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Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

• Reducing aqueous secretions with acetazolamide and inducing pupillary constriction with topical pilocarpine. • Pilocarpine should not be the initial treatment as it is ineffective at pressures above 40 mmHg. • Mannitol is typically reserved for refractory cases, not responding to the initial medical treatment. Top Tips Do not use mydriatic drugs (e.g., atropine and epinephrine) during ophthalmologic examination in patients with acute angle-closure glaucoma! Moreover, do not cover the eye, since darkness induces mydriasis and worsens the condition

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Primary open-angle glaucoma (POAG) Epidemiology: • The most common type of glaucoma. present in 2% of people older than 40 years. • Second leading cause of blindness following age-related macular degeneration (AMD). Pathophysiology • Secondary clogging of the trabecular meshwork or reduced drainage → gradual ↑ in IOP → vascular compression → ischemia to the optic nerve → progressive visual impairment. Risk factors: age, family history, black patients, myopia, hypertension, diabetes mellitus Features: • bilateral, progressive visual field loss (from peripheral to central) (Loss of nasal visual field) progressing to 'tunnel vision' • Fundoscopy: cupping and pallor of optic disc Management: • Eye drops to lower intra-ocular pressure (IOP) • Laser trabeculoplasty An alternative first-line treatment refractory to pharmacotherapy

Chapter 12

Ophthalmology

Medication Mode of action Notes Prostaglandin analogues (e.g. Latanoprost) Increases uveoscleral outflow hypokalemia, renal stones, acidosis, and aplastic anemia. Beta-blockers (e.g. Timolol) Reduces aqueous production Should be avoided in asthmatics and patients with heart block Sympathomimetics (e.g. brimonidine, an alpha2adrenoceptor agonist) Reduces aqueous production and increases outflow Avoid if taking MAOI or tricyclic antidepressants Adverse effects include hyperaemia Carbonic anhydrase inhibitors (e.g. acetazolamide) Reduces aqueous production Systemic absorption may cause sulphonamide-like reactions Miotics (e.g. pilocarpine, a muscarinic receptor agonist) Increases uveoscleral outflow Adverse effects included a constricted pupil, headache and blurred vision

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Age related macular degeneration (AMD) Epidemiology • The most common cause of blindness Pathophysiology • progressive degenerative changes in the central part of the retina (macula) → visual impairment Risk factors: • Advanced age, smoking Classification Dry AMD (nonexudative, atrophic) Prevalence ~90% ~10% Pathophysiology Deposition of drusen (yellow round spots) in the retinal pigment epithelium. Choroidal neovascularization (between the retinal pigment epithelium and Bruch's membrane) Onset slow progressive visual impairment (usually over decades) acute or insidious onset (over weeks to months) Presentation Bilateral manifests in one eye Fundoscopy Drusen (a small, yellowish, granular, subretinal deposits that are age related). Subretinal and intraretinal hemorrhage and/or exudate. if neovascularisation is present fluorescein angiography is performed Treatment Supportive: stop smoking Diet: high dose of beta-carotene, vitamins C and E, and zinc. Supplements should be avoided in smokers due to an increased risk of lung cancer Symptoms Reduced visual acuity: 'blurred', 'distorted' vision, central vision is affected first (central scotomas) Notes & Notes for MRCP
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Once daily administration Preferred first-line therapy. should be used first-line in patients with a history of asthma. Adverse effects: brown pigmentation of the iris, growth of eyelashes Epithelial keratopathy Systemic: paresthesia, Wet AMD (exudative , neovascular) First-line: injection of VEGF inhibitors (ranibizumab, bevacizumab, pegaptanib) into the vitreous body.

Differential diagnosis Differential diagnosis of vision loss Condition Clinical features Fundoscopy Age related macular degeneration • May be insidious (dry AMD) or rapid (wet AMD) onset • Impairment of central vision only (vision loss is rare) Open-angle glaucoma • Insidious onset • Peripheral vision loss (tunnel vision) Central Vessel occlusion (retinal artery) • Acute or subacute onset • Complete vision loss • Swollen disc • Retinal haemorrhages • Cotton wool spots Retinal detachment • Acute onset • Partial or complete vision loss (falling curtain) Cataract • Insidious onset • Blurred, dim vision, and a glare • Absent or opacified red-reflex Top tips Notes & Notes for MRCP
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• Drusen • Macula depigmentation • Disc cupping with high intraocular pressure • Detached or floating retina • Retina may not be visible (in advanced disease) The fundus shows small pale dots over the macular area typical of drusen. This is macular degeneration and one of the commonest causes of blindness.

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Cataracts Definition • opacification of the lens Causes • Majority age related (Senile cataracts) the most common cause 17% of people older than 40 years 50% of people older than 75 years UV light • Systemic diabetes mellitus steroids Inhaled steroids can cause cataracts infection (congenital rubella) metabolic: diabetes hypocalcaemia, galactosaemia but if the galactosaemia is treated, the cataract is reversible. myotonic dystrophy, Down's syndrome • Ocular trauma uveitis high myopia topical steroids Feature • Symptoms painless,

progressive, and slow vision loss
• Physical exam absent red reflex Classification • Nuclear sclerosis: the most common type of cataract, involves the central or 'nuclear' part of the lens. common in old age reduction of vision is the major symptom.

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 change lens refractive index, often leads to an increase in refractive power of the lens causing nearsightedness (problems with distance vision). • Polar: localized, commonly inherited, lie in the visual axis • Subcapsular: glare is the major symptom Glare is difficulty seeing in the presence of bright light such as direct or reflected sunlight or artificial light such as car headlamps at night. due to steroid use, just deep to the lens capsule, in the visual axis Posterior subcapsular cataracts are associated with: retinitis pigmentosa chronic steroid use. Anterior subcapsular cataracts are associated with: idiopathic or secondary to trauma and iotragenic causes. • Dot opacities common in normal lenses, also seen in: diabetes myotonic dystrophy

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Diabetic retinopathy See endocrinology

Angioid retinal streaks • Angioid retinal streaks are seen on fundoscopy as irregular dark red streaks radiating from the optic nerve head. They are caused by degeneration, calcification and breaks in Bruch's membrane . Causes A useful mnemonic for angioid retinal streak is SLAPPERS: • S - Sickle-cell anaemia • L - Lead poisoning • A - Abetalipoproteinaemia/acromegaly • P - Paget's disease /phacomatoses (tuberous, sclerosis, neurofibromatosis, SturgeWeber) • P - Pseudoxanthoma elasticum • E - Ehlers-Danlos syndrome • R - Raised calcium or phosphate • S - Short people (dwarfism).

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Mydriasis Causes of mydriasis (large pupil) • third nerve palsy • Holmes-Adie pupil • traumatic iridoplegia • phaeochromocytoma • congenital • Drug causes of mydriasis topical mydriatics: tropicamide, atropine sympathomimetic drugs: amphetamines, pseudoephedrine, amphetamines and cocaine, anticholinergic drugs: eg antihistamines, atropine and tricyclic antidepressants Poisons (atropine, CO, ethylene glycol).

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Miosis Causes of small pupils include: • Horner's syndrome • Old age • Pontine haemorrhage • Argyll Robertson pupil • Drugs, and • Poisons (opiates, organophosphates).

Holmes-Adie pupil Abnormally dilated pupil (mydriasis) which does not constrict in response to light, loss of deep tendon reflexes, and abnormalities of sweating. Holmes-Adie pupil is a benign condition most commonly seen in women. It is one of the differentials of a dilated pupil. Overview • unilateral in 80% of cases • dilated pupil (tonically dilated pupil) • slowly reactive to accommodation but very poorly (if at all) to light • once the pupil has constricted it remains small for an abnormally long time • associated

with absent ankle/knee reflexes and impaired sweating

The cause of the associated arreflexia is unknown. Pathophysiology • Viral or bacterial infection causes damage to neurons in the ciliary ganglion, located in the posterior orbit, that provides parasympathetic control of eye constriction. • damage to the dorsal root ganglia of the spinal cord problems with autonomic control of the body. Diagnosis • testing with low dose (1/8%) pilocarpine may constrict the tonic pupil due to cholinergic denervation super-sensitivity. A normal pupil will not constrict with the dilute dose of pilocarpine.

Argyll-Robertson pupil • the prostitute's pupil - accommodates but doesn't react. • Another mnemonic used for the Argyll-Robertson Pupil (ARP) is Accommodation Reflex Present (ARP) but Pupillary Reflex Absent (PRA) Features • small, irregular pupils • no response to light but there is a response to accommodate Causes • diabetes mellitus • syphilis (neurosyphilis)

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Anisocoria • is a condition characterized by an unequal size of the eyes' pupils. • Affecting 20% of the population, • it can be an entirely harmless condition or a symptom of more serious medical problems • The history of anisocoria, with headaches and diplopia should ring alarm bells, in that a life-threatening posterior communicating artery aneurysm/berry aneurysm needs to be excluded urgently.

Optic atrophy • Optic atrophy is a descriptive term, it is the optic neuropathy that results in visual loss • Usually bilateral and causes a gradual loss of vision. • On fundoscopy optic atrophy is seen as pale, well demarcated disc. • Causes may be acquired or congenital Acquired causes • multiple sclerosis • papilloedema (longstanding) • raised intraocular pressure (e.g. glaucoma, tumour) • retinal damage (e.g. choroiditis, retinitis pigmentosa) • ischaemia • toxins: tobacco amblyopia, quinine, methanol, arsenic, lead • nutritional: vitamin B1, B2, B6 and B12 deficiency Congenital causes • Friedreich's ataxia • mitochondrial disorders e.g. Leber's optic atrophy usually affects young men. It causes sequential optic neuropathies in days to weeks. It is typically painless and severe. Visual acuity fails to improve. Mutations in the MT-ND1, MT-ND4, MT-ND4L, and MT-ND6 genes These genes are contained in mitochondrial DNA. Specifically, more than 50% of males with a mutation and more than 85% of females with a mutation never experience vision loss or related medical problems. • DIDMOAD - the association of cranial Diabetes Insipidus, Diabetes Mellitus, Optic Atrophy and Deafness (also known as Wolfram's syndrome)

Optic neuritis

The patient sees nothing and the doctor sees nothing • Optic neuritis is a broad term which can be used to describe inflammation, degeneration or demyelination of the optic nerve. • Optic neuritis is very rare in people over the age of 50. • It encompasses a number of conditions, including: Papillitis (anterior optic neuritis) - the intraocular portion of the nerve is affected, and the optic disc is swollen It is important to note that the disc changes in papilloedema may closely resemble those of papillitis but visual acuity is markedly reduced in papillitis and not papilloedema. Retrobular neuritis - the distal portion of the optic nerve is affected, and the disc is therefore not swollen Neuroretinitis - optic disc and adjacent temporal retina are affected. Causes • multiple sclerosis • diabetes • syphilis Features • unilateral decrease in visual acuity over hours or days Visual loss typically occurs over days rather than hours. Sudden visual loss due to optic neuritis is very unusual. Optic neuritis presents with a particular type of central visual loss - a central scotoma. • poor discrimination of colours, 'red desaturation' - ie when red looks paler to one eye than the other - • The retrobulbar neuritis seen with ethambutol may be unilateral or bilateral; as such unilateral symptoms do not preclude the diagnosis. Notes & Notes for MRCP
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This patient has optic atrophy as revealed by a particularly pale disc. Causes include: • Glaucoma • External compression of the optic nerves, for example, pituitary tumour, and • Multiple sclerosis.

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• pain worse on eye movement • relative afferent pupillary defect during the 'swinging flashlight test'. • central scotoma • Most cases of optic neuritis are retrobulbar and hence there are no abnormalities on fundoscopy. the most likely finding on fundoscopy Normal optic disc Diagnosis • MRI with gadolinium of the brain will likely show enhancement of the optic nerve • Abnormal visual evoked potentials (VEP) Management • high-dose steroids Methylprednisolone pulse therapy is the standard treatment slightly shortens the time of recovery but does not prevent neurodegeneration and persistent visual impairment. • recovery usually takes 4-6 weeks • erythropoietin may have neuroprotective effects in autoimmune optic neuritis Prognosis • MRI: if > 3 white-matter lesions, 5-year risk of developing multiple sclerosis is c. 50% • Retrobulbar neuritis has the same systemic implications as optic neuritis, in that an episode of optic or retrobulbar neuritis can contribute to a diagnosis of multiple sclerosis

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Relative afferent pupillary defect • Also known as the Marcus-Gunn pupil, a relative afferent pupillary defect is found by the 'swinging light test'. • It is caused by a lesion anterior to the optic chiasm i.e. optic nerve or retina Causes • retina: detachment • optic nerve: optic neuritis e.g. multiple sclerosis Pathway of pupillary light reflex • afferent: retina → optic nerve → lateral geniculate body → midbrain • efferent: Edinger-Westphal nucleus (midbrain) → oculomotor nerve

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Swinging flashlight test & Relative afferent pupillary defect RAPD (Marcus Gunn pupil) • The Marcus Gunn pupil is a relative afferent pupillary defect indicating a decreased pupillary response to light in the affected eye • In the swinging flashlight test, a light is alternately shone into the left and right eyes. • A normal response would be equal constriction of both pupils, regardless of which eye the light is directed at. This indicates an intact direct and consensual pupillary light reflex. • When the test is performed in an eye with an afferent pupillary defect, light directed in the affected eye will cause only mild constriction of both pupils (due to decreased response to light from the afferent defect), while light in the unaffected eye will cause a normal constriction of both pupils (due to an intact efferent path, and an intact consensual pupillary reflex). Thus, light shone in the affected eye will produce less pupillary constriction than light shone in the unaffected eye. • A positive RAPD is due to retinal or optic nerve disease. due to the consensual response of the pupillary light reflex, shining light in the unaffected eye will produce bilateral miosis. shining light in the affected eye will not produce miosis because the afferent limb of the pupillary light reflex pathway is damaged (eg: optic neuritis) However, due to the bilateral projections of nerves from the Edinger-Westphal nucleus, light shined in the unaffected eye will produce bilateral miosis. This phenomenon is called a consensual response.

Herpes simplex keratitis • Herpes simplex keratitis most commonly presents with a dendritic corneal ulcer Features • red, painful eye • photophobia • epiphora • visual acuity may be decreased • fluorescein staining may show an epithelial ulcer (dendritic corneal ulcer) Management • immediate referral to an ophthalmologist • topical aciclovir

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Herpes zoster ophthalmicus • Herpes zoster ophthalmicus (HZO) describes the reactivation of the varicella zoster virus in the area supplied by the ophthalmic division of the trigeminal nerve. • It accounts for around 10% of case of shingles. Features • vesicular rash around the eye, which may or may not involve the actual eye itself • Hutchinson's sign: rash on the tip or side of the nose. Indicates nasociliary involvement and is a strong risk factor for ocular involvement Management • Oral antiviral treatment for 7-10 days, ideally started within 72 hours. Topical antiviral treatment is not given in HZO • oral corticosteroids may reduce the duration of pain but do not reduce the incidence of postherpetic neuralgia • ocular involvement requires urgent ophthalmology review Complications • ocular: conjunctivitis, keratitis, episcleritis, anterior uveitis • ptosis • post-herpetic neuralgia

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Blepharitis • Blepharitis is inflammation of the eyelid margins. • It may due to either meibomian gland dysfunction (common, posterior blepharitis) or seborrhoeic dermatitis/staphylococcal infection (less common, anterior blepharitis). • Blepharitis is also more common in patients with rosacea • The meibomian glands secrete oil on to the eye surface to prevent rapid evaporation of the tear film. Any problem affecting the meibomian glands (as in blepharitis) can hence cause drying of the eyes which in turns leads to irritation Features • symptoms are usually bilateral •

grittiness and discomfort, particularly around the eyelid margins • eyes may be sticky in the morning • eyelid margins may be red. Swollen eyelids may be seen in staphylococcal blepharitis • styes and chalazions are more common in patients with blepharitis • secondary conjunctivitis may occur Management • softening of the lid margin using hot compresses twice a day • mechanical removal of the debris from lid margins - cotton wool buds dipped in a mixture of cooled boiled water and baby shampoo is often used* □ *an alternative is sodium bicarbonate, a teaspoonful in a cup of cooled water that has recently been boiled • artificial tears may be given for symptom relief in people with dry eyes or an abnormal tear film

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Keratitis Definition • Keratitis refers to inflammation of one or more of the three corneal layers, the most common of which is epithelial keratitis. This is characterised by dendritic ulcers. Rarer forms involve the stroma or endothelium. Causes • Pseudomonas aeruginosa is commonly associated with contact lens related infections. • The management must also include advising the patient to discontinue wearing contact lenses and referral to a specialist ophthalmic unit. • Recurrence is common.

Keratitis overview Characteristic features Therapy Bacterial keratitis □ Most common form of keratitis (typically Staph. aureus □ ↑ Risk with wearing contact lenses Pseudomonas is seen □ Purulent discharge and/or hypopyon in contact lens □ Round corneal infiltrate or ulcer wearers.) Herpes zoster keratitis □ ↓ Corneal sensation □ Punctate lesions on the corneal surface (early disease) □ Vesicular eruption on forehead, bridge, and tip of the nose Herpes simplex Dendritic or geographic corneal ulcer Topical trifluridine or ganciclovir keratitis Acanthamoeba □ ↑ Risk with wearing contact lenses keratitis □ Corneal ring infiltrate Features • Red eye: pain and erythema (sharp ocular pain) • photophobia • blurred vision (in many cases). • Microbial keratitis, causing a white corneal infiltrate • foreign body, gritty sensation • hypopyon may be seen Dendritic ulcers • caused by herpes simplex virus. • Presentation is usually with pain, photophobia, blurred vision, conjunctivitis and chemosis. • Steroid eye drops are contraindicated as they may induce massive amoeboid ulceration and blindness. • treated with aciclovir eye drops, which should be continued for three days after the ulcer has healed. Notes & Notes for MRCP
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Topical broad-spectrum antibiotics (e.g., ciprofloxacin) □ Oral acyclovir, valacyclovir, or famciclovir □ Topical steroids Topical antiseptic (e.g., chlorhexidine) with propamidine

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Red eye There are many possible causes of a red eye. It is important to be able to recognise the causes which require urgent referral to an ophthalmologist. Below is a brief summary of the key distinguishing features Acute angle closure glaucoma • severe pain (may be ocular or headache) • decreased visual acuity, patient sees haloes • semi-dilated pupil • hazy cornea Anterior uveitis • Features □ acute onset □ pain □ blurred vision and photophobia □ small, fixed oval pupil, ciliary flush □ sign on ocular examination →Hypopyon • Iritis is associated with conditions such as: □

Reiter's Behcet's Psoriatic arthropathy (about 20%) inflammatory bowel disease. • Signs of anterior uveitis Keratic precipitates: (opaque aggregates of inflammatory cells deposited on the endothelium in anterior uveitis. They are typically located inferiorly. Cells +/- flare +/- fibrin in the anterior chamber Ciliary injection - localised conjunctival injection (redness) around the limbus Posterior synechiae - where part of the pupil margin becomes stuck to the lens Hypopyon (in severe anterior uveitis).

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Scleritis Definition • inflammation that occurs throughout the entire thickness of the sclera, Aetiology • may be underlying autoimmune disease e.g. rheumatoid arthritis Around 50% of patients with scleritis have an underlying disease, of which the majority are connective tissue disorders. Rheumatoid arthritis is the most common. Features • severe pain (may be worse on movement) and tenderness pain in scleritis is more evident and severe than episcleritis. Tenderness to palpation of the globe can differentiate it from episcleritis. After asking the patient to look down with eyelids closed, the physician gently presses the globe. Patients with scleritis have tenderness on palpation, while those with episcleritis do not. Unlike scleritis, patients with episcleritis do not complain of blurred vision or photophobia. Studies have shown that patients with RA-associated scleritis have more widespread systemic disease and a higher mortality rate than those episcleritis. • 50% of cases are bilateral. • Pain often radiates to the forehead, brow and jaw. This pain worsens with movement of the eye, and is classically worse at night. • There is associated watering, photophobia and a gradual decrease in vision (sometimes with diplopia). • Systemic symptoms such as fever, headache and vomiting can occur. • On examination the globe is tender, and the sclera can have a bluish tinge. • visual acuity is normal • there is marked dilatation of the deep and superficial scleral vessels. • Scleritis may cause thinning of the sclera (scleromalacia) and subsequent perforation. Treatment • Management ultimately depends on the underlying cause, but includes NSAIDs and prednisolone. • The patient should be referred urgently to the ophthalmology clinic • Application of topical phenylephrine 2.5% leads to blanching of episcleral vessels in episcleritis but not in scleritis.

Episcleritis Scleritis is painful, episcleritis is not painful • Results in ocular irritation with nodules. • acute in onset, with mild pain or discomfort / grittiness. • can be unilateral or bilateral, with localised or diffuse red eye. • There may be mild photophobia and watering. The lack of photophobia and discharge, and normal vision, makes episcleritis the most likely option

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Ocular manifestation of rheumatoid arthritis (see rheumatology)

Conjunctivitis • Purulent discharge if bacterial, clear discharge if viral • Viral conjunctivitis causes redness, soreness and watering. In severe cases it can cause a keratitis which may affect vision. It is highly contagious so patients should be advised to practise strict hand hygiene, to avoid sharing towels and to take time off work. It is a self-limiting disease which may take several weeks to resolve. Patients are treated with topical lubricants and some ophthalmologists give topical chloramphenicol to protect against secondary bacterial infections.

Subconjunctival haemorrhage • history of trauma or coughing bouts • adverse effect of aspirin therapy (and other antiplatelets). • It usually resolves over 10-14 days. • If the haematoma is large it may be worth considering prophylactic antibiotic eyedrops.

Posterior uveitis • Posterior uveitis describes inflammation of the choroid, which can involve the retinal vessels. • presents with gradual visual loss and floaters, which is often bilateral. • Discomfort and erythema are rare. • Slit light examination can demonstrate inflammatory lesions on the retina or choroid, with inflammation of the retinal vessels and oedema of the optic nerve.

Retinitis CMV Retinitis: causes hemorrhage at the edge of the area of retinal necrosis • Retinitis is inflammation of the retina in the eye, which may lead to blindness. • may be caused by several infectious agents, toxoplasmosis, cytomegalovirus and candida. • Cytomegalovirus retinitis is the most common cause of vision loss in AIDS patients. Toxocara retinitis • In retinitis due to Toxocara canis, there is usually only a single, well demarcated lesion.

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The slide shows the typical appearance of Toxocara retinitis with a lesion at the macula.

Retinitis pigmentosa Definition • Retinitis pigmentosa is a degenerative disease involving retinal receptors and pigment cells. Pathophysiology • degeneration of rod photoreceptor cells in the retina night blindness and low peripheral vision There are two types of photoreceptors, called rods and cones. Rods are in the outer regions of the retina, and allow us to see in dim and dark light. Died early night blindness Cones reside mostly in the central portion of the retina, and allow us to perceive fine visual detail and color. Died in the late stages Features • night blindness is often the initial sign • funnel vision (the preferred term for tunnel vision) • fundoscopy: black bone spicule-shaped pigmentation in the peripheral retina, mottling of the retinal pigment epithelium Associated diseases • Refsum disease: cerebellar ataxia, peripheral neuropathy, deafness, ichthyosis • Usher syndrome • abetalipoproteinemia • Lawrence-Moon-Biedl syndrome • Kearns-Sayre syndrome

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• Alport's syndrome • mitochondrial myopathy • drug-induced Thioridazine (typical antipsychotic drug belonging to the phenothiazine group and was previously widely used in the treatment of schizophrenia and psychosis; withdrawn worldwide in 2005 because it caused severe cardiac arrhythmias,) It is important to differentiate this from corneal deposits that may develop with the use of chlorpromazine. Thioridazine retinal deposits (retinitis pigmentosa). chlorpromazine corneal deposits Fundus showing changes secondary to retinitis pigmentosa

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Sudden painless loss of vision Causes Notes Central retinal vein occlusion • Incidence increases with age • More common than arterial occlusion • Causes : glaucoma, polycythaemia, hypertension, DM • Features: afferent pupillary defect On fundoscopy: widespread dot-and-blot and/or flame-shaped hemorrhages in all four retinal quadrants Cotton wool spots characterized by yellow-white deposits on the retina caused by swelling of retinal nerve fibers due to ischemia Severe macular edema and papilledema • Fluorescein angiography: in order to differentiate ischemic from nonischemic forms of retinal vein occlusion

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Causes Notes Branch retinal vein occlusion • Features: Usually asymptomatic No afferent pupillary defect the hemorrhages are found in a single zone. Central Retinal artery occlusion • Causes: thromboembolism (from atherosclerosis) or arteritis (e.g. temporal arteritis) • Features: Sudden, painless loss of vision in one eye (often described as a “descending curtain”) afferent pupillary defect, history of amaurosis fugax, often describes as a 'black curtain' descending over the vision. On fundoscopy: 'cherry red' spot on a pale retina, Grayish-white (cloudy) discoloration of the entire retina Cherry-red spot at the fovea centralis Branch retinal artery occlusion • Features: Sudden onset of visual field defects (scotomas) in the affected eye No afferent pupillary defect, On fundoscopy: Grayish-white discoloration of the retinal quadrant supplied by the affected vessel Retinal detachment • Risk factors: Previous intraocular surgery (e.g., cataract surgery), posterior vitreous detachment • Most commonly due to retinal tears → retinal fluid, which is formed by vitreous degeneration, seeps into the subretinal space → retinal detachment • Features: Prodromal symptoms: result from posterior vitreous detachment (floaters, flashes of light (photopsia) Localized retinal detachment: scotoma (visual field defect): Dense shadow that starts peripherally progresses towards the central vision Straight lines appear curved Extensive retinal detachment and/or macular involvement: Central visual loss (often described by patients as a curtain descending over their field of vision) Fundoscopy: A freshly detached retina has a grey color instead of the normal pink color and may appear crinkled. A retinal tear may be visible Vitreous haemorrhage • Causes : bleeding disorders, DM → Proliferative retinopathy → rupture fragile neovascular vessels (most common cause) • Features: Large bleeds cause sudden visual loss Moderate bleeds may be described as numerous dark spots Small bleeds may cause floaters Fundoscopy: inability to visualise the retina Amaurosis fugax • Definition: sudden, painless loss of vision that lasts for seconds to minutes and is followed by spontaneous recovery (mostly unilateral) • Cause: retinal ischemia following transient occlusion of the central retinal artery by microemboli • Complications: Transient ischemic attacks (TIA)

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Posterior vitreous detachment • Occur in up to 50-75% of the population over 65 years • Features: Flashes of light (photopsia) - in the peripheral field of vision Floaters, often on the temporal side of the central vision • Complications: Retinal tears/holes, retinal detachment, Vitreous hemorrhage Images Central vein occlusion: Flame-shaped hemorrhage is visible in all four retinal quadrants. Central retinal artery occlusion: Narrow retinal arteries and a pale retina with early signs of nerve fiber layer edema are visible. The fovea centralis appears red (cherry-red spot; due to the transparency of the well-vascularized choroid, as no nerve fibers are present in the fovea avascular zone. Therefore, there is no edema. Retinal detachment: The retina is visible as a yellow-grey, bullous elevation in the upper part of the image.

- Green overlay: detached retina
- Red overlay: tear Fundus Photograph of Vitreous Haemorrhage

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An elderly patient with acute visual loss has giant cell arteritis until proved otherwise The history of diabetes, complete loss of vision in the affected eye and inability to visualise the retina point towards a diagnosis of vitreous haemorrhage. Nasal branch retinal vein occlusion sudden blurring (not total visual loss) of the temporal field in the affected eye. Of all types of retinal vessel occlusion, ischemic Central Retinal Vein Occlusion is most commonly associated with neovascularization.

Tunnel vision Tunnel vision (also known as Kalnienk vision) is the loss of peripheral vision with retention of central vision, resulting in a constricted circular tunnel-like field of vision. Causes • papilloedema • glaucoma • retinitis pigmentosa • choroidoretinitis • optic atrophy secondary to tabes dorsalis • hysteria

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Ectopia lentis Ectopia lentis/subluxation of the lens is associated with: • Ehlers-Danlos syndrome • Marfan's syndrome • Weill-Marchesani syndrome (short stature, skeletal abnormalities and ectopia lentis), and • Refsum's disease.

Fundoscopic features in eye infections • Cytomegalovirus (CMV) retinitis secondary to human immunodeficiency virus (HIV) Fundoscopy of the left eye revealed an extensive 'brushfire-like' lesion in the major superior temporal arcade with a large patch of white fluffy lesion mixed with extensive retinal haemorrhages. • Ocular histoplasmosis and syphilitic choroiditis would give a fundus picture of multiple whitish lesions. • Syphilitic neuroretinitis would normally give a picture of a macular star exudation. • Tuberculous periphlebitis gives a picture of perivenous sheathing and minimal retinal haemorrhages.

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Eye signs in Systemic diseases • Lisch nodules of the iris are golden nodules occurring bilaterally in the teenage years onwards in Neurofibromatosis type 1 (NF-1). Axillary freckles appear at 10 years of age, while cafe au lait spots increase in size and number throughout childhood. • Brushfield spots of the iris are found in people with Down syndrome. • Kayser-Fleischer rings are due to copper deposition in Descemet's membrane of the cornea. • Band keratopathy is caused by calcium deposition in Bowman's layer of the cornea. Patients who present with band keratopathy should have a serum calcium and phosphate level • Ectopia lentis with aortic regurgitation Marfan syndrome (Lens dislocation (classically upwards)). Inferior dislocated lens consistent with a diagnosis of homocytinstinuria. • Roth's spots haemorrhages in the retina associated with subacute bacterial endocarditis. also, seen in leukaemia. • 'black sunburst' - a chorioretinal scar, which is one of the commoner retinal manifestations of Sickle cell disease (SCD) and pathognomonic.

Hyphaema Overview • Occurs when bleeding from iris vessels fills the anterior chamber with blood and if there is enough blood • the main risk in the acute stage is of raised intraocular pressure (IOP). • It is usually caused by trauma - often small objects (champagne corks, squash balls) hitting the eye. Treatment • Strict rest is vital if a hyphaema is present, as there is an increased risk of a second bleed in the initial period. • Intravenous carbonic anhydrase inhibitors is the most appropriate treatment • Aspiration may be required to prevent loss of vision. • avoid drops that dilate the pupil (such as anticholinergics) the iris remains stable and a second bleed is therefore less likely. The slide shows hyphaema: blood in the anterior chamber.

Third edition

Notes & Notes

For MRCP part 1 & 11

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Pharmacology

Updated

By

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Pharmacology

Basic pharmacology

Pharmacokinetics: metabolism

• Drug metabolism usually involves two types of biochemical reactions - phase I and phase II reactions. • The majority of phase I and phase II reactions take place in the liver • Phase I reactions: oxidation, reduction, hydrolysis.
 Mainly performed by the P450 enzymes but some drugs are metabolised by specific enzymes, for example alcohol dehydrogenase and xanthine oxidase.
 Products of phase I reactions are typically more active and potentially toxic • Phase II reactions: conjugation.
 Products are typically inactive and excreted in urine or bile.
 Glucuronyl, acetyl, methyl, sulphate and other groups are typically involved

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Drug absorption • Diffusion.
 Most drug absorption in the gastrointestinal tract occurs by diffusion.
 For diffusion to occur: the drug must be dissolved so that individual drug molecules come into contact with the gut epithelium,
 the drug must be lipid soluble so that it can cross the cell membrane. Because the cell membrane is lipoid, lipid-soluble drugs diffuse most rapidly.
 Drugs that are not ionized are lipid soluble and most likely to be well absorbed from the gastrointestinal tract. The ionized form has low lipid solubility (but high water solubility—ie, hydrophilic) and high electrical resistance and thus cannot penetrate cell membranes easily. • Theoretically, weakly acidic drugs (eg, aspirin) are more readily absorbed from an acid medium (stomach) than are weakly basic drugs (eg, quinidine). However, whether a drug is acidic or basic, most absorption occurs in the small intestine because the surface area is larger and membranes are more permeable

In the elderly population, phase I reactions will usually become impaired before phase II reactions.

Lipid soluble drug vs lipid insoluble drug lipid soluble drug lipid insoluble drug have good gastrointestinal absorption have poor gastrointestinal absorption can be given orally may need to be given parenterally will be widely distributed in the body (large volume of distribution) has limited distribution (may not cross bloodbrain barrier or placenta and less likely to be stored in fat tissue) usually requires metabolism before elimination (to decrease lipid solubility) often have a long plasma half-life (prolonged by ‘reservoir’ of drug in tissues and by requirement for metabolism). MRCPUK-part-1-Sep 2017: What is the mechanism that make salmeterol acts as a LABA? Its long duration results from its high lipid solubility Lipophilic, Hydrophilic and Amphiphilic Chemical nature Clinical significance Example • can easily diffuse across the lipid bilayer of the cell membrane. Lipophilic • Predominantly nonpolar compounds can be administered topically can across the bloodbrain barrier • Metabolised in the liver and then excreted through the bile duct • can only cross the lipid bilayer via facilitated transport • Smaller hydrophilic molecules can diffuse along a concentration gradient through pores in the membrane • eliminated by the kidneys Hydrophilic • Predominantly polar compounds Amphiphilic • Both lipophilic and hydrophilic Local anesthetics, e.g., lidocaine Drug metabolism in patients with advanced liver disease • Plasma

proteins fall in liver disease and may negatively affect drug distribution • Both intrahepatic and extrahepatic cholestasis may affect the metabolism of drugs that are actively secreted into bile, eg ciprofloxacin • Conjugation reactions are affected to a lesser extent by advanced liver disease and only occur in very late stage disease Pharmacokinetics in chronic renal failure • Renal failure disturbs virtually every kinetic parameter including: gastric absorption hepatic metabolism of some drugs protein binding volume of distribution Notes & Notes for MRCP
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may be eliminated without metabolism often have a short plasma half-life as elimination does not require metabolism. • Scopolamine (hydroscine) Tertiary amine Used to treat motion sickness • Butylscopolamine (hyoscine butylbromide) Quarternary amine Used as an antispasmodic to treat GI colic

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