Diabetic retinopathy (DR)

It's a micro-angiopathy affecting the retinal arterioles, capillaries & venules. Always bilateral but asymmetrical.

**Etiology:** Unknown (DM more than 10yrs, so DR is related to duration of DM not related to severity of DM, so DR is common in insulin dependent = Juvenile DM).

**Risk factors:** - Young age & long duration.
  - Poor control of DM.
  - Coexisting hypertension.
  - High blood lipid level.
  - Pregnancy.

**Pathogenesis:**

**Micro-vascular leakage:**

Due to: loss of pericytes due to sorbitol $\rightarrow$ disturbance of blood retinal barrier

**Micro-vascular occlusion:** caused by:

1- Thickening of basement membrane.

2- Aggregation & stickiness of platelets.

3- Endothelial cell damage & proliferation

4- RBCs changes
Resulting in:

Retinal ischemia which release chemical (vasogenic factor which stimulate new vessels formation) → proliferative.

Clinical picture:

1- Non – proliferative (simple or Background) DR

Retinal Hemorrhage: dot Hemorrhage (Deep, rounded)

Retinal exudate: Deep hard exudates arranged in clumps or rings called circinate + retinal edema.

Micro-aneurysms.

2- Pre-proliferative DR:

Retinal Hemorrhage: Dots & blot More than 2 quadrants.

Retinal exudates: soft (cotton wool spots) and hard exudate & edema.

3- Proliferative DR:

Retinal exudates: Cotton wool.

Retinal vessels: neo-vessels (NV):

1- At the Disc → NVD (neovessels at disc).

2- At the Retina → NVE (neovessels at elsewhere).

Pre-retinal & Vitreous hemorrhage: Bleeding from the neo vessels which may organize → vitreous fibrosis & epi-retinal membranes

Tractional retinal detachment: Due to pull by the vitreous fibrosis & contraction of the epi-retinal membranes.
4- Advanced Diabetic Eye

Tractional RD (Retinitis proliferans).

Rubeosis Iridis that may lead to neo-vascular glaucoma.

5- Diabetic Maculopathy

Either:

a) Focal edema: from microaneurysm.

b) Diffuse edema (Cystoid Macular Edema): Fluorescence Angiography → flower petals.

c) Ischemic maculopathy

Treatment:

1- Medical Treatment:

Preproliferative (background)

- Control DM & hypertension.

- Prevent platelet stickiness → Aspirin (in small doses).

2- Argon Laser Treatment:

Indications:

- In pre. and proliferative DR & robeiosis iridis → do PRP (panretinal photocoagulation).

- In diabetic macular edema do

  - Focal laser (in focal edema).

  - Grid laser (in diffuse edema).
3- Intravitreal Triamcinolone acetonide injection.

4. Intravitreal AntiVEGF injection eg. Avastine or Lucentis

5- Vitrectomy+laser endophotocoagulation:
   Indications:
   1- Persistent vitreous hemorrhage for 1 month.
   2. Tractional RD.
   3- Persistent macular edema.

6- Neovascular glaucoma: as in CRVO.

Retinal detachment (RD)

It's a condition in which the retina is separated into 2 layers:

1- Retinal pigment epithelium. (RPE).

2- Sensory retina (as there is an embryological potential space between these 2 layers)

1. Rhegmatogenous RD

It's due to formation of a retinal tear, which allow liquefied vitreous to enter between the retinal layers causing retinal separation.

Risk Factors of Rhegmatogenous RD:

(a) Chorio-retinal degeneration as in high myopia.

(b) Blunt trauma.

(c) Aphakia.
(d) Family history of RD or history of RD in the fellow eye.

**Shape of retinal tears:**

1. Horse-shoe tear (the most common).
2. Retinal hole.
3. Arrow head tear.
4. Giant tear.

**Clinical picture:**

**Early:**

- **Flashes of light (photopsia):** Due to mechanical irritation of rods & cones by vitreous traction.

  - Floaters: *(Musca volitans):* Due to vitreous degeneration → opacities, minute hemorrhage from the tear into the vitreous.

  - Metamorphopsia, micropsia, macropsia.

**Late:**

- **Field defect (black curtain).**

- **Failure of vision (HM or PL vision):** (Painless & rapid) Due to foveal involvement (foveal detachment).

**Longstanding (Old)RD:** Shows

- Retinal thinning due to atrophy.
Treatment:

Prophylaxis:

Retinal tear → sealing.

-Indications:

  a) If patient is:

    - Myopic.
    - Aphakic.
    - R.D. in the others eye.

  b) Break:

    - Large.

    - Superior → spread rapidly by gravity.

Technique of sealing:

Most breaks are adequately treated by:

  * Argon laser:

    Especially if the media is clear.

  * Cryotherapy: (trans-scleral) especially if the media is Hazy or peripheral tear).

Curative:

  I-Sealing of the break:

    By -Cryotherapy.  -Laser
II: Approximation of the retina & choroid (reposition of the retina):

*Evacuate the Subretinal fluid (SRF):

*Scleral buckling:

Pushing the sclera by:

- Silicone implant: it is sutured on the sclera over the tear

*Intra-vitreal injection of air or expandable gases like sulphur hexafluoride

III: pars plane Vitrectomy + Intra-vitreal injection of silicon oil

2- Tractional RD

In which retina is pulled by vitreous fibrosis, as in:

- Cyclitic membrane, organized vitreous hemorrhage.

- Proliferative diabetic retinopathy (PDR).

- Retinopathy of prematurity (ROP).

- Penetrating post. Segment trauma: with vitreous loss & vitreous hemorrhage.

3- Exudative RD:

In which the retina is pushed by fluid derived from the choroid which gain access to the subretinal space through damaged RPE, as in:

- Choroiditis as in "Harada disease".

- Posterior scleritis.
- Neoplasm (M.M. of choroid or secondary's).

- Coat's disease.

- Systemic causes: toxaemia of pregnancy, malignant hypertension.

**TREATMENT:**

1. Tractional: Vitrectomy.

2. Exudative: Inflammatory (choroiditis & post. Scleritis) → give cortisone.

   - Malignant → Enucleation.

**Degeneration of the Retina**

**(I) Retinitis Pigmentosa**

It is heredo-familial, bilateral, progressive, pigmentation retinal degeneration of unknown etiology.

**Etiology:**

Unknown, may be:

1. Abiotrophy: ischemia & Vit A decrease.

2. Phototoxicity.

3. Hereditary: mode of inheritance may be,
   - Autosomal dominant.
   - Autosomal recessive.
   - X-linked (so common in boys).
Clinical pictures:

Symptoms:

- Night blindness (defective dark adaptation) due to affection of rods.
- Progressive visual field contraction.
- Finally, Complete loss of vision.

Signs:

1-Fundus Picture:

- Retina: Spider (Bone corpuscle) like pigmented spots at equator, then spread ant. & post.
- Vessels: Markedly attenuated.
- Disc: Consecutive optic atrophy (Waxy or pale disc).

2-Field changes:

- Early: ring (annular) scotoma: due to equatorial pigmentary degeneration

Investigations:

- ERG: is markedly diminished especially Scotopic ERG, it's affected early while photopic REG is affected later)
- EOG: subnormal.

Treatment:

1- Vitamin A. 2- Vasodilators.
3- Placental extract.
4. Low vision aid.

5. Genetic counseling.

**AGE REALTED MACULAR DEGENERATION (ARMD)**

It's a macular disease leads to severe affection of the central vision in old age above 50 yrs. Macular degeneration is diagnosed as either dry (non-neovascular) or wet (neovascular) which is refers to growth of new blood vessels in central part of retina called macula.

**Dry macular degeneration (non-neovascular)**- Dry AMD is an early stage of the disease and may result from the aging and thinning of macular tissues, depositing of pigment in the macula.

**Dry macular degeneration** is diagnosed when yellowish spots known as **drusen**, a small, yellow or white deposit in the eye.

**Wet macular degeneration (neovascular)**- with wet macular degeneration, new blood vessels grow beneath the retina and leak blood and fluid.

**Clinical pictures:**

1. **Age related maculopathy = drusen:**

   Small, discrete, yellow white, slightly elevated bilateral symmetrical spots.

2. **AMD with 2 main types:**

   1. **Dry(atrophic) type:**

      - There is slowly progressive geographic atrophy of photoreceptors, RPE & chorio capillaries.

      - Clinical pictures: gradual impairment of vision.
Fluorescence Angiography → Window defect.

Treatment: No effective treatment (stop smoking + treat hypertension) low vision aid may be helpful.

2-Wet type: due to abnormal neovascularization from the choroids (CNV) under retina grow through a defect in the Bruchs membrane (appears as gray-green lesion) → leakage.

Clinical pictures: sudden impairment of vision.

Treatment:

1) Argon laser photocoagulation: for well-defined extrafoveal CNV.

The aim it to destroy the CNV without damage to the fovea.

2) PTD (Photodynamic therapy): for subfoveal CNV.

3) Intravitreal injection of Avastin (Anti VEGF) & Triamcinolone actinoide.