# Posterior segment trauma

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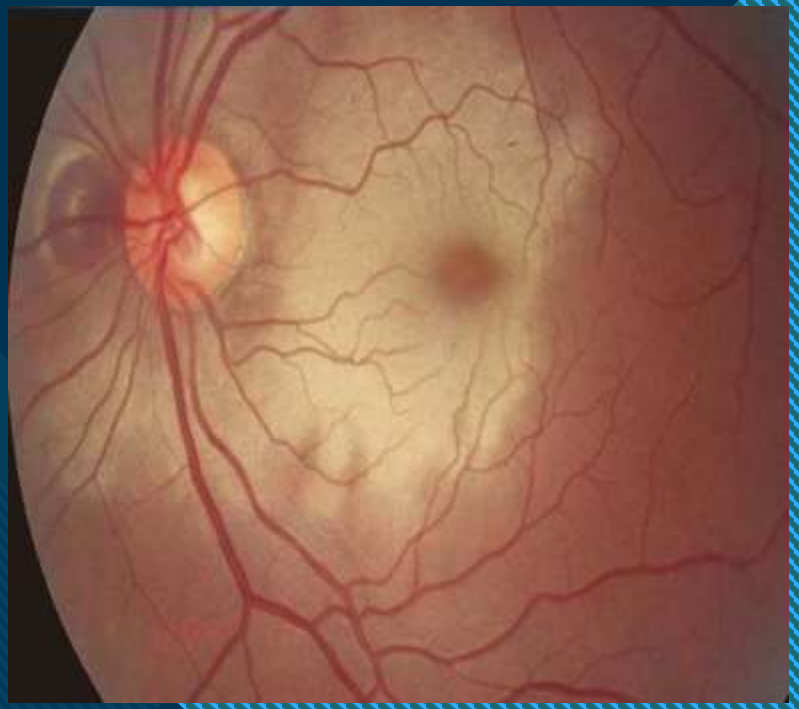
# Commotio Retinae/Berlins Edema



It is a transient opacification of the retina post-blunt trauma. It can be located at the posterior pole or in retinal periphery. When it involves the macula it is known as Berlin's edema

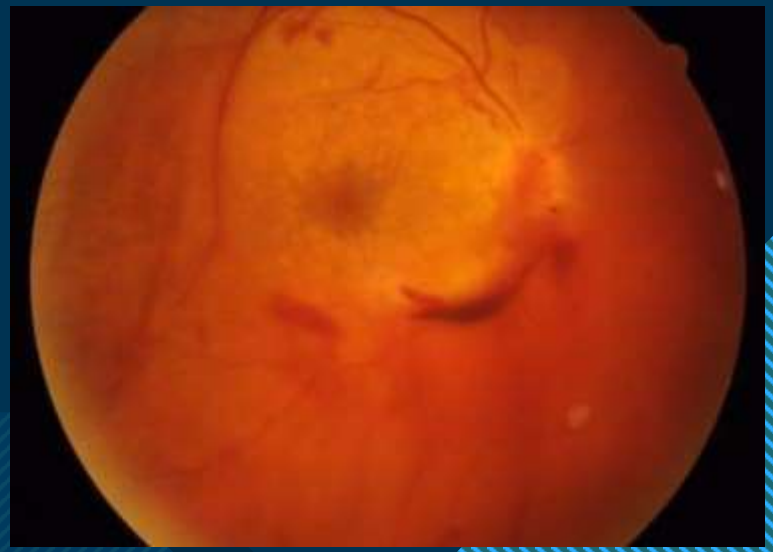
Various theories have been proposed for its pathogenesis. Though originally postulated to be due to extracellular edema, there are recent reports on the histopathological studies which show disruption or fragmentation of the photoreceptor outer segment of the retina as the most common finding in patients with commotio retinae.

The OCT :increased retinal thickness with disruption of the IS/OS junction and corresponding hyperreflectivity and damage to the external limiting membrane. Those with IS/ OS junction defects are more likely to have irreversible photoreceptor damage

There is no specific treatment for this condition. Oral steroids may be given to reduce the edema.

The visual prognosis is usually good except in cases of choroidal ruptures, retinal detachments or atrophy and hypertrophy of RPE.

# Vitreous Hemorrhage



It usually occurs due to traumatic disruption of the blood vessels in anterior segment or posterior segment due to external mechanical forces.

The visual acuity can vary from 20/200 or better to hand movements or even light perception.

Those presenting with low visual acuity and cataract or aphakia are considered as risk factors for poor prognosis

Ultrasound (USG) is essential to rule out associated retinal detachments (RD), intraocular foreign body (IOFB), and occult scleral ruptures along with the position of the dislocated lens in which cases, urgent surgical intervention is required. Posterior vitreous detachment (PVD) and associated choroidal detachment can also be noted in USG.

There is no consensus regarding the timing of intervention. One can wait for up to 2–3 months for spontaneous resolution of the hemorrhage (head elevation and avoidance of strenuous activities are advised in this period).

The prognosis is usually good in simple cases of VH. However, in those associated with RD, IOFB, and globe ruptures, the visual prognosis may be affected depending on the extent of the effect.

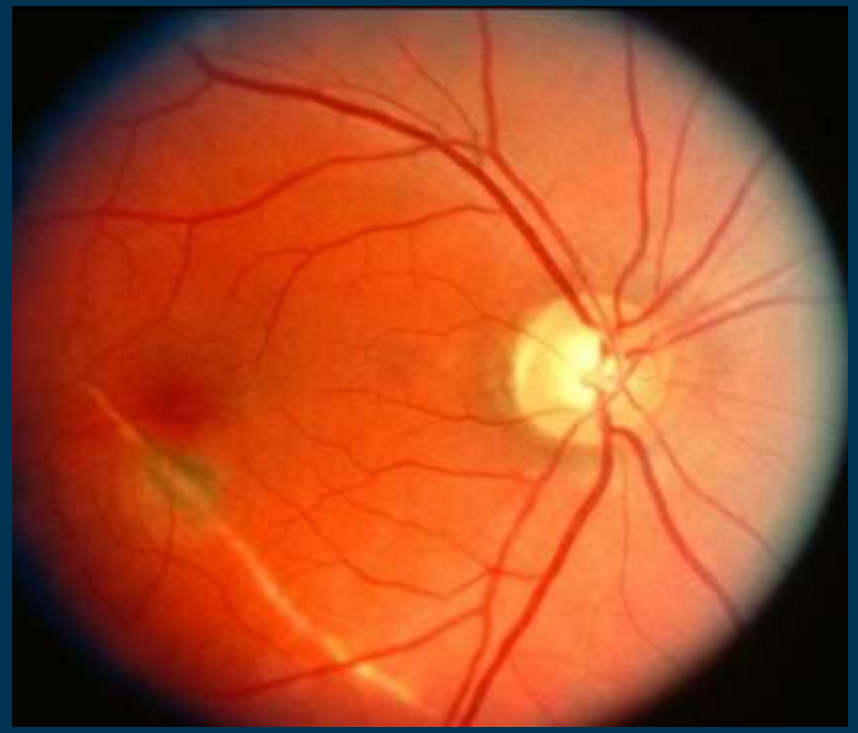
# Posterior Dislocation of Lens

Anteroposterior force generated due to blunt trauma leading to equatorial expansion can cause disruption of zonular fibers and hence dislocation of lens/IOL. It may be associated with VH or RD, secondary glaucoma and traumatic vitreoretinopathy Ultrasound can aid in diagnosis. Surgery involves complete vitrectomy to free the nucleus/IOL of surrounding vitreous followed by its removal.

# Choroidal Rupture

It is a break in the choroid, Bruch's membrane, and RPE due to an expansile force created rapid compression of the globe following blunt trauma. Sclera being inelastic resists the expansion but retina stretches during the injury.  Bruch's membrane breaks due to lack of elasticity . It occurs in 5–8% of all closed globe injuries.

Choroidal ruptures are of two types—direct and indirect. Those following direct trauma, i.e., directly at the site of trauma tend to be anterior and parallel to the ora serrata. The indirect ruptures are caused due to countercoup mechanism and usually involve the posterior pole most commonly located temporal to the optic nerve and affect macula. These account for nearly 60% of the cases



The symptoms depend on the area affected. Macula involving cases usually present with decreased vision, scotomas, enlargement of the blind spot, or metamorphopsia.

It appears as a crescent-shaped curvilinear reddish-yellow line usually parallel to the optic nerve. At times, it may not be visible initially due to overlying hemorrhage. The rupture can be partial thickness or full thickness.

The late complication frequently include CNV (5–10%). Those closer to fovea are more prone to CNV. The visual prognosis for central ruptures is usually poor, especially if associated with retinal or subretinal hemorrhages, vitreous hemorrhage, or CNV.

- Diagnosis is usually based on the history of blunt ocular trauma along with clinical findings.
- FFA shows early hypofluorescence due to rupture of choroidal vessels at the site followed by late staining due to leakage from adjacent capillaries. Hyperflourescence increasing in size and intensity can be seen in cases with CNVM.
- OCT shows discontinuity of the RPE along with thinning of the underlying choroid.
- Treatment usually involves observation along with Amsler's monitoring. Anti-VEGFs are used in cases complicated with CNVM.

# Optic Nerve Avulsion

- Avulsion of optic nerve is one of the severe complications of ocular trauma. It is characterized by disinsertion of optic nerve from retina, choroid, and vitreous at the level of lamina cribrosa without rupture of nerve sheath . Common sites include intraorbital and intracanalicular areas. The lamina cribrosa is more prone to injury due to absence of myelin and other supportive connective tissues . Although rare, it should be kept in suspicion when the vision loss is severe and dense . Although rare, it should be kept in suspicion when the vision loss is severe and dense

- The vascular changes are commonly associated due to close relation of optic nerve with retinal vasculature . The visual prognosis depends on the initial vision after trauma. The visual loss is due to damage and break of the nerve fibers. Partial avulsions usually carry better prognosis.
- The diagnosis can be made clinically by seeing a cavity at the site of optic disc but the view is frequently obliterated due to associated vitreous or retinal hemorrhages. USG and CT can help in diagnosing such cases.

- USG shows hypolucent area in the region of optic nerve head
- CT scan shows vitreous prolapse into optic nerve sheath in a mushroom pattern.
- VEP can also point toward optic nerve involvement.

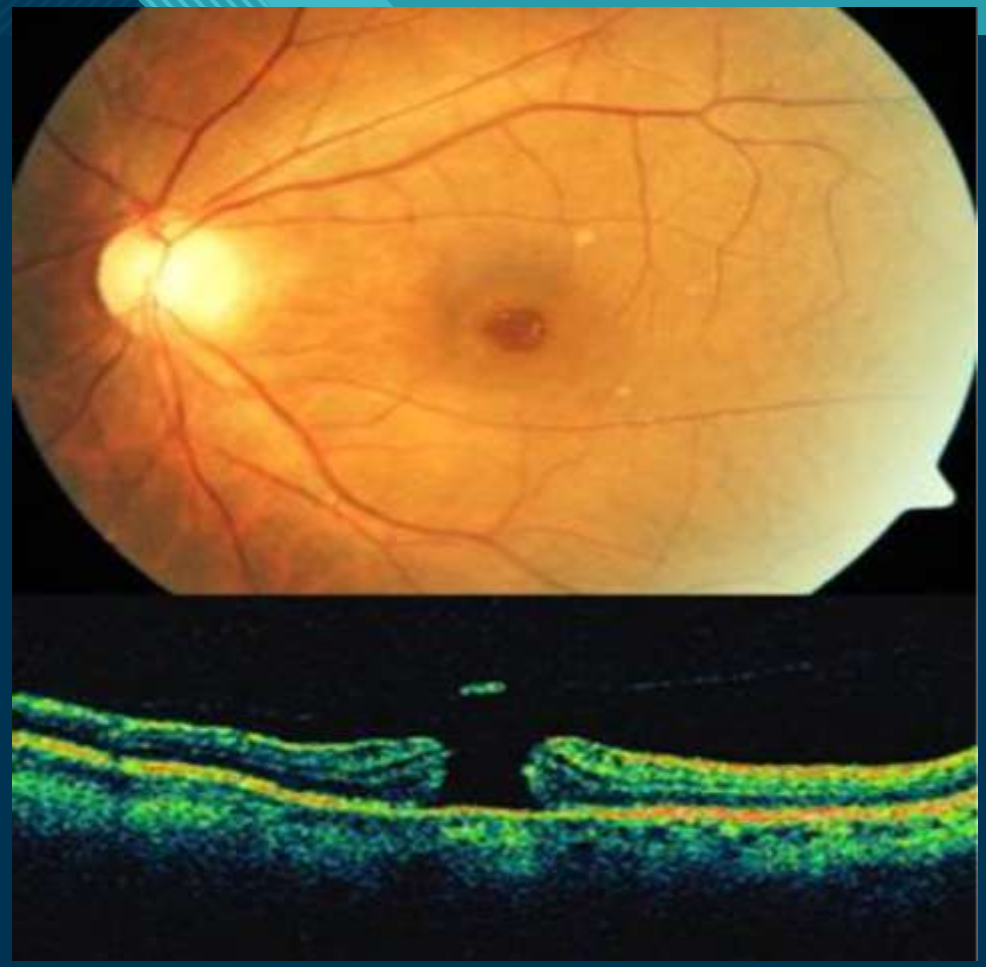
- Vitrectomy can be done in cases with dense vitreous hemorrhage but the outcome is limited due to optic nerve involvement.

# Traumatic Macular Holes

- The incidence of traumatic macular hole is close to 1.4% among closed globe injury . It is more common in the younger population and can lead to visual loss, especially if associated with other retinal pathologies like commotio retinae, retinal or vitreous hemorrhage, choroidal ruptures, retinal tears, or dialysis. Traumatic macular holes tend to have a larger base diameter and thinner average retinal thickness than idiopathic ones and are usually not associated with PVD.

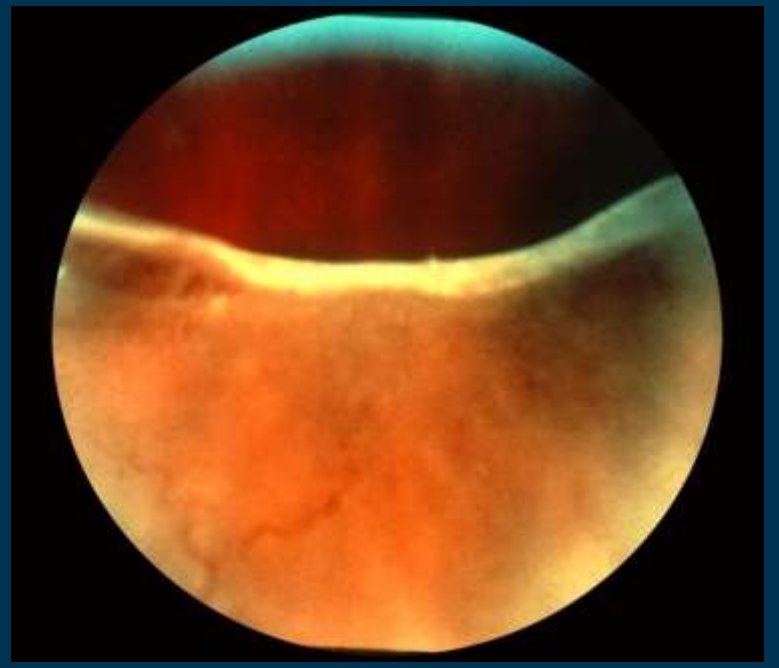
- The exact causative mechanism is not known. Yokotsuka et al. postulated sudden vitreous separation as the primary cause  while Johnson et al. proposed the countercoup mechanism in 2001.

- Several case reports show high rate of spontaneous closure of traumatic macular holes usuall between 2 weeks and 12 months .
- The visual prognosis is usually good after spontaneous closure. The mechanism of closure is proliferation of glial cells or RPE from the hole’s edge and stimulation of astrocytes migration to heal the hole . Observation is advised for 3–6 months due to high rate of spontaneous closure. However, early vitrectomy can be planned in children to avoid amblyopia.



# Retinal Detachment

- Traumatic RD usually occurs due to abrupt globe deformity following the impact of OBT. The force of the injury is an important predictor of the extent of vitreoretinal damage. Those with high myopia, aphakia, and fellow eye history of RD are more prone to such consequences.
- Retinal dialysis is the most common retinal finding in cases of blunt trauma and the most common being that by a fist. It is described as disinsertion of retina from the ora serrata. The most common site is inferotemporal followed by superonasal.
- Apart from retina dialysis, retinal tears are also noted, which can eventually lead to rhegmatogenous RD. Giant retinal tears (GRT) can also be present which can be either circumferential or radial.

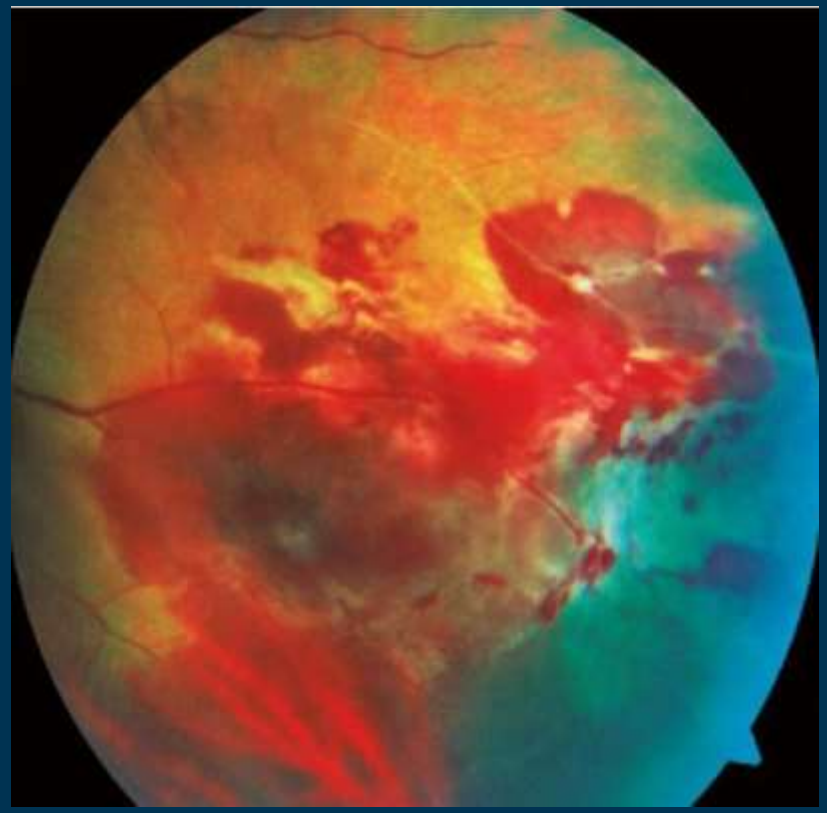


- Many patients are asymptomatic initially and present only after vision loss due to retinal detachments. Others may present with flashes, floaters, loss of peripheral visual field, or dimness of vision. The absence of symptoms is related to the fact that many retinal dialyses occur in the absence of a PVD with a slow progression of subretinal fluid, and there is characteristic involvement of the superior visual field. Thus, a thorough retinal peripheral examination with scleral depression is essential to identify dialysis before it progresses to retinal detachment. The dialysis typically opens up on scleral depression. Repeated examinations may be required in cases of VH or preretinal blood, which can obscure the view.

- There may be other associated findings like avulsion of the vitreous base, VH, traumatic cataract, retinal demarcation lines, and pars plana detachments. According to a study, demarcation lines were more common in inferotemporal dialyses suggesting some chronicity, whereas vitreous base avulsion and pars plana detachments were more common with superonasal dialyses suggesting more acute and severe trauma
- The diagnosis is usually by thorough clinical examination and can be aided by USG and wide field imaging. The dialysis and tears with no or minimal subretinal fluid can be safely treated with photocoagulation or cryopexy. The treatment should be initiated as soon as possible. In cases with detachments, an urgent surgical intervention is warranted.

# Chorioretinitis Sclopetaria

- Sclopetaria is a secondary outcome of a decelerating object passing at a high velocity adjacent to the sclera.This results in concussion injury to the retina and choroid. The mechanism is thought to be the energy released by the projectile while passing close to the eyeball . There is typically no penetration of the particle into the globe.
- The area affected due to direct injury is usually the area adjacent to the path of the projectile. While indirect effect can be caused in other areas of choroid and retina due to propagation of the shock waves. Multiple areas of affection can be present.

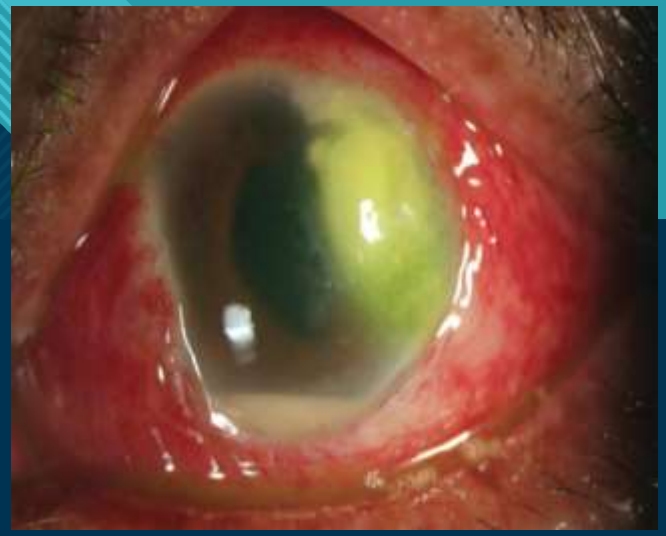


- The visual acuity at presentation depends on the area involved. The condition is frequently associated with ocular motility disorders, IOP changes due to intraocular inflammation, retinal, and macular edema. It is usually associated with poor visual prognosis. The retina is rarely detached in such conditions due to inflammation in the necrotic edges of the rupture, which causes chorioretinal scar formation. Hence, prophylactic cryopexy or photocoagulation is usually not required. The late complication of this condition involves formation of CNV.

# Subretinal Hemorrhages

- Visual loss is profound and immediate in cases with submacular hemorrhages (SMH) . Permanent damages to neurosensory retina and retinal pigment epithelium are likely to occur due to iron toxicity to photoreceptors from hemoglobin breakdown.
- Better visual outcomes are expected in cases with good initial visual acuity, duration <2 weeks and hemorrhage not larger than three disc diameters.

# Posttraumatic Endophthalmitis



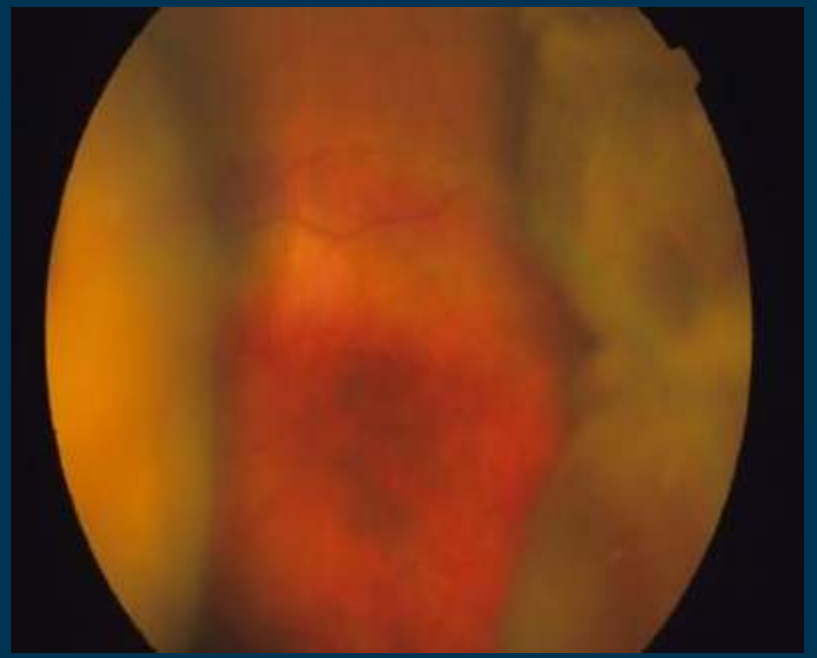
- Posttraumatic infectious endophthalmitis occurs in 2–7% of all penetrating intraocular injuries and 7–31% of injuries with retained intraocular foreign bodies.
- Due to initial injury, delay in primary repair and more virulent organisms (Bacillus or/staphylococcus species), they have worse outcomes than other types of endophthalmitis.
- Presentation >24 h after injury, IOFBs composed of steel, and organic or soil-contaminated IOFBs, large wounds, vitreous prolapse through the wound are all risk factors for endophthalmitis.

- The clinical signs include eyelid edema, congestion, purulent discharge, corneal edema, anterior chamber reaction, hypopyon, vitritis. The patient may present with out of proportion pain. The progression and severity depend on the virulence of the organism.
- Pars plana core vitrectomy with preoperative and postoperative antibiotics on an urgent basis is needed. Empirical intravitreal vancomycin (1 mg/0.1 mL) and ceftazidime (2.25 g/0.1 mL) are used. Intravitreal amphotericin B (0.005 mg/0.1 mL) is reserved for infections with a strong suspicion of fungal infections. The use of adjunctive intravitreal steroids is controversial.

- The purpose of vitrectomy is to reduce the load of inflammation and infection, obtaining a sample for culture studies, irrigating toxins, and allowing better distribution of antibiotics.
- The most common species involved are Bacillus, Staphylococcus epidermidis, and Streptococcus species. Bacillus, in particular, tends to be aggressive and frequently cause rapid destruction of ocular tissues .
- The prognosis is usually poor and depends on the virulence of the causative organism, timing and extent of the surgery, and presence of retinal detachment.

# Suprachoroidal Hemorrhage

- An expulsive choroidal hemorrhage is a dreadful complication rarely associated with traumatic globe rupture. Predisposing factors are age, systemic hypertension, diabetes, glaucoma, myopia, and recent intraocular surgery.
- The clinical signs include low IOP, forward bulge of the iris and pupillary dilatation.
- The diagnosis can be made on USG that shows a round mound with low-to-medium point echoes. When choroidal mounds touch each other they are called kissing choroidals.



# Sympathetic Ophthalmia

- The incidence of SO ranges from 0.2 to 0.5% after penetrating ocular injuries. It is a bilateral diffuse granulomatous inflammation that usually occurs within days or months of penetrating ocular injuries (about 80% occurring in first 3 months and 90% within first year)
- The injured eye is called the exciting eye and the other eye developing inflammation later is called the sympathizing eye. There is no age or racial predisposition but it occurs more commonly in males following ocular trauma. The possible mechanism is thought to be of autoimmune origin.

- The patient presents with pain, photophobia, and blurring of the vision in the sympathizing eye. The clinical signs include bilateral diffuse panuveitis showing mutton fat KPs on corneal endothelium and/or posterior synechae. The IOP may be raised secondary to inflammation. The patient may have associated vitritis, retinal vasculitis, papillitis, and choroiditis (seen as whitish lesions called “Dalen Fuch’s nodules” in peripheral fundus).
- The predisposing factors include penetrating trauma, trauma near ciliary body, interval between repair more than 48 h, and larger wound.

- The diagnosis is usually based on history taking and clinical examination. However, OCT, FFA, and ICGA can help in the diagnosis.
- FFA shows multiple hyperfluorescent spots seen in venous phase till late phases. In severe cases, there may be pooling of exudates suggestive of exudative retinal detachment.
- ICGA shows multiple hypofluorescent spots that become more distinct in late phases. B scan shows choroidal thickening and exudative detachment of the retina.

- Systemic corticosteroids are the mainstay of treatment. Additionally, topical steroids and cycloplegics are used to control the anterior chamber reaction and prevent posterior synechae formation. The steroids are followed by systemic immunomodulatory agents like cyclosporine, azathioprine, or cyclophosphamide.
- Secondary enucleation of the exciting eye to reduce inflammation in the sympathizing eye does not necessarily lead to a better visual outcome or to a reduced need for medical therapy.

# PURTSCHER RETINOPATHY

- Acute compression injuries to head and thorax initiates endothelial damage and complement mediated leuko embolization, which causes granulocyte aggregation.
- Fundus : Large cotton wool spots, hemorrhages , retinal edema mainly surrounding the optic disc, polygonal shaped areas of retinal whitening (Purtscher flecken).

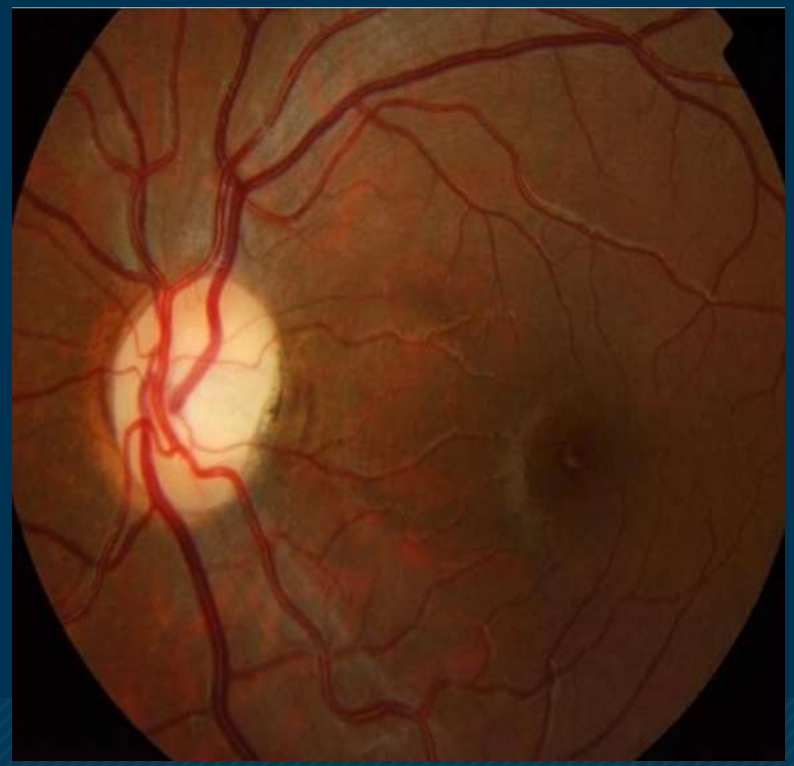
# Terson Syndrome

- Terson syndrome is recognized as a vitreous and sub-ILM or subhyaloid hemorrhage caused by an abrupt intracranial hemorrhage.
- Approximately one-third of patients with subarachnoid or subdural hemorrhage have associated intraocular hemorrhage, which may include intraretinal and subretinal bleeding.
- Terson syndrome occurs primarily in individuals between 30 and 50 years old, but it canoccur at any age.
- In most cases, visual function is unaffected once the hemorrhage clears. Spontaneous improvement generally occurs, although vitrectomy is occasionally required to clear the ocular media.

# TRAUMATIC OPTIC NEUROPATHY:

- Direct traumatic optic neuropathy (TON): may be caused by injury to the nerve itself or by laceration with bone fragments or other foreign bodies. Injuries may also cause compressive optic neuropathy secondary to intraorbital or intrasheath hemorrhage.
- Indirect TON :may occur even with relatively minor head injury. The trauma involves the frontal or maxillary bone, and the transmitted forces damage the optic nerve at the orbital apex. Avulsion of the nerve may also occur. The pathophysiology presumably involves shear forces on the nerve and possibly its vascular supply in the optic canal.

- Vision loss is typically immediate and often severe (24%–86% of patients have no light perception at presentation). External evidence of injury may be scarce. An RAPD is invariably present if there is unilateral vision loss. Although the ONH usually appears normal at onset, it becomes atrophic within 4–8 weeks.



# Thanks for your attention