



Episode 9 – Non-traumatic Eye Emergencies

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General Approach:

Characterization of a patient's eye pain helps determine the location of the pathology:

(a) up-front, scratchy, 'sand-in-the-eye' pain relates to anterior structures such as the lids, conjunctivae, cornea, and foreign bodies;

(b) deep, boring, 'behind-the-eye' toothache pressure-type pain relates to posterior structures;

(c) photophobia relates to uveal tract pathology such as iris, ciliary body and choroid

Systematic eye exam for every patient with an eye complaint: 6 parts - visual acuity (the '6th vital sign'), visual fields by confrontation, size and reactivity of pupil, extraocular movements (for cranial nerve palsies), anterior segment (slit-lamp exam) and posterior segment (fundoscopy)

The painful red eye

Acute angle-closure glaucoma:

Characterized by at least 2 of the following symptoms – acute onset ocular pain with nausea vomiting, intermittent blurring of vision with halos seen around lights (due to corneal edema), photophobia, and visual loss – along WITH at least 3 of the following signs – intraocular pressure (IOP) >21 mmHg (often 40-80 mmHg), conjunctival injection, corneal epithelial edema causing haziness, fixed and mid-dilated pupil, and shallow anterior chamber (on *oblique flashlight test* with shadow medial to pupil caused by the bulging anterior chamber when the light is flashed from the patient's temporal side)

Risk factors include elderly patients who are far-sighted (hyperopic), and *triggers* include transition from light to dark environment (causing mydriasis) as well as many medications – anticholinergic, antimuscarinic, antidepressant, antipsychotic, antihistaminic, and sympathomimetics (most of them through mydriasis)

ED management (re-assess IOP q15min in the early phases of treatment):

Topical beta-blocker (e.g. 1 drop of Timolol 0.25-0.5%) to decrease aqueous humour production if no contraindications present (COPD, asthma, bradycardia)

Topical cholinergic (1-2 drops of Pilocarpine q15min x2 doses) to cause myosis, decrease angle of the anterior chamber at the trabecular meshwork level, and increase drainage

Parenteral agents: Acetazolamide 500mg IV (or PO) to decrease aqueous humour production, and add Mannitol 1.25-2g/kg IV of 20% solution to exert an osmotic diuresis if a response with other drugs does not occur

When is acute angle-closure glaucoma missed? (a) Elderly, disabilities, dementia, psychiatric pts where a thorough history and physical may be challenging (b) pts who present primarily with headache or abdo pain and vomiting

Consider acute angle-closure glaucoma in any pt who has had recent eye surgery

Anterior uveitis or iritis:

Symptoms – deep, aching pain radiating to periorbital or temporal area, worse with eye movements and accommodation, and with PHOTOPHOBIA (very important)

Signs – *ciliary flush* (erythema that is worse closer to the iris, and not in the periphery as in conjunctivitis), pain relief with cyclolegics but not with topical anesthetics, and *consensual photophobia* (light in the unaffected eye, because it causes bilateral myosis, causes pain), and abnormal slit lamp exam with *keratitic precipitates* or *hypopyon*, and *cells and flare* in the anterior chamber, which scintillate for milliseconds at a time like ‘snowflakes in the car’s headlights’ when driving at night under an oblique slit lamp light

Important diagnosis to make in order to prevent long-term vision loss consequences due to scarring, malfunction in the shape of the iris, and the increased likelihood of retinal detachment, and also to screen for the 50% of cases that have underlying infectious or autoimmune diseases such as herpes, tuberculosis, syphilis, toxoplasmosis, as well as the seronegative spondyloarthropathies – inflammatory bowel disease, ankylosing spondylitis, reactive arthritis, psoriatic arthritis; also Behçet, rheumatoid arthritis, SLE, sarcoidosis, and scleroderma

ED management:

Cycloplegic drops to prevent myosis, which acts as both pain control and breaking up of the synechiae caused around the iris – Homatropine (1 drop TID of 2-5% solution) lasts a few days and is probably best, or Cyclopentolate (1 drop TID of 0.5-2% solution), which lasts 1 day; Mydriacyl, which lasts only 2-3hrs, is good for diagnosis but not for treatment

Steroids should only be used in conjunction with ophthalmologist consultation due to the concern for worsening of underlying infection or development of corneal ulcer, both of which may lead to loss of vision

Ophthalmological follow-up within 24hrs

The painless red eye

Kawasaki disease:

Acute systemic vasculitis of mucosal vessels of unknown etiology in children <5yo leading to coronary artery aneurysm if not treated acutely – the criteria is remembered with the mnemonic *Warm C-R-E-A-M*

Warm - Fever >39°C for 5 days, PLUS 4 of the 5 following signs (or less in ‘atypical’ form of the disease):

C - Conjunctivitis, bilateral non-exudative

R – Rash, polymorphic (e.g. urticarial, maculopapular) and on the body (not vesicular nor bullous)

E – Edema or erythema of the hands, eventually leading to desquamation

A - Adenopathy with at least one anterior cervical lymph node >1.5cm (not generalized over the whole body)

M – Mucosal involvement: fissured red lips, strawberry tongue

Incomplete Kawasaki: American Guidelines - “If the typical clinical findings of Kawasaki disease are present in a child with fever for less than five days, or if three classic features plus coronary artery findings on echocardiography are found, the diagnosis still can be made and treatment initiated”

Features that go *against* the diagnosis of Kawasaki: exudative conjunctivitis or pharyngitis, discrete intra-oral lesions, bullous or vesicular rash and generalized lymphadenopathy

Pearls: almost always look unwell/very irritable, fever is minimally responsive to antipyretics, often have peri-anal erythema, and there is often a sharp demarcation at the wrist and ankles from the redness in the extremities

Abnormal tests – High platelets, WBC, ESR and CRP, LFTs (especially AST/ALT), sterile pyuria in urine (WBC present but no bacteria), as well as normochromic, normocytic anemia and hyponatremia

Treatment – High dose ASA and IVIG, best if treat within 10d of onset of symptoms (lower complication rate)

Painless loss of vision

Ddx of painless acute visual loss not related to trauma includes vascular occlusion, retinal detachment, vitreous hemorrhage, macular disorders, neuro-ophthalmologic disease, hysteria, and secondary to methanol or viagra

Pearls – loss of vision over seconds is usually due to a vascular etiology, such as ischemia of the retina; posterior vitreous detachment will often cause debris to obstruct the light but the patient can see through it, vs. retinal detachment whereby there is a frank field deficit

Temporal arteritis:

Vasculitis of autoimmune etiology affecting the arterial branches of the internal carotid artery (ophthalmic and temporal arteries) that is often preceded by features of polymyalgia rheumatica (PMR)

Definition from the American College of Rheumatologists: age over 50, new headache, abnormality of the temporal artery (tenderness or pulselessness), ESR >50, and positive temporal artery biopsy; 3 out of 5 positive criteria result in a sensitivity of 93% for the diagnosis, and a specificity of 91%

Symptoms – jaw claudication and diplopia are most predictive, but also temporal area pain and subacute peripheral vision loss, low grade fever, constitutional symptoms lasting weeks to months (anorexia, fatigue, myalgias, night sweats, weight loss), ataxia, decreased hearing

Signs – beading, prominence or tenderness of the temporal artery is the most predictive, but also temporal scalp area tenderness, hyperesthesia and hypersensitivity, and vestibular or hearing impairment at time of presentation

Think of temporal arteritis in elderly patients who present 'Weak & Dizzy' because often symptoms are vague

ESR and CRP – Normal ESR values increase with age ($\text{age} \div 2$ for men, and $[\text{age} + 10] \div 2$ in women), and biopsy-positive temporal artery cases with normal ESR values are not uncommon, but the combination of normal ESR *and* CRP has a sensitivity of 99% in ruling out the diagnosis

Do not rely on a normal ESR alone to rule out temporal arteritis

Treatment – Steroids, with PO formulation if no ocular symptoms are present and IV if they are; do not delay treatment even if biopsy cannot be performed for a few days as earlier treatment has been shown to lead to better improvement (60% if treated within 24hrs of symptoms, vs. only 5% if treated after 24hrs)

Central Retinal Artery Occlusion (CRAO) – first image:

Sudden abrupt painless vision loss (i.e. stroke of the retina, with the same car-diovascular risk factors), either complete (with light-dark perception only over the whole visual field), or with central vision retained (due to the different vas-cular distribution of the fovea)

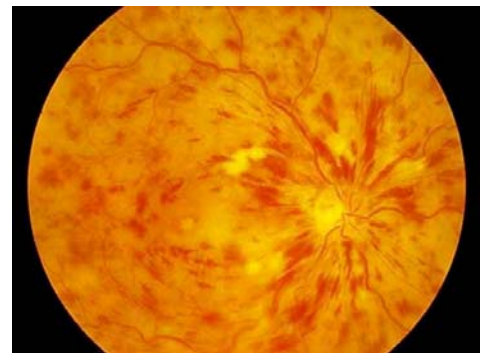
Signs – Positive RAPD (rapid afferent pupillary defect) with a blanched, non-perfused retina with the pathognomonic cherry-red spot, i.e. the red fovea underlying the almost transparent retina, lateral to optic disk

Management – Time-sensitive goal to restore blood flow and remove obstruction through vigorous massage of eyeball to break the clot down and send it downstream to convert it to a branch occlusion, antiplatelet therapy (ASA), and decrease IOP (timolol, acetazolamide, and mannitol), possibly thrombolytics on a case-by-case basis, and breathing into a paper bag 10min each hour to increase the CO₂ with resultant arterial vasodilation



Central Retinal Venous Occlusion (CRVO) – second image:

“DVT of the eye”, vascular engorgement (gradual changes); tell-tale – big angry stormy eye on fundoscopic exam; hyperaemic, ‘blood and thunder’ exam



Painful loss of vision

Optic neuritis:

Inflammation of the optic nerve (due to demyelination), usually in women 15-45, causing the classic triad of unilateral loss of vision, eye pain (especially with eye movement) and dyschromatopsia (washed out colors, especially red, as if looking through frosted glass)

Signs – Markedly decreased visual acuity (often to light perception only), with a normal eye and fundoscopic exam – “the patient sees nothing, and you see nothing on exam” (may sometimes see blurred disk margins and swollen veins), with positive RAPD (the pupils paradoxically dilate when the flashlight is swung from the unaffected to the affected eye due to the decreased light perception; also seen in vitreous hemorrhage, retinal detachment or ischemia, and retrobulbar hemorrhage); If plaques are seen on MRI, more likely to develop multiple sclerosis

Treatment – The Optic Neuritis Treatment Trial showed that IV methylprednisolone 250mg qid x 3d followed by an oral prednisone taper over 1wk restores vision more quickly, and delays the development of multiple sclerosis by 2yrs

Pearls from the experts

Infectious conjunctivitis: The most important part of management is irrigation and washing out of the eye

Contact lens wearers should be prescribed anti-pseudomonals such as Tobramycin (aminoglycoside), or Ciprofloxacin

Suspect gonococcal infection with recent unprotected sexual intercourse, especially if there is substantial purulent discharge that re-accumulates rapidly after wiping, or in infants 3-6 days of life with red eyes after vaginal birth, which require admission and IV antibiotics

4 non-traumatic eye emergencies that require prompt ophthalmological consultation:

Acute angle-closure glaucoma, severe uveitis, acute loss of vision (from CRAO, temporal arteritis, retinal detachment or optic neuritis), and significant corneal ulceration of >1mm in length

Terson syndrome - third image: In subarachnoid hemorrhage (SAH), the sudden rise in intracranial pressure may lead to retinal veins rupture, resulting in a subhyloid hemorrhage with boat shape from central disk on funduscopy (pathognomonic for SAH)

