Diabetic retinopathy

Diabetic retinopathy is the most common cause of blindness in adults aged 35-65 years-old. Hyperglycaemia is thought to cause increased retinal blood flow and abnormal metabolism in the retinal vessel walls. This precipitates damage to endothelial cells and pericytes

Endothelial dysfunction leads to increased vascular permeability which causes the characteristic exudates seen on fundoscopy. Pericyte dysfunction predisposes to the formation of microaneurysms. Neovasculization is thought to be caused by the production of growth factors in response to retinal ischaemia

In exams you are most likely to be asked about the characteristic features of the various stages/types of diabetic retinopathy. Recently a new classification system has been proposed, dividing patients into those with non-proliferative diabetic retinopathy (NPDR) and those with proliferative retinopathy (PDR):

Traditional classification

Background retinopathy

- microaneurysms (dots)
- blot haemorrhages (<=3)
- hard exudates

Pre-proliferative retinopathy

- cotton wool spots (soft exudates; ischaemic nerve fibres)
- > 3 blot haemorrhages
- venous beading/looping
- deep/dark cluster haemorrhages
- more common in Type I DM, treat with laser photocoagulation

New classification

Mild NPDR

- 1 or more microaneurysm

Moderate NPDR

- microaneurysms
- blot haemorrhages
- hard exudates
- cotton wool spots, venous beading/looping and intraretinal microvascular abnormalities (IRMA) less severe than in severe NPDR

Severe NPDR

- blot haemorrhages and microaneurysms in 4 quadrants
- venous beading in at least 2 quadrants
- IRMA in at least 1 quadrant

Proliferative retinopathy

- retinal neovascularisation - may lead to vitrous haemorrhage
- fibrous tissue forming anterior to retinal disc
- more common in Type I DM, 50% blind in 5 years

Maculopathy

- based on location rather than severity, anything is potentially serious
- hard exudates and other 'background' changes on macula
- check visual acuity
- more common in Type II DM

Primary open-angle glaucoma

Glaucoma is a group disorders characterised by optic neuropathy due, in the majority of patients, to raised intraocular pressure (IOP). It is now recognised that a minority of patients with raised IOP do not have glaucoma and vice versa

Primary open-angle glaucoma (POAG, also referred to as chronic simple glaucoma) is present in around 2% of people older than 40 years. Other than age, risk factors include:

- family history
- black patients
- myopia
- hypertension
- diabetes mellitus

POAG may present insidiously and for this reason is often detected during routine optometry appointments. Features may include

- peripheral visual field loss - nasal scotomas progressing to 'tunnel vision'
- decreased visual acuity
- optic disc cupping

Red eye

There are many possible causes of a red eye. It is important to be able to recognise the causes which require urgent referral to an ophthalmologist. Below is a brief summary of the key distinguishing features

Acute angle closure glaucoma
- severe pain (may be ocular or headache)
- decreased visual acuity, patient sees haloes
- semi-dilated pupil
- hazy cornea

Anterior uveitis
- acute onset
- pain
- blurred vision and photophobia
- small, fixed oval pupil, ciliary flush

Scleritis
- severe pain (may be worse on movement) and tenderness
- may be underlying autoimmune disease e.g. rheumatoid arthritis

Conjunctivitis
- purulent discharge if bacterial, clear discharge if viral

Subconjunctival haemorrhage
- history of trauma or coughing bouts

Age related macular degeneration

Age related macular degeneration is the most common cause of blindness in the UK. Degeneration of the central retina (macula) is the key feature with changes usually bilateral.

Traditionally two forms of macular degeneration are seen:

- dry (geographic atrophy) macular degeneration: characterised by drusen - yellow round spots in Bruch's membrane
- wet (exudative, neovascular) macular degeneration: characterised by choroidal neovascularisation. Leakage of serous fluid and blood can subsequently result in a rapid loss of vision. Carries worst prognosis

Recently there has been a move to a more updated classification:

- early age related macular degeneration (non-exudative, age related maculopathy): drusen and alterations to the retinal pigment epithelium (RPE)
- late age related macular degeneration (neovascularisation, exudative)

Risk factors

- age: most patients are over 60 years of age
- smoking
- family history
- more common in Caucasians
- high cumulative sunlight exposure
- female sex

Features

- reduced visual acuity: 'blurred', 'distorted' vision, central vision is affected first
- central scotomas
- fundoscopy: drusen, pigmentary changes

Investigation and diagnosis

- optical coherence tomography: provide cross sectional views of the macula
- if neovascularisation is present fluorescein angiography is performed

General management

- stop smoking
- high dose of beta-carotene, vitamins C and E, and zinc may help to slow down visual loss for patients with established macular degeneration. Supplements should be avoided in smokers due to an increased risk of lung cancer

Dry macular degeneration - no current medical treatments

Wet macular degeneration

- photocoagulation
- photodynamic therapy
- anti-vascular endothelial growth factor (anti-VEGF) treatments: intravitreal ranibizumab

Primary open-angle glaucoma: management

The majority of patients with primary open-angle glaucoma are managed with eye drops. These aim to lower intra-ocular pressure which in turn has been shown to prevent progressive loss of visual field.

Medication	Mode of action	Notes
Prostaglandin analogues (e.g. Latanoprost)	Increases uveoscleral outflow	Once daily administration Adverse effects include brown pigmentation of the iris
Beta-blockers (e.g. Timolol)	Reduces aqueous production	Should be avoided in asthmatics and patients with heart block
Sympathomimetics (e.g. Brimonidine, an alpha2-adrenoceptor agonist)	Reduces aqueous production and increases outflow	Avoid if taking MAOI or tricyclic antidepressants Adverse effects include hyperaemia
Carbonic anhydrase inhibitors (e.g. Dorzolamide)	Reduces aqueous production	Systemic absorption may cause sulphonamide-like reactions
Miotics (e.g. Pilocarpine)	Increases uveoscleral outflow	Adverse effects included a constricted pupil, headache and blurred vision

Sudden painless loss of vision

The most common causes of a sudden painless loss of vision are as follows:

- ischaemic optic neuropathy (e.g. temporal arteritis or atherosclerosis)
- occlusion of central retinal vein
- occlusion of central retinal artery
- vitreous haemorrhage
- retinal detachment

Ischaemic optic neuropathy

- may be due to arteritis (e.g. temporal arteritis) or atherosclerosis (e.g. hypertensive, diabetic older patient)
- due to occlusion of the short posterior ciliary arteries, causing damage to the optic nerve
- altitudinal field defects are seen

Central retinal vein occlusion

- incidence increases with age, more common than arterial occlusion
- causes: glaucoma, polycythaemia, hypertension
- severe retinal haemorrhages are usually seen on fundoscopy

Central retinal artery occlusion

- due to thromboembolism (from atherosclerosis) or arteritis (e.g. temporal arteritis)
- features include afferent pupillary defect, 'cherry red' spot on a pale retina

Vitreous haemorrhage

- causes: diabetes, bleeding disorders
- features may include sudden visual loss, dark spots

Retinal detachment

- features of vitreous detachment, which may precede retinal detachment, include flashes of light or floaters (see below)

Differentiating posterior vitreous detachment, retinal detachment and vitreous haemorrhage

Posterior vitreous detachment	Retinal detachment	Vitreous haemorrhage
• Flashes of light (photopsia) - in the peripheral field of vision • Floaters, often on the temporal side of the central vision	• Dense shadow that starts peripherally progresses towards the central vision • A veil or curtain over the field of vision • Straight lines appear curved • Central visual loss	• Large bleeds cause sudden visual loss • Moderate bleeds may be described as numerous dark spots • Small bleeds may cause floaters

Thyroid eye disease

Thyroid eye disease affects between 25-50% of patients with Graves' disease. It is thought to be caused by an autoimmune response against an autoantigen, possibly the TSH receptor, which in turns causes retro-orbital inflammation. The patient may be eu-, hypo- or hyperthyroid at the time of presentation

Prevention

- smoking is the most important modifiable risk factor for the development of thyroid eye disease
- radioiodine treatment may increase the inflammatory symptoms seen in thyroid eye disease. In a recent study of patients with Graves' disease around 15% developed, or had worsening of, eye disease. Prednisolone may help reduce the risk

Features

- exophthalmos
- conjunctival oedema
- optic disc swelling
- ophthalmoplegia
- inability to close the eye lids may lead to sore, dry eyes. If severe and untreated patients can be at risk of exposure keratopathy

Management

- topical lubricants may be needed to help prevent corneal inflammation caused by exposure
- steroids
- radiotherapy
- surgery