Ocular Emergencies
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- Differentiate “Emergency” vs. “Urgency”
- Proper Triage necessary (Front desk, Doctor away, After hours)
- Understand the “10 A Club”

- Papilledema
- Giant Cell Arteritis
- Aneurysm
- Pituitary Apoplexy
- Carotid Artery Dissection
- Central Retinal Artery Occlusion
- Perforated Globe
- Acute Angle Closure Glaucoma
- Acid / Alkaline Chemical Burn
- HyphemA
• Rapid unilateral painless vision loss – count fingers / light perception visual acuity in 90% of eyes

Etiology:
• Arterial emboli (cholesterol, fibro-platelet, calcific) 20% cases
• Thrombus formation secondary to arteriosclerosis
• Arteritis – Giant cell arteritis, Lupus, Polyarteritis nodosa
• Blood dyscrasias (hypercoagulation disorder)
• Migraine
• Optic nerve drusen

Presentation:
• Superficial retinal whitening with a "cherry red spot" in the fovea
• Retinal whitening within 30 minutes to 1 hour from ischemic necrosis
• Narrowed retinal arterioles with segmentation
• Cilioretinal artery may be spared the fovea
• Relative Afferent Pupillary Defect
• Optic Nerve pallor weeks to months later

Treatment:
• Ocular Massage (digital pressure) – 10 sec on/10 sec off x 5 minutes
• Vasodilation (increase CO2 by breathing into a paper bag)
• Lowering of intraocular pressure (IOP) – beta blocker, Diamox, etc.
• Anterior chamber paracentesis
• Sublingual Nitroglycerin / Intra-Arterial fibrinolysis with (tPA)

Systemic Testing:
• Immediate Westergren Sed. Rate if patient is > 55 years old; R/O GCA
• Carotid artery evaluation, blood pressure, lipid profile, blood sugar
• Echocardiogram – cardiac evaluation
• Fluorescein angiography (FA)
Work-Up:

- Cardiovascular Evaluation
  - Echocardiogram (Transesophageal vs. Transthoracic)
    - To find congenital/acquired valvular anomalies
    - To evaluate for cardiac myxoma, atrial fibrillation
    - Congenital heart defects such as a patent foramen ovale
    - R/O endocarditis (infectious process)-Heart murmur 90%

- Hyperviscosity Evaluation
  - Multiple Myeloma, leukemias, and Waldenstroms
  - Macroglobulenia

- Hypercoaguable Evaluation
  - Factor V Leiden, Hyperhomocysteine,
    - Antiphospholipid syndrome, birth control pills, pregnancy

- Hemoglobinopathies
  - Sickle Cell disease and trait

Follow Up:

- RTC at 1 month to check for neovascularization of disc/iris
- RTC at 3 months to check for neovascularization of disc/iris
- Neo of iris = 20% of patients at about 4 weeks
- Neo of disc = 3% of patients
- Extremely important to perform a complete medical work-up to stop progression of the disease along with any systemic sequelae
Arteritic Anterior Ischemic Optic Neuropathy

- Must be differentiated from "Non-Arteritic" AION

- Sudden painless loss of vision
- Females > Males (2:1 ratio)
- Patients usually > 55 years of age

Presentation: Ocular "Giant Cell Arteritis"
- Acute painless vision loss (VA loss is usually permanent)
- Pale swelling of the optic nerve head with flame shaped hemes
- Central retinal artery occlusion may occur
- Cranial nerve palsy (CN 3,4,6) may also be present, CWS

Etiology

Occlusion of the "short posterior ciliary" arteries with giant white blood cells

Presentation: Systemic

- Headache
- Scalp tenderness
- Jaw claudication
- Night sweats
- Weight loss
- Fever
- Polymyalgia rheumatica
- Depression

Laboratory Testing:

- Immediate Erythrocyte Sedimentation Rate (Westergren ESR)
- Immediate C-reactive protein (Acute Phase Reactant) >2.45 mg/dl
- Platelet count (Thrombocytosis) = risk for permanent visual loss
- CBC with differential = anemia of chronic inflammation
• A normal ESR does not R/O GCA; Normal in ~13% of GCA
• 20% of GCA patients do not have systemic symptoms

Giant Cell Arteritis
- Temporal Arteritis
- Cranial Arteritis
- Granulomatous Arteritis

White Blood Cells
- T-cells infiltrate arteries
- Cytokines IL, TNF, IFN

Attacks medium and large sized arteries
- Superficial Temporal Artery
- Coronary Artery
- Subclavian Artery
- Facial Artery

Possible association with “Polymyalgia Rheumatica” (PMR)
- Stiffness in the neck, shoulder, and hip
- 50% of Giant Cell patients have PMR
- Is there a link between GCA and PMR??

Treatment:
- Immediate IV steroid therapy – IVMP x 3 days
  Oral Prednisone x 24 months
- Possible temporal artery biopsy
  • Done within a week of starting steroids
  • Specimen is 2.5 cm long
  • If biopsy is negative but suspicion high, then biopsy the opposite side
  • ~13% of cases will be positive on the opposite side
- Methotrexate with Prednisone
- Always suspect recurrence
The "One Third Rule" in Giant Cell Arteritis

- 1/3 of optic nerves in the fellow eye will become infarcted within 48 hours in untreated patients.
- 1/3 of optic nerves in the fellow eye will become infarcted within 1 month in untreated patients.
- Second eye infarctions are rare after more than 1 month

"Malignant Hypertension" defined as Blood Pressure > 210/120

Ocular Presentation:
- Disc edema with or without exudate
- Arterio-venous crossing changes
- Nerve fiber layer infarcts (cotton wool spots)
- Macular edema
- Hard exudates / flame shaped hemis
- Choroidal ischemia

Treatment:
- Blood pressure measurement
- Immediate referral to emergency room or PCP for slow lowering of the the blood pressure

General Rule:
- Chest Pain
- Difficulty breathing
- Immediate Attention

Grade 4 HR

BP 225 / 125

BP 235 / 130
"Malignant Hypertension"

Hypertensive Choroidopathy

Hypertensive Choroidopathy
Orbital Cellulitis

- Inflammation of orbital soft tissue posterior to the orbital septum
- May get a direct extension to the brain – may lead to death

Systems of Classification

- Stage 1: Preseptal Cellulitis
- Stage 2: Orbital Cellulitis
- Stage 3: Subperiosteal abscess
- Stage 4: Intraorbital abscess
- Stage 5: Cavernous Sinus Thrombosis – usually has fatal outcome

Etiology:

- Eyelid infection (Hordeolum)
- Sinus infection (paranasal sinusitis extension in ~90% of cases)
- Dental infection
- Ocular trauma or surgery
- Orbital infection (dacryocystitis)
- Upper respiratory infection
- Otitis Media

Presentation Ocular:

- Eyelid edema (absence of a lid crease) - Painful
- Conjunctival chemosis
- Proptosis / Globe displacement
- Restricted motility - may have associated pain (60%)
- Visual Acuity decrease

Sinusitis

- S. pneumoniae
- other streptococci
- S. aureus
- H. influenzae (esp. in children)
- anaerobes less common

Post-traumatic and post surgical

- S. aureus most common
- anaerobes less common
Usually, orbital cellulitis occurs in the childhood years which has been attributed to the relatively incomplete development of immunity in this age group. In these patients, sinus disease has been found to be the most common predisposing factor. Over 90% of these patients have radiologically confirmed sinusitis, the most common being ethmoidal and maxillary. Ethmoidal sinusitis has been demonstrated to be the source of infection in significantly large number of cases.

Hordeolum

Preseptal Cellulitis
### Presentation: Systemic
- Fever (as high as 104)
- Headache
- Malaise
- Nausea / Vomiting

### Preseptal Cellulitis Lab Work
- Complete Blood Count (CBC)
- CT if unable to tell orbital involv.

### Treatment:
- Immediate referral to emergency room or to Ophthalmology
- Broad spectrum coverage with **IV antibiotics** after culture
- MRI or **orbital CT** (Contrast not needed)
- Hospitalization
- Consultation with ENT, PCP and Neurosurgery if intracranial infection

### Preseptal Cellulitis Lab Work
- Complete Blood Count (CBC)
- CT if unable to tell orbital involv.

### Inflammation of orbital soft tissue posterior to the orbital septum

### Oral antibiotics → Dacryocystis with Preseptal Cellulitis

### IV antibiotics → Orbital Cellulitis

### Oral antibiotics → Preseptal Cellulitis
Open Globe Injuries

- In the United States alone ~ 2,500,000 eye injuries per year
- United States Eye Injury Registry (USEIR) was established in 1988
- Goal is collect and document information on serious eye injuries.

Data from USEIR shows the following:
- Mean age of 29 years old
- Median age of 26 years old
- 57% of patients usually < 30 years old
- 80% are males

Classification System for Ocular Trauma

Type: Open Globe
- Rupture
- Penetrating
- Perforating
- Intraocular FB
- Mixed

Open globe = patient kept nil per os (NPO), pain meds given, contact MD!

Type: Closed Globe
- Hyphema, Subluxated lens, Retinal tear, Choroidal rupture

Open globe injuries classification and treatment guidelines.
Subconjunctival Hemorrhage

Fox Shield
The incidence of endophthalmitis following penetrating injuries is between 5% to 14%.

- The USEIR incidence is 2.6% and more common in males.
- The incidence is more common in rural settings (30%) or involves an IOFB (15%)
- Infections with more than one organism are common (48%)
- Bacillus and staph are most prevalent.

Intraocular Foreign Bodies – iron and copper are toxic. Aluminum metal alloys, plastics are non-toxic
Cranial Nerve 3 palsy – Pupil involved ophthalmoplegia

Medical emergency if pupil is dilated

Ophthalmoplegia caused by the following:

- Aneurysm
- Microvascular disease (Infarction)
- Tumor
- Trauma
- Infection (syphilis)
- Idiopathic

The most common cause of motility restriction after orbital trauma is orbital soft tissue swelling. Orbital compartment syndrome = true emergency.
• Complete pupil involvement – Send to ER – 20% die within 48 hrs
• Relative Pupil involvement
• Pupil spared

Presentation: Ocular

Ptosis – Levator
Limited ocular motility
Complete or relative sparing of the pupil
Aneurysmal Third-Nerve Palsies

- 90% of aneurysmal third-nerve palsies have the pupil affected.
- Half of patients diagnosed with aneurysmal third-nerve palsies have a subarachnoid hemorrhage within 2 weeks.
- Half of those patients who hemorrhage will die.
- Catheter Cerebral Angiography is the best imaging tool, however, it will cause stroke or myocardial infarction in 1%-2% of patients.
- The lack of anisocoria in an isolated third-nerve palsy does not exclude an aneurysm or compressive lesion.

Terson’s Syndrome
Retinal Detachment – Threatening the fovea
Treatment:

- Referral to a retinal specialist
- Laser retinopexy
- Scleral buckle
- Cryotherapy
- C3F8 or SF6 "gas bubble" as a tamponade

Horner’s Syndrome

- Patient has headache and vision loss in one eye
- Funny taste on the back of the tongue
- Hemispheric neurologic deficits
- Trauma or pseudoaxanthoma elasticum
- Neurologic consultation
- Carotid artery imaging
- MRA / MRI / CT
- Anticoagulants to prevent stroke
Horner's Syndrome:

- New Diagnostic test
  - 0.5% or 1.0% Apraclonidine (Iopidine)
- Alpha agonist
  - Dilates a Horner pupil (supersensitivity)
  - No affect on normal pupil

Digital infra-red photos taken under scotopic illumination. Note reversal of anisocoria after use of Apraclonidine, indicative of a right Horner syndrome.
Pituitary Apoplexy

70 year old white male – Emergency visit
Reduced vision OU – Prior diagnosis of "dry" ARMD (OU)
(OD) 20/400  (OS) 20/50

Soft confluent drusen (OU)

Pituitary Adenoma
Pituitary Adenoma

Microadenomas - < 10 mm in diameter and confined to the sella turcica

Macroadenomas - > 10 mm in size and extends beyond the sella turcica

Symptoms:
- Vertical and horizontal diplopia
- Pain (aneurysm) ... variable with diabetic 3rd
- Acute = aneurysm or ischemic vascular
- Progressive = infiltrative or compressive

Treatment:
- Referral to Neuro-Ophthalmologist immediately
- Possible MRI / MRA / CT
- Catheter Cerebral angiography – if aneurysm suspected ****
- ESR if Giant Cell Arteritis is suspected

Ocular Emergency:
- A sudden onset of ophthalmoplegia, vision loss, nausea, vomiting, and severe headache = Pituitary Apoplexy

Pituitary Apoplexy = Acute hemorrhagic infarction of a pituitary adenoma

- Visual Fields: Bitemporal Defect
- Diagnosis made by MRI or CT scan
- Treatment: Surgery, Radiation, Medication
Ocular Emergencies

The End
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