Diabetic Retinopathy

Ahmad Rimawi

Epidemiology

(The World Health Organisation (1992) definition of blindness is vision less than 3/60 in the better eye with best available spectacle correction.)

- Diabetes is therefore one of the most serious challenges to health care world-wide. According to recent projections it will affect 239 million people by 2010- doubling in prevalence since 1994. Diabetes will affect 28 million in western Europe, 18.9 million in North America 138.2 million in Asia, 1.3 million in Australasia.

- **Diabetes mellitus is the most common cause of blindness amongst individuals of working-age (20-65 years)**. The prevalence of blindness due to DR in Western Communities is estimated as between 1.6-1.9/ 100,000

Epidemiology

- **During the first two decades of disease, nearly all patients with type 1 diabetes and over 60% of patients with type 2 diabetes have retinopathy.[1]**
- WESDR demonstrated that type 1 patients experience a 25% rate of retinopathy after 5 years of disease, and 80% at 15 years of disease[2]
- **About 2% of type 2 diabetics have CSME at diagnosis and** up to 21% of newly diagnosed type 2 patients have some degree of retinopathy at time of diagnosis[1]

[1]American Diabetes Association: Retinopathy in Diabetes (Position Statement). *Diabetes Care* 27 (Suppl.1): S84-S87, 2004
2 Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR)

Pathogenesis of Diabetic Microangiopathy

- Hyperglycaemia causes
  - BM thickening
  - non enzymaitc glycosylation
  - increased free radical activity
  - increased flux through the polyol pathway
  - osmotic damage

- Haemostatic abnormalities of the microcirculation;
  It has also been said that platelet abnormalities in diabetics may contribute to diabetic retinopathy. There are three steps in platelet coagulation: initial adhesion, secretion, and further aggregation. It has been shown that the **platelets in diabetic patients are "stickier"** than platelets of non-diabetics They secrete prostaglandins that cause other platelets to adhere to them (aggregation) and blockage of the vessel and endothelial damage.

Diabetic retinopathy

Two types of diabetic retinopathy:

- Nonproliferative diabetic retinopathy (NPDR)
  - Early stage diabetic retinopathy
- Proliferative diabetic retinopathy (PDR)
  - Later stage diabetic retinopathy

A classification of diabetic retinopathy

Non-proliferative diabetic retinopathy (NPDR)

Mild non-proliferative diabetic retinopathy

- Microaneurysms
- Dot and blot haemorrhages
- Hard (intra-retinal) exudates

Moderate-to-severe non-proliferative diabetic retinopathy

The above lesions, usually with exacerbation, plus:

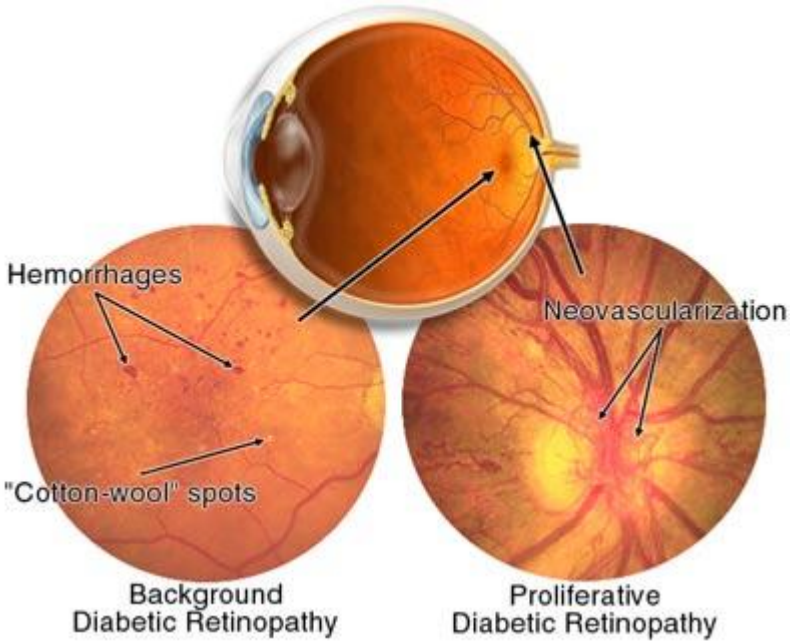
- Cotton-wool spots
- Venous beading and loops
- Intraretinal microvascular abnormalities (IRMA)

Proliferative diabetic retinopathy

- Neovascularization of the retina, optic disc or iris
- Fibrous tissue adherent to vitreous face of retina
- Retinal detachment
- Vitreous haemorrhage
- Pre retinal haemorrhage

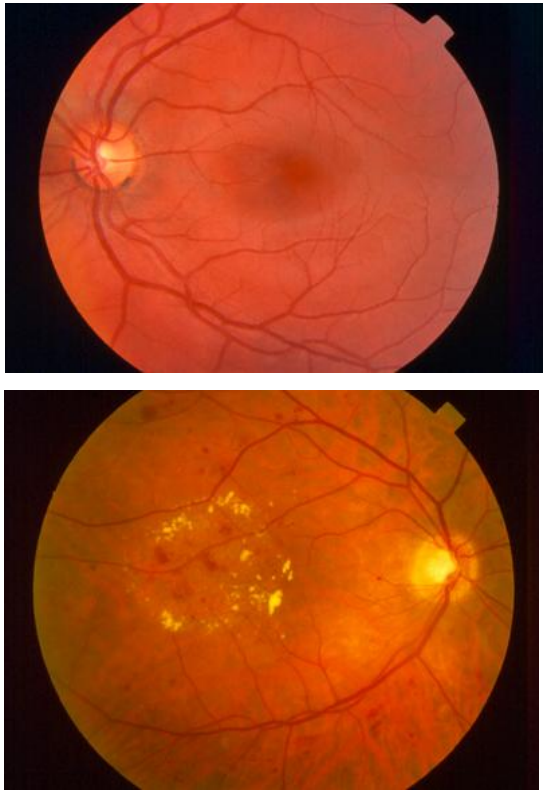
Maculopathy

- Clinically significant macular oedema (CSME)
- Ischaemic Maculopathy



Nonproliferative diabetic retinopathy (NPDR)

- Also called background diabetic retinopathy.
- Earliest stage of diabetic retinopathy.
- Damaged blood vessels in the retina leak extra fluid and small amounts of blood into the eye.
- Cholesterol or other fat deposits from blood, called hard exudates, may leak into retina.



Top: Healthy retina

Bottom: Retina with NPDR, containing hard exudates

Nonproliferative diabetic retinopathy

With NPDR, the central vision is affected by any of the following:

- Hard exudates on the central retina (macula).
- Microaneurysms (small bulges in blood vessels of the retina that often leak fluid).
- Retinal hemorrhages (tiny spots of blood that leak into the retina).
- Macular edema (swelling/thickening of macula).
- Macular ischemia (closing of small blood vessels/capillaries).

Microaneurysms

- Retinal microaneurysms are focal dilatations of retinal capillaries, 10 to 100 microns in diameter, and appear as red dots.
- They are usually seen at the posterior pole, especially temporal to the fovea.
- **Microaneurysms are the first ophthalmoscopically detectable change in diabetic retinopathy.**
- **Beginning as dilatations in areas in the capillary** wall where pericytes are absent, microaneurysms are initially thin-walled**. Later, endothelial cells proliferate and lay down layers of basement membrane material around themselves**.
- Fibrin and erythrocytes may accumulate within the aneurysm. Despite multiple layers of basement membrane, they are permeable to water and large molecules, allowing the accumulation of water and lipid in the retina.
- Since fluorescein passes easily through them, many more microaneurysms are usually seen on fluorescein angiography than are apparent on ophthalmoscopy

Retinal Haemorrhages

- When the wall of a capillary or microaneurysm is sufficiently weakened, it may rupture, giving rise to an intraretinal haemorrhage. **If the hemorrhage is deep (i.e., in the inner nuclear layer or outer plexiform layer), it usually is round or oval ("dot or blot")**

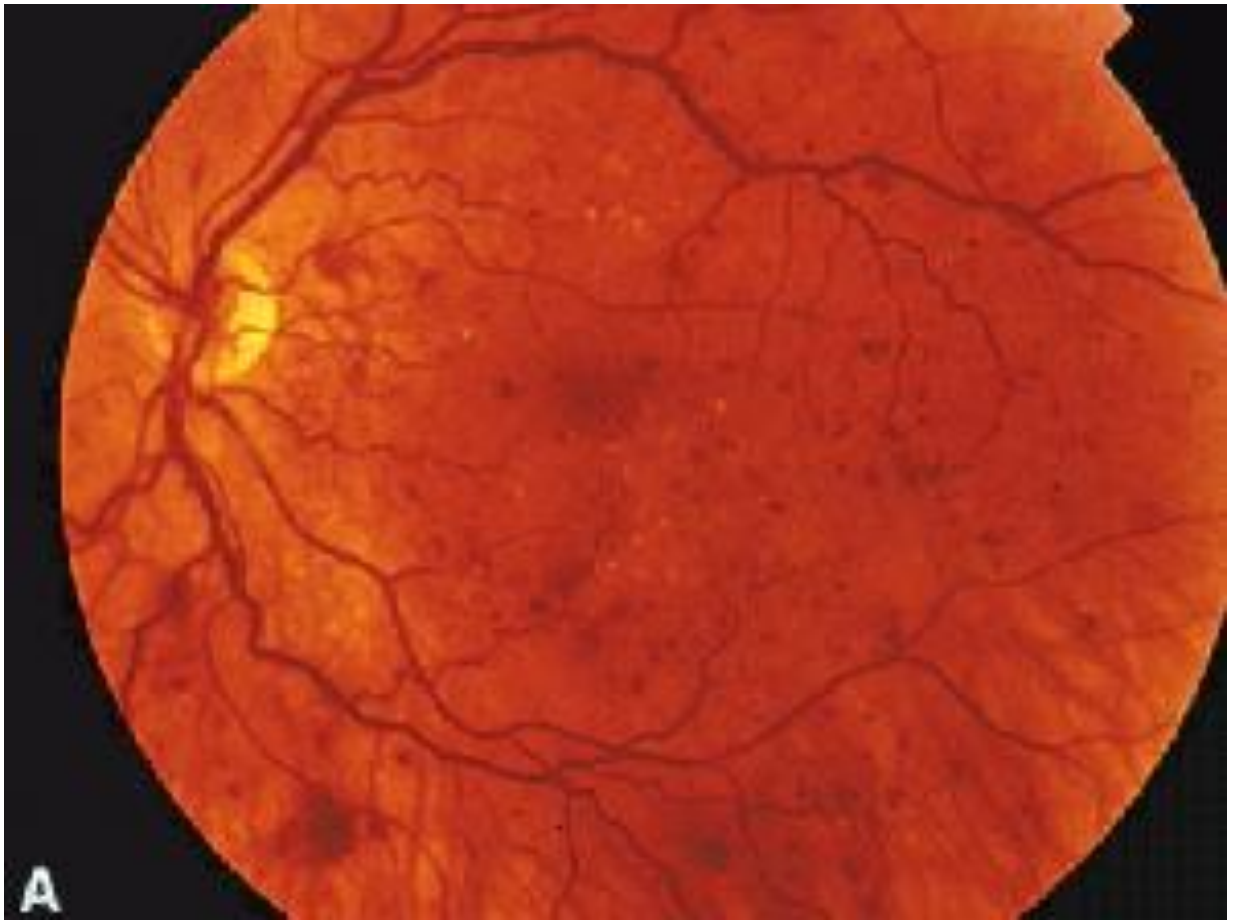
- Dot haemorrhages appear as bright red dots and are the same size as large microaneurysms.

- Blot haemorrhages are larger lesions they are located within the mid retina and often within or surrounding areas of ischaemia.

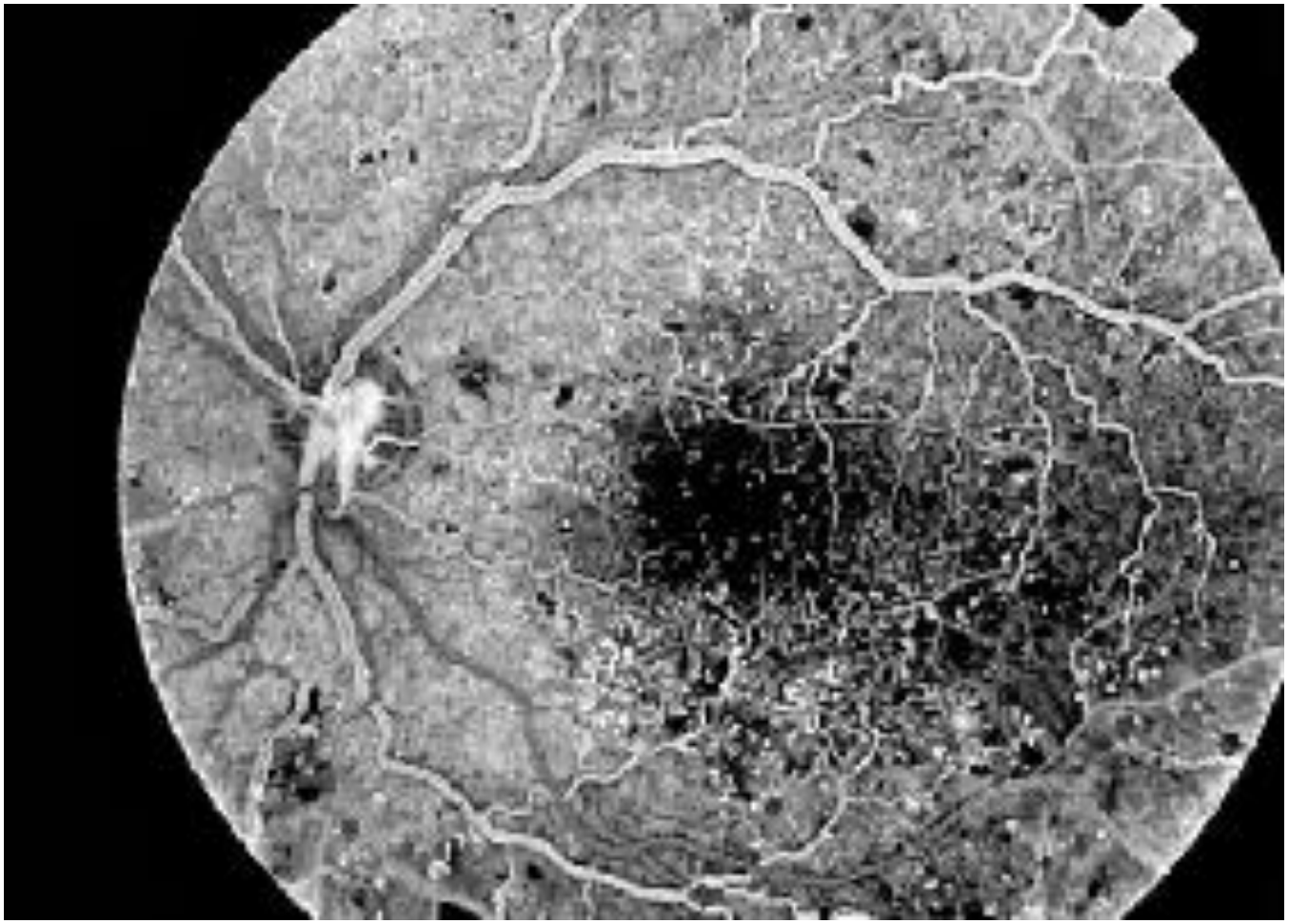
- **If the hemorrhage is more superficial and in the nerve fiber layer, it takes a flame or splinter shape, which is indistinguishable from a hemorrhage seen in hypertensive retinopathy.** They often absorb slowly after several weeks. Their presence strongly suggests the co-existence of systemic hypertension.

- **Diabetics with normal blood pressure may have multiple splinter haemorrhages. Nevertheless, when an ophthalmologist sees numerous splinter haemorrhages in a diabetic patient, the patient's blood pressure must be checked because a frequent complication of diabetes is systemic hypertension.**

Non-proliferative diabetic retinopathy (NPDR)



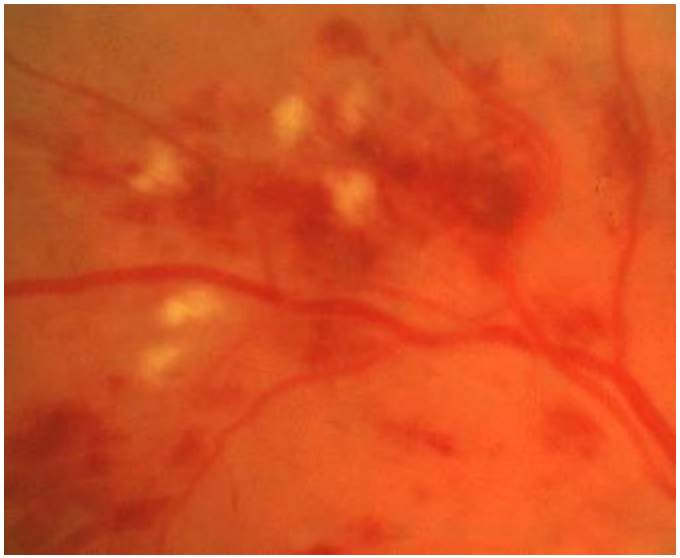
Non-proliferative diabetic retinopathy (NPDR)



Cotton Wool Spots

- Cotton wool spots result from occlusion of retinal pre-capillary arterioles supplying the nerve fibre layer with concomitant swelling of local nerve fibre axons. Also called "soft exudates" or "nerve fibre layer infarctions" they are white, fluffy lesions in the nerve fibre layer. Fluorescein angiography shows no capillary perfusion in the area of the soft exudate. They are very common in DR, especially if the patient is also hypertensive.

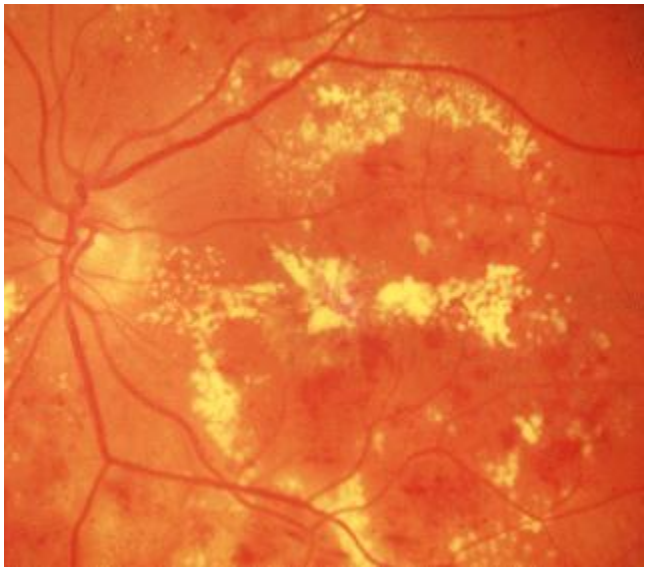
Cotton Wool Spots



Hard exudates (Intra-retinal lipid exudates)

- Hard exudates (Intra-retinal lipid exudates) are yellow deposits of lipid and protein within the sensory retina. Accumulations of lipids leak from surrounding capillaries and microaneuryisms, they may form a circinate pattern. Hyperlipidaemia may correlate with the development of hard exudates.

Hard exudates (Intra-retinal lipid exudates)



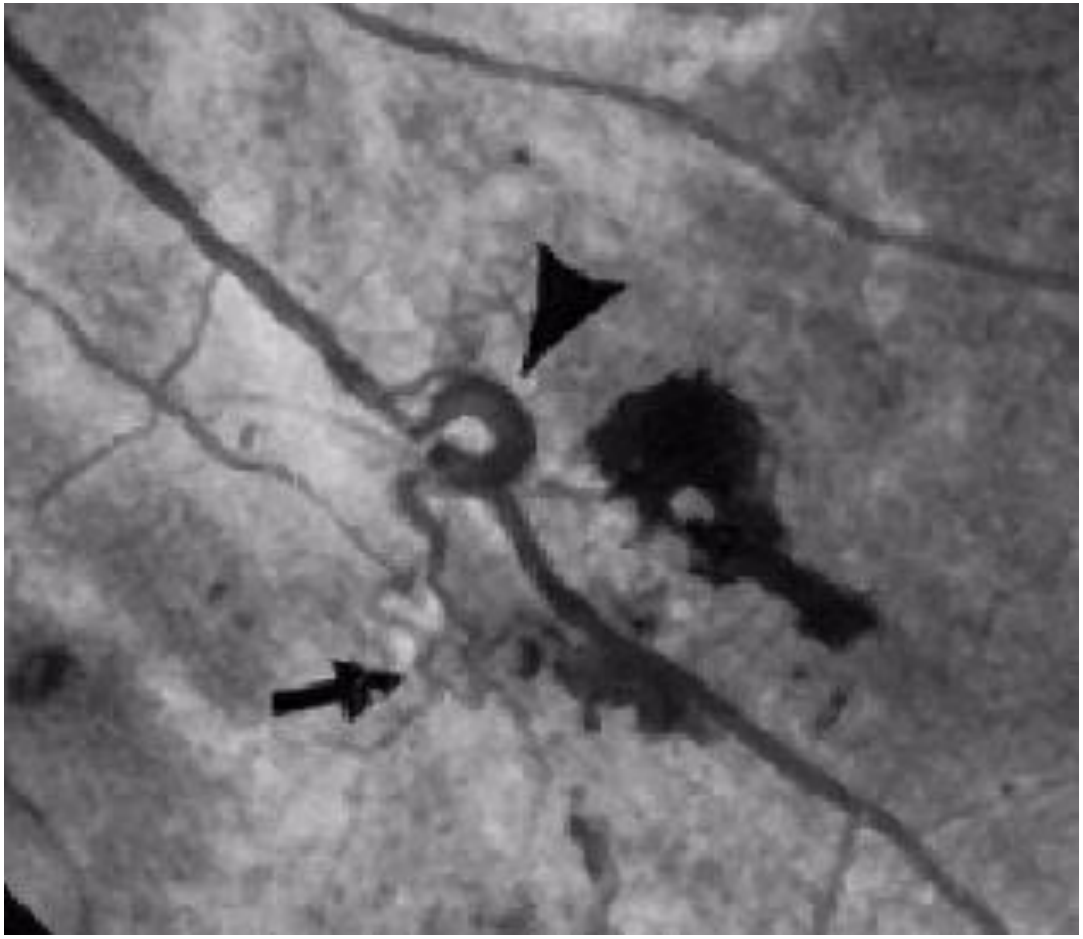
- Accumulations of lipids leak from surrounding capillaries and microaneuryisms, they may form a circinate pattern.

Late non proliferative changes

Intra-retinal microvascular abnormalities (IRMA) are abnormal, dilated retinal capillaries or may represent intraretinal neovacularization which has not breached the internal limiting membrane of the retina.

- They indicate severe non-proliferative diabetic retinopathy that may rapidly progress to proliferative retinopathy. Venous beading has an appearance resembling sausage-shaped dilatation of the retinal veins. It is another sign of severe non proliferative diabetic retinopathy.

Late non proliferative changes



Nonproliferative diabetic retinopathy

Macular edema

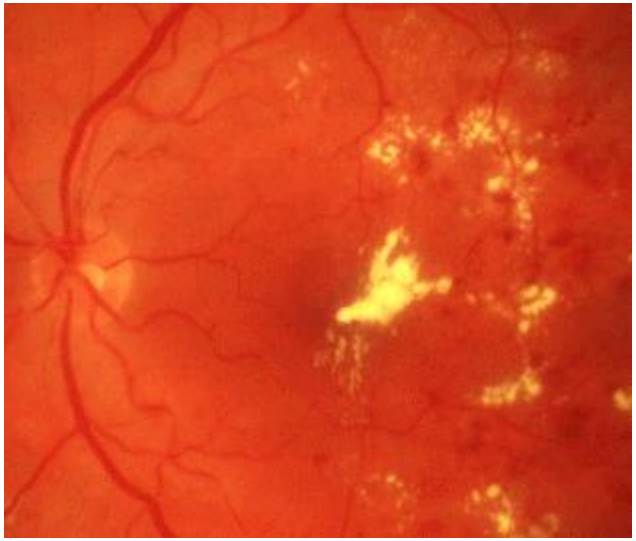
- Macular edema can be present at this stage and is defined as retinal thickening within 3,000 microns of the fovea (center of vision).
- Macula thickens or swells, affecting vision.
- Most common cause of vision loss in diabetes.
- Vision loss may be mild to severe.
- Peripheral (side) vision remains.
- Laser treatment may help to stabilize vision.
  - If CSME present: color fundus photography, fluorescein angiography, and photocoagulation
- May occur at any stage of retinopathy

Characteristics of Clinically Significant Macular (O)Edema (CSME)

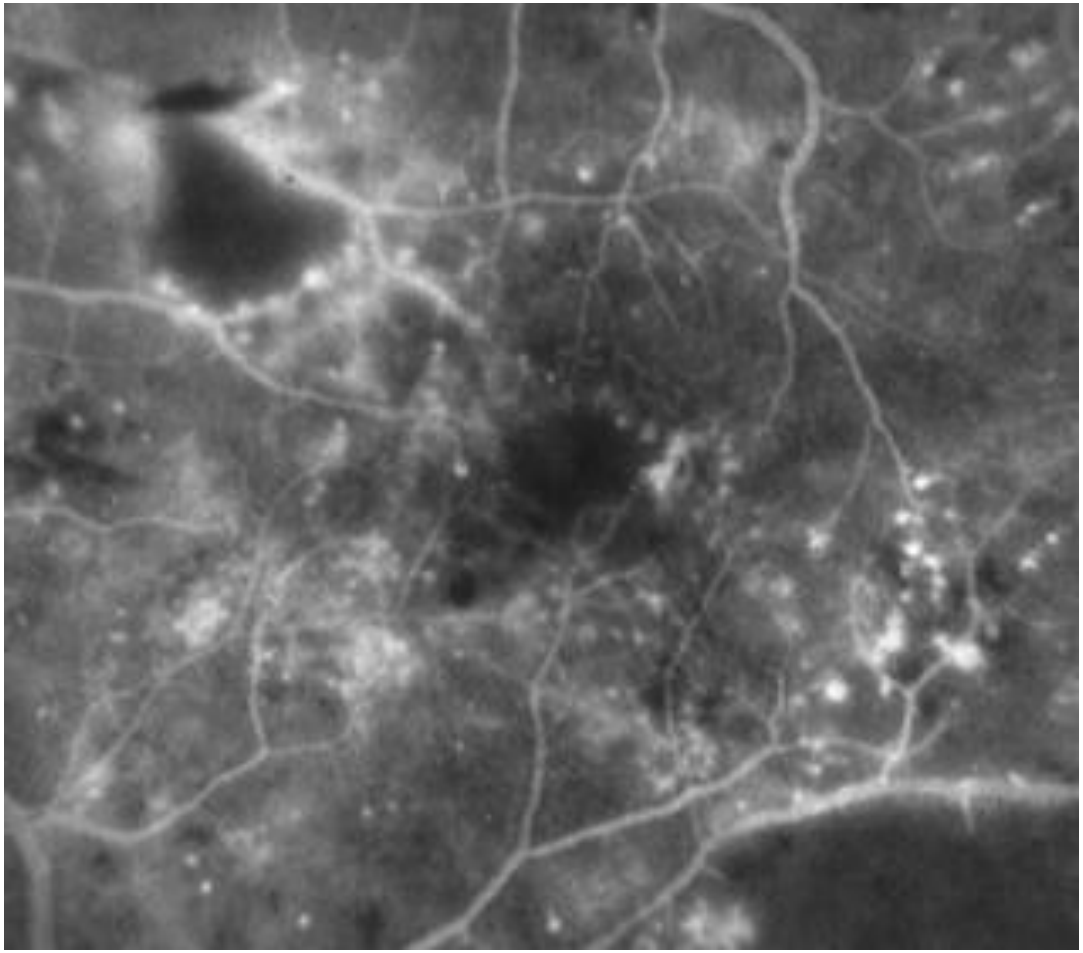
- The leading cause of visual loss amongst diabetics. Diagnosed by stereoscopic assessment of retinal thickening, usually by slit lamp biomicroscopy.

- Defined as the presence of one or more of the following, (Modified Airlie -House Criteria)
  - Retinal oedema within 500 microns of the centre fovea.
  - Hard exudates within 500 microns of fovea if associated with adjacent retinal thickening
  - Retinal oedema that is one disc diameter or larger, any part of which is within one disc diameter of the centre of the fovea.
  - Laser grid photocoagulation reduces the risk of visual loss by 50% at 2 years

CSME



CSME



Nonproliferative diabetic retinopathy

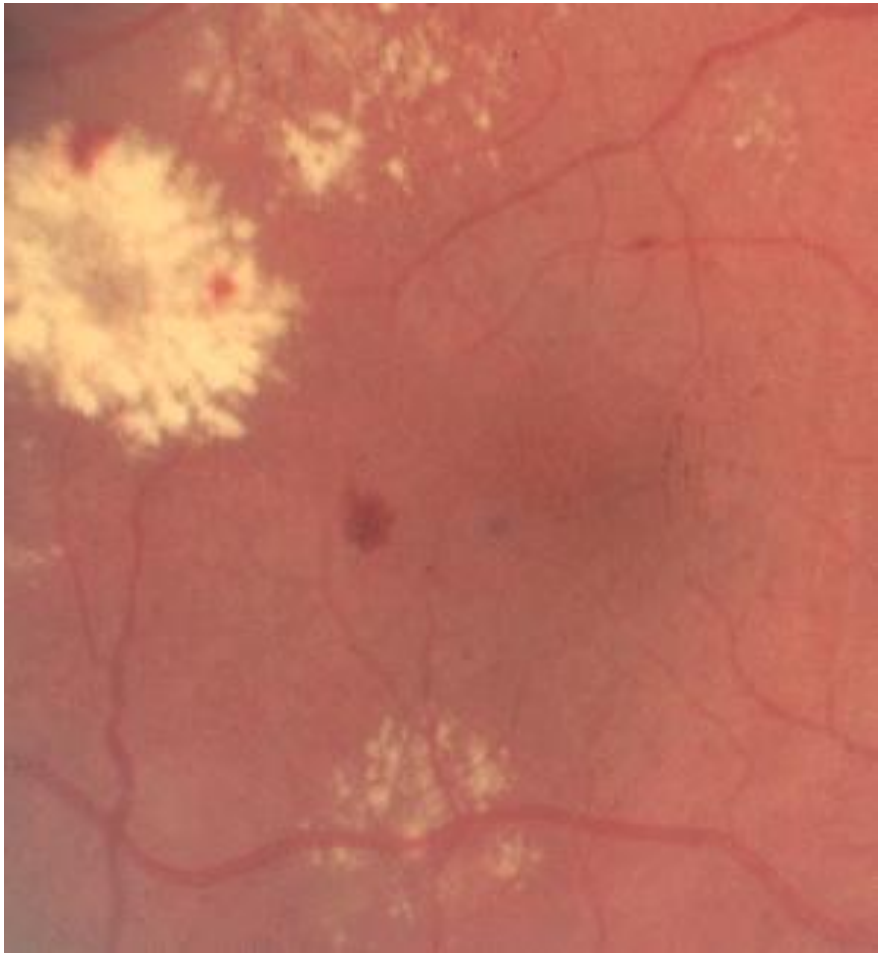
Macular ischemia

- Small blood vessels, or capillaries, close, blurring vision.
- Macula no longer receives enough blood to work properly.
- Currently no effective treatment for macular ischemia.

Ischaemic Maculopathy

- Maculopathy in type 1 diabetics is often due to drop out of the perifoveal capillaries with non perfusion and the consequent development of an *ischaemic maculopathy*.
- Enlargement of the foveal avascular zone (FAZ) is frequently seen on fluorescein angiography. Ischaemic maculopathy is not uncommon in type 2 diabetics, maculopathy in this group may show both changes due to ischaemia but also retinal thickening.

Ischaemic Maculopathy



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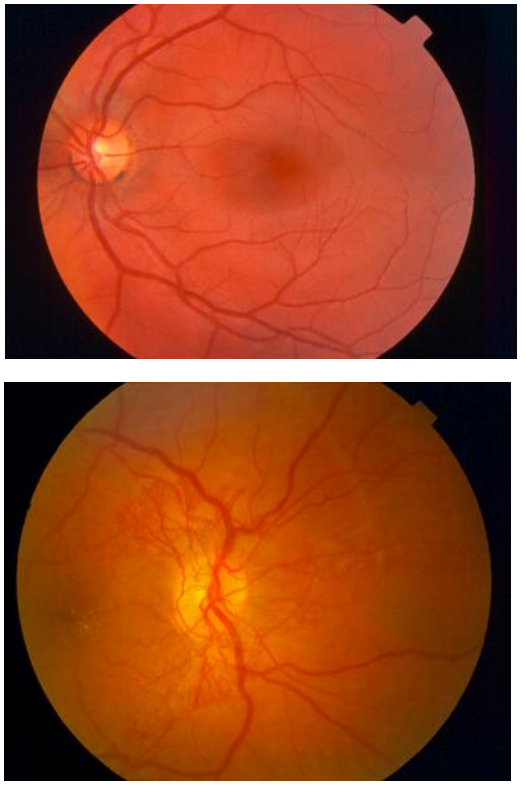
Proliferative diabetic retinopathy (PDR)

- Later stages of diabetic retinopathy.
- Abnormal blood vessels begin to grow on surface of retina or optic nerve; can't provide retina with normal blood flow (neovascularization).
- PDR can cause severe visual loss and other serious complications, such as neovascular glaucoma and loss of the eye.



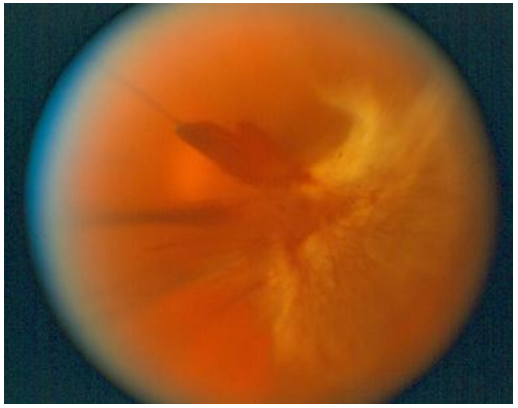
Top: Healthy retina

Bottom: Retina with PDR and neovascularization

Proliferative diabetic retinopathy

With PDR, vision is affected when any of the following occur:

- Vitreous hemorrhage (new, abnormal blood vessels bleed into vitreous gel in center of eye, preventing light rays from reaching the retina).
- Traction retinal detachment (new, abnormal blood vessels begin to shrink and tug on retina; may cause retina to detach).
- Neovascular glaucoma (neovascularization occurs in the iris, causing pressure to build up in the eye, damaging the optic nerve).



Vitreous hemorrhage

Proliferative diabetic retinopathy

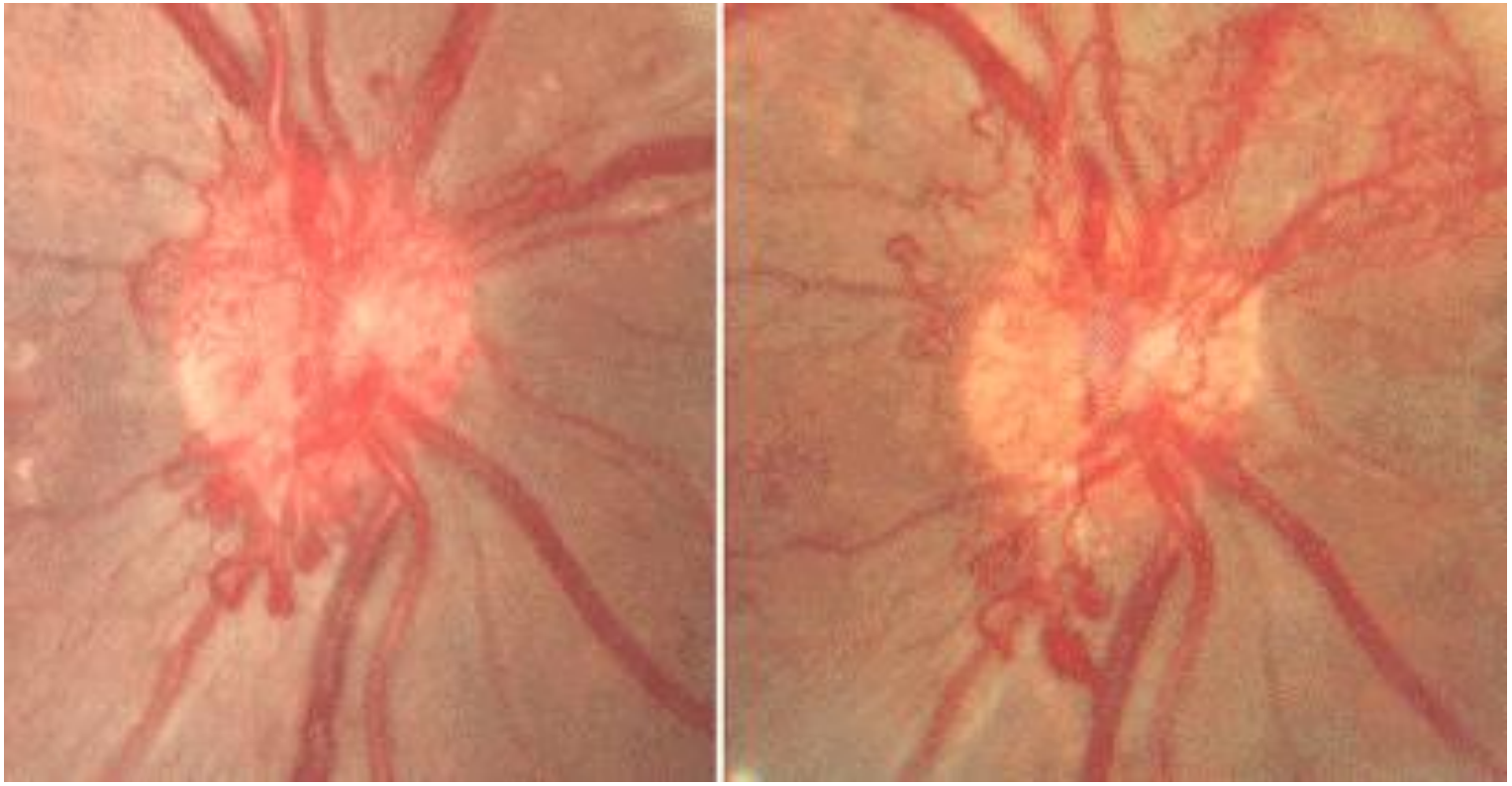
- Retinal ischaemia due to widespread capillary non perfusion results in the production of vasoproliferative substances and to the development of neovascularization. Neovascularization can involve the retina, optic disc or the iris*(rubeosis iridis).*

- Rubeosis iridis is a sign of severe proliferative disease, it may cause intractable glaucoma.

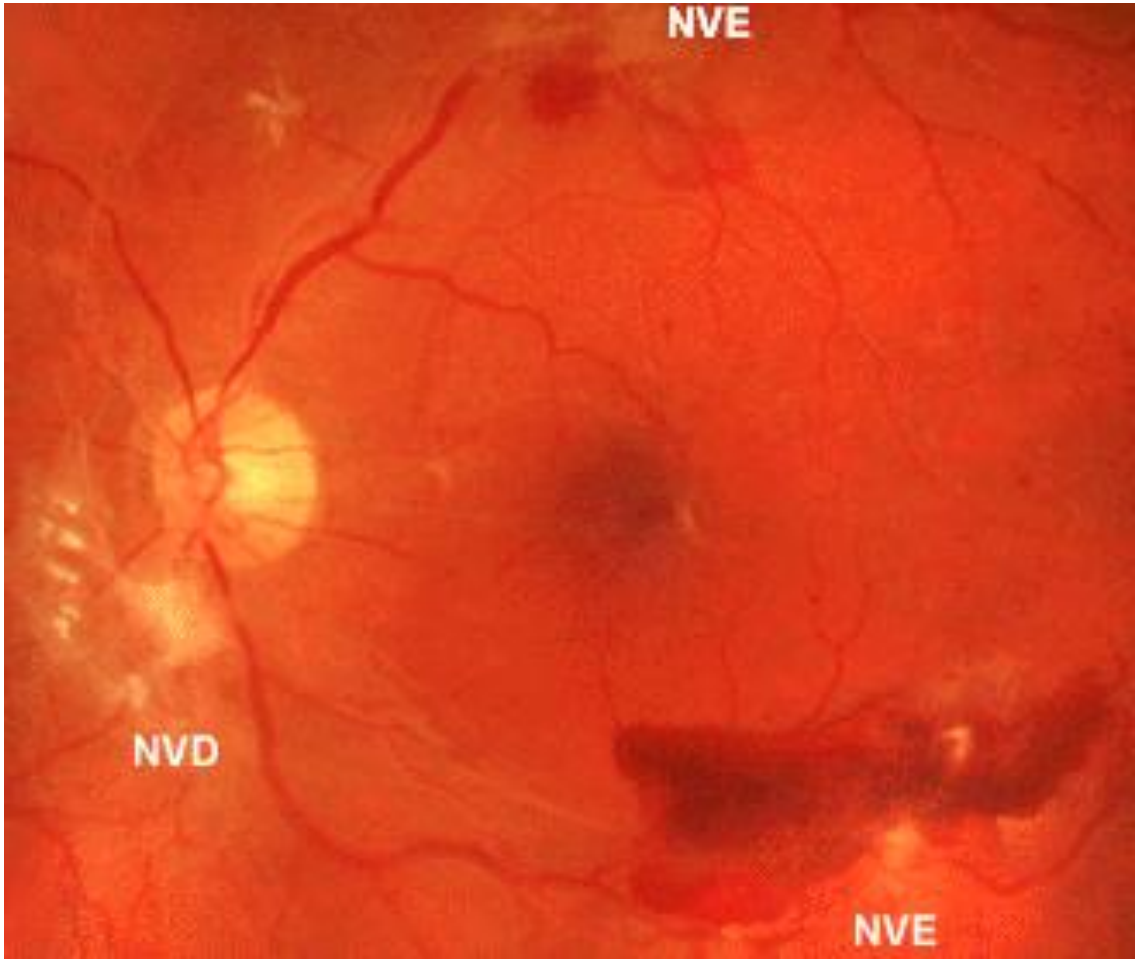
- Bleeding from fragile new vessels involving the retina or optic disc can result in vitreous or retinal haemorrhage. Retinal damage can result from persistent vitreous haemorrhage.

- **Pre-retinal haemorrhages are often associated with retinal neovascularization,** they may dramatically reduce vision within a few minutes.

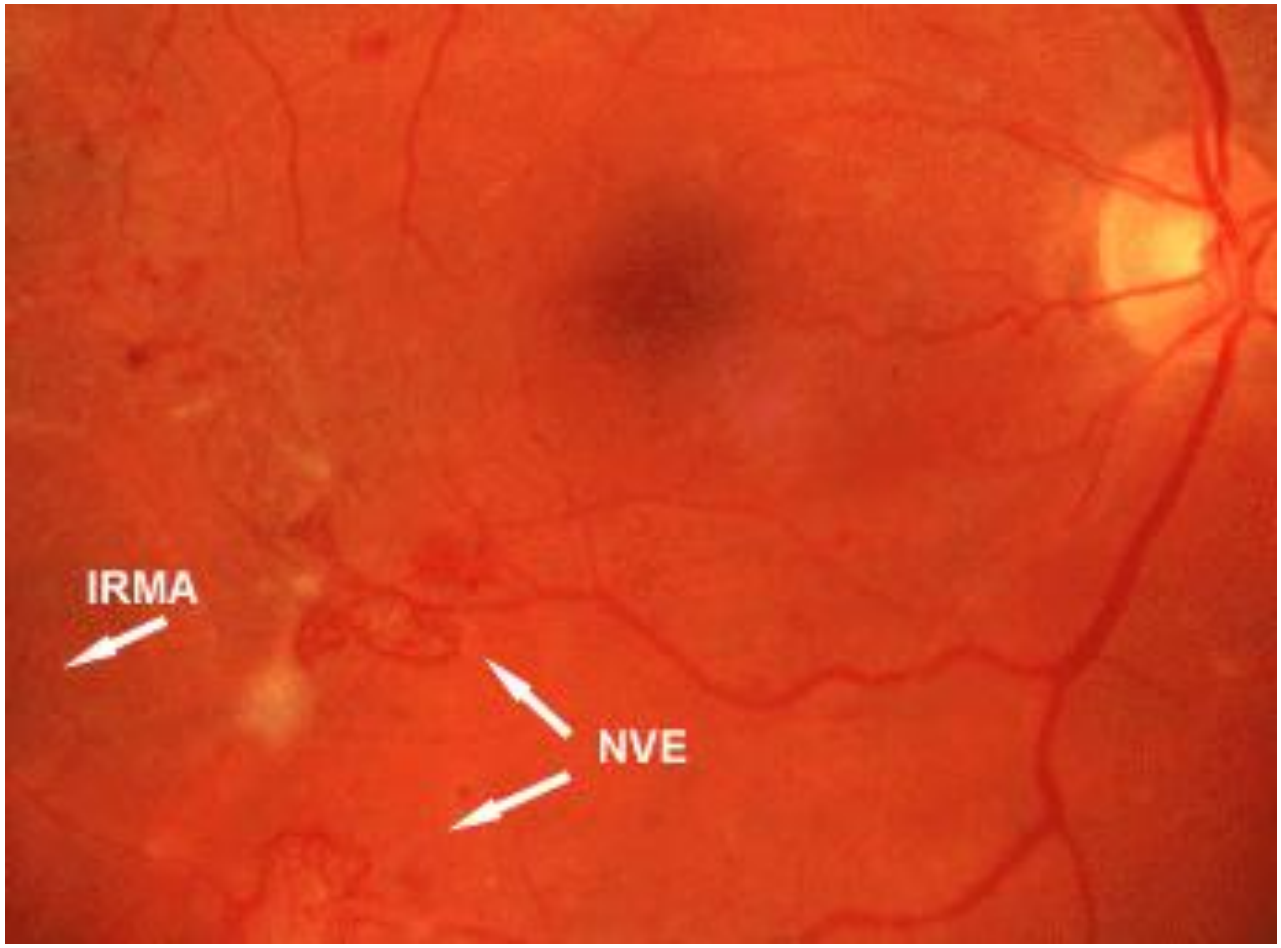
Proliferative diabetic retinopathy



Proliferative diabetic retinopathy



Proliferative diabetic retinopathy

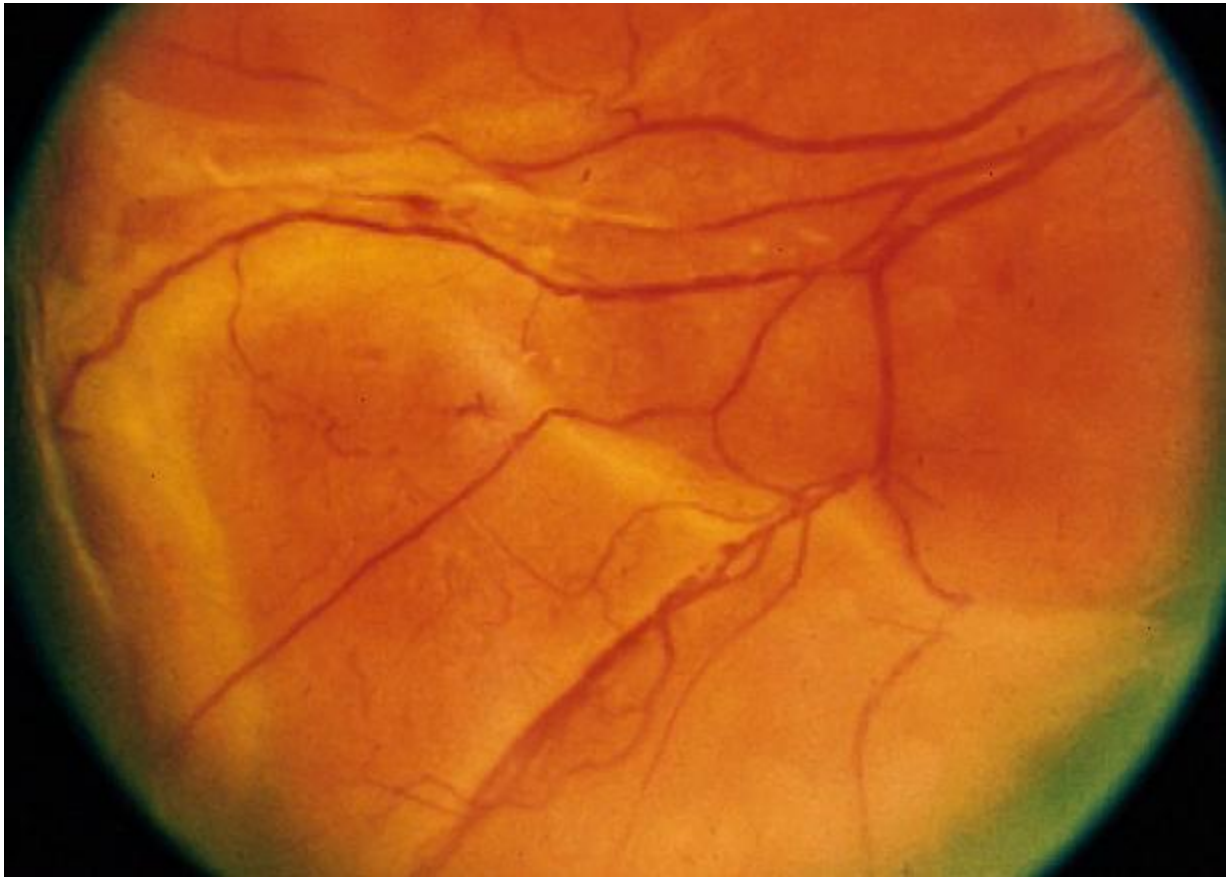


Late Disease

- Contraction of associated fibrous tissue formed by proliferative disease tissue can result in deformation of the retina and tractional retinal detachment

- Rhegmatogenous Vs Nonrhegmatogenous

Late Complications



SIGNS AND SYMPTOMS

- Some common symptoms of diabetic retinopathy are listed below, however, diabetes may cause other eye symptoms.
  - Blurred vision (this is often linked to blood sugar levels)
  - Floaters and flashes
  - Sudden loss of vision

Normal vision

Vision with diabetic retinopathy

Screening for diabetic eye problems should ideally include the following,

1. The history of any visual symptoms or changes in vision

2. Measurement of visual acuity (unaided, with spectacles / pinhole as necessary)

3. Iris examination by slit lamp biomicroscopy prior to pupil mydriasis.

4. Pupil mydriasis. (tropicamide 0.5 %) -the risk of precipitating angle closure glaucoma is actually very small. Patients should be accompanied by a relative and instructed not to drive home.

5. Examination of the crystalline lens by slit lamp biomicroscopy.

6. Fundus examination by slit lamp biomicroscopy using diagnostic contact lens or slit lamp indirect ophthalmoscopy.

Slit Lamp Biomicroscopy

Slit lamp examination (using either indirect ophthalmoscopy with a convex aspheric lens or diagnostic contact lens) yields much more information by providing stereoscopic assessment of retinal thickening and proliferative retinopathy, particularly important when assessing **possible retinal traction.**

Slit lamp biomicroscopy of the fundus : For detection and monitoring of diabetic eye disease.

Photoscreening

- An alternative to slit lamp biomicroscopy is the photoscreening of diabetic patients with a fundus camera.

- Photoscreening **will not always detect subtle signs of DR , such as retinal thickening, but a success rate of 80-92% in detecting DR** is claimed by researchers. There are numerous photographic techniques used ranging from a single photograph to a 9 photograph collage.

- Three photographs spread across the posterior pole are now widely regarded as being most cost efficient.

A protocol for diabetic screening and Monitoring

Type 2 diabetic patients without retinopathy should be assessed at the time of diagnosis and bi-annually thereafter.

- Patients with diabetes and mild non-proliferative retinopathy should be assessed every 12 months by a suitably experienced practitioner.

- Screening doctors should always look, in particular, for the onset of clinically significant macular oedema (CSME).

Type 1 diabetics rarely develop retinopathy until after eight years of diabetic life. The current recommendation is that screening is unnecessary for at least the first five years of the disease and that patients without retinopathy should be screened annually after the onset of puberty until the onset of non-proliferative diabetic retinopathy (NPDR).

General aspects of the ocular care of diabetics

- Factors that can worsen diabetic retinopathy- and indeed the general prognosis of diabetes, include poor diabetic control, systemic hypertension,hyperlipidaemia, cigarette smoking, diabetic nephropathy, anaemia, pregnancy and cataract surgery

Glycaemic control

- It is now proven that good diabetic control may slow the development and progression of diabetic retinopathy in both type 1 and type 2 diabetes.

- For example, the United Kingdom Prospective Diabetes Study 1998 (UKPDS) followed 5,102 newly diagnosed type 2 diabetics prospectively since 1977. Those diabetics who were intensively treated and achieved tight control with either insulin or suphonylurea had diabetic endpoints 12% lower than less well controlled diabetics.

  Overall there was a 25% reduction in microvascular end points in the group exhibiting good glycaemic control.

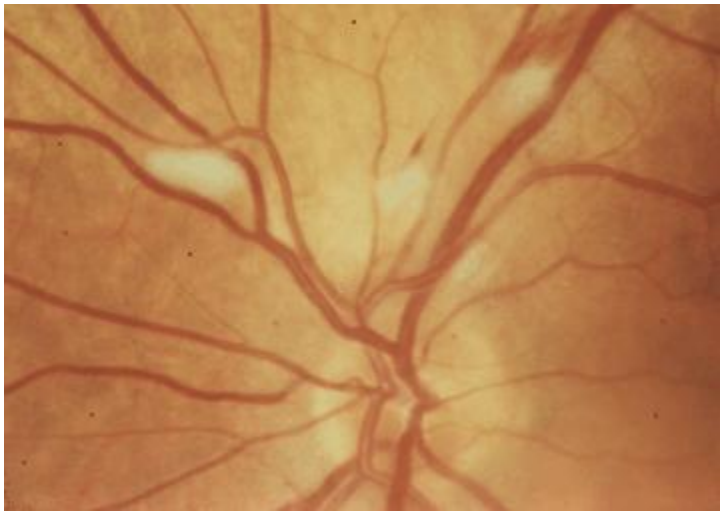
Pregnancy

- **Diabetic retinopathy may worsen during pregnancy.** Screening should therefore be undertaken at confirmation of pregnancy and **every two months during pregnancy** if no retinopathy is present, or monthly, if retinopathy is present.

Systemic hypertension and DR in type 2 diabetes

- Recent literature indicates that there is a striking correlation between the presence of systemic hypertension and progression of diabetic retinopathy. Recent studies have delineated **the role of treating associated hypertension and the slowing of the progress of DR**. It is important to note that many type 2 diabetics will need a combination of anti-hypertensive agents to lower their blood pressure.

Hypertension



Systemic hypertension and DR in type 2 diabetes

- The risk of reduction of visual acuity was lowered by 47%.

- Atenolol and Captopril were equally effective in reducing the risk of progression of retinopathy in type 2 diabetics.

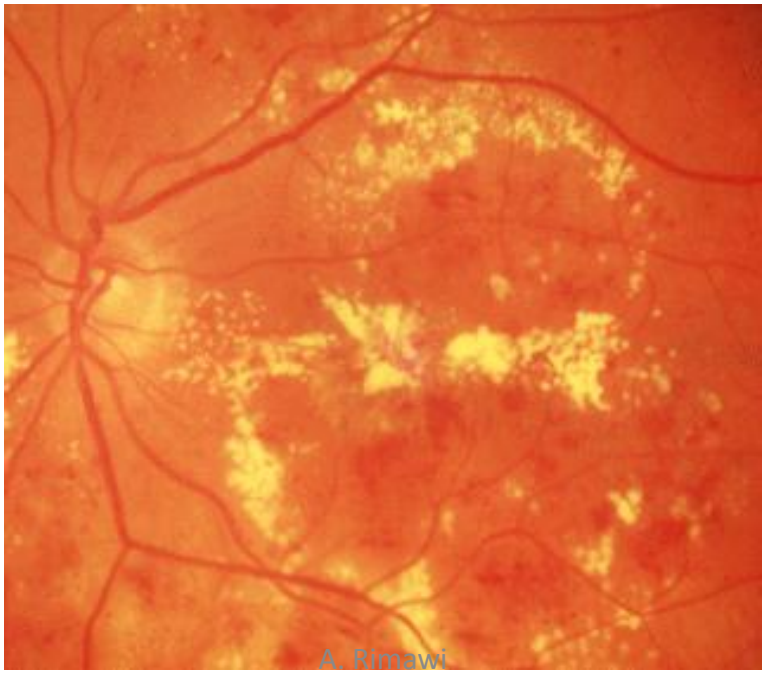
Angiotensin Converting Enzyme (ACE) inhibitors in Type 1 diabetes

- The EUCLID study is currently investigating the prophylactic treatment of type 1 diabetics with the Angiotensin Converting Enzyme (ACE) Inhibitor Lisinopril and the progression of nephropathy and other microvascular disease including DR . Preliminary reports are of a specific benefit are encouraging, with a claimed 50% reduction in progression of DR in type 1 diabetics.

- The study did not look at maculopathy- so that implications are unclear for type 2 diabetics, although **no specific advantage of ACE inhibitors (Captopril) over Atenolol was seen in UKPDS.**

Hyperlipidaemia and diabetic maculopathy

- There is evidence in the literature that diabetics who have exudative maculopathy with extensive lipid exudes benefit from active treatment of hyperlipidaemia



Diabetic nephropathy

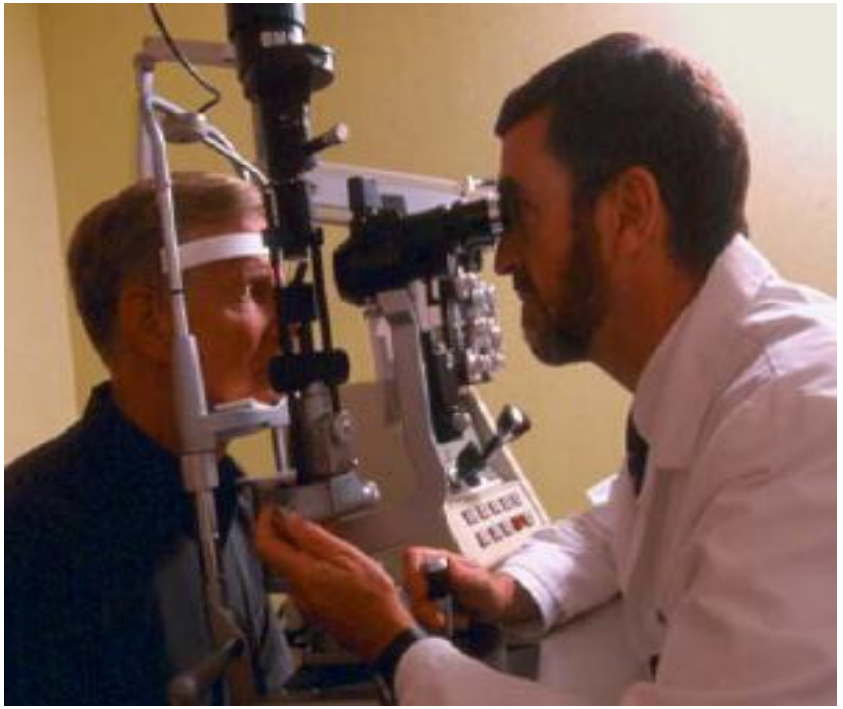
- Diabetic nephropathy accelerates the progression of retinopathy, especially macular oedema, *inter alia* via increased levels of fibrinogen and lipoprotein and associated hypertension.
- **The visual prognosis is often better if the nephropathy is treated by renal transplantation rather than by dialysis**
- Any anaemia resulting from renal disease must be aggressively treated.
- **Diabetic retinopathy is a common prelude to the development of renal disease.**

Cataract surgery

- Cataract surgery may lead to progression of pre-existing macular oedema and proliferative diabetic retinopathy. However, cataracts may impede fundoscopy and therefore interfere with the treatment of diabetic retinopathy. **If possible, diabetic retinopathy should be treated prior to cataract surgery**

Diagnosing diabetic retinopathy

- **Diabetes can cause vision in both eyes to change, even if you do not have retinopathy.**
- **Rapid changes in your blood sugar alter the shape of the lens of the eye; the image on the retina will become out of focus.**
- **You can reduce episodes of blurred vision by maintaining good control of your blood sugar.**



Diagnosing diabetic retinopathy

- **People with diabetes should see their ophthalmologist immediately if they have visual changes that:**
  - affect only one eye
  - last more than a few days
  - are not associated with a change in blood sugar.
- **Ideally, your blood sugar levels should be consistently controlled for several weeks prior to seeing your ophthalmologist for an exam.**
  - Erratic blood sugar control causes a change in the focusing power of the eye, interfering with eyeglasses prescription measurements.

When to schedule an eye exam

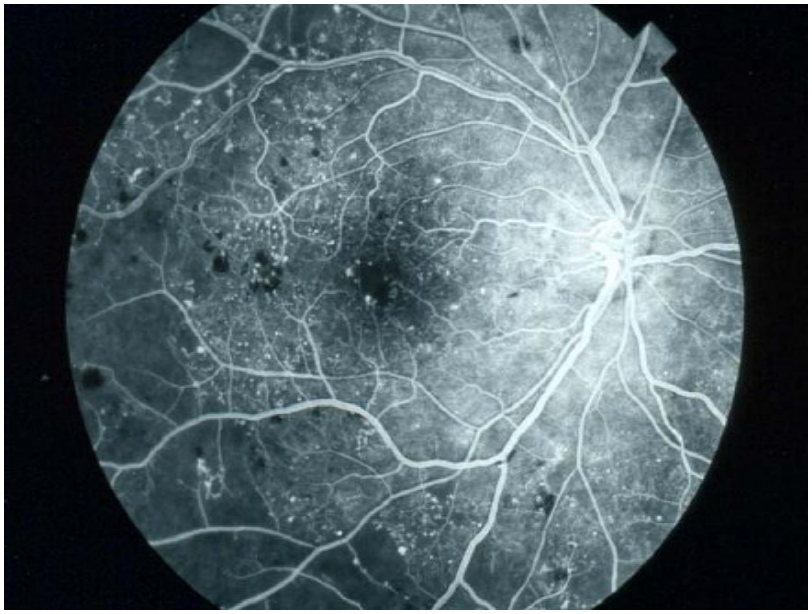
- **If you were 30 years old or younger when your diabetes was first detected, you should have your first eye exam within five years after that diagnosis.**
- **If you were 30 years old or older, your first exam should be within a few months of the diabetes diagnosis.**
- **If you are pregnant, you should have an exam within the first trimester.**
- **If you already have experienced a high-risk condition, such as kidney failure or amputation related to diabetes, schedule an eye exam immediately.**

What happens during an eye exam

- **Fluorescein angiography: a diagnostic procedure using a special camera to take photographs of the retina after a small amount of water soluble dye (fluorescein) is injected into a vein in the arm.**

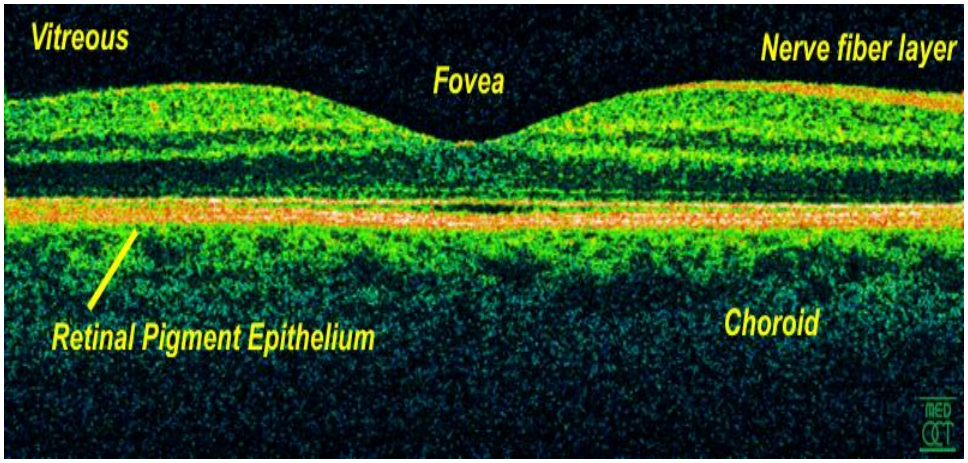
- **The photographs of fluorescein dye traveling throughout the retinal vessels show:**
  - which blood vessels are leaking
  - the severity of the leakage
  - which blood vessels are closed
  - whether or not neovascularization is present

- **Fluorescein angiography helps in determine:**
  - **Why vision is blurred.**
  - **Whether or not laser treatment should be started.**
  - **Where to apply laser treatment.**
  - **How effective laser treatment may be.**

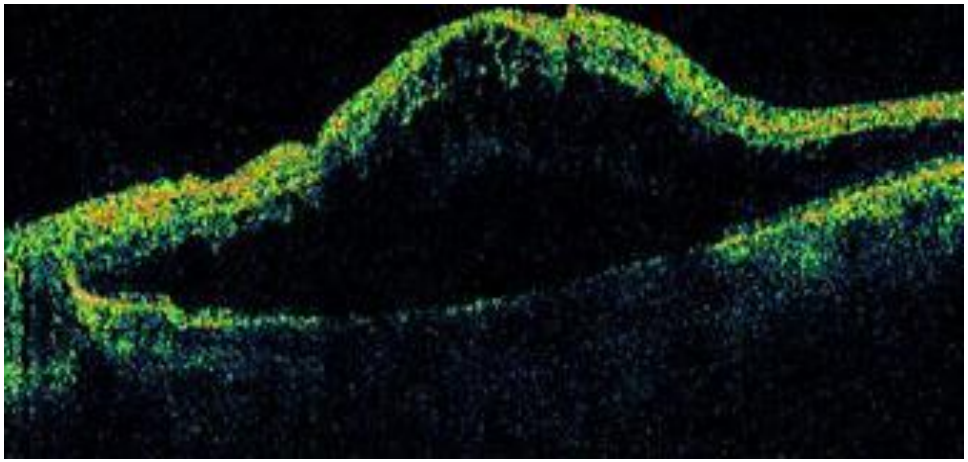


Optical Coherence Tomography (OCT)

- OCT is a diagnostic tool for diseases of the **macula** (central retina) and **optic nerve.**
- OCT uses non-invasive coherent light (in which the electromagnetic waves maintain a fixed phase relationship with each other) to create a microscopic cross sectional image of the macula and optic nervehead capable of resolution to between 8 and 10 microns.
- In diabetics, OCT can “map” areas of macular edema (“swelling”) thus facilitating fluorescein angiography in guiding laser treatment of the macula. Repeat OCT studies may assist the ophthalmologist in assessing response to treatment and recurrence of macular edema.

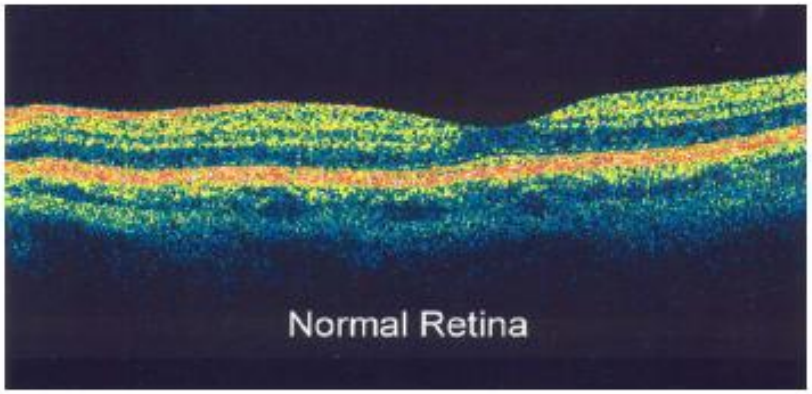


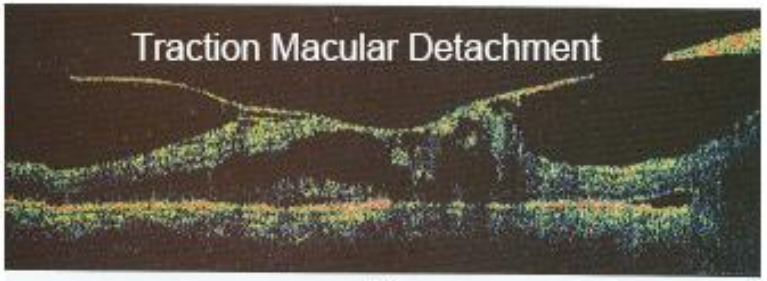
- Normal OCT demonstrating normal retinal contour.



- OCT of a patient with diabetic macular oedema. The retinal layers are swollen.

OCT


Normal Retina


Traction Macular Detachment

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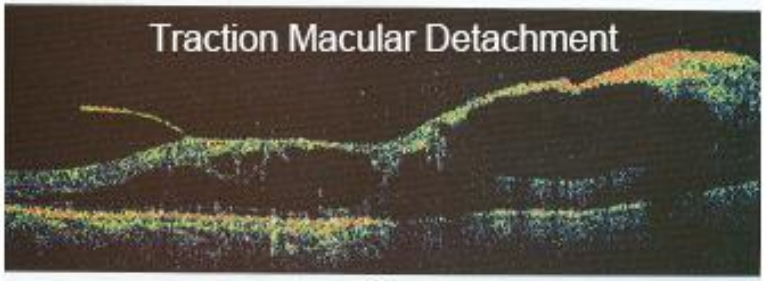
Traction Macular Detachment

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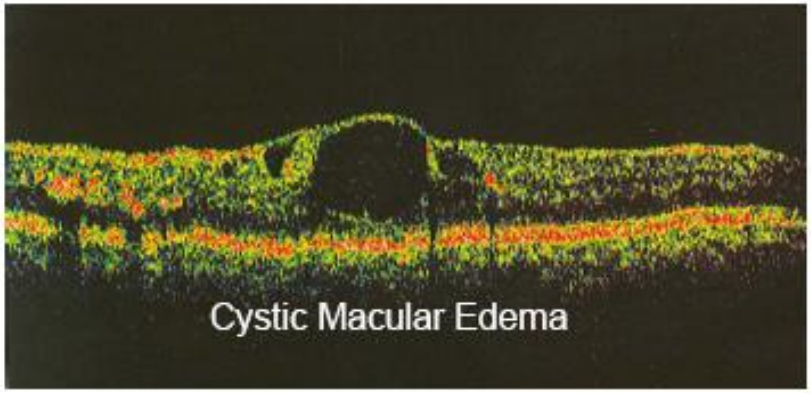
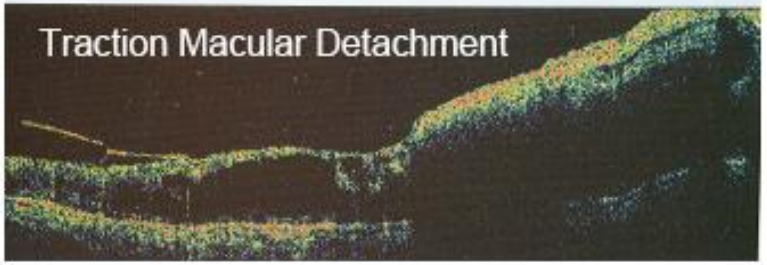

Cystic Macular Edema

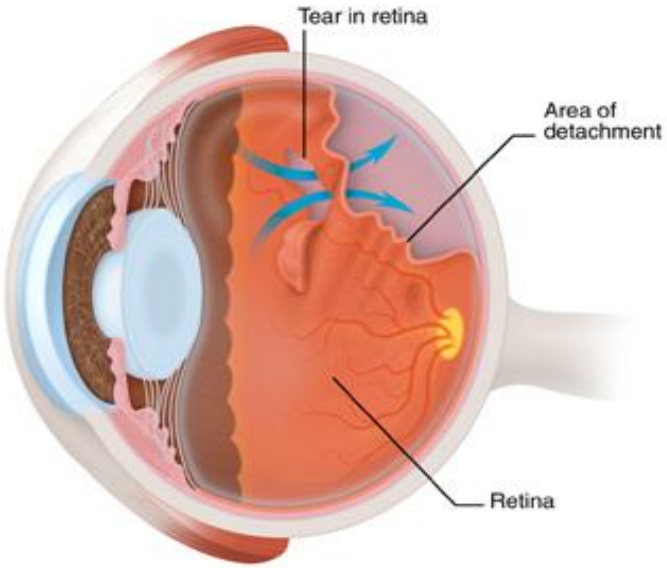

Traction Macular Detachment

What happens during an eye exam

Ultrasound

- The ultrasound “sees” through the blood to determine if the retina has detached.
- If there is **detachment** near the macula, prompt surgery may be necessary.

- After evaluation, the ophthalmologist / retinal specialist will decide whether to treat or re-examine.



Retinal detachment

Treating diabetic retinopathy

“An ounce of prevention is worth a pound of cure”.

- **Strict control of your blood sugar will significantly reduce the longterm risk of vision loss from diabetic retinopathy.**
- **Laser treatment (photocoagulation) is often recommended for people with macular edema, PDR, and neovascular glaucoma**

United Kingdom Prospective Diabetes Study (UKPDS)

- The UKPDS, the largest clinical study of diabetes ever attempted, has shown for the first time that the life-threatening complications of type 2 (non-insulin dependent) diabetes, often regarded as inevitable, can be reduced by more intensive management. The 20-year study recruited over 5,000 patients with type 2 diabetes in England, Northern Ireland and Scotland.
- **The UKPDS has revealed that better blood glucose control reduces the risk of**
  - diabetic retinopathy & cataract by 25%
  - early kidney disease by 30-35%.
- **The UKPDS has also demonstrated that better blood pressure control (in the many patients who have high BP) reduces the risk of:**
  - serious deterioration of vision by more than a third,
  - death from long-term complications of diabetes by a third,
  - strokes by more than a third.

Treating diabetic retinopathy

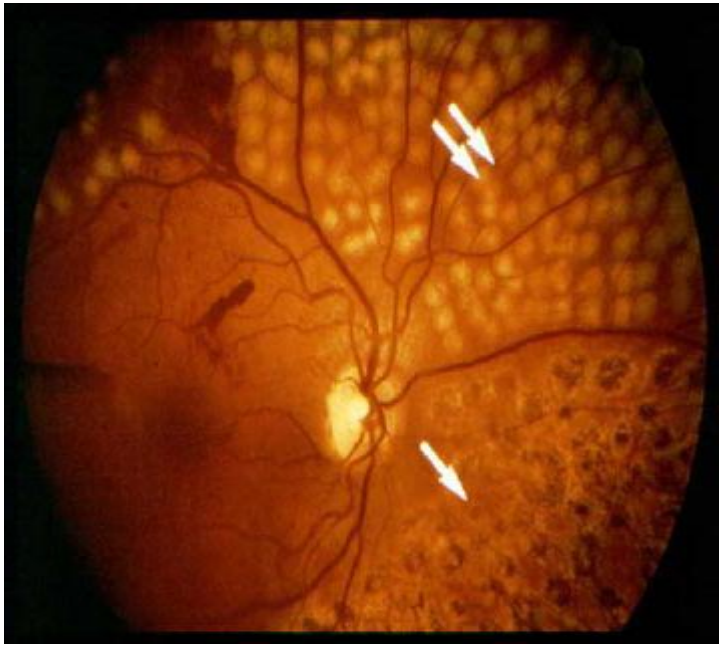
Laser surgery for macular edema

- **Low energy laser is focused onto microaneursysms in the macula to decrease leakage.**
- **Patients may see laser spots near the center of their vision following treatment; usually fade with time, but may not entirely disappear.**
- **Uncommon for people who have blurred vision from macular edema to recover normal vision, although some may experience partial improvement.**
- **Main goal of treatment: prevent further loss of vision.**

Treating diabetic retinopathy

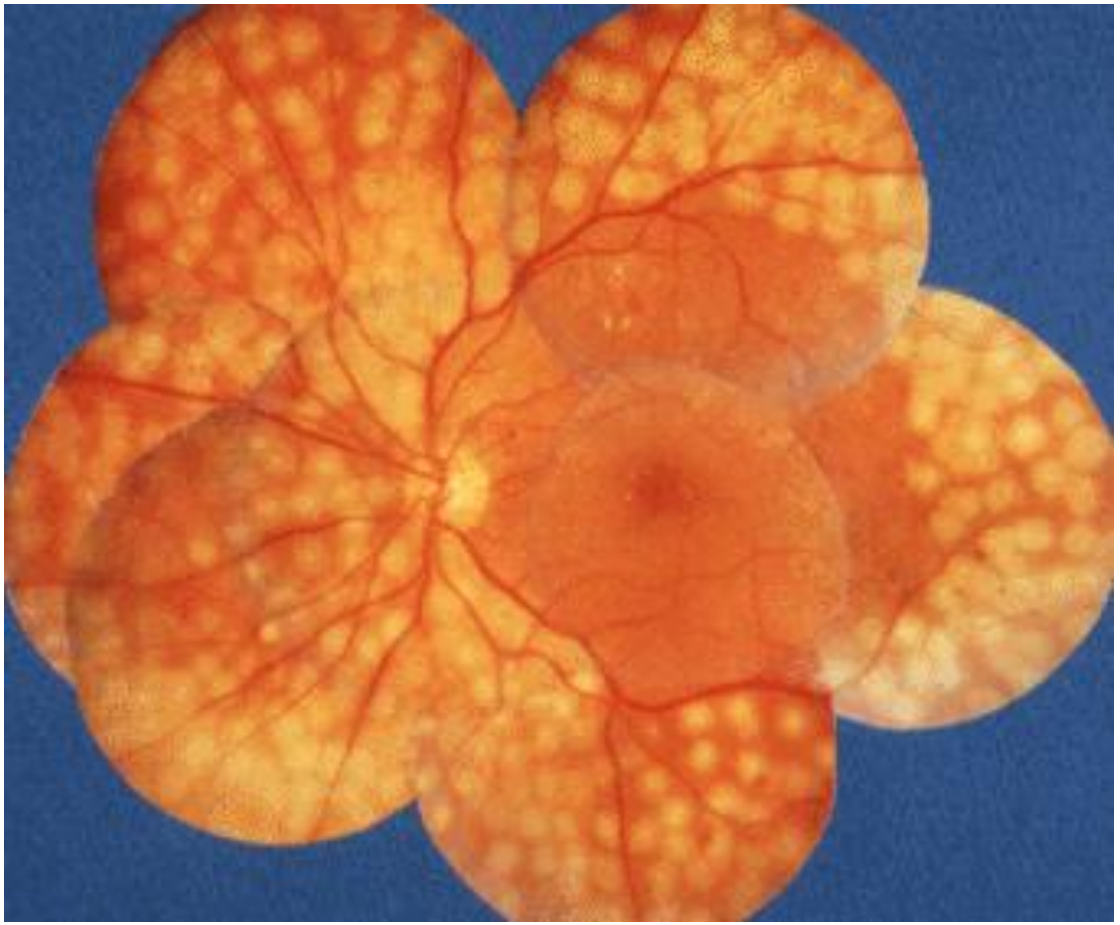
Laser surgery for PDR

- **Laser is focused on all parts of the retina except the macula.**
- **This "panretinal" photocoagulation treatment causes abnormal new vessels (neovascularization) to shrink; often prevents them from recurring.**
- **Treatment decreases the chance that vitreous bleeding or retinal detachment will occur.**
- **Multiple laser treatments over time are usually necessary.**

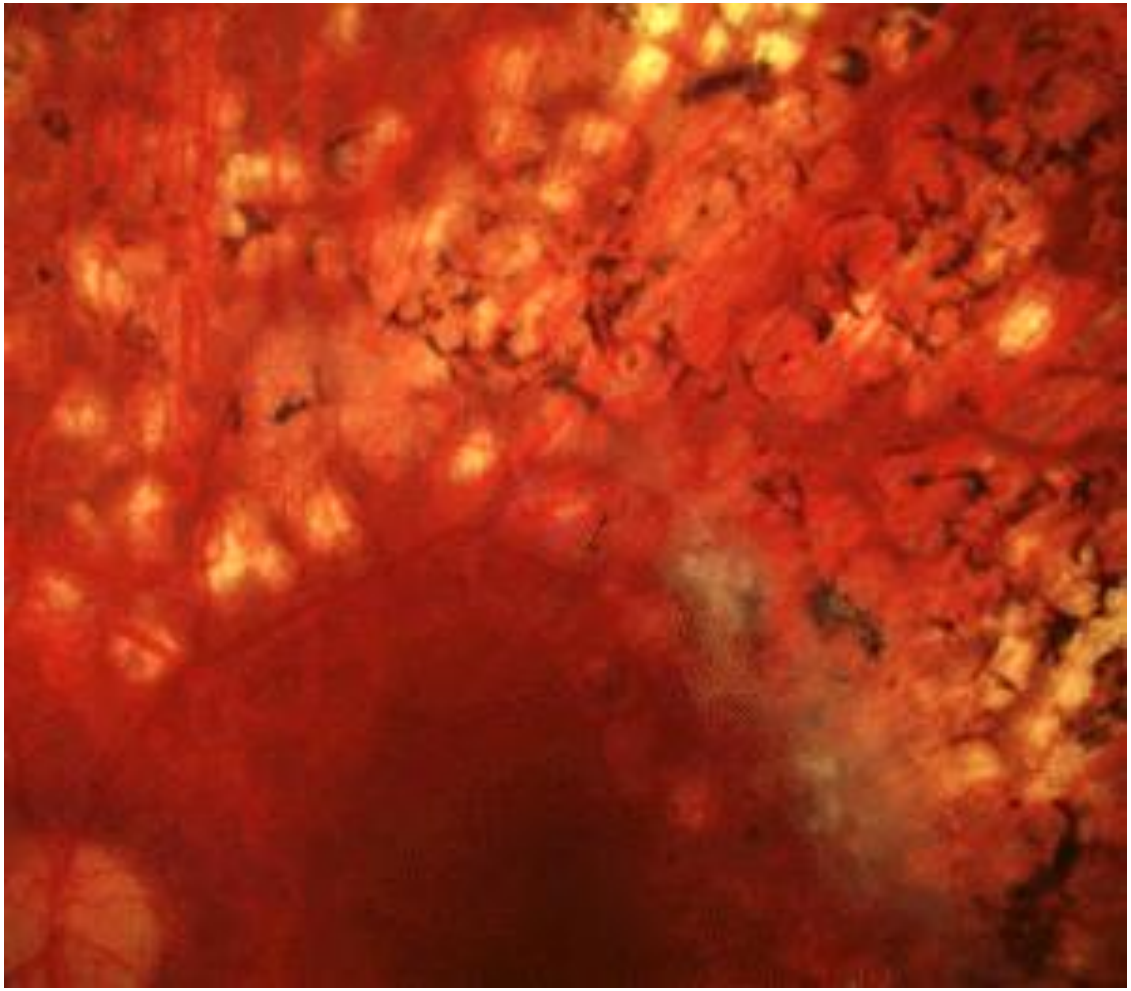


laser panretinal photocoagulation treatment (arrows show laser spots on the retina)

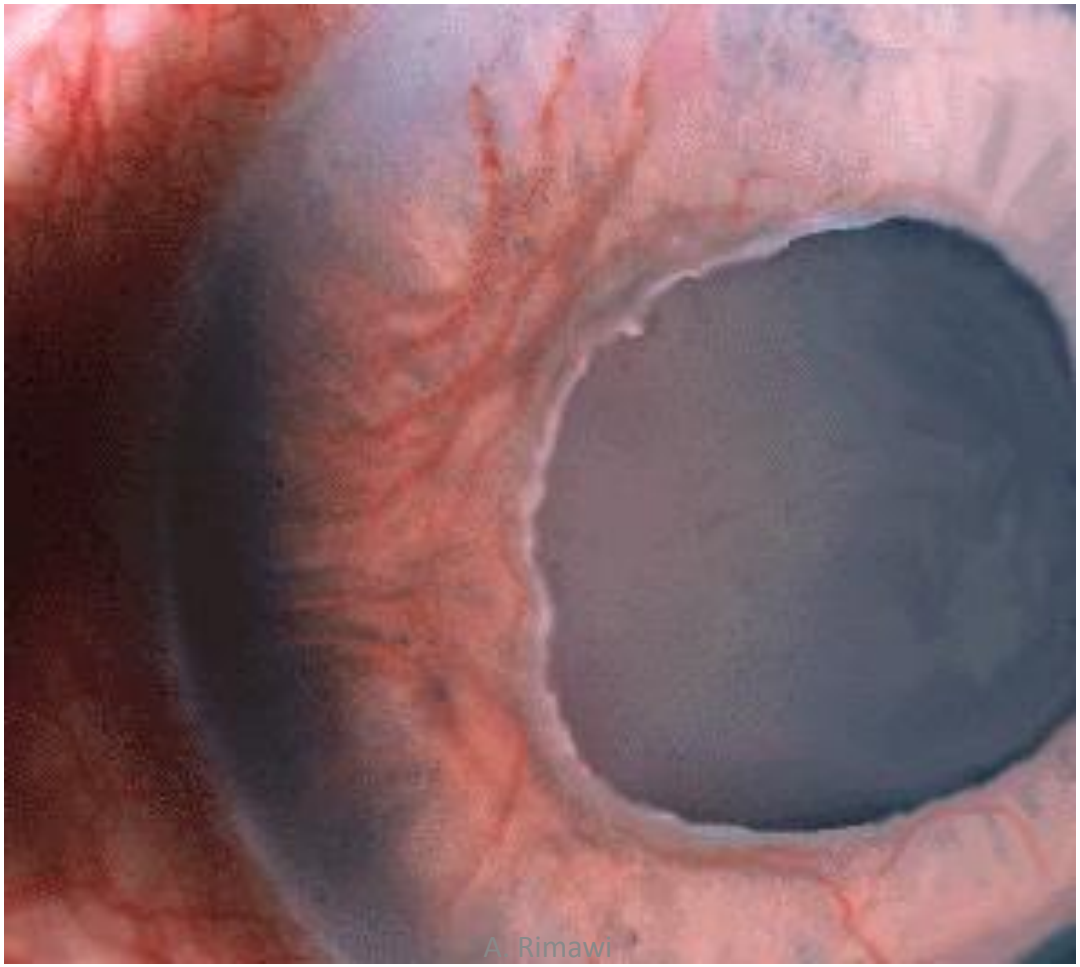
Panretinal laser photocoagulation



Panretinal laser photocoagulation for proliferative DR



Iris Neovascularisation



Complications of laser photocoagulation

Although laser therapy can be highly effective in preventing blindness, it is associated with numerous complications.

- **Retinal vein occlusion** can follow inadvertent photocoagulation of a retinal vein. Rarely, there may be loss of central acuity from inadvertent photocoagulation of the fovea.
- **Vitreous haemorrhage** can follow photocoagulation of retinal or choroidal vessels.
- There may be visual field restriction, **decreased contrast sensitivity, impaired night vision or impaired colour vision.**
- **Headache** can sometimes follow laser therapy. The headache is usually relieved with rest and simple analgesia. *Glaucoma* must be excluded if the headache is severe or persistent.

Drug Therapy for Diabetic Retinopathy

Intravitreal drug injections:

- **triamcinolone (a corticosteroid; mechanism of action unclear)**
- **Macugen, Lucentis, & Avastin (drugs primarily used to manage neovascular (“wet”) age-related macular degeneration (reduction in the severity of retinopathy by inhibiting VEGF).**
- **Intravitreal drug injections are usually perfomed with laser procedures in order to enhance response to treatment**

Treating diabetic retinopathy with Vitrectomy surgery for advanced PDR

Indications: vitreous hemorrhage (clear, gel-like substance in middle of eye) fills with non-clearing blood and **traction retinal detachment.**

Performed in the operating room, this microsurgical procedure involves removing the blood-filled vitreous and removal of neovascular (new vessel) membranes (fibrovascular proliferation)

Improves vision by re-establishing clear vitreous fluids and lowers the probability of future bleeding by removing the neovascular membranes.

Removal of scar-like (fibrovascular) membranes results in retinal

reattachment and possible improvement in vision.

ARTERIAL OCCLUSION

ARTERIAL OCCLUSION

PATHOGENESIS:

- Central and branch retinal artery occlusions are usually embolic in origin.

- Three types of emboli are recognized:
  - ***fibrin-platelet emboli commonly from diseased carotid arteries.***
  - ***cholesterol emboli commonly from diseased carotid arteries.***
  - ***Calcific emboli from diseased heart valves.***

HISTORY:

The patient complains of **a sudden painless loss of all or part** of the vision.

- Fibrin platelet emboli typically cause a fleeting loss of vision as the emboli passes through the retinal circulation (*amaurosis fugax). This may last for* some minutes and then clears.

- Cholesterol and calcific emboli may result in permanent obstruction with no recovery in vision (they may also be seen in the retinal vessels of asymptomatic individuals).

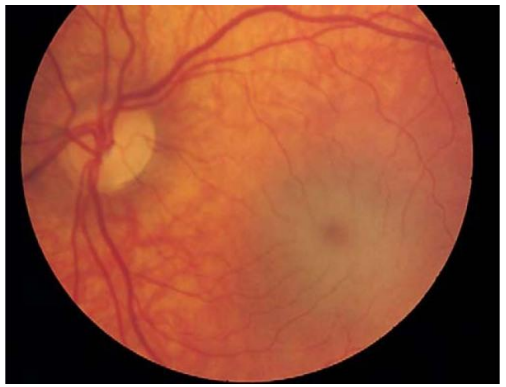
- A central retinal artery obstruction is frequently caused by an embolus, although as it lodges further back in the arterial tree behind the optic nerve head, it cannot be seen.

- In young patients, transient loss of vision may be caused by migraine.

SIGNS

- Occasionally, a series of white platelet emboli can be seen passing rapidly through a vessel; more often a bright yellow, reflective cholesterol embolus is noted occluding an arterial branch point.

- The acutely affected retina is swollen and white (*oedematous), while the fovea is red (cherry red spot) as it has no supply from the retinal circulation, is not swollen, and the* normal choroid can be seen through it.



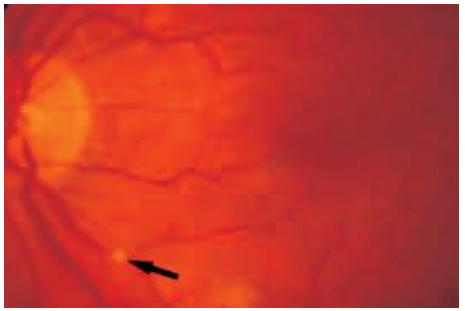
- After several weeks the disc becomes pale (*atrophic) and the arterioles attenuated. The condition may* also occasionally be caused by vasculitis, such as giant cell arteritis.

INVESTIGATION

- Patients require a careful vascular work-up since disease in the eye may reflect systemic vascular disease.
- A search for carotid artery disease should be made by assessing the strength of carotid pulsation and listening for bruits.
- Ischaemic heart disease, peripheral claudication and hypertension may also be present.

- Appearance of a cholesterol embolus (arrow).

They appear to sparkle when viewed with a direct ophthalmoscope.



- A carotid endarterectomy may be indicated to prevent the possibility of a cerebral embolus if a stenosis of the carotid artery greater than 75% is present.

- Doppler ultrasound allows non-invasive imaging of both the
- carotid and vertebral arteries to detect such a stenosis.

TREATMENT & PROGNOSIS

- Acute treatment of central and branch artery occlusions is aimed at dilating the arteriole to permit the embolus to pass more distally.
- Results are usually disappointing although a trial is worthwhile if the patient is seen within 24 hours of onset of the obstruction.
- The patient is referred to an eye unit where the following measures may be tried:
  - lowering the intraocular pressure with intravenous acetazolamide;
  - ocular massage
  - paracentesis (a needle is inserted into the anterior chamber to release aqueous and lower the intraocular pressure rapidly).
  - getting the patient to rebreath into a paper bag firmly applied around the mouth and nose to use the vasodilatatory effect of raised carbon dioxide levels.

- **The prognosis:** Full visual recovery occurs with amaurosis fugax but more prolonged arterial occlusion results in severe unrecoverable visual loss.