RETINAL VASCULAR DISEASES

DR KAUSHIKI BADVE

TOPICS

Vascular occlusions – venous & arterial

Diabetic retinopathy

Hypertensive retinopathy

Retinopathy of Prematurity

RETINAL VENOUS OCCLUSIVE DISEASE

Central Retinal Vein Occlusion (CRVO)

Branch Retinal Vein Occlusion (BRVO)

INTRODUCTION

Most common vascular disorder
Elderly (6^{th} – 7^{th} decade)
Due to thrombus formation

PREDISPOSING FACTORS

- Age
- Diabetes & Hypertension
- Increased Blood Viscosity
- Leukemia, Polycythemia
- Macroglobulinaemia
- Oral contraceptive pills
- Hyperlipidemia
- Raised IOP (POAG)
- Smoking

PATHOGENESIS

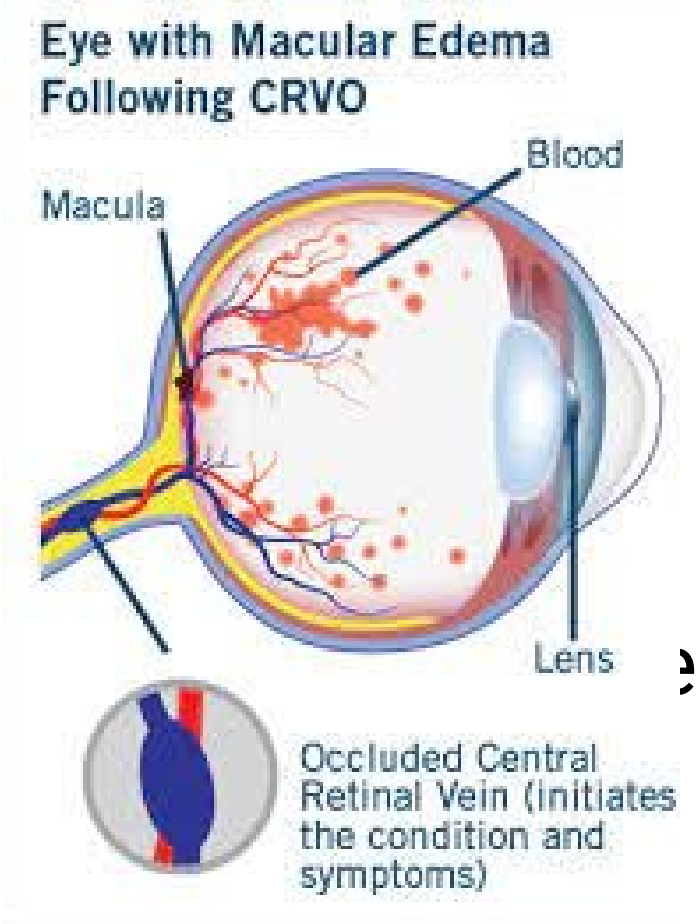
Disease of Vessel wall-

Vasculitis

Eales Disease

Pressure over veins by atherosclerotic retinal artery causes thrombosis in the lumen of the vein – Venous Occlusion

PATHOPHYSIOLOGY



Vein occlusion

Stagnation of Blood flow

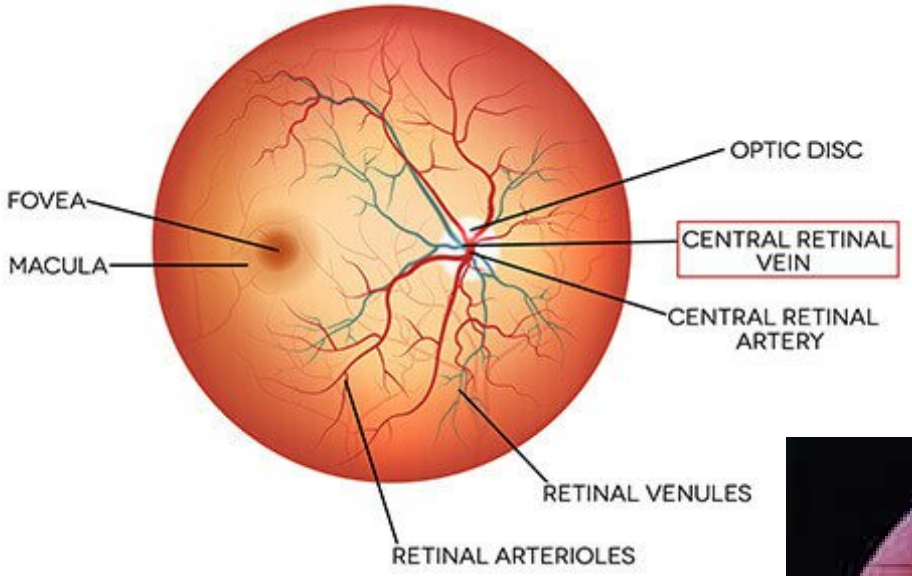
Hypoxia

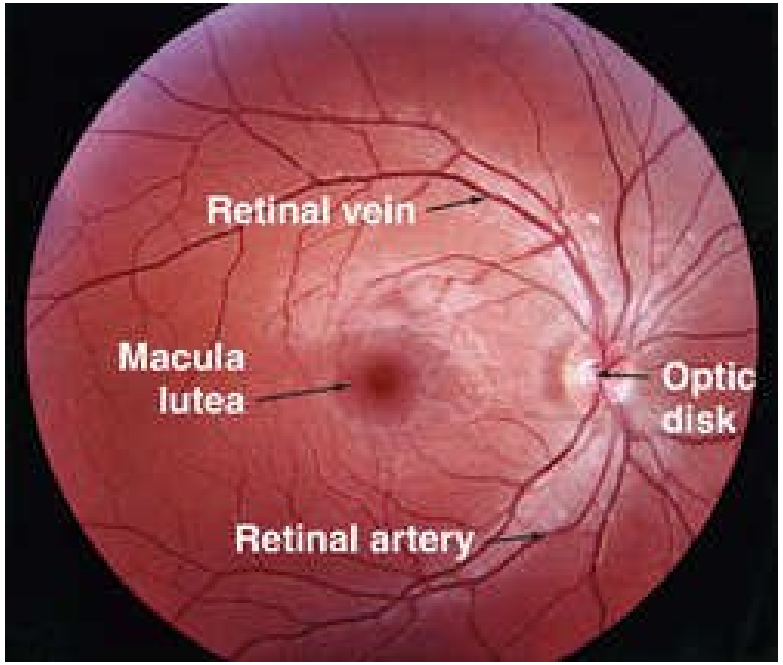
emorrhage

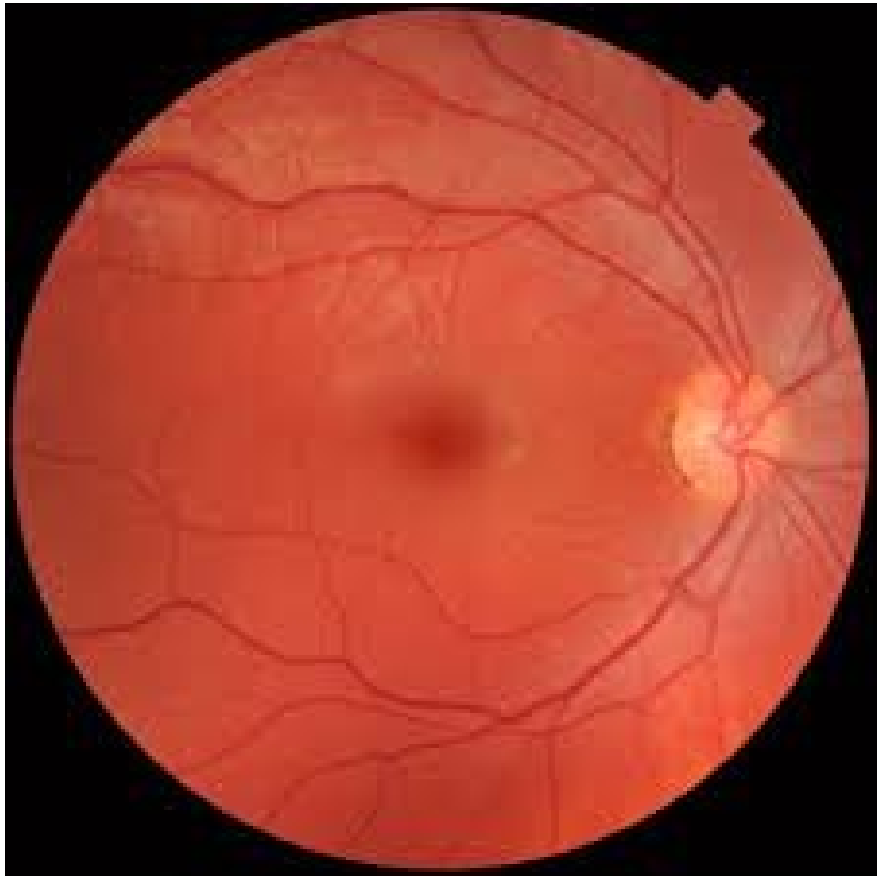
oedema

Neovascularization

NORMAL FUNDUS





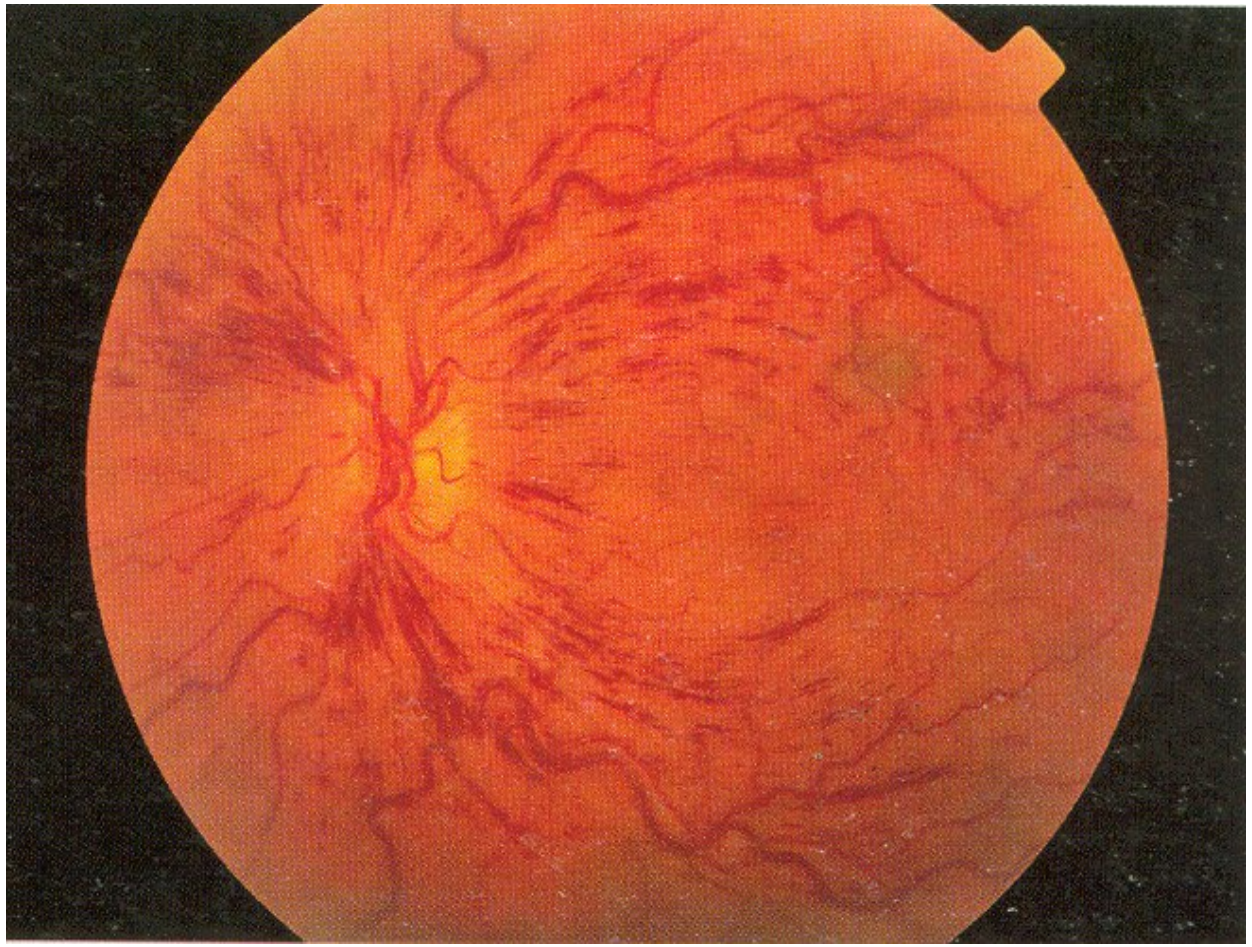


CENTRAL RETINAL VEIN OCCLUSION

2 TYPES

Non Ischaemic CRVO

Ischaemic CRVO



NON ISCHAEMIC CRVO

More common 75%

Clinical Features

Mild – moderate Diminution of vision

RAPD absent

NON ISCHAEMIC CRVO

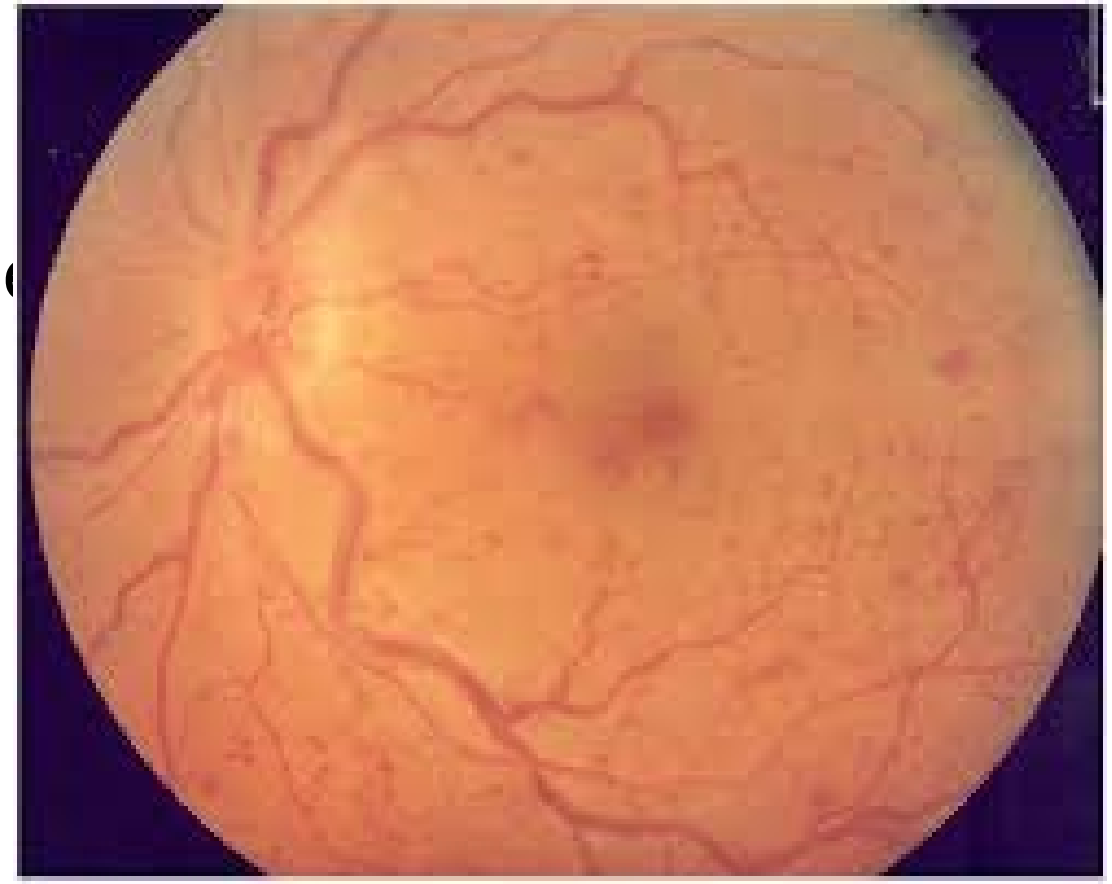
Fundus Exam :-

Mild venous congestion

Flame shaped Retinal Haemorrhage in periphery

Mild disc oedema

No macular oedema



ISCHAEMIC CRVO

due to acute complete occlusion of Central Retinal Vein

Clinical Features

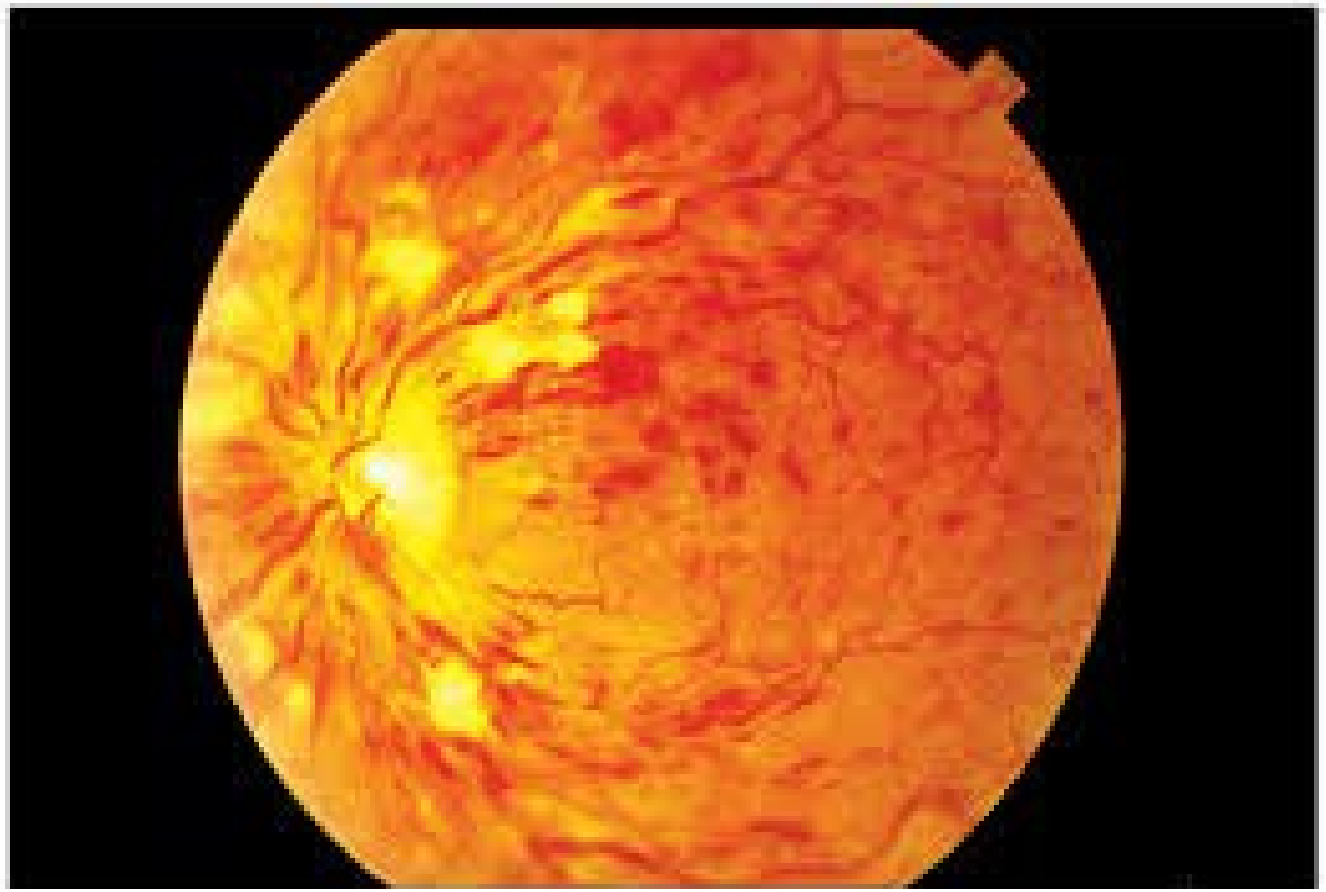
Sudden Gross Diminution of vision

RAPD present

ISCHAEMIC CRVO

Fundus Exam :-

- Optic Disc Congested, Margins Blurr
- Veins engorged & Tortuous
- Multiple Retinal Haemorrhages
- Splashed tomato appearance
- Numerous cotton wool spots
- Macular haemorrhages & oedema



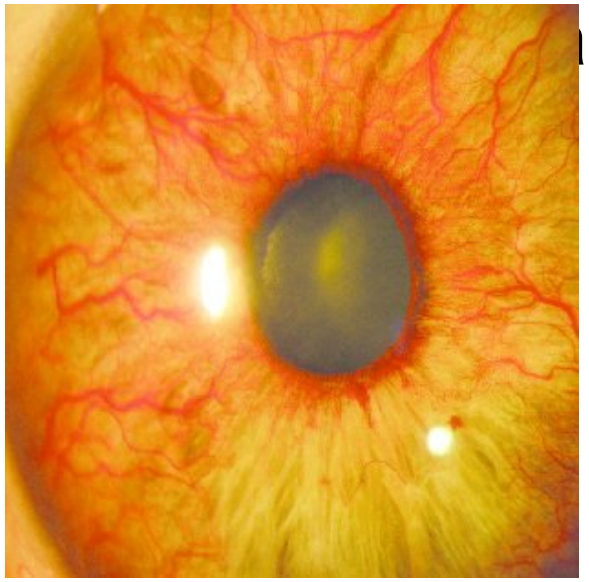
ISCHAEMIC CRVO

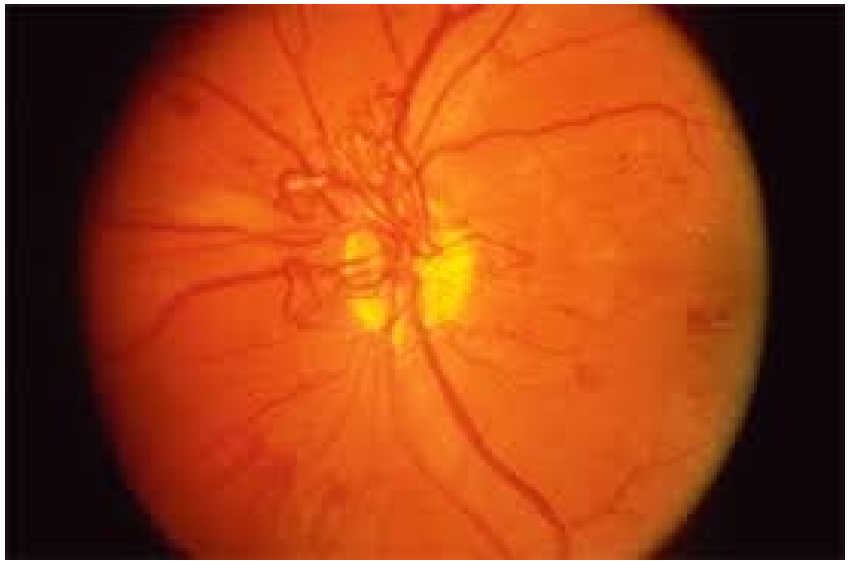
Later stages/ complications:

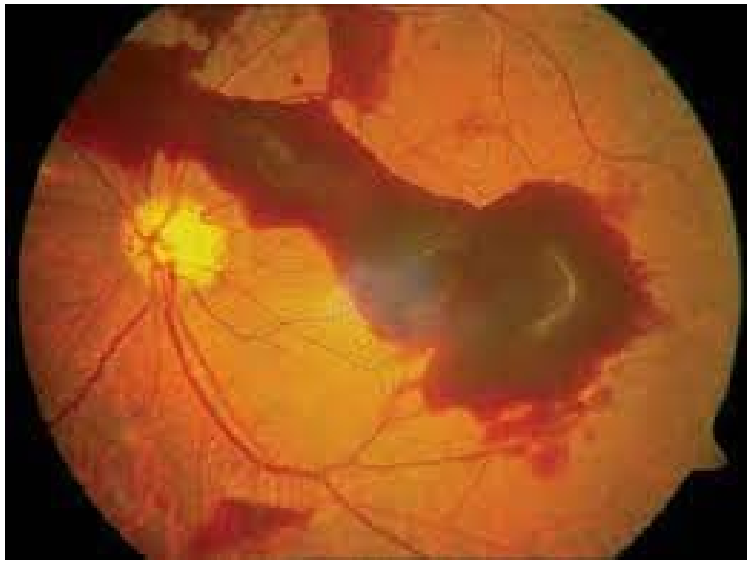
Neovascularization (NVD, NVE, NVI) – 90 day Neovascular Glaucoma

Chronic Macular Oedema

hem







BRANCH RETINAL VEIN OCCLUSION

Clinical Features (BRVO)

Changes Limited to area drained by the obstructed vein

Upper Temporal Branch is more common

Vision only affected if macula is involved

Complications like neovascularization and chronic macular oedema in <30% of cases

BRVO

MANAGEMENT

Investigations

- Recording of Blood Pressure
- Recording of IOP
- Haemogram – Lipid profile
- Blood Sugar estimation
- STS & Mantoux Test in young patients
- FFA – after haemorrhages resolve
- OCT – macular oedema

TREATMENT

Treat the predisposing conditions

Observation & monitoring for 6 – 8 weeks

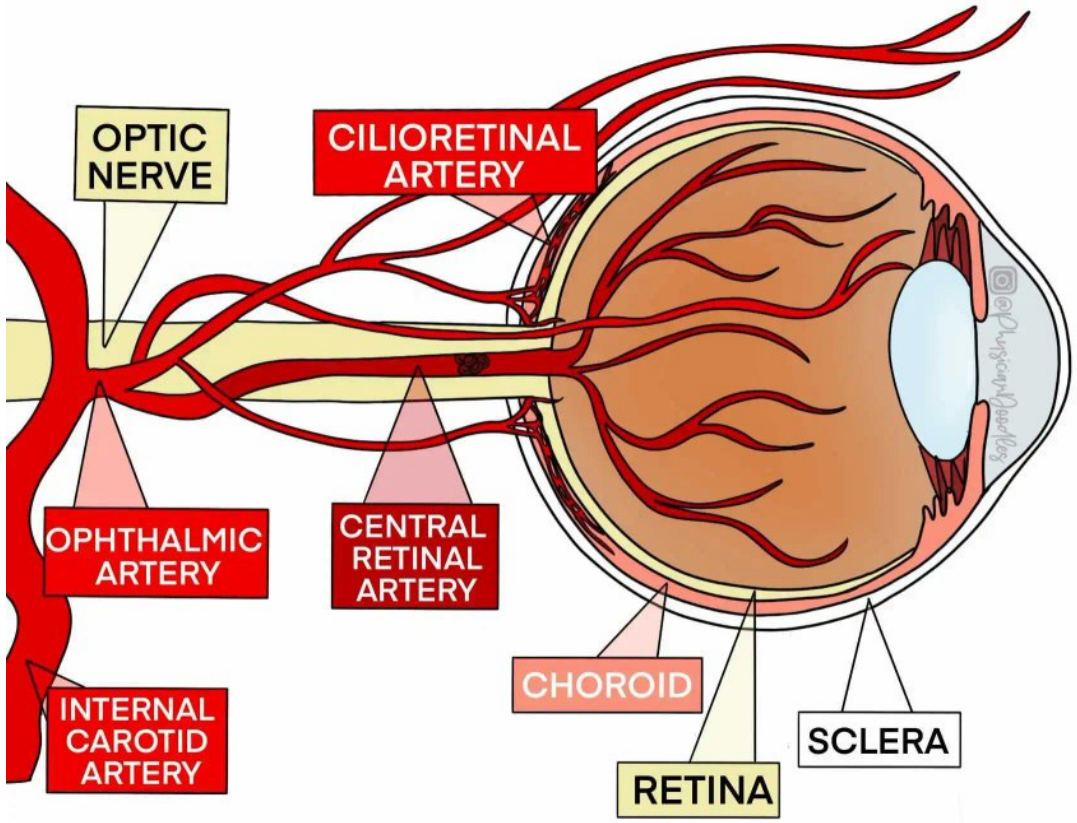
Medical therapy – Intra vitreal Anti VEGF, steroids (ozurdex)

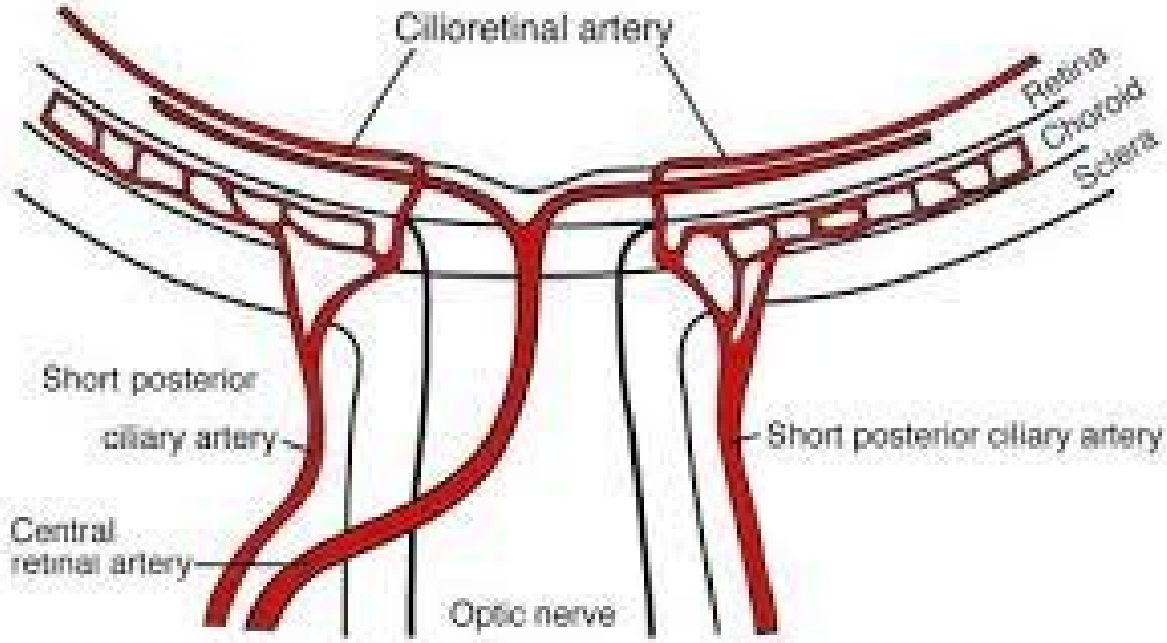
Laser treatment- Pan Retinal Photocoagulation If NVE / NVD occurs

Macular Grid – Macular oedema in BRVO

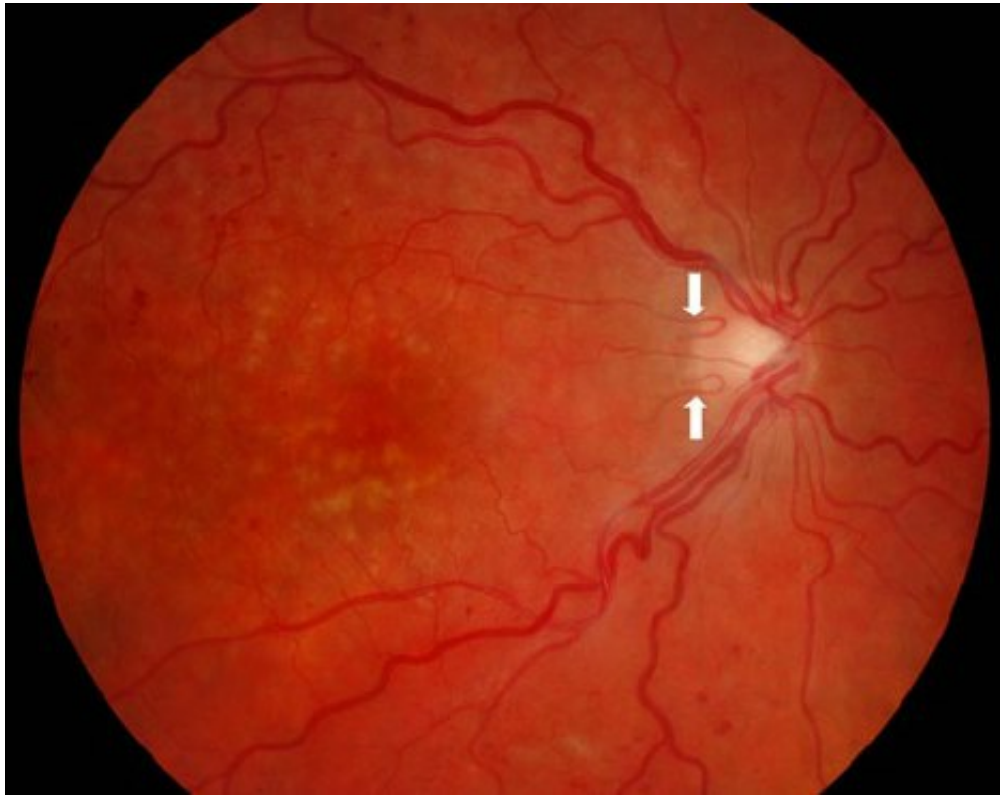
Surgical Treatment – Pars plana vitrectomy

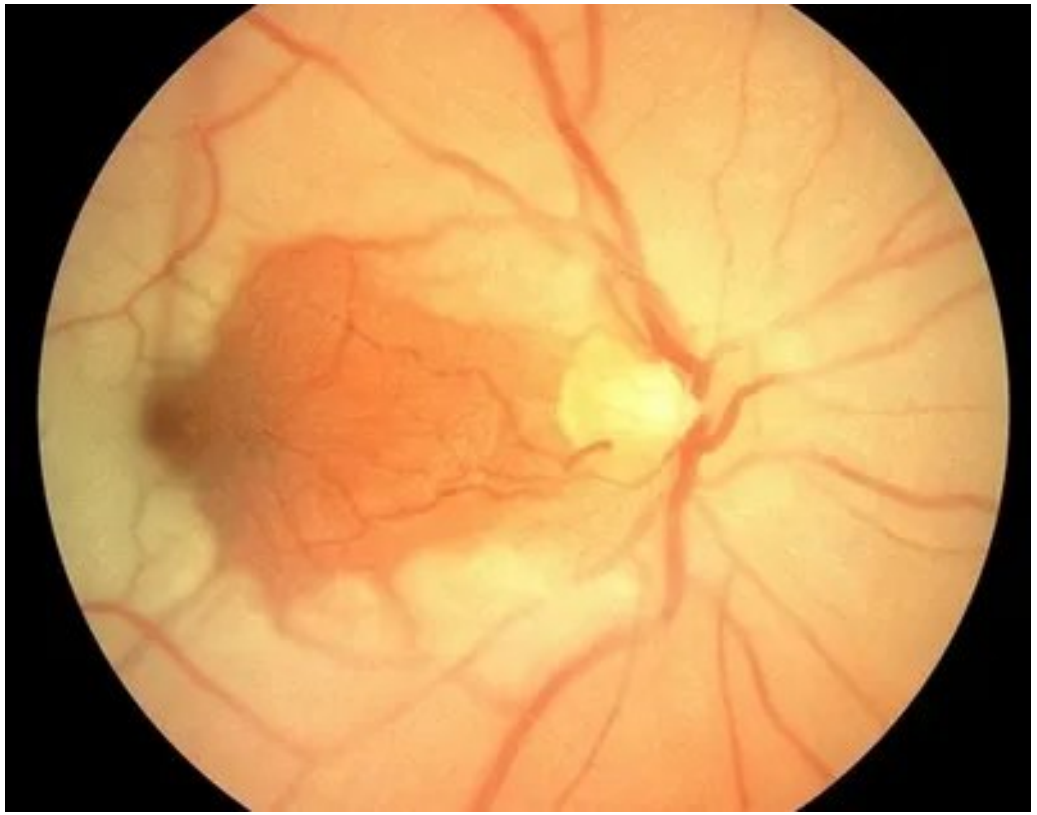
BLOOD SUPPLY OF RETINA





CILIORETINAL ARTERY





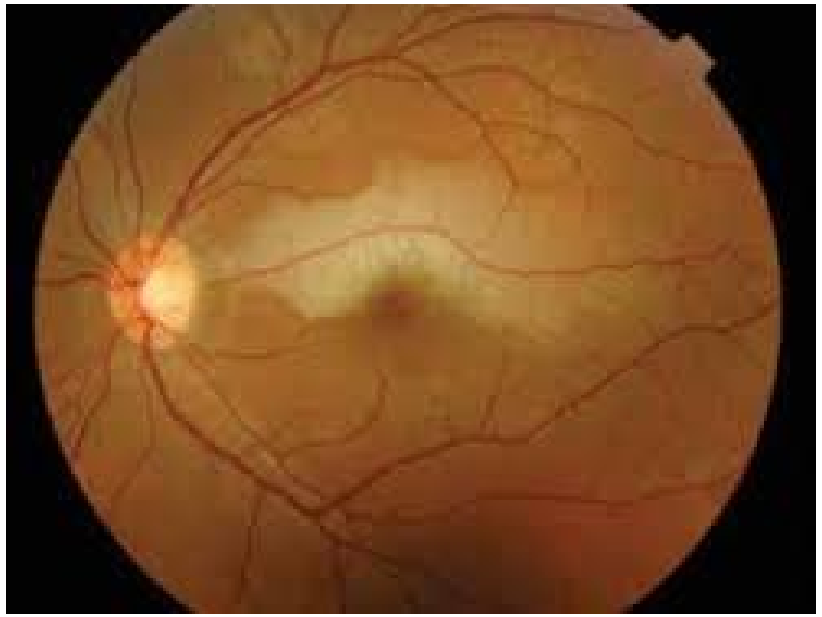
MACULA SPARING CRAO

RETINAL ARTERY OCCLUSIONS

CRAO (60%)

BRAO (35%)

Cilioretinal A occlusion (5%)



Ocular Emergency

Often leads to Blindness

May be presenting symptom of systemic disease

Central Retinal artery is an End artery

Ganglion cells can withstand ischemia for only 5 min

AETIOLOGY

Emboli – Heart/carotid artery

Thrombus

Retinal arteritis with obliteration

Giant Cell Arteritis

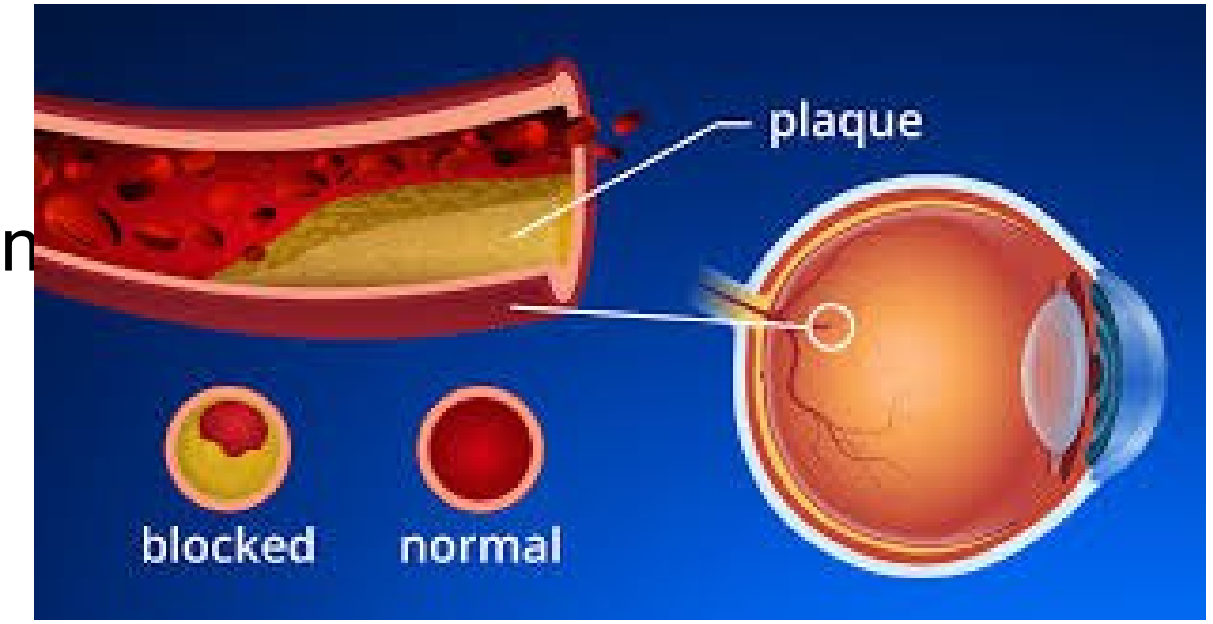
Takayasu disease

Polyarteritis Nodosa

Angiospasm

Pressure from Outside

Increased IOT/ Pressure over Globe



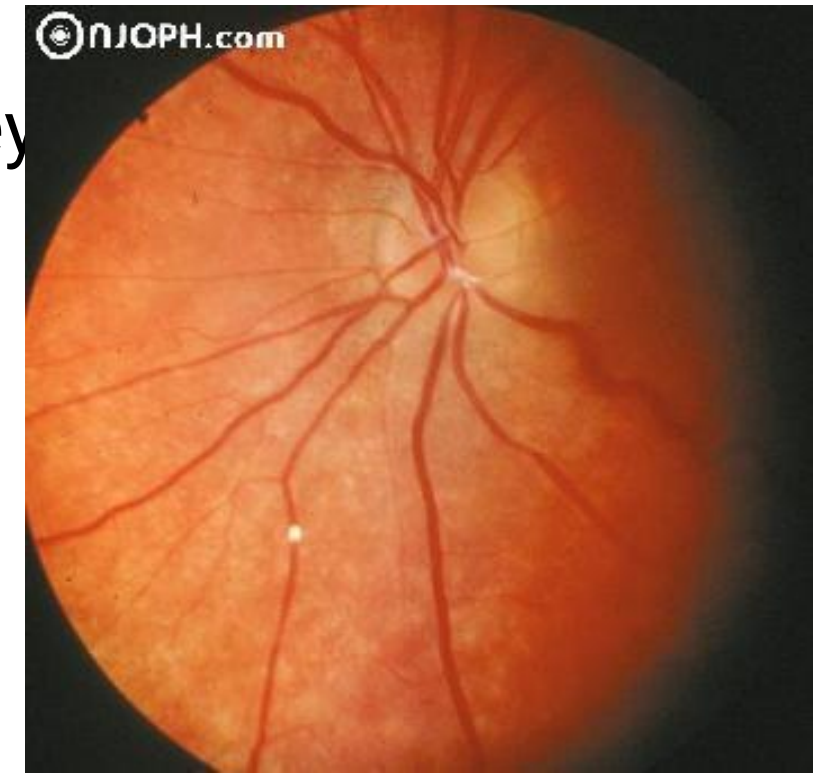
CENTRAL RETINAL ARTERY OCCLUSION

Clinical Features-

Sudden painless loss of vision in affected ey

No perception of light

Afferent Pupillary defect



CENTRAL RETINAL ARTERY OCCLUSION

Fundus Exam-

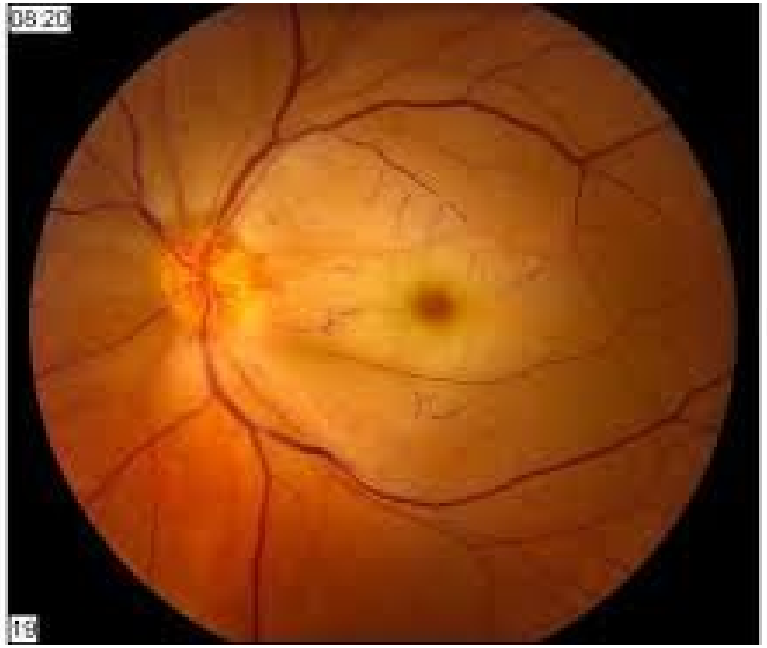
Retina looks milky white

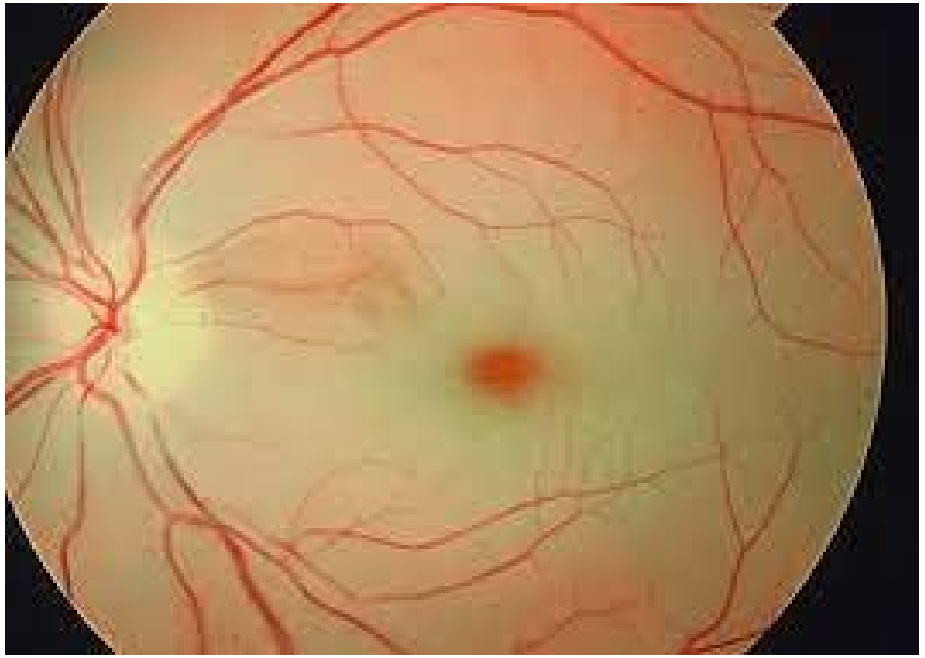
Cherry red spot at Fovea

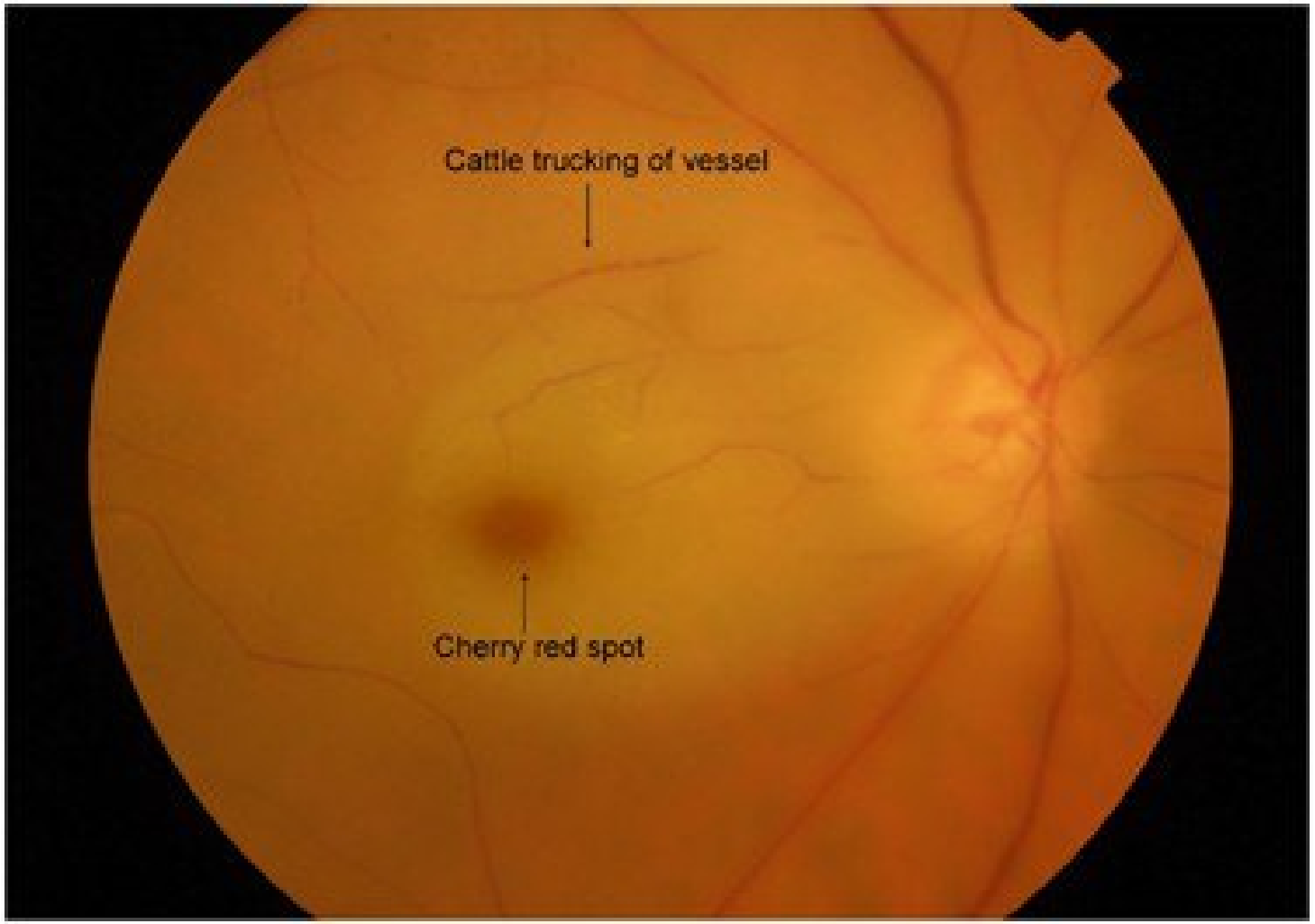
Marked narrowing of retinal arterioles

Cattle Track appearance Segmentation of Blood column on pressure over globe

End result – Optic atrophy in 4- 6 weeks






Cattle trucking of vessel
Cherry red spot

TREATMENT OF CRAO

Lower IOP

Ocular Massage

IV Acetazolamide 500mg/ Mannitol

AC Paracentesis

Inhalation of 95% O2 + 5% CO2 – relieve angiospasm

Vasodilators

Fibrinolytic therapy

MANAGEMENT OF CRAO

All patients with CRAO should be investigated fully

Lipid Profile

Carotid artery doppler

Examination of Heart – Echo

Temporal Artery biopsy

ESR

DIABETIC RETINOPATHY

Microangiopathy affecting retinal precapillary arterioles , capillaries and venules

DIABETIC RETINOPATHY

Approximately 2% of all Diabetics become blind

Incidence blindness is 20 times greater in Diabetics

Incidence of Diabetic Retinopathy is related to duration
Diabetes Usually occurs after 15 – 20 years of Diabetic age

PATHOGENESIS

Microvascular Occlusion

Thickening of Capillary Basement Mn

Endothelial damage & proliferation

Changes in RBCs

Increased stickiness of Platelets

Retinal Ischaemia

AV Shunts — IRMA

Neovascularization — NVD/ NVE

PATHOGENESIS (contd)

Microvascular Leakage

Loss of Pericytes

Retinal Haemorrhage

Retinal Oedema

Hard exudates

Microaneurysms

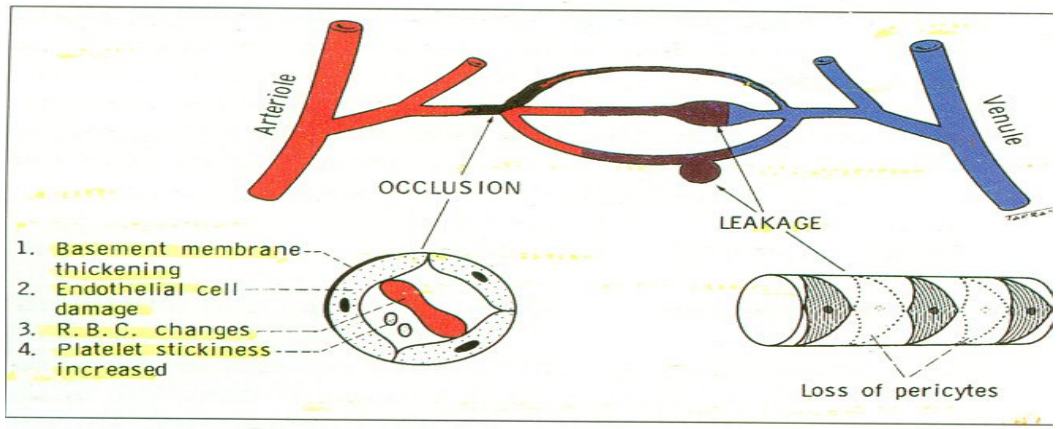


Figure 12.1 Pathogenesis of diabetic retinopathy

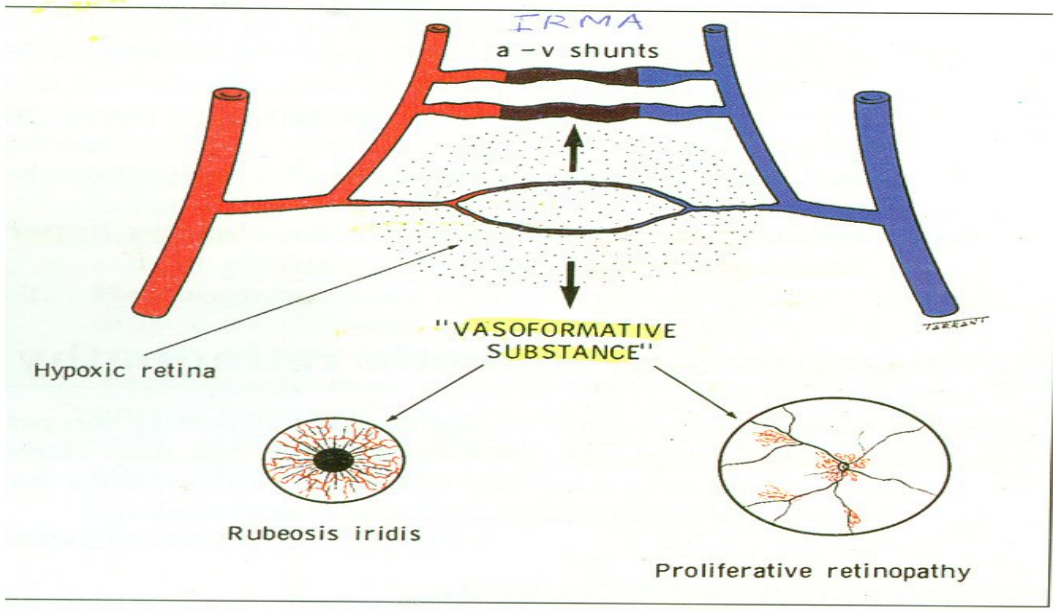
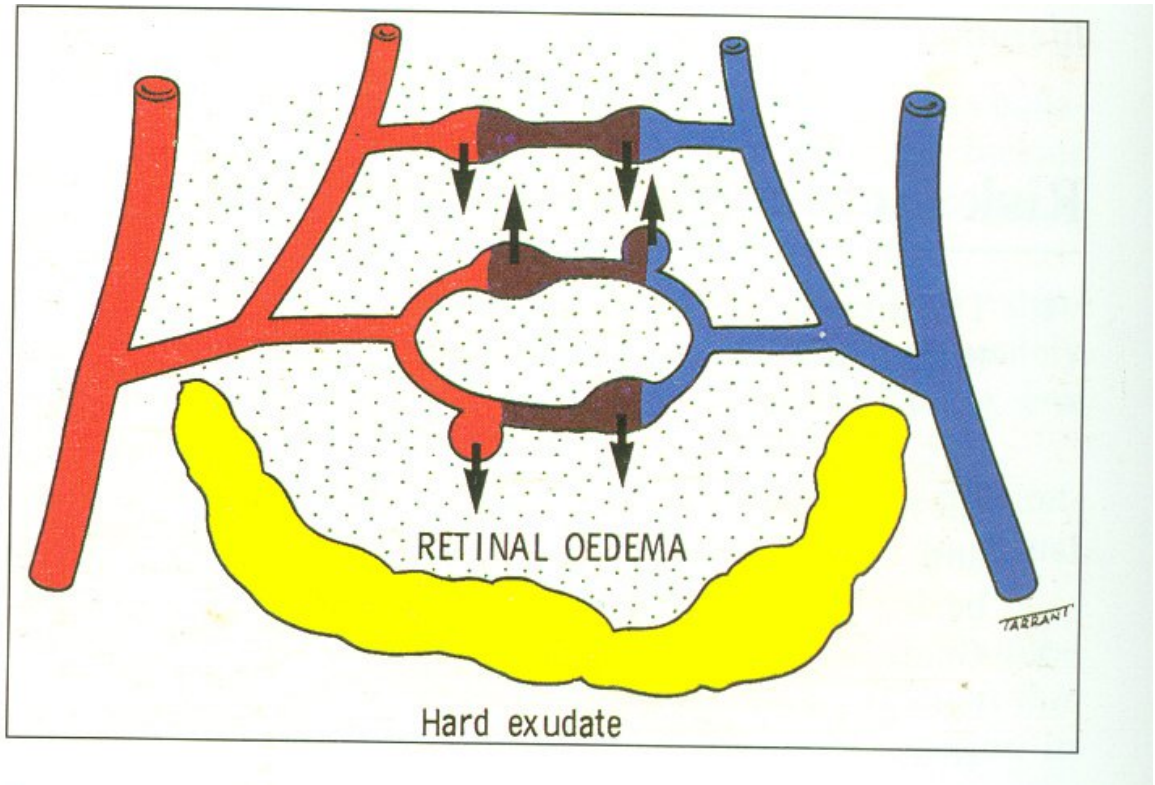


Figure 12.2 Consequences of retinal ischaemia in diabetic retinopathy



DIABETIC RETINOPATHY CLASSIFICATION

ETDRS (Early Treatment Diabetic Retinopathy Study)

Non Proliferative Diabetic Retinopathy (NPDR)
(Mild, Moderate & Severe)

Proliferative Diabetic Retinopathy (PDR)
Diabetic Maculopathy
Advanced Diabetic Eye disease

Non Proliferative Diabetic Retinopathy

Fundus Findings:

Microaneurysms

Dot & Blot Haemorrhages

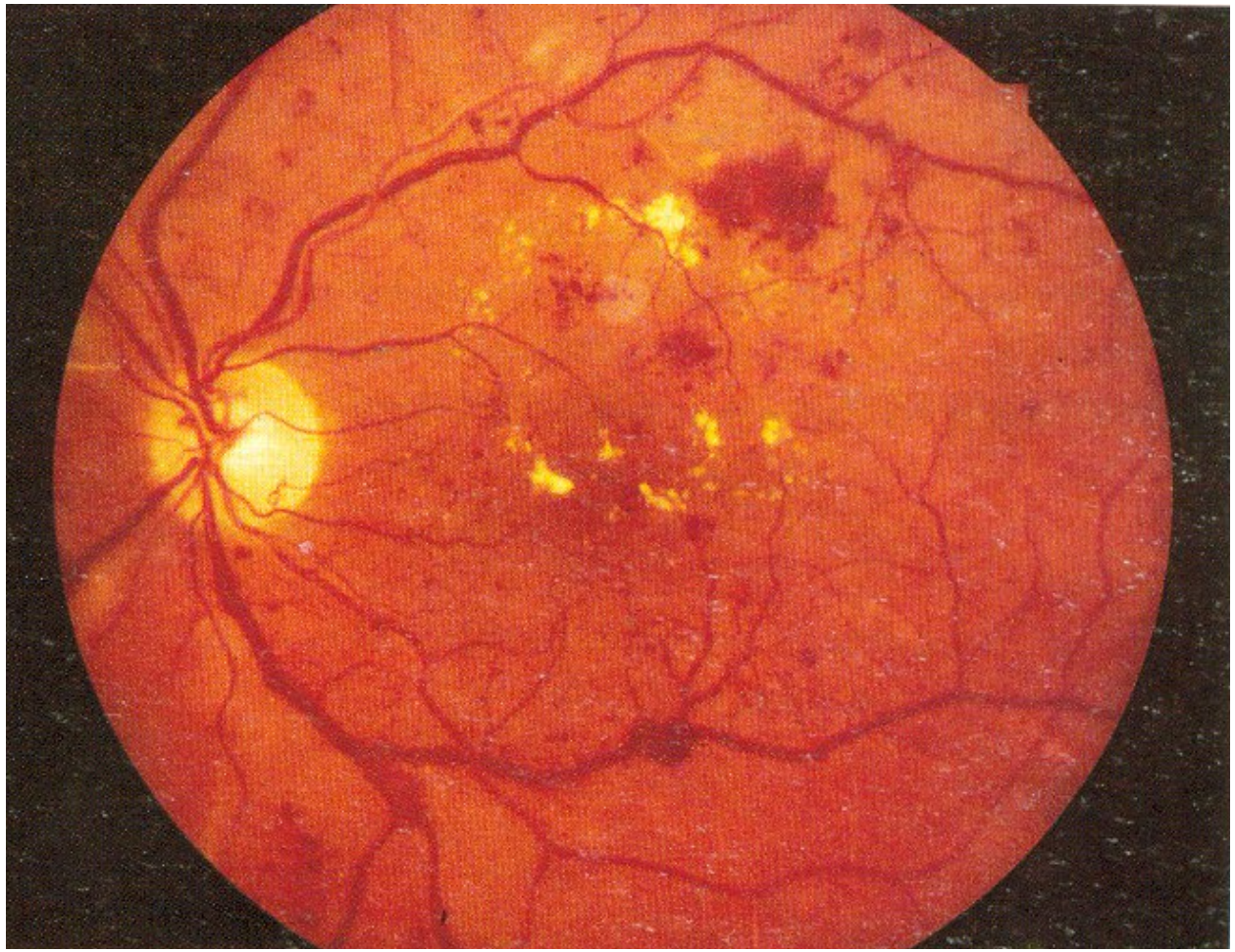
Hard Exudates

Severe cases:

Flame Shaped Haemorrhages

Soft Exudates

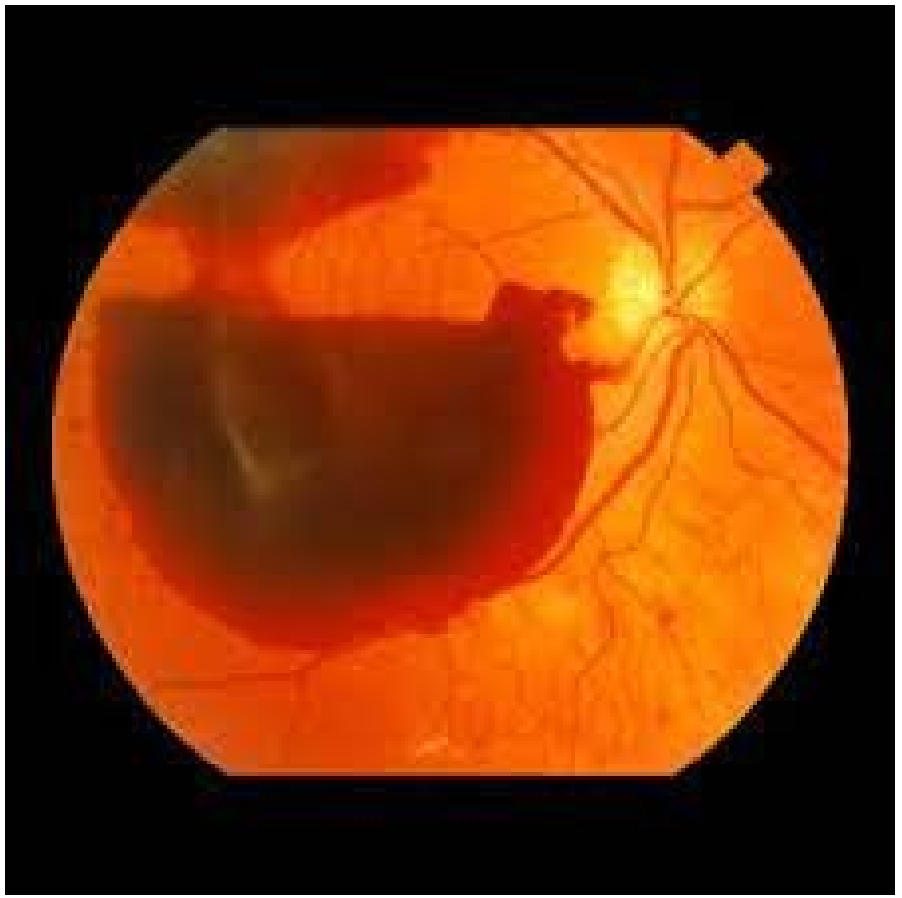
IRMA

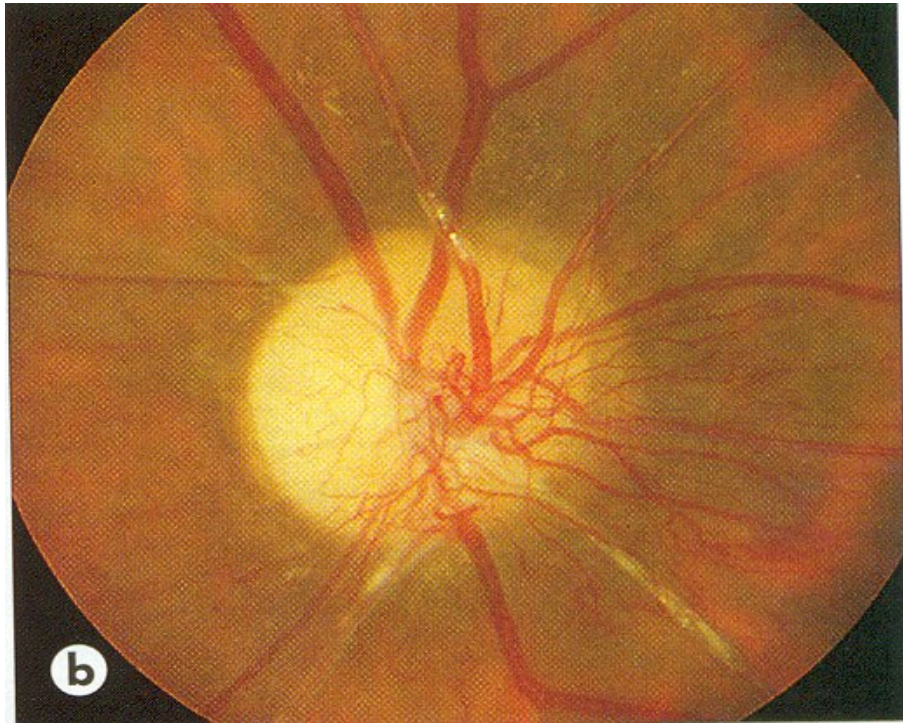


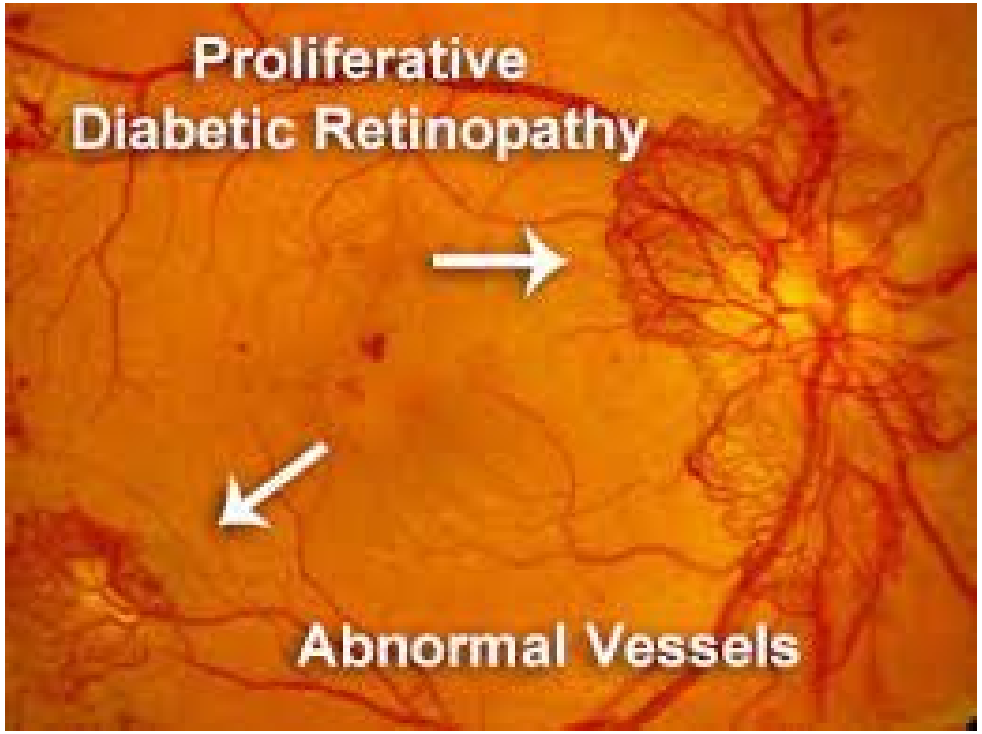
Proliferative Diabetic Retinopathy

Characterized by

- Neovascularisation
- NVD / NVE
- Vitreous Haemorrhage



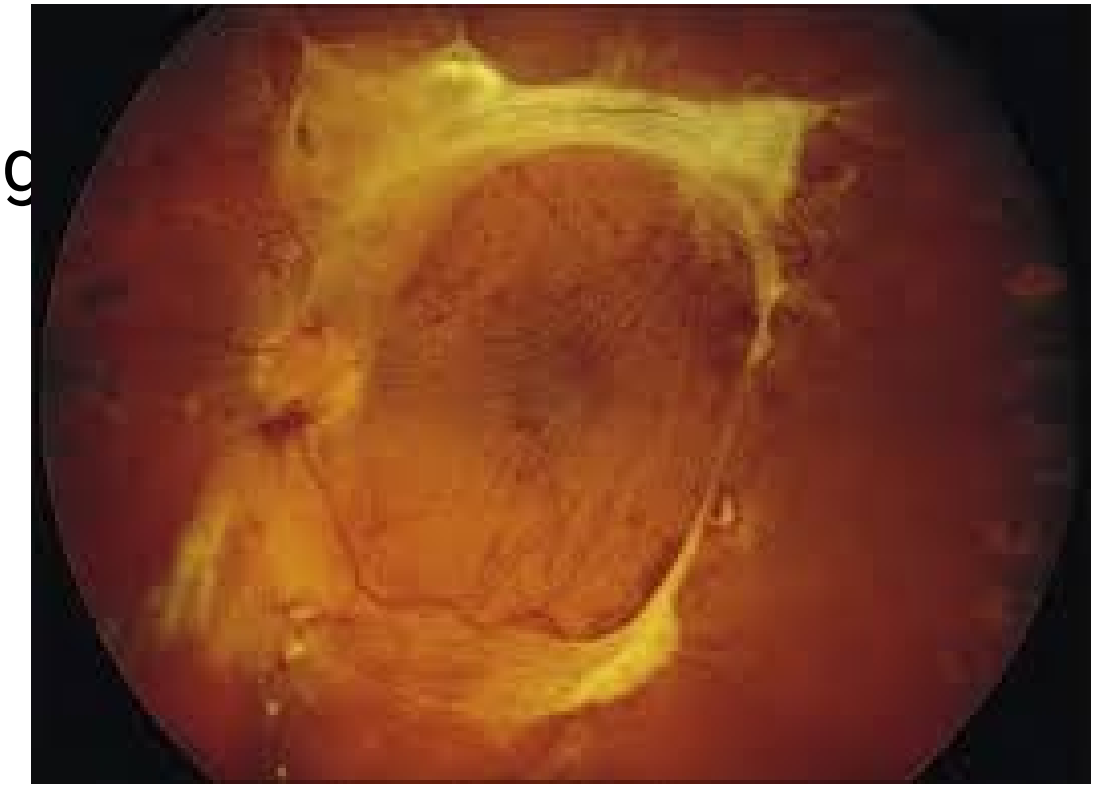

b


Proliferative
Diabetic Retinopathy
Abnormal Vessels

ADVANCED DIABETIC EYE DISEASE

Complications:

- Persistent Vitreous Haemorrhag
- Traction Retinal Detachment
- Neovascular Glaucoma



DIABETIC RETINOPATHY TREATMENT

Non Proliferative Diabetic Retinopathy

Good Diabetic control

Regular Fundus examination:

3 yr after diagnosis in type1/ on diagnosis in type 2 DM

Then yearly fundus exam

Macular Grid photocoagulation for macular oedema

DIABETIC RETINOPATHY TREATMENT

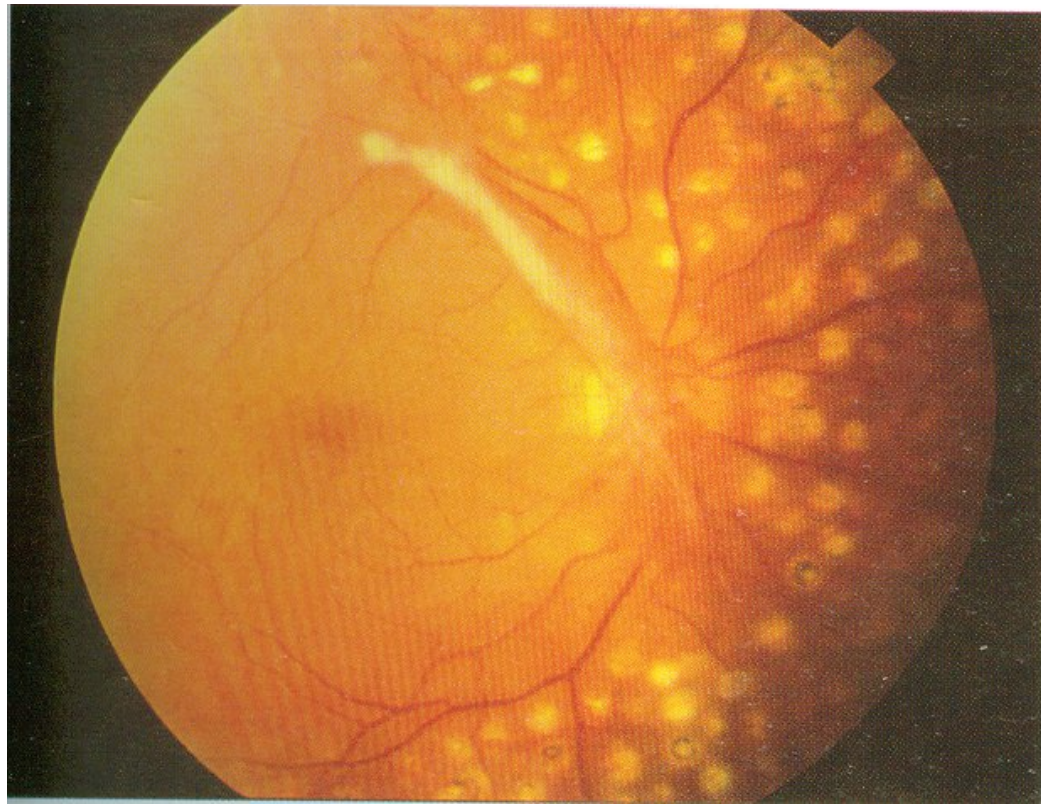
Proliferative Diabetic Reatinopathy

Intravitreal Anti VEGF drugs

√ Bevacizumab, Ranibizumab

√ In macular Oedema

√ Repeated after 4- 6 weeks



Laser Therapy:

√ PRP (Pan retinal photocoagulation)

√ Macular Grid laser

Vitreo Retinal surgery

√ Treatment of vitreous haemorrhage/

HYPERTENSIVE RETINOPATHY

Central Retinal artery is a branch of ophthalmic artery which is a branch of Internal Carotid artery

Mirrors the cerebral circulation

Medium sized artery which can be visualized

HYPERTENSIVE RETINOPATHY

Response of arteries to increased blood pressure

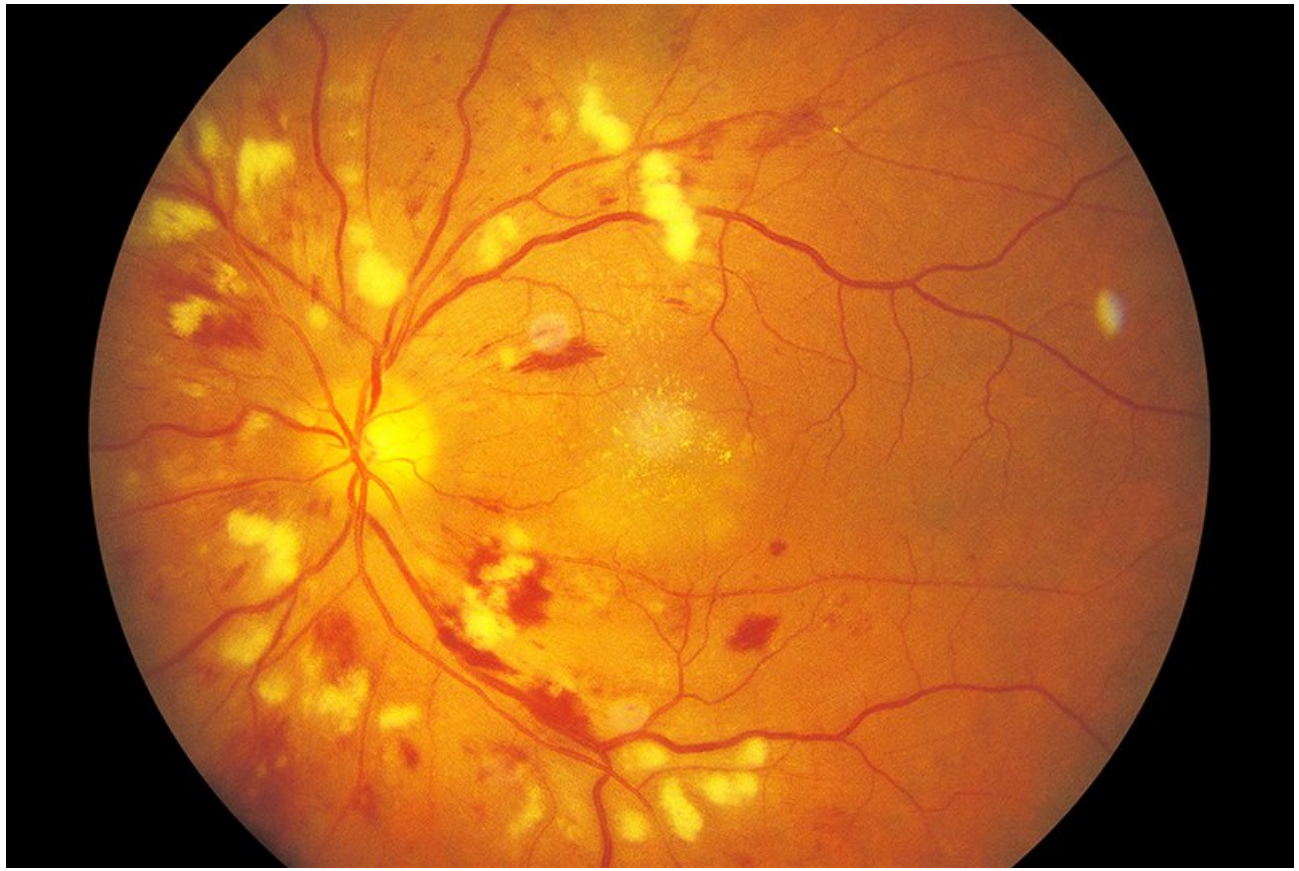
In young individual , recent hypertension, Spasm of arterioles

Long standing Hypertension - Arteriosclerosis

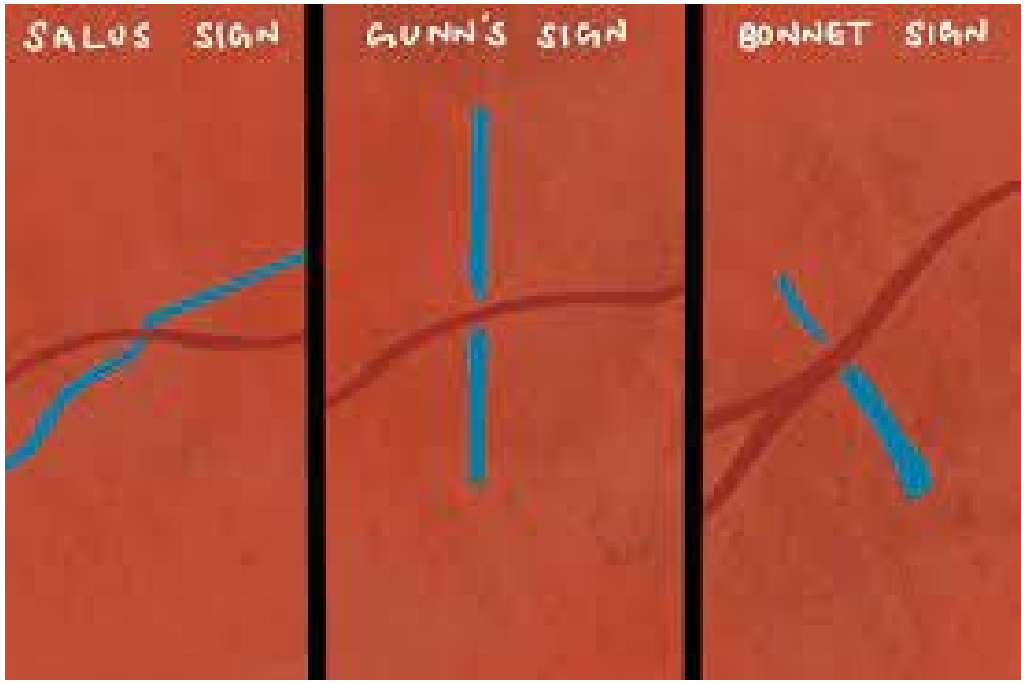
HYPERTENSIVE RETINOPATHY

Fundus Picture

- Generalized narrowing & Focal Narrowing of arterioles
- Flame shaped haemorrhages
- Hard Exudates – Macular star
- Cotton wool spots
- Papilloedema – Malignant hypertension
- Ischaemic Choroidal Infarct- Elschnigs spots



HYPERTENSIVE RETINOPATHY



Arteriosclerotic changes

-Broadening of light reflex

-Artereo venous crossing change

Concealment of vein-

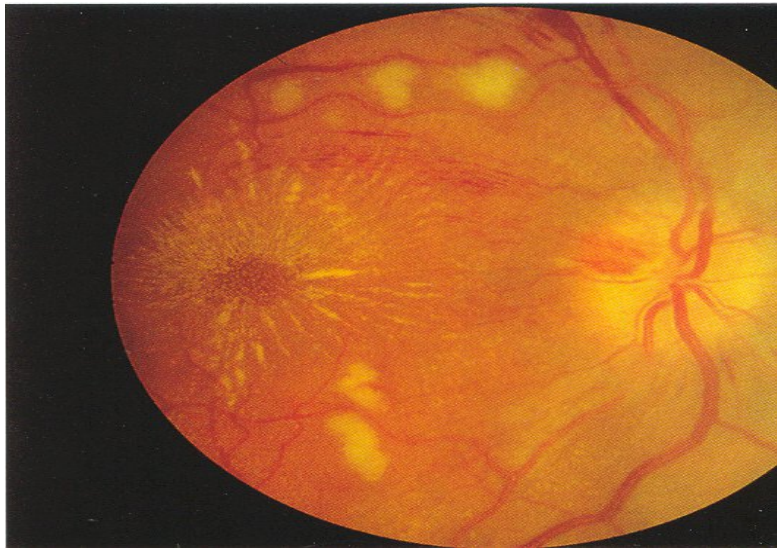
deflection of vein- Salus sign

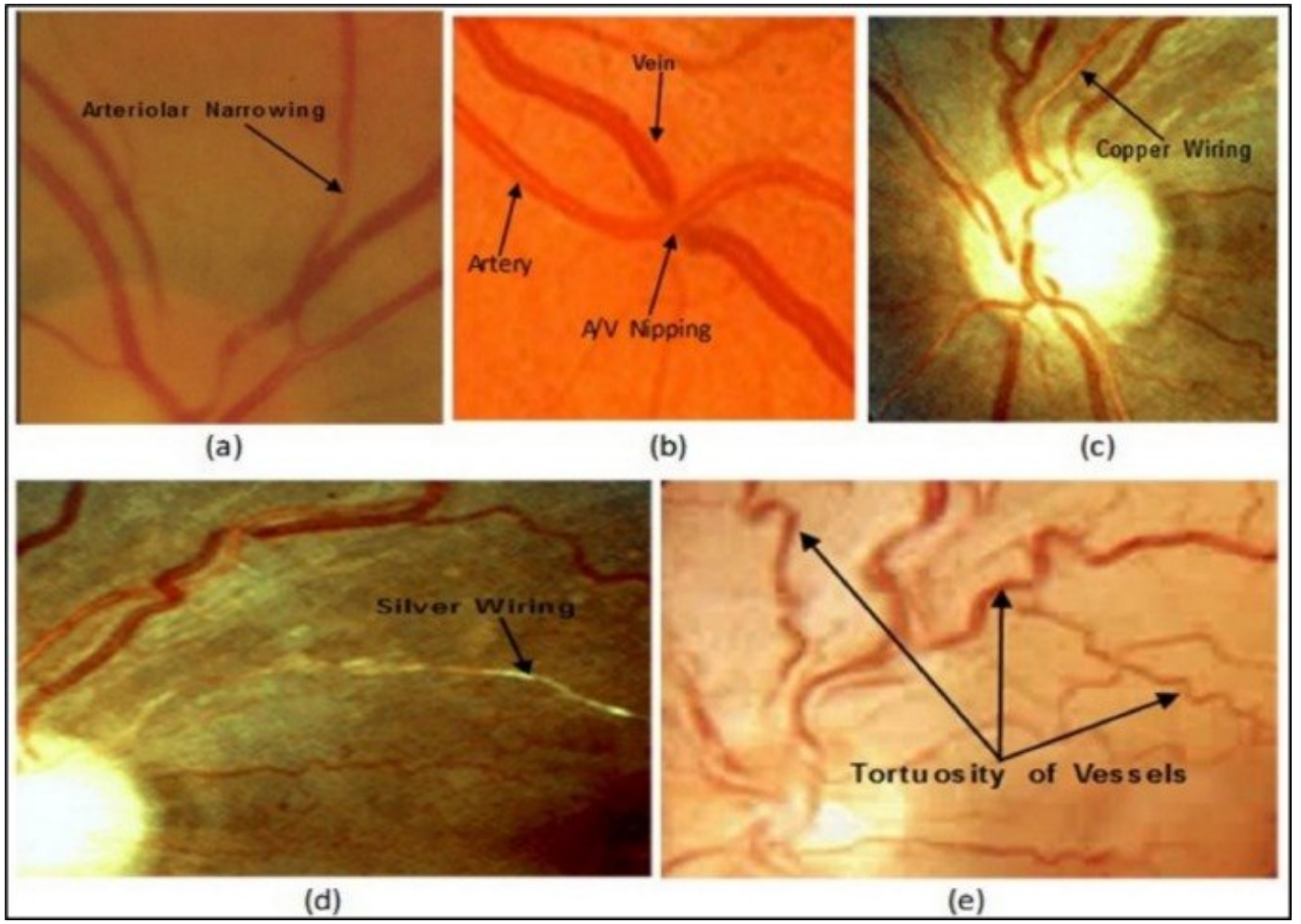
Banking of vein- Bonnet sign

Tapering of veins on either side- Gunns sign

-Copper wire arteries

-Silver wire arteries




Arteriolar Narrowing
Vein
Artery
A/V Nipping
Copper Wiring
(a)
(b)
(c)
Silver Wiring
Tortuosity of Vessels
(d)
(e)

KEITH WAGNER CLASSIFICATION

Grade- I Generalized narrowing of arterioles

Concealment of Veins

Grade- II Focal narrowing of arterioles

Deflection of Veins- Salu's sign

Grade- III Marked AV crossing changes

Copper / silver wire arteries

Retinal haemorrhages, Hard exudates

Cotton wool spots

Grade – IV Papilloedema

MODIFIED SCHEIES CLASSIFICATION

Staging of retinopathy

Grade- I Barely detectable narrowing of arterioles

Grade- II Obvious Focal narrowing of arterioles

Grade- III Grade II plus Retinal haemorrhages & exudates

Grade – IV Grade III plus disc oedema

MODIFIED SCHEIES CLASSIFICATION

Grading of light reflex/ Arteriosclerotic changes

Grade – I Increased of light reflex

Grade – II Marked AV crossing change

Grade – III Copper wire arteries

Grade - IV Silver wire arteries

MANAGEMENT

Complications – BRVO/ CRVO

Treatment – Good control of Hypertension

RETINOPATHY OF PREMATURITY

Bilateral proliferative retinopathy

Earlier called Retrolental fibroplasia

Risk Factors:

- √ Prematurity <32 weeks
- √ Low birth Weight < 1500 gm
- √ Supplemental oxygen therapy
- √ Others – Vit E deficiency, Respiratory distress, asphyxia, shock

PATHOGENESIS

Normally due to VEGF Retinal vessels reach:

Nasal periphery - 8 months POG

Temporal periphery – 1month post birth

Prematurity VEGF ↓ Vessels halted – after birth VEGF ↑ causes:

Neovascularization

Fibrous proliferation

INTERNATIONAL CLASSIFICATION OF ROP

Based on:

- Stage
- Zone
- Extent

STAGING OF ROP

STAGE 1 – Demarcation line

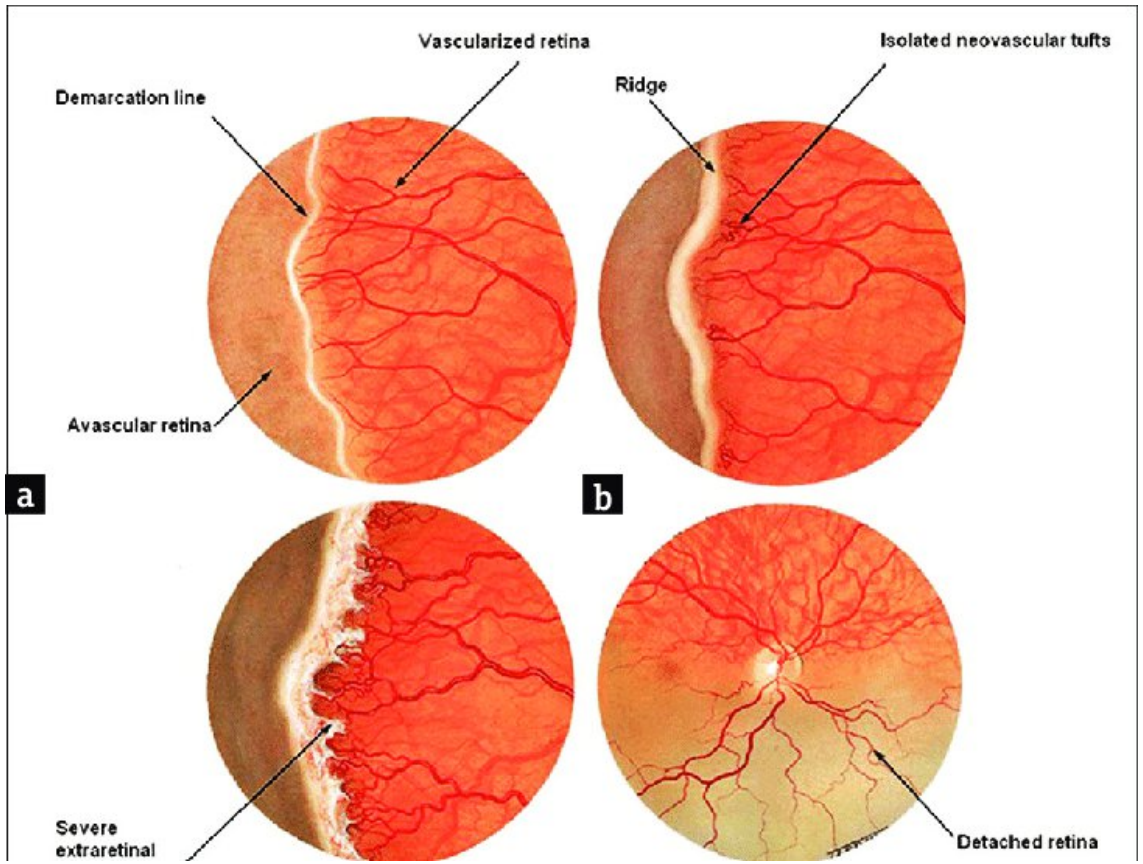
STAGE 2 – Ridge formed

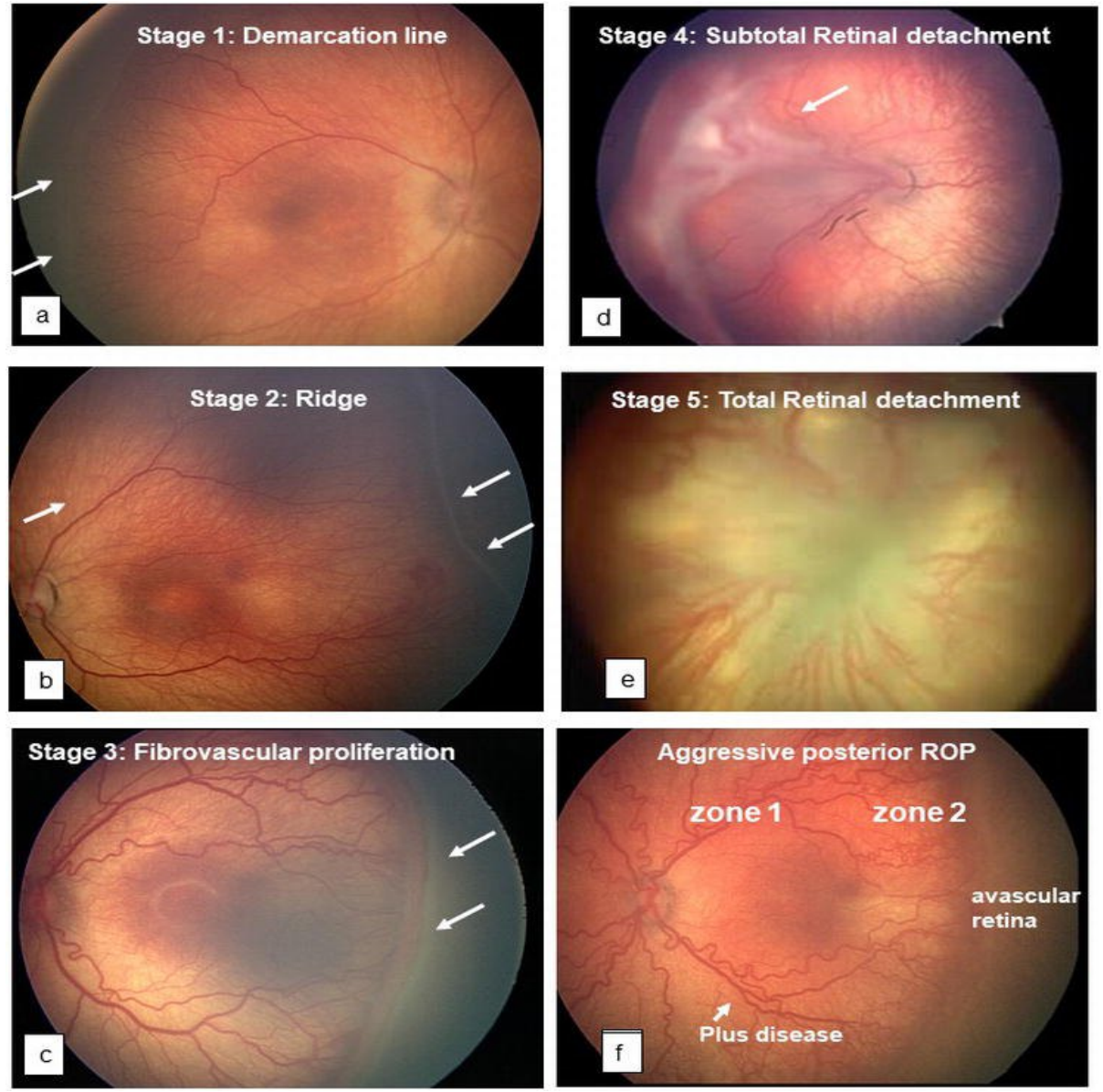
STAGE 3 – Ridge with extraretinal fibrovascular proliferation

STAGE 4a – Subtotal RD not involving macula

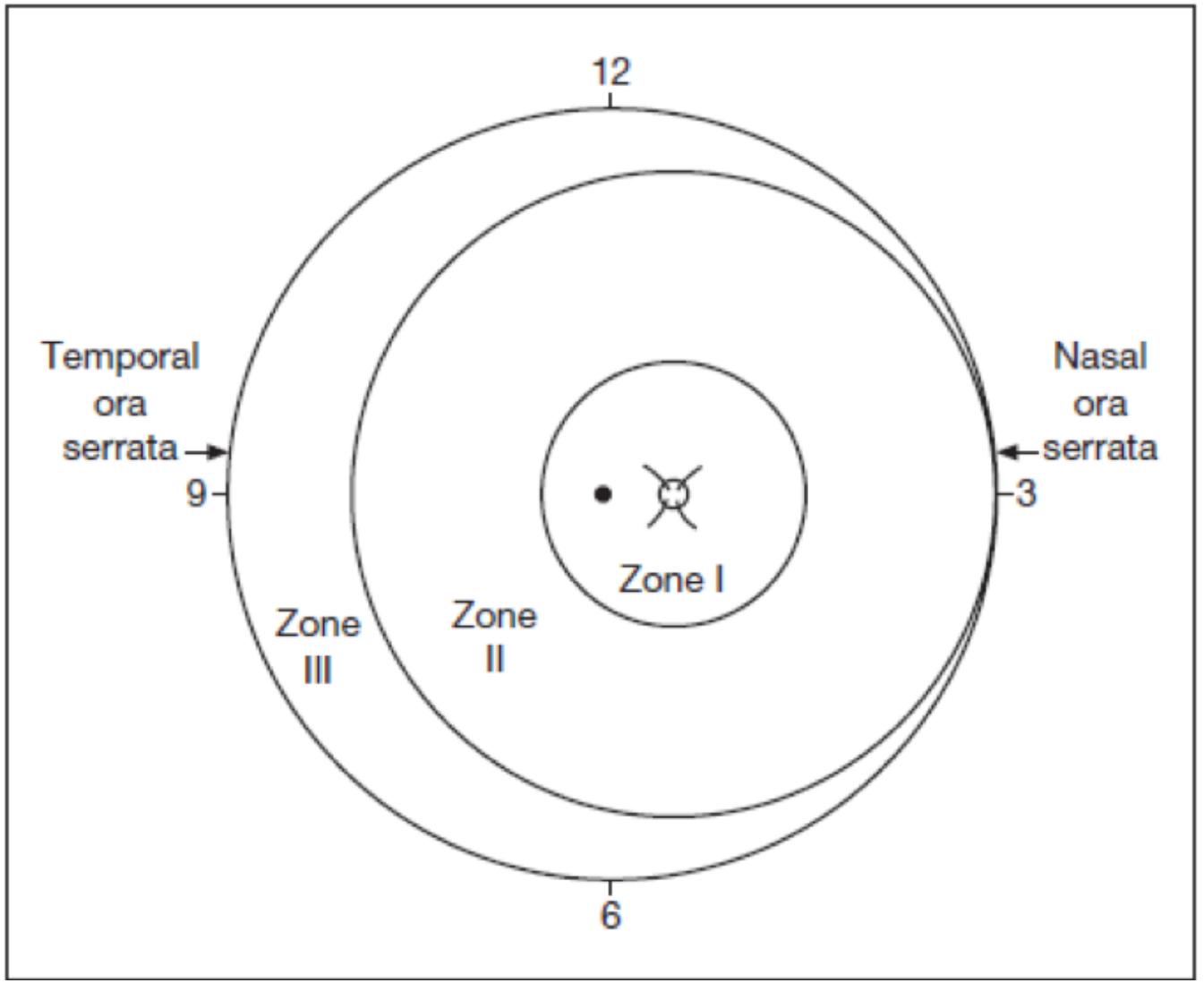
STAGE 4b – Subtotal RD involving macula

STAGE 5 – Total RD


Vascularized retina
Demarcation line
Avascular retina
a
Ridge
Isolated neovascular tufts
b
Severe
extraretinal
Detached retina

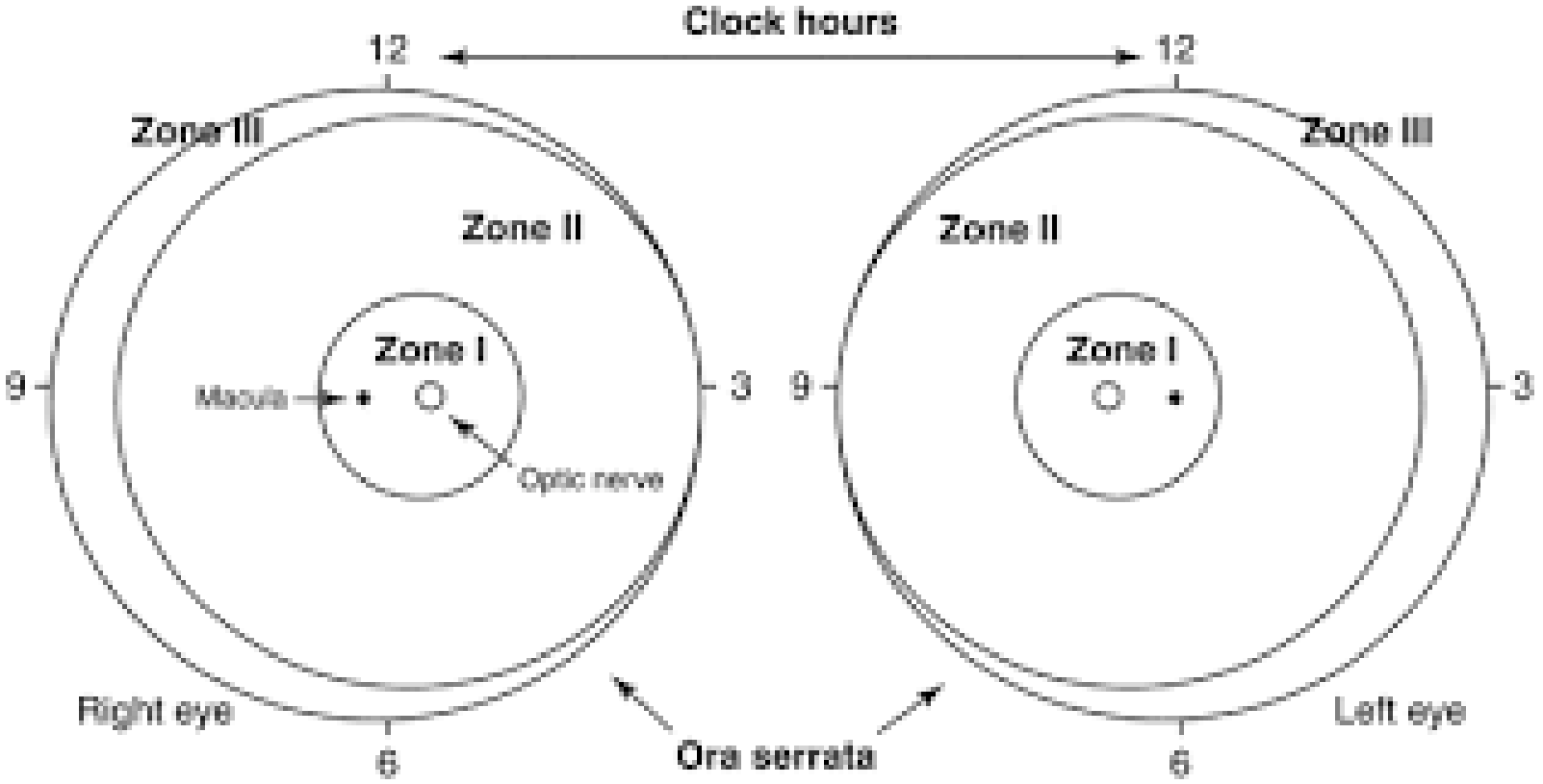

Stage 1: Demarcation line
a
Stage 4: Subtotal Retinal detachment
d
Stage 2: Ridge
b
Stage 5: Total Retinal detachment
e
Stage 3: Fibrovascular proliferation
c
Aggressive posterior ROP
zone 1
zone 2
avascular
retina
Plus disease
f

ZONES OF ROP


12
Temporal
ora
serrata
9
Nasal
ora
serrata
3
6
Zone I
Zone
II
Zone
III

EXTENT OF INVOLVEMENT

Denoted by the number of clock hours involved in a particular zone



MANAGEMENT

Prophylaxis

Early diagnosis and treatment

SCREENING PROTOCOL

In NICU for preterm babies 3- 4 weeks after birth/ 32 weeks post conception

Repeat examination every 1- 2 weeks till complete retinal vascularization

TREATMENT

LASER

532 nm frequency doubled Yag laser

INTRAVITREAL ANTI VEGF

No retinal destruction as in laser

Recurrence higher

VITREO RETINAL SURGERY for retinal detachment


THANK YOU