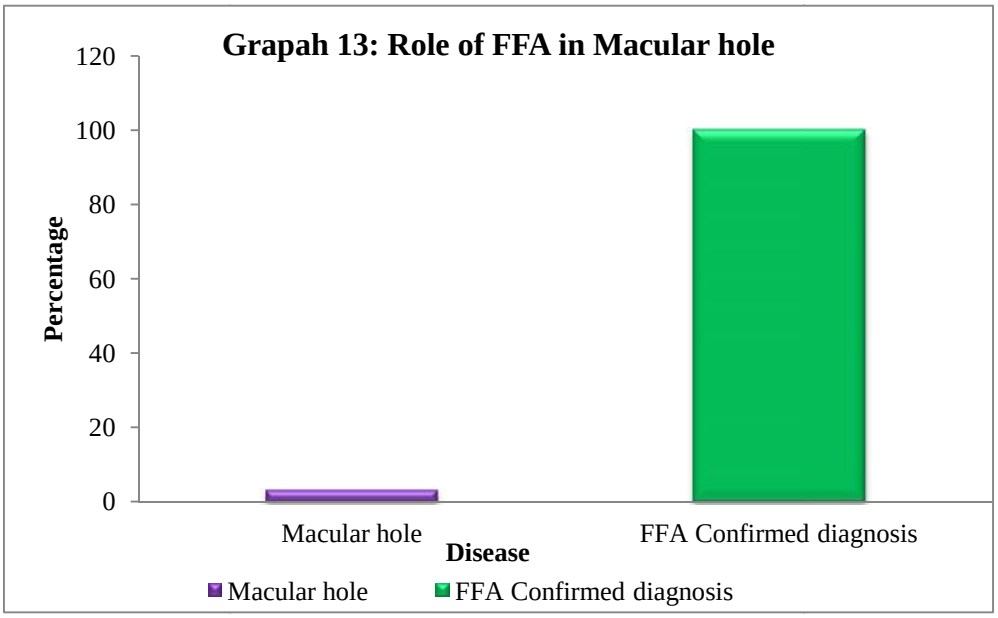

Table 15 and Graph 13 represents the role of FFA in Macular Hole. Out of 2 cases of full thickness macular hole, FFA has confirmed diagnosis in all the cases. FFA can be used as a diagnostic modality in the analysis macular hole cases.

16. INFLAMMATORY CAUSES

Table 16: Distribution of Inflammatory causes

Disease	Number of patient	Prevalence(%)
Inflammatory causes(Total)	03	04.54
Types of Inflammations		
Choroiditis	02	66.66
CMV retinitis	01	33.33

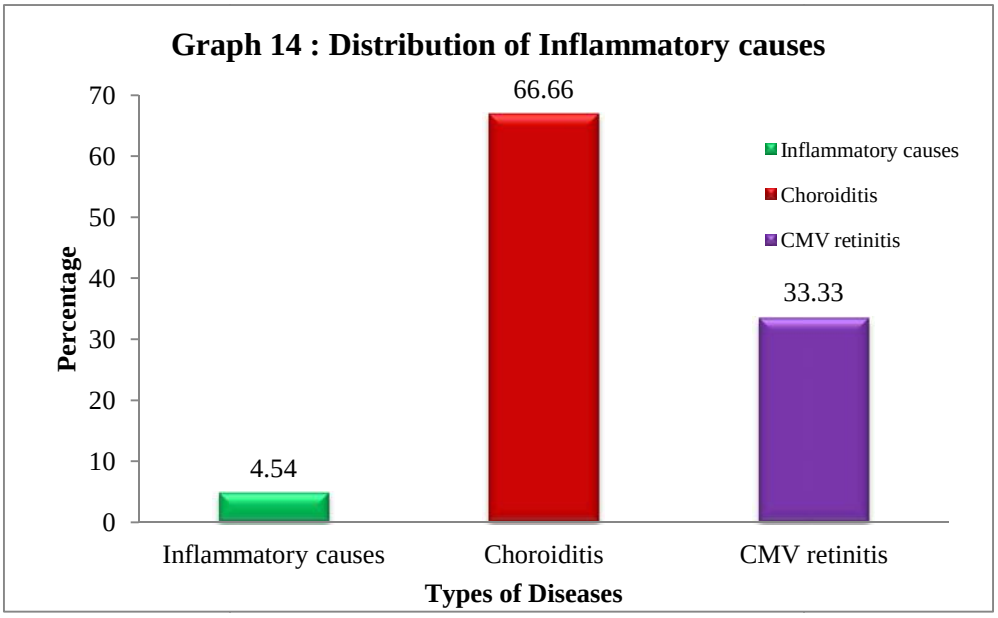

Table 16 and Graph 14 represents the distribution of inflammatory causes. Out of 3 majority (66.66%) of the cases were having choroiditis followed by CMV retinitis seen in least cases.

17. ROLE OF FFA IN INFLAMMATORY CAUSES

Table 17: Role of FFA in Inflammatory causes

Disease	Number of patient	Prevalence(%)
Inflammatory causes(Total)	03	04.54
FFA Confirmed diagnosis	02	66.66
FFA altered diagnosis	01	33.33

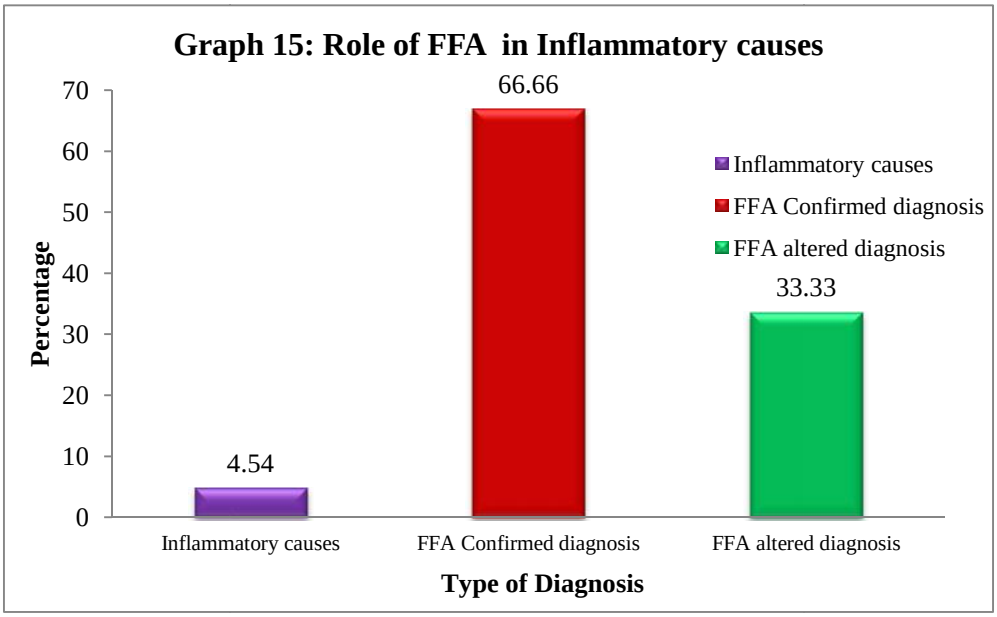

Table 17 and Graph 15 represents the role of FFA in inflammatory causes. Out of 3 cases, FFA confirmed the diagnosis in 66.66% of cases and altered the diagnosis in 33.33% of cases. Macular edema due to vascular occlusions was confirmed only by FFA.

18. ROLE OF FFA IN MACULAR DISEASES

Table 18: Efficacy of FFA diagnosis in relation to Clinical evaluation of Macular Diseases

Role of FFA	Number of patient (n = 66)	Prevalence (%)
FFA Confirmed	41	62.12
FFA Altered	25	37.87
Total	66	100

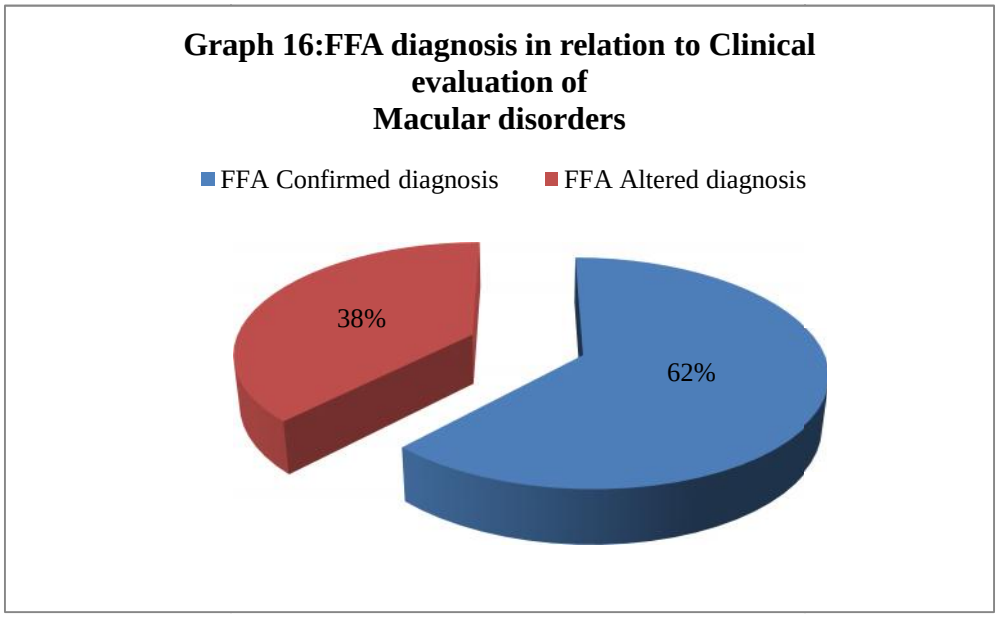

Table 18 and Graph 16 represents the efficacy of FFA in the diagnosis of macular diseases. In our study out of 66 cases of macular diseases , FFA confirmed the diagnosis in 62% of the cases and altered the diagnosis in 38% of cases. According to binomial test P<0.001 which is found to be statistically significant.

Therefore FFA has proved to be a superior diagnostic modality as compared to clinical ophthalmoscopy alone in the diagnosis of macular diseases.

ON ANALYSIS OF 66 CASES:

1) 45 (66%) cases were detected positive by both clinical ophthalmoscopy and FFA
2) 21 (33%) cases were altered in their diagnosis after doing FFA
3) FFA played a major role in the confirmation and classification of the lesions diagnosed by clinical ophthalmoscopy in majority of the cases

The clinical ophthalmoscopy alone has a low negative predictive value compared to FFA. Therefore FFA is a superior diagnostic modality and a diagnostic tool must for all macular diseases for detailed clinical evaluation and planning for further management.

19. ROLE OF FFA IN CLASSIFICATION MACULAR DISEASES

Table 19: Efficacy of FFA diagnosis in relation to classification of macular diseases Macular disorders in patients studied

Role of FFA	Number of patient (n = 66)	Prevalence (%)
FFA Classified	42	63.63
FFA not classified	24	36.36
Total	66	100

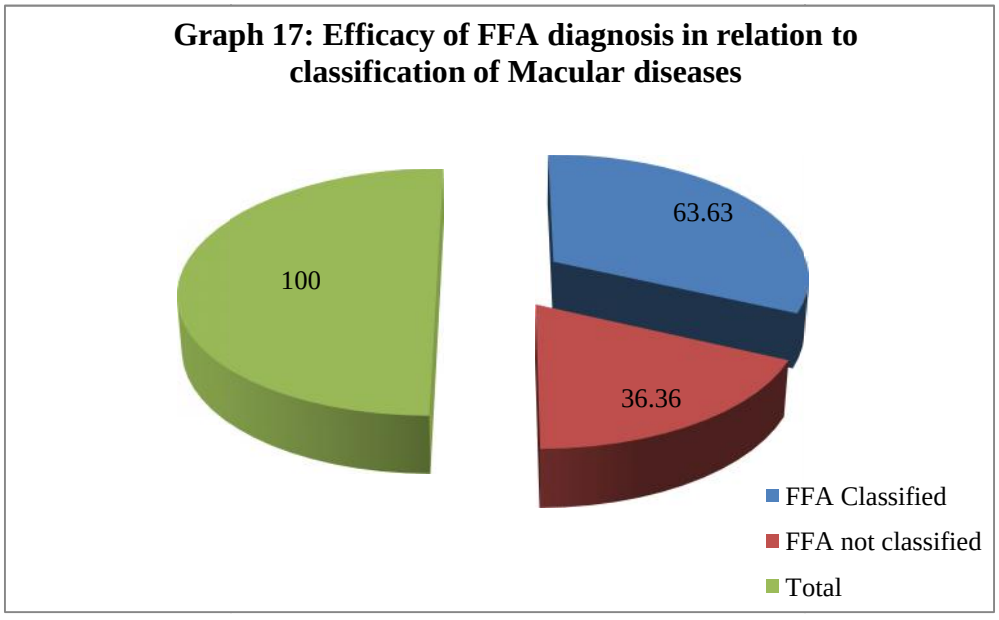

Table 19 and Graph 17 represents the efficacy of FFA in classification of macular diseases. Out of 66 cases studied, FFA has classified the lesions in 64% of cases which is found to be statistically significant with P<0.001.

20. ADVERSE REACTIONS OF FFA

Table 20: Distribution of Adverse Reactions to Fluorescein Dye

Reaction to Fluorescein dye	Number of participants	Percentage (%)
Nil	63	95.31
Present	03	4.68
Nausea	03	4.68

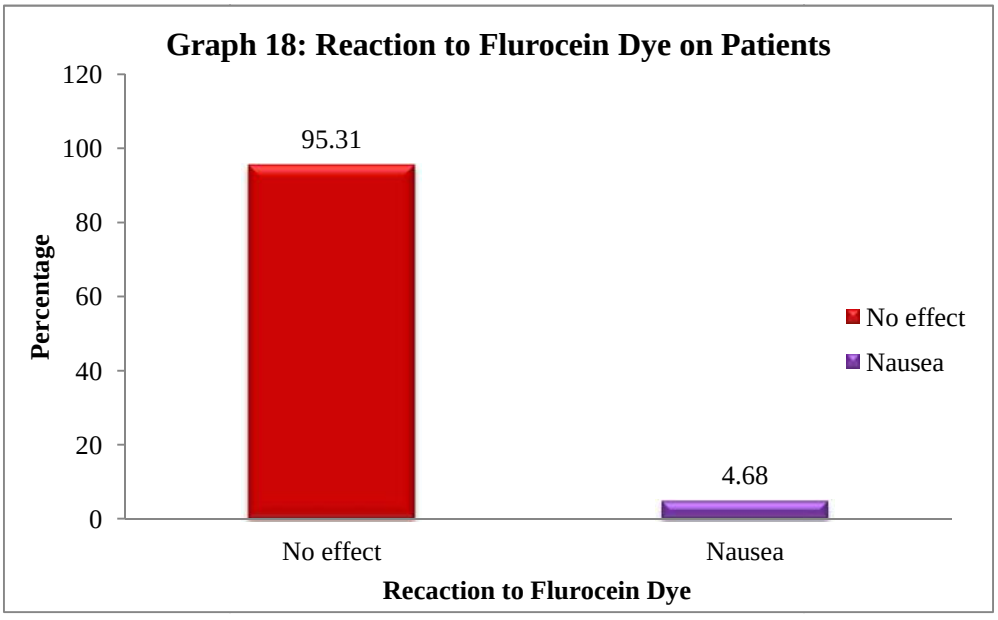

Table 20 and Graph 18 represents the reaction to Fluorescein dye. In present study Out of 66 participants who underwent FFA, Nausea was the most common side effect seen only in 4.68% of cases. No other cases experienced any anaphylactic reactions in our study.

21. STATISTICAL ANALYSIS OF OUR STUDY

Table 21

Agreement	Kappa	Std. Err.	Z-value	P-values
90.91%	0.8908	0.0645	13.8200	<0.001

In present study there was 90.91% agreement between Clinical Ophthalmoscopy and Fundus Fluorescein Angiography(FFA) which is found to be statistically significant (P<0.001).This study demonstrated that FFA is a reliable diagnostic modality for the diagnosis of macular diseases.

DISCUSSION

The present study was conducted on 66 participants with suspected macular pathologies at KLEs Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi during the study period of 1 year from 1st January 2016 to 31st December 2016.

The trend of retinal blindness has changed its pattern over the years in developing countries. Diabetic retinopathy, age-related macular degeneration (ARMD) and retinopathy of prematurity (ROP) have become the important and increasing causes of retinal blindness.

FFA was introduced in clinical use in 1961 by Novotny and Alvis who perfect the photographic study of the human retinal circulation. For over few decades fundus photography and fluorescein angiography have been extremely valuable for expanding our knowledge of anatomy and pathophysiology of the retina and choroid.FFA helps in the diagnosis and monitoring of the treatment of various retinal and choroidal diseases.

In our study total 66 participants with macular pathologies were evaluated in detail both by clinical ophthalmoscopy and Fundus Fluorescein angiography.Among 66 cases, 20(30.30.%) cases of ARMD, 21(31.81%) cases of diabetic Maculopathy, 8(12.12%) cases of vascular occlusions , 10(15.15%) cases of CSCR, 02(3.03%) cases of macular dystrophies , 3(4.54%) cases of inflammatory causes and 2(3.03%) case of macular hole were studied.

The mean age of the study population was 58.95 years. Males were more 63.3% compared to females 36.4%.

1. AGE RELATED MACULAR DEGENERATION(ARMD):

The present study showed the majority of cases were ARMD (30.30%).On analysis of 20 ARMD cases , Dry ARMD cases were 50% and 50% cases were of Wet ARMD. The result of present study are in agreement with observed value of Gatut Suhendro et al on Fundal fluorescein angiography in ARMD who found wet ARMD in 47.3% and dry ARMD in 52.7% of cases.[53]

In our study all the cases of dry ARMD cases were confirmed by FFA. Among Wet ARMD cases Classic CNVM was found in 30% cases and Occult CNVM in 70% cases .Talks J et al in their cross sectional study showed that occult CNVM was seen in 40.5% cases and predominantly classic CNV was seen in 19.8% of cases .[77]

In our study out of 10 cases of Wet ARMD, FFA confirmed the diagnosis in 20% cases and altered the diagnosis in 80% of cases. Talks J et al in their retrospective study showed that 81% of wet ARMD cases could be diagnosed only by FFA.[77]

On analysis of total 20 cases of ARMD, FFA confirmed the diagnosis in 60% of cases and altered the diagnosis in 40% of cases. The results of our study are in agreement with study conducted by Sanjeev K Nainiwal et al who concluded that FFA confirmed the diagnosis in 68.42% of ARMD cases, altered the diagnosis in 31.57% cases .[78]

In our study FFA classified the ARMD lesions in 50% of cases into occult CNVM and classic CNVM .

Arvind et al in their study on FFA in posterior segment diseases concluded that among ARMD cases FFA categorized the lesion in 47% of cases. [55.]

Thus FFA played an immense role in confirming and classifying the lesions in Wet ARMD into different categories which could not be made by clinical ophthalmoscopy alone.

2. DIABETIC MACULOPATHY:

In our study majority of cases were of Diabetic Maculopathy(31.81%). Among 21 cases, 33.33% cases of Clinically significant macular edema,28.57% cases of focal Maculopathy, 23.80% cases of diffuse Maculopathy and 14.28% cases of ischemic Maculopathy were analysed .

Sarfaraz Hussain et al on Incidence of Angiographic Patterns of Diabetic Maculopathy in 115 eyes found that Diffuse Maculopathy and Focal diabetic Maculopathy was seen predominantly followed by Ischaemic type which was seen in less cases.[64]

Qamar et al on diagnosis of diabetic macular edema (DME) based on fundus fluorescein angiography (FFA) findings found three different FFA patterns. Diffuse maculopathy was seen predominantly followed by Focal type and ischaemic type.[61]

Among 21 diabetic Maculopathy cases in our study, FFA confirmed the diagnosis only in 33.33% of cases and altered the diagnosis in 66.66% of cases. The results are in agreement with Suresha et al in their study who showed that FFA has confirmed type of diabetic maculopathy only in 24% cases and has altered diagnosis in 76% of cases.[56]

Out of 21 cases of Diabetic Maculopathy FFA classified the lesions into diffuse maculopathy, focal maculopathy, ischaemic maculopathy and CSME in 66.66% cases.

Arvind et al in their study on FFA in diabetic maculopathy found that FFA is instrumental in categorizing the lesion of diabetic retinopathy and also helpful in identification of clinically significant macular oedema and foveal avascular zone.[55]

In this study, we found that all the cases of ischaemic maculopathy were diagnosed only by FFA by detecting areas of capillary non-perfusion which are not easily recognised by ophthalmoscopy.

Thus FFA is a superior diagnostic modality in confirming and classifying the diabetic Maculopathy cases.FFA is the gold standard in diagnosing Ischaemic Maculopathy cases.

Syed SH et al in their interventional study on diabetic retinopathy concluded that for the proper diagnosis and management only clinical examination cannot be relied upon. FFA must be performed before deciding the appropriate mode of treatment[79]

3. CENTRAL SEROUS CHORIORETINOPATHY(CSCR)

FFA has confirmed the diagnosis in all cases of CSCR and classified the CSCR into ink blot type and smoke stack type based on the pattern of leakage. Among 10 cases majority were of ink blot type constituiting 60% of cases and followed by smoke stack type seen in 40% of cases.

Alicia CSW et al on Angiographic Characteristics of Acute Central Serous Chorioretinopathy in an Asian population confirmed that the inkblot pattern of leakage was the most common pattern seen on angiography. [69]

Maha M et al on Angiographic characteristics of central serous chorioretinopathy in an Egyptian population showed that the inkblot pattern was most common found in 53% of patients.[71]

Thus FFA helps in classifying the type of CSCR based on the pattern of leakage and aids in planning for further management

4. VASCULAR OCCLUSIONS

In our study out of 8 cases of vascular occlusions majority were due to BRVO and one case of CRVO and Cilioretinal artery occlusion each were included. The results of our study are in agreement with Sanjeev K Nainiwal et al who found that Branch Retinal Vein Occlusion was more prevalent followed by Central Retinal Vein Occlusion.[78]

On analysis of 8 cases of vascular occlusions 62.5% of cases were found to have macular edema and 37.5% cases had macular ischemia due to vascular occlusions.

In our study FFA has confirmed the diagnosis in 62.5% cases and altered the diagnosis in 37.5% cases. Macular ischaemia due to vascular occlusions was confirmed only by FFA .

A study by Suresha et al concluded that FFA has confirmed diagnosis in 57.14% cases and altered its diagnosis in 42.85% cases of vascular occlusions.[56]

Thus FFA remains a valuable tool in differentiating macular edema from macular ischemia in vascular occlusions and helps in further management and predicting prognosis.

5. INFLAMMATORY DISEASES

Out of 3 inflammatory causes, 2 cases of choroiditis and one case of CMV retinitis were included..

Vishali Gupta et al in their study on Retinal imaging in uveitis concluded that FFA is useful in differentiating active from inactive uveitis and also confirming the diagnosis of co-existent pathologies like cystoid macular edema, choroidal neovascularization, subtle retinal vasculitis. FFA helped to monitor response to therapy and identifying the areas of capillary non-perfusion as well as retinal neovascularization.[80]

In our study FFA has confirmed the diagnosis in 66.66% cases and altered the diagnosis in 33.33% cases

Sanjeev K et al in their study concluded that on analyzing inflammatory chorioretinal disorders FFA has confirmed diagnosis in 73.33% cases and has altered its diagnosis in 26.67% cases[78]

6. MACULAR DYSTROPHY

In our study Among 2 cases of macular dystrophy, one case of Stargadt's disease and one case of adult onset Vitelliform dystrophy was included .

FFA has confirmed the diagnosis in all the cases of macular dystrophies which were clinically diagnosed by ophthalmoscopic examination.

Wykes et al in their study concluded that FFA confirmed 100% cases of hereditary macular degeneration which was diagnosed by clinical examination [62]

A study conducted by Arvind et al in their study on macular dystrophies concluded that FFA helped in confirming the diagnosis of Macular dystrophies. [55]

7. MACULAR HOLE

In our study 2 cases of full thickness macular hole have been studied. FFA has confirmed the diagnosis in both the cases according to pattern of leakage.

John T. Thompson et al in their study found that Fluorescein angiography is an important tool in the evaluation of macular holes and is most useful when the diagnosis of a full-thickness macular hole is in question. The angiographic findings helped in analyzing successful or unsuccessful closure of the macular hole.[81]

8. ADVERSE REACTIONS OF FFA

In our study among 66 FFAs performed 4.68% cases experienced nausea as the most common side effect. No other cases experienced any anaphylactic reactions.

A prospective study by José M. Beleña1 et al on adverse reactions of fluorescein angiography showed that major adverse reaction was nausea which occurred in only 0.35 of patients [82]

Kunyong Xu et al in their study on adverse reactions during FFA concluded that only 3.3% cases experienced adverse reactions. The most common adverse reaction was nausea and vomiting .[83]

Kwan et al in a recent survey of 11,898 instances of FFA in Australia only 1% adverse reactions were recorded. Most common side effect was nausea and vomiting. No serious adverse reactions or deaths occurred.[84]

9. SATATISTICAL ANALYSIS OF OUR STUDY

In our study out of 66 cases,FFA confirmed the diagnosis in 62.12% of cases and altered the diagnosis in 37.87% cases. FFA played an important role in classifying the macular diseases in 63.63% of cases which is statistically significant.

On statistical analysis by applying binomial-test, analysis of comparison between clinical ophthalmoscopy and FFA was found to be significant. There was 90.91% agreement between clinical ophthalmoscopy and FFA with P value <0.001 which was found to be statistically significant. Thus FFA can be used as a superior diagnostic modality in classification and diagnosis of macular diseases.

Arvind R et al on Role of Fluorescein angiography in evaluation of posterior segment disorders concluded that FFA is a superior diagnostic tool and is a necessity for evaluating, localizing and categorization of lesions in Retinal, Macular and Choroidal pathologies[55]

Verma et al in their study at RP centre, AIIMS Delhi concluded that FFA is the most useful and practical approach for tracing the macular lesions with findings conclusive in 80% of cases".[85]

CONCLUSION

The present study conducted on 66 participants with suspected macular pathologies at KLEs Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi during the study period of 1 year from 1st January 2016 to 31st December 2016.

In this study, we examined all the participants by conventional methods of ophthalmoscopy followed by Fundus Fluorescein Angiography. The findings were analysed and categorized

The following conclusions were drawn from the study.

- FFA confirmed the diagnosis in all the Dry ARMD cases which were diagnosed by clinical ophthalmoscopy.
- FFA played a definitive role in diagnosing wet ARMD by early detection and localization of site of CNVM in relation to the foveal avascular zone.
- FFA classified the Wet ARMD cases into Occult CNVM and Classic CNVM. Thus FFA is an important investigation for diagnosis of wet ARMD.
- FFA confirmed and classified the Diabetic Maculopathy into focal maculopathy, diffuse maculopathy, ischaemic maculopathy and CSME in all the cases. The predominant type of diabetic maculopathy was found to be CSME followed by focal and diffuse diabetic maculopathy .
- The ischaemic diabetic maculopathy cases were diagnosed only by FFA, thus FFA is gold standard in the diagnosis of ischaemic diabetic Maculopathy cases.

- FFA must be performed before deciding the appropriate mode of treatment in diabetic maculopathy especially during laser therapy for detecting areas of macular edema.
- Among vascular occlusions majority of the cases were due to BRVO.FFA played a major role in differentiating macular oedema from macular ischemia which helped in the prognosis of these conditions.
- In the study of macular dystrophy FFA confirmed the diagnosis in all the cases.FFA can be used as a diagnostic modality in confirmation of macular dystrophy.
- FFA gave a definitive diagnosis in CSCR cases by detecting the number of leakage points and the exact site of leakage.FFA classified the leakage pattern into inkblot and smoke stack pattern and played an immense role in laser photocoagulation by detecting exact site of leakage points in CSCR.
- FFA confirmed the diagnosis in full thickness macular hole by detecting the pattern of leakage.FFA can be used as a diagnostic modality in confirming macular hole cases.
- Macular edema due to inflammatory cause was confirmed by FFA in majority of the cases which helped in the prognosis of these conditions.
- In our study nausea was the most common adverse effect seen only in 3 cases with no anaphylactic reactions seen.Therefore FFA can be used as a safe and valuable tool in diagnosis and management of macular disorders.

In conclusion FFA can be used as a superior diagnostic modality in classification and diagnosis of macular diseases.

SUMMARY

Retinal disease is the primary cause of 12.7% of blindness in a population based surveys in India. Macular diseases are the most frequent constituiting 35.6% of all posterior segment disease. Macular diseases like Age Related Macular Degeneration, Central Serous Retinopathy, Diabetic Macular Edema, Vascular occlusive diseases can cause irreversible blindness and thus requires detailed evaluation and management. Fundus Fluorescein Angiography helps us to examine structures in macular region which are beyond the reach of direct ophthalmoscopy and fundus photography. The purpose of this study was to assess the role of FFA in classification and diagnosis of macular diseases.

The present study conducted on 66 participants with suspected macular pathologies at KLEs Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi during the study period of 1 year from 1st January 2016 to 31st December 2016. In this study we examined all the participants by conventional methods of ophthalmoscopy followed by Fundus Fluorescein Angiography. The findings were analysed and categorized.

The mean age of the study population was 58.95 years. Majority of the participants were males (63.6%). Majority of the participants were having ARMD (30.30%) and Diabetic Maculopathy (31.81%) where as 15.15% participants were having CSCR and 12.12% were having vascular occlusions. The least participants were having inflammatory causes followed by macular dystrophy and macular hole.

On the analysis of 20 cases of ARMD, FFA confirmed the diagnosis in 60% of cases and altered the diagnosis in 40% of cases.FFA classified the lesions in 50%

of cases of ARMD .FFA was definitive in the diagnosis of Wet ARMD and classified the lesions into Occult CNVM and Classic CNVM which could not be made by clinical ophthalmoscopy alone.

In our study out of 21 cases of Diabetic Maculopathy, majority of participants were having CSME (33.33%) and Focal Diabetic Maculopathy (28.57%) followed by Diffuse Maculopathy seen in 23.80% cases. The least participants (14.28%) were having ischaemic maculopathy.FFA has confirmed the diagnosis only in 33.33% cases and has altered diagnosis in 66.66% of cases. FFA is the gold standard in diagnosing ischaemic maculopathy cases. FFA classified the lesions in 66.66% of cases. Thus FFA played a major role in classification and diagnosis of Diabetic Maculopathy and played an important role in planning of further management.

In the study of vascular occlusions majority (75%) of the participants were having BRVO. FFA has confirmed the diagnosis in 62.50% cases and altered the diagnosis in 37.50% cases. Macular ischaemia due to vascular occlusion was confirmed only by FFA. FFA remains a valuable tool in differentiating macular edema from macular ischemia in vascular occlusions.

Out of 2 macular dystrophy cases FFA confirmed the diagnosis in all the cases according to the pattern of leakage.FFA also confirmed the diagnosis in macular hole cases and helped in planning for further management.

The present study showed that 15.15% participants were diagnosed as CSCR. Out of which 60% were diagnosed as Ink Blot type and 40% diagnosed as smoke stack type.FFA played a definitive role in the diagnosis of CSCR by detecting the exact site of leakage.

Among inflammatory occlusions majority (66.66%) of the participants were having choroiditis followed by CMV retinitis. Macular edema due to vascular occlusions was confirmed only by FFA.

Out of 66 FFAs performed, only 4.68% participants experienced nausea as the most common side effect. None of the cases experienced any anaphylactic reactions. Thus FFA can be used as a safe diagnostic modality in the evaluation of macular diseases.

In our study out of 66 cases of macular diseases,FFA confirmed the diagnosis in 62.12% of cases and altered the diagnosis in 37.87% of cases. On statistical analysis there was 90.91% correlation found between clinical ophthalmoscopy and FFA with $P<0.001$,which is found to be statistically significant. Out of 66 cases, FFA classified the lesions in 63.63% of cases which is also statistically significant.

Thus FFA can be used as a superior diagnostic modality in the classification and diagnosis of macular diseases.

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ANNEXURE-I

CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mr/Mrs/Ms_____You are invited to participate in our research study **titled " HOSPITAL BASED STUDY ON THE ROLE OF FUNDUS FLUORESCEIN ANGIOGRAPHY IN CLASSIFICATION AND DIAGNOSIS OF MACULAR DISEASES"** at KLES Dr.Prabhakar Kore Hospital, Belagavi." Conducted by J.N. Medical College, Belagavi.

Respected Sir/Madam we request you to enroll yourself to participate in our study as you are eligible for doing so.

Your participation in the study is voluntary. Your decision whether or not to participate in the study will not affect your relationship with J.N. Medical College. If you decide to participate you are free to withdraw at any time.

The purpose of the research is to assess the role of fundus fluorescein angiography(FFA) in classification and diagnosis of macular diseases. If you agree to enroll yourself in this study, you will be asked to give detailed history. Then you will be clinically examined in detail by slit-lamp examination, Clinical ophthalmoscopy. Investigations like Blood Pressure measurement, Random Blood sugar, renal function tests(Blood urea and Serum Creatinine),HbA1C,Urine albumin, sugar and microscopy will be done. Then you will be undergoing fundus fluorescein angiography (FFA).Precautions will be taken during the procedure. Your participation may benefit

you and others and others suffering from same ailment in future, by helping us learn more about the disease process and better treatment modalities.

There will not be any extra cost incurred by the participant. The participant will however have to pay for the investigations which are the part of the existing management protocol for this ailment. There is no commitment for any reimbursement or and other compensation for the participant.

The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to others without your written permission.

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

If you have any question about your rights as a study participant, you may also contact **Dr. GANGA S. PILLI** **MD.DCP.DPM** Chairman, institutional ethics committee on Human Subjects Research, Professor Department of pathology JNMC, Belagavi. Contact no. 08312471350/09480275601

Consent for participation in research trial

I, Mr./Ms./ Mrs _____ voluntarily agree for the participation as a subject of the present study. By signing this consent form I am not giving up any of my legal rights, I may withdraw from the study at anytime. I am signing the consent form after having read or been read for me in vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name: _____

Signature or the Left Thumb Print of Subject: _____

Witness Name: _____

Signature of Witness: _____

Investigators Name: _____

Signature of Investigator: _____

Date: _____

Place: _____

ANNEXURE-II

PROFORMA

PATIENTS ID NO: ☐☐☐

NAME: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

AGE: ☐☐ Years

SEX: ☐ (1-Male; 2-Female)

ADDRESS: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐ ☐

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

CONTACT NUMBER :- ☐☐☐☐☐☐☐☐☐☐

OP NUMBER: ☐☐☐☐☐☐☐☐

IP NUMBER: ☐☐☐☐☐☐☐☐

DATE OF ADMISSION: ☐☐ / ☐☐/ ☐☐☐☐

DATE OF DISCHARGE: ☐☐ / ☐☐ / ☐☐☐☐

IS THE PATIENT ELIGIBLE FOR STUDY? (1-YES; 2-NO) ☐

HAS INFORMED CONSENT BEEN GIVEN? (1-YES; 2-NO) ☐

FINAL RESULT INFORMATION ☐

1- INELIGIBLE
2- ELIGIBLE- REFUSAL
3- ELIGIBLE- PARTICIPATING

SURGEON’S NAME: ☐☐☐☐☐☐☐☐☐☐☐☐☐

SURGEON’S SIGNATURE: ____________________

DATE: ☐☐/ ☐☐/ ☐☐☐☐

CHIEF COMPLAINTS:

DIMINUTION OF VISION RE ☐

Duration: _____ days/ months/years

LE ☐

Duration: _____ days/ months/years

HISTORY OF PRESENT ILLNESS:

1 .DIMINUTION OF VISION 1- Gradual; 2- Sudden ☐

1- Progressive; 2- Static ☐

1- Painless; 2- Painful ☐

1- For distance; 2- For near ☐

2. DIPLOPIA/POLYOPIA 1- Present; 2- Absent ☐

3. COLOURED HALOS 1- Present; 2- Absent ☐

4. BLACK SPOTS BEFORE THE EYES 1- Present; 2 - Absent ☐

5. WATERING 1- Present; 2 - Absent ☐

6. REDNESS 1- Present; 2 - Absent ☐

7. DISCHARGE 1- Present; 2 - Absent ☐

8. H/O WEARING GLASSES (1-Distance; 2-Near; 3-Both) ☐

Duration: ☐☐ months/years

PAST HISTORY:

TRAUMA TO THE EYE: 1- Present; 2- Absent ☐

OCULAR SURGERY: 1- Present; 2- Absent ☐

Type of surgery: _____

Duration: ☐☐ months/years

DIABETES: 1- Present 2- Absent ☐

Duration: ☐☐ months/years

HYPERTENSION: 1- Present 2- Absent ☐

Duration: ☐☐ months/years

ANY OTHER MEDICAL DISORDERS: _____

PERSONAL HISTORY:

SMOKING: 1- Present; 2- Absent ☐

Duration: ☐☐ months/years

ALCOHOLISM: 1- Present; 2- Absent ☐

Duration: ☐☐ months/years

ANY OTHER ADDICTIONS: _____ ☐

Duration: ☐☐ months/years

GENERAL PHYSICAL EXAMINATION:

General Appearance:

1- Well built ,2- Moderately built, 3- Poorly built, 4- emaciated ☐

Pallor: 1- Present 2- Absent ☐

If present 1- Mild 2- Moderate 3- Severe ☐

Pulse: ☐☐☐/minute

BP:- ☐☐☐/ ☐☐☐mm of hg

Temperature: ☐☐☐gree Fahrenheit

Respiratory rate: ☐☐inute

SYSTEMIC EXAMINATION:

CVS: 1- Normal 2- Abnormal ☐
if 2, specify :_____

RS: 1- Normal 2- Abnormal ☐
if 2, specify: _____

CNS: 1- Normal 2- Abnormal ☐
if 2, specify :_____

Per Abdomen: 1- Normal 2- Abnormal ☐
if 2, specify :_____

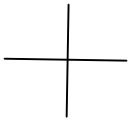
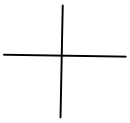
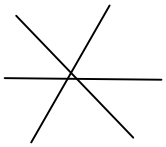
OCULAR EXAMINATION:

Head posture: 1- Erect ,2- Tilted ☐

Visual Axis: 1- Parallel, 2- Deviated ☐

Facial Symmetry: 1- Symmetrical, 2- Asymmetrical ☐

Extraocular movements:

RE-  LE-  Binocular :- 

(N- Normal, R- Restricted) ☐

1) Visual Acuity:

	RE	LE
DISTANT		
PINHOLE		
NEAR		
AIDED		

2. Adnexa (1- Normal; 2-Abnormal)	☐	☐
3. Sclera (1- Normal; 2- Congested)	☐	☐
4. Conjunctiva (1-normal; 2-conjunctival congestion; 3-ciliary congestion; 4-chemosis)	☐	☐
5. Cornea (1- normal; 2-opacity; 3-vascularisation)	☐	☐
6. Anterior chamber (1- normal depth; 2-shallow; 3-deep)	☐	☐
7. Iris (1-normal colour & pattern; 2-Abnormal)	☐	☐

8. Pupil: Size- ____ in mm Shape- 1- Round & Regular; 2-Abnormal Reaction: Direct (1. Present, 2. Absent) Indirect (1. Present, 2. Absent) Near reflex (1. Present, 2. Absent)	☐ ☐ ☐ ☐	☐ ☐ ☐ ☐
9. Lens Clarity- 1. Clear, 2. Opaque Cataract - (1) , PCIOL - (2) Cataract if present- 1.immature 2.mature 3. hyper mature	☐ ☐ ☐	☐ ☐ ☐

FUNDUS	RE	LE
GLOW		
MEDIA		
DISC		
C:D RATIO		
BLOODVESSELS		
BACKGROUND		
MACULA		

DIAGNOSIS:-

IMPRESSION:-

INVESTIGATIONS:

1. Ocular

A) IOP:

RE: ☐ mm of hg
LE : ☐ mm of hg

AMSLER RE: ☐

LE : ☐

B) Blood sugar: _____mg%

C) Blood Pressure:_____mm of hg

D) COMPLETE HAEMOGRAM:

E) HbA1C:

F) LIPID PROFILE:

G) BLOOD UREA:

H) SERUM CREATININE:

I)URINE

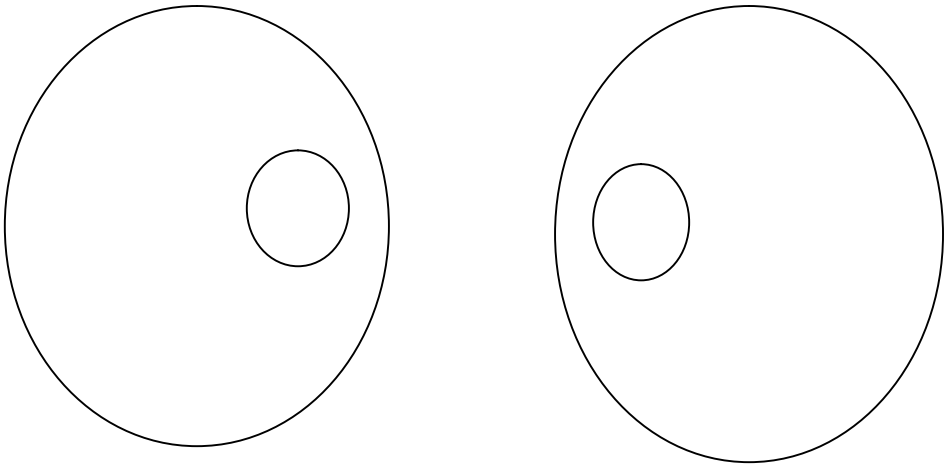
ALBUMIN :

SUGAR: :

MICROSCOPY:

FFA :

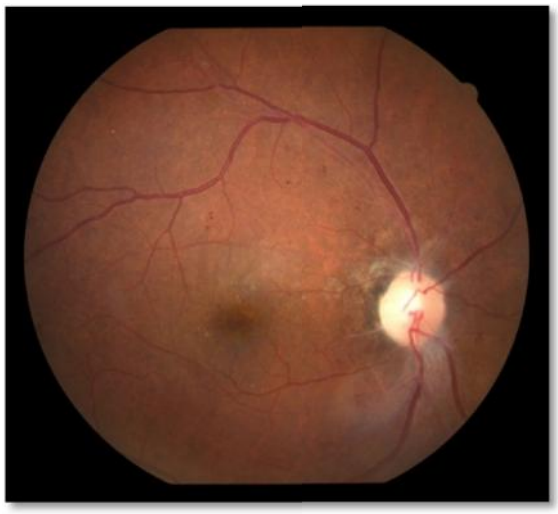
RE **LE**



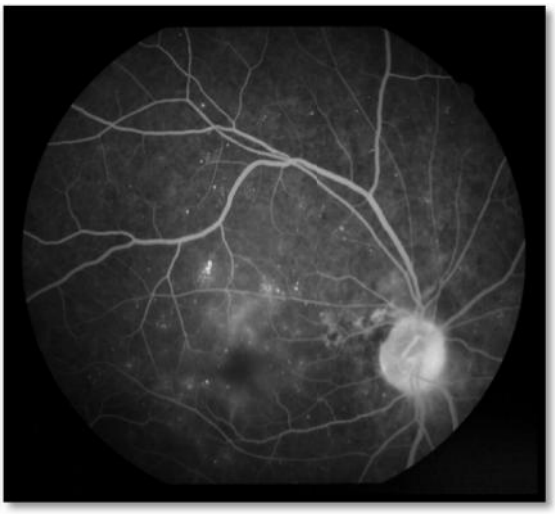
ANNEXURE III – PHOTOGRAPHS

PHOTOGRAPH 1:FUNDUS CAMERA

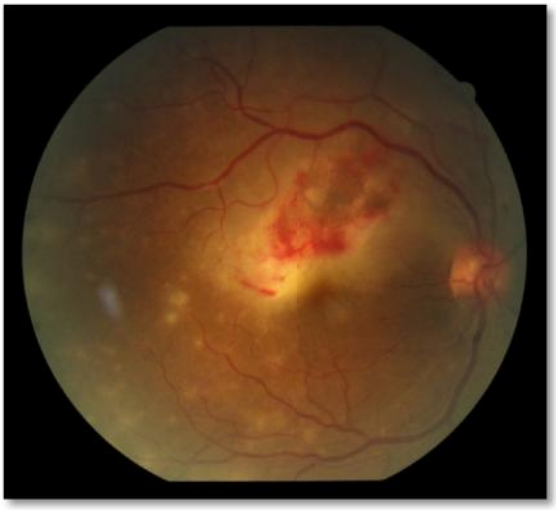
PHOTOGRAPH 2:SODIUM FLUORESCEIN DYE



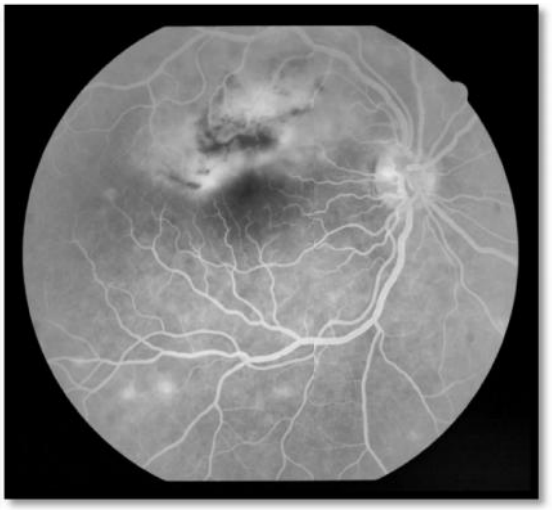
PHOTOGRAPH 3: MILD NPDR



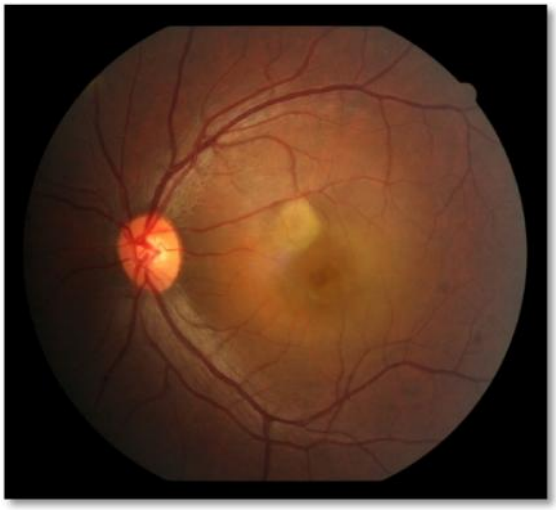
PHOTOGRAPH 4: FFA - DIFFUSE DIABETIC MACULOPATHY



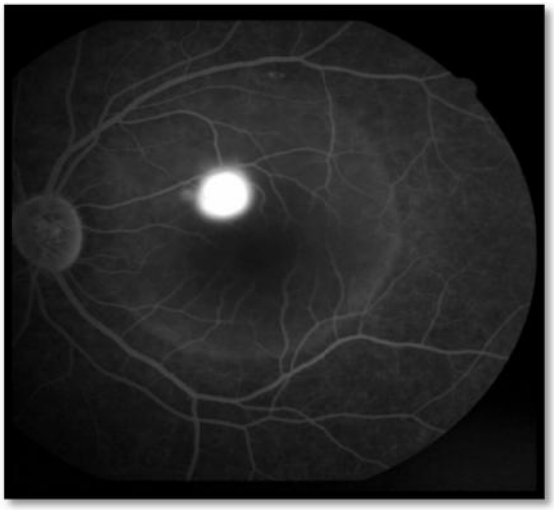
PHOTOGRAPH 5: CMV RETINITIS



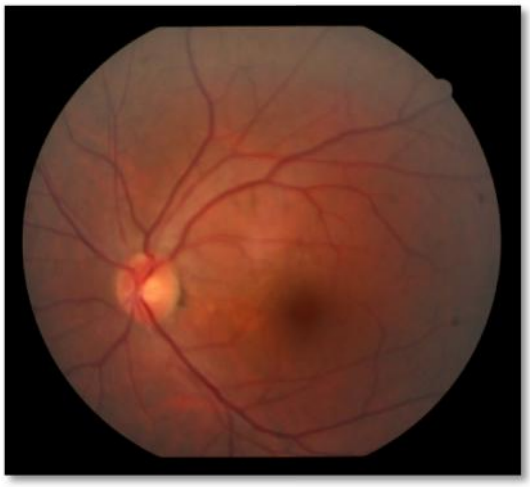
PHOTOGRAPH 6: FFA CMV RETINITIS WITH MACULAR EDEMA



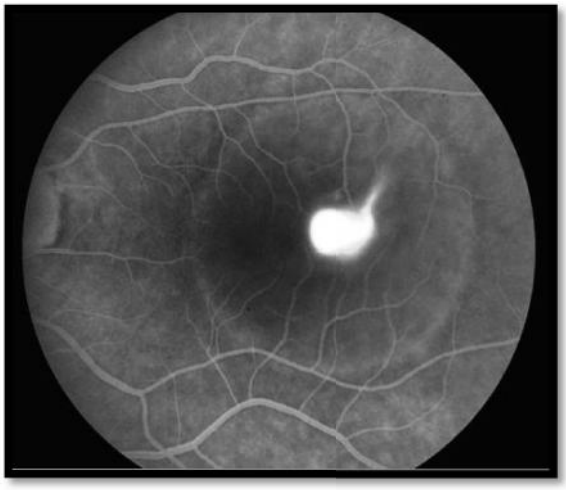
PHOTOGRAPH 7: CENTRAL SEROUS RETINOPATHY



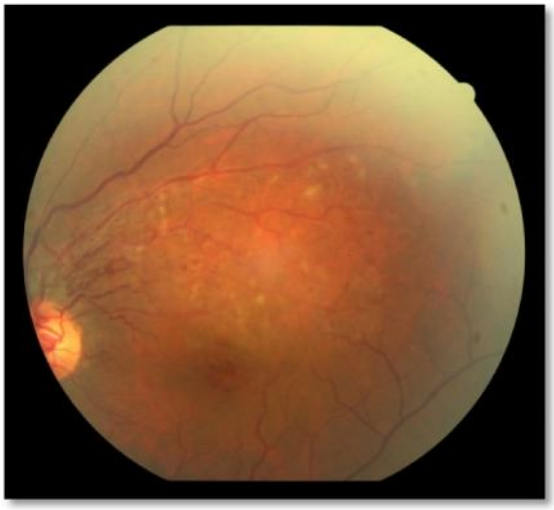
PHOTOGRAPH 8: : FFA - INK BLOT TYPE OF CENTRAL SEROUS RETINOPATHY



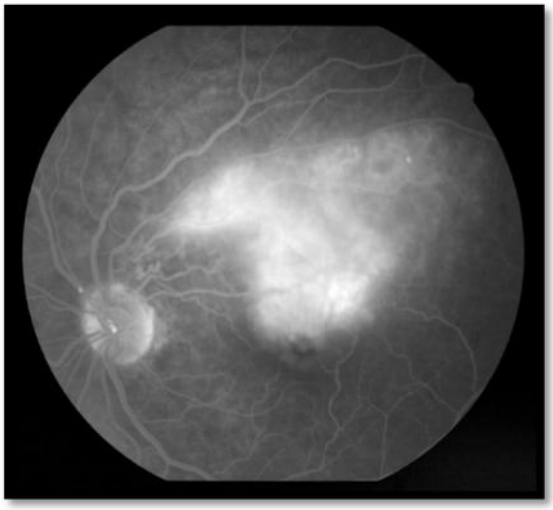
PHOTOGRAPH 9 : CENTRAL SEROUS RETINOPATHY



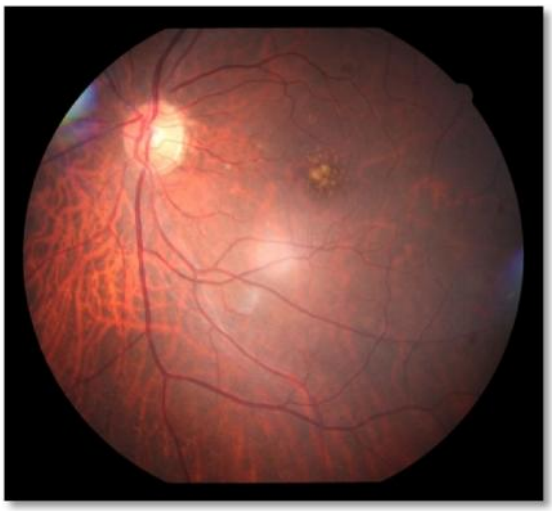
PHOTOGRAPH 10: FFA SMOKE STACK TYPE OF CENTRAL SEROUS RETINOPATHY



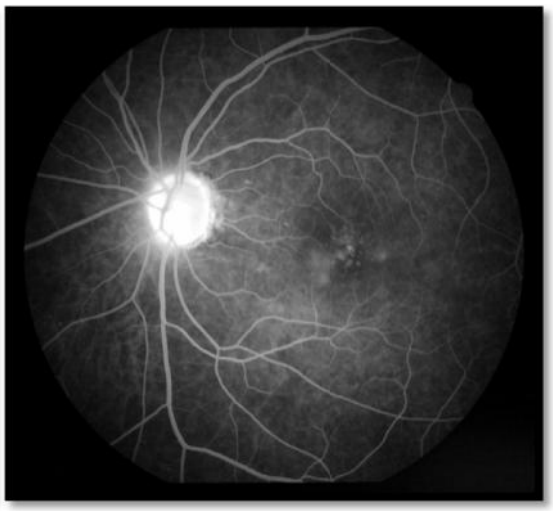
PHOTOGRAPH 11: LEFT EYE SUPERO TEPORAL(ST) BRVO



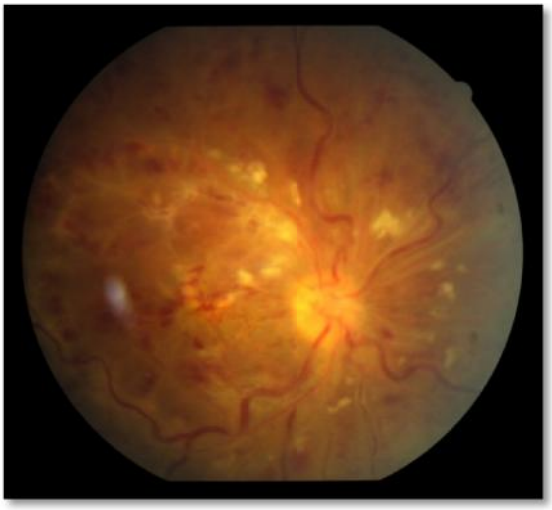
PHOTOGRAPH 12: FFA-LEFT EYE ST BRVO WITH MACULAR EDEMA



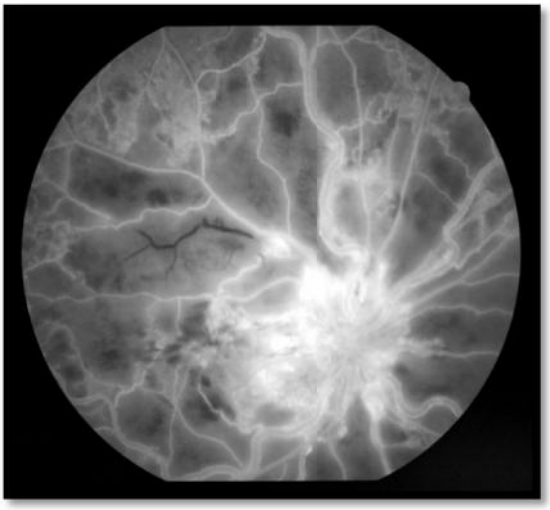
PHOTOGRAPH 13: DRY ARMD



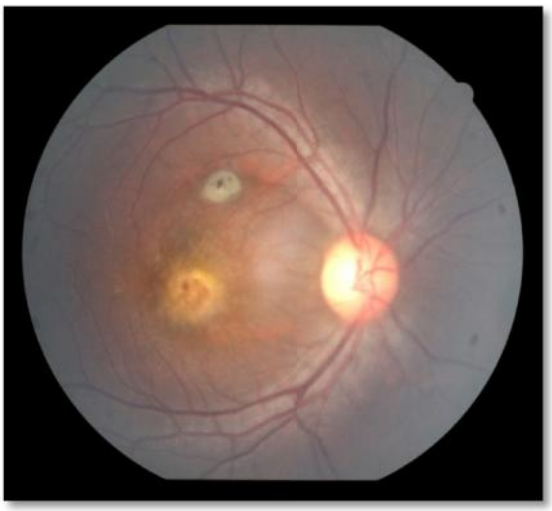
PHOTOGRAPH 14: FFA-DRY ARMD



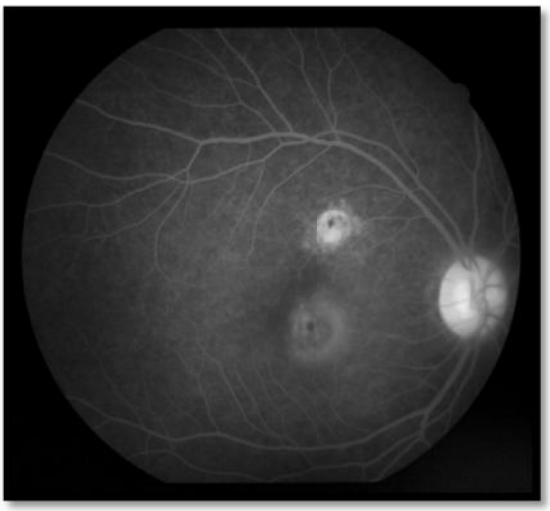
PHOTOGRAPH 15: CRVO



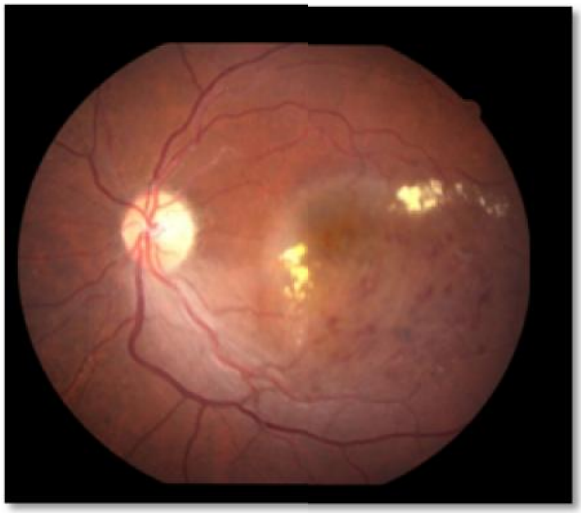
PHOTOGRAPH 16: FFA-NON ISCHAEMIC CRVO WITH MACULAR EDEMA



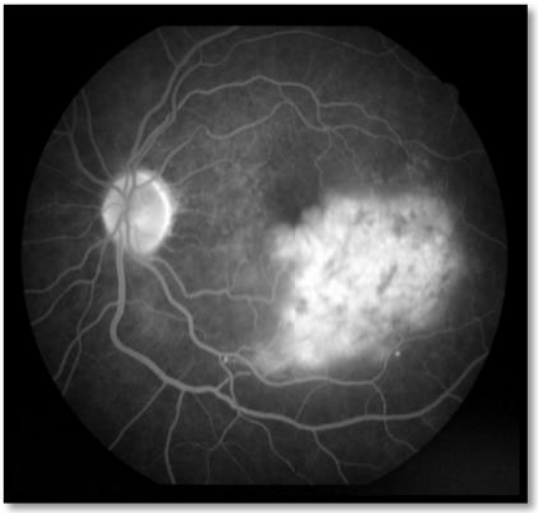
PHOTOGRAPH 17: CHOROIDITIS WITH ?MACULAR EDEMA



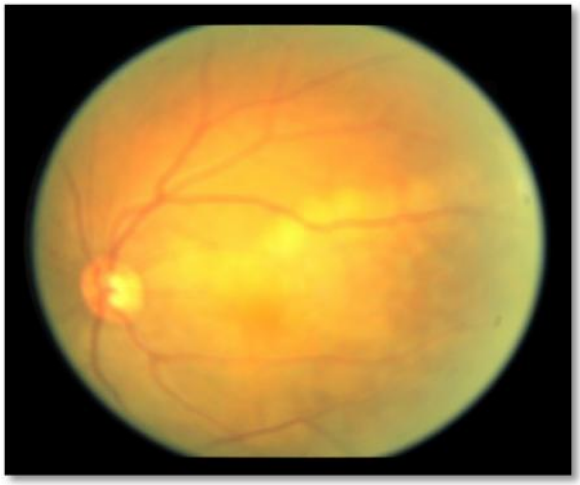
PHOTOGRAPH 18: FFA-CHOROIDITIS WITH MACULAR EDEMA



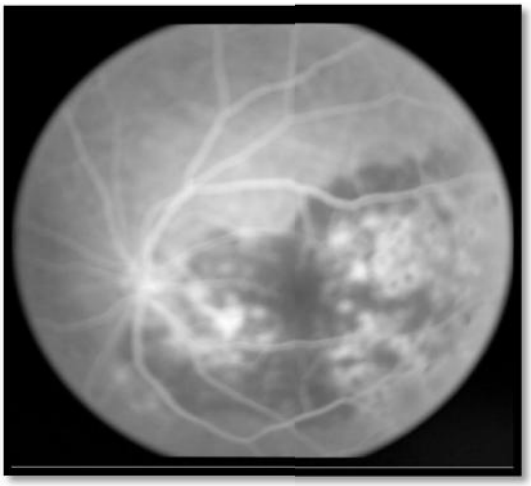
PHOTOGRAPH 19: INFERO TEMPORAL BRVO WITH MACULAR EDEMA



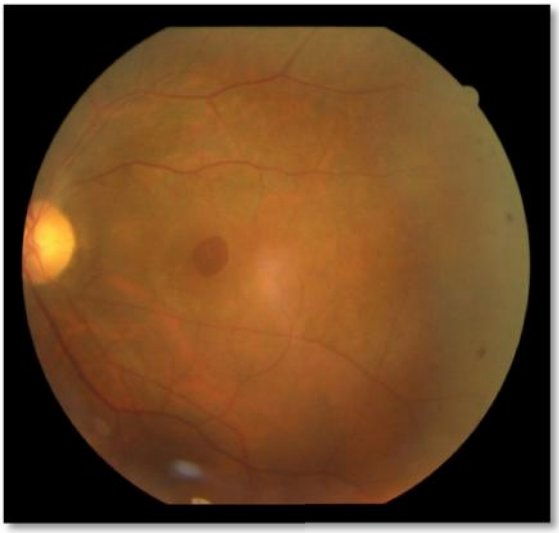
PHOTOGRAPH 20: FFA-INFERO TEMPORAL BRVO WITH MACULAR EDEMA



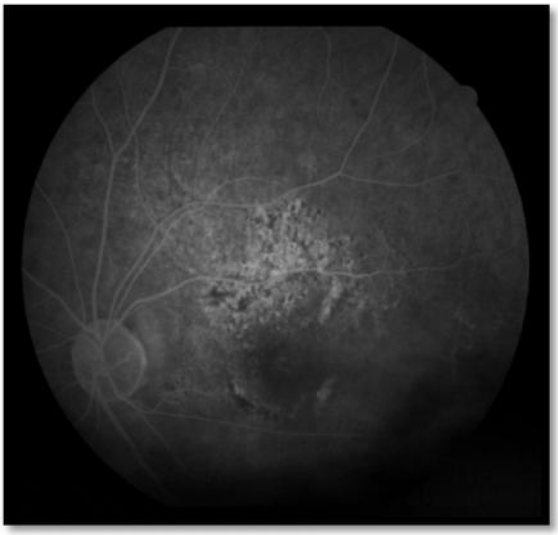
PHOTOGRAPH 21: CILIORETINAL ARTERY OCCLUSION



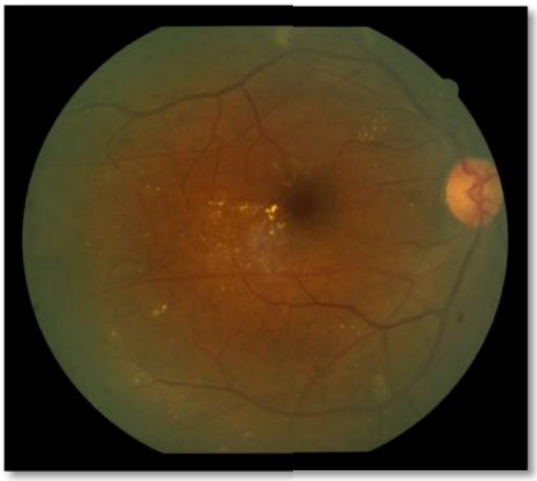
PHOTOGRAPH 22: FFA-CILIORETINAL ARTERY OCCLUSION WITH MACULAR EDEMA



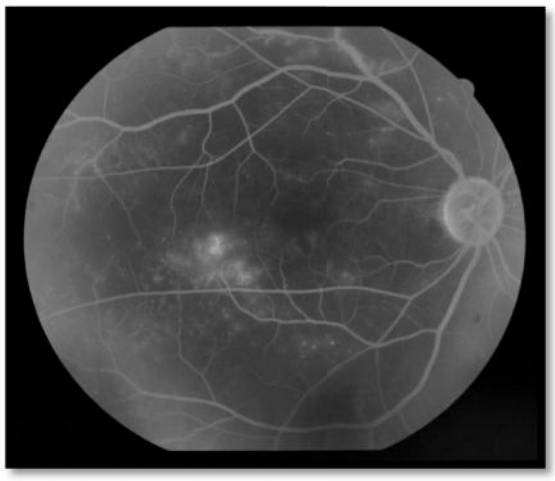
PHOTOGRAPH 23: LEFT EYE FULL THICKNESS MACULAR HOLE



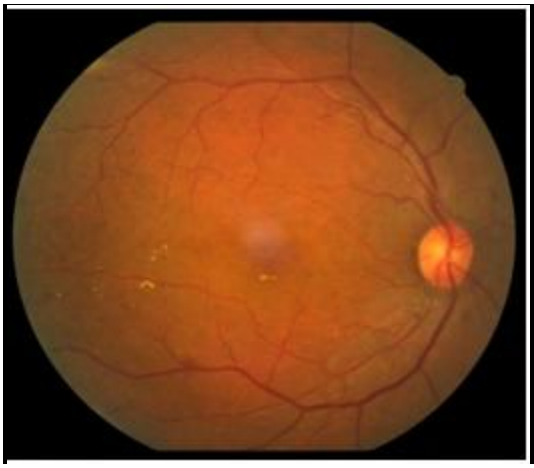
PHOTOGRAPH 24: FFA -LEFT EYE FULL THICKNESS MACULAR HOLE



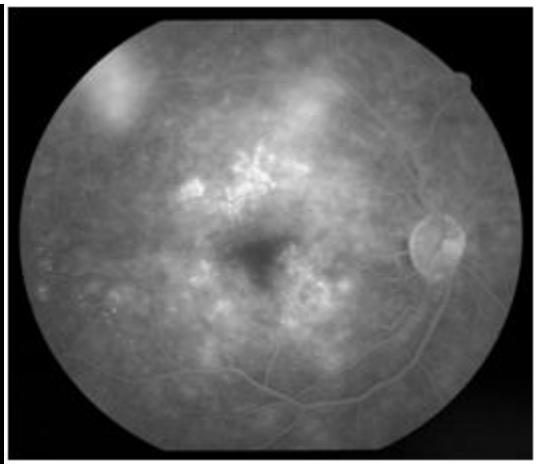
PHOTOGRAPH 25: RIGHT EYE MODERATE NPDR



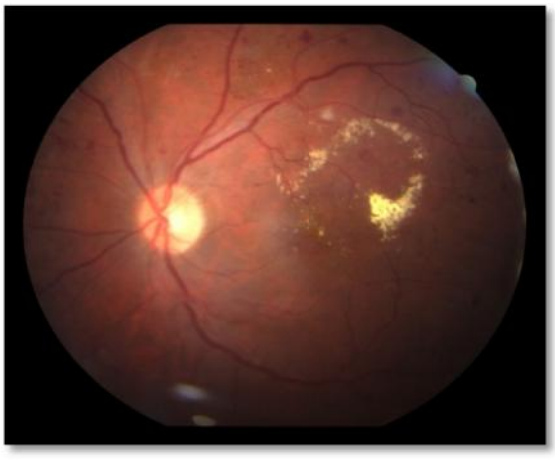
PHOTOGRAPH 26: FFA-RIGHT EYE FOCAL DIABETIC MACULOPATHY



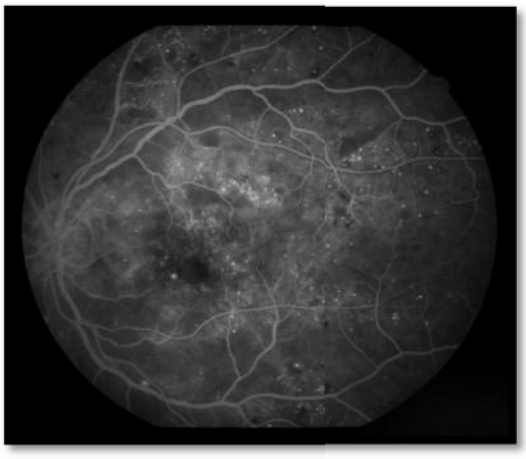
PHOTOGRAPH 27: RIGHT EYE MILD NPDR



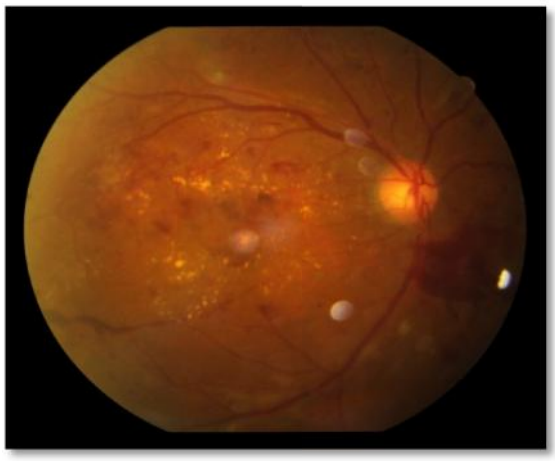
PHOTOGRAPH 28: FFA-RIGHT EYE DIFFUSE DIABETIC MACULOPATHY



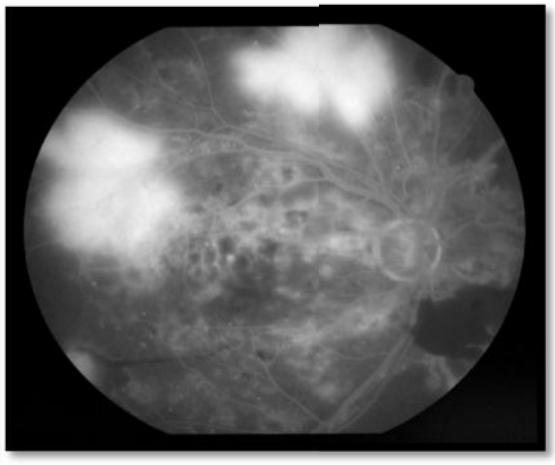
PHOTOGRAPH 29: RE CSME



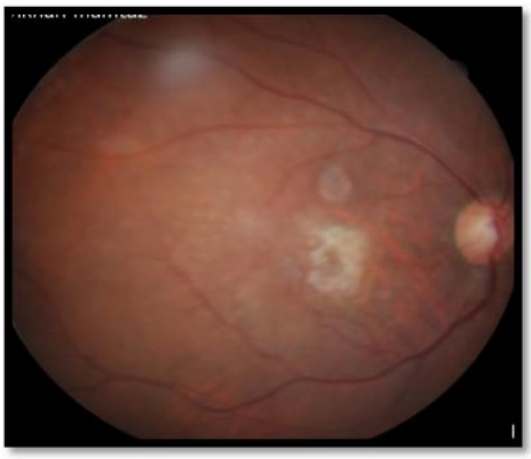
PHOTOGRAPH 30: FFA-RE MODERATE NPDR WITH CSME



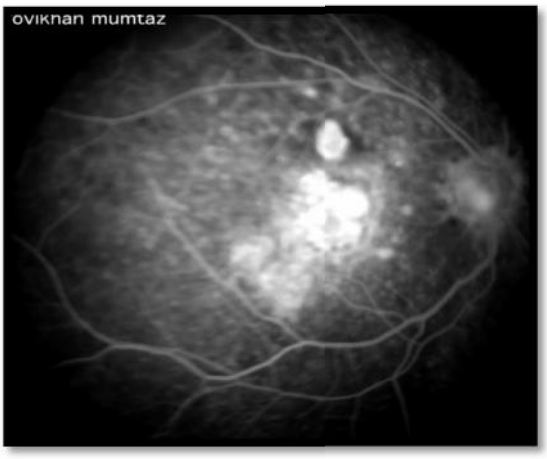
PHOTOGRAPH 31: RE PROLIFERATIVE DIABETIC RETINOPATHY



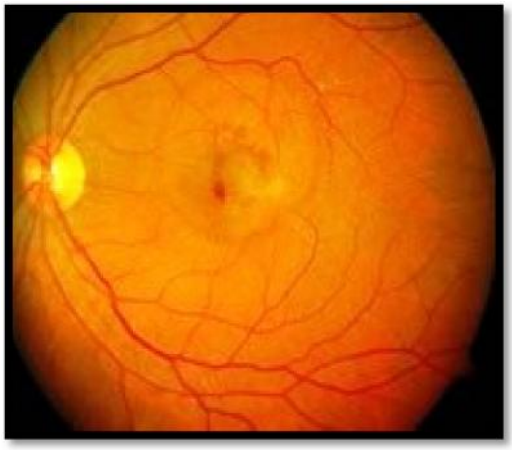
PHOTOGRAPH 32: FFA-PDR WITH NVE WITH DIFFUSE DIABETIC MACULAR EDEMA



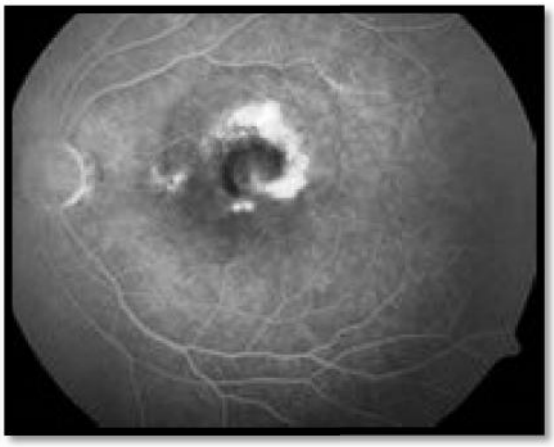
PHOTOGRAPH 33: CLASSIC SUBFOVEAL CNVM



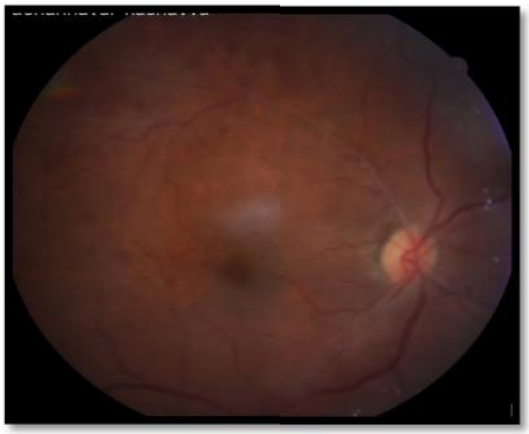
PHOTOGRAPH 34: FFA-SHOWING LACY HYPERFLUORESCENCE IN CLASSIC SUBFOVEAL CNVM



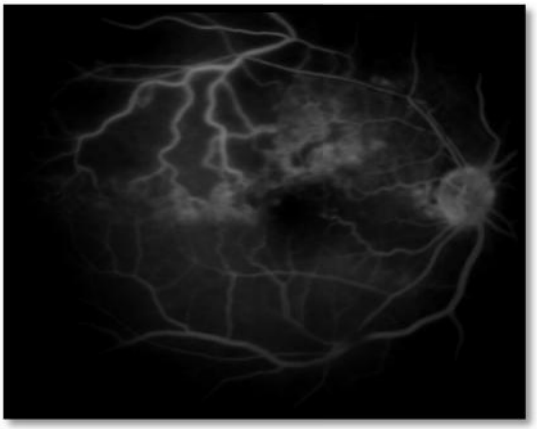
PHOTOGRAPH 35:WET ARMD ?OCCULT CNVM



PHOTOGRAPH 36: FFA-OCCULT CNVM

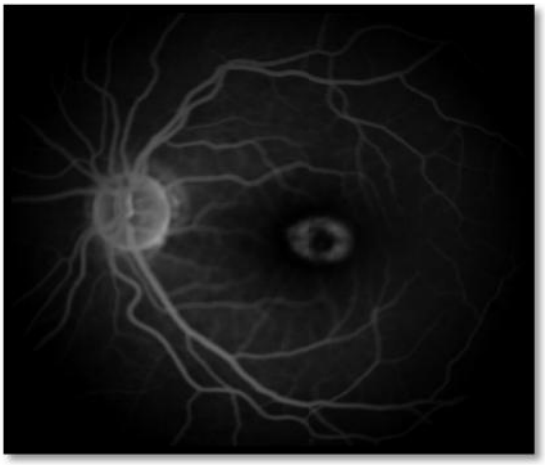


PHOTOGRAPH 37: RIGHT EYE SUPERO TEMPORAL BRVO



PHOTOGRAPH 38: FFA-ISCHAEMIC BRVO WITH AREAS OF CAPILLARY NON PERFUSION

PHOTOGRAPH 39: STARGADT'S DISEASE



PHOTOGRAPH 40: FFA- STARGADT'S DISEASE

S.NO	NAME	AGE	SEX	IP/OP NO.	SYSTEMIC DISEASES RISK FACTOR		VA	VA	PH	PH	NV	NV	CV	CV	AMSLER	AMSLER	FUNDUS FINDINGS(Direct,Indirect ,90D)	DIAGNOSIS SUSPECTED	FFA INTERPRETATION	FFA DIAGNOSIS	DIAGNOSIS CONFIRMED BY FFA	DIAGNOSIS ALTERED BY FFA	CLASSIFICATION/ CATEGORIZATIO N BY FFA	REACTION TO FLUORESCEI N
							RE	LE	RE	LE	RE	LE	RE	LE	RE	LE								
1	SUNITA PATIL	44	F	3954237			6\6	6\60	6\6	6\36	N6	N18	N	N	N	N	Left eye chorioretinal atrophic patch present	Left eye choroiditis	Hyperfluorescence in the early phase increased in intensity in late phase	Macular edema due to choroiditis		Yes		Nil
2	YALLUBAI KESAREKAR	70	F	3737047			CF1M	CF1M	NI	NI							Both eyes well defined,round and thickened lesion in the macula	Both eyes macular hole	Window defect in early phase followed by hyperfluorescent halo in late phase	Both eyes full thickness macular hole	Yes			Nausea
3	CHANNABASAPPA	80	M	4117216	Hypertension	Smoking	CF2M	CF2M	NI	NI	N36	N36	N	N	D	D	Yellowish lesion at the macula with neovascularisation	Both eyes ? wet armd	Lacy hyperfluorescence in the arteriovenous phase which increased in late phase	Both eyes occult cnvm		yes	Occult cnvm	Nil
4	SHIVABASAPPA N	65	M	1820881		Smoking	6\24	6\18	6\12	6\9	N8	N6	N	N	N	N	Soft drusens with RPE changes are seen	Both eyes dry armd	Hyperfluorescence noted only in late phase of ffa	Both eyes dry armd	Yes			Nil
5	DATTATREYA PATIL	79	M	3633817	DM & HTN	Smoking	6\60	6\36	6\60	6\18	N10	N10	N	N	D	N	soft drusens at the macula are seen	Re dry armd	Diffuse hyperfluorescence in the early phase increased in intensity in late phase	Right occult cnvm		Yes	Occult cnvm	Nil
6	NARASAGOWDA	60	M	3265771	HTN		CF2M	6\60	CF2M	6\24	N8	N6	N	N	D	N	Re confluent large soft drusens with elevated areas	?Re cnvm	Pathcy hyperfluorescence noted in early phase with late staining at the macula	Right occult cnvm	Yes		Occult cnvm	Nil
7	GEETA BADAVANE	50	F	3566403	DM		6\24	6\18	6\18	6\9	N8	N8	N	N	D	D	Both eyes microaneurysms and exudates are seen	Both eyes mild npdr	Focal hyperfluorescence noted in the late arteriovenous phase due to leakage	Both eyes focal diabetic maculopathy		Yes	Focal diabetic maculopathy	Nil
8	LEELA	32	F	3652818	Stress		6\6	CF1M	6\6	NI	N6	N36	N		N	D	Left eye round circular ring reflex seen at the macula with neurosensory detachment	Left eye cscr	Early hyperfluorescent spot progressing in a vertical column like a smoke stack pattern	Left eye cscr	Yes		Smoke stack type cscr	Nil
9	YALLUBAI KILLEKAR	61	F	3765808	DM & HTN		6\12	CF1M	6\9	CF1M	N8	N18	N	N	N	D	Left eye Attenuation of the artery with cloudy appearance at the distribution of cilioretinal artery	Left eye cilioretinal artery occlusion	Early phase showing filling deffect with enlarged faz with late leakage	Macular edema due to cilioretinal artery occlusion		Yes	Macular edema	Nil
10	RAJASHREE C	45	F	3774378	Hyperlipidemia & HTN		CF1/2M	6\6	CF1/2M	6\6		N6		N	D	N	Re dilated and tortuous veins with extensive flame shaped haemorrhages	Re crvo with macular edema	Delayed venous filling with hyperfluorescence in the late phase with good capillary perfusion is seen	Right macular edema due to old non ischeamic crvo	Yes		Macular edema	Nil
11	AKBAR	65	M	3443458	DM	Smoking	6\60	CF3M	6\24	6\60	N10	N18	N	N	N	N	Microaneurysms and hard exudates seen in all quadrants	Both eyes moderate NPDR	Focal hyperfluorescence noted in the late arteriovenous phase due to leakage	Both eyes focal diabetic maculopathy		Yes	Focal diabetic maculopathy	Nil
12	BHIMAVVA	49	F	3781252	HTN		CF1M	6\18	CF1M	6\9		N8		N	D	D	Re-retinal heamorrhages with well defined geographic areas of opcification	Be cmv retinitis ?macular edema	Both eyes focal areas of hyperfluorescence at macular area in the late phase	Both eyes CMV retinitis with macular edema	Yes		Macular edema	Nil
13	LEELA DINAKAR PATIL	51	F	3805740			6\36	CF1M	6\24	CF1M	N8	N36	N	N	N	D	Left eye Round circular ring reflex seen at the macula with neurosensory detachment	Left eye CSCR	Left eye Early hyperfluorescent spot progressing in a ink blot patter	Left eye ink blot type of CSCR	Yes		Ink blot type of cscr	Nil
14	MUGATSAB PEERAPUR	78	M	3595571		Smoking	CF4M	CF4M	CF4M	CF4M	N18	N18	N	N	D	D	Both eyes localized sub retinal fluid with heamorrhages at the foveal area	Both eyes ?wet armd	Both eyes hyperfluorescence noted in the early arteriovenous phase in a lacy pattern which increased in intensity in late phase	Both eyes classic cnvm		Yes	Classic cnvm	Nil
15	YASHODHA B	70	F	3813104	HTN & Hypercholesterolemia		6\18	6\24	6\12	6\18	N12	N8	N	N	N	D	Left eye Dilated and tortous vessels ,collaterals and haemorrhages seen in the superotemporal quadrant	Left eye st brvo ?macular edema	Left eye Capillary non perfusion areas noted delayed venouis filling	Le st-brvo with macular ischamia		Yes	Macular ischamia	Nausea
16	SHAKUNTALA NAIK	62	F	3818348	HTN		6\18	6\18	6\12	6\9	N10	N10	N	N	N	N	Soft drusens with RPE changes are seen	Both eyes dry armd	Hyperfluorescence at the macular area only iin the late phase of ffa noted	Both eyes dry armd	Yes		Both eyes dry armd	Nil
17	MAHADEVI	55	F	332674	HTN		6\24	6\36	6\6	6\24	N6	N12	N	N	N	D	Dilated and tortous vessels ,collaterals and haemorrhages seen in the superotemporal quadrant	Le superotemporal brvo ?maculae edema	Delayed venouis filling with focal areas of hyperfluorescence in the in the macular area in early phase which increased in intensity in late phase	Left eye st-brvo with macular edema	Yes		macular edema	Nil
18	SADASHIV KAMBLE	22	M	3156362			CF2M	6\6	CF2M	6\6	N36	N6	N	N	D	N	Chorioretinal atrophic patch with elevated granular area at the macula	Re choroiditis with ?macular edema	Hyperfluorescence due to leakage of the dye only in late phase noted	Re choroiditis with macular edema	Yes		macular edema	
19	VIRUPAXI SANGATE	75	M	3256580	HTN	Smoking	6\12	6\24	6\6	6\18	N6	N12	N	N	N	D	Dilated and tortous vessels ,collaterals and haemorrhages seen in the superotemporal quadrant	Le st-brvo ?macular edema	Delayed venouis filling with areas of capillary non perfusion are noted	Le st-brvo with macular ischamia		Yes	macular ischaemia	Nil
20	NANDEPPA M	57	M	720760	DM		6\9	6\18	6\9	6\12	N8	N8	N	N	N	N	Microaneyrysms and exudates are seen at the macular area	Re mild npdr	Focal areas of hyperfluorescence due to leakage at the late phase seen	Right eye focal diabetic maculopathy		Yes	Focal maculopathy	Nil
21	GANGUBAI M	73	F	3845245	DM		6\9	6\12	6\9	6\12	N8	N8	N	N	N	N	Left eye exudates,microaneurysms and haemorrhages are seen	Left eye moderate npdr	Early hyperfluorescent spots due to leakage of the dye progressing to a characteristic petalloid pattern	Left eye diffuse diabetic maculopathy		Yes	Diffuse diabetic maculopathy	Nil
22	FATHIMA BEGAM	78	F	2630637			6\9	6\9	6\6	6\6	N6	N6	N	N	N	N	Soft drusens seen at the macula	Both eyes dry armd	Both eyes hyperfluorescence was noted at the macular area only late phase of ffa	Both eyes dry armd	Yes		Dry armd	Nil
23	CHANDRASHEKHAR	70	M	2117116	DM & HTN		6\12	6\12	6\9	6\9	N8	N8	N	N	N	N	Both eyes heamorrhages and cotton wool spots seen in all quadrants exudates surrounding the macula are seen	Both eyes moderate NPDR with CSME	Both eyes hyperfluorescent areas were noted at macula in early phase which increased in intensity in late phase	Both eyes moderate NPDR with CSME	Yes		CSME	
24	PANDURANG R	68	M	3765087	DM & HTN	Smoking	6\60	CF3M	6\24	6\60	N10	N12	N	N	N	N	Right eye haemorrhages and cotton wool spots are noted,exudates surrounding the macula are seen	Right eye mild npdr with csme	Re-scattered areas of hyperfluorescence noted in early phase increased in intensity in late phase	Re mild npdr with csme	Yes		CSME	Nil
25	NILAVVA R GUDIMANI	65	F	3904051	DM		6\24	CF3M	6\12	CF3M	N8	N18	N	N	N	N	Left eye heamorrhages and cotton wool spots seen in all quadrants	Left eye severe npdr	Re-scattered areas of hyperfluorescence noted in early phase increased in intensity in late phase	Left eye diffuse diabetic maculopathy		Yes	Diffuse diabetic maculopathy	Nil
26	BASAVANTAPPA	60	M	3412566	HTN	Smoking	6\18	6\12	6\12	6\9	N8	N8	N	N	N	N	Confluent drusens are seen at the macular area	Both eyes dry armd	Both eyes hyperfluorescence was noted at the macular area only late phase of ffa	Both eyes dry armd	Yes		Dry armd	Nil
27	SHRENIK KAGE	55	M	388374	DM	Smoking	6\12	6\12	6\9	6\9	N8	N8	N	N	N	N	Both eyes heamorrhages and cotton wool spots seen in all quadrants	Both eyes moderate npdr	Both eyes hyperfluorescent areas were noted at macula in early phase which increased in intensity in late phase	Both eyes moderate npdr with csme		Yes	CSME	Nil
28	CHINGLU BADAVANE	75	M	3895297		Smoking	6\36	6\36	6\18	6\12	N8	N8	N	N	N	N	Both eye soft drusens with RPE changes are seen	Both eyes dry armd	Both eyes hyperfluorescence was noted at the macular area only late phase of ffa	Both eyes dry armd	Yes		Dry armd	Nil
29	PREMILA K	65	F	3940368			6\12	6\24	6\9	6\18	N8	N10	N	N	N	N	Soft drusens seen at the macula	Both eyes dry armd	Both eyes hyperfluorescence was noted at the macular area only late phase of ffa	Both eyes dry armd	Yes		Dry armd	Nil
30	DEVAGOUDA PATIL	51	M	3918231			6\6	CF1M	6\6	CF1M	N6		N		N	D	Well defined,round and thickened lesion in the macula	Left eye full thickness macular hole	Window defect in early phasefollowed by hyperfluorescent halo in late phase	Left eye full thickness macular hole	Yes			Nil
31	SHARADA SOLANKI	66	F	3900358	DM		6\9	6\12	6\9	6\9	N8	N8	N	N	N	N	Re few microaneyrysms and soft exudates are seen	Re mild npdr	Both eyes hyperfluorescent areas were noted at macula in early a-v phase which increased in late phase extensively due to leakage of the dye	Right eye focal maculopathy		Yes	Focal diabetic maculopathy	Nil
32	MAHADEVAPPA	60	M	744628	DM	Smoking	6\24	6\18	6\12	6\9	N10	N8	N	N	N	N	Both eyes dot and blot haemorrhages and cotton wool spots are present	Both eyes severe npdr	Right eye hypofluorescence in the late a-v phase with capillary non perfusion areas are noted enlarged faz noted in the right eye	Right eye ischamic maculopathy		Yes	Ischeaimic maculopathy	Nil
33	VIJAY	39	M	3950032	Type A Personality		6\6	6\24	6\6	6\12	N10	N8	N	N	N	D	Round circular ring reflex seen at the macula with neurosensory detachment	Left eye CSCR	Early phase hyperfluorescent spot at the macula is seen which increased in fluorescence like a ink blot	Left eye ink blot type of cscr	Yes		Ink blot type of cscr	Nil
34	FRANCIS MAHURE	51	M	3851357	DM & HTN		6\9	6\60	6\9	6\60	N8	N18	N	N	N	N	Le microaneyrysms and soft exudates are seen in all quadrants	Le moderate npdr	Left eye hypofluorescence in the late a-v phase with capillary non perfusion areas are noted enlarged faz noted in the right eye	Left eye ischaemic maculopathy		Yes	Ischeaimic maculopathy	Nausea
35	MANIK PATIL	80	M	3954033	HTN	Smoking	6\60	6\36	6\36	6\24	N18	N18	N	N	N	N	Multiple soft drusens are seen at the macular area	Both eyes dry armd	Both eyes hyperfluorescence was noted at the macular area only late phase of ffa	Both eyes dry armd	Yes		Dry armd	Nil
36	KULDEEP SHER SINGH	39	M	3980517	Occupational Stress		6\9	6\60	6\9	6\36	N8	N12	N	N	N	D	Round circular ring reflex seen at the macula with neurosensory detachment	Left eye CSCR	Early phase hyperfluorescent spot at the macula is seen which increased in fluorescence like a ink blot	Left eye ink blot type of cscr	Yes		Ink blot type of cscr	Nil

S.NO	NAME	AGE	SEX	IP/OP NO.	SYSTEMIC DISEASES RISK FACTOR		VA	VA	PH	PH	NV	NV	CV	CV	AMSLER	AMSLER	FUNDUS FINDINGS(Direct,Indirect ,90D)	DIAGNOSIS SUSPECTED	FFA INTERPRETATION	FFA DIAGNOSIS	DIAGNOSIS CONFIRMED BY FFA	DIAGNOSIS ALTERED BY FFA	CLASSIFICATION/ CATEGORIZATIO N BY FFA	REACTION TO FLUORESCEI N
37	NARENDRA JADHAV	63	M	3971344	DM & HTN		6\36	6\24	6\24	6\18	N18	N12	N	N	N	N	Both eyes few microaneyrysms and soft exudates are seen at the macular area	Both eyes mild npdr with csme	Tiny hyperfluorescent dots were noted in early phase which increased in intensity in late phase	Both eyes mild npdr with csme	Yes		CSME	Nil
38	KISHORE RAKSHE	50	M	3996705	DM		6\24	6\18	6\12	6\12	N10	N8	N	N	N	N	Both eyes dot and blot haemorrhages and cotton wool spots are present in all quadrants	Both eyes severe npdr	Both eyes hypofluorescence in the late a-v phase with capillary non perfusion areas are noted enlarged faz noted in the right eye	Right eye ischaemic diabetic maculopathy		Yes	Ischeaimic maculopathy	Nil
39	SUMAN MUGAL	62	F	3991907			6\60	6\24	6\36	6\24	N18	N10	N	N	N	N	Both eye soft drusens with rpe changes are seen	Both eyes dry armd	Both eyes hyperfluorescence was noted at the macular area only late phase of ffa	Both eyes dry armd	Yes		Dry armd	Nil
40	YALLAPPA KAMBLE	55	M	3885671	HTN		6\24	6\24	6\18	6\18	N12	N12					Both eyes round,elevated subfoveal lesion is seen about 1/2 disc diameter	Both eyes ?Macular dystrophy	Small irregular hyperfluorescent ring noted in both eyes	Adult onset macular vitelliform dystrophy	Yes			Nil
41	NEMU MANNUR	80	M	3917402	DM	Smoking	6\24	6\60	6\18	6\36	N10	N36	N		N	D	Le soft drusens with elevated areas	? Le cnvm	Pathcy hyperfluorescence noted in early phase with late staining at the macula	Le classic cnvm	Yes		CNVM	Nil
42	VASANT K	66	M	4046444	DM		6\18	6\24	6\12	6\18	N8	N8	N	N	N	N	Both eye soft drusens are seen	Both eyes dry armd	Both eyes hyperfluorescence was noted at the macular area only late phase of ffa	Both eyes dry armd	Yes		Dry armd	Nil
43	SHOBHATAI KATE	65	F	4053162	DM		6\24	6\36	6\18	6\24	N12	N12	N	N	D	D	Both eyes localized sub retinal fluid with heamorrhages at the foveal area	Both eyes? wet armd	Both eyes hyperfluorescence noted in the early arteriovenous phase in a lacy pattern which increased in intensity in late phase	Both eyes classis cnvm		Yes	Classic cnvm	Nil
44	MARIYAMMA NAIDU	60	F	3471950	DM		6\60	6\36	6\36	6\24	N36	N18			D	D	Right eye exudates presentwith dot blot haemorrhages in all quadrants	Right eye severe npdr	Right eye hyperfluorescent areas were noted at macula in early a-v phase which increased in late phase extensively	Right eye diffuse diabetic maculopathy		Yes	Diffuse diabetic maculopathy	Nil
45	MALLAPPA KUDRI	50	M	4051725	HTN		6\60	6\18	6\36	6\12	N12	N8	N	N	D	N	Dilated and tortous vessels ,collaterals and haemorrhages seen in the superotemporal quadrant	Right eye superotemporal brvo ?macular edema	Macular ischaemia capillary non perfusion areas noted in early phase delayed venouis filling are noted	RE ST-BRVO with macular ischaemia		Yes	Macular ischaemia	Nil
46	RAMAPPA B	75	M	4055227	DM		6\24	6\18	6\18	6\12	N12	N10	N	N	N	N	Both eyes heamorrhages and cotton wool spots seen in all quadrants exudates surrounding the macula are seen	Both eyes moderate NPDR with CSME	Both eyes hyperfluorescent areas were noted at macular area in early a-v phase,which increased in intensity in late phase with IRMA noted	Both eyes moderate npdr with csme	Yes		CSME	Nil
47	SUNITA BISTE	39	F	4022682			6\24	6\36	6\9	6\36	N10	N18	N	N	N	D	Round circular ring reflex seen at the macula with neurosensory detachment	Left eye cscr	Tiny hyperfluorescent spot at the macula which increased in intensity in a smoke stack pattern	Left eye smoke stack type of cscr	Yes		Smoke stack type	Nil
48	MAHAVEER	61	M	4090322	HTN		6\12	6\24	6\9	6\18	N8	N10	N	N	N	D	Dilated and tortous vessels ,collaterals and haemorrhages seen in the superotemporal quadrant	Left eye superotemporal brvo ?macular edema	Capillary non perfusion areas noted in early phase delayed venouis filling with focal areas of hyperfluorescence in the macula at the in the the late phase noted	Left eye st-brvo with macular edema	Yes		Macular edema	Nil
49	SHANKRAYYA U	70	M	4091998	DM		CF1M	6\18		6\12		N12		N	D	N	Right eye exudates and microaneurysms seen in all quadrants and ring of hard exudates seen temporal to the macula	Right eye ?diabetic maculopathy	Multiple hyperfluorescent spots at the early phase which increased in intensity in late phase in a petalloid pattern	Right eye diffuse diabetic maculopathy	Yes		Diffuse diabetic maculopathy	Nil
50	NARAYAN PATIL	45	M	4094873		Type A Personality	6\6	6\12	6\6	6\12	N6	N12	N	N	N	D	Round circular ring reflex seen at the macula with neurosensory detachment	Left eye cscr	Tiny hyperfluorescent spot at the macula which increased in intensity in a smoke stack pattern	Left eye smoke stack type of cscr	Yes		Smoke stack type	Nil
51	SUNDRABAI PATIL	60	F	4120235	DM		6\18	6\60	6\12	6\36	N10	N18	N	N	N	N	Both eyes heamorrhages and cotton wool spots seen in all quadrants exudates surrounding the macula are seen	Both eyes moderate npdr with csme	Both eyes hyperfluorescent areas were noted at macular area in early a-v phase,which increased in intensity in late phase with irma noted	Both eyes moderate npdr with csme	Yes		CSME	Nil
52	RADHA MORABAD	50	F	4033024	DM		6\36	6\9	6\24	6\6	N18	N6	N	N	N	N	Right eye-microaneurysms and few exudates are seen	Right rye mild npdr	Tiny hyperfluorescent dots were noted in early phase which increased in intensity in late phase	Right eye focal diabetic maculopathy		Yes	Focal diabetic maculopathy	Nil
53	BABUSAHEB PATIL	40	M	4176897			6\6	6\60	6\6	6\60	N6		N	N	N	D	Round circular ring reflex seen at the macula with neurosensory detachment	Left eye cscr	Early phase hyperfluorescent spot at the macula is seen which increased in fluorescence like a ink blot	Left eye ink blot type of cscr	Yes		Ink blot type of csr	Nil
54	RAJENDRA H	48	M	416872		Smoking	6\18	6\9	6\12	6\6	N12	N6	N	N	D	N	Round circular ring reflex seen at the macula with neurosensory detachment	Right eye cscr	Tiny hyperfluorescent spot at the macula which increased in intensity in a smoke stack pattern	Right eye smoke stack type of cscr	Yes		Smoke stack type	Nil
55	ISHWAR BAGOJI	70	M	4186988		Smoking	6\60	6\60	6\18	6\18	N18	N10	N	N	D	N	Confluent drusens at the macula	Right eye dry armd	Lacy hyperfluorescence in the arteriovenous phase which increased in late phase	Right eye occult cnvm		Yes	Occult cnvm	Nil
56	FAKIRAPPA	70	M	779192	DM		6\24	6\60	6\9	6\36	N10	N18	N	N	N	D	Yellowish lesion at the macula with drusens	Left eye armd	Left eye hyperfluorescence noted in the early arteriovenous phase increased in intensity in late phase with subfoveal neovascularisation	Left eye occult cnvm		Yes	Occult cnvm	Nil
57	MALLIKARJUN	54	M	649052	DM	Smoking	6\9	6\36	6\6	6\36	N6	N18	N	N	N	D	Yellowish lesion at the macula with subfoveal neovascularisation	Left eye wet armd	Lacy hyperfluorescence in the arteriovenous phase which increased in late phase	Left eye occult cnvm		Yes	Occult cnvm	Nil
58	MEHROON M PATHAN	65	M	4224687	DM	Smoking	6\18	6\36	6\12	6\18	N10	N12	N	N	N	N	Both eyes few microaneyrysms and soft exudates are seen	Both eyes mild npdr	Tiny hyperfluorescent dots were noted in early phase which increased in intensity in late phase	Both eyes mild npdr with focal diabetic maculopathy		Yes	Focal diabetic maculopathy	Nil
59	LEELA SUNTAKAR	32	F	3354107			6\6	CF1M	6\6	CF1M	N6		N		N	D	Round circular ring reflex seen at the macula with neurosensory detachment	Left eye cscr	Early phase hyperfluorescent spot at the macula is seen which increased in fluorescence like a ink blot pattern	Left eye ink blot type of cscr	Yes		Ink blot type	Nil
60	MEHABOOBSAB S	50	M	4186736	HTN		6\6	CF3M	6\6	CF3M	N6		N		N	D	Left dilated and tortous vessels ,collaterals and haemorrhages seen in the superotemporal quadrant	Left eye superotemporal brvo ?macular edema	Capillary non perfusion areas noted in early phase delayed venouis filling with focal areas of hyperfluorescence in the macula in a petalloid pattern in the the late phase noted	Left eye st-brvo with cystoid macular edema	Yes		Macular edema	Nil
61	CHANNABASAPPA N	60	M	420857	DM		CF3M	6\18	CF3M	6\9	N36	N8		N	N	N	Both eyes dot blot haemorrhages seen in all quadrants with irma is seen	Both eyes severe npdr	Both eyes scattered areas of hyperfluorescence noted in early phase increased in intensity in late phase	Both eyes severe npdr with diffuse diabetic maculopathy		Yes	Diffuse diabetic maculopathy	Nil
62	SHOBHA SHRINGERI	62	F	3746567	HTN		6\24	6\9	6\18	6\6	N8	N6	N	N	N	N	soft drusens are seen at the macular area	Both eyes dry armd	Both eyes hyperfluorescence was noted at the macular area only late phase of ffa	Both eyes dry armd	Yes		Dry armd	Nil
63	MARUTI GHADI	80	M	4284068	HTN	Smoking	CF2M	CF2M	6\24	6\60	N18	N12	N	N	D	D	Yellowish lesion at the macula with subfoveal neovascularisation	Both eyes wet armd	Both eyes hyperfluorescence noted in the early phase increased in intensity in late phase with subfoveal neovascularisation	Both eyes occult cnvm		Yes	Occult cnvm	Nil
64	KALIMULLA J	36	M	4288318		Smoking	6\12	6\6	6\9	6\12	N8	N6	N	N	D	N	Round circular ring reflex seen at the macula with neurosensory detachment	Right eye cscr	Early phase hyperfluorescent spot at the macula is seen which increased in fluorescence like a ink blot pattern	Right eye ink blot type of cscr	Yes		Ink blot type	Nil
65	SOMAPPA KORI	60	M	4297507	DM	smoking	6\36	6\36	6\18	6\24	N12	N12	N	N	D	D	Both eyes dot blot haemorrhages seen in all quadrants with irma is seen	Both eyes severe npdr with csme	Both eyes hyperfluorescent areas were noted at macular area in early a-v phase,which increased in intensity in late phase with irma noted	Both eyes severe npdr with csme	Yes		CSME	Nil
66	FIRDOUS J	35	M	4320078			CF4M	CF1M	6\60	CF3M	N36	N36					Beaten bronze appearance at the macular area	? Macular dystrophy	Hyperfluorescence due to window deffect with dark choroid	Be macular dystrophy Stargadt's disease	Yes			

ANNEXURE-V

KEY TO MASTER CHART

S.No	–	Serial number
VA	–	Visual Acuity
PH	–	Pin Hole
CF	–	Counting Fingers
CV	–	Colour Vision
NV	–	Near Vision
N	–	Normal
D	–	Distorted
CNVM	–	Choroidal Neovascular Membrane
DM	–	Diabetes Mellitus
HTN	–	Hypertension
RPE	–	Retinal Pigment Epithelium
NPDR	–	Non Proliferative Diabetic Retinopathy
BRVO	–	Branch Retinal Vein Occlusion
CRVO	–	Central Retinal Vein Occlusion
ARMD	–	Age Related Macular Degeneration
CSCR	–	Central Serous Chorio Retinopathy