BRADFORD VTS TEACHING AIDS

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Eyes in Primary Care

A Comprehensive Guide for GP Trainees and IMGs

Contents

2 million
People in the UK have a sight problem

1.5%
Of all GP consultations are eye-related

50%
Of glaucoma cases remain undiagnosed

60+
AMD is the leading cause of blindness over age 60

Before You Start — Three Rules to Live By

- Visual acuity is the vital sign of the eye — always measure it first.
- Any painful red eye in a contact lens wearer needs urgent same-day referral.
- Sudden painless visual loss is a medical emergency until proven otherwise.

1. Eye Anatomy & Physiology

A solid grasp of eye anatomy lets you interpret symptoms rapidly. Think of the eye as a camera: the **cornea and lens** focus light, the **retina** is the film, the **optic nerve** sends the image to the brain, and the **aqueous humour** maintains the pressure that keeps the whole structure inflated.

Structure	Function	Clinical Relevance
Cornea	Refracts ~70% of incoming light; more than the lens	Must stay clear; ulcers and abrasions reduce vision
Lens	Fine-tunes focus by changing shape (accommodation)	Becomes rigid with age (presbyopia); cloudy = cataract
Aqueous humour	Produced by ciliary body; drains via Canal of Schlemm	Blocked drainage → raised IOP → glaucoma
Retina	Photoreceptors convert light to nerve signals	Macula = central vision; periphery = movement detection
Choroid	Highly vascular layer between sclera and retina	Supplies blood to outer retina; involved in uveitis
Vitreous	Gel filling the main chamber; firm in youth, liquefies with age	Detachment causes floaters/flashes; can pull on retina
Optic nerve	Carries visual signals to the visual cortex	RAPD indicates optic nerve or severe retinal disease

Refractive Errors at a Glance

Condition	What happens	Correction
Myopia (short sight)	Image focused in front of retina	Concave (minus) lens
Hypermetropia (long sight)	Image focused behind retina	Convex (plus) lens
Astigmatism	Cornea shaped like a spoon — different axes focus differently	Cylindrical lens
Presbyopia	Lens loses elasticity — near focus fails (universal after ~45 yrs)	Reading glasses

Anatomy Pearls

• Pre-auricular lymphadenopathy accompanies adenoviral and chlamydial conjunctivitis — always palpate the pre-auricular nodes when examining a red eye.

• Corneal sensation is carried by CN V (trigeminal). Loss of corneal sensation is seen in herpes simplex keratitis and can lead to neurotrophic ulcers.

2. Red Eye & Allergic Disease

Red eye is one of the most common eye presentations in primary care. Your job is to distinguish the **benign and self-limiting** (e.g. viral conjunctivitis) from the **sight-threatening emergencies** (e.g. acute angle-closure glaucoma, keratitis). The history and a systematic examination will almost always give you the answer.

Differential Diagnosis of Red Eye

Diagnosis	Pain/Discomfort	Discharge	Vision	Urgency
Viral conjunctivitis	Gritty, watery	Watery	Normal	Routine
Bacterial conjunctivitis	Gritty	Mucopurulent, lids sticky	Normal	Routine
Allergic conjunctivitis	ITCH — the key symptom	Watery/mucoid	Normal	Routine
Blepharitis	Burning, grittiness — lid margins	Crusting on lids	Normal	Routine
Episcleritis	FB sensation, focal ache	None	Normal	Routine
Subconjunctival haemorrhage	None (alarming appearance)	None	Normal	Routine (check BP)
Anterior uveitis (iritis)	Deep ache, photophobia	None	Reduced	**Same-day referral**
Keratitis / Corneal ulcer	Severe pain, photophobia	Watery/purulent	Reduced	**URGENT REFERRAL**
Acute angle-closure glaucoma	Severe, nausea/vomiting, haloes	None	Severely reduced	**999 / EMERGENCY**

Systematic Examination of the Red Eye

Visual acuity (VA) — ALWAYS check VA first — reduced VA signals serious pathology

Inspection — Discharge, lid swelling, periorbital erythema, proptosis

Pattern of redness — Diffuse = conjunctivitis; Circumcorneal (ciliary flush) = iritis/keratitis; Localised = episcleritis

Corneal clarity — Haze or opacity = keratitis, glaucoma, or corneal oedema

Pupil — Irregular/small = iritis; fixed mid-dilated = acute glaucoma

Fluorescein staining — Epithelial defects stain bright green under cobalt blue light — essential for detecting abrasions, ulcers, and dendritic patterns (HSV)

Lid eversion — Evert the upper lid to look for foreign bodies, cobblestones (giant papillary conjunctivitis), or follicles

IOP (digital) — A rock-hard eye = acute glaucoma until proven otherwise

Management by Diagnosis

Bacterial Conjunctivitis

Most cases are self-limiting within 1–2 weeks. Topical antibiotics are not always necessary. If treatment is warranted (symptoms >5 days, persistent discharge, or patient preference):

- **First line:** Chloramphenicol 0.5% drops QDS for 5 days, or 1% ointment TDS (broad-spectrum; available OTC). Continue until 48 hours after resolution.
- **Alternative:** Fusidic acid 1% drops BD (note: limited Gram-negative cover — use only when chloramphenicol is contraindicated or not tolerated).

Source: NICE CKS Infective Conjunctivitis; BNF. Note: most mild acute bacterial conjunctivitis resolves without antibiotics.

Conjunctivitis: Key Distinctions

- Bacterial conjunctivitis — lids stuck together on waking, mucopurulent discharge, usually bilateral (one eye then the other).
- Viral conjunctivitis — watery discharge, often follows URTI, palpable pre-auricular node, highly contagious. No antibiotic treatment; reassure and use lubricants and cold compresses.
- Contact lens wearer with red eye — treat as keratitis until proven otherwise. You must refer same-day.

Allergic Conjunctivitis

ITCH is the cardinal symptom. Often associated with allergic rhinitis (rhinoconjunctivitis). Can be seasonal (pollens) or perennial (dust mite, animal dander).

- **Step 1:** Topical antihistamine (e.g. antazoline/xylometazoline) for short-term relief.
- **Step 2:** Mast cell stabiliser — sodium cromoglicate 2% QDS (works best as prevention; needs regular use).
- **Step 2 (alternative):** Olopatadine 0.1% BD — dual-action (antihistamine + mast cell stabiliser); preferred by many.

Do NOT use topical steroids in primary care for allergic eye disease — risk of glaucoma, cataract, and masking infection.

Blepharitis

A chronic, relapsing condition — think of it like eczema of the eyelid margins. It is incurable but controllable. Due to meibomian gland dysfunction or staphylococcal colonisation.

- Warm compresses to the lids for 5–10 minutes, twice daily.
- Lid margin hygiene: gently scrub the lash line with diluted baby shampoo or proprietary lid wipes.
- Preservative-free lubricants for comfort.

Anterior Uveitis (Iritis)

Deep aching pain, photophobia, circumcorneal redness, and reduced VA. The pupil may be small and irregular due to posterior synechiae (iris adhesions). Keratic precipitates (white cells on corneal endothelium) and hypopyon (pus level in anterior chamber) may be seen on slit lamp.

■ Anterior Uveitis — Refer Same Day

- ■ Anterior uveitis requires same-day ophthalmology referral.
- ■ Requires topical steroids and cycloplegics — never prescribe these without ophthalmology review.
- ■ Investigate for systemic cause if recurrent or bilateral: HLA-B27, sarcoidosis, IBD, ankylosing spondylitis.

Herpes Zoster Ophthalmicus — What You Must Know

✓ Herpes Zoster Ophthalmicus (shingles affecting CN V1): treat with oral aciclovir 800mg 5× daily for 7 days (within 72 hours of rash onset).

✓ If the EYE becomes red, refer urgently to ophthalmology — do not manage in the community.

✓ If the tip of the nose is involved (Hutchinson's sign), intraocular involvement is more likely.

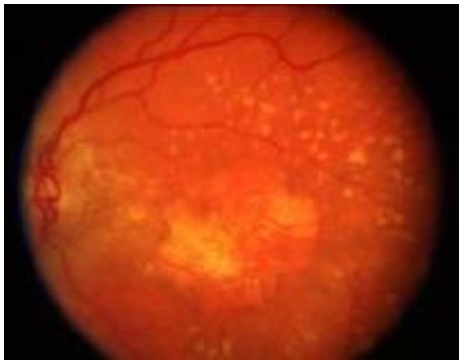
✓ If the tip of the nose is NOT involved, ocular involvement is less likely — but still examine the eye carefully.

3. Age-Related Macular Degeneration (AMD)

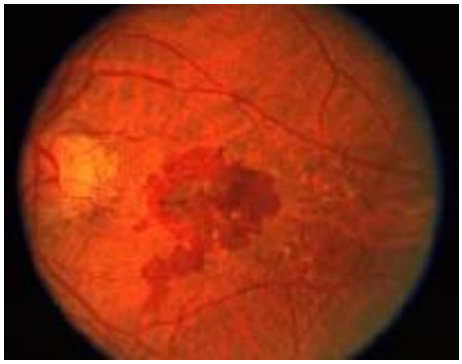
AMD is the leading cause of irreversible visual loss in the UK in people over 60, with approximately 40,000 new cases per year. It affects the macula — the central area of the retina responsible for reading, recognising faces, and fine detail. Think of the macula as the "HD zoom lens" of the eye. When it fails, central vision blurs or distorts, but peripheral vision is usually preserved.

Dry AMD vs Wet AMD

Feature	Dry AMD (Early/Non-exudative)	Wet AMD (Late/Neovascular)
Proportion	~90% of AMD	~10% of AMD
Pathology	Drusen (yellow-white deposits) and RPE atrophy	Choroidal neovascularisation (CNV) — new leaky blood vessels
Symptoms	Often asymptomatic early; mild blur or colour change	Rapid central distortion, straight lines appear wavy (metamorphopsia), scotoma
Visual loss	Gradual over years	Can be rapid — days to weeks
Treatment	No pharmacological treatment; lifestyle modification	Anti-VEGF injections — urgent referral needed (window of opportunity)
Prognosis	Slow, variable progression	80% develop fibrotic scar if untreated; 20% geographic atrophy



Dry AMD: drusen (yellow deposits) and geographic atrophy of the RPE. Note large choroidal vessels visible through the RPE defect.



Wet AMD: classic choroidal neovascular membrane (CNVM) causing subretinal haemorrhage at the fovea — a dense central scotoma results.

The Amsler Grid — Your Primary Care Tool

The Amsler grid is a simple self-test for macular function. A grid of horizontal and vertical lines is viewed at reading distance (30 cm) with reading glasses on, one eye at a time. Any distortion, waviness, or missing area in the central grid is highly suspicious for wet AMD.

Amsler Grid — Key Instructions

• Any new distortion of the Amsler grid in a patient with known drusen = urgent ophthalmology referral within days (not weeks). This is the window of opportunity for anti-VEGF treatment.

• Patients with dry AMD can be given an Amsler grid to monitor at home and instructed to contact the surgery immediately if distortion develops.

Anti-VEGF Treatment for Wet AMD

The breakthrough in wet AMD is anti-VEGF therapy: intravitreal injections that block vascular endothelial growth factor (VEGF), the molecule that drives the growth of leaky new blood vessels. Think of VEGF as the "build more pipes" signal — anti-VEGF blocks that signal before the pipes cause irreversible damage.

Drug	Trade Name	NICE Status
Ranibizumab	Lucentis	NICE approved (TA155)
Aflibercept	Eylea	NICE approved (TA294)
Brolucizumab	Beovu	NICE approved (TA672)
Faricimab	Vabysomo	NICE approved (TA897, 2023) — dual VEGF-A/Ang-2 inhibitor
Bevacizumab gamma	Lytenava	NICE final draft approved (2024) — first licensed bevacizumab for wet AMD

Source: NICE Technology Appraisals; Macular Society 2024. Avastin (bevacizumab) remains unlicensed for intravitreal use in the UK.

Prevention & Lifestyle Advice

AMD Prevention — Evidence-Based Advice

✓ Smoking cessation is the single most important modifiable risk factor for AMD — smokers have 2–4× the risk of AMD.

✓ Diet rich in leafy green vegetables (lutein and zeaxanthin) and oily fish is associated with reduced AMD progression.

✓ AREDS-formulation supplements (vitamins C, E, zinc, copper, lutein/zeaxanthin) are recommended for intermediate or advanced AMD in one eye (e.g. PreserVision).

✓ Statins and aspirin have no demonstrated effect on AMD progression.

4. Neuro-Ophthalmology: Acute Visual Loss & Diplopia

Acute visual loss is a medical emergency until proven otherwise. Speed of diagnosis and referral determines whether vision is saved. Your first question must always be: **monocular or binocular?** This single answer directs the entire diagnostic pathway.

Monocular loss	Binocular loss
Problem in the eye or optic nerve on that side	Problem behind the optic chiasm (brain)
CRAO, CRVO, retinal detachment, vitreous haemorrhage, optic neuritis, GCA	Stroke, TIA (amaurosis fugax from carotid), occipital lesion

Causes of Acute Visual Loss — Know These Cold

Central Retinal Artery Occlusion (CRAO)

Sudden, painless, profound monocular visual loss. "Cherry red spot" on fundoscopy. First branch of carotid. Treat as stroke — look for embolic source (carotid, cardiac).

Giant Cell Arteritis (GCA)

You must exclude GCA in any patient >50 years with sudden visual loss. Ask about headache, scalp tenderness, jaw claudication, shoulder girdle aching. ESR and CRP urgently. Start high-dose prednisolone IMMEDIATELY if suspected — do NOT wait for biopsy results. Delay = permanent blindness.

Retinal Detachment

New onset flashing lights and floaters = vitreoretinal traction. Curtain or shadow across vision = detachment. Same-day ophthalmology referral. Myopia increases risk tenfold.

Optic Neuritis

Painful eye movement, reduced colour perception (especially red), reduced VA over days. RAPD positive. Often the first presentation of MS (but may not be). Refer to ophthalmology. Investigate with MRI brain.

Vitreous Haemorrhage

Sudden shower of floaters, reduced vision. In a known diabetic = proliferative retinopathy until proven otherwise. Urgent ophthalmology referral.

Amaurosis Fugax

Transient monocular visual loss — like a curtain falling and lifting over one eye (minutes). This is a TIA equivalent affecting the retinal artery. Manage as TIA: antiplatelet therapy, urgent carotid Doppler, cardiology review.

Relative Afferent Pupillary Defect (RAPD)

An RAPD (Marcus Gunn pupil) tells you there is a defect **in front of** the optic chiasm — i.e. in the retina or optic nerve on that side. The swinging torch test: shine a bright light alternately between eyes. In a normal person, both pupils constrict equally. In an RAPD, when the light moves to the affected eye, both pupils DILATE (paradoxical dilatation) because the afferent signal from that eye is weaker.

Diplopia (Double Vision)

Nerve	Muscle affected	Diplopia direction	Key associations
3rd nerve palsy	All except SO and LR	Eye down and out; ptosis	Pupil-sparing = ischaemic (DM, HTN) — recovers 3 months. Pupil-involved = surgical emergency (PCA aneurysm)
4th nerve palsy	Superior oblique	Vertical diplopia worse on downgaze	Often post-traumatic
6th nerve palsy	Lateral rectus	Horizontal diplopia — cannot abduct	Raised ICP, DM, stroke

■ Diplopia Red Flags

■ 3rd nerve palsy with pupil involvement (dilated, unreactive pupil) = posterior communicating artery aneurysm until proven otherwise — this is a neurosurgical emergency. Call 999.

■ Horner's syndrome (ptosis + miosis + anhidrosis) = sympathetic chain disruption. Carotid dissection must be excluded urgently.

5. How to Examine the Eye

A systematic eye examination can be performed in primary care with basic equipment. You do not need a slit lamp to make most diagnoses — you need a systematic approach.

Equipment Checklist

- Snellen chart (6m or 3m equivalent)
- Pinhole occluder
- Bright pen torch (LED)
- Ophthalmoscope
- Fluorescein strips and cobalt blue light
- Topical anaesthetic (e.g. proxymetacaine 0.5% — onset 30s, lasts ~10–15 min)
- Tropicamide 1% for mydriasis (warn: cannot drive; very small risk of precipitating acute angle closure in predisposed patients)

Step-by-Step Examination

Step 1: Visual acuity — Use Snellen chart at correct distance. Test each eye separately (cover the other). Use pinhole if VA is reduced — improvement with pinhole suggests refractive error rather than pathology. Record as e.g. 6/12 pinhole 6/6.

Step 2: Visual fields — Confrontation testing. Use a red pin (not just a finger) — red desaturation is an early sensitive sign of optic nerve or chiasmal disease.

Step 3: Pupils — Check size, shape, direct and consensual reflex. Perform swinging torch test for RAPD. An irregular, small pupil = iritis. Fixed mid-dilated = acute glaucoma.

Step 4: Eye movements — Ask the patient to follow your finger in an H-pattern. Pain on movement = orbital cellulitis, scleritis, or optic neuritis.

Step 5: Anterior segment — Examine with a pen torch: lids, lashes, conjunctiva, cornea, anterior chamber depth, iris. Evert the upper lid. Apply fluorescein and use cobalt blue light.

Step 6: Fundoscopy — Start with the ophthalmoscope at +10 to check the red reflex (absence = cataract, vitreous haemorrhage, or other opacity). Move closer; examine disc (cup-to-disc ratio, rim, vessels), then follow arcades to the macula. Ask patient to look at the light to centre on the fovea.

Key History Questions

Question	Why it matters
One eye or both?	Monocular loss = ocular/optic nerve; binocular = post-chiasmal/brain
Onset: sudden or gradual?	Sudden = vascular (CRAO, vitreous haemorrhage, RD); gradual = cataract, glaucoma, AMD
Pain?	Aching = iritis, scleritis; sharp = corneal; none = vascular, retinal
Floaters/flashing lights?	Flashes = vitreoretinal traction; shower of floaters = vitreous haemorrhage or RD
Haloes around lights?	High IOP — acute angle-closure glaucoma until proven otherwise

Contact lenses?	Must treat as keratitis — same-day referral
Myopia?	10× increased risk of retinal detachment
Family history?	Glaucoma in first-degree relative = 1 in 10 lifetime risk; squint
Diabetes / hypertension?	Diabetic retinopathy, vascular occlusions, NAION

6. Diabetic Eye Disease

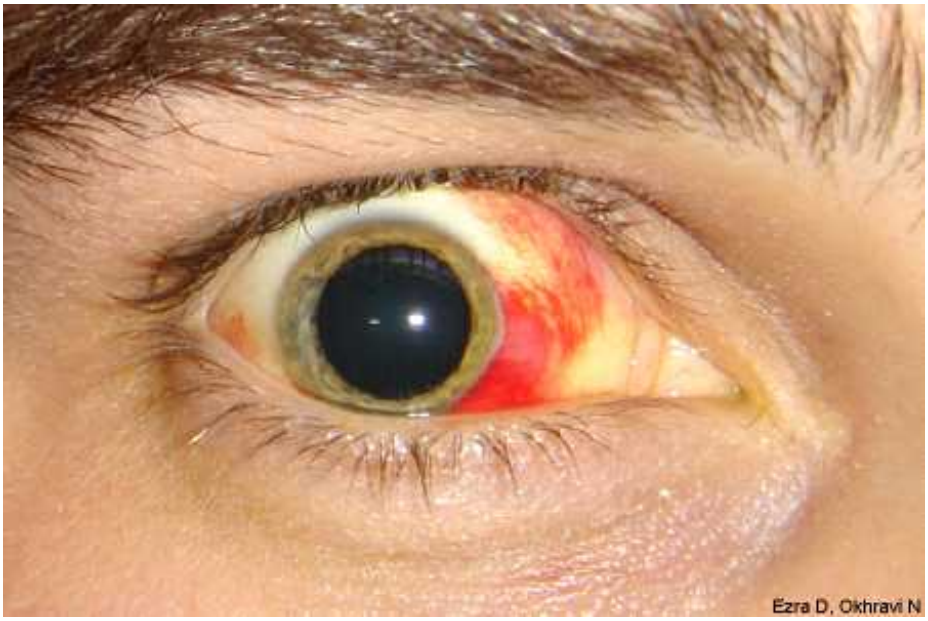
Diabetic retinopathy is the most common cause of preventable sight loss in working-age adults (20–55 years) in the UK. After 20 years of diabetes, over 90% of people with type 1 diabetes and over 60% with type 2 will have some degree of retinopathy.

Crucially, early diabetic retinopathy causes no symptoms. By the time the patient notices blurred vision, significant damage may already have occurred. This is why the NHS Diabetic Eye Screening Programme is vital.

NHS Diabetic Eye Screening Grading System

Grade	Name	Features	Action
R0	No DR	Normal retina	Routine annual screening
R1	Mild non-proliferative	Microaneurysms only	Routine annual screening
R1*	Moderate non-proliferative	Extensive microaneurysms, intraretinal haemorrhages, hard exudates	Refer to hospital eye service
R2	Severe non-proliferative (pre-proliferative)	Venous beading, venous loops, IRMA, multiple deep blot haemorrhages, cotton wool spots	Refer to hospital eye service
R3	Proliferative	New vessels at disc (NVD) or elsewhere (NVE), pre-retinal or vitreous haemorrhage, fibrovascular proliferation, tractional retinal detachment	**URGENT referral to hospital eye service**
M	Maculopathy	Exudates, thickening, microaneurysms, or haemorrhage within 1 disc diameter of the fovea centre; VA ≤6/12 associated	Refer to hospital eye service (urgent if VA affected)

Source: NHS Diabetic Eye Screening Programme; NICE NG242 (August 2024). R3 and ungradeable images require urgent review.



Fundus photograph: Diabetic macular oedema (DMO). Hard exudates, microaneurysms, and retinal thickening at the macula — the primary cause of visual loss in type 2 diabetes.

Treatment of Diabetic Eye Disease

Retinopathy: Panretinal photocoagulation (PRP / laser) is used for proliferative retinopathy. It halts progression in about 50% of cases. Anti-VEGF agents (intravitreal ranibizumab, aflibercept, or faricimab) are now standard of care for diabetic macular oedema (DMO). (Source: NICE NG242, August 2024)

Your Role in Diabetic Eye Disease

✓ Optimise HbA1c, blood pressure, and lipids — these are the most important things you can do in primary care to prevent and slow diabetic eye disease.

✓ ACE inhibitors have evidence for reducing the incidence of diabetic retinopathy beyond blood pressure control alone.

✓ Rapid improvement in glycaemic control can paradoxically worsen retinopathy in the short term — warn patients, and involve ophthalmology before aggressive glucose lowering in those with significant retinopathy (NICE NG242).

✓ Ensure all diabetic patients are enrolled in the NHS Diabetic Eye Screening Programme — this is YOUR responsibility as the GP.

Retinopathy vs Maculopathy — Know the Difference

• Peripheral retinopathy (R-grading) and maculopathy (M-grading) are independent conditions — peripheral disease does NOT progress to maculopathy.

• The majority of visual loss in type 2 diabetes is from maculopathy (central oedema), not from proliferative retinopathy.

• A patient can have severe maculopathy but minimal peripheral retinopathy — VA may be the only clue in primary care.

7. Cataract & Refractive Surgery

A cataract is any clouding of the lens. It is the most common cause of correctable visual loss worldwide. Think of it as the lens slowly becoming frosted glass — light still enters but is scattered rather than focused.

Symptoms of Cataract

- Gradual blurring of vision (most common)
- Glare or haloes around lights — particularly problematic with oncoming headlights at night
- Reduced colour perception (colours appear washed out or yellowed)
- Reduced contrast sensitivity and depth perception
- Monocular diplopia (double vision in one eye only)
- Frequent changes in glasses prescription

Cataract Surgery

Modern cataract surgery is performed using phacoemulsification — a small incision technique. The natural lens is emulsified ultrasonically, removed, and replaced with an artificial intraocular lens (IOL). This is the most commonly performed surgical procedure in the UK. Refer when the cataract is affecting the patient's daily activities — there is no "minimum VA" threshold for referral.

Refractive Surgery (LASIK/LASEK)

Laser refractive surgery reshapes the cornea using an excimer laser to correct myopia, hypermetropia, and astigmatism. Wavefront-guided technology maps the prescription at every point across the cornea for maximum precision.

Advantages	Risks & Complications
Freedom from glasses/contact lenses	Dry eyes (very common — warn patients)
Improved night vision possible	Halos, glare, and starbursts (especially at night)
Contact lens users >0.5 yrs have HIGHER infective risk than LASIK	Under- or over-correction
Correction of full refractive spectrum	Rare: keratectasia, uveitis, endophthalmitis, retinal detachment

Contact Lens Safety — Critical Points

• Contact lens wearers who develop a painful red eye must be referred same-day to ophthalmology — presume corneal ulcer until ruled out.

• Pseudomonas aeruginosa keratitis is a devastating but preventable complication of extended-wear soft contact lenses — it can destroy the cornea within 24 hours.

• Warn all contact lens users: never sleep in lenses, always clean with sterile solution, replace lens cases monthly.

8. Glaucoma

Glaucoma is **optic nerve damage with characteristic visual field loss**, usually (but not always) associated with raised intraocular pressure (IOP). It is often called "the silent thief of sight" — peripheral vision is lost so gradually that patients do not notice until a large proportion is gone.

2nd	50%	4×	5×
Leading cause of irreversible blindness worldwide	Of cases in the UK remain undiagnosed	Increased risk of falls in patients with glaucoma	Increased odds of a car crash (important for DVLA)

Risk Factors

Risk Factor	Detail
Age	Risk increases significantly over age 60
Race	Afro-Caribbean origin: 4–5× increased risk and younger onset; East Asian: higher risk of normal-tension and angle-closure glaucoma
Family history	First-degree relative with glaucoma = ~1 in 10 lifetime risk. Entitled to free NHS sight tests.
Raised IOP	IOP >21 mmHg is a major risk factor (though some glaucoma occurs at normal IOP)
Myopia	High myopia increases risk of primary open-angle glaucoma
Corticosteroid use	Topical, inhaled, or systemic steroids can raise IOP

Diagnosis

Glaucoma is diagnosed on three pillars: **optic disc changes** (vertical cup-to-disc ratio ≥0.6 is suspicious, especially with notching of the neuroretinal rim), **visual field loss** (characteristic patterns on Humphrey or Goldmann perimetry), and **raised or borderline IOP**. Visual fields are the most important measurement — they reflect what function remains, and what we are trying to preserve.

Why Patients Miss Their Own Glaucoma

• The cortex "fills in" blind spots — patients with significant glaucomatous field loss may not notice it until it is severe. Do not be reassured by "I haven't noticed anything wrong with my vision".

• When assessing field loss, compare BOTH eyes — a large scotoma in one eye may be compensated by the other. Bilateral inferior field loss is particularly significant for mobility and driving.

Treatment

Lowering IOP is the only intervention with robust evidence for preventing progression — even in patients with normal-tension glaucoma. The goal is to maximise quality of vision over the patient's remaining lifetime.

Treatment	Class	Notes

Prostaglandin analogues (latanoprost, bimatoprost, travoprost)	1st line medical treatment	Most effective IOP reduction. Once daily (usually evening). Side effects: iris colour change, eyelash growth, periorbital fat atrophy. NICE NG81; also latanoprost–netarsudil now NICE approved (TA1009, Oct 2024) when prostaglandin analogue alone is insufficient.
Beta-blockers (timolol, betaxolol)	2nd line / adjunct	Contraindicated in asthma, COPD, bradycardia. Betaxolol is cardioselective — safer in respiratory disease but less effective.
Carbonic anhydrase inhibitors (dorzolamide, brinzolamide)	Adjunct (topical)	Avoid in sulphonamide allergy. Available in fixed combination with timolol.
Alpha-2 agonists (brimonidine)	Adjunct	Avoid in patients on MAOIs. Risk of drowsiness.
Selective laser trabeculoplasty (SLT)	1st line option (NICE NG81)	NICE NG81 now recognises SLT as a first-line option — discuss with patient. Can delay or reduce need for drops.
Surgical (trabeculectomy, tubes)	When drops/laser insufficient	For progressive disease despite maximum medical treatment.

Sources: NICE NG81 (Glaucoma: diagnosis and management); NICE TA1009 (Latanoprost–netarsudil, October 2024). Prostaglandin analogues are first-line; choose lowest acquisition cost when no clinical factors favour one over another.

Functional Impact — Ask Your Patients These

Patients may not volunteer functional difficulties. Ask directly:

- Do you trip over things or bump into door frames?
- Do you have difficulty on stairs?
- Do you have difficulty following a line of print?
- Do you have difficulty finding things on a supermarket shelf?
- Have you had any near-misses while driving?

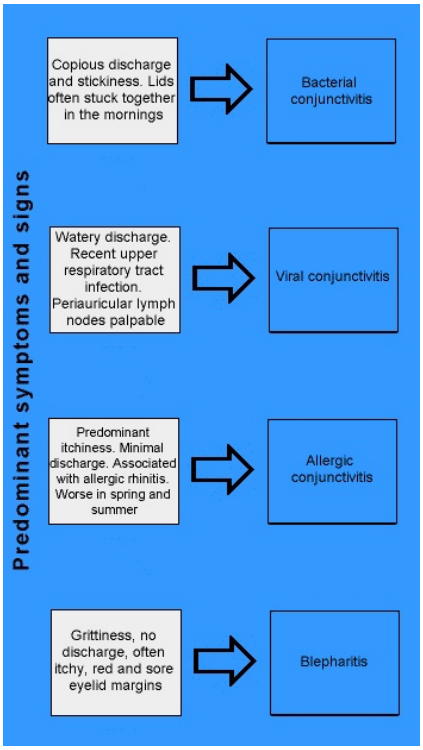
■ Glaucoma Emergencies & Legal Duties

■ Acute angle-closure glaucoma: severe eye pain, headache, nausea/vomiting, halos around lights, rock-hard eye, fixed mid-dilated pupil. Call 999 / emergency ophthalmology immediately.

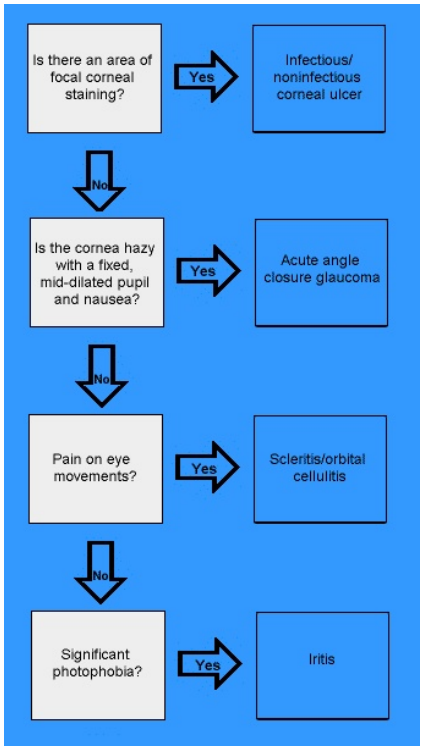
■ Any patient with acutely raised IOP must NOT be given mydriatics — this will precipitate or worsen angle closure.

■ DVLA notification: patients with significant bilateral field loss may be unfit to drive — discuss and document.

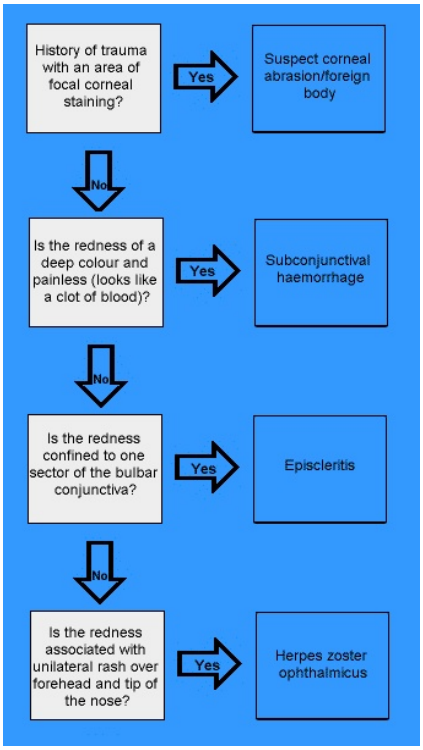
Visual Representations of Eye Conditions



Diabetic Retinopathy Grading Chart (R-grades)



Maculopathy Reference Guide (M-grades)



Combined Grading Reference

9. MCQ Practice Cases

Test your knowledge with these clinical scenarios. Think about the diagnosis before reading the answer.

Case 1

A 17-year-old presents with bilateral discomfort developing over six months. He describes itchiness, sneezing, and a runny nose. His vision is normal when he wipes his eyes. The conjunctivae are injected.

What is the most likely diagnosis?

a) Bacterial conjunctivitis

✓ **b) Allergic conjunctivitis (rhinoconjunctivitis)**

c) Viral conjunctivitis

d) Blepharitis

Explanation:

The key symptom is ITCH, combined with nasal symptoms (rhinoconjunctivitis). Allergic conjunctivitis is often seasonal (pollens) or perennial (dust mite, animal dander). VA is normal after wiping away excess tearing — reduced VA would suggest a more serious diagnosis. Treatment: topical antihistamine or mast cell stabiliser (olopatadine preferred for combined action). Do NOT use topical steroids.

Case 2

A 28-year-old man presents with a three-day history of a painless red right eye. Onset was sudden. VA is 6/9 bilaterally. No corneal staining. Fundoscopy is normal. There is a solid red area with a sharply defined posterior edge.

What is the most likely diagnosis?

a) Corneal foreign body

b) Central retinal artery occlusion

✓ **c) Subconjunctival haemorrhage**

d) Episcleritis

Explanation:

Subconjunctival haemorrhage produces a solid red haematoma with a well-defined posterior border. It is painless and vision is unaffected. Causes include coughing, straining, hypertension, and anticoagulants. No treatment is required — it resolves in 2–3 weeks. CHECK BLOOD PRESSURE. Refer only if associated with head injury (possible base of skull fracture if the posterior limit of the haematoma cannot be seen laterally).

Case 3

A 42-year-old nurse wearing extended-wear soft contact lenses presents with one day of pain and blurred vision in the right eye. On examination, there is focal fluorescein staining with an area of corneal opacity.

What is the single most suggestive feature of a corneal ulcer?

a) Fixed mid-dilated pupil

✓ **b) Focal fluorescein staining with corneal opacification**

c) Normal visual acuity

d) Palpebral conjunctival injection

Explanation:

A focal area of fluorescein staining combined with corneal opacity indicates epithelial disruption plus stromal involvement — this is a corneal ulcer until proven otherwise. CONTACT LENS + RED PAINFUL EYE = same-day urgent ophthalmology referral. This could be Pseudomonas keratitis — a sight-threatening emergency. A fixed mid-dilated pupil = acute angle-closure glaucoma. Palpebral injection alone = conjunctivitis.

Case 4

A 28-year-old woman with a three-day history of a red eye. She has intense photophobia, deep aching eye pain, and reduced VA. The pupil appears small and slightly irregular.

Which feature most strongly suggests uveitis?

✓ **a) Intense photophobia and deep pain with reduced VA**

b) Purulent discharge with lids stuck in the morning

c) Marked bilateral itchiness

d) Gritty discomfort at the lid margins

Explanation:

Deep aching pain and intense photophobia with reduced VA strongly suggest anterior uveitis (iritis). The small, irregular pupil is caused by posterior synechiae (adhesions between iris and lens). This patient needs SAME-DAY referral to ophthalmology. Purulent discharge with sticky lids = bacterial conjunctivitis. Bilateral itch = allergic. Lid margin grittiness = blepharitis.

Case 5

A 34-year-old primary school teacher presents with four-day history of bilateral red eyes following a URTI. There is watery discharge and mild crusting in the morning. VA is 6/6 bilaterally. Palpable pre-auricular lymph nodes. The conjunctivae are diffusely inflamed.

What is the most appropriate management?

a) Chloramphenicol drops QDS for 5 days

✓ b) Supportive care with lubricants and cold compresses; safety-net

c) Olopatadine drops BD for 2 weeks

d) Urgent ophthalmology referral

Explanation:

This is viral conjunctivitis: bilateral, post-URTI, watery discharge, palpable pre-auricular lymphadenopathy, and lids NOT firmly stuck. There is no specific treatment — antibiotics are not indicated. Management: preservative-free lubricants, cold compresses, and patient education about contagiousness (stay off work if possible while actively tearing; avoid sharing towels). Most episodes resolve within 1–2 weeks. Safety-net: if corneal involvement develops (reduced VA, staining), refer urgently — adenoviral keratoconjunctivitis can be severe.

Quick Reference: When to Refer

Condition	Urgency	Key Feature
Acute angle-closure glaucoma	**999 / EMERGENCY**	Severe pain, nausea, haloes, rock-hard eye
3rd nerve palsy (pupil involved)	**999 / EMERGENCY**	Pupil-involving 3rd nerve palsy = PCA aneurysm until proven otherwise
GCA with visual symptoms	**SAME DAY + immediate steroids**	Do NOT wait for ESR/CRP or biopsy before starting prednisolone
Orbital cellulitis	**SAME DAY**	Proptosis, pain on eye movement, unwell
Corneal ulcer / keratitis	**Same day**	Especially in contact lens wearers
Retinal detachment (suspected)	**Same day**	Curtain/shadow, new floaters + flashes, myopia
Anterior uveitis	**Same day**	Deep pain, photophobia, circumcorneal redness, reduced VA
Wet AMD (new distortion)	**Within days (urgent)**	Window of opportunity for anti-VEGF; do not delay
Proliferative DR (R3)	**Within 2 weeks**	New vessels on disc or elsewhere; vitreous haemorrhage
HZO with eye involvement	**Urgent**	Red eye in shingles of V1 distribution
Subconjunctival haem + head injury	**A&E;**	Cannot see posterior limit laterally = possible base of skull fracture
Cataract (symptomatic)	**Routine**	Affecting daily activities — no minimum VA for referral