Ophthalmology

TRAUMA

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EYELID TRAUMA

1- Haematoma (Black eyes) (Panda eyes):

It is the *most common* result of blunt injury to the eyelid or forehead (due to continuous space below the tense aponeurosis of scalp that extends to the loose space around the eye) and it is generally innocuous. It is important to exclude the following serious associated conditions:

a- Trauma to the globe.
b- Orbital walls fracture.
c- Basal skull fracture.

2- Laceration:

Two types of eyelid laceration:

a- Superficial lacerations: they are parallel to the lid margin without gaping.
Treatment: suturing.
b- Lid margin lacerations: which are invariably gape and must therefore be carefully sutured with perfect alignment to prevent notching.
***** Improper suturing may end with notching or fibrosis (scars) that causes foreign body sensation and then might end with corneal abrasion and its consequences.

ORBITAL FRACTURES

- Blow-out floor fracture:

It is typically caused by sudden increase in the orbital pressure by a striking object such as a fist or tennis ball. Since the bones of the lateral wall and roof are usually able to withstand such trauma, the fracture most frequently involves the floor and occasionally, the medial orbital wall may also be fractured by such type of trauma.

Signs:

1- Periocular signs: include ecchymosis, oedema and subcutaneous emphysema.

2- Infraorbtal nerve anesthesia: involving the lower lid, cheek, side of the nose, upper lip, upper teeth and gums.

3- Vertical diplopia: happens due to:

a- Hemorrhage and oedema of the orbit restricting the movements of the globe.

b- Mechanical entrapment of the inferior rectus or inferior oblique muscle or both within the fracture.

c- Direct extraocular muscle injury.

4- Enophthalmos may be present if the fracture is large.

5- Ocular damage, e.g. hyphaema, angle recession and retinal dialysis.

Treatment of Blow out floor fracture:

Initially, it is *conservative* with systemic antibiotics; the patient should be instructed not to blow the nose to avoid transmission of bacteria from maxillary sinus to the orbit.

Subsequently, it is aimed at prevention of permanent vertical diplopia and/or cosmetically unacceptable enophthalmos.

Indications of surgery:

1- Wait for 2 weeks (not more as fibrosis make the surgery difficult or impossible) until hemorrhage, edema and inflammation settles, then check for diplopia in primary position and down gaze, if the diplopia still exists after 2 weeks then, surgery is indicated to release the muscles and to cover the defective fractured bone by bone graft or synthetic materials.

2- Enophthalmos more than 2 mm which causing cosmetic blemish.

TRAUMA TO THE GLOBE

- Closed injury: it is commonly seen due to blunt trauma. The outer corneoscleral wall of the globe is intact; however, intraocular damage may be present.

- Open injury: it involves a full-thickness wound of the corneoscleral wall.

Open injury can occur by the following *mechanisms*:

1- Blunt trauma: can lead to a full-thickness wound at its weakest point. This is called **rupture globe**.

2- Trauma by sharp object: e.g. knife can cause a full-thickness wound which is called **laceration**.

3- Trauma by high velocity sharp object: e.g., shell injury, small foreign bodies scattering from hammer or other material, which can

cause ***single*** full-thickness wound without an exit wound (there is intraocular retention of the foreign body), this type of wound is called **"Penetration wound"**. If it cause ***two*** full-thickness wounds, one entry and one exit, which is usually caused by a missile (no retention of the foreign body), this type of wound called **"Perforation wound"**.

General principles of management:

1- Initial assessment:

a- Determination of any **associated life-threatening problems,** and general condition should be stabilized.

b- History: circumstances, timing and likely object.

c- Thorough **examination** of both eyes and orbits.

2- Special investigations:

a- Plain radiographs: when a foreign body is suspected, to localize it and plan for the surgery.

b- CT: superior to plain x ray in detection and localization of intraorbital foreign body. It is also used in determining the integrity of intracranial, facial and intraocular structures.

*** NB:** MRI should never be performed if a metallic foreign body is suspected as this may induce more traumas and damage by its movement again.

c- Ultrasound: detection of intraorbital foreign body, globe rupture (as the rupture may be posteriorly hidden), retinal detachment.

d- Electrophysiological tests (VEP, EOG, ERG) in assessing the integrity of the optic nerve and retina.

BLUNT TRAUMA

Causes: squash balls, luggage straps and champagne corks.

Complications:

1- Anterior segment complications:

a- Corneal abrasion: epithelial loss, which stains with fluorescein, treated by pressure bandage for 24 to 48 hours.

b- Hyphaema: hemorrhage in the anterior chamber usually occurs in children and young persons. The source of bleeding is the iris or ciliary body. Secondary bleeding can occur during the first week and is more serious than initial bleeding.

***** Hyphaema may cause secondary glaucoma by three ways: EITHER through occluding of the trabecular meshwork by blood

cells and proteins, OR by pupillary block (due to clot that occludes pupil) OR by the associated iritis and its complications e.g. Anterior and posterior synechia. Corneal staining (haemosiderosis) can occurs duo to persistent Hyphaema specially if associated with rising IOP. It is due to deposition of iron on corneal endothelium which leads to sever affection of VA where penetrating keratoplasty indicated.

***** If hyphaema fills more than half of the anterior chamber, the patient should be admitted to hospital with complete bed rest, and if it is mild hyphaema and fills less than half of the anterior chamber, the patient is discharged but with complete bed rest in home. Bed rest is important step in treatment of hyphaema to avoid secondary bleeding.

***** Surgery ("**Paracentesis**") is indicated when there is:

1- persistent total hyphema.

2- sever and persistent rising IOP.

3- corneal staining.

In paracentesis, washing of AC is usually done with replacement of blood by a visco-elasitc substance or fluid e.g. normal saline, ringer solution or balance salts solution (BSS).

When sugery not indicated **cycloplegia, topical steroids, bed rest, IOP monitoring & follow up** are required.

c- Traumatic mydriasis: it is often permanent due to damage to the iris sphincter muscles. Permanent large mydriasis lead to photophobia an blurred vision.

d- Iridodialysis: is a dehiscence of the iris from the ciliary body at its root. Usually the pupil has a D shape and the vertical part of D is toward the dehiscent. It is innocuous and asymptomatic or occasionally can cause monocular diplopia (2 pupils).

e- Ciliary body: - Ciliary shock (ocular hypotonia).

- Anterior chamber angle recession (lead to glaucoma).

*** AC angle recession:** recession of the angle between the periphery of the iris and anterior face of ciliary body, which seen by gonioscopy. Angle recession per se is an innocuous thing, but may indicate severe trauma and associated with damage to the trabecular meshwork that may cause "Angle recession glaucoma". This type

of secondary glaucoma might occur after months or even a long time (years).

f- Lens: cataract. R: surgery

g- Rupture of the globe: usually anterior with prolapse of intraocular tissues, but occasionally posterior (occult).

2- Posterior segment complications:

a- PVD (posterior vitreous detachment): it may be associated with vitreous hemorrhage or retinal tear (where pigment cells similar to tobacco dust may be seen floating in the anterior vitreous).

b- Commotio retinae: concussion of the sensory retina resulting in cloudy swelling area of retina due to damage of inner part of blood retinal barrier. If the oedema is persists and involving the macula, it will cause cystoid macular edema (CME) and permanent diminish of VA.

c- Choroidal rupture.

d- Retinal break: retinal dialysis, tears and holes.

*** Retinal dialysis:** dis insertion of part of the extreme periphery of sensory retina from its attachment to the non-pigmented epithelium of ciliary body.

e- Optic neuropathy: is an uncommon but often devastating cause of permanent visual loss.

f- Optic nerve avulsion: is rare and typically occurs when an object intrudes between the globe and the orbital wall, displacing the eye.

PENETRATING TRAUMA

Causes:

Penetrating trauma is three times more common in males than in females, and in younger age group than in old age group. The most frequent causes are assault, domestic accidents and sort. The extent of the injury is determined by the size of the object, its speed at the time of impact and its composition.

Complications:

1- Anterior segment complications:

a- Small corneal lacerations: with formed anterior chamber, it does not require suturing as it heals spontaneously.

b- Medium-sized corneal lacerations: usually require suturing to reform the anterior chamber, especially if the anterior chamber is shallow or flat.

c- Corneal lacerations with iris prolapse:

In the 1st 24h, reposition of the iris (still viable) and suturing of lacerations.

After the 1st 24h, the iris should be excised and then suture the lacerations.

d- Corneal lacerations with lenticular (lens) damage:

Suturing of the laceration and removing of the damaged lens and replaced by IOL.

e- Anterior scleral laceration ± Iridociliary prolapse and vitreous incarceration:

If **(-)** i.e. Anterior scleral laceration only, then suturing only,

If **(+)**, then reposition of exposed viable uveal tissue and cut prolapsed vitreous flush within the wound otherwise subsequent vitreoretinal traction occur and lead to retinal detachment.

2- Posterior segment complications:

- **Posterior scleral lacerations:** usually associated with retinal breaks unless very superficial. The sclera should be sutured with treatment of retinal break prophylactically by cryotherapy to avoid rhegmatogenous R.D.

INTRAOCULAR FOREIGN BODIES

An Intraocular foreign body may traumatize the eye by the following mechanisms:

1- Mechanically (laceration).

2- Introduce infection.

3- Toxic effects on the intraocular structures.

Stones and organic foreign bodies are prone to result in infections.

Glass, plastics, gold and silver are inert and can be left with no harmful effect.

Iron and copper foreign bodies undergo dissociation and result in siderosis and chalcosis respectively. These foreign bodies should be removed immediately or within few days.

Siderosis:

Intraocular ferrous foreign body undergoes dissociation resulting in the deposition of Iron in the intraocular epithelial cells (especially in lens and retina) that leads to toxic effect on cellular enzymes that leads to cell death.

Features of siderosis:

Which are: cataract, reddish-brown staining of the iris, secondary glaucoma (due to trabecular meshwork deposition) and pigmentary retinopathy (blindness).

Treatment: iron foreign body should be removed.

Chalcosis:

The ocular reaction to an intraocular foreign body with a high copper content involves a violent endophthalmitis-like picture which often progress to phthisis bulbi.

Treatment: Copper foreign body should be removed.

***** Endophthalmitis means that there is inflammation of all intraocular structures except the sclera, but if inflammation involves the sclera it is called "**Panophthalmitis**".

ENUCLEATION (EXCISION OF THE EYEBALL)

Primary enucleation: should be performed only for sever injuries, with no prospect of retention of vision when it is impossible to repair the sclera.

It has been recommended that enucleation should be performed within 10 days of the original injury in order to prevent the very remote possibility of sympathetic ophthalmitis.

Secondary enucleation: may be considered following primary repair if the eye is severely and irreversibly damaged, particularly if it is also unsightly and uncomfortable.

SYMPATHETIC OPHTHALMITIS

It is a very rare, bilateral, granulomatous panuveitis which occurs after **open** ocular injuries usually associated with uveal prolapse or less frequently following intraocular surgery, when the uveal tissue came in contact with conjunctiva. It occurs due to antibody formation against the uveal tract lead to severe immunological inflammation of the injured eye and the fellow eye.

The traumatized eye is referred to as the **"exciting eye"**, and the fellow eye, which also develop uveitis, is called **"Sympathizing eye"**.

Presentation:

65% of cases present between 2 weeks to 3 months after initial injury, 90% of all cases occur within the first year but it can occurs later on after many years e.g. 20 years.

Signs:

- The exciting eye shows evidence of the initial trauma and is frequently very red and irritable.
- The sympathizing eye becomes photophobic and irritable.
- Both eyes then develop a chronic granulomatous anterior uveitis with iris nodules and large keratic precipitates.
- Bilateral disc swelling and multifocal choroiditis.

Course:

Rarely, the uveitis is mild and self-limiting, but **usually**, intraocular inflammation becomes chronic and if not treated appropriately, it may lead to cataract, glaucoma and phthisis bulbi in both eyes.

Treatment:

1- Systemic steroid.
2- Topical steroid.
3- Short acting mydriatics.
4- Systemic Immunosuppressive agents in resistant cases.

But the prognosis is usually poor

Chemical injuries

Causes

Chemical injuries range in severity from the trivial to the potentially blinding. The majority are accidental, and a few due to assault. Two-thirds of accidental burns occur at work and the remainder at home. Alkali burns are twice as common as acid burns since alkalis are more widely used both at home and in industry. The severity of a chemical injury is related to the properties of the chemical, the area of affected ocular surface, duration of exposure (including retention of particulate chemical on the surface of the globe or under the upper

lid) and related effects such as thermal damage. Alkalis tend to penetrate more deeply than acids, as the acids coagulate surface proteins, forming a protective barrier. The most common involved alkalis are ammonia, sodium hydroxide and lime. The commonest acids implicated are sulphuric, sulphurous, hydrofluoric, acetic, chromic and hydrochloric. Ammonia and sodium hydroxide may produce severe damage because of rapid penetration. Hydrofluoric acid used in glass etching and cleaning also tends to rapidly penetrate the eye, whilst sulphuric acid may be complicated by thermal effects and high velocity impact after car battery explosions.

Management

a) Emergency treatment: should started as soon as possible (before history & exam)

1 Copious irrigation is crucial to minimize duration of contact with the chemical and normalize the pH in the conjunctival sac as soon as possible, and the speed and efficacy of irrigation is the most important prognostic factor following chemical injury. A sterile balanced buffered solution, such as normal saline or Ringer lactate should be used to irrigate the eye for 15–30 minutes or until pH is neutral (tap water should be used if necessary to avoid any delay). A topical anaesthetic should be instilled prior to irrigation, as this dramatically improves comfort and facilitates cooperation. A lid speculum may be helpful.

2 Double-eversion of the upper eyelid should be performed so that any retained particulate matter trapped in the fornices is identified and removed.

3 Debridement of necrotic areas of corneal epithelium should be performed to promote re-epithelialization and remove associated chemical residue.

4 Admission to hospital will usually be required for severe injuries (grade 4 ± 3) in order to ensure adequate eye drop instillation in the early stages.

b) Detailed history, examination & grading of severity:

Grading is performed on the basis of corneal clarity and severity of limbal ischaemia (Roper-Hall system); the latter is assessed by observing the patency of the deep and superficial vessels at the limbus

Grade 1 is characterized by clear cornea (epithelial damage only) and no limbal ischaemia (**excellent prognosis**).
Grade 2 shows hazy cornea but with visible iris details and less than one-third of the limbus being ischaemic **(good prognosis).**
Grade 3 manifests total loss of corneal epithelium, stromal haze obscuring iris details and between one-third and half limbal ischaemia (**guarded prognosis**)**.**
Grade 4 shows opaque cornea and more than half limbal ischaemia (**very poor prognosis**).

c) Medical treatment:

Most mild (grade 1 and 2) injuries are treated with topical antibiotic ointment for about a week, with topical steroids and cycloplegics if necessary. The main aims of treatment of more severe burns are to **reduce inflammation, promote epithelial regeneration and prevent corneal melting**. For moderate-severe injuries, preservative-free drops should be used.

1. **Steroids & NSAIDs.** steroids reduce inflammation and neutrophil infiltration, and address anterior uveitis. However, they also impair stromal healing by reducing collagen synthesis and inhibiting fibroblast migration. For this reason topical steroids may be used initially but must be tailed off after 7–10 days when sterile corneal ulceration is most likely to occur. Steroids may be replaced by topical NSAIDs, which do not affect keratocyte function.
2. **Cycloplegia** to decrease pain.
3. **Topical antibiotic** drops are used for prophylaxis of bacterial infection (e.g. chloramphenicol q.i.d.).
4. **Ascorbic acid** (for healing &scar maturation) reverses a localized tissue scorbutic state and improves wound healing, promoting the synthesis of mature collagen by corneal fibroblasts. Topical sodium ascorbate 10% is given 2-hourly in addition to a systemic dose of 1–2 g vitamin C (L-ascorbic acid) q.i.d. (not in patients with renal disease).
5. **Citric acid** (Anti-inflammatory Anti collagenase) is a powerful inhibitor of neutrophil activity and reduces the intensity of the inflammatory response. Chelation of extracellular calcium by citrate also appears to inhibit collagenase. Topical sodium citrate 10% is given 2-hourly for about 10 days, and may also

be given orally (2 g four times daily). The aim is to eliminate the second wave of phagocytes, which normally occurs about 7 days after the injury. Ascorbate and citrate can be tapered as the epithelium heals.

6 **Tetracyclines** (Anti collagenase Anti-inflammatory) are effective collagenase inhibitors and also inhibit neutrophil activity and reduce ulceration. They should be considered if there is significant corneal melting and can be administered both topically (tetracycline ointment q.i.d.) and systemically (doxycycline 100 mg b.d. tapering to once daily). Acetylcysteine 10% drops 6 times daily are an alternative topical anticollagenase agent.

d) Surgery:

1 Early surgery may be necessary to promote revascularization of the limbus, restore the limbal cell population and re-establish the fornices. One or more of the following procedures may be used:

- Advancement of Tenon's capsule and suturing to the limbus is aimed at re-establishing limbal vascularity thus preventing the development of corneal ulceration.
- Limbal stem cell transplantation from the patient's other eye (autograft) or from a donor (allograft) is aimed at restoring normal corneal epithelium.
- Amniotic membrane grafting to promote epithelialization and suppression of fibrosis.
- Gluing or keratoplasty may be needed for actual or impending perforation.

2 **Late surgery** may involve the following procedures:

- Division of conjunctival bands and treating symblepharon
- Conjunctival or mucous membrane grafts.
- Correction of eyelid deformities .
- Keratoplasty should be delayed for at least 6 months and preferably longer to allow maximal resolution of inflammation.
- Keratoprosthesis may be required in very severely damaged eyes because the results of conventional grafting are poor.

References:

Clinical Ophthalmology A Systematic Approach ; Jack J Kanski & Brad Bowling.

Learning objectives:

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