433 Teams

OPHTHALMOLOGY

Ocular manifestations of systemic diseases

Color index:

432 Team – **Important** – **433 Notes** – **Not important**

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1- Diabetic retinopathy:

Introduction:

- Systemic diseases involved in Retina can lead to blindness. This can be prevented by early treatment and control of the systemic diseases.
- The most common disease that involved the eye (particularly Retina) is DIABETES.
- DM can cause retinopathy, acute angle closure glaucoma, cataract and III, IV and VI cranial nerve palsy.
- Most common ocular manifestation is retinopathy.
- Most common cause of blindness, diabetics have more than 25 times chance of getting blind than the normal people do.
- Almost in all people with type 1 diabetes mellitus. 'due to early onset'.
- Microvascular complications correlate significantly with DM.
- Hypertension, obesity and the control of blood sugar are risk factors that could be modified.
- Diabetic retinopathy is **most common microvascular complication of DM** and can lead to retinal degeneration.
- Because diabetic retinopathy often goes unnoticed until vision loss occurs, people with diabetes should get a comprehensive dilated eye exam at least once a year.
- Diabetic macular edema is a consequence of diabetic retinopathy that causes swelling in the area of the retina called the macula.
- Controlling diabetes—by taking medications, staying physically active, and maintaining a healthy diet—can prevent or delay or vision loss.
- The only means of preventing blindness due to diabetic retinopathy is through screening and early treatment.

Risk Factors:

1- **Duration of diabetes** (non-modifiable risk factor): The longer the duration of diabetes, the more risk of DR (By 15 years of diabetes, almost all patients come with DR).
2- **Poor control of diabetes** (most important modifiable risk factor): Raised HbA1c is associated with an increased risk of PDR.
3- **Pregnancy** **(poor pre-pregnancy control, too rapid tightening of control during early stages).**
4- **Hypertension:** should be controlled (<140/80 mmHg).
5- **Nephropathy** (if sever, it is associated with worsening of retinopathy): Renal transplantation may improve DR
6- **Others:** Hyperlipidemia, smoking, cataract surgery, obesity and anemia.

Pathogenesis:

Development of progressive vasculopathy characterized by dysfunction of endothelial cells lining retinal capillaries and the loss of the inner Blood-Retina-barrier. The inner Blood-Retina-barrier contains tight- junctions that reduce the space between adjacent endothelial cells lining the fine capillaries of the retinal microvasculature to form a selective and regulatable barrier. So, if there is a breakdown of the blood-retinal barrier, which normally prevents water movement in the retina, fluid (lipoprotein) will accumulate in the retinal tissue leading to macular edema, thickening and swelling which distorts central vision.
This will also result in the occlusion of the capillaries causing to hypoxia and ischemia. The retina will react by producing angiogenesis factors like the Vascular Endothelial Growth factor (VEGF) and they will induce more breakdown of inner blood retinal-barrier (increasing the hyper permeability) and induce the formation of new vessels.
At this stage of new vessels, the disease called proliferative disease. These new vessels are not healthy; they can bleed causing loss the vision from hemorrhage in the eye (Vitreous hemorrhage) and can lead to the formation of fibrous tissue causing traction in the retina (Retinal detachment). At the level of retinal microvasculature, the endothelial cells lining the capillaries become activated and they express adhesion of molecules (intercellular adhesion molecules (ICAMs) allowing the circulating leukocytes to come and adhere to the endothelial cells of the capillaries. This is known as leukostasis (inflammation in the diabetic retina). In the case of sever ischemia; new vessels not only form in the retina, but in the iris as well (Rubeosis iridis). This results in neovascular glaucoma.

Summary of pathogenesis: ☺

- Hyperglycemia lead to dysfunction of endothelial cells lining retinal capillaries and the loss of the inner Blood-Retina-barrier lead to edema.
- Occlusion of vessels produce VGEF and lead to neovascularization

Ocular complications of diabetes:

• Common

- **Retinopathy:** **Most common one**
- Iridopathy (minor iris transillumination defects). Unstable refraction.
- **Iridocyclitis** (another term of uveitis)
- **Optic neuropathy:** diabetics have more than 25 times chance to get blindness

• Uncommon

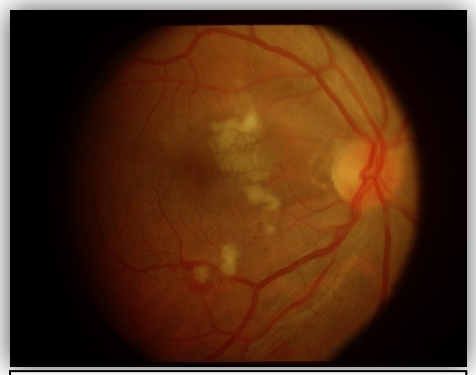
- Recurrent styes.
- Xanthelasma.
- Accelerated senile cataract.
- **Rubeosis iridis** (neovascularization in iris) "May lead to Neovascular glaucoma (NVG)"
- Ocular motor **nerve palsies (3^{rd},4^{th},6^{th}).**
- Reduced corneal sensitivity.

• Rare

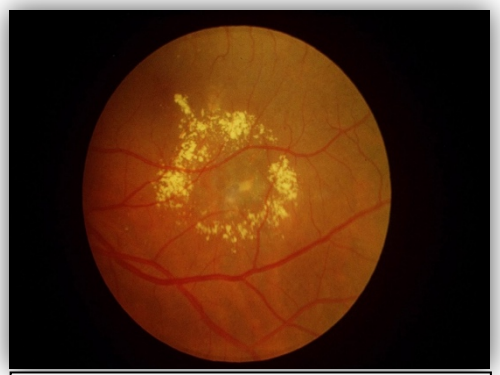
- Papillopathy, pupillary light-near dissociation.
- Wolfram syndrome (progressive optic atrophy and multiple).
- Neurological and systemic abnormalities, acute-onset.
- **Cataract**, rhino-orbital mucormycosis.

RETINAL CHANGES:

1- **Hard exudates** on the retina and **macular edema** resulted from the leakage.
2- **microaneurysms** 'blots and dots'
3- **soft exudates** 'cotton-wool spots'.
4- **Venous changes:** beading vs looping (The most reliable signs of retinal ischemia in diabetic retinopathy).
5- **hemorrhage** if neovascularization results in weak vessels and easy to break.
6- **Neovascularization**



Cotton wall spot



Hard exudate

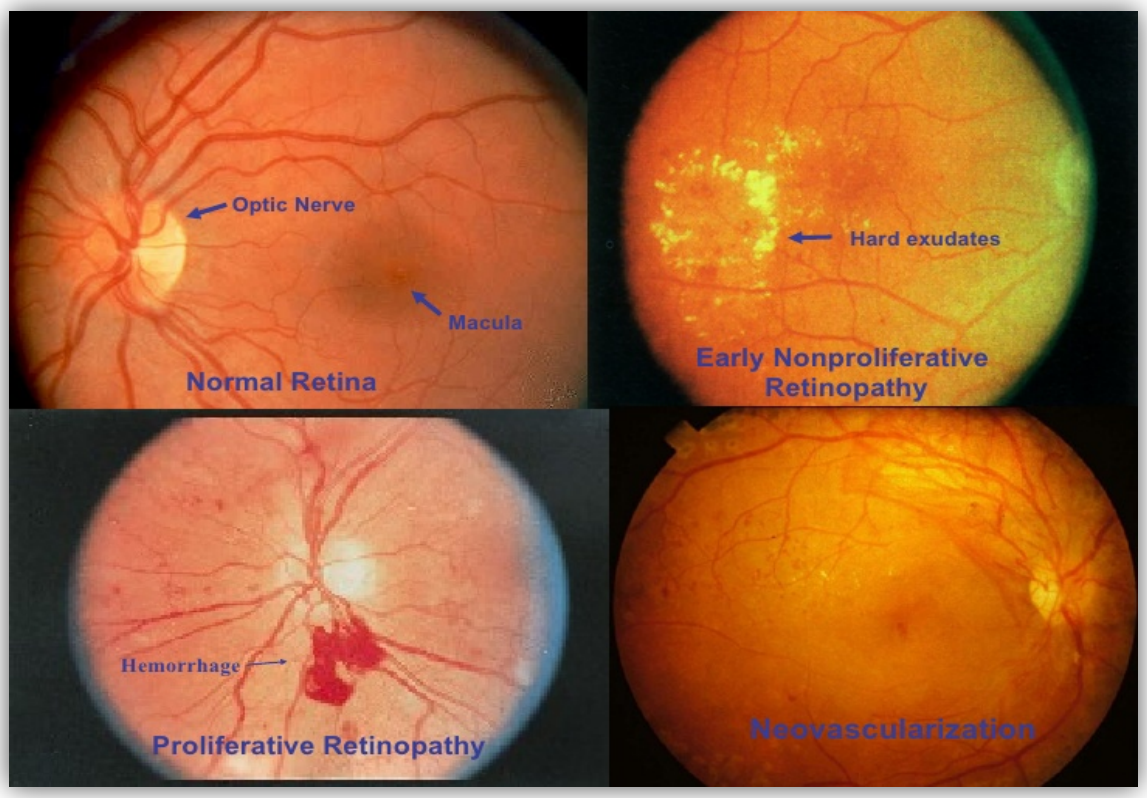


image 1: show the difference between normal eye and stages of diabetic retinopathy

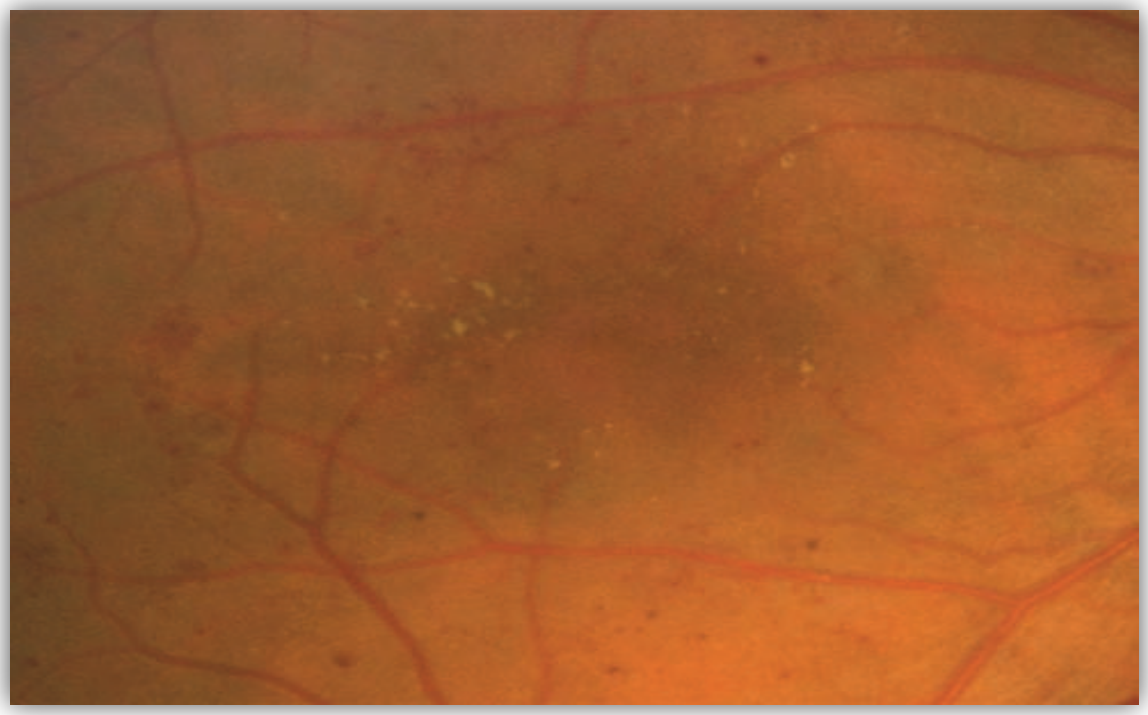


image2: we can see that there are small exudates with microaneurysims.

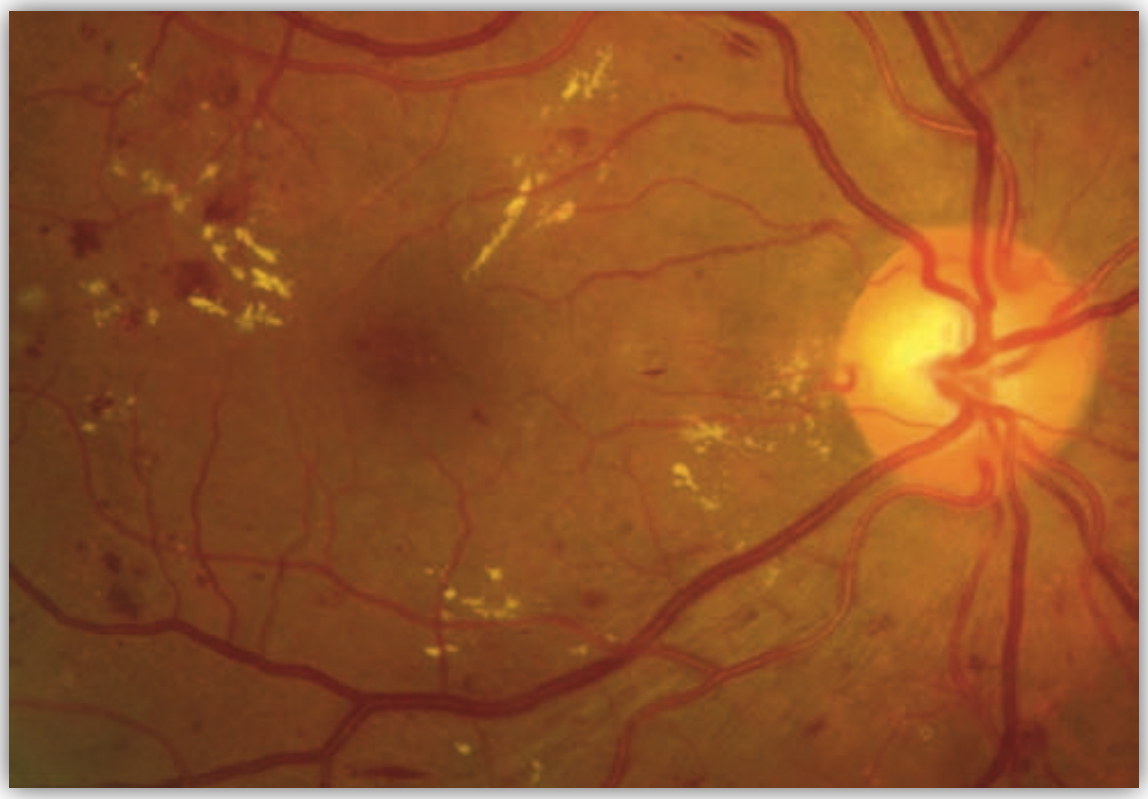


image3: more extensive exudates some associated with microaneurysims.

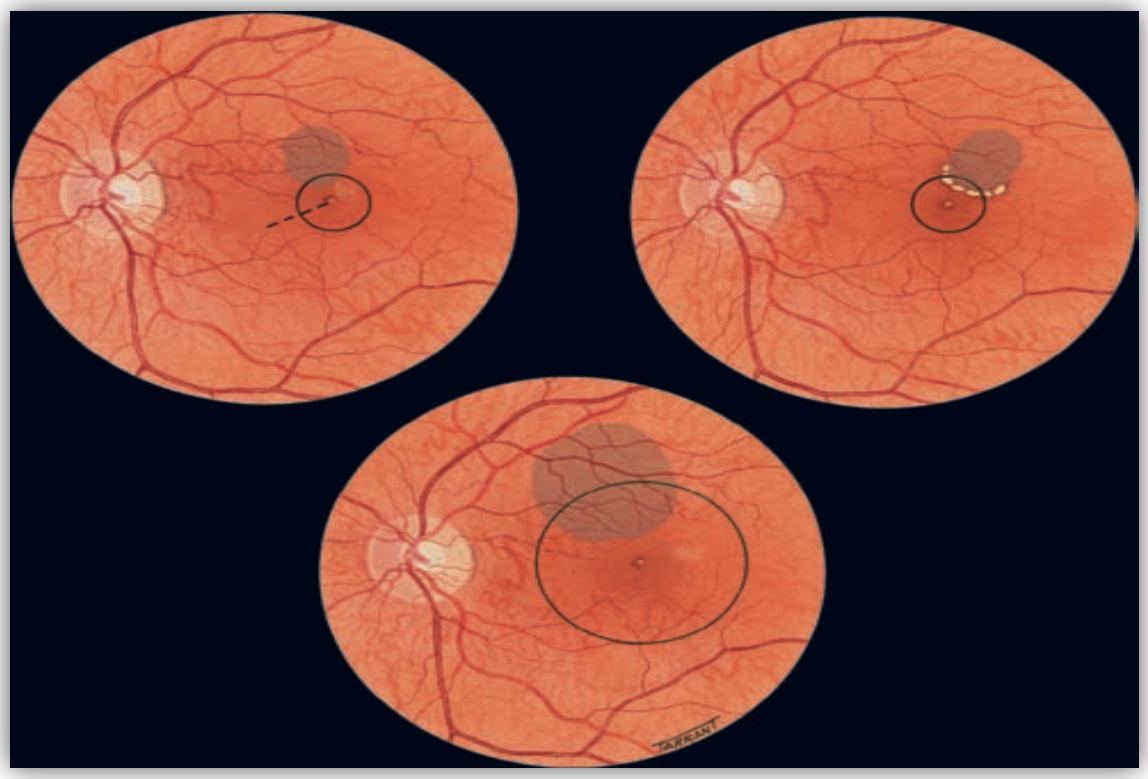


image4: we can see significant macular edema progression.

Non-proliferative diabetic retinopathy:

Mild NPDR: (Control blood sugar)
Microaneurysmus only
Moderate NPDR: (Control blood sugar)
Microaneurysms – retinal haemorrhages - circumstances exudates – cotton wool spots – minimal intraretinal microvascular anomalies (arterio-venous shunt) – minimal venous changes (looping and beading)
Severe NPDR (important): (Control blood sugar + Consider panretinal laser coagulation)
All the above + severe intraretinal microvascular anomalies (arterio-venous shunt) – severe venous changes (looping and beading)
Does not cause hemorrhage (no new vessels); we see only sign of ischemia.

Diabetic macular edema 'DMO':

-The hard exudate here is in the center of the macula (can cause a loss of central vision). It's a sign of macular edema.

-Nowadays, Optic coherence tomography (OCT) is used to diagnose early macular edema even in patients with very mild edema that you can't see it.

-Treatment of diabetic macular edema (DMO):

1- **Focal laser photocoagulation:**

Treatment of micro aneurysms with laser (After 6 months there will be a scar from the laser treatment but no hard exudate). **Very effective in controlling diabetic macular edema.**

2- **Intravitreal injection of anti-VEGF agents** (anti- vascular endothelial growth factor): **(Ranibizumab or Bevacizumab)** In addition to laser, we frequently inject Intravitreal injection of anti-VEGF agents to help control diabetic macular edema.

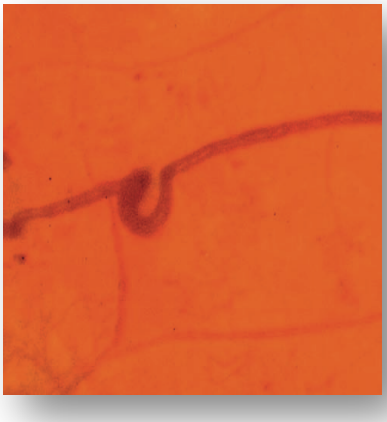
* Diabetic retinopathy patients have progressive macular vascular occlusion which will cause ischemia of retina (hypoxia).

Signs of Retinal Ischemia (Patient is at high risk to develop new vessels):

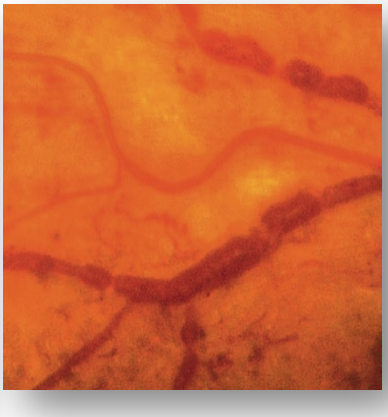
At this stage, sever non-proliferative diabetic retinopathy (Non-PDR). Patient should receive laser Panretinal photocoagulation.

- **Cotton wool spots** (less important sign and not used in the classification of non-PRD).
- **Venous changes** (The most reliable signs of retinal ischemia in diabetic retinopathy).
- **Intraretinal microvascular abnormalities (IRMA):** are arteriolar–venular shunts that run from retinal arterioles to venules, thus bypassing the capillary bed and are therefore often seen adjacent to areas of marked capillary hypoperfusion. * Dilated telangiectatic capillaries.
- **Intraretinal Hemorrhage:** The extent of involvement is a significant marker of the likelihood of progression to proliferative diabetic retinopathy.

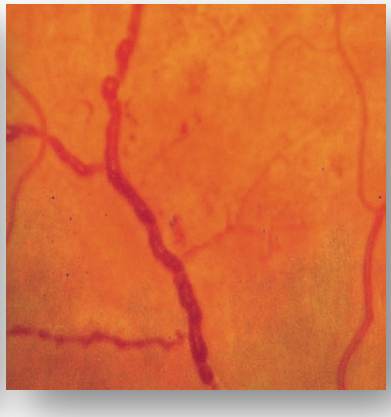
Venous changes:



Looping



Beading



Severe segmentation

Proliferative diabetic retinopathy: (important)

There are new vessels. *The only means of **preventing blindness** due to diabetic retinopathy is through **screening and early treatment.**

Fibrous tissue, initially fine, gradually develops in association as vessels increase in size. They are most commonly seen at the posterior pole:

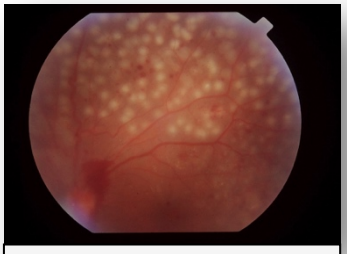
- Ø **New vessels at the disc (NVD):** describes neovascularization on or within one disc diameter of the optic nerve head.
- Ø **New vessels elsewhere (NVE):** describes neovascularization further away from the disc; it may be associated with fibrosis if long-standing.

  Ø **New vessels on the iris (NVI):** also known as rubeosis iridis, carry a high likelihood of progression to neovascular glaucoma.

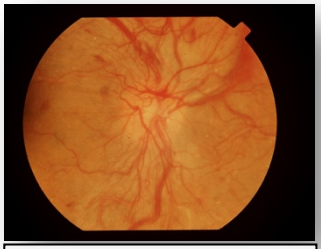
-Treatment:

1- Scatter laser treatment **pan-retinal photocoagulation.** (The mainstay of PDR treatment) *Protect the patient from blindness. *Apply scattered laser beams throughout the retina starting from the vascular arched, don't reach the macula and extend up to the periphery.

2- Intravitreal anti-VEGF injection.



Pan-retinal photocoagulation



Neovascularization

Complications of Retinal photocoagulation:

1- Anterior segment complications such as corneal or lenticular opacification.
2- Transient visual loss.
3- Photocoagulation of the fovea.
4- Macular edema.
5- Hemorrhage.
6- Choroidal Effusion.
7- Color vision alterations.
8- Visual field defects and night vision problems.
9- Hemeralopia.

❖ The three most common causes of intraocular inflammation in the kingdom:

1) Vogt–Koyanagi–Harada (VKH) syndrome
2) Tuberculosis
3) Behçet disease

2-Grave's disease:

Introduction:

- Most common cause of both **bilateral and unilateral proptosis** in an adult

Pathogenesis:

- Autoimmune disease characterized with serum IgG antibodies bind to TSH receptors in the thyroid and causes overstimulation and high thyroid hormone production.
- Autoimmune antibodies infiltrate eye, **cause inflammation of extraocular muscles and associated with increased secretion of glycosaminoglycans and osmotic imbibition of water.**

Risk factors:

Smoking (most important) – family history

Systematic manifestation:

Pretibial myxedema, heat intolerance, weight loss etc…

Ocular manifestation:

Lid retraction - Proptosis and exophthalmos - Lid lag – restrictive myopathy Limitation of extraocular muscles movements - Infiltration of hormones in the globe.

*Also other symptoms and signs of hyperthyroidism e.g. pretibial myxedema, heat itolerance, weight loss etc…

Investigations:

1- Thyroid function test: High T3, T4 and low TSH

2- Visual evoked potential: To exclude Optic neuropathy

Treatment:

1- Anti-thyroid medications or Thyroid ablation with radioactive iodine **(for disease itself)**

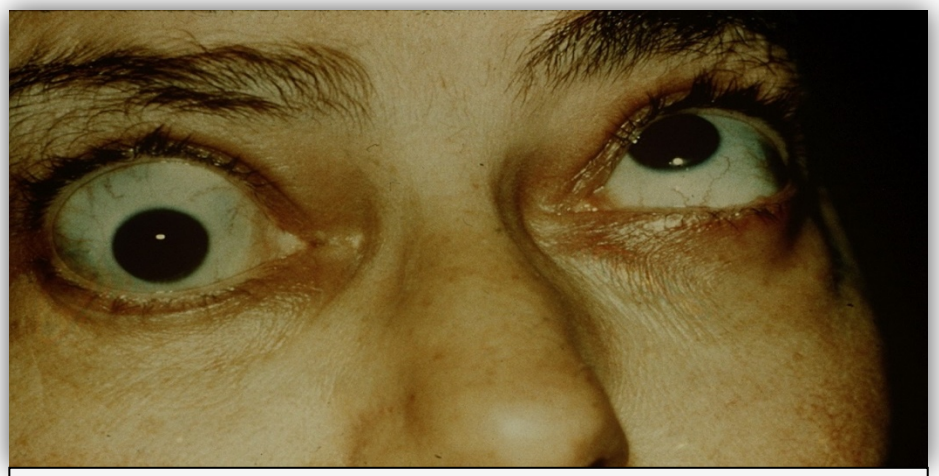
2- Steroid, lubricants and eye protection before sleep **(for eye symptoms)**

3- If there is restrictive myopathy. Surgical intervention is required

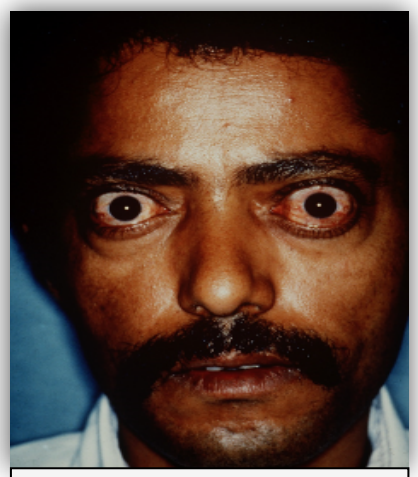
Complications:

1- Optic neuropathy

2- Exposure or bacterial keratitis



Limitations of extraocular muscle



Exophthalmos with lid retraction

2- Tuberculosis:

Introduction:

- TB is a chronic **granulomatous infection** usually caused in humans by **Mycobacterium tuberculosis.**
- TB is primarily a pulmonary disease but may spread by the bloodstream to other sites; ocular involvement commonly occurs without clinically overt systemic disease.

Ocular manifestation:

- Phlyctenular keratoconjunctivitis (small, circumscribed lesions at the corneal limpus) - Interstitial keratitis – Vitritis - Choroidal granuloma
- **Retinal vasculitis 'Eales disease'**
- **Anterior uveitis (Granulomatous uveitis):** causes blindness if not treated
- **Mutton-fat keratic precipitation:** collection of inflammatory cells on the corneal endothelium appear large with yellowish color.

Investigations:

1- PCR and the interferon-gamma release assay (IGRA)

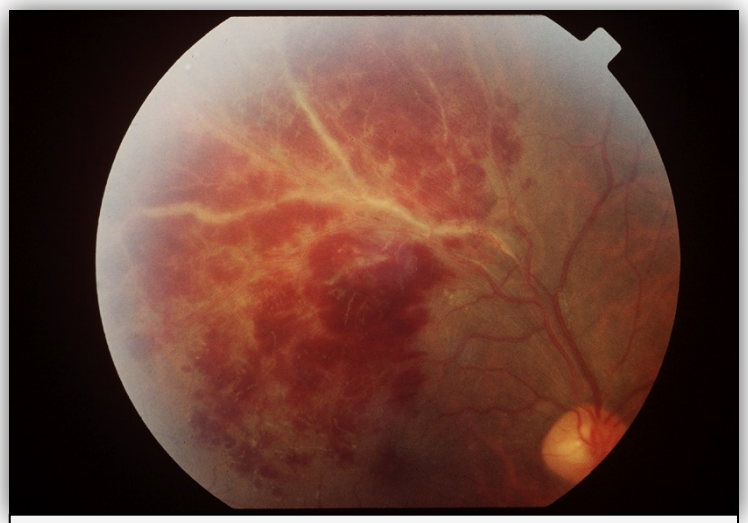
2- Aqueous or vitreous sampling rarely yields demonstrable (smear – acid-fast bacilli on Ziehl–Neelsen staining – or culture – Lowenstein–Jensen medium)

Treatment:

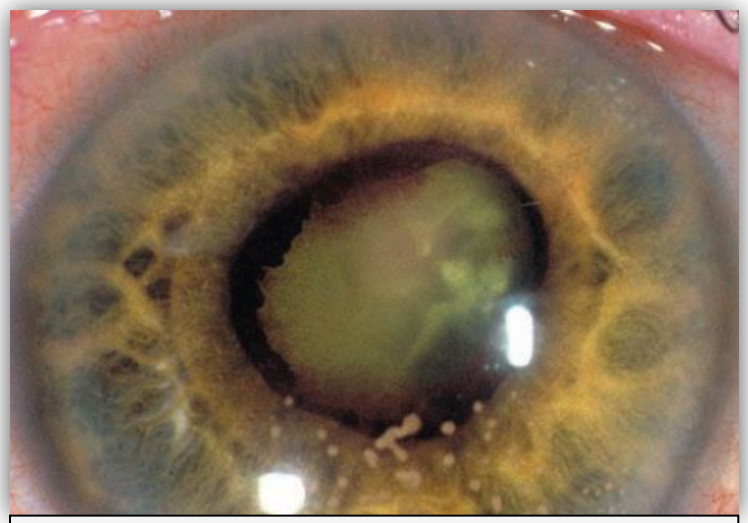
1- Prolonged Anti-TB therapy *multi-drug therapy (4 drugs in 2 months, then continue for 6 months with 2 drugs)

- Isoniazid (with Vitamin B6 (pyridoxine) to prevent the development of peripheral neuropathy), rifampicin, pyrazinamide and ethambutol
- **ethambutol can cause optic neuropathy**

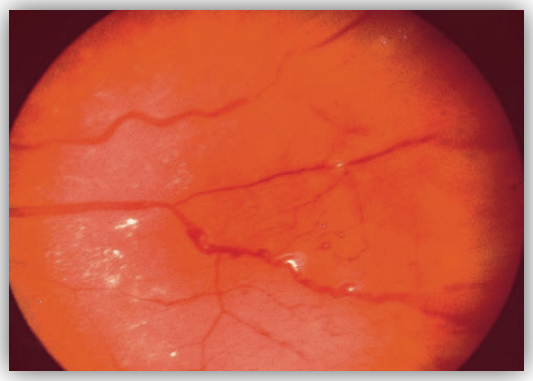
2- Topical and systemic steroids may be used concomitantly to reduce inflammation- induced damage



Retinal vasculitis



Mutton-fat keratic precipitation



Eales disease: Fundus photo of the peripheral retina, revealing vascular tortuosity and peripheral retinal neovascularization.

4- Syphilis: not common in our region

- could be either congenital syphilis 'transplacental infection' or acquired.
- Congenital syphilis:
  - Interstitial Keratitis
  - Chorioretinitis
- Acquired syphilis:
  - Ocular chancre.
  - Iridocyelitis.
  - Interstitial Keratitis.
  - Chorioretinitis.
  - Neuro-ophthalmic (nerve palsies, optic neuritis...).

5- Sarcoidosis:

Introduction:

- It is a chronic disorder of unknown cause, manifesting with **non-caseating granulomatous inflammatory foci.**
- It more frequently affects Japanese people, it increases with good hygiene and it is one of the most common systemic associations of uveitis.

Systematic manifestation:

The triad: **erythema nodosum - bilateral hilar lymphadenopathy - polyarthralgia**

Ocular manifestation:

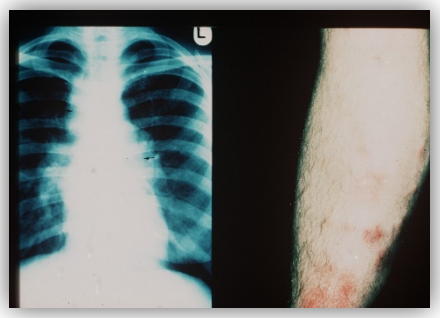
- Candle-wax exudate in the retina
- Optic nerve, Retinal, Choroidal Lid margin and conjunctival granulomas.
- **Mutton-fat keratic precipitates**
- Acute and Chronic granulomatous iridocyclitis.
- Peripheral retinal periphlebitis.

Investigations:

1. **Tuberculin skin test:** negative in sarcoidosis
2. **Chest X-ray:** showing bilateral hilar lymphadenopathy (BHL)
3. **Elevated serum ACE levels and/or elevated serum lysozyme**
4. Ca and liver function (**Abnormal liver enzyme tests).**

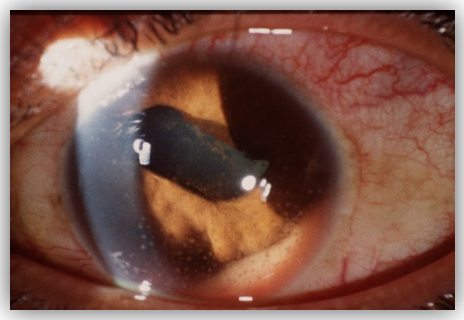
Treatment:

Steroid and NSAID's

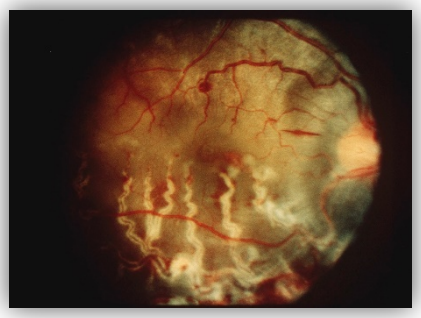


CXR of Bilateral hilar lymph nodes in patient with granulomatous uveitis

and erythema nodusm



Granuloma with posterior synechia



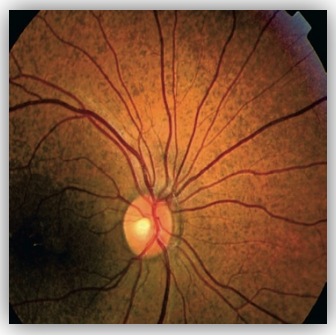
Candle-wax exudate

6- Rubella:

❖ Trans placental transmission could lead to congenital abnormalities.

Ocular manifestation:

- Congenital Cataract and Glaucoma
- **Microphthalmos (small eye)**
- **Pigmentary retinopathy: salt and pepper**
- **Anterior uveitis: unresponsive to steroids**

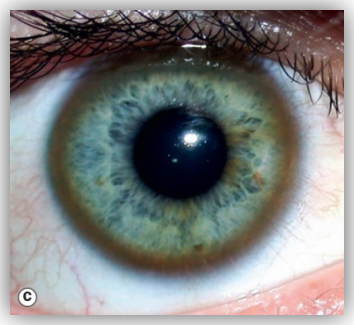


Rubella retinopathy

7- Wilson's disease 'hepatic degeneration':

Introduction:

- A rare condition involving the widespread **abnormal deposition of copper** in tissues. It is caused **by a deficiency of alpha 2 caeruloplasmin**, the major copper-carrying blood protein.



Kayser-Fleischer ring

Systematic manifestation:

- Liver disease.
- Basal ganglia dysfunction.
- Psychiatric disturbances.

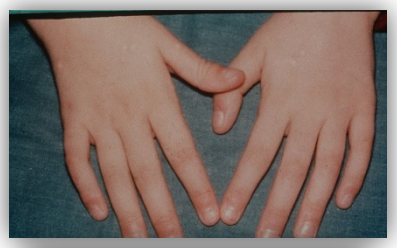
Ocular manifestation:

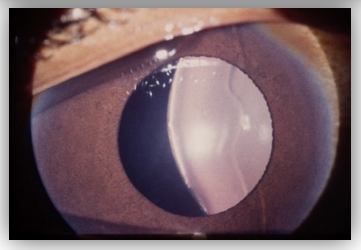
- **Kayser-Fleischer ring**: cons**ists** of a **brownish-yellow zone**
- **of fine copper dusting in peripheral descemet membrane**
- detected with gonioscopy (Important sign).
- **Anterior capsular 'sunflower' cataract**

Treatment:

Penicillamine

8- Marfan's syndrome:





Introduction:

It is an autosomal dominant disease.

Systematic manifestation:

- **Arachnodactyly (Long fingers)**
- Heart diseases.
- Bone deformities

Ocular manifestation:

- **Lens subluxation:** due to weakness of the lens zonulles (the most important feature in the eye in this patient)
- **Retinal detachment**
- **Axial myopia**
- **Angle anomaly lead to glaucoma**

9- Systemic Lupus Erythematosus:

- Autoimmune disease characterized by increased Anti-DNA, ANA, antiphospoholipid antibodies and decreased C3 & C4.
- **Retinal pathology:** cotton wool spots

10- Rheumatoid Arthritis:

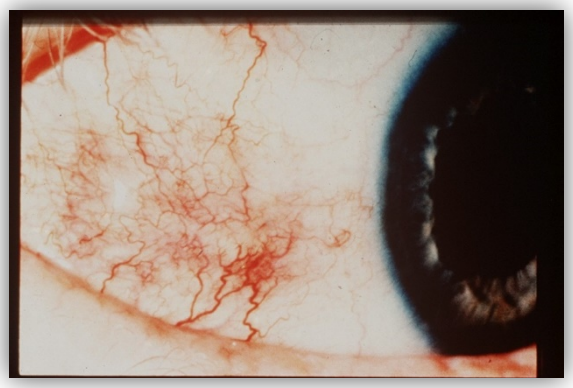
Not common here in our region, it's rare. The patient had positive Rheumatoid factor

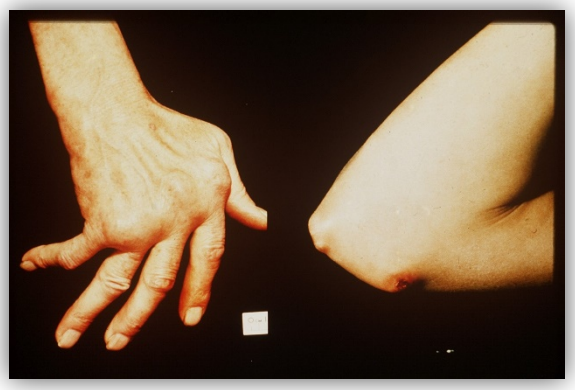
Ocular manifestation:

- **keratoconjunctivitis sicca, dryness and keratitis**

Investigation:

1. Positive Rose Bengal staining (for keratoconjunctivitis sicca)
2. RF positive





11- Ankylosing spondylitis:

Introduction:

- Ankylosing spondylitis (AS) is characterized by inflammation, calcification and finally ossification of ligaments and capsules of joints with resultant bony ankylosis of the axial skeleton.
- It more commonly affects males, of whom 90% are HLA-B27-positive.
- Unilateral usually
- Radiological changes often predate clinical symptoms
- **HLA positive disease:** IBD, psoriasis, Reiter and ankylosing spondylitis

Systematic manifestation:

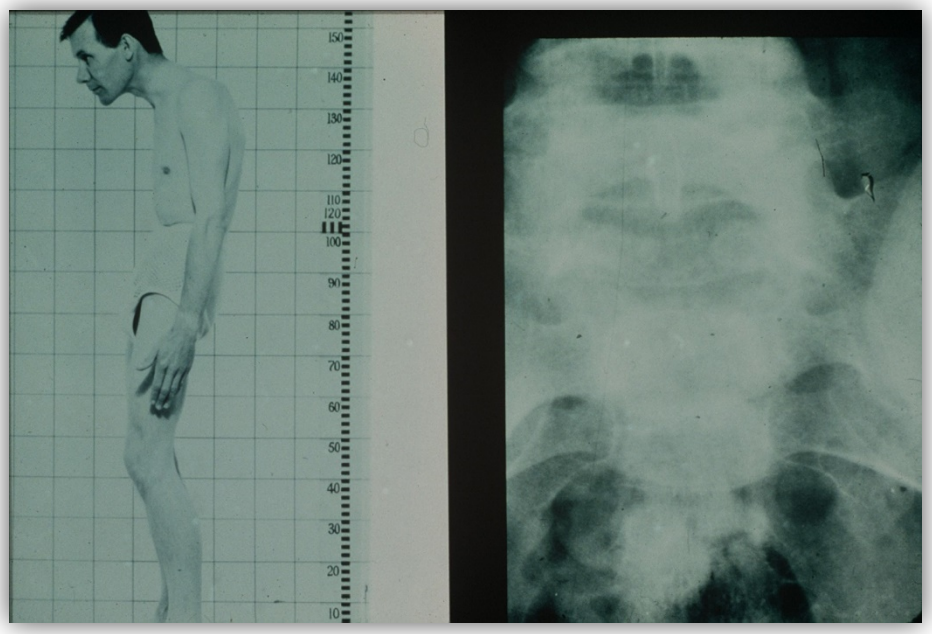
- Pain and stiffness in the lower back with limitation of movement
- Calcification of spinal ligaments gives rise to a **'bamboo spine'.**

Ocular manifestation:

- Acute **non- granulomatous** anterior uveitis

Investigations:

1. **HLA-B27-positive**
2. **X-ray: sacroiliac joints shows juxta-articular osteoporosis in the early stages**



12- Juvenile chronic arthritis:

Introduction:

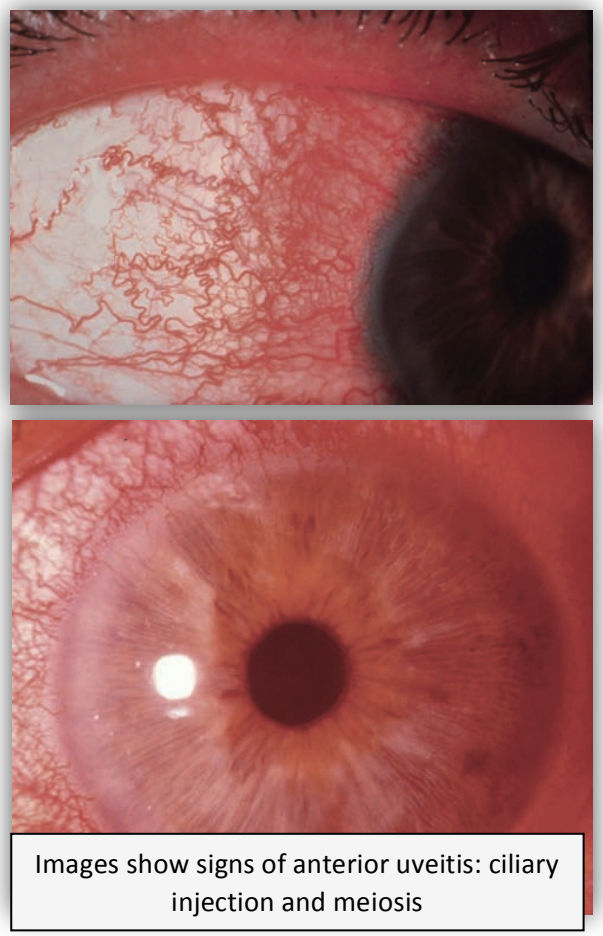
- It is defined as **arthritis of unknown etiology** that begins **before the age of 16 years and persists for at least 6 weeks.**
- **Most common cause of anterior uveitis in children** in western countries.
- The patient may get no complains until they get blind.

Risk factors of uveitis:

-Oligoarticular onset
-Being a girl
-Young age < 4
-Positive antinuclear antibody

Systematic manifestation:

a) **Pauciarticular form:** four or less joints are affected **(associated with 20% uveitis)**
b) **Polyarticular form:** five or more joints are affected. **(rare uveitis)**
c) **Stills disease:** fever, episodic erythematous maculopapular rash, lymphadenopathy and hepatosplenomegaly. **(extremely rare uveitis)**



Images show signs of anterior uveitis: ciliary injection and meiosis

Ocular manifestation:

- Chronic non-granulomatous uveitis
- Band keratopathy
- Posterior synechiae

Investigations:

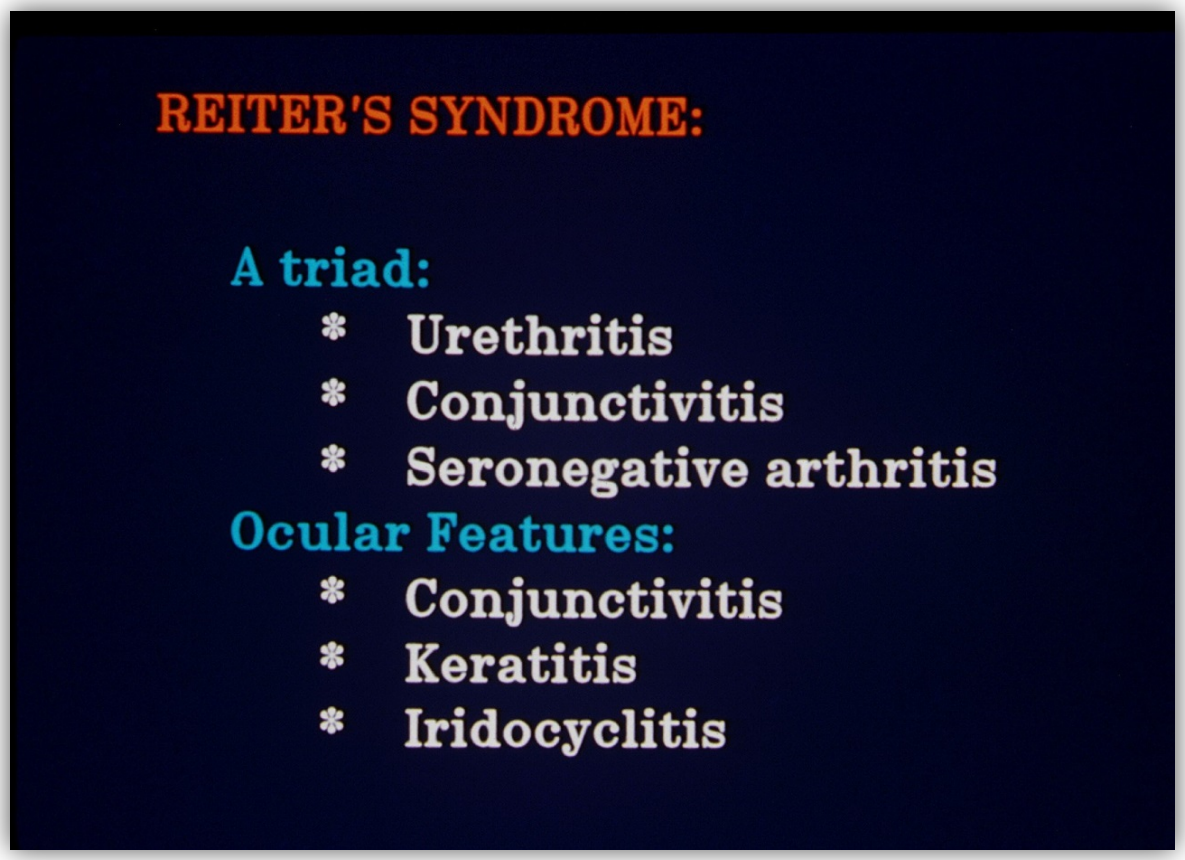
1. **Anti-ANA antibodies:** will be positive in majority of pauciarticular type
2. **Rehumatoid factor:** positive in some polyarticular type
3. **HLA-B27:** it will be positive in some patient

Complications:

- glaucoma
- amblyopia
- maculopathy
- phthisis

Treatment:

Topical and systematic Steroid and mydriatic agent to prevent posterior synechiae



13- Behçet disease:

Introduction:

- Behçet disease (BD) is an idiopathic, multisystem syndrome characterized by recurrent aphthous oral ulcers, genital ulceration and uveitis. It's not in whites but is common here.
- It is non-granulomatous with transient mobile hypopyon in a relatively white eye Hypopyon in HLA-B27 is solid " doesn't move".
- Patients with relapsing/remitting acute onset of bilateral panuveitis with retinal vasculitis and often spontaneous resolution even without treatment

Systematic manifestation:

- Vasculitis
- Recurrent aphthous oral ulcers
- genital ulceration

Ocular manifestation:

- Anterior non-granulomatous uveitis
- Transient hypopyon
- Retinal vasculitis
- Optic disc hyperemia

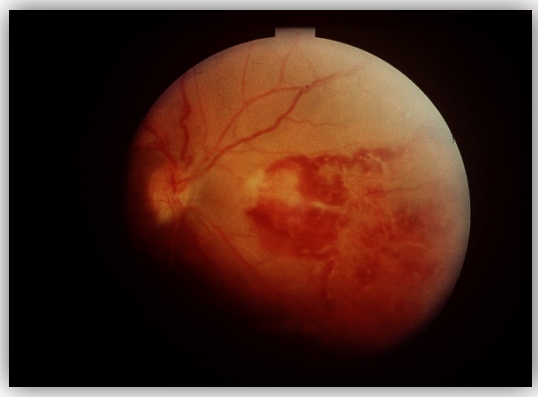
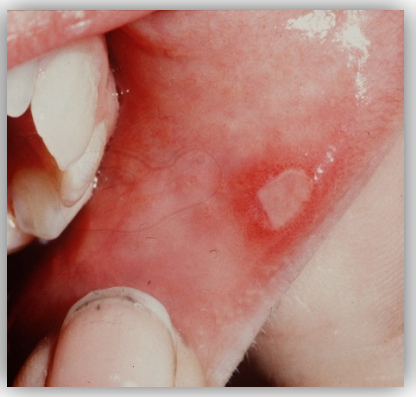
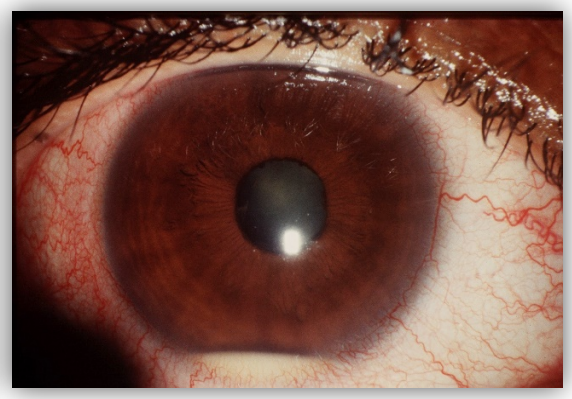
Investigations:

1- HLA-B51 is positive

2- Pathergy test: pustule 24–48 hours after a sterile needle prick

Treatment:

- Steroid for a short time then Cyclosporine
- Anti-Alpha tumor necrosis agents "infliximab" if case is resistant

14- Toxiplasmosis:

Introduction:

- Toxoplasmosis is caused by Toxoplasma gondii after eating raw meat
- Can be congenital: in the third trimester.
- Most common cause of infectious uveitis in many countries. Common in France due to their habits." they eat raw meat"
- Bilateral in most cases.
- Recurrent retinitis may cause blindness

Systematic manifestation:

- Congenital: Convulsions and intracranial calcification

Ocular manifestation:

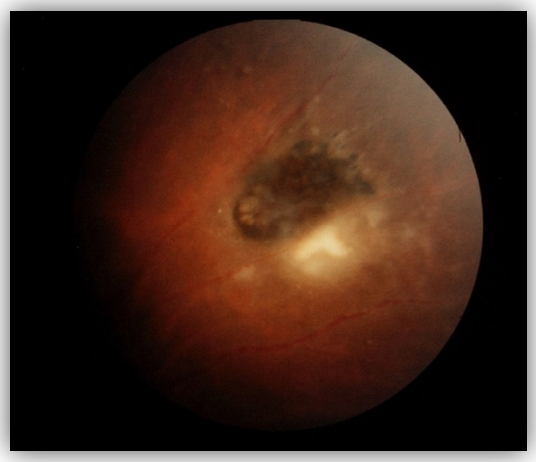
- posterior uveitis (Constitutes 20-60% of all posterior uveitis).
- Macular lesion
- Retinochoroiditis ”fluffy white with pigmented scar” which has a predilection for the posterior pole.
- Unilateral floaters, blurring and photophobia.

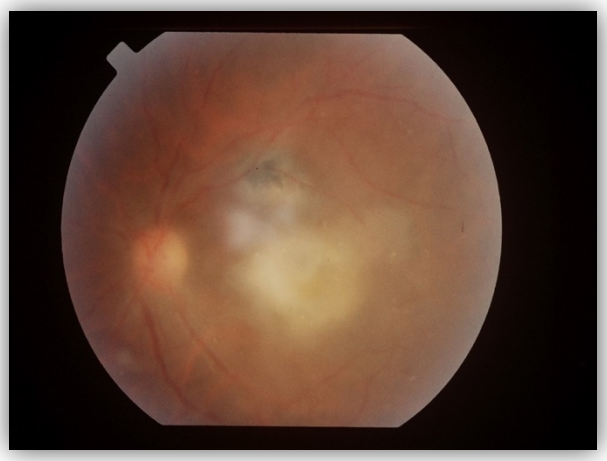
Investigations:

PCR and serology

Treatment:

Clindamycin and Co- trimoxazole, 48hr later add steroid to suppress inflammation.





15- sjogren's syndrome:

Systematic manifestation:

Dryness of skin and mouth and arthralgia and polyneuropathy.

Ocular manifestation:

keratoconjunctivitis sicca "dryness of eye"

Investigation:

1. Schirmer tear test
2. Positive Rose Bengal staining (for keratoconjunctivitis sicca"
3. ANA, RF positive
4. Associated with **HLA-B8/DR3**

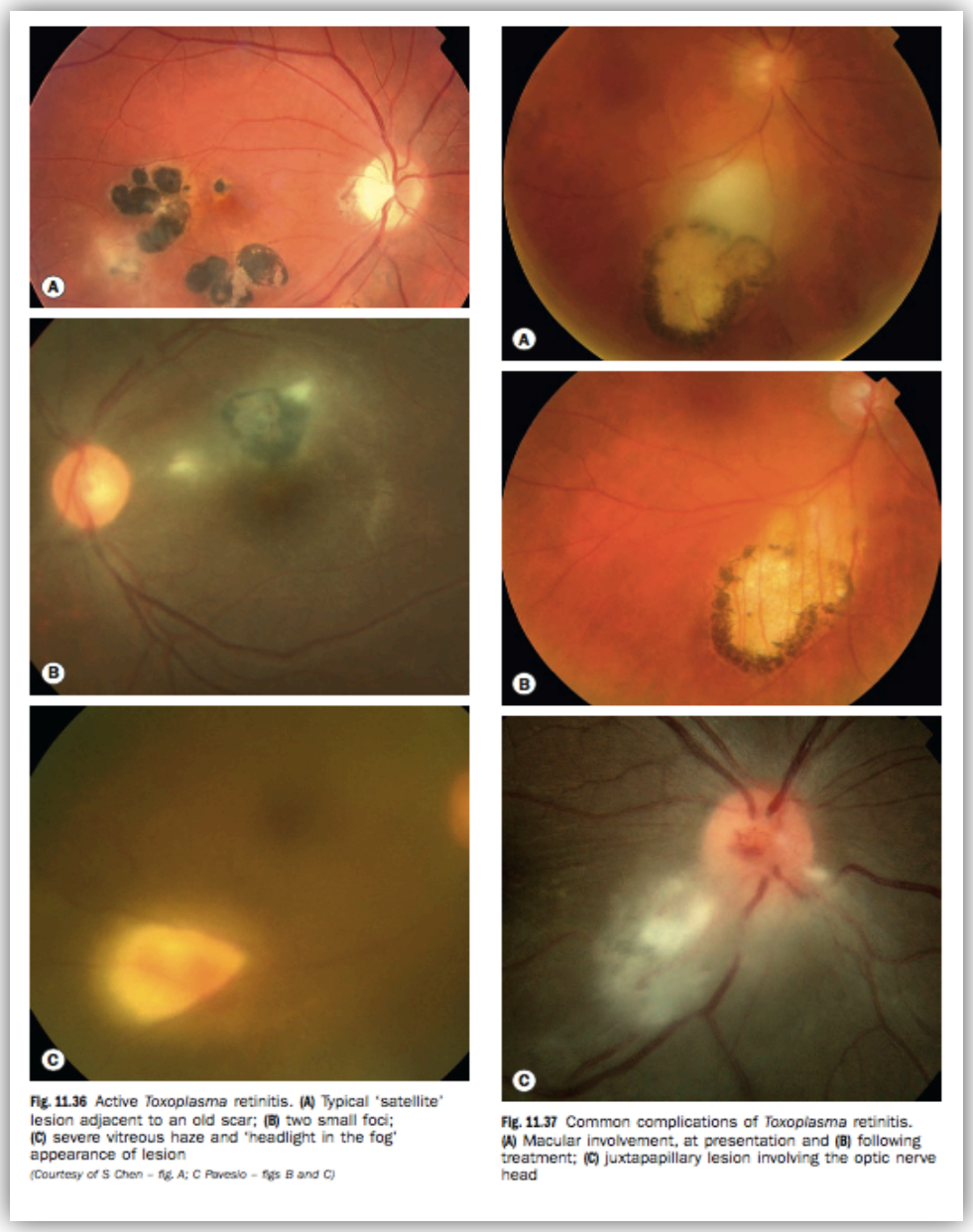


Fig. 11.36 Active *Toxoplasma* retinitis. (A) Typical 'satellite' lesion adjacent to an old scar; (B) two small foci; (C) severe vitreous haze and 'headlight in the fog' appearance of lesion
(Courtesy of S Chen – fig. A; C Pavesio – figs B and C)

Fig. 11.37 Common complications of *Toxoplasma* retinitis. (A) Macular involvement, at presentation and (B) following treatment; (C) juxtapapillary lesion involving the optic nerve head

16- Vogt- koyangi Harada disease:

Introduction:

- Idiopathic Multisystem, autoimmune disease directed against melanocyte-containing tissues such as the uvea, ear and meninges.
- Most common cause of uveitis in the **kingdom of Saudi Arabia.**
- Mean age is 28 year old
- Most common cause of intraocular inflammation

Systematic manifestation:

- Alopecia
- Vitiligo
- **Poliosis**: absence or decreased melanin in head hair, eyebrows or eyelashes.
- Deafness and vertigo
- Meningismus

Ocular manifestation:

- Bilateral granulomatous anterior uveitis
- Bilateral multifocal posterior uveitis
- Dalen–Fuchs nodules
- 'sunset glow' fundus

Investigations:

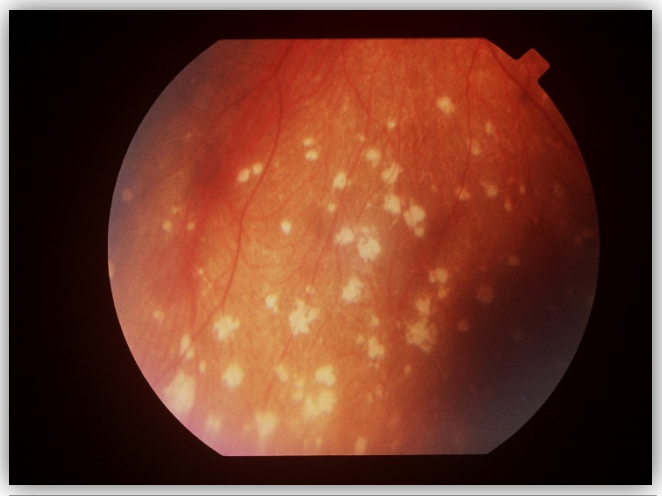
1. Associated HLA-DR1 and HLA-DR4
2. Lumbar puncture if diagnosis uncertain; CSF shows a transient lymphocytic pleocytosis, and melanin-containing macrophages.

Complications:

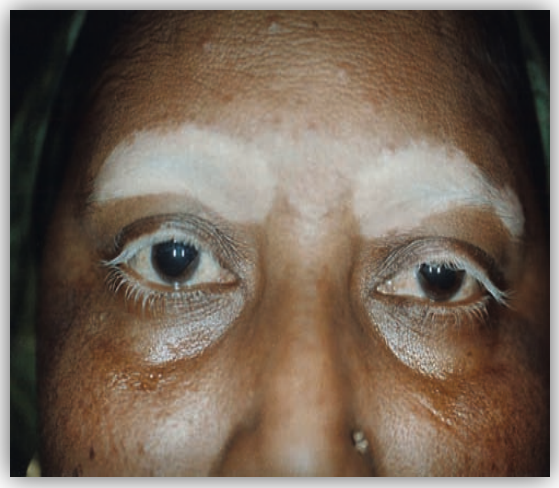
1. Glaucoma
2. cataract
3. choroidal neovascularization,
4. subretinal fibrosis,
5. retinal atrophy

Treatment:

High-dose steroid or infliximab in case of steroid resistance



Dalen–Fuchs nodules

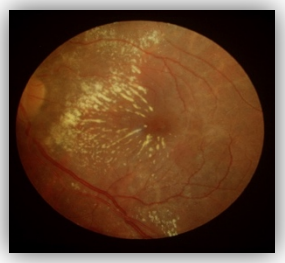


Vitiligo and poliosis in Vogt–Koyanagi–Harada syndrome

17- Sickel cell disease: The doctor passed the slides and said not important

- Retinopathy due to sickling of RBC causing peripheral vascular occlusion of the circulation and neovasculization in the periphary. Looks like "Sea fans"

18- Hypertensive retinopathy:



Introduction:

- the primary response of the retinal arterioles to systemic hypertension is vasoconstriction; this is less marked in older individuals due to involutional sclerosis conferring increased rigidity. Arteriolosclerosis refers to hardening and loss of elasticity of small vessel walls, manifested most obviously by arteriovenous (AV) Nipping(nicking) at crossing points; its presence makes it probable that hypertension has been present for many years, even if the BP is currently controlled. Nipping: Thinning of the vein after the artery and it's changing direction, to avoid pressure by the artery.

Ocular manifestation:

Keith-wagener grouping:

Stage I & II: arteriolar attenuation (increase light reflex as a result of chronic hypertension there will be thickening of arterioles giving the appearance of **silver wire and copper wire in the artery**)

Stage III: Cotton-wall spots – Hard exudate – **Macular star** if deposited around the fovea – retinal edema – Retinal hemorrhage

Stage IV: All the above + Bilateral edema of the optic nerve - Edema of optic disc seen in malignant hypertension

19- Giant cell arteritis:

Introduction:

- Affect medium and large vessels. Anterior ischemic optic neuropathy is divided into two types: Arteritic , Non-Arteritic.

❖ **Non-arteritic anterior ischaemic optic neuropathy** (NAION): more common, caused by occlusion of the short posterior ciliary arteries resulting in partial or total infarction of the optic nerve head. More common, comes with people with diabetes, hypertension, hyperlipidaemia, collagen vascular disease, antiphospholipid antibody syndrome, hyperhomocysteinaemia, sudden hypotensive events, cataract surgery, sleep apnoea syndrome and erectile dysfunction."Patient complains of sudden painless monocular visual loss; this is frequently discovered on awakening, suggesting a causative role for nocturnal hypotension.

❖ **Arteritic anterior ischaemic optic neuropathy** (AAION): Caused by giant cell arteritis (GCA). About 50% of patients with GCA have polymyalgia rheumatica (PMR)" pain and stiffness in proximal muscle groups, typically the shoulders and biceps, that is worse on waking and **jaw claudication**".

- Once the vision is lost you cannot restore it.

Risk Factors:

Old age, Females, smoking, low body mass index and early menopause.

Investigations:

1. Erythrocyte Sedimentation Rate 'ECR' ,
2. CBC
3. temporal artery biopsy.

Ocular manifestation:

1. **Ocular motor palsies, including a pupil-involving third nerve palsy,**
2. **Sudden, profound unilateral visual loss** not uncommonly preceded by transient visual obscurations (amaurosis fugax) (Latin fugax meaning fleeting, Greek amaurosis meaning darkening, dark, or obscure) which is a painless transient monocular visual loss. And sometimes by double vision. Periocular pain is also common.
3. **strikingly pale 'chalky white' edematous disc** is particularly suggestive of GCA.

Treatment:

High-dose of steroid and protect the eye

Summary:

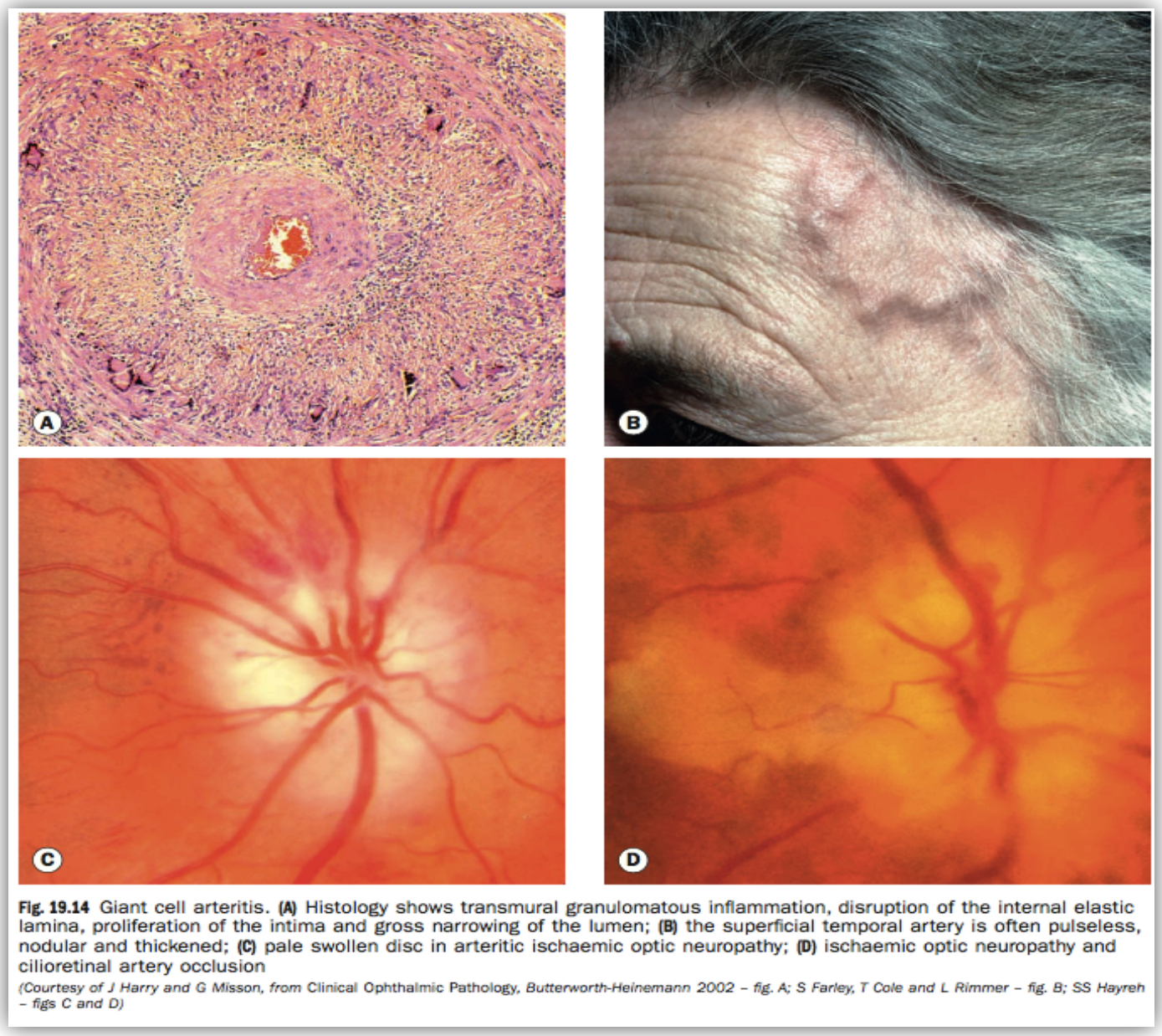


Fig. 19.14 Giant cell arteritis. **(A)** Histology shows transmural granulomatous inflammation, disruption of the internal elastic lamina, proliferation of the intima and gross narrowing of the lumen; **(B)** the superficial temporal artery is often pulseless, nodular and thickened; **(C)** pale swollen disc in arteritic ischaemic optic neuropathy; **(D)** ischaemic optic neuropathy and cilioretinal artery occlusion

(Courtesy of J Harry and G Misson, from Clinical Ophthalmic Pathology, *Butterworth-Heinemann 2002 – fig. A; S Farley, T Cole and L Rimmer – fig. B; SS Hayreh – figs C and D)*

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