

## OPHTHALMOLOGY-System Wise 1700-by Sush and Team. 2016

Susmita, Asad, Manu, Saima, Zohaib, Savia, Shanu, Mona, Manisha, Sitara, Samreena, Sami and Komal

**Dear Plabber,**

- This first ever System Wise 1700 document was created thanks to 3 months of daily hard work by the PLAB Skype group 'Unity' which was brought together by Dr Susmita Chowdhury.
- Please ignore the old versions posted by my new skype member Murtaza as he did so without permission.

*The team members were:*

- 📖 **Susmita** (Lead/most ignorant as she is working full time in public health for 13 years)
- 📖 **Asad** (Invaluable in IT and all types of support/the heart of the group)
- 📖 **Manu** (Volunteered to solve more questions/pathologist/amazing genuine person)
- 📖 **Saima** (Most concise clear notes/ photographic memory)
- 📖 **Zohaib** (Great research/a surgeon)
- 📖 **Savia** (Great research/multi-tasker with two little ones)
- 📖 **Shanu** (Very helpful after her March exam for those appearing in June)
- 📖 **Mona** (Great contributor in discussions)
- 📖 **Manisha** (Gyne/great discussion contributor)
- 📖 **Sitara** (Good discussion contributor)
- 📖 **Samreena** (Stayed a shorter time but great)
- 📖 **Sami** (Contributed the most early on but too brilliant for the group/still great friends)
- 📖 **Komal** (Knowledgeable sweet supportive girl)

- The main purpose was to break down the 1700 Q Bank System wise.
- We did our own reliable research for the options (OHCM/Patient info etc.) and concluded these keys below on skype. This can save you 100s of hours of research. But I suggest you also do your own.
- 90% of the document consists of Unity research. We also added information from other circulating documents and they are referenced as Dr Khalid/Dr Rabia (and her Team).
- However, several keys may be 'incorrect' and so please use your own judgment as we take no responsibility. I suggest cross checking with Dr Khalid's latest keys (a few of which are still debatable). Finally decide on your own key.
- Sorry if some members failed to make their answers thorough. The highlights are mostly as per what the team members wanted to highlight. Blank tables to be ignored.
- Note that some 1700 Questions are *missing* from here (when members did not do their share). Questions may not be in order due to merging of documents and there is excess information than required. Read as much as needed.
- This has been circulated by our team as a generous contribution to the Plabbers' success and must not be 'sold'.

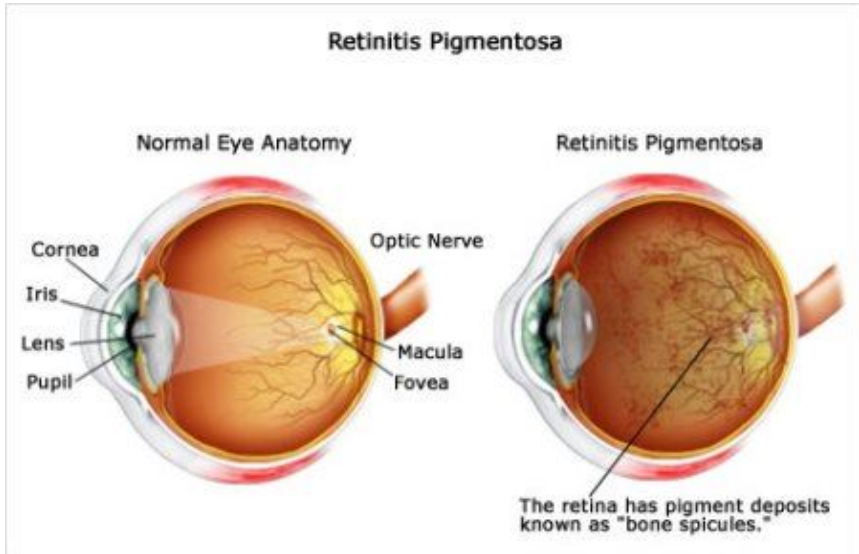
***Good luck and best wishes: Sush and Team***

<b>Q: 78</b>	<p>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?</p> <p>a. B2 agonist b. Corticosteroid c. Diuretic d. Theophylline</p>
<b>Clincher(s)</b>	<b>COPD, multiple drug therapy, deteriorating vision in left eye</b>
A	S/E → trembling (hands), nervous tension, headaches, palpitations, muscle cramps
B	S/E → <b>cataracts</b> and glaucoma
C	Can cause blurred vision (furosemide)
D	S/E → palpitations, headaches, diarrhea, vomit/nausea, arrhythmia, tachycardia
E	
<b>KEY</b>	<b>B</b>
<b>Additional Information</b>	<p><b>Steroid tablets</b></p> <p>Short, occasional courses of steroid tablets taken for no longer than three weeks are very unlikely to cause troublesome side effects.</p> <p>It's sometimes necessary, however, for them to be taken for longer periods. In these cases, you're more likely to develop troublesome side effects, although this is not inevitable.</p> <p>Steroid tablets taken for longer than three weeks can potentially cause:</p> <ul style="list-style-type: none"> <li>• increased appetite – which can potentially lead to weight gain if you find it difficult to control what you eat</li> <li>• <b>acne</b></li> <li>• rapid mood swings and mood changes – such as becoming aggressive, irritable and short-tempered with people</li> <li>• thin skin that bruises easily</li> <li>• muscle weakness</li> <li>• delayed wound healing</li> <li>• a combination of fatty deposits that develop in the face, stretch marks across the body and acne – known as <b>Cushing's syndrome</b></li> <li>• weakening of the bones (osteoporosis)</li> <li>• <b>diabetes</b> (or they may worsen existing diabetes)</li> <li>• high blood pressure</li> <li>• <b>glaucoma and cataracts (eye conditions)</b></li> <li>• <b>stomach ulcers</b> – you may be prescribed an additional medication called a proton pump inhibitor (PPI) to reduce this risk</li> <li>• mental health problems, such as <b>depression</b>, suicidal thoughts, <b>anxiety</b>, confusion and <b>hallucinations</b> – see your GP if you experience any of these</li> </ul> <p><u>Management of COPD (partially)</u></p> <pre> graph TD     A["Controlled oxygen therapy if SaO<sub>2</sub> &lt;88% or PaO<sub>2</sub> &lt;7 kPa Start at 24-28%, aim sats 88-92% (94-98% if no hypercapnia on ABG) Adjust according to ABG, aim PaO<sub>2</sub> &gt;8.0kPa with a rise in PaCO<sub>2</sub> &lt;1.5kPa<sup>25</sup>"] --&gt; B["Steroids iv hydrocortisone 200mg and oral prednisolone 30mg od (continue for 7-14d)"]     B --&gt; C["Antibiotics"]     </pre>

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Reference	<a href="http://patient.info/medicine/furosemide-frusol-lasix">http://patient.info/medicine/furosemide-frusol-lasix</a> 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.

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 (loss of peripheral vision) e. Keratitis
Clincher(s)	<b>30yr old woman, gradual decrease visual acuity</b>
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	<b>D</b>
Additional Information	 <p>The diagram illustrates the difference between a normal eye and an eye affected by Retinitis Pigmentosa. On the left, 'Normal Eye Anatomy' shows a cross-section of a healthy eye with labels for the Cornea, Iris, Lens, Pupil, Optic Nerve, Macula, and Fovea. On the right, 'Retinitis Pigmentosa' shows a cross-section of an eye where the retina is affected. A label points to the retina, stating: 'The retina has pigment deposits known as "bone spicules."'.</p>

## Retinitis pigmentosa

There is generalised retinal pigment degeneration with decreased vision. Patients have difficulty seeing at night

### About

- RP is also the manifestation of several rare neurological diseases
- Usual form is severe and AR but AD inheritance is seen and sex linked too

### Aetiology

- Most cases are due to a defect in the gene for rhodopsin
- More common in males 3:1

### Conditions associated with RP

- Usher syndrome
- Bardet-Biedl syndrome
- Mitochondrial diseases
- Abetalipoproteinaemia
- Refsum disease - elevated serum phytanic acid
- Kearns-Sayre syndrome
- Lowe syndrome

### Clinical

- Night blindness (nyctalopia) progressing to total visual loss (can see well in day but not at night)
- May begins in childhood and as time goes on progresses to tunnel vision
- Appearance on fundoscopy of bony spicules, attenuated vessels and disc pallor

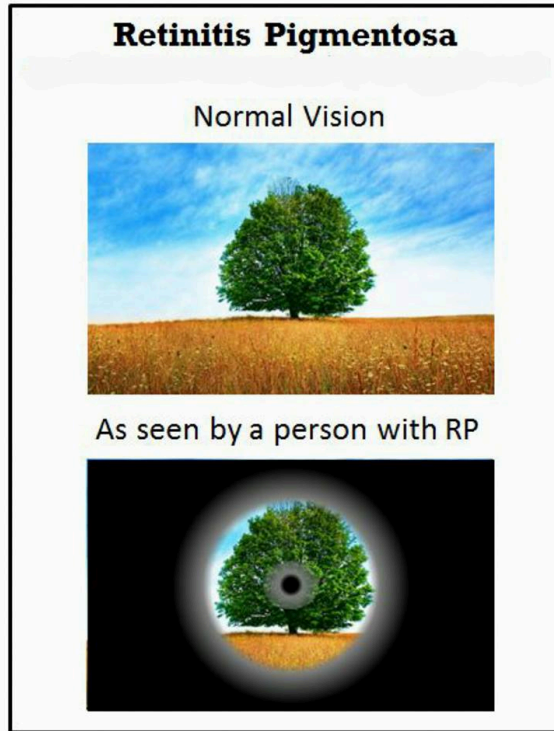
### Investigations

- The electroretinogram signal is diminished - it is normal in Leber's type of optic atrophy
- Perimetry shows tunnel vision

### Management

- End result is often blindness
- Vitamin A has been used to treat with fairly poor results

RP combined with progressive deafness is called Usher syndrome.



**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 mitochondrial 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.

	<p><b>Kearne-Sayre syndrome</b></p> <ul style="list-style-type: none"> <li>• Key features, <b>TRIAD</b> of <ul style="list-style-type: none"> <li>• Ptosis</li> <li>• Chronic progressive external ophthalmoplegia</li> <li>• Heart block</li> </ul> </li> </ul> <p><b>Bassen-Kornzweig syndrome</b></p> <ul style="list-style-type: none"> <li>• Key features, <b>TRIAD</b> of <ul style="list-style-type: none"> <li>• Ataxia</li> <li>• Acanthocytosis (red blood cell abnormality)</li> <li>• Abetalipoproteinemia (fat malabsorption)</li> </ul> </li> <li>• Treatment <ul style="list-style-type: none"> <li>• Vitamins A and E may be beneficial (page 413)</li> </ul> </li> </ul> <p><b>Refsum's syndrome</b></p> <ul style="list-style-type: none"> <li>• Key features, <b>TRIAD</b> of <ul style="list-style-type: none"> <li>• Phytanic acid metabolic defect</li> <li>• Peripheral neuropathy</li> <li>• Palpitations (cardiac arrhythmia)</li> </ul> </li> </ul> <p><b>Usher's syndrome</b></p> <ul style="list-style-type: none"> <li>• Key features, <b>TRIAD</b> of <ul style="list-style-type: none"> <li>• Deafness</li> <li>• Ataxia (vestibular dysfunction)</li> <li>• Neurological abnormalities</li> </ul> </li> </ul> <p><b>Bardet-Biedl syndrome</b></p> <ul style="list-style-type: none"> <li>• Key features, <b>TRIAD</b> of <ul style="list-style-type: none"> <li>• Obesity</li> <li>• Hypogenitalism</li> <li>• Polydactyly</li> </ul> </li> </ul> <p><b>Laurence-Moon syndrome</b></p> <ul style="list-style-type: none"> <li>• Key features, <b>TRIAD</b> of <ul style="list-style-type: none"> <li>• Spastic paraplegia</li> <li>• Hypogenitalism</li> <li>• Mental handicap</li> </ul> </li> </ul>
<b>Reference</b>	
Dr Khalid/Rabia	<p>Retinitis pigmentosa primarily affects the peripheral retina resulting in funnel vision</p> <p>Features</p> <ul style="list-style-type: none"> <li>• night blindness is often the initial sign</li> <li>• funnel vision (the preferred term for tunnel vision)</li> <li>• funduscopy: black bone spicule-shaped pigmentation in the peripheral retina, mottling of the retinal pigment epithelium</li> </ul>



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	<p>Associated diseases</p> <ul style="list-style-type: none"> <li>• Refsum disease: cerebellar ataxia, peripheral neuropathy, deafness, ichthyosis</li> <li>• Usher syndrome</li> <li>• abetalipoproteinemia</li> <li>• Lawrence-Moon-Biedl syndrome</li> <li>• Kearns-Sayre syndrome</li> <li>• Alport's syndrome</li> </ul>
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<b>Q: 121</b>	<p>An 8yo (M) 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?</p> <p>a. Fragile X syndrome b. Prader-willi syndrome c. DiGeorge syndrome d. Marfans syndrome</p>
<b>Clincher(s)</b>	
A	Mental retardation-
B	Short stature and small hands and feet- binge eating- hyperphagia, usually obese,
C	Absence of p3rd and 4rth pharyngeal pouch, thymus, aortic arch, absence of T cells, prone to infections, absent parathyroid- hypocalcemia
D	Tall for his age and chest pain likely due to aortic dissection
E	
<b>KEY</b>	<b>D</b>
Additional Information	<p>Marfan's syndrome is a connective tissue disease with an autosomal dominant inheritance and an incidence of 4-6 per 100 000.</p> <p>People with Marfan's syndrome used to have a life expectancy reduced by 50% but this is now changing because of improved treatment of cardiovascular abnormalities.</p> <p>Typical musculoskeletal features include limbs disproportionately long for the trunk, scoliosis (in particular pectus excavatum or carinatum), and a high-arched narrow palate with laxity of the joints.</p>

## Marfan's syndrome

- AD inherited connective tissue with distinctive appearance and risk of death from aortic disease
- A type 1 fibrillinopath

### Aetiology

- Autosomal dominant disease
- Mutation in the gene for fibrillin 1 on chromosome 15
- Fibrillin 1 is a component of elastin
- Diagnosis is based on clinical criteria

### Clinical

- Tall thin wide arm span - long thin arms and legs called dolichostenomelia
- Arm span to height > 1.05
- Arachnodactyly - long spidery fingers
- Pectus excavatum
- Eyes - Ectopia lentis - lens dislocation upwards, myopia, (fibrillin in suspensory ligament of eye)
- Aortic aneurysm formation and possible dissection (fibrillin in Aorta)
- Mitral valve prolapse - mid-systolic click murmur syndrome
- Aortic regurgitation - early diastolic murmur
- Spontaneous pneumothorax and apical blebs
- Unstable joints, dislocation of the patella, progressive kyphoscoliosis
- Inguinal and femoral herniae

### Differential with Marfanoid Habitus

- Homozygous Homocystinuria
- Lujan-Fryns syndrome
- MEN2B

### Investigations

- Echocardiography to assess mitral valve and Aortic root
- Genetics for FBN1 mutation

### Management

- Prophylactic Beta blockade from early age reduces aortic dilatation
- Surgery for dissection - some may need insertion of a Dacron graft to the aortic valvular ring after excision of a terminally dilated aorta when diameter reaches 5 cm. This also requires reimplantation of the coronary arteries
- Life expectancy approaches that of normals

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

	<ul style="list-style-type: none"> <li>• mental retardation - the IQ is usually in the range 50 to 70</li> <li>• dysregulated temperature control resulting in hypothermia</li> <li>• non-insulin-dependent diabetes</li> <li>• sleep apnoea and cor pulmonale</li> </ul> <p>The <b>DiGeorge syndrome</b> is an example of a selective T-cell deficiency caused by the failure of development of the <b>third and fourth pharyngeal pouches</b>. These pouches give rise to the following structures:</p> <ul style="list-style-type: none"> <li>• thymus</li> <li>• parathyroids</li> <li>• aortic arch</li> <li>• portions of the lips and ears</li> </ul> <p>Consequently, DiGeorge syndrome may present with as immune deficiency state - usually T cells, but sometimes B cells, and also aberrant calcium metabolism, congenital heart disease and abnormal facies.</p> <p>Clinical features relate closely to the structural defects present in the DiGeorge syndrome:</p> <p>Thymic hypoplasia:</p> <ul style="list-style-type: none"> <li>• low T cell counts</li> <li>• susceptibility to mycobacteria, viruses and fungi, for example chronic mucocutaneous candidiasis</li> <li>• compensatory B cell leukocytosis</li> <li>• peripheral lymphocytes do not respond to polyclonal T cell activators</li> <li>• organ specific autoimmune disorders</li> </ul> <p>Absent parathyroid glands:</p> <ul style="list-style-type: none"> <li>• low PTH levels</li> <li>• low plasma calcium</li> <li>• muscle twitching or tetany</li> <li>• basal ganglia calcification and intellectual retardation</li> <li>• hypocalcaemic cataract</li> </ul> <p>Congenital malformations of the great vessels</p> <p>Facial abnormalities</p> <p>Thymic hypoplasia with retention of parathyroid function is termed Nezelof syndrome.</p>
<b>Reference</b>	
Dr Khalid/Rabia	<p>Ans. 1. The key is D. Marfans syndrome.</p> <p>Ans. 2. Cause of severe crushing chest pain may be aortic dissection.</p> <p>Ans. 3. Most common cardiac abnormalities in Marfans syndrome are: dilatation of the aorta and mitral regurgitation.</p> <p>Marfans syndrome diagnosis:</p>

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	<p>Major criteria (diagnostic if &gt;2): Lens dislocation (ectopia lentis) aortic dissection or dilatation; dural ectasia; skeletal features: arachnodactyly (long spidery fingers), armspan &gt; height, pectus deformity, scoliosis, pes planus. Minor signs: Mitral valve prolapse, high-arched palate, joint hypermobility. Diagnosis is clinical.</p> <p><b>DANGER IS AORTIC DISSECTION.</b> Surgery is done when aorta &gt;5cm</p> <p>Can also cause pneumothorax.</p>
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<b>Q: 167</b>	<p>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?</p> <p>a. Reassurance and annual screening only- no blood he,  b. Urgent referral to ophthalmologist- neovascularization  c. Laser therapy  d. Non-urgent referral to ophthalmologist  e. Nothing can be done- macular degeneration</p>
<b>Clincher(s)</b>	<b>Dot and blot haemorrhage , multiple cotton wool spots</b>
A	
B	Proliferative with neovascularization)
C	
D	Pre-proliferative retinopathy so non-urgent referral.
E	
<b>KEY</b>	<b>D</b>
Additional Information	<p><b>Diabetic retinopathy consists of various types:</b></p> <ul style="list-style-type: none"> <li>▷ background diabetic retinopathy</li> <li>▷ diabetic maculopathy</li> <li>▷ pre-proliferative diabetic retinopathy</li> <li>▷ proliferative diabetic retinopathy</li> </ul> <p><b>Diabetics are more prone to other eye disorders, including:</b></p> <ul style="list-style-type: none"> <li>▷ glaucoma</li> <li>▷ cataract</li> </ul>

## treatment principles - diabetic retinopathy

Medical Search

Go

- ▷ co-existing conditions may lead to an acceleration of retinopathy (pregnancy, anaemia, hypertension, renal disease) and these should be treated; treat any hyperlipidaemia

NICE have issued guidance regarding the management of diabetic retinopathy:

- ▷ Eye care for all people with diabetes
  - ▷ maintain good blood pressure control (at or below 140/80 mmHg) (1)
  - ▷ maintain good blood glucose levels (preferably below HbA1c 6.5-7.5%, according to the individual's target) (1)
  - ▷ check visual acuity
  - ▷ refer for specialist opinion if cataracts are interfering with vision or the retina is obscured (1)
  - ▷ NICE guidance states criteria for timing of review for diabetic eye disease (2)
    - ▷ **emergency review by an ophthalmologist is indicated for either:**
      - ▷ sudden loss of vision
      - ▷ rubeosis iridis
      - ▷ pre-retinal or vitreous haemorrhage
      - ▷ retinal detachment
    - ▷ rapid review by an ophthalmologist should be arranged for new vessel formation
    - ▷ ophthalmologist referral, in accordance with the National Screening Committee criteria and timelines if any of these features is present, is indicated if either:
      - ▷ referable maculopathy:
        - ▷ exudate or retinal thickening within one disc diameter of the centre of the fovea
        - ▷ circinate or group of exudates within the macula (the macula is defined here as a circle centred on the fovea, with a diameter the distance between the temporal border of the optic disc and the fovea)
        - ▷ any microaneurysm or haemorrhage within one disc diameter of the centre of the fovea, only if associated with deterioration of best visual activity to 6/12 or worse
      - ▷ referable pre-proliferative retinopathy (if cotton wool spots are present, look carefully for the following features, but cotton wool spots themselves do not define pre-proliferative retinopathy):
        - ▷ any venous beading
        - ▷ any venous loop or reduplication
        - ▷ any intraretinal microvascular abnormalities
        - ▷ multiple deep, round or blot haemorrhages
    - ▷ any unexplained drop in visual acuity.

	<p>Diabetic retinopathy is a microangiopathy affecting the retinal precapillary arterioles, the capillaries and the venules. Diabetic retinopathy has features of microvascular occlusion and leakage.</p> <p>Diabetics have a 20 times greater risk of becoming blind than non-diabetics:</p> <ul style="list-style-type: none"> <li>▷ diabetic retinopathy is the commonest cause of blindness in individuals between the ages of 20 and 65</li> <li>▷ the risk of developing diabetic retinopathy is greater the longer the diagnosis of diabetes</li> <li>▷ good diabetic control slows the development of retinopathy</li> </ul> <p>The early clinical signs of diabetic retinopathy are</p> <ul style="list-style-type: none"> <li>▷ microaneurysms and dot intraretinal hemorrhages <ul style="list-style-type: none"> <li>▷ present in almost all who have had type 1 diabetes for 20 years</li> <li>▷ present in nearly 80 percent of those with type 2 disease for 20 years (1)</li> </ul> </li> <li>▷ microaneurysms are the earliest signs: <ul style="list-style-type: none"> <li>▷ they are outpouchings of the capillaries, which appear as minute, sharply circumscribed, red dots, and are distinguished from haemorrhages by their usual location far from blood vessels and their sharp border</li> <li>▷ many are too small to be visualised with the ophthalmoscope and are apparent only following fluorescein angiography</li> </ul> </li> <li>▷ dot and blot haemorrhages: <ul style="list-style-type: none"> <li>▷ occur within the inner nuclear layer of the retina where cells are so compactly arranged that the (intraretinal) haemorrhage cannot spread</li> </ul> </li> <li>▷ flame shaped haemorrhages: <ul style="list-style-type: none"> <li>▷ in the nerve fibre layer - their shape reflects the nerve fibre distribution</li> </ul> </li> <li>▷ retinal oedema: <ul style="list-style-type: none"> <li>▷ due to capillary leakage - with a predilection for the macula</li> </ul> </li> <li>▷ hard exudates: <ul style="list-style-type: none"> <li>▷ yellow, lipid precipitates which result from resorption of retinal oedema</li> </ul> </li> <li>▷ in general vision is normal; however central vision is variably impaired if changes affect the fovea - note the fovea is capillary free</li> <li>▷ visual loss results mainly from <ul style="list-style-type: none"> <li>▷ macular edema,</li> <li>▷ macular capillary nonperfusion,</li> <li>▷ vitreous hemorrhage, and</li> <li>▷ distortion or traction detachment of the retina (2)</li> </ul> </li> </ul>
<p><b>Reference</b></p>	
<p>Dr Khalid/Rabia</p>	<p><b>Diabetic retinopathy</b> 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.</p> <ul style="list-style-type: none"> <li>• <b>Background retinopathy:</b> Microaneurysms (dots), haemorrhages (blots) and hard exudates (lipid deposits). Refer if near the macula, eg for intravitreal triamcinolone.</li> <li>• <b>Pre-proliferative retinopathy:</b> Cotton-wool spots (eg infarcts), haemorrhages, venous beading. These are signs of retinal ischaemia. Non urgent Refer to a specialist.</li> <li>• <b>Proliferative retinopathy:</b> New vessels form. <b>Needs urgent referral.</b></li> <li>• <b>Maculopathy:</b> (hard to see in early stages). Suspect if acuity. Prompt laser, intra vitreal steroids or anti-angiogenic agents may be needed in macular oedema.</li> </ul>

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<b>Q: 335</b>	<p>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?</p> <p>a. Inhaled salbutamol b. Inhaled steroids c. Aminophylline d. Beta-blockers e. Oral steroids</p>
<b>Clincher(s)</b>	<b>Asthma medication, decreased vision.</b>
A	
B	
C	
D	
E	Correct answer
<b>KEY</b>	<b>E</b>
Additional Information	Prolonged steroid use → Cataract formation
<b>Reference</b>	
Dr Khalid/Rabia	
<b>Q:938</b>	<p>A 33yo female complains of <b>diplopia on upright gaze</b>. Exam: <b>ptosis</b> can be seen. There are no other complains or any significant PMH. What is the most appropriate inv for him?</p> <p>a. Ophthalmoscopy – not in inside of eye- b. Visual field test – does not talk about visual field problem c. TFT – thyroid- lid retraction d. CT – if nerve problem e. Checking red reflex – cataract</p>
<b>Clincher(s)</b>	<b>Ptosis +</b>
A	
B	
C	
D	<p>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.</p> <p><b>3rd nerve palsy</b></p> <p>Remember pneumatic S04LR6(superior oblique by 4<sup>th</sup> nerve and lateral reclusive by 6<sup>th</sup> nerve rest is all supplied by 3<sup>rd</sup> nerve )</p>
E	
<b>KEY</b>	<b>D</b>



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Additional Information	External Ocular Paralysis			
	Muscle	Direction of pull	Result of paralysis	Cranial nerve
	Medial rectus	Medially	Lateral	III
	Superior rectus	Upwards	Downwards	III
	Lateral rectus	Laterally	Medial	VI
	Inferior rectus	Downwards	Upwards	III
	Superior oblique	Down and out	Up and in	IV
	Inferior oblique	Up and out	Down and in	III
Reference				
Dr Khalid/Rabia	Rabia			

Q:997	<p>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?</p> <ol style="list-style-type: none"> <li>Pan retinal photocoagulation – if neovascularisation - DM</li> <li>Corticosteroids – only 2 cond: GCA, Uveitis, MS(optic Neuritis) – never in keratitis</li> <li>Scleral buckling – in retinal detachment</li> <li>Surgical extraction of lens</li> <li>Pressure over eyeball – to relieve embolus</li> </ol>
Clincher(s)	
A	
B	
C	
D	
E	<p><b>Causes of cherry red spot at macula:</b></p> <p><b>Metabolic Storage Diseases:</b></p> <p>Hurler's disease</p> <p>Tay-Sachs disease</p> <p>MPS VII (Sly syndrome)</p> <p>Farber's disease</p> <p>GM1 gangliosidosis</p> <p>Niemann Pick's disease</p> <p>Sandhoff disease</p> <p>Shprintzen-Goldberg syndrome</p> <p><b>Lysosomal Storage Diseases</b></p>

	<p>Congenital Developmental Diseases : Leber's Congenital Amaurosis</p> <p>Hereditary/ Familial:</p> <p>Hallervorden Spatz disease</p> <p>Krabbe disease</p> <p><b>Degenerative:</b> Metachromatic leukodystrophy</p> <p><b>Vascular:</b> Central retinal artery occlusion</p> <p><b>Drugs:</b></p> <p>Quinine toxicity</p> <p>Dapsone toxicity</p> <p><b>Poisoning:</b></p> <p>Carbon monoxide</p> <p>Methanol</p> <p><b>CENTRAL RETINAL ARTERY OCCLUSION</b> (Severe visual loss to finger counting or hand movement)</p> <ul style="list-style-type: none"> <li>- Usually elderly patient -Sudden onset of visual loss</li> <li>-Associated with hypertension and diabetes On examination: plus or minus carotid bruit</li> </ul> <p><b>Fundoscopy:</b> Optic disc is pale due to ischaemia. Cherry red spots on macula. Sometimes cholesterol is visible in the retinal arteries</p> <p><b>Causes:</b> Arteriosclerosis, Emboli Investigations:</p> <ul style="list-style-type: none"> <li>-Slit lamp examination -Fundoscopy (Pale Optic Disc) -Carotid Doppler- If carotid bruit</li> </ul>
<b>KEY</b>	<b>E</b>
Additional Information	<p><b><u>Reason:</u></b> Presentaion in Central Retinal Artery occlusion is painless loss of vision unilaterally over a few seconds.Can be a HX of Amaurosis Fugax.</p> <p><b><u>Exam:</u></b>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</p> <p><b><u>Management:</u></b> Presentation within 90-100 min Occular massage can be tried.</p> <p><b><u>Paracentesis and acetazolamide to reduce intraocular pressure</u></b></p> <p><b><u>Sublingual isosorbide dinitrate.</u></b></p> <p><b><u>Oral pentoxifyphylline</u></b></p>

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<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:1003</b>	<p>A 30yo woman presents with acute headache (acute close angle). She complains of seeing halos especially at night (GLAUCOMA key). What is the single most likely defect?</p> <ol style="list-style-type: none"> <li>Paracentral scotoma – open angle glaucoma first symptom</li> <li>Monocular field loss – MS, amorgas fugax</li> <li>Tunnel vision – closed angle glaucoma, rentinitis pigmentation, macular degeneration</li> <li>Central scotoma – in macular degeneration, optic neuritis</li> <li>Cortical blindness – stroke, (homonymous heminopia</li> </ol> <p>PITS</p> <p>Parietal- inferial quandri</p> <p>Temporal – superior quandrianopia (Eye)</p>
<b>Clincher(s)</b>	<b>Halos at night</b>
A	
B	
C	
D	
E	
<b>KEY</b>	<b>C</b>
Additional Information	<p><u><b>This is glaucoma</b></u></p> <p><b>paracentral scotoma first, f/b a SEIDEL'S scotoma, f/b an arcuate and a double arcuate scotoma and finally</b></p> <p><b>a tunnel vision leading to blindness.</b></p> <p><b>HALOES:</b></p> <p><u>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:</u></p> <p><u>Excessive formation of tears.</u></p> <p><u>Oedema of the corneal epithelium from any cause (eg, contact lens</u></p>

	<p><u>overwear).</u></p> <p><u>Corneal dystrophies in their later stages.</u></p> <p><u>Chronic open-angle glaucoma.</u></p> <p><u>Early cataracts (glare of headlights making nighttime driving impossible).</u></p> <p><u>Pigment dispersion syndrome.</u></p> <p><u>Vitreous opacities.</u></p> <p><u>Drugs (eg, digitalis and chloroquine).</u></p>
<b>Reference</b>	NHS
Dr Khalid/Rabia	

<b>Q:1010</b>	<p>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?</p> <ol style="list-style-type: none"> <li>Paracentral scotoma</li> <li>Mono-ocular field loss (in MS)</li> <li>Homonymous upper quadrantanopia – temporal</li> <li>Central scotoma – optic neuritis</li> <li>Homonymous lower quadrantanopia – parietal</li> </ol>
<b>Clincher(s)</b>	<b>MS with visual problems</b>
A	Same as D for explanation see below
B	<p><b>Reason :</b> 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.</p> <p><b>This is also called amaurosis fugax</b></p> <p><b>Ocular causes include:</b></p> <p>Iritis</p> <p>Keratitis</p> <p>Blepharitis</p> <p>Optic disc drusen</p> <p>Posterior vitreous detachment</p> <p>Closed-angle glaucoma</p>

	<p>Transient elevation of intraocular pressure  Intraocular hemorrhage  Coloboma  Myopia  Orbital hemangioma  Orbital osteoma  Keratoconjunctivitis sicca  Neurologic origin      Edit  Neurological causes include:</p> <p><b>Optic neuritis</b>  Compressive optic neuropathies  And pappilodema</p>
C	<p>upper homonymous quadrantanopia describes the loss of the same upper quadrant from each visual field.</p> <p>Upper homonymous quadrantanopias are usually caused by damage to the optic radiation as it passes through the temporal lobes.</p> <p>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.</p> <p><b>Left superior quadrantanopia can result from damage to the right hemisphere's lateral geniculate nucleus (LGN)</b>, 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). <b>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</b> (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.</p>

D	<p align="center"><b>Central Visual Field Defects</b></p> <p align="center">Conditions Causing central visual field defects</p> <ul style="list-style-type: none"> <li>▪ Optic neuritis</li> <li>▪ Age related Macular degeneration</li> <li>▪ Optic atrophy</li> <li>▪ Macular hole/lesions</li> <li>▪ Myopic maculopathy</li> <li>▪ Occipital cortex lesions</li> <li>▪ Stroke and trauma</li> <li>▪ Central serous retinopathy</li> <li>▪ Best's and Vitelliform diseases</li> <li>▪ Cone dystrophies</li> <li>▪ Berlin's edema</li> <li>▪ Macular Coloboma</li> <li>▪ Drug e.g. ethanol, lead, methanol, syphilis,</li> <li>▪ Glaucoma</li> </ul> <p align="right">6</p>
E	<p>A lower homonymous quadrantanopia describes the loss of the same upper quadrant from each visual field.</p> <p>Lower homonymous quadrantanopias are usually caused by damage to the optic radiation as it passes through the parietal lobes.</p> <p>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.</p> <p>Homonymous inferior quadrantanopia may originate from tumors, vascular lesions, or infection</p>
<b>KEY</b>	<b>B</b>
Additional Information	<p><b>MS causes</b> 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)</p> <p><b>colour blindness ,eye pain; usually made worse when moving the eyelashes of light when moving the eye</b></p> <p><b>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.</b></p> <p><b>Other visual problems</b> that can occur in MS include:</p> <p><b>double vision</b></p>



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	<b>eye pain in both eyes</b>
	<b>involuntary eye movements (usually from side to side), known as nystagmus</b>
<b>Reference</b>	GP note book ,NHS
Dr Khalid/Rabia	Rabia

<b>Q:1022</b>	A 39yo male presents with visual symptoms. Ophthalmoscopy shows Papilloedema (I. Which anatomical site is most likely to be affected? a. Optic nerve - MS b. Optic disc – papilloedema c. Optic radiation d. Occulomotor nerve – no nerve symptoms e. Optic chiasma- bitemp hemianopia
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	<b>B</b>
Additional Information	<b><u>Optic disc swelling can be caused by a number of conditions; papilloedema relates more specifically to optic disc swelling secondary to raised intracranial pressure.</u></b>  <b><u>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)</u></b>
<b>Reference</b>	
Dr Khalid/Rabia	Rabia

<b>Q:1038</b>	A pt with flame shaped hemorrhage on long term tx with nifedipine. What is the single most likely dx? a. Macular degeneration -
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	<p>b. HTN retinopathy (flame shaped hgs)</p> <p>c. MS</p> <p>d. DM background – cotton wool spots and hard exudates</p> <p>e. Proliferative DM retinopathy – neovascularisation</p> <p>f. SLE</p>
<b>Clincher(s)</b>	<b>Treatment on nifedipine and flame shaped haemorrhages</b>
A	<p><b>DRY AGE-RELATED MACULAR DEGENERATION</b></p> <p>Risk factors: Increasing age, smoking, alcohol and female sex.</p> <p><b>Symptoms:</b></p> <ul style="list-style-type: none"> <li>-Gradual onset of decreased vision, initially to read and recognise faces due to loss of central field of vision. -Central Scotoma (walking into desks)</li> <li>-It is due to photoreceptors</li> <li>-<b>Patient sees wavy lines</b> -</li> <li>-Macula has pigmentation geographically</li> <li>-Bumps into objects -30% inheritance</li> </ul> <p><b>Investigation: Fundoscopy</b> or slit lamp examination you see a large area of geographical atrophy at the macula and pigmentation.</p> <p><b>Treatment</b> is nothing just to reduce age related risk factors</p>
B	On fundoscopy grade 3 shows flame shaped haemorrhages
C	<p>They causes symptoms of <b>optic neuritis</b> like blurred vision,fraying of vision,pain and vision loss gets worse before it gets better for 4-12 weeks</p> <p>Double vision, uncontrolled eye moments occur</p> <p>Treated with steroids</p>
D and e explanation	<p><b>Retinopathy Pathogenesis:</b> microangiopathy in capillaries, precapillary arterioles and venules causes occlusion ± leakage. Vascular occlusion causes ischaemia ± new vessels in the retina, optic disc, and iris, ie <b>proliferative retinopathy</b>. New vessels can bleed (<b>vitreous haemorrhage</b>). Retraction of fibrous tissue running with new vessels heightens risk of retinal detachment. Occlusion also causes <b>cotton wool spots</b> (ischaemic nerve fibres). Vascular leakage: As pericytes are lost, capillaries bulge (<b>microaneurysms</b>) and there is oedema &amp; hard exudates (lipoprotein &amp; lipid filled macrophages). Rupture of microaneurysms at the nerve fibre level causes <b>flame shaped haemorrhages</b>; when deep in the retina, <b>blot haemorrhages</b> 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. <b>Non-proliferative diabetic retinopathy (NPDR)</b> is rated as mild, moderate or severe depending on the degree of ischaemia. Signs comprise <b>microaneurysms</b> (seen as 'dots'), <b>haemorrhages</b> (flame shaped or 'blots') <b>hard exudates</b> (yellow patches), engorged tortuous veins, cotton wool spots, large blot haemorrhages (the latter 3 are signs of significant ischaemia). NPDR can progress to sight-threatening proliferative retinopathy. Proliferative diabetic retinopathy</p>

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	<p>(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 significantly threaten vision (clinically significant macular oedema). It can exist with otherwise mild retinopathy. <input type="checkbox"/> Refer those with maculopathy, severe NPDR, or proliferative retinopathy urgently for assessment and treatment (eg photocoagulation) to protect vision.</p> <p><b>Treatment</b> Good control of diabetes prevents new vessels forming. 'Metabolic memory' effects mean that early good control of diabetes with insulin pays dividends later. 124 Pregnancy, dyslipidaemia, BP, renal disease, smoking, and anaemia may accelerate retinopathy. <b>Photocoagulation</b> by laser is used to treat both maculopathy (focal or grid) and proliferative retinopathy (panretinal). <b>Intravitreal triamcinolone</b> and anti-VEGF drugs (p439) are used with laser to treat diabetic macular oedema. See figs 1 &amp; 2. If vitreous haemorrhage is massive and does not clear, <b>vitrectomy</b> may be needed.</p>
f	SLE causes mainly inflammation conjunctivitis and episcleritis
<b>KEY</b>	<b>B</b>
Additional Information	<p>pt is on Ca channel blocker indicating he is hypertensive.</p> <p>Fundoscopy findings in hypertensive retinopathy:</p> <p>Grade 1: tortuous arteries with thick shiny walls, silver copper wiring</p> <p>Grade 2: AV nicking</p> <p>Grade 3: flame hemorrhages, dot and blot, hard soft exudates</p> <p>Grade 4: papilledema</p> <p>Rx. Control Bp</p>
<b>Reference</b>	
Dr Khalid/Rabia	OHCM

<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	
<b>Clincher(s)</b>	
A	

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B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:635</b>	<p>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?</p> <p>a. Acute conjunctivitis b. Acute dacrocystitis c. Acute iritis d. Retrobulbar neuritis e. Scleritis</p>
<b>Clincher(s)</b>	
A	Acute Conjunctivitis
B	Acute Dacryocystitis
C	Acute Iritis
D	Retrobulbar Neuritis
E	Scleritis
<b>KEY</b>	
Additional Information	<p><b>Ohcs- 435</b> <u>Acute dacryocystitis</u> (is acute inflammation of the tear sac which is located medial to the medial canthus. This may spread to surrounding tissues</p>

(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 inflamed, and the hyperaemic vessels may be moved over the sclera, by gentle pressure on the globe. Acuity, pupillary responses, and corneal lustre are unaffected. 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 ciprofloxacin 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 inflammation will involve the ciliary body, the best term is anterior uveitis, but note that anterior and posterior components may be affected 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 affects 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 inflammation (disrupts flow of aqueous ( } glaucoma } adhesions between iris & lens).

	<p>Drops: 0.5–1% prednisolone/2h, to ↓ inflammation (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 inflammation.</p> <p><i>Intravitreal and biological agents</i> show promise eg anti-TNF and anti-CD20 if HLAB27+ve (most with anterior uveitis are).<sup>55</sup> Adalimumab has a role.<sup>56</sup></p> <p><b>Types &amp; causes of uveitis</b></p> <p><b>Anterior uveitis:</b></p> <ul style="list-style-type: none"> <li>• Ank. spond.; Still's<sup>2</sup>,</li> <li>• Sarcoid; Behcet's, etc<sup>3</sup></li> <li>• Crohn's/UC; Reiter's<sup>2</sup></li> <li>• Herpes, TB, syphilis, HIV</li> </ul> <p><b>Intermediate uveitis:</b></p> <ul style="list-style-type: none"> <li>• MS; lymphoma; sarcoid</li> </ul> <p><b>Posterior &amp; panuveitis:</b></p> <ul style="list-style-type: none"> <li>• Herpes simplex + zoster</li> </ul> <p>toxoplasmosis; TB; CMV; endophthalmitis</p> <ul style="list-style-type: none"> <li>• Lymphoma; sarcoidosis</li> <li>• Behcet's</li> </ul>
<b>Reference</b>	
Dr Khalid/Rabia	<p><b>b. Acute dacryocystitis</b></p> <p>Dacryocystitis is infection of the lacrimal sac</p> <p>Features</p> <ul style="list-style-type: none"> <li>• watering eye (epiphora)</li> <li>• swelling and erythema at the inner canthus of the eye</li> </ul> <p>Management is with systemic antibiotics. Intravenous antibiotics are indicated if there is associated periorbital cellulitis</p>

<b>Q:653</b>	<p>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?</p> <p>a. CRAO b. CRVO c. Branch RAO d. Branch RVO e. Circumciliary vein occlusion</p>
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<b>Clincher(s)</b>	
A	<p><b>Central retinal artery occlusion</b></p> <p>There is dramatic visual loss within seconds of occlusion. In 90% acuity is finger counting or worse. An afferent pupil defect (p424) appears within seconds and may precede retinal changes by 1h. The retina appears white, with a cherry red spot at the macula (figs 1-2, p436). Exclude temporal arteritis. Occlusion is often thrombo-embolic (clot, tumour, infective<sup>etc</sup>). Look for signs of atherosclerosis (bruits; BP↑), heart valve disease, diabetes, smoking, or lipidst.<sup>72</sup> ▶▶ If seen within 6h of onset aim is to increase retinal blood flow by reducing intraocular pressure by ocular massage, surgical removal of aqueous from the anterior chamber or the use of antihypertensive treatment. NB: fluorescein angiography may show branch retinal artery occlusion.<sup>73</sup> Hyperbaric oxygen has been tried (~70% get improved acuity).<sup>74</sup></p> <p>If a single branch of the retinal artery is occluded, the retinal and visual changes relate only to the part of the retina supplied.</p>
B	<p><b>Retinal vein occlusions: central or branch vein?</b></p> <p><b>Central retinal vein occlusion (crvo)</b> Incidence increases with age. It is commoner than arterial occlusion. Causes/associations: arteriosclerosis, BP↑, diabetes and polycythaemia; glaucoma (all types). If the whole central retinal vein is thrombosed, there is visual loss (eg acuity reduced to finger counting). It is less sudden than central retinal artery occlusion. Visual loss may be perceived as sudden by the patient but the mechanism is of visual loss is due to the development of ischaemia and macular oedema.</p> <p>crvo is divided into non-ischaemic and ischaemic (with cotton wool spots, swollen optic nerve, macular oedema, and risk of neovascularization; hence need for follow-up). Non-ischaemic forms have better acuity (even 6/6) and prognosis (signs are less dramatic too). But this can convert to the ischaemic form in 30%; hence the need for follow-up. A fundus fluorescein angiogram is used to determine the degree of ischaemia and pan-retinal photocoagulation is given to prevent or treat neovascularization. Unfortunately even if the macular oedema resolves anatomically visual prognosis is poor. Aim to prevent rubeotic glaucoma and a painful eye (beracizumab and ranbizumab (Lucentis® (p439)) can treat the macular oedema),<sup>75</sup> as can lasers and dexamethasone intravitreal implants.<sup>NICE 2011</sup></p>
C	<p><b>Branch retinal vein occlusion Signs: Unilateral visual loss and fundal appearances</b></p> <p>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</p>
D	<p><b>Diagnosis and differential diagnosis</b> Other causes of sudden loss of vision:</p> <ul style="list-style-type: none"> <li>• Retinal detachment (p444)</li> <li>• Acute glaucoma (painful, p430)</li> <li>• Migraine.</li> </ul> <p>Stroke patients may complain of monocular blindness but visual field testing will usually reveal a homonymous hemianopia. Sudden bilateral visual loss is</p>

	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	<p>Sudden painless loss of vision  Urgent help is needed in: retinal artery occlusion of &lt;6h; any sudden visual loss of &lt;6h if the cause is unknown, or giant cell arteritis (GCA). <i>5 questions:</i> • Headache associated? (GCA, ESR) (do this test urgently in all cases &gt;50yrs old) • Eye movements hurt? (optic neuritis) • Lights/flashes preceding visual loss? (detached retina) • Like a curtain descending? amaurosis fugax  may precede permanent visual loss, eg from emboli/GCA) • Poorly controlled DM: fig 1 shows vitreous haemorrhage (bottom left) from new vessels (top right). <i>Check:</i> Acuity, pupil reaction, fundi. Then refer</p> <p><b>Anterior ischaemic optic neuropathy (AION)</b> The optic nerve is damaged if posterior ciliary arteries are blocked by inflammation or atheroma. <i>Fundoscopy:</i> pale/swollen optic disc. <b>Arteritic AION (giant cell arteritis):</b> The other eye is at risk until steroids are given. <i>Symptoms:</i> malaise, jaw claudication (chewing pain) tender scalp and temporal arteries (thickened absent pulses), neck pain. <i>Tests:</i> ESR (&gt;47) &amp; CRP (&gt;2.45mg/dL) preferably before steroids; temporal artery biopsy within 1 week of starting prednisolone; may miss affected 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 &gt;1yr. <b>67,68 Nonarteritic AION:</b> Associations: BP; lipids; DM, smoking. Treating these protects vision in the other eye. Histology: necrosis &amp; apoptosis at the photoreceptor level</p> <p><b>Vitreous haemorrhage (VH)</b> 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 floaters, (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 reflex 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.</p> <p><b>Subacute loss of vision Optic neuritis (fig 2)</b> Unilateral loss of acuity occurs over hours or days. Colour vision is affected (dyschromatopsia): reds appear</p>

less red, 'red desaturation'—and eye movements hurt. The pupil shows an afferent 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 deficiency. **?** High-dose methyl prednisolone for 72h (250mg/6h IV), then prednisolone (1mg/kg/d PO) for 11 days may briefly delay onset of MS (no change to long-term disability).

**Transient visual loss** **?** Always think of vascular causes, such as platelet–fibrin/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 likely to 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 inflammatory disorders affecting the uvea may also affect 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 & fig 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 fluid 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 flecks in the retina. This condition was the first to be treated with embryonal stem cells.

**Tobacco–alcohol amblyopia** From cyanide radicals, when smoking and alcohol excess are combined. Signs: optic atrophy (fig 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 correlate 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 bisulphide.

**Other causes:** Leber's optic atrophy (p648), multiple sclerosis (MS), syphilis, external pressure on the nerve (intraorbital or intracranial tumours, Paget's disease affecting 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).

	<p><b>Typical causes</b></p> <ul style="list-style-type: none"> <li>• Cataract</li> <li>• Macular degeneration</li> <li>• Glaucoma (p440)</li> <li>• Diabetic retinopathy</li> <li>• Hypertension (p448)</li> <li>• Optic atrophy (below)</li> <li>• Slow retinal detachment</li> </ul>
<b>Reference</b>	
Dr Khalid/Rabia	<p><b>a. CRAO</b> pale optic disc, cherry red spot on macula</p> <p>The most common causes of a sudden painless loss of vision are as follows:</p> <ul style="list-style-type: none"> <li>• ischaemic optic neuropathy (e.g. temporal arteritis or atherosclerosis)</li> <li>• occlusion of central retinal vein</li> <li>• occlusion of central retinal artery</li> <li>• vitreous haemorrhage</li> <li>• retinal detachment</li> </ul> <p>Ischaemic optic neuropathy</p> <ul style="list-style-type: none"> <li>• may be due to arteritis (e.g. temporal arteritis) or atherosclerosis (e.g. hypertensive, diabetic older patient)</li> <li>• due to occlusion of the short posterior ciliary arteries, causing damage to the optic nerve</li> <li>• altitudinal field defects are seen</li> </ul> <p>Central retinal vein occlusion</p> <ul style="list-style-type: none"> <li>• incidence increases with age, more common than arterial occlusion</li> <li>• causes: glaucoma, polycythaemia, hypertension</li> <li>• severe retinal haemorrhages are usually seen on fundoscopy</li> </ul> <p>Central retinal artery occlusion</p> <ul style="list-style-type: none"> <li>• due to thromboembolism (from atherosclerosis) or arteritis (e.g. temporal arteritis)</li> <li>• features include afferent pupillary defect, 'cherry red' spot on a pale retina</li> </ul> <p>Vitreous haemorrhage</p> <ul style="list-style-type: none"> <li>• causes: diabetes, bleeding disorders</li> <li>• features may include sudden visual loss, dark spots</li> </ul> <p>Retinal detachment</p> <ul style="list-style-type: none"> <li>• features of vitreous detachment, which may precede retinal</li> </ul>

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	detachment, include flashes of light or floaters
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<b>Q:654</b>	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 (CA glaucoma- eye is hard) b. Relieve pain with aspirin c. 100% oxygen (cluster headache) d. CT (cerebral/neuro logical) e. Relieve pain with sumatriptan (migraine)
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
<b>Additional Information</b>	In <i>acute angle closure glaucoma</i> 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 suffered from migraine. OHCS 479
<b>Reference</b>	
Dr Khalid/Rabia	<p><b>a. Measure IOP</b></p> <p>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</p> <p>Features</p> <ul style="list-style-type: none"> <li>• severe pain: may be ocular or headache</li> <li>• decreased visual acuity</li> <li>• symptoms worse with mydriasis (e.g. watching TV in a dark room)</li> <li>• hard, red eye</li> <li>• haloes around lights</li> <li>• semi-dilated non-reacting pupil</li> <li>• corneal oedema results in dull or hazy cornea</li> <li>• systemic upset may be seen, such as nausea and vomiting and even abdominal pain</li> </ul>



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	<p>Management</p> <ul style="list-style-type: none"> <li>• urgent referral to an ophthalmologist</li> <li>• management options include reducing aqueous secretions with acetazolamide and inducing pupillary constriction with topical pilocarpine</li> </ul>
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<b>Q:737</b>	<p>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?</p> <p>a. Cataract- posterior capsule cataract</p> <p>b. Glaucoma - notjing</p> <p>c. Retinal detachment- flashes</p> <p>d. Iritis- painful</p> <p>e. GCA- unilateral</p>
<b>Clincher(s)</b>	
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<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	<p>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.</p> <p>Many causes of gradual visual loss can be diagnosed on history and examination alone with only the most basic additional investigations. For any</p>

patient with a gradual loss in visual acuity, the following protocol should be followed to reach to an initial diagnosis.

### Assessment

#### History

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).

	<ul style="list-style-type: none"> <li>○ If the opacity 'moves' in the same direction, it is anterior to the lens (cornea).</li> <li>○ If the opacity 'moves' in the opposite direction, it is posterior to the pupil (posterior lens or vitreous).</li> <li>○ If the media is clear, it is more likely to be a retinal or optic nerve disorder.</li> <li>○ If there is a normal red reflex, take a good look at the fundus.</li> <li>○ Do a functional testing of visual field, pupils, optic nerve and macula.</li> <li>○ Use an Amsler grid to look for distortion of straight lines - if present, this indicates serious macular pathology and needs urgent referral</li> </ul> <p><b>Interpreting the findings - some clinical patterns</b></p> <ul style="list-style-type: none"> <li>• Loss of vision in one eye - implies the problem is in the eye itself or in the optic nerve <i>before</i> it reaches the optic chiasm.</li> <li>• Loss of vision in both temporal fields (bitemporal hemianopia) - occurs from lesions compressing the optic chiasm (eg, pituitary tumour or craniopharyngioma).</li> <li>• Loss of left or right visual field (homonymous hemianopia) - implies the lesion is somewhere between the optic chiasm and the occiput.</li> <li>• Chronic glaucoma tends to cause tunnel vision (loss of peripheral visual field).</li> <li>• Macular degeneration causes loss of central vision (central scotoma) and may cause distortion of straight lines.</li> <li>• <b>Cataracts</b> - 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.</li> </ul>
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<b>Q:807</b>	<p>807. A 52yo man has a <b>painful, red, photophobic</b> right eye with slightly blurred vision and watering for 3 days. He has had no such episodes in the past. On <b>slit lamp examination there are cells and flare in the ant chamber</b> (ant uveitis) and pupil is sluggish to react. What is the single most appropriate clinical dx?</p> <ol style="list-style-type: none"> <li>Acute close-angle glaucoma- sudden</li> <li>Acute conjunctivitis- not photophobia</li> <li>Acute dacrocystitis- inflammation of lacrimal sac</li> <li>Acute iritis- painful, photophobia</li> </ol>
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	e. Corneal foreign body- no mention of trauma
<b>Clincher(s)</b>	
A	
B	
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E	
<b>KEY</b>	<b>D</b>
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	<p>KEY is D This is a clear Picture of Acute Iritis. Iritis forms the part of anterior Uveitis.</p> <p><b>Anterior uveitis</b> 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</p> <p><b>SYMPTOMS AND SIGNS:</b> 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.</p> <p><b>FINDINGS on SLIT LAMP EXAMINATION:</b> The diagnosis of iritis is confirmed by examining the eye with a slit lamp (a special microscope designed for <a href="#">eye exams</a>). Your ophthalmologist can see cells (white <a href="#">blood cells</a>) and flare (particles of protein) in the fluid that is produced in the eye.</p> <p>Two other <a href="#">physical exam</a> findings aid your eye doctor in diagnosing iritis. They include:</p> <ul style="list-style-type: none"> <li>• Topical anesthetics do not relieve the pain associated with iritis.</li> <li>• 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</li> </ul>

<b>Q:</b>	
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<b>Clincher(s)</b>	
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<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	
<b>Clincher(s)</b>	
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<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	
<b>Clincher(s)</b>	
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<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	

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<b>Clincher(s)</b>	
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B	
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D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:835</b>	<p>A 30yo man comes with hx of early morning back pain and stiffness. Exam: red eyes. What is the single most appropriate option?</p> <p>a. Iris b. Ciliary body c. Cornea d. Conjunctivitis e. Sclera</p>
	Scleritis painful, but not episcleritis
<b>Clincher(s)</b>	<b>Young man,morning backache,stiff ness and red eye</b>
A	
B	
C	
D	
E	
<b>KEY</b>	<b>A</b>
Additional Information	<p><b>Ankylosing Spondylitis:</b> 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.</p> <p><b>Presentation</b></p> <ul style="list-style-type: none"> <li>• Symptoms may be subtle in early stages or mild disease, with an insidious onset over several months to years.</li> <li>• AS usually presents before the age of 30 years.</li> <li>• Most patients have mild chronic disease or intermittent flares with periods of remission.</li> <li>• Systemic features are common. Fever and weight loss may occur during periods of active disease. Fatigue is also prominent.</li> <li>• Morning stiffness is characteristic.</li> <li>• <b>Inflammatory back pain:</b> <ul style="list-style-type: none"> <li>◦ Often improves with moderate physical activity.</li> <li>◦ Unlike mechanical back pain, patients often experience stiffness and pain which awaken them in the early morning hours.</li> </ul> </li> </ul>

- 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<sup>[5]</sup>**
  - **Eye involvement**
  - Acute anterior uveitis occurs in 20-30% of patients. Of all

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	<p>patients presenting with acute anterior uveitis, a third to a half have or will go on to develop AS.</p> <ul style="list-style-type: none"> <li>○ Acute anterior uveitis presents with an acutely painful red eye and severe photophobia and requires emergency treatment to prevent visual loss.</li> <li>○ There could be cardiopulmonary, neurological And Renal involvement too.</li> <li>○</li> </ul> <p><b>Treatment:</b> 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</p>
<b>Reference</b>	Patient.info
Dr Khalid/Rabia	

<b>Q:841</b>	<p>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?</p> <ol style="list-style-type: none"> <li>a. Iris</li> <li>b. Ciliary body</li> <li>c. Cornea</li> <li>d. Conjunctivitis</li> <li>e. Sclera</li> <li>f. Lichen planus</li> </ol>
	Red painful: scleritis (epi- is not) Rx- steroids
<b>Clincher(s)</b>	<b>36year old ,morning stiffness of small joints and red painfull eye</b>
A	
B	
C	
D	
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.
<b>KEY</b>	<b>E</b>
Additional Information	<p><b>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.</b></p> <p><b>Scleritis may be the initial or only presenting clinical manifestation of these potentially lethal disorders. The correct and rapid diagnosis and the</b></p>



	<p><b><u>appropriate systemic therapy can halt the relentless progression of both ocular and systemic processes, thus preventing destruction of the globe and prolonging survival</u></b></p> <p><b><u>The extra-articular manifestations of rheumatoid arthritis affect the following systems:</u></b></p> <ul style="list-style-type: none"> <li>• <u>respiratory</u></li> <li>• <u>haematological</u></li> <li>• <u>neurological</u></li> <li>• <u>lymphoreticular</u></li> <li>• <u>ocular</u></li> <li>• <u>cardiac</u></li> <li>• <u>systemic</u></li> </ul> <p>The ocular manifestations of rheumatoid arthritis include:</p> <ul style="list-style-type: none"> <li>• keratoconjunctivitis sicca</li> <li>• episcleritis</li> <li>• scleritis and scleromalacia perforans</li> <li>• iritis</li> <li>• tenosynovitis of the ocular muscles</li> <li>• Sjogren's Syndrome</li> </ul> <p><b>Scleritis</b> (Vasculitis of the sclera.)<sup>59</sup> Rarely, the sclera itself is inflamed and pain is significant. There is generalized inflammation with oedema of the conjunctiva and scleral thinning (if necrotizing, globe perforation is a risk). Association: connective tissue disorders; infections. Refer to a specialist. Acuity may be ↓ (esp. if associated with ocular hypertension, a systemic disease, uveitis, or there is posterior scleritis). Tests: ESR, ANCA for AAV <sup>antineutrophil cytoplasmic antibody-associated vasculitis</sup> (Wegener's *+polyangiitis). Most need oral steroids/immunosuppression (ciprofloxacin, topical fortified amikacin, and vancomycin drops if staphylococcal).<sup>60,61</sup></p>
<b>Reference</b>	Gp notebook
Dr Khalid/Rabia	

<b>Q:842</b>	<p>A 23yo man comes with 2d hx of sticky greenish discharge from the eyes with redness. What is the single most appropriate option?</p> <p>a. Iris</p> <p>b. Ciliary body</p> <p>c. Cornea</p> <p>d. Conjunctivitis</p> <p>e. Sclera</p>
<b>Clincher(s)</b>	<b>Sticky greenish discharge from eyes and redness</b>
A	
B	

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C	
D	
E	
<b>KEY</b>	<b>D</b>
Additional Information	<p><b>Conjunctivitis</b> The conjunctiva is red and inflamed, and the hyperaemic vessels may be moved over the sclera, by gentle pressure on the globe. Acuity, pupillary responses, and corneal lustre are unaffected. 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 ciprofloxacin is spreading, and, to a lesser extent, to gentamicin.<sup>62</sup> 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). <i>Rx for allergic conjunctivitis:</i> Try antihistamine 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.<sup>63</sup></p>
<b>Reference</b>	Ohcs 432
Dr Khalid/Rabia	

<b>Q:870</b>	<p>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?</p> <p>a. Iris b. Ciliary body c. Ant chamber d. Post chamber e. Cornea</p>
<b>Clincher(s)</b>	<b>Visual acuity checked,12hr later severe pain and redness in eye</b>
A	
B	
C	
D	
E	
<b>KEY</b>	<b>C</b>
Additional Information	.
<b>Reference</b>	
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

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	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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<b>Q:917</b>	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
<b>Clincher(s)</b>	<b>Fullness in the ear, recurrent vomiting, tinnitus</b>
A	
B	
C	
D	
E	
<b>KEY</b>	<b>A</b>
<b>Additional Information</b>	<p><u><b>MENIERES DISEASE (source : nhs)</b></u></p> <p><u><b>Its a case of menieres disease.</b></u></p> <p><u><b>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.</b></u></p> <p><u><b>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.</b></u></p> <p><u><b>Ménière's disease most commonly affects people aged 20-60 and it's thought to be slightly more common in women than men.</b></u></p> <p><u><b>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.</b></u></p> <p><u><b>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</b></u></p>
<b>Reference</b>	

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Dr Khalid/Rabia	
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<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

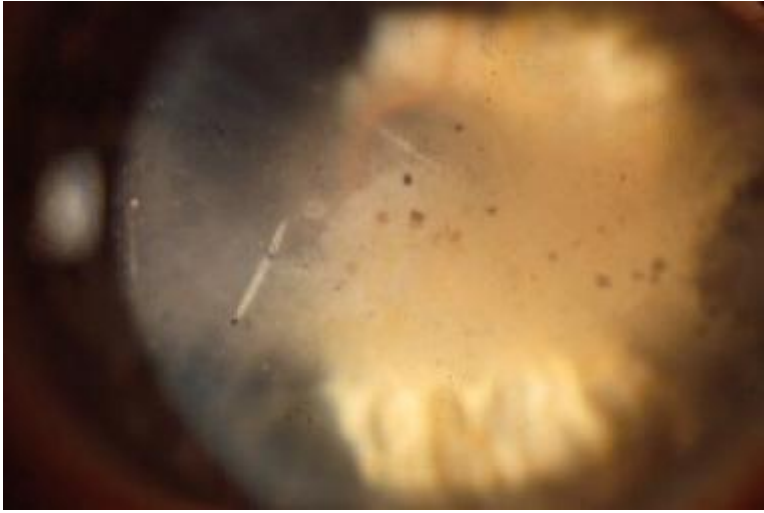
<b>Q: 1049</b>	A 32yo man suffering from MS presents with blurring of vision. Ophthalmoscopy shows pallor of the optic disc. Which anatomical site is most likely to be affected? a. Optic nerve b. Optic disc- in ICP, MS, c. Optic radiation d. Trigeminal e. Oculomotor nerve
<b>Clincher(s)</b>	<b>MS!</b>
A	
B	
C	
D	
E	

KEY	A
Additional Information	<p><b><u>Optic disc pallor shows optic nerve damage.</u></b></p> <p><b><u>Optic neuritis occurs in MS patients. Pain on eye movement, rapid and decreased central vision</u></b></p> <p>Optic neuritis usually affects one eye. Symptoms might include:</p> <ul style="list-style-type: none"> <li>• <b>Pain.</b> 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.</li> <li>• <b>Vision loss.</b> 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.</li> <li>• <b>Loss of color vision.</b> Optic neuritis often affects color perception. You might notice that colors appear less vivid than normal.</li> <li>• <b>Flashing lights.</b> Some people with optic neuritis report seeing flashing or flickering lights.</li> </ul>
Reference	
Dr Khalid/Rabia	<p>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.</p> <p>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</p> <ul style="list-style-type: none"> <li>· Electrophysiology: can detect demyelination in apparently unaffected pathways with characteristic delays. Visual evoked potential studies should be the first choice.</li> <li>· 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.</li> <li>· Cerebrospinal fluid: rise in total protein with increase in immunoglobulin concentration with presence of oligoclonal cases.</li> </ul> <p>Rx:</p>

	<p>Decrease stress</p> <p>Steroid</p> <p>Interferon</p> <p>Monoclonal antibody, alemtuzumab</p> <p>AZT for relapsing and remitting MS</p>
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<b>Q: 1056</b>	<p>. Which of the following is not a degenerative corneal disease?</p> <p>a. Band keratopathy -</p> <p>b. Marginal dystrophy</p> <p>c. Fatty/lipid degeneration</p> <p>d. Mooren's ulcer- due to foreign trauma- not degenerative, autonomic</p> <p>e. Keratoconus</p>
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<b>Clincher(s)</b>	
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A	<p>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).<sup>[1]</sup> 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.)</p>  <p>Band keratopathy.</p> <p>Note the bandlike whitish-grey lesion across the corneal surface, sparing the superior and inferior cornea.</p>
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B	<p><b>Pellucid marginal degeneration (PMD; keratotorus)</b>, is a degenerative <a href="#">corneal</a> condition, often confused with <a href="#">keratoconus</a>. It is typically characterized by a clear, bilateral thinning (<a href="#">ectasia</a>) in the inferior and peripheral region of the cornea, although some cases affect only one eye. The cause of the disease remains unclear (<a href="#">idiopathic</a>).</p> <p>The ocular condition present in the patient whose case is to be reported has</p>
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	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.
<b>KEY</b>	
<b>Additional Information</b>	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.
<b>Reference</b>	<a href="http://www.ncbi.nlm.nih.gov/pubmed/9848831">http://www.ncbi.nlm.nih.gov/pubmed/9848831</a> . Medspace, jama ophthalmology
<b>Dr Khalid/Rabia</b>	<p>Dx is Mooren's ulcer.</p> <p>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.</p> <p>Pellucid marginal degeneration is a degenerative corneal condition, often confused with keratoconus.</p> <p>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.</p> <p>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.</p> <p>Fatty/lipid degeneration is degenerative.</p> <p>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-inflammatory drops, cytotoxics (topical and systemic), conjunctivectomy, and cornea debridement (superficial keratectomy). None is known to be superior</p>

<b>Q: 1090</b>	<p>A 29yo woman presents with lid lag, lid retraction and diplopia. What is the most appropriate next step?</p> <p>a. TFT b. Tensilon test- in myasthenia graves- lid lag c. Fundoscopy d. Autoantibodies e. EMG- used in myasthenia graves</p>
	Graves disease
<b>Clincher(s)</b>	<b>Lid lag, lid retraction, diplopia</b>
A	
B	The Tensilon test is a method to help diagnose <b>myasthenia gravis</b> .
C	
D	
E	Electromyography ( <b>EMG</b> ) 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.
<b>KEY</b>	<b>D</b>
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	<p>Scenario suggestive of Graves disease, hyperthyroidism. Graves' disease:[9]</p> <ul style="list-style-type: none"> <li>* 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: <ul style="list-style-type: none"> <li>* Thyroglobulin.</li> <li>* Thyroid peroxidase (or antimicrosomal antibodies).</li> <li>* Sodium-iodide symporter.</li> <li>* TSH receptor.</li> </ul> </li> <li>* The condition is characterised by a small to moderate diffuse, firm goitre with 50% of these showing ophthalmopathy.</li> <li>* There may be a personal or family history of autoimmune disease.</li> <li>* &lt;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.</li> <li>* There may also be lymphoid hyperplasia including splenomegaly and an enlarged thymus. * Associated with other autoimmune conditions - eg, pernicious anaemia, type 1 diabetes mellitus.</li> </ul> <p>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</p>



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	<p>needed (T3 toxicosis occurs in 5% of patients).</p> <ul style="list-style-type: none"> <li>* Autoantibodies - these are most commonly seen in Graves' disease:</li> <li>* Antimicrosomal antibodies - against thyroid peroxidase.</li> <li>* Antithyroglobulin antibodies.</li> <li>* TSH-receptor antibodies which are commonly present in Graves' disease but are not routinely measured.</li> <li>* TSI if elevated helps to establish a diagnosis of Graves' disease</li> </ul>
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<b>Q: 1144</b>	<p>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?</p> <p>a. Cataract</p> <p>b. DM retinopathy</p> <p>c. Hypermetropia</p> <p>d. Macular degeneration- occurs in older</p> <p>e. HTN retinopathy</p>
<b>Clincher(s)</b>	
A	
B	Patient not diabetic
C	<b>Hypermetropia</b> (Hyperopia) - Long Sighted. <b>Hypermetropia</b> means long sight and is where the image of a nearby object is formed behind the retina.
D	
E	Patient not hypertensive
<b>KEY</b>	<b>A</b>
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	<p>absent red reflex + tx for RA for 3yrs+ gradual loss of vision in both eyes.</p> <p>the treatment plan of rheumatoid arthritis includes : corticosteroids which induce cataract formation with long term use</p> <ul style="list-style-type: none"> <li>- Cataracts are cloudy (opaque) areas that develop in the lens of an eye and affects vision</li> <li>- Vision becomes gradually worse over the years.</li> <li>- Most affected people develop a cataract for no apparent reason. Factors that may increase the chance of developing cataracts include:</li> </ul>

	<ul style="list-style-type: none"> <li>- Having a poor diet.</li> <li>- Smoking.</li> <li>- Being exposed to a lot of ultraviolet light.</li> <li>- Diabetes.</li> <li>- Steroid medicines.</li> <li>- Having a family history of cataracts</li> <li>- 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</li> </ul>
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<b>Q: 1222</b>	<p>A 45yo man has had impaired vision and pain on eye movement in his left eye over the last 5 days. He also notes <b>loss of color vision</b> 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?</p> <ol style="list-style-type: none"> <li>Acute dacryocystitis</li> <li>Acute iritis-</li> <li>Papillitis</li> <li>Retrobulbar neuritis – optic nerve</li> <li>Scleritis</li> </ol>
<b>Clincher(s)</b>	
A	Acute inflammation of the tear sac
B	<p>Pain in the eye or brow region</p> <p>Worsened <b>eye pain</b> when exposed to bright light</p> <p>Reddened eye, especially adjacent to the iris</p> <p>Small or funny shaped pupil</p> <p>Blurred <b>vision</b></p> <p><b>Headache</b></p>
C	<p><b>Optic papillitis</b> is a specific type of <b>optic neuritis</b>. Inflammation of the <b>optic nerve head</b> is called "papillitis" or "intraocular optic neuritis"; inflammation of the <b>orbital</b> portion of the nerve is called "retrobulbar optic neuritis" or "orbital optic neuritis".<sup>[1]</sup> 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 <b>multiple sclerosis</b>.<sup>[2]</sup></p> <p>Papillitis may have the same appearance as papilledema. However, papillitis</p>

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	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.
<b>KEY</b>	<b>d. Retrobulbar neuritis</b>
Additional Information	
<b>Reference</b>	Webmd.ohcs.
Dr Khalid/Rabia	<p>Optic neuritis or retrobulbar neuritis is inflammation of the optic nerve. It consists of the classic triad -</p> <ol style="list-style-type: none"> <li>1. Reduced vision</li> <li>2. Eye pain, particularly on movement</li> <li>3. Impaired colour vision</li> </ol> <p>Common causes are multiple sclerosis, giant cell arteritis.</p> <p>Typical signs - decreased pupillary light reaction in affected eye, or Marcus Gunn Pupil</p>

<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	
<b>Clincher(s)</b>	

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A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:372</b>	<p>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: <b>flame shaped hemorrhages. PALE FUNDUS</b> The right eye is normal.</p>
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	<p>What is the cause of this pts unilateral blindness?</p> <p>a. HTN retinopathy b. CRA thrombosis c. CRV thrombosis d. Background retinopathy e. Retinal detachment</p>
<b>Clincher(s)</b>	<b>HTN male loses vision in left eye. Funds exam reveal flame shape haemorrhages.</b>
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.
<b>KEY</b>	<b>C</b>
<b>Additional Information</b>	<i>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.</i>
<b>Reference</b>	
Dr Khalid/Rabia	<p>unilateral blindness with flame shaped hemorrhages are characteristic of CRVO).</p> <p>Flame shaped hemorrhages are seen in HTN and diabetic retinopathy too but they will cause bilateral damage.</p>

<b>Q:397</b>	<p>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?</p> <p>a. Cataract b. Glaucoma c. Retinal detachment d. Iritis e. GCA</p>
<b>Clincher(s)</b>	<b>Changes of glasses due to Dec vision but no improvement.</b>
A	Opacity of lens and cause Dec vision and changes of glasses doesn't help in correcting the vision.
B	<p>Glaucoma is inc in intra ocular pressure and visual acuity is decreased</p> <p>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.</p>

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C	Sudden loss of painless vision
D	Iritis is part of Ant uveitis and present in AS, Reiters syndrome, UC, Cihns, 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.
<b>KEY</b>	<b>A</b>
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	<p>old age and progressive weakness supports Cataract</p> <p>Not glaucoma...as pupil would be mid dilated and sluggish reaction and in acute attack corneal edema</p> <p>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</p> <p>Not iritis..as pupil would be constricted and cornea would have precipitation on its back (keratic precipitate)</p> <p>Not GCA(giant cell arteritis) as vision on it is suddenly dropped to HM up to LP and vision dropped day and night</p>

<b>Q:398</b>	<p>A pt comes with sudden loss of vision. On fundoscopy the optic disc is normal. What is the underlying pathology?</p> <p>a. Iritis</p> <p>b. Glaucoma</p> <p>c. Vitreous chamber</p> <p>d. Retinal detachment</p>
	Sudden loss of vision but on fundoscopy the disc is normal
<b>Clincher(s)</b>	
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	
<b>KEY</b>	<b>D</b>
Additional Information	<i>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.</i>
<b>Reference</b>	
Dr Khalid/Rabia	Causes of sudden painless loss of vision:

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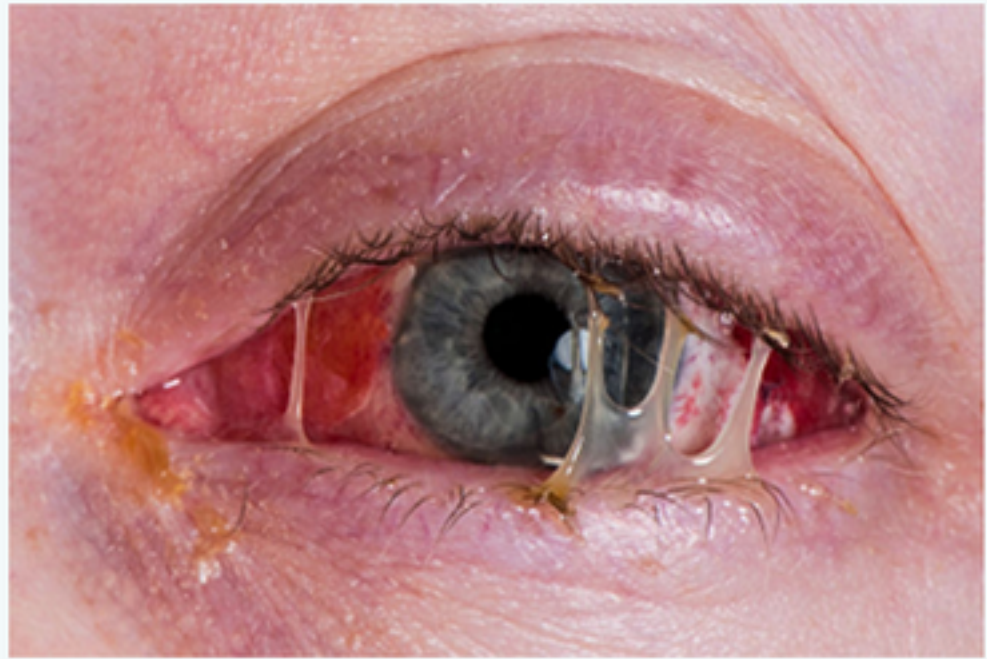
	<ol style="list-style-type: none"> <li>1. Retinal detachment</li> <li>2. Vitreous haemorrhage</li> <li>3. Retinal vein occlusion</li> <li>4. Retinal artery occlusion</li> <li>5. Optic neuritis</li> <li>6. Cerebrovascular accident (GCA)</li> </ol>
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<b>Q:419</b>	<p>A 54yo myopic develops flashes of light and then sudden loss of vision. That is the single most appropriate tx?</p> <ol style="list-style-type: none"> <li>a. Pan retinal photo coagulation</li> <li>b. Peripheral iridectomy</li> <li>c. Scleral buckling</li> <li>d. Spectacles</li> <li>e. Surgical extraction of lens</li> </ol>
<b>Clincher(s)</b>	<b>Tx of retinal detachment</b>
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.
<b>KEY</b>	<b>C</b>
Additional Information	<p><b><u>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.</u></b></p> <p><b><u><a href="http://www.surgeryencyclopedia.com">www.surgeryencyclopedia.com</a></u></b></p>
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:429</b>	<p>. A pt presents with a painful, sticky red eye with a congested conjunctiva. What is the most suitable tx?</p> <ol style="list-style-type: none"> <li>a. Antibiotic PO</li> <li>b. Antihistamine</li> <li>c. Antibiotic drops</li> <li>d. Steroid drops</li> <li>e. IBS</li> </ol>
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<b>Clincher(s)</b>	<b>Tx of conjunctivitis</b>
A	No role
B	Given in allergic conjunctivitis
C	Right option
D	Can be given in severe inflammatory conditions.
E	?
<b>KEY</b>	<b>C</b>
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	<p><b>Painful eye, usually bilateral.</b> Smearing of vision on waking up. Mild photophobia. If severe, indicates corneal involvement or adenoviral conjunctivitis.</p> <p><b>Thick yellowish-white mucopurulent discharge.</b> Visual acuity is normal</p> <p>Symptoms- Red eye, difficult to open in the morning, glued together by discharge.</p> <p>Presence of follicles on the conjunctiva- More likely viral conjunctivitis.</p>





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.

<b>Q:443</b>	<p>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?</p> <ol style="list-style-type: none"> <li>Pan retinal photo coagulation</li> <li>Pilocarpine eye drops</li> <li>Corticosteroids</li> <li>Peripheral iridectomy</li> <li>Surgical extraction of lens</li> </ol>
<b>Clincher(s)</b>	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.

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C	Right option
D	Tx of acute angle closure glaucoma
E	Tx of cataract.
<b>KEY</b>	<b>C</b>
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	<p>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.</p> <p>&gt; Pan retinal photocoagulation is done for diabetic retinopathy where parts on the retina are burned in order to reduce the Oxygen demand.</p> <p>Lens extraction is done mainly for cataract to remove the opacified lens that disturbs the vision</p> <p>&gt; 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.</p> <p>&gt; 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</p> <p>&gt; Surgical extraction of the lens is done for cataract where the opacified lens that disturbs the vision is removed</p>

<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	

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D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q: 443</b>	A 38yo female presents with sudden loss of vision but fundoscopy is normal. She 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
<b>Clincher(s)</b>	<b>38yo, female, sudden loss of vision, fundoscopy normal, h/o 1yr ago, weakness in RUL and exaggerated reflexes.</b>
A	Pan retinal photocoagulation is done for <b>diabetic retinopathy</b> where parts on the retina are burned in order to reduce the Oxygen demand.
B	Pilocarpine is a parasympathomimetic given for <b>open angle glaucoma</b> 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 <b>open angle glaucoma</b> 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 <b>for cataract</b> where the opacified lens

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	that disturbs the vision is removed
<b>KEY: C</b>	<b>Corticosteroid.</b> 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	<div style="background-color: red; color: white; padding: 5px;"><b>500 Multiple sclerosis (MS)</b></div> <p><b>Cause</b> Discrete plaques of demyelination occur at multiple CNS sites, from T-cell-mediated immune response (trigger is unknown, but see vit. D, below). Demyelination heals poorly, causing <b>relapsing and remitting symptoms</b>. Prolonged demyelination causes axonal loss and clinically <b>progressive symptoms</b>. <b>Prevalence:</b> commoner in temperate areas (England ≥42:100,000; SE Scotland 200:100,000;<sup>248</sup> rarer in Black Africa/Asia). Lifetime UK risk 1:1000. Adult migrants take their risk with them; children acquire the risk of where they settle. <b>Mean age of onset:</b> 30yrs. ♀:♂ ≥3:1.<sup>249</sup></p> <p>Early exposure to sunlight/vit. D is important, and vit. D status relates to prevention of MS, and fewer symptoms and fewer new lesions on MRI in established MS.<sup>250, 251</sup></p> <p><b>Presentation</b> Usually monosymptomatic: <b>unilateral optic neuritis</b> (pain on eye movement and rapid ↓ central vision); numbness or tingling in the limbs; <b>leg weakness</b>; brainstem or cerebellar symptoms (eg diplopia, ataxia). Other signs: see box. Symptoms may worsen with heat (eg hot bath) or exercise. Rarely polysymptomatic.</p> <p><b>Progression:</b> Early on, relapses (which can be stress induced)<sup>252</sup> may be followed by remission and full recovery. With time, remissions are incomplete, so disability accumulates. Steady progression of disability from the outset also occurs, while</p>
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q: 460</b>	A 55yo man presents with mild headache. He has changed his spectacles thrice in 1 yr. there is <b>mild cupping</b> > <b>glaucoma</b> 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 (constrictor not dilator) c. Corticosteroids d. Scleral buckling e. Analgesics alone
<b>Clincher(s)</b>	<b>sickle shaped scotoma, mild cupping present in the disc, spectacles thrice in 1 yr, headache</b>
A	
B	
C	
D	Scleral buckling is done for retinal detachment to put the retina back in place.
E	
<b>KEY: B</b>	<b>Pilocarpine.</b>  Sickle-shaped scotoma or siesel 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,

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	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	

<b>Q: 473</b>	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
	LR6SO4
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY: B</b>	<b>Trochlear Nerve</b> 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	

<b>Q: 487</b>	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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<b>Clincher(s)</b>	
A	
B	
C	
D	
E	IOP (Intraocular pressure) is used to investigate glaucoma.
<b>KEY: A</b>	<b>ESR</b> 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	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q: 518</b>	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 (all dry)
<b>Clincher(s)</b>	
A	Autosomal dominant, Clinically the patient suffers oedema of the skin and mucosal surfaces
B	
C	Most commonly in children There is an incubation period of 14 to 25 days. A prodrome of non specific symptoms like fever, malaise myalgias, and anorexia may be followed by enlargement of one or both parotid glands, developing over a period of 1 to 3 days. <ul style="list-style-type: none"> <li>▷ older children may complain of tenderness over the parotid gland and occasionally ear ache before actual swelling becomes evident (2)</li> <li>▷ the parotid enlargement may displace the ear lobe upwards and obliterate the space between the mandible and the sternomastoid muscle</li> <li>▷ the swelling may even impede chewing or pronunciation of words (3)</li> <li>▷ other salivary glands, namely the submandibular and sublingual salivary glands, may also become inflamed.</li> <li>▷ upon looking in the mouth, the tonsils may be displaced towards the midline.</li> </ul>
D	
E	
<b>KEY: E</b>	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	<p><b>Sjögren's syndrome</b> A chronic inflammatory autoimmune disorder, which may be primary (♀:♂≈9:1, onset 4<sup>th</sup>-5<sup>th</sup> decade) or secondary, associated with connective tissue disease (eg RA, SLE, systemic sclerosis). There is lymphocytic infiltration and fibrosis of exocrine glands, especially lacrimal and salivary glands. <b>Features:</b> ↑tear production (dry eyes, keratoconjunctivitis sicca), ↓salivation (xerostomia—dry mouth, caries), parotid swelling. Other glands are affected causing vaginal dryness, dyspareunia, dry cough and dysphagia. Systemic signs include polyarthritis/arthritis, Raynaud's, lymphadenopathy, vasculitis, lung, liver and kidney involvement, peripheral neuropathy, myositis and fatigue. It is associated with other autoimmune diseases (eg thyroid disease, autoimmune hepatitis, PBC) and an ↑risk of non-Hodgkin's B-cell lymphoma. <b>Tests:</b> Schirmer's test measures conjunctival dryness (&lt;5mm in 5min is +ve). Rose Bengal staining may show keratitis (use a slit-lamp). Anti-RO (SSA; in 40%) &amp; anti-La (SSB; in 26%) antibodies may be present (in pregnancy, these cross the placenta and cause fetal congenital heart block in 5%). ANA is usually +ve (74%); rheumatoid factor is +ve in 38%.<sup>112</sup> There may be hypergammaglobulinaemia. Biopsy shows focal lymphocytic aggregation. <b>Rx:</b> Treat sicca symptoms: eg hypromellose (artificial tears), frequent drinks, sugar-free pastilles/gum. NSAIDs and hydroxychloroquine are used for arthralgia. Immunosuppressants may be indicated in severe systemic disease.<sup>113</sup> Henrik Conrad Samuel Sjögren, 1899-1986 (Swedish ophthalmologist)</p>
Reference	
Dr Khalid/Rabia	<p>e. Sjogrens syndrome main symptoms of xerophthalmia (dry eyes), xerostomia (dry mouth) and enlargement of the parotid glands.</p> <ul style="list-style-type: none"> <li>• Difficulty eating dry food, typically cracker biscuits.</li> <li>• Difficulty with dentures.</li> <li>• Complaint of the tongue sticking to the roof of the mouth.</li> <li>• Speaking for long periods of time causes hoarseness.</li> <li>• Oral candidiasis and angular cheilitis.</li> </ul> <p>Dry eyes tend to cause a gritty sensation. There is a predisposition to blepharitis and the eyes may be sticky in the morning.</p> <ul style="list-style-type: none"> <li>• There may be recurrent parotitis, usually bilateral. Glands are usually enlarged but this is not often the presenting feature.</li> <li>• Dryness of the mucosa of the trachea and bronchi may present as a dry cough.</li> <li>• Dryness of the pharynx and oesophagus may cause difficulty in swallowing, and lack of saliva and secretions may predispose to gastro-oesophageal reflux.</li> <li>• There can be dry skin and vaginal dryness causing dyspareunia</li> <li>• 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.</li> <li>• Fatigue is a common feature.</li> <li>• About 20% have Raynaud's phenomenon.</li> </ul> <p>Associated diseases</p> <p>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 recurrent miscarriage with antiphospholipid</p>



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	syndrome. Investigations • Rheumatoid factor • Antinuclear antibodies • Schirmer test
<b>Q:1287</b>	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
<b>Clincher(s)</b>	<b>Sudden loss of vision , high bp</b>
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 hypercoagulable 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
<b>KEY</b>	<b>d-CRVO</b>
Additional Information	
<b>Reference</b>	
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. <b>CRVO: tomato splash, swollen huge optic disc, congested fundus</b> <b>CRAO: white retina, pale fundus, cherry red spot</b>

<b>Q:1294</b>	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
<b>Clincher(s)</b>	<b>Drusen yellow spots</b>
A	<i>Maculopathy:</i> Leakage from the vessels close to the macula cause oedema and can significantly threaten vision (clinically significant macular oedema). It can exist with otherwise mild retinopathy.

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B	
C	<i>MS –hard exudates , macular edema, hemorrhage</i>
D	<p><i>Non-proliferative diabetic retinopathy (NPDR)</i> is rated as mild, moderate or severe depending on the degree of ischaemia. Signs comprise microaneurysms (seen as 'dots'), haemorrhages (flame shaped or 'blots') hard exudates (yellow patches), engorged tortuous veins, cotton wool spots, large blot haemorrhages (the latter 3 are signs of significant ischaemia). NPDR can progress to sight-threatening proliferative retinopathy.</p>
E	<i>Proliferative diabetic retinopathy (PDR):</i> Fine new vessels appear on the optic disc, retina and can cause vitreous haemorrhage.
KEY	a. Macular degeneration
Additional Information	<ul style="list-style-type: none"> <li>· dry (geographic atrophy) macular degeneration: characterised by drusen - yellow round spots in Bruch's membrane</li> <li>· 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</li> </ul> <p>Features</p> <ul style="list-style-type: none"> <li>· reduced visual acuity: 'blurred', 'distorted' vision, central vision is affected first</li> <li>· central scotomas</li> <li>· fundoscopy: drusen, pigmentary changes</li> </ul>
Reference	
Dr Khalid/Rabia	

<b>Q:1295</b>	<p>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?</p> <p>a. CRAO b. Branch RAO c. CRVO d. Optic atrophy</p>
<b>Clincher(s)</b>	<b>Headache , blurring of vision , acuity loss</b>
A	<p>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</p>
B	
C	Retinal vein occlusion is one of the most common causes of sudden painless unilateral

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	loss of vision. Loss of vision is usually secondary to macular oedema
D	
E	
<b>KEY</b>	C – CRVO
Additional Information	.
<b>Reference</b>	
Dr Khalid/Rabia	<p>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).</p> <p>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.</p> <p>Central retinal vein occlusion</p> <ul style="list-style-type: none"> <li>· incidence increases with age, more common than arterial occlusion</li> <li>· causes: glaucoma, polycythaemia, hypertension</li> <li>· severe retinal haemorrhages are usually seen on fundoscopy</li> </ul>

<b>Q:1296</b>	<p>A 64yo DM has come for a routine eye check up. Fundoscopy: new vessels all over the retina. What is the most appropriate management?</p> <ol style="list-style-type: none"> <li>a. Strict sugar control</li> <li>b. Regular eye check ups</li> <li>c. Non urgent referral to specialist</li> <li>d. Laser photocoagulation</li> <li>e. Insulin</li> </ol>
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	<p>d. Laser photocoagulation</p> <p>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.</p>
Additional Information	
<b>Reference</b>	
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	Traditional classification	New classification
	<p>Background retinopathy</p> <ul style="list-style-type: none"> <li>• microaneurysms (dots)</li> <li>• blot haemorrhages (<math>\leq 3</math>)</li> <li>• hard exudates</li> </ul> <p>Pre-proliferative retinopathy</p> <ul style="list-style-type: none"> <li>• cotton wool spots (soft exudates; ischaemic nerve fibres)</li> <li>• <math>&gt; 3</math> blot haemorrhages</li> <li>• venous beading/looping</li> <li>• deep/dark cluster haemorrhages</li> <li>• more common in Type I DM, treat with laser photocoagulation</li> </ul>	<p>Mild NPDR</p> <ul style="list-style-type: none"> <li>• 1 or more microaneurysm</li> </ul> <p>Moderate NPDR</p> <ul style="list-style-type: none"> <li>• microaneurysms</li> <li>• blot haemorrhages</li> <li>• hard exudates</li> <li>• cotton wool spots, venous beading/looping and intraretinal microvascular abnormalities (IRMA) less severe than in severe NPDR</li> </ul> <p>Severe NPDR</p> <ul style="list-style-type: none"> <li>• blot haemorrhages and microaneurysms in 4 quadrants</li> <li>• venous beading in at least 2 quadrants</li> <li>• IRMA in at least 1 quadrant</li> </ul>

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

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

<b>Q:</b>	
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	
Additional Information	
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q:1457</b>	<p>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?</p> <p>a. Ciliary body</p>
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	<p>b. Sclera</p> <p>c. Conjunctivitis</p> <p>d. Cornea</p> <p>e. Iris</p>
<b>Clincher(s)</b>	
A	
B	
C	
D	
E	
<b>KEY</b>	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	<p><b>Corneal injuries</b> may be physical, chemical, environmental (eg, ultraviolet (UV) damage) or infective.</p> <p>Corneal abrasions are common. There is usually a history of minor trauma from a scratch, grit or contact lens problem.</p> <p>If there is no history of injury, consider infection - eg, herpes simplex infection, chemical injury and corneal foreign bodies.</p> <p><b>Symptoms of superficial corneal abrasion or corneal FB</b> : Redness, pain, watering (usually). FB sensation (usually), Blurred vision, Photophobia, Pain on eye movement, Patients are fairly reliable at locating the FB.</p>

	<p>Investigations: are not required if you can be sure that the injury is superficial. Plain X-rays of</p> <p>_____</p> <p>of the orbit/face can be used to exclude known radiopaque FBs</p> <p>Treatment= Removal of Foreign body. Use a topical anesthetic</p> <p>Management of corneal abrasion= refer if large abrasion otherwise: Analgesia= Paracetamol</p> <p>_____</p> <p>or ibuprofen are first line. topical antibiotics for 7 days= Chloramphenicol for 1st line.</p>
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q: 1559</b>	<p>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?</p> <p>a. CRVO</p> <p>b. Acute closed angle glaucoma</p> <p>c. Uveitis</p> <p>d. Iritis</p> <p>e. Open angle glaucoma</p>
<b>Clincher(s)</b>	<b>Painful eye with visual blurring and acuity of only one finger.</b>
A	

B	
C	
D	
E	
<b>KEY</b>	<b>B- acute angle glaucoma,</b>
Additional Information	<p>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.</p> <p>Treatment</p> <ul style="list-style-type: none"> <li>* Pilocarpine 2 drops x 2 hourly</li> <li>* Acetazolamide 500mg IV x STAT OR Mannitol IV</li> <li>* Good analgesics</li> <li>* Anti-emetics</li> </ul>
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q: 1612</b>	<p>A 75yo man with declining vision, cornea and pupils are normal, fundus shows obscured margins. What is the single most likely dx?</p> <ul style="list-style-type: none"> <li>a. Macular degeneration</li> <li>b. HTN retinopathy</li> <li>c. MS</li> <li>d. DM background</li> <li>e. Proliferative DM retinopathy</li> </ul>




<b>Clincher(s)</b>	<b>Declining vision, cornea and normal pupils with obscured fundus.</b>
A	
B	
C	
D	
E	
<b>KEY</b>	<b>A</b>
Additional Information	The age of the patient, normal ophthalmological examination and <b>obscured margins of the fundus</b> 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.
<b>Reference</b>	
Dr Khalid/Rabia	

<b>Q: 1625</b>	<p>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?</p> <p>a. Chorioretinitis – bilat cataracts</p> <p>b. Conjunctivitis</p> <p>c. Episcleritis</p> <p>d. Iritis</p> <p>e. Keratitis</p>
<b>Clincher(s)</b>	<b>Acute painful, red right eye with blurring of vision for one eye and backache with stiffness- ankylosing spondylitis.</b>
A	Inflammation of iris, retina and choroid
B	Inflammation of conjunctiva.
C	Inflammation below the conjunctiva in the sclera

D	
E	
KEY	<b>D- Iritis.</b>
Additional Information	<p><b>Anterior uveitis/iritis</b> 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 inflammation will involve the ciliary body, the best term is anterior uveitis, but note that anterior and posterior components may be affected together. <i>The patient:</i> 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). <i>Talbot's test</i> is +ve.<sup>4</sup> <i>Slit lamp:</i> White precipitates on the back of the cornea; anterior chamber cells (pus = 'hypopyon'). It typically affects those of working age. <i>Causes:</i> see MINIBOX; often none is found (∴ 'autoimmune'). It may relapse so regular eye clinic care and follow-up is vital. <i>R:</i> Aim to prevent damage from prolonged inflammation (disrupts flow of aqueous (→glaucoma ± adhesions between iris &amp; lens). Drops: 0.5–1% prednisolone/2h, to ↓ inflammation (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 inflammation. <i>Intravitreal and biological agents</i> show promise eg anti-TNF and anti-CD20 if HLAB27+ve (most with anterior uveitis are).<sup>55</sup> Adalimumab has a role.<sup>56</sup></p> <p><b>Types &amp; causes of uveitis</b></p> <p><b>Anterior uveitis:</b><sup>53,54</sup></p> <ul style="list-style-type: none"> <li>• Ank. spond.; Still's<sup>2</sup>, p654</li> <li>• Sarcoid; Behçet's, etc<sup>3</sup></li> <li>• Crohn's/uc; Reiter's<sup>4</sup></li> <li>• Herpes, TB, syphilis, HIV</li> </ul> <p><b>Intermediate uveitis:</b></p> <ul style="list-style-type: none"> <li>• MS; lymphoma; sarcoid</li> </ul> <p><b>Posterior &amp; panuveitis:</b></p> <ul style="list-style-type: none"> <li>• Herpes simplex + zoster</li> <li>• toxoplasmosis; TB; CMV; endophthalmitis</li> <li>• Lymphoma; sarcoidosis</li> <li>• Behçet's</li> </ul>
Reference	
Dr Khalid/Rabia	

Q: 1689	<p>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?</p> <p>a. Pseudotumor –</p> <p>b. Orbital teratoma</p>
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	<p>c. Optic neuritis (for MS)</p> <p>d. Sarcoidosis</p> <p>e. Optic glioma – unilat, no flashes involved</p> <p>f. Lymphangioma – no lymphandep</p> <p>g. Rhabdomyosarcoma – no muscle</p> <p>h. Retinal vascular shunts</p> <p>i. Retinoblastoma – infantdisease</p> <p>j. Mucormycosis – fungal infection</p>
<b>Clincher(s)</b>	<b>Previous history of flashes and inflammation of optic nerve</b>
A	
B	
C	
D	
E	
<b>KEY</b>	<b>C- optic neuritis.</b>
Additional Information	<p><b>Subacute loss of vision Optic neuritis (fig2)</b>            Unilateral loss of acuity occurs over hours or days. Colour vision is affected (dyschromatopsia): reds appear less red, 'red desaturation'—and eye movements hurt. The pupil shows an afferent 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 deficiency. <b>Rx:</b> High-dose methylprednisolone for 72h (250mg/6h iv), then prednisolone (1mg/kg/d po) for 11 days may briefly delay onset of MS (no change to long-term disability).<sup>70</sup></p> <p><b>Transient visual loss</b> ▶ Always think of vascular <b>Typical causes:</b></p>
	 <p><b>Fig 2.</b> Optic neuritis</p>
<b>Reference</b>	
Dr Khalid/Rabia	