RETINOPATHIES

DIABETIC RETINOPATHY

DIABETIC RETINOPATHY

- It is the "disease of the retina" caused by microangiopathy due to long term effect of diabetes leading to progressive damage of the retina & blindness.

- Most common cause of severe bilateral visual loss in working age group.

Risk factors

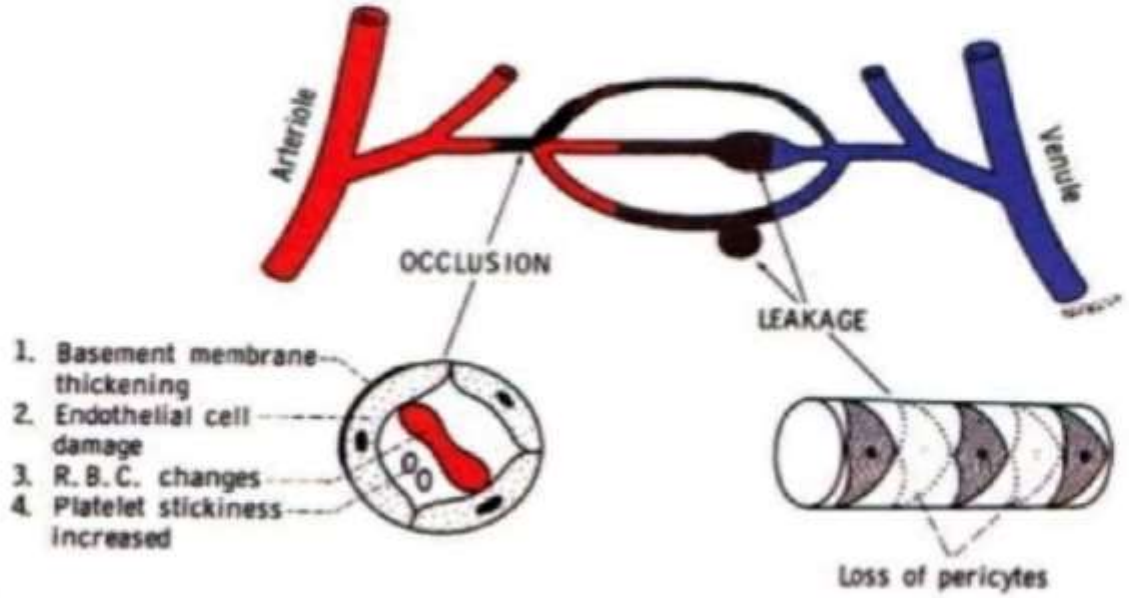
- Age at diagnosis of diabetes
- Duration of diabetes
- Poor metabolic control
- Pregnancy
- Hypertension
- Nephropathy
- Others - smoking, obesity, hyperlipidemia, anemia

Pathophysiology

- DR is a microangiopathy primarily affecting the precapillary arterioles, capillaries, venules and post capillary venules.

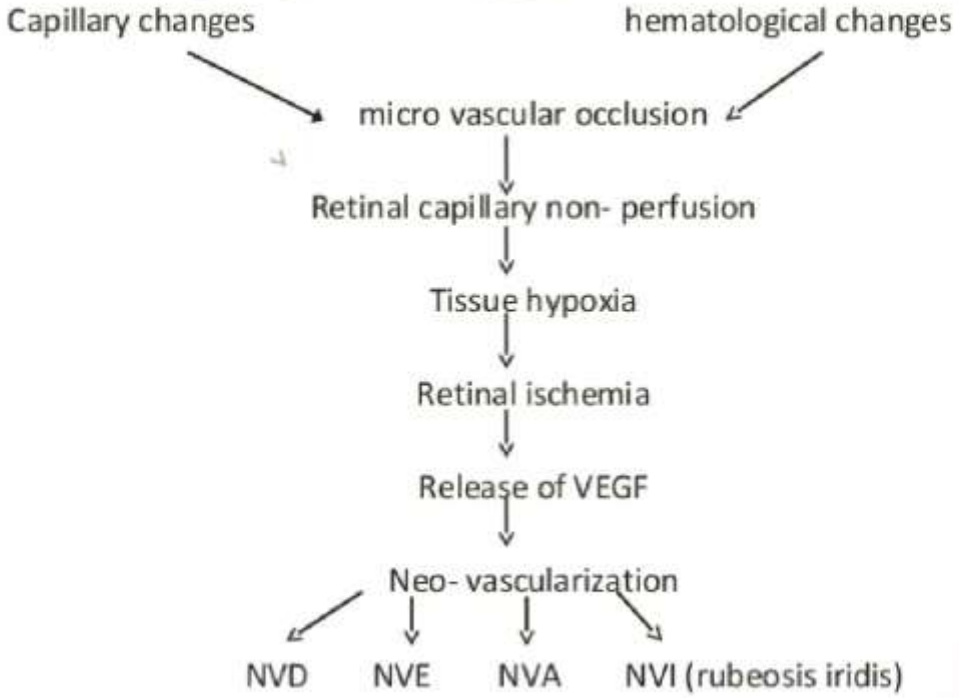
- The basic component of damaging process are microvascular occlusion & microvascular leakage.

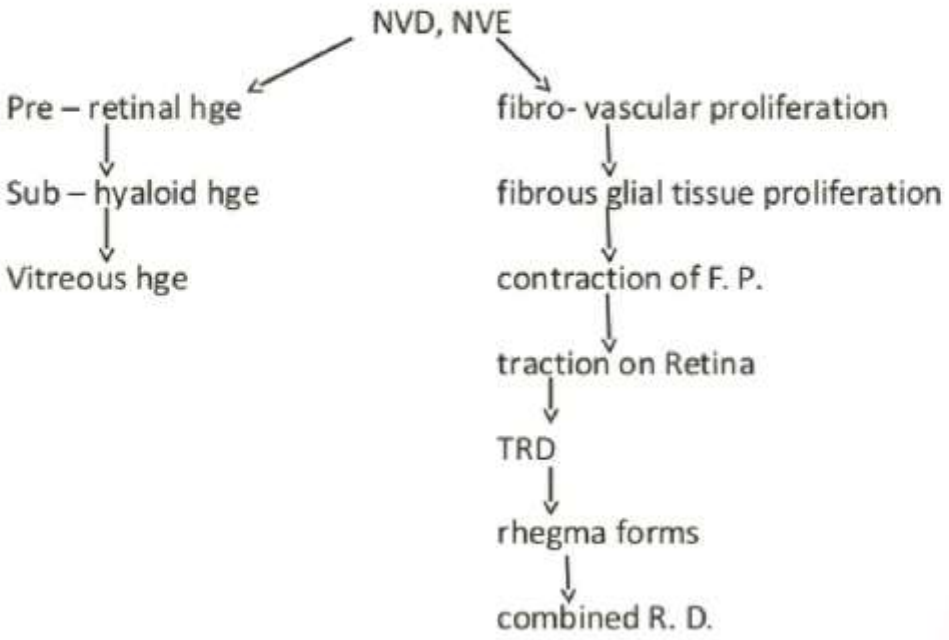
- Microvascular leakage
- Microvascular occlusion

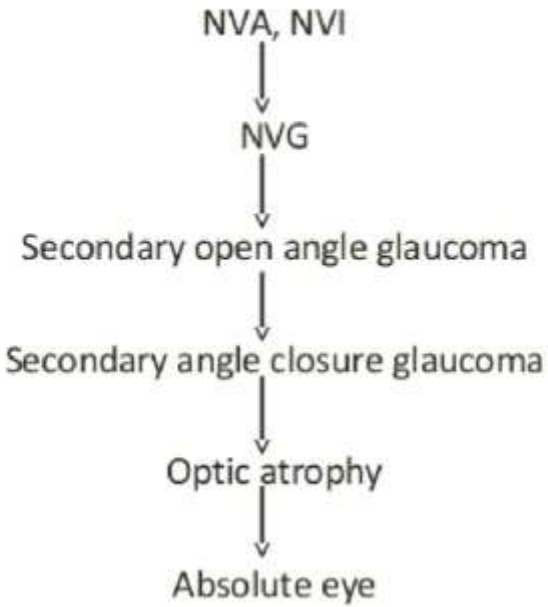


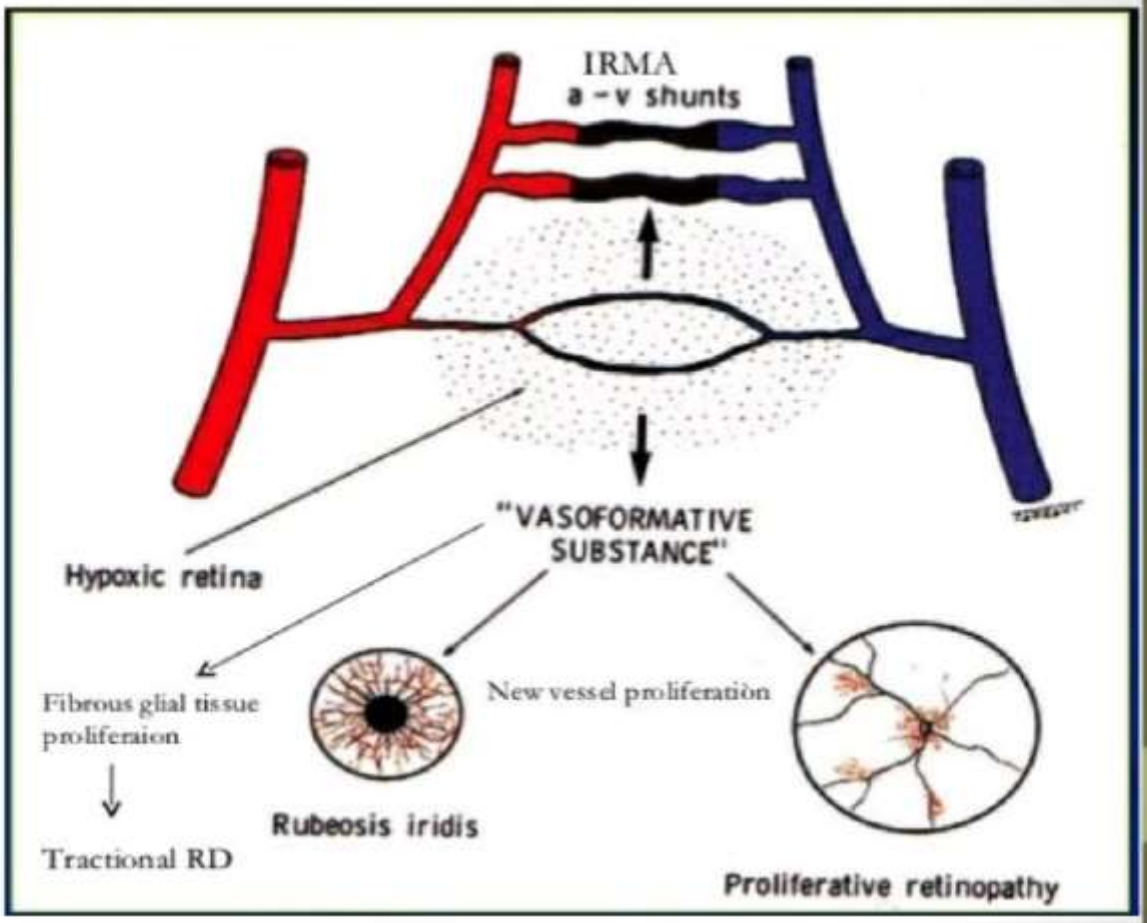
- **Capillary changes:**
  - Degeneration & loss of pericytes
  - Thickening of Basement membrane
  - Damage & proliferation of endothelial cells
- **Hematological changes:**
  - Deformation of RBC & rouleaux formation
  - Increased plasma viscosity
  - Increased platelet stickiness & aggregation

Pathophysiology

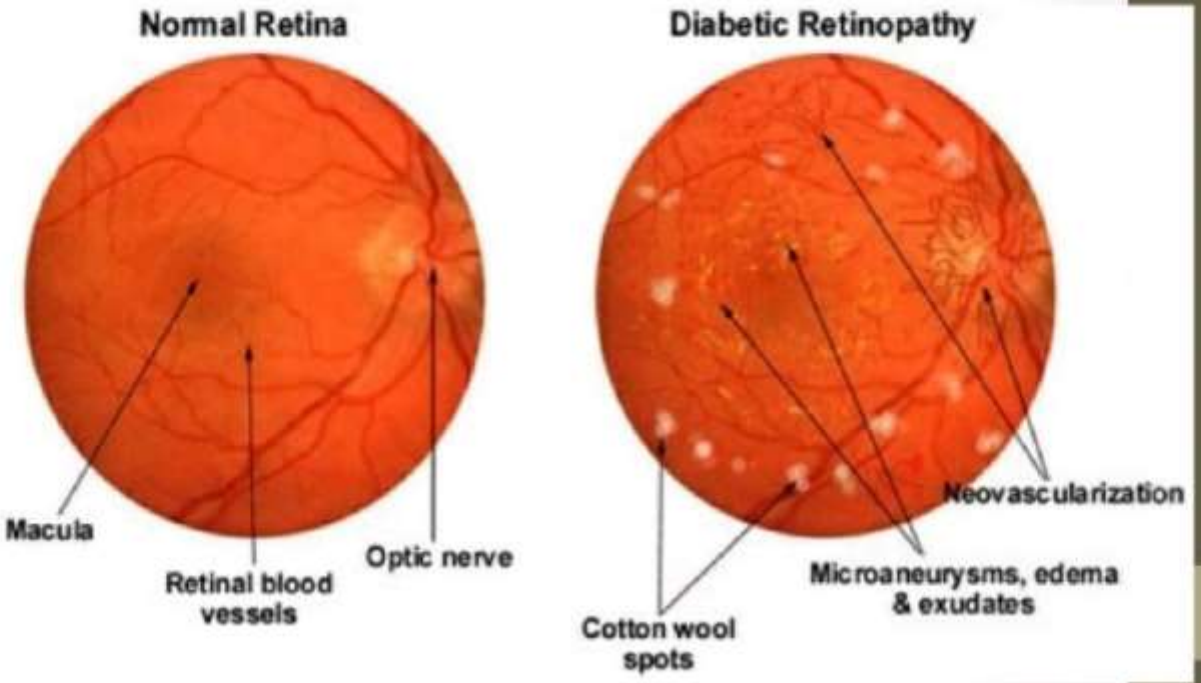



NVD, NVE
Pre – retinal hge
Sub – hyaloid hge
Vitreous hge
fibro- vascular proliferation
fibrous glial tissue proliferation
contraction of F. P.
traction on Retina
TRD
rhegma forms
combined R. D.


NVA, NVI
NVG
Secondary open angle glaucoma
Secondary angle closure glaucoma
Optic atrophy
Absolute eye

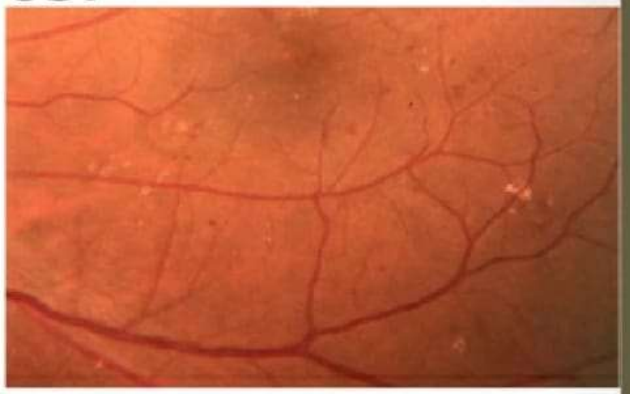

IRMA
a – v shunts
"VASOFORMATIVE SUBSTANCE"
Hypoxic retina
Fibrous glial tissue proliferaion
Tractional RD
Rubeosis iridis
New vessel proliferation
Proliferative retinopathy

Clinical Features:



Clinical features:

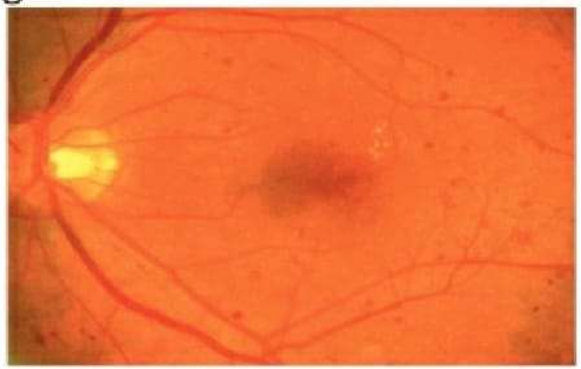
Microaneurysm:



- Earliest sign of D. R.
- Appear as tiny red dots.
- It is focal saccular dilatation of capillary walls where pericytes are absent.
- Located in the outer plexiform layer & inner nuclear layer.
- Usually at posterior pole, especially temporal to the fovea.
- In FFA – present at the edge of area of capillary non- perfusion (hyper fluorescence dots)

Retinal Hemorrhages:

Dot & blot hge:



- Due to rupture of wall of capillary or Microaneurysm, giving rise to intra retinal hge.
- If the hge is deep (i.e. in the inner layer of OPL) it is usually is round or oval (dot/ blot hge)

Retinal Hemorrhages:

Flame shaped Hge:

- Arise from superficial precapillary arterioles.
- the hge is more superficial & in the nerve fiber layer, it takes a flame/ splinter shape as they follow the architecture of the nerve fiber layer.

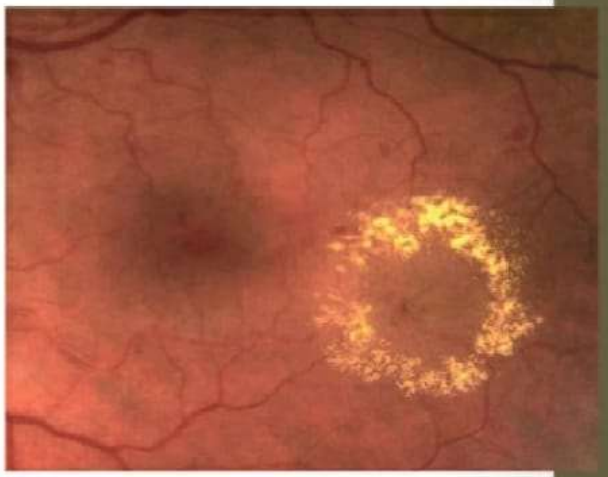
Exudates

Hard exudates:

- These are yellow deposits of lipid, lipoprotein & lipid filled macrophages within the outer plexiform & inner nuclear layer.
- They are arranged in clumps or form circinate pattern around the Microaneurysm frequently at posterior pole.
- These are sign of current/ previous Macular edema.

Cotton wool Spot (soft exudates):

- They are white fluffy lesion in nerve fiber layer.
- Caused by capillary occlusion at NFL due to infarction.

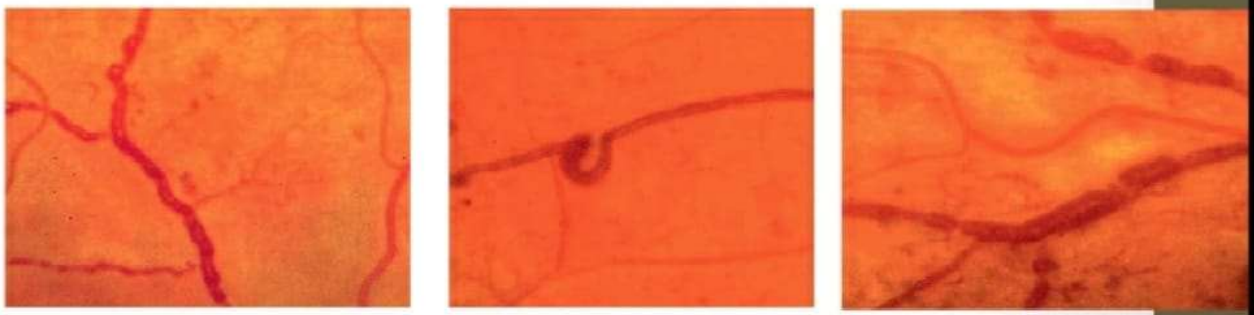


Intraretinal microvascular abnormality (IRMA):

- Abnormal dilated, tortuous retinal capillaries that act as a **shunt** between arterioles & venules.
- It's within the internal limiting membrane.

Venous changes:

In the form of "beading", "looping" & "severe segmentation" due to venous stagnation.



Classification

ETDRS CLASSIFICATION:

NON PROLIFERATIVE DIABETIC RETINOPATHY

MILD NPDR	MA, retinal hemorrhage, hard exudates
MODERATE NPDR	Mild NPDR plus cotton wool spots, venous changes & IRMA
SEVERE NPDR **4:2:1 rule**	Moderate NPDR plus one of – •MA, retinal hge in all four quadrants •Venous changes in 2/ more quadrants •IRMA atleast in 1 quadrant
VERY SEVERE NPDR	Two or more of the above features on severe NPDR

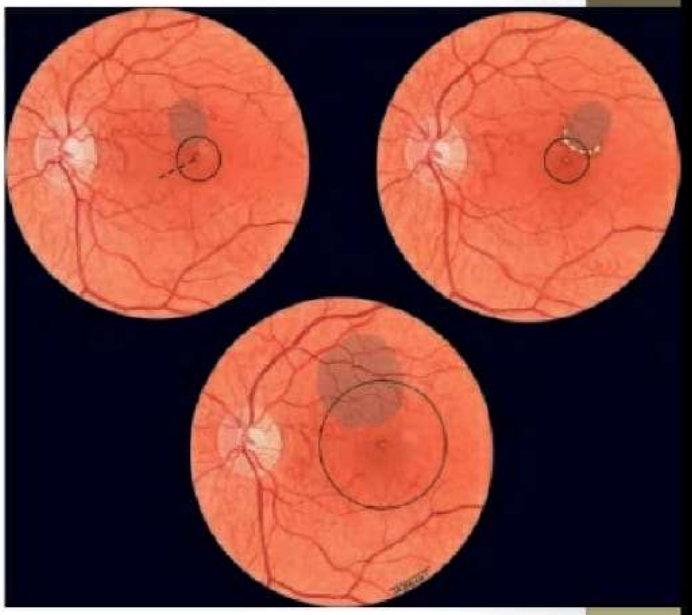
Proliferative diabetic retinopathy:

- early/ mild/ non – high risk PDR
- High risk:
  - New vessel on Disc:
    - More than 1/3rd of Optic disc diameter with/ without hge
    - Less than 1/3rd of optic disc diameter with hge (preretinal & vitreous hge)
  - New vessel elsewhere in fundus:
    - More than ½ of Optic disc diameter with/ without hge
    - Less than ½ of Optic disc diameter with hge. (preretinal & vitreous hge)

CLINICALLY SIGNIFICANT MACULAR EDEMA:

Modified Airlie- House criteria:

- Retinal edema within 500 micron of central fovea
- HE within 500 micron of fovea centralis associated with adjacent retinal thickening
- Retinal edema that is 1 disc diameter or larger, any part of which is with in 1 disc diameter of the fovea centralis.



Management

1) History from patient
2) Clinical features
3) Ocular examination
   - BCVA
   - Slit lamp examination
     - Cornea
     - Iris
     - lens
   - Funduscopy

Investigation:

- Laboratory test :
  - Blood sugar- FBS, RBS, 2HPPBS
  - Serum lipid profile
  - Medical evaluation of HbA1c
- Ancillary test
  - Color fundus photograph
  - Fundus Fluorescein Angiography
  - OCT

Treatment:

- LASER TREATMENT:
  - ✓ Focal laser
  - ✓ Grid laser
  - ✓ Pan retinal photocoagulation
- INTRA VITREAL INJECTION:
  - ✓ Anti- VEGF injection

Laser

Indication:

- Grid + focal laser – CSME
- PRP:
  - PDR --
  - ✓ High risk
  - ✓ Early PDR –
    - In pts with poor compliance
    - During pregnancy
    - Pt with systemic disease
    - Pending cataract surgery/ YAG laser capsulotomy
    - Rubeosis iridis
    - Severe/ very severe NPDR (irregular F/U)

Laser (M/A)

Focal laser:

- Burns only Microaneurysm

Grid laser:

- Around the edema except the Foveolar avascular zone.

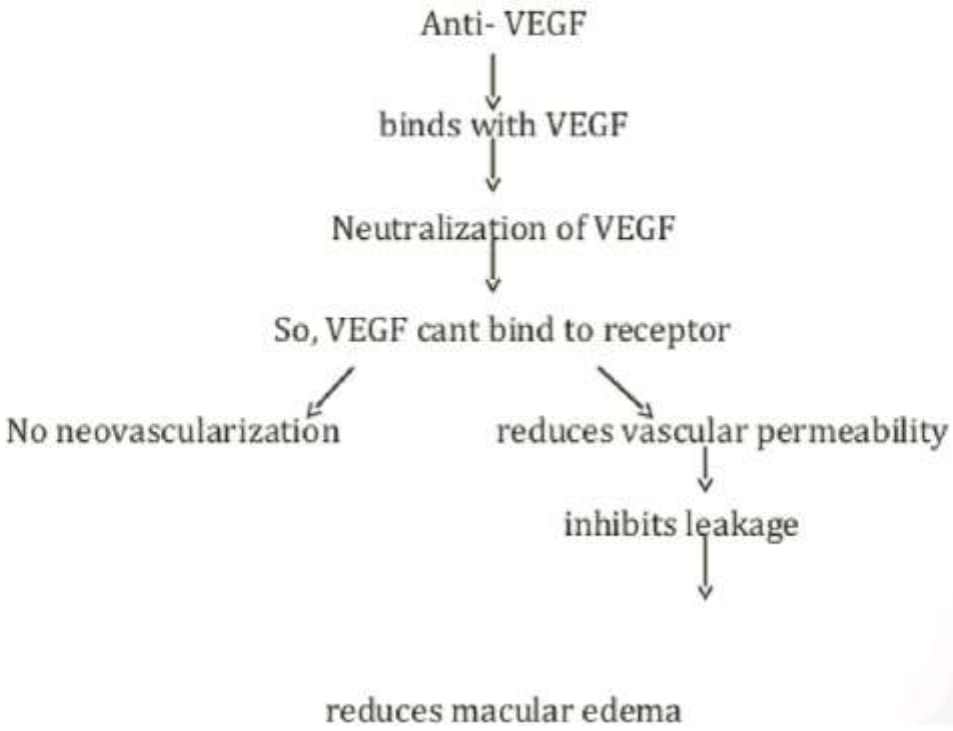
PRP:

- It burns the viable retina which is suffering from ischemia, thus reduces the oxygen demand of retina & prevents hypoxia.
- As it burns the ischemic retina, so reduces VEGF secretion by the ischemic retina.

Anti- VEGF

It's a humanized monoclonal antibody fragment.

M/A:



Anti- VEGF

Commonly used:

Bevacizumab (Avastin): dose – 1.25 mg &

Ranibizumab (Lucentis) : dose – 0.5 mg

Indication:

- NPDR with CSME
- PDR with CSME
- ARMD with CNVM
- CME
- CSCR
- NVG
- CRVO, BRVO

Complication:

- SCH
- Cataract
- Glaucoma
- Vitreous hemorrhage
- Endophthalmitis
- RD

"Diabetes can be controlled and does not have to keep people from achieving their dreams"

Michael Hunter

Conclusion

Diabetic Retinopathy is preventable through strict glycemic control and annual dialated eye exams by an opthalmologist.

VASCULAR DISORDERS OF RETINA

- **RETINAL ARTERY OCCLUSIONS**:
- ETIOLOGY-
- 1.Emboli- Emboli from carotid artery and those of cardiac origin are the most common cause of CRAO

  -cholesterol emboli
- -calcium emboli
- -platelet fibrin emboli
- 2.atherosclerotic –related thrombosis
- 3.retinal arteritis with obliteration
- 4.angiospasm
- 5.raised IOP
- 6.thrombophilic disorders

- **1.Central retinal artery occlusion:**
- It occurs due to obstruction at the level of lamina cribrosa
- Symptoms: sudden painless loss of vision occuring over seconds. Patient may give history of transient visual loss(amaurosis fugax).

- Signs:
- Visual acuity is markedly reduced.
- Direct pupillary reflex is absent and RAPD is positive.
- Fundus examination-
- Narrowing of retinal arteries
- Retina becomes milky white due to ischaemic oedema
- Cherry red spots in the centre of macula
- Cattle tracking
- Atrophic changes

- FFA (fundus fluoroscein angiography) -shows delay in arterial filing and masking of choroidal vasculature due to retinal oedema.

Fig. 12.7 *Fundus photograph showing marked retinal pallor in acute central retinal artery occlusion (CRAO): A, with sparing of the territory supplied by cilioretinal artery; and B, cherry-red spot in the absence of cilioretinal artery*

- **2.Branch retinal artery occlusion(BRAO)**
- It occurs following lodgment of embolus at a bifurcation.
- Retina distal to occlusion becomes oedematous with narrowed arterioles.
- Later on the involved area is atrophied leading to permanent sectoral visual field defect.

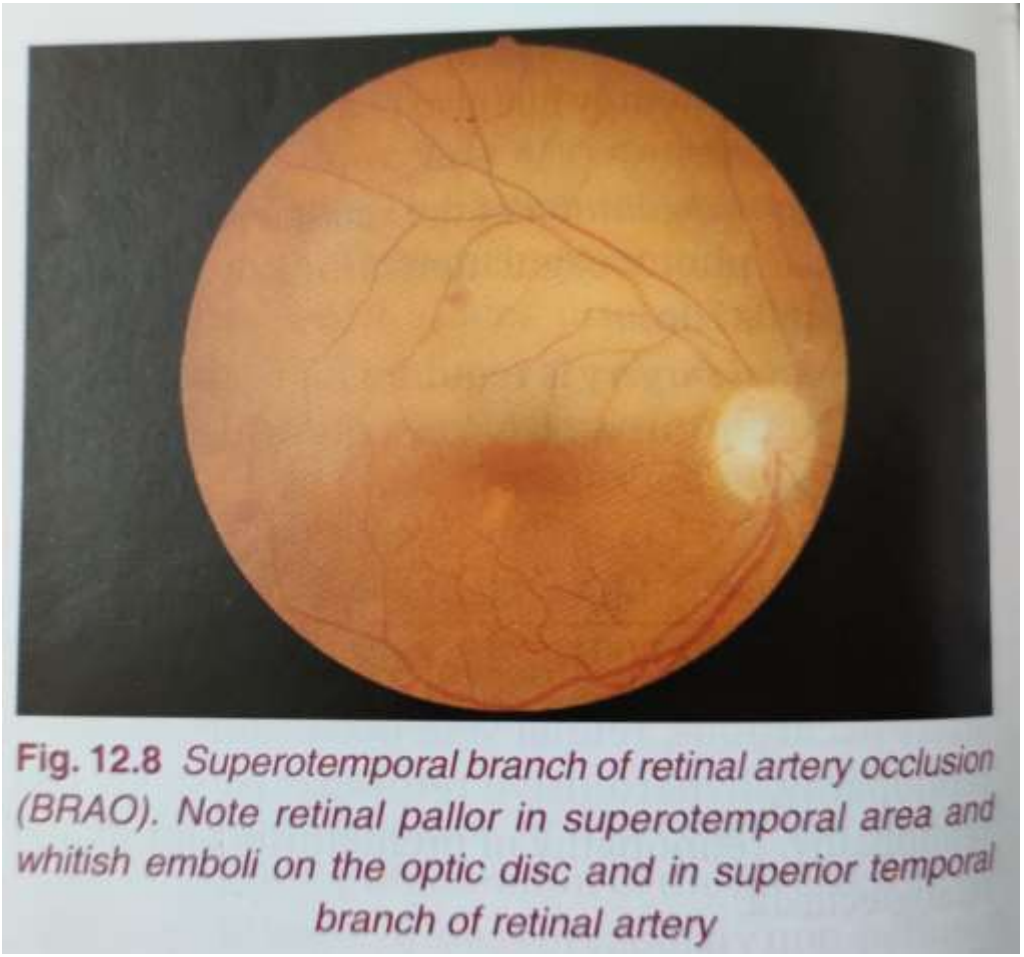


Fig. 12.8 *Superotemporal branch of retinal artery occlusion (BRAO). Note retinal pallor in superotemporal area and whitish emboli on the optic disc and in superior temporal branch of retinal artery*

- **MANAGEMENT**
- Treatment of CRAO is unsatisfactory, as retinal tissue cannot survive ischaemia for more than 90-100mins.
- A. Aggressive treatment of acute episode of CRAO

  should be done in all cases within 24 hrs of onset

- Immediate lowering of IOP
- Vasodilators and inhalation of mixture of 5% CO2 and 95% O2
- Fibrinolytic therapy
- Intravenous steroids
- Laser photo disruption
- **Complications**- neovascular glaucoma

- **Retinal vein occlusions**
- More common than artery occlusion
- Typically affects elderly patients.

- Etiology:
- 1.pressure on the vein by an atherosclerotic retinal artery
- 2.Hypertension and diabetes mellitus
- Hyperviscocity of blood
- Periphlebitis retinae
- Raised IOP
- Local causes

- Classification
- 1.CRAO –Ischaemic or non-ischaemic
- 2.BRVO

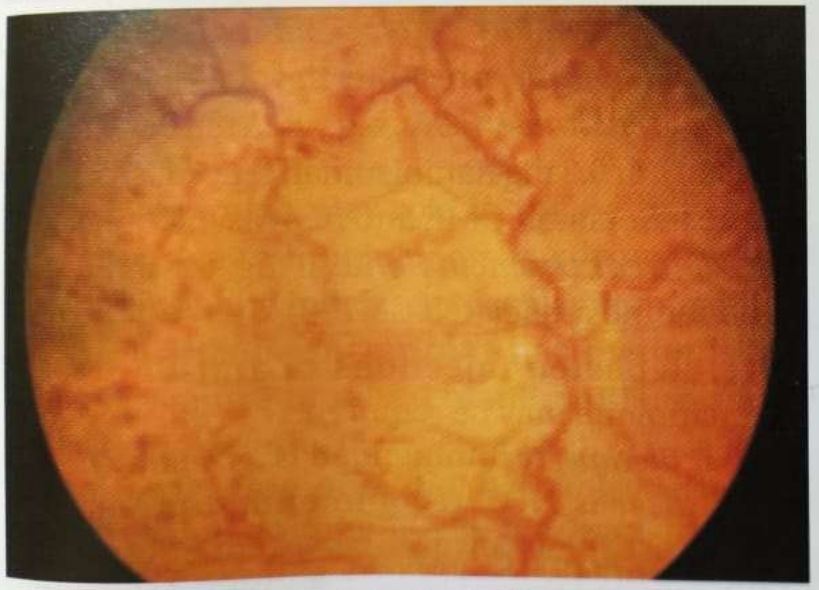


Fig. 12.9 *Central retinal vein occlusion (non-ischaemic)*

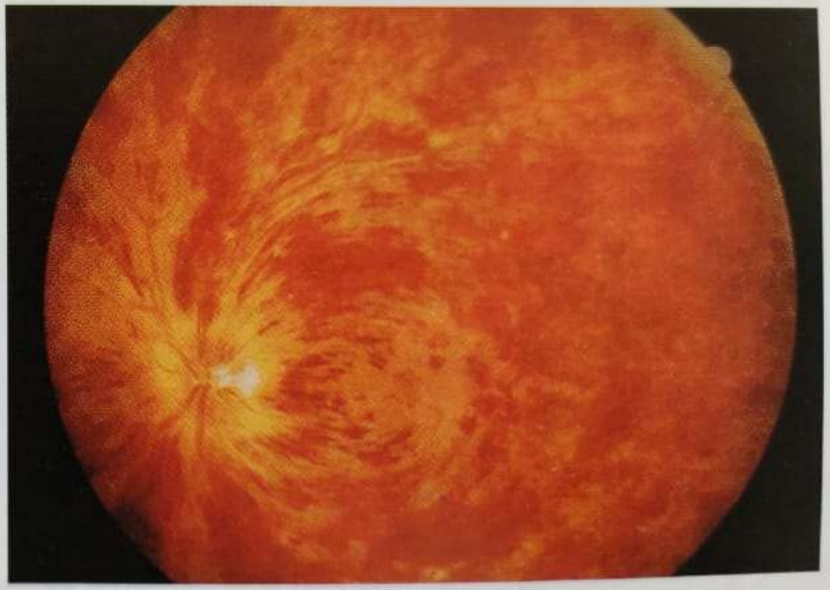


Fig. 12.10 *Central retinal vein occlusion (ischaemic)*

- **CRVO (central retinal vein occlusion)**
- **1.Non-ischaemic CRVO-**
- It is venous stasis retinopathy and characterized by mild to moderate visual loss and no RAPD.
- Early cases on fundus examination reveal mild venous congestion and tortuosity, few flame shaped haemorrhages,mild papilledema and mild or no macular edema.

- In late stages (after 6-9 months), there appears sheathing around the main veins, and few chorioretinal collaterals around the disc.
- Haemorrhages are partly absorbed .macula may show cystoid oedema.

- **2.Ischaemic CRVO**
- Ischaemic CRVO (haemorhagic retinopathy) refers to acute (sudden) complete occlusion of central retinal vein.
- It is characterized by marked sudden visual loss and RAPD.

- Early cases reveals:
- Massive engorgement, congestion and tortuosity of retinal veins
- Massive retinal haemorrhages (“splashed tomato” appearance).
- Cotton wool spots
- Disc shows oedema and hyperemia
- Macular area shows haemorrhages and sever edema
- Vitreous haemorrhages.

- In late stages, marked sheathing around veins and collaterals around the disc.
- Neovascularisation at the disc (NVD) or in the periphery (NVE).
- Macula shows marked pigmentary changes and cystoid edema.

- Pathognomic features for ischaemic CRVO differentiating it from non-ischaemic CRVO:

1. Presence of RAPD
2. Visual field defect
3. Reduced amplitude of b- wave of electroretinogram

Complications-Rubeosis iridis and neovascular glaucoma

- **Branch retinal vein occlusion (BRVO)**
- It is more common than the central retinal vein occlusion..it may occur as:
- Hemispheric occlusion
- Quandrantic occlusion
- Small branch occlusion

- Features:
- Retinal edema and haemorrhages
- Secondary glaucoma
- Chronic macular edema.

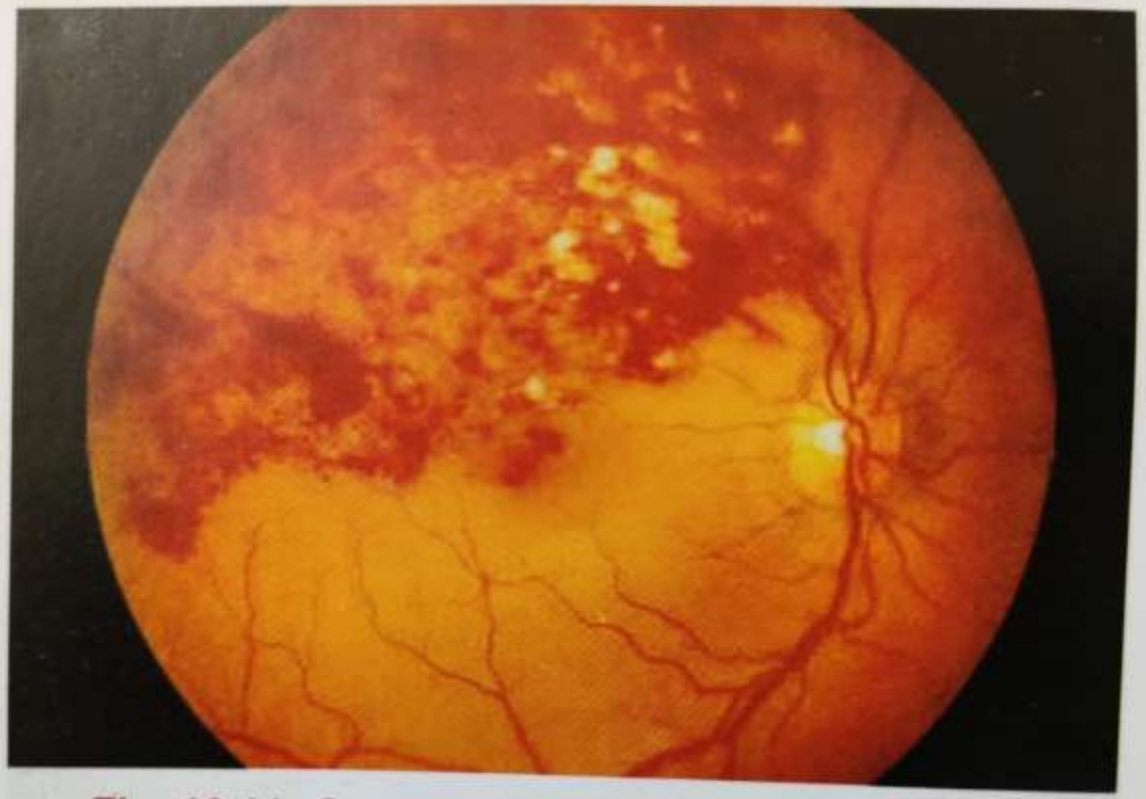


Fig. 12.11 *Superotemporal branch of retinal vein occlusion (BRVO)*

- **Management of retinal vein occlusion**
- OCULAR EXAMINATION:
- Visual acuity
- IOP recording
- undilated slit lamp examination to detect neovascularization of iris
- Gonioscopy
- Fundus examination

- Ocular investigations:
- Goldmann perimetry and ERG
- FFA
- OCT

SYSTEMIC EXAMINATIO AND INVESTIGATIONS:
Hypertension,DM, heart diseases, dyslipidaemia
Hypercoagulative conditions
homocysteinosis

- Differential diagnosis:
- Diabetic retinopathy is generally bilateral and CRVO is usually unilateral.

- Ocular ischaemic syndrome due to carotid occlusive diseasehas only dilated veins without tortuosity (in CRVO tortuosity is also seen) and retinal h’ges seen in mid periphery.

- Treatment:
- Treatment of systemic and ocular associations
- Observation and monitoring
- Ocular therapy
- Medical therapy-

  intravitreal anti-VEGF ; 1.25 mg Bevacizumab (avastin)

  intravitreal triamcinolone (1mg/0.1ml)

- Laser therapy:
- Grid laser
- Panretinal photocoagulation (PRP)
- Surgical therapy:

  Pars plana vitrectomy-

  persistant vitreous haemorrhages

  tractional retinal detachment

  ERM

  intractable neovascular glaucoma

HYPERTENSIVE RETINOPATHY

- Hypertensive retinopathy refers to fundus changes occurring in patients suffering from systemic HTN.

Clinical presentation include-

Retinopathy

Choroidopathy

Optic neuropathy

- Pathogenesis:
- Vasoconstriction- of retinal arterioles
- -of choroidal vessels
- - of peripapillary choroid
- Arteriosclerotic changes
- Increased vascular permeability

- Clinical types:

Chronic hypertensive retinopathy

Malignant or acute hypertensive retinopathy

- **Chronic hypertensive retinopathy**
- 1.hypetension with senile sclerosis
- 2.chronic hypertension with compensatory arteriolar sclerosis

- Fundus changes:
- Generalized arterial narrowing-
  vasoconstrictive phase
  sclerotic phase
- Focal arteriolar narrowing

- Arteriovenous nicking-
- this is the hallmark of hypertensive retinopathy. It occurs when arteriole crosses and compresses vein. also known as A-V crossing.
- Salu's sign-deflection of veins at the arteriovenous crossing.
- Bonnet sign-banking of veins distal to a-v crossing
- Gunn sign- tapering of veins in either side of crossing.

- Arteriolar reflex changes-
- Bright and thin, linear blood reflex
- More diffuse and less bright reflex
- Copper wiring (due to progressive sclerosis)
- Silver wiring(continued sclerosis)
- Superficial retinal haemorrhages (flame shaped)
- Hard exudates (yellowish waxy spots due to lipid deposits in the outer plexiform layer of retina)
- Cotton wool spots (fluffy white lesions which represent the areas of infarct in the nerve fiber layer)

- **Malignant acute hypertensive retinopathy**:
- There is an expression of its rapid progression to a serious degree in a patient with relatively young arterioles undefended by fibrosis.

- Fundus picture shows changes of acute hypertensive retinopathy, choroidopahty and optic neuropathy

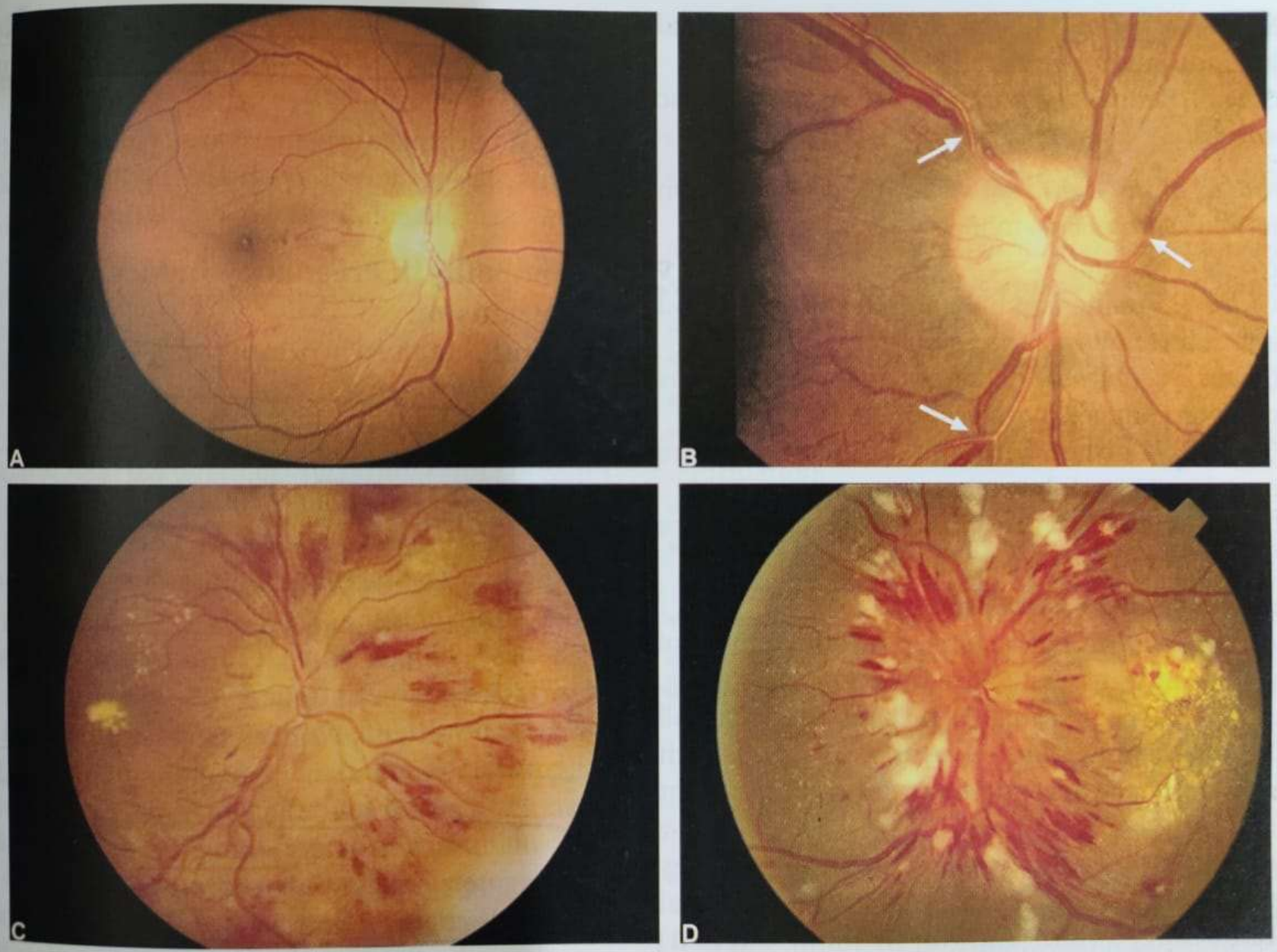


Fig. 12.12 *Hypertensive retinopathy: A, grade I; B, grade II; C, grade III; D, grade IV*

1 Acute hypertensive retinopathy

- Marked arteriolar narrowing
- Superficial retinal h’ges
- Focal intraretinal pariarteriolar transudates
- Cotton wool spots

2.Acute hypertensive choroidopaty

Acute focal retinal pigment epitheliopathy

Elsching’s spots

Siegrist streaks

Serous neurosensory retinal detachment

- 3.acute hypertensive optic neuropathy
- Disc edema and h’ges on optic disc and peripapillary retina
- Disc pallor

Staging of hypertensive retinopathy

- **Keith and wagner classification**

Grade I : mild generalised arteriolar attenuation,particularly of small branches, with broadening of the arteriolar light reflex and vein concealment.

Grade II : marked generalised narrowing and focal attenuation of arterioles associated with deflection of veins at a-v crossing (Salu's sign).

Grade III : grade II changes plus copper wiring of arterioles,banking of veins distal to a-v crossing (bonnet sign) ,tapering of veins on either side of the crossing (Gunn sign) and right angle deflection of veins(Salu's sign) .

flame shaped h'ges,cotton wool spots and hard exudates are also present

- Grade IV : all changes of grade III plus silver wiring of arterioles and papilledema.

- **Grading of the light reflex changes** resulting from arteriosclerosis is as follows-
- Grade 0 . Normal
- Grade1 . Broadening of light reflex with minimum a-v compression
- Grade2 . Light reflex changes & a-v crossing changes more prominent
- Grade3 . Copper wiring & more prominent a-v compression
- Grade4 . Silver wiring & sever a-v crossing

Management

- Mild hypertensive retinopathy requires blood pressure control only
- Moderate patients in addition to bp control may require cholesterol lowering agents
- Accelerated hypertensive retinopathy requires urgent antihypertensive management

Retinopathy in pregnancy induced HTN (PIH)

- PIH previously known as “toxaemia of pregnancy” is a disease of unknown etiology characterized by raised blood pressure, proteinuria and generalised edema.

- Retinal changes are liable to occur when BP is above 160/100 mmhg & are marked when BP raises above 200/130 mmhg.

- Earliest changes-narrowing of nasal arterioles f/b generalized narrowing.
- Severe persistent spasm of vessels causes retinal hypoxia gives appearance of ‘cotton wool spots’ & superficial h’ges
- Further progression of retinopathy occurs rapidly if pregnancy is allowed to continue
- Retinal edema and exudation may associated with ‘macular star’ or ‘flat macular detachment’
- Prognosis of retinal reattachment is good as it occurs spontaneously within few days of termination of pregnancy.

- Management :
- Changes of retinopathy are reversible and disappear after the delivery, unless organic vascular disease is established.

In preorganic stage- when patient responds well to conservative treatment, the pregnancy may justifiable be continued under close observation.

- Advent of hypoxic retinopathy should be considered an indication for termination of pregnancy; otherwise, permanent visual loss or even loss of life (of both mother and foetus) may occur.

THANK YOU