28 Posterior Segment Complications of Ocular Trauma

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28.1 Open Globe Injuries

Open globe injuries (OGIs) are a major cause of vision-threatening ophthalmic emergency, especially in working age population. The mean estimated incidence of OGIs is ~3.5 cases in 100,000 annually, leading to 200,000 OGIs per year worldwide [1]. They are further classified as globe rupture, or laceration which involves penetrating and perforating injuries and intraocular foreign body.

28.1.1 Globe Rupture

A globe rupture occurs when there is full-thickness defect of cornea, sclera, or both the structures by blunt or penetrating trauma (Fig. 28.1). Globe rupture usually results from high-velocity blunt objects striking the eye which eventually may raise IOP leading to tissue tearing and decompression via a rupture of the eye wall [2]. The rupture site is most commonly near the globe's equator, posterior to the insertion of the rectus muscles as the sclera here is weakest and thinnest. In eyes with a previous surgical incision, the rupture usually occurs at the previous incision. It is necessary to keep a high index of suspicion for occult globe rupture in cases of OBT. Signs of high suspicion include low IOP of 5 or less at presentation, visual acuity of light perception or less, abnormally shallow or deep anterior chamber, dislocation of lens, presence of RAPD, dense VH preventing the view of the fundus or an RD [3]. The visual prognosis is usually

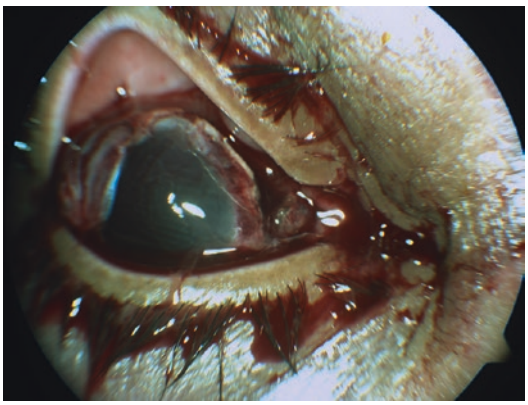

Fig. 28.1 Slit lamp photograph showing conjunctival chemosis and globe rupture post-blunt ocular trauma. Multiple scleral ruptures were discovered intraoperatively on globe exploration

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poor. Poor visual acuity at presentation is a predictor of poor visual outcome. An urgent intervention in form of globe exploration and closure of the wound is required. The rupture can be associated with conditions like hyphema, dislocation of lens, traumatic cataract, vitreous hemorrhage, or retinal detachments. The primary closure of the wound can be combined with lensectomy and vitrectomy as required or a secondary surgery can be planned at a later date. Pre- and postoperative antibiotic covers are essential to prevent endophthalmitis.

28.1.2 Penetrating and Perforating Injuries

By definition, penetrating injuries are those with one entry wound, i.e., the causative object does not pass through and through, while perforating injuries have both entry and exit wounds. The incidence is higher in males [4]. The causative agent is usually a high velocity or sharp object. The most important primary prevention includes wearing adequate and appropriate protective eyewear while doing vision-threatening activities.

Prompt evaluation and management are warranted. A thorough history regarding the type of object and mode and timing of injury and whether any protective eyewear was being worn at the time of injury should be recorded. In case the view of posterior segment is jeopardized due to media opacity like hyphema, cataract, or VH, a gentle USG and CT scan should be done to rule out retained intraocular foreign bodies (IOFB). MRI is contraindicated where a metallic foreign body is suspected.

The factors accounting for poor visual outcomes are poor visual acuity at time of presentation, presence of RAPD, time involved between injury and repair, severity of injury, size and location of wound, presence of VH, RD, and IOFB [5].

A prompt globe exploration and closure with or without vitrectomy should be done [6]. A single-step approach of wound closure and comprehensive reconstruction can be undertaken. Such an approach is specifically indicated in cases with IOFB and endophthalmitis. A two-step approach consists of watertight primary closure followed by vitrectomy and globe reconstruction at a second stage usually after 10–14 days. Strict asepsis has to be followed. General anesthesia is preferred. The goals of vitrectomy are to remove the media opacities as far as possible, remove the focus of infection or IOFB if any, remove the damaged vitreous and hence reduce the scaffold for further fibrocellular proliferation leading to PVR. The timing of vitrectomy varies from surgeon to surgeon. Early intervention in cases of traumatic detachments reduces the incidence of endophthalmitis and proliferative vitreoretinopathy (PVR). However, a later intervention may reduce chances of intraoperative hemorrhage and postoperative inflammation. Pre- and postoperative antibiotic coverage is a must. The infusion port during the vitrectomy is placed in the healthy sclera and should be free from incarceration. The flow in vitreous cavity should be free flow to avoid secondary retinal or choroidal detachments. An adequate visualization of posterior segment is essential. In cases of severe corneal trauma, a temporary keratoprosthesis or endoscope can be used. Vitrectomy is done with high-speed cutters and with low-to-moderate suction. The lens should be removed in case it is cataractous or required for anterior vitreous dissection. It is necessary to do a complete vitrectomy to prevent further fibrovascular proliferations. Special attention should be paid for any retinal incarceration. Such cases require complete relief of traction at incarcerated site. A relaxing retinectomy (RR) can also be done which is extended up to normal retina to ensure complete relaxation of the retina. The anterior incarcerated retina is then completely excised.

Severely injured eye might require enucleation, especially to prevent sympathetic ophthalmia (SO). However, prompt and tight surgical wound closure and adequate management of postoperative inflammation have decreased the incidence of SO and hence this argument is no

more justified [7]. Despite repeated surgeries, the risk of SO has not risen above 0.1–0.3% during the last four decades [8]. It was only 20 years ago that Morris et al. reported on their attempts to reconstruct 11 eyes with severe ocular trauma that could not perceive light. The authors succeeded in restoring some function in 7 of 11 cases [9].

Although there may be cases that are not salvageable, an attempt to salvage the eye by primary repair rather than primary enucleation must be done. Primary enucleation should never be the first approach.

28.2 Closed Globe Injuries

28.2.1 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 (Fig. 28.2). It was first described by Berlin in 1873 [10].

Various theories have been proposed for its pathogenesis. Though originally postulated to be due to extracellular edema [10], 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 [11].

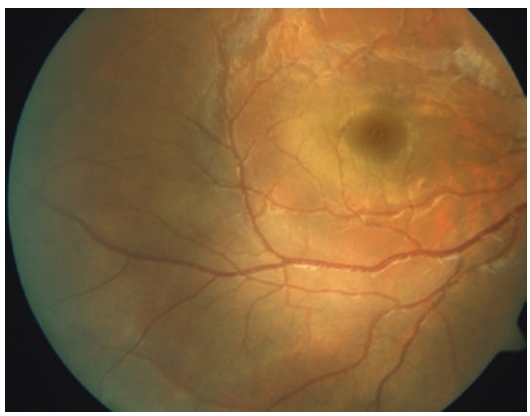

Fig. 28.2 Fundus image showing edema at the macular region (Berlin's edema) seen post blunt trauma to the eye

The OCT shows increased retinal thickness with disruption of the IS/OS junction and corresponding hyperreflectivity [11] and damage to the external limiting membrane. Those with IS/OS junction defects are more likely to have irreversible photoreceptor damage [12]. 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.

28.2.2 Vitreous Hemorrhage

It usually occurs due to traumatic disruption of the blood vessels in anterior segment or posterior segment due to external mechanical forces [13]. Among all traumatic causes, OBT accounts for the majority of traumatic VH being responsible for 29.6–64% of such cases with a male predominance [13, 14].

The visual acuity can vary from 20/200 or better to hand movements or even light perception [14]. Those presenting with low visual acuity and cataract or aphakia are considered as risk factors for poor prognosis [14]. 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). Small gauge transconjunctival pars planavitrectomy (PPV) can be planned in cases of nonresolving vitreous hemorrhage. Initially the vitreous from central region is removed anteriorly and then extending the excision up to the posterior hyaloid. A complete vitrectomy with PVD induction should be done. Long-standing VH can be associated with

spontaneous PVD. A fresh hemorrhage appears reddish or yellowish while old blood is brownish. Active bleeding points if visualized can be managed by raising the IOP and fluid air exchange or with the help of endocautery. A thorough peripheral examination of the retina with the help of wide-angle viewing systems should be done intraoperatively to rule out any retinal breaks or dialysis in which case endolaser/cryo can be done simultaneously. Cryotherapy is preferred in case of anterior breaks and when residual vitreous obscures its view. It is necessary to clear the vitreous around the retinal breaks thoroughly in order to avoid undue traction around them later, which can lead to retinal detachments. Removal of subretinal blood also warrants elimination of the vitreous surrounding it. The blood can then be aspirated from the existing tear or a retinotomy with an extrusion cannula. The retinotomy is later treated with laser. 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.

28.2.3 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 [15]. 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. Details of the surgery have been covered in Chap. 37.

28.2.4 Choroidal Rupture

It was first described by Von Graefe in 1854 [16]. It is a break in the choroid, Bruch's membrane, and RPE due to an expansile force created by 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 [17]. It occurs in 5–8% of all closed globe injuries [18].

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 [19].

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%) [17]. 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 [17].

28.2.5 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 [20]. Common sites include intraorbital and intracanalicular areas [21]. The lamina cribrosa is more prone to

injury due to absence of myelin and other supportive connective tissues [22]. Although rare, it should be kept in suspicion when the vision loss is severe and dense [22].

The vascular changes are commonly associated due to close relation of optic nerve with retinal vasculature [21]. The visual prognosis depends on the initial vision after trauma. The visual loss is due to damage and break of the nerve fibers [22]. 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 [23] and 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.

28.2.6 Traumatic Macular Holes

The incidence of traumatic macular hole is close to 1.4% among closed globe injury [24]. It is more common in the younger population [24] 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 [25].

The exact causative mechanism is not known. Yokotsuka et al. postulated sudden vitreous separation as the primary cause [26] while Johnson et al. proposed the countercoup mechanism in 2001 [27].

Several case reports show high rate of spontaneous closure of traumatic macular holes usually between 2 weeks and 12 months [28]. 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 [29]. 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.

Vitrectomy and ILM peeling with intraocular tamponade is the treatment of choice for non-closing and large macular holes. Details of the procedure have been covered in Chap. 22. The final visual outcome depends on the size and duration of the macular hole.

28.2.7 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 [7]. Retinal dialysis is the most common retinal finding in cases of blunt trauma and the most common being that by a fist. In a series of 5360 eyes with RD, dialysis was the most frequent type of tear in detachments related to trauma [30]. It is described as disinsertion of retina from the ora serrata. The most common site is inferotemporal followed by superonasal. Apart from retinal 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 [30]. 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 [31]. 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 [30].

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 [32] (Fig. 28.3). The treatment should be initiated as soon as possible.

In cases with detachments, an urgent surgical intervention is warranted. Scleral buckling is usually preferred in such cases because PVD induction, which is difficult in young subjects, can be avoided by this procedure. Also, chances of iatrogenic breaks, secondary cataract formation, and complications related to intraocular tamponade of gas/oil can be avoided [33]. Circumferential silicon buckles are usually used based on the location and size of the break. Subretinal fluid may or may not be drained. Absorption of SRF in cases of chronic detachments tends to be slower.

The indications for primary vitrectomy in traumatic RD include VH, dislocated lens, PVR, giant retinal tear with everted flap, posterior tear, and subretinal hemorrhage. It is necessary to rule out choroidal hemorrhage prior to the surgery with USG. PPV helps in removal of media opacities like VH and allows a better view of the retina. The timing of vitrectomy is still controversial. Early vitrectomy poses a higher risk of intraoperative complications, bleeding, wound leakage, and bad visualization. Conversely, a delayed vitrectomy have a higher incidence and severity of postoperative complications, mainly PVR [34].

The prognosis is usually not very good due to longer intervals in treatment, frequent occurrence of PVR, and frequent macular involvement. Visual outcomes are better if the macula is attached at the time of repair. Worse presenting visual acuity, RAPD, presence of submacular hemorrhages, foveal choroidal rupture, macular hole, globe rupture, PVR, endophthalmitis, or IOFBs are all associated with poor visual outcome.

28.2.8 Traumatic PVR

PVR may develop in up to 70% of cases in open globe injuries especially in case of subretinal or vitreous hemorrhage, GRT, or large wound. It develops due to chronicity of the detachments along with fibrous tissue changes in the vitreous and usually occurs inferiorly first as cells gather in between the retina and oil bubble due to gravity. It eventually causes a pull on the retina and leads to retinal detachments (Fig. 28.4).

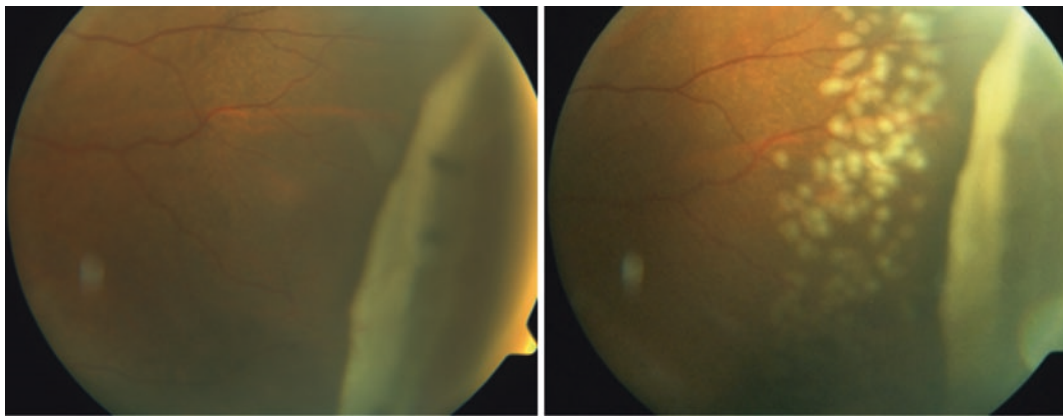

Fig. 28.3 Fundus image on the left shows retinal dialysis post blunt ocular trauma which was lasered as seen in the image on the right. The patient was sequentially observed and the retina remained stable post laser

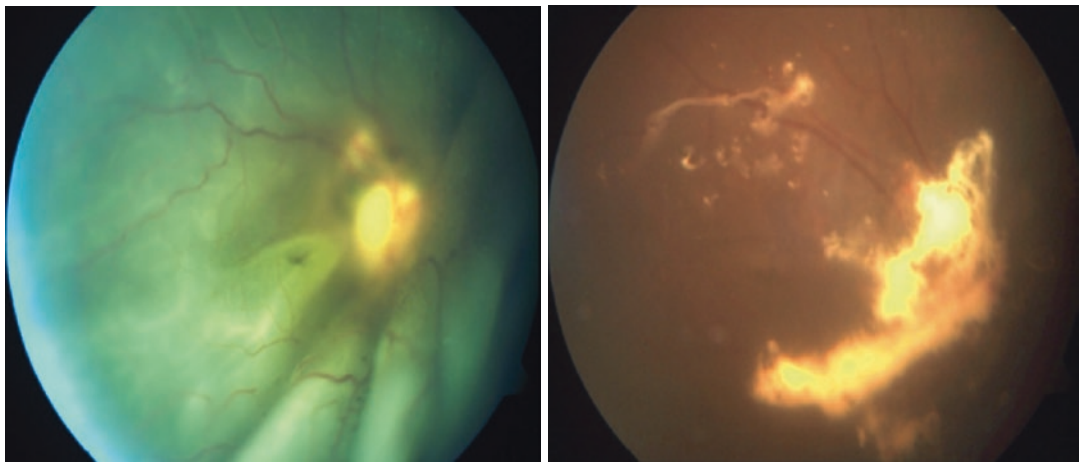

Fig. 28.4 Fundus image on the left shows traumatic retinal detachment with proliferative vitreoretinopathy in a patient with blunt ocular trauma. The right image is the postoperative fundus image with attached retina and silicone oil in situ

PFCL is an invaluable tool in these cases as it allows flattening of retina, facilitate intraocular manipulations, removal of epiretinal and fibroproliferative membranes, and provide adequate intraoperative retinal tamponade. It also helps in draining the SRF by anterior retinal breaks and stabilizes large breaks. Meticulous peeling of the epiretinal membranes and removal of any subretinal bands and gliosis with help of forceps and scissors is essential for successful reattachment of the retina. If needed, bimanual vitrectomy and membrane dissection should be done with the help of chandelier illumination.

In cases with persistent traction, relaxing retinotomy (RR) should be done. Almost 40% of the cases will need RR to reattach the retina [35]. Circumferential RR is preferred. Endodiathermy is applied to the edge of the retina to be incised prior to excision. Then, the retina is incised with help of microscissors or vitrectomy cutters up to the site of normal retina to relieve complete traction. The anterior retina is then excised. Injection of PFCL is then done to drain the remaining SRF and flatten the retina. Endophotocoagulation is done in 2–3 rows along the edge of RR. Slippage of retina in these cases can be avoided by direct PFCL–SOI exchange. Gases like SF6 or C3F8 and silicone oil are lighter tamponades with lower specific gravity and hence support the superior retina well but can pose a risk of inferior and posterior pole PVR due to accumulation of fibrous tissues [36]. Heavy tamponades in form of 5000 cst silicone oil or densiron are often required. They are especially preferred in inferior PVR. However, there are cases of recurrent superior detachments in a densiron filled eyes. Sandwich technique (use of densiron with silicone oil) is useful in such cases. Densiron is used in place of balanced salt solution in the infusion port using the dual-mode injection/extraction available in Alcon constellation vitrectomy system in complicated cases. This helps in draining of the persistent SRF while injecting densiron simultaneously. Densiron being heavier settles inferiorly under the silicone oil without mixing with it or losing its unique physical property. Hence, complete tamponade is obtained.

28.2.9 Chorioretinitis Sclopetaria

This relatively rare condition was first described in 1901 by Goldzieher [37]. Sclopetaria is a secondary outcome of a decelerating object passing at a high velocity adjacent to the sclera [38]. 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 [37]. 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 [37]. Hence, prophylactic cryopexy or photocoagulation is usually not required. The late complication of this condition involves formation of CNV.

28.2.10 Subretinal Hemorrhages

Visual loss is profound and immediate in cases with submacular hemorrhages (SMH) (Fig. 28.5). Permanent damages to neurosensory retina and retinal pigment epithelium are likely to occur due to iron toxicity to photoreceptors from hemoglobin breakdown. Early removal of SMH from the macular area is associated with better visual outcomes. Vitrectomy with modifications like a retinotomy, mechanical removal of subretinal clot, tissue-type plasminogen activator (tPA) use has been tried with variable final visual outcomes [39].

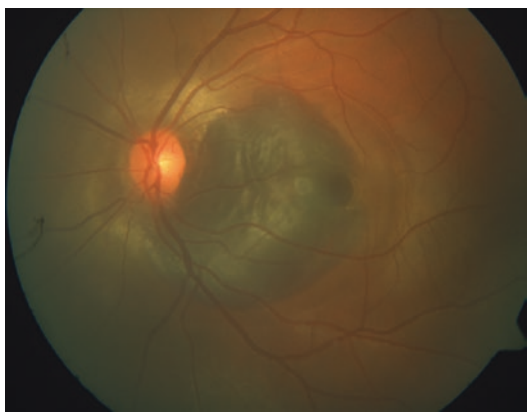

Fig. 28.5 Fundus image showing dense submacular bleed following trauma by a cricket ball

tPA along with pneumatic displacement (SF6 or C3F8) of the blood from fovea or pneumatic displacement alone is associated with better anatomical outcomes. Appropriate postoperative positioning also helps in faster displacement of blood. Better visual outcomes are expected in cases with good initial visual acuity, duration <2 weeks and hemorrhage not larger than three-disc diameters.

28.2.11 Posttraumatic Endophthalmitis

Posttraumatic infectious endophthalmitis occurs in 2–7% of all penetrating intraocular injuries and 7–31% of injuries with retained intraocular foreign bodies [40]. 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.

The indications for pars plana vitrectomy from the Endophthalmitis Vitrectomy Study were limited to postoperative infections and do not apply in cases of trauma and IOFBs associated with endophthalmitis [41]. Pars plana core vitrectomy along 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. In cases with corneal

opacity/abscess, vitrectomy can be done open sky or needs to be combined with temporary keratoprosthesis which can then be replaced with donor cornea at the end of the surgery. 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 peripheral vitreous removal and base excision is avoided in such cases due to friable nature of the retina. The topicals involve instillation of fortified antibiotics started on first postoperative day. Repeat intravitreal injections can be safely performed 48 h after the first injection. In cases of worsening inflammation or pain, repeat intravitreal injections or vitrectomy has to be considered.

Adequate intraocular samples (AC tap, vitreous tap, and vitrectomy cassette fluid) and IOFBs should be sent for microbiology investigations (Gram stain, aerobic, anaerobic, and fungal cultures). 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 [42]. 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.

28.2.12 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.

Symptoms are pain, headache, nausea, and vomiting. 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. USG also notes clot lysis and hence guides in timing and site of drainage during the surgery. The largest choroidal is the optimum site for drainage.

The ideal timing of vitrectomy is debatable. The surgery can be delayed for about 2 weeks. However, in cases of kissing choroidals and uncontrolled IOP one can intervene earlier. One should watch for liquefaction of clot lysis on USG, which is evident as reduced echogenicity. Surgical intervention is done in presence of anterior chamber infusion. The surgery includes choroidal drainage 4 mm posterior to the limbus in the quadrants having hemorrhage by either scleral cutdown or by an angled insertion of 23 or 25 gauge trochar cannula. Oozing out of dark chocolate brown fluid occurs suggestive of suprachoroidal hemorrhage. After choroidal drainage, a long infusion cannula is put and infusion is started only after seeing the tip. The goals of hemorrhagic choroidal drainage are to eliminate appositional or large, nonappositional choroidals, normalize IOP, and eliminate posterior pressure on the lens–iris diaphragm. Concurrent vitrectomy with PFCL and silicone oil tamponade is also required, especially in cases associated with vitreous hemorrhage or retinal detachments.

28.2.13 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) [43]. 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 [44].

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