Eye Emergencies

Azadeh doozandeh, MD

Torfeh Medical Center

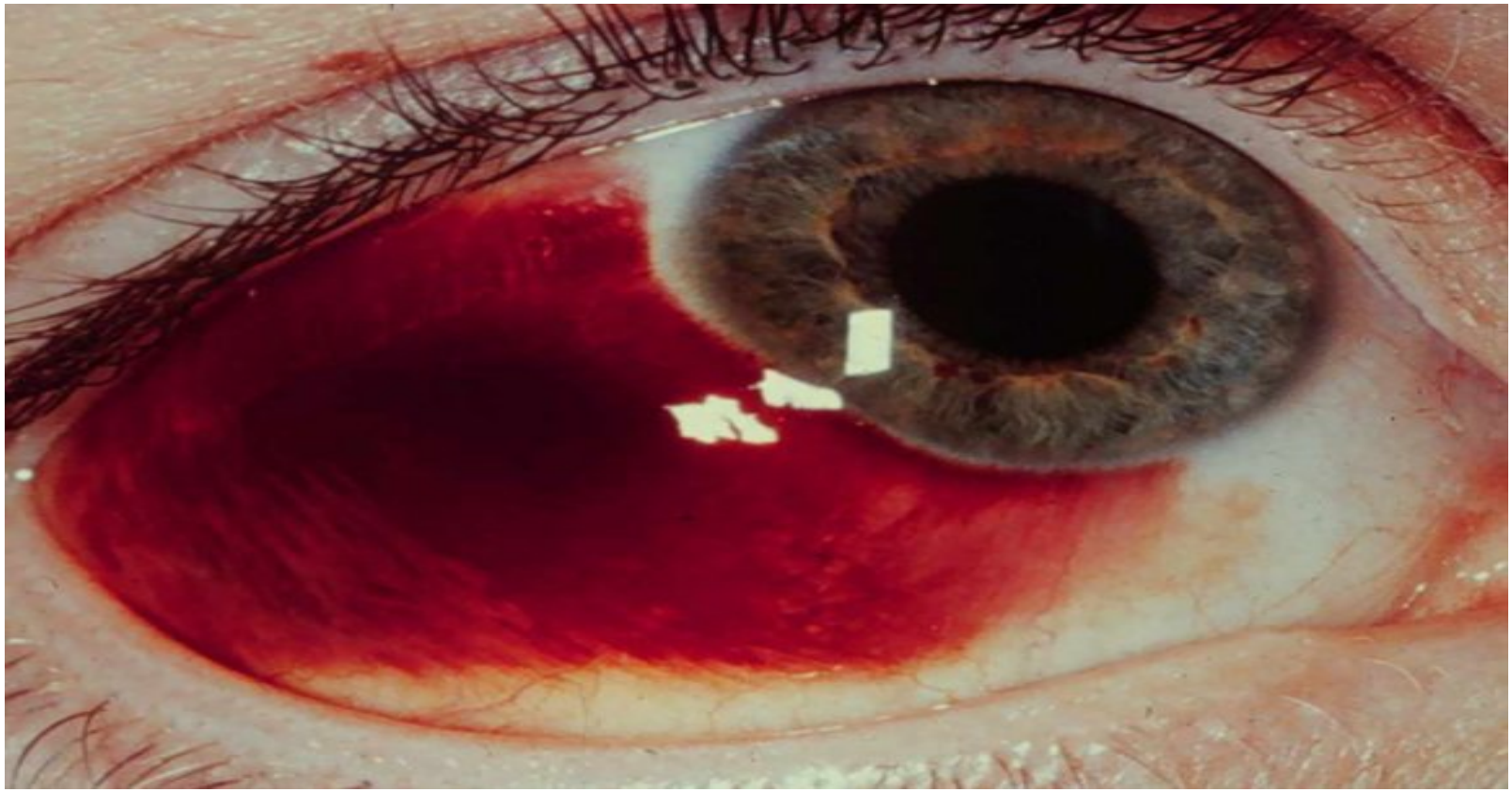
Traumatic Eye Injuries

- Subconjunctival Hemorrhage
  - Disruption of conjunctival blood vessel
  - Etiology
    - Trauma
    - Sneezing
    - Gagging
    - Valsalva
  - Will resolve spontaneously within 2 weeks

  *If dense, circumferential bloody chemosis is present, must rule out globe rupture

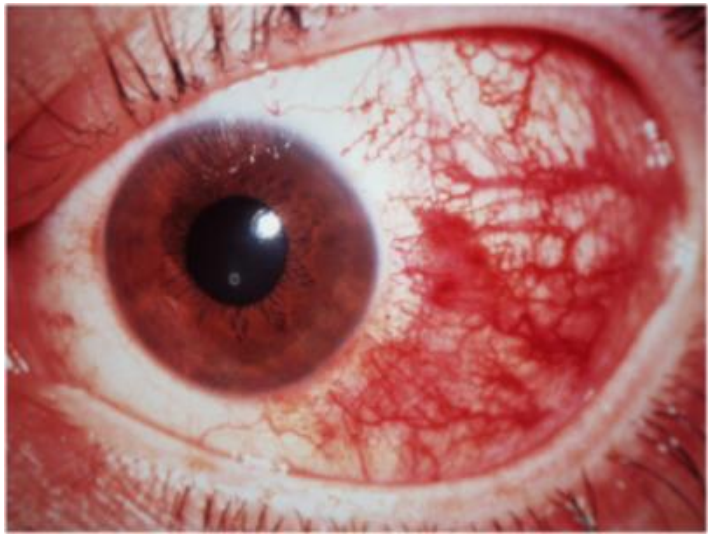
Traumatic Eye Injuries

- Subconjunctival Hemorrhage



Traumatic Eye Injuries

- Conjunctival Abrasion
  - Superficial abrasions
    - Treatment: 2-3 days of erythromycin ointment
  - Ocular foreign body should be excluded

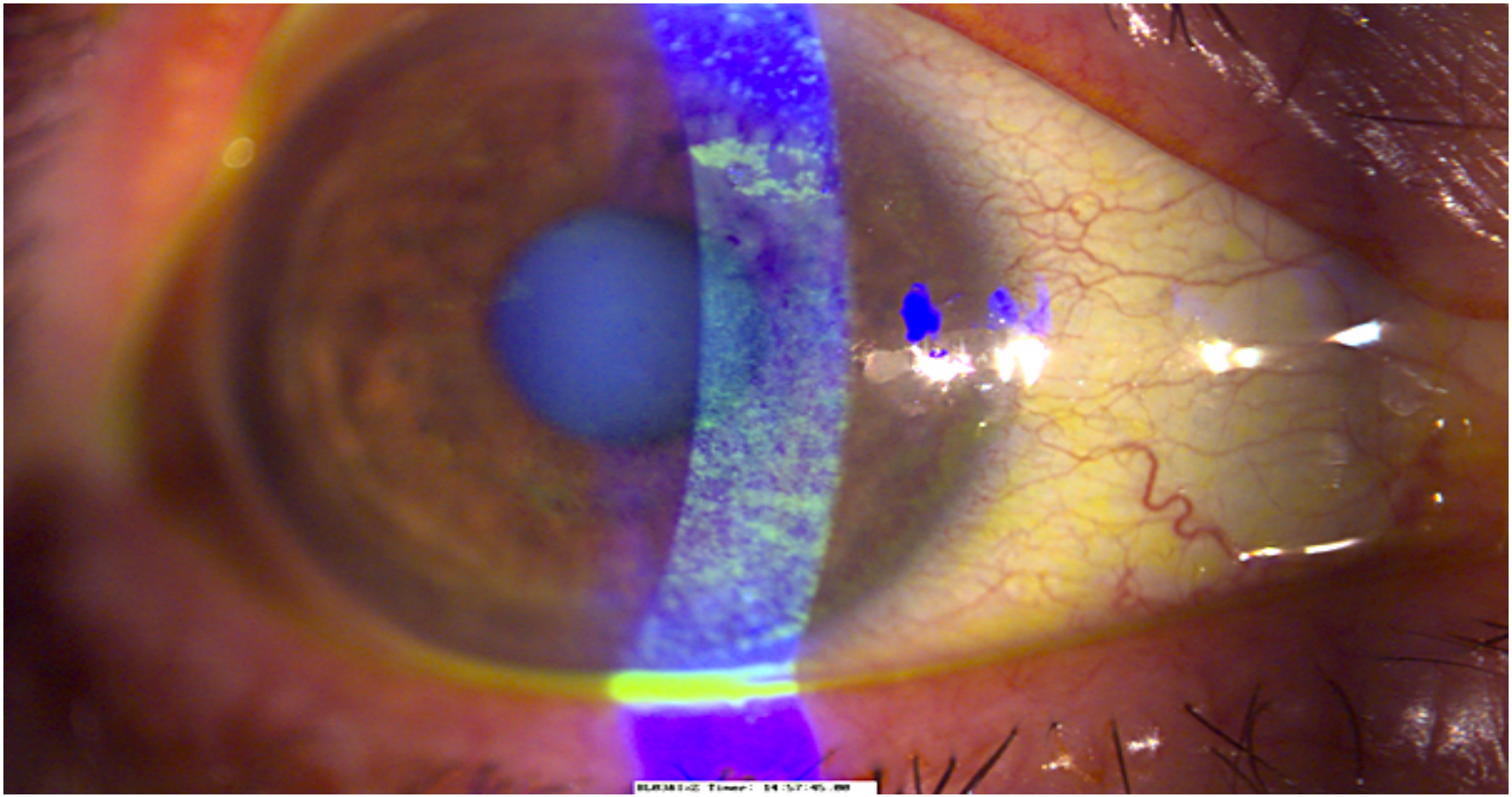


Traumatic Eye Injuries

- Corneal Abrasion
  - Tearing, photophobia, blepharospasm, severe pain
  - Fluorescein: dye uptake at defect site
  - Rule out foreign body
  - Treatment:
    - Cycloplegic
    - Topical Tobramycin, Erythromycin, or Bacitracin/polymyxin drops
    - Contact lens wearers: Cipro, Ofloxacin, or Tobramycin drops
    - Ophthalmology consult within 24 hours

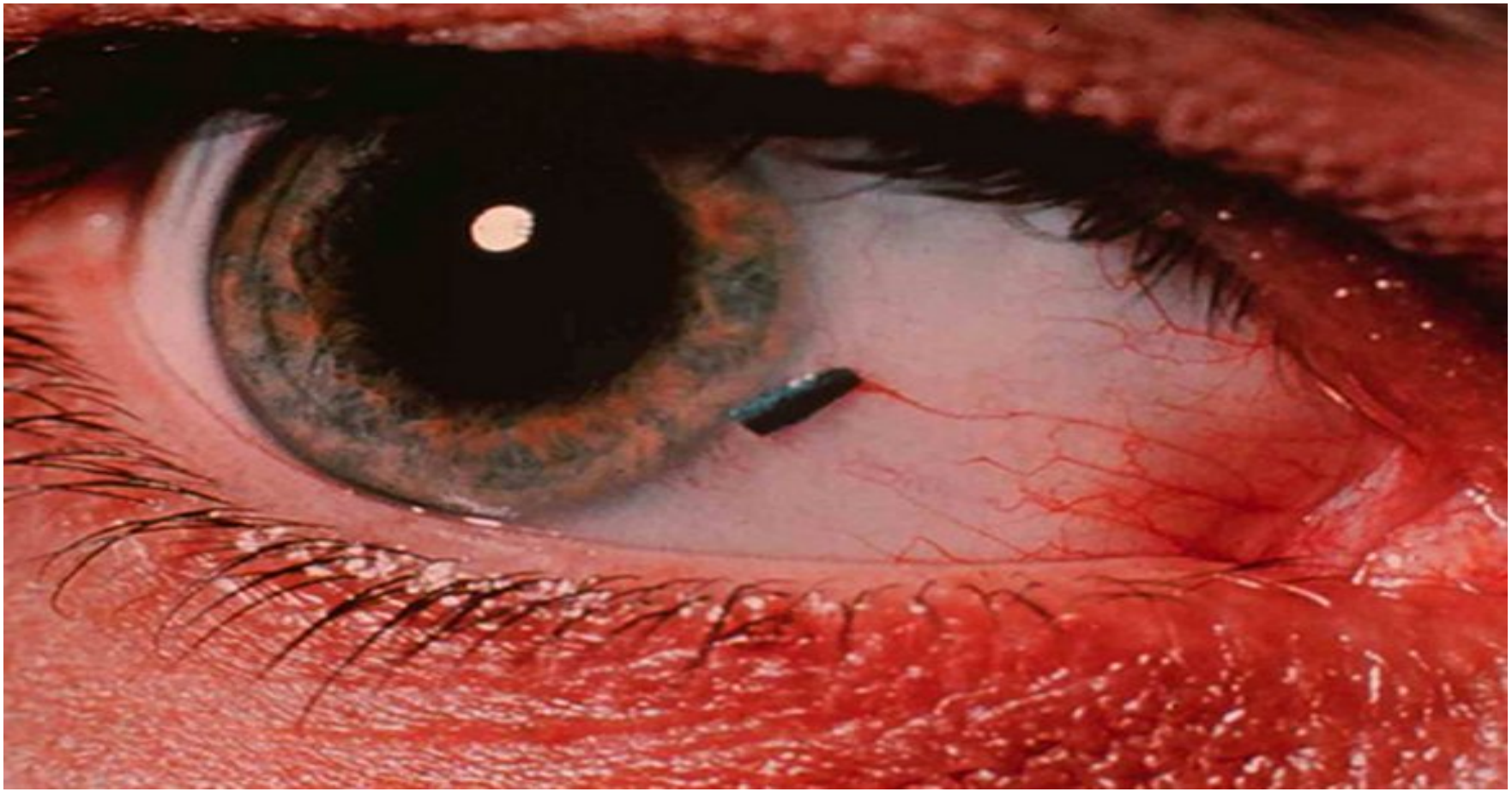
Traumatic Eye Injuries

- Corneal Abrasion



Traumatic Eye Injuries

- Conjunctival Foreign Bodies

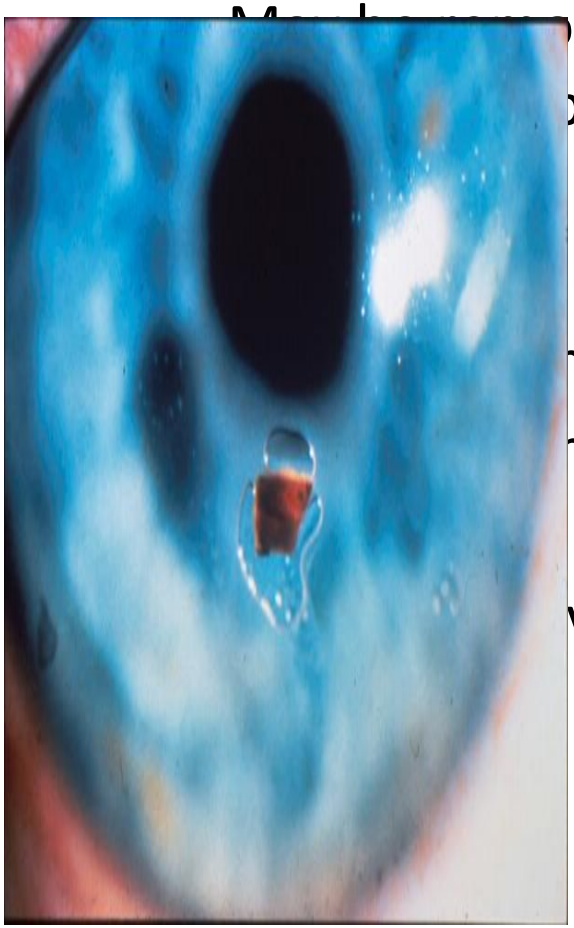


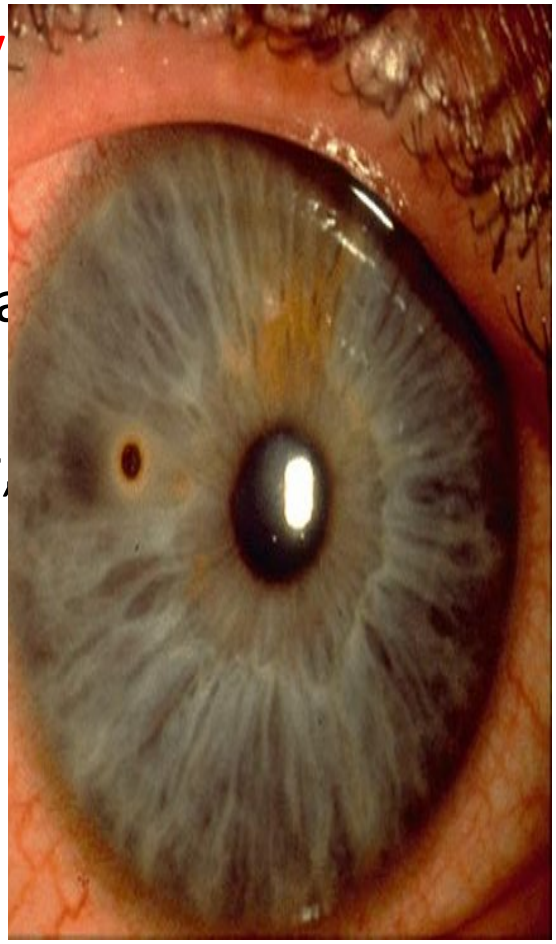
Traumatic Eye Injuries

- Corneal Foreign Bodies

Traumatic Eye Injuries

- Corneal Foreign Bodies





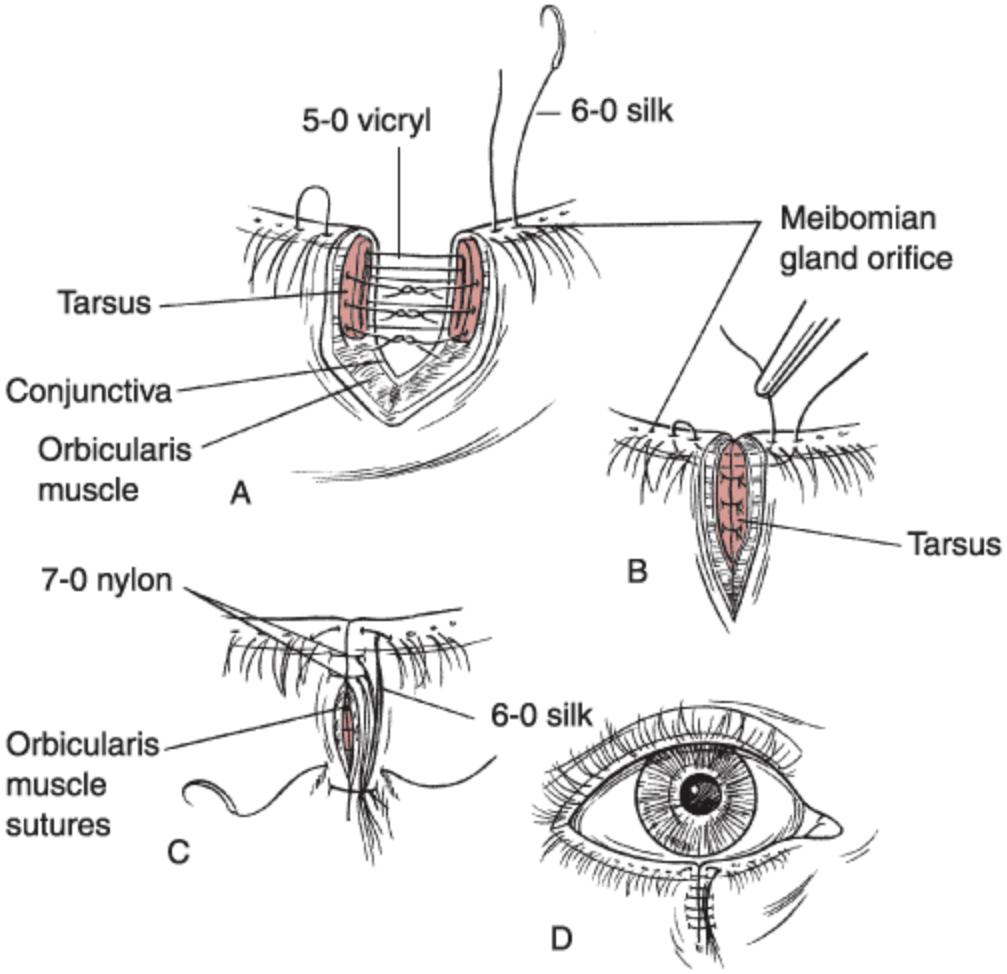
Traumatic Eye Injuries

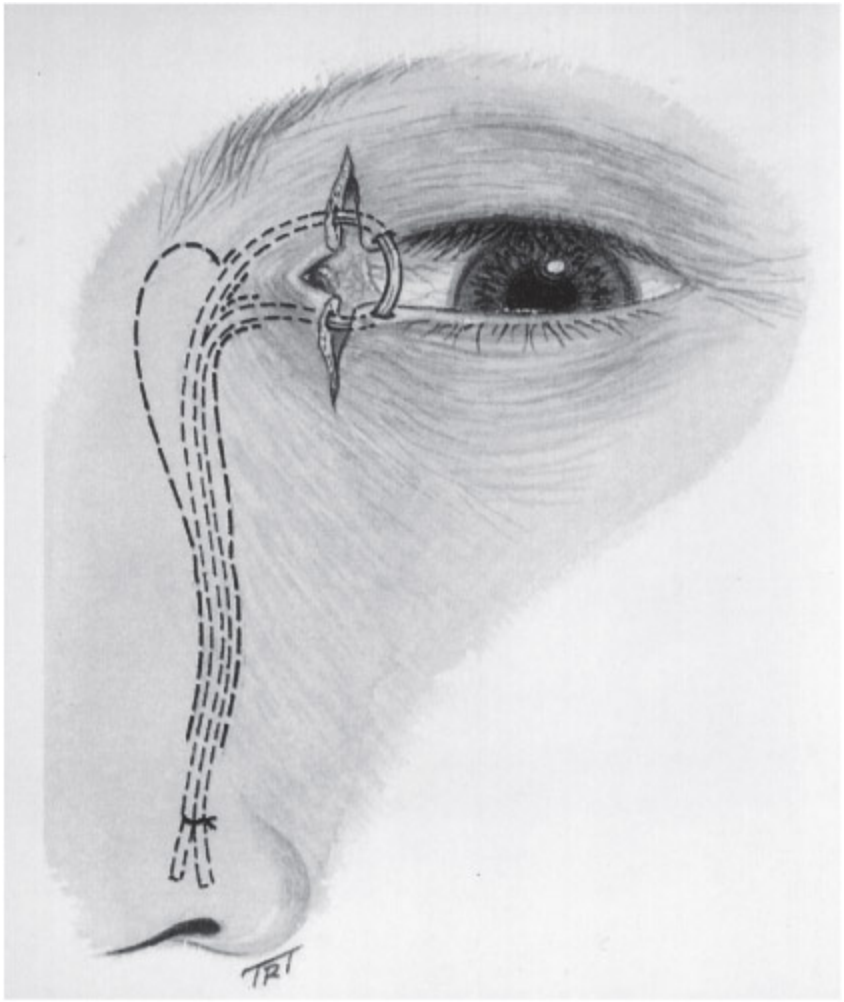
- Lid Lacerations
  - Must exclude damage to eye and nasolacrimal system
  - Fluorescein staining in the tear layer that appear in the adjacent lac confirm nasolacrimal involvement
  - Most require ophtho consult

Traumatic Eye Injuries

- Lid Lacerations

Traumatic Eye Injuries



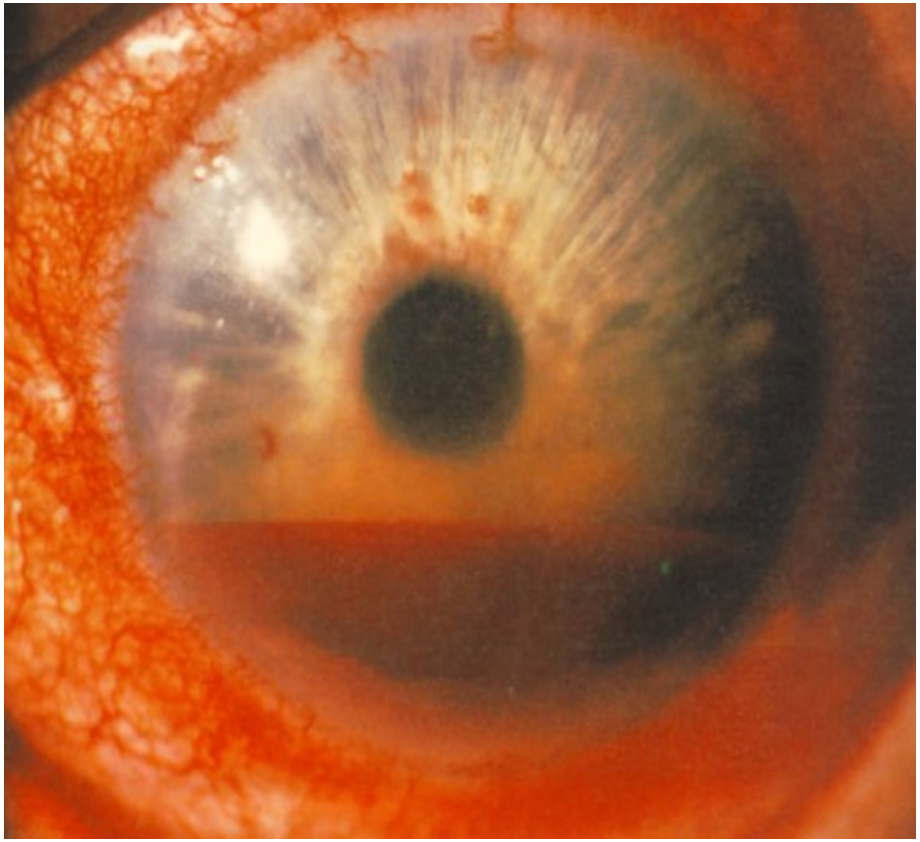


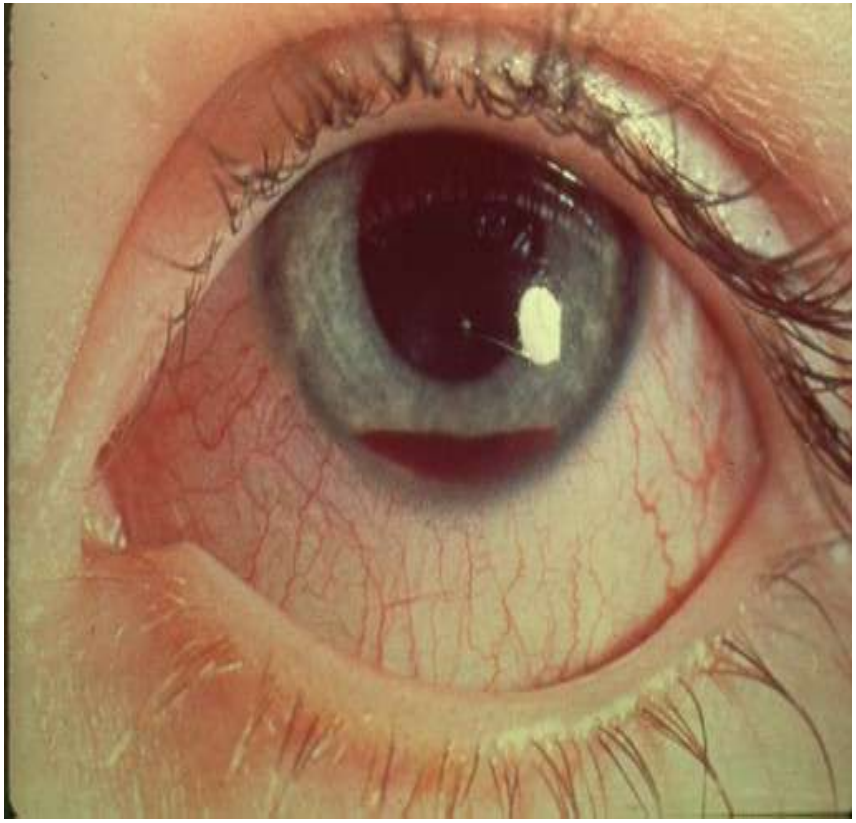
Traumatic Eye Injuries

- Blunt Trauma
  - Immediately assess integrity of globe and visual acuity
  - Eval depth of anterior chamber, pupil size, monocular blindness ruptured globe

Traumatic Eye Injuries

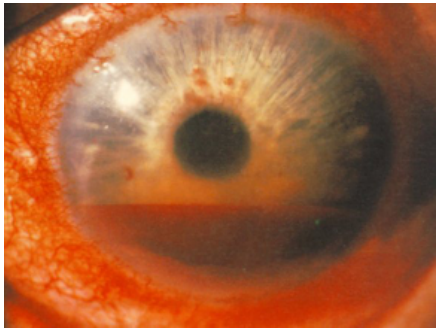
- Hyphema

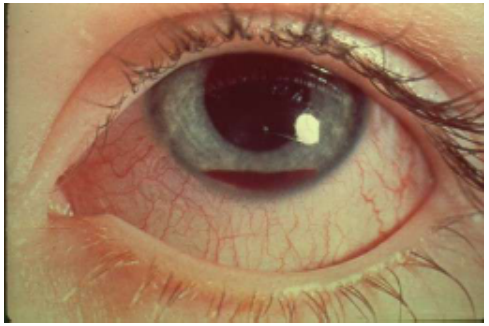




Traumatic Eye Injuries

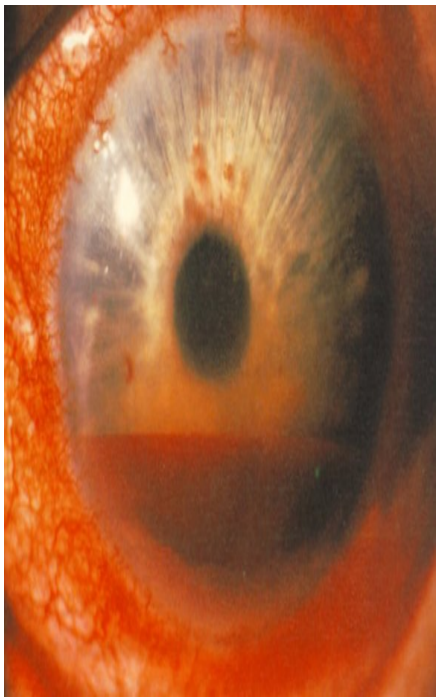
- Hyphema
  - Blood in the anterior chamber
  - Spontaneous or post-trauma
  - Treatment:
    - Place the pt upright to allow inferior settling of blood
    - Exclude ruptured globe
    - Dilate the pupil with atropine
    - Measure intraocular pressure – if > 30 mmHg apply topical Timolol
    - Emergent Optho eval

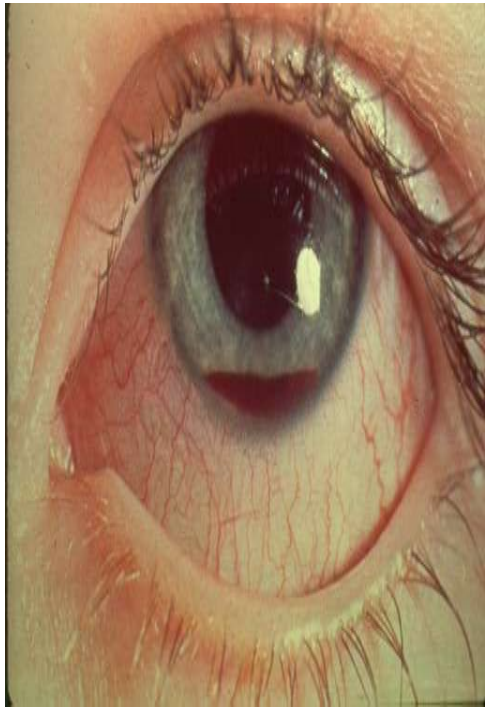




Traumatic Eye Injuries

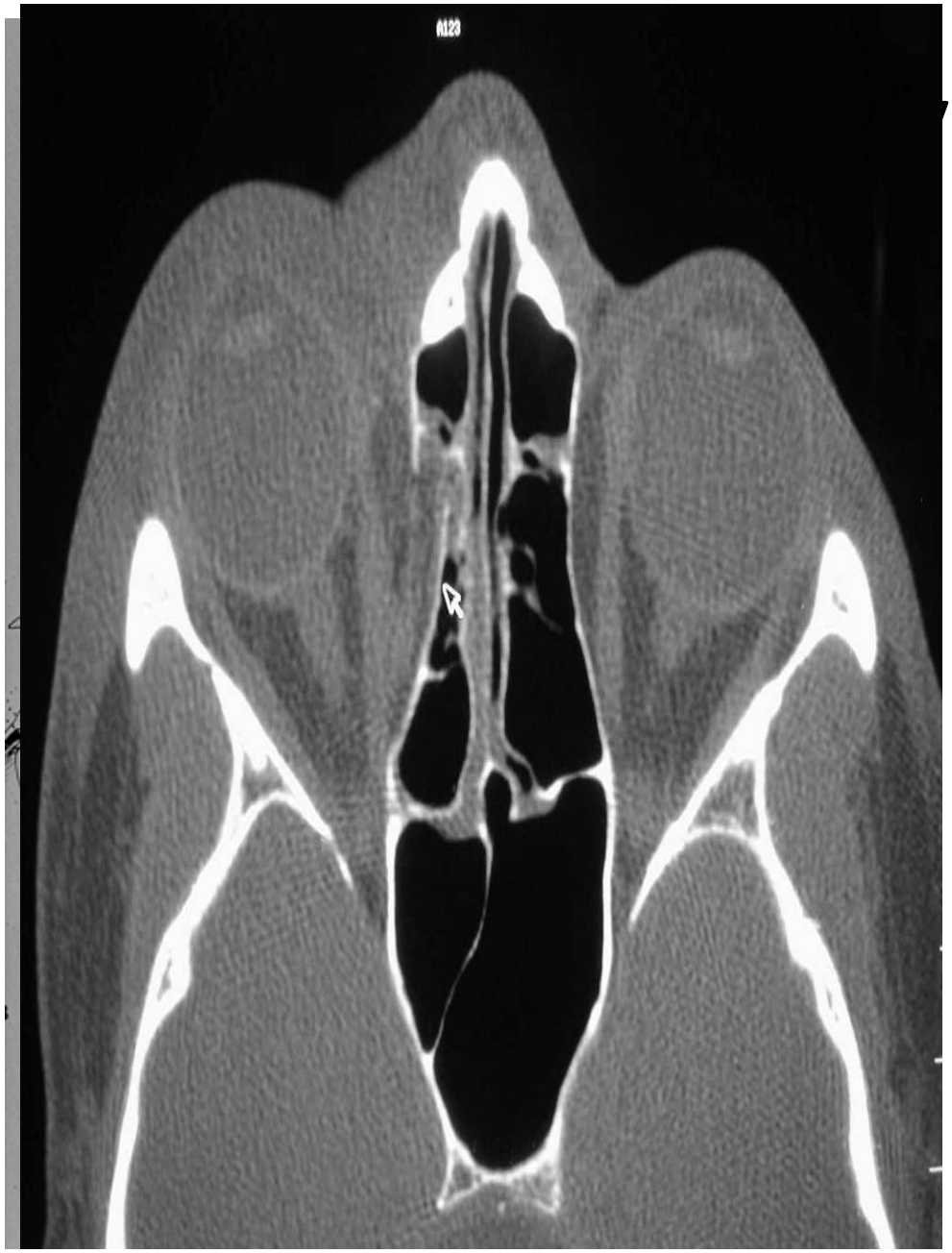
- Hyphema
  - Risk for worse rebleed in the next 2-5 days is very high





Traumatic Eye Injuries

- Blowout Fractures
  - Inferior and medial wall most at risk
  - Evaluate for
    - inferior rectus entrapment (diplopia on upward gaze)
    - infraorbital nerve paresthesia
    - subcutaneous emphysema (when blowing the nose)
  - Orbital cut CT scan
  - Treatment: rule out ocular trauma and give oral Keflex
  - Isolated blowout fracture – ophtho eval in 3 – 10 days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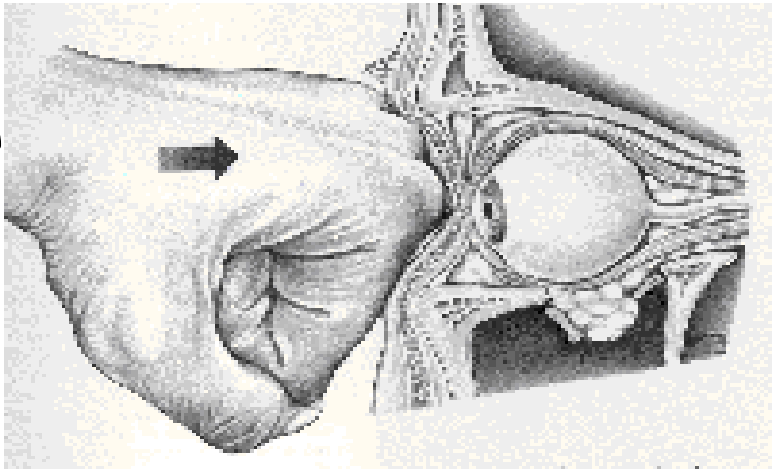

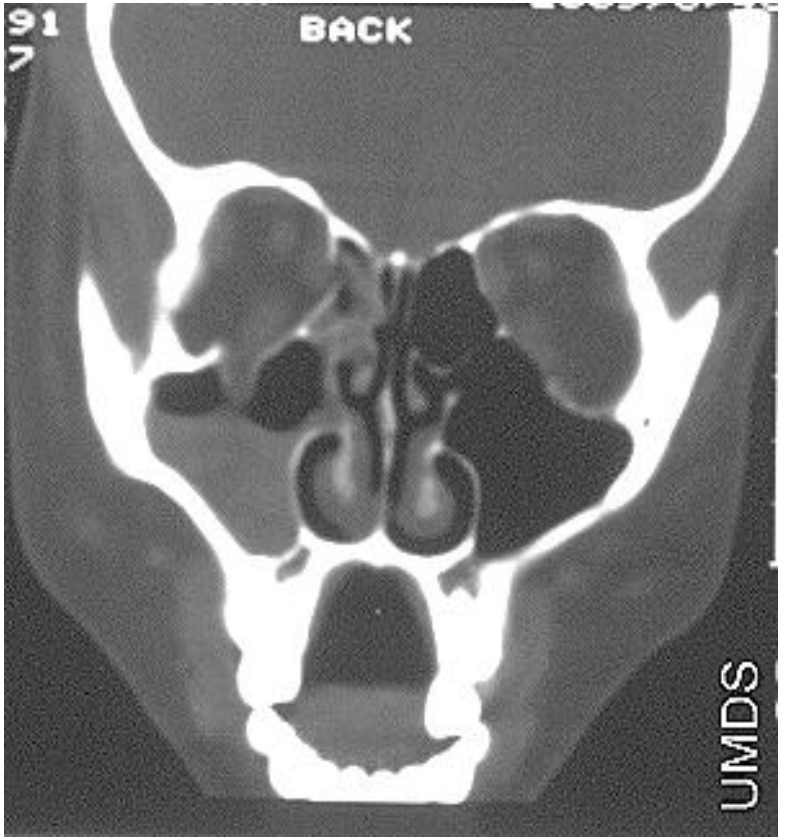
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BACK
UMDS

Traumatic Eye Injuries

- Penetrating Trauma/Ruptured Globe
  - Severe subconjunctival hemorrhage
  - Shallow or deep anterior chamber in one eye
  - Hyphema
  - Tear-drop shaped pupil

Traumatic Eye Injuries

- Penetrating Trauma/Ruptured Globe
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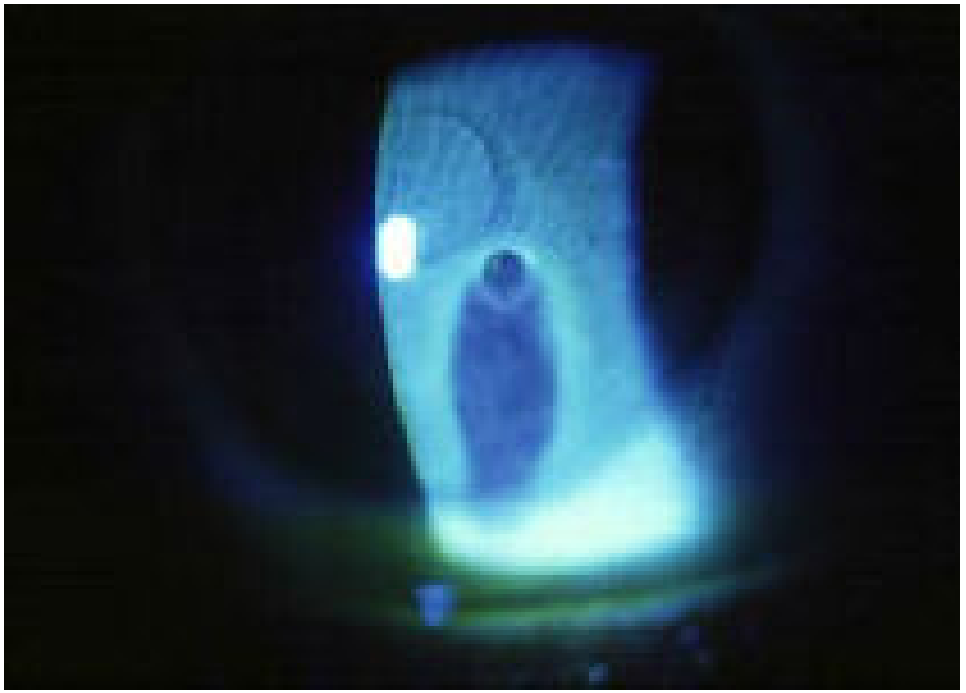
Traumatic Eye Injuries

- Penetrating Trauma/Ruptured Globe
  - Severe subconjunctival hemorrhage
  - Shallow or deep anterior chamber in one eye
  - Hyphema
  - Tear-drop shaped pupil
  - Limited extraocular motility
  - Extrusion of globe contents
  - Significant reduction in visual acuity

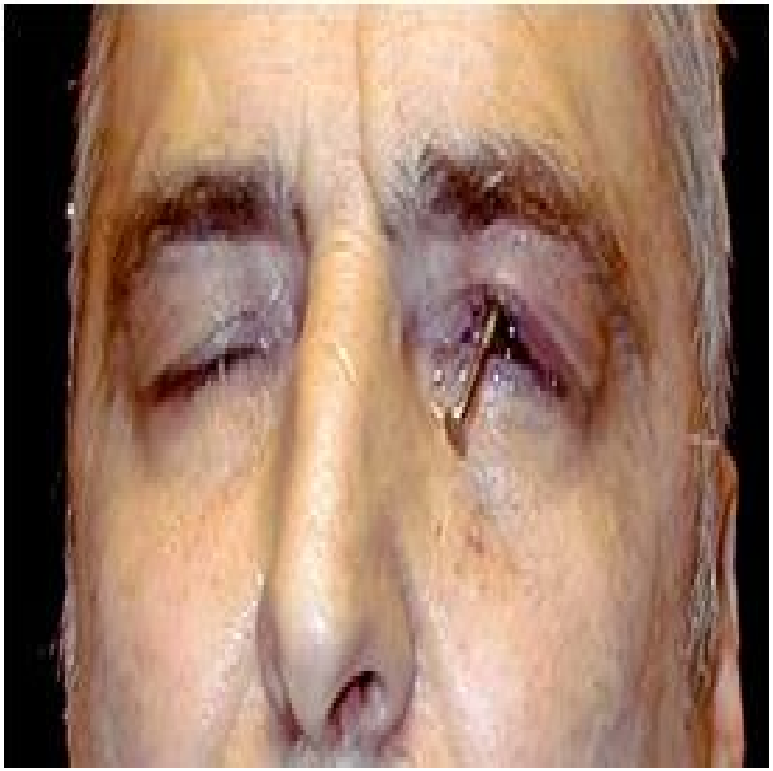
Traumatic Eye Injuries

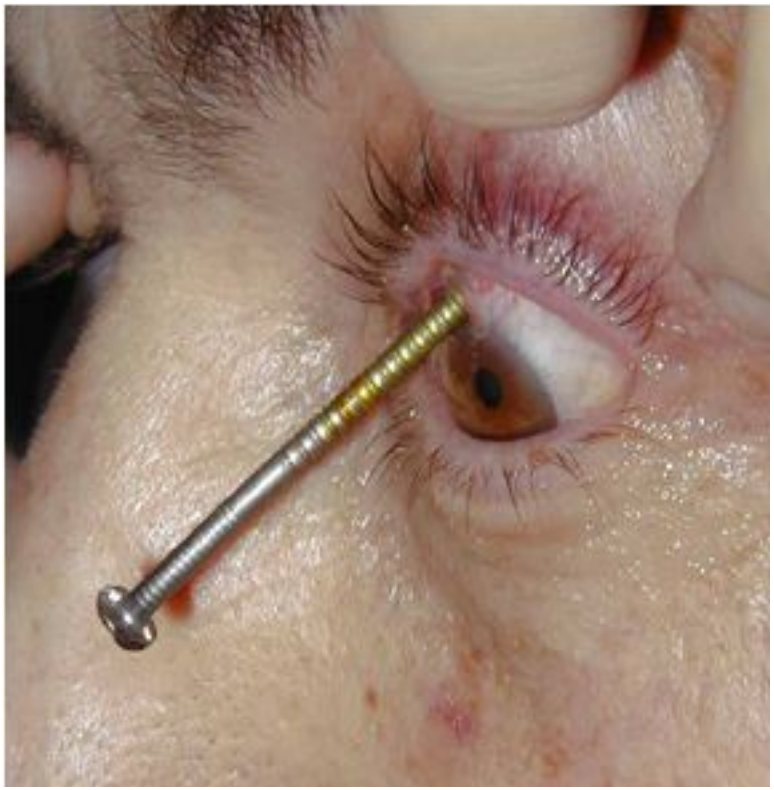
- Penetrating Trauma/Ruptured Globe
  - Seidel's test
    - Fluourescein streaming



Traumatic Eye Injuries

- Penetrating Trauma/Ruptured Globe





Traumatic Eye Injuries

- Penetrating Trauma/Ruptured Globe
  - If a globe injury is suspected:
    - Don’t manipulate the eye any more
    - …Step away from the eye
    - Place the pt upright
    - NPO
    - Protective eye shield
    - Administer IV cephazolin and antiemetic
    - Tetanus

Traumatic Eye Injuries

- Penetrating Trauma/Ruptured Globe
  - Orbital CT
    - If intraocular foreign body suspected
  - Call Ophtho right away

Traumatic Eye Injuries

- Chemical Ocular Injury
  - Acid or alkali – treat the same
  - Immediately flush (at the scene)
  - Continue to flush until pH is normal (7.0)
    - Check with urine dipstick
  - Recheck pH after sweeping the fornices for retained particles
  - Measure IOP

Traumatic Eye Injuries

- Chemical Ocular Injury

  Chemical injuries of the eye may produce extensive damage to the ocular surface epithelium, cornea & anterior segment, resulting in permanent unilateral or bilateral visual impairment

- 80% of ocular chemical burns were due to industrial and/or occupational exposure
- Ocular burns are more common in males than in females

ETIOLOGY- ALKALI

- **Ammonia-**---Fertilizers, Refrigerants, cleaning agents
- **Lye** (NaOH)- Drain cleaners
- **Potassium hydroxide-** Caustic potash
- **Magnesium Hydoxide** –Sparklers
- **Lime**-($Ca(OH)_2$- Plaster, whitewash, cement

ETIOLOGY-ACID

- **Sulfuric acid-** Industrial cleaners, Battery acid
- **Sulfurous acid**-Bleach, Refigerants
- **Hydrofluoric acids**- Glass polishing
- **Acetic acids**- Vinegars

- Alkali substances are lipophilic and penetrate more rapidly than acids. Saponification and Liquefaction

- Alkali substances pass into the anterior chamber rapidly (5-15 min) exposing the iris, ciliary body, lens, and trabecular network to further damage.

- Irreversible damage occurs at a pH value above 11.5.

- Acid burns cause **protein coagulation** in the corneal epithelium, which limits further penetration.
- Acid burns usually are **non progressive** and **superficial**.

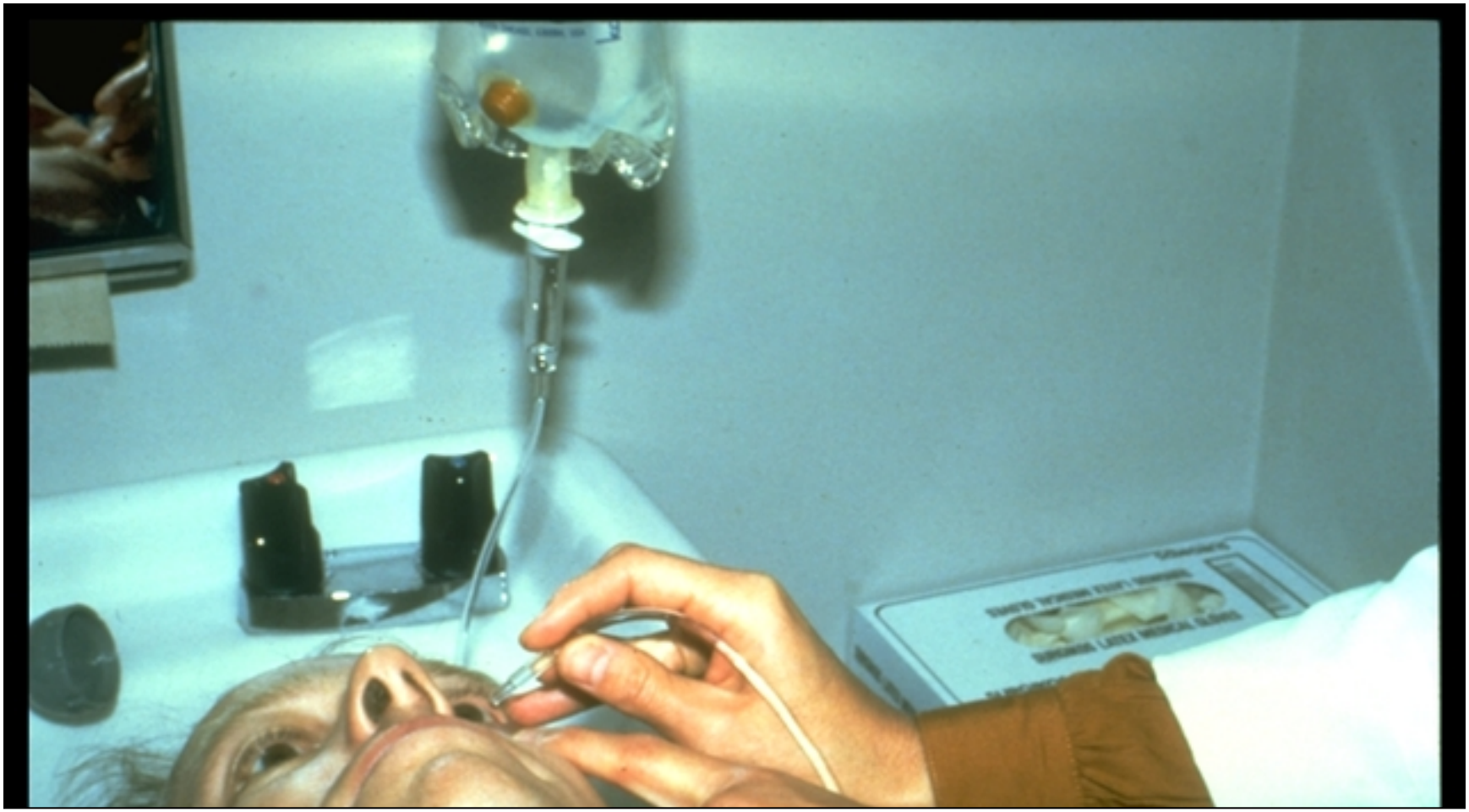
Hydrofluoric acid is an exception

Traumatic Eye Injuries

- Chemical Ocular Injury
  - Treatment:
    - Cycloplegic
    - Erythromycin ointment
    - Narcotic pain meds
    - Tetanus
  - Immediate ophtho eval if not completely normal after initial measures

Copious Irrigation

Immediate, copious
30 minutes – Morgan Lens
lactated Ringer's solution
Normal pH—between 7.3 to 7.6



Traumatic Eye Injuries

- Crazy Glue!

Traumatic Eye Injuries

- Crazy Glue!
  - Injury occurs only as a result of hard particles that form after drying
  - Ophtho uses crazy glue as treatment in clinic
  - Treatment:
    - Erythromycin ointment
    - Remove pieces that are easy to remove
    - Optho can remove residual glue within 48 hours

Traumatic Eye Injuries

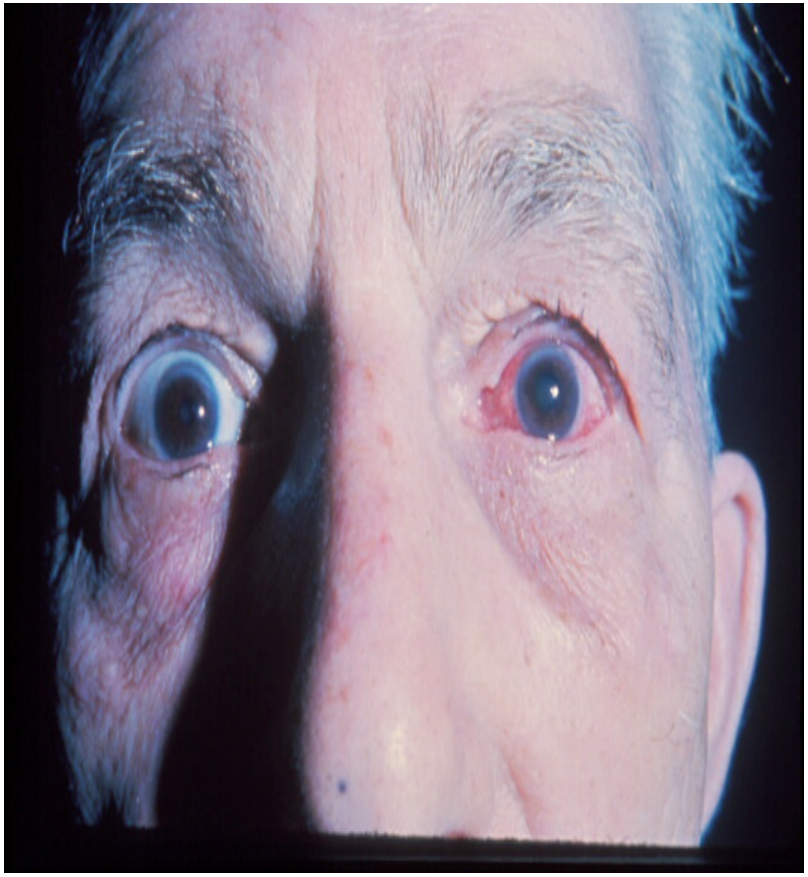
- Crazy Glue!
  - Mineral oil may help separate the lids
  - Never use acetone or other substance that breaks up glue

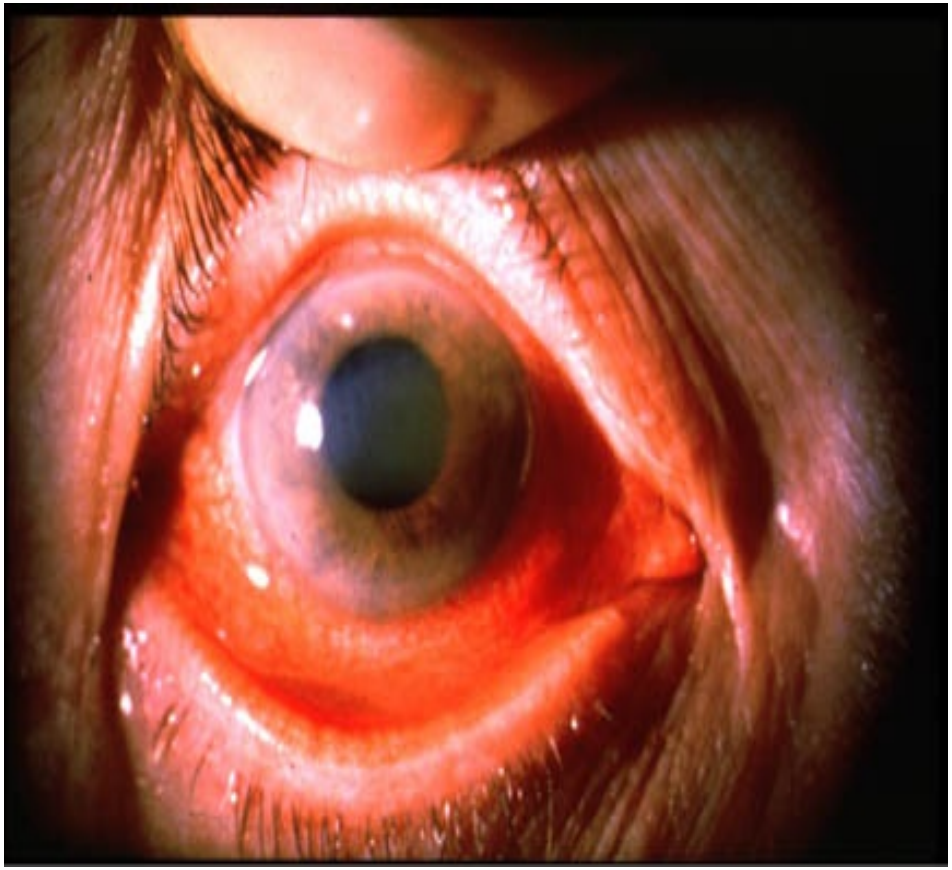
Acute Vision Loss

- Acute Angle Closure Glaucoma
  - Eye pain, headache, cloudy vision, colored halos around lights, conjunctival injection
  - Fixed, mid-dilated pupil
  - Increased IOP (40-70 mm Hg)
    - Normal range is 10 – 20 mm Hg
  - Nausea, vomiting

Acute Vision Loss

- Acute Angle Closure Glaucoma





Acute Vision Loss

- Acute Angle Closure Glaucoma
  - Immediate treatment:
    - Timolol
    - Apraclonidine
    - Prednisolone acetate
  - If IOP > 50 mm Hg or severe vision loss:
    - Acetazolamide 500mg IV
  - If no decrease in IOP or vision improvement:
    - IV Mannitol
  - Pilocarpine 1-2% in affected eye, pilocarpine 0.5% in contralateral eye (after IOP < 40 mm Hg)
  - Immediate Ophtho consult

Acute Vision Loss

- Optic Neuritis
  - Inflammation of the optic nerve
    - Infection, demyelination, autoimmune dx
  - Presentation:
    - Vision reduction (poor color perception)
    - Pain with extraocular movement
    - Afferent pupillary defect
  - Swelling of the optic disc may be seen

Acute Vision Loss

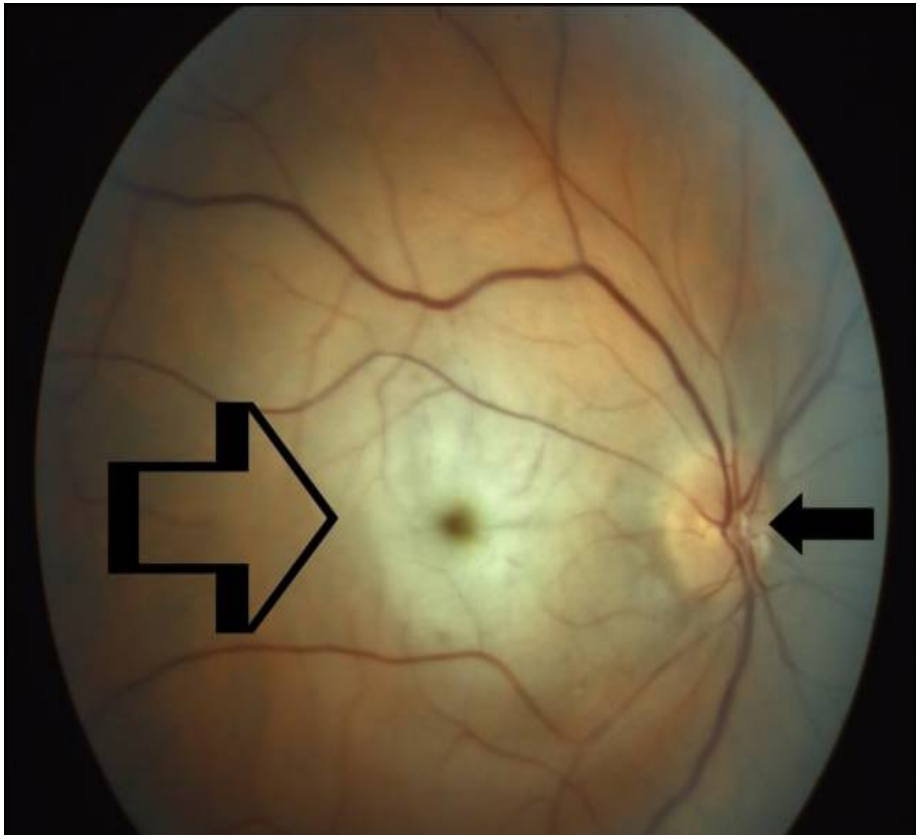
- Optic Neuritis
  - Diagnosis
    - Red Desaturation Test
      - Stare at bright red object with normal eye only
      - Object will appear pink or light red in affected eye
  - Treatment
    - Discuss with Ophtho

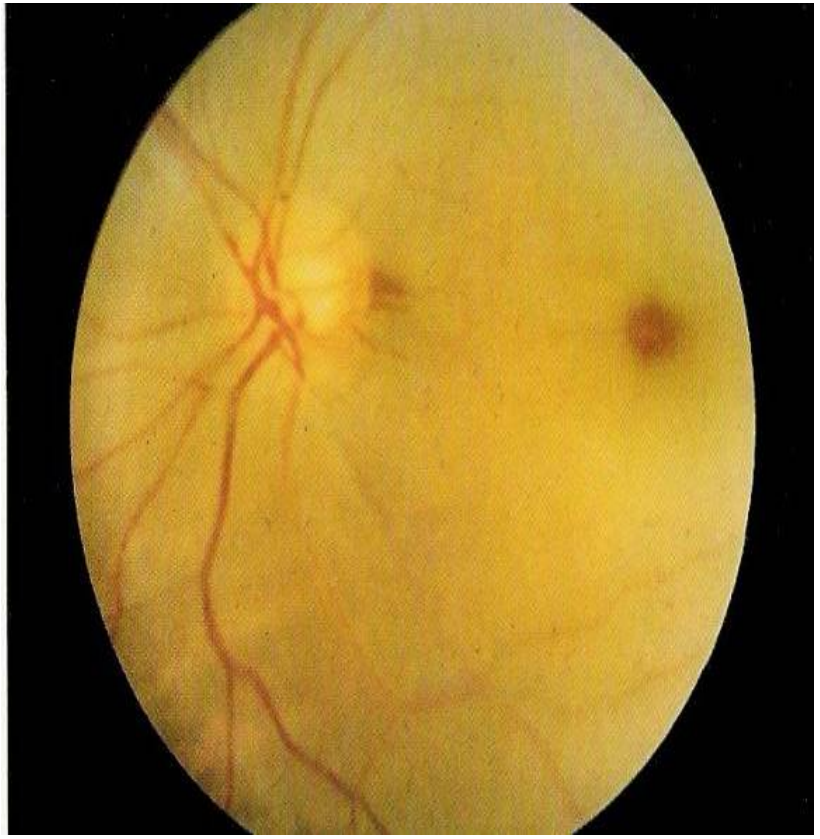
Acute Vision Loss

- Central Retinal Artery Occlusion
  - Causes
    - Thrombosis, embolus, giant cell arteritis, vasculitis, sickle cell disease, trauma
  - Preceded by amaurosis fugax
  - Painless vision loss
    - May be complete or partial
  - Afferent pupillary defect
  - Pale fundus with narrowed arterioles and segmented flows (boxcars) and bright red macula (cherry red spot)

Acute Vision Loss

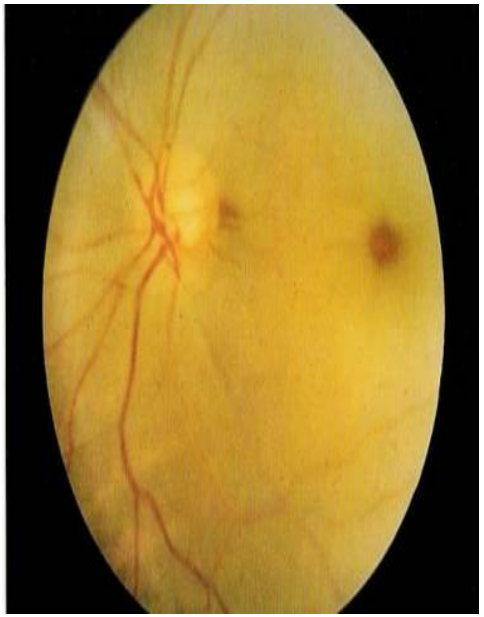
- Central Retinal Artery Occlusion





Acute Vision Loss

- Central Retinal Artery Occlusion
  - Treatment:
    - Ocular massage!
      - 15 seconds of direct pressure with sudden release
    - Topical timolol or IV acetazolamide
    - Emergent Optho eval

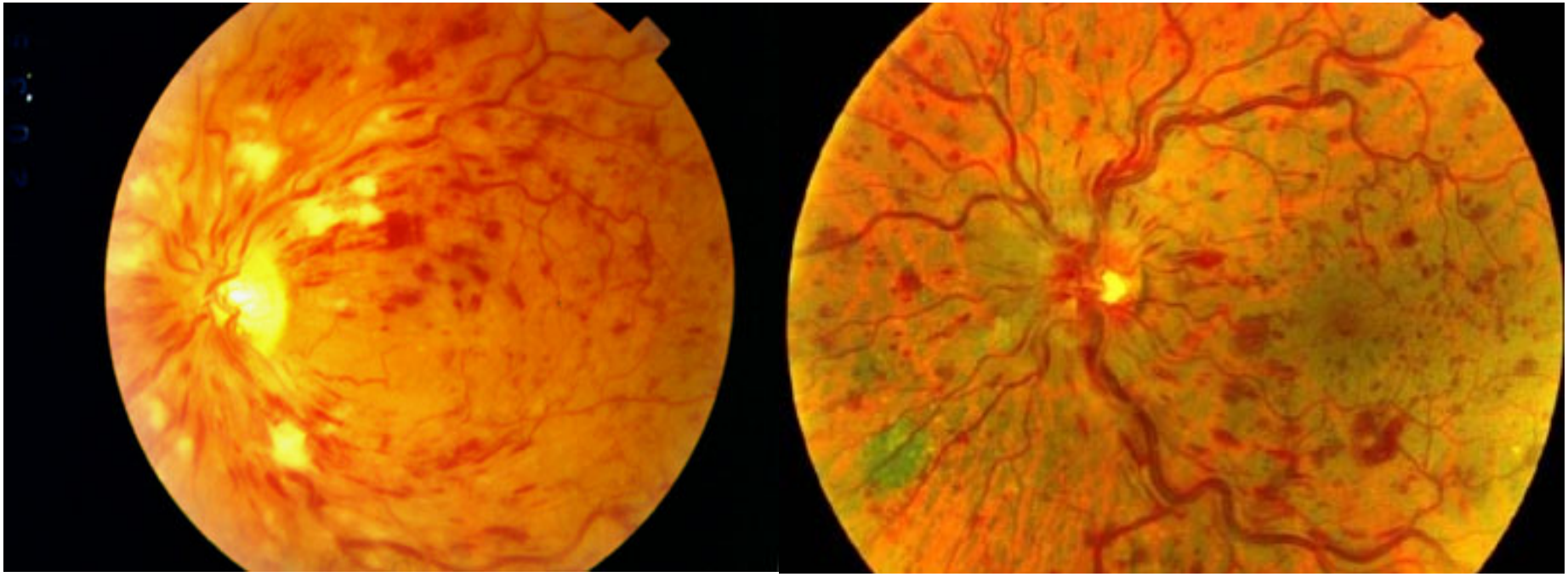


Acute Vision Loss

- Central Retinal Vein Occlusion
  - Thrombosis – diuretics and oral contraceptives predispose
  - Painless, rapid monocular vision loss
  - Fundoscopy:
    - Diffuse retinal hemorrhage
    - Cotton wool spots
    - Optic disc edema
    - “Blood and thunder”

Acute Vision Loss

- Central Retinal Vein Occlusion



Acute Vision Loss

- Central Retinal Vein Occlusion
  - Treatment:
    - ASA 325
    - Ophtho referral

Anterior Ischemic Optic Neuropathy (AION)

- Includes syndromes involving the optic nerve head, with visible optic disc edema.

- typically classified as:

- Arteritic AION (usually due to GCA)
- Nonarteritic AION

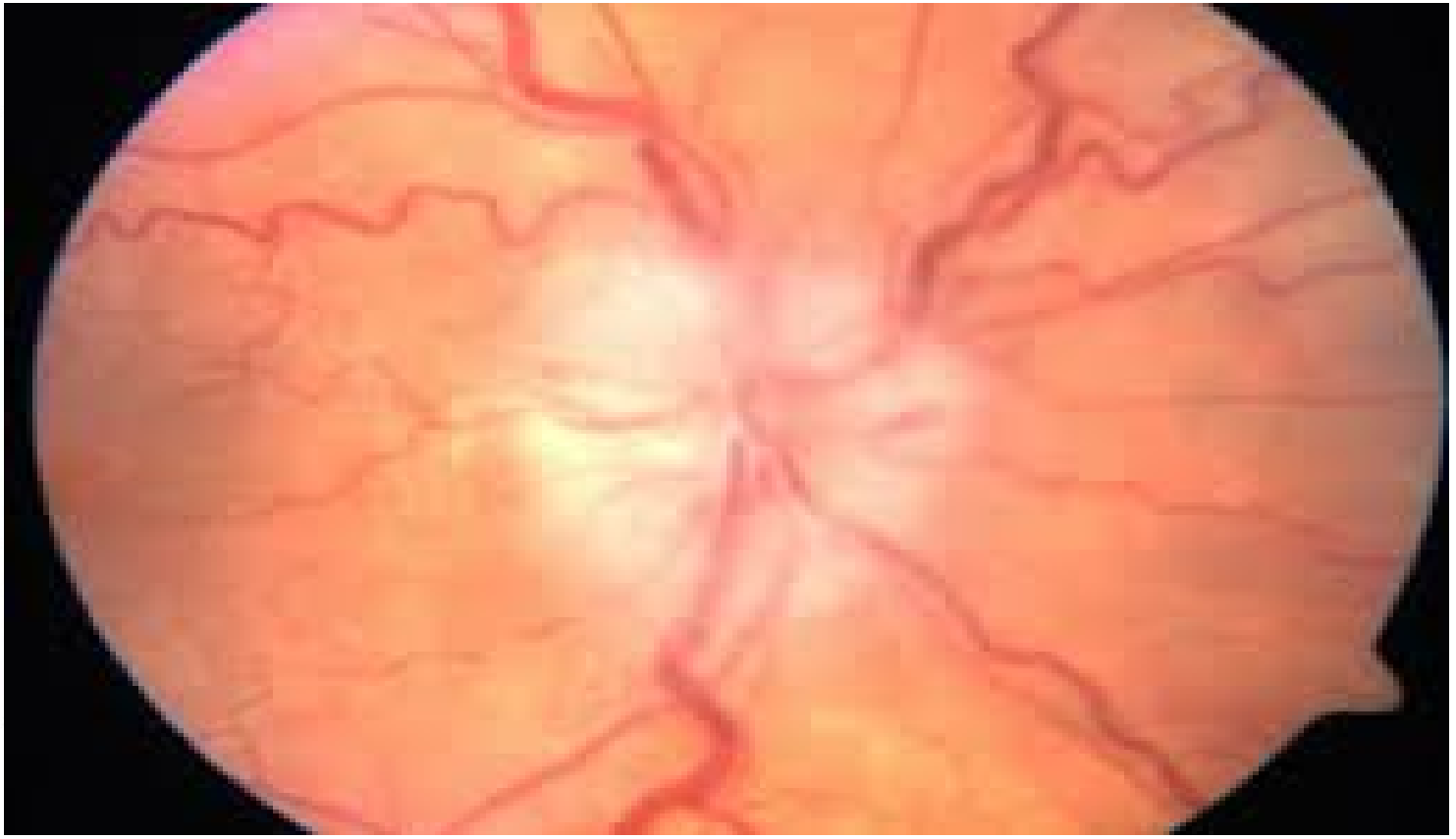
- The most important initial step in the management of AION is the assessment for evidence of GCA.

- Most cases of active GCA show markedly elevated ESRs (mean 70 mm/hr , often above 100 mm/hr).

- the level may be normal in up to 22% of patients with GCA .
- The test is nonspecific, elevation confirming only the presence of any active inflammatory process or other disorder affecting red cell aggregation. (C-reactive protein)

Acute Vision Loss

- Temporal Arteritis (Giant Cell Arteritis)



Acute Vision Loss

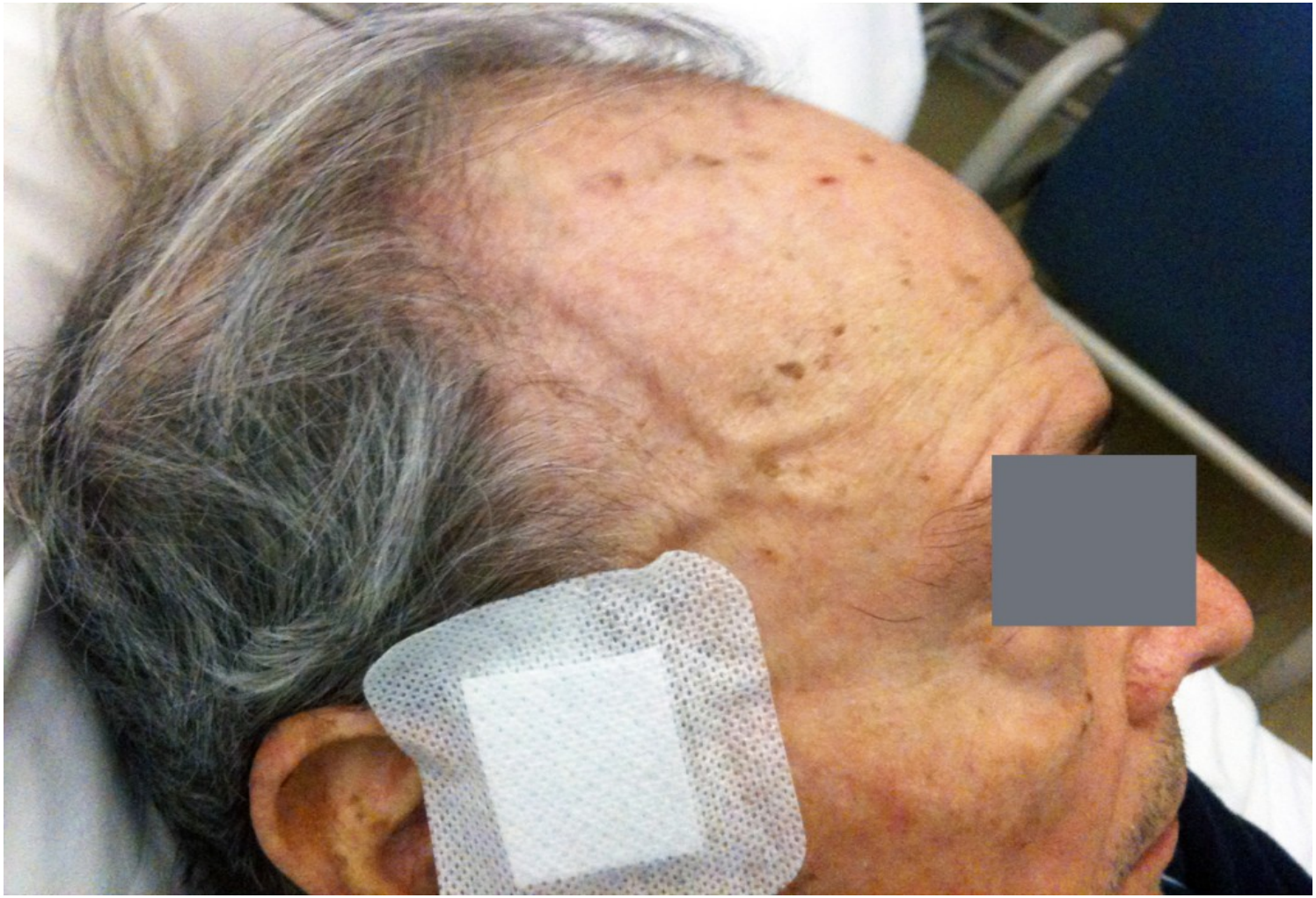
- Temporal Arteritis (Giant Cell Arteritis)
  - Systemic vasculitis that can cause ischemic optic neuropathy
  - Usually
    - > 50 years old
    - Female
    - Polymyalgia rheumatica

Acute Vision Loss

- Temporal Arteritis (Giant Cell Arteritis)
  - Presentation:
    - Headache
    - Jaw claudication
    - Myalgias, fatigue
    - Fever, anorexia
    - Temporal artery tenderness
    - TIA or stroke?
    - Afferent pupillary defect

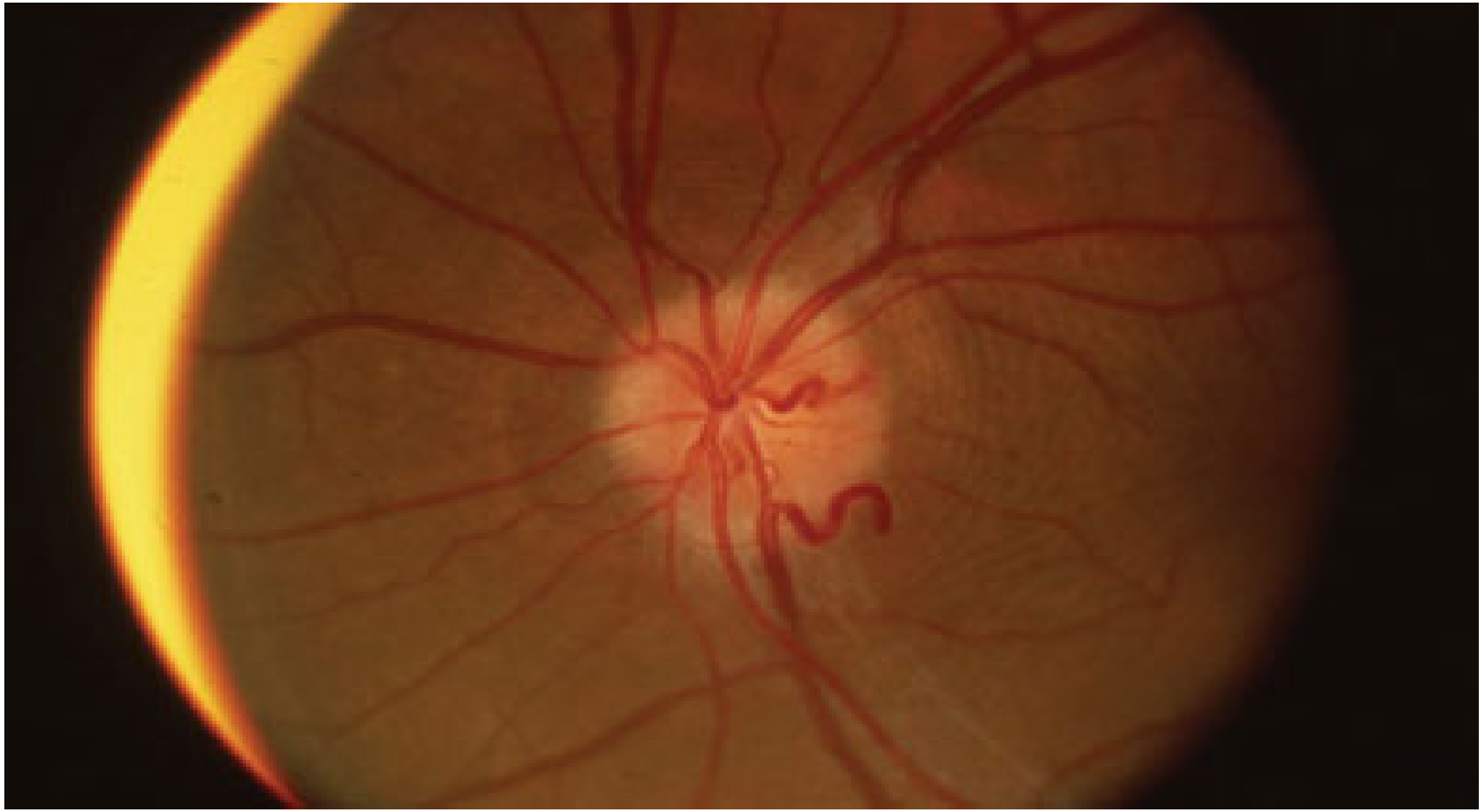
Acute Vision Loss

- Temporal Arteritis (Giant Cell Arteritis)
  - Diagnosis
    - Don’t waste your time if you suspect diagnosis
    - ESR, CRP
    - Temporal artery biopsy (gold standard)
  - Treatment
    - IV steroids and Ophtho consult



Nonarteritic anterior ischemic optic neuropathy

- More common (accounting for 90%-95% of AION cases) and occurs in a relatively younger age group (mean age, 60 years).
- NAION is presumed to be related to compromise of the optic disc microcirculation in the setting of structural "**crowding**" of the disc.



typically small in diameter and demonstrates a small or absent physiologic cup ("**disc at risk**").

Characteristic	Arteritic	Nonarteritic
Age	Mean, 70 years	Mean, 60 years
Sex	F > M	F = M
Associated symptoms	Headache, scalp tenderness, jaw claudication, transient visual loss	Usually none
Visual acuity	<20/200 in >60% of cases	>20/200 in >60% of cases
Disc	Pale swelling common Cup normal	Pale or hyperemic Cup small
Erythrocyte sedimentation rate	Mean, 70 mm/hr	Mean, 20–40 mm/hr
Fluorescein angiography	Disc delay and choroid delay	Disc delay
Natural history	Rarely improve Fellow eye, 54%–95%	16%–42.7% improve Fellow eye, 12%–19%
Treatment	Systemic steroids	None proven

Toxic/nutritional optic neuropathy

- Optic neuropathy resulting from toxic exposure or nutritional deficiency usually presents as a gradually progressive, bilaterally symmetrical, painless visual loss affecting central vision and causing central or cecocentral scotoma.
- Methanol and ethylene glycol toxicity result in rapid onset of severe bilateral visual loss with prominent disc edema.

- ***Thank you.***