

Definition

- Ocular trauma is defined as the result of mechanical, electrical, thermal or chemical energy damage to the eye.

Epidemiology-

Globally

Incidence - 55 million ocular injuries /year

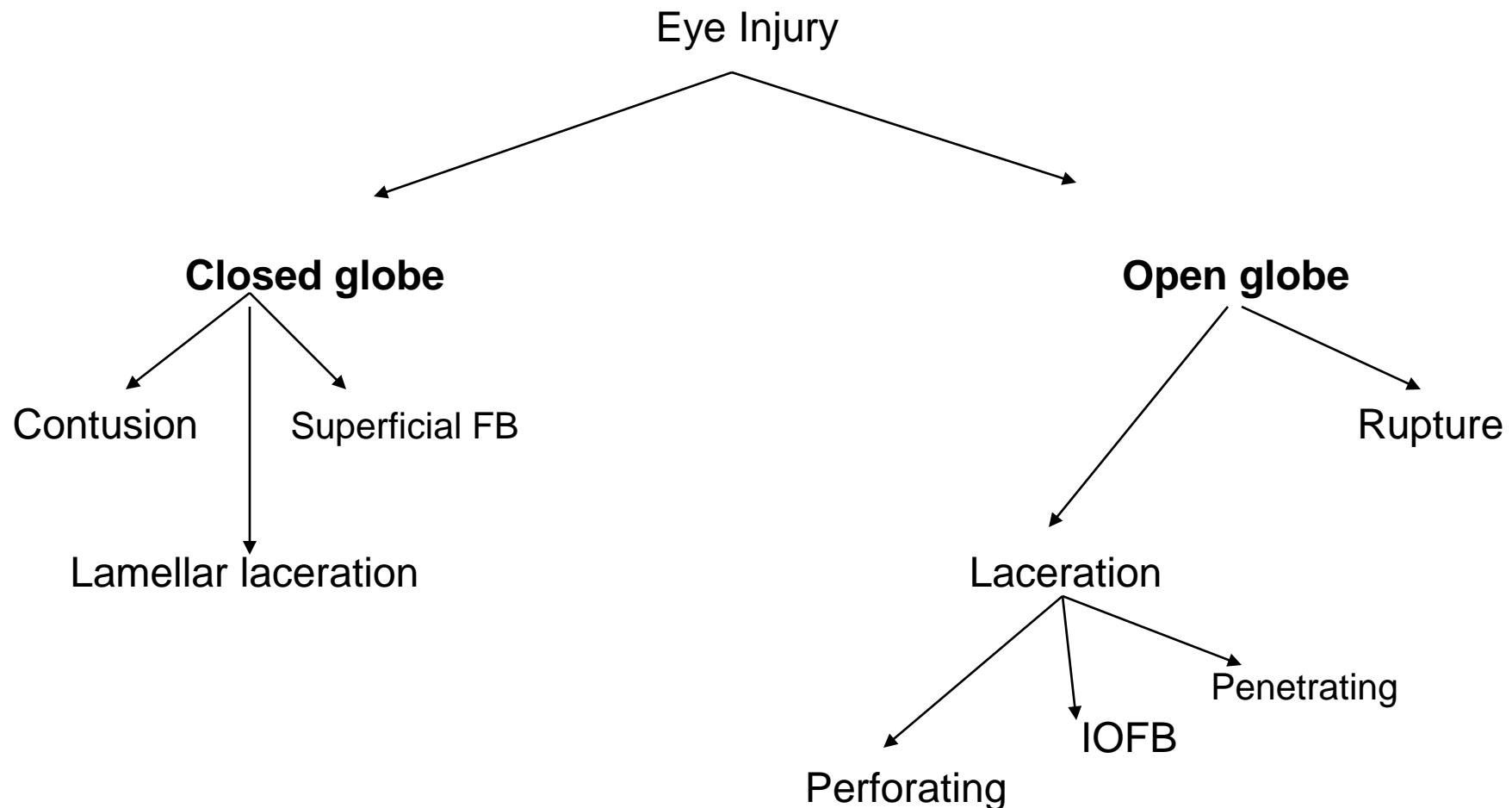
Prevalence-

Blindness due to injuries-1.6 million

(V/A<3/60)

Visual impairment - 19 million

International Classification



Closed globe Injury

- Grade-Presenting V/A
- 1. $\geq 20/40$
 - 2.20/50 to 20/100
 - 3.19/100 to 5/200
 - 4.4/200 to Light perception
 - 5.No light perception of bright light

Pupil - RAPD Positive
Negative

- Zone –
- I .External bulbar conjunctiva,cornea,sclera
 - II. Anterior segment including posterior lens capsule & pars plicata
 - III. Posterior segment past posterior lens capsule

Open globe Injury

Type/ Mechanism of injury-

Grade-Presenting V/A 1. \geq 20/40

2.20/50 to 20/100

3.19/100 to 5/200

4,4/200 to Light perception

5.No light perception of bright light

Pupil - RAPD Positive

Negative

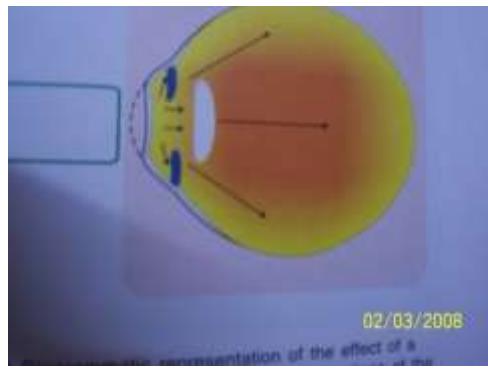
Zone – I. Isolated to cornea may include limbus

II, Corneo scleral limbus to 5mm posterior in sclera

III. Full thickness scleral injuries more than 5mm
posterior to the limbus.

Blunt Trauma-

Mechanism –



Lid injuries-



Lacerations



Black Eye

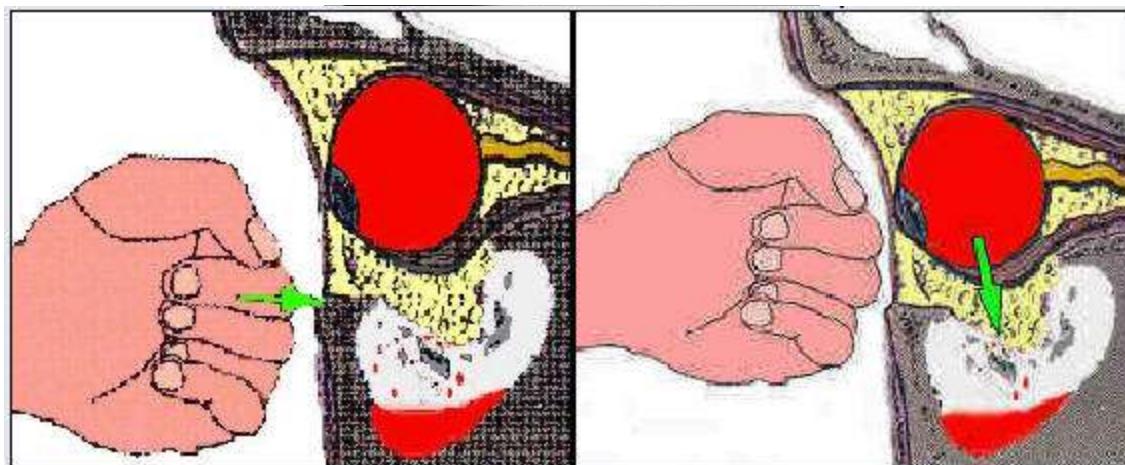
Orbital Injuries



Orbital Emphysema



Blow out #



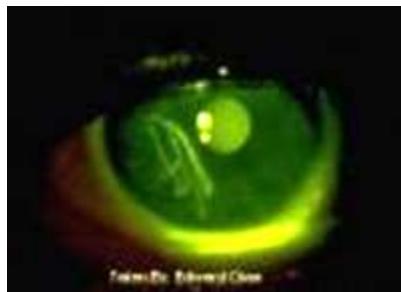
Tear –Drop sign



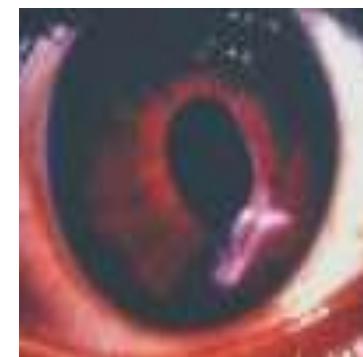
Injuries of Conjunctiva , Cornea & Sclera



Laceration& H-age



Abrasions



Rupture

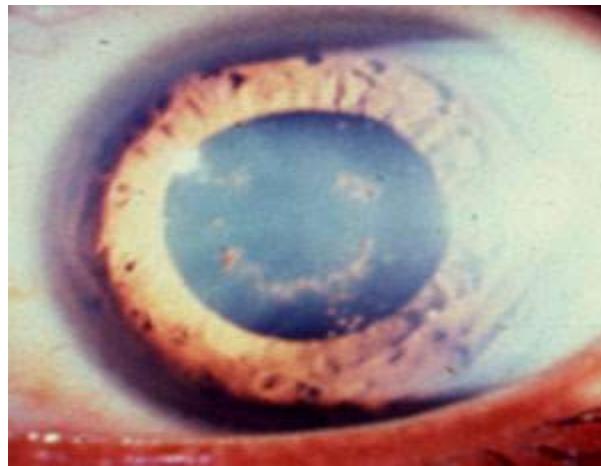
Injury to Iris, Angle & Traumatic glaucoma



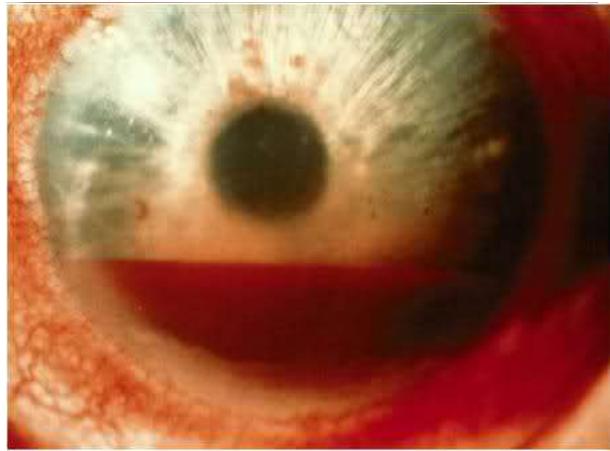
Iridodialysis



Angle recession



Vossius ring



Hyphaema



8Ball hyphaema

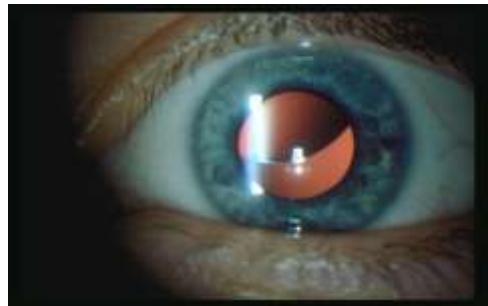


Corneal staining

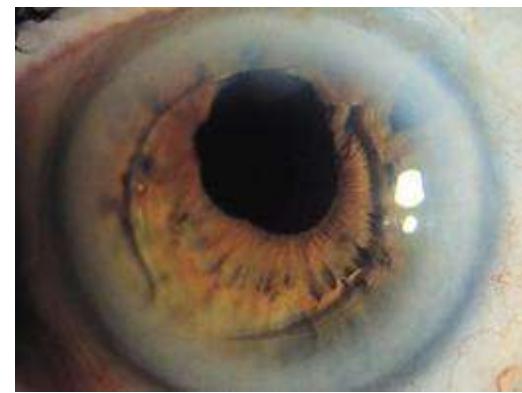
Lens injuries



Rosette cataract



Posterior
dislocation



Anterior dislocation

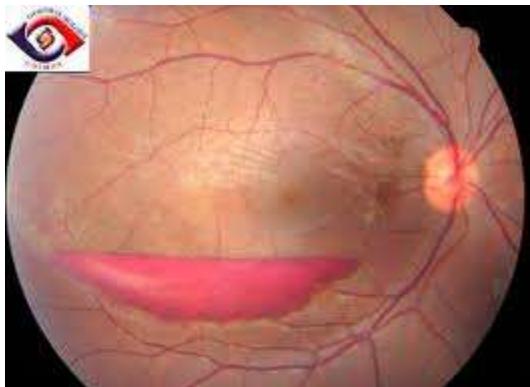
Vitreous & Retinal injuries



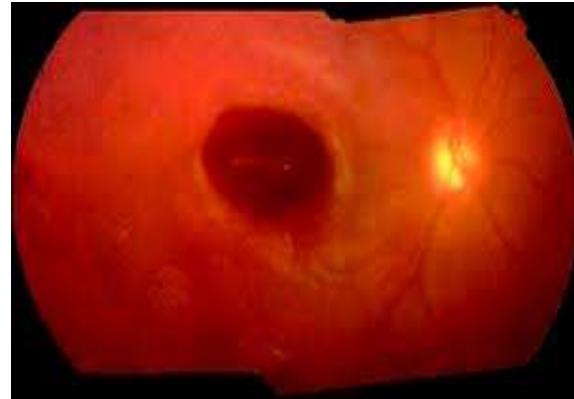
Vitreous base detachment & Retinal Dialysis



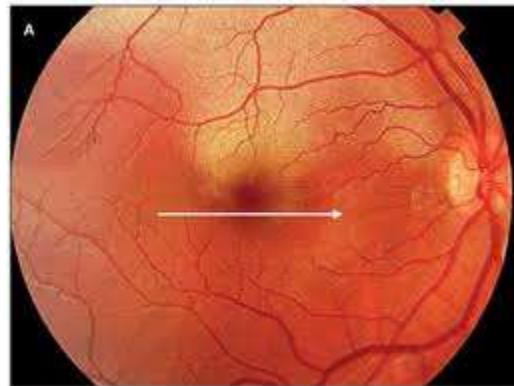
PVD



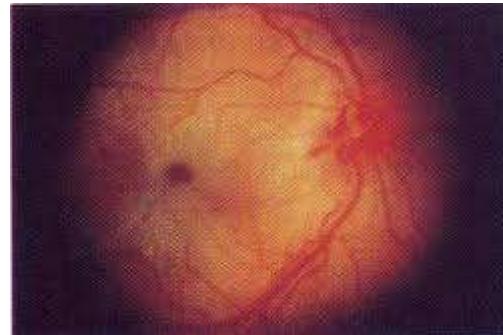
Subhyaloid H-age



Macular H-age



Commotio Retinae

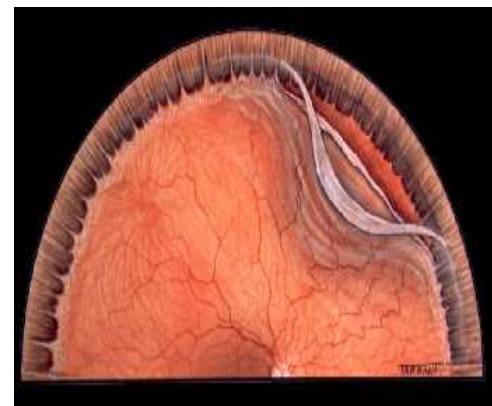




Choroidal Rupture



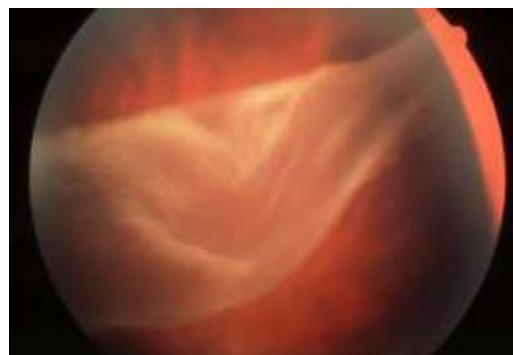
PVR



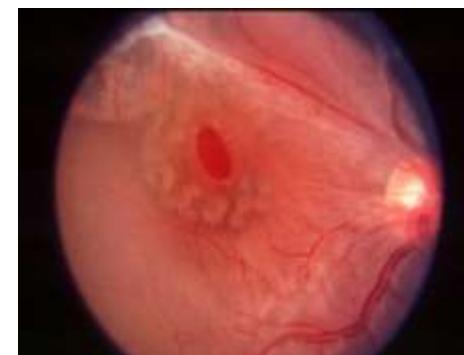
Avulsion of vitreous
base & retinal dialysis



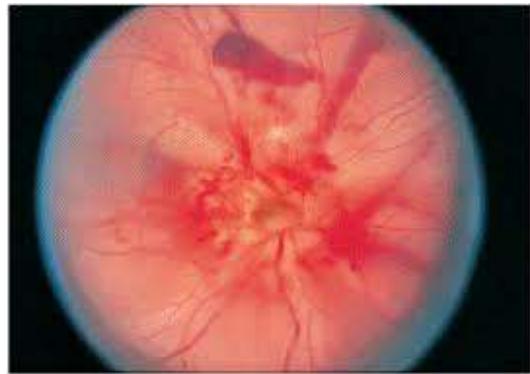
Retinitis sclopetaria



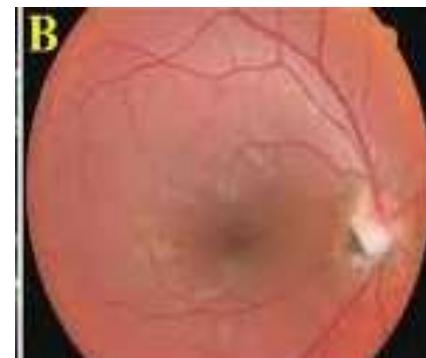
Equitorial tears



Macular hole

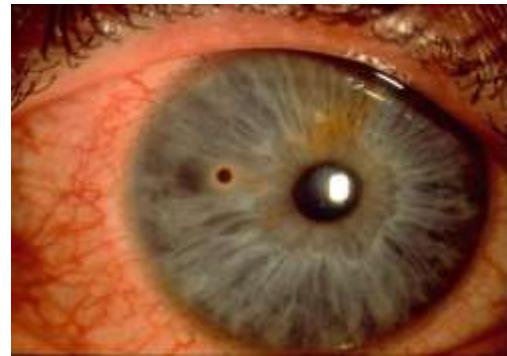


Optic nerve head avulsion



Optic atrophy

Superficial FBs



Penetrating Injury

Corneal Perforation with/without iris prolapse





Peaked pupil in corneo-scleral tear

Consequences-

Post traumatic iridocyclitis

Infection

Sympathetic Ophthalmitis

Endophthalmitis



Panophthalmitis





Cataract

Penetrating wounds with retention of foreign body(RIOFB)

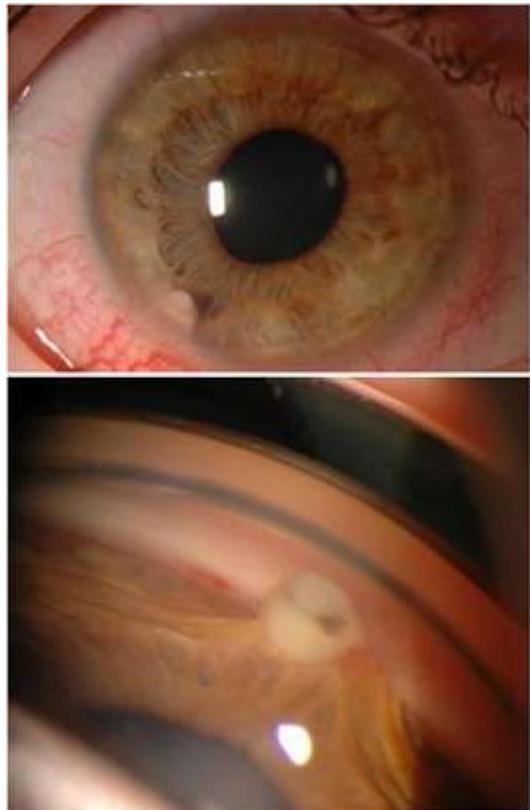
- 90% iron/steel (Industrial injuries)
- stone
- glass
- lead pellets
- Cu percussion caps

Size & Velocity of missile is important



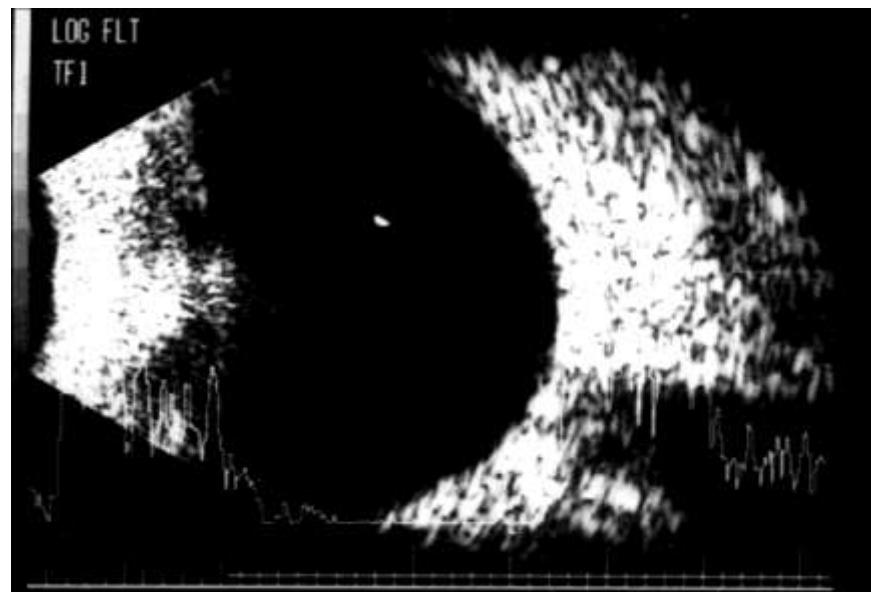
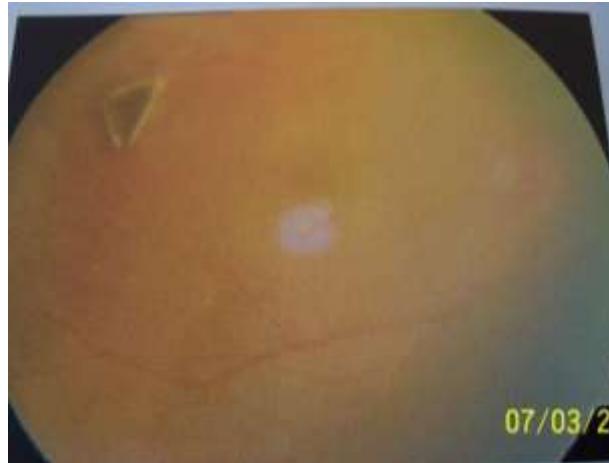
Tract in the Iris & lens



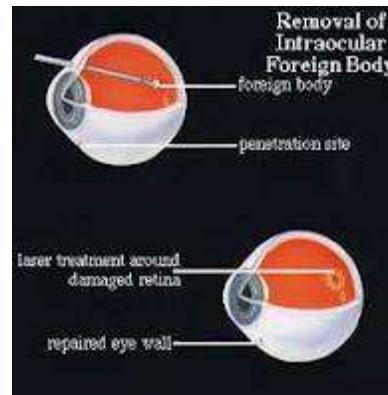


FB in the angle

Tract in the vitreous & liquefaction of vitreous



FB on the retina



Double perforation –FB in the orbit



Foreign body removal forceps

FB causes damage by:-

Mechanical effects

Introduction of infection

Specific action on the intraocular tissues

Lodgment of FB in the Posterior Segment can cause-
Widespread degeneration

Pigment disturbance at the macula

Concussion effects

Vitreous liquefaction

Vitreous H'ge

Fibrous proliferation in the vitreous

Retinal detachment

Infection-

Small flying metallic objects are rendered sterile.

Infection occurs because of wood/stone.

Prognosis bad despite prophylactic antibiotics.

Reaction of ocular tissues to FB-

Non organic FB-

Inert- delayed onset iridocyclitis

Glass, plastic, porcelain

Gold, silver, platinum, titanium

Lead-Coated with carbonate → less reaction

Fibrosis & Encapsulation

Aluminium- powdered → local reaction

Suppuration-Zn, Ni, Hg

Degeneration-Fe&Cu are widely distributed in the eye due to electrolytic dissociation.

Iron/Steel depending on ferrous content- **Siderosis**

Iron combines with cellular proteins → damage esp to epithelial cells
→ atrophy.

Earliest Manifestation-Deposition in the anterior lens capsular cells



Oval patches of rusty deposits
of the size of dilated pupil.



Iris heterochromia



Sphincter atrophy- Mydriasis

Retinal blood vessels
attenuation.

RP like pigmentation.

ERG- ↑ amplitude of a
wave as

condition
progresses b wave also ↓

ERG becomes flat

Prussian blue stain.

Copper-

Pure Cu- violent reaction → profuse fibrosis
→ encapsulation/Suppuration → phthisis

Alloy-Chalcosis

Cu is deposited where resistance to migration occurs due to continuous membranes like DM

Kayser Flaisher ring



Sunflower cataract



History

Thorough exam

Wound of entry

Gonioscopy

Tracks in lens

Fundus exam

Radiography-Caldwell and Lateral view

Limbal ring/ Contact lens- Meridian
Distance from limbus

CT

MRI-wood and vascular changes detected

Contraindicated in metallic FB

USG

Locater

Treatment –

Removal

Intravitrial magnets

Lens extraction

Sympathetic Ophthalmitis



Dalen Fuch's nodules

Chemical Injuries Of The Eye

Common causes of chemical injuries

Class	Compound	Sources	Comments
Alkali	Ammonia $[NH_4]$	Fertilizers Cleaning agents, refrigerant	Combines with water In tear film to form NH_4OH Rapid deep entry
	Lye $NaOH$	Drain Cleaner	Rapid deep entry
	$Mg(OH)_2$	Sparklers	Thermal&Alkali injury
	Lime $Ca(OH)_2$	Plaster Cement White Wash	Most common cause Poor penetration Toxicity ↑ by retained particles

Acid	Sulphuric	Battery acid	Combines with water to produce charring may be associated with FB/laceration
	Sulphurous	SO ₂ Fruit/Veg preservative Bleach Refrigerant	Penetrates more easily than other acids
	Hydro fluoric acid	Glass etching	Penetrates easily Causes most severe acid injury
	Acetic	Vinegar	Mild with lesser severe with higher concentration

Pathophysiology-

Severity related to surface area of contact & degree of penetration.

Alkalies penetrate deeper

Depending on degree of penetration there may be damage to

Corneal & conjunctival epithelium, limbal stem cells,

keratocytes, stromal nerve endings,

endothelium, lens, ciliary body and vascular endothelium

of conjunctiva, episclera, iris, and ciliary body.

The depth of ocular surface penetration & possible limbal stem cell damage can be evaluated indirectly by

Vascular ischaemia

and

Necrosis of limbal & bulbar conjunctiva.

Thoft classification-

Grade I- Little/no loss of limbal stem cells and presents with little/no ischaemia

Grade II- Subtotal loss of limbal stem cells and ischaemia of less than one half of limbus

Grade III- Total loss of limbal stem cells with preservation of proximal conjunctival epithelium and presents with ischaemia of one half to entire limbus.

Grade IV- Total limbal stem cell loss as well as loss of proximal conjunctival epithelium presents with extensive damage to the entire anterior segment.

Grading of severity of chemical injuries

Grade I (excellent prognosis)

- Clear cornea
- Limbal ischaemia - nil

Grade II (good prognosis)



Grade III (guarded prognosis)



Grade IV (very poor prognosis)



- Cornea hazy but visible iris details
- Limbal ischaemia < 1/3

- No iris details
- Limbal ischaemia - 1/3 to 1/2

- Opaque cornea
- Limbal ischaemia > 1/2

The principles guiding evaluation and management are based on addressing the following pathophysiologic mechanisms-

1. Regeneration of ocular surface epithelium and its state of differentiation
2. Stromal matrix remodelling including repair and degradation
3. Infection.

Pathophysiologic & Clinical course-McCully

Phase	Period	Patho physiology	Evaluation	Treatment
Immediate			Extent Depth Toxicity Concentration	Thorough wash for 30 min till neutralization Removal of particles under L.A Debridement
Acute	0 - 7 days	Within 0 -24 hrs Peripheral PMN Infiltration ↑IOP Re epithelization ↑MMP-9 Degradation MMP1,8 &Remodelling of stroma.	Re epithelization IOP Progressive inflammation	Topical- corticosteroids 2 hrly 10% Na ascorbate 2hrly 10 % Na citrate 2 hrly 1% Tetracycline ointment Qid Antiglaucoma T/t Cycloplegics Lubricants Oral-Na ascorbate 1g Qid Doxycycline 100 mg Bid Gr-IV-Conjunctival & Tenon'advancement

Early repair	7 -21 days	<p>Epithelial migration in less severe injuries</p> <p>14 -21 days maximum collagen synthesis</p> <p>Collagenolysis</p> <p>2nd wave of inflammatory cell infiltration & persistance till lack of epithelization</p> <p>&/stimulus from necrotic conjunctival tissue</p>	<p>Epithelization defects</p> <p>& change in ischaemic pattern.</p>	<p>Taper topical steroids after close monitoring</p> <p>Topical progestational steroids</p> <p>NSAIDs/both 2 hrly</p> <p>Continue other T/t</p>
Late repair	21 days – several months	Balance of degradation and collagen synthesis depends on epithelization	Healing pattern	<p>Taper medical therapy after reepithelization (Gr-II)</p> <p>Perform ocular surface transplantation(Gr-III/IV)</p> <p>Perform tectonic procedures</p> <p>tissue adhesive</p> <p>Keratoplasty</p> <p>Large diameter keratoplasty</p> <p>Keratoprosthesis only if absolutely necessary.</p> <p>Treat sequelae</p>

Revision

- What are the effects of blunt trauma on the anterior segment of the eye?

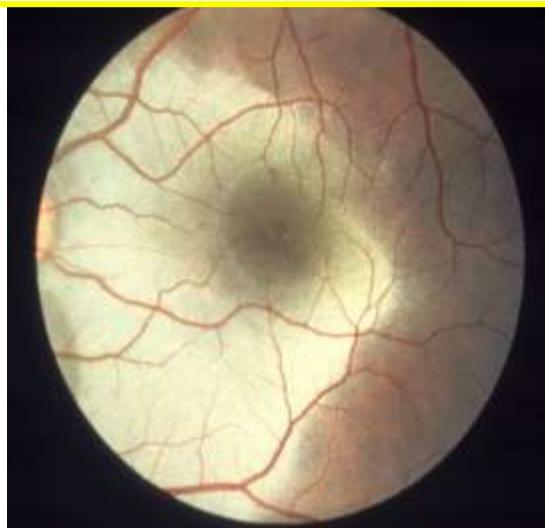
Anterior segment complications of blunt tra

			
Hyphaema	Sphincter tear	Iridodialysis	Vossius ring
			
Cataract	Lens subluxation	Angle recession	Rupture of globe

Q-2

- What are the effects of blunt trauma on the posterior segment of the eye?

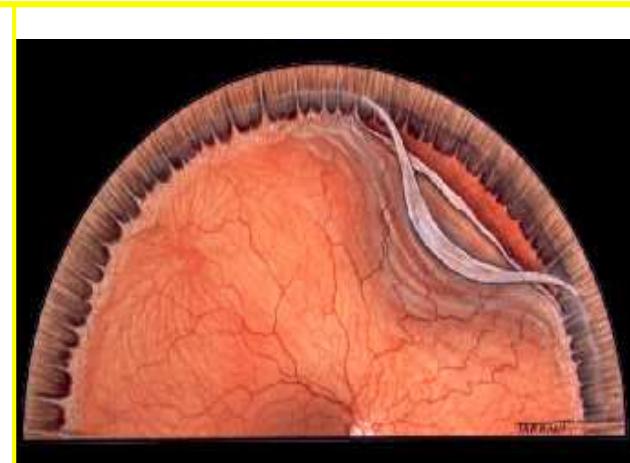
Posterior segment complications of blunt tra



Commotio retinae



Choroidal rupture and haemorrhage



Avulsion of vitreous base and retinal dialysis



Equatorial tears



Macular hole



Optic neuropathy

Q-3

- What are the complications of penetrating trauma?

Complications of penetrating trauma



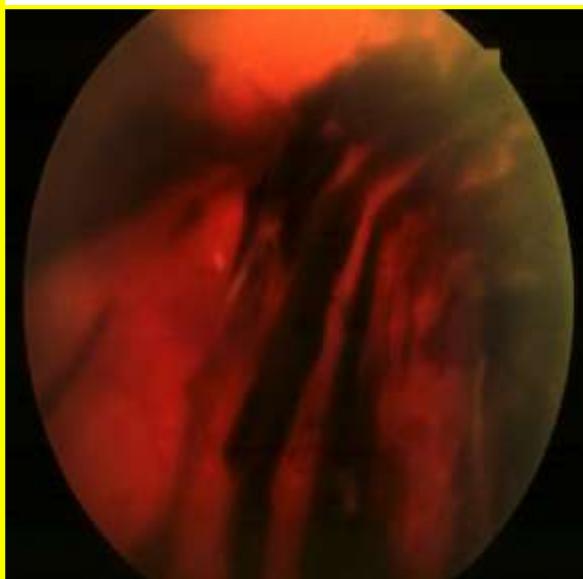
Flat anterior chamber



Uveal prolapse



Damage to lens and iris



Vitreous haemorrhage



Tractional retinal detachment



Endophthalmitis

Q-4

- What are the grades of chemical trauma?

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