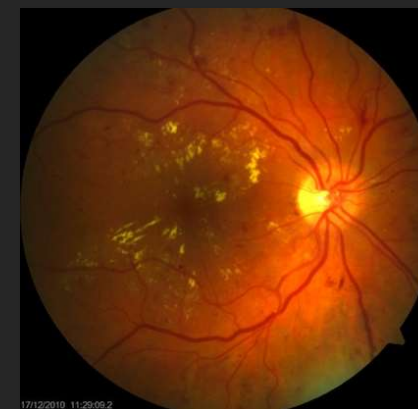
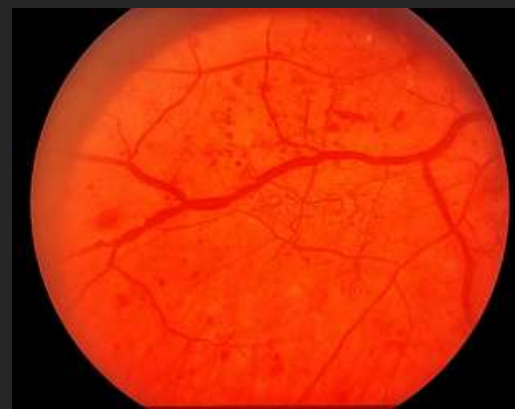
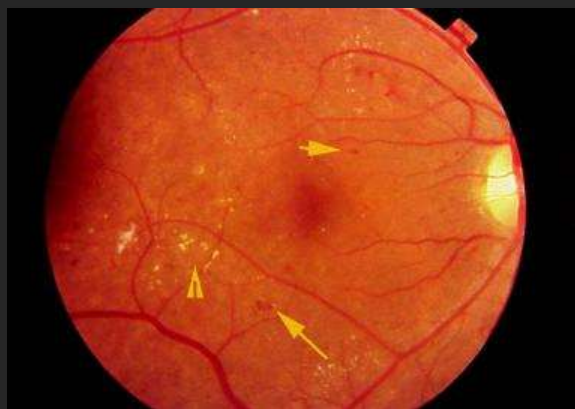


Diabetic Retinopathy (DR)



- In developed countries, one of most common causes of blindness in 30 - 65 years old
- Prevalence of DR **increases with the duration of diabetes**
- **After 20 years** of disease, almost all Type 1 diabetes and the majority of Type 2 diabetic patients have some degree of DR

Pathogenesis

Hyperglycemia

↑ Retinal
Blood Flow

Disruption of metabolism in
Endothelial cells & Pericytes

Impaired vascular
Autoregulation

↑ Vasoactive substance
production &
Endothelial cell proliferation

Capillary - reduced lumen
& hypoperfusion

Chronic retinal ischemia

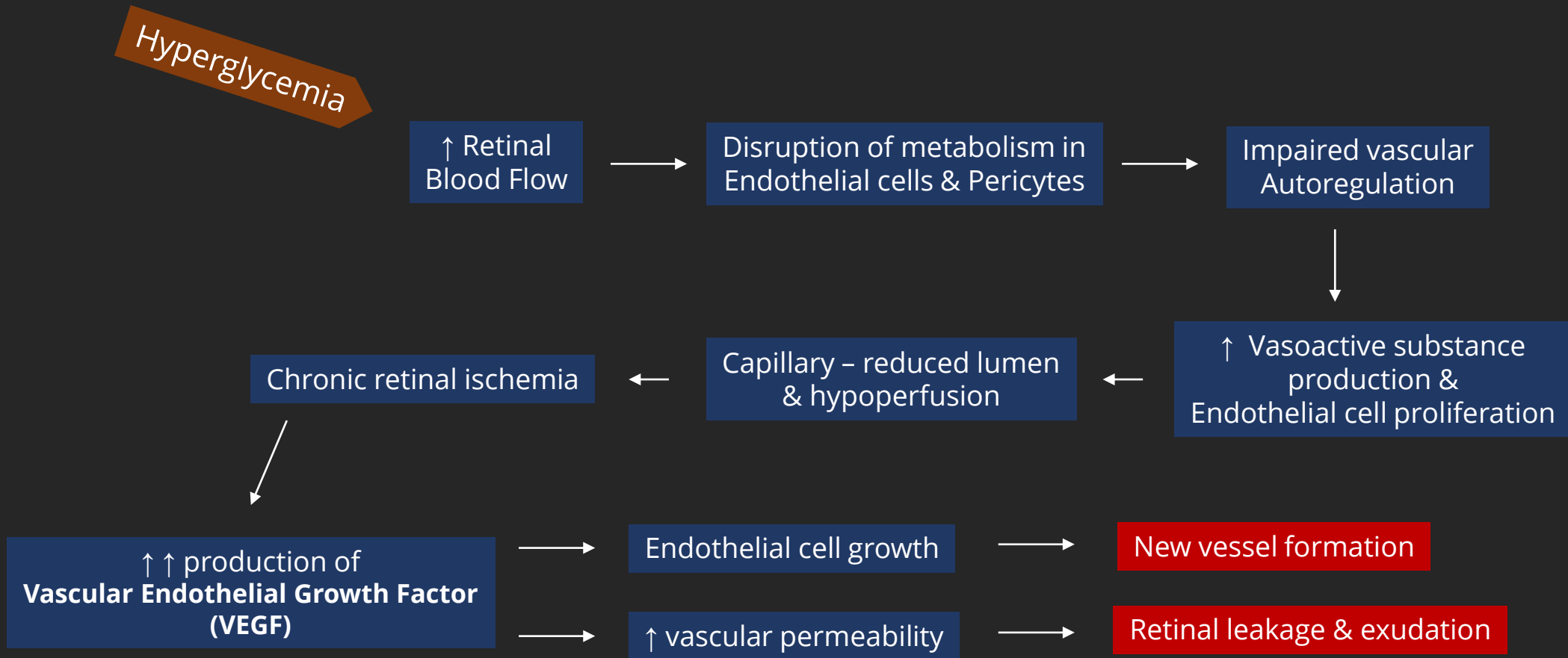
↑ ↑ production of
**Vascular Endothelial Growth Factor
(VEGF)**

Endothelial cell growth

New vessel formation

↑ vascular permeability

Retinal leakage & exudation



Stages

Non-proliferative DR

- Background DR
- Pre-proliferative DR

Proliferative DR

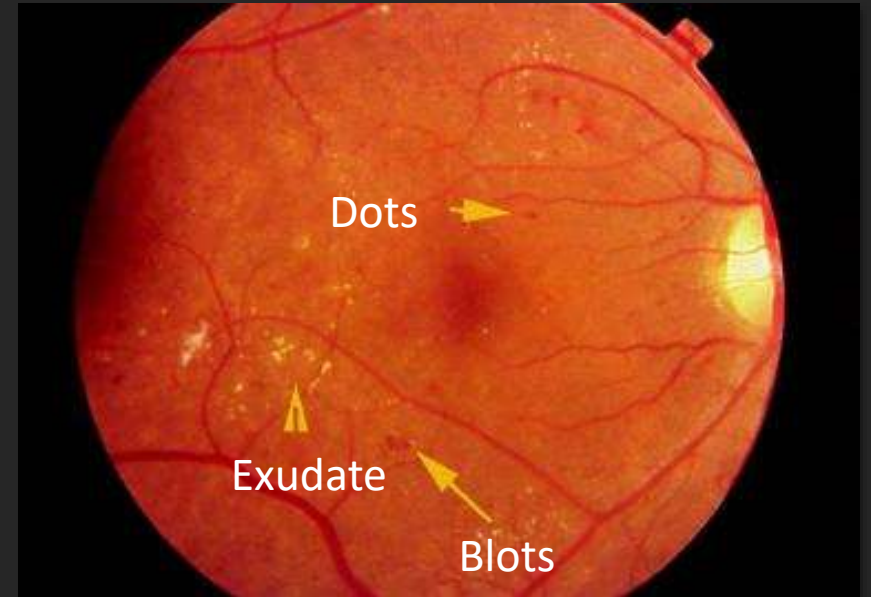
Maculopathy

Background Retinopathy

'Dots' – *Microaneurysms* appearing as dots. Usually the earliest clinical abnormality

'Blot' hemorrhages – Rupture of microaneurysm

'Hard exudates' – Capillary leakage & resultant lipid deposition

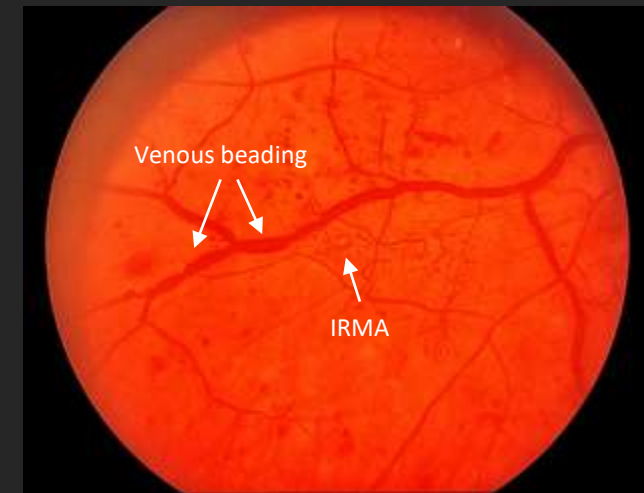
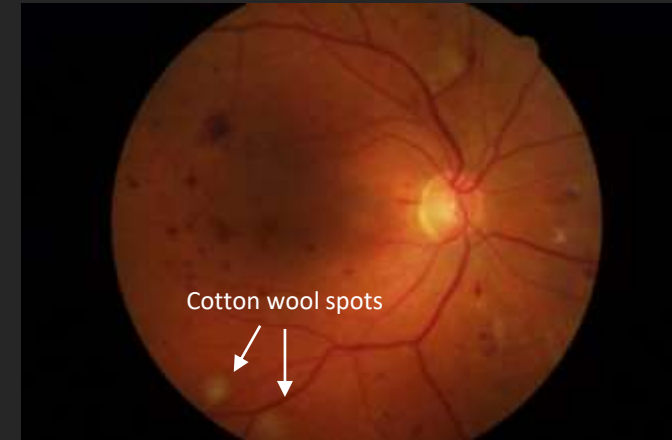


Pre-proliferative Retinopathy

'Cotton-wool' spots – *appears due to retinal ischemia*

'Venous beading' – appears as Sausage shape

'Intra-retinal Microvascular Abnormalitis' (IRMA) – Spidery vessels, often with sharp corners that indicates dilatations of pre-existing capillaries



Proliferative Retinopathy

New vessel formation or '**Neovascularization**' (NV)

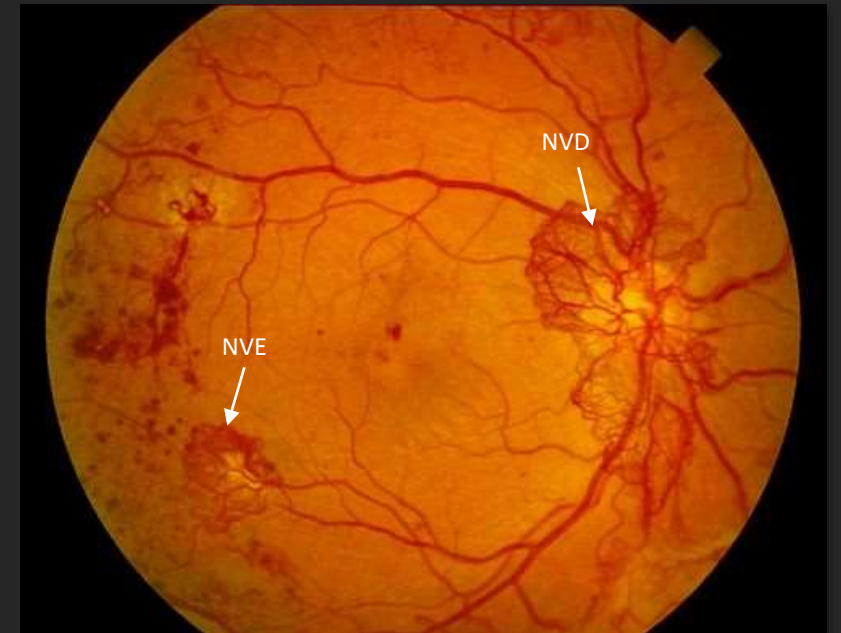
NVD – if new vessels appear on optic disc

NVE – if it appears Elsewhere on retina

Initially, fine vessels form on surface of retina & later extend forward on the posterior surface of vitreous

Serous product leak from these vessels stimulates a connective tissue reaction with gliosis & fibrosis – putting traction pull on retina

'Rubeosis iridis' → Secondary Glaucoma



Maculopathy

Not a separate group in classification

Maculopathy or Clinically Significant Macular Oedema (CSMO) occur when hard exudates form on macula associated with oedema

It causes decreased visual acuity & is sight threatening



Loss of visual acuity

Visual Impairment

No significant effect if involvement is in peripheral retina

Suspect CSMO, if changes found near macula and accompanying loss of visual acuity

Macular oedema can cause impairment of visual acuity even if there is only mild peripheral non-proliferative retinopathy and no other obvious pathology

Vision Loss in DR

Occurs due either to *Vitreous haemorrhage* or *Retinal detachment*

Important

In pre-proliferative and proliferative retinopathy, whether or not visual acuity is impaired, prompt laser treatment is important to reduce the risk of haemorrhage, fibrosis/gliosis and severe irreversible visual impairment

Screening of DR

- Annual screening for retinopathy in all diabetic patients, as the disease is asymptomatic in the early stages, when treatment is most effective.
- Preferred method - *Digital photographic system for retinal imaging*
- Refer patients with sight-threatening retinopathy to ophthalmologist for examination with *Slit lamp biomicroscopy*
- If direct ophthalmoscopy is used, the pupils should be dilated for adequate examination

Prevention of DR

- Control Diabetes
 - **Transient deterioration of retinopathy** may occur due to loss of hyperglycaemia-induced hyperperfusion in the retinal circulation and a consequent increase in ischaemia
 - This effect wears off within 18 months
 - Improvement in glycaemic control should therefore be gradual in patients with retinopathy, particularly when glycaemic control is initially poor
- Control Blood Pressure - < 130/80 mmHg
- Hyperlipidemia treatment ?

Management of DR

- a. Metabolic Control
- b. Antiangiogenic Factor
- c. Retinal Photocoagulation
- d. Vitrectomy

- Glycaemic control - HbA1C around 53 mmol/mol or 7%
- Blood pressure - < 130/80 mmHg

- Antiangiogenic factors

Ranibizumab (monoclonal antibody fragment that binds to VEGF-A) is used for diabetic macular oedema

Management of DR

- a. Metabolic Control
- b. Antiangiogenic Factor
- c. Retinal Photocoagulation
- d. Vitrectomy

- **Indications**

- Severe proliferative or very severe non-proliferative retinopathy
- New vessels elsewhere with vitreous haemorrhage
- New vessels without vitreous haemorrhage in type 2 diabetes
- CSMO
- Rubeosis Iridis

Argon laser photocoagulation is the usual method

Patients should be reviewed regularly

Extensive bilateral photocoagulation may interfere with driving ability and reduce night vision

- *Rubeosis iridis* is a severe complication requiring early and extensive Pan-retinal Photocoagulation (PRP)

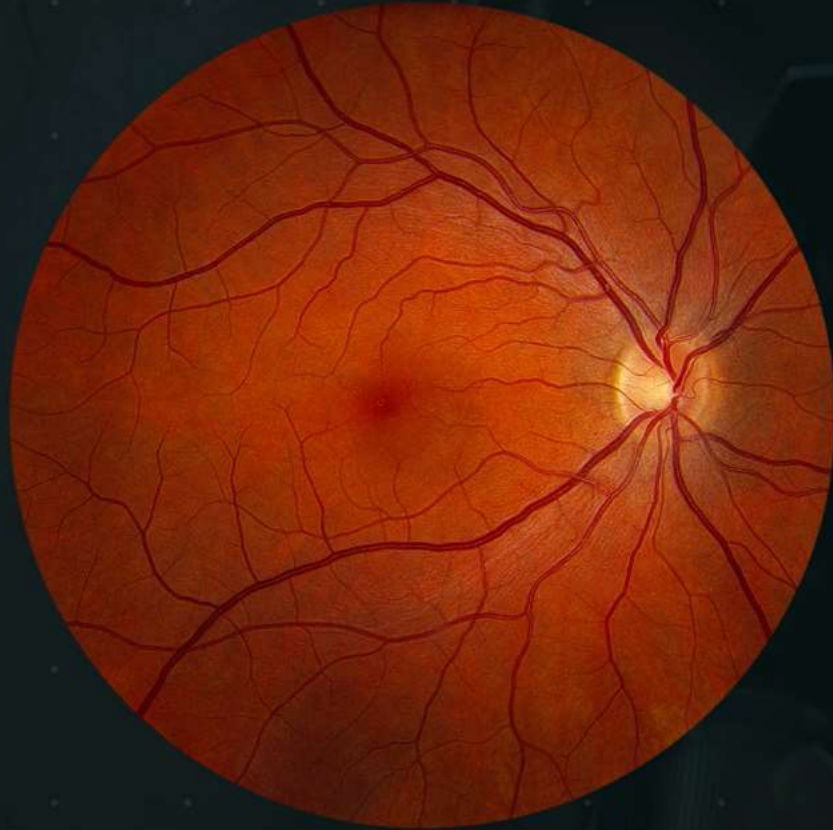
Management of DR

- a. Metabolic Control
- b. Antiangiogenic Factor
- c. Retinal Photocoagulation
- d. Vitrectomy
 - Selective cases of Type 1 diabetics with diabetic eye disease causing visual loss by *recurrent vitreous haemorrhage* that has failed to clear or by *tractional retinal detachment* threatening the macula
 - Value uncertain in Type 2 DM

Comparison Slides

Normal vs Diabetic Retinopathy

COMPARISON WITH NORMAL FUNDUS



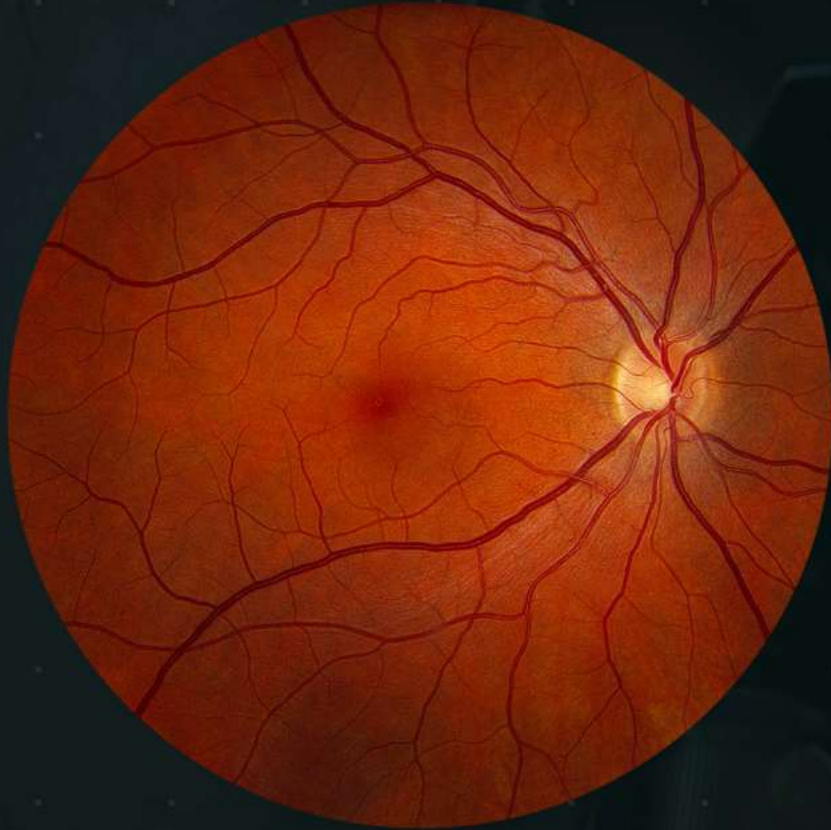
NORMAL FUNDUS



BACKGROUND RETINOPATHY

(MICROANEURYSMS, BLOT HEMORRHAGES, EXUDATES)

COMPARISON WITH NORMAL FUNDUS



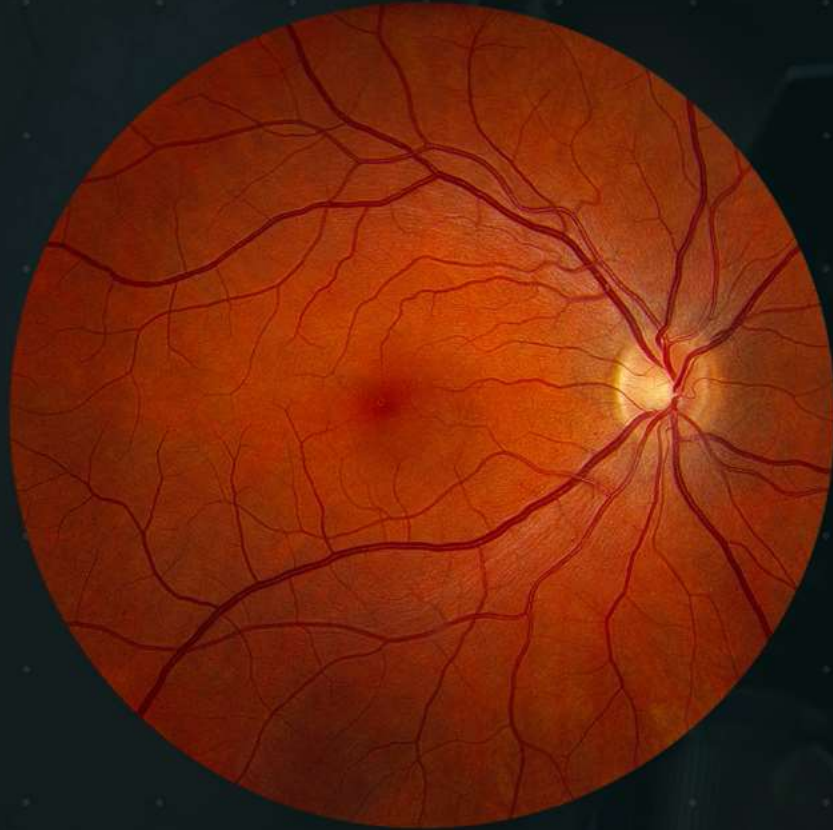
NORMAL FUNDUS



PREPROLIFERATIVE RETINOPATHY

COTTON WOOL SPOTS VISIBLE IN PERIPHERAL FIELDS
ALONGWITH MULTIPLE DOT & BLOT HEMORRHAGES

COMPARISON WITH NORMAL FUNDUS



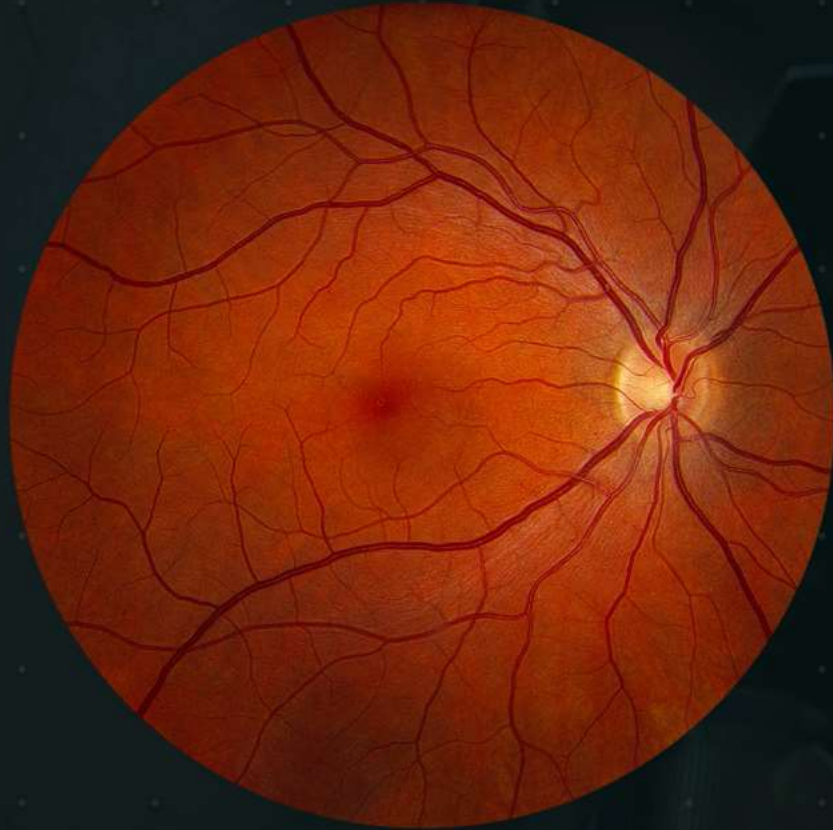
NORMAL FUNDUS



PROLIFERATIVE RETINOPATHY

NEOVASCULARISATION ON OPTIC DISC

COMPARISON WITH NORMAL FUNDUS



NORMAL FUNDUS



MACULOPATHY

HARD EXUDATES INVOLVING MACULA

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