Update on Ophthalmology Basics

Neeti Alapati, MD
KU EYE
Glaucoma / Anterior Segment Services
neetid@kumc.edu
Objectives

• Review of common ophthalmic diseases
• Initial treatment options
• Identify when to refer to Ophthalmology Clinic
Basic Eye Anatomy
Basic Eye Anatomy

https://www.researchgate.net/figure/The-anatomy-of-ocular-system-the-anterior-segment-involves-conjunctiva-ciliary-body_fig1_325339781
Cornea and External Disease: Preseptal Cellulitis

- Erythema, edema, and warmth of the eyelid
- Absence of Orbital Signs: Normal vision, EOM, pupillary responses, quiet conjunctiva, red color saturation, and absence of proptosis

https://www.reviewofoptometry.com/article/put-a-lid-on-preseptal-cellulitis
Cornea and External Disease: Orbital Cellulitis

- Erythema, edema, warmth of eyelid

- +Orbital signs: decreased vision, restrictive EOM + pain, APD, Chemosis, red color desaturation

- 10% of patients develop subperiosteal abscess or orbital abscess

Cornea and External Disease: Preseptal/Orbital Cellulitis

• Etiology: Most often bacterial
  – Gram Positive Cocci – Staph / Strep
  – Less commonly viral: HSV, VZV, Adenovirus
  – Children <9 typically one organism, >9 y/o polymicrobial

• Work-up: CT Orbits + Max/Face
Cornea and External Disease: Preseptal Cellulitis

• Treatment:
  • Mild cases: outpatient, daily monitoring
    – Ampicillin, Amoxicillin-clavulanate, Fluoroquinolones, Azithromycin, Clindamycin
    – If concern for MRSA: Bactrim, Rifampin, Clindamycin, Fluoroquinolones
  • If no response within 48 hours, development of orbital signs, children <2, or severe cases should be imaged and treated inpatient with IV antibiotics
Cornea and External Disease: Orbital cellulitis

- Treatment
  - Admission for broad spectrum IV antibiotics
    - Drug choice depends on age and organism of concern
  - If no improvement within 24-48 hours, reimage with contrast enhanced CT to look for subperiosteal or orbital abscess

http://www.ajnr.org/content/cow/07132009
Cornea and External Disease: Corneal Abrasion

- Corneal epithelial defect often associated with pain and at times decreased vision, conjunctival injection, photosensitivity, tearing

- Causes:
  - Mechanical
  - Dryness related
  - Neurotrophic disease
  - Post-surgical
  - Chemical injury
Cornea and External Disease: Corneal Abrasion

• **Exam:**
  - Focal area of fluorescein uptake
  - Conjunctival Injection
  - Corneal edema

• **Treatment:**
  - Antibiotic ointment
  - Antibiotic drops
  - Bandage contact lens
  - Pressure Patching
  - *Avoid topical anesthetics*

https://www.guidelinesinpractice.co.uk/eye-ear-nose-and-throat/red-eye-whats-the-diagnosis/454421.article
Cornea and External Disease: Corneal Abrasion

- Prevention:
  - Encourage patient to use protective eye wear with high risk activities: lawn mowing, painting, working with cars

- Beware: given mechanism of injury, always keep in mind that patient could have corneal laceration (ruptured globe) or corneal ulcer
  - Peaking of pupil, significant vision changes, large area of bullous subconjunctival hemorrhage

https://www.potthoffeyecare.com/2018/10/05/can-an-eye-explode/
Cornea and External Disease: Chemical Burn

- Injury to the conjunctiva and/or cornea by a chemical

- An ophthalmic emergency, requires immediate attention

- Early intervention can prevent possible blindness

Cornea and External Disease: Chemical Burn

- Represents 11.5%-22.1% of all ocular traumas
- Two third of cases are young men and children 1-2 years of age
- Alkali agents more common in building materials and cleaning agents
  - most common culprit

http://morancore.utah.edu/basic-ophthalmology-review/chemical-burns/
Cornea and External Disease: Chemical Burn

• Alkali —
  – Lipophilic and can penetrate tissues more rapidly
  – Saponify fatty acids of cell membranes, can penetrate corneal stroma

• Acids —
  – Less harmful
  – Denature and precipitate proteins in tissues
  – Coagulated proteins act as barrier and prevent further penetration
Cornea and External Disease: Chemical Burn

- Prevention is key!!!!!! Protective eyewear!!!

- Before exam, check pH – normal 7.0-7.2

- Wait at least 5 minutes post irrigation to re-check pH

https://www.ecmag.com/section/safety/easy-eyes-eye-protection
Cornea and External Disease: Chemical Burn

• Have patient up right and tilt head towards affected side

• Instill topical anesthetic

• Administer fluid nasal to lateral, pouring away from non-affected eye

• Have patient blink frequently during irrigation and to look in all directions to ensure all areas are irrigated
Cornea and External Disease: Chemical Burn

- Morgan Lens: IV tubing can connect to this lens and allow easier irrigation

https://www.youtube.com/watch?v=zmXqunkuVR4
Cornea and External Disease: Chemical Burn

- Refer to eye clinic or send to ER with ophthalmologist present same same day.
- Treat with Antibiotics, Cycloplegics, Artificial Tears, Steroid Drops.
- In severe cases: Ascorbic Acid (promotes collagen synthesis), Doxycycline (reduces MMP which degrade collagen), Medroxyprogesterone.
Corneal and External Disease: Chemical Burn

- Debridement of Necrotic Epithelium
- Amniotic Membrane Transplant
- Limbal Stem Cell Transplant
- Oral Mucosa Transplant
- Boston Keratoprosthesis
Anterior Segment and Lens

- **Uveitis** – inflammation within the uveal tract (iris, ciliary body, choroid)

- Caused by a conglomerate of etiologies
  - Inflammatory
  - Infectious
  - Traumatic
  - Malignancy
  - Medication Induced
  - Idiopathic

Anterior Segment and Lens

• Classified by location of involvement:
  – Anterior
  – Intermediate
  – Posterior

• Symptoms:
  – Pain
  – Redness
  – Photophobia
  – Decreased vision
  – Tearing

https://www.healthline.com/health/uveitis#pictures
Anterior Segment and Lens

- **Exam Findings:**
  - Anterior chamber cell
  - Keratic precipitates
  - Flare
  - Hypopyon
  - Iris nodules
  - Posterior Synechiae

- **Posterior / Intermediate segment findings**
  - Vitritis (snowballs/snowbanking)
  - Retinal hemorrhages
  - Retinal Whitening
  - Vitreous Hemorrhage
  - Macular Edema
  - Retinal Detachment
Anterior Segment and Lens

- Work-up depends on location of involvement and risk factors

- Infectious: Syphillis, HSV, Tb, CMV, Toxo, Rubella, Lyme

- Inflammatory: Sarcoidosis, RA, HLA-B27 associated, Behcets, Vogt-koyangi-harda Syndrome, TINU, Psoriatic Arthritis

- Malignancy – lymphoma, retinoblastoma, iris melanoma, etc
Anterior Segment and Lens

• Treatment:
  – Topical, periocular/intravitreal, and or oral steroids
  – Cycloplegia
  – Treatment of underlying disease!!!!

• If unable to get off topical steroids / posterior involvement, coordination with rheumatology:
  – Systemic immunosuppression: Azathioprine, Methotrexate, Tacrolimus
  – Biologics: Adalimumab, Infliximab, Etanercept
Anterior Segment and Lens

- Cataract: most common types
  - Nuclear sclerosis
  - Cortical
  - Posterior Subcapsular
    - Can have more acute onset

- Common Symptoms
  - Decreased vision
  - Decreased contrast sensitivity
  - Haloes and/or glare around lights

https://www.webmd.com/eye-health/cataracts/ss/slideshow-cataracts
Anterior Segment and Lens

- Risk Factors
  - Age
  - Diabetes
  - Chronic steroid use
  - Radiation
  - Ultraviolet light exposure
  - Ocular trauma
  - Prior ocular surgery
  - Genetic Predisposition

https://www.diabetes.co.uk/diabetes-causes.html

https://www.valleymed.com/ct_detail.html?pgguid=26f09ba1-c7c1-48ea-a624-f0e4860cd64a
Anterior Segment and Lens

- Treatment: most often outpatient surgery, one eye done at a time
- Most often done under topical anesthesia with MAC
- Rarely need to stop blood thinners
- Patients on chemotherapy are ideally done on non-infusion weeks

https://www.corneaconsultants.com/cataracts/
Posterior Segment

• Diabetic Retinopathy
  – Microvascular end organ disease due to diabetes
  – Leading cause of blindness worldwide in 25-74 year olds
  – Wisconsin Epidemiologic Study of Diabetic Retinopathy showed after 20 years
    • Type 1: 99% will have some degree of retinopathy
    • Type 2: 60% will have some degree of retinopathy

https://www.frontiersin.org/files/Articles/374639/fphys-09-00820-HTML/image_m/fphys-09-00820-g003.jpg
Risk Factors for diabetic retinopathy:

- Duration of disease
- Uncontrolled glucose and blood pressure control
- HTN
- Dyslipidemia
- Ethnicity
- Pregnancy
- Smoking

https://www.eyecarevisioncenter.com/eye-health/diabetic-retinopathy/
Posterior Segment

• Two Primary Types:
  – Proliferative (PDR) vs Non-Proliferative Diabetic Retinopathy (NPDR)
    • Distinguishing feature is presence of ischemia resulting in proliferation of abnormal blood vessels
  – Exam findings:
    • NPDR: Retinal Microaneurysms, Intraretinal Hemorrhages, Exudates, Cotton Wool Spots
    • PDR: above + neovascularization (iris, retina, optic disc)
NPDR

https://imagebank.asrs.org/file/5343/severe-npdr
PDR

https://retinavitreous.com/diseases/dm_pdr.php

Posterior Segment

• Diabetic Retinopathy:

• Prevention: optimizing glycemic and blood pressure control

• Every 1 point reduction in HbA1c results in 21% reduction in any end point related to diabetes
Posterior Segment

• Treatment:
  – Mostly for PDR, Diabetic Macular Edema, and High-risk NPDR
    • Laser – panretinal photocoagulation (PRP), focal-grid laser
    • Intravitreal Injections – Anti-VEGF (Bevacizumab, Ranibizumab, Afibercept)
    • Topical, periocular, intravitreal steroids
    • Surgical – Pars Plana Vitrectomy

https://en.wikipedia.org/wiki/Diabetic_retinopathy#/media/File:Fundus_photo_showing_scat
tter_laser_surgery_for_diabetic_retinopathy_EDA09.JPG
Posterior Segment

• Hypertensive Retinopathy
  – Typically asymptomatic, occasional blurred/decreased vision

• Signs:
  – Arteriolar narrowing, AV nicking, microaneurysms, dot-blot hemorrhages, cotton wool spots, exudates, optic disc edema

• Treatment: control blood pressure
Posterior Segment

http://morancore.utah.edu/basic-ophthalmology-review/hypertensive-retinopathy/
Optic Nerve

• Glaucoma: Progressive optic neuropathy associated with visual field changes due to retinal ganglion cell loss

• *Intraocular pressure is a risk factor but is not mandatory*

• A large array of diseases, divided into primary vs secondary glaucoma and open vs closed angle
Optic Nerve

– Typically asymptomatic until disease advanced

– Risk factors: age, race, intraocular pressure, central corneal thickness, family history

– Low or normal pressure glaucoma association:
  • Migraines, OSA, Anemia, Nocturnal Hypotension, Raynauds
Optic Nerve

- Exam findings: enlargement of the optic cup, vessel bayonetting, disc hemorrhages

https://www.glaucoma.org/glaucoma/optic-nerve-cupping.php
Optic Nerve

- Diagnosis is made using a combination of:
  - Exam findings
  - Visual field changes
  - Optical coherence tomography (OCT)

https://www.hopkinsmedicine.org/wilmer/services/glaucoma/book/ch06s04.html
Optic Nerve: Glaucoma

- **Treatment:**
  - **Topical Ocular Anti-hypertensives:**
    - **Beta-blockers:** timolol, betaxolol, cartelol etc
      - avoid in those w/ asthma/COPD, can cause bradycardia, arrhythmia, impotence, fatigue and increased falls
    - **Alpha-agonists:** brimonidine, apraclonidine
      - Can cause dry mouth and fatigue
      - Hypertension, tachycardia, arrhythmia
      - *Avoid in infants and children:* associated with CNS depression
    - **Prostaglandins:** latanoprost, bimatoprost, travaprost
      - Least systemic side effects, occasional nightmares / psych changes in elderly patients
    - **Carbonic Anhydrase Inhibitors:** dorzolamide, brinzolamide
      - Sulfa derivatives
Optic Nerve: Glaucoma

• Treatment:
  – Systemic:
    • Oral Carbonic Anhydrase Inhibitors: acetazolamide, methazolamide
      – Decrease aqueous production
      – Metabolic acidosis, potassium depletion, fatigue, parenthesis, metallic taste with soda, fatigue, kidney stones, GI symptoms
    • Hyperosmotic Agents – oral glycerine and mannitol IV
      – Decreases vitreous volume
      – Can cause headaches, back pain, diuresis, circulatory overload with angina, pulmonary edema, heart failure, seizure, and cerebral hemorrhage
Optic Nerve: Glaucoma

• Treatment:
  – Lasers
  – Minimally Invasive Procedures: iStent Inject / Goniotomy, etc
  – Filtration Procedures: trabeculectomy, tube shunt

  – *Some procedures do necessitate cessation of anticoagulation!*

    • Without anticoagulation, there can be uncontrollable intraoperative bleeding. Most concerning would be suprachoroidal hemorrhage that can rapidly lead to blindness
Optic Nerve: Anterior Ischemic Optic Neuropathy

- Ischemia of the optic nerve caused by a myriad of etiologies, exact mechanism uncertain
  - Non-arteritic (NAION) – associations: OSA, Nocturnal Hypotension, Meds (PDE5, INF-alpha), optic disc drusen
  - Arteritic – most often Giant Cell Arteritis (GCA) – inflammation and thrombosis of the short posterior ciliary arteries

Optic Nerve: Anterior Ischemic Optic Neuropathy

- NAION: Unilateral, painless vision loss

- AAION: Acute, painful vision loss, typically in those older than 50 y/o

- Exam: visual field defects, reduced color vision, +APD
Optic Nerve: Anterior Ischemic Optic Neuropathy

- NAION: Hyperemic disc edema

Optic Nerve: Anterior Ischemic Optic Neuropathy

- AAION: Pallid disc edema, diplopia, nystagmus, ptosis, pain/nodularity on palpation of temporal artery

Optic Nerve: Anterior Ischemic Optic Neuropathy

- **AAION Systemic Symptoms:**
  - Headache (most common)
  - Scalp and temporal artery tenderness
  - Jaw claudication (most specific)
  - Fatigue
  - Weight loss
  - Anorexia
  - Fever
  - Joint and muscle pain (Polymyalgia Rheumatic in 50%)

- **Occult GCA without overt systemic symptoms in 20%**

[Temporal Artery Biopsy for Giant Cell Arteritis](https://creakyjoints.org/diagnosis/temporal-artery-biopsy-to-diagnose-giant-cell-arteritis/)
Optic Nerve: Anterior Ischemic Optic Neuropathy

- **AAION:**
  - Elevated ESR and/or CRP
  - Thrombocytosis
  - Temporal Artery Biopsy (TAB)
  - Color Doppler Ultrasounds (CDUS) – non-invasive, real time imaging of multiple arteries, shows “halo sign” adjacent to arterial wall
  - MRI – thickening and/or enhancement

[Image: http://www.ajnr.org/content/28/9/1722]
Optic Nerve: Anterior Ischemic Optic Neuropathy

• NAION Treatment:
  – Avoid Nocturnal Hypotension and treat OSA
  – Topical antihypertensive to optimize ocular perfusion
  – No clear benefit from steroids or anticoagulation

• Prognosis:
  – 50%: 20/30 or better, 25%: 20/200 or worse
  – Younger patient typically recover better
  – Recurrence rates over 5 year
    – same eye: 3-7%
    – Contralateral eye: 15-24%

https://en.wikipedia.org/wiki/Continuous_positive_airway_pressure
Optic Nerve: Anterior Ischemic Optic Neuropathy

• AAION Treatment:
  – Prompt initiation of high-dose corticosteroids
    • Typically Solumedrol IV 1g/day x 3 days then oral
    • Can start with Prednisone PO 60-100 mg/day
    • SLOW gradual taper in conjunction with Rheum

  – Tocilizumab – monoclonal antibody binds to human IL-6 receptor

  – Steroid Sparing Agents:
    • Methotrexate

https://www.eurekalert.org/multimedia/pub/230582.php
Optic Nerve: Anterior Ischemic Optic Neuropathy

• Prognosis:
  – Untreated 54-95% patients will have visual loss within 4 months
  – Worsening vision in 9-17% despite therapy
  – Bilateral vision loss can proceed quickly in up to 50% cases

– Systemic Complications
  • Aortic aneurysms or dissection
  • Increased risk of angina or MI
  • Stroke

https://medlineplus.gov/ency/article/000181.htm
Question 1

- What is the first line treatment for a chemical burn to the cornea?
  a. Topical antibiotics
  b. Topical steroids
  c. Copious saline irrigation with Morgan lens
  d. Bandage contact lens
Answer 1

• C: Copious saline irrigation with Morgan lens
Question 2

• What is the leading cause of preventable blindness in the United States in those 24-75 years old?
  a. Hypertensive retinopathy
  b. Diabetic retinopathy
  c. Anterior ischemic optic neuropathy
  d. Primary open angle glaucoma
Answer 2

- Diabetic Retinopathy
References


Thank you!

- Questions
  - neetid@kumc.edu