

# RETINAL VASCULAR DISEASES

Supervised by :

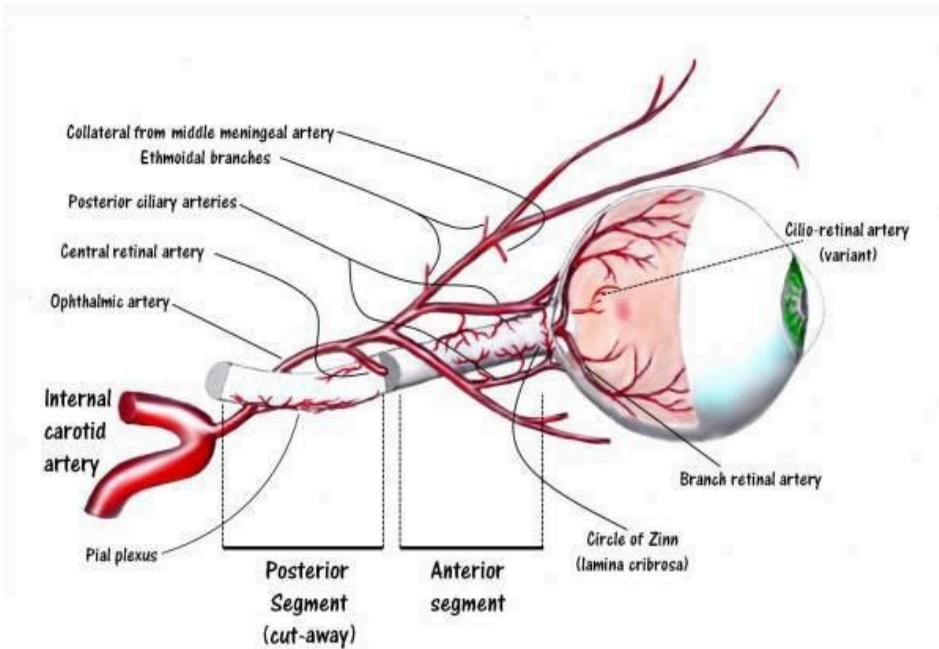
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# RETINAL VASCULAR ANATOMY



**Internal Carotid  
Artery**

**Ophthalmic Artery**

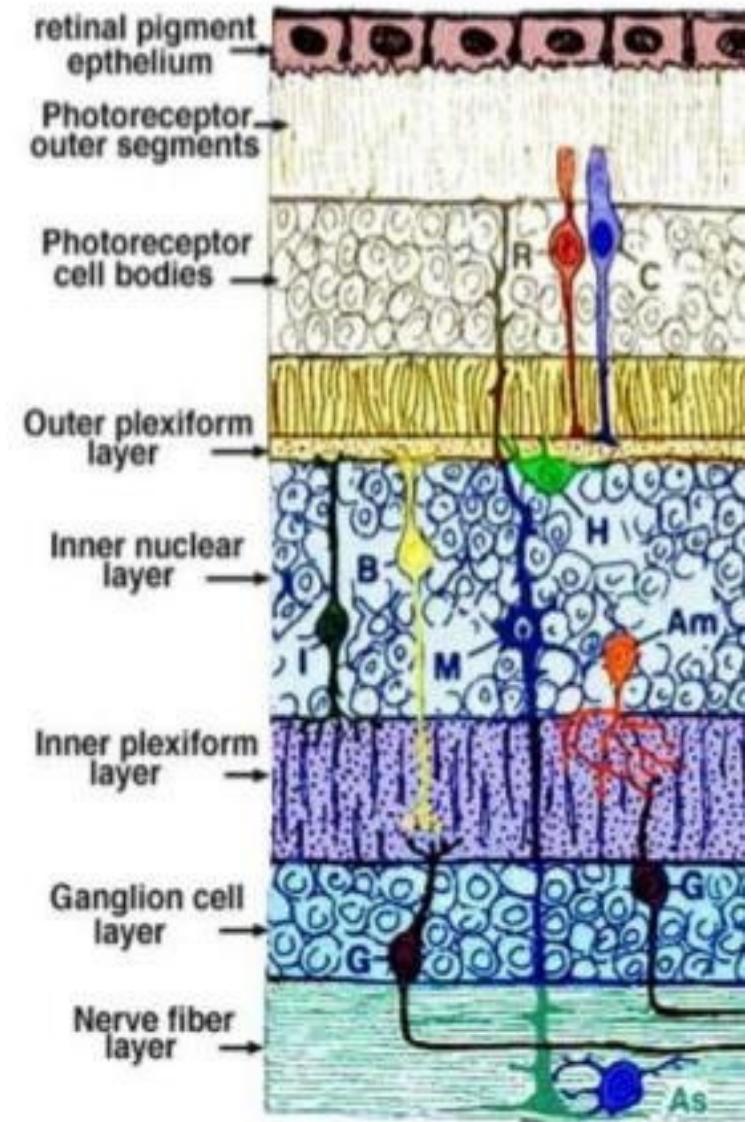
**Short and  
Long  
Posterior  
Ciliary**

**CRA**

**Muscular**

# RETINAL VASCULAR ANATOMY

- Central retinal artery (CRA) supplies the inner 2/3 of the retina through direct capillary networks (perfusion).
- Posterior ciliary artery supplies the outer 1/3 through the choroidal circulation (diffusion).



# RETINAL PATHOLOGY

**Signs of retinal vascular diseases are due to:**

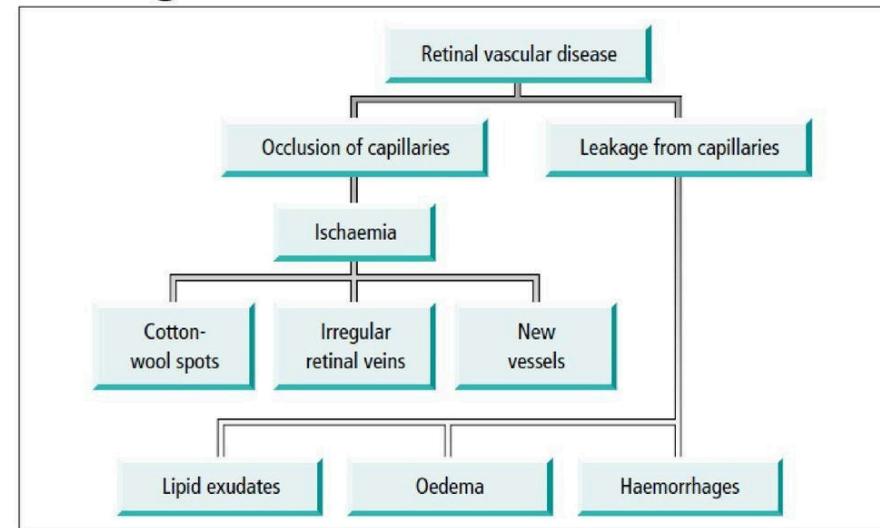
• **Leakage:**

- H e m o r r h a g e
- Edema
- Ex u d a t i o n

• **Occlusion:**

- Neovascularization
- Cotton wool spots

## Signs of retinal vascular disease

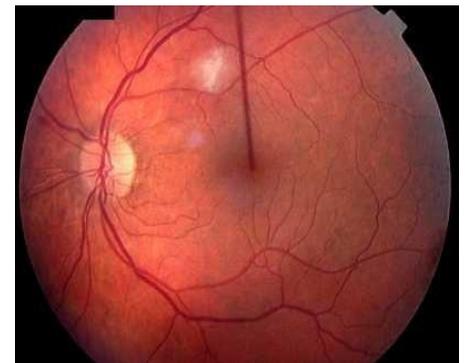


**Figure 12.1** The building blocks of retinal vascular disease. Capillary leakage and occlusion often occur together.

# OCCLUSION

## **Ischemia:**

- Build up of the axonal debris in the nerve fibre layer resulting from the reduced axoplasmic flow due to ischemia giving a cotton wool appearance.
- The histological hallmark of cotton wool spots is considered by many authors to be cytooid bodies, that of which are eosinophilic segments of ganglion cell axons that are swollen because of defective axoplasmic flow.



# OCCLUSION

## **Neovascularization:**

- Elevated levels of hypoxia-inducible factor-1 (HIF-1) stimulate the expression of vascular endothelial growth factor (VEGF), platelet-derived growth factor-B (PDGF-B), and other vasogenic factors.

# DIABETIC RETINOPATHY

**Is most common in type I (40%) than type II (20%)**

**It's the common cause of legal blindness between ages of 20 - 65.**

**Risk factors:**

**-Duration of DM**

- after 20 years- 99% of type I and 60% of type II DM have
- some form of DR
  - type I- 50% of PDR, type II-25% PDR

**-Poor metabolic control**

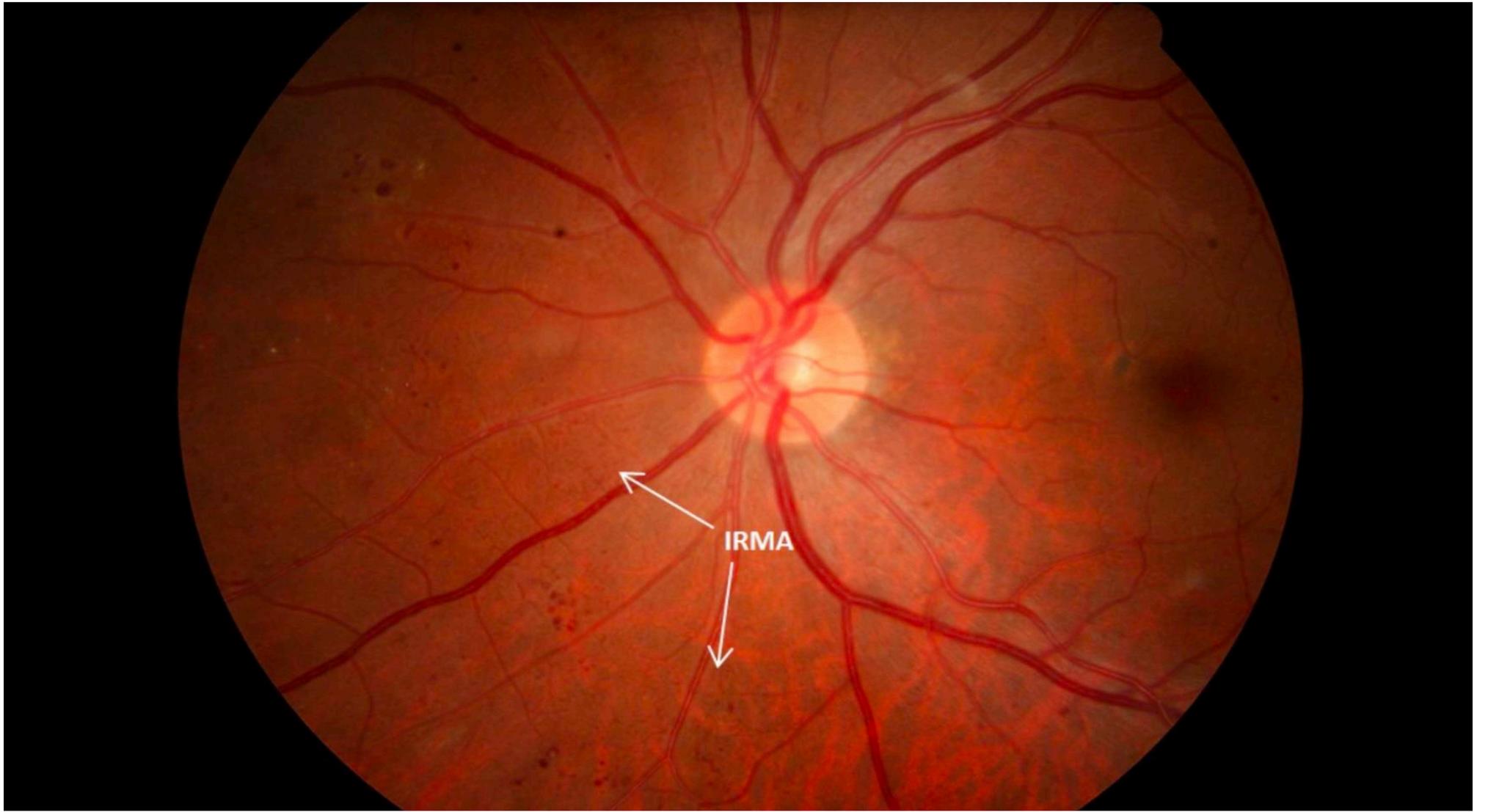
**-Pregnancy, HTN, nephropathy, obesity, hyperlipidemia and anemia**

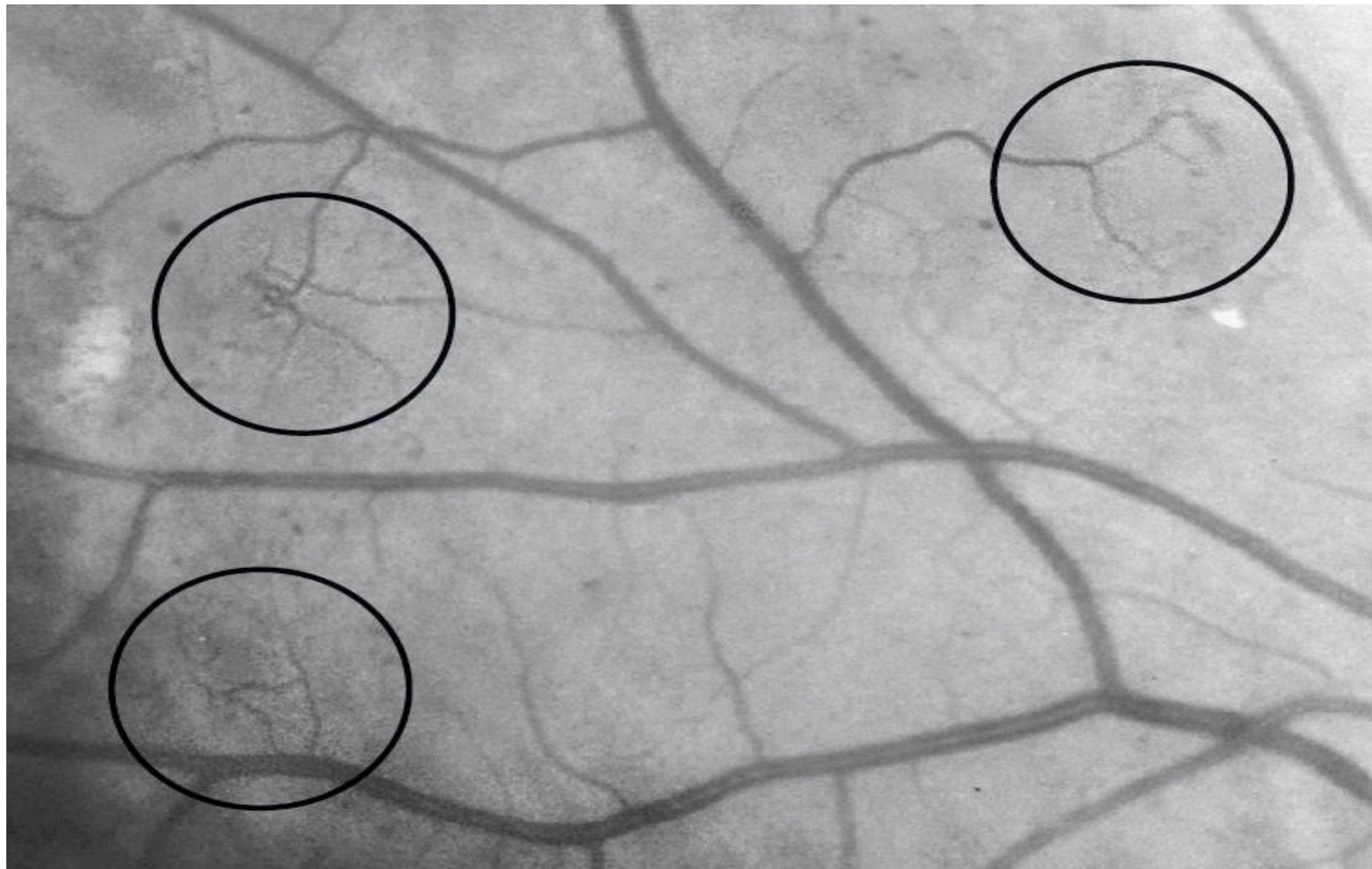
**-Smoking**

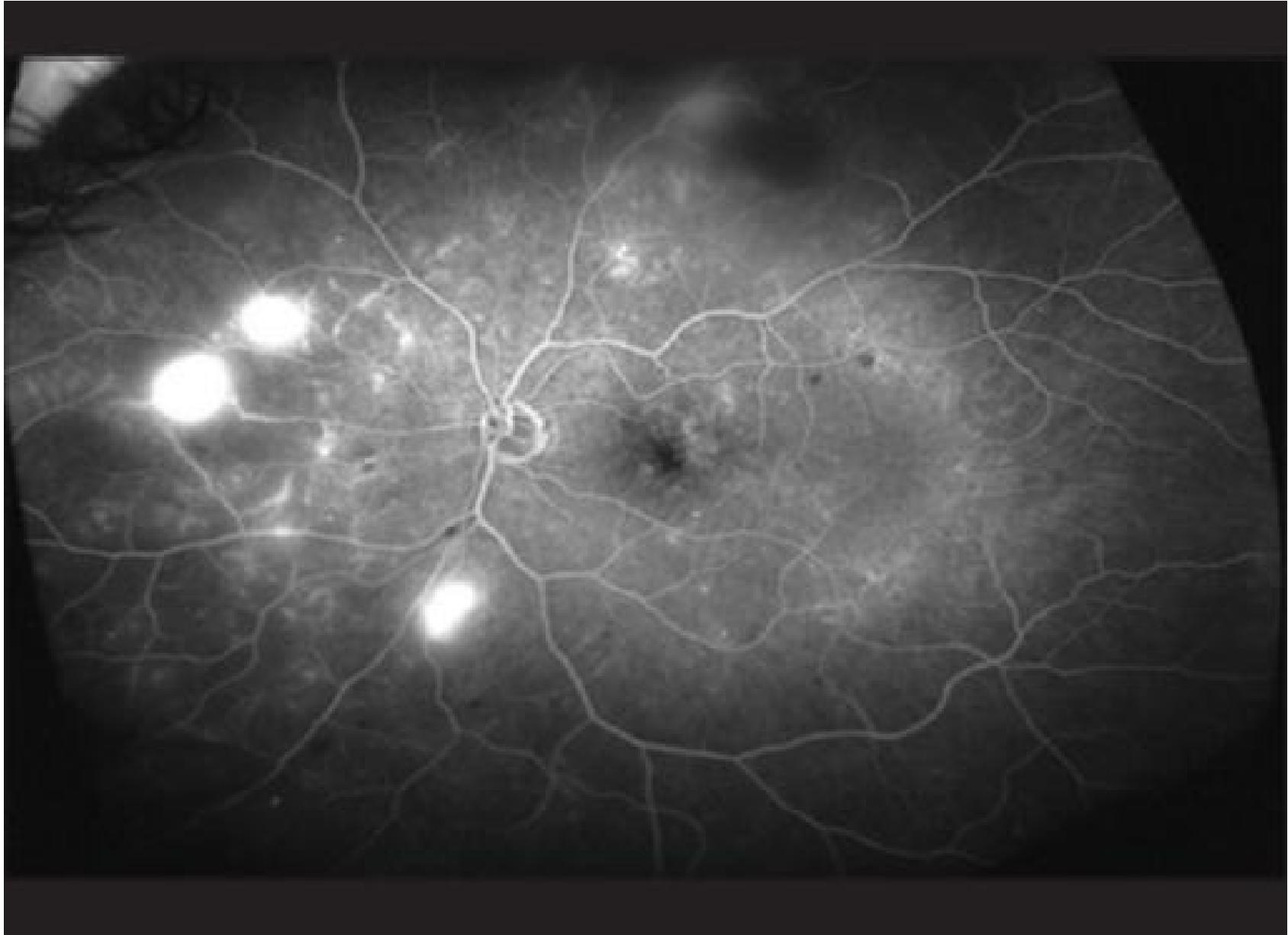
**-Family history**

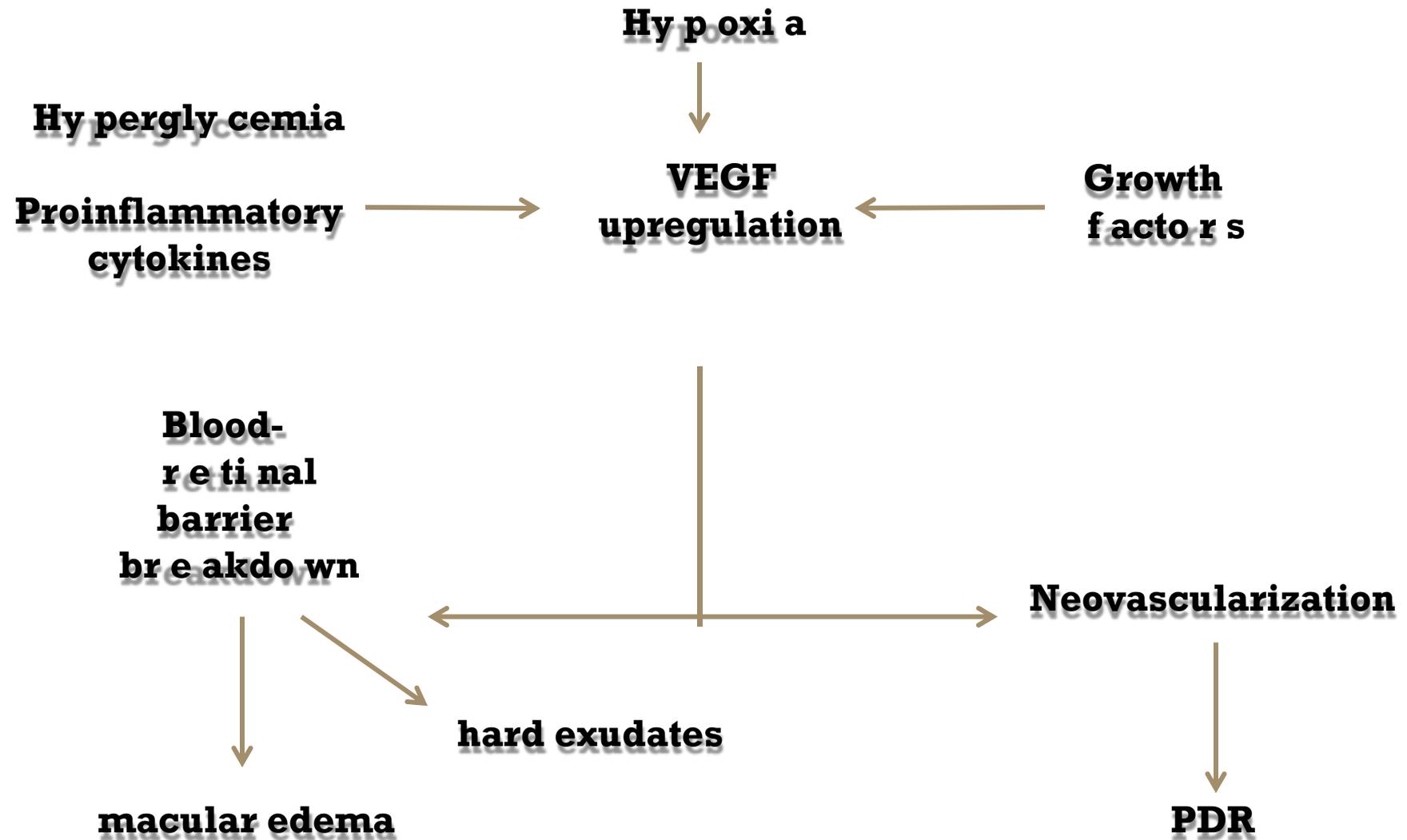
# PATHOGENESIS

- **Microangiopathy characterized by:**
  - loss of pericytes
  - proliferation of the endothelial cells
- **Microvascular occlusion results in:**
  - A-V shunts
    - IRMA (intra-retinal microvascular abnormalities)
  - Neovascularization







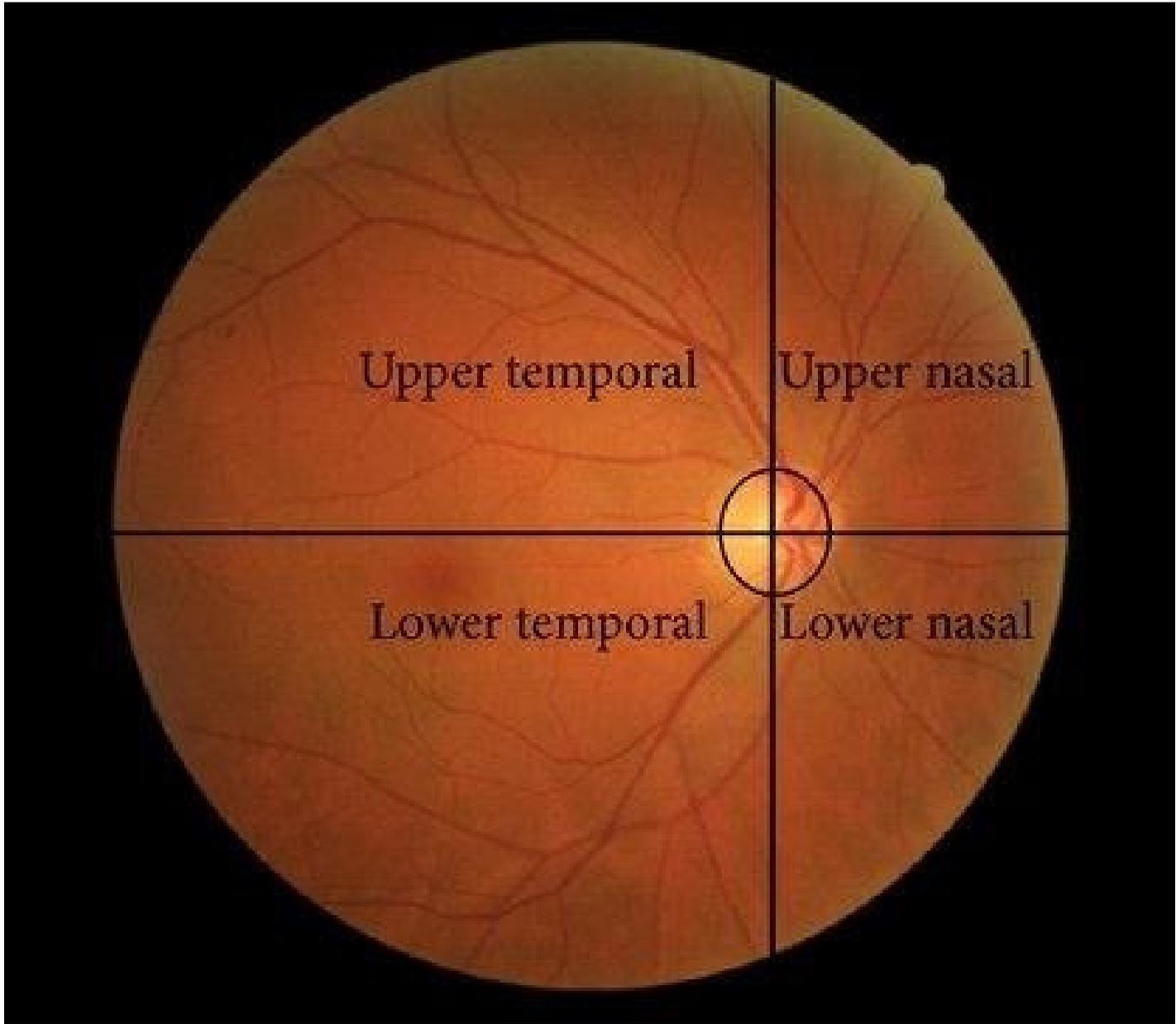


# History

Diabetic retinopathy should be diagnosed before it is symptomatic. All diabetics should have fundoscopy performed at least yearly. Screening for sight-threatening retinopathy (maculopathy and proliferative retinopathy) should begin by 5 years after diagnosis in patients with type I disease, and may be from the time of presentation in type II disease, since its time of onset is unknown. **Gradual visual loss:** in maculopathy  
**Sudden visual loss:** in vitreous hemorrhage

# CLASSIFICATION

- **Diabetes without retinopathy**
- **NPDR**
  - Mild
  - Moderate
  - Severe
- **PDR Low Risk**
  - High Risk
  -
- **Maculopathy**



Upper temporal

Upper nasal

Lower temporal

Lower nasal

# NONPROLIFERATIVE RETINOPATHY

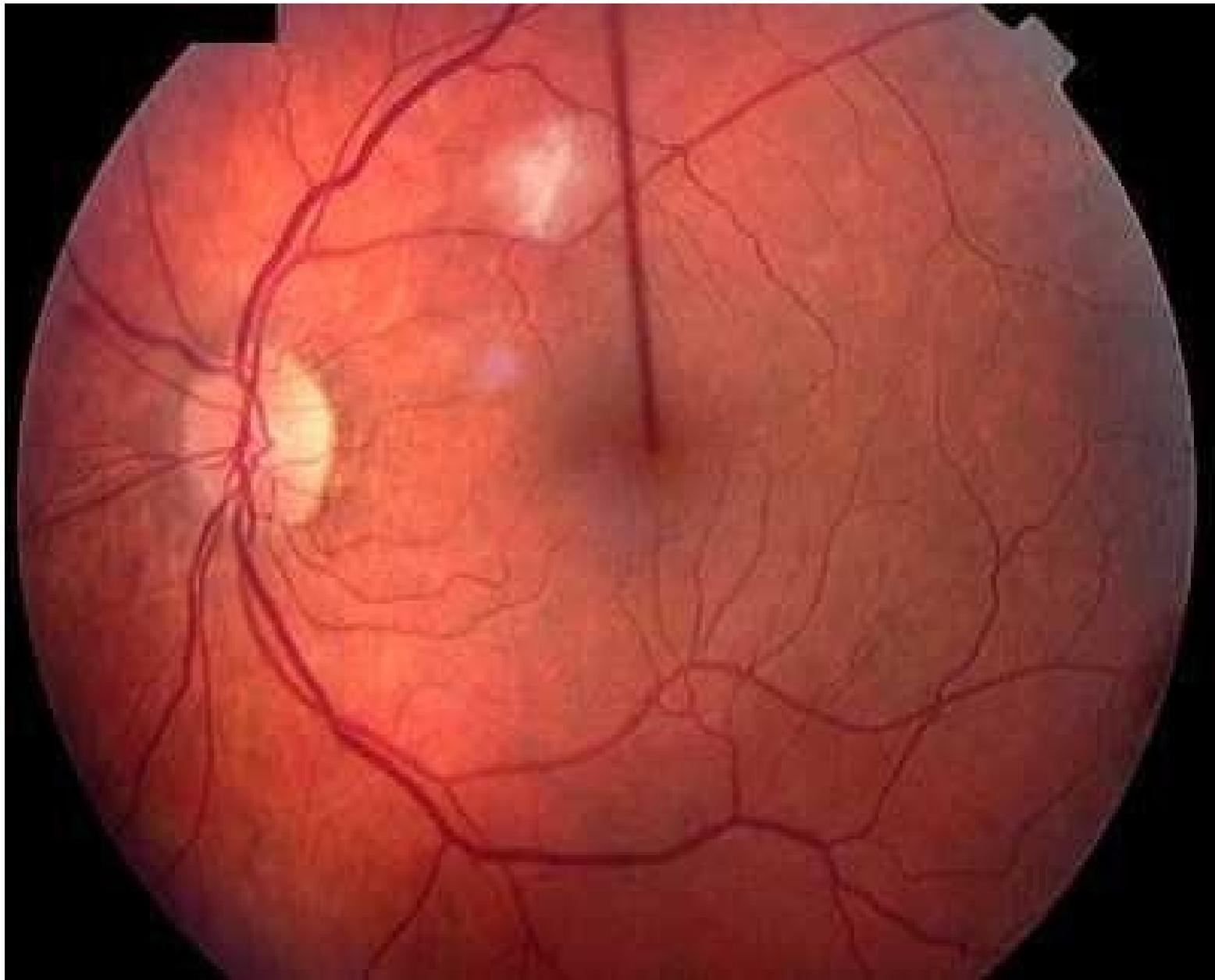
## Classification (with or without maculopathy):

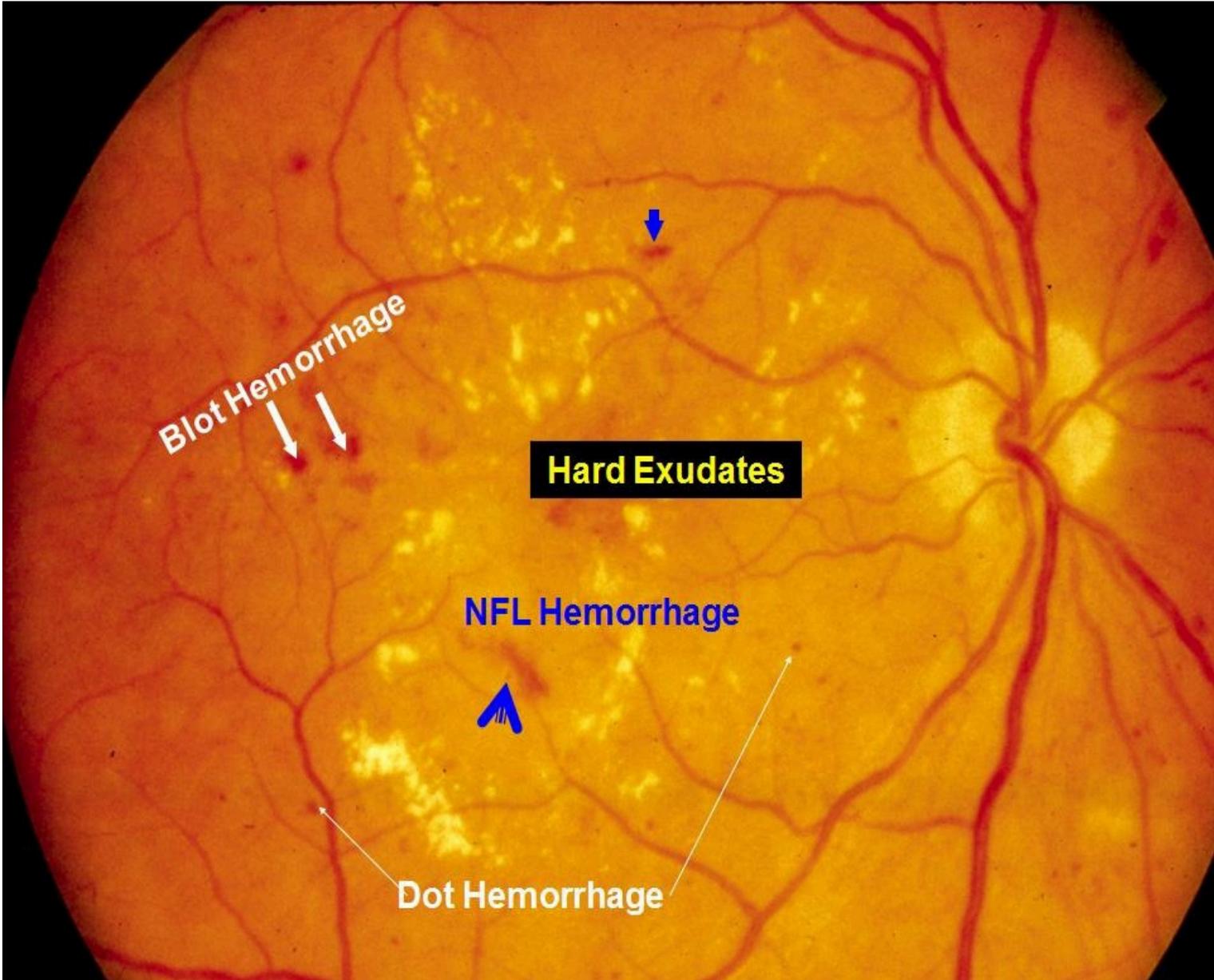
- **Mild:** MAs only
- **Moderate:** MA and DBH (<20/field)
- **Severe:** 4/2/1 rule
  - 4 quadrants of diffuse MA/DBH (>20/field)
  - 2 quadrants with venous bleeding
  - 1 IRMAs

## Present as:

- Microaneurysms
- Hard exudates
- Dot and Blot hemorrhage
- Cotton wool spots
- Venous changes
- IRMAs

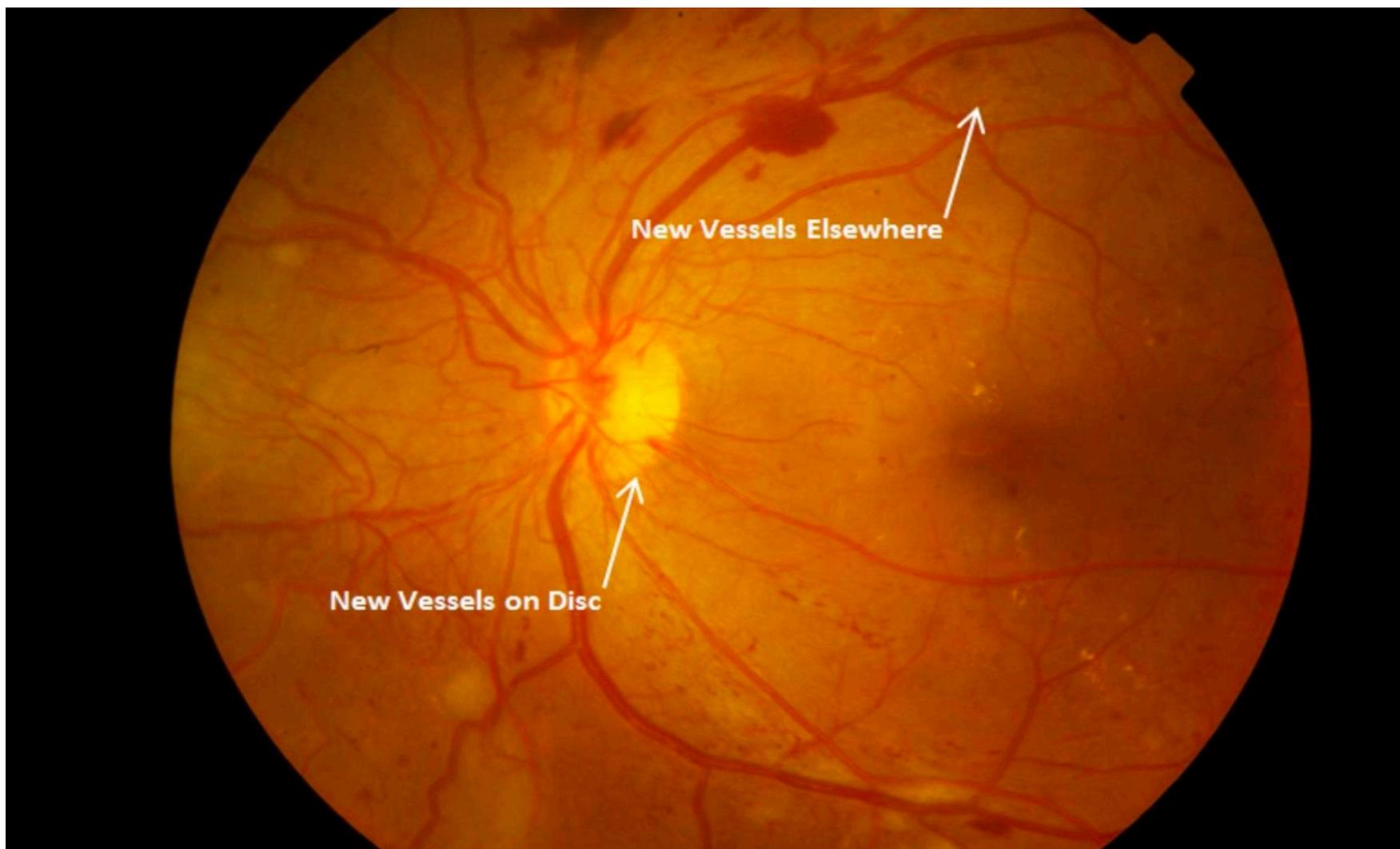






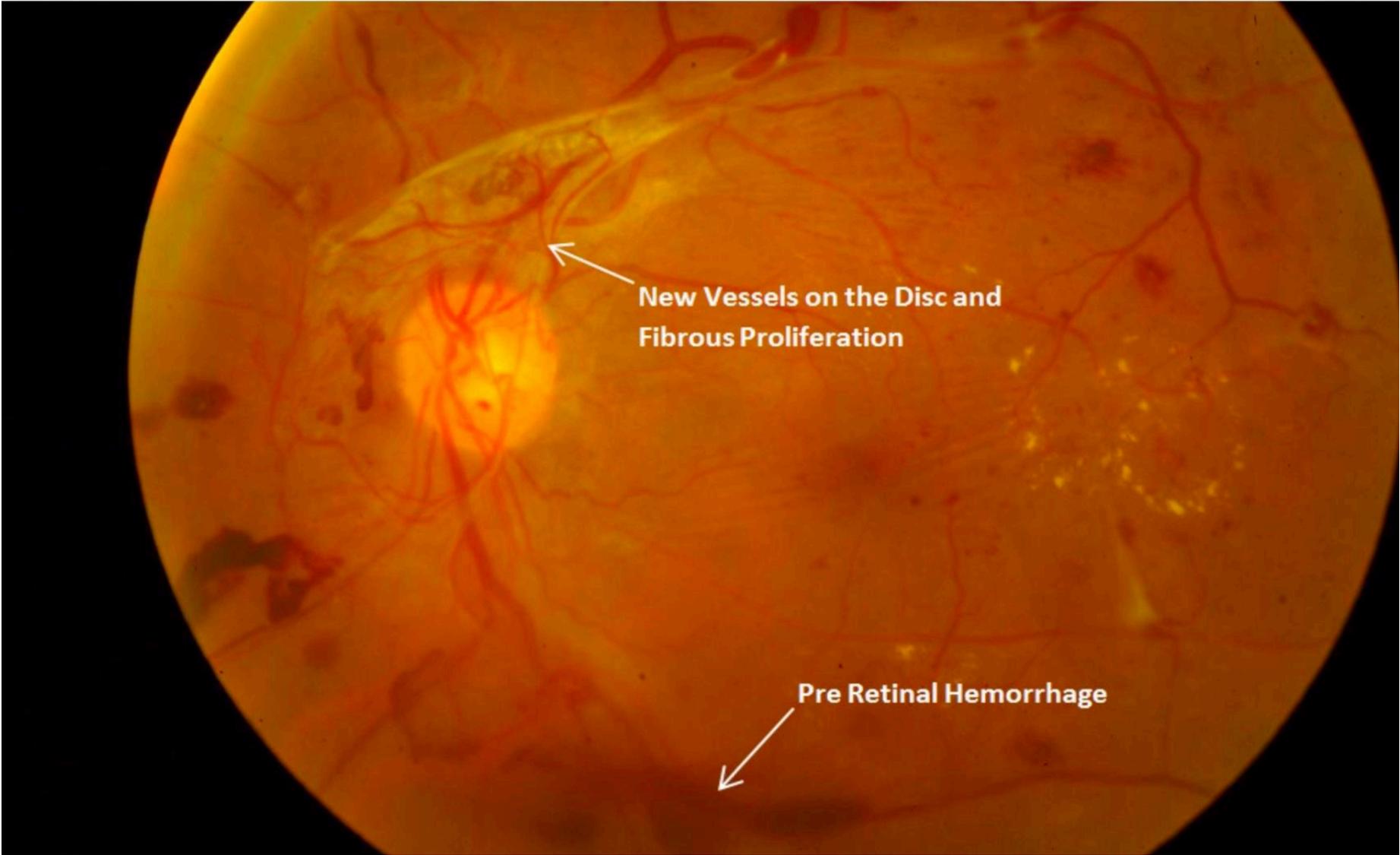
# PROLIFERATIVE RETINOPATHY

- **Neovascularization in:**
  - Disc (NVD)
  - Elsewhere (NVE)
  - Iris (NVI)
  - Angle (NVA)
- Three stages of evolution:
  1. New vessels with minimal fibrous tissue appear
  2. The new vessels increase in size and extend with an increased fibrous component
  3. Regression, leaving residual fibrovascular proliferation along the posterior hyaloid which can create traction



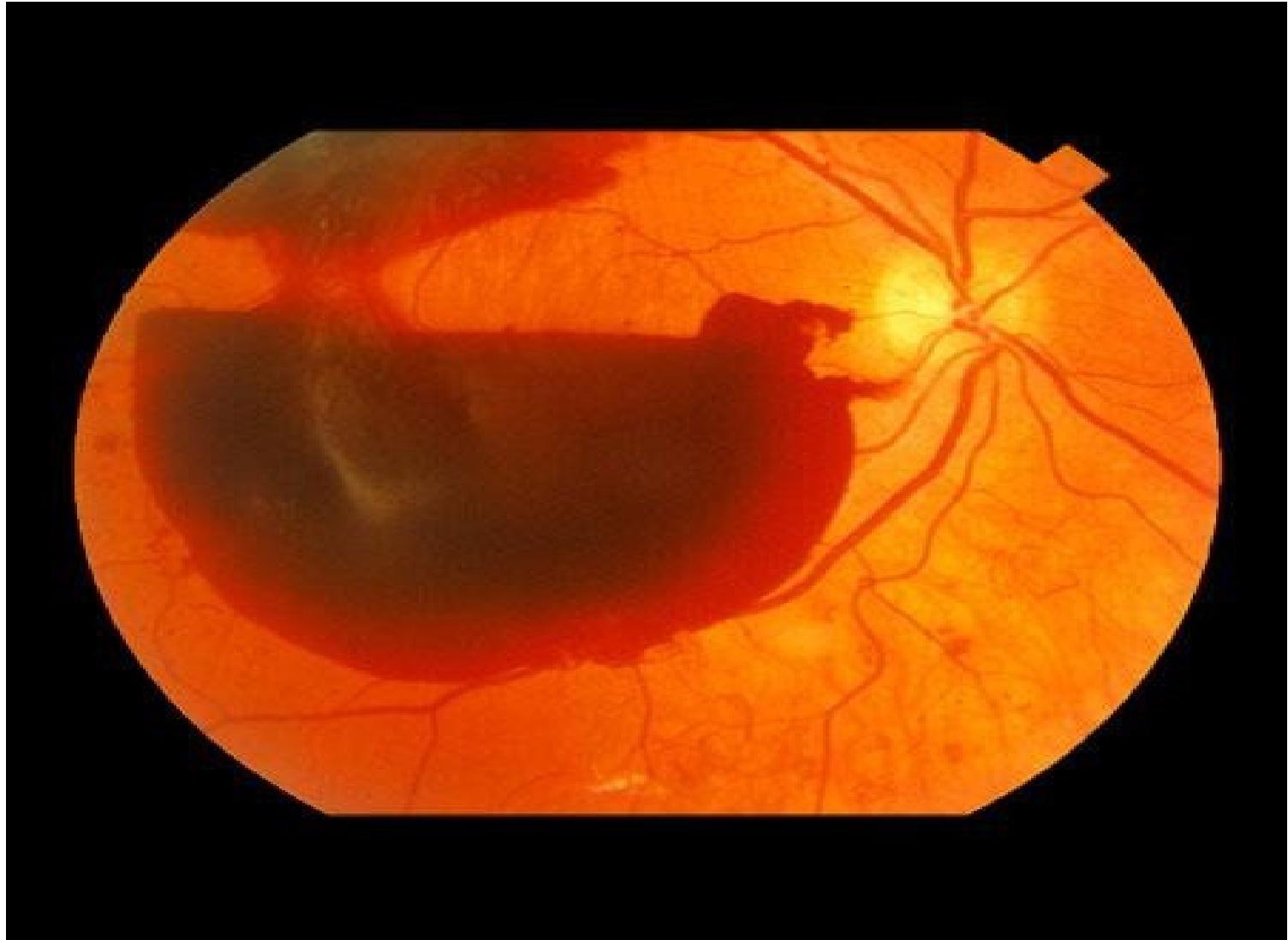
New Vessels Elsewhere

New Vessels on Disc



**New Vessels on the Disc and  
Fibrous Proliferation**

**Pre Retinal Hemorrhage**



# Proliferative Retinopathy

- **High Risk PDR:**
  - NVD >1/3 of the disc area
  - NVD + vitreous or preretinal hemorrhage
  - NVE >1/2 disc area + vitreous or preretinal hemorrhage
- **Non-high risk PDR:**
  - NVD or NVE

# T R E A T M E N T

- Fluorescein angiography could be used to assess the degree of ischemia, edema and sub-clinical diabetic changes.

**Medication:** Intravitreal anti-VEGF and

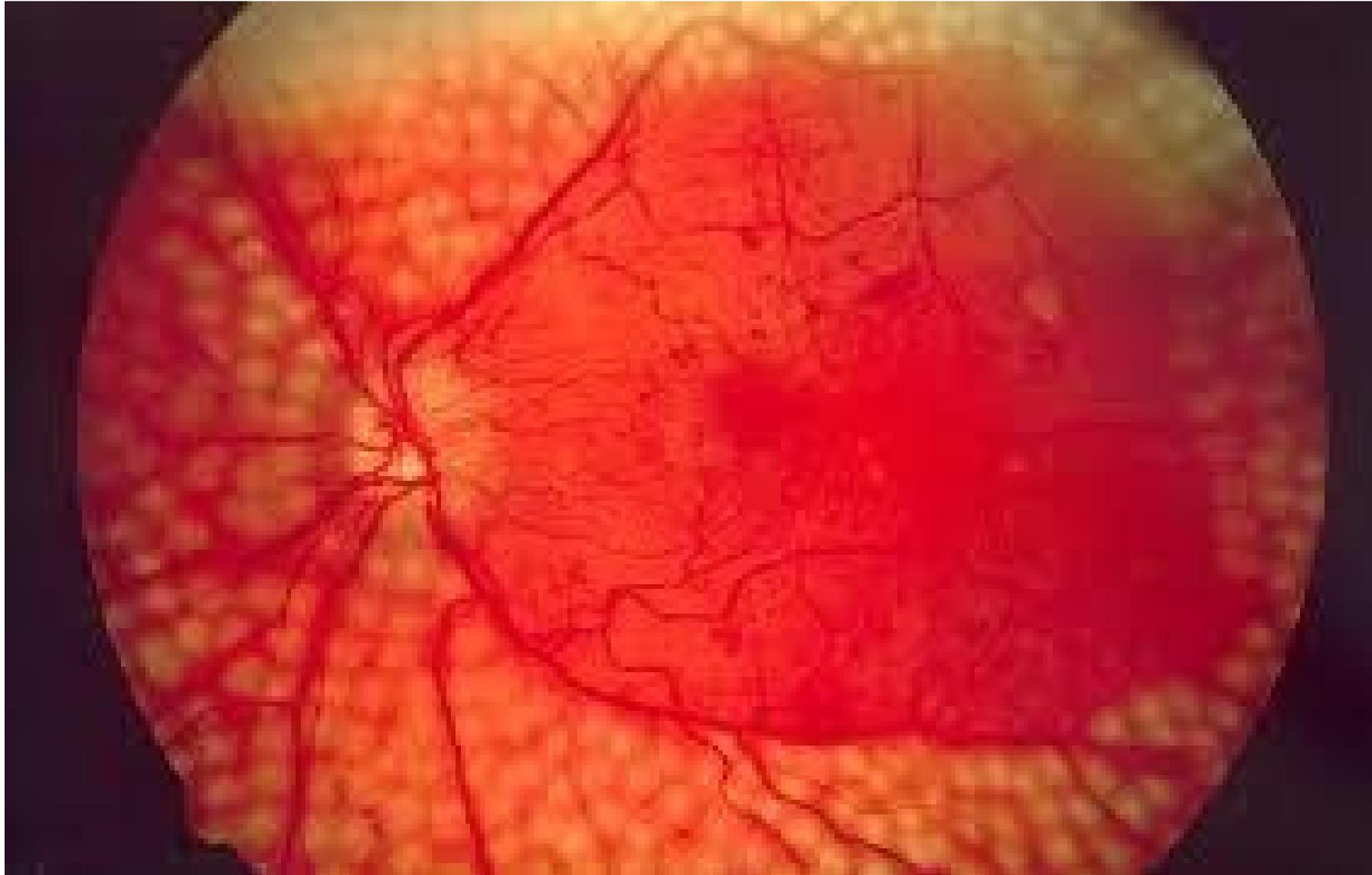
- steroids

**Laser** is the mainstay of treatment:

- \_\_\_\_\_
  - Focally (in the macula)
  - PRP (Pan Retinal Photocoagulation)
- **Surgery:** advanced PDR (Vitreous hemorrhage and traction RD)

**Prognosis** is based on:

- control of blood sugar
- time of treatment
- family history
- stage of DR



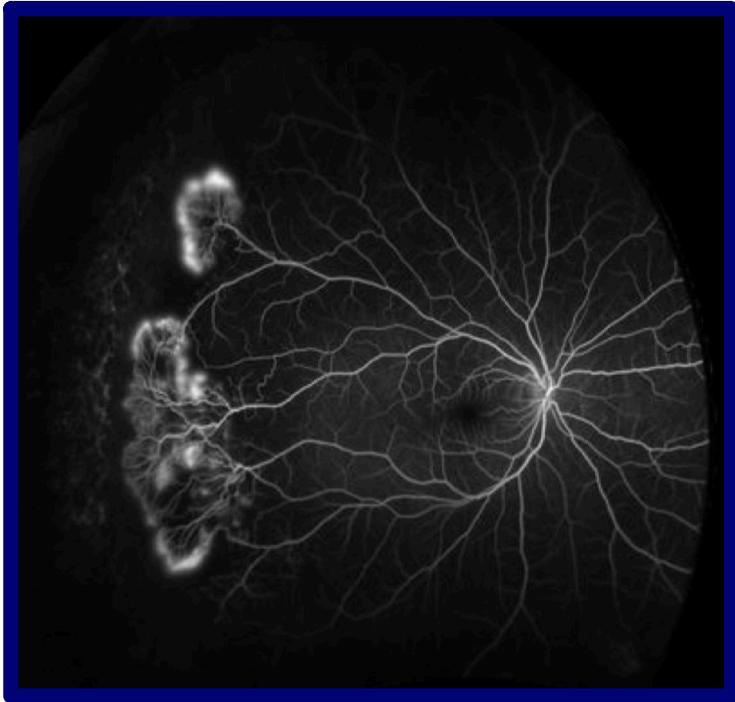


Can occur in any sickling hemoglobinopathies with greater risk seen in those with HbSC and SThal and a lower risk with HbSS.

- 
- Blind spots
  - Sudden onset of floaters or blurred vision
  - Flashes of light
  - Loss of side vision or curtains

- 
- 
- Tortuous veins
  - Peripheral hemorrhage
  - Capillary non-perfusion
  - Pigmented spots (salmon coloured spots)
  - Proliferative sickle retinopathy (sea fan pattern)

- Laser
- Surgery



- **Arterial occlusion**
- **Venous occlusion**
- **Hypertensive retinopathy**
- **Retinopathy of prematurity**





## Risk factors:

Hypercholesterolemia

Obesity

Old age >60

DM

Temporal arteritis

Atrial fibrillation

Thrombophilia(OCPs)

Sudden, painless, unilateral, often severe visual loss.

Fugax :caused by Fibrin-platelet emboli typically cause a fleeting loss of vision as the emboli pass through the retinal circulation.

Migraine: could be transient.

Patient may be known to have carotid or valve disease.

Acute changes include: retinal opacification, whitening

oedema;

cherry-red spot at the macula;

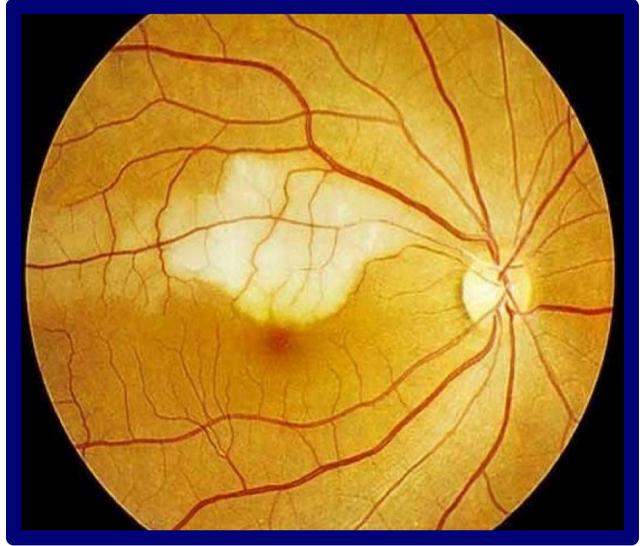
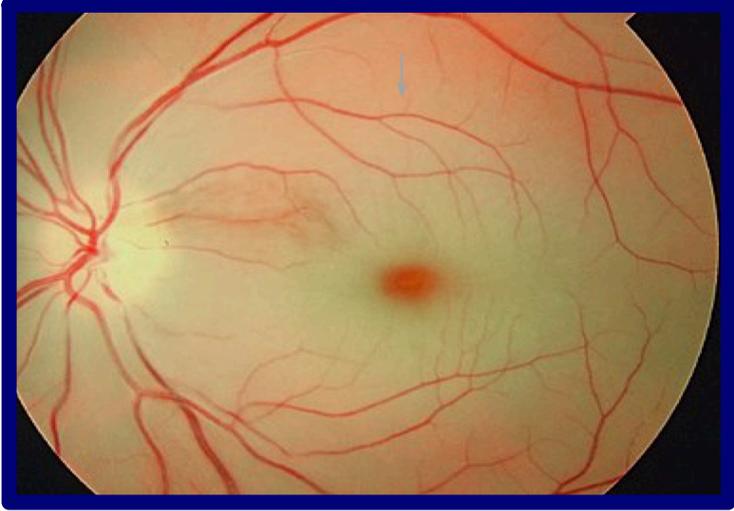
intra-arteriolar blood column segmentation (box-carring);

pale OD and retina, and attenuated blood vessels.

Ask about transient ischaemic attacks, cerebrovascular accidents, symptoms of giant cell arteritis, or amaurosis fugax, drugs,...

Auscultate the carotids for bruits using the stethoscope bell, check heart sounds for a valvular murmur, and radial pulse for atrial fibrillation

Look for intra-arteriolar calcific, cholesterol, or fibrinoplatelet emboli



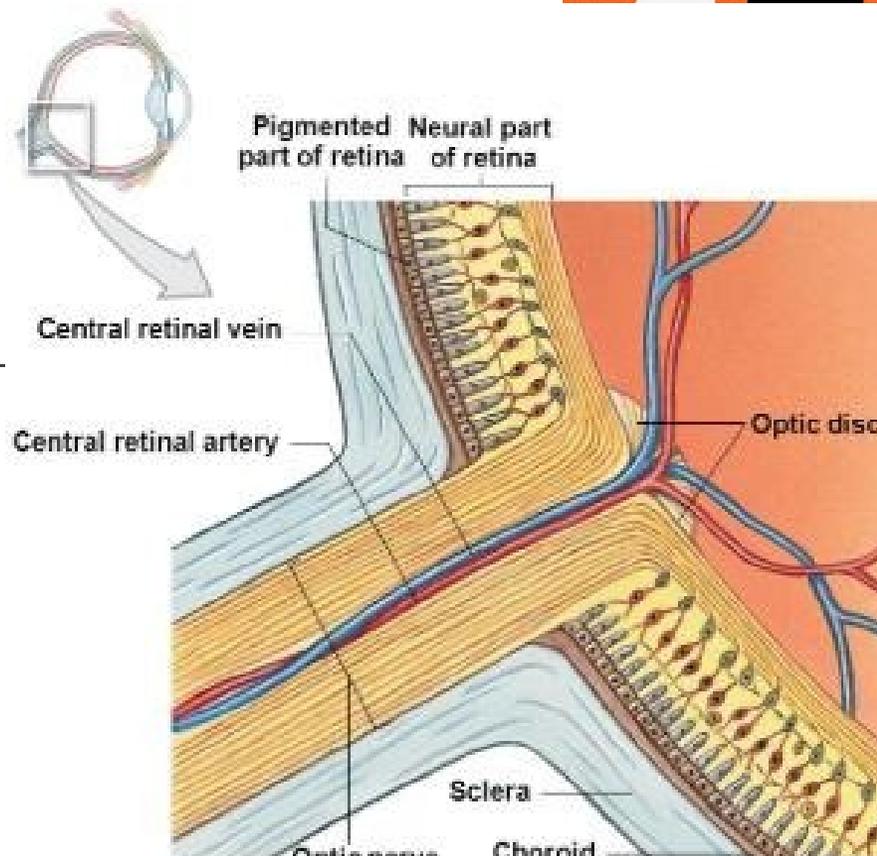
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- Arrange BP.
  - urgent ESR and CRP.
  - blood sugar.
  - Lipids.
  - ECG and echo.
  - Coagulation profile.
  - carotid artery : Doppler.
  - fluorescein angiogram and cardiac examination for embolic source.

- 
- Acute treatment especially if in the 1st 24 h, **ADMISSION**
  - High concentration O2 inhalation.
  - IV Acetazolamide (reduce IOP).
  - Ocular massage OR AC paracentesis may be needed.

- 
- **Treat the underlying cause.**
  - **Immediat anticoagulant & antiplatlet (Aspirin).**
  - **Carotid endarterectomy :**  
**indicated if a stenosis of the carotid artery is greater than 75% to prevent the possibility of cerebral embolus.**
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# *Central retinal vein occlusion (CRVO)*

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Risk factors: diabetes, hyperlipidemia, DM and glaucoma,

Predisposing factors:

### 1) Blood dyscrasias

\*hyper viscosity due to:

- a) use OCPs
- b) sickle cell disease
- c) changes in plasma proteins: macroglobulinemia
- d) hypercellularity: polycythemia

\*coagulation disorders

### 2) Changes in the wall of the vein:

- \*inflammation associated with sarcoidosis, Behcet syndrome
- \*congenital narrowing of central retinal vein

### 3) Changes outside the vein

- \* increase intraocular pressure
- \* rigid artery (atherosclerosis) compresses the vein

CRVO typically occurs in Patients > 45 years 2ry to retinal vein thrombosis

CRVO in patients < 45 years may suggest a clotting disorder

# ***Clinical picture:*** 1) ***painless visual loss in one eye*** 2) ***slower onset than retinal artery occlusion***

## Non- ischemic



- 1) retinal hemorrhages distributed in all quadrants of the retina
- 2) Macular edema may or may not be present

## Ischemic



- 1) Four quadrant retinal hemorrhage
- 2) Cotton wool spot
- 3) Optic disc swelling + macular oedema → exudate
- 4) Dilated tortuous retinal veins
- 5) Neovascularization of iris, retina or disc

# *Management*

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## INVESTIGATIONS

- 1) check BP, fasting blood glucose, lipid profile, thyroid function & ESR
- 2) Plasma protein electrophoresis & thrombophiliascreen
- 3) fluorescein angiogram if ischemia is suspected
- 4) ocular OCT

- 1) retinal laser if the retina is ischemic, to prevent development of retinal & iris new vessel
- 2) intravitreal steroid therapy
- 3) intravitreal anti VEGF

# ***HYPERTENSIVE RETINOPATHY***

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1) definition: spectrum of retinal vascular related to elevation in blood pressure

2) pathophysiology:

A) systemic HTN → hypertonus of retinal arterioles (primary response) → narrow straight arterioles

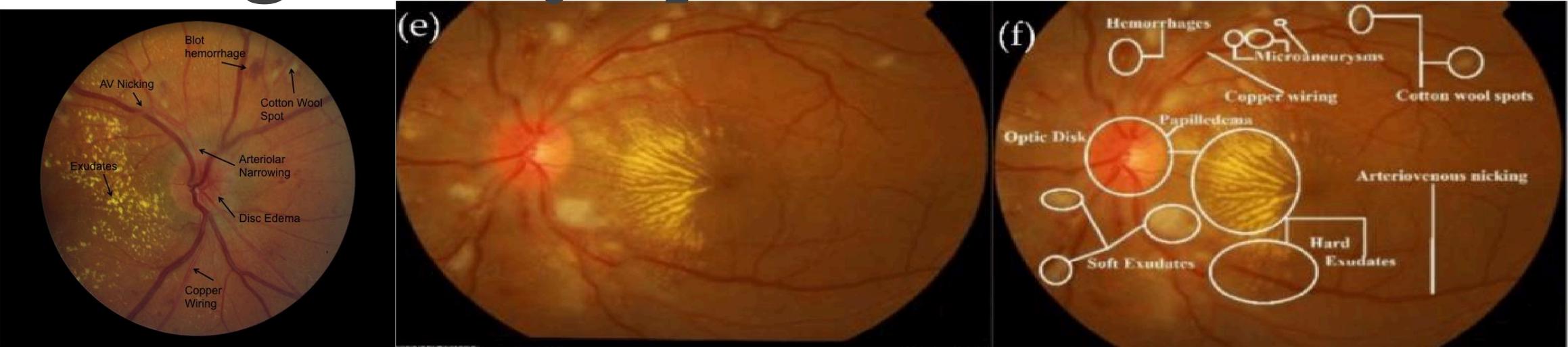
B) prolonged sustained systemic HTN → 2ry arterio- sclerosis ( intimal hyalinization, medial hypertrophy & endothelial hyperplasia) → vessel wall is thickened

3) classification:

Acute changes are more often symptomatic but not always

Chronic changes are typically asymptomatic until late stage

# Signs & symptoms



- 1) Blurred vision
- 2) Episodes of temporary vision loss

- 1) A-V narrowing/ attenuation
- 2) A-V crossing changes (AV nicking)
- 3) Arteriolar color changes (silver wiring / copper wiring)
- 4) Vessel sclerosis
- 5) Occasional hemorrhage
- 6) Cotton wool spot

# *Management*

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1) control blood pressure

2) life style modifications

3) **Regular Monitoring:** Regular eye exams are essential to monitor the progression of hypertensive retinopathy. The frequency of these exams depends on the severity of the condition and how well the hypertension is controlled

# *Retinopathy of prematurity*

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ROP: is a disease of the eye affecting prematurely born babies generally having received intensive neonatal care, in which oxygen therapy is used on them due to the premature development of their lungs

\*\* Initial failure of normal retinal vascularization → phase of aggressive new vessel formation extending forward into the vitreous → traction detachment

\*\* ↓↓ risk of occurrence by regulating the level of oxygen exposure

# ***Risk factors***

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1) GA < 32 weeks 2) birth weight < 1500g

3) Apnea

4) sepsis 5) duration of ventilation

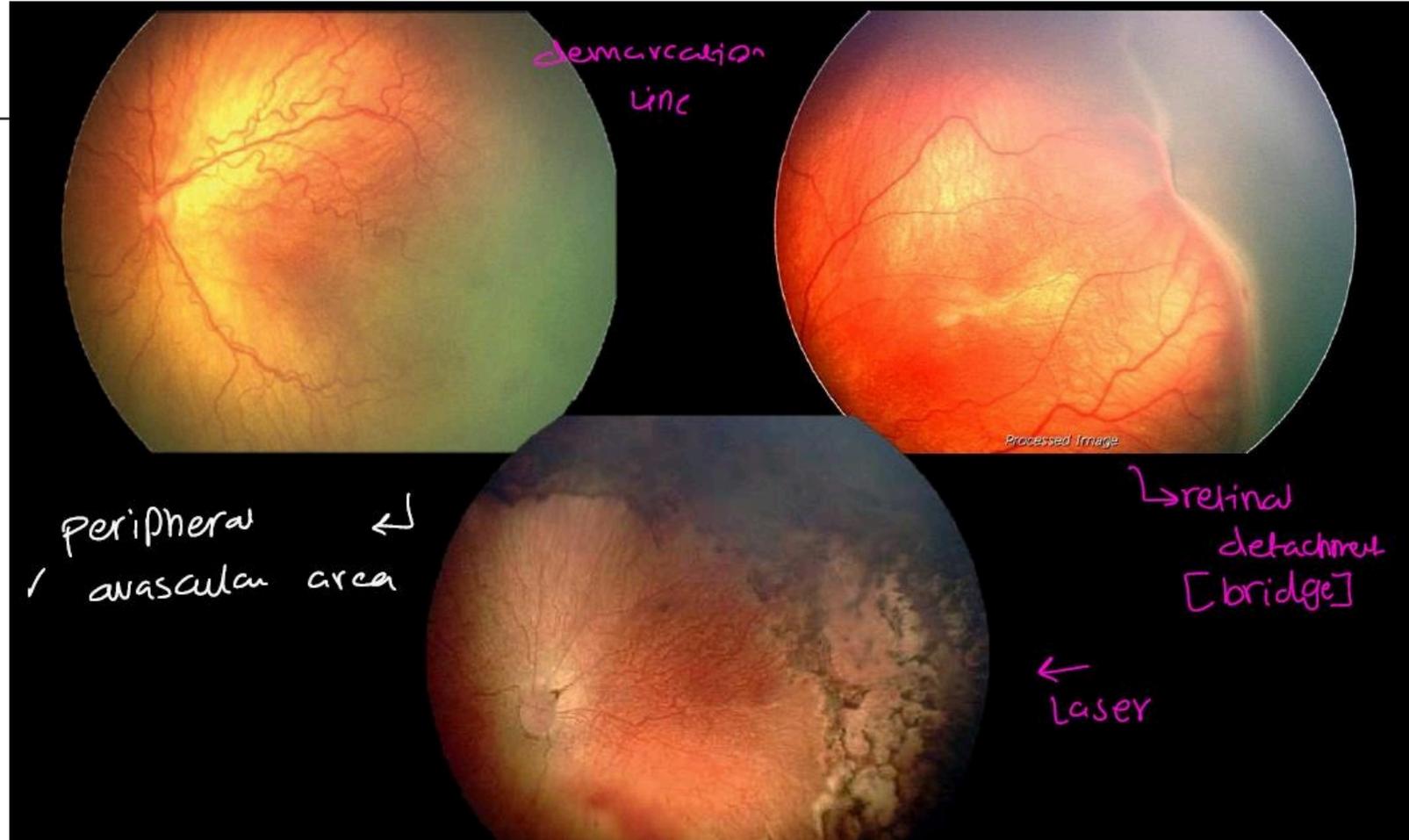
6) blood transfusion 7) retinal light exposure

8) presence of intraventricular hemorrhage

# ROP signs

\*\*depending on severity

- 1) small avascular ridge / zone
- 2) new vessels
- 3) retinal hemorrhage
- 4) ↑ tortuosity
- 5) vitreous hemorrhage
- 6) retinal detachment



# ***ROP management***

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- 1) regular screening of infants at risk
- 2) laser / cryotherapy to ischemic area
- 3) retinal surgeries