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| **Q: 78** | A 67yo man has deteriorating vision in his left eye. He has longstanding COPD and is on multiple drug therapy. What single medication is likely to cause this visual deterioration?  a. B2 agonist  b. Corticosteroid  c. Diuretic  d. Theophylline |
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| **Clincher(s)** | **COPD, multiple drug therapy, deteriorating vision in left eye** |
| A | S/E 🡪 trembling (hands), nervous tension, headaches, palpitations, muscle cramps |
| B | S/E 🡪 cataracts and glaucoma |
| C | Can cause blurred vision (furosemide) |
| D | S/E 🡪 palpitations, headaches, diarrhea, vomit/nausea, arrhythmia, tachycardia |
| E |  |
| **KEY** | **B** |
| Additional Information | Management of COPD (partially) |
| **Reference** | <http://patient.info/medicine/furosemide-frusol-lasix>  corticosteroids nhs side effects |
| Dr Khalid/Rabia | Q. 1. What is the key?  Q. 2. What is the cause of deteriorating vision?  Ans. 1. The key is B. Corticosteroid.  Ans. 2. Prolonged corticosteroids [also topical i.e. eye drop] can cause cataract. |

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| **Q: 99** | A 30yo woman had a gradual decrease of visual acuity since the last 3 years. Now she has a disability due to very low vision. What’s the dx?  a. Glaucoma  b. Cataract  c. Macular degeneration  d. Retinitis pigmentosa  e. Keratitis |
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| **Clincher(s)** | **30yr old woman, gradual decrease visual acuity** |
| A | Usually occurs after the age of 50 and its not gradual loss of vision |
| B | Cataract 🡪 Nuclear (central) type 🡨 Usually old age |
| C | Central vision (focus) lost (not blindness)🡪 cant identify people and read |
| D | Peripheral retina affected 🡪 tunnel vision. Gradual loss of vision. |
| E | Vision not effected but pain, redness and photophobia |
| **KEY** | **D** |
| Additional Information | Usher's syndrome is inherited in an autosomal recessive manner, and is the association of:   * sensorineural deafness * retinitis pigmentosa   It is the most frequent cause of deaf-blindness.  Lowe's syndrome is an X linked condition in which there is a Fanconi syndrome with decreased urinary ammonia, organic aciduria, and sometimes a heavy proteinuria.  The Kearns-Sayer syndrome is caused by a somatic insertion or deletion mutation in the mitochodrial genome.  The severity of the syndrome is very variable, depending on the proportion of mitochondria containing mutated DNA. Invariably the disease becomes more severe with age.  KSS may be considered as a severe form of chronic external ophthalmoplegia. |
| **Reference** |  |
| Dr Khalid/Rabia | Retinitis pigmentosa primarily affects the peripheral retina resulting in funnel vision  Features   * night blindness is often the initial sign * funnel vision (the preferred term for tunnel vision) * fundoscopy: black bone spicule-shaped pigmentation in the peripheral retina, mottling of the retinal pigment epithelium   Associated diseases   * Refsum disease: cerebellar ataxia, peripheral neuropathy, deafness, ichthyosis * Usher syndrome * abetalipoproteinemia * Lawrence-Moon-Biedl syndrome * Kearns-Sayre syndrome * Alport's syndrome |

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| **Q: 121** | An 8yo child who is tall for his age and has a refractory error for which he wears glasses has presented with severe crushing chest pain. What is the most likely dx?  a. Fragile X syndrome  b. Prader-willi syndrome  c. DiGeorge syndrome  d. Marfans syndrome |
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| **Clincher(s)** |  |
| A | Mental retardation |
| B | Short stature and small hands and feet |
| C |  |
| D | Tall for his age and chest pain likely due to aortic dissection |
| E |  |
| **KEY** | **D** |
| Additional Information | The fragile X syndrome is said to be the most common heritable cause of mental retardation after Down's syndrome. It is caused by a dominant X-linked gene with a penetrance of only 50% in females.  The gene which is most commonly responsible is FMR-1 (familial mental retardation 1). The disease occurs when the expression of FMR-1 is disrupted by:   * a large number, more than 230, of trinucleotide repeats * a deletion (rare)   It seems that loss of fmr-1 results in the fragile X syndrome.  Pre-pubertal features:   * normal growth but large head - greater than 50th percentile * delayed attainment of developmental milestones * tantrums, hyperactivity and autism   Post-pubertal   * low IQ (20-70) * long face, prominent forehead, large ears, large jaw * macroorchidism   Other features   * ophthalmologic - strabismus * orthopaedic - pes planus & joint hyperextension * dermatologic - soft, smooth skin * cardiac - mitral valve prolapse   Fragile X should be considered for all children with developmental delay of unknown cause.  Prader-Willi syndrome is a disorder which affects 1 in 15000 births and is characterised by:   * neonatal hypotonia * behavioural disturbance, particularly hyperphagia * mild-to-moderate mental retardation   60% of cases have a small deletion of the paternal chromosome 15q11-13. The majority of the remainder have two apparently normal apparently normal (but maternally inherited) chromosome 15s (See genetics section for more details).  Infants are hypotonic and have swallowing difficulties.  During childhood the following features may be noted:   * cryptorchidism * genital hypoplasia * dolichocephaly * almond-shaped eyes * narrow face * small mouth with down-turned corners   In later life there is:   * short stature * small hands and feet * severe obesity - evident from 9 to 15 months, with severe hyperphagia * mental retardation - the IQ is usually in the range 50 to 70 * dysregulated temperature control resulting in hypothermia * non-insulin-dependent diabetes * sleep apnoea and cor pulmonale   The DiGeorge syndrome is an example of a selective T-cell deficiency caused by the failure of development of the third and fourth pharyngeal pouches.  These pouches give rise to the following structures:   * thymus * parathyroids * aortic arch * portions of the lips and ears   Consequently, DiGeorge syndrome may present with as immune deficiency state - usually T cells, but sometimes B cells, and also aberrant calcium metabolism, congential heart disease and abnormal facies.  Clinical features relate closely to the structural defects present in the DiGeorge syndrome:  Thymic hypoplasia:   * low T cell counts * susceptibility to mycobacteria, viruses and fungi, for example chronic mucocutaneous candidiasis * compensatory B cell leukocytosis * peripheral lymphocytes do not respond to polyclonal T cell activators * organ specific autoimmune disorders   Absent parathyroid glands:   * low PTH levels * low plasma calcium * muscle twitching or tetany * basal ganglia calcification and intellectual retardation * hypocalcaemic cataract   Congental malformations of the great vessels  Facial abnormalities  Thymic hypoplasia with retention of parathyroid function is termed Nezelof syndrome. |
| **Reference** |  |
| Dr Khalid/Rabia | Ans. 1. The key is D. Marfans syndrome.  Ans. 2. Cause of severe crushing chest pain may be aortic dissection.  Ans. 3. Most common cardiac abnormalities in Marfans syndrome are: dilatation of the aorta and mitral regurgitation.  Marfans syndrome diagnosis:  Major criteria (diagnostic if >2): Lens dislocation (ectopia lentis) aortic dissection or  dilatation; dural ectasia; skeletal features: arachnodactyly (long spidery fingers), armspan > height, pectus deformity, scoliosis, pes planus. Minor signs: Mitral valve prolapse, high-arched palate, joint hypermobility. Diagnosis is clinical.  **DANGER IS AORTIC DISSECTION**. Surgery is done when aorta >5cm  Can also cause pneumothorax. |

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| **Q: 167** | A 45yo T1DM had an annual check up. Ophthalmoscopy showed dot and blot hemorrhage + hard exudate and multiple cotton wool spots. What is the next step in management?  a. Reassurance and annual screening only  b. Urgent referral to ophthalmologist  c. Laser therapy  d. Non-urgent referral to ophthalmologist  e. Nothing can be done |
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| **Clincher(s)** | **Dot and blot haemorrhage , multiple cotton wool spots** |
| A |  |
| B | Proliferative with neovascularization) |
| C |  |
| D | Pre-proliferative retinopathy so non-urgent referral. |
| E |  |
| **KEY** | **D** |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia | **Diabetic retinopathy** Blindness is preventable. Annual retinal screening mandatory  for all patients not already under ophthalmology care. Pre-symptomatic screening enables laser photocoagulation to be used, aimed to stop production of angiogenic factors from the ischaemic retina. Indications: maculopathy or proliferative retinopathy.  • **Background retinopathy:** Microaneurysms (dots), haemorrhages (blots) and  hard exudates (lipid deposits). Refer if near the macula, eg for intravitreal triamcinolone.  • **Pre-proliferative retinopathy**: Cotton-wool spots (eg infarcts), haemorrhages,  venous beading. These are signs of retinal ischaemia. Non urgent Refer to a specialist.  • **Proliferative retinopathy**: New vessels form. **Needs urgent referral**.  • **Maculopathy**: (hard to see in early stages). Suspect if acuity. Prompt laser, intra vitreal steroids or anti-angiogenic agents may be needed in macular oedema. |

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| **Q: 335** | A 48yo woman who has been taking medications for asthma for a long time has now presented with decreasing vision. What is the most probable cause for her decrease in vision?  a. Inhaled salbutamol  b. Inhaled steroids  c. Aminophylline  d. Beta-blockers  e. Oral steroids |
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| **Clincher(s)** | **Asthma medication, decreased vision.** |
| A |  |
| B |  |
| C |  |
| D |  |
| E | Correct answer |
| **KEY** | **E** |
| Additional Information | Prolonged steroid use 🡪 Cataract formation |
| **Reference** |  |
| Dr Khalid/Rabia |  |
| **Q:938** | A 33yo female complains of diplopia on upright gaze. Exam: ptosis can be seen.  There are no other complains or any significant PMH. What is the most appropriate inv  for him?  a. Ophthalmoscopy  b. Visual field test  c. TFT  d. CT  e. Checking red reflex | |
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| **Clincher(s)** | **Ptosis +** | |
| A |  | |
| B |  | |
| C |  | |
| D | ptosis can be due to neurological causes of muscle weakness in this case , but there is  associated diplopia so it’s better to exclude any nerve lesion through ct.  3rd nerve palsy  Remember pneumonic S04LR6(superior oblique by 4th nerve and lateral reclusive by 6th nerve rest is all supplied by 3rd nerve ) | |
| E |  | |
| **KEY** | **D** | |
| Additional Information |  | |
| **Reference** |  | |
| Dr Khalid/Rabia | Rabia | |

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| **Q:997** | A 55yo man who is hypertensive suddenly lost his vision. The retina  is pale and fovea appears as a bright cherry red spot. What is the single most  appropriate tx?  a. Pan retinal photocoagulation  b. Corticosteroids  c. Scleral buckling  d. Surgical extraction of lens  e. Pressure over eyeball |
|  |  |
| **Clincher(s)** |  |
| A |  |
| B |  |
| C |  |
| D |  |
| E | Causes of cherry red spot at macula:  Metabolic Storage Diseases:  Hurler's disease  Tay-Sachs disease  MPS VII (Sly syndrome)  Farber's disease  GM1 gangliosidoses  Niemann Pick's disease  Sandhoff disease  Shprintzen-Goldberg syndrome  Lysosomal Storage Diseases  Congenital Developmental Diseases : Leber's Congenital Amaurosis  Hereditary/ Familial:  Hallervorden Spatz disease  Krabbe disease  Degenerative: Metachromatic leukodystrophy  Vascular: Central retinal artery occlusion  Drugs:  Quinine toxicity  Dapsone toxicity  Poisoning:  Carbon monoxide  Methanol  CENTRAL RETINAL ARTERY OCCLUSION (Severe visual loss to finger counting or hand movement)  - Usually elderly patient -Sudden onset of visual loss  -Associated with hypertension and diabetes On examination: plus or minus carotid bruit  Fundoscopy: Optic disc is pale due to ischaemia. Cherry red spots on macula. Sometimes cholesterol is visible in the retinal arteries  Causes: Arteriosclerosis, Emboli Investigations:  -Slit lamp examination -Fundoscopy (Pale Optic Disc) -Carotid Doppler- If carotid bruit |
| **KEY** | **E** |
| Additional Information | **Reason: Presentaion in Central Retinal Artery occlusion is painless loss of**  **vision unilaterally over a few seconds.Can be a HX of Amaurosis Fugax.**  **Exam:afferent pupillary defect, a pale retina with attenuation of the vessels.**  **segmentation of the blood column in the arteries ('cattle-trucking') and the**  **centre of the macula (supplied by the intact underlying choroid) stands out**  **as a cherry-red spot**    **Management: Presentation within 90-100 min Occular massage can be tried.**  **Paracentesis and acetazolamide to reduce intraocular pressure**  **Sublingual isosorbide dinitrate.**  **Oral pentoxyphylline** |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q:1003** | A 30yo woman presents with acute headache. She complains of seeing  halos especially at night. What is the single most likely defect?  a. Paracentral scotoma  b. Monocular field loss  c. Tunnel vision  d. Central scotoma  e. Cortical blindness |
|  |  |
| **Clincher(s)** | **Halos at night** |
| A |  |
| B |  |
| C |  |
| D |  |
| E |  |
| **KEY** | **C** |
| Additional Information | **This is glaucoma**  **paracentral scotoma first, f/b a SEIDEL'S scotoma, f/b an arcuate and a double arcuate scotoma and finally**  **a tunnel vision leading to blindness.**  **HALOES:**  **These are caused by light, passing through water in or on the surface of the eye, being broken down into its spectral colours. This results in rainbow-like coloured rings around lights or bright objects. Acute angle-closure glaucoma is the most common and the most clinically significant cause. It is a sight-threatening condition. However, there are a number of other causes:**  **Excessive formation of tears.**  **Oedema of the corneal epithelium from any cause (eg, contact lens overwear).**  **Corneal dystrophies in their later stages.**  **Chronic open-angle glaucoma.**  **Early cataracts (glare of headlights making nighttime driving impossible).**  **Pigment dispersion syndrome.**  **Vitreous opacities.**  **Drugs (eg, digitalis and chloroquine).** |
| **Reference** | NHS |
| Dr Khalid/Rabia |  |

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| **Q:1010** | A 50yo man complains of visual prbs and dull pain in the left eye.  Fundoscopy reveals papilloedema. He was dx with MS 2yrs ago. There is no  consensual light reflex of the right eye. What is the single most likely  defect?  a. Paracentral scotoma  b. Mono-ocular field loss  c. Homonymous upper quadrantanopia  d. Central scotoma  e. Homonymous lower quadrantanopia |
|  |  |
| **Clincher(s)** | **MS with visual problems** |
| A | Same as D for explanation see below |
| B | Reason : As the Pt wa diagnosed with M.S, M.S mostly affects vision  unilaterally, with optic neuritis,papilloedema ,painful eye , decreased vision,  blindness or hemianopia.thus loss of consensual light reflex in the opposite  eye.  This is also called amaurosis fugax  Ocular causes include:  Iritis  Keratitis  Blepharitis  Optic disc drusen  Posterior vitreous detachment  Closed-angle glaucoma  Transient elevation of intraocular pressure  Intraocular hemorrhage  Coloboma  Myopia  Orbital hemangioma  Orbital osteoma  Keratoconjunctivitis sicca  Neurologic origin Edit  Neurological causes include:  Optic neuritis  Compressive optic neuropathies  And pappiloedema |
| C | upper homonymous quadrantanopia describes the loss of the same upper quadrant from each visual field.  Upper homonymous quadrantanopias are usually caused by damage to the optic radiation as it passes throught the temporal lobes.  For example a lesion in the left temporal lobe will cause a right homonymous upper quadrantanopia, that is the loss of the upper nasal quadrant from the left eye and the upper temporal quadrant from the right eye.  Left superior quadrantanopia can result from damage to the right hemisphere’s lateral geniculate nucleus (LGN), which carries visual information to the striate cortex. Right superior quadrantanopia is opposite to left superior quadrantanopia (i.e., visual field loss is in the upper temporal field of the right eye and the upper nasal field of the left eye, with damage occurring at the LGN). Damage to the LGN can be caused by a partial lesion, which could be due to ischemia (a deficiency in blood supply) of the LGN .Lesions to the optic radiation of the temporal lobe in a region called Myer’s loop (the anterior region of the optic radiation) may also be involved in causing homonymous superior quadrantanopia. Myer’s loop travels from the LGN to the striate cortex carrying visual information. Lesions in the right temporal lobe that affect Myer’s loop will cause visual field loss in the upper temporal quadrant of the left eye and upper nasal quadrant of the right eye, and vice versa when the lesion is located on the left temporal lobe. |
| D |  |
| E | A lower homonymous quadrantanopia describes the loss of the same upper quadrant from each visual field.  Lower homonymous quadrantanopias are usually caused by damage to the optic radiation as it passes throught the parietal lobes.  For example a lesion in the left parietal lobe will cause a right homonymous lower quadrantanopia, that is the loss of the lower nasal quadrant from the left eye and the lower temporal quadrant from the right eye.  Homonymous inferior quadrantanopia may originate from tumors, vascular lesions, or infection |
| **KEY** | **B** |
| Additional Information | **MS causes some loss of vision in the affected eye – this can range from mild to severe (total loss of vision occurs in one in 35 cases)**  **colour blindness ,eye pain; usually made worse when moving the eyeflashes of light when moving the eye**  **These symptoms are the result of optic neuritis, which is inflammation (swelling) of the optic nerve that transmits visual information to the brain. This normally only affects one eye.**  **Other visual problems that can occur in MS include:**  **double vision**  **eye pain in both eyes**  **involuntary eye movements (usually from side to side), known as nystagmus** |
| **Reference** | GP note book ,NHS |
| Dr Khalid/Rabia | Rabia |

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| **Q:1022** | A 39yo male presents with visual symptoms. Ophthalmoscopy shows  papilloedema. Which anatomical site is most likely to be affected?  a. Optic nerve  b. Optic disc  c. Optic radiation  d. Occulomotor nerve  e. Optic chiasma |
|  |  |
| **Clincher(s)** |  |
| A |  |
| B |  |
| C |  |
| D |  |
| E |  |
| **KEY** | **B** |
| Additional Information | **Optic disc swelling can be caused by a number of conditions; papilloedema**  **relates more specifically to optic disc swelling secondary to raised**  **intracranial pressure.**    **Disc swelling is distinct from disc atrophy which refers to a loss of nerve**  **fibres at the optic nerve head and which results in a pale disc. Atrophy may**  **be primary (where it occurs without prior disc swelling) or secondary (where**  **it is preceded by disc swelling)** |
| **Reference** |  |
| Dr Khalid/Rabia | Rabia |

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| **Q:1038** | A pt with flame shaped hemorrhage on long term tx with nifedipine. What is the  single most likely dx?  a. Macular degeneration  b. HTN retinopathy  c. MS  d. DM background  e. Proliferative DM retinopathy  f. SLE |
|  |  |
| **Clincher(s)** | **Treatment on nifedipine and flame shaped haemorrhages** |
| A | DRY AGE-RLATED MACULAR DEGENERATION  Risk factors: Increasing age, smoking, alcohol and female sex.  Symptoms:  -Gradual onset of decreased vision, initially to read and recognise faces due to loss of central field of vision. -Central Scotoma (walking into desks)  -It is due to photoreceptors  -Patient sees wavy lines -  -Macula has pigmentation geographically  -Bumps into objects -30% inheritance  Investigation: Fundoscopy or slit lump examination you see a large area of geographical atrophy at the macula and pigmentation.  Treatment is nothing just to reduce age related risk factors |
| B | On fundoscopy grade 3 shows flame shaped haemorrhages |
| C | They causes symptoms of optic neuritis like blurred vision,fraying of vision,pain and vision loss gets worse before it gets better for 4-12 weeks  Double vision, uncontrolled eye moments occur  Treated with steroids |
| D and e explanation | Retinopathy Pathogenesis: microangiopathy in capillaries, precapillary arterioles and venules causes occlusion ± leakage. Vascular occlusion causes ischaemia ± new vessels in the retina, optic disc, and iris, ie proliferative retinopathy. New vessels can bleed (vitreous haemorrhage). Retraction of f i brous tissue running with new vessels heightens risk of retinal detachment. Occlusion also causes cotton wool spots (ischaemic nerve fi bres). Vascular leakage: As pericytes are lost, capillaries bulge (microaneurysms) and there is oedema & hard exudates (lipoprotein & lipid fi lled macrophages). Rupture of microaneurysms at the nerve fi bre level causes fl ame shaped haemorrhages; when deep in the retina, blot haemorrhages form. Presymptomatic screening enables timely laser photocoagulation. Screen by regular eye exam or retinal photography. Screen all diabetic patients annually in the community, by dilated fundus photography which is then reviewed by a trained screening service. Referrals are then made accordingly. Lesions are mostly at the posterior pole and can be easily seen by ophthalmoscope. Non-proliferative diabetic retinopathy (NPDR) is rated as mild, moderate or severe depending on the degree of ischaemia. Signs comprise microaneurysms (seen as ‘dots’), haemorrhages (fl ame shaped or ‘blots’) hard exudates (yellow patches), engorged tortuous veins, cotton wool spots, large blot haemorrhages (the latter 3 are signs of signifi cant ischaemia). NPDR can progress to sight-threatening proliferative retinopathy. Proliferative diabetic retinopathy (PDR): Fine new vessels appear on the optic disc, retina and can cause vitreous haemorrhage. Maculopathy: Leakage from the vessels close to the macula cause oedema and can signifi cantly threaten vision (clinically signifi cant macular oedema). It can exist with otherwise mild retinopathy. Refer those with maculopathy, severe NPDR, or proliferative retinopathy urgently for assessment2 and treatment (eg photocoagulation) to protect vision. Treatment Good control of diabetes prevents new vessels forming. ‘Metabolic memory’ eff ects mean that early good control of diabetes with insulin pays dividends later. 124 Pregnancy, dyslipidaemia, BP, renal disease, smoking, and anaemia may accelerate retinopathy. Photocoagulation by laser is used to treat both maculopathy (focal or grid) and proliferative retinopathy (panretinal). Intravitreal triamcinolone and anti-VEGF drugs (p439) are used with laser to treat diabetic macular oedema. See fi gs 1 & 2. If vitreous haemorrhage is massive and does not clear, vitrectomy may be needed. |
| f | SLE causes mainly inflammation conjunctivitis and episcleritis |
| **KEY** | **B** |
| Additional Information | pt is on Ca channel blocker indicating he is hypertensive.  Fundoscopic findings in hypertensive retinopathy:  Grade 1: tortuous arteries with thick shiny walls, silver copper wiring  Grade 2: AV nipping  Grade 3: flame hemorrhages, dot and blot, hard soft exudates  Grade 4: papilledema  Rx. Control Bp |
| **Reference** |  |
| Dr Khalid/Rabia | OHCM |

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| **Q:** |  |
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| **Clincher(s)** |  |
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| B |  |
| C |  |
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| **KEY** |  |
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| **Reference** |  |
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| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q:635** | A 40yo man has pain, redness and swelling over the nasal end of his right lower eyelid. The eye is watery with some purulent discharge. The redness extends on to the nasal peri-orbital area and mucoid discharge can be expressed from the lacrimal punctum. What is the single most appropriate clinical dx?  a. Acute conjunctivitis  b. Acute dacrocystitis  c. Acut iritis  d. Retrobulbar neuritis  e. Scleritis |
|  |  |
| **Clincher(s)** |  |
| A | Acute Conjuctivitis |
| B | Acute Dacryocystitis |
| C | Acute Iritis |
| D | Retrobulbar Neuritis |
| E | Scleritis |
| **KEY** |  |
| Additional Information | **Ohcs- 435** Acute dacryocystitis (is acute infl ammation of the tear sac which is located medial to the medial canthus. This may spread to surrounding tissues (cellulitis) and result in systemic upset. Immediate antibiotic therapy may resolve the infection. Failure leads to local abscess formation  Chronic dacryocystitis This typically occurs in the middle-aged and elderly.The lacrimal sac distends, discharges mucopus (=gound) into the eye } nasolacrimal duct block. Treat any infection promptly. CT of the orbit is needed if orbital cellulitis. If the nasolacrimal duct is permanently blocked, dacryocystorhinostomy (DCR) establishes communication between the lacrimal sac and the nasal cavity.  *:* Squamous cell ca of the lacrimal drainage system.  Conjunctivitis The conjunctiva is red and infl amed, and the hyperaemic vessels may be moved over the sclera, by gentle pressure on the globe. Acuity,pupillary responses, and corneal lustre are unaff ected. Eyes itch, burn, and  lacrimate. There may be photophobia. It is often bilateral with discharge sticking lids together. Causes: adenoviruses (small lymphoid aggregates appear as follicles on conjunctiva), bacteria (purulent discharge more prominent), or allergic.  Chloramphenicol 0.5% drops/4–6h is often used (or fusidic acid drops).Staphs are common causes—and resistance to ciprofl oxacin is spreading, and,to a lesser extent, to gentamicin. 62 It is usually self-limiting (more prolonged if  allergic). In prolonged conjunctivitis, esp. in young adults or those with sexual diseases, consider chlamydial infection (get expert help; see ophthalmia neonatorum, p36).  *for allergic conjunctivitis:* Try anti histamine drops, eg emedastine or olopatadine refer if not settling in a few days. Sodium cromoglicate and steroid drops (after advice from an ophthalmologist) may help.  Anterior uveitis/iritis The uvea is the pigmented part of the eye (iris, ciliary body, choroid). The iris and ciliary  body are called the anterior uvea; as iris infl ammation will involve the ciliary body, the best term is anterior  uveitis, but note that anterior and posterior components may be aff ected together. *The patient:* Acute  pain, photophobia, acuity ( aqueous precipitates),lacrimation (no sticky discharge, unlike in conjunctivitis),  circumcorneal redness (ciliary congestion), small pupil, initially from iris spasm; later it may be irregular  or dilate irregularly due to adhesions between lens and iris (synechiae). *Talbot’s test* is +ve:4 *Slit lamp:* White  precipitates on the back of the cornea; anterior chamber cells (pus =‘hypopyon’). It typically aff ects those of working age. *Causes:* see MINIBOX; often none is found ( ‘autoimmune’). It may relapse so regular eye clinic  care and follow-up is vital. *:* Aim to prevent damage from prolonged infl ammation  (disrupts fl ow of aqueous (glaucoma } adhesions between iris & lens). Drops:  0.5–1% prednisolone/2h, to  infl ammation (hence pain, redness, and exudate). To  prevent adhesions between lens and iris (synechiae) keep pupil dilated with cyclopentolate  0.5%/8h, unless very mild. Use the slit lamp to monitor infl ammation.  *Intravitreal and biological agents* show promise eg anti-TNF and anti-CD20 if  HLAB27+ve (most with anterior uveitis are).55 Adalimumab has a role.56  **Types** & **causes of uveitis**  ***Anterior uveitis:***  • Ank. spond.;Still’s2,  • Sarcoid; Behcet’s, etc3  • Crohn’s/UC; Reiter’s  • Herpes, TB, syphilis, HIV  ***Intermediate uveitis:***  • MS; lymphoma; sarcoid  ***Posterior*** *&* ***panuveitis:***  • Herpes simplex + zoster  toxoplasmosis; TB; CMV;  endophthalmitis  • Lymphoma; sarcoidosis  • Behcet’s |
| **Reference** |  |
| Dr Khalid/Rabia | **b. Acute dacryocystitis**  Dacryocystitis is infection of the lacrimal sac  Features   * watering eye (epiphora) * swelling and erythema at the inner canthus of the eye   Management is with systemic antibiotics. Intravenous antibiotics are indicated if there is associated periorbital cellulitis |

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| **Q:653** | 653. A 52yo male presents with sudden complete loss of vision from right eye. He also had been complaining of right sided headaches which would come up more on chewing. On fundoscopy, the retina was pale and a cherry red spot could be seen in the macular region. What caused this vision loss?  a. CRAO  b. CRVO  c. Branch RAO  d. Branch RVO  e. Circumciliary vein occlusion |
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| **Clincher(s)** |  |
| A |  |
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| C | Branch retinal vein occlusion Signs: Unilateral visual loss and fundal appearances  in the corresponding area. Retinal ischaemia leads to release of  vascular endothelial growth factor (VEGF) and retinal new vessel formation.  Treatment of neovascularization is with laser photocoagulation. Macular  oedema persisting for months without improvement may receive grid pattern  argon laser photocoagulation (} arterial crimping). 7n6=7 |
| D | Diagnosis and diff erential diagnosis Other causes of sudden loss of vision:  • Retinal detachment (p444) • Acute glaucoma (painful, p430) • Migraine.  Stroke patients may complain of monocular blindness but visual fi eld testing  will usually reveal a homonymous hemianopia. Sudden bilateral visual loss is  unusual (may be CMV infection in HIV patients, p448). |
| E | If a single branch of the retinal artery is occluded, the retinal and visual  changes relate only to the part of the retina supplied. |
| **KEY** |  |
| Additional Information | Sudden painless loss of vision  Urgent help is needed in: retinal artery occlusion of <6h; any sudden visual loss of <6h if the  cause is unknown, or giant cell arteritis (GCA). *5 questions:* • Headache associated? (GCA, ESR  (do this test urgently in all cases 50yrs old) • Eye movements hurt? (optic neuritis) • Lights/  fl ashes preceding visual loss? (detached retina) • Like a curtain descending? amaurosis fugax  may precede permanent visual loss, eg from emboli/GCA) • Poorly controlled DM: fi g 1 shows  vitreous haemorrhage (bottom left) from new vessels (top right). *Check:* Acuity, pupil reaction, fundi. Then refer  Anterior ischaemic optic neuropathy (AION) The optic nerve is damaged if posterior ciliary arteries are blocked by infl ammation or atheroma. *Fundoscopy:*pale/swollen optic disc. *Arteritic* ***AION*** *(giant cell arteritis): The other eye*  *is at risk* until steroids are given. *Symptoms:* malaise, jaw claudication (chewing pain) } tender scalp and temporal arteries (thickened } absent pulses), neck pain. *Tests:* ESR (>47) & CRP (>2.45mg/dL) preferably before steroids; temporal artery biopsy within 1 week of starting prednisolone; may miss aff ected sections of artery (skip lesions). *:* Start prednisolone 80mg/24h PO promptly (some advocate higher IV doses if visual failure is occurring). Tailing off steroids as ESR and symptoms settle may take >1yr. 67,68 *Nonarteritic* ***AION****:* Associations: BP; lipids; DM, smoking. Treating these protects vision in the other eye. Histology: necrosis & apoptosis at the photoreceptor level  Vitreous haemorrhage (VH) Source from retinal new vessels (diabetes,branch or central retinal vein occlusion; see BOX), retinal tears, retinal detachment or trauma. Small extravasations of blood produce vitreous fl oaters,(seen by the patient as small black dots or tiny ring-like forms with clear centres) which may not greatly obscure vision. With a large enough bleed to obscure vision, there is no red refl ex and the retina may not be seen. A B-scan  ultrasound is needed to identify a cause in this situation. VH undergoes spontaneous absorption. In dense VH a vitrectomy is done to remove the blood in the vitreous if the retina is torn/detached or the patient needs treatment of new vessels. In diabetic patients who have previously had photocoagulation for new vessels with recurrent VH, it is acceptable to wait 3 months for resolution.  Subacute loss of vision *Optic neuritis* (fi g 2) Unilateral loss of acuity occurs over hours or days. Colour vision is aff ected (dyschromatopsia): reds appear less red, ‘red desaturation’—and eye movements hurt. The pupil shows an aff erent defect (p424). The disc is normal in ~60%, swollen (papillitis) in 23%, blurred and/or hyperaemic in 18% (+haemorrhages in 2%). Temporal pallor occurs in 10% suggesting a past attack of optic neuritis in the same eye.  Recovery is usual over 2–6 weeks, but 45–80% develop multiple sclerosis (MS) in the next 15yrs. Other causes: syphilis, Devic’s demyelination, Leber’s optic atrophy, diabetes, vitamin defi ciency. *:* High-dose methyl prednisolone for  72h (250mg/6h IV), then prednisolone (1mg/kg/d PO) for 11 days may briefl y delay onset of MS (no change to long-term disability).  Transient visual loss Always think of vascular causes, such as platelet–fi brin/cholesterol microemboli  from atherosclerotic plaques in the heart or carotid arteries (any stenosis or bruit?). 71 Be cautious  in diagnosing migraine for the 1st time if aged >50yrs.  **Typical causes:**  • Vascular; TIA; migraine  • Multiple sclerosis  • Subacute glaucoma  (not always painful)  • Papilloedema  **Gradual Loss of Vision**  Be aware that for many, the chief question is likelyto be "will I go blind?": be optimistic where possible.  Patients may *not* tell you that they also fear they are going mad, having complex visual hallucinations,  often of faces. These occur without psychiatric signs and are often related to failing  vision in the elderly: the Charles Bonnet syndrome (p463). 77  Choroiditis (choroidoretinitis) The choroid is part of the uvea (iris, ciliary  body and choroid), and infl ammatory disorders aff ecting the uvea may  also aff ect the choroid. The retina may be invaded by organisms which set  up a granulomatous reaction (which can be mistaken for a retinoblastoma).  Toxoplasmosis and toxocara are more common than TB. Sarcoidosis is another  cause. *Tests:* CXR; Mantoux; serology. In the acute phase, vision may be  blurred, a grey–white raised patch is seen on the retina, vitreous opacities occur,  and there may be cells in the anterior chamber. Later, a choroidoretinal  scar (white patch with pigmentation around) will be seen, these being symptomless  unless involving the macula. Treat the cause.  Choroid melanomas are the commonest malignant tumour of the eye. Appearing  as mottled grey/black on the fundus, they can cause retinal detachment  over the growth. Spread is haematogenous or by local orbit invasion.  *Treatment:* Enucleation, plaque radiotherapy, local tumour irradiation, photocoagulation,  transpupillary thermotherapy & microsurgical resection.  Age-related macular degeneration (ARMD) is the chief cause of registrable  blindness.UK Cause: behavioural/nutritional factors (eg B12) 78 and (epi)genetic  mechanisms.2 It occurs in the elderly who present with deteriorating central  vision. There is pigment, drusen (BOX & fi g 2) and sometimes bleeding at the  macula. ARMD is categorized as dry or wet. Dry ARMD shows mainly drusen and  degenerative changes at the macular. It progresses slowly. Wet ARMD occurs  when aberrant vessels grow from the choroid into the neuro-sensory retina  and leak (choroidal new vessels: CNV). Vision deteriorates rapidly and distortion  is a key feature. Ophthalmoscopy shows fl uid exudation, localized detachment  of the pigment. Treatment is available for wet ARMD (BOX). Be prompt  as substantial visual loss may occur while the patient waits. 79 Patients are  advised to stop smoking and have a diet rich in green vegetable  Gradual loss of vision in teenagers Think of Stargardt macular degeneration  and look for prominent yellow fl ecks in the retina. This condition was the  fi rst to be treated with embryonal stem cells.  Tobacco–alcohol amblyopia From cyanide radicals, when smoking and alcohol  excess are combined. Signs: optic atrophy (fi g 1); loss of red/green discrimination  (early) scotomata. Vitamins *may* help (B1, B2, B6, B12, folic acid). 80  Optic atrophy Discs are pale (degree doesn’t  cor relate with visual loss). It may be from intraocular  pressure (glaucoma), or retinal damage  (choroiditis, retinitis pigmentosa, cerebromacular  degeneration), or be due to ischaemia (retinal  artery occlusion). *Causative toxins:* Tobacco;  methanol; lead; arsenic; quinine; carbon bisulfi de.  *Other causes:* Leber’s optic atrophy (p648), multiple  sclerosis (MS), syphilis, external pressure on  the nerve (intraorbital or intracranial tumours,  Paget’s disease aff ecting the skull). Examine the cerebellum and eye movements:  nystagmus in the abducting eye suggests MS (or stroke or DM); in the elderly  look for temporal artery pulselessness (or a scar from a previous biopsy).  **Typical causes**  • Cataract  • Macular degeneration  • Glaucoma (p440)  • Diabetic retinopathy  • Hypertension (p448)  • Optic atrophy (below)  • Slow retinal detachment |
| **Reference** |  |
| Dr Khalid/Rabia | **a. CRAO**  pale optic disc, cherry red spot on macula  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 |

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| **Q:654** | 654. A 48yo woman presents with left-sided severe headache. She also has a red, watering eye and complains of seeing colored haloes in her vision. What is the most appropriate next step?  a. Measure IOP  b. Relieve pain with aspirin  c. 100% oxygen  d. CT  e. Relieve pain with sumatriptan |
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| Additional Information | In *acute angle closure glaucoma* it is corneal oedema that causes them as  intraocular pressure rises with pupillary dilatation. If haloes are accompanied  by eye pain consider this diagnosis and refer immediately. Jagged haloes  which change shape are usually due to migraine. Beware labelling haloes as  migrainous in those >50yrs who have not previously suff ered from migraine. OHCS 479 |
| **Reference** |  |
| Dr Khalid/Rabia | **a. Measure IOP**  Red watery eye point to cluster headache but that's occur in young males...all other points to glaucoma  haloes seen in glaucoma. Measure iop  Features   * severe pain: may be ocular or headache * decreased visual acuity * symptoms worse with mydriasis (e.g. watching TV in a dark room) * hard, red eye * haloes around lights * semi-dilated non-reacting pupil * corneal oedema results in dull or hazy cornea * systemic upset may be seen, such as nausea and vomiting and even abdominal pain   Management   * urgent referral to an ophthalmologist * management options include reducing aqueous secretions with acetazolamide and inducing pupillary constriction with topical pilocarpine |

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| **Q:737** | 737.   An old woman having decreased vision can’t see properly at night. She has changed her glasses quite a few times but with no effect. She has normal pupils and cornea. What is the most likely dx?  a. Cataract  b.     Glaucoma  c.      Retinal detachment  d.     Iritis  e.      GCA |
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| Dr Khalid/Rabia | Vision loss among the elderly is a major health care problem. Approximately one person in three has some form of vision-reducing eye disease by the age of 65. The most common causes of vision loss among the elderly are age-related macular degeneration, glaucoma, cataract and diabetic retinopathy. Age-related macular degeneration is characterized by the loss of central vision. Primary open-angle glaucoma results in optic nerve damage and visual field loss. Because this condition may initially be asymptomatic, regular screening examinations are recommended for elderly patients. Cataract is a common cause of vision impairment among the elderly, but surgery is often effective in restoring vision. Diabetic retinopathy may be observed in the elderly at the time of diagnosis or during the first few years of diabetes. Patients should undergo eye examinations with dilation when diabetes is diagnosed and annually thereafter.    Many causes of gradual visual loss can be diagnosed on history and examination alone with only the most basic additional investigations. For any patient with a gradual loss in visual acuity ,the following protocol should be followed to reach to an initial diagnosis. AssessmentHistory A routine history is mandatory and will often guide you to a possible cause. Specifically ask about:   * The nature of the problem:   + Unilateral versus bilateral.   + Blurred vision: whether this is the whole field, close, distance or both.   + Restricted visual field: often noted following difficulties in driving, knocking into things at the periphery of vision   + Distorted rather than blurred vision (eg, dent in printed words, door/window frames, objects appearing smaller or larger). If so, check with an Amsler grid (see under 'Further reading & references', below). **Distortion of straight lines indicates serious macular pathology and needs urgent referral**.   + Bits of visual field missing altogether: central versus peripheral; establish what the remainder of the vision is like. * Progression: ask whether there has been a slow and steady decline; whether there have been step-wise drops in visual acuity, or whether the problem has been intermittent. If intermittent, think of transient ischaemic attacks (TIAs) or impending acute angle-closure glaucoma. * Ask whether there have been any associated factors. For example, pain (very important diagnostically, see 'Painful loss', below), redness, or visual phenomena - eg, haloes, flashes of light, new floaters. **These symptoms usually merit an urgent referral**. * Ask whether there are any precipitating factors. Specifically ask about changes over the course of a day and whether vision is better in the day or at night.  Examination Important points to note on examination are:   * The visual acuity of both eyes. Note whether this improves using a pinhole. * The red reflex: a media opacity (appears black against the red reflex) suggests a corneal, lens or vitreous problem. To localise the site of the opacity with respects to the pupil (lens):   + Slowly shift the direction of your ophthalmoscope light.   + Look at the direction in which the opacity appears to move in relationship to the pupillary (central) axis.   + If there is no 'movement' of the opacity, it lies within the pupil (lens).   + If the opacity 'moves' in the same direction, it is anterior to the lens (cornea).   + If the opacity 'moves' in the opposite direction, it is posterior to the pupil (posterior lens or vitreous).   + If the media is clear, it is more likely to be a retinal or optic nerve disorder.   + If there is a normal red reflex, take a good look at the fundus.   + Do a functional testing of visual field, pupils, optic nerve and macula.   + Use an Amsler grid to look for distortion of straight lines - if present, this indicates serious macular pathology and needs urgent referral  Interpreting the findings - some clinical patterns  * Loss of vision in one eye - implies the problem is in the eye itself or in the optic nerve *before* it reaches the optic chiasm. * Loss of vision in both temporal fields (bitemporal hemianopia) - occurs from lesions compressing the optic chiasm (eg, pituitary tumour or craniopharyngioma). * Loss of left or right visual field (homonymous hemianopia) - implies the lesion is somewhere between the optic chiasm and the occiput. * Chronic glaucoma tends to cause tunnel vision (loss of peripheral visual field). * Macular degeneration causes loss of central vision (central scotoma) and may cause distortion of straight lines. * [**Cataracts**](http://patient.info/doctor/cataracts-and-cataract-surgery) - the patient often complains of glare in dark conditions (and so difficulty in driving at night) and may complain that colours appear more dull than they used to. There may be an abnormal red reflex and, in advanced cases, the cataract may be visible to the naked eye (this is increasingly rare these days). Other aspects of the examination should be normal unless there is concurrent pathology. Refer routinely. |

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| **Q:807** | 807.   A 52yo man has a painful, red, photophobic right eye with slightly blurred vision and watering for 3 days. He has had no such episodes in the past. On slit lamp examination there are cells and flare in the ant chamber and pupil is sluggish to react. What is the single most appropriate clinical dx?  a.     Acute close-angle glaucoma  b.     Acute conjunctivitis  c.      Acute dacrocystitis  d. Acute iritis  e.      Corneal foreign body |
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| **KEY** | **D** |
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| Dr Khalid/Rabia | KEY is D  This is a clear Picture of Acute Iritis. Iritis forms the part of anterior Uveitis.  **Anterior uveitis** is the term for inflammation which affects the eye's front (anterior) part of the uveal tract. This can include the iris (iritis) or the iris and the ciliary body (iridocyclitis). It is the most common type of uveitis  SYMPTOMS AND SIGNS:  this usually affects one eye. The common symptoms are eye pain (usually felt as a dull ache in and around the eye), redness of your eye, and photophobia (which means you do not like bright light). You may develop blurred vision or even some visual loss (usually temporary). You may develop headaches and notice that the pupil of the affected eye may change shape slightly. The pupil may not react to light (normally becomes smaller) or it may lose its smooth round shape. Your eye may become watery. The symptoms tend to develop over a few hours or days.  FINDINGS on SLIT LAMP EXAMINATION:  The diagnosis of iritis is confirmed by examining the eye with a slit lamp (a special microscope designed for [eye exams](http://www.webmd.com/eye-health/eye-tests-exams)). Your ophthalmologist can see cells (white[blood cells](http://www.webmd.com/heart/anatomy-picture-of-blood)) and flare (particles of protein) in the fluid that is produced in the eye.  Two other [physical exam](http://www.webmd.com/a-to-z-guides/annual-physical-examinations) findings aid your eye doctor in diagnosing iritis. They include:   * Topical anesthetics do not relieve the pain associated with iritis. * Shining light in the normal, unaffected eye causes pain in the affected eye if iritis is present. This is because shining light in one eye causes both pupils to constrict. Movement of the affected iris causes pain |

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| **Q:835** | A 30yo man comes with hx of early morning back pain and stiffness. Exam: red eyes. What is the single most appropriate option?  a. Iris  b. Ciliary body  c. Cornea  d. Conjunctivitis  e. Sclera |
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| **Clincher(s)** | **Young man,morning backache,stiff ness and red eye** |
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| **KEY** | **A** |
| Additional Information | **Ankylosing Spondylitis:**  Ankylosing spondylitis (AS) is a chronic seronegative spondyloarthropathy which primarily involves the axial skeleton (ie sacroiliitis and spondylitis). The aetiology is unknown but involves the interaction of genetic and environmental factors.  **Presentation**   * Symptoms may be subtle in early stages or mild disease, with an insidious onset over several months to years. * AS usually presents before the age of 30 years. * Most patients have mild chronic disease or intermittent flares with periods of remission. * Systemic features are common. Fever and weight loss may occur during periods of active disease. Fatigue is also prominent. * Morning stiffness is characteristic. * **Inflammatory back pain**:   + Often improves with moderate physical activity.   + Unlike mechanical back pain, patients often experience stiffness and pain which awaken them in the early morning hours.   + The spinal disease starts in the sacroiliac joints (bilateral lumbosacral region) and may be felt as diffuse nonspecific buttock pain.   + On examination there is often tenderness of the sacroiliac joints or a limited range of spinal motion.   + In the advanced stages, patients develop loss of lumbar lordosis, buttock atrophy, and an exaggerated thoracic kyphosis with a stooped forward neck sometimes referred to as a 'question mark posture'.  * **Peripheral enthesitis**:   + Occurs in approximately a third of patients.   + Common sites - behind the heel (Achilles tendonitis), the heel pad (plantar fasciitis) and the tibial tuberosity.   + Lesions tend to be painful, especially in the morning. There may be associated swelling of the tendon or ligament insertion. * **Peripheral arthritis**:   + Also occurs in about a third of patients.   + Joint involvement is usually asymmetric, involving the hips, shoulder girdle (glenohumeral, acromioclavicular, and sternoclavicular joints), joints of the chest wall (costovertebral joints, costosternal junctions) and symphysis pubis.   + Other peripheral joints are less often and less severely affected, usually as asymmetrical oligoarthritis.   + In children, AS tends to commence with arthritis prior to spinal disease developing.   + Temporomandibular joints are occasionally involved.   + **Extra-articular manifestations[**[**5**](http://patient.info/doctor/ankylosing-spondylitis-pro#ref-5)**]**   + **Eye involvement**   + Acute anterior uveitis occurs in 20-30% of patients. Of all patients presenting with acute anterior uveitis, a third to a half have or will go on to develop AS.   + Acute anterior uveitis presents with an acutely painful red eye and severe photophobia and requires emergency treatment to prevent visual loss.   + There could be cardiopulmonary,neurological And Renal involvement too.   **Treatment**: The objectives of treatment for patients with uveitis include the reduction of inflammation (corticosteroids), the relief of symptoms (cycloplegics and anti-inflammatories), and the preservation or restoration of visio |
| **Reference** | Patient.info |
| Dr Khalid/Rabia |  |

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| **Q:841** | A 36yo lady comes with hx of early morning stiffness of her small joints and with red and painful eye. What is the single most appropriate option?  a. Iris  b. Ciliary body  c. Cornea  d. Conjunctivitis  e. Sclera  f. Lichen planus |
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| **Clincher(s)** | **36year old ,morning stiffness of small joints and red painfull eye** |
| A |  |
| B |  |
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| E | **Scleritis often appears in association with other inflammatory diseases such**  **as rheumatoid arthritis and granulomatosis -the histopathological changes**  **are characteristic of a chronic granulomatous disease.** |
| **KEY** | **E** |
| Additional Information | **Scleritis is a chronic, painful, and potentially blinding inflammatory disease that is characterized by edema and cellular infiltration of the scleral and episcleral tissues. Scleritis is commonly associated with systemic autoimmune disorders, including rheumatoid arthritis, systemic lupus erythematosus, relapsing polychondritis, spondyloarthropathies, Wegener granulomatosis, polyarteritis nodosa, and giant cell arteritis.**  **Scleritis may be the initial or only presenting clinical manifestation of these potentially lethal disorders. The correct and rapid diagnosis and the appropriate systemic therapy can halt the relentless progression of both ocular and systemic processes, thus preventing destruction of the globe and prolonging survival**  **The extra-articular manifestations of rheumatoid arthritis affect the following systems:**   * **respiratory** * **haematological** * **neurological** * **lymphoreticular** * **ocular** * **cardiac** * **systemic**   The ocular manifestations of rheumatoid arthritis include:   * keratoconjunctivitis sicca * episcleritis * scleritis and scleromalacia perforans * iritis * tenosynovitis of the ocular muscles * Sjogren's Syndrome   . |
| **Reference** | Gp notebook |
| Dr Khalid/Rabia |  |

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| **Q:842** | A 23yo man comes with 2d hx of sticky greenish discharge from the eyes with redness. What is the single most appropriate option?  a. Iris  b. Ciliary body  c. Cornea  d. Conjunctivitis  e. Sclera |
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| **Clincher(s)** | **Sticky greenish discharge from eyes and redness** |
| A |  |
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| **Reference** | Ohcs 432 |
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| **Q:870** | A 45yo woman had her visual acuity checked at her local optician.12h later she presents to the ED with severe pain and redness in her eye. What is the single most appropriate option?  a. Iris  b. Ciliary body  c. Ant chamber  d. Post chamber  e. Cornea |
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| **Clincher(s)** | **Visual acquity checked,12hr later severe pain and redness in eye** |
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| Dr Khalid/Rabia | The key is C. [In acute angle closure glaucoma half-dilated pupil is the most likely position which precipitates an acute attack as the trabecular meshworks are mostly closed by peripheral anterior synechia of peripheral iris in this position. And mild illuminated darkened room like of an optician (also use of mydriatics accelerates this) or opera (cinema hall) are culprit to make this! As the block occurs in anterior chamber it is the likely option here]. |

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| **Q:917** | A 49yo man complains of fullness in his left ear, recurrent vomiting  and tinnitus. What is the most appropriate med?  a. Buccal prochlorperazine  b. Oral chlorpheniramine  c. Oral flupenphenazine  d. Buccal midazolam  e. IV rantidin |
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| **Clincher(s)** | **Fullness in the ear,recurrent vomiting,tinnitus** |
| A |  |
| B |  |
| C |  |
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| **KEY** | **A** |
| Additional Information | **MENIERES DISEASE (source : nhs)**  **Its a case of menieres disease.**  **Ménière's disease is a rare disorder that affects the inner ear. It can cause vertigo, tinnitus, hearing loss, and a feeling of pressure deep inside the ear.**  **Ménière's disease often progresses through different stages. In the early stages, most people have sudden and unpredictable attacks of vertigo, accompanied by nausea, vomiting and dizziness. During the later stages, the episodes of vertigo tend to occur less frequently and sometimes stop altogether over time. However, the tinnitus and hearing loss often become worse and you may be left with permanent balance and hearing problems.**  **Ménière's disease most commonly affects people aged 20-60 and it's thought to be slightly more common in women than men.**  **During an attack of Ménière's disease, you may be prescribed medication to treat the symptoms of vertigo, nausea and vomiting.This is usually prochlorperazine or an antihistamine.If these work, you may be given a supply to keep, so you can take them quickly during an attack.**  **If you experience vomiting during your attacks, you can take a type of prochlorperazine called Buccastem. This comes as a tablet that you place between your gums and your cheek, on the inside of your mouth. The tablet dissolves and is absorbed into your** |
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| **Q: 1049** | A 32yo man suffering from MS presents with blurring of vision. Ophthalmoscopy shows pallor of the optic disc. Which anatomical site is most likely to be affected?  a. Optic nerve  b. Optic disc  c. Optic radiation  d. Trigeminal  e. Oculomotor nerve |
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| **Clincher(s)** | **MS!** |
| A |  |
| B |  |
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| D |  |
| E |  |
| **KEY** |  |
| Additional Information | **Optic disc pallor shows optic nerve damage.**  **Optic neuritis occurs in MS patients. Pain on eye movement, rapid and decreased central visiom**  Optic neuritis usually affects one eye. Symptoms might include:   * **Pain.** Most people who develop optic neuritis experience eye pain that's worsened by eye movement. Sometimes the pain feels like a dull ache behind the eye. * **Vision loss.** Most people experience at least some temporary reduction in vision, but the extent of vision loss varies. Noticeable vision loss usually develops over hours or days. Exercise or a hot bath or shower may exaggerate the vision loss. Vision loss is permanent in some cases. * **Loss of color vision.** Optic neuritis often affects color perception. You might notice that colors appear less vivid than normal. * **Flashing lights.** Some people with optic neuritis report seeing flashing or flickering lights. |
| **Reference** |  |
| Dr Khalid/Rabia | Optic neuritis is an acute, sometimes painful, reduction or loss of vision in one eye, and is a relatively common presenting symptom of MS. Optic neuritis (ON) is inflammation of the optic nerve. Classically there is a triad of clinical features - reduced vision (of varying severity), eye pain (particularly on movement) and impaired colour vision.  Double vision  Facial weakness  Deafness  Depression  Taste and smell alteration  Loss of sensation in legstr. Myelitis  Urgency and frequency in passing urine  Impotence  Loss of thermoregulation  INV  · Electrophysiology: can detect demyelination in apparently unaffected pathways with characteristic delays. Visual evoked potential studies should be the first choice.  · MRI scan: 95% of patients have periventricular lesions and over 90% show discrete white matter abnormalities. Areas of focal demyelination can also be seen as plaques in the optic nerve, brainstem and spinal cord.  · Cerebrospinal fluid: rise in total protein with increase in immunoglobulin concentration with presence of oligoclonal cases.  Rx:  Decrease stress  Steroid  Interferon  Monoclonal antibody, alemtuzumab  AZT for relapsing and remitting MS |

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| **Q: 1056** | . Which of the following is not a degenerative corneal disease?  a. Band keratopathy  b. Marginal dystropathy  c. Fatty/lipid degeneration  d. Mooren’s ulcer  e. Keratoconus |
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| **Clincher(s)** |  |
| A | Band keratopathy is characterized by the appearance of a band across the central cornea, formed by the precipitation of calcium salts on the corneal surface (directly under the epithelium).[[1]](javascript:showrefcontent('refrenceslayer');)This form of corneal degeneration can result from a variety of causes, either systemic or local, with visual acuity decreasing in proportion to the density of the deposition (see the image below). (See Etiology.)  Band keratopathy. Note the bandlike whitish-grey lesion across the corneal surface, sparing the superior and inferior cornea. |
| B | **Pellucid marginal degeneration** (PMD; **keratotorus**), is a degenerative [corneal](https://en.wikipedia.org/wiki/Cornea) condition, often confused with [keratoconus](https://en.wikipedia.org/wiki/Keratoconus). It is typically characterized by a clear, bilateral thinning ([ectasia](https://en.wikipedia.org/wiki/Ectasia)) in the inferior and peripheral region of the cornea, although some cases affect only one eye. The cause of the disease remains unclear ([idiopathic](https://en.wikipedia.org/wiki/Idiopathic)).  The ocular condition present in the patient whose case is to be reported has been described under various names, furrow keratitis, marginal degeneration of the cornea, ectatic marginal dystrophy of the cornea and other variants. Marginal dystrophy of the cornea seems most appropriate, as this feature is common in all cases and the term is in keeping with modern nomenclature. |
| C |  |
| D |  |
| E | keratoconus is an eye condition in which the normally round dome-shaped clear window of the eye (cornea) progressively thins causing a cone-shaped bulge to develop. Exactly why this happens is unknown, but genetic factors play a role and it is more common in people with allergic diseases such as asthma, in Down's syndrome and in some disorders of connective tissue such as Marfan's disease. It affects up to one in 1,000 people and is more common in people of Asian heritage. It is usually diagnosed in teenagers and young people. |
| **KEY** |  |
| Additional Information | Mooren's ulcer is a rapidly progressive, painful, ulcerative keratitis which initially affects the peripheral cornea and may spread circumferentially and then centrally. Mooren's ulcer can only be diagnosed in the absence of an infectious or systemic cause and must be differentiated from other corneal abnormalities, such as Terrien's degeneration. Although the etiology remains unknown, recent research has proposed an underlying immune process and a possible association with the hepatitis C virus. The response to medical and surgical intervention is typically poor, and the visual outcome can be devastating. |
| **Reference** | <http://www.ncbi.nlm.nih.gov/pubmed/9848831>. Medspace, jama ophthalmology |
| Dr Khalid/Rabia | Dx is Mooren’s ulcer.  Band keratopathy is characterized by the appearance of a band across the central cornea, formed by the precipitation of calcium salts on the corneal surface (directly under the epithelium). This form of corneal degeneration can result from a variety of causes, either systemic or local, with visual acuity decreasing in proportion to the density of the deposition.  Pellucid marginal degeneration is a degenerative corneal condition, often confused with keratoconus.  Keratoconus is a degenerative disorder of the eye in which structural changes within the cornea cause it to thin and change to a more conical shape than the more normal gradual curve.  Mooren’s ulcer is a chronic, painful peripheral corneal ulcer of unknown cause that easily leads to loss of vision. Severe pain, red, tearing and photophobic.  Fatty/lipid degeneration is degenerative.  In mooren’s ulcer severe pain is common and eye(s) may be very red, photophobic, and tearing. It is more common in southern and central Africa, China, and India. Treatments tried: steroidal and nonsteroidal anti-infl ammatory drops, cytotoxics (topical and systemic), conjunctivectomy, and cornea debridement (superficial keratectomy). None is known to be superior |

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| **Q: 1090** | A 29yo woman presents with lid lag, lid retraction and diplopia. What is the most appropriate next step?  a. TFT  b. Tensilon test  c. Fundoscopy  d. Autoantibodies  e. EMG |
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| **Clincher(s)** | **Lid lag, lid retraction, diplopia** |
| A |  |
| B | The Tensilon test is a method to help diagnose [myasthenia gravis](https://www.nlm.nih.gov/medlineplus/ency/article/000712.htm). |
| C |  |
| D |  |
| E | Electromyography (**EMG**) is a diagnostic procedure to assess the health of muscles and the nerve cells that control them (motor neurons). Motor neurons transmit electrical signals that cause muscles to contract. |
| **KEY** | **D** |
| Additional Information |  |
| **Reference** | Should be tft |
| Dr Khalid/Rabia | Scenario suggestive of Graves disease, hyperthyroidism. Graves' disease:[9]  \* This is the most common cause of hyperthyroidism and has an autoimmune basis. It is mediated by B and T lymphocytes, characterised also by the presence of thyroid-  stimulating immunoglobulins (TSIs). These are directed at four different thyroid antigens:  \* Thyroglobulin.  \* Thyroid peroxidase (or antimicrosomal antibodies).  \* Sodium-iodide symporter.  \* TSH receptor.  \* The condition is characterised by a small to moderate diffuse, firm goitre with 50% of these showing ophthalmopathy.  \* There may be a personal or family history of autoimmune disease.  \* <5% have pretibial myxoedema called thyroid dermopathy (as can occur anywhere, particularly following trauma). This is usually associated with moderate to severe ophthalmopathy. 10-20% have clubbing (thyroid acropathy). Thyroid dermopathy usually appears as non-pitting plaques with pink/purple colour. There are also nodular and generalised forms.  \* There may also be lymphoid hyperplasia including splenomegaly and an enlarged thymus. \* Associated with other autoimmune conditions - eg, pernicious anaemia, type 1diabetes mellitus.  Investigations \* Thyroid function tests (TFTs): serum TSH can exclude primary thyrotoxicosis. Confirm the diagnosis with free T4 levels. If TSH is suppressed but free T4 levels are normal, then if not previously supplied, free T3 level is needed (T3 toxicosis occurs in 5% of patients).  \* Autoantibodies - these are most commonly seen in Graves' disease:  \* Antimicrosomal antibodies - against thyroid peroxidase.  \* Antithyroglobulin antibodies.  \* TSH-receptor antibodies which are commonly present in Graves' disease but are not routinely measured.  \* TSI if elevated helps to establish a diagnosis of Graves' disease |

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| **Q: 1144** | A 28yo woman has been on tx for RA for 3yrs. She has gradual loss of vision in both eyes. Her  IOP is normal. Red reflex is absent in both eyes. What is the single most likely dx?  a. Cataract  b. DM retinopathy  c. Hypermetropia  d. Macular degeneration  e. HTN retinopathy |
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| **Clincher(s)** |  |
| A |  |
| B | Patient not diabetic |
| C | **Hypermetropia** (Hyperopia) - Long Sighted. **Hypermetropia** means long sight and is where the image of a nearby object is formed behind the retina. |
| D |  |
| E | Patient not hypertensive |
| **KEY** | **A** |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia | absent red reflex + tx for RA for 3yrs+ gradual loss of vision in both eyes.  the treatment plan of rheumatoid arthritis includes : corticosteroids which induce cataract formation with long term use  - Cataracts are cloudy (opaque) areas that develop in the lens of an eye and affects vision  - Vision becomes gradually worse over the years.  - Most affected people develop a cataract for no apparent reason. Factors that may increase the chance of developing cataracts include:  - Having a poor diet.  - Smoking.  - Being exposed to a lot of ultraviolet light.  - Diabetes.  - Steroid medicines.  - Having a family history of cataracts  - There are no medicines, eye drops or lasers that can treat cataracts. The only way of treating cataracts is with an operation. This is a very common operation |

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| **Q: 1222** | A 45yo man has had impaired vision and pain on eye movement in his left eye over the last 5 days. He also notes loss of color vision in the same eye. In the left eye, the visual acuity is up to  counting fingers. When the pupil is stimulated with light, it dilates. His fundus is normal. What is  the single most appropriate clinical dx?  a. Acute dacryocystitis  b. Acute iritis  c. Papillitis  d. Retrobulbar neuritis  e. Scleritis |
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| **Clincher(s)** |  |
| A | Acute inflammation of the tear sac |
| B | * Pain in the eye or brow region * Worsened [eye pain](http://www.webmd.com/eye-health/eye-pain-causes-symptoms-diagnosis-treatment) when exposed to bright light * Reddened eye, especially adjacent to the iris * Small or funny shaped pupil * Blurred [vision](http://www.webmd.com/eye-health/default.htm) * [Headache](http://www.medicinenet.com/script/main/art.asp?articlekey=20628) |
| C | **Optic papillitis** is a specific type of [optic neuritis](https://en.wikipedia.org/wiki/Optic_neuritis). Inflammation of the [optic nerve head](https://en.wikipedia.org/wiki/Optic_nerve_head) is called "papillitis" or "intraocular optic neuritis"; inflammation of the [orbital](https://en.wikipedia.org/wiki/Orbit_(anatomy)) portion of the nerve is called "retrobulbar optic neuritis" or "orbital optic neuritis".[[1]](https://en.wikipedia.org/wiki/Optic_papillitis#cite_note-1) It is often associated with substantial losses in visual fields, pain on moving the globe, and sensitivity to light pressure on the globe. It is often an early sign of [multiple sclerosis](https://en.wikipedia.org/wiki/Multiple_sclerosis).[[2]](https://en.wikipedia.org/wiki/Optic_papillitis#cite_note-2)  Papillitis may have the same appearance as papilledema. However, papillitis may be unilateral, whereas papilledema is almost always bilateral. Papillitis can be differentiated from papilledema by an afferent pupillary defect (Marcus Gunn pupil), by its greater effect in decreasing visual acuity and color vision, and by the presence of a central scotoma. |
| D |  |
| E | Scleritis is a chronic, painful, and potentially blinding inflammatory disease that is characterized by edema and cellular infiltration of the scleral and episcleral tissues. |
| **KEY** | **d. Retrobulbar neuritis** |
| Additional Information |  |
| **Reference** | Webmd.ohcs. |
| Dr Khalid/Rabia | Optic neuritis or retrobulbar neuritis is inflammation of the optic nerve. It consists of the classic triad -   1. Reduced vision 2. Eye pain, particularly on movement 3. Impaired colour vision   Common causes are multiple sclerosis, giant cell arteritis.  Typical signs - decreased pupillary light reaction in affected eye, or Marcus Gunn Pupil |

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| **Q:372** | A HTN male loses vision in his left eye. The eye shows hand  movement and a light shined in the eye is seen as a faint light.  Fundus exam: flame shaped hemorrhages. The right eye is normal.  What is the cause of this pts unilateral blindness?  a. HTN retinopathy  b. CRA thrombosis  c. CRV thrombosis  d. Background retinopathy  e. Retinal detachment |
|  |  |
| **Clincher(s)** | **HTN male loses vision in left eye. Funds exam reveal flame shape haemorrhages.** |
| A | HTN retinopathy should be bilateral |
| B | CRAO will present with sudden loss of vision and optic disc will be pallor with cherry red macula. |
| C | CRVO can also cause sudden painless vision but slower then CRAO and fundus will show stormy Sunset picture due to haemorrhages. |
| D | Related with DM |
| E | Sudden loss of vision here remember four P’s floaters, field loss, flashes, fall in acuity. |
| **KEY** | **C** |
| Additional Information | *Central retinal vein thrombosis is common in obese and with diabetic, HTN hx. Also present in polycythemia, atherosclerosis and glaucoma all types. It causes sudden painless visual loss although it is less sudden then CRAO but most common.* |
| **Reference** |  |
| Dr Khalid/Rabia | unilateral blindness with flame shaped hemorrhages are  characteristic of CRVO).  Flame shaped hemorrhages are seen in HTN and diabetic  retinopathy too but they will cause bilateral damage. |

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| **Q:397** | An old woman having decreased vision cannot see properly at night. She has changed her glasses quite a few times but to no effect. She has normal pupil and cornea. What is the most likely dx?  a. Cataract  b. Glaucoma  c. Retinal detachment  d. Iritis  e. GCA |
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| **Clincher(s)** | **Changes of glasses due to Dec vision but no improvement.** |
| A | Opacity of lens and cause Dec vision and changes of glasses doesn't help in correcting the vision. |
| B | Glaucoma is inc in intra ocular pressure and visual acuity is decreased  With acute close angle it's painful red eye with fixed dilated oval pupil. With chronic/open angle glaucoma no pain but optic disc cupping. |
| C | Sudden loss of painless vision |
| D | Iritis is part of Ant uveitis and present in AS, Reiters syndrome, UC, Crihns, sarcoidosis, behcets etc. It cause fall in visual acuity and red eye |
| E | GCA will give hx of headache, jaw claudication and sudden loss of vision. |
| **KEY** | **A** |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia | old age and progressive weakness supports Cataract  Not glaucoma...as pupil would be mid dilated and sluggish reaction and in acute attack corneal edema    Not RD...as pupil would be yellowish in color and there would be RAPD in massive RD and vision would be dropped in day and night    Not iritis..as pupil would be constricted and cornea would have precipitation on its back (keratic precipitate)    Not GCA(giant cell arteritis) as vision on it is suddenly dropped to HM up to LP and vision dropped day and night |

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| **Q:398** | A pt comes with sudden loss of vision. On fundoscopy the optic disc is normal. What is the underlying pathology?  a. Iritis  b. Glaucoma  c. Vitreous chamber  d. Retinal detachment |
|  | Sudden loss of vision but on fundoscopy the disc is normal |
| **Clincher(s)** |  |
| A | Complicated and untreated iritis can cause closed angle glaucoma but not painless sudden vision loss. |
| B | It's a gradual loss of vision |
| C | Vitreous chamber involvement such as haemorrhage there's no red reflex and retina can not be seen. |
| D | Reyinal detachment again cause sudden painless vision. |
| E |  |
| **KEY** | **D** |
| Additional Information | *I think the option of stroke or TIA is missing as retinal detachment doesnot present with normal disc but here no option given for stroke so might be a very minute one which can be missed on optic disc I suppose in retinal detachment.* |
| **Reference** |  |
| Dr Khalid/Rabia | Causes of sudden painless loss of vision:  1. Retinal detachment  2. Vitreous haemorrhage  3. Retinal vein occlusion  4. Retinal artery occlusion  5. Optic neuritis  6. Cerebrovascular accident (GCA) |

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| **Q:419** | A 54yo myopic develops flashes of light and then sudden loss of vision. That is the single most appropriate tx?  a. Pan retinal photo coagulation  b. Peripheral iridectomy  c. Scleral buckling  d. Spectacles  e. Surgical extraction of lens |
|  |  |
| **Clincher(s)** | **Tx of retinal detachment** |
| A | PRP is an extensive laser treatment applied to the peripheral retina inside of your eye. This treatment is recommended when abnormal blood vessels are growing inside of your eye |
| B | Done in acute close angle glaucoma |
| C | Tx for retinal detachment. Other options are vitrectomy, cryo or laser therapy to secure the retina. |
| D | For myopic/hyper entropic/astigmatics |
| E | For cataracts. |
| **KEY** | **C** |
| Additional Information | **Scleral buckling is a surgical procedure in which a piece of silicone plastic or sponge is sewn onto the sclera at the site of a retinal tear to push the sclera toward the retinal tear. The buckle holds the retina against the sclera until scarring seals the tear. It also prevents fluid leakage which could cause further retinal detachment.**  **www.surgeryencyclopedia.com** |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q:429** | . A pt presents with a painful, sticky red eye with a congested conjunctiva. What is the most suitable tx?  a. Antibiotic PO  b. Antihistamine  c. Antibiotic drops  d. Steroid drops  e. IBS |
|  |  |
| **Clincher(s)** | **Tx of conjunctivitis** |
| A | No role |
| B | Given in allergic conjunctivitis |
| C | Right option |
| D | Can be given in severe inflammatory conditions. |
| E | ? |
| **KEY** | **C** |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia | Painful eye, usually bilateral. Smearing of vision on waking up.Mild photophobia. If severe, indicates corneal involvement or adenoviral conjunctivitis.  Thick yellowish-white mucopurulent discharge. Visual acuity is normal  Symptoms- Red eye, difficult to open in the morning, glued together by discharge. Presence of follicles on the conjunctiva- More likely viral conjunctivitis.  Treatment:  Topical broad spectrum antibiotics. Drug of choice is chloramphenicol drops. If pregnant, intolerant to chloramphenicol or history of aplastic anemia or blood dyscrasia, use fusidic acid. |

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| **Q:443** | A 38yo female presents with sudden loss of vision but fundoscopy is normal. She had a similar episode about 1 y ago which resolved completely within 3m. Exam: mild weakness of right upper limb and exaggerated reflexes. What is the single most appropriate tx?  a. Pan retinal photo coagulation  b. Pilocarpine eye drops  c. Corticosteroids  d. Peripheral iridectomy  e. Surgical extraction of lens |
|  |  |
| **Clincher(s)** | **Sudden loss of vision with relapsing, remitting hx. Mild weakness of right upper limb and exaggerated reflexes all point towards MS.** |
| A | Done for new abnormal vessels formed. |
| B | No help here it's given In glaucoma. |
| C | Right option |
| D | Tx of acute angle closure glaucoma |
| E | Tx of cataract. |
| **KEY** | **C** |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia | This is a case of optic neuritis caused by Multiple sclerosis. Steroids are the answer here. They are given during acute symptomatic attacks of MS. During relapse or remission, disease modifying agents like interferons are given.  > Pan retinal photocoagulation is done for diabetic retinopathy where parts on the retina are burned in order to reduce the Oxygen demand.  Lens extraction is done mainly for cataract to remove the opacified lens that disturbs the vision  > Peripheral iridectomy is done by making a hole in the iris for open angle glaucoma in order to provide an alternative drainage for the fluid accumulating inside the eye, thus decreasing the IOP.  > Pilocarpine is a parasympathomimetic given for open angle glaucoma in order to contract the ciliary muscles and to open the trabecular meshwork, allowing increased outflow of the aqueous humour  >Surgical extraction of the lens is done for cataract where the opacified lens that disturbs the vision is removed |

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|  |  |
| **Clincher(s)** | **38yo, female, sudden loss of vision, fundoscopy normal, h/o 1yr ago, weakness in RUL and exaggerated reflexes.** |
| A | Pan retinal photocoagulation is done for **diabetic retinopathy** where parts on the retina are burned in order to reduce the Oxygen demand. |
| B | Pilocarpine is a parasympathomimetic given for **open angle glaucoma** in order to contract the ciliary muscles and to open the trabecular meshwork, allowing increased outflow of the aqueous humour |
| C |  |
| D | Peripheral iridectomy is done by making a hole in the iris for **open angle glaucoma** in order to provide an alternative drainage for the fluid accumulating inside the eye, thus decreasing the IOP. |
| E | Surgical extraction of the lens is done **for cataract** where the opacified lens that disturbs the vision is removed |
| **KEY: C** | **Corticosteroid.**  the term rapid loss of vision is more appropriate than sudden loss of vision in multiple sclerosis. Remission and relapse of optic neuritis and focal neurological symptoms and exaggerated reflexes all points towards multiple sclerosis. Treatment option is corticosteroids. |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q: 460** | A 55yo man presents with mild headache. He has changed his spectacles thrice in 1 yr. there is mild cupping present in the disc and sickle shaped scotoma present in both eyes. What is the single most appropriate tx?  a. Pan retinal photo coagulation  b. Pilocarpine eye drops  c. Corticosteroids  d. Scleral buckling  e. Analgesics alone |
|  |  |
| **Clincher(s)** | **sickle shaped scotoma,** **mild cupping present in the disc, spectacles thrice in 1 yr, headache** |
| A |  |
| B |  |
| C |  |
| D | Scleral buckling is done for retinal detachment to put the retina back in place. |
| E |  |
| **KEY: B** | **Pilocarpine.**  Sickle-shaped scotoma or siedel sign is often seen in glaucoma. That along with the fact that he keeps changing his spectacles denotes that this is a case of progressive open angle glaucoma. It can also present with nausea, vomiting, headache and ocular pain. Treated with Carbonic anhydrase inhibitors like acetazolamide, Miotic agents (parasympathomimetics) such as pilocarpine, Alpha2-adrenergic agonists like brimonidine, or Prostaglandin analogs like latanoprost. |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q: 473** | A 62yo male comes to the GP complaining of double vision while climbing downstairs. Which of the following nerve is most likely involved?  a. Abducens nerve  b. Trochlear nerve  c. Oculomotor nerve  d. Optic nerve  e. Trigeminal nerve |
|  |  |
| **Clincher(s)** |  |
| A |  |
| B |  |
| C |  |
| D |  |
| E |  |
| **KEY: B** | **Trochlear Nerve**  This is a lesion in the Trochlear nerve affecting the Superior oblique muscle.  All extrinsic muscles of the eye are supplied by the Oculomotor nerve except the Lateral rectus by the Abducens nerve and the Superior oblique by the trochlear (mnemonic LAST).  Oculomotor nerve may cause palsy of inferior rectus, medial rectus and superior rectus manifesting as double vision in multiple gaze. But trochlear involving superior oblique only causes diplopia in downgaze only. |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q: 487** | A 62yo lady presents with right sided headache and loss of vision. What is the single most inv?  a. ESR  b. BUE  c. CT head  d. XR orbit  e. IOP |
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| **Clincher(s)** |  |
| A |  |
| B |  |
| C |  |
| D |  |
| E | IOP (Intraocular pressure) is used to investigate glaucoma. |
| **KEY: A** | **ESR**  This is most probably Giant cell arteritis/Temporal arteritis. It is common in females and elderly people and should always be considered in cases of new-onset headache in patients 50 years of age or older. Initial investigation is ESR which will be raised (>40mm/hr), and confirmatory diagnosis is temporal artery biopsy. Patient should be started on steroids immediately if GCA is suspected, even if diagnosis is not confirmed, as delay in treatment might lead to blindness due to occlusion of the ophthalmic artery. |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q: 518** | A 70yo woman presents with recurrent episodes of parotid swelling. She complains of difficulty in talking and speaking and her eyes feel gritty on waking in the morning. What is the single most likely dx?  a. C1 esterase deficiency  b. Crohns disease  c. Mumps  d. Sarcoidosis  e. Sjogrens syndrome |
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| **Clincher(s)** |  |
| A | Autosomal dominant, Clinically the patient suffers oedema of the skin and mucosal surfaces |
| B |  |
| C | Most commonly in children |
| D |  |
| E |  |
| **KEY: E** | Sjogrens syndrome.  parotid swelling, difficulty talking and speaking (?) (or chewing and swallowing) due to dry mouth and eyes feeling gritty on waking in the morning due to dryness of eye are suggestive of Sjogrens syndrome |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia | e. Sjogrens syndrome main symptoms of xerophthalmia (dry eyes), xerostomia (dry mouth) and enlargement of the parotid glands.  • Difficulty eating dry food, typically cracker biscuits.  • Difficulty with dentures.  • Complaint of the tongue sticking to the roof of the mouth.  • Speaking for long periods of time causes hoarseness.  • Oral candidiasis and angular cheilitis.  Dry eyes tend to cause a gritty sensation. There is a predisposition to blepharitis and the eyes may be sticky in the morning.  • There may be recurrent parotitis, usually bilateral. Glands are usually enlarged but this is not often the presenting feature. • Dryness of the mucosa of the trachea and bronchi may present as a dry cough. • Dryness of the pharynx and oesophagus may cause difficulty in swallowing, and lack of saliva and secretions may predispose to gastro-oesophageal reflux. • There can be dry skin and vaginal dryness causing dyspareunia  • Disease of the pancreas can lead to malabsorption and even acute pancreatitis or chronic pancreatitis but a more likely cause of elevated serum amylase is parotitis.  • Fatigue is a common feature.  • About 20% have Raynaud's phenomenon.  Associated diseases  There may be a number of associated autoimmune conditions, such as the variant of scleroderma: calcinosis, Raynaud's phenomenon, (o)esophageal motility disorder, sclerodactyly and telangiectasia (CREST). There may be joint pain, swelling and fatigue rrecurrent miscarriage with antiphospholipid syndrome.  Investigations • Rheumatoid factor • Antinuclear antibodies • Schirmer test |

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| **Q:1287** | A pt comes with sudden loss of vision. Exam: high BP. Fundoscopy: retina appears swollen.  Which blood vessel occlusion is involved?  a. Branch RVO  b. Branch RAO  c. CRAO  d. CRVO |
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| **Clincher(s)** | **Sudden loss of vision , high bp** |
| A |  |
| B |  |
| C | In CRAO you'd see a pale white retina,and is usually secondary to a thromboembolus ie in people with condition predisposing to a hypercoagulative state,and yes HTN is one such state... |
| D | a swollen retina indicates blood stasis,ie congestion in the retina due to outflow obstruction due to blockage of the main blood outflow channel from the retina ie CRV |
|  |  |
| **KEY** | **d-CRVO** |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia | In CRAO retina would be pale and you'd see a cherry red macula  In Non-ischaemic - mild defect. There are widespread dot-blot and flame haemorrhages throughout the fundus and some disc oedema. In Ischaemic - severe visual impairment, the fundus looks similar to the non-ischaemic picture but disc oedema is more severe.  CRVO: tomato splash, swollen huge optic disc, congested fundus  CRAO: white retina, pale fundus, cherry red spot |

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| **Q:1294** | An 84yo woman with drusen and yellow spots in the center of retina. What is the single most likely dx?  a. Macular degeneration  b. HTN retinopathy  c. MS  d. DM background  e. Proliferative DM retinopathy |
|  |  |
| **Clincher(s)** | **Drusen yellow spots** |
| A | *Maculopathy:* Leakage from the vessels close  to the macula cause oedema and can signifi cantly threaten vision (clinically  signifi cant macular oedema). It can exist with otherwise mild retinopathy. |
| B |  |
| C | *MS –hard exudates , macular edema, hemorrhage* |
| D | *Non-proliferative diabetic retinopathy (****NPDR****)* is rated as mild, moderate  or severe depending on the degree of ischaemia. Signs comprise microaneurysms  (seen as ‘dots’), haemorrhages (fl ame shaped or ‘blots’) hard  exudates (yellow patches), engorged tortuous veins, cotton wool spots, large  blot haemorrhages (the latter 3 are signs of signifi cant ischaemia). NPDR can  progress to sight-threatening proliferative retinopathy. |
| E | *Proliferative diabetic retinopathy (****PDR****):* Fine new vessels appear on the optic disc, retina and cancause vitreous haemorrhage. |
| **KEY** | a. Macular degeneration |
| Additional Information |  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  Features   reduced visual acuity: 'blurred', 'distorted' vision, central vision is affected first   central scotomas   fundoscopy: drusen, pigmentary changes |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q:1295** | 1295. A pt presents with headache, blurring of vision and acuity loss. On fundoscopy, dots and blots  were noted with huge red swollen optic disc. What is the most probable dx?  a. CRAO  b. Branch RAO  c. CRVO  d. Optic atrophy |
|  |  |
| **Clincher(s)** | **Headache , blurring of vision , acuity loss** |
| A | In CRAO retina would be pale and you'd see a cherry red macula  In Non-ischaemic - mild defect. There are widespread dot-blot and flame haemorrhages throughout the fundus and some disc oedema. In Ischaemic - severe visual impairment, the fundus looks similar to the non-ischaemic picture but disc oedema is more severe.  CRVO: tomato splash, swollen huge optic disc, congested fundus  CRAO: white retina, pale fundus, cherry red spot |
| B |  |
| C | Retinal vein occlusion is one of the most common causes of sudden painless unilateral loss of vision. Loss of vision is usually secondary to macular oedema |
| D |  |
| E |  |
| **KEY** | C – CRVO |
| Additional Information | . |
| **Reference** |  |
| Dr Khalid/Rabia | Branch RVO: unilateral, painless blurred vision, metamorphopsia (image distortion) ± a field defect. Fundoscopy will reveal vascular dilatation and tortuosity of the affected vessels, with associated haemorrhages in that area only (look for an arc of haemorrhages, like a trail left behind a cartoon image of a shooting star).  Retinal vein occlusion is one of the most common causes of sudden painless unilateral loss of vision. Loss of vision is usually secondary to macular oedema.  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 |

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| **Q:1296** | A 64yo DM has come for a routine eye check up. Fundoscopy: new vessels all over the retina. What is the most appropriate management?  a. Strict sugar control  b. Regular eye check ups  c. Non urgent referral to specialist  d. Laser photocoagulation  e. Insulin |
|  |  |
| **Clincher(s)** |  |
| A |  |
| B |  |
| C |  |
| D |  |
| E |  |
| **KEY** | d. Laser photocoagulation  This has been the mainstay of treatment for a period of 25 years: the aim is to induce regression of new blood vessels and reduce central macular thickening. |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia | |  | | --- | |  |  |  |  | | --- | --- | | Traditional classification | New 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 | 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 | |

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| **Q:1351** | A 4yo boy ingested his grandmother’s medicine and has developed dilated pupil. What is the  cause?  a. Amitryptiline  b. Paracetamol  c. Iron  d. Digoxin |
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| **Clincher(s)** |  |
| A | Tcricyclic antidepressant |
| B | Liver affected mainly |
| C | Liver - |
| D | **Yellow halos around eye** |
| E |  |
| **KEY** | * 1. Amtriptyline has side effects common to anticholinergics which include mydriasis. |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q:1390** | A 7yo boy presents with proptosis and periorbital edema. What is the immediate action that needs to be taken?  a. IV morphine and immediate ophthalmoscopy  b. IV morphine  c. Observation only |
|  |  |
| **Clincher(s)** | **Proptosis and periorbital edema.** |
| A |  |
| B |  |
| C |  |
| D |  |
| E |  |
| **KEY** | **Best choice is A IV morphine and immediate ophthalmoscopy.** |
| Additional Information | **This patient is suffering from orbital cellulitis. Urgent ophthalmogist opnion is needed to save his eye.** |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q:** |  |
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| **Clincher(s)** |  |
| A |  |
| B |  |
| C |  |
| D |  |
| E |  |
| **KEY** |  |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q:1457** | A 37yo laborer comes with hx of redness of left eye with foreign body sensation in the same eye. What is the single most appropriate option?  a. Ciliary body  b. Sclera  c. Conjunctivitis  d. Cornea  e. Iris |
|  |  |
| **Clincher(s)** |  |
| A |  |
| B |  |
| C |  |
| D |  |
| E |  |
| **KEY** | D Cornea. Injury is caused by a foreign body and foreign bodies most like injure cornea leading to corneal ulcer if not removed |
| Additional Information | **Corneal injuries** may be physical, chemical, environmental (eg, ultraviolet (UV) damage) or  infective.  Corneal abrasions are common. There is usually a history of minor trauma from a scratch,  grit or contact lens problem.  If there is no history of injury, consider infection - eg, herpes simplex infection, chemical  injury and corneal foreign bodies.  **Symptoms of superficial corneal abrasion or corneal FB :** Redness, pain, watering  (usually). FB sensation (usually), Blurred vision, Photophobia,Pain on eye movement,  Patients are fairly reliable at locating the FB.  Investigations: are not required if you can be sure that the injury is superficial.Plain X-rays of    of the orbit/face can be used to exclude known radiopaque FBs  Treatment= Removal of Foreign body. Use a topical anesthetic  Management of corneal abrasion= refer if large abrasion otherwise: Analgesia= Paracetamol    or ibuprofen are first line. topical antibiotics for 7 days= Chloramphenicol for 1st line. |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q: 1559** | A pt with sudden severe eye pain, red eye, visual blurring, acuity of only finger counting, nausea, vomiting with a shallow ant chamber that is hazy on shining a torch. What is the dx?  a. CRVO  b. Acute closed angle glaucoma  c. Uveitis  d. Iritis  e. Open angle glaucoma |
|  |  |
| **Clincher(s)** | **Painful eye with visual blurring and acuity of only one finger.** |
| A |  |
| B |  |
| C |  |
| D |  |
| E |  |
| **KEY** | **B- acute angle glaucoma,** |
| Additional Information | The painful presentation rules out CRVO and Open Angle Glaucoma. The characteristic nausea, vomiting and shallow anterior chamber signifies an acute event, which in this case would be Acute Closed Angle Glaucoma. Uveitis and Iritis will not have nausea or vomiting which signify markedly raised intraocular pressure.  Treatment  \* Pilocarpine 2 drops x 2 hourly  \* Acetazolamide 500mg IV x STAT OR Mannitol IV  \* Good analgesics  \* Anti-emetics |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q: 1612** | A 75yo man with declining vision, cornea and pupils are normal, fundus shows obscured margins. What is the single most likely dx?  a. Macular degeneration  b. HTN retinopathy  c. MS  d. DM background  e. Proliferative DM retinopathy |
|  |  |
| **Clincher(s)** | **Declining vision, cornea and normal pupils with obscured fundus.** |
| A |  |
| B |  |
| C |  |
| D |  |
| E |  |
| **KEY** | **E** |
| Additional Information | The age of the patient, normal opthalmological examination and **obscured margins of the fundus** all point towards age related macular degeneration. HTN would have other findings on the ocular exam, MS presents with optic neuritis and RAPD +ve with red colour blindness developing, DM and proliferative DM would present with other fundus findings according to degree and stage of diabetic retinopathy. |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q: 1625** | A 22yo man has had an acute, painful, red right eye with blurring of vision for one day. He had a similar episode 1y ago and has had episodic back pain and stiffness relieved by exercise and diclofenac for four years. What is the SINGLE most likely cause of his red eye?  a. Chorioretinitis  b. Conjunctivitis  c. Episcleritis  d. Iritis  e. Keratitis |
|  |  |
| **Clincher(s)** | **Acute painful, red right eye with blurring of vision for one eye and backache with stiffness- ankylosing spondylitis.** |
| A | Inflammation of iris, retina and choriod |
| B | Inflammation of conjunctiva. |
| C | Inflammation below the conjunctiva in the sclera |
| D |  |
| E |  |
| **KEY** | **D- Iritis.** |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia |  |

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| **Q: 1689** | A 30yo caucasian man presented with a 2wk hx of gradually worsening vision in his left eye. The patient had been seen once by a neurologist 2yrs prv for flashes. At that time a head CT was normal. The patient was lost to follow up with the neurologist, but the flashes had continued for the 2yr period. The patient did not experience visual changes with activity or movement. The patient reported continued decreasing vision. Goldmann visual fields were done and showed a central scotoma. A MRI was done at this time and showed inflammation of the left optic nerve. A likely diagnosis is?  a. Pseudotumor  b. Orbital teratoma  c. Optic neuritis  d. Sarcoidosis  e. Optic glioma  f. Lymphangioma  g. Rhabdomyosarcoma  h. Retinal vascular shunts  i. Retinoblastoma  j. Mucormycosis |
|  |  |
| **Clincher(s)** | **Previous history of flashes and inflammation of optic nerve** |
| A |  |
| B |  |
| C |  |
| D |  |
| E |  |
| **KEY** | **C- optic neuritis.** |
| Additional Information |  |
| **Reference** |  |
| Dr Khalid/Rabia |  |