Uveitis

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Abbreviations

CT Computed tomography
LIU Lens-induced uveitis

Traumatic Uveitis

Traumatic uveitis typically occurs following blunt trauma to the globe. Previously termed traumatic iritis or traumatic iridocyclitis, it can be a transient, self-limited event, or a prolonged inflammatory response resistant to typical anti-inflammatory therapy [1]. Traumatic uveitis is also frequently seen in association with other injuries following penetrating ocular trauma. This is a common presentation in patients following war injuries, terror attacks, and natural disasters. In these situations, the uveitis is often obscured by other traumatic injuries such as hyphema, iris trauma, or iris incarceration into a wound.

Isolated traumatic uveitis is less common in combat and civilian casualty situations compared to other ocular blunt injuries. In many cases, the object is larger than the globe and tends to be a lower velocity impact compared to penetrating wounds [2]. However, smaller, higher velocity objects such as BB and plastic pellets may produce blunt trauma and traumatic uveitis [3]. Hands, feet, other body parts, balls, paintballs, rackets, and various other blunt objects can strike the globe during assaults, home-related or sporting activities, or work-related activities [4–7].

Traumatic uveitis may also occur as a result of primary blast injury to the eye. Primary blast injury is the result of a strong overpressurization following the blast or shock wave that leads to deformation, disruption, and possible rupture of tissues. Less powerful overpressurization may cause disruption of internal ocular structures [8]. Secondary blast injuries (flying debris and/or fragments) produce most of the ocular injuries following explosions and bombings [8, 9]. Review of the literature does not reveal any reports of traumatic uveitis as an isolated injury following a primary blast injury. However, traumatic uveitis may be one of several closed-globe injuries that occur following explosive blasts and may be underreported or undetected due to presence of these other injuries such as hyphema [10–15].

Symptoms of isolated traumatic uveitis include blurry vision, photophobia, redness, and occasionally pain. Some patients may have an associated corneal abrasion. Traumatic corneal endothelial rings may be visible [16–18]. All patients exhibit anterior chamber cells and flare. Pigmented cells are often seen in the anterior

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chamber and a microhyphema may be present. In severe cases, fibrin and posterior synechiae may be present. In some patients, small tears of the iris along the pupil margin are visible, as well as other signs of iris trauma [4]. Intraocular pressure may be increased or decreased. Rarely, glaucoma may develop as a result of prolonged inflammation or therapy with corticosteroids.

Initial treatment of isolated traumatic uveitis is based upon the patient's level of discomfort and the severity of the inflammation. In most patients, a topical cycloplegic will reduce ciliary spasm and relieve pain and photophobia. In more severe cases, a cycloplegic agent is often useful for prevention of posterior synechiae formation. Topical corticosteroids are the mainstay of therapy in most patients with mild to severe forms of traumatic uveitis. In cases with severe inflammation with or without fibrin, hourly prednisolone acetate 1% is recommended. Periocular triamcinolone acetonide injections may be beneficial for those patients with severe inflammation unresponsive to topical corticosteroids. Topical corticosteroid dosage is reduced and tapered off beginning after a 50% reduction in the amount of cell and flare is noted. Patients with severe inflammation may require prolonged treatment for several months with topical corticosteroids before tapering, and discontinuation may occur. Topical glaucoma therapy may be required if the intraocular pressure is elevated. Topical beta-blockers, carbonic anhydrase inhibitors, and alpha-agonists are useful options for patients who are at risk for vision loss due to elevated intraocular pressure [19, 20].

In patients with traumatic uveitis and other anterior segment traumatic injuries, the diagnosis and management of the uveitis is more complex. Most of these cases occur in the setting of penetrating trauma with coexisting pathology such as corneal laceration, iris incarceration and/or prolapse, hyphema, iris trauma, lens capsule disruption, lens dislocation, and other anterior segment abnormalities. These injuries often limit evaluation of the anterior chamber and any underlying uveitis. Therefore, careful evaluation of the anterior chamber following repair of any corneal wounds or removal of lens material is essential to determine the severity of the uveitis and initial therapeutic approach. Similar to isolated traumatic uveitis, topical prednisolone acetate 1% is prescribed based upon the severity of the inflammation. A topical cyloplegic agent may also be considered based upon the clinical situation. In most cases of penetrating trauma requiring surgical repair, a slow taper of corticosteroids over several months may be necessary based upon the severity of uveitis during follow-up evaluations. Elevated intraocular pressure may require treatment with topical glaucoma medications, as discussed previously.

Complications of mild isolated traumatic uveitis without additional intraocular damage are uncommon. Some patients with isolated traumatic uveitis may have a severe prolonged inflammatory response requiring months of topical corticosteroid therapy and slow taper. These patients may be at increased risk for development of cataract as well as secondary glaucoma. Patients with severe blunt trauma often have concomitant injuries such as hyphema, angle recession, inflammatory trabeculitis, or other conditions that may lead to complications. In these patients, synechiae formation, secondary glaucoma, and traumatic cataract may occur as a result of these additional injuries.

Patients with traumatic uveitis and globe trauma are at increased risk for complications, typically due to the perforating/penetrating injuries and subsequent inflammatory response. Complications resulting from the globe trauma may result in synechiae formation, secondary glaucoma, cataract, as well as cyclitic membrane formation. Some patients may experience unremitting anterior uveitis following successful repair of the corneal and/or scleral wounds. In these cases, an occult foreign body should be suspected. Ultrasound is often useful to locate the foreign body, although in some patients computed tomography (CT) may be necessary [21–23]. Magnetic resonance imaging may also be useful for detection of occult non-metallic foreign bodies if initial CT scanning is negative [24]. Persistent inflammation may also occur in patients with chronic endophthalmitis following trauma. This is an atypical presenta-

tion of post-traumatic endophthalmitis, and the diagnosis is often delayed. In these patients, samples of aqueous and vitreous are typically obtained for culture and sensitivities. The etiology in these unusual cases may include bacteria as well as fungi. Treatment is guided by culture and sensitivity results (see section "Traumatic Endophthalmitis").

Lens-Induced Uveitis

Lens-induced uveitis (LIU) includes several types of uveitis that differ in etiology and pathogenesis. In this chapter, the term LIU will be used to describe any inflammatory process resulting from trauma to the lens. This includes blunt trauma as well as perforating and penetrating injuries to the lens. Older terms for these types of uveitis include phacoanaphylaxis, phacoanaphylactic endophthalmitis, phacoantigenic, and phacogenic uveitis.

The pathogenesis of LIU is uncertain; however, older theories are primarily based upon an autoimmune response to released lens proteins [25–28]. The proposed triggering mechanism in virtually all cases is traumatic rupture of the lens capsule [29]. Histopathology reveals an area of zonal granulomatous inflammation surrounding the lens material and ruptured lens capsule. Epithelioid and giant cells are also present in the zone of inflammation with a surrounding mononuclear cell infiltrate. The uvea is also infiltrated with lymphocytes as well as plasma cells [25, 29, 30].

Blunt trauma to the anterior segment can produce rapid posterior movement of the lens-iris diaphragm without apparent damage to the lens. However, in some cases, the trauma may cause rupture of the lens capsule with release of lens proteins into the anterior chamber [16, 31, 32]. Anterior chamber cells and flare are visible and a hypopyon may be present. Moderate to severe anterior uveitis is typical and lens material may be seen in the anterior chamber in some cases (Fig. 12.1). In most patients, fine or mutton-fat keratic precipitates are noted. A tear in the anterior lens capsule may be visible, although most cases will have a wrinkled anterior lens capsule surface. Additional injuries such as corneal abrasions or hyphema are common and may obscure assessment of the anterior segment structures and the lens-induced inflammatory response.

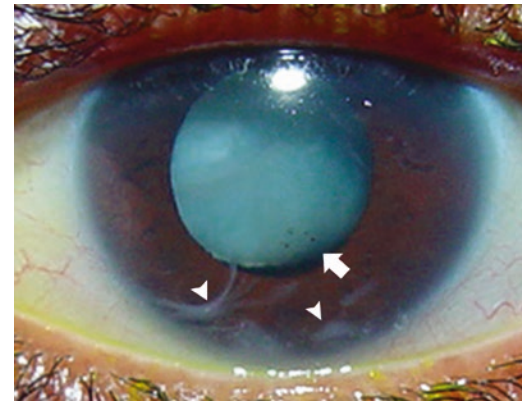

Fig. 12.1 Lens-induced uveitis following blunt trauma to the eye. Lens material (arrowheads) is visible in the inferior anterior chamber. Superior dislocation of lens (arrow) is also noted

Perforating or penetrating injuries to the lens may also damage the lens with liberation of lens protein into the anterior chamber as well as release of cortical and/or nuclear lens material [33]. The resultant inflammatory response may be mild to severe depending upon the amount of material released [34]. In many cases, multiple anterior segment structures are damaged at the time of injury. As a result, a mixed inflammatory reaction is often present in the anterior chamber with inflammatory cells and blood as well as tissue fragments and possibly foreign bodies. With most of these severe injuries, lens material may not be visible initially and only later discovered as the inflammation and blood clears. In some cases, the disorder is unrecognized and phthisis may eventually result [16, 25, 29, 30].

Treatment of LIU whether from blunt or penetrating injury is surgical removal of all lens material [25, 34, 35]. The timing of surgical removal is based upon several factors, including the presence of visible lens material in the anterior chamber, extent of lens damage, concomitant anterior segment injuries, visibility of the anterior chamber, as well as availability of equip-

ment for lens removal. In cases with extensive lens damage and large amounts of lens material in the anterior chamber, surgical removal should be performed at the same time as other injuries such as corneal laceration are repaired [29, 36, 37]. Removal of lens material may be delayed in patients without other injuries requiring urgent surgical repair [38]. Patients with small corneal lacerations and minimal lens trauma or cases with severe corneal edema or large hyphemas are examples of situations in which delayed surgical removal of lens material may be considered [37]. However, surgery should be performed as soon as possible in most patients to prevent worsening of lens-induced inflammation with time. Topical corticosteroids should be utilized to control the inflammation prior to and following surgery. Systemic corticosteroids may be necessary for some patients with severe inflammation inadequately controlled with topical therapy [25, 29].

Complications are relatively common, especially if there is a delay in diagnosis and treatment of traumatic LIU. Corneal edema may develop as a result of retained lens material in the anterior chamber or if there is an increase in intraocular pressure. Posterior synechiae as well as pupillary membrane formation are frequent in cases with uncontrolled inflammation. Some patients may develop secondary glaucoma due to the uveitis or chronic use of corticosteroids. In severe cases, a cyclitic membrane may develop leading to hypotony and ultimately phthisis [39, 40].

Traumatic Endophthalmitis

Infectious endophthalmitis is a rare but potentially devastating complication of penetrating and perforating injuries to the globe. Virtually all of these cases are exogenous endophthalmitis following trauma to the globe, although in combat and civilian disasters, multiple systemic injuries are common which may result in systemic infections leading to endogenous endophthalmitis [41–48]. In this chapter, traumatic endophthalmitis will refer to only those cases which are exogenous in nature, resulting from penetrating or perforating wounds.

The incidence of post-traumatic endophthalmitis has been estimated to be 0.9–17% [49–59]. Following combat-related injuries, the estimated incidence is 2–7.5% [12, 33, 60–64]. During the most recent US Military conflicts in Afghanistan and Iraq, no cases of traumatic endophthalmitis have been reported [64]. The incidence of endophthalmitis following open-globe injuries varies by several factors, including location (urban, rural, combat zone, etc.), presence of retained intraocular foreign body, lens capsule rupture, and delay in wound closure [50, 52, 56, 65]. In addition, estimated incidence rates may be less accurate in reports describing civilian or military mass casualty events due to the initial chaos of the event, limited time for meticulous data collection, and sparsity of follow-up data [66–71]. Rapid evacuation of casualties to higher levels of care at distant locations also contributes to the limited access to follow-up data [12, 64, 69].

The prognosis in cases of post-traumatic endophthalmitis varies by the virulence of the causative organism, extent of ocular injury, timing of treatment, presence of an intraocular foreign body, and presence of a retinal detachment [72–75]. Patients with poor initial visual acuity may also have a worse prognosis [75, 76]. Delays in primary repair greater than 24–72 hours are associated with worse visual outcomes [74, 77]. Compared with earlier studies, recent reports suggest that delayed removal of intraocular foreign bodies may not be associated with a worse prognosis in some situations [53, 63, 73, 78–80]. Overall, post-traumatic fungal endophthalmitis has a worse prognosis than bacterial endophthalmitis with over two-thirds of eyes having no light perception and many ultimately requiring evisceration or enucleation [81, 82].

Clinical manifestations of traumatic endophthalmitis are highly variable and may be masked by the underlying injury. As a result, delays in diagnosis are not uncommon. Patients typically complain of increasing pain with decreased vision during the first few days following injury or repair of the ruptured globe. However, in patients with fungal endophthalmitis, the onset of pain may be delayed for weeks to months following the injury. Common signs of endophthalmitis

include conjunctival hyperemia, severe anterior chamber cells and flare, corneal edema, and vitritis out of proportion to the underlying injury. Additional findings include purulent discharge from the site of injury, anterior chamber fibrin, hypopyon, infiltrates in the anterior chamber, or vitreous and retinal perivasculitis [74, 83–85]. In severe cases of post-traumatic endophthalmitis, panophthalmitis may develop if the infection spreads to the adjacent orbital structures. In these patients, eyelid edema and erythema, proptosis, pain with eye movement, and limited ocular motility are common [74]. Patients with *Bacillus cereus* endophthalmitis may have several unique clinical features, including severe eyelid edema, proptosis, corneal ring infiltrate or abscess, and in some cases fever and leukocytosis. These eyes may also demonstrate rapid progression of the infection with devastating visual loss. *Clostridium perfringens* is a much less common cause of endophthalmitis following trauma, but it too may have unusual clinical manifestations. Severe ocular pain, a green-brown hypopyon, and possibly intraocular gas bubbles are characteristic of infection with *Clostridium perfringens* [74, 86]. Rapid progression to panophthalmitis is not uncommon in these patients.

If traumatic endophthalmitis is suspected, specimens should be quickly obtained for stains, cultures, and antimicrobial sensitivities. If possible, specimens should be obtained prior to beginning antimicrobial therapy. However, in combat or civilian disasters, many patients have multiple systemic injuries and likely have received prophylactic antibiotics prior to the suspicion of endophthalmitis. Samples from the ocular wound, anterior chamber, and vitreous should be immediately inoculated onto appropriate microbiologic media. Vitreous specimens may be obtained by vitreous tap or biopsy [87]. Blood agar is used for detection of aerobic bacteria and fungi, while chocolate agar is utilized for aerobic bacteria as well as *Moraxella* and *Haemophilus* species. Thioglycolate broth is useful for anaerobic bacteria as well as microaerophilic bacteria. Sabouraud's dextrose agar is typically used for isolation of fungi [88–93]. Microscopic examination of Gram's stained specimens may reveal inflammatory cells as well as bacteria and their morphology and staining patterns. Staining with calcofluor-KOH should be performed in cases suspicious for fungal endophthalmitis [93, 94].

The causative organisms in most cases of traumatic endophthalmitis are bacteria. However, the organisms vary by location, contamination of the wound, and presence or absence of intraocular foreign bodies. Furthermore, infection with multiple organisms is not uncommon following traumatic injuries [50–52, 92, 95, 96]. Overall, gram-positive organisms are the most common pathogens in post-traumatic endophthalmitis accounting for approximately 75% of all cases [74, 97]. In many studies, *Staphylococcal epidermidis* is the most common gram-positive organism [52, 72, 74, 98–101]. This is not unexpected since *S. epidermis* is a normal constituent of the skin flora. Less common gram-positive bacteria include *S. aureus*, Streptococcal species, *Bacillus cereus*, other *Bacillus* species, and *Clostridium* species [51, 72, 74, 98, 101–106]. *Bacillus* species have been reported to account for up to 20% of post-traumatic endophthalmitis cases [74, 97, 102, 104, 105, 107]. *Bacillus cereus* is especially virulent with a rapid onset, usually within 24 hours following injury and often with devastating outcomes [108–111]. *Clostridium* species are gram-positive anaerobic bacteria which have the ability to form spores. *Clostridium perfringens* is found in soil throughout the world and is a known cause of post-traumatic endophthalmitis. Most cases of *Clostridium perfringens* endophthalmitis are associated with retained intraocular foreign bodies and have a very poor prognosis [74]. Gram-negative organisms are a less common cause of endophthalmitis accounting for up to 33% of cases of post-traumatic endophthalmitis [74, 98, 109]. Gram-negative organisms associated with endophthalmitis following trauma include *Pseudomonas* species, *Proteus mirabilis*, *Stenotrophomonas maltophilia*, *Acinetobacter*, and *Moraxella* [50, 72, 112–114]. Fungi are rare causes of post-traumatic endophthalmitis occurring in up to 15% of cases [72, 91]. Wounds contaminated by vegetable matter, mud, or stones may be at increased risk for fungal endophthalmitis [85, 115]. *Candida* species are the most

common fungal organisms isolated following globe trauma [90]. Specific fungal etiologies reported following globe trauma include *Candida albicans*, *Candida parapsilosis*, *Aspergillus fumigatus*, *Aspergillus flavus*, *Fusarium solani*, *Exophilia jeanselmei*, *Paecilomyces lilacinus*, *Acremonium curvulum*, *Alternaria infectoria*, *Scopulariopsis brevicaulis*, and *Cylindrocarpon tonkinese* [74, 85].

Medical management of post-traumatic endophthalmitis includes the use of prophylactic antibiotics following globe trauma as well as specific antimicrobial therapy of suspected and confirmed cases of endophthalmitis. Antibiotic prophylaxis includes antimicrobials administered prior to any clinical manifestations suggesting endophthalmitis [50]. Despite no widely accepted guidelines, prophylactic antibiotics are often recommended for patients with penetrating or perforating injuries to the globe [49, 56, 74, 116, 117]. Broad-spectrum, systemic, topical, and subconjunctival antibiotics are typically used in these cases. The contribution of systemic prophylactic measures to prevention of endophthalmitis is uncertain; however, systemic antibiotics are typically initiated if there is suspected or confirmed penetrating or perforating injury to the globe [49, 83, 118, 119]. Prophylactic intravenous systemic antibiotics are commonly used following combat injuries and civilian mass casualty events due to the presence of non-ocular injuries [120–124]. The choice of specific systemic antibiotics is based upon a number of factors, including the patient's overall condition, setting of the trauma, wound contamination, and presence of intraocular foreign bodies. Intravenous vancomycin is often used with ceftazidime to provide broad-spectrum antibiotic prophylaxis [49, 56, 74, 125]. Vancomycin is effective against most gram-positive organisms, including *Bacillus* species, while ceftazidime provides good gram-negative coverage. For patients with normal renal function, vancomycin 1 g every 12 hours and ceftazidime 1 g every 8 hours is a commonly used regimen [49, 126]. Clindamycin 300 mg every 8 hours may be used in patients allergic to vancomycin, while a fluoroquinolone may be substituted for ceftazidime in patients with a penicillin allergy. The optimal duration of intravenous antibiotic administration has not been established and varies among reports from 2 to 10 days [49, 56, 127].

Topical and subconjunctival antibiotics are begun after primary repair of open-globe injury. Despite no specific guidelines for the use of specific topical and/or subconjunctival antibiotics following surgical repair of globe trauma, most studies describe the use of at least topical antibiotics following primary repair of the wound. Various non-fortified topical antibiotics have been used following repair of globe trauma, including ciprofloxacin, moxifloxacin, levofloxacin, tobramycin, gentamicin, or chloramphenicol [73, 100, 118, 128, 129]. Prophylactic subconjunctival antibiotics include cefazolin, gentamicin, clindamycin, vancomycin, and ceftazidime [100, 116, 119, 130]. The use of prophylactic intravitreal antibiotics at the time of surgical repair is controversial, yet patients at high risk for traumatic endophthalmitis may benefit from prophylactic intravitreal antibiotics [49, 50, 83, 90, 106, 109, 117, 131, 132]. Criteria for these high-risk cases varies but probably should include patients with intraocular foreign bodies, lens disruption, injury in a rural setting, and wounds contaminated by vegetable or organic matter [56]. Intravitreal vancomycin 1 mg/0.1 ml and ceftazidime 2.25 mg/0.1 ml are most commonly used for prophylaxis in these situations [51, 56, 83, 125, 129, 131].

Management of cases of suspected traumatic endophthalmitis begins with obtaining specimens for culture and sensitivities as described previously. Broad-spectrum antibiotics are started immediately thereafter while awaiting the results of cultures. Empiric intravenous antibiotics should have good intravitreal penetration and sensitivity against the spectrum of bacteria commonly associated with ocular trauma. Sensitivity against *Bacillus* species should also be considered when choosing systemic antibiotics due to the high incidence of *Bacillus* species endophthalmitis following trauma [52, 56, 72, 74, 107, 116]. Intravenous vancomycin 1 g every 12 hours and ceftazidime 1 g every 8 hours provide good coverage against most organisms encountered following globe trauma including *Bacillus* spe-

cies [56, 74, 98, 126]. Intravenous clindamycin 300 mg every 8 hours may be used if vancomycin is contraindicated; especially if there is a clinical suspicion of *Bacillus* species infection [74, 105]. Several fluoroquinolones including ciprofloxacin, levofloxacin, and moxifloxacin achieve good aqueous and vitreous levels and may be useful alternative agents, but there are limited data regarding their use in traumatic endophthalmitis [49, 73, 101, 119, 133]. Intravenous voriconazole is recommended for all patients with post-traumatic fungal endophthalmitis based upon its coverage against *Candida* and *Aspergillus* species [74, 82, 134].

Intravitreal antibiotics are administered immediately after intraocular specimens are obtained. Initial antibiotic therapy should be broad spectrum to cover both gram-positive and gram-negative organisms. In most cases of trauma, infection with *Bacillus* species should be considered when selecting antibiotics [56, 74]. Intravitreal vancomycin 1 mg/0.1 ml and ceftazidime 2.25 mg/0.1 ml are typically used to provide broad-spectrum coverage following trauma [56, 126, 128]. In patients with suspected fungal endophthalmitis, intravitreal amphotericin B 5 μg/0.1 ml or voriconazole 100 μg/0.1 ml is typically used as initial therapy [56, 135]. Intravitreal corticosteroids may be considered in the management of post-traumatic bacterial endophthalmitis; however, their use remains somewhat controversial. On the contrary, intravitreal corticosteroids should not be used in cases of suspected or proven fungal endophthalmitis [56]. Corticosteroids modulate the immune response and limit tissue damage by inflammatory cells as well as bacterial toxins [136–139]. Nevertheless, it is important to recognize that intravitreal corticosteroids may exacerbate the infection if the infecting organism is not sensitive to the empiric intravitreal antibiotics [74]. Dexamethasone 0.4 mg/0.1 ml is the most commonly used intravitreal corticosteroid in cases of bacterial endophthalmitis [140, 141].

Topical broad-spectrum antibiotics are typically used in combination with intravitreal antibiotics while awaiting culture results. The most commonly used regimen in cases of suspected traumatic endophthalmitis is fortified vancomycin hydrochloride (50 mg/ml) with ceftazidime (100 mg/ml) [74]. Both of these drugs are administered every hour. An alternative combination consists of fortified gentamicin or tobramycin 14 mg/ml with fortified cefazolin 50 mg/ml. Topical fluoroquinolones including ciprofloxacin 0.3% or moxifloxacin 0.3% are additional options for initial therapy [74]. Topical antibiotics can be modified following results of microbiology cultures based upon sensitivities of the isolated organism. Topical corticosteroids as well as cycloplegics are also frequently administered in conjunction with antibiotic therapy [56].

Subconjunctival antibiotics are often used in the management of endophthalmitis. In patients with suspected traumatic endophthalmitis, subconjunctival vancomycin 25 mg and ceftazidime 100 mg are commonly used [56]. Other subconjunctival antibiotic options include tobramycin, gentamicin, or amikacin, although there are limited data regarding their use following globe trauma [116, 129, 142]. Subconjunctival injection of aminoglycosides should be done with extreme caution to avoid intraocular injection. Additionally, aminoglycosides should not be injected into the subconjunctival space if there is any evidence or suspicion of an open wound [126, 143].

If cultures identify a specific organism or multiple organisms, the sensitivities should be reviewed to determine the most appropriate antimicrobial therapy. In some cases, this may require switching to a different antimicrobial agent and/or discontinuation of broad-spectrum drugs. Repeat intravitreal injections may be necessary in some patients based upon culture results or those with no improvement or worsening inflammation. Repeat intravitreal antibiotics or antifungals might also be considered in eyes with highly virulent or resistant organisms such as *Bacillus* species [56, 144].

Vitrectomy may be utilized in selected cases of traumatic endophthalmitis. Pars plana vitrectomy is often necessary for management of retinal detachment, intraocular foreign bodies, and posterior dislocated lens material. Recommendations regarding timing of vitrectomy in patients with

intraocular foreign bodies differ but some surgeons advocate early removal of foreign bodies to avoid complications such as endophthalmitis and proliferative vitreoretinopathy [53, 79]. On the other hand, recent studies suggest that delayed removal may not increase the risk of endophthalmitis, especially if patients receive prophylactic antibiotics prior to surgery [53, 73, 78–80, 145]. Pars plana vitrectomy may also be useful in patients with severe inflammation and no response to intravitreal antibiotics within 48 hours or marked worsening of inflammation after 24 hours following initial therapy [50, 72, 74]. Possible benefits of vitrectomy in these cases are obtaining additional vitreous for culture, reduction in the pathogen load, and partial clearing of vitreous opacities [75, 97, 146]. However, there are no widely accepted recommendations regarding timing, surgical techniques, or use of antibiotics in the irrigating solutions during vitrectomy in patients with traumatic endophthalmitis. Bhagat and coworkers suggest a limited vitrectomy in these situations based upon the suboptimal view of the posterior segment and high risk for retinal tears and detachment associated with the severe inflammation [74].

References

1. Duke-Elder S, MacFaul PA. Concussions and contusions. In: Duke-Elder S, editor. System of ophthalmology, Vol. XIV, Injuries, Part 1: Mechanical injuries. St. Louis: Mosby; 1972. p. 63–149.
2. Duma SM, Ng TP, Kennedy EA, Stitzel JD, Herring IP, Kuhn F. Determination of significant parameters for eye injury risk from projectiles. J Trauma. 2005;59(4):960–4.
3. Ramstead C, Ng M, Rudnisky CJ. Ocular injuries associated with Airsoft guns: a case series. Can J Ophthalmol. 2008;43(5):584–7.
4. Canavan YM, Archer DB. Anterior segment consequences of blunt ocular injury. Br J Ophthalmol. 1982;66(9):549–55.
5. Schein OD, Hibberd PL, Shingleton BJ, Kunzweiler T, Frambach DA, Seddon JM, et al. The spectrum and burden of ocular injury. Ophthalmology. 1988;95(3):300–5.
6. Macewen CJ. Eye injuries: a prospective survey of 5671 cases. Br J Ophthalmol. 1989;73(11):888–94.
7. Capao Filipe JA. Modern sports eye injuries. Br J Ophthalmol. 2003;87(11):1336–9.
8. Morley MG, Nguyen JK, Heier JS, Shingleton BJ, Pasternak JF, Bower KS. Blast eye injuries: a review for first responders. Disaster Med Public Health Prep. 2010;4(2):154–60.
9. Mines M, Thach A, Mallonee S, Hildebrand L, Shariat S. Ocular injuries sustained by survivors of the Oklahoma City bombing. Ophthalmology. 2000;107(5):837–43.
10. Quere MA, Bouchat J, Cornand G. Ocular blast injuries. Am J Ophthalmol. 1969;67(1):64–9.
11. Beiran I, Miller B. Pure ocular blast injury. Am J Ophthalmol. 1992;114(4):504–5.
12. Wong TY, Seet MB, Ang CL. Eye injuries in twentieth century warfare: a historical perspective. Surv Ophthalmol. 1997;41(6):433–59.
13. Abbotts R, Harrison SE, Cooper GL. Primary blast injuries to the eye: a review of the evidence. J R Army Med Corps. 2007;153(2):119–23.
14. Blanch RJ, Bindra MS, Jacks AS, Scott RA. Ophthalmic injuries in British armed forces in Iraq and Afghanistan. Eye (Lond). 2011;25(2):218–23.
15. Scott R. The injured eye. Philos Trans R Soc Lond Ser B Biol Sci. 2011;366(1562):251–60.
16. Banitt MR, Malta JB, Mian SI, Soong HK. Rupture of anterior lens capsule from blunt ocular injury. J Cataract Refract Surg. 2009;35(5):943–5.
17. Maloney WF, Colvard M, Bourne WM, Gardon R. Specular microscopy of traumatic posterior annular keratopathy. Arch Ophthalmol. 1979;97(9):1647–50.
18. Cibis GW, Weingeist TA, Krachmer JH. Traumatic corneal endothelial rings. Arch Ophthalmol. 1978;96(3):485–8.
19. De Leon-Ortega JE, Girkin CA. Ocular trauma-related glaucoma. Ophthalmol Clin N Am. 2002;15(2):215–23.
20. Milder E, Davis K. Ocular trauma and glaucoma. Int Ophthalmol Clin. 2008;48(4):47–64.
21. Deramo VA, Shah GK, Baumal CR, Fineman MS, Corrĕa ZM, Benson WE, et al. The role of ultrasound biomicroscopy in ocular trauma. Trans Am Ophthalmol Soc. 1998;96:355–65; discussion 365–7.
22. Deramo VA, Shah GK, Baumal CR, Fineman MS, Corrêa ZM, Benson WE, et al. Ultrasound biomicroscopy as a tool for detecting and localizing occult foreign bodies after ocular trauma. Ophthalmology. 1999;106(2):301–5.
23. Patel SN, Langer PD, Zarbin MA, Bhagat N. Diagnostic value of clinical examination and radiographic imaging in identification of intraocular foreign bodies in open globe injury. Eur J Ophthalmol. 2012;22(2):259–68.
24. Moisseiev E, Last D, Goez D, Barak A, Mardor Y. Magnetic resonance imaging and computed tomography for the detection and characterization of nonmetallic intraocular foreign bodies. Retina. 2015;35(1):82–94.
25. Marak GE. Phacoanaphylactic endophthalmitis. Surv Ophthalmol. 1992;36(5):325–39.
26. Gery I, Nussenblatt R, BenEzra D. Dissociation between humoral and cellular immune responses

to lens antigens. Invest Ophthalmol Vis Sci. 1981;20(1):32–9.
27. Goldschmidt L, Goldbaum M, Walker SM, Weigle WO. The immune response to homologous lens crystallin. I. Antibody production after lens injury. J Immunol. 1982;129(4):1652–7.
28. Lai JC, Lobanoff MC, Fukushima A, Wawrousek EF, Chan CC, Whitcup SM, Gery I. Uveitis induced by lymphocytes sensitized against a transgenically expressed lens protein. Invest Ophthalmol Vis Sci. 1999;40(11):2735–9.
29. Perlman EM, Albert DM. Clinically unsuspected phacoanaphylaxis after ocular trauma. Arch Ophthalmol. 1977;95(2):244–6.
30. Thach AB, Marak GE, McLean IW, Green WR. Phacoanaphylactic endophthalmitis: a clinicopathologic review. Int Ophthalmol. 1991;15(4):271–9.
31. Zabriskie NA, Hwang IP, Ramsey JF, Crandall AS. Anterior lens capsule rupture caused by air bag trauma. Am J Ophthalmol. 1997;123(6):832–3.
32. Dezhagah H. Circular anterior lens capsule rupture caused by blunt ocular trauma. Middle East Afr J Ophthalmol. 2010;17(1):103–5.
33. Dansey-Browning GC. The value of ophthalmic treatment in the field. Br J Ophthalmol. 1944;28(2):87–97.
34. Chandler PA. Problems in the diagnosis and treatment of lens-induced uveitis and glaucoma. AMA Arch Ophthalmol. 1958;60(5):828–41.
35. Muga R, Maul E. The management of lens damage in perforating corneal lacerations. Br J Ophthalmol. 1978;62(11):784–7.
36. Lamkin JC, Azar DT, Mead MD, Volpe NJ. Simultaneous corneal laceration repair, cataract removal, and posterior chamber intraocular lens implantation. Am J Ophthalmol. 1992;113(6):626–31.
37. Moisseiev J, Segev F, Harizman N, Arazi T, Rotenstreich Y, Assia EI. Primary cataract extraction and intraocular lens implantation in penetrating ocular trauma. Ophthalmology. 2001;108(6):1099–103.
38. Tanito M, Kaidzu S, Katsube T, Nonoyama S, Takai Y, Ohira A. Diagnostic Western blot for lens-specific proteins in aqueous fluid after traumatic lens-induced uveitis. Jpn J Ophthalmol. 2009;53(4):436–9.
39. Inazumi K, Gentile RC, Lee KY, Ishikawa H, McCormick SA, Liebmann JM, Ritch R. Ultrasound biomicroscopic diagnosis of cyclitic membranes. Am J Ophthalmol. 2001;131(4):446–50.
40. Chan CC, Fujikawa LS, Rodrigues MM, Stevens G, Nussenblatt RB. Immunohistochemistry and electron microscopy of cyclitic membrane. Report of a case. Arch Ophthalmol. 1986;104(7):1040–5.
41. Petersen K, Riddle MS, Danko JR, Blazes DL, Hayden R, Tasker SA, Dunne JR. Trauma-related infections in battlefield casualties from Iraq. Ann Surg. 2007;245(5):803–11.
42. Murray CK. Infectious disease complications of combat-related injuries. Crit Care Med. 2008;36(7 Suppl):S358–64.
43. Murray CK. Epidemiology of infections associated with combat-related injuries in Iraq and Afghanistan. J Trauma. 2008;64(3 Suppl):S232–8.
44. Murray CK, Hinkle MK, Yun HC. History of infections associated with combat-related injuries. J Trauma. 2008;64(3 Suppl):S221–31.
45. Kluger Y. Bomb explosions in acts of terrorism--detonation, wound ballistics, triage and medical concerns. Isr Med Assoc J. 2003;5(4):235–40.
46. Kluger Y, Peleg K, Daniel-Aharonson L, Mayo A, Israeli Trauma Group. The special injury pattern in terrorist bombings. J Am Coll Surg. 2004;199(6):875–9.
47. Bartels SA, VanRooyen MJ. Medical complications associated with earthquakes. Lancet. 2012;379(9817):748–57.
48. Keven K, Ates K, Sever MS, Yenicesu M, Canbakan B, Arinsoy T, et al. Infectious complications after mass disasters: the Marmara earthquake experience. Scand J Infect Dis. 2003;35(2):110–3.
49. Andreoli CM, Andreoli MT, Kloek CE, Ahuero AE, Vavvas D, Durand ML. Low rate of endophthalmitis in a large series of open globe injuries. Am J Ophthalmol. 2009;147(4):601–608.e2.
50. Essex RW, Yi Q, Charles PG, Allen PJ. Post-traumatic endophthalmitis. Ophthalmology. 2004;111(11):2015–22.
51. Boldt HC, Pulido JS, Blodi CF, Folk JC, Weingeist TA. Rural endophthalmitis. Ophthalmology. 1989;96(12):1722–6.
52. Thompson JT, Parver LM, Enger CL, Mieler WF, Liggett PE. Infectious endophthalmitis after penetrating injuries with retained intraocular foreign bodies. National eye trauma system. Ophthalmology. 1993;100(10):1468–74.
53. Jonas JB, Knorr HL, Budde WM. Prognostic factors in ocular injuries caused by intraocular or retrobulbar foreign bodies. Ophthalmology. 2000;107(5):823–8.
54. Duch-Samper AM, Menezo JL, Hurtado-Sarrió M. Endophthalmitis following penetrating eye injuries. Acta Ophthalmol Scand. 1997;75(1):104–6.
55. Verbraeken H, Rysselaere M. Post-traumatic endophthalmitis. Eur J Ophthalmol. 1994;4(1):1–5.
56. Ahmed Y, Schimel AM, Pathengay A, Colyer MH, Flynn HW. Endophthalmitis following open-globe injuries. Eye (Lond). 2012;26(2):212–7.
57. Ahmed SA, Zaki RG. Forensic analysis of ocular injuries during the 2011 revolution in Egypt. Forensic Sci Int. 2013;233(1–3):348–54.
58. Muzaffar W, Khan MD, Akbar MK, Malik AM, Durrani OM. Mine blast injuries: ocular and social aspects. Br J Ophthalmol. 2000;84(6):626–30.
59. Parke DW, Pathengay A, Flynn HW, Albini T, Schwartz SG. Risk factors for endophthalmitis and retinal detachment with retained intraocular foreign bodies. J Ophthalmol. 2012;2012:758526.
60. Treister G. Ocular casualties in the six-day war. Am J Ophthalmol. 1969;68(4):669–75.
61. Anderson WD. Prophylactic antibiotics and endophthalmitis in Vietnam. Am J Ophthalmol. 1973;75(3):481–5.

62. Lashkari K, Lashkari MH, Kim AJ, Crane WG, Jalkh AE. Combat-related eye trauma: a review of 5,320 cases. Int Ophthalmol Clin. 1995;35(1):193–203.
63. Thach AB, Ward TP, Dick JS, Bauman WC, Madigan WP, Goff MJ, Thordsen JE. Intraocular foreign body injuries during Operation Iraqi Freedom. Ophthalmology. 2005;112(10):1829–33.
64. Weichel ED, Colyer MH, Ludlow SE, Bower KS, Eiseman AS. Combat ocular trauma visual outcomes during operations Iraqi and Enduring Freedom. Ophthalmology. 2008;115(12):2235–45.
65. Yang CS, Lu CK, Lee FL, Hsu WM, Lee YF, Lee SM. Treatment and outcome of traumatic endophthalmitis in open globe injury with retained intraocular foreign body. Ophthalmologica. 2010;224(2):79–85.
66. Klein JS, Weigelt JA. Disaster management. Lessons learned. Surg Clin North Am. 1991;71(2):257–66.
67. Singer AJ, Singer AH, Halperin P, Kaspi G, Assaf J. Medical lessons from terror attacks in Israel. J Emerg Med. 2007;32(1):87–92.
68. McAlister CN, Murray TJ, Lakosha H, Maxner CE. The Halifax disaster (1917): eye injuries and their care. Br J Ophthalmol. 2007;91(6):832–5.
69. Thach AB, Johnson AJ, Carroll RB, Huchun A, Ainbinder DJ, Stutzman RD, et al. Severe eye injuries in the war in Iraq, 2003-2005. Ophthalmology. 2008;115(2):377–82.
70. Butler FK, Blackbourne LH. Battlefield trauma care then and now: a decade of tactical combat casualty care. J Trauma Acute Care Surg. 2012;73(6 Suppl 5):S395–402.
71. Sobaci G, Mutlu FM, Bayer A, Karagül S, Yildirim E. Deadly weapon-related open-globe injuries: outcome assessment by the ocular trauma classification system. Am J Ophthalmol. 2000;129(1):47–53.
72. Brinton GS, Topping TM, Hyndiuk RA, Aaberg TM, Reeser FH, Abrams GW. Posttraumatic endophthalmitis. Arch Ophthalmol. 1984;102(4):547–50.
73. Colyer MH, Weber ED, Weichel ED, Dick JS, Bower KS, Ward TP, Haller JA. Delayed intraocular foreign body removal without endophthalmitis during Operations Iraqi Freedom and Enduring Freedom. Ophthalmology. 2007;114(8):1439–47.
74. Bhagat N, Nagori S, Zarbin M. Post-traumatic infectious endophthalmitis. Surv Ophthalmol. 2011;56(3):214–51.
75. Cornut PL, Youssef el B, Bron A, Thuret G, Gain P, Burillon C, et al. A multicentre prospective study of post-traumatic endophthalmitis. Acta Ophthalmol. 2013;91(5):475–82.
76. Das T, Kunimoto DY, Sharma S, Jalali S, Majji AB, Nagaraja Rao T, et al. Relationship between clinical presentation and visual outcome in postoperative and posttraumatic endophthalmitis in south central India. Indian J Ophthalmol. 2005;53(1):5–16.
77. Nicoară SD, Irimescu I, Călinici T, Cristian C. Outcome and prognostic factors for traumatic endophthalmitis over a 5-year period. J Ophthalmol. 2014;2014:747015.
78. Wani VB, Al-Ajmi M, Thalib L, Azad RV, Abul M, Al-Ghanim M, Sabti K. Vitrectomy for posterior segment intraocular foreign bodies: visual results and prognostic factors. Retina. 2003;23(5):654–60.
79. Erakgun T, Egrilmez S. Prognostic factors in vitrectomy for posterior segment intraocular foreign bodies. J Trauma. 2008;64(4):1034–7.
80. Choovuthayakorn J, Hansapinyo L, Ittipunkul N, Patikulsila D, Kunavisarut P. Predictive factors and outcomes of posterior segment intraocular foreign bodies. Eye (Lond). 2011;25(12):1622–6.
81. Wykoff CC, Flynn HW, Miller D, Scott IU, Alfonso EC. Exogenous fungal endophthalmitis: microbiology and clinical outcomes. Ophthalmology. 2008;115(9):1501–7, 1507.e1–2
82. Silva RA, Sridhar J, Miller D, Wykoff CC, Flynn HW. Exogenous fungal endophthalmitis: an analysis of isolates and susceptibilities to antifungal agents over a 20-year period (1990–2010). Am J Ophthalmol. 2015;159(2):257–264.e1.
83. Reynolds DS, Flynn HW. Endophthalmitis after penetrating ocular trauma. Curr Opin Ophthalmol. 1997;8(3):32–8.
84. Thompson WS, Rubsamen PE, Flynn HW, Schiffman J, Cousins SW. Endophthalmitis after penetrating trauma. Risk factors and visual acuity outcomes. Ophthalmology. 1995;102(11):1696–701.
85. Gupta A, Srinivasan R, Kaliaperumal S, Saha I. Post-traumatic fungal endophthalmitis--a prospective study. Eye (Lond). 2008;22(1):13–7.
86. Abu el-Asrar AM, al-Amro SA, al-Mosallam AA, al-Obeidan S. Post-traumatic endophthalmitis: causative organisms and visual outcome. Eur J Ophthalmol. 1999;9(1):21–31.
87. Han DP, Wisniewski SR, Kelsey SF, Doft BH, Barza M, Pavan PR. Microbiologic yields and complication rates of vitreous needle aspiration versus mechanized vitreous biopsy in the endophthalmitis vitrectomy study. Retina. 1999;19(2):98–102.
88. Allansmith MR, Skaggs C, Kimura SJ. Anterior chamber paracentesis. Diagnostic value in postoperative endophthalmitis. Arch Ophthalmol. 1970;84(6):745–8.
89. Forster RK. Endophthalmitis. Diagnostic cultures and visual results. Arch Ophthalmol. 1974;92(5):387–92.
90. Peyman GA, Carroll CP, Raichand M. Prevention and management of traumatic endophthalmitis. Ophthalmology. 1980;87(4):320–4.
91. Rowsey JJ, Newsom DL, Sexton DJ, Harms WK. Endophthalmitis: current approaches. Ophthalmology. 1982;89(9):1055–66.
92. Affeldt JC, Flynn HW, Forster RK, Mandelbaum S, Clarkson JG, Jarus GD. Microbial endophthalmitis resulting from ocular trauma. Ophthalmology. 1987;94(4):407–13.
93. Baron EJ, Miller JM, Weinstein MP, Richter SS, Gilligan PH, Thomson RB, et al. A guide to utilization of the microbiology laboratory for diagnosis of infectious diseases: 2013 recommendations by the Infectious Diseases Society of America (IDSA) and

the American Society for Microbiology (ASM)(a). Clin Infect Dis. 2013;57(4):e22–e121.
94. Thomas PA. Current perspectives on ophthalmic mycoses. Clin Microbiol Rev. 2003;16(4):730–97.
95. Nobe JR, Gomez DS, Liggett P, Smith RE, Robin JB. Post-traumatic and postoperative endophthalmitis: a comparison of visual outcomes. Br J Ophthalmol. 1987;71(8):614–7.
96. Jindal A, Moreker MR, Pathengay A, Khera M, Jalali S, Majji A, et al. Polymicrobial endophthalmitis: prevalence, causative organisms, and visual outcomes. J Ophthalmic Inflamm Infect. 2013;3(1):6.
97. Alfaro DV, Roth D, Liggett PE. Posttraumatic endophthalmitis. Causative organisms, treatment, and prevention. Retina. 1994;14(3):206–11.
98. Chhabra S, Kunimoto DY, Kazi L, Regillo CD, Ho AC, Belmont J, et al. Endophthalmitis after open globe injury: microbiologic spectrum and susceptibilities of isolates. Am J Ophthalmol. 2006;142(5):852–4.
99. Rubsamen PE, Cousins SW, Martinez JA. Impact of cultures on management decisions following surgical repair of penetrating ocular trauma. Ophthalmic Surg Lasers. 1997;28(1):43–9.
100. Sabaci G, Bayer A, Mutlu FM, Karagül S, Yildirim E. Endophthalmitis after deadly-weapon-related open-globe injuries: risk factors, value of prophylactic antibiotics, and visual outcomes. Am J Ophthalmol. 2002;133(1):62–9.
101. Kunimoto DY, Das T, Sharma S, Jalali S, Majji AB, Gopinathan U, et al. Microbiologic spectrum and susceptibility of isolates: part II. Posttraumatic endophthalmitis. Endophthalmitis research group. Am J Ophthalmol. 1999;128(2):242–4.
102. David DB, Kirkby GR, Noble BA. Bacillus cereus endophthalmitis. Br J Ophthalmol. 1994;78(7):577–80.
103. Long C, Liu B, Xu C, Jing Y, Yuan Z, Lin X. Causative organisms of post-traumatic endophthalmitis: a 20-year retrospective study. BMC Ophthalmol. 2014;14:34.
104. Miller JJ, Scott IU, Flynn HW, Smiddy WE, Murray TG, Berrocal A, Miller D. Endophthalmitis caused by bacillus species. Am J Ophthalmol. 2008;145(5):883–8.
105. Schemmer GB, Driebe WT. Posttraumatic bacillus cereus endophthalmitis. Arch Ophthalmol. 1987;105(3):342–4.
106. Zhang Y, Zhang MN, Jiang CH, Yao Y, Zhang K. Endophthalmitis following open globe injury. Br J Ophthalmol. 2010;94(1):111–4.
107. O'Day DM, Smith RS, Gregg CR, Turnbull PC, Head WS, Ives JA, Ho PC. The problem of bacillus species infection with special emphasis on the virulence of bacillus cereus. Ophthalmology. 1981;88(8):833–8.
108. Davey RT, Tauber WB. Posttraumatic endophthalmitis: the emerging role of bacillus cereus infection. Rev Infect Dis. 1987;9(1):110–23.
109. Lieb DF, Scott IU, Flynn HW, Miller D, Feuer WJ. Open globe injuries with positive intraocular cultures: factors influencing final visual acuity outcomes. Ophthalmology. 2003;110(8):1560–6.
110. Wiskur BJ, Robinson ML, Farrand AJ, Novosad BD, Callegan MC. Toward improving therapeutic regimens for bacillus endophthalmitis. Invest Ophthalmol Vis Sci. 2008;49(4):1480–7.
111. Bottone EJ. Bacillus cereus, a volatile human pathogen. Clin Microbiol Rev. 2010;23(2):382–98.
112. Berrocal AM, Scott IU, Miller D, Flynn HW. Endophthalmitis caused by moraxella species. Am J Ophthalmol. 2001;132(5):788–90.
113. Lai TY, Kwok AK, Fung KS, Chan W-M, Fan DS, Lam DS. Stenotrophomonas maltophilia endophthalmitis after penetrating injury by a wooden splinter. Eye. 2001;15(3):353–4.
114. Patton N. Post-traumatic endophthalmitis caused by xanthomonas maltophilia. Eye (Lond). 2001;15(Pt 6):801–2.
115. Pflugfelder SC, Flynn HW, Zwickey TA, Forster RK, Tsiligianni A, Culbertson WW, Mandelbaum S. Exogenous fungal endophthalmitis. Ophthalmology. 1988;95(1):19–30.
116. Parrish CM, O'Day DM. Traumatic endophthalmitis. Int Ophthalmol Clin. 1987;27(2):112–9.
117. Mittra RA, Mieler WF. Controversies in the management of open-globe injuries involving the posterior segment. Surv Ophthalmol. 1999;44(3):215–25.
118. Ariyasu RG, Kumar S, LaBree LD, Wagner DG, Smith RE. Microorganisms cultured from the anterior chamber of ruptured globes at the time of repair. Am J Ophthalmol. 1995;119(2):181–8.
119. Colyer MH, Chun DW, Bower KS, Dick JS, Weichel ED. Perforating globe injuries during operation Iraqi Freedom. Ophthalmology. 2008;115(11):2087–93.
120. Hospenthal DR, Murray CK, Andersen RC, Blice JP, Calhoun JH, Cancio LC, et al. Guidelines for the prevention of infection after combat-related injuries. J Trauma. 2008;64(3 Suppl):S211–20.
121. Turégano-Fuentes F, Caba-Doussoux P, Jover-Navalón JM, Martín-Pérez E, Fernández-Luengas D, Díez-Valladares L, et al. Injury patterns from major urban terrorist bombings in trains: the Madrid experience. World J Surg. 2008;32(6):1168–75.
122. Aschkenasy-Steuer G, Shamir M, Rivkind A, Mosheiff R, Shushan Y, Rosenthal G, et al. Clinical review: the Israeli experience: conventional terrorism and critical care. Crit Care. 2005;9(5):490–9.
123. Yang C, Wang HY, Zhong HJ, Zhou L, Jiang DM, Du DY, et al. The epidemiological analyses of trauma patients in Chongqing teaching hospitals following the Wenchuan earthquake. Injury. 2009;40(5):488–92.
124. Miskin IN, Nir-Paz R, Block C, Merin O, Burshtein S, Pirogovsky S, et al. Antimicrobial therapy for wound infections after catastrophic earthquakes. N Engl J Med. 2010;363(26):2571–3.

125. Lorch A, Sobrin L. Prophylactic antibiotics in posttraumatic infectious endophthalmitis. Int Ophthalmol Clin. 2013;53(4):167–76.
126. Kresloff MS, Castellarin AA, Zarbin MA. Endophthalmitis. Surv Ophthalmol. 1998;43(3): 193–224.
127. Faghihi H, Hajizadeh F, Esfahani MR, Rasoulinejad SA, Lashay A, Mirshahi A, et al. Posttraumatic endophthalmitis: report no. 2. Retina. 2012;32(1):146–51.
128. Narang S, Gupta V, Gupta A, Dogra MR, Pandav SS, Das S. Role of prophylactic intravitreal antibiotics in open globe injuries. Indian J Ophthalmol. 2003;51(1):39–44.
129. Soheilian M, Rafati N, Mohebbi MR, Yazdani S, Habibabadi HF, Feghhi M, et al. Prophylaxis of acute posttraumatic bacterial endophthalmitis: a multicenter, randomized clinical trial of intraocular antibiotic injection, report 2. Arch Ophthalmol. 2007;125(4):460–5.
130. Mieler WF, Ellis MK, Williams DF, Han DP. Retained intraocular foreign bodies and endophthalmitis. Ophthalmology. 1990;97(11):1532–8.
131. Seal DV, Kirkness CM. Criteria for intravitreal antibiotics during surgical removal of intraocular foreign bodies. Eye (Lond). 1992;6(Pt 5):465–8.
132. Meredith TA. Posttraumatic endophthalmitis. Arch Ophthalmol. 1999;117(4):520–1.
133. Hariprasad SM, Shah GK, Mieler WF, Feiner L, Blinder KJ, Holekamp NM, et al. Vitreous and aqueous penetration of orally administered moxifloxacin in humans. Arch Ophthalmol. 2006;124(2):178–82.
134. Hariprasad SM, Mieler WF, Holz ER, Gao H, Kim JE, Chi J, Prince RA. Determination of vitreous, aqueous, and plasma concentration of orally administered voriconazole in humans. Arch Ophthalmol. 2004;122(1):42–7.
135. Kernt M, Neubauer AS, De Kaspar HM, Kampik A. Intravitreal voriconazole: in vitro safety-profile for fungal endophthalmitis. Retina. 2009;29(3):362–70.
136. Meredith TA, Aguilar HE, Miller MJ, Gardner SK, Trabelsi A, Wilson LA. Comparative treatment of experimental staphylococcus epidermidis endophthalmitis. Arch Ophthalmol. 1990;108(6):857–60.
137. Schulman JA, Peyman GA. Intravitreal corticosteroids as an adjunct in the treatment of bacterial and fungal endophthalmitis. A review. Retina. 1992;12(4):336–40.
138. Park SS, Samiy N, Ruoff K, D'Amico DJ, Baker AS. Effect of intravitreal dexamethasone in treatment of pneumococcal endophthalmitis in rabbits. Arch Ophthalmol. 1995;113(10):1324–9.
139. Kernt M, Kampik A. Endophthalmitis: pathogenesis, clinical presentation, management, and perspectives. Clin Ophthalmol. 2010;4:121–35.
140. Das T, Jalali S, Gothwal VK, Sharma S, Naduvilath TJ. Intravitreal dexamethasone in exogenous bacterial endophthalmitis: results of a prospective randomised study. Br J Ophthalmol. 1999;83(9): 1050–5.
141. Albrecht E, Richards JC, Pollock T, Cook C, Myers L. Adjunctive use of intravitreal dexamethasone in presumed bacterial endophthalmitis: a randomised trial. Br J Ophthalmol. 2011;95(10):1385–8.
142. Smiddy WE, Smiddy RJ, Ba'Arath B, Flynn HW, Murray TG, Feuer WJ, Miller D. Subconjunctival antibiotics in the treatment of endophthalmitis managed without vitrectomy. Retina. 2005;25(6): 751–8.
143. Judson PH. Aminoglycoside macular toxicity after subconjunctival injection. Case report. Arch Ophthalmol. 1989;107(9):1282–3.
144. Shaarawy A, Grand MG, Meredith TA, Ibanez HE. Persistent endophthalmitis after intravitreal antimicrobial therapy. Ophthalmology. 1995;102(3):382–7.
145. Hutton WL, Fuller DG. Factors influencing final visual results in severely injured eyes. Am J Ophthalmol. 1984;97(6):715–22.
146. Duch-Samper AM, Chaqués-Alepuz V, Menezo JL, Hurtado-Sarrió M. Endophthalmitis following open-globe injuries. Curr Opin Ophthalmol. 1998;9(3):59–65.