# UNDERSTANDING AND INTERPRETATION OF FUNDUS FLUORESCEIN ANGIOGRAM

**INTRODUCTION:** 

Fundus Fluorescein Angiography(FFA) is one of the most informative investigative procedures to evaluate & assess selected choroidal, retinal, optic nerve head(ONH), macular & vascular abnormalities. It is extremely valuable for expanding our knowledge of the anatomy, pathology & pathophysiology of the retina & choroid.

#### **BASIC PRINCIPLES**

Luminescence is the emission of light from any source other than high temperature. Luminescence occurs when energy in the form of electromagnetic radiation is absorbed & then re-emitted at another frequency. It always entails a shift from shorter wavelength to a longer wavelength.

Fluorescence is luminescence that is maintained only by continuous excitation.

# PROPERTIES OF FLUORESCEIN SODIUM

- Fluorescein sodium (C20H1005Na) is a highly fluorescent chemical compound synthesized from the petroleum derivatives resorcinol & Phthalic anhydride.
- It absorbs blue light, with peak absorption & excitation occurring at wavelengths between 465-490 nm.
- □ Fluorescence occurs at the yellow-green wavelengths of 520 to 530 nm.(figure 1)
- **I**t diffuses through choriocapillaris, but not normal

Dr. G.Nageswar Rao, MD Asst. Prof. Ophthalmology, KIMS Dr. Gayatri Kanungo, MS Consultant Ophthalmologist, KIDS

retinal vascular endothelium or intact retinal pigment epithelium.

- 80% of injected dye binds to proteins, leaving 20%
  unbound dye available for fluorescence.
- □ The retina is illuminated with 465-490mm wavelength through the use of exciter filters.
- □ Fluorescein absorbs this wavelength & re-emits wave length of 520-530 mm.
- □ Barrier filters are used in the camera so that only the re-emitted wavelength of 520-530mm exposes the film.(figure 2)
- □ The dye is metabolized by the kidneys & is eliminated through urine within 24 to 36 hours of administration.

#### **THE PROCEDURES :**

#### **Preparation of the Patient**

Patient should preferably be on empty stomach for 3-4 hours before initiating the procedure.

The patient's pupils are dilated fully with 2% tropicamide.

An informed consent should be signed by the patient highlighting the following:

- **Explain the procedure in detail**
- Describe Possible risks & their probability
- Possible staining of conjunctiva & dark colouration of urine.
- In patients of diabetes, false positive urine glucose reduction test.

#### Photographic technique

- **D** The patient is seated in front of the fundus camera.
- Colour & red free control photographs are taken prior to injection.
- 500mg of dye (5ml. of 10% solution or 2ml of 25% solution) is injected into the antecubital vein ( second choice: vein at back of hand) over 2-4 sec..
- Photographs are taken at approximately 1-second intervals, for 5-25 seconds after injection.
- Late stage photos are taken 5 to 15 mins later.
- NaFl typically reaches the central retinal artery 10-15second after injection. This ranges from 5 to 30 sec. depending on cardiac disease, cardiac output, blood viscocity, vascular occlusion, hypertension, renal disease, vessel caliber etc.

#### **ADVERSE REACTIONS**

Mild	Moderate	Severe
Transient nausea	Extravasation of dye	Bronchospasm
Vomitting	Syncope	Laryngeal Edema
Flushing of the skin	Thrombophlebitis	Anaphylaxis
Itching	Transient N.Palsy	Myocardial infarction
Urticaria		

# PHASES OF THE ANGIOGRAM

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.

# THE NORMAL ANGIOGRAM

 Baseline photos (Autofluorescence/ Pseudofluorescence) are taken.

- □ Transition time / Arm to Retina time- The time it takes fluorescein to reach central retinal artery after injection (5-10 sec.).
- Choroidal (Pre-arterial Phase): It occurs 10 seconds after dye injection & is characterized by patchy filling of the choroids-typically described as a ground-glass appearance.
- Retinal arterial phase shows arterial filling & continuation of choridal filling (10-12 sec after injection).(figure 3)
- □ The arteriovenous (Capillary phase) shows complete filling of the arteries & capillaries & early flow in veins(13 sec. after injection.)
- Early venous phase (laminar phase) 1-2 seconds after arterial phase- the veins have a laminar appearance The Central Lumen remains dark while the wall fluoresce(14-15 sec).(figure 4). This is because the fluorescein from the venules enters the veins along their walls and also vascular flow is faster in the center of a lumen( tube) than on the sides, the fluorescein seems to stick to the sides, creating the laminar pattern of retinal venous flow.
- Mild venous phasedisplays almost complete venous filling(16-17 sec.).(figure 5)
- □ Late venous phase shows complete venous filling within reducing concentration of dye in the arteries(18-20 sec.).(figure 6)
- Peak phase of Angiogram- Macular phase when the foveal capillaries are well filled(25-30 seconds).
- □ The late (elimination) phase displays choroidal pooling & scleral staining (5min).
- □ Macular hypofluorescence: It is due to
- □ Avascularity of foveal avascular zone.

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Block of background choroidal fluorescence due to xanthophyll pigments & retinal pigment epithelium(RPE)

cells at the fovea.

# **THEABNORMAL FLUORESCEIN ANGIOGRAM**

# **HYPERFLUORESCENCE**

Leakage of the dye (Microaneurysm, Intraretinal microvascular abnormalities, Capillary leakage, neovascularisaton).(figure 7,8,9,10)

> In the subretinal space (CSR).(figure 11,12)

Pooling of the dye 

In the sub-RPE space (Pigment epithelial detachment).

Transmited hyperfluorescence(window defect) due to RPE atrophy.

Staining of tissues as a result of prolonged retention of dye (drusen, sclera & ONH).

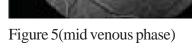
# **HYPOFLUORESCENCE:**

- Blockage of retinal fluorescence
- Vitreous opacities & preretinal lesion such as blood.(figure 13,14)
- Deep retinal lesions intraretinal haemorrhage \_ and hard exudates.
- Blockage of choroidal fluorescence
- Sub retinal /sub RPE lesion such as blood \_ (figure 15,16)
- Increased density of RPE -Congenital hypertrophy of RPE
- Choroidal leisions such as naevi.
- Filling Defects:
- Vascular Occulusion (figure 17,18)
- Loss of vascular bed (Myopic degeneration/ choriodermia).

# Figure 1 Figure 3 Figure 2 Excitation & emission of fluorescene Photographic principles of FA (Choroidal & arterial phase) for the second Blue faits Rive starting (the Conversition's Solid Tokos-press light Centers camers ( 600 Weveleigh - new



Figure 4 (laminar phase)



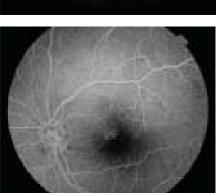


Figure 6(late venous phase)

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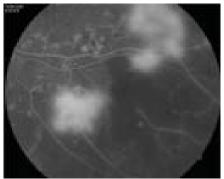


Figure 10 Hyperfluorescence due to leaking new vessels in proliferative diabetic retinopathy



Figure 13

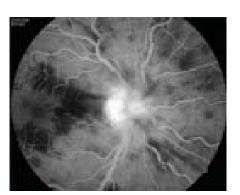


Figure 16 Blockage of background choroidal fluorescence due to the subretinal blood

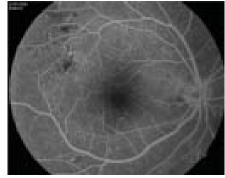


Figure 8 Hyperfluorescence due to leakage of micro-aneurisms

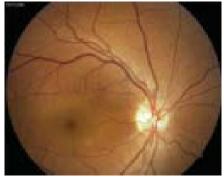


Figure 11

Figure 17

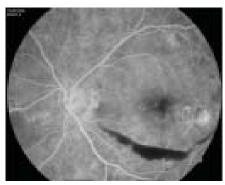


Figure 14 Hypofluorescence( blocked fluorescence) due to pre-retinal haemorrhage

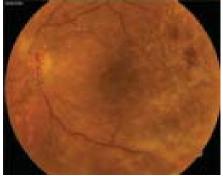


Figure 9



Figure 12 Hyperfluorescence due to Pooling Of the dye into subretinal space in CSR

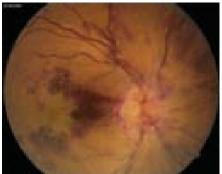


Figure 15

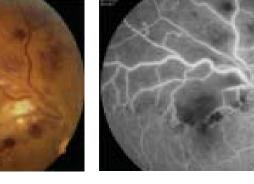


Figure 18 Filling defect due to vascular occlusion causing hypofluorescence

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