Ocular Trauma
Series Editor: Hua Yan

Hua Yan *Editor*

Management on Complicated Ocular Trauma

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Ocular Trauma

Series Editor

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Ocular trauma can be a serious threat to vision, especially if not medically intervened appropriately and in a timely fashion. Immediate and accurate diagnosis and effective treatment is the key to save the eyes and visual function, as well as a great challenge to ophthalmologists, especially emergency doctors. This book series is designed to help the doctors and clinical practitioners have a thorough understanding of ophthalmic emergencies and a mastery of every details of ocular trauma. To do the best, it is required that the ER doctors have solid theoretical knowledge about the anatomy of the eye and basic skills in ophthalmic operations. For that reason, "Anatomy and examination of ocular trauma" is believed to be necessary and fundamental for this book series. Beyond this, familiarity with the emergency room and efficient protocol will be helpful for the doctors to give treatment in the first time, and it will also be an important part of this book series. Almost all the aspects and details of ocular trauma will be covered in this book series, including mechanical and non-mechanical ocular trauma. Special topics of complicated situations, such as ciliary body impairment, will also be introduced in this book series. Hopefully the readers will enjoy it and find it helpful for them to provide better care to the patients and save vision.

More information about this series at http://www.springer.com/series/15621

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Foreword

Although ocular trauma has been reported as the third most important cause of vision loss worldwide, it is the leading cause of monocular blindness, especially in the young group. Trauma occurrence shows high unpredictability, and ocular trauma represents a huge challenge for the treating ophthalmologist due to the severity of many of the injuries. Because of the extreme variations in how medical service is organized (infrastructure of the facility, training experience, equipment-availability, etc.), it is critical to unify and refine the criteria for ocular trauma treatment and enhance the emergency education of all ophthalmologists. The series of the books put forward a series of novel concepts, mechanisms, and technique about ocular trauma on the examinations, diagnosis, treatment, and rehabilitation.

Professor Hua Yan is the Chairman of the Department of Ophthalmology, and President of Tianjin Medical University, Member of the Medical Commission of the Association of National Olympic Committee, Vice President of the Asian Pacific Ophthalmic Trauma Society, and President of the Chinese Ocular Trauma Society. He has been contributing to the field of ocular traumatology for decades. In the past several years, he was invited by Springer to serve as the chief editor for a series of books on ocular trauma, collaborating with world-famous ophthalmologists. The titles include *Anatomy and Examination of Ocular Trauma*, *Ocular Emergency*, *Mechanical Ocular Trauma*, *Atlas of Ocular Trauma*, *Sports-related eye injuries, and Management on Complicated Ocular Trauma*. The edition on ocular emergencies won in 2019 the most outstanding book published by Springer, and the one on *Sports-related eye injuries* is the world's first professional work on the subject.

These high-quality books are well organized, providing comprehensive basic knowledge along with vivid clinical cases using high-definition photographs. These cases illustrate both those cases where consensus on ocular trauma management exists and where controversies persist. The series of the books put forward a series of novel concepts, mechanisms, and techniques about ocular trauma on the examination, diagnosis, treatment, and rehabilitation. Since personal wellness has attracted increasing attention from the general public in recent years, people participate in a variety of fitness activities and sports sometimes close to the realm of professional athletes, the risk of sustaining (eye) injuries has also increased. This calls for an adequate response from the medical community in terms of assessment and treatment. The book on *Sports-related eye injuries* is, therefore, timely not only for the sports enthusiasts to increase their awareness of injury prevention, but also

for ophthalmologists who treat patients with sports-related eye injuries. To improve the results in the treatment of these patients, the book named *Management on complicated ocular trauma* is in the process of being published.

This series of books on ocular traumatology is the first such book series in the world. This book series will be an invaluable resource for physicians in general and surgeons in particular to help them provide the best possible care to their patients and save the vision of every eye where salvage is possible, as well as advance the field of ocular traumatology worldwide.

October 7, 2021

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Foreword

It is my pleasure to write about your series of books written by you on *Anatomy and Examination in Ocular Trauma, Ocular Emergency, Mechanical Ocular Trauma, Atlas of Ocular Trauma, Sports-related eye injuries, and Management on Complicated Ocular Trauma.*

Ocular trauma (OT) is an important preventable cause of blindness and visual impairment over the world, with an incidence of estimated over 50 million annually, nearly one third of which could result in severe vision loss even blindness. As far as we know, ocular trauma occurs in combat, explosion, sports, working, traffic accident, home, fireworks, and other kinds of accident commonly due to the specific position of the eyes in our body. Although the whole society pays high attention to ocular trauma with tons of investigations and research devoted, prevention and treatment of ocular trauma are varied in different areas where doctors lack comprehensive training. Meanwhile, there is a short of frontier reference books published recently, hitherto, a series of books on ocular trauma could improve the global medical service of ocular trauma to a large extent.

Professor Hua Yan is the President of the Tianjin Medical University, Chairman of the Department of Ophthalmology at Tianjin Medical University General Hospital, Professor of Tianjin Medical University, Member of the Medical Commission of the Association of National Olympic Committee, Vice President of the Asia Pacific Ophthalmic Trauma Society, President of the China Ocular Trauma Society, and Executive Committeeman of the Chinese Ophthalmological Society. Yan has published over 200 book chapters, journal papers, and conference papers. His contributions have been vital to the development of ophthalmology, especially the ocular trauma, for decades. Professor Yan is capable of taking on the exciting challenge of inviting a group of world-renowned experts on ocular trauma to work on this important series of books about ocular trauma step by step, including *Anatomy and Examination of Ocular trauma, Ocular Emergence, Mechanical Ocular Trauma, Atlas of Ocular trauma, Sport-related Eye Injury and Management on Complicated Ocular Trauma*, which is the first and unique series of books for ocular trauma worldwide.

The series of the books give comprehensive introduction about ocular trauma systematically. The book named "*Anatomy and Examination of Ocular Trauma*" introduces anatomy, examinations, and symptoms of ocular trauma and presents an anatomy-based and practical book about ocular trauma. The book named "*Ocular Emergence*" provides symptom-based summarization of diseases in ocular emergency and facilitates the readers to

make the most appropriate decision on urgent ocular cases. The book named "*Mechanical Ocular Trauma*" addresses hot topics in mechanical ocular trauma and presents a balanced mix of consensus and controversies with advice about how to make appropriate decisions on the management. The book named "*Atlas of Ocular Trauma*" presents hundreds of comprehensive pictures of all types of ocular trauma to illustrate signs and examination of ocular trauma and explains the key points of treatment and surgical procedures. The book named "*Sport-related Eye Injury*" provides essential know-how and standard procedures for the management of sports-related eye injuries and discusses various sports-related eye injuries in detail on a symptom-based review. The book named "*Management on Complicated Ocular Trauma*" introduces the clinically unmanageable complicated ocular trauma with typical cases including signs, examinations, surgical procedures, and conservative management by experienced experts. The series of the books on ocular trauma illustrate a series of innovative concepts, mechanisms, and techniques on ocular trauma related ocular structure, examination, diagnosis, and treatment.

Professor Yan has accomplished the high qualified series of book on ocular trauma, which is the first and unique series on this topic. The books will be huge wealth for medical students and clinicians to help them give best care to the patients suffered from ocular trauma and rescue their vision at the most extent. Professor Yan' work contributes to the management and training on ocular trauma significantly.

October 7, 2021

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Preface

It is of great honor for me to rise to the challenge of inviting a group of experts to compile the comprehensive book on complicated ocular injury with great threaten to the vision of people at working age. Prior to this book, we have issued a series book on ocular trauma, which received high quality of appraisal over the world. However, there are various complicated ocular trauma being significant concerned associated with high morbidity and disability around the world, which attracts the ophthalmologists' attention in the clinic due to difficult management. Treatments to those injuries are hard, complicated, long-term, and costly. Hence, this book is aimed at illustrating the common complicated ocular trauma with typical signs, examinations, surgical procedures, and conservative management to clinical practitioners including the nurses, medical students, residents, fellows, and ophthalmologists, and helps them make the most appropriate decision on the diagnosis and management of such patients.

The overall organization of the book has been divided into 23 chapters encompassing the Complicated Corneal and Scleral Rupture, Traumatic Cyclodialysis, Traumatic Aniridia, Traumatic Dislocation of Lens, Intraocular hemorrhage, Open Globe Injury with Choroidal and Retinal Detachment, Choroidal and Retinal Detachment combined with Cyclodialysis in Open Globe Injury, Repairment of Traumatic Choroidal Tear, Complicated Ocular Trauma with Corneal Opacity, Traumatic Giant Macular Hole, Posterior Polar Ocular Perforating Injury, Giant Intraocular Foreign Body, Orbital Foreign Body, Traumatic Glaucoma, Traumatic Endophthalmitis, Permanent Silicon Oil Tamponade, Traumatic Optic Neuropathy, Severe Orbital Fracture, Traumatic Globe Luxation, Complicated Blepharal Trauma, Severe Ocular Chemical Injury, Ocular Laser Burns, Pediatric Ocular Trauma. This book consists of all types of complicated ocular trauma with brief case reports combined with the most comprehensive pictures. For each disease, a brief introduction, explanation as well as management are offered to the readers. With the illustrative figures and explanations, the goal of making the right diagnosis, offering the best advice or treatment to the patients, and understanding surgical procedures would be easily achieved. Hopefully, this book may help the readers to be fully prepared for the challenge of complicated ocular traumatic cases.

Tianjin, China — Hua Yan, MD, PhD

Contents

Complicated Corneal and Scleral Rupture

1

Kristina Stanfield and Bo Huang

Abstract

Complicated corneal and scleral injuries have been shown to be a significant proportion of annual emergency room visits worldwide. In this chapter, the types and definitions of these injuries were described. Furthermore, important clinical signs, examinations, diagnosis of these complicated ocular injuries, as well as their management and postoperative complications, were discussed in detail.

Keywords

Corneal laceration · Scleral laceration
Corneal rupture · Scleral rupture · Open globe injury · Corneal laceration repair · Scleral laceration repair

1.1 Introduction

A significant proportion of visits to the emergency room annually, eye injuries are far too common. One study showed an incidence of 4.49 open globe injury cases per 100,000 population in the USA from 2006 to 2014 (mean [SD] age of study participants, 37.7 [22.5] years; 75.3% male) [1]. Less wealthy and less educated individuals tend to take part in riskier activities, thus increasing their risk for eye injury [2, 3]. There is a 40–60% higher risk in nonwhites [4, 5], and there is increased risk with those that report dangerous self-behaviors like traffic violations, illicit drug and alcohol use, unemployment, and unsettle social environments [3, 6].

Blunt and sharp articles are frequently implicated, with the most common cause for ocular trauma due to blunt objects [7]. In decreasing order of frequency in the USA, rocks, fists, baseballs, lumber, fishing weights are responsible for the majority of ocular injury; in contrast, fists, wood branches, rocks, and champagne corks are the more common culprits in Hungary [7]. Assault, work-related events, sports, and recreational events [8–12], motor vehicle crashes, and falls also contribute a substantial portion of eye injuries [2, 5, 13]. Younger adults sustain open globe injury most commonly through assault and working with metal; falls are responsible for globe rupture in older age groups, with 82% reporting a history of previous intraocular surgery [14]. The cornea is involved in 92% of pediatric open globe injuries [7]. As can be surmised, globe rupture and perforating injuries notably result in poorer visual outcomes than eyes without such injuries. Work-related globe injuries tend to have a higher incidence of intraocular foreign bodies and cataracts when compared to those open globe injuries not occurring at work.

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The cornea is the most frequently involved tissue in ocular injuries, followed by the retina, vitreous, and sclera [7].

The majority of serious eye injuries sustained are by persons with no eye protection, meaning that most of these ocular insults are avoidable. Prevention is key to avoiding globe injury [15]. Since the introduction of seat belt laws, eye injuries have been reduced to 47–65% [13, 16, 17]. Polycarbonate shield wear has been shown to be protective during combat [18, 19] and many sport activities [20–22]. With the target population skewed, further educational efforts should continue to be concentrated on young males in high-risk occupations, as eye protection and educational programs have been shown to be successful in increasing eye protection compliance [18, 23].

1.2 Definition

Classification of traumatic globe injury can be stratified into the type of wound inflicted upon the eye. Penetrating injuries penetrate the eye but do not form an exit wound, and perforating injuries have both an entrance and exit wound.

Penetrating injuries usually partially cut or tear the surface of the cornea or sclera as they make contact with the globe. Sources of these injuries include Hymenoptera insect stings, repair, and maintenance work, wood chopping, machine use, simple instrument use, and falls. These injuries do not have an exit wound.

Perforating injuries completely cut or tear through the surface of the globe. Endophthalmitis follows such injuries that penetrate or perforate the globe, with a much higher infection rate than endophthalmitis after intraocular surgery. Risk factors include retained intraocular foreign body, rural setting at the time of injury, disruption of the crystalline lens, and delay in primary wound closer [7].

A third type of open globe injury includes contusion, where the globe directly or indirectly ruptures from pressure. Direct rupture occurs most commonly at the limbus, cornea, or sclera at the impact site. Indirect rupture results from redistributed force via impact on an incompressible globe. This occurs most frequently at the limbus and area of the sclera posterior to recti insertions, both of which are ruptured perpendicularly to the original line of contusion force; countercoup injury in a parallel line of force can cause posterior scleral rupture adjacent to the optic nerve. All of these locations are among the globe's thinnest areas.

Sometimes, corneal perforation can occur in predisposed eyes. Ehlers-Danlos is one such condition with the abnormal organization of collagen bundle crosslinking, predisposing the eye to injury with minor trauma [24].

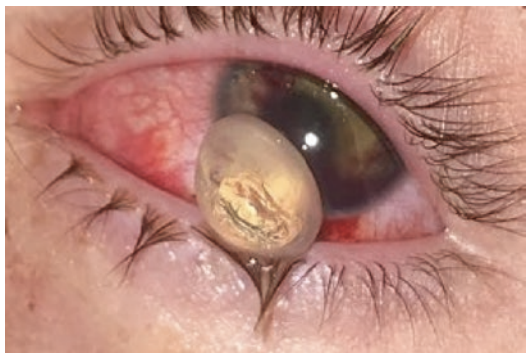
Eye injuries can be caused and complicated by intraocular foreign bodies, accounting for 18–41% of all open globe injuries [7]. Some inorganic metals like gold, silver, platinum, aluminum, and glass are inert substances, thereby causing little to no reaction if left undisturbed. Other metals such as lead and zinc can cause a chronic nongranulomatous reaction that is overall well tolerated by the eye. In contrast, iron has a high affinity for epithelial cells, and once ionized, can diffuse throughout the eye to deposit in an assortment of ocular structures, causing the condition known as *siderosis bulbi*. The trivalent form of iron, ferric, is not near as toxic as the bivalent ferrous form. Similarly, copper can also ionize and deposit in many ocular structures, with its preference being basement membranes; this condition is called *chalcosis*. In the core of golf balls are barium sulfate and zinc disulfide, both of which do not cause inflammation but are birefringent to polarized light [24]. Intraocular foreign bodies are identified frequently in combat ocular trauma and are found to be associated with more severe injuries [25].

Organic matter like cilia, grass clippings, tree, and bone causes a striking granulomatous reaction, sometimes carrying secondary microbial or fungal contamination into the eye.

1.3 Case

A 17-year old male was playing a drinking game that consisted of hammering nails into a stump while intoxicated. He presented to the emer-

gency department the following morning with his mother, complaining of eye pain and decreased vision. His injured eye had light perception vision with a peaked pupil. The eye was palpated and found to be soft. On exam, there was an inferonasal limbal rupture with protrusion of natural crystalline lens. The anterior chamber was diffusely shallow and collapsed with heme stranding and iris peaking. The patient was taken urgently to the operating room for lens removal with primary open globe repair. In the operating room, the lens was resected in one piece while carefully repositioning prolapsed uveal tissue back into the eye using viscoelastic agents. An inferior conjunctival peritomy revealed an underlying scleral laceration inferonasal. Interrupted 9-0 nylon suture was used to close the scleral wound using partial-thickness bites, and 10-0 nylon suture was used to close the corneal wound. All knots were rotated and buried. The conjunctiva was reapproximated at the limbus using 8-0 vicryl suture. It was decided to leave the patient aphakic and implant an intraocular lens in subsequent surgery in the future.



1.4 Important Signs, Examinations, Diagnosis, Surgical Procedures and Skills, or Postoperative Treatment for Complications

According to recent epidemiological studies, risk factors include male gender [11], notably those aged in the fourth decade of life [1, 26]. Most serious eye injuries are sustained by persons that fail to wear sufficient eye protection during high-risk situations that occur at home or at work, with construction considered one of the most high-risk professions. Substance abuse has also been shown to contribute as a factor in eye injury from trauma.

Prevention is the most successful means to avert ocular injury. Primary prevention includes wearing correct eye protection such as polycarbonate lenses while engaging in visually threatening actions.

Cornea and scleral ruptures commonly present to the emergency room after the initial injury, and a thorough history is the first step in evaluation [27]. Ordinarily, patients will report pain, blurred or double vision, or foreign body sensation in the area of injury. It is important to note the time of injury. Mechanism of injury is important to ascertain as well, as it will guide the surgical plan. One should suspect foreign bodies and their potential for ocular toxicity; most intraocular foreign bodies reside in the posterior segment, which can possibly necessitate the need for a vitrectomy. The amount of energy transferred during the injury may decide how much damage the globe has endured, with rupture extending externally or internally. Inquire about prior vision and if there has been a decline; it is important to take note of amblyopia and glaucoma. Ask about the use of safety or prescription glasses worn during injury. Relevant ocular surgical history should be elicited, as a patient with a prior corneal transplant may require a new cornea from a transplant bank before proceeding to the operating room for repair. A patient with previous incisional keratotomy or globe repair is much more prone to rupture along with these areas of previous incisions. Medical history, including medications, allergies, prior reaction to anesthesia, and timing of last meal, should be elicited. Tetanus status should be determined and treated accordingly.

Examination of the traumatized eye should be assumed with care. Externally, the patient should be checked for any external facial or eyelid laceration as well as scattered debris that may warrant a search for an intraocular foreign body. Edematous eyelids can be cautiously retracted using Desmarres, taking care not to exert undue

pressure on the globe. Check visual acuity and pupils, taking care not to exert any extra pressure on the globe if rupture is suspected. Baseline best-corrected visual acuity is of the utmost importance in determining the prognosis of vision after globe repair in the setting of trauma. Pupils may signify direct injury to the optic nerve or deeper intracranial pathology. If a pupil is unable to be examined in the injured eye, the other pupil should be checked for a reverse afferent pupillary defect to gleam some insight into the injured eye's optic nerve function. Intraocular pressure can be cautiously assessed by gently palpating the globe with fingertips; while it is normally common practice to measure pressure using handheld tonometry in the emergency room setting, in the case of a suspected open globe, it is not necessary to quantify a numerical value for fear of expulsing intraocular contents. Epithelial defects can be easily seen by light reflex off the anterior cornea; abnormalities in this light reflex can point to epithelial or stromal loss from lack of normal high luster. Take note of any anterior segment trauma, including conjunctival laceration, corneal abrasion, corneal foreign bodies, subconjunctival hemorrhage, shallow or collapsed anterior chamber, hyphema [28], iris peaking or irregularities, or focal lens opacification due to disruption. A peaked pupil may signify vitreous or uveal prolapse through the wound. Seidel testing should be employed to discover the depth of corneal laceration if one exists. If a wound has self-sealed, gonioscopy can be performed. The eye should be dilated and the posterior segment examined for intraocular foreign body. In the posterior segment, look for vitreous and retinal hemorrhages as well as retinal tears. If there is no view posteriorly due to obscured media, a CT scan can aid in the search for an intraocular foreign body. MRI should be avoided in the initial management if the metallic intraocular foreign body is suspected [29–31]. Ultrasonography is generally avoided in the acute setting as well in order to not dislodge intraocular contents.

Metallic intraocular foreign bodies are most common [32, 33], and usually are extricated to avoid toxicity. Siderosis results from iron compounds and copper compounds can lead to chalcosis. Zinc and aluminum should be removed as well. Inert substances like glass, stone, sand, and plastic can be left alone without removal, as they do not normally incite an inflammatory reaction.

After inspection and examination, the surgeon must formulate a surgical plan. The primary outcomes of closing the laceration include reestablishment of vision, normalizing anatomic relationships, preventing infection, and halting secondary disease processes like glaucoma and amblyopia. When discussing the plan for surgical repair, these goals must be stressed to the patient. The most important prognostic indicator for vision is preoperative visual acuity testing after injury.

If there is a rupture that is indeed found on exam or imaging, the globe should be urgently closed. A small perforation less than 2 mm in diameter can sometimes be closed with tissue glue, such as cyanoacrylate, which works by polymerizing on contact with liquid. Tissue glue only sticks to dry lacerations free of epithelium, and it can be applied to the globe using a syringe, a drip technique, or by applying glue to the opposite side of a polyethylene disk placed on the end of the applicator stick. A therapeutic contact lens follows glue application to ease discomfort and avoid contact of glue with tarsal conjunctiva. Because tissue glue has antimicrobial action, corneal infection in rare; however an adjacent inflammatory infiltrates can be noted. Constant broad-spectrum topical antibiotic drops should be employed while tissue glue and contact lens are in place. If the tissue glue is applied generously to a descemetocele, the glue may come off while stuck to underlying corneal tissue.

More extensive lacerations should be taken urgently to the operating room for exploration and repair. General anesthesia is the preferred method for sedation as a corneoscleral laceration is a contraindication for retrobulbar anesthesia. If local injection into the retrobulbar or peribulbar space occurs, then intraocular pressure may increase and thus extrude intraocular contents. A nondepolarizing muscle relaxant is also used to decrease the risk of extraocular muscle contraction, which may further increase intraocular pres-

sure. Postoperatively, the patient must be very carefully extubated after general anesthesia to avoid bucking and emesis, as these events too can increase intraocular pressure.

When prepping and draping the eye preoperatively, care must be taken to avoid undue pressure on the globe. An adhesive barrier is used to expose the eye and shield the lashes from the operating field. A wire lid speculum can be placed over this barrier, avoiding pressure on the globe during application. Irrigation is favored over povidone-iodine solution after the speculum has been positioned. If the lid tissue is macerated or a speculum is unable to provide adequate exposure of the field, 4-0 silk traction sutures may be placed through the lids. Bridal sutures through the extraocular muscles are generally avoided as they can cause anterior chamber narrowing and tissue incarceration from external pressure. The wound should be cultured and then surveyed to remove any foreign bodies.

Once under the microscope, the laceration should be explored fully yet gentle enough not to expel any further intraocular contents. Corneoscleral lacerations extending from cornea past limbus into sclera should be tracked posteriorly (but not beyond the equator) to view their extent. A conjunctival peritomy can be useful, sometimes encompassing the full 360-degrees, in order to find the furthest course of the laceration. The limbus should be first realigned using 8-0 or 9-0 nylon sutures, taking care to reposition any uvea that has prolapsed; this task can be aided by the use of a viscoelastic agent and a cyclodialysis spatula. The laceration should be examined for vitreous prolapse or foreign bodies. Vitreous prolapse is recognized using a dry cellulose sponge, gently lifting upwards from the wound and trimming flush with sclera in order to avoid further traction on the remaining vitreous.

Corneal wound edges must be approximated using suture to facilitate watertight closure; corneal tissue is avascular and will not stretch or replicate, so correct alignment of wound edges is paramount. Although likely unavoidable, astigmatism, and postoperative scarring should be minimized at best while restoring the contour and integrity of the corneal tissue. Monofilament 10-0 nylon suture on a fine spatula microsurgical needle is most commonly used for corneal sutures. Bicurved needles are employed with small or large radii of curvatures; the former helps to pass little deep bites centrally while the latter is used in the periphery for greater lengthier bites [36]. Corneal sutures should not be placed full thickness in order to minimize the risk of endophthalmitis due to direct microbial tracking into the anterior chamber; instead, the suture should be placed at 90% stromal depth equally on both sides of the wound edges. If the sutures are placed more superficially, they will result in wound gape. An unequal approximation may allow the wound edges to override. Avoid suture through the central visual axis but if necessary, use little small bites to limit scarring. If possible, avoid corneal tissue removal; this can unnecessarily create force on non-elastic corneal tissue through the use of tight sutures (Fig. 1.1).

Place corneal sutures by inserting the needle tip perpendicular to the corneal surface, then rotate the needle along its curve through the wound edges using a twisting wrist motion, and finally exit again perpendicular to the other side of the wound. The needle passes, and depths should be equal on either side of the wound edges in order to achieve an ideal approximation. If a linear wound is found to have a perpendicular component, it must be closed first before the shelved portion of the laceration. The suture placement should enter and exit equidistant to the posterior (not anterior) aspect of the wound in order for the wound edges to approximate appropriately and not override their margins. The perpendicular lacerations are repaired first using interrupted sutures in order to reform the anterior chamber; once in place, the shelved areas of the laceration will oppose automatically, thus requiring less suture and minimizing tension. Because it can be difficult to evaluate the correct tension for initial sutures, slipknots like the granny-style slipknot can be loosened or tightened after all the sutures are in place. The granny-style slipknot allows for controlled tension by pulling one end vertically while the other is pulled horizontally.

With jagged lacerations, slipknot sutures are used to close each linear segment first in the

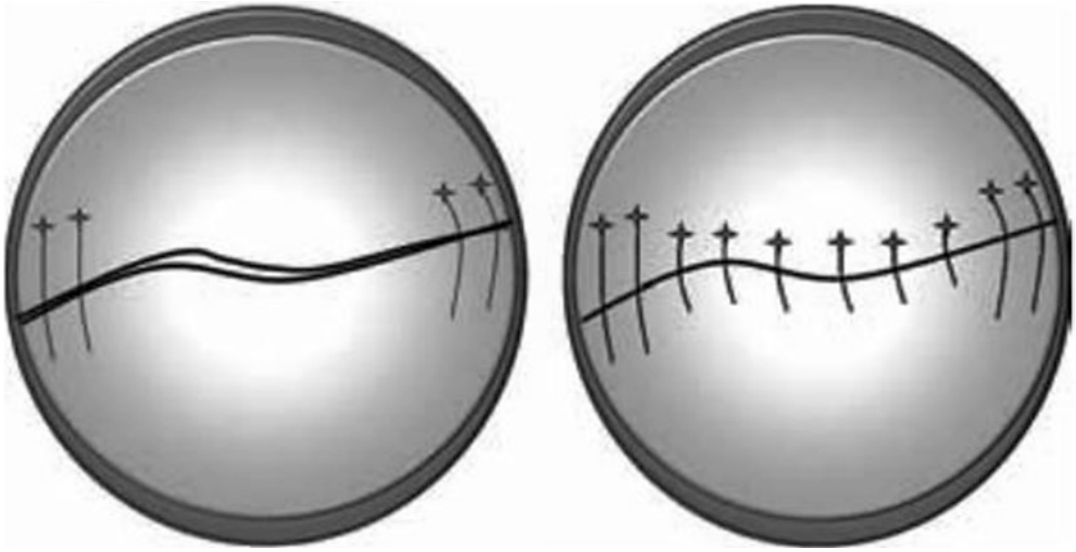


Fig. 1.1 Partial-thickness corneal suture placement. Peripheral corneal sutures should be long, while central sutures are small and deep

hopes that the apices might self-seal. A mattress suture can be employed to close the apex if necessary and left to degrade over time. A purse-string suture can be useful to close a stellate laceration by passing intralamellar sutures through a partial-thickness incision made by a guarded diamond knife (Fig. 1.2).

Sometimes, the laceration cannot be approximated due to tissue loss. In nonnecrotic stroma measuring less than 5 mm, a circular autograft from the peripheral cornea measuring 1 mm larger than the area of tissue loss may be trephined and placed over the deficit using interrupted 10-0 nylon sutures. If there is not enough peripheral cornea to graft, donor cornea or sclera should be used. In the case of graft loss in traumatized eyes with a history of keratoplasty, a donor cornea will be needed. Peripheral compression sutures can be passed in the corneal periphery in order to steepen the central cornea and thus decrease postoperative astigmatism. The ends of all knots are trimmed short and rotated posteriorly into the stroma to avoid discomfort to the tarsal conjunctiva.

Injured sclera should be reapproximated using 7-0 or 8-0 polyglactin (Vicryl, Ethicon) absorbable suture, making sure to place suture as soon as a fresh area of laceration is exposed but before the area distal to the laceration is explored; this "close-as-you-go" technique will help steady the globe and prevent further uveal and vitreous prolapse. Scleral sutures should begin anteriorly and work their way posteriorly. Conjunctiva and Tenon's capsule should only be opened enough to visualize the laceration and not be extensively dissected. When the laceration is found to extend past an extraocular muscle, the muscle can first be secured using a double-armed 6-0 polyglactin suture then disinserted to allow for more exposure of the wound. After the laceration is reapproximated, the muscle can then easily be reattached at its insertion site. If the wound is found to extend posterior to the equator and cannot be safely appreciated, it is best to let that portion of the wound heal secondarily and leave it unsutured (Fig. 1.3).

Some scleral defects cannot be closed primarily, thus necessitating the need for a scleral patch graft. The most commonly used, the homologous sclera is stored in glycerin or alcohol, or it is frozen in antibiotic solution. Glycerin-dehydrated and alcohol-fixed sclera can be used up to 1 year, while frozen sclera can be used up to 3 months from their dates of preservation. Before placing a scleral graft, episcleral, and choroidal tissues are removed, and the scleral defect is exposed by conjunctival peritomy and dissection. Permanent

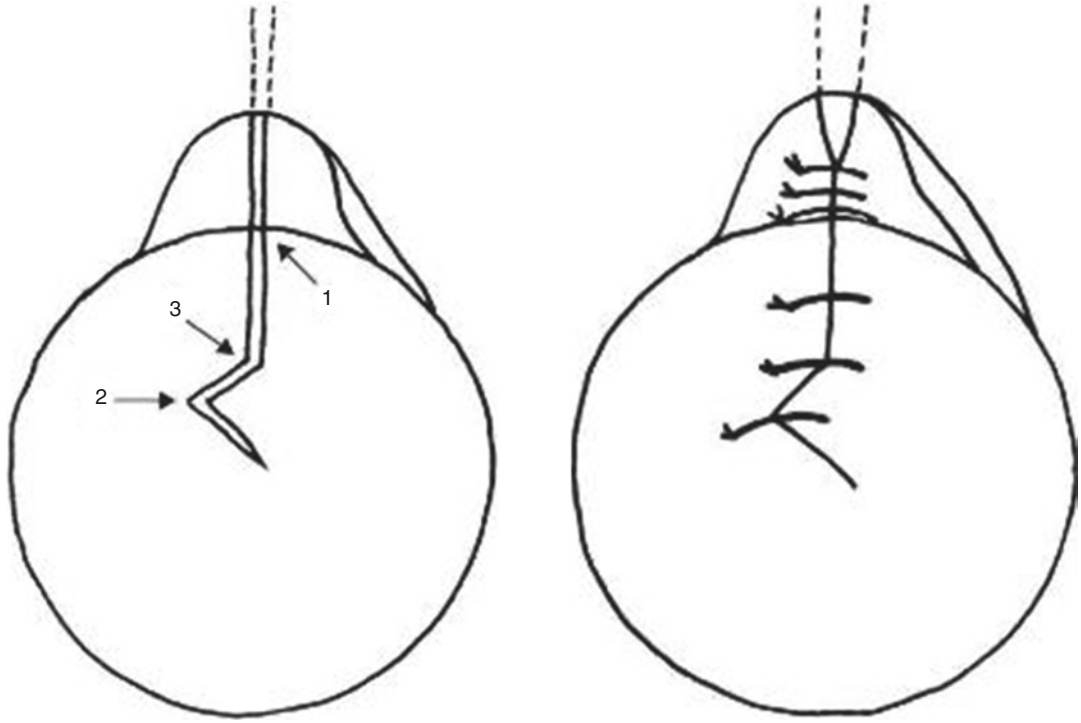


Fig. 1.2 Corneoscleral wound repair. The limbus (1) is approximated first followed by corneal then scleral closure (2, 3)

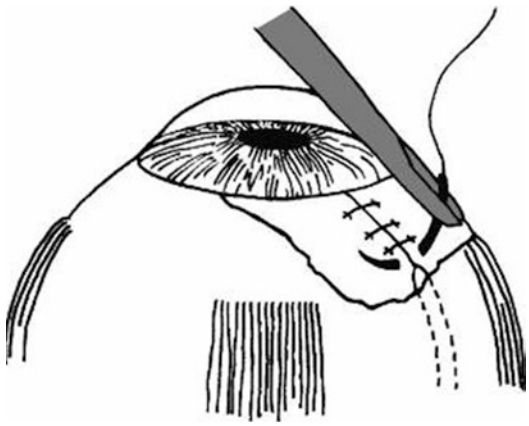


Fig. 1.3 Scleral wound repair using the "close-as-you-go" technique. Limited dissection through conjunctiva and Tenon's capsule allow enough visualization of the laceration barely to prevent further intraocular contents from expulsing while providing overall stabilization to the globe

sutures (silk, Dacron) are preferred to allow for extending wound support while healing. The graft should overly the defect by a few millimeters. Other scleral grafts can be fashioned from conjunctiva or Tenon's capsule, tarsoconjunctival flap, autologous sclera, fascia lata or periosteum, or split-thickness dermis.

If there is an intraocular foreign body, vitreous hemorrhage, or retinal detachment, a vitrectomy may be required. A pars plana approach is generally employed.

A paracentesis track can be made through the anterior chamber to check for leaks and verify watertight wound closure using gentle pressure and Seidel test if needed.

Endophthalmitis is of paramount concern, so prophylaxis should be initiated. At the termination of the procedure, periocular subconjunctival injections of vancomycin 25 mg or ceftazidime 100 mg should be administered. Because the suspicion of postoperative endophthalmitis is extremely low, subconjunctival dexamethasone 12–24 mg is often used. Intraocular injections of vancomycin 1 mg/0.1 mL or ceftazidime 2 mg/0.1 mL can also be used. Systemically, vancomycin 1 g intravenously every 12 h or ceftazi-

dime 1–2 g intravenously every 8 h can be used for a total of 4 days. Topically, vancomycin 50 mg/mL, ceftazidime 50 mg/mL, and a cycloplegic should be used once an hour for the first 48 h, then tapered pending clinical course. A topical steroid like prednisolone acetate 1% four times daily is often added.

The patient should be instructed on the strict usage of a Fox shield over the eye at bedtime for the first few weeks postoperatively, with the understanding to use safety glasses made of thick polycarbonate lenses to protect the eye during day further.

Corneal sutures are generally removed 3 months postoperatively in the adult patient and weeks later in the pediatric patient due to quicker wound healing. Loose sutures should be removed to prevent epithelial breakdown with a subsequent ulcer or abscess formation. If there is vascularization under the suture, it should be removed. Alternate sutures are first removed, followed by topical antibiotics four times daily for 5–7 days.

Best visual acuity is first undertaken by spectacle correction. Corneal topography can help to find tight sutures causing residual astigmatism. Once healed, compressive sutures are removed to help with astigmatism. If spectacles cannot fully correct vision, large rigid contact lenses with high oxygen permeability are used to treat irregular astigmatism; soft contact lenses are generally avoided due to the risk of vascularization of the corneal scar. As a last resort, penetrating keratoplasty may be warranted for further visual rehabilitation.

The repaired globe is serially examined postoperatively. Visual acuity, pupils, and intraocular pressure are measured. Seidel test is used to note any leakage from the wound. Ultrasonography can aid in visualizing the posterior segment if the media is not transparent.

1.5 Personal Experience or Matters That Need Attention

Although the occurrence of postoperative endophthalmitis is fairly low, it should be treated with intravitreal antibiotics as outlined above, and cultures taken from the operating room should be followed for microbial growth.

With corneoscleral rupture comes other traumatic injuries to the globe. Hyphemas are possible after postoperative repair, and although they are resorbed in the first week, they can elevate intraocular pressure from trabecular meshwork blockage. This pressure spike can be managed with topical acetazolamide, beta-blockers, and alpha agonists. Both topical and systemic steroids can further help to calm inflammation. If medical management does not work, the patient may need to be taken back to the operating room for anterior chamber washout in order to prevent corneal blood staining and endothelial damage.

On the other hand, the intraocular pressure may be low, which most often indicates a wound leak. This can be confirmed by the Seidel test and treated with pressure patching, aqueous suppressants, and a bandage contact lens. Cyanoacrylate tissue glue can be used but make sure tissue is dry and is not directly applied over suture, as the suture-glue bond is permanent. If these measures do not stop the leak, again, the patient may need to return to the operating room for additional 10-0 nylon sutures. Glaucoma can result from peripheral anterior synechiae into the leaking wound if this complication is not recognized early.

Epithelial downgrowth is essentially proliferating epithelium tracking into the anterior chamber through a previous wound. Spots of epithelium on the iris stroma will burn white with low-power argon laser. If advanced, the epithelium may need to be removed by surgery.

Sympathetic ophthalmia is a bilateral, diffuse, granulomatous, T-cell mediated uveitis that can occur anywhere from 2 weeks to many years after uveal prolapse or incarceration, most commonly secondary to perforating or penetrating ocular injury. Visual prognosis is more promising with early enucleation 3–6 months after injury.

Phacoantigenic endophthalmitis is a zonal granulomatous inflammation secondary to a ruptured lens capsule. Activated immune cells can clog the trabecular meshwork, causing glaucoma. Angle recession glaucoma from trauma can develop from posteriorly displaced iris root and

inner pars plicata from a laceration. When the entire pars plicata is dislodged from the scleral spur, a cyclodialysis cleft can result. An iridodialysis can result from a tear at the iris root.

The posterior segment is often involved with globe trauma. Retinal detachments can develop following contusion, as well as macular holes, likely because of the inelastic characteristic of the vitreous that does not allow for stretching. Chorioretinitis sclopetaria manifests following shockwave-like penetrating (not perforating) globe damage. Choroidal hemorrhage and rupture can occur from either indirect or direct trauma. In the retina, commotion retinae and retinal hemorrhages can occur at the site of injury. The optic nerve can endure avulsion, hemorrhage, and edema.

Foreign body granulomas can form around intraocular foreign bodies that are lodged within the eye at the time of initial injury; this can be avoided by removal.

1.6 Specific Challenges of Corneal and Scleral Rupture

Open globe repair should be undertaken within 24–48 h upon presentation in order to salvage vision and minimize infection risk. Often patients present for treatment after a substantial portion of time has passed since the initial injury. It can be a challenge to navigate a macerated globe back to its original anatomical structure, and not uncommonly, primary enucleation must be undertaken.

Often the traumatized globe has multiple other eye injuries that need to be mended, usually at a later date after the initial repair and most times requiring multiple surgeries. Postoperatively, the repaired globe is examined posteriorly using B-scan to look for retinal detachments or choroidal damage that may benefit from repair in the operating room. Secondary cataracts can develop either from direct lens trauma or from intraoperative repair of retinal damage; cataract extraction may need to be performed if visually significant. Secondary glaucoma can develop from trabecular meshwork damage. Medical management using topical ocular antihypertensives is usually tried first before proceeding to surgical management by tube shunt or filtering surgery.

Visual prognosis following repair is always guarded. Prognostic factors indicating good visual prognosis (as defined as visual acuity 20/60 or better) include presenting acuity after injury 20/200, wound location anterior to pars plana and measuring 10 mm or less, intraocular foreign body in the posterior segment, and sharp mechanism of injury [26, 34]. Age <5 years appears to be an independent risk factor for poorer visual outcome [35].

References

1. Mir TA, Canner JK, Zafar S, Srikumaran D, Friedman DS, Woreta FA. Characteristics of open globe injuries in the United States from 2006 to 2014. JAMA Ophthalmol. 2020;138(3):268–75.
2. Liggett P, Pince K, Barlow W, Ragen M, Ryan S. Ocular trauma in an urban population. Ophthalmology. 1990;97:581–4.
3. Glynn R, Seddon J, Berlin B. The incidence of eye injuries in New England adults. Arch Ophthalmol. 1988;106:785–9.
4. Katz J, Tielsch J. Lifetime prevalence of ocular injuries from the Baltimore Eye Survey. Arch Ophthalmol. 1993;111:1564–8.
5. Tielsch J, Parver L, Shankar B. Time trends in the incidence of hospitalized ocular trauma. Arch Ophthalmol. 1989;107:519–23.
6. Negrel A, Thylefors B. The global impact of eye injuries. Ophthalmic Epidemiol. 1998;5:143–69.
7. Kuhn F, Pieramici DJ. Ocular trauma principles and practice. Thieme; 2002.
8. May DR, et al. The epidemiology of serious eye injuries from the United States Eye Injury Registry. Graefe's Arch Clin Exp Ophthalmol. 2000;238(2):153–7.
9. Karlson T, Klein B. The incidence of acute hospital-treated eye injuries. Arch Ophthalmol. 1986;104:1473–6.
10. Schein OD, Hibberd PL, Shingleton BJ, et al. The spectrum and burden of ocular injury. Ophthalmology. 1988;95:300–5.
11. Bauza AM, et al. Work-related open-globe injuries: demographics and clinical characteristics. Eur J Ophthalmol. 2013;23(2):242–8.
12. White M, Morris R, Feist R, Witherspoon C, Helms H, John G. Eye injury: prevalence and prognosis by setting. South Med J. 1989;82:151–8.
13. Chapman-Smith JS. Eye injuries produced by vehicle safety glass. N Z Med J. 1978;88:239.
14. Beshay N, et al. The epidemiology of open globe injuries presenting to a tertiary referral eye hospital in Australia. Injury. 2017;48(7):1348–54.

15. Abbott J, Shah P. The epidemiology and etiology of pediatric ocular trauma. Surv Ophthalmol. 2013;58(5):476–85.
16. Cole MD, Clearkin L, Dabbs T, Smerdon D. The seat belt law and after. Br J Ophthalmol. 1987;71:436–40.
17. John G, et al. Field evaluation of polycarbonate versus conventional safety glasses. Southern Med J. 1988;81(12):1534–6.
18. Belkin M, Treister G, Dotan S. Eye injuries and ocular protection in the Lebanon War, 1982. Isr J Med Sci. 1984;20:333–8.
19. Thomas R, et al. Ocular injury reduction from ocular protection use in current combat operations. J Trauma Injury Infect Crit Care. 2009;66.
20. Easterbrook M. Eye protection in racquet sports. Curr Ther Sports Med. 1990;2:356–62.
21. Pashby T, Pashby R, Chisholm L, Crawford J. Eye injuries in Canadian hockey. Can Med Assoc J. 1975;113:663.
22. Pashby T. Eye injuries in Canadian hockey. Phase II. Can Med Assoc J. 1977;117:671.
23. Desai P, et al. Incidence of cases of ocular trauma admitted to hospital and incidence of blinding outcome. Br J Ophthalmol. 1996;80(7):592–6.
24. Yanoff M, Sassani JW. Ocular pathology. Elsevier; 2020.
25. Justin GA, et al. Intraocular foreign body trauma in operation Iraqi freedom and operation enduring freedom. Ophthalmology. 2018;125(11):1675–82.
26. Mohan S, et al. Repair of corneoscleral perforations. Delhi Ophthalmological Soc Times. 2008;14(2).
27. Krachmer JH, et al. Cornea. Fundamentals, diagnosis and management. Elsevier Mosby; 2005.
28. Maltzman B, Pruzon H, Mund M. A survey of ocular trauma. Surv Ophthalmol. 1976;21:285–90.
29. Loporchio D, et al. Intraocular foreign bodies: a review. Surv Ophthalmol. 2016;61(5):582–96.
30. Patel SN, et al. 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.
31. Dunkin JM, et al. Globe trauma. Semin Ultrasound CT MRI. 2011;32(1):51–6.
32. Baillif S, Paoli V. Open-globe injuries and intraocular foreign bodies involving the posterior segment. Journal Français D'Ophtalmologie. 2012;35(2):136–45.
33. Yigit O, et al. Foreign body traumas of the eye managed in an emergency department of a single-institution. Turk J Trauma Emerg Surg. 2012;18(1):75–9.
34. Zhang Y, et al. Intraocular foreign bodies in China: clinical characteristics, prognostic factors, and visual outcomes in 1421 eyes. Am J Ophthalmol. 2011;152(1).
35. Rostomian K, Thach AB, Isfahani A, Pakkar A, Pakkar R, Borchert MJ. Open globe injuries in children. JAA-POS. 1998;2(4):234–8.
36. Rowsey JJ, Hays JC. Refractive reconstruction for acute eye injuries. Ophthalmic Surg. 1984;15:569.

Traumatic Cyclodialysis

2

Yujie Yan and Zhijun Wang

Abstract

Traumatic cyclodialysis is a condition in which the longitudinal fibers of the ciliary muscle are disinserted from the scleral spur, creating an abnormal communication between the anterior chamber and the suprachoroidal space by different kinds of trauma. This chapter specifically focuses on its definition, epidemiology, pathogenesis, clinical manifestations, investigations, and managements. This chapter also provides clinical cases of complicated traumatic cyclodialysis.

Keywords

Traumatic cyclodialysis · Clinical manifestation · Management

2.1 Introduction

Cyclodialysis is a condition in which the longitudinal fibers of the ciliary muscle are disinserted from the scleral spur, creating an abnormal communication between the anterior chamber and the suprachoroidal space, which leads to chronic ocular hypotony, corneal edema, shallowing of the anterior chamber, cataract, choroidal effusion, optic nerve edema, retinal and choroidal folds, hypotonus maculopathy, retinal pigment epithelium (RPE) atrophy and loss of vision [1]. It usually occurs due to ocular trauma or as complications following ophthalmic procedures, which involves iris manipulation such as extracapsular cataract surgery, phacoemulsification, intraocular lens insertion, after phakic IOL removal and displacement of an angle supported anterior chamber IOL etc [2]. Classically, Gonioscopy remains the gold standard for the identification of cyclodialysis clefts; However, ultrasound biomicroscopy [UBM], anterior segment optical coherence tomography [OCT], magnetic resonance imaging [MRI] have proven to be effective in diagnosing as well. Although medical treatment can help to close small clefts, laser treatment or surgery is usually needed to seal the fistula.

This chapter will specifically focus on traumatic cyclodialysis, including its definition, epidemiology, pathogenesis, clinical manifestations, investigations, and management.

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2.2 Definition and Epidemiology

Traumatic Cyclodialysis is defined as longitudinal fibers of the ciliary muscle disinserted from the scleral spur, as a result of ocular trauma. It can be accompanied by traumatic hyphema, lens dislocation, traumatic mydriasis, iridocyclitis, and with severe vitreous or chorioretinal pathology [3]. Although cyclodialysis has been described after penetrating ocular trauma [4], most of the cases are followed by blunt ocular trauma, with the reported rates ranging between 1 and 11% [5]. It is more frequent in men, who are more prone to suffer trauma. A variety of agents like balls, stones, punches, airbags, firecrackers and elastic cords have been reported to cause cyclodialysis [1].

2.3 Pathogenesis

Cyclodialysis results from momentary axial compression and rapid equatorial expansion that stretches the ocular tissue, leading to the separation of the meridional ciliary muscle fibers from their attachment in the scleral spur [6]. The detachment of the ciliary body from the scleral spur seems to be the most common mechanism for persistent ocular hypotension after blunt trauma. There does not seem to be a preferential location for cyclodialysis, superior and inferior locations have all been reported.

The direct communication between the anterior chamber and the ciliochoroidal space allows an unrestricted flow from the anterior chamber to the Suprachoroidal space. The choroidal detachment thus produced would decrease the production of aqueous humor in the ciliary epithelium. All these mechanisms contribute to hypotony. However, the intraocular pressure drop is not proportional to the cleft size, which indicates that factors such as decreased aqueous production might also influence the outcome [7].

2.4 Clinical Manifestations

The ocular hypotony syndrome is considered the most important and devastating clinical feature of traumatic cyclodialysis. Ocular hypotony is defined as an IOP of 5 mmHg or lower. Clinical manifestations of chronic hypotony include corneal edema, shallowing of the anterior chamber, cataract, choroidal effusion or detachment, retinal and choroidal folds, optic disk swelling, venous tortuosity, and maculopathy. All these conditions may lead to visual loss, which may become permanent if the hypotony is not resolved promptly. Angular recession is accompanied by traumatic cyclodialysis in most patients. However, secondary open-angle glaucoma is not frequent. A series even found that the presence of a cyclodialysis was a protective factor against the development of glaucoma [8].

2.5 Investigations

Locating the cleft is essential for treatment. Gonioscopy has been considered the gold standard for the diagnosis of cyclodialysis. However, it may not be an optimal choice when there are corneal folds, hazy media, or a shallow anterior chamber. Moreover, microclefts and oblique channels may be particularly difficult to detect using gonioscopy. It is reported that gonioscopy will open the cleft and cause a transient flow of aqueous into the suprachoroidal space, resulting in post gonioscopy hypotony and inducing a significant reduction of IOP and the appearance of visible folds in Bowman's membrane [9].

UBM is considered more useful and sensitive in localizing, measuring, and monitoring cyclodialysis compared with gonioscopy. It allows a cross-sectional image of the cleft (Fig. 2.1) and

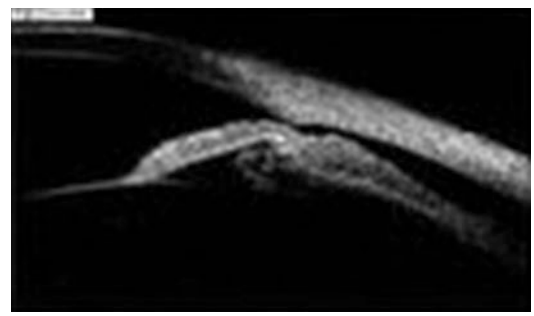

Fig. 2.1 A cross-sectional UBM image of traumatic cyclodialysis, showing ciliary muscle disinserted from the scleral spur, and communication between the anterior chamber and the suprachoroidal space

has the ability to detect when there are corneal folds, hyphema, a shallow anterior chamber or iris bowing. It also has the ability to detect tractional ciliary body detachment, ciliary body atrophy, suprachoroidal effusion, which is an essential tool in the differential diagnosis of ocular hypotony.

OCT is a noninvasive, painless, noncontact technique that provides reproducible images of the anterior segment and can be used in the diagnosis of cyclodialysis as well. It is performed in an upright position (while UBM is usually performed in a supine position), so the findings may more likely reflect gonioscopic and slit-lamp biomicroscopic clinical findings. However, the structure posterior to the iris is not visible with OCT, and traditionally its sensitivity has been considered inferior to UBM. Other ancillary techniques such as transillumination [10], magnetic resonance imaging [11] have also been reported but not widely used in clinical settings.

2.6 Management

Treatment of traumatic cyclodialysis should be started if hypotony is associated with morphological and functional complications. Several options have been used for treating this condition but should concerning about the numbers and the extent of the cleft to choose the optimal treatment.

Medical treatment should be employed first because some cyclodialysis resolves spontaneously, and a slight delay in IOP restoration does not change the visual prognosis. Medical treatment includes administrating atropine and steroids(topical and systemic) for several weeks and quick tapering of steroids. Steroids should not be used for extended periods, for it is believed that they can hinder the healing process that closes the fistula. For a small cleft, if ocular hypotony maculopathy persists after 2 months of stopping steroids, then Invasive or semi-invasive methods should be considered. However, for medium or large clefts, it is recommended to take the invasive methods with no delay.

If the cleft is a small cleft (<3 clock-hours), a laser should be tried. Argon laser is always useful when the cleft is easy to locate. The laser should be applied on both sides of the cleft, and bubble formation is considered a sign of good tissue response. Zeiss four-mirror gonioscopy is recommended in order to deepen the angle. Pilocarpine and viscoelastic are considered able to open the cleft and make Argon laser photocoagulation easier. Swelling of the choroid following laser treatment closes the cleft. Nd-YAG laser and laser endo-photocoagulator have also been used. If the cleft cannot be located, transscleral methods (such as transscleral diode laser) allow treating a wider area. Diathermy is not recommended because it could damage the lens and induce scleral ectasia [12]. Cryotherapy has shown only modest results, and it induces severe inflammation, so it should only be used when the laser is not available or preferably used combined with a gas injection or vitrectomy. If medical and laser treatment have failed in small clefts, direct cyclopexia should be performed(illustrated in the coming paragraph).

If medical treatment is not effective in medium-size clefts (3–6 clock-hours), direct cyclopexia should be considered. This procedure starts with a dissection of limbal based sclera lamella, with the thickness of 1/2–2/3 of the total sclera thickness and 3–3.5 mm behind the limbus (Fig. 2.2); then penetrate the sclera 1.5 mm behind the limbus (Fig. 2.3)and finally, suturing the insertion of the ciliary muscle to the scleral spur with 10/0 nylon. Locate the cleft and determine its extension before surgery is crucial in this procedure. However, "real time" locating the cleft during surgery and check the effect after suturing using a gonioscopy or a Goldmann

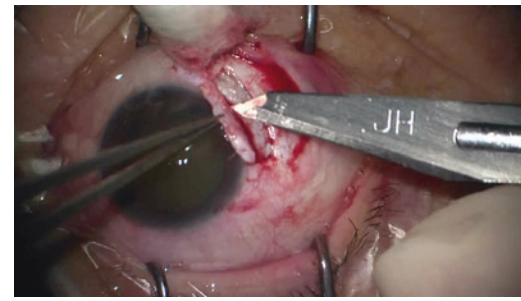

Fig. 2.2 Dissection of limbal based sclera lamella, with the thickness of 1/2–2/3 of the total sclera thickness, and 3–3.5 mm behind the limbus

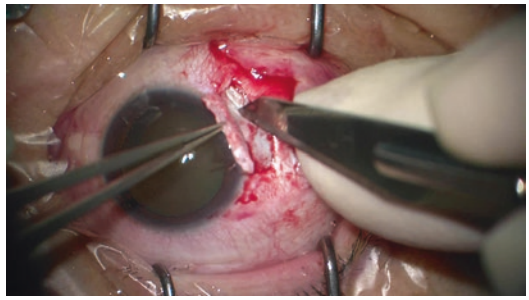

Fig. 2.3 Penetrating the sclera 1.5 mm behind the limbus

three-mirror contact lens is even more important. To locate 1–2 mm beyond the area of cyclodialysis is suggested. It can be challenging to suture a hypotonic eye. Using an anterior chamber viscoelastic or posterior infusion cannula to restore ocular pressure can sometimes be useful. Potential complications include intraocular hemorrhage, endophthalmitis, cataract, vitreous loss, retinal detachment, wound dehiscence, and secondary glaucoma due to peripheral anterior synechiae. For multiple or very extensive clefts, direct cyclopexy is not recommended because dissection of an extensive scleral lamella could affect the anterior segment. Closure of the cleft is followed by an IOP spike. It is considered to be the result of the restoration of aqueous humor production by the ciliary body with incomplete recovery of trabecular meshwork function. IOP returns to normal after a few hours, and this spike occurs in more than half the patients [13]. This spike can be controlled with topical medication except for miotics for they could lead to reopening of the cleft.

Vitrectomy with gas tamponade or silicone oil seems to be the safest option in clefts with a bigger size of (>6 clock-hours), or multiple medium-size clefts, or clefts with various retinal or choroid damage that need vitrectomy as well. Vitrectomy may also be necessary if direct cyclopexy is not successful in small or medium-size clefts. Cryotherapy can be accompanied by vitrectomy to facilitate the closure rate. Endoscopic-guided suture of the ciliary body detachment can also be performed though it is difficult to separate the effects of gas and the suture, as gas tamponade by itself is an effective method to treat cyclodialysis.

Other options have been reported. One of them is Internal tamponade using a capsular tension ring (CTR) or an intraocular lens (IOL) when cataract surgery is necessary. An IOL is inserted in the sulcus and carefully oriented until one of the haptics faces the site of the cyclodialysis. If the cleft is the extent, placing a CTR in the sulcus would be a better option. The scarring process induced by postoperative inflammation and a moderate internal compression by the haptic or the CTR, results in closure of the fistula. However, long-term outcomes are still uncertain. Potential risks include ciliary body damage, erosion, hemorrhage, and pain from the compressive effects of the haptics or the CTR [14]. Scleral buckling and pneumocyclopexy have also been reported, with the efficiency of which is under debate.

2.7 Specific Challenges and Personal Experience

Treating traumatic cyclodialysis in blunt trauma to large extent and with severe retinal and choroid involvement can be challenging. Vitrectomy and silicone oil tamponade are needed in most cases. A posterior infusion cannula to restore ocular pressure should always be made first, however, sometimes it is hard to position it into the right place in the vitreous cavity, whether because of the low IOP, choroidal detachment or hemorrhage, or the large extent of cyclodialysis. Anterior chamber infusion can then be an alternative way to restore IOP. In cases with large traumatic cyclodialysis at the position of trocar insertion, the cannula cannot penetrate the detached ciliary body. It is better to drainage the suprachoroidal fluid or hemorrhage and then suture the cleft first (see Case 1). Further steps can then be done to manage the retinal or choroidal damage. For an extremely detached ciliary body with a wide extent, an iris hooker can be used to maintain the ciliary body in position temporarily and make the suturing easier (Fig. 2.4).

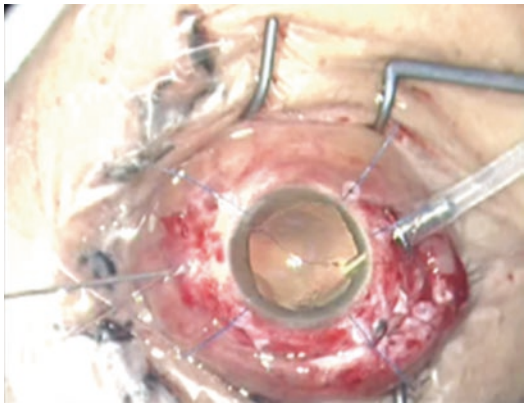

Fig. 2.4 Iris hooker can be used to maintain the ciliary body in position and make the suturing easier

If the posterior cannula can easily be placed in position, then suturing is not always necessary. Cryotherapy along the extent of the cleft can be made to facilitate the closure of the cleft (see Case 2). Perfluoro-N-octane (PFO) assisted total gas-fluid exchange to squeeze the suprachoroidal fluid is always vital before silicone tamponade. A face-down position is recommended after surgery. Silicone oil can be removed depending on how well the retina recover, but for most cases with such severe trauma, silicone oil retention would probably be the result.

2.8 Case 1

Patient with blunt trauma of the right eye causing vitreous hemorrhage, traumatic cataract, lens dislocation and traumatic cyclodialysis was present. Anterior chamber infusion was used to retain the IOP, cataract, and vitreous hemorrhage were removed successfully. PFO was used to retain the retina. Cyclodialysis was right at the position of trocar insertion; thus the cannula cannot penetrate the detached ciliary body. After drainage of the suprachoroidal fluid, the ciliary body was sutured using the bimanual suturing technique. The total gas-fluid exchange was then made, and silicone oil was tamponade (Video 2.1). Six months after surgery, silicone oil was removed, and retinal was in position. The anterior chamber

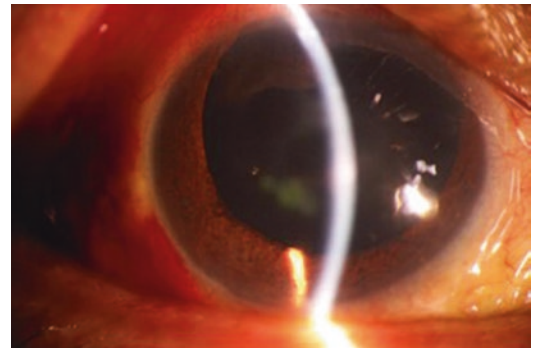

Fig. 2.5 Slip lamp photo showing anterior chamber was deep, and pupil was almost round

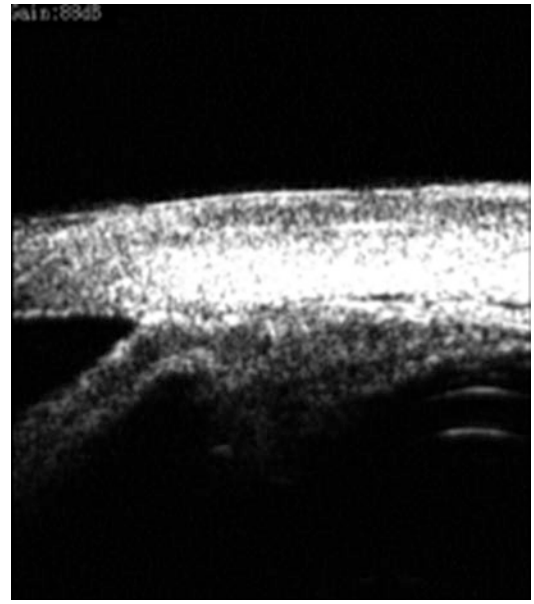

Fig. 2.6 Cross-sectional UBM image showed the ciliary body was reattached

was deep, and the pupil was almost round (Fig. 2.5). UBM showed the ciliary body was reattached (Fig. 2.6), and IOP was maintained in normal status.

2.9 Case 2

Patient with blunt trauma of the left eye causing vitreous hemorrhage, traumatic cataract, and traumatic cyclodialysis was present. After

cataract and vitreous hemorrhage were removed, cryotherapy along the extent of the cleft was made to facilitate the closure of the cleft (Video 2.2). Total gas-fluid exchange with PFO was used to stabilize the retina, and total gas-fluid exchange was made. Silicone oil was then tamponade. Six months after surgery, silicone oil was removed, and the retinal was in position. The anterior chamber was deep, and pupil was round (Fig. 2.7). UBM showed the ciliary body was reattached (Fig. 2.8), and IOP was maintained in normal status.

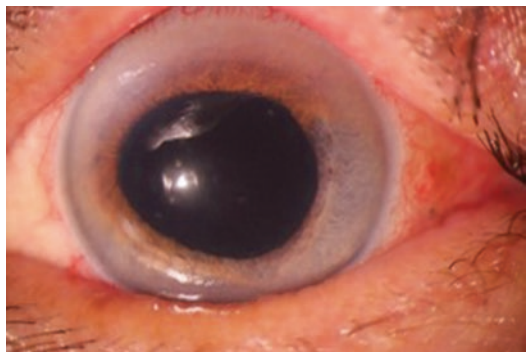

Fig. 2.7 Slip lamp photo showing anterior chamber was deep and pupil was round

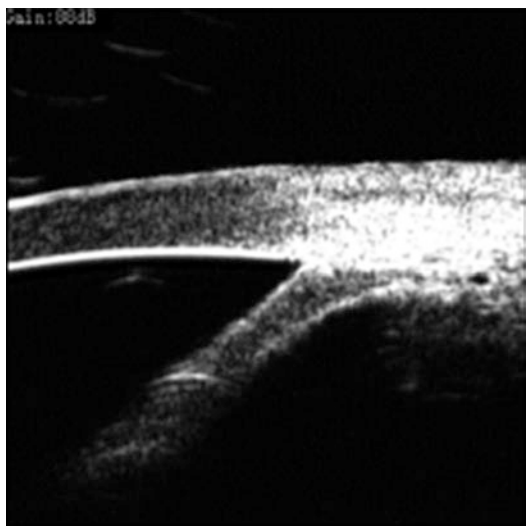

Fig. 2.8 Cross-sectional UBM image showed the ciliary body was reattached

References

1. González Martín Moro J, Contreras Martín I, Muñoz Negrete FJ, et al. Cyclodialysis: an update. Int Ophthalmol. 2017;372(2). https://doi.org/10.1007/s10792-016-0282-8.
2. Ioannidis AS, Barton K. Cyclodialysis cleft: causes and repair. Curr Opin Ophthalmol. 2010;212(2):151–4. https://doi.org/10.1097/ICU.0b013e3283366a4d.
3. Guluma K, Jeffrey E. Lee Rosen's emergency medicine: concepts and clinical practice. Chapter 61, 790–819.e3.
4. Aviel E, Avisar R. Traumatic cyclodialysis. A case report. Br J Ophthalmol. 1976;6011(11):748–9. https://doi.org/10.1136/bjo.60.11.748.
5. Grosskreutz C, Aquino N, Dreyer EB. Cyclodialysis. Int Ophthalmol Clin. 1995;351(1):105–9. https://doi.org/10.1097/00004397-199503510-00011.
6. Murta F, Mitne S, Allemann N, Paranhos A Jr. Direct cyclopexy surgery for post-traumatic cyclodialysis with persistent hypotony: ultrasound biomicroscopic evaluation. Arq Bras Oftalmol. 2014;77(1):50–3. https://doi.org/10.5935/0004-2749.20140013.
7. Malandrini A, Balestrazzi A, Martone G, Tosi GM, Caporossi A. Diagnosis and management of traumatic cyclodialysis cleft. J Cataract Refract Surg. 2008;34(7):1213–6. https://doi.org/10.1016/j.jcrs.2008.02.038S0886-3350(08)00412-4.
8. Sihota R, Kumar S, Gupta V, Dada T, Kashyap S, Insan R, Srinivasan G. Early predictors of traumatic glaucoma after closed globe injury: trabecular pigmentation, widened angle recess, and higher baseline intraocular pressure. Arch Ophthalmol. 2008;126(7):921–6. https://doi.org/10.1001/archopht.126.7.921126/7/921.
9. Andreatta W, Agrawal P, Shah P. Identification of post-gonioscopy hypotony: a simple clinical test to help diagnose occult cyclodialysis clefts. Ophthalmic Physiological Optics. 2015;35(2):242. https://doi.org/10.1111/opo.12173.
10. Jewelewicz DA, Liebmann JM, Ritch R. The use of scleral transillumination to localized the extent of a cyclodialysis cleft. Ophthalmic Surg Lasers. 1999;30(7):571–4.
11. Johnson SM, Cheng HM, Pineda R, Netland PA. Magnetic resonance imaging of cyclodialysis clefts. Graefes Arch Clin Exp Ophthalmol. 1997;235(7):468–71. https://doi.org/10.1007/BF00947068.
12. Trikha S, Turnbull A, Agrawal S, Amerasinghe N, Kirwan J. Management challenges arising from a traumatic 360 degree cyclodialysis cleft. Clin Ophthalmol. 2012;6:257–60. https://doi.org/10.2147/OPTH.S29123opth-6-257.
13. Agrawal P, Shah P. Long-term outcomes following the surgical repair of traumatic cyclodialysis clefts. Eye (Lond). 2013;27(12):1347–52. https://doi.org/10.1038/eye.2013.183.
14. Ioannidis AS, Barton K. Cyclodialysis cleft: causes and repair. Curr Opin Ophthalmol. 2010;21(2):150–4. https://doi.org/10.1097/ICU.0b013e3283366a4d.

Traumatic Aniridia

3

Andrés M. Rousselot Ascarza and Diego Desio

Abstract

The aim of this chapter is to describe the nature of traumatic aniridia and the currently available therapeutic options.

Keywords

Eye injuries · Aniridia · Trauma

3.1 Introduction

The iris is a considerably vascularized smooth muscle sphincter covered with varying degrees of pigmentation. Its immediate and most evident function is to *regulate* the amount of light that enters the eyeball through the aperture of its central space, the pupil. Under high luminosity, the sphincter contracts and opens in dim conditions. Failure in this basic physiological effect results in glare and photophobia but also reduced depth of focus due to the loss diaphragm effect, much like the one used in pinhole visualization.

Alongside the main aid, the iris provides in the regulation of light entrance to the eye, and there are other more subtle but no less important aspects to iridian anatomy. Autonomic responses in the iris tissue cause it to dilate under stress or arousal situations alongside facial expressions and vascular dilatation (blushing); these involuntary responses carry much of the subtext in nonverbal communication, often being unnoticed under normal conditions but detected immediately under malfunction with an instinctive disgust response in the observer. The cosmetic importance of the iris is paramount to the patient and should thus be taken into consideration alongside functionality.

The Iris also has a particular property among the eye tissues in its ability to stick to damaged and inflamed structures generating synechiae which are undesirable to the ophthalmologist but act as a natural closing mechanism with tamponade and vascularization to the area essential if no medical treatment is available.

Partial or total traumatic aniridia constitutes an important challenge to the ophthalmologist, who must balance all of the aspects mentioned above with the different technical options for resolution on a case-by-case basis, as we will discuss in this chapter.

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3.2 Definition of the Disease

Traumatic iris defects can be classified into four large groups:

Classification	Characteristics
Pupillary sphincter defects	Paralytic intermediate mydriasis.
Radial iridian defects	Wedge-shaped iridan tissue loss usually under 90°.
Iridodialisis	Iris separation at its base.
Aniridia	Large or total loss of iris tissue.

They can be produced by penetrating or blunt eye trauma. Iridian defects due to penetrating trauma can be repaired in the same surgical act or in a delayed manner. But as in all-penetrating ocular trauma, the primary objective is the hermetic closure of the eyeball; it might be advisable to defer the surgical repair of the iris to a second procedure when the best decision can be made with good visualization, the right tools, equipment, and properly trained personnel are present.

There is a wide range of possibilities in the resolution of iris defects. Some are easier and widespread, others more expensive or non-available due to government entities pending approval. These go from non-surgical to surgical approaches.

Non-surgical choices may offer the benefit of being reversible and economically accessible while usually providing only partial resolution to these cases. The use of spectacles might be an interesting recommendation, especially for patients until they receive their final surgical procedure since they can provide some alleviation to glare while masking the cosmetic issues. They may be simple sunshades, photochromatic glasses, and some patients might even prefer pinhole spectacles not only for their level of occlusion but also their added depth of field (Fig. 3.1). Cases with total aniridia and aphakia might choose to have a full occlusion with an opaque shade in the affected eye.

Another more invasive but reversible approach is the use of contact lenses. These have the bene-

Fig. 3.1 The use of slits and pinholes to reduce glare has been documented amongst the peoples of the north (left). Modern and commercially available examples exist available for the general public (right)

fit of tackling both optical solutions and cosmetic compensation while reducing the amount of light that enters the eye. They do, however, present the disadvantage of eye manipulation, tolerance requirement from the patient and a reasonably smooth cornea to be applicable.

Corneal tattooing (D Hirsbein [1]) provides an interesting alternative to intraocular surgery for localized iris defects and might even be a valid cosmetic approach, especially for patients without visual prognosis and severely damaged corneas since a trained physician might even imitate the characteristics of the fellow healthy eye with the use of various pigments. This is not, however, the best approach for full aniridia and also lacks the reversibility of the non-surgical methods.

In the repair of the ruptured sphincter of the iris, iridodialysis and in radial defects of the iris under 90°, the surgical technique of choice is suturing. Repair of the iris sphincter due to traumatic mydriasis using the loop technique with 10-0 polypropylene suture is the most used approach. The most popular surgical repair of radial defects of the iris and iridodialysis is the McCannel technique with 10-0 polypropylene double-stitched suture (Fig. 3.2).

In many cases of large defects of the iris, tissue retracts and becomes attached to the chamber angle, producing peripheral anterior synechiae, which can alter the flow of aqueous humor. Thus, through goniosynechialysis, in a careful way, the iris can be released for its subsequent iridoplasty with sutures. In those cases where repair of the iris is not possible due to great loss of iris tissue, there are different intraocular devices for its treatment.

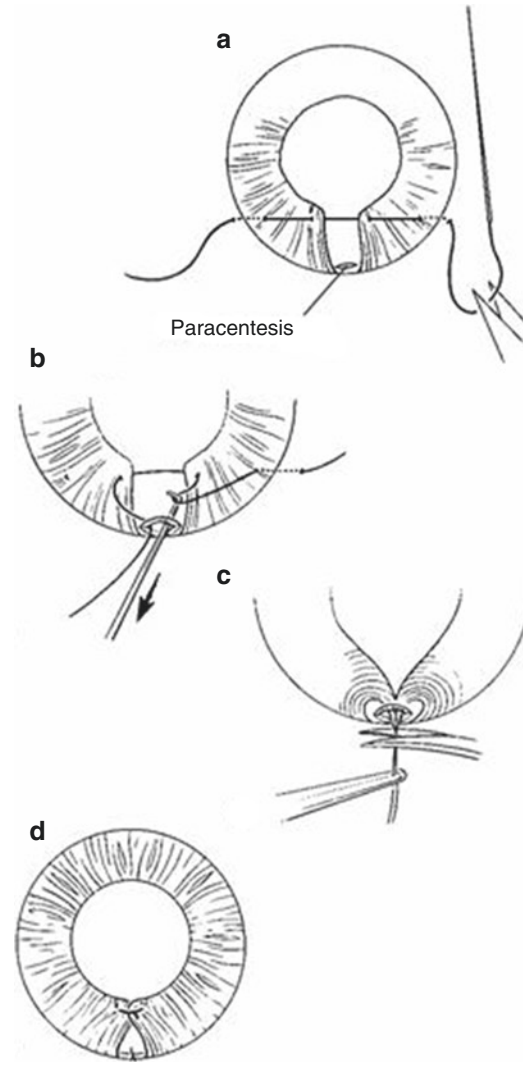

Fig. 3.2 The McCannel technique for repairing iris lacerations. With large lacerations, multiple sutures may be used. (a) A limbal paracentesis is made over the iris discontinuity. Then a long Drews needle with 10-0 polypropylene is passed through the peripheral cornea, the edges of the iris, and the peripheral cornea opposite, and the suture is cut. (**b**) A Sinskey hook, introduced through the paracentesis and around the suture peripherally, is drawn back out through the paracentesis. (**c**) The suture is securely tied. (**d**) After the suture is secure, it is cut, and the iris is allowed to retract. (Pending permission Reproduced with permission from Hamill MB. Repair of the traumatized anterior segment. Focal Points: Clinical Modules for Ophthalmologists. San Francisco: American Academy of Ophthalmology; 1992, module 1. Illustrations by Christine Gralapp)

3.3 Implants for Iris Defects

In 1964, Peter Choyce [2] reported the use of the first intraocular anterior chamber prosthetic iris device (PID). Clinically, PID can be categorized into three groups: (1) iris-lens diaphragm, (2) endocapsular capsular tension ring (CTR)-based PID, and (3) customized artificial iris [3].

There are several case series in the literature with follow-up for the treatment of large iris defects of different types, and their choice will depend on each case [4–6]. In the majority of penetrating or blunt ocular trauma in which there has been great damage to the iris, other intraocular structures will surely have been affected, such as the lens, chamber angle and retinal injuries. That said, most ocular trauma patients have or will develop a lens opacification and therefore, when deciding to implant an intraocular device for the treatment of large iris defects, the extraction of the lens is often essential. When deciding to treat an iris defect, we must evaluate the size of the defect to be corrected. As previously mentioned, radial defects smaller than 90° should, as far as possible, be sutured. For those cases where the iris cannot be sutured and for defects greater than 90° there are different devices for aniridia (partial or total) on the market, and the choice will depend on whether the patient is phakic, pseudophakic, or aphakic. Currently, there are several implants available to treat aniridia, as well as aphakia or cataracts, marketed by Morcher GMBH (Germany), HumanOptics (Germany) [7] and Ophtec Inc. (USA) [8] (see Fig. 3.3 below). All companies present different models of implants to treat iris defects.

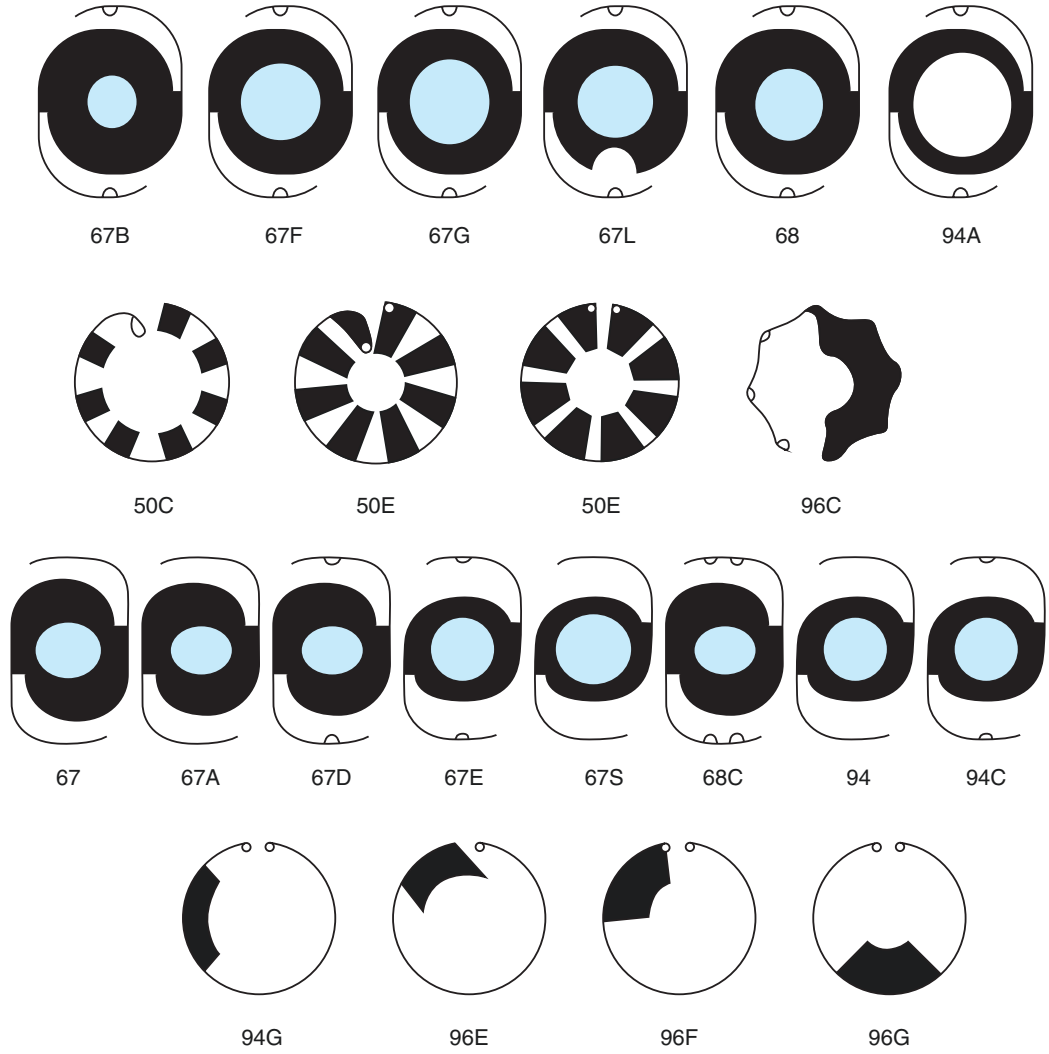

Fig. 3.3 Currently available models of aniridia implants from Morcher GMBH (Stuttgart, Germany) [9]

In phakic patients with iris defects under 180°, a surgical option is cataract surgery with routine intraocular lens implantation and placement in the capsular bag of one or two partial rings for aniridia with a 90° diaphragm (Morcher GMBH), positioning them in such a way as to cover the iris defect (see Case 2). Another option is to place a 180° diaphragm ring in the sulcus [10]. The size of the incision for the insertion of these devices will depend on the chosen model, ranging from 3 mm to 4.5 mm, approximately. In pseudophakic patients with iris defects, the same options can be chosen with the placement of one or two implants at 90° or 180°, depending on the iris defect to be covered.

In cases where aphakia occurs, there are several paths to choose from. It is possible to opt for the placement of an implant for aniridia with optics with a 360° diaphram of black PEMA sutured to the sclera (see Case 1). Ophtec USA Inc. has iris prostheses (with 360° diaphragms) of different colors with the possibility of incorporating a lens for optical correction through a 3.5 mm incision assisted by an injector. The HumanOptics CUSTOM FLEX artificial iris is a custom foldable iris prosthesis, without optics, for insertion in the posterior chamber sutured to the intraocular lens or sclera (see Case 3).

In all cases of partial or total iris defects, the choice of the type of implant and the surgical technique will depend on the ophthalmologist, as well as their experience in these cases.

3.4 Brief Case Reports

3.4.1 Case 1

Fifty-four-year-old male with work-related blunt ocular trauma to his OS 2 years prior with sphincter iris rupture, generalized iris atrophy, low inferior lens subluxation and mild cataract.

Preoperative uncorrected visual acuity OD 10/10 OS 2/10.

Best-corrected visual acuity improving to 8/10 in OS. (sph. +1.25 cil. −2.00 at 70°).

Normal IOP and fundus examination in both eyes.

The patient's main source of discomfort was a glare in his left eye.

Our first approach was the placement of a corrected iris print contact lens (CL) for refractive, cosmetic, and functional purposes, reaching a 8/10 VA with reduced glare. Unfortunately, the patient reported intolerance to the CL, choosing not to use it.

Six months later, the patient's cataract worsened into a mature white cataract, reducing the glare but increasing the esthetic concerns. With a normal ultrasound evaluation, the surgical option was presented to remove the cataract and place a lens with aniridia implant 67F from Morcher GMBH (Stuttgart, Germany) (Fig. 3.4).

A superior scleral approach was used to perform the phacoemulsification of the lens with the posterior widening of the incision for placement within the lens bag. The implant's wide nature allowed for proper tension without the need for fixation despite some superior zonular rupture.

Final visual acuity in the left eye was 10/10 with sph +1.00 cyl. −2.50 at 10° ADD +3.00.

Best tolerance and glare reduction were obtained with multifocal progressive aerial spectacles with photochromatic transition treatment.

3.4.2 Case 2 (Courtesy of Dr. Ariel Blanco)

A 22-year-old male suffering a penetrating corneal wound to his right eye with iris tissue loss. First surgical intervention achieved the correct closure of the eye. A secondary procedure was indicated when a white cataract developed.

Phacoemulsification of the lens was performed with the assistance of ocular tension rings placement. Two 50C aniridia implants from Morcher GMBH (Stuttgart, Germany) were placed in the bag to achieve occlusion alongside an aspheric foldable IOL (Fig. 3.5).

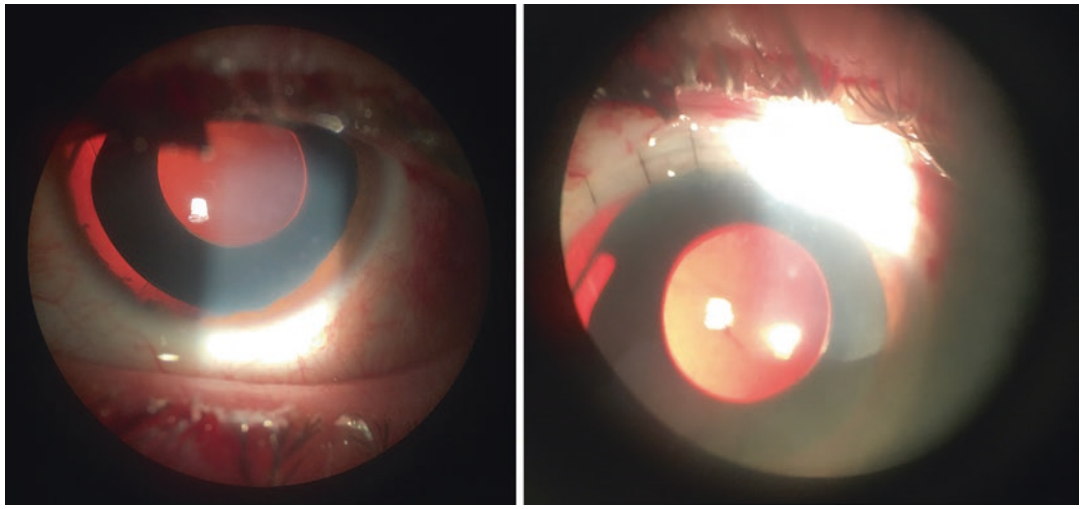

Fig. 3.4 Postop 67F lens from Morcher GMBH (Stuttgart, Germany)

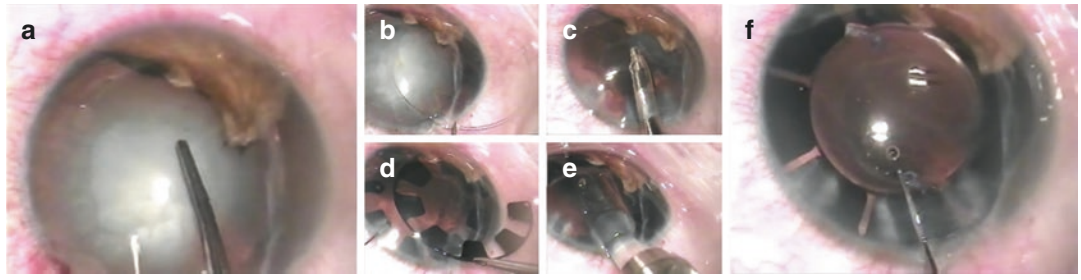

Fig. 3.5 (**a**) Initial conditions, (**b**) Capsular tension ring placement, (**c**) phacoemulsification, (**d**) Morcher implants, (**e**) IOL placement, (**f**) final results. Images Courtesy of Dr. Ariel Blanco

3.4.3 Case 3 (Courtesy of Dr. Ariel Blanco)

A 50-year-old male suffering post-traumatic aniridia with iris atrophy in his left eye.

After lens removal and IOL placement in the bag the Artificial Iris is cropped to size, and a peripheral "artificial iridectomy" is performed to guarantee aqueous flow (see Fig. 3.6a–d). Two points of fixation from prostheses to sclera are taken with polypropylene suturue before the implantation through the main corneal incision (see Fig. 3.6e, f). After implantation, the sutures are tightened and fixed, correcting for centration of the prostheses (see Fig. 3.6g, h).

3.5 Important Signs, Examinations, Diagnosis, Surgical Procedures and Skills, or Postoperative Treatment for Complications

Appearances might be deceiving, and a trauma case severe enough to cause some degree of aniridia is not to be underestimated. Whether the iris is lost due to extraction in prolapsed tissue, atrophied, desinserted, or ruptured, the energy involved in any of these traumatic mechanisms rarely limits its damage solely to the sphincter.

A thorough fundus examination is mandatory in any, and all trauma cases, second only to ultra-

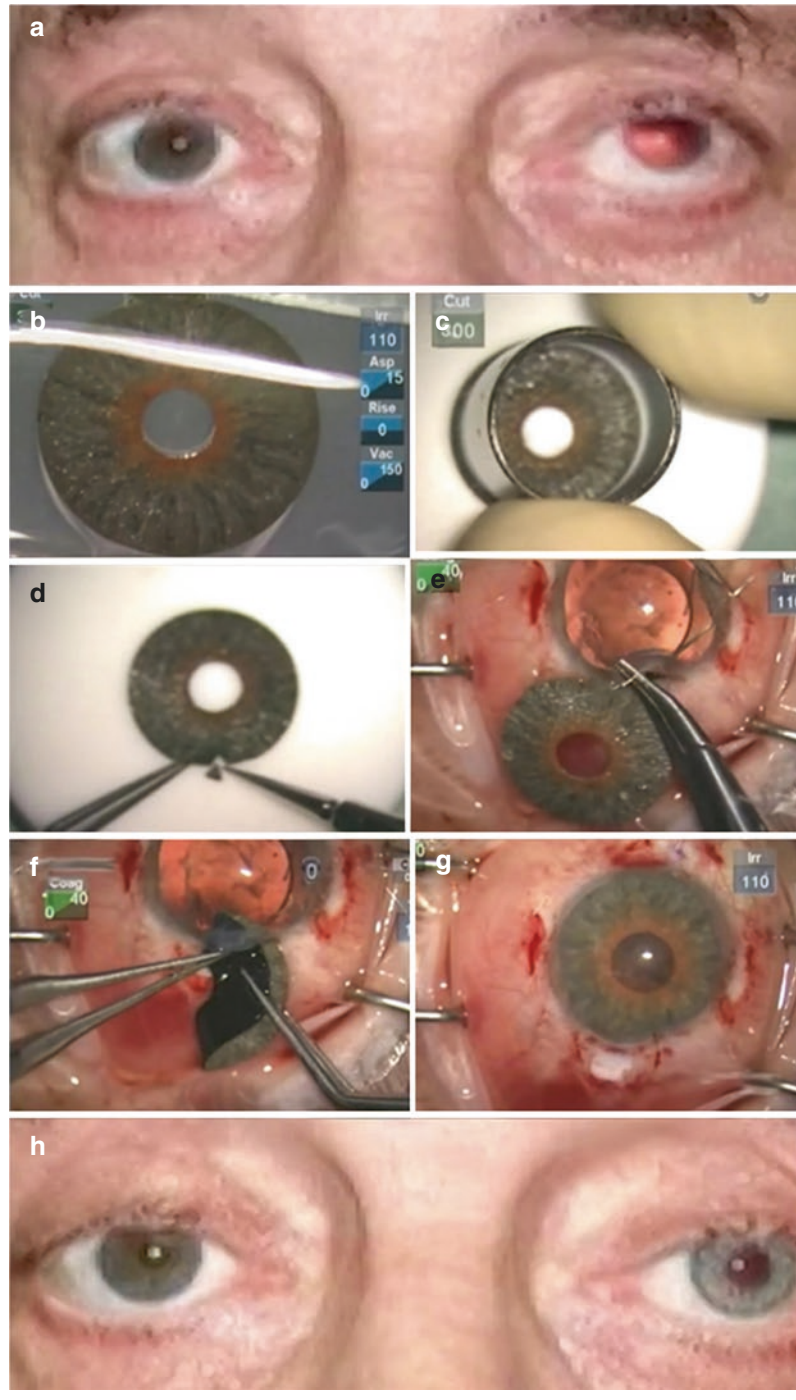

Fig. 3.6 Implantation procedure for CUSTOMFLEX® ARTIFICIALIRIS by HumanOptics. (**a**) Pre-operatory, (**b**) iris implant, (**c**) implant size adjustment, (**d**) suture fixation, (**e**) first intraocular suture placement, (**f**) folded implant insertion, (**g**) fully placed implant, (**h**) post-operatory. Images Courtesy of Dr. Ariel Blanco

sound evaluation (in closed globe scenarios). If no retinal detachment (RD) is present special attention must be taken to examine both the posterior pole for macular holes and the periphery for any retinal tears. Retinal damage takes priority after eye closure is obtained when needed, and all anterior segment interventions are secondary to the retina expert. Mixed procedures such as phacovitrectomy with anterior segment repair can be attempted under the proper conditions of visual-

ization, resources, and personnel available. Posterior segment evaluation does not stop if the initial evaluation is normal since some patients might experience RD in mid to late terms after ocular trauma. Patients should be observed in all follow-ups as well as educated in alarm signs such as novel floaters, metamorphopsia, and photopsia.

Intraocular pressure (IOP) increases, and glaucoma secondary to anterior segment trauma is frequent. Pigment dispersion, synechiae, angular scarring, and hyphema are possible causal mechanisms and need to be addressed since the initial evaluation all the way to postop follow-up. Same as with retinal damage, not all ocular trauma patients who develop glaucoma experience it since their initial exam and most are only detected if examined.

When examining the anterior segment, phacodonesis is one of the most informative signs to be looked for. Even if no evident subluxation is observed, knowing the zonular structure might be damaged can make the difference in proper planning for intervention with alternative IOLs and capsular support such as tension rings. A simple way to examine the inevident cases is, under slit lamp evaluation with the patient's head weight fully rested on the supports, to knock on top of the examination table upon which the slit lamp is placed. The vibrations will travel through the lamp's structure into the eye and cause a subtle but observable movement of the lens. Whenever in doubt, repeat the process observing the healthy eye and confirm no such changes occur.

As in all intraocular procedures, follow-up consultations need to evaluate for endophthalmitis and RD with prompt referral to retina experts if any of these are detected. A common complication, especially with in bag or sulcus placement of prostheses, is the luxation to the posterior segment. In such cases, a full vitrectomy is indicated with removal and/or scleral fixations of the devices.

3.6 Personal Experiences

As suggested at the beginning of this chapter, there are multiple sources of discomfort for the patient suffering aniridia. Especially in post-trauma patients who experienced a totally normal life until the event and now have to cope with the consequences. A sincere and thorough interrogation will go a long way in identifying the main concern with each patient for whom functionality might not be the top priority.

Some patients pose glare as their main concern without much care for esthetics and even stating that the functionality of their fellow healthy eye is compromised since they can no longer work comfortably outdoors. It is often useful to consider the need for fundus examination while tackling glare with occlusive means since the "new pupil" will be static; therefore some compromise with minor glare reduced by opaque or changing spectacles might be advisable.

Other patients want to regain visual acuity at all costs and as soon as possible, especially if the fellow eye is not at 20/20 for some other reason. Patients with a relatively healthy posterior segment and corneal transparency might achieve good visual outcomes provided the correct IOL measurements are obtained, and the prosthesis is correctly placed. But, for most, there needs to be serious talk before surgery to manage expectations. A post-traumatic cataract with aniridia is not a run of the mill procedure, lens measurements can be troublesome and sometimes can only be estimated via ultrasonography, the anatomy of the anterior segment is drastically changed, leading to corrective mistakes, and the zonular diaphragm might not resist the in bag option leading to sulcus placement or scleral fixation (all less precise). An honest talk with the patient and their family needs to address these issues and explain the case at hand with the myriad of alternatives both in treatment and possible complications, always keeping the door open for ulterior interventions or the assistance of contact lenses and/or glasses (this is particularly true for younger patients who are not used to reading glasses).

Finally but not least important is the esthetic aspect concerning the patient. This often disregarded issue by the physician might be the center of the patient's psychological discomfort and is a subject that needs to be handled with every bit of honesty as the ones mentioned above with an

extra layer of tact not only with young and impressionable patients but also with the occasional heavy-hearted parent. When available, offer the cosmetic contact lens option as soon as possible, even when surgical treatment is under consideration or scheduled for the intermediate future. This will provide not only the alleviation of the patient's concern; but also a valid alternative in case surgical outcomes do not meet the subject's expectations.

As far as surgical options, the CUSTOMFLEX® ARTIFICIALIRIS by HumanOptics provides one of the best alternatives in handling these two aspects (no commercial interest from any of the authors). We do find, however, two limitations to be considered with this alternative.

1. *Pupil Size*: ArtificialIris is 360°, 12.8-mm diameter disks with fixed pupils of 3.35 mm, which present a limitation in the fundus examination. However, modern Non-Mydriatic Fundus Cameras can help overcome this issue, and an ultrasound evaluation can provide a comprehensive assessment of the posterior segment.
2. *Price and Availability:* the implant was granted approval by the FDA on May 30, 2018, extending its legality to other, but not all, regulatory agencies around the world that follow the American standards. Yet, price can be a significant limiting factor.

References

1. Hirsbein D, et al. Corneal tattooing for iris defects. J Fr Ophtalmol. 2008;31(2):155–64.
2. Choyce P. Inter-ocular lenses and implants. London: Lewis; 1964.
3. Ayres SB. Management of aniridia and iris defects: an update. Curr Opin Ophthalmol. 2016;27:244–9.
4. Mayer CS, et al. Challenges and complication management in novel artificial iris implantation. J Ophthalmol. 2018, epub.
5. Li J, et al. Modified implantation of black diaphragm intraocular lens in traumatic aniridia. J Catar Refrac Surg. 2013;39(6):822–5.
6. Mavrikakis I, et al. Surgical management of iris defects with prosthetic iris devices. Eye (London). 2005;19:205–9.
7. HumanOptics. humanoptics.com. 2020. https://www.humanoptics.com/en/physicians/artificialiris/
8. Inc., O. U. ophtec.com. 2020. https://www.ophtec.com/products/trauma-surgery/implants/iris-prosthesis#downloads
9. Srinivasan S, et al. Prosthetic irisdevices. Can J Ophthalmol. 2014;49:6–17.
10. Miller K, Lin S. Lessons learned from implantation of Morcher 50D and 96S artificial iris diaphragms. Case Rep Ophthalmol. 2017;8:527–34.

Traumatic Dislocation of the Lens

4

Hong Yan, Chenjun Guo, and Huping Song

Abstract

The condition of traumatic lens dislocation is complex and diverse, and the diagnosis can be confirmed based on clinical manifestations and imaging examinations. The challenge is formulating a unique treatment plan based on the degree, location of the dislocation, and whether it is combined with other intraocular tissue damage, to achieve a more ideal treatment effect which may require two or more operations. The symptoms and examination of lens dislocation, the choice of intraocular lens, various surgical methods and surgical strategies for different dislocations are discussed.

Keywords

Trauma · Lens dislocation · Symptoms
Examination · Surgical treatment

Supplementary Information The online version of this chapter (https://doi.org/10.1007/978-981-16-5340-7_4) contains supplementary material, which is available to authorized users.

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4.1 Introduction

Ectopia lentis is defined as the dislocation of the natural crystalline lens. Many conditions can lead to ectopia lentis, such as trauma, pseudoexfoliation syndrome, Marfan syndrome, Weill–Marchesani syndrome, intraocular neoplasia, and aniridia [1]. Traumatic injury is the most common cause of subluxated or dislocated lenses [2]. The most common cause of lens luxation–subluxation is blunt trauma, accounting for approximately 53% of all cases [3].

Dislocation of the lens can occur secondary to ocular trauma in both open and closed globe injury subgroups and can be associated with other sequelae in both the anterior and posterior segments.

Retinal break or detachment is often associated with traumatic dislocation of the lens, especially in some serious trauma. Retinal injuries occurred in approximately one-third of patients with traumatic dislocation of the lens [1]. Cataract was the most frequent ocular association (53.5%) [4].

4.2 Definition

Traumatic dislocation of the lens refers to incomplete and total dislocation of the lens due to rupture of zonules caused by ocular trauma, which is the most common reason for lens dislocation.

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Subluxation of the lens refers to that the zonules around the lens are not completely broken, and the lens is still in the posterior chamber (PC) (Fig. 4.1). Luxation of the lens is the rupture of the zonules. The lens may fall into the anterior chamber (AC) (Fig. 4.2), fall into the vitreous cavity, be stuck in the pupil, or even enter the bulbar conjunctiva. Both lens subluxation and luxation can result from either open or closed globe trauma [5]. Lens dislocation is more common in patients with closed ocular trauma than that in patients with open ocular trauma.

Blunt trauma causes anteroposterior shortening of the globe with equatorial expansion, stretching the zonules asymmetrically depending on the dominant mechanical forces of coup/contrecoup, resulting in luxation. A penetrating object produces zonulolysis of the corresponding sector as it ruptures the zonules during its passage.

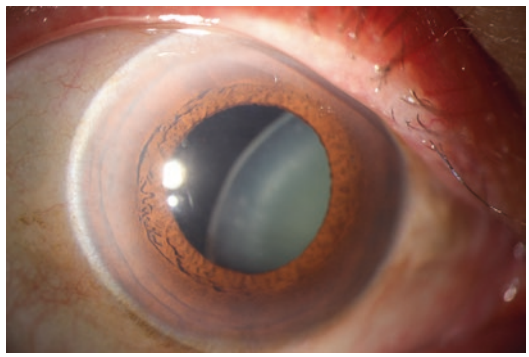

Fig. 4.1 Incomplete dislocation of lens

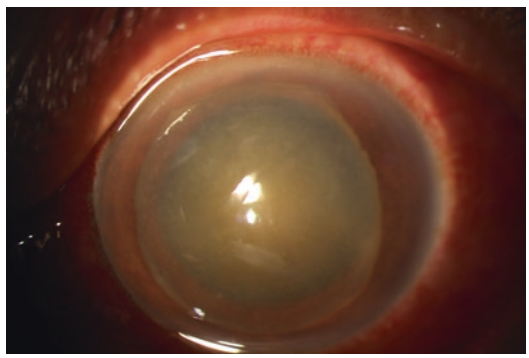

Fig. 4.2 The lens may fall into the anterior chamber

4.3 Case 1

A 52-year-old female presented with vision loss and pain in her right eye for 3 days. Her eye was hit by her grandson with a water pistol accidently. She initially visited a local clinic closer to home, and was prescribed some topical antibiotic eye drops, without any efficacy, so she was transferred to Ocular Trauma Center, Shaanxi Eye Hospital for further care. She had hypertension for 10 years, and did not take medicine regularly.

The visual acuity was hand motion/30 cm, the intraocular pressure was 7 mmHg. The cornea edema and Descemet's folds were found, anterior chamber was deep with a little hyphema interiorly. Pupil was 5 mm irregular, poorly reactive. Lens was opacified and dislocated into vitreous cavity inferiorly (Fig. 4.3). Fundus could not be seen clearly with vitreous hemorrhage. B scan confirmed the lens luxation and vitreous hemorrhage, with no retinal detachment. CT also demonstrated cataract and lens luxation. So the diagnosis was lens luxation, vitreous hemorrhage of left eye.

The pars plana vitrectomy (23G) and lensectomy were performed, retinal was attached, and an intraocular lens was suspended by 10-0 polypropylene at 6 and 12 o'clock (Fig. 4.4). The visual acuity was 20/80 on the first day postoperation, with normal intraocular pressure, the cornea was clear, anterior chamber was deep, IOL position was well, and fundus was normal.

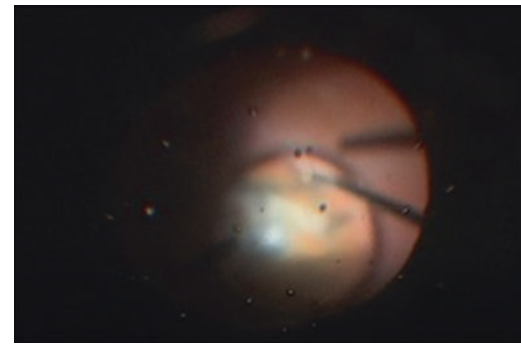

Fig. 4.3 Lens was opacified and dislocated into vitreous cavity inferiorly

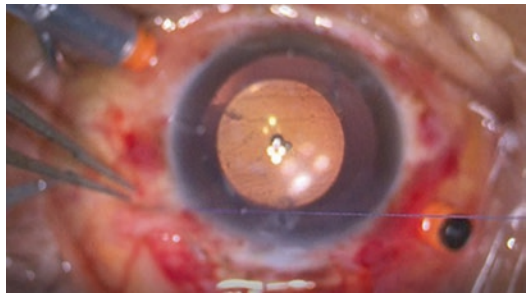

Fig. 4.4 After the pars plana vitrectomy (23G) and lensectomy were performed, an IOL was suspended by 10-0 polypropylene at 6 and 12 o'clock

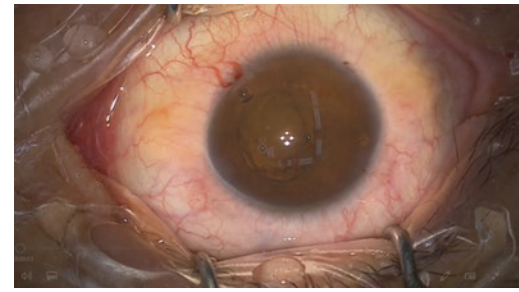

Fig. 4.5 The cornea was clear, anterior chamber was deep, pupil was 3 mm and irregular, and lens was luxated

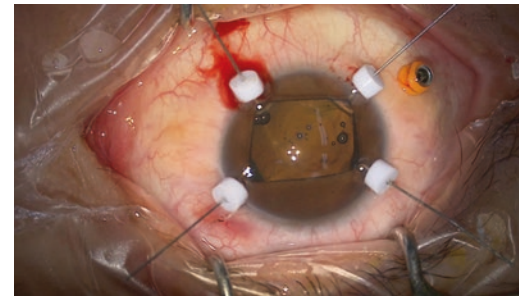

Fig. 4.6 Using iris hooks, the pars plana vitrectomy (23G) and lensectomy were performed

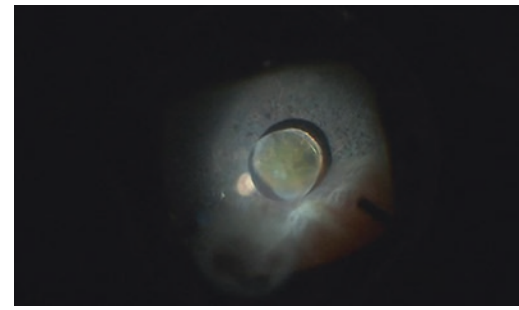

Fig. 4.7 The lens was opacified and dislocated into vitreous cavity inferiorly, retina was pigmented, and retinal detachment was noted from 9 to 2 o'clock superiorly, with lattice degeneration of retina and holes

4.4 Case 2

A 32-year-old man presented with blurred vision in his right eye for 1 week without trauma. Fourteen years ago, retinal detachment was found in his left eye, and vitrectomy combined with silicone oil tamponade, lensectomy were performed, without IOL implantation. He had congenital heart disease nearly 30 years, and mitral valve was replaced for 5 years.

The visual acuity of right eye was 20/120, the intraocular pressure was 13.9 mmHg. The cornea was clear, anterior chamber was deep, pupil was 3 mm, irregular (Fig. 4.5), lens was opacified and dislocated into vitreous cavity inferiorly, retinal detachment was found. B scan confirmed the lens luxation and retinal detachment. The diagnosis of lens luxation, retinal detachment, Mafan's syndrome was made.

The pars plana vitrectomy (23G) and lensectomy were performed, with the help of iris hooks (Fig. 4.6). The retina was pigmented and retinal detachment was noted from 9 to 2 o'clock superiorly, with lattice degeneration of retina and holes (Fig. 4.7). Photocoagulation was made around lattice degeneration of retina and holes, and silicone oil was tamponaded. The visual acuity was 20/200 on the first day post-operation, with normal intraocular pressure, retina was attached.

4.5 Important Signs

Common symptoms of lens dislocation include decreased vision, ametropia (myopia, hyperopia, or severe astigmatism), monocular diplopia, glare, and functional aphakia [6].

The symptoms of lens subluxation depend on the degree of displacement. If lens subluxation remains on the optic axis, myopia caused by the change in lens thickness is possible. If the lens is inclined horizontally, vertically, or obliquely, severe astigmatism could occur, which is difficult to correct. If the lens is displaced so that it occupies only half of the pupil, monocular diplopia may occur.

According to the different positions of lens dislocation, the clinical symptoms are different. Pupillary block caused by the lens incarcerated in the pupil area may lead to pupillary block glaucoma. If the lens completely leaves the pupil area, it will be aphakic, the AC will become deeper, the iris will tremble, and the dislocated lens will move with the body position in the early stage. If the lens is located in the AC, it will sink below the deeper AC. The lens of the AC may cause serious iridocyclitis, corneal dystrophy, and acute glaucoma due to repeated contact with the cornea, iris, and ciliary body. There is no inflammatory reaction in the early stage and tissue damage in the later stage when the lens falls into the vitreous body.

4.6 Examinations

Slit lamp is the most common detection method for lens dislocation. Through a slit lamp, the position of the lens can be observed. In addition, lens position can be viewed using orbital CT and B-scan ocular ultrasonography. If there is an open trauma and B-scan ocular ultrasonography cannot be used, orbital CT can be used instead [7].

4.7 Treatment

The effect of treatment on lens dislocation caused by trauma is different. The best-corrected distance visual acuity changes from blindness to good vision, depending on the severity of the injury and the severity of the combined injury.

The treatment of lens dislocation depends on many factors, including the position of the lens, hardness of the nucleus, visual acuity, complications, and the degree of compound injury.

For mild lens dislocation, there is no obvious visual loss and anterior or posterior injury, which does not need surgery. For existing complications such as high intraocular pressure (IOP), drug control should be considered first, and glucocorticoids should be used for uveitis. For lens subluxation without other combined injuries and complications, the primary treatment is to correct the ametropia in the lens or aphakic area and restore proper vision.

The surgical indications of lens dislocation were as follows: the dislocation of the lens seriously damaged the vision, accompanied by a cataract, the lens falling into the AC, phakic glaucoma, lens allergic uveitis, or pupillary block glaucoma; conservative treatment or simple glaucoma surgery cannot reduce the IOP; and lens opacity affects the examination and operation of retinal detachment [8].

Lens trauma can be treated using various surgical procedures and fixation techniques. A careful surgical strategy should be established for globe reconstruction after trauma with secondary lens implantation.

4.8 Surgical Treatment

4.8.1 Lens Extraction

1. Anterior surgery includes phacoemulsification, aspiration, extracapsular extraction, and intracapsular extraction.

If the posterior capsule is intact and there is no vitreous prolapse into the AC, the treatment could consist of phacoemulsification or extracapsular cataract extraction. When all zonules are torn, another therapeutic option is intracapsular cataract extraction. Phacoemulsification is suitable for less than 1/4 lens dislocation without a too hard lens nucleus.

Lensectomy is recommended in cases of documented vitreous prolapse into the lens and can be combined with intraocular lens (IOL) implantation.

If the posterior capsule is intact, femtosecond laser is used to remove the anterior capsule without traction of the zonules. Femtosecond laser is not suitable for patients with a large lens tilt, a large range of lens dislocation, serious lens shaking, or pupil dilation [9, 10].

A luxated lens could be located subconjunctivally, anteriorly, or posteriorly or extruded completely. The subconjunctival site can be reached using a pair of forceps or a cryoapplicator. If the lens is luxated into the AC, immediate surgical intervention is obligatory to preclude further endothelial cell injury.

2. For posterior surgery, pars plana lensectomy is indicated when there is incomplete lens dislocation greater than 180° with the lens entering the vitreous cavity, the vitreous body falling into the AC, or vitreous hemorrhage or retinal detachment. Vitrectomy could be performed in children or young individuals.

If the nucleus of the lens is hard, during vitrectomy, a viscoelastic agent and heavy water can be used to hold up the lens to avoid damage to the retina caused by phacoemulsification and the vitrectomy head [11]. If the lens nucleus is too hard, limbal incision could be considered.

Iridotomy can prevent pupillary block and acute glaucoma.

4.8.2 IOL Selection

In the case of post-traumatic uveitis, silicone IOLs should not be used because inflammatory reaction substances may deposit on the surface of the lens. Acrylic lenses are preferred in eyes that require vitreoretinal lenses in the future or those with a history of uveitis.

In 1972, Worst [9] has first proposed the iris claw-type IOL. The iris claw-type IOL is a monolithic IOL made of polymethylmethacrylate. The loops on both sides of the optical part have small cracks, which can be fixed on the inactive peripheral iris matrix.

A three-piece PC IOL can be placed in the ciliary sulcus of the eyes with an intact anterior capsule; however, the posterior capsule is compromised. Without capsular support, a three-piece lens can be sutured to the sclera [12].

PC IOLs are the standard therapeutic approach.

The selection of the right type of lens depends on the individual surgeon's experience and acquaintance with the variant implantation techniques [8].

4.8.3 IOL Implantation

If there is capsular bag support, a secondary IOL can be placed in the sulcus. If there is no capsular bag support, a secondary IOL can be fixated to the AC angle, iris, or sclera.

4.8.3.1 Anterior Chamber IOL Implantation

The advantages of AC IOLs are low implantation difficulty, short operation time, and safety. Its disadvantages include that this IOL could easily damage the corneal endothelium, resulting in corneal endothelium decompensation. In addition, fibrin around the loop of this IOL accumulates in the atrial angle, leading to choroidal tissue necrosis, blood–ocular barrier damage, the release of several inflammatory mediators [13], secondary chronic ocular inflammation, and glaucoma. With the advancement of the new open-loop AC IOLs, the incidence of complications has decreased, the operating time has shortened, and lens insertion has been simplified.

PC IOLs are preferred for aphakic eyes with Fuchs' endothelial dystrophy, a low endothelial cell count, or cystoid macula edema, rather than AC IOLs.

4.8.3.2 Iris-Fixed IOL Implantation

The *iris claw-type IOL* can be fixed into the inactive peripheral iris matrix. This part of the iris has less blood vessels and does not easily react [10–16]. In addition, the iris claw-type IOL has a bridging arc structure, which can eliminate the occurrence of pupil edge erosion. The concave–convex lens design can increase the distance between the IOL and corneal endothelium. Complications of this IOL including dislocation

decreased corneal endothelial cell count, transient high IOP, wound leakage needing to be resutured, and retinal detachment. In addition, the iris claw-type IOL can be fixed on the PC, which can ensure the depth of the AC and the distance from corneal endothelial cells. Meanwhile, it does not affect the iris function and is not easy to cause pupil block and secondary glaucoma. In some patients, mild oval pupils, cystoid macular edema, iris clip shedding, and iris depigmentation can occur [14].

The iris claw-type IOL can be used as an alternative for patients with a shallow AC angle in whom an AC-fixed IOL cannot be used [15]; however, this method is not suitable for patients with obvious iris trauma and anterior segment structure damage. In addition, because the iris claw-type IOL is not foldable, the main incision is larger, and two auxiliary incisions are needed. Therefore, the incision leakage should be carefully checked during the operation.

The *fixation of PC IOLs on the iris by suture* is another simple method. For patients with aphakia with partial loss of the iris after ocular trauma, the IOL complex can be fixed on the residual iris. The complications of this IOL type are dislocation, iritis, abnormal pupil function, and iris pigment dissemination. It is not suitable for patients with severe iris injury and obvious damage to the anterior segment structure. Iris-sutured posterior IOLs require less surgical time than the transcleral approach. Certain complications such as postoperative inflammation, uveitis, and iris atrophy with pigment-dispersion syndrome and secondary glaucoma can appear following iris fixation.

4.8.3.3 Posterior Chamber Intraocular Lens Implantation

Presence of Capsular Bag Support

If the capsule bag is intact, the IOL can be implanted into the bag. If the anterior capsule is intact and the posterior capsule is torn, the IOL can be placed in the ciliary sulcus. If a part of the anterior capsule is intact, one haptic of the IOL can be placed in the ciliary sulcus and the other fixed in the sclera or iris.

If zonule rupture is less than 120°, the IOL can be fixed using a capsular tension ring (CTR). After the CTR is placed, it can help reset the dislocation bag and make the tension evenly distributed. Simultaneously, it can prevent fibroblasts and lens epithelial cells from migrating to the posterior capsule.

The CTR should be used carefully when there is a hole in the posterior capsule. The CTR can make the posterior capsule hole bigger, increasing the risk of the IOL falling into the vitreous cavity. In addition, the tension segment of the bag along with the CTR or alone was used to assist IOL implantation.

Absence of Capsular Bag Support

Scleral fixation of sutured intraocular lens

Scleral suture fixation is one of the most commonly used secondary IOL implantation methods. In 1988, Girard [16] has first proposed the design of fixing IOLs to the sclera using a suture. The key operation procedure is as follows: make a lamellar scleral flap, clip out the suture through the PC from the corneal lamellar incision, cut it, fix the haptics of the IOL, and implant them into the PC. Adjust the suture tension to a suitable position and bury the suture knot in the lamellar sclera.

This method is *applicable* because of its long distance from the cornea, iris and other tissues. Therefore, the pupil function will not be affected, the postoperative visual acuity will be improved and stable, and various complications caused by the AC IOL can be avoided [13]. The operation is relatively difficult and requires high suturing and surgical skills.

This method can cause *complications*, including suture fusion, rupture, IOL deviation, displacement, fibrin reaction, vitreous hernia, retinal detachment, vitreous hemorrhage, suprachoroidal hemorrhage, and cystoid macular edema [17]. Suture-related complications include suture rupture leading to IOL dislocation, endophthalmitis, suture fusion, and lens tilt. Presently, the scleral fixation IOL technique is suitable for patients with corneal injury, shallow AC, peripheral angle adhesion, glaucoma, and other diseases.

Presently, there are many new methods of IOL suture fixation to improve the stability and accuracy of IOLs in the eye. Mambran's technique described a two-point sclera fixation. Furthermore, one-point, three-point, and four-point fixations have been reported [18]. Szurman [19] has described a new knotless Z-suture technique without the need for protective sclera flaps or lamellar grooves. Lens subluxation was performed using a single-loop suture fixation technique.

Sutureless intrascleral posterior chamber intraocular lens fixation

The intrascleral IOL fixation technique has become a popular procedure because of its advantages over conventional transcleral suturing of IOLs [20, 21]. In 2006, Gabor and Pavlidis [12] have proposed the sutureless scleral interlaminar fixation technique. A standard three-piece PC IOL was used to fix in the ciliary sulcus, and the haptics of the IOL were fixed in the scleral tunnel parallel to the corneoscleral margin (Fig. 4.8). Subsequently, the doctors at home and abroad made various attempts and improvements.

Yamane has proposed a new technique for scleral IOL fixation [22]. Two angled incisions parallel to the limbus were made using 30-gauge thin-wall needles. The haptics of an IOL were externalized using the needles and cauterized to make a flange of the haptics. The flange of the haptics were pushed back and fixed into the scleral tunnels (Fig. 4.9).

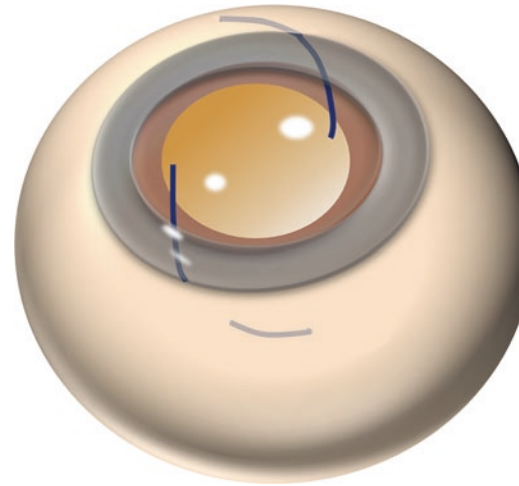

Fig. 4.8 Gabor and Pavlidis sutureless scleral interlaminar fixation of IOL

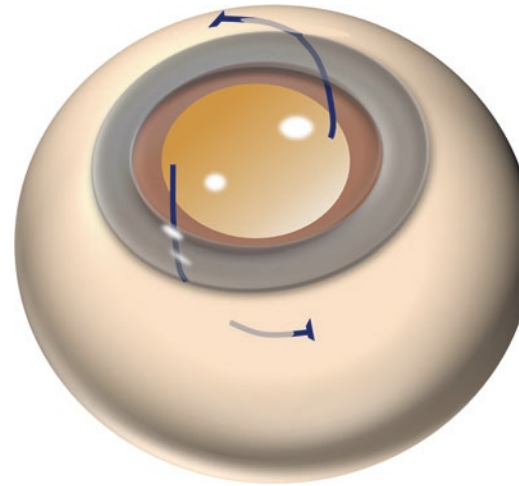

Fig. 4.9 Yamane's new technique for scleral IOL fixation

Scleral fixation using fibrin glue

The fibrin glue method, proposed by Agarwal et al. [23] in 2008, is another relatively new scleral fixation method without the use of sutures. Agarwal et al. have used the design of the scleral flap to take out the haptics of the IOL and fix it to scleral flaps using fibrin glue (Fig. 4.10). Common complications include tilt and dislocation of the IOL, IOL haptics prolapse, corneal endothelial injury, and secondary glaucoma [24].

Personal experience or matters need attention

In treating lens dislocation, we must judge the degree of lens dislocation and observe the lens position using CT and a slit lamp. We should pay attention to the patient's position (sitting or supine position). According to the degree of lens dislocation, different treatment methods could be used.

Lens hardness is an important factor to take out the lens. If the nucleus of the lens is soft, it can be directly aspirated. If the segment of hard nucleus falls into the vitreous cavity, heavy water may be used to float the lens and remove it by phacoemulsification; if the nucleus is too hard,

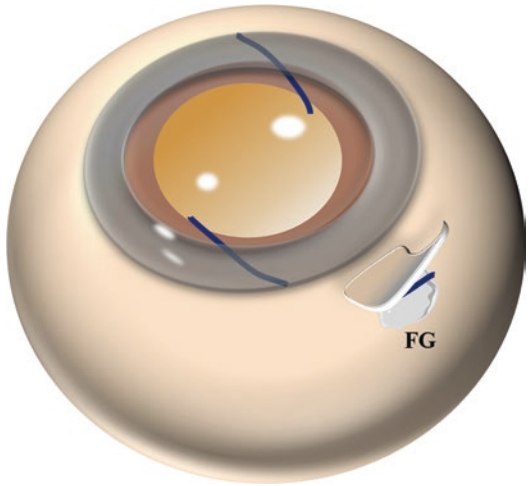

Fig. 4.10 Scleral fixation with bio glue of IOL. *FG* fibrin glue

smashing guided by ultrasound is difficult, and causing retinal damage is easy. We may use heavy water to remove the lens nucleus from the limbal incision.

For lens dislocation caused by trauma, a clear understanding of the injury is the basis of a good treatment design. Traumatic lens dislocation is often associated with other ocular injuries. It should be led or participated by experienced cataract surgeons and retinal specialist surgeons.

Surgical Video

See Videos 4.1 and 4.2.

References

1. Ke G, Zhou E, Zhu K, Wei Y, Wang Z, Jia Y, Wang S, Gu Y. Retinal break associated with traumatic lens dislocation or subluxation requiring vitrectomy[J]. Graefes Arch Clin Exp Ophthalmol. 2020;258(3):693–697.
2. Jarrett WH II. Dislocation of the lens. A study of 166 hospitalized cases[J]. Arch Ophthalmol. 1967;78(3):289–96.
3. Choi DY, Kim JG, Song BJ. Surgical management of crystalline lens dislocation into the anterior chamber with corneal touch and secondary glaucoma[J]. J Cataract Refract Surg. 2004;30(3):718–21.
4. Khokhar S, Agrawal S, Gupta S, et al. Epidemiology of traumatic lenticular subluxation in India[J]. Int Ophthalmol. 2014;34(2):197–204.
5. Viestenz A, Küchle M. Retrospective analysis of 417 cases of contusion and rupture of the globe with frequent avoidable causes of trauma: the Erlangen Ocular Contusion-Registry (EOCR) 1985–1995[J]. Klin Monbl Augenheilkd. 2001;218(10):662–9.
6. Salehi-Had H, Turalba A. Management of traumatic crystalline lens subluxation and dislocation[J]. Int Ophthalmol Clin. 2010;50(1):167–79.
7. Lee S, Hayward A, Bellamkonda VR. Traumatic lens dislocation[J]. Int J Emerg Med. 2015;8:16.
8. Fiorentzis M, Viestenz A, Heichel J, Seitz B, Hammer T, Viestenz A. Methods of fixation of intraocular lenses according to the anatomical structures in trauma eyes[J]. Clin Anat. 2018;31(1):6–15.
9. Kandar AK. Combined special capsular tension ring and toric IOL implantation for management of post-DALK high regular astigmatism with subluxated traumatic cataract[J]. Indian J Ophthalmol. 2014;62(7):819–22.
10. Dalma-Weiszhausz J, Franco-Cardenas V, Dalma A. A modified technique for extracting a dislocated lens with perfluorocarbon liquids and viscoelastics[J]. Ophthalmic Surg Lasers Imaging. 2010;41(5):572–4.
11. Grewal DS, Basti S, Singh Grewal SP. Femtosecond laser-assisted cataract surgery in a subluxated traumatic cataract[J]. J Cataract Refract Surg. 2014;40(7):1239–40.
12. Gabor SG, Pavlidis MM. Sutureless intrascleral posterior chamber intraocular lens fixation[J]. J Cataract Refract Surg. 2007;33(11):1851–4.
13. Wagoner MD, Cox TA, Ariyasu RG, et al. Intraocular lens implantation in the absence of capsular support: a report by the American Academy of Ophthalmology[J]. Ophthalmology. 2003;110(4):840–59.
14. Gonnermann J, Klamann MK, Maier AK, et al. Visual outcome and complications after posterior iris-claw aphakic intraocular lens implantation[J]. J Cataract Refract Surg. 2012;38(12):2139–43.
15. Koss MJ, Kohnen T. Intraocular architecture of secondary implanted anterior chamber iris-claw lenses in aphakic eyes evaluated with anterior segment optical coherence tomography[J]. Br J Ophthalmol. 2009;93(10):1301–6.
16. Girard LJ. PC-IOL implantation in the absence of posterior capsular support[J]. Ophthalmic Surg. 1988;19(9):680. 682
17. Melamud A, Topilow JS, Cai L, et al. Pars plana vitrectomy combined with either secondary scleral-fixated or anterior chamber intraocular lens implantation[J]. Am J Ophthalmol. 2016;168:177–82.
18. Nakashizuka H, Shimada H, Iwasaki Y, et al. Pars plana suture fixation for intraocular lenses dislocated into the vitreous cavity using a closed-eye cow-hitch technique[J]. J Cataract Refract Surg. 2004;30(2):302–6.
19. Szurman P, Petermeier K, Aisenbrey S, et al. Z-suture: a new knotless technique for transscleral suture fixation of intraocular implants[J]. Br J Ophthalmol. 2010;94(2):167–9.
20. Bading G, Hillenkamp J, Sachs HG, et al. Long-term safety and functional outcome of combined pars plana vitrectomy and scleral-fixated sutured poste-

rior chamber lens implantation[J]. Am J Ophthalmol. 2007;144(3):371–7.
21. Zhu X, Zhang Y, He W, Ye H, Jiang C, Lu Y. Tilt, Decentration, and Internal Higher-Order Aberrations of Sutured Posterior-Chamber Intraocular Lenses in Patients with Open Globe Injuries[J]. J Ophthalmol. 2017;2017:3517461.
22. Yamane S, Sato S, Maruyama-Inoue M, et al. Flanged intrascleral intraocular lens fixation with double-needle technique[J]. Ophthalmology. 2017;124(8):1136–42.
23. Agarwal A, Kumar DA, Jacob S, et al. Fibrin glue-assisted sutureless posterior chamber intraocular lens implantation in eyes with deficient posterior capsules[J]. J Cataract Refract Surg. 2008;34(9):1433–8.
24. Narang P, Agarwal A. Glued intrascleral haptic fixation of an intraocular lens[J]. Indian J Ophthalmol. 2017;65(12):1370–80.

Intraocular Hemorrhage

5

Juliana Mascato, Guilherme Guedes, Pedro Rebello, Daniel Lani, and Rodrigo Brant

Abstract

Post-traumatic intraocular hemorrhage is a major challenge for the ophthalmologist. Hyphema and vitreous hemorrhage are frequent presentations in ocular trauma. They occur most commonly in young males. When treated properly, they may not leave severe sequelae. However, complications such as increased intraocular pressure, hematic corneal impregnation, retinal detachment, vitreoretinal proliferation, macular hole, choroidal ruptures, among others, can result in low final visual acuity. The intensity of the trauma along with its mechanism are directly related to the visual prognosis. Management can be expectant, clinical or surgical, depending on the severity of the case. In this chapter, a series of trauma cases with intraocular hemorrhage and their respective therapeutic approaches will be presented.

Keywords

Eye trauma · Traumatic hyphema · Vitreous hemorrhage · Pars plana vitrectomy

5.1 Introduction

Post-traumatic intraocular hemorrhage is a major challenge for the ophthalmologist. Hyphema and vitreous hemorrhage are frequent presentations in ocular trauma. They occur most commonly in young males [1]. When treated properly, they may not leave severe sequelae [2].

However, complications such as increased intraocular pressure, hematic corneal impregnation, retinal detachment, vitreoretinal proliferation, macular hole, choroidal ruptures, among others, may result in low final visual acuity. The intensity of the trauma and the related mechanism are directly related to the visual prognosis. The management can be expectant, clinical or surgical, depending on the severity [3, 4].

5.2 Definition

Post-traumatic intraocular hemorrhage can affect the anterior and/or posterior segment of the eye. Traumatic hyphema may occur due to damage to the iris and/or the ciliary body [4]. Traumatic vit-

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reous hemorrhage may be the result of trauma to the ciliary body, choroid, and/or retina.

5.2.1 Hyphema

The term Hyphema describes the presence of red blood cells inside the anterior chamber, usually deposited in the inferior portion of the eye due to gravity. The main causes for the appearance of this pathology are ocular trauma and recent eye surgery. However, spontaneous hyphema may occur from other causes, such as neovascular glaucoma, or blood dyscrasias. It is classified into four grades that correspond to the level filled in the anterior chamber (AC): grade I—<1/3 of the AC; grade II—from 1/3 to 1/2 of AC; grade III—>1/2 AC; grade IV—AC fully covered [5].

In the setting of trauma leading to hyphema, patients may complain of decreased visual acuity, pain, headache, and photophobia. It is important to initially follow the patient for the first 48 to 72 h after the trauma, during which secondary bleeding can occur. It can cause permanent low visual acuity if not handled correctly. The main complications are corneal hematic impregnation, central retinal artery occlusion, and glaucoma [6–9].

If hematic corneal impregnation occurs, it can last for a long period of time, gradually disappearing through a process of phagocytosis of the deposited blood pigment, and the total clearing process can take up to 2 or 3 years. Usually the cornea clearing is incomplete, resulting in a permanent corneal opacity [6–9]. In cases of hyphema grade IV, surgical anterior chamber aspiration should be indicated in the first days of the trauma to avoid permanent corneal clouding.

Increase of intraocular pressure (IOP) can occur through several mechanisms, such as angular recession, anterior or posterior peripheral synechiae and ghost cell glaucoma. In cases that do not improve with drug treatment, surgical management is indicated for pressure control. Mild cases can be managed clinically, and the treatment includes elevated head position (over 30°), increased fluid intake, use of corticosteroids, cycloplegics, and fibrinolytic agents, and suspension of anticoagulants drugs [10].

However, surgical approach should be considered in cases in which the intraocular pressure remains high, there is hematic impregnation in the cornea or persistent blood clot. Surgical treatment is indicated if the IOP is higher than 50 mmHg for 5 days or over 35 mmHg for 7 days. Cases of corneal endothelium dysfunction or previous glaucoma deserve special attention [11].

Anterior chamber blood aspiration, with or without the use fibrinolytic agents, must be indicated early in the treatment of Hyphema grade IV. The treatment goal is clear the visual axis and remove floating debris. Excessive removal of the hyphema or blood clots may result in persistent bleeding, and should be avoided [12].

5.2.2 Vitreous Hemorrhage

Vitreous hemorrhage may be due to bleeding from the ciliary body, choroid, and/or retina. Carefull retinal examination with scleral indentation should be performed, looking for retinal tears that may require treatment [13].

When fundus examination is not possible due to media opacity, ultrasonography (USG) must be performed. If there is no documented injury, observation with serial Ultrasound examinations may be considered. If USG detect any retinal tear and laser treatment is impossible due to vitreous hemorrhage, PPV should be indicated in order to avoid the progression of the lesion to a retinal detachment [14, 15].

When pars plana vitrectomy is indicated in patients with dense vitreous hemorrhage, it is recommended to start the surgery with the infusion in the anterior chamber to ensure that it is not in the suprachoroidal space. If there is Choroidal detachment, drainage should be considered before the PPV. The infusion can be safely positioned into pars plana as soon as the surgeon achieves adequate visualization for the posterior pole and the sites of trocar insertion [16].

The clots in the hemorrhagic choroidal detachment or in the subretinal space will be best managed after total liquefaction, which

is achieved after 14 days of the bleeding. Serial ultrasound examination can help the surgeon to identify the liquefaction of the blood clots, providing the best timing to indicate the secondary surgical procedure [17–19].

When hemorrhagic choroidal detachment is limited to one quadrant and is only slightly elevated, drainage may not be necessary and spontaneous absorption may occur in weeks or months. However, when the detachment is extensive or presenting "kissing choroidals," surgical drainage is mandatory to avoid complications like secondary retinal detachment due to vitreoretinal proliferation [19].

Anterior vitrectomy should be performed in the presence of dislocated lens or identification of the vitreous in the anterior chamber [20].

The surgical procedure starts with the conjunctival peritomy followed by pexy and exposure of the rectus muscles of the quadrant that will be drained. The drainage can be done through a full thickness scleral flap or using the vitrectomy valves in cases of bulous choroidal. The entrance in the suprachoroidal space should be at least 8 mm from the limbus. Before the drainage begins, the infusion is positioned in the anterior chamber through a limbic paracentesis and opened to pressurize the eye [20, 21].

Excessive compression of the edges adjacent to the scleral flap should be avoided, as it predisposes retinal incarceration [20, 21].

After draining, the infusion is removed and the sclerotomy may or may not be sutured. The sutures are removed and the conjunctiva is closed. Visual prognosis depends on multiple factors, such as: retinal detachment, choroidal rupture, retinal incarceration, vitreoretinal proliferation, macular hole, and Berlin edema. Some of these conditions will only be recognized during or after vitrectomy and may require several surgeries later [20, 21].

In the presence of corneal or scleral laceration, associated with vitreous hemorrhage, ocular closure should be done immediately; however, the posterior segment approach can be done at first or delayed, depending on the surgeon's preference. 72-h vitrectomy is associated with a better prognosis. Delaying the secondary procedure after trauma is related to a reduction in the chance of severe intraocular bleeding, mainly due to less uveal congestion, in addition to facilitating the detachment of the posterior hyaloid and removal of the liquefied blood clots [22]. In the presence of intraocular foreign body (IOFB), there is an increased chance of endophthalmitis if the IOFB remains in the eye for more than 24-h, in addition to facilitating the formation of a fibrous capsule around it, which is difficult to remove surgically. Perforations that extend to the posterior pole, with difficult surgical access, may have spontaneous closure with the proliferation of fibroblastic tissue after a few days. Delayed surgery in those cases reduced the chance of iatrogenic tissue prolapse during vitrectomy [23].

Retinal detachment after trauma can be hemorrhagic (when heavy bleeding from the choroid creates a blood pocket and detaches the retina), rhegmatogenous (when there is an associated retinal tear), or tractional (when there is vitreoretinal proliferation or retinal incarceration in the wound, for example) [24].

In the first case, the removal of subretinal blood can be quite difficult if the surgery is perfomed early. In the second, the use of scleral buckle associated with PPV can lead to best post operative anatomical results, due to the lower chance of redettachment. Finally, in the third, PVR peeling and techniques to relieve tractions, such as relaxing retinotomies or retinectomies, in addition to scleral buckle at the traction site, are indicated [25].

5.3 Cases

Case 1

A 7-year-old boy came to the hospital 2 weeks after a penetrating trauma with a BB gun pellet in the right eye. He denied low vision acuity. The visual acuity was 20/20 in both eyes with preserved photomotor reflexes. Slit-lamp examination of the right eye demonstrated edema of the eyelid, hyposphagma and the other eye structures were preserved (Fig. 5.1–1). Funduscopy of the left eye was normal (Fig. 5.1–2), however, on the temporal retinal of the right eye a subretinal hemorrhage and choroidal rupture was identified (Fig. 5.1–3). The cranial X rays showed the presence

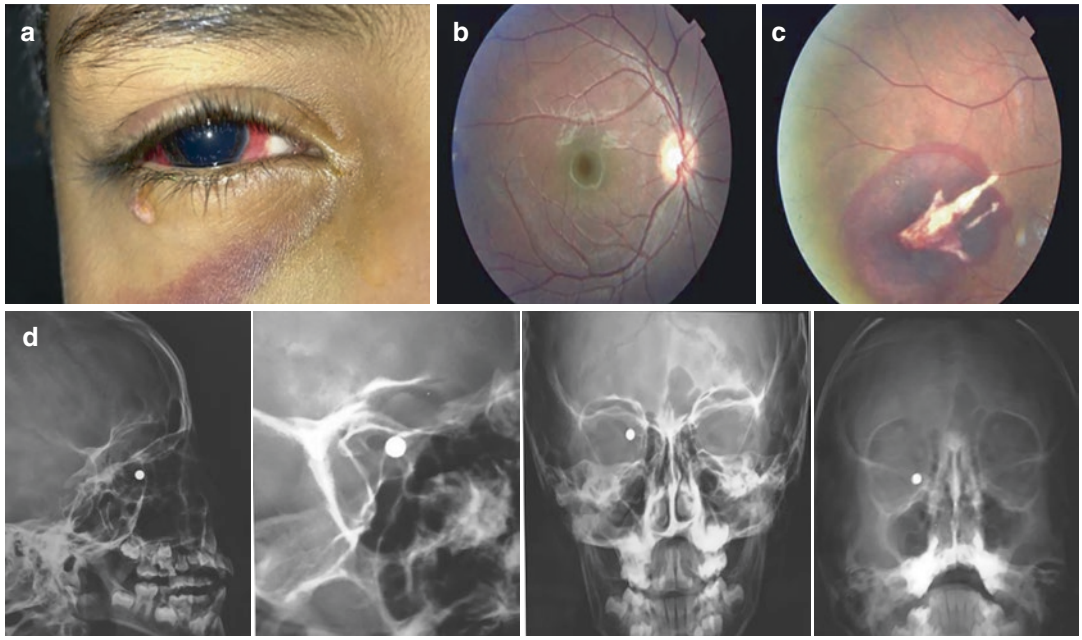

Fig. 5.1 (**a**) A 7-year-old boy with edema of eyelid, hyphosphagma; anterior chamber exam was normal. (**b**) Posterior pole of the right eye. (**c**) Subretinal hemorrhage and choroidal rupture resulting in the temporal retina. (**d**) Cranial X ray showing foreign body in posterior to the eye wall

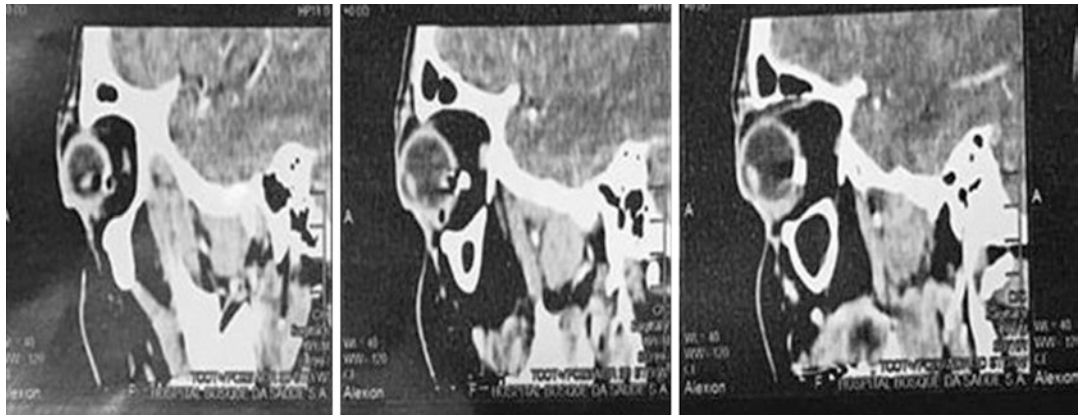

Fig. 5.2 9.3 × 4.4 × 6.1 mm metallic foreign body, located in the right retro-orbital region inferior to the optic nerve

of an foreign body (Fig. 5.1–4). The observation was indicated due to the object's proximity to the optic nerve.

Case 2

A Male patient, 50 years old, reports low visual acuity in the right eye after penetrating trauma in his workplace. Scleral sutures were performed in another service and an orbital CT scan was requested to assess the possibility of intraocular foreign body. A 9.3 × 4.4 × 6.1 mm metallic foreign body, located in the right retro-orbital region inferior to optic nerve was visualized (Fig. 5.2). Ocular ultrasound was performed, which showed that the object had partially transfixed the sclera (Fig. 5.3).

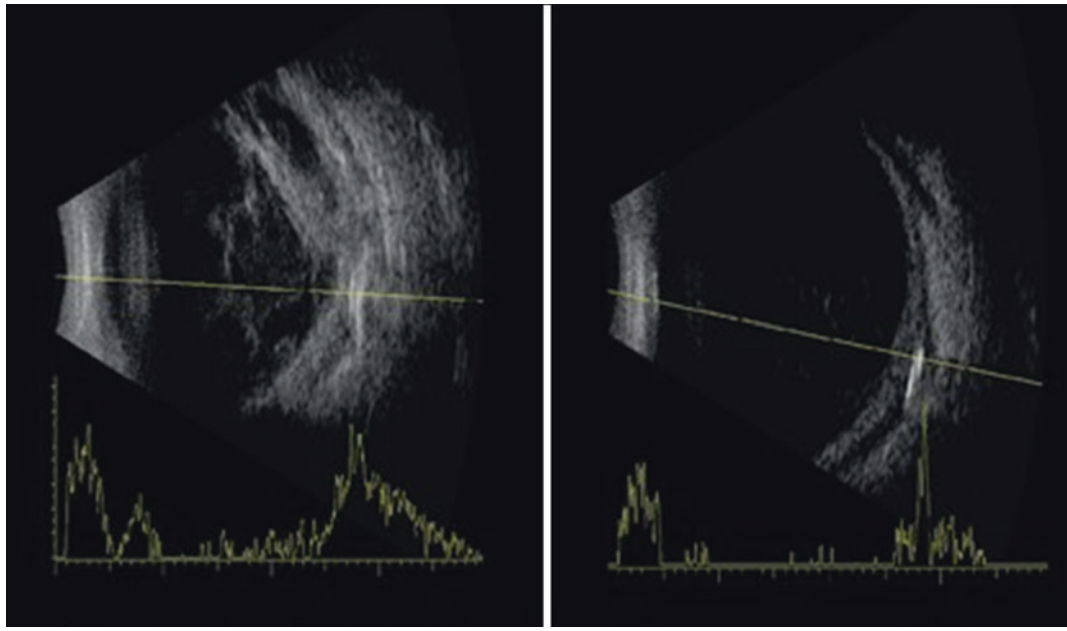

Fig. 5.3 Partial detachment of posterior hyaloid and adhesions in the papilla, equator in nasal meridians and in the periphery of temporal meridians. Hemorrhagic and/or inflammatory vitreous and subhyaloid process. Image showing inferior temporal juxtapapillary metallic IOFB (7:00 AM meridian), which partially transfixes the ocular wall (sclera and choroid)

The surgical procedure was delayed for 14 days to allow for clot liquefaction. PPV with Phacoemulsification and intraocular lens implantation was indicated. Dense vitreous opacities can make it difficult to visualize the anterior capsule during capsulorexis. The use of accessory posterior lighting facilitates capsular visualization by backlighting (moonlight capsulorexis). Phacoemulsification was performed without complications; however, there was a lower temporal zonular disinsertion. Due to the possibility of removing the intraocular foreign body through the anterior chamber, it was decided not to implant the intraocular lens at the end of the vitrectomy.

PPV was perfomed, followed by shaving of the vitreous base. A dense plaque of inferior hemorrhage, which extended from the vitreous base to the posterior pole was observed, which was cautionsly removed.

The fibrotic membranes were removed at its periphery with the aid of retinal forceps, without causing iatrogenic tear or retinal detachment. It was impossible to visualize the foreign body, which was probably lodged in the sclera. The removal attempt was aborted to avoid iatrogenic damage to the eye. Endolaser was performed at the edges and in the center of the lesion, and the IOFB was left in place since it was not the the intraocular space.

The three-piece intraocular lens was implated without complications in the ciliary sulcus. Fluid-air exchange was carried out and it was not necessary to add a tamponade agent, such as silicone oil or gas (Fig. 5.4).

Case 3

A male patient, 35 years old, admitted to our emergency room, after a blunt traumatic accident in the left hemiface. On ophthalmological examination, he presented laceration of the lower eyelid and left lower temporal sclera. During surgical exploration of the lower eyelid region, a 6 × 13 mm metallic foreign body was detected and removed, followed by eyelid reconstruction (Fig. 5.5). Shortly thereafter,

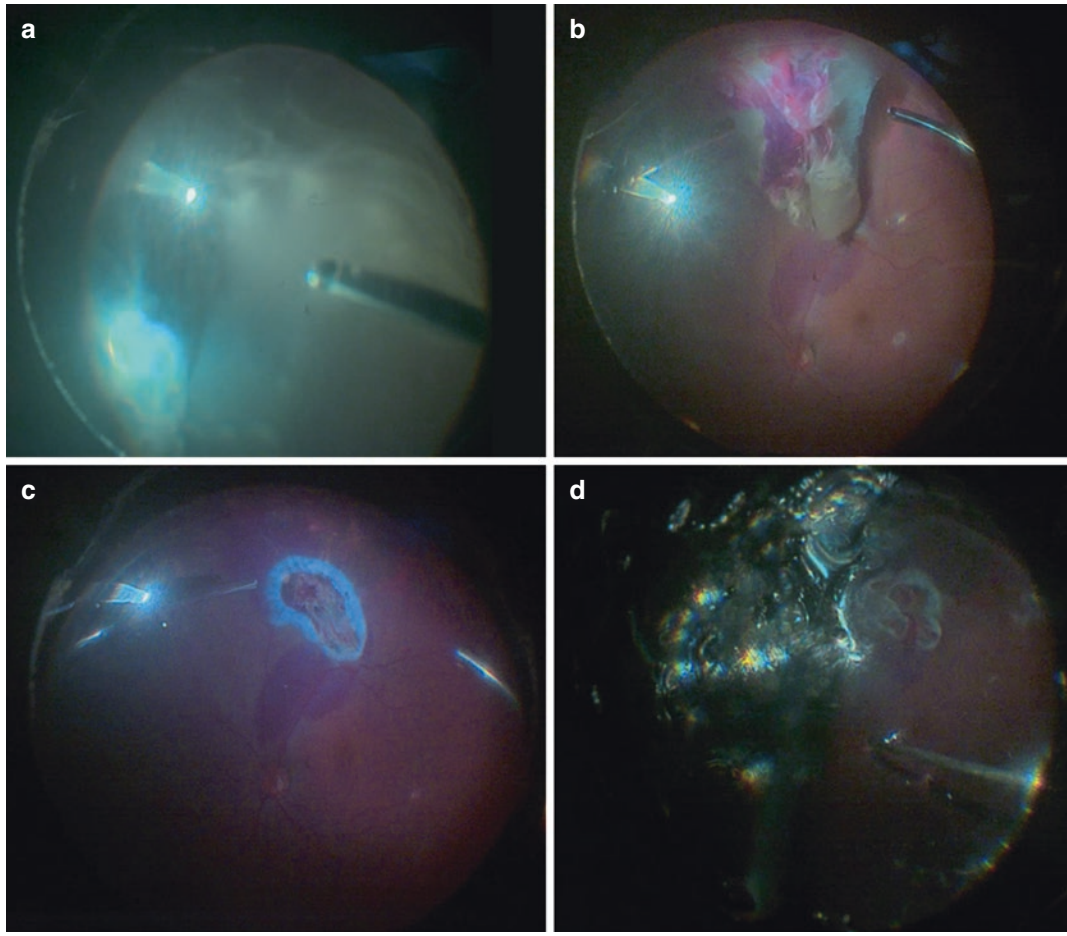

Fig. 5.4 (**a**) Removal of vitreous membranes. (**b**) Dissection of the adherent vitreous. (**c**) Endolaser enclosing the entire trauma area. (**d**) Air fluid exchange

conjunctival peritomy and exploration of the extent of the lesion was performed and scleral suture made. It evolved on the first postoperative day with an important hyphema, making it impossible to evaluate the posterior segment, and intraocular pressure of 12 mmHg. We opted for conservative management through the use of topical corticosteroids, cycloplegics, topical antibiotics, and serial ultrasounds. Ocular ultrasound revealed hyperreflective echoes in the vitreous cavity (probable vitreous hemorrhage) with retinal detachment and retinal incarceration in the lower temporal region (laceration site) (Fig. 5.6). A new surgical approach was performed 14 days after the trauma, in which the anterior chamber was washed, comprising of phacoaspiration associated with posterior pars plana vitrectomy.

At the surgery beginning, a corneal paracentesis was performed and an infusion line was placed in the anterior chamber. As the blood was not clearing with online the saline irrigation, it was decided to cut and aspirate the clot with vitrectomy probe. After that, the ocular media became clear. Phacoemulsification was perfomed, and hydrophobic acrylic intraocular lens

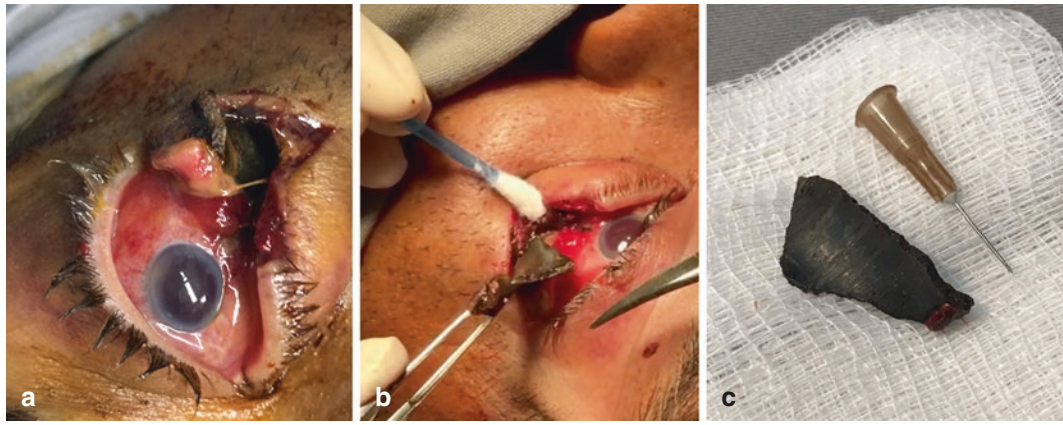

Fig. 5.5 (**a**) Laceration of the lower eyelid and left lower temporal sclera. (**b**, **c**) During surgical exploration of the lower eyelid region, a 6 × 13 mm metallic foreign body was detected and removed followed by eyelid reconstruction

was implant in the capsular bag. The infusion line was placed in the vitreous cavity. After entering the vitreous cavity, blood adhered to a fibrous membrane, was visualized and an orifice was created in this region, in order to visualize the dissection plane. The retina was incarcerated in the scleral wall, through the primary trauma wound. 360º retinectomy was perfomed to removed the retina from the incarceration. Perfluorocarbon liquid was placed, flattening the entire retina. Endolaser was them performed and silicone oil implanted at the end of the surgery (Fig. 5.7).

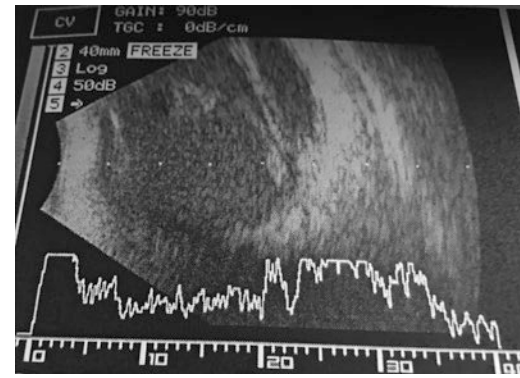

Fig. 5.6 Ocular ultrasound revealed hyperreflective echoes in the vitreous cavity (probable vitreous hemorrhage) with detachment and retinal incarceration in the lower temporal region (laceration site)

Case 4

An 85-year-old woman presented with low visual acuity and pain after a complicated cataract surgery. During the procedure, the patient had posterior capsular rupture, with dropped lens fragments. A PPV was perfomed to remove the lens fragments, and at the end of the procedure, after IOL positioning in the ciliary sulcus, the surgeon noticed a red reflex loss immediately suturing the corneal incisions (Fig. 5.8–1). An ocular ultrasound evidenced retinal and choroidal detachment almost forming "kissing choroidals." Anterior chamber aspiration and drainage of the choroidal detachment was indicated after 14 days of the first surgery. After the AC aspiration with an anterior chamber infusion (Fig. 5.8–2), limbal conjunctival peritomy was performed and traction sutures placed in the rectus muscles. Scleral flaps were constructed 8 mm from the limbus, in the region of the most bullous choroidal detachments (Fig. 5.8–3). Once the sclerotomy was done, liquefied blood flowed out the incision (Fig. 5.8–4). PPV was perfomed to attach the retina, and silicone was left as tamponade.

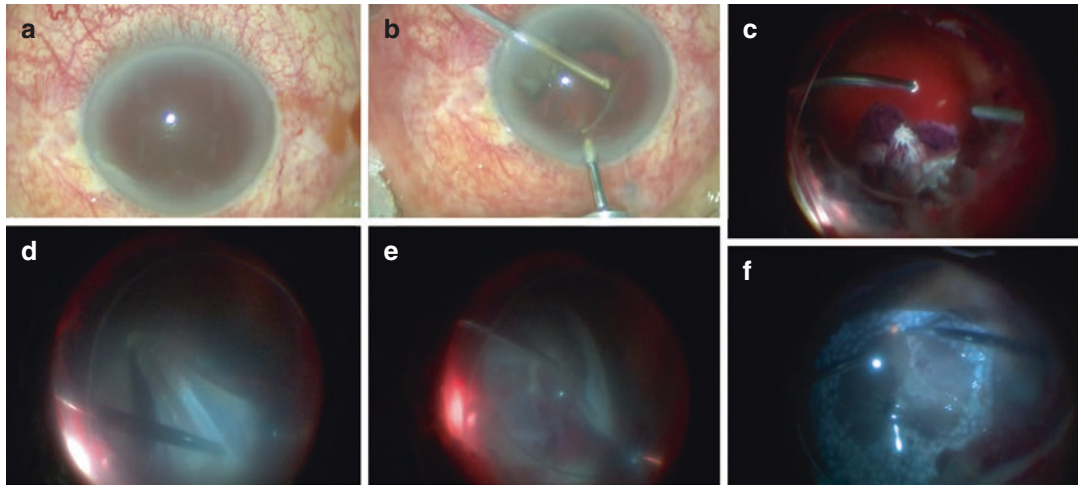

Fig. 5.7 (**a**, **b**) Total AC clot and its removal. (**c**, **d**) PPV and visualisation of dissection plane. (**e**, **f**) Retinectomy of the edges. Flattening of the entire retina. Endolaser 360º performed and silicone oil implanted

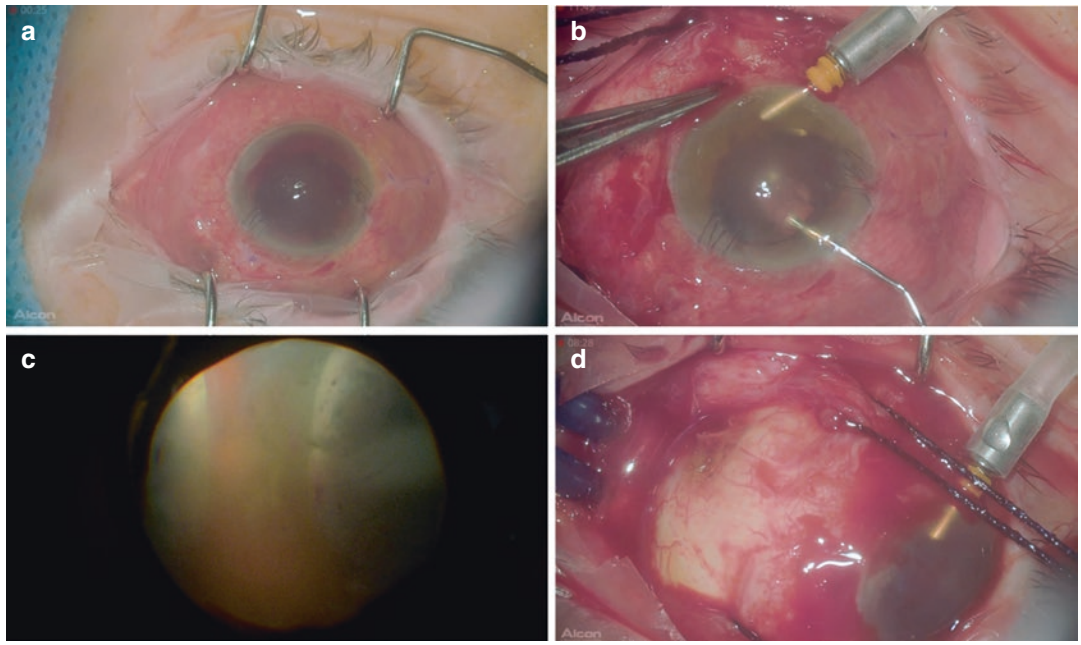

Fig. 5.8 (**a**) Hyphema in the anterior chamber with hemorrhagic choroidal detachment secondary to filtering surgery. (**b**) AC lavage with irrigation maintainer. (**c**) "kissing choroidals" detachment. (**d**) 0-0 cotton thread to exposure scleral flap region

5.4 Important Signs, Examinations, Diagnosis, Surgical Procedures, or Postoperative Treatment for Complications

It is important to define the mechanism of the trauma (whether blunt, penetrating, or perforating), the extent of the damage (if it has damaged extraocular structures), and the presence of risk factors for bleeding (use of anticoagulants, for example).

A complete anamnesis and physical examination are always necessary, especially in elderly patients, in whom a mild head trauma can lead to subdural hematoma and progressive lowering of the level of consciousness [26].

Visual acuity and pupillary examination can provide evidence of visual prognosis, such as traumatic optic neuropathy, suprachoroidal hemorrhage, and occult scleral rupture. Imaging exams are used to assess the integrity of the eyeball and its adjacent structures, as well as the presence of a foreign body [27].

Computarized Tomography (CT Scan) is the exam of choice, as it can be used safely both to detect a foreign body location and to assess adjacent eye structures, even when it is compromised. Despite being of great value for metallic objects, wooden or plastic components are difficult to visualize in the CT Scan. Magnetic Resonance Imaging (MRI) is not recommended for primary trauma assessment. If there is a metallic IOFB, MRI can cause its movement and lead to iatrogenic damage. However, if mettalic IOFBs are discarded, MRI is the best exam to locate IOFBs made of wood or plastic [27].

Ultrasonography is not indicated as the first complementary exam in the Emergency room, because the ocular manipulation involvedin the aquisition of the images in cases of penetrating or perforating trauma, can lead to extrusion of intraocular content. However, it is useful in the evaluation of vitreous hemorrhage, retinal detachment, or choroidal detachment, after the first week of primary repair [28].

5.5 Personal Experience

Surgical treatment of trauma-induced intraocular hemorrhage requires great expertise from the surgeon in view of the challenges that may arise in each case, which sometimes are not previously identified. We opted, in most cases, to perform the reconstruction of the eyeball, with corneal and/or scleral suture as a primary procedure, if needed.

Posteriorly, usually in 14 days, we perform the vitrectomy of cases with dense vitreous hemorrhage and retinal detachment. We also wait to perform the choroidal drainage of liquefied blood in cases of hemorrhagic choroidal detachment, after performing serial ocular ultrasonography. In the presence of an intraocular foreign body, it is preferable to perform vitrectomy earlier, to avoid retinal induced IOFB toxicity, associated with reconstruction of the eyeball, if necessary.

The exact location of the foreign body remains obscure, even with imaging exams, the exact location of the foreign body (intra ocular, orbital or mixed location) may be discovered only during the surgical procedure for removal. Objects that are mobile in the vitreous are removed easiely than those that are attached to the sclera. In some cases, it is debatable if the IOFB should or not be removed, especially when retro orbital or close to the optic nerve and macular area.

Prognosis orientation to the patient and family is important. We avoid eviscerate the eyeball at first. The attempt to preserve the eye and possible future complications, such as endophthalmitis and sympathetic ophthalmia, are discussed. The visual result depends not only on the preservation of the anatomy, but also on the quality of the remaining

tissue. Multiple surgeries may be necessary, despite of the good initial recovery.

5.6 Specific Challenges

The intraocular pressure increase results from a possible secondary glaucoma, that can elevate the chance of expulsive hemorrhage, specially if not handled correctly. This should be identified as early as possible and requires the ability to finish the surgery quickly with sutures of the incisions.

Intense intraocular hemorrhage may occur, requiring maneuvers such as the use of endolaser or endodiathermy to cauterize the bleeding focus, if possible. In addition, the increased infusion pressure or the use of perfluorocarbon can assist in this process and reduce bleeding, even if temporary.

The presence of extensive subretinal hemorrhage in the macular region implies a poor prognosis. Its removal can be done with an extensive retinectomy and aspiration of the blood with vitreophage or by a less aggressive management, such as injection of subretinal tissue plasminogen activator, for example, according to the extent of the bleeding or the surgeon's preference. Choroidal tears or Berlin edema can compromise visual acuity despite good bleeding resolution and surgical outcome.

References

1. Tongu MT, Bison SH, Souza LB, Scarpi MJ. Aspectos epidemiológicos do traumatismo ocular fechado. Arq Bras Oftalmol. 2001;64:57–61.
2. Berríos RR, Dreyer EB. Traumatic hyphema. Int Ophthalmol Clin. 1995;35:93–103.
3. Shingleton BJ, Hersh PS. Traumatic hyphema. In: Shingleton BJ, Hersh PS, Kenyon KR, editors. Eye trauma. St Louis: Mosby; 1998. p. 104–14.
4. Walton W, Hagen SV, Grigorian R, Zarbin M. Management of traumatic hyphema. Surv Ophthalmol. 2002;47:297–334.
5. Sankar PS, Chen TC, Grosskreutz CL, Pasquale LR. Traumatic hyphema. Int Ophthalmol Clin. 2002;42:57.
6. Crouch ER Jr, Crouch ER. Management of traumatic hyphema: therapeutic options. J Pediatric Ophthalmol Strabismus. 1999;36:238.
7. Shiuey Y, Lucarelli MJ. Traumatic hyphema: outcomes of outpatient management. Ophthalmology. 1998;105:851.
8. Gharaibeh A, Savage HI, Scherer RW, et al. Medical interventions for traumatic hyphema. Cochrane Database Syst Rev. 2019;1:CD005431.
9. Shiuey Y, Lucarelli MJ. Traumatic hyphema: outcomes of outpatient management. Ophthalmology. 1998;105:851.
10. Witteman GJ, Brubaker SJ, Johnson M, Marks RG. The incidence of rebleeding in traumatic hyphema. Ann Ophthalmol. 1985;17:525.
11. Graul TA, Ruttum MS, Lloyd MA, Radius RL, Hyndiuk RA. Trabeculectomy for traumatic hyphema with increased intraocular pressure. Am J Ophthalmol. 1994;117:155–9.
12. Read J. Traumatic hyphema: surgical vs medical management. Ann Ophthalmol. 1975;7:659.
13. Jena S, Tripathy K. Vitreous hemorrhage. [Updated 2020 Aug 8]. In: StatPearls [Internet]. Treasure Island, FL: StatPearls Publishing; 2020. https://www.ncbi.nlm.nih.gov/books/NBK559131/
14. Dana MR, Werner MS, Viana MA, Shapiro MJ. Spontaneous and traumatic vitreous hemorrhage. Ophthalmology. 1993.
15. Berdahl JP, Mruthyunjaya P. Vitreous hemorrhage: diagnosis and treatment. In: [Internet]. EyeNet Magazine Publishing; 2007. https://www.aao.org/eyenet/article/vitreous-hemorrhage-diagnosis-treatment-2
16. Learned D, Eliott D. Management of delayed suprachoroidal hemorrhage after glaucoma surgery. Semin Ophthalmol. 2018;33(1):59–63.
17. Birt CM, Berger AR. Anterior chamber maintenance during drainage of a suprachoroidal hemorrhage in two phakic eyes. Ophthalmic Surg Lasers. 1996;27(9):739–45.
18. Wirostko WJ, Han DP, Mieler WF, Pulido JS, Connor TB Jr, Kuhn E. Suprachoroidal hemorrhage: outcome of surgical management according to hemorrhage severity. Ophthalmology. 1998;105(12):2271–5.
19. Mei H, Xing Y, Yang A, Wang J, Xu Y, Heiligenhaus A. Suprachoroidal hemorrhage during pars plana vitrectomy in traumatized eyes. Retina. 2009;29(4):473–6.
20. Vuković D, Petrović S, Paović P. Secondary surgical management of suprachoroidal hemorrhage during pars plana vitrectomy. Vojnosanit Pregl. 2015;72(3):287–90.
21. Feretis E, Mourtzoukos S, Mangouritsas G, Kabanarou SA, Inoba K, Xirou T. Secondary management and outcome of massive suprachoroidal hemorrhage. Eur J Ophthalmol. 2006;16(6):835–40.
22. Kathryn C. Management of open globe injuries. Int Ophthalmol Clin. 1999;39(1):59–69.
23. Loporchio D, Mukkamala L, Gorukanti K, Zarbin M, Langer P, Bhagat N. Intraocular foreign bodies: a review. Surv Ophthalmol. 2016;61(5):582–96.
24. Stryjewski TP, Andreoli CM, Eliott D. Retinal detachment after open globe injury. Ophthalmology. 2014;121(1):327–33. https://doi.org/10.1016/j.ophtha.2013.06.045. Epub 2013 Sep 4.

25. Orban M, Islam YF, Haddock LJ. Timing and outcomes of vitreoretinal surgery after traumatic retinal detachment. J Ophthalmol. 2016;2016:4978973.
26. Wirbelauer C. Management of the red eye for the primary care physician. Am J Med. 2006;119(4):302–6.
27. Parke DW, Flynn HW, Fisher YL. Management of intraocular foreign bodies: a clinical flight plan. Can J Ophthalmol. 2013;48:8–12.
28. Lahham S, Shniter I, Thompson M, Le D, Chadha T, Mailhot T, Kang TL, Chiem A, Tseeng S, Fox JC. Point-of-care ultrasonography in the diagnosis of retinal detachment, vitreous hemorrhage, and vitreous detachment in the emergency department. JAMA Netw Open. 2019;2(4):e192162.

Open Globe Injury with Choroidal and Retinal Detachment

6

S. Natarajan, Sneha Makhija, and Astha Jain

Abstract

Traumatic retinal detachments can result from blunt or penetrating trauma. Suprachoroidal hemorrhage is a painful condition often seen in association with trauma. Diagnosis of retinal and choroidal detachments often requires a Bscan ultrasonic examination as vitreous hemorrhage precludes the view of the posterior segment and thus the clinical diagnosis. The presence of a retinal detachment along with choroidal detachment in the case of open globe injury is an indication for early intervention.

Keywords

Retinal detachment · Choroidal detachment
Choroidal drainage

6.1 Introduction

The international society of ocular trauma in their BETTS classification [1] classifies open globe injuries into rupture and laceration. Both of these may be associated with devastating complications of choroidal and retinal detachments. The reported incidence of retinal detachment in patients with rupture injuries stands at 42% [2]. Lacerations are further divided into Penetrating injuries, Intraocular foreign body, and Perforating injuries. Nineteen percent of laceration injuries are associated with traumatic retinal detachment, of which perforating injuries have the maximum chances of having a retinal detachment associated with it [2]. The crude incidence rate of retinal detachment after open globe trauma was 29% [2]. In a study done on 210 patients with open globe injuries, 91 (43.33%) patients showed choroidal detachment [3]. These were further divided into serous choroidal detachment, hemorrhagic choroidal detachment, and kissing choroidal detachment, serous detachments being the most common of these.

6.2 Definition

Traumatic retinal detachments can result from blunt or penetrating trauma. In literature, retinal detachments are said to be present at presentation in 3.4–35% of the cases [4–12]. Stryjewski TP et al. [2] reported that of the 255 cases of retinal detachments that they studied, only 27% were identified within the first 24 h of injury. A total of 47% were identified within 1 week and 72%

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within 1 month. Five percent detachments occurred 1 year after injury. 9% of the ones diagnosed were on the table in the operating room at the time of vitrectomy for another indication, such as nonclearing hemorrhage. Rhegmatogenous type of retinal detachment is most commonly associated with trauma.

The suprachoroidal space is normally virtual as the choroid is in close apposition to the sclera. Accumulation of serous fluid or blood in this space is called choroidal effusion or suprachoroidal hemorrhage, respectively. Suprachoroidal hemorrhage is a painful condition often seen in association with trauma or intraoperatively in some patients.

Diagnosis of retinal and choroidal detachments often requires a Bscan ultrasonic examination as vitreous hemorrhage precludes the view of the posterior segment and thus the clinical diagnosis.

Pieramici DJ et al. [13] had classified ocular injuries into three zones. A zone I injury is isolated to the cornea (including the limbus), a zone II injury involves the sclera no more than 5 mm posterior to the limbus, and a zone III injury involves the sclera more than 5 mm posterior to the limbus (Fig. 6.1).

Eyes presenting with vitreous hemorrhage, zone III injuries, low visual acuity of light perception, or no light perception are reported to have a notably high risk of detachments [2, 11, 14, 15].

6.3 Case Report

A 47-year-old male with a history of road traffic accident causing blunt trauma to his right eye presented to the emergency care unit of the ophthalmology department with complaint of pain, redness, diminution of vision, and bleeding from the right eye. On examination, the visual acuity of the right eye was the perception of light. Slit-lamp examination showed a corneoscleral tear along with uveal tissue prolapse. The lens status and posterior segment were not visualized. A right lower lid laceration was also noted. Left eye examination was within normal limits. He was taken up for emergency corneoscleral tear repair with anterior vitrectomy with intracameral antibiotic injection.

Postoperative anterior segment examination showed total hyphema with intact corneal sutures. Intraocular pressure was digitally high, and posterior details were not visualized. The lid wound was found to be healing well. Postoperative B-scan showed evidence of traumatic retinal detachment with choroidal detachment. The suprachoroidal space appeared to have a fluid with high reflectivity and irregular internal struc-

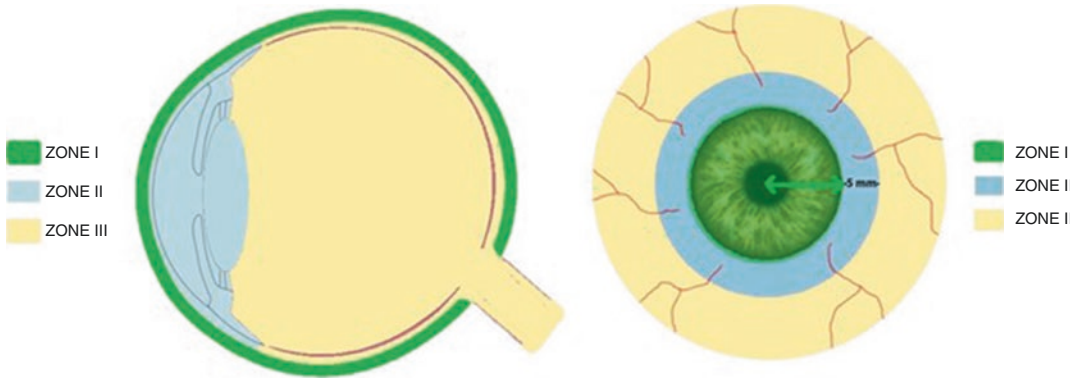

Fig. 6.1 Schematic representation of three zones of ocular injury [13]

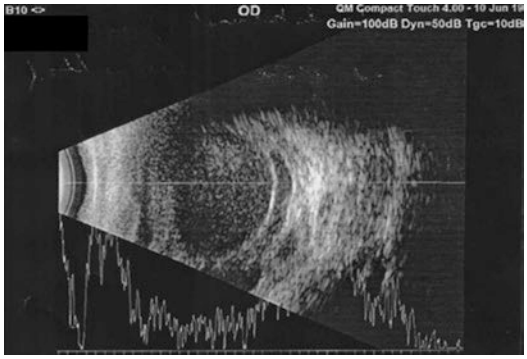

Fig. 6.2 B-scan in a case of blunt trauma showing evidence of vitreous hemorrhage with retinal detachment with hemorrhagic choroidal detachment with low to moderate spikes on A-Scan suggestive of liquified blood

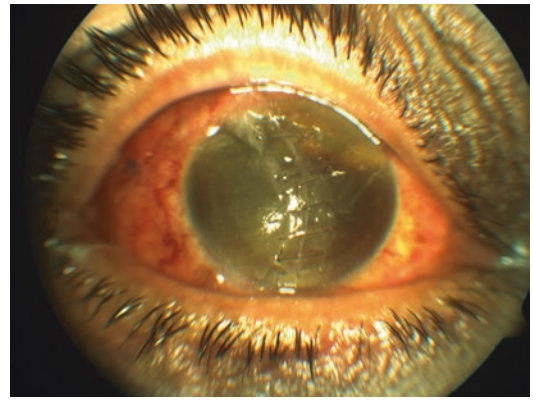

Fig. 6.3 Anterior segment photo showing repaired corneal tear with corneal sutures

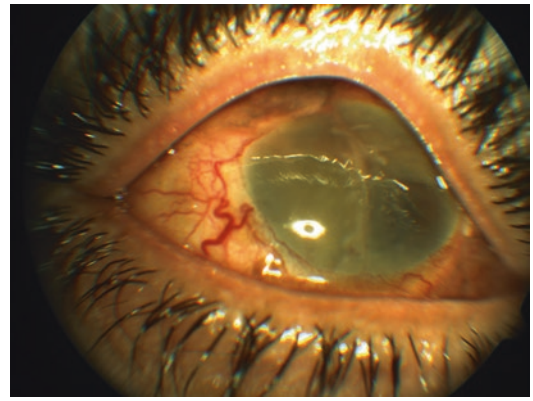

Fig. 6.4 At the final visit: corneal sutures were removed and a corneal scar was noted, the anterior chamber well formed

ture with low to moderate spikes on A-Scan suggestive of liquified blood. The vitreous cavity also showed multiple echoes with low to moderate spikes on the A-scan suggestive of vitreous hemorrhage. Lens echo was present (Fig. 6.2). Topical and systemic antibiotics, steroids, and cycloplegic agents were continued.

He underwent an anterior chamber wash with choroidal drainage with 23G Pars plana vitrectomy with silicone oil injection with intravitreal and sub-tenon steroid injection under local anesthesia. Medical and superior recti were tagged. Anterior chamber wash was done, followed by placing an anterior chamber maintainer. Choroidal drainage was done supero-nasally where there was maximum choroidal detachment. A scleral window was made to drain the choroidals. Pars plana vitrectomy was done to clear the vitreous hemorrhage. Severe proliferative vitreoretinopathy changes were noted. Fluid air exchange was carried out, followed by silicone oil injection. Poor posterior segment visualization due to corneal haze made the surgical maneuvering challenging.

Postoperatively, anterior segment examination revealed a well-formed anterior chamber (Fig. 6.3). The posterior segment was not clearly visible. At 1-month follow-up post-surgery, visual acuity of the right eye was the perception of light. Corneal sutures were removed and a corneal scar was noted; the anterior chamber was clear and well formed (Fig. 6.4). The retina appeared attached. Left eye examination was unremarkable.

Take away messages from this case:

1. Traumatic retinal + choroidal detachment has a poor prognosis in most cases.

2. The patient must be counseled about the need for multiple surgeries.
3. The presence of a concurrent corneal scar can make visualization of the posterior segment difficult.
4. Realistic expectations about the outcome of such a condition must be borne in mind by the patient, their relatives, and the treating team.

6.4 Approach to a patient with Traumatic Choroidal and Retinal Detachment

6.4.1 Preoperative Evaluation

6.4.1.1 History

A detailed history is of utmost importance while examining a patient of trauma. The circumstances of the injury in terms of the time since injury, mode of injury, use, and breakage of spectacles, any projectiles to the eye, prior medication or treatment taken elsewhere help plan and prognosticate the injury. It is also important to rule out and associated head or bodily injuries. One must notify the local police in case of assault or road traffic accidents, or any medicolegal cause of injury.

6.4.1.2 Examination

Examination of a patient of trauma starts with the patient entering the clinic. Watching the patient's gait, ability to maneuver around the place, ability to open the eye can give some signs to the ophthalmologist about the nature, severity, and urgency of the condition. A general systemic examination must be done to rule out an altered sensorium secondary to an associated head injury.

After taking a detailed history and a brief torchlight examination, the patient's visual acuity must be assessed. In cases where the lids are swollen, or conjunctival chemosis is present, one must mechanically retract the lids gently to record the visual acuity and examine the patient.

On slit-lamp examination, one must assess the extent of the injury and look for the extent of the corneal or scleral tear present. The status of the crystalline lens must be noted if possible.

The posterior segment examination may not always be possible in the presence of open globe injuries. Hence B-Scan may be helpful.

6.4.2 Imaging

Ultrasonography can be used in the office by the ophthalmologist and aids in planning the further course of treatment. In case of an open globe injury, one must be extremely careful about performing a sterile and gentle B-scan. Some authors believe it is best deferred until after a primary repair has been done. A retinal detachment on B-scan is seen as a membrane attached to the optic disc with limited after movements. The A-scan shows a corresponding high amplitude peak. A giant retinal tear may be seen as inward rolling of the retina with an associated retinal detachment. In long-standing cases or cases of glaucoma, a deep optic cup can be visualized on B-scan as a bean pot-like configuration.

Multiple mobile point echoes on B-scan are suggestive of vitreous hemorrhage. The amplitude on A-scan of these echoes is usually mild to moderate. It is important to keep the gain in the optimal setting(around 70 decibels) as high gains (100 decibels) result in many echoes even in normal eyes and may give a false impression of vitreous hemorrhage.

A highly mobile membrane not attached to the optic nerve head is suggestive of posterior vitreous detachment.

A choroidal detachment is visualized as a round mound that is limited by the strong adhesions at the scleral spur, vortex veins and optic nerve. It has limited mobility. The corresponding peak on A-scan is a double-peaked or an "M shaped" peak. The suprachoroidal space may show mild to moderate amplitude point echoes on A-Scan suggestive of hemorrhagic fluid. When the two domes of the choroidal detachment touch each other, it is known as "kissing choroidals." These cases may lead to permanent adhesion and are an indication for urgent surgical intervention. A B-scan also notes clot lysis, and the location of the suprachoroidal hemorrhage guides the site of drainage.

An ultrasound biomicroscopy can detect very small effusions over the ciliary body without clinically detectable choroidal detachment. A detached ciliary body may also be picked up on this imaging modality.

Lastly, a computed tomography scan can also demonstrate a retinal or choroidal detachment when done in cases of complex head and neck trauma.

6.4.3 Management

The presence of a retinal detachment along with choroidal detachment in the case of open globe injury is an indication for early intervention. It can be done as a two-staged procedure with primary repair of the open wound carried out first followed by management of retinal and choroidal detachment.

The presence of a choroidal detachment with retinal detachment makes the management more challenging. Drainage of choroidals is undertaken first. In the case of a pseudophakic or aphakic eye, an anterior chamber maintainer is placed. In the case of a phakic eye, a 6 mm infusion cannula can be used 2–2.5 mm behind the limbus with an entry that is perpendicular to the sclera. The crystalline lens may have to be removed at times if one is dealing with anterior PVR.

Choroidal drainage can be done by either a scleral cut down or a trocar insertion. The area of maximum choroidals is identified, and peritomy is done in that quadrant. A bridle suture is passed through the rectus muscle in that quadrant. A radial sclerotomy is created around 8 mm posterior to the limbus. The choroidal space can then be entered by a bent needle or an MVR blade. When the suprachoroidal space is reached, a gush of liquefied hemorrhage appears. A cyclodialysis spatula can be inserted in the suprachoroidal space to keep the cut down open, the anterior lip of the wound can be pressed, and a cotton-tipped applicator can be used to massage the globe to maximize the egress of fluid. Alternatively, a 23G non-valved cannula can be positioned in the suprachoroidal space at around 20 degree. The procedure can be repeated in a different quadrant if significant choroidal detachment persists.

Vitrectomy is then carried out to manage the retinal detachment. If there is persisting choroidal detachment, PFCL can be used to displace the choroidals anteriorly. Retinal detachment in such severe eye injuries is usually associated with proliferative vitreoretinopathy changes. Meticulous membrane dissection needs to be carried out. Brilliant blue dye can be used to stain the membranes to ensure complete removal. Relaxing retinotomy may be required in cases of retinal incarceration or shortening. Silicone oil or gas can be used as a tamponading agent.

6.5 Prognosis and Long-Term Outcome

The presence of open globe injury with choroidal detachment and retinal detachment makes the visual and anatomical prognosis very poor. These eyes stand a high risk of going into phthisis despite bet of efforts. In a study of 242 cases of globe rupture, the presence of closed-funnel retinal detachment, proliferative vitreoretinopathy grade C or more, and choroidal damage were the risk factors for unfavorable outcomes [16]. Another study on 33 traumatized eyes with no light perception, presence of ciliary body damage, see intraocular hemorrhage, closed-funnel RD, and choroidal damage was associated with poor visual and anatomical prognosis [17].

6.6 Specific Challenges from Personal Experience

1. One must have a high index of suspicion for retinal and choroidal detachments in open globe trauma.
2. A B-scan ultrasonography must be done in all cases of trauma with conditions such as a corneal tear, corneal edema, hyphema, or vitreous hemorrhage that preclude posterior segment examination.

3. The presence of a coexisting corneal tear makes the visualization of the posterior segment difficult.
4. These cases have high chances of postoperative hypotony due to coexistent ciliary body damage.

References

1. Kuhn F, Morris R, Witherspoon CD. Birmingham Eye Trauma Terminology (BETT): terminology and classification of mechanical eye injuries. Ophthalmol Clin North Am. 2002;15:139–43.
2. Stryjewski TP, Andreoli CM, Eliott D. Retinal detachment after open globe injury. Ophthalmology. 2014;121(1):327–33. https://doi.org/10.1016/j.ophtha.2013.06.045.
3. Andreoli MT, Yiu G, Hart L, Andreoli CM. B-scan ultrasonography following open globe repair. Eye (Lond). 2014;28(4):381–5. https://doi.org/10.1038/eye.2013.289.
4. Kim JH, Yang SJ, Kim DS, et al. Fourteen-year review of open globe injuries in an urban Korean population. J Trauma. 2007;62:746–9.
5. Bauza AM, Emami P, Soni N, et al. A 10-year review of assault-related open-globe injuries at an urban hospital. Graefes Arch Clin Exp Ophthalmol. 2013;251:653–9.
6. Bauza AM, Emami P, Son JH, et al. Work-related open–globe injuries: demographics and clinical characteristics. Eur J Ophthalmol. 2012;23:242–8.
7. Yalcin Tök O, Tok L, Eraslan E, et al. Prognostic factors influencing final visual acuity in open globe injuries. J Trauma. 2011;71:1794–800.
8. Schmidt GW, Broman AT, Hindman HB, Grant MP. Vision survival after open globe injury predicted by classification and regression tree analysis. Ophthalmology. 2008;115:202–9.
9. Rahman I, Maino A, Devadason D, Leatherbarrow B. Open globe injuries: factors predictive of poor outcome. Eye (Lond). 2005;20:1336–41.
10. Gupta A, Rahman I, Leatherbarrow B. Open globe injuries in children: factors predictive of a poor final visual acuity. Eye (Lond). 2008;23:621–5.
11. Lesniak SP, Bauza A, Son JH, et al. Twelve-year review of pediatric traumatic open globe injuries in an urban U.S. population. J Pediatr Ophthalmol Strabismus. 2011;49:73–9.
12. Han SB, Yu HG. Visual outcome after open globe injury and its predictive factors in Korea [report online]. J Trauma. 2010;69:E66–72.
13. Pieramici DJ, Sternberg P, Aaberg TM, et al. Ocular Trauma Classification Group A system for classifying mechanical injuries of the eye (globe). Am J Ophthalmol. 1997;123:820–31.
14. Thakker MM, Ray S. Vision-limiting complications in open-globe injuries. Can J Ophthalmol. 2006;41:86–92.
15. Cardillo JA, Stout JT, LaBree L, et al. Post-traumatic proliferative vitreoretinopathy. The epidemiologic profile, onset, risk factors, and visual outcome. Ophthalmology. 1997;104:1166–73.
16. Feng K, Wang CG, Hu YT, Yao Y, Jiang YR, Shen LJ, Pang XQ, Nie HP, Ma ZZ. Clinical features and prognosis of eyeball rupture: eye injury vitrectomy study. Clin Exp Ophthalmol. 2015;43(7):629–36. https://doi.org/10.1111/ceo.12534. Epub 2015 Jun 19.
17. Feng K, Hu YT, Ma Z. Prognostic indicators for no light perception after open-globe injury: eye injury vitrectomy study. Am J Ophthalmol. 2011;152(4):654–662.e2. https://doi.org/10.1016/j.ajo.2011.04.004.

7 Choroidal and Retinal Detachment Combined with Cyclodialysis in Open Globe Injury

Su Jin Park and Dong Ho Park

Abstract

Open globe injury (OGI) is defined as a full-thickness defect of the cornea or sclera. Depending on the severity of the trauma, the involvement of vitreoretinal lesion varies, and examination may be difficult. Therefore, to better understand the patient's severity before surgery, various examination methods such as computed tomography, B-scan ultrasonography, gonioscopy, and anterior segment optical coherence tomography should be appropriately used. In most cases of OGI, the primary suture is performed immediately after the trauma, and the secondary surgery is planned. In this chapter, we reviewed the retinal detachment, choroidal detachment, and cyclodialysis that could accompany open globe injury and summarized the diagnosis, treatment and postoperative complications.

Keywords

Open globe injury · Choroidal detachment
Cyclodialysis · Retinal detachment

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7.1 Introduction

Ocular trauma causing open globe injury (OGI) is an important cause of vision loss, and more than 200,000 OGI occur worldwide every year [1]. In OGI, vitreoretinal involvement causes significant vision loss or blindness. However, depending on the severity of the trauma, the examination may be difficult, especially predicting hidden wounds. Therefore, in this chapter, among the various event that may accompany OGI, we will discuss particularly retinal detachment (RD), choroidal detachment, and cyclodialysis.

7.2 Definition

According to the Birmingham Eye Trauma Terminology (BETT) criteria [2], an OGI is defined as a full-thickness defect of the cornea or sclera. According to the injury mechanism, the OGI is divided into ruptures or lacerations*:* ruptures are caused by blunt objects and lacerations are caused by sharp ones. Lacerations are further subdivided into *penetrating* injury, *intraocular foreign body* (IOFB) injury, and *perforating* injury. A penetrating injury has only an entrance wound, an IOFB injury has an entrance wound and a retained IOFB, and a perforating injury has an entrance and an exit wound. According to the location relative to the limbus, injuries are

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divided into three zones: a zone I injury is isolated to the cornea (including the limbus), a zone II injury involves the sclera no more than 5 mm posterior to the limbus, and a zone III injury involves the sclera more than 5 mm posterior to the limbus [3].

Severe globe rupture caused incarcerated retina to the lesion site. The residual retina was detached and funnel-like detachment will result in poor visual acuity and severe proliferative vitreoretinopathy (PVR). Retinal dialysis is the most common cause of traumatic RD. Retinal tears are the second-most common predisposing lesion, responsible for about 20% of the traumatic RD [4].

A cyclodialysis cleft is the most common complication in closed globe injuries, with an incidence of 1–11% [5]; it occasionally occurs in open globe injuries and iatrogenic injuries. A cyclodialysis is the disinsertion of the longitudinal ciliary muscle fibers from the scleral spur resulting in a cleft [6]. One of the consequences of cyclodialysis is the opening of communication between the anterior chamber and the suprachoroidal space. Then, ocular hypotony occurs, which is accompanied by choroidal detachment and chorioretinal folds, which affect the macula and cause visual loss [7].

A choroidal detachments are rare conditions that occur when there is an accumulation of fluid or blood in the suprachoroidal layer, located between the choroid and sclera [8]. The pathogenesis of traumatic ciliochoroidal detachment is an increase in the permeability of the ciliary vessels after blunt injury, leading to extravascular leakage of the plasma components [9].

7.3 Case

A 43-year-old male visited the emergency room because of a traumatic right eye injury by a metal hook. The initial best-corrected visual acuity (BCVA) was hand motion (HM) in the right eye and 20/50 in the left eye. Slit-lamp examination showed that scleral laceration of about 1 cm or more in length and uveal and vitreous tissues prolapsed from the lesion site. The anterior chamber was collapsed, and iris defect was accompanied from 12 to 3 o'clock (Fig. 7.1). Since fundus was not visible, B-scan ultrasonography and computed tomography (CT) were additionally performed (Figs. 7.2 and 7.3). B-scan and CT showed hemorrhagic choroidal detachment and no IOFB.

Under the diagnosis of scleral laceration with iridodialysis and suprachoroidal hemorrhage, the patient had an emergency operation. After uveal reposition, the scleral wound was sutured with 8-0 ethilon, and the anterior chamber was irri-

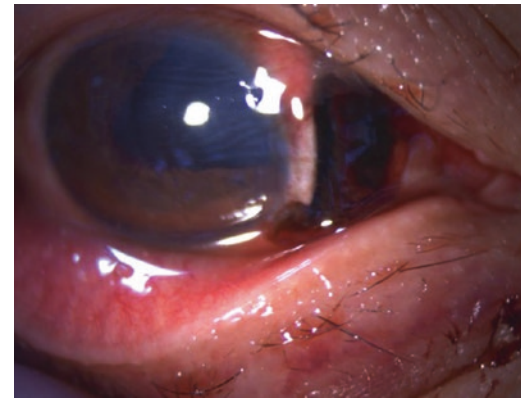

Fig. 7.1 Slit-lamp photograph of the right eye. Scleral laceration with prolapse of vitreous and uveal tissue on the nasal side of the eye

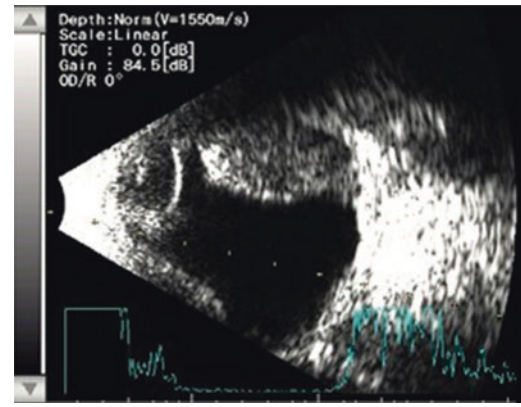

Fig. 7.2 B-scan ultrasonography showed hyperechogenic lesions in the suprachoroidal space, indicating hemorrhagic choroidal detachment

gated with antibiotics, including vancomycin and ceftazidime.

At 1 month after the surgery, though suprachoroidal hemorrhage disappeared on the B-scan, the fundus was not observed due to traumatic cataract, and visual acuity was HM (Fig. 7.4). The second operation of cataract surgery was completed by putting the intraocular lens in the bag with normal fundus findings. One month after the second surgery, BCVA was 20/100 in the right eye.

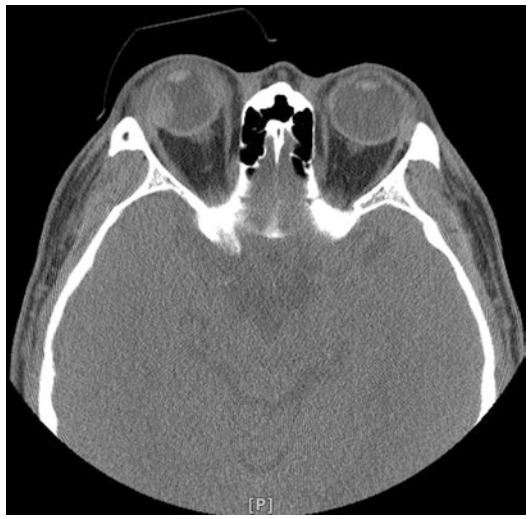

Fig. 7.3 Axial computed tomography of paranasal sinuses. Hyperdense lesions involving the periphery of the right orbit, but with sparing the posterior third on the axial image

7.4 Important Signs, Examinations, Diagnosis, Surgical Procedures, and Postoperative Treatment for Complications

Due to the severe pain, clinical examination of traumatic injuries is more difficult than other diseases. The factors that make the posterior segment examination difficult are as follows: anterior segment injury, including hyphema, corneal edema, or opacification, and posterior segment pathology, including vitreous hemorrhage.

7.4.1 Important signs

The OGI is accompanied by many signs, including hyphema, which is the most common sign on admission (76.7%), iris prolapse (57.9%), vitreous hemorrhage (52.2%), laceration on the eyelid and/or eyebrow (34%), and retinal detachment (29.6%) [10]. Hongsheng et al. reported that among the patients with rupture injuries, 17.3% had a ciliary injury, 62.7% had choroidal injuries, and ciliary and choroidal injuries were both detected in 20% of patients[11].

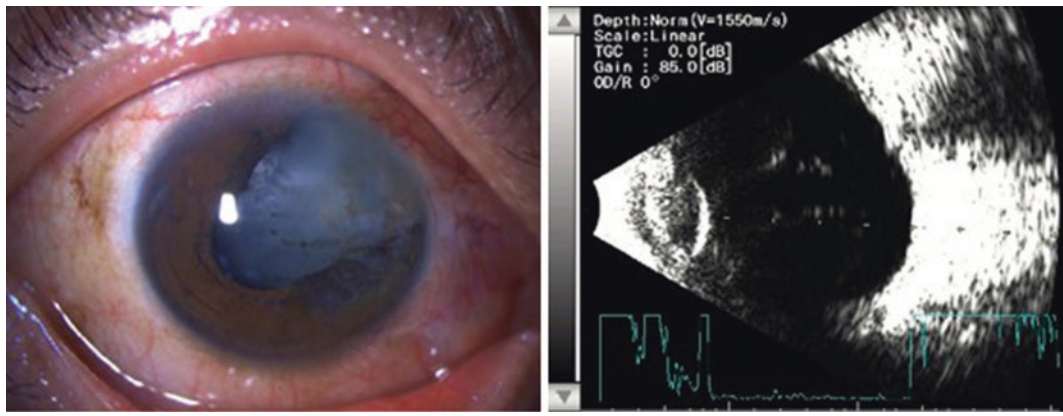

Fig. 7.4 Slit-lamp photograph and B-scan ultrasonography image 1 month after surgery. Traumatic cataract and iris defects are seen on a slit-lamp examination, and posterior vitreous detachment is confirmed by B-scan

7.4.2 Examination and Diagnosis

7.4.2.1 Slit-Lamp Examination

When patients with trauma visit, the slit-lamp examination is performed first. At this time, damage to the vitreoretinal area should be suspected if there are signs such as severe hemorrhagic chemosis, shallow anterior chamber, prolapsed uvea and vitreous, and eccentric pupil.

7.4.2.2 Computed Tomography

The CT scan should be performed to determine the IOFB and occult OGI. In case of traumatic ocular injury, the findings that can be observed on CT are as follows: change in globe contour, volume loss, intraocular air, change in anterior chamber depth, dislocated or absent lens, vitreous hemorrhage, and retinal or choroidal detachment [12].

Ora serrata is an inferred reference point in distinguishing between RD and choroidal detachment on imaging. On the axial plane, ora serrata is located at the 2 and 10 o'clock position of the globe immediately posterior to the ciliary bodies [13].

RD with bilateral V-shaped leaflets converging towards the optic disc. Importantly the anterior margin of the leaflets does not extend beyond the ora serrata reference point at the 2 and 10 o'clock position. However, choroidal detachment with convex lens shape leaflets anteriorly extending beyond the ora serrata reference point and posteriorly limited by insertions of the vortex veins [14].

7.4.2.3 B-Scan Ultrasonography

B-scan ultrasonography is an important diagnostic tool for providing information on diagnosis and prognosis in OGI. In particular, RD and choroidal detachment are mainly diagnosed by B-scan ultrasonography when the funduscopic examination is not possible due to hemorrhage. In OGI, the first b-scan is performed the day after primary closure of the globe due to ocular instability. However, most retinal detachments (53%) were not diagnosed until a week after the trauma. Considering the delayed occurrence of RD, careful follow-up is necessary after the primary closure of the OGI [15].

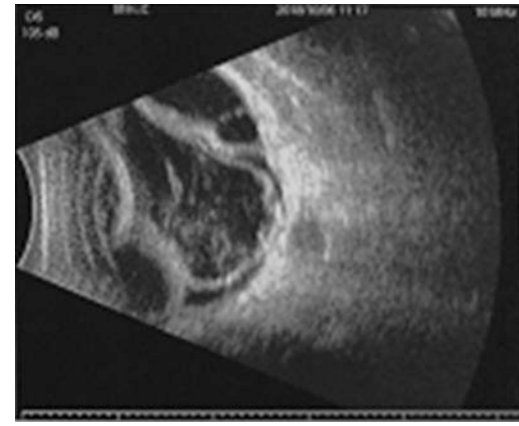

Fig. 7.5 Choroidal detachment with retinal detachment in B-scan ultrasonography. By courtesy of professor Hua Yan from Tianjin Medical University, China

In partial detachment, thin and mobile linear echogenic membrane is observed and usually extends to the optic nerve head, but not across it. In the case of total RD, it is observed in the vitreous cavity in a "V" shape because the retina is attached to the ora serrata anteriorly and the optic nerve head posteriorly. On the other hand, thick and rigid echogenic bands are observed in the choroidal detachment and protrude convexly into the vitreous. These end at the level of the exit foramina of the vortex veins and do not extend to the optic disc [16] (Fig. 7.5).

7.4.2.4 Gonioscopy, Ultrasound Biomicroscopy and Anterior Segment Optical Coherence Tomography

A slit lamp gonioscopy is useful in cases with clear media. However, cyclodialysis clefts are often missed gonioscopically for various reasons such as hyphema, iris bowing, extreme hypotony, distorted anatomy or Descemet's folds [17, 18]. Ultrasound biomicroscopy (UBM) has been described in these situations as supplementing gonioscopic findings. UBM is a noninvasive method that uses high-frequency (50–60 MHz) transducers to image anterior segment structures [19] (Fig. 7.6).

However, since UBM also requires an anesthetic gel and needs to be in contact with the patient's eyes, it is difficult to use immediately

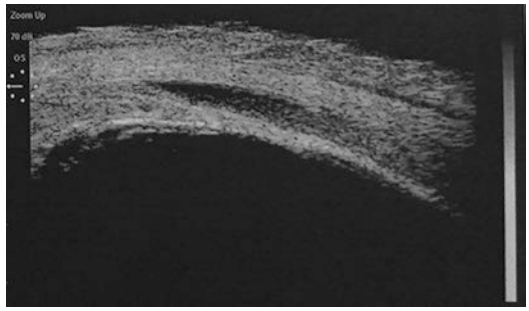

Fig. 7.6 A cyclodialysis cleft confirmed by ultrasound biomicroscopy. Expansion of suprachoroidal space due to detachment of the ciliary body. By courtesy of professor Hua Yan from Tianjin Medical University, China

after OGI. Therefore, in open injuries, anterior segment optical coherence tomography (AS-OCT) can be usefully used in that contact is not required. AS-OCT provided high-resolution images of iridocorneal angle abnormalities in the presence of abnormal anterior segment anatomy [20].

7.4.3 Treatment

Multiple operations are required for patients with OGI. In the open-wound state, the eye is soft, and the intraocular tissues are mostly prolapsed. Therefore, first, primary suture and intraocular tissue reposition are required. The prolapsed iris and choroid should be repositioned after complete irrigation, and the prolapsed vitreous body can be removed.

The second surgery is usually planned according to the condition of the eye after the first emergency surgery. Because the OGI is characterized as a multiple damaged state, meticulous and thorough examinations at each step are required to plan the most effective and the least invasive surgery.

7.4.3.1 Treatment for Retinal Detachment and Choroidal Detachment

In most cases, patients with OGI underwent primary closure immediately. However, the timing of vitreoretinal intervention is varied. In addition, the type of vitreoretinal intervention varies depending on the condition of the eye. Several authors report early vitreoretinal intervention to be performed between 3 and 14 days after OGI [21–26]. In animal study, posterior vitreous detachment occurred at 1–2 weeks after injury, and retinal detachment occurred between 7 and 11 weeks [27]. In a histological study, PVR formation was found between 1 and 2 weeks after severe penetrating trauma in eyes with an RD [28]. Therefore early vitreoretinal intervention reduces the risk of the formation of extensive PVR

7.4.3.2 Cyclodialysis Treatment

Cyclodialysis in small sizes can be treated with medical management such as 1% atropine or laser photocoagulation. If medical or laser treatment does not respond, several surgical treatments can be considered: transscleral diathermy, cryotherapy, and direct cyclopexy [17]. The principle of surgical repair includes the obliteration of the cyclodialysis space and apposition between the sclera and the wall of the ciliary body [29].

Recently, several reports have proposed alternative novel techniques. Portney and Purcell proposed the idea of anterior buckling using a silicone rod under a partial thickness scleral flap [30]. Meanwhile, methods of using a capsular tension ring (CTR) or haptics of large IOLs for internal apposition (internal cerclage) of the ciliary body to sclera have been reported [31–33].

7.4.4 Postoperative Treatment for Complication

There are several postoperative complications of OGI repair. The delayed presentation was an important risk factor for endophthalmitis, microbial keratitis and postoperative wound leak [34]. Possible complications can be divided according to the anatomical wound position. Traumatic cataracts and corneal scarring were the most prevalent vision-limiting complications in patients with zone I (cornea-only) lacerations. The most common vision-limiting factors in eyes with zones II and III lacerations (involving sclera) were cataracts and RD detachments [35].

Several studies reported complications following vitreoretinal surgery for traumatic RD. Surgery mostly accompanies cataracts in the phakic eyes [36–38]. Secondary glaucoma was also a significant complication and, in some cases, required surgical intervention [39, 40]. Patients developed phthisis bulbi, with up to 30% of patients experiencing this complication in certain studies [36, 39, 41–43]. PVR was a significant finding postoperatively, as it was found in up to 56% of patients [40–43].

Very high IOP may follow after successful cyclodialysis cleft surgery. In most cases, the IOP can be controlled through medical treatment, and long-term treatment is not usually required if glaucoma has not been present [29].

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References

1. Négrel AD, Thylefors B. The global impact of eye injuries. Ophthalmic Epidemiol. 1998;5(3):143–69. https://doi.org/10.1076/opep.5.3.143.8364.
2. Kuhn F, Morris R, Witherspoon CD. Birmingham Eye Trauma Terminology (BETT): terminology and classification of mechanical eye injuries. Ophthalmol Clin N Am. 2002;15(2):139–43, v. https://doi.org/10.1016/s0896-1549(02)00004-4.
3. Pieramici DJ, Sternberg P Jr, Aaberg TM Sr, Bridges WZ Jr, Capone A Jr, Cardillo JA, et al. A system for classifying mechanical injuries of the eye (globe). The Ocular Trauma Classification Group. Am J Ophthalmol. 1997;123(6):820–31. https://doi.org/10.1016/s0002-9394(14)71132-8.
4. Sheard RM, Mireskandari K, Ezra E, Sullivan PM. Vitreoretinal surgery after childhood ocular trauma. Eye (London, England). 2007;21(6):793–8. https://doi.org/10.1038/sj.eye.6702332.
5. Grosskreutz C, Aquino N, Dreyer EB. Cyclodialysis. 1995;35(1):105–9.
6. Ormerod LD, Baerveldt G, Sunalp MA, Riekhof FT. Management of the hypotonous cyclodialysis cleft. Ophthalmology. 1991;98(9):1384–93. https://doi.org/10.1016/S0161-6420(91)32121-3.
7. Cerio-Ramsden CD, Muñoz-Negrete FJ, Rebolleda G. Post-traumatic cyclodialysis cleft treated with transscleral diode laser. Archivos de la Sociedad Espanola de Oftalmologia. 2009;84(1):47–50. https://doi.org/10.4321/s0365-66912009000100008.
8. Diep MQ, Madigan MC. Choroidal detachments: what do optometrists need to know? Clin Exp Optom. 2019;102(2):116–25. https://doi.org/10.1111/cxo.12807.
9. Yang J, Liu Q, Li X, Zhou L, Sun P, Wang X. Clinical evaluation of traumatic ciliochoroidal detachment with surgical treatment. Eye Sci. 2013;28(3):124–8. 39
10. Sahin Atik S, Ugurlu S, Egrilmez ED. Open globe injury: demographic and clinical features. 2018;29(3):628–31. https://doi.org/10.1097/scs.0000000000004156.
11. Bi H, Cui Y, Li Y, Wang X, Zhang J. Clinical characteristics and surgical problems of ruptured globe injury. Curr Therap Res. 2013;74:16–21. https://doi.org/10.1016/j.curtheres.2012.10.002.
12. Arey ML, Mootha VV, Whittemore AR, Chason DP, Blomquist PH. Computed tomography in the diagnosis of occult open-globe injuries. Ophthalmology. 2007;114(8):1448–52. https://doi.org/10.1016/j.ophtha.2006.10.051.
13. Kubal WS. Imaging of orbital trauma. Radiographics: a review publication of the Radiological Society of North America, Inc. 2008;28(6):1729–39. https://doi.org/10.1148/rg.286085523.
14. Roy AA, Davagnanam I, Evanson J. Abnormalities of the globe. Clin Radiol. 2012;67(10):1011–22. https://doi.org/10.1016/j.crad.2012.03.006.
15. Stryjewski TP, Andreoli CM, Eliott D. Retinal detachment after open globe injury. Ophthalmology. 2014;121(1):327–33. https://doi.org/10.1016/j.ophtha.2013.06.045.
16. De La Hoz PM, Torramilans Lluís A, Pozuelo Segura O, Anguera Bosque A, Esmerado Appiani C, Caminal Mitjana JM. Ocular ultrasonography focused on the posterior eye segment: what radiologists should know. Insights Imaging. 2016;7(3):351–64. https://doi.org/10.1007/s13244-016-0471-z.
17. Küchle M, Naumann GO. Direct cyclopexy for traumatic cyclodialysis with persisting hypotony. Report in 29 consecutive patients. Ophthalmology. 1995;102(2):322–33. https://doi.org/10.1016/s0161-6420(95)31021-4.
18. Bhende M, Lekha T, Vijaya L, Gopal L, Sharma T, Parikh S. Ultrasound biomiscroscopy in the diagnosis and management of cyclodialysis clefts. Indian J Ophthalmol. 1999;47(1):19–23.
19. Pavlin CJ, Harasiewicz K, Sherar MD, Foster FS. Clinical use of ultrasound biomicroscopy.

Ophthalmology. 1991;98(3):287–95. https://doi.org/10.1016/s0161-6420(91)32298-x.
20. Mateo-Montoya A, Dreifuss S. Anterior segment optical coherence tomography as a diagnostic tool for cyclodialysis clefts. Archiv Ophthalmol (Chicago, IL: 1960). 2009;127(1):109–10. https://doi.org/10.1001/archophthalmol.2008.561.
21. 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. https://doi.org/10.1016/s0002-9394(99)00254-8.
22. Hui Y, Wang L, Shan W. Exploratory vitrectomy for traumatized eyes with no light perception and dense vitreous hemorrhage. [Zhonghua yan ke za zhi] Chin J Ophthalmol. 1996;32(6):450–2.
23. Brinton GS, Aaberg TM, Reeser FH, Topping TM, Abrams GW. Surgical results in ocular trauma involving the posterior segment. Am J Ophthalmol. 1982;93(3):271–8. https://doi.org/10.1016/0002-9394(82)90524-4.
24. Coleman DJ. Early vitrectomy in the management of the severely traumatized eye. Am J Ophthalmol. 1982;93(5):543–51. https://doi.org/10.1016/s0002-9394(14)77367-2.
25. Mieler WF, Mittra RA. The role and timing of pars plana vitrectomy in penetrating ocular trauma. Archiv Ophthalmol (Chicago, IL: 1960). 1997;115(9):1191–2. https://doi.org/10.1001/archopht.1997.01100160361017.
26. de Bustros S, Michels RG, Glaser BM. Evolving concepts in the management of posterior segment penetrating ocular injuries. Retina (Philadelphia, PA). 1990;10(Suppl 1):S72–5. https://doi.org/10.1097/00006982-199010001-00012.
27. Cleary PE, Ryan SJ. Method of production and natural history of experimental posterior penetrating eye injury in the rhesus monkey. Am J Ophthalmol. 1979;88(2):212–20. https://doi.org/10.1016/0002-9394(79)90468-9.
28. Winthrop SR, Cleary PE, Minckler DS, Ryan SJ. Penetrating eye injuries: a histopathological review. Br J Ophthalmol. 1980;64(11):809–17. https://doi.org/10.1136/bjo.64.11.809.
29. Ioannidis AS, Barton K. Cyclodialysis cleft: causes and repair. Curr Opin Ophthalmol. 2010;21(2):150–4. https://doi.org/10.1097/ICU.0b013e3283366a4d.
30. Portney GL, Purcell TW. Surgical repair of cyclodialysis induced hypotony. Ophthalmic Surg. 1974;5(1):30–2.
31. Yuen NS, Hui SP, Woo DC. New method of surgical repair for 360-degree cyclodialysis. J Cataract Refract Surg. 2006;32(1):13–7. https://doi.org/10.1016/j.jcrs.2005.05.035.
32. Mardelli PG. Closure of persistent cyclodialysis cleft using the haptics of the intraocular lens. Am J Ophthalmol. 2006;142(4):676–8. https://doi.org/10.1016/j.ajo.2006.05.027.
33. Hoerauf H, Roider J, Laqua H. Treatment of traumatic cyclodialysis with vitrectomy, cryotherapy, and gas endotamponade. J Cataract Refract Surg. 1999;25(9):1299–301. https://doi.org/10.1016/s0886-3350(99)00160-1.
34. Kong GY, Henderson RH, Sandhu SS, Essex RW, Allen PJ, Campbell WG. Wound-related complications and clinical outcomes following open globe injury repair. Clin Exp Ophthalmol. 2015;43(6):508–13. https://doi.org/10.1111/ceo.12511.
35. Thakker MM, Ray S. Vision-limiting complications in open-globe injuries. Can J Ophthalmol. 2006;41(1):86–92. https://doi.org/10.1016/s0008-4182(06)80074-8.
36. Rouberol F, Denis P, Romanet JP, Chiquet C. Comparative study of 50 early- or late-onset retinal detachments after open or closed globe injury. Retina (Philadelphia, PA). 2011;31(6):1143–9. https://doi.org/10.1097/IAE.0b013e3181f9c22e.
37. Sisk RA, Motley WW III, Yang MB, West CE. Surgical outcomes following repair of traumatic retinal detachments in cognitively impaired adolescents with self-injurious behavior. J Pediatr Ophthalmol Strabismus. 2013;50(1):20–6. https://doi.org/10.3928/01913913-20121002-01.
38. Kolomeyer AM, Grigorian RA, Mostafavi D, Bhagat N, Zarbin MA. 360° retinectomy for the treatment of complex retinal detachment. Retina (Philadelphia, PA). 2011;31(2):266–74. https://doi.org/10.1097/IAE.0b013e3181eef2c7.
39. Wang NK, Chen YP, Yeung L, Chen KJ, Chao AN, Kuo YH, et al. Traumatic pediatric retinal detachment following open globe injury. Ophthalmologica Journal international d'ophtalmologie International journal of ophthalmology Zeitschrift fur Augenheilkunde. 2007;221(4):255–63. https://doi.org/10.1159/000101928.
40. Lesniak SP, Bauza A, Son JH, Zarbin MA, Langer P, Guo S, et al. Twelve-year review of pediatric traumatic open globe injuries in an urban U.S. population. J Pediatr Ophthalmol Strabismus. 2012;49(2):73–9. https://doi.org/10.3928/01913913-20110712-02.
41. Nashed A, Saikia P, Herrmann WA, Gabel VP, Helbig H, Hillenkamp J. The outcome of early surgical repair with vitrectomy and silicone oil in open-globe injuries with retinal detachment. Am J Ophthalmol. 2011;151(3):522–8. https://doi.org/10.1016/j.ajo.2010.08.041.
42. Ehrlich R, Polkinghorne P. Small-gauge vitrectomy in traumatic retinal detachment. Clin Exp Ophthalmol. 2011;39(5):429–33. https://doi.org/10.1111/j.1442-9071.2011.02485.x.
43. Eliott D, Hauch A, Kim RW, Fawzi A. Retinal dialysis and detachment in a child after airbag deployment. J AAPOS. 2011;15(2):203–4. https://doi.org/10.1016/j.jaapos.2010.11.021.

8 Repairment of Traumatic Choroidal Tear

Yuntao Hu and Mengda Li

Abstract

Choroidal injury is a common and serious complication of ocular trauma, which can occur not only in blunt ocular trauma, but also in laceration injuries. Any form of traumatic injury to the eye can result in choroidal injuries. A comprehensive understanding of the classification, presentation, and treatment of choroidal injury is necessary to the prognosis of the disease. The first chapter briefly reviews the anatomy of the choroid, and then the causes, classification, and mechanism of choroidal injury are summarized in the second chapter. The clinical presentation and treatment of choroidal injury are presented in the third and fourth chapter, respectively.

A choroidal rupture is a break in the choroid, the Bruch membrane, and retinal pigment epithelium (RPE). The wound of choroidal ruptures requires no treatment. However, the most troublesome chronic complication of choroidal rupture is choroidal neovascularization (CNV) that can lead to hemorrhagic or serous macular detachment. Therefore, during one to several years after injury, patients should have regularly examinations to detect CNV, and should be treated in time if necessary.

Choroidal detachment can be categorized in to *serous choroidal detachment* and *hemorrhagic choroidal detachment* based on the type of fluid that accumulates in the space between the choroid and the sclera, which can be distinguished by B-scan ultrasonography. Most cases occur due to post-operative hypotony, and will resolve spontaneously as the intraocular pressure (IOP) rising. Conservative management, such as cycloplegics and steroids, leads to better visual outcomes.

Expulsive choroidal hemorrhage is the acute nature of a suprachoroidal hemorrhage that breaks through the choroid, expulses the intraocular contents, and often results in total loss of vision. When it occurs, the key to saving the eye is to stop the bleeding which is achieved by immediate wound closure; the IOP elevates and the bleeding soon tamponades itself. Most retinal surgeons will wait 7–14 days for the choroidal hemorrhage to liquefy and perform a drainage surgery, or combined vitrectomy.

Choroidal avulsion is a serious subtype of choroidal injury, defined as a severe separation of choroid from the sclera and irregular choroidal residuals on the scleral wall. It should be managed by surgery to reduce the incidence of the intraocular low pressure, and to facilitate the reattachment of retina. We

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recommend a simple technique, internal continuous mattress suture, to repair the choroidal avulsion.

Keywords

Choroidal injury · Choroidal rupture
Choroidal detachment · Expulsive choroidal hemorrhage · Choroidal avulsion

8.1 Anatomy of the Choroid

The choroid is the posterior segment of the uveal tract, between the retinal pigment epithelium (RPE) and the sclera. Anteriorly, the choroid joins with the ciliary body, and posteriorly, it is firmly attached to the margins of the optic nerve. The thickness of the choroid is probably dependent on blood flow dynamics and has a diurnal variation, being approximately 220μm at the posterior pole and 100μm anteriorly. Histologically the choroid consists of Bruch's membrane, the choriocapillaris, the vascular layer, and the suprachoroid. The choroid is composed of three layers of choroidal blood vessels: large, medium, and small. The deeper the vessels are placed in the choroid, the wider their lumens.

8.1.1 Bruch's Membrane

It is a connective tissue layer with 2–4μm, and histologically appears as an acellular glassy membrane beneath the RPE. It comprises five layers: the RPE basal lamina, an inner collagenous zone, a middle elastic layer, an outer collagenous zone, and the basement membrane of the endothelial cells in the choriocapillaris.

8.1.2 Choriocapillaris

The internal portion of the choroid vessels is known as the choriocapillaris, which is highly anastomosed and fenestrated on the retinal aspect to facilitate efficient metabolic exchange with the outer retina. The choriocapillaris is fed from the arterioles from the medium vessel layer (Sattler's layer) in a lobular pattern. Smooth muscle cells are not usually present in this layer.

8.1.3 Vascular Layer

This layer lies beneath the choriocapillaris and can be subdivided into an inner layer of medium vessel (arterioles and venules, Sattler's layer) and an outer layer of large vessel (major arteries and veins, Haller's layer).

8.1.4 Suprachoroid

It is the transitional zone between the choroid and the sclera, consisting of numerous fibrous lamellae of varying thicknesses, melanocytes, and flattened processes of fibroblasts. The anterior and posterior ciliary vessels and ciliary nerves traverse through the suprachoroidal space. The vessels have no branch in this space; however, the nerves have branches and form networks. In pathological conditions, suprachoroid may be separated by fluid and blood.

8.1.5 Blood Supply of the Choroid

The choroidal vasculature is supplied by both the long and short posterior ciliary arteries of the ophthalmic artery. Venous drainage occurs via the vortex veins (usually four, but may be up to six), each draining a sector of the choroid into the superior and inferior ophthalmic veins, and ultimately ending up in the pterygoid plexus and cavernous sinus.

8.1.6 Nerve Supply of the Choroid

The sensory, sympathetic, and parasympathetic nerves of choroid are innervated by the long and short ciliary nerves.

8.1.7 Physiological Function of the Choroid

The aggregate of choroidal blood vessels serves to nourish the outer portion of the underlying retina. The capillaries are very permeable to glucose and amino acids, which support the transport of nutrients across the retinal pigment epithelium and Bruch's membrane. The high concentration of protein causes a high oncotic pressure gradient which may be responsible for transporting fluid out of the retina and the eye. It also acts as a conduit for vessels traveling to other parts of the eye and may also have a thermoregulatory role. Furthermore, absorption of light by choroidal pigment aids vision by preventing unwanted light from reflecting back through the retina. The regulation of blood flow in the choroid may also influence intraocular pressure by affecting perfusion rates of the ciliary processes.

8.2 Causes, Classification, and Mechanism of Choroidal Injury

8.2.1 Causes of Choroidal Injury

According to the Birmingham Eye Trauma Terminology System classification, mechanical eye injuries were classified into *closed globe injuries* and *open globe injuries* based on the integrity of the eyewall (sclera and cornea). Open globe injuries (OGIs) were categorized into ruptures or lacerations depending on the mechanism of injury. *Ruptures* are caused by blunt objects; *lacerations* are caused by sharp ones. Lacerations were further classified as *penetrations* when there was an entrance wound alone, as *intraocular foreign bodies (IOFBs)* when there was an entrance wound and part or all of the lacerating object remained in the eye, and as *perforations* when the lacerating object resulted in entrance and exit wounds [1].

Choroidal injury is a common and serious complication of ocular trauma, which can occur not only in blunt ocular trauma, but also in laceration injuries. Any form of injury to the eye can result in choroidal injuries, for example, hitting the eye on the floor, sports injuries, traffic accidents, gun-shot-related injuries, etc. [2]. Choroidal injuries may also occur during ocular surgeries, for example, penetrating keratoplasty, cataract surgery (especially extracapsular cataract extraction), and trabeculectomy [3].

8.2.2 Classification and Mechanism of Choroidal Injury

A choroidal injury can be classified into three types; *choroidal rupture*, *choroidal detachment*, and *choroidal avulsion* depending on the different injury mechanisms and clinical manifestations. Choroidal detachment is further categorized into *hemorrhagic choroidal detachment* and *serous choroidal detachment* based on the type of fluid that accumulates in the space between the choroid and the sclera. According to the extent of hemorrhage, hemorrhagic choroidal detachment can be subdivided into *limited suprachoroidal hemorrhage* and *expulsive choroidal hemorrhage*.

8.2.2.1 Choroidal Rupture

Choroidal rupture is a break in the choroid, Bruch membrane, and retinal pigment epithelium (RPE), which was first described by von Graefe in 1854. Choroidal ruptures have been reported in up to 5–10% of cases of blunt ocular trauma but can also occur in the setting of penetrating or perforating injuries [2].

In blunt ocular trauma, choroidal rupture may be direct, occurring at the site of impact, or indirect, occurring remotely from the site of impact (countercoup). Eighty percent of choroidal ruptures are indirect. In direct injuries, the choroidal rupture produces ruptures that are located anteriorly and oriented parallel to the ora serrata. Indirect injuries produce ruptures that are located distant from the strike site-more posteriorly and often concentric to the optic disc in a crescent shape. Any of the three layers of the eyeball can

rupture, but commonly only the choroid ruptures. The retina is often spared due to its inherent elasticity, whereas the sclera is spared due to its great tensile strength [2, 4].

The predominant theory regarding the mechanism of choroidal rupture attributes to a mechanical injury. During high-speed orbital injuries, the protective ocular reflexes position the eye in an elevated and abducted position. At this point in time, the anteroposterior compressive forces on to the globe create an eccentrically positioned circle of damaging currents along the posterior ocular coats against a relatively static optic nerve. Because of this eccentricity, a longer radius of curvature is expected to lie along the temporal half of the globe leading to an elastic recoil of the retinal and scleral layers and a fracture along the RPE–Bruch's–Choriocapillaris complex manifesting clinically as choroidal rupture [5].

Histopathology of ruptures reveals early hemorrhage, followed by fibrovascular tissue proliferation and RPE hyperplasia, accomplished within 2–3 weeks after the trauma, leading ultimately to a well-defined scar in the area of the choroid–RPE–Bruch membrane defect. In some cases, the scar tissue extends into the subretinal space, the retina, and occasionally even as far as the inner retina and/or vitreous. Atrophy and thinning of the retina may occur, in an outer to inner retinal sequence. There is often hyperplasia of RPE at the margins of the rupture [2, 6, 7].

8.2.2.2 Choroidal Detachment

Choroidal detachment is defined as a separation of the choroid from the sclera; the choroid is only loosely connected to the sclera at the sites of scleral spur, vortex veins, and posterior pole [8]. The common cause of choroidal detachment includes ocular injury, open globe surgery, inflammation, infection, drug-induced processes, and growth of neoplasms. In ocular injury or surgery, the detachment is mainly caused by serous or hemorrhage [9].

Serous choroidal detachment is a result of effusion through an unruptured vessel wall. It has been associated with ocular trauma, intraocular surgery, hypotony, systemic hypertension, glaucoma, and inflammation [10]. Serous choroidal detachment has been shown in a multicenter randomized clinical trial to be as frequent as 11% after trabeculectomy [11].

The main significance of choroidal effusion is that it may lead to *hemorrhagic choroidal detachment*, which is defined as the accumulation of blood between the choroid and the sclera [12]. It has been reported to occur in 3.1% of intracapsular cataract extractions [13], in 2.2% of extracapsular extractions [14], in 1.9% of pars plana vitrectomy [15], and in 30% open globe injuries [16]. If adequate measures are not taken, the limited suprachoroidal hemorrhage is able to rapidly develop into expulsive choroidal hemorrhage [12].

Expulsive choroidal hemorrhage is the acute nature of a suprachoroidal hemorrhage that breaks through the choroid, expulses the intraocular contents, and often results in total loss of vision. It is one of the most dramatic, catastrophic complications of intraocular surgery [17].

The most common element in serous or hemorrhagic choroidal detachment development is hypotony. The normal environment for choroidal vessels is intraocular pressure (IOP) exceeding the atmospheric pressure by 15 mmHg to 18 mmHg, which can force blood from the choroid into the draining vortex veins. A sudden drop of this external pressure in intraocular surgeries or ocular injuries therefore can cause an increase in permeability of the choroidal capillaries, and this subsequently leads to a positive flow of fluid out of the capillaries and into the suprachoroidal space and leads to serous choroidal detachment. As the IOP drops to zero once the globe is opened, there is anterior displacement of the retina and choroid, placing traction on the already weakened posterior ciliary arteries at their point of emergence from the intrascleral canals and rupturing them, leading the occurrence of hemorrhagic choroidal detachment [12, 18]. Histological reports confirm the necrotic posterior ciliary arteries are responsible for the hemorrhage [19, 20]. This occurs because the length of the arterial connections between the choroid and the sclera is short [17, 21].

If appropriate measures on the surgeon's part are not taken and blood keeps on accumulating in the suprachoroidal space, it causes a choroidal detachment that progressively enlarges, pushing the choroid and the retina centrally and then anteriorly. Eventually, the opposing retinal surfaces meet, giving rise to clinical condition called kissing choroidal detachment. As the vitreous is pushed toward the surgical wound, it extrudes the iris, and, if present, the crystalline lens or the intraocular lens, and the retina. Vitreous hemorrhage is probably a marker for a more severe and explosive hemorrhage, since the blood probably gains access to the vitreous cavity by breaking through retina or ciliary body [22, 23]. The suprachoroidal blood can break through and present in the surgical incision. Bleeding, however, does not necessarily offer protection against retinal extrusion [12].

A rabbit experimental model was used to determine the sequence of histopathologic events that lead to expulsive choroidal hemorrhage. The right eyes were proposed, and the central cornea, lens, and anterior vitreous were removed. After surgery, all eyes developed choroidal effusion, choroidal hemorrhage, or expulsive hemorrhage. Histologic examination revealed four sequential stages of expulsive hemorrhage as follows: (1) There was engorgement of the choriocapillaris. (2) Suprachoroidal effusion occurred mainly near the posterior pole. (3) As the effusion enlarged, stretching and tearing of choroidal vessels as well as tearing of the vessels and attachments at the base of the ciliary body occurred. (4) Massive extravasation of blood, primarily from the torn vessels at the ciliary body base, resulted in suprachoroidal hemorrhage and expulsion of blood through the surgical wound [17].

8.2.2.3 Choroidal Avulsion

Choroidal avulsion is a serious subtype of choroidal injury, defined as a severe separation of choroid from the sclera and irregular choroidal residuals on the scleral wall, usually accompanied by the detachment or avulsion of ciliary body at the corresponding quadrant, caused by a sudden external force burst [24]. Choroidal avulsion is also described as "choroidodialysis", "choroidal separation", and "ruptured choroidal detachment". The inner surface of scleral always can be seen during surgery in choroidal avulsion eye.

During the severe ocular trauma, based on the principle of the external force delivery along the eyeball wall, when an external force leads to a drastic deformation of the eyeball, the maximal tension will focus on the peripheral part of the optic disc and vitreous base, resulting in an avulsion or even a retro-equatorial detachment of the choroid from the sclera. This detachment will lead to an immediate damage of the choroidal blood supply. Depending on a vast number of capillaries and the abundant interstitial substance it has, as well as the participation of outer choroidal brown lamina and the inner choroidal pigment epithelia into the tissue repair, a good many fibrous connective tissues were produced during the repair, leading to a choroidal atrophy or sclerosis, which hinders the choroidal reattachment (Even heavy water and scleral buckling cannot fulfill the attachment of choroid to the sclera). Therefore, the outcome of the choroidal avulsion will be determined by the treatment timing, method, and technique [25].

8.3 Clinical Presentation of Choroidal Injury

8.3.1 Choroidal Rupture

Clinical Findings

Consistent with most traumatic ocular injury, 80% choroidal ruptures occur in male [26]. Visual acuity at presentation can range from 20/20 to light perception, depending on the location of the rupture, the degree of associated vitreous, subretinal, or choroidal hemorrhage, hemorrhagic detachment of the pigment epithelium, macular hole formation, and other associated ocular injuries [27, 28]. Typically, a subretinal hemorrhage obscures the rupture early on (Fig. 8.1a). With resorption of the blood, the choroidal rupture is subsequently noted, which clinically appears as white to yellow-red lesions 1–5 disc diameters in length and one-fifth to one-third of a disc diameter in width with tapered

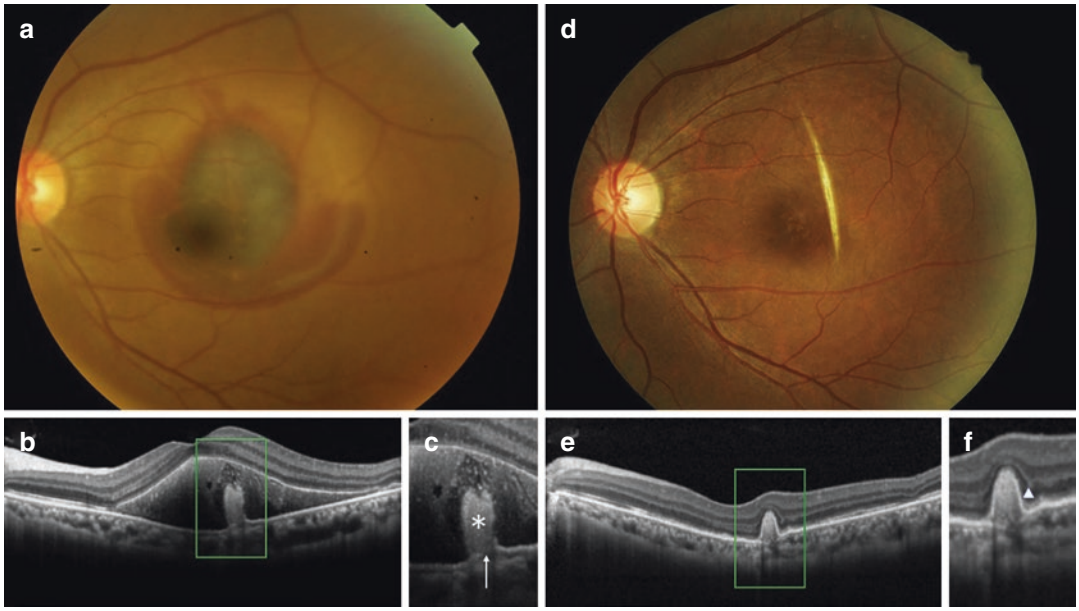

Fig. 8.1 A 48-year-old woman sustained a blunt injury to her left eye, decreasing visual acuity to finger counting. Examination revealed submacular hemorrhage with an underlying choroidal rupture (**a**). Spectral-domain OCT demonstrated diffuse subretinal hyperreflective material associated with hyperreflective protrusion (asterisk) from disruption (arrow) of the retinal pigment epithelium (RPE)–Bruch membrane complex (**b** and **c**). Six months later her vision was 20/100, and examination showed choroidal rupture with resolution of submacular hemorrhage (**d**). Spectral-domain OCT demonstrated partial loss of the ellipsoidal zone and external limiting membrane and RPE migration (arrowhead) adjacent to the hyperreflective fibrotic tissue (**e** and **f**). Green boxes indicate areas of magnification [29]

or forked ends (Fig. 8.1d) [27]. Direct injuries typically produce peripheral ruptures parallel to the ora, whereas indirect injuries result in crescent-shaped lesions concentric to the disc. Ruptures are more commonly located temporal to the disc than nasal. In most instances ruptures are single, but about 25% of affected eyes will contain multiple ruptures [27]. A wide range of visual field defects may be noted. The defects may correspond to the location of the rupture, or to nerve fiber layer damage, or due to an associated traumatic optic neuropathy [30–32].

Choroidal neovascularization (CNV)

Studies reported that 10–20% patients developed CNV anywhere after choroidal rupture, which is the most troublesome chronic complication of choroidal rupture that can lead to hemorrhagic or serous macular detachment [28, 33]. This most frequently occurs during the first year (81.2%) after the injury but has been reported up to 5 years after injury [4, 33]. CNV mostly develops in the foveal or macular area, which may be associated with the regional imbalance in secretion of pro-angiogenic and anti-angiogenic cytokine [34]. There can be a spontaneous involution of the CNV within weeks following injury, but, unfortunately, in some eyes, the CNV is aggressive or gets reactivated, subsequently leading to visual loss in about 5% of ruptures [29, 35]. Risk factors for development of CNV include macula involving, longer length of ruptures, ruptures closer to the center of the fovea, and those occurring in those of older age [29, 35]. In most cases, the CNV appeared consistent with a subretinal (type 2) CNV [36].

Fundus Autofluorescence

In choroidal ruptures, fundus autofluorescence reveals a well-defined linear hypoautofluorescent defect, which is surrounded by a hyperautofluorescent rim that is indicative of RPE hyperplasia

during healing. In some cases, near-infrared autofluorescence may better delineate ruptures through hemorrhage [34, 37].

Fluorescein Angiography (FA)
Typical findings on FA include early hypofluorescence due to disruption of the choroid and choriocapillaris in the area of the rupture. The normal choriocapillaris near the margin of the rupture subsequently leaks into the scar, which eventually becomes hyperfluorescent due to staining [30]. Findings of CNV include early leakage of dye near the area of rupture, which increases and persists into the late phases of the angiogram [27] (Fig. 8.2).

Indocyanine Green Angiography (ICG)
ICG typically exhibits hypofluorescence at the rupture site throughout all phases. ICG can identify ruptures that are imperceptible clinically or on FA [39]. CNV on ICG shows a late hyperfluorescence [39].

Optical Coherence Tomography (OCT)
Two distinct tomographic patterns of choroidal ruptures can be identified on OCT. The first type is a forward protrusion of the retinal pigment epithelium–choriocapillaris (RPE-CC) layer with an acutely angled pyramid or dome shape. This was associated with either a small loss of continuity of the retinal pigment epithelium layer or elevated RPE-CC projection accompanied by a significant quantity of subretinal hemorrhage (Fig. 8.1b, c, e, f). The second type observed was a larger area of disruption of the RPE-CC layer, photoreceptor inner segment/outer segment junction, and external limiting membrane, with a posteriorly directed concave contour depression at that area and downward sliding of tissues into the defect. CNV presents subretinal hyperreflectivity on OCT, and a well-defined high-flow neovascular network on OCTA [40].

8.3.2 Choroidal Detachment

There are two recognized forms of choroidal detachment: serous and hemorrhagic. Serous choroidal detachments occur when there is leakage of serum from the choroidal blood vessels into the suprachoroidal space. Hemorrhagic choroidal detachments occur when there is abnormal blood accumulation in the suprachoroidal space secondary to rupture of a ciliary blood vessel.

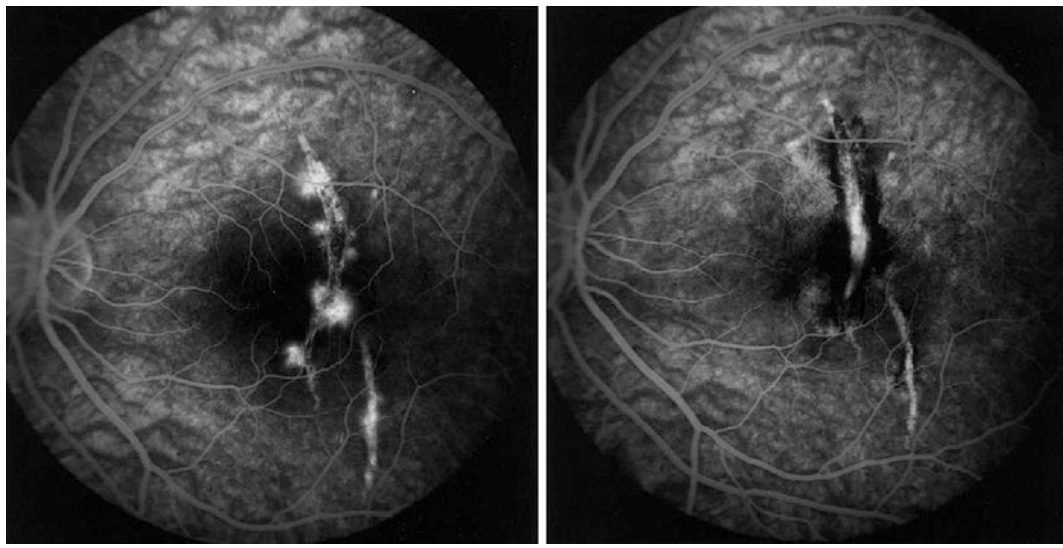

Fig. 8.2 (Left) Digital intravenous fluorescein angiography of a 16-year-old patient showing choroidal neovascularization (CNV) secondary to traumatic choroidal rupture. (Right) After two photodynamic therapy treatments, leakage from the CNV had ceased [38]

The most common cause of choroidal detachment is secondary to open globe surgery, which always occur between the second and fifth day after surgery [22]. Trauma has also been documented to cause choroidal detachment. Retrospective studies found choroidal detachment occurring in 18–38% open globe trauma cases [41, 42].

Risk factors of choroidal detachment include: hypotony, topical use of antimetabolites (such as mitomycin C) [43], nanophthalmos and high hypermetropia [44], increased age, arterial hypertension, tachycardia, atherosclerosis and diabetes [22, 45], anti-hypertensive medications [46], antiplatelet and anticoagulant medications [47, 48].

Serous choroidal detachment

Patients with small, peripheral serous detachments mostly occur postoperatively, and may be completely asymptomatic, or display a small myopic shift, and angle closure from anterior displacement of the ciliary body, lens, and iris. Typically, there may be up to four smooth lobes of fluid accumulation which characteristically extend up to the vortex veins, as the vortex veins are firmly attached to the choroid and sclera. Hypotony maculopathy may also be present, with subretinal fluid, macula striae, and retinal vessel distortion [9].

Hemorrhagic choroidal detachment

Individuals experiencing a hemorrhagic choroidal detachment may have sudden decreased visual acuity and onset of pain due to stretching of the ciliary nerves, which is always companied by headache, nausea, and vomiting. The lobes of hemorrhagic detachments do not transilluminate as is seen with serous detachments [45, 49].

Post-operative hemorrhages are usually limited, and not associated with any expulsion of intraocular contents, as these occur within an enclosed eye, but may eventually lead to apposition of the retina in the posterior chamber if severe. The pathophysiology for intraoperative and post-operative suprachoroidal hemorrhages is the same but the visual outcomes differ due to the open or closed nature of the eye [45].

Expulsive suprachoroidal hemorrhage (ESH)

Intraoperative or open globe traumatic hemorrhagic detachments are referred to as expulsive suprachoroidal hemorrhages. In a study on eyes undergoing drainage for suprachoroidal hemorrhage 33% of the cases were caused by trauma and 60% occurred during open globe surgery [50]. The visual acuity is usually poor or even no light perception (NLP). The IOP at presentation is low due to destruction of the eyewall; and it may maintain at a low, normal, or high range after the wound or incision water-tight closure [16, 51]. It can be complicated by progressive corneal edema, iris incarceration, vitreous incarceration, pupillary block, lens damage, lens-cornea touch, chorioretinal apposition to the posterior lens capsule, central retinal apposition, giant retinal tear, rhegmatogenous retinal detachment, retinoschisis, retinal incarceration, vitreous base avulsion, vitreous hemorrhage, complete disorganization or herniation of intraocular contents, intraocular foreign body, and phthisis [12].

During the ocular surgery, the early signs of expulsive choroidal hemorrhage include: patient's complaint of severe pain of the eye; tactile of the eyeball is firmness because of the elevated IOP; dark mounds of the choroid can be seen through the pupil; loss of red reflex; anterior chamber bleeding/shallowing; bulging of lens, etc. It is important for the surgeon to be alert for the early signs of an expulsive hemorrhage, because the eye frequently can be saved if the wound can be closed [12].

During a choroidal detachment, a shallow anterior chamber is commonly seen. The shallow anterior chamber can be caused by a sagittal compression of the vitreous from the anterior displacement of the choroid, which then causes a blockage of the pupil by the vitreous or lens, and anterior rotation of the ciliary body base due to the detachment. Accumulation of fluid in the supraciliary space appears to be the main cause of an associated secondary angle closure, and this observation has been supported with ultrasound biomicroscopic studies [22, 52].

B-scan ultrasonography

It can be used to document not only the location and height of the detachment, and is useful in differentiating between serous and hemorrhagic choroidal detachment. The fluid in a serous detachment is much less echo-dense than blood [45] (Figs. 8.3 and 8.4). In expulsive hemorrhagic choroidal detachment, massive dome-shaped or "kissing" choroidal hemorrhage with high reflection occupying the eye ball can be detected in the ultrasonography (Fig. 8.5). It can also be used to determine the dynamics of blood liquefaction to determine proper timing for drainage. Initially, fresh blood and clots are imaged as a large mass of highly reflective material. Over time, as the blood in the suprachoroidal space begin to liquefy, the reflections on ultrasonography become lower and more regular, eventually ending up with diffuse low reflective opacities that may be observed to move on dynamic examination. The average time of blood liquefaction is 15 days [45, 51, 53, 54].

Orbital Magnetic resonance imaging (MRI) and computed tomography scan (CT)

Orbital MRI and CT may also be used to determine the presence and underlying cause of choroidal detachment; however, they may be less accurate than B-scan ultrasonography and ultrasound biomicroscopy [9, 55].

8.3.3 Choroidal Avulsion

Choroidal avulsion is usually secondary to a severe ocular trauma, particularly an open one. It can be caused by explosive injury, blunt object injury, sharp object injury, and traffic accident.

Ophthalmic examination shows low visual acuity ranges from NLP to LP; low intraocular pressure; corneal striae; disarrangement of intraocular structure; massive vitreous or choroidal hemorrhage; detachment of the retina and/or choroid, and/or cyclodialysis. Intraoperative findings of choroidal avulsion indicate severe separation of choroid and several choroid residuals with different densities attached on the sclera at the choroidal detachment area [25, 56] (Fig. 8.6).

B-scan ultrasound of choroidal avulsion reveals subretinal sonographic signal emerging from the ocular wall and extending into the center of the vitreous cavity, with or without connection with the ciliary body [25, 56].

8.4 Treatment of Choroidal Injury

8.4.1 Choroidal Rupture

For the treatment of choroidal rupture, there are currently no medications or surgical procedures available that particularly effective. However, a

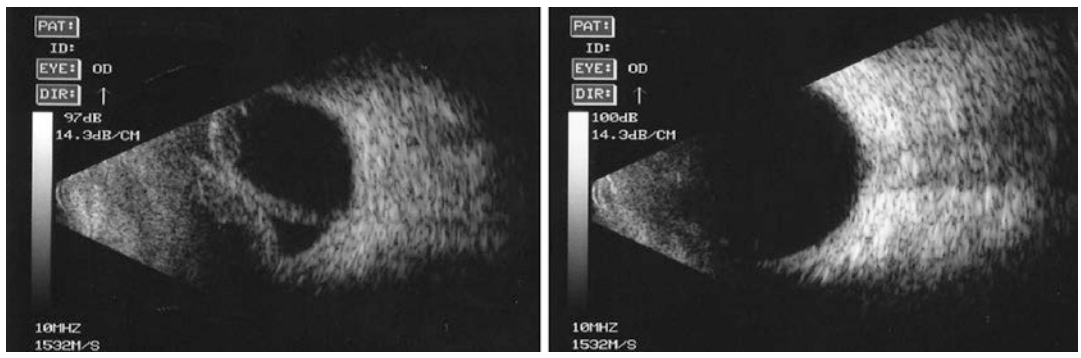

Fig. 8.3 B-scan ultrasonography of serous choroidal detachment after glaucoma surgery. (Left) Preoperative B-scan ultrasonography of an appositional bullous serous choroidal detachment with low echo-dense. The case was drained with anterior chamber infusion and transconjunctival cannula placement at the suprachoroidal space. (Right) 1 week postoperatively the choroidal detachment was completely resolved [53]

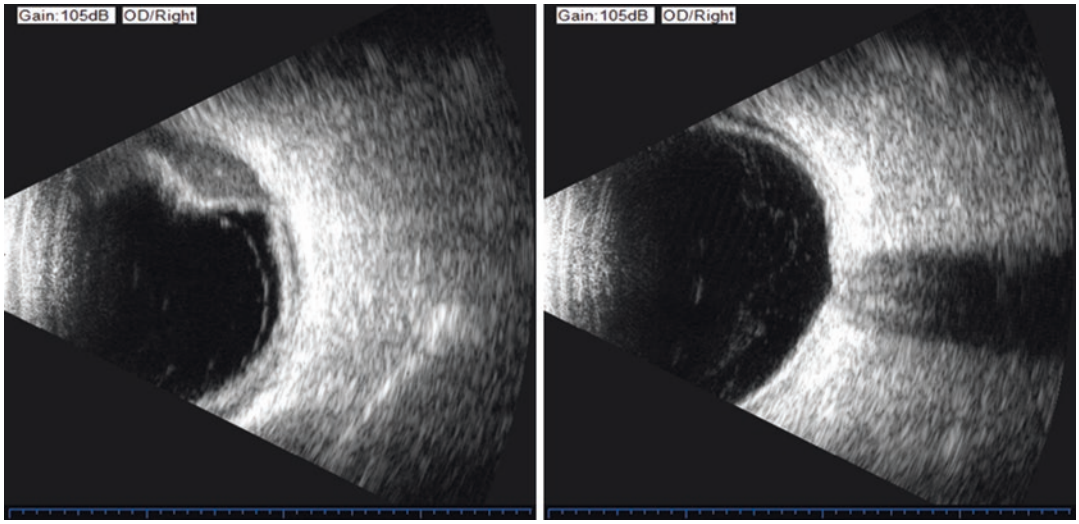

Fig. 8.4 (Left) B-scan ultrasonography of limited hemorrhagic choroidal detachment after trabeculectomy with highly reflective material. (Right) The choroidal detachment was almost resolved 10 days after drainage surgery

few case reports have suggested that the intravitreal injection of expandable gases, such as SF6 or C3F8, or along with tissue plasminogen activator can have a positive therapeutic effect on treating choroidal ruptures with newly incurred subretinal hemorrhage [57–59]. This treatment is performed in the hope that the expandable gas in combination with the prone position would push the hemorrhage beneath the macula to the periphery of the eye in order to restore the patient's central vision [57].

The wound of choroidal ruptures requires no treatment. Fibrovascular proliferation seals the rupture, and a hyperplastic pigment epithelial scar forms [6]. Neovascularization occurs in the area of rupture, but should regress over the first few weeks. The healing process is complete in 3 weeks. Instead, during one to several years after injury, patients should have regularly scheduled fluorescein examinations to detect CNV, after which one or more of several treatment modalities may be considered [4].

Laser Photocoagulation

Laser was used in the treatment of extrafoveal CNV from choroidal ruptures as early as the 1970s. The limited case reports in the literature, however, point to early resolution of subretinal fluid and hemorrhage, followed by a high rate of recurrence [2, 60–62]. The majority of CNVs were subfoveal, making laser photocoagulation less attractive as a treatment.

Photodynamic Therapy (PDT)

In a study of 26 young patients with CNV following traumatic choroidal rupture, PDT shows encouraging results in stabilizing CNV with absence of leakage on FFA, and improving visual acuity with a follow-up period of 30 months [38] (Fig. 8.2). However, several case reports and small case series consistently demonstrate that PDT produces a short-lived resolution of leakage and CNV regression, with subsequent recurrent CNV, resulting frequently in vision loss over the long term [28, 63].

Pharmacologic Therapies

The advent of anti-vascular endothelial growth factor (VEGF) agents in ophthalmology offered a novel approach to the treatment of choroidal rupture-related CNV. But there was no standardized protocol for initiation or re-treatment with injections. Studies reported that monoclonal anti-VEGF (bevacizumab) intravitreal injection can produce an improvement in visual acuity and resolution of leakage within the posterior pole during the follow-up period of 6-month and 2-month, 6-week, respectively [64–66]. A case series reported that 8 of 101 eyes developed CNV after choroidal ruptures were treated with

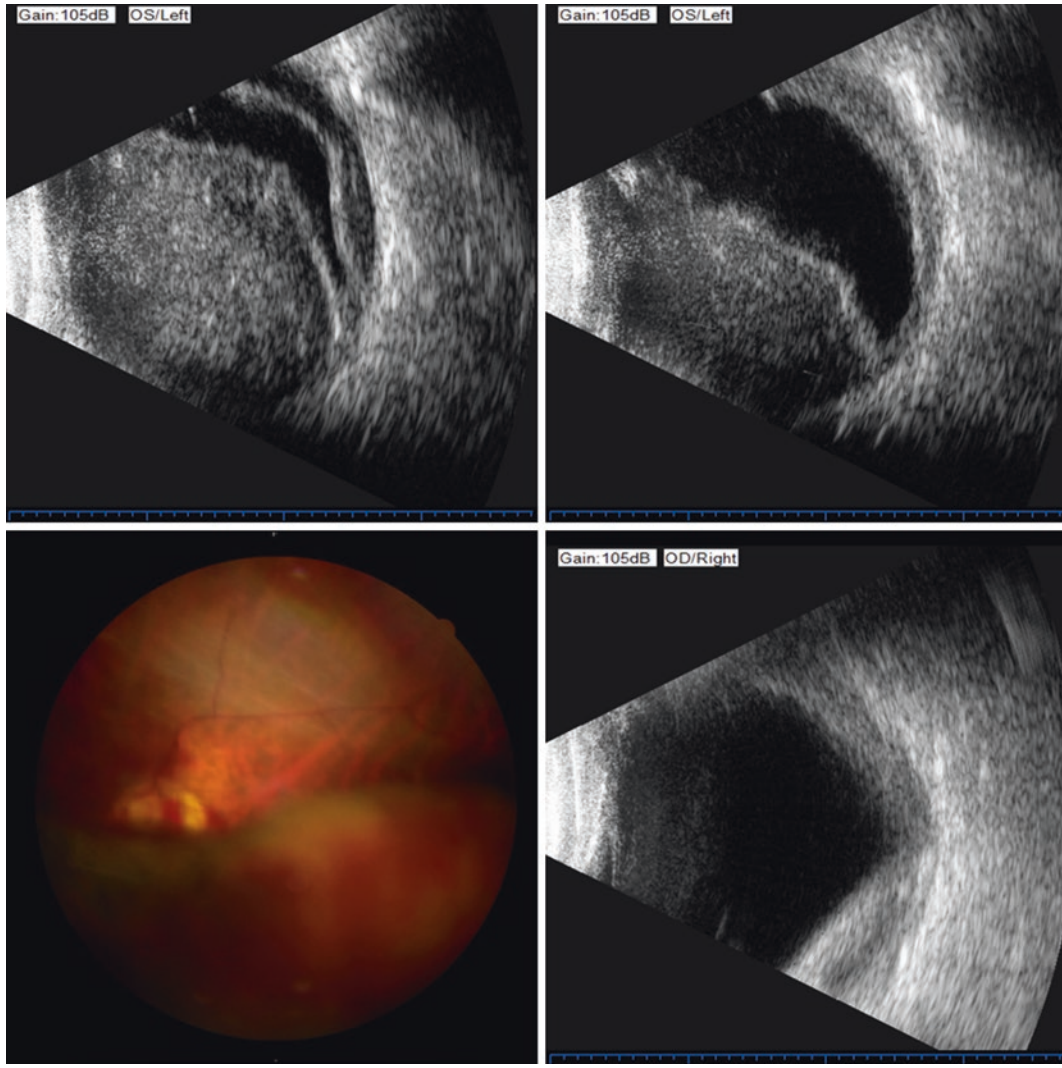

Fig. 8.5 B-scan ultrasonography of expulsive suprachoroidal hemorrhage (ESH) after cataract surgery. (Top left) Preoperative B-scan ultrasonography showed massive dome-shaped choroidal hemorrhage with high reflection occupying the eye ball. Suprachoroidal injection of tPA was performed on the 6th day of ESH. (Top right) One day postoperatively the blood liquefied, and the reflections on ultrasonography become lower and more regular, so drainage surgery combined with vitrectomy was performed. (Bottom left) 10 days after the drainage surgery, a small range of hemorrhagic choroidal detachment still left in the inferior quadrant, a secondary drainage surgery was conducted for a more complete removal of the hemorrhage. (Bottom right) The hemorrhage decreased remarkably 1 month postoperatively. The visual acuity improved from light perception at the base line to 0.2, 8 months after the surgery

intravitreal anti-VEGF injections (either bevacizumab or aflibercept). The mean follow-up interval was 2.9 years. Patients responded well anatomically and vision remained stable after a median of two intravitreal anti-VEGF injections [36]. Early intravitreal anti-VEGF injection could also be an effective treatment option for patients with vision loss associated with traumatic choroidal rupture and subretinal fluid within the posterior pole before development of CNV [67]. Although there are no reports of more long-term follow-up, the limited data suggest that anti-VEGF agents may increase the likelihood of photoreceptor recovery and may produce a more durable response when compared with laser or PDT [2, 68].

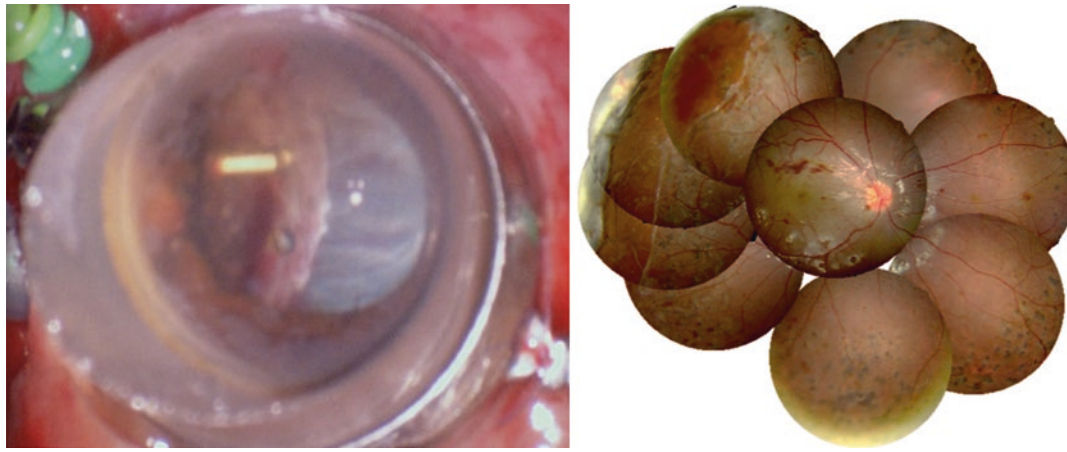

Fig. 8.6 (Left) Fundus picture taken during the surgery showed temporal choroidal avulsion ranged from 8 to 11 o'clock and close funnel-like retinal detachment after ocular rupture of the right eye by traffic accident. The patient underwent choroid suture combined with vitrectomy and silicone oil tamponade. (Right) Fifteen months after the surgery, the choroid and retina reattached well, and the best visual acuity improved from no light perception to 0.05

Surgery

Limited case reports and case series report some successful surgeries (pars plana vitrectomy, access retinotomy, and extraction of subfoveal CNV membrane) for CNV with associated subretinal hemorrhage. Visual acuities can be stable at 20/15–20/50 up to 7–39 months after surgery [28, 69–71].

Prognosis

Certain factors have been identified that adversely affect visual recovery in patients with choroidal ruptures. Associated macular hole, macular pigmentary disturbance, choroidal neovascularization, and optic atrophy generally result in lesser amounts of visual recovery [28, 69–71]. According to the Massachusetts Eye and Ear Infirmary study of 111 cases, most patients with traumatic choroidal rupture do not achieve final VA of 20/40 or better [28].

8.4.2 Choroidal Detachment

Conservative Treatment for Serous Choroidal Detachment and Limited Hemorrhagic Choroidal Detachment

Most serous choroidal detachments and limited hemorrhagic choroidal detachments occur due to post-operative hypotony. If post-operative hypotony is secondary to a wound leak, Seidel's test should be performed to confirm the presence and location of the aqueous leak. If it is a mild leak, the wound will likely self-resolve. If it is a moderate wound leak, but the anterior chamber is still of adequate depth, a bandage contact lens can be applied to promote reepithelialization and prevent lid interaction with the cornea [49, 72]. If the anterior chamber is shallow, or the intraocular pressure continues to be low despite treatment, surgical repair is essential [9].

Post-operative choroidal detachments are usually localized and have little effect on visual acuity. They will resolve spontaneously as the intraocular pressure (IOP) rises. Management is usually conservative and includes cycloplegics and steroids [23, 73]. Cycloplegia is used to prevent iridocorneal interactions from an anteriorly shifted lens–iris diaphragm, as well as to rotate the ciliary body posteriorly to deepen the anterior chamber [9, 49]. Topical steroids are used to reduce inflammation and stop transudation of fluid from choroidal capillaries in to the suprachoroidal space [74]. Oral steroids may be commenced if the topical therapy does not improve the condition, or if it is a severe detachment [9]. In some cases of hemorrhagic detachments, intraocular pressure may be elevated and thus topical

beta blocker and oral carbonic anhydrase inhibitor medications are recommended [45]. Most cases will resolve within 7–10 days as the intraocular pressure normalizes [46]. Conservative management leads to better visual outcomes [75].

Surgery for choroidal drainage is indicated when there is non-resolving detachment with conservative management, when severe anterior chamber shallowing occurs, or when vitreous or retinal incarceration is present [50, 76, 77] (Figs. 8.3 and 8.4).

Emergency Management of Expulsive Choroidal Hemorrhage

When *expulsive choroidal hemorrhage* occurs in open globe injury or during ocular surgery, the key to saving the eye is to stop the bleeding which is achieved by immediate wound closure; the IOP elevates and the bleeding soon tamponades itself [12]. Several methods can be conducted to close the eye. Suture the wound if there is time for the introduction of sutures [18]. If there is no preplaced suture and no time to introduce sutures, apply digital pressure or use strong forceps to approximate the wound lips. Temporary keratoprosthesis can be used to close even large wounds, such as penetrating keratoplasty [12]. Ideally wound closure should be water-tight without tissue incarceration. If incarceration exists, it is advisable to defer repositioning of ocular tissues until the time of secondary repair. Primary drainage of hemorrhagic choroidal detachment should be avoided if possible [78]. High-dose systemic and topical corticosteroids are beneficial during the immediate post-operative period, which may decrease ocular inflammation and may help prevent permanent apposition of the retina to the iris and other anterior structures [79].

Drainage Surgery and Vitrectomy of Serous and Hemorrhagic Choroidal Detachment In eyes with serous choroidal detachments, the indications for drainage were persistent (≥3 months) and nearly appositional serous choroidal detachments [53]. As for hemorrhagic choroidal detachment, although the optimal timing for secondary drainage is controversial, most retinal surgeons will wait 7–14 days for the choroidal hemorrhage to liquefy, which can be determined by B-scan ultrasound [78]. Too early drainage prevents thrombus formation and may exacerbate the condition [12]. For patients with severe open globe injuries, 7–14 days after the injury is also an optimal timing, which gives the advantage of performing vitrectomy in a quite eye mostly with a clear cornea, posterior hyaloidal detachment, less proliferative vitreoretinopathy, and without any new hemorrhage and leakage [80].

We prefer a suprachoroidal injection with tissue plasminogen activator (tPA) (10μg/0.2 ml) at the site of hemorrhagic choroidal detachment 1 day before the drainage surgery for a complete hemorrhagic liquefication [81].

An important principle in the management of serous or hemorrhagic choroidal detachment is the avoidance of hypotony and the maintenance of constant infusion pressure in the eye. Often the infusion port cannot be placed through the pars plana site because of choroidal edema and anterior displacement of the retina. The infusion port usually is moved anteriorly to the limbus. The optimal site of draining is at the highest point of choroidal detachment, as determined by echography. The traditional method to drainage the suprachoroidal liquid is through an adequate radial scleral incision. Vitrectomy can be conducted at the limbus or through the par plana. Liquid perfluorocarbon could be used to expand the intraocular space, and its specific gravity allows drainage of the hemorrhage to proceed from posterior to anterior for a more complete drainage of the hemorrhage. If necessary, a new infusion port can be placed through the pars plana for further vitrectomy [12, 78] (Fig. 8.7).

With the development of surgical technique, transconjunctival drainage of serous or hemorrhagic choroidal detachment with a 20-gauge, 23-gauge, or 25-gauge trocar/cannula system seems to be a feasible and simple surgical option with minimal scleral and conjunctival damage.

Tommaso et al. placed an infusion line in the anterior chamber and a 23-gauge vitrectomy cannula in 3.5 mm from the limbus. The cannula was inserted along the meridians where the ultrasound showed the most prominent choroidal detachment and to angle them as much as possible in the insertion maneuver. As the blood flowed

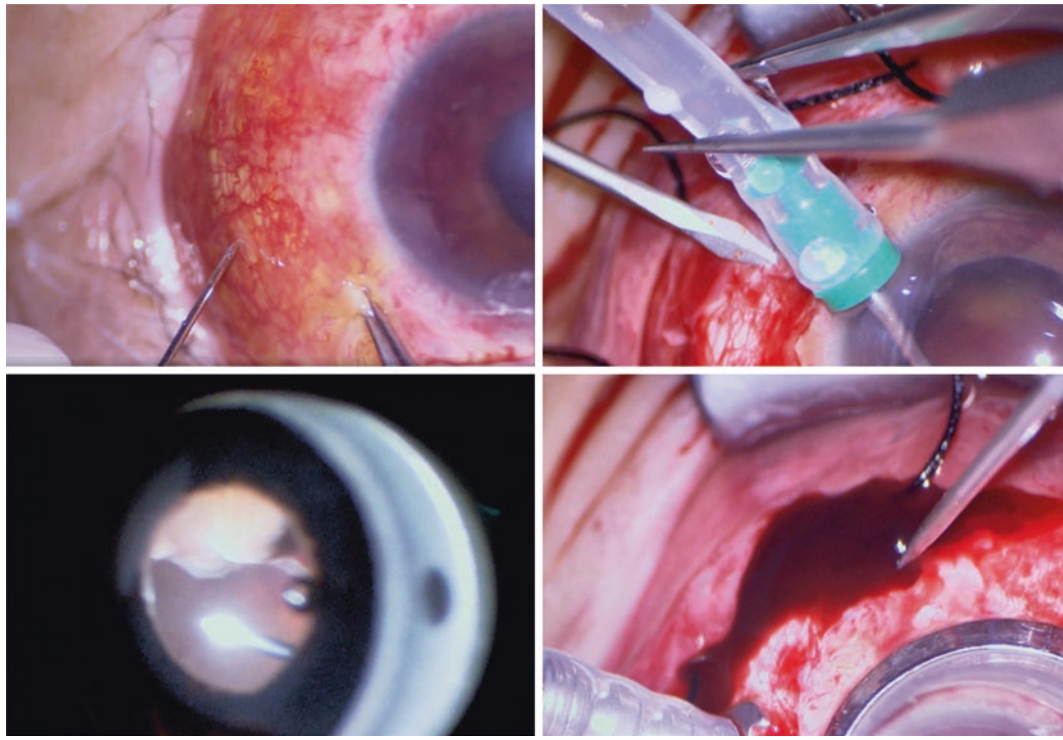

Fig. 8.7 tPA assisted vitrectomy in expulsive choroidal hemorrhage complicating cataract surgery. (Top left) tPA (10μg/0.2 ml) was injected into the suprachoroidal space; Surgery was performed the next day after tPA injection. (Top right) Sclerotomy was performed at the highest point of choroidal detachment to drain the liquefied hemorrhage with anterior chamber maintainer. (Below left) Vitrectomy was conducted with a pars plana infusion, fundus examination showed hemorrhagic choroidal detachment left in the inferior quadrant. (Below right) Sclerotomy at a new site was conducted for a more complete drainage of the hemorrhage

out, the choroidal detachment visibly recessed. At this point, 23G vitrectomy can be performed through the limbus, and then through the pars plana when the choroidal detachment almost resolved, while the drainage cannula is left open to drain any additional choroidal blood. Perfluorocarbons was used for further complete drainage, and finally, the vitreous cavity was filled with silicone oil [51].

To avoid the potential risks of pars plana drainage to the retina and choroid, Flavio A. et al. used 25-gauge cannulas for serous choroidal and 20-gauge cannulas for hemorrhagic choroidal detachment drainage, respectively. They chose to place them further posteriorly (7.0 mm from limbus), guided by ultrasonography, to achieve a more complete drainage without the need for pars plana vitrectomy and use perfluorocarbons/silicone oil to push fluid/hemorrhage posteroanteriorly. In this manner, they assure that the pars plana is reattached and only need to then perform pars plana incisions in cases that have other concomitant comorbidities that require vitrectomy. They suggest that 23-gauge may work just as well as the 25-gauge system for serous drainage; but in eyes with hemorrhagic choroidal detachment, 20-gauge can be used to benefit a wider section and to allow a better flux and easier release of small clots compared with 23- and 25-gauge cannulas [53].

Combined vitrectomy and additional procedures are indicated when there is vitreous or retinal

incarceration, vitreous hemorrhage, retinal breaks, and rhegmatogenous retinal detachment, etc.

Prognosis

An expulsive choroidal hemorrhage has a historically poor prognosis, much worse than a postoperative serous or limited hemorrhagic choroidal detachment has. In globe traumatic ruptures complicated by massive choroidal hemorrhage, uveal prolapse and retinal detachment, visual acuities sometimes can improve from LP preoperatively to 20/70–1/200 postoperatively after treated with choroidal hemorrhage drainage combined with vitrectomy and silicone oil injection [82].

8.4.3 Choroidal Avulsion

Choroidal avulsion should be managed by surgery to reduce the incidence of the intraocular low pressure, and to facilitate retinal reattachment, which is essential for visual improvement and appearance of the eyeball.

Repairment of choroidal avulsion can be conducted at the same time as the pars plana vitrectomy at 7–14 days after the injury. At this time, the choroid is not atrophic and stiff. However, the detached choroid could not be mobilized and could not be reattached to the inner surface of the sclera, either by intraocular perfluorocarbon liquid or by scleral buckling. Several methods were reported to reattach the choroid. According to the scope of the avulsion, Jin M et al. conducted scleral incisions involving the whole scleral layer, and sutured the avulsive choroid back to the inner surface of the sclera using 10-0 nylon suture before the scleral incision was closed. Then perfluorocarbons and silicone oil are injected into the vitreous cavity for further retinal and choroidal reattachment [25].

YR Jiang et al. injected fibrin glue (TianXiu Co., Guangzhou, China) into the suprachoroidal space between the detached choroid and the sclera after an air-fluid exchange was performed to prevent the glue from overflowing from the suprachoroidal space into the vitreous cavity. Five minutes later, when the fibrin clot started to form, silicone oil was injected into the vitreous cavity to attach the retina and to indirectly press the choroid against the sclera [56].

We use a simple technique, continuous mattress suture to repair the choroidal avulsion. During pars plana vitrectomy, a 27-gauge needle with a 10-0 suture enters the vitreous cavity from pars plana on the opposite side of the choroidal avulsion, and passes through the area of avulsive choroid and sclera to outside of eyeball wall. The top of the 10-0 suture is pulled and held. The needle tip is pulled back into vitreous cavity and then passes through the choroid and sclera repeatedly with half o'clock interval. In every procedure of threading the 10-0 suture forms a loop, and top of the suture passes through the loops continuously. When choroidal and scleral suturing finished, the locking-suture loop is adjusted and tied. More than one suture entrance from the pars plana maybe need if the scale of choroidal avulsion larger than three quarters. However, this method is difficult to reattach avulsing choroid in posterior, because it is difficult to get the needle out at the posterior pole (Fig. 8.8).

For patients with cornea opacity, such as cornea blood staining, choroidal and ciliary body suture also can be conducted under direct vision or under temporary keratoprosthesis before penetrating keratoplasty (Fig. 8.8 bottom right).

Prognosis

Choroidal avulsion is still a severe condition in ocular trauma. There is no perfect method to repair serious choroidal avulsion yet. Low IOP is always an annoying situation after choroidal avulsion repairment.

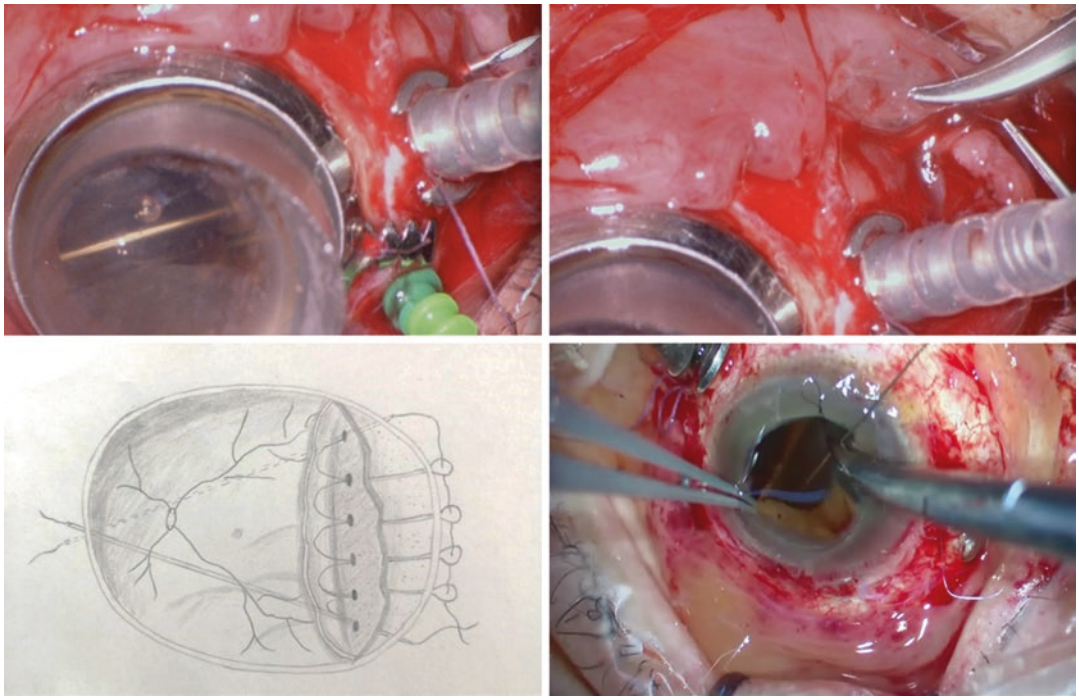

Fig. 8.8 Surgery for choroidal avulsion. (Top left) During pars plana vitrectomy, a 27-gauge needle with a 10-0 suture enters the vitreous cavity from the pars plana at the opposite side of the choroidal avulsion, and passes through the area of avulsive choroid and sclera to outside of eyeball wall. (Top right) The top of the 10-0 suture is pulled and held. The needle tip is pulled back into vitreous cavity and then passes through the choroid and sclera repeatedly with half o'clock interval. (Bottom left) Diagram of continuous mattress suture of choroid and scleral. (Bottom right) For patients with cornea opacity choroidal avulsion suture can be conducted under direct vision by open sky before penetrating keratoplasty

References

1. Kuhn F, Morris R, Witherspoon CD. Birmingham Eye Trauma Terminology (BETT): terminology and classification of mechanical eye injuries. Ophthalmol Clin N Am. 2002;15:139–43.
2. Patel MM, Chee YE, Eliott D. Choroidal rupture: a review. Int Ophthalmol Clin. 2013;53:69–78.
3. Bhagat N, Turbin R, Langer P, Soni NG, Bauza AM, Son JH, Chu D, Dastjerdi M, Zarbin M. Approach to management of eyes with no light perception after open globe injury. J Ophthalmic Vis Res. 2016;11:313–8.
4. Dubinski W, Sharma S. Ophthaproblem. Choroidal rupture. Canadian Family Physician/Medecin de famille canadien. 2006;52:1071, 1079.
5. Pujari A, Chawla R, Agarwal D, Gagrani M, Kapoor S, Kumar A. Pathomechanism of traumatic indirect choroidal rupture. Med Hypothes. 2019;124:64–6.
6. Aguilar JP, Green WR. Choroidal rupture. A histopathologic study of 47 cases. Retina (Philadelphia, PA). 1984;4:269–75.
7. Kempster RC, Green WR, Finkelstein D. Choroidal rupture. Clinicopathologic correlation of an unusual case. Retina (Philadelphia, PA). 1996;16:57–63.
8. Greenberger R, Khangure MS. Exudative choroidal detachment–C.T. features. Australasian Radiol. 1987;31:355–6.
9. Diep MQ, Madigan MC. Choroidal detachments: what do optometrists need to know? Clini Exp Optometry. 2019;102:116–25.
10. Maumenee AE, Schwartz MF. Acute intraoperative choroidal effusion. Am J Ophthalmol. 1985;100:147–54.
11. Jampel HD, Musch DC, Gillespie BW, Lichter PR, Wright MM, Guire KE. Perioperative complications of trabeculectomy in the collaborative initial glaucoma treatment study (CIGTS). Am J Ophthalmol. 2005;140:16–22.
12. Kuhn F, Morris R, Mester V. Choroidal detachment and expulsive choroidal hemorrhage. Ophthalmol Clin North Am. 2001;14:639–50.
13. Hoffman P, Pollack A, Oliver M. Limited choroidal hemorrhage associated with intracapsular cataract

extraction. Archiv Ophthalmol. (Chicago, IL: 1960). 1984;102:1761–5.
14. Bukelman A, Hoffman P, Oliver M. Limited choroidal hemorrhage associated with extracapsular cataract extraction. Archiv Ophthalmol. (Chicago, IL: 1960). 1987;105:338–41.
15. Piper JG, Han DP, Abrams GW, Mieler WF. Perioperative choroidal hemorrhage at pars plana vitrectomy. A case-control study. Ophthalmology. 1993;100:699–704.
16. Ung C, Stryjewski TP, Eliott D. Indications, findings, and outcomes of pars plana vitrectomy after open globe injury. Ophthalmology. Retina. 2020;4: 216–23.
17. Beyer CF, Peyman GA, Hill JM. Expulsive choroidal hemorrhage in rabbits. A histopathologic study. Archiv Ophthalmol. (Chicago, IL: 1960). 1989;107: 1648–53.
18. Cantor LB, Katz LJ, Spaeth GL. Complications of surgery in glaucoma. Ophthalmology. 1985;92:1266–70.
19. Manschot WA. The Pathology of Expulsive Hemorrhage* *This paper was read in abstract form before the XVII International Congress of Ophthalmology New York, 1954. Am J Ophthalmol. 1955;40:15–24.
20. Wolter JR. Expulsive hemorrhage: a study of histopathological details. Graefe's archive for clinical and experimental ophthalmology = Albrecht von Graefes Archiv fur klinische und experimentelle Ophthalmologie. 1982;219:155–8.
21. Wolter JR, Garfinkel RA. Ciliochoroidal effusion as precursor of suprachoroidal hemorrhage: a pathologic study. Ophthalmic Surg. 1988;19:344–9.
22. Saxena RC, Kumar D. Choroidal detachment (a clinico-aetiopathological study). Indian J Ophthalmol. 1983;31:238–241.
23. Brubaker RF, Pederson JE. Ciliochoroidal detachment. Survey Ophthalmol. 1983;27:281–9.
24. Bordeianu CD. The appropriateness of our therapeutic attitude to expulsive hemorrhage. Journal Francais D Ophtalmologie. 1984;7:313–20.
25. Jin M, Yuhua T, Zhaomeng S, et al. Efficacy of combined vitreous surgery and choroidal suture fixation on choroidal avulsion. 眼科学报. 2011:143–7.
26. Raman SV, Desai UR, Anderson S, Samuel MA. Visual prognosis in patients with traumatic choroidal rupture. Canadian J Ophthalmol./Journal canadien d'ophtalmologie. 2004;39:260–266.
27. Wyszynski RE, Grossniklaus HE, Frank KE. Indirect choroidal rupture secondary to blunt ocular trauma. A review of eight eyes. Retina (Philadelphia, PA). 1988;8:237–43.
28. Ament CS, Zacks DN, Lane AM, Krzystolik M, D'Amico DJ, Mukai S, Young LH, Loewenstein J, Arroyo J, Miller JW. Predictors of visual outcome and choroidal neovascular membrane formation after traumatic choroidal rupture. Archiv Ophthalmol. (Chicago, IL: 1960). 2006;124:957–66.
29. Chen KJ. OCT in choroidal rupture with submacular hemorrhage. Ophthalmology. Retina. 2018.
30. Shakin JL, Yannuzzi LA. Posterior segment manifestations of orbital trauma. Adv Ophthalmic Plastic Reconstruct Surg. 1987;6:115–35.
31. Jones WL. A case of an irregular altitudinal field defect associated with a traumatic choroidal rupture. Am J Optometry Physiological Optics. 1981;58: 1134–1137.
32. Glazer LC, Han DP, Gottlieb MS. Choroidal rupture and optic atrophy. Br J Ophthalmol. 1993;77:33–35.
33. Secrétan M, Sickenberg M, Zografos L, Piguet B. Morphometric characteristics of traumatic choroidal ruptures associated with neovascularization. Retina (Philadelphia, PA). 1998;18:62–6.
34. Shin JY, Chung B, Na YH, Lee J, Chung H, Byeon SH. Retinal pigment epithelium wound healing after traumatic choroidal rupture. Acta ophthalmologica. 2017;95:e582–e586.
35. Hart CD, Raistrick R. Indirect choroidal tears and late onset serosanguinous maculopathies. Graefe's archive for clinical and experimental ophthalmology = Albrecht von Graefes Archiv fur klinische und experimentelle Ophthalmologie. 1982;218:206–10.
36. Russell JF, Albini TA, Berrocal AM, Dubovy SR, Rosenfeld PJ, Smiddy WE, et al. Anti-vascular endothelial growth factor therapy for choroidal rupture-associated choroidal neovascularization. Ophthalmol Retina. 2020;4:226–8.
37. Lavinsky D, Martins EN, Cardillo JA, Farah ME. Fundus autofluorescence in patients with blunt ocular trauma. Acta ophthalmologica. 2011;89:e89–94.
38. Harissi-Dagher M, Sebag M, Gauthier D, Marcil G, Labelle P, Arbour JD. Photodynamic therapy in young patients with choroidal neovascularization following traumatic choroidal rupture. Am J Ophthalmol. 2005;139:726–8.
39. Yilmaz G, Aydin P. Visualization of choroidal rupture with indocyanine green angiography. Retina (Philadelphia, PA). 2000;20:315–6.
40. Benillouche J, Astroz P, Ohayon A, Srour M, Amoroso F, Pedinielli A, Mouallem A, Souied EH. Optical coherence tomography angiography imaging of choroidal neovascularization secondary to choroidal rupture treated by intravitreal ranibizumab. Retinal Cases & Brief Reports. 2019.
41. Sheng I, Bauza A, Langer P, Zarbin M, Bhagat N. A 10-year review of open-globe trauma in elderly patients at an urban hospital. Retina (Philadelphia, PA). 2015;35:105–10.
42. Almendárez JE, Vargas DM, González C, Takane M, Koga W. Ultrasound findings in ocular trauma. Arch Soc Esp Oftalmol. 2015;90:572–7.
43. Bindlish R, Condon GP, Schlosser JD, D'Antonio J, Lauer KB, Lehrer R. Efficacy and safety of mitomycin-C in primary trabeculectomy: five-year follow-up. Ophthalmology. 2002;109:1336–42.
44. Yalvac IS, Satana B, Ozkan G, Eksioglu U, Duman S. Management of glaucoma in patients with nanophthalmos. Eye (Lond). 2008;22:838–43.

45. Chu TG, Green RL. Suprachoroidal hemorrhage. Survey Ophthalmol. 1999;43:471–86.
46. Nakakura S, Noguchi A, Tabuchi H, Kiuchi Y. Bimatoprost-induced late-onset choroidal detachment after trabeculectomy: a case report and review of the literature. Medicine (Baltimore). 2017;96:e5927.
47. Law SK, Song BJ, Yu F, Kurbanyan K, Yang TA, Caprioli J. Hemorrhagic complications from glaucoma surgery in patients on anticoagulation therapy or antiplatelet therapy. Am J Ophthalmol. 2008;145: 736–46.
48. Ichhpujani P, Gupta SK, Sood S. Management of choroidal detachment and shallow anterior chamber. J Curr Glaucoma Pract. 2011;5:39–43.
49. Schrieber C, Liu Y. Choroidal effusions after glaucoma surgery. Curr Opin Ophthalmol. 2015;26:134–42.
50. Wirostko WJ, Han DP, Mieler WF, Pulido JS, Connor TB Jr, Kuhn E. Suprachoroidal hemorrhage: outcome of surgical management according to hemorrhage severity. Ophthalmology. 1998;105:2271-2275.
51. Rossi T, Boccassini B, Iossa M, Lesnoni G, Tamburrelli C. Choroidal hemorrhage drainage through 23-gauge vitrectomy cannulas. Retina (Philadelphia, PA). 2010;30:174–6.
52. Yang JG, Li JJ, Tian H, Li YH, Gong YJ, Su AL, He N. Uveal effusion following acute primary angle-closure: a retrospective case series. Int J Ophthalmol. 2017;10:406–412.
53. Rezende FA, Kickinger MC, Li G, Prado RF, Regis LG. Transconjunctival drainage of serous and hemorrhagic choroidal detachment. Retina (Philadelphia, PA). 2012;32:242–9.
54. Shiraki N, Wakabayashi T, Sato T, Sakaguchi H, Nishida K. Intraoperative B-scan ultrasonography and pars plana vitrectomy for severe open globe injury with hemorrhagic retinal and choroidal detachment. Graefe's archive for clinical and experimental ophthalmology = Albrecht von Graefes Archiv fur klinische und experimentelle Ophthalmologie. 2017;255:2287–91.
55. Chee YE, Kanoff JM, Eliott D. Remarkable visual recovery after severe open globe injury. Am J Ophthalmol Case Rep. 2016;3:34–35.
56. Jiang YR, Tao Y, Jonas JB. Traumatic choriodialysis treated by intraocular fibrin glue. Acta ophthalmologica. 2010;88:e129–e130.
57. Wang SH, Lim CC, Teng YT. Airbag-associated severe blunt eye injury causes choroidal rupture and retinal hemorrhage: a case report. Case Rep Ophthalmol. 2017;8:13–20.
58. Holland D, Wiechens B. Intravitreal r-TPA and gas injection in traumatic submacular hemorrhage. Ophthalmologica. Journal international d'ophtalmologie. Int J Ophthalmol. Zeitschrift fur Augenheilkunde. 2004;218:64–9.
59. Goldman DR, Vora RA, Reichel E. Traumatic choroidal rupture with submacular hemorrhage treated with pneumatic displacement. Retina (Philadelphia, PA). 2014;34:1258–60.
60. Hilton GF. Late serosanguineous detachment of the macula after traumatic choroidal rupture. Am J Ophthalmol. 1975;79:997–1000.
61. De Laey JJ. Choroidal neovascularisation after traumatic choroidal rupture. Bulletin de la Societe belge d'ophtalmologie. 1986;220:53–9.
62. Fuller B, Gitter KA. Traumatic choroidal rupture with late serous detachment of macula. Report of successful argon laser treatment. Archiv Ophthalmol. (Chicago, IL: 1960). 1973;89:354–5.
63. Conrath J, Forzano O, Ridings B. Photodynamic therapy for subfoveal CNV complicating traumatic choroidal rupture. Eye (Lond). 2004;18:946–7.
64. Chanana B, Azad RV, Kumar N. Intravitreal bevacizumab for subfoveal choroidal neovascularization secondary to traumatic choroidal rupture. Eye (Lond). 2009;23:2125–6.
65. Puklin JE, Prasad A, Patel CC. Intravitreal bevacizumab in the treatment of choroidal neovascularization from a traumatic choroidal rupture in a 9-year-old child. Retinal Cases Brief Rep. 2009;3:125.
66. Lorusso M, Micelli Ferrari L, Nikolopoulou E, Micelli Ferrari T. Optical coherence tomography angiography evolution of choroidal neovascular membrane in choroidal rupture managed by intravitreal bevacizumab. Case Rep Ophthalmological Med. 2019;2019:5241573.
67. Kim M, Kim JH, Seo Y, Koh HJ, Lee SC. Intravitreal bevacizumab for traumatic choroidal rupture. Optometry Vis Sci: official publication of the American Academy of Optometry. 2015;92:e363–7.
68. Francis JH, Freund KB. Photoreceptor reconstitution correlates with visual improvement after intravitreal bevacizumab treatment of choroidal neovascularization secondary to traumatic choroidal rupture. Retina (Philadelphia, PA). 2011;31:422–4.
69. Gotzaridis EV, Vakalis AN, Sethi CS, Charteris DG. Surgical removal of sequential epiretinal and subretinal neovascular membranes in a patient with traumatic choroidal rupture. Eye. 2003;17:790–1.
70. Abri A, Binder S, Pavelka M, Tittl M, Neumüller J. Choroidal neovascularization in a child with traumatic choroidal rupture: clinical and ultrastructural findings. Clin Exp Ophthalmol. 2010;34:460–3.
71. Gross JG, King LP, De Juan E, Powers T. Subfoveal neovascular membrane removal in patients with traumatic choroidal rupture. Ophthalmology. 1996;103:579–85.
72. Sulewski ME, Kracher GP, Gottsch JD, Stark WJ. Use of the disposable contact lens as a bandage contact lens. Archiv Ophthalmol. (Chicago, IL: 1960). 1991;109:318.
73. Bellows AR, Chylack LT Jr, Hutchinson BT. Choroidal detachment. Clinical manifestation, therapy and mechanism of formation. Ophthalmology. 1981;88:1107–15.
74. Krishnan M, Baskaran R. Management of postoperative choroidal detachment. Indian J Ophthalmol. 1985;33:217.

75. de Barros DS, Navarro JB, Mantravadi AV, Siam GA, Gheith ME, Tittler EH, et al. The early flat anterior chamber after trabeculectomy: a randomized, prospective study of 3 methods of management. J Glaucoma. 2009;18:13–20.
76. WuDunn D, Ryser D, Cantor LB. Surgical drainage of choroidal effusions following glaucoma surgery. J Glaucoma. 2005;14:103–8.
77. Allen JC. Surgical treatment of choroidal detachment. Ophthalmic Surg. 1976;7:95–7.
78. Glazer LC, Williams GA. Management of expulsive choroidal hemorrhage. Semin Ophthalmol. 1993;8:109–13.
79. Lambrou FH Jr, Meredith TA, Kaplan HJ. Secondary surgical management of expulsive choroidal hemorrhage. Archiv Ophthalmol. (Chicago, IL: 1960). 1987;105:1195–8.
80. Ozdek S, Hasanreisoglu M, Yuksel E. Chorioretinectomy for perforating eye injuries. Eye (Lond). 2013;27:722–7.
81. Fei P, Jin HY, Zhang Q, Li X, Zhao PQ. Tissue plasminogen activator-assisted vitrectomy in the early treatment of acute massive suprachoroidal hemorrhage complicating cataract surgery. Int J Ophthalmol. 2018;11:170–1.
82. Liggett PE, Mani N, Green RE, Cano M, Ryan SJ, Lean JS. Management of traumatic rupture of the globe in aphakic patients. Retina (Philadelphia, PA). 1990:S59–64.

9 Complicated Ocular Trauma with Corneal Opacity

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Abstract

Eye trauma is a relevant cause of blindness and visual impairment worldwide. The cornea and the corneoscleral region are the most common places of injury. The main etiologies of corneal injuries are corneal abrasion, corneal foreign bodies, corneal perforation, and eye burns. Damage to the cornea leads to swelling, increased thickness, and development of opacification due to the healing process. Therefore, a normal cornea allows visualization of intraocular surgical maneuvers in the anterior and posterior segment of the eye during eye surgery. In situations requiring immediate action by the ocular surgeon, numerous abnormalities in the anterior segment (blood in the anterior chamber, iris deformities, pupil membranes, and traumatic cataracts) can be managed if the cornea is transparent. However, the presence of important corneal opacities (such as intense or rapidly progressing corneal edema, large and thick scars, hematic impregnation, and neovascularization of the cornea) impairs a safe and effective vitrectomy, limiting the possibility of reconstruction of the anterior and posterior segments. The posterior segment surgeon is faced with the following choices when dealing with eyes with corneal opacities: postponing posterior segment surgery while waiting for corneal clearing; performing immediate surgery with temporary keratoprosthesis (TKP) combined with pars plana vitrectomy (PPV) or performing endoscopy-guided vitrectomy. The decision will be guided by the urgency to approach the posterior segment and the availability of materials. TKP or endoscopic vitrectomy is ideal in urgent cases that require immediate surgical approach. Furthermore, two cases of trauma with opacities, one with blood in the anterior chamber (hyphema) and the other with corneal opacity.

Keywords

Ocular trauma · Corneal opacity
Vitrectomy · Hyphema · Keratoprosthesis

9.1 Introduction

Eye trauma is a relevant cause of blindness and visual impairment worldwide, with around 1.6 million people bilaterally blind and an additional 19 million suffering from monocular blindness [1]. This pathology affects economically active

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young people, with significant societal, human, and socioeconomic losses due to these events. The cornea and the corneoscleral region are the most common places of injury due to the greater exposure of these structures to impact, and knowledge of the anatomy of the anterior segment and the trauma mechanism are fundamental to its correct therapeutic approach [3]. The morbidity of corneal injuries can vary greatly, from small (such as corneal abrasions and foreign bodies) up to large, vision threatening injuries. Penetrating trauma with damage to the anterior and posterior segment and eye burns that affect the limbus are the most serious corneal injuries, due to the potential for irreversible functional sequelae such as glaucoma and tractional detachment of the retina [4, 5].

9.2 Etiology

The main etiologies of corneal injuries are corneal abrasion, corneal foreign bodies, corneal perforation (Fig. 9.1), and eye burns (greater potential for morbidity). Eye burns can be chemical (acidic and alkaline), thermal, or by ultraviolet radiation (UV).

Alkaline chemical burns are the most common due to the prevalence of household agents containing caustic soda and ammonia.

Thermal burns are rare, usually related to accidents involving fire or fireworks. Corneal laceration and perforation are usually accidental or work related, involving direct impact of objects at high speed.

When collecting the anamnesis, it is essential to know the type of object that impacted the globe and the type of activity that was being performed, in order to investigate the presence of intraocular foreign body, which has its incidence higher in cases involving metal x metal impact [6].

9.3 Pathophysiology

The pathophysiology of corneal trauma involves the mechanism of corneal injury and its healing in response to trauma. The corneal epithelium quickly regenerates in the face of corneal abrasion (24–48 h). For chemical burns, the composition of the substance is what determines the extent of the injury. Corneal lesions due to alcohol cause immediate de-epithelialization, but there is spontaneous re-epithelialization without sequelae. Injuries by acidic substances, on the other hand, cause coagulation and tissue necrosis, causing greater morbidity to ocular tissues. However, the alkali lesion has the highest morbidity as it causes hydrophilic and lipophilic degeneration. This is due to the saponification process of cell membranes in contact with alkali, leading to immediate cell penetration and tissue degradation.

In the corneal healing process, cell migration occurs initially, followed by cell proliferation and differentiation. Limbic stem cells are essential for the regeneration of the corneal epithelium, with migration and remodeling of the extracellular matrix. These cells migrate through a single layer to cover an epithelial defect, being this phe-

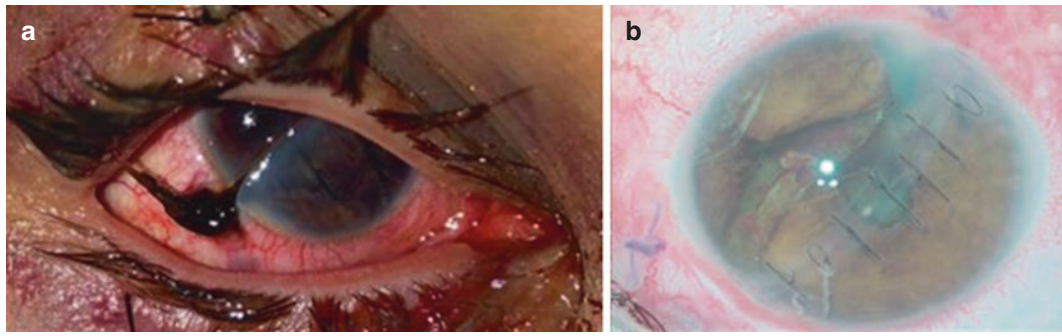

Fig. 9.1 Corneal trauma. Before (**a**) and after (**b**) first surgery

nomenon essential to cure corneal abrasions. The complete process of epithelial regeneration, with restoration of the original epithelial thickness by the basal cells takes up to 6 weeks and recurrent corneal erosions can occur in cases where this process is incomplete. However, in deeper traumas, with stromal involvement, cell differentiation of keratocytes into myofibroblasts and fibroblasts occurs, leading to corneal opacity [7].

Penetrating corneoscleral traumas can trigger a cascade of inflammation that leads to retinal detachment. Several studies of penetrating trauma in animal models have evaluated the wound healing process associated with the development of tractional retinal detachment and Proliferative Vitreoretinopathy (PVR) [8–11]. Studies have also emphasized the importance of the presence of vitreous hemorrhage in trauma in the pathophysiology of traction retinal detachment and development of PVR, being a more significant factor than the rupture of the lens [12, 13].

The location of the penetrating lesion is also important in the development of PVR and retinal detachment in trauma. A study using a penetrating trauma model in rabbits showed that injury through the equatorial retina resulted in 16% retinal detachment, injury at ora serrata level resulted in 78% retinal detachment, and injury through peripheral retina with involvement of the ciliary body resulted in 14% retinal detachment. This result emphasizes the role of the vitreous base as a framework for cell proliferation and the development of traction, culminating in detachment of the traction retina [14].

9.4 Evaluating Corneal Opacities

The Cornea is an ocular tissue of the anterior segment whose main function depends on its transparency, which allows the transmission of light to the retina. Normal hydration of the cornea is vital to maintain its transparency. Damage to the epithelium, stroma, or endothelium, for a variety of causes, leads to swelling, increased thickness, and the development of opacification due to the healing process. Therefore, a normal cornea, that is, a transparent cornea is of utmost importance for individual visual function and to allow visualization of intraocular surgical maneuvers in the anterior and posterior segment of the eye during eye surgery (Fig. 9.2) [15–17].

There are numerous causes that lead to loss of corneal clarity (such as infections, trauma, dystrophies). Such conditions culminate in a common clinical denominator: corneal opacity. Such conditions lead to visual disturbances and difficulty in potential anterior and posterior segment surgical approaches [16, 17].

Transparency is defined by relating the amount of light that spreads and/or is absorbed when passing through an object. Upon entering the cornea, light follows two main paths: direct line through the eye or it is dispersed. The greater the light scattering, the more opaque the cornea is and the less transparent it is. In normal individuals, transparency depends on the transmission of light in each layer of the cornea, especially the stroma, in such a way that the uniform organization of collagen fibrils minimizes light scattering [16, 17].

In clinical practice, corneal opacities are usually assessed subjectively by the ability to visualize structures posterior to the cornea (such as the anterior chamber and iris). There is a grade used in corneal transplants, which subjectively ranks from 0+ to 5+, with 0+, clear, 1+ Minimum opacity (pupil margin and iris vessels clearly seen through the cornea); and 5+ (maximum stromal

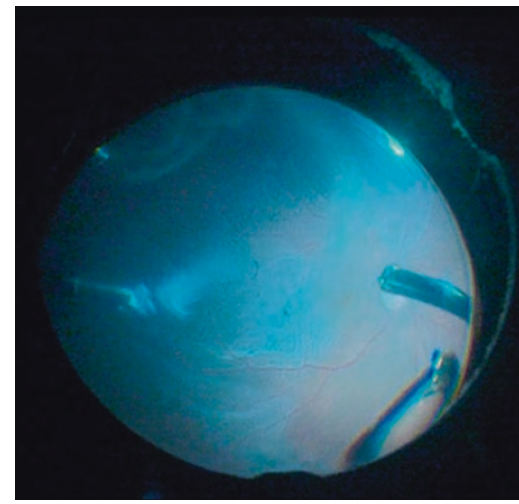

Fig. 9.2 Vitrectomy through the transparent cornea

opacity, anterior chamber not visible) [18]. Another way of assessing common clinical use is describing the size and location of the opacities. Such qualitative description allows serial longitudinal tracking through the photographic record through the slit lamp in order to monitor changes. One of the problems with subjective evaluation is that you are a dependent examiner. Therefore, more objective methods are imperative in order to assist clinical decision and standardize language among researchers [17].

The first method measures the amount of light transmitted by the cornea, measuring the corneal function. Spectrophotometry directly quantifies the corneal transparency with the emission of light at a predefined intensity through the cornea, measuring how much of the predefined light has passed. Its limitations are related to the use in corneal ex vivo and the equipment is not widely available [19–21].

Commercially available equipment such as anterior segment Optical Coherence Tomography (OCT) or the Scheimpflug, can quantify the scattering of light by the cornea. They measure corneal opacity through the estimation of the amount of light that has dispersed, that is, the amount of light that does not pass through the cornea, and is lost or dissipated. This device can detect how much of the light has returned towards the observer. The Pentacam system can measure the scattering of light from the cornea in a 0–100 scale. Such information can create a densitometry map representing the grade of corneal opacity [22–24].

9.5 Treatment

Visualization is the paramount for ocular surgery. Faced with a medium opacity that makes visualization difficult, providing a clear visual axis will be the surgeon's first step during ocular trauma surgery.

In situations requiring immediate action by the ocular surgeon, numerous abnormalities in the anterior segment (blood in the anterior chamber, iris deformities, pupil membranes, and traumatic cataracts) can be managed if the cornea is transparent. Small and thin corneal scars usually allow good visualization so Pars Plana Vitrectomy(PPV) is made in the usual way.

However, the presence of important corneal opacities (such as intense or rapidly progressing corneal edema, large and thick scars, hematic impregnation, and neovascularization of the cornea) impairs a safe and effective vitrectomy, limiting the possibility of reconstruction of the anterior and posterior segments.

The posterior segment surgeon is faced with the following choices when dealing with eyes with corneal opacities: postponing posterior segment surgery while waiting for corneal clearing; performing immediate surgery with temporary keratoprosthesis (TKP) combined with Pars Plana vitrectomy (PPV) or performing endoscopy-guided vitrectomy. The decision will be guided by the urgency to approach the posterior segment and the availability of materials. TKP or endoscopic vitrectomy is ideal in urgent cases that require immediate surgical approach [25, 26].

The development of the temporary keratoprosthesis (TKP) allowed posterior segment view for severely traumatized eyes. Landers, 1981, described use TKP with PPV [27].

The TKP is attached to the sclera, providing good wide-field visualization of the posterior pole and the periphery of the retina [28]. In all cases, the first step is to perform the reconstruction of the eyeball (corneal suture). TKP/PVV is performed in a second moment.

A corneal trepanation is performed to fit the TKP, and phacoemulsification or open sky removal of the lens with intraocular lens implantation is performed as needed. Landers TKP is positioned in the corneal bed and sutured in the sclera with 6.0 silk or nylon. Then, surgical vitreoretinal maneuvers are done according to the underlying pathology. At the end of the case, TKP is removed and penetrating keratoplasty (PKP) is performed (Fig. 9.3) [25].

Endoscopic vitrectomy was developed to remove an intraocular foreign body in its first

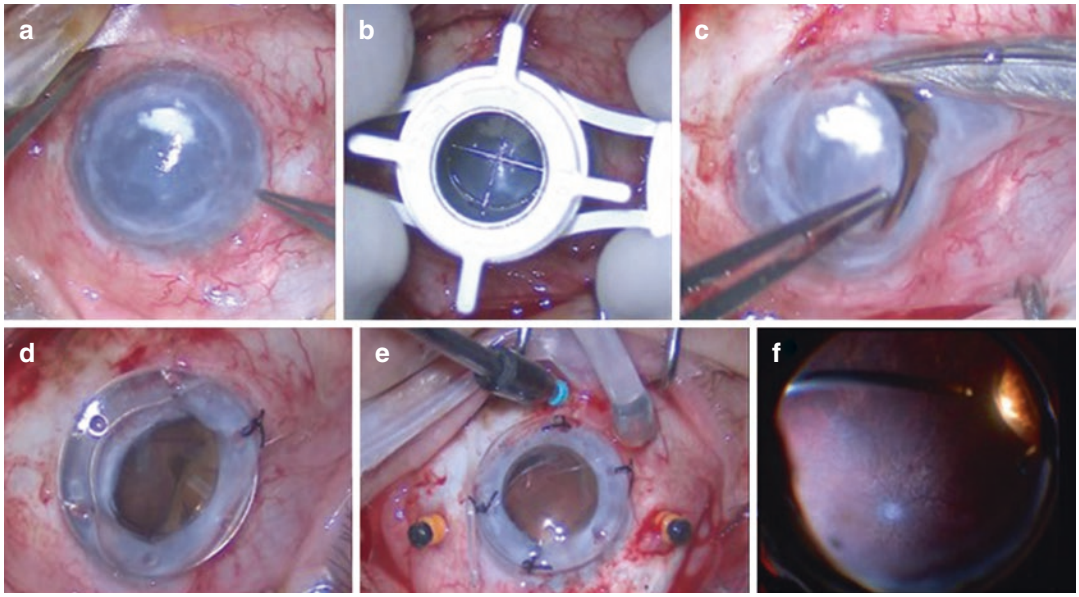

Fig. 9.3 Corneal opacity (**a**); corneal trepanation (**b**); keratectomy (**c**); Landers TKP (**d**, **e**); PPV (**f**)

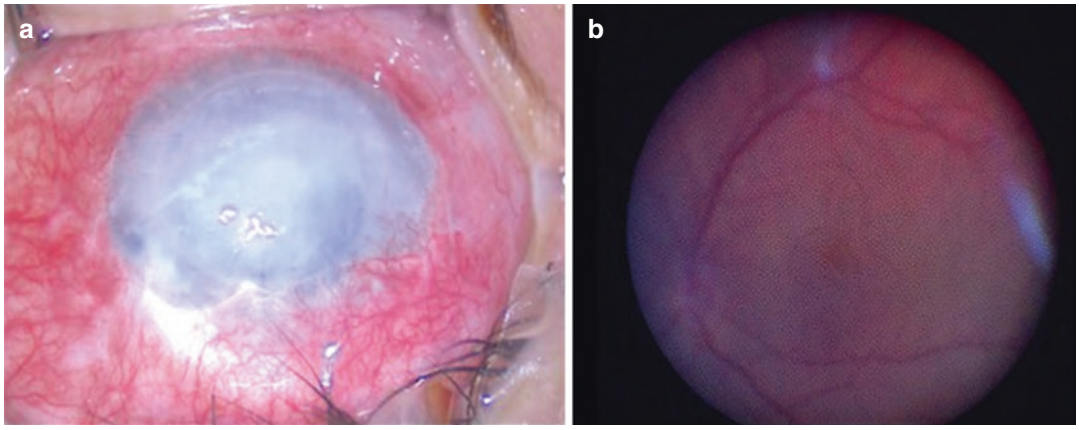

Fig. 9.4 Corneal opacity (**a**) and endoscopic view (**b**) to assess viability of the retina

description in the literature [29]. In 1990, flexible 20G endoscopes were developed to treat numerous posterior segment diseases [30]. Soon after, video endoscopes appeared, with a camera for remote real-time viewing of both the microscope and endoscope images [31]. Current equipment contains a 23G, Xenon light source, a fiber optic video camera connected to a monitor, and an endolaser unit (810 nm Diode). The 20 gauge probes provide 90–140 degrees of visual field. Endoscopic probes can be straight or curved.

Its ability to bypass the anterior segment allows immediate vitreoretinal interventions without dependency on the transparency of the cornea. In this way, it is a very important tool in opaque corneal situations. The learning curve for mastering endoscopic vitrectomy is slow (Fig. 9.4).

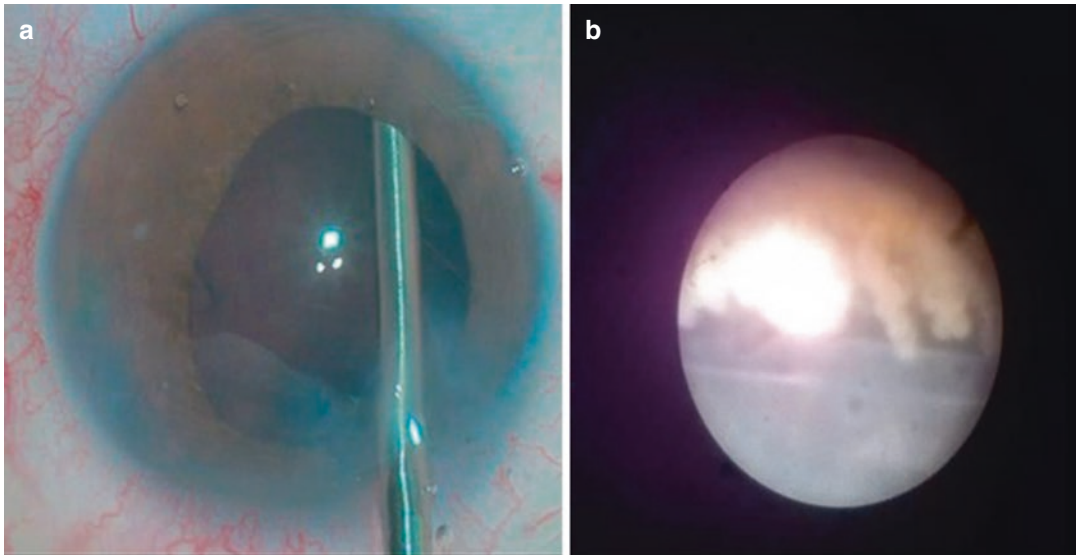

Fig. 9.5 Simultaneous microscopic (**a**) and endoscopic (**b**) visualization to perform endocyclophotocoagulation

The surgery is performed using a separate monitor which nowadays can be attached to a 3D surgical visualization system (Fig. 9.5) [26].

9.6 Surgical Case 1

A 48-year-old man suffered penetrating trauma to his left eye with a piece of glass at work. The patient removed the object from the own eye immediately after the trauma, suffering a corneal laceration from limbus to limbus. On initial examination, in addition to the corneal lesion, the eye presented a total hyphema, making it impossible to assess the anterior and posterior segments.

The patient's visual acuity was 20/20 in the right eye and light perception in the left eye. Orbital CT Scans were performed, ruling out the presence of an intraocular foreign body.

The patient was immediately submitted to corneal suture and aspiration of the intraocular blood in the anterior chamber (Fig. 9.6). In the following week, ocular ultrasound was performed,

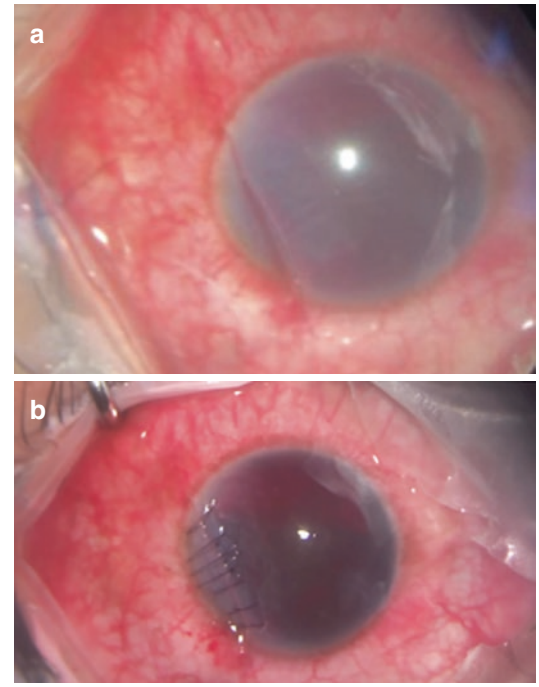

Fig. 9.6 (**a**) Immediate pre-operative appearance of the corneal laceration from limbus to limbus and total hyphema. (**b**) After the corneal suture, with 10-0 nylon

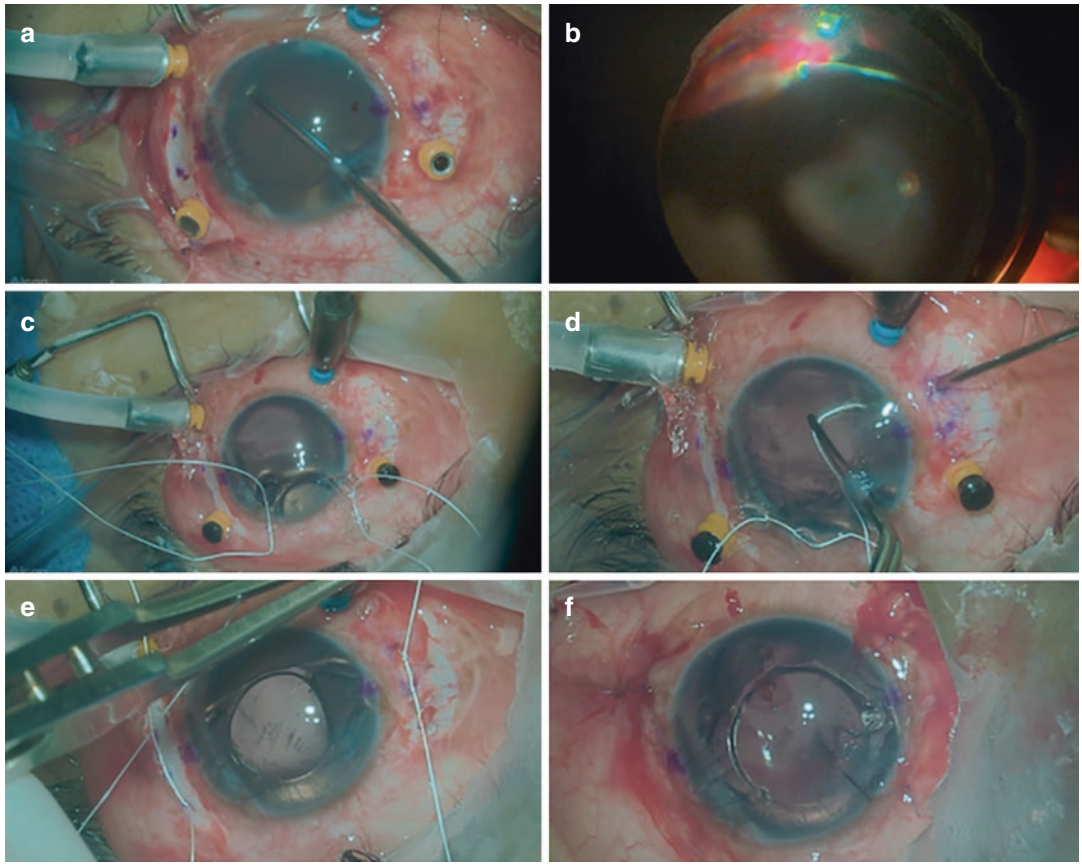

Fig. 9.7 Scleral IOL fixation with GORE-TEX suture cv-8. (**a**) Anterior vitrectomy; (**b**) dense vitreous hemorrhage deposited inferiorly; (**c**) passage of the suture thread through the haptics of the Akreos lens; (**d**) Retinal forceps are used to expose each end of the suture to the level of the sclerotomy previously marked at the beginning of the surgery in order to ensure the proper positioning of the IOL. (**e**) Suture placed in the sclera. (**f**) Final aspect of the surgery

revealing aphakia and vitreous hemorrhage, with the retina applied.

After 14 days of the first surgical intervention, Pars Plana Vitrectomy with scleral fixation of Akreos IOL with GORE-TEX suture was performed (Fig. 9.7).

During surgery, it was observed, in addition to the aphakia, aniridia and dense vitreous hemorrhage, without retinal tears or detachment.

Despite the temporal corneal opacity, good visualization of the retina was obtained through the use of the BIOM non-contact system. The patient evolved with a best corrected visual acuity of 20/25 with correction, centered IOL and a colored contact lens was adapted postoperatively to address photophobia (Fig. 9.8).

9.7 Surgical Case 2

An 8-year-old female patient suffered a sharp trauma in the right eye with a key.

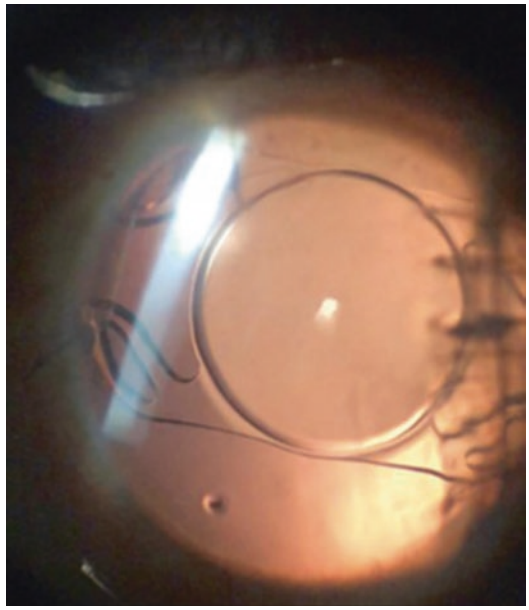

Fig. 9.8 Late 3-month postoperative period. Centralized IOL, visual acuity of 20/20 with best correction. The patient underwent a colored contact lens adaptation in order to reduce the photophobia caused by aniridia

The patient presented extensive corneal laceration with herniated iris and lens material in the anterior chamber. She was immediately submitted to a corneal suture procedure with lensectomy and anterior vitrectomy; the crystalline material was aspirated with the vitrectomy probe (Fig. 9.9).

As it was not possible to observe capsular support for IOL implantation, it was decided not to implant it at this time. In the first week after surgery, the patient underwent ocular ultrasound, which revealed a clear vitreous and an applied retina, and a new surgical procedure was scheduled for a secondary IOL implantation 2 weeks after the first surgery.

During the second surgical procedure, anterior and posterior synechiae were undone and IOL implanted in the ciliary sulcus, with good centralization (Fig. 9.10). The patient presented a good postoperative evolution, with transparency of the visual axis despite the nasal corneal opacity and good centralization of the intraocular lens after 1 month of surgery (Fig. 9.11).

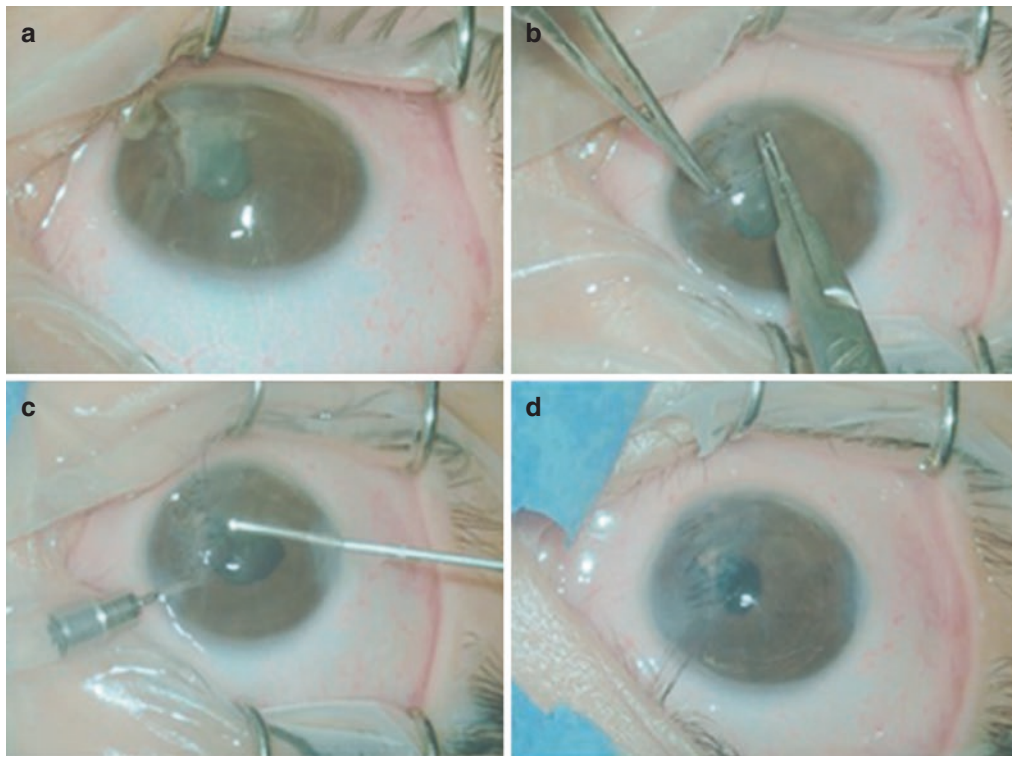

Fig. 9.9 (**a**) Intraoperative image showing an extensive, irregular laceration, tamponaded by the iris, with crystalline material in the anterior chamber. (**b**) Corneal suture with 10-0 nylon after filling the anterior chamber with methylcellulose. (**c**) Anterior vitrectomy with vitrectomy probe was performed, with aspiration of the lens material and with the infusion in the anterior chamber. (**d**) Final aspect of the surgery

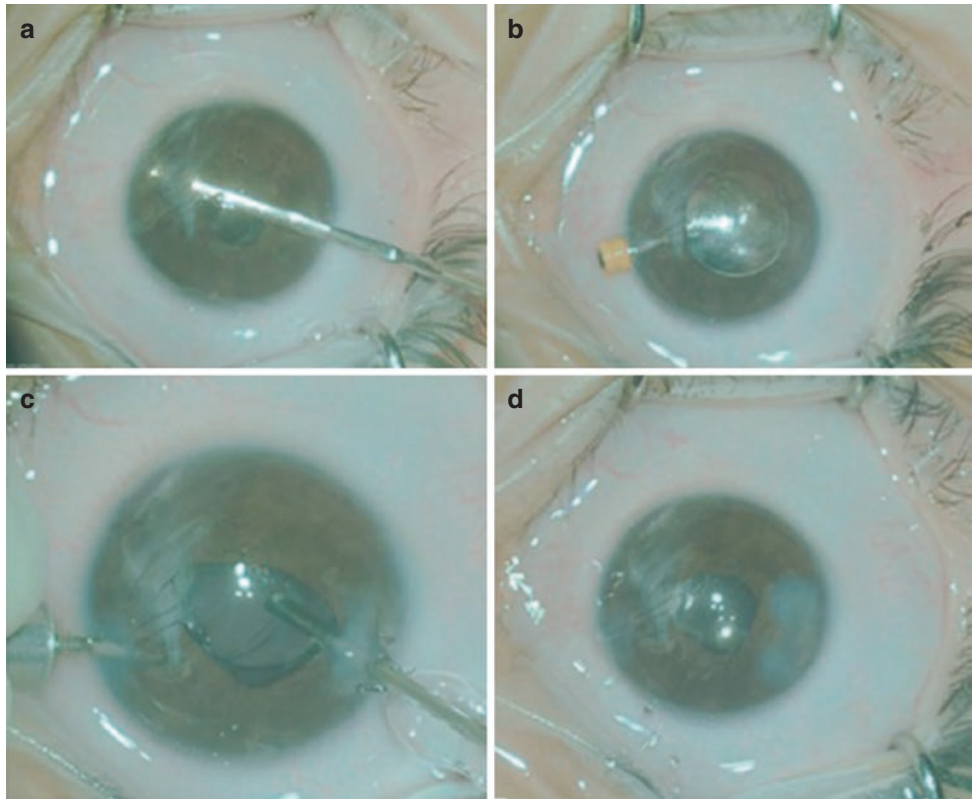

Fig. 9.10 IOL implantation 15 days later. (**a**) After filling the anterior chamber with methylcellulose, anterior and posterior synechiae were undone with an iris spatula. It was also used to certify that there was 360 degree capsular support for the lens implant in the ciliary sulcus. (**b**) The intraocular lens was implanted through a 2.75 mm incision and initially positioned over the iris and then placed in the ciliary sulcus. (**c**) Anterior vitrectomy was performed. (**d**) Final aspect of the surgery, with good IOL centering

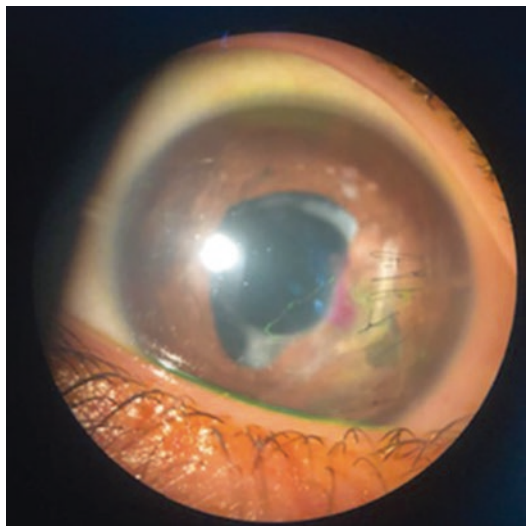

Fig. 9.11 Appearance of the 30-day postoperative period: corectopy, nasal corneal opacity, centered intraocular lens with free visual axis and 20/30 visual acuity with best correction

References

1. Lima-Gómez V, Blanco-Hernández DM. Expected effect of treatment on the rate of visual deficiency after ocular trauma. Cir Cir. 2010;78:302–9.
2. Tielsch JM, Parver LM. Determinants of hospital charges and length of stay for ocular trauma. Ophthalmology. 1990;97:231–7.
3. Kuhn F, Morris R, Witherspoon CD, Mester V. The Birmingham Eye Trauma Terminology system (BETT). J Fr Ophtalmol. 2004;27:206–10.
4. Saka ES, Monsudi KF, Olatuji V. TRAUMATIC CORNEAL LACERATION IN NORTHWESTERN NIGERIA. J West Afr Coll Surg. 2017;7(4):72–84.
5. Bremond-Gignac D, Copin H, Benkhalifa M. Corneal epithelial stem cells for corneal injury. Expert Opin Biol Ther. 2018;18(9):997–1003.
6. Hossain RR, Papamichael E, Coombes A. East London deliberate corrosive fluid injuries. Eye (Lond). 2020;34(4):733–9.
7. Barrientez B, Nicholas SE, Whelchel A, Sharif R, Hjortdal J, Karamichos D. Corneal injury: clinical and molecular aspects. Exp Eye Res. 2019;186:107709.

8. Cleary PE, Ryan SJ. Experimental posterior penetrating eye injury in the rabbit. II. Histology of wound, vitreous, and retina. Br J Ophthalmol. 1979;63:312–21.
9. Cleary PE, Ryan SJ. Method of production and natural history of experimental posterior penetrating eye injury in the rhesus monkey. Am J Ophthalmol. 1979;88:212–20.
10. Cleary PE, Jarus G, Ryan SJ. Experimental posterior penetrating eye injury in the rhesus monkey: vitreous-lens admixture. Br J Ophthalmol. 1980;64:801–8.
11. Cleary PE, Minckler DS, Ryan SJ. Ultrastructure of traction retinal detachment in rhesus monkey eyes after a posterior penetrating ocular injury. Am J Ophthalmol. 1980;90:829–45.
12. Hsu HT, Ryan SJ. Natural history of penetrating ocular injury with retinal laceration in the monkey. Graefes Arch Clin Exp Ophthalmol. 1986;224:1–6.
13. Hsu HT, Ryan SJ. Lens trauma in experimental penetrating eye injury. In: Henkind P, editor. Acta: XXIV International Congress of Ophthalmology. Philadelphia: JB Lippincott; 1983.
14. Hsu HT, Ryan SJ. Experimental retinal detachment in the rabbit: penetrating ocular injury with retinal laceration. Retina. 1986;6:66–9.
15. Freegard TJ. The physical basis of transparency of the normal cornea. Eye. 1997;11(4):465–71. https://doi.org/10.1038/eye.1997.127.
16. Maurice DM. The structure and transparency of the cornea. J Physiol. 1957;136:263–86. https://doi.org/10.1113/jphysiol.1957.sp005758.
17. Dohlman TH, Yin J, Dana R. Methods for assessing corneal opacity. Semin Ophthalmol. 2019;34(4):205–10. https://doi.org/10.1080/08820538.2019.1620796.
18. Sonoda Y, Streilein JW. Orthotopic corneal transplantation in mice–evidence that the immunogenetic rules of rejection do not apply. Transplantation. 1992;54(4):694–704. https://doi.org/10.1097/00007890-199210000-00026.
19. Doutch J, Quantock AJ, Smith VA, Meek KM. Light transmission in the human cornea as a function of position across the ocular surface: theoretical and experimental aspects. Biophys J. 2008;95:5092–9. https://doi.org/10.1529/biophysj.108.132316.
20. Christens-Barry WA, Green WJ, Connolly PJ, Farrell RA, McCally RL. Spatial mapping of polarized light transmission in the central rabbit cornea. Exp Eye Res. 1996;62:651–62. https://doi.org/10.1006/exer.1996.0076.
21. Doutch JJ, Quantock AJ, Joyce NC, Meek KM. Ultraviolet light transmission through the human corneal stroma is reduced in the periphery. Biophys J. 2012;102:1258–64. https://doi.org/10.1016/j.bpj.2012.02.023.
22. Rose JS, Eldrina J, Joshua A, et al. Objective quantification of corneal haziness using anterior segment optical coherence tomography. J Curr Ophthalmol. 2018;30:54–7. https://doi.org/10.1016/j.joco.2017.08.001.
23. Wang J, Simpson TL, Fonn D. Objective measurements of corneal light-backscatter during corneal swelling, by optical coherence tomography. Invest Opthalmol Vis Sci. 2004;45(10):3493. https://doi.org/10.1167/iovs.04-0096.
24. Ni Dhubhghaill S, Rozema JJ, Jongenelen S, Ruiz Hidalgo I, Zakaria N, Tassignon M-J. Normative values for corneal densitometry analysis by Scheimpflug optical assessment. Invest Ophthalmol Vis Sci. 2014;55:162–8. https://doi.org/10.1167/iovs.13-13236.
25. Nowomiejska K, Haszcz D, Forlini C, et al. Wide-field Landers temporary keratoprosthesis in severe ocular trauma: functional and anatomical results after one year. J Ophthalmol. 2015;2015:163675. https://doi.org/10.1155/2015/163675.
26. Yonekawa Y, Papakostas TD, Marra KV, Arroyo JG. Endoscopic pars plana vitrectomy for the management of severe ocular trauma. Int Ophthalmol Clin. 2013;53(4):139–48. https://doi.org/10.1097/IIO.0b013e3182a12b1f.
27. Landers MB III, Foulks GN, Landers DM, Hickingbotham D, Hamilton RC. Temporary keratoprosthesis for use during pars plana vitrectomy. Am J Ophthalmol. 1981;91(5):615–9.
28. Toth A, Landers MB III. A new wide-field temporary keratoprosthesis. Retina. 1993;13(4):353–5.
29. Thorpe H. Ocular endoscope: instrument for removal of intravitreous nonmagnetic foreign bodies. Trans Am Acad Ophthalmol Otolaryngol. 1934;39:422–4.
30. Volkov VV, Danilov AV, Vassin LN, Frolov YA. Flexible endoscopes. Ophthalmoendoscopic techniques and case reports. Arch Ophthalmol. 1990;108(7):956–7. https://doi.org/10.1001/archopht.1990.01070090058039.
31. Eguchi S, Araie M. A new ophthalmic electronic videoendoscope system for intraocular surgery. Arch Ophthalmol. 1990;108(12):1778–81. https://doi.org/10.1001/archopht.1990.01070140132046.

Traumatic Giant Macular Hole

10

Tingkun Shi, Qi Zhang, and Haoyu Chen

Abstract

Traumatic giant macular hole is the full-thickness defect of fovea caused by blunt trauma and with a diameter of more than 1500 μm. The pathogenesis may be vitreofoveal traction caused by global expansion during contusion, or the inflammation and necrosis of the retina after injury. Optical coherence tomography can confirm the diagnosis and quantitatively measure the diameter. Traumatic giant macular hole is difficult to close, even after surgery. Several surgical techniques, including inverted internal limiting membrane (ILM) flap, autologous ILM transplant, autologous platelet implantation, amniotic membrane plug, and autologous retinal transplant have been shown to promote the closure of giant macular hole.

Keywords

Giant macular hole · Traumatic macular hole Optical coherence tomography · Internal limiting membrane flap

10.1 Introduction

Traumatic giant macular hole is a rare clinical condition. It usually refers to the macular hole caused by blunt trauma and with a diameter of more than 1500 μm. Traumatic giant macular hole is difficult to close spontaneously, even after an operation. Several surgical techniques may promote the closure of a traumatic giant macular hole. But the visual outcome may still be not improved, which remains a challenge. This chapter discusses the definition, clinical manifestation, diagnosis, and management of traumatic giant macular hole.

10.2 Definition

Macular hole is the full-thickness neurosensory retina defect at the fovea. Although most macular hole patients are idiopathic, there are several causes of secondary macular hole—ocular trauma, high myopia, proliferative diabetic retinopathy, Alport syndrome, etc. Most traumatic macular hole cases occur in ocular contusions; a small number of cases happened in open globe injury, laser and solar damage [1–3]. Unlike idiopathic macular hole, traumatic macular hole more commonly occurs in adolescents, more male than female, with age ranges from 15- to 36 years old, mean 22 years. Most of the injuries are small spherical subjects, such as tennis, badminton, plastic bullets, and golf balls. It may occur

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either at the time of injury or following the resolution of commotio retinae [4]. The mechanism of traumatic macular hole from a blunt ocular trauma was theorized that a sudden compression and expansion of the globe that produces significant stress on the fovea, which would lead to severe traction from vitreous to the fovea, and formation of the macular hole [5]. Another possible mechanism was thought due to inflammation and necrosis of the retina after blunt contusion. They are often concurrent with other changes, such as choroidal rupture, contusions of the retinal neurosensory and pigmentary epithelial layers, subretinal hemorrhage, equatorial breaks, and retinal dialysis [6].

The size of a macular hole is an important parameter that affects the visual acuity and the outcome after surgical intervention. The larger the diameter, the more difficult the macular hole closes after surgical intervention. However, there was no consistent definition of "large macular hole." Some articles used >400 μm as the criteria [7], while some others used >700 μm [8]. Also, the definition of a giant macular hole is not consistent. Few articles used the basal diameter as the measurement [9], but most accepted opinion is the minimal diameter of the hole should be measured and used as the criteria. In an article by Dr. Lyu, minimal diameter ≥700 μm was used to define a giant macular hole [10]. While some other articles used the criteria of 1500 μm [11, 12]. In our opinion, minimal diameter >1500 μm is a good definition for a giant macular hole. The optic disc's diameter is about 1500 μm, and it can be easily used as a reference.

Giant macular hole is a rare condition. There are no large case series, but only some case reports in the literature. The cause of giant macular hole in the case reports includes Alport syndrome [11], choroiditis [13], retinal pigment epithelial tear [14], and blunt trauma [9, 12, 15–17].

10.3 Symptoms and Signs

The patient sustained with traumatic macular hole mostly complained of severe decreased or loss of central vision, metamorphopsia or central darkness and combined other traumatic symptoms, such as pain, photophobia, tears, and blepharospasm. On biomicroscopic examination, macular hole is a round or oval hole with sharp edges in the fovea. But some traumatic macular holes have more irregular edges and sharp boundaries [5]. If the macular hole's size is larger than the optic disc, it is likely a giant macular hole. The bottom of the hole is crimson (the color of deep choroidal vascular). Light band interruption can be visible under the ophthalmoscope with slit light, and a white halo around the hole can be seen when subretinal fluid is present. Optical coherence tomography (OCT) provides in vivo high-resolution cross-sectional images of retinal tissues' microstructure. OCT can not only demonstrate the full-thickness defect at the fovea, but also quantitatively measure the diameter and show the accompanied changes such as intraretinal cyst and commotio retinae.

10.4 Diagnosis

The diagnosis criteria mainly depend on the history of trauma or exposure to solar or laser and full thickness of neurosensory retina defect at the fovea. OCT could confirm the diagnosis and measure the minimal diameter of the macular hole. When the minimal diameter ≥1500 μm, giant macular hole can be diagnosed.

10.5 Treatment

1. Follow-up and observation

It was reported that a significant proportion of traumatic macular holes could close spontaneously (25–66.7%) [18–21]. Our previous study found that 37% of traumatic macular holes closed spontaneously. Smaller minimal diameter and fewer intraretinal cysts are the predictive factors for spontaneous closure. Therefore, the giant macular hole cannot close spontaneously. There was no literature report on this case.

While, on the other hand, a giant macular hole is difficult to close even after surgery. Therefore,

some doctors choose not to operate [16]. If there is no retinal detachment, observation may be a good option for a giant macular hole. However, some giant macular hole may develop to retinal detachment [17]. Therefore, the patients should be followed up and informed of the risk of retinal detachment. And in case retinal detachment occurs, vitreoretinal surgery is recommended to prevent the development of phthisis bulbi.

2. Drugs

Inflammation and macular edema play a role in the pathogenesis of macular hole formation. Therefore, modalities to reduce inflammation and eliminate edema may promote the closure of the hole. There are some case reports that macular hole closed after topical corticosteroid [22], non-steroidal anti-inflammatory drugs [23], and carbonic anhydrase inhibitor [24], peribulbar steroid injection [25] treatment. However, these cases are small. Giant macular holes are unlikely to close with these drugs. There was no report of giant macular hole treated with medicine.

3. Surgery

Since giant traumatic macular hole cannot close spontaneously or by medicine, vitreoretinal surgery is the only way to close the hole. Traditionally, the macular hole surgery approach includes posterior vitreous vitrectomy, ILM peeling, gas tamponade, and face-down position. However, the anatomical and functional outcome of this technique for large macular hole is limited. Additional approaches have been applied to close large or refractory macular holes. Since a giant macular hole is rare, it is difficult to recruit enough cases for clinical trials. Therefore, the evidence from large or refractory macular hole was adopted.

Michalewska et al. [26] compared inverted ILM flap with standard 3-port pars plana vitrectomy and found that inverted ILM flap technique improved the closure rate (98% vs 86%) and postoperative visual acuity (Mean 0.17 vs 0.28) in macular holes with a diameter greater than 400 μm. They hypothesized that the inverted ILM flap technique stimulates the proliferation of glial cells that fill macular holes, thereby enhancing closure and improving postoperative visual acuity. This is a very useful technique and can be used in large or giant macular hole cases. It was reported that a macular hole with 2845 μm in diameter was closed by the inverted ILM flap technique [27].

Inverted ILM flap may be difficult in the cases with macular neurosensory retinal atrophy or previous macular surgery whose foveal ILM was already removed. ILM transplantation can be used in these situations. Morizane et al. [28] reported that autologous transplantation of ILM may contribute to improved anatomic and visual outcomes in the treatment of refractory macular holes caused by idiopathic or traumatic or other etiologies.

Autologous platelet implantation is another strategy to promote the closure of macular hole. It was reported that 62 macular holes operated with autologous platelet-rich plasma closed compared to 11 in 152 macular hole remains open after the conventional technique [29]. There was a report of chronic and giant traumatic macular hole with a base diameter of 3000 μm closed with autologous platelet implantation [9]. Another literature reported a giant traumatic macular hole with retinal detachment closed with a combination of inverted ILM flap, autologous platelet implantation, and silicone oil tamponade [17].

Besides, the human amniotic membrane plug was also used to treat failed macular holes resulting in closure and visual improvement in 36/36 (100%) patients [30]. But there was no report of an amniotic membrane plug for giant macular hole.

Recently, autologous retinal transplant was used to treat macular holes and results in 89.2% closure [30]. It was even reported that the autologous retinal transplant vascularization and reperfusion were observed using optical coherence tomography angiography for giant macular hole [31]. There was a case report using autologous retinal graft to close a giant macular hole [15].

10.6 Case

A 34-year-old female presented to our hospital emergency department with sudden decreased vision and pain after hit by a fist in her left eye 2 h ago. On examination, her left eye best-corrected visual acuity was HM, and her right eye was 0.5 after corrected by −22.0 DS/−2.5 DC × 180. Anterior segment and intraocular pressure were normal in both eyes. Fundus examination revealed mild vitreous hemorrhage and a 1836 μm diameter full-thickness macular hole with surround retinal detachment confirmed by spectral-domain optical coherence tomography in the left eye (Fig. 10.1). Fundus examination was normal in the right eye except for extensive tessellated changes.

Surgical repair was performed with 23-gauge pars plana vitrectomy, posterior hyaloid detachment. Liquid perfluorocarbon (PFCL) was injected over the macular area, and peripheral vitreous was removed. The flap of the inner limiting membrane was inverted and covered the macular hole, air-fluid exchange was applied to remove the PFCL, and silicone oil tamponade.

Postoperative topical use of 0.5% ofloxacin eye drops to prevent infection and anti-inflammatory treatment with 1% prednisone acetate eye drops four times a day for 2 weeks. Prone poisoning was strictly maintained at the first week after surgery. One week after the surgery,

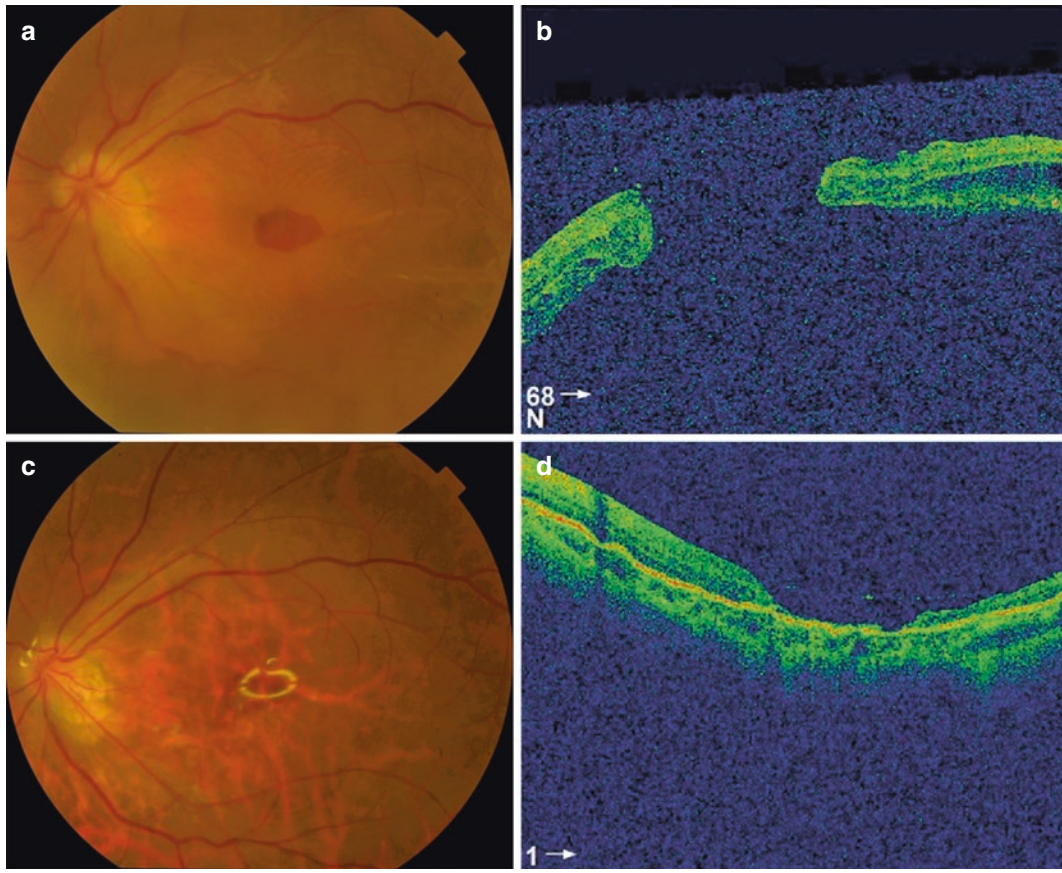

Fig. 10.1 High myopia patient with traumatic macular hole. (**a**) Color fundus photograph shows a giant full thickness and irregular circle macular hole combined with posterior retinal detachment; (**b**) Optical coherence tomography confirmed the giant macular hole; (**c**) Color fundus photograph shows the macular hole closed and retinal reattached tamponade with silicone oil 1 week after the vitreoretinal surgery. (**d**) Corresponding optical coherence tomography shows the retinal reattached and the macular hole closed

the macular hole had closed with the retina reattached under silicone oil. After 10 months, her best-corrected visual acuity was improved to 0.16 and the macular hole remained closed.

10.7 Personal Experience

The counseling before the operation should be detailed. The doctors should provide the following information to the patient: (1) The incidence of giant traumatic macular hole is low. And therefore, there is no strong evidence in the literature on any treatment of natural history; (2) Although about 1/3 of traumatic macular hole may close spontaneously, giant traumatic macular hole is unlikely to heal itself; (3) Giant macular hole may progress to retinal detachment; (4) Drug is unlikely effective on giant macular hole; (5) Operation may close the macular hole and improve visual acuity, but the larger the macular hole is, the lower possibility of macular hole closure after operation; (6) The patient needs to keep a prone position for some time after operation; (7) There may be intraoperative and postoperative complications. Then, discuss whether he/she receive a vitreoretinal operation to close the hole with the patient. If the patient decides not to receive an operation, he/she should be followed up, and when there is retinal detachment, operation is indicated.

Several techniques may promote the closure of the macular hole. Peeling of the inner limiting membrane can release the centrifugate tangential traction force. It may be difficult peeling of ILM in traumatic giant macular hole due to the ILM adhesion to neurosensory retina tightly after injury, especially the cases with neuroretina atrophy. Staining with indocyanine green (ICG) or brilliant blue G can facilitate ILM peeling. However, caution must be taken to avoid the toxicity of the dye, especially ICG. Use of perfluorocarbon liquid or viscoelastic may prevent the contact of dye with retinal pigment epithelium. Besides ILMP, several techniques can be used to provide a bridge for the proliferation of retinal glia to heal the macular hole, including inverted ILM flap, free ILM flap, posterior capsule, and amniotic membrane patch. In our opinion, the inverted ILM flap is recommended because it is relatively easier and unlikely to dislocate during the following procedures and after the operation. Other techniques can be used if the ILM around the macular hole has been removed or very difficult to peel. Retinal transplant is a recently introduced technique and has shown promising efficiency.

The tamponade at the end of surgery would be performed using C3F8 or air or silicone oil. For traumatic giant macular holes, we prefer silicone oil tamponade. Silicone oil can last a longer time, sufficient for the long time duration of giant macular hole healing. The patients are instructed to keep a prone position after the operation. The duration of the prone position is controversial in the literature. We suggest monitoring the morphology of the macular hole after operation using OCT. When OCT found that the macular hole has closed, the patient does not need to keep the prone position.

10.8 Specific Challenges

Most macular holes can be closed with the advantage of surgical techniques, even some giant macular holes. However, the visual outcome is still a challenging problem in giant macular hole. Some patients may get functional improvement, while some may still suffer a central scotoma. There are some possible explanations. First, the retinal tissue may be insufficient to heal the giant macular hole, especially the photoreceptor. The hole is healed mostly by the proliferation of retinal glia. Second, the free ILM flap inside the macular hole may prevent the complete recovery of the photoreceptor. Third, the dye used for ILM staining may cause toxicity of retinal pigment epithelium and photoreceptor. Forth, ILM peeling may damage the retinal nerve fiber layer.

Retinal transplant may provide sufficient retinal tissue to the macular hole. And a case report showed that there was vascular reperfusion of the graft. However, we still do not know how the graft's neuron reconnect with the surrounding retinal tissue. And therefore, the functional recovery may still be limited.

References

1. Johnson R, McDonald H, Lewis H, Grand M, Murray T, Mieler W, Johnson M, Boldt H, Olsen K, Tornambe P, et al. Traumatic macular hole: observations, pathogenesis, and results of vitrectomy surgery. Ophthalmology. 2001;108(5):853–7.
2. Sou R, Kusaka S, Ohji M, Gomi F, Ikuno Y, Tano Y. Optical coherence tomographic evaluation of a surgically treated traumatic macular hole secondary to Nd:YAG laser injury. Am J Ophthalmol. 2003;135(4):537–9.
3. Lin L, Liang C, Chiang S, Yang H, Chang C. Traumatic macular hole secondary to a Q-switch Alexandrite laser. Retina (Philadelphia, PA). 2005;25(5):662–5.
4. Huang J, Liu X, Wu Z, Lin X, Li M, Dustin L, Sadda S. Classification of full-thickness traumatic macular holes by optical coherence tomography. Retina (Philadelphia, PA). 2009;29(3):340–8.
5. Oehrens A, Stalmans P. Optical coherence tomographic documentation of the formation of a traumatic macular hole. Am J Ophthalmol. 2006;142(5):866–9.
6. Miller J, Yonekawa Y, Eliott D, Kim I, Kim L, Loewenstein J, Sobrin L, Young L, Mukai S, Vavvas D. Long-term Follow-up and Outcomes in Traumatic Macular Holes. Am J Ophthalmol. 2015;160(6):1255–1258.e1251.
7. Boral SK, Agarwal D, Das A, Chakraborty D, Sinha TK. A novel video overlay guided enlargement of area of ILM peeled versus inverted flap technique: a long-term study in large macular holes. Eur J Ophthalmol. 2020:1120672120979904.
8. Mahalingam P, Sambhav K. Surgical outcomes of inverted internal limiting membrane flap technique for large macular hole. Indian J Ophthalmol. 2013;61(10):601–3.
9. Coca M, Makkouk F, Picciani R, Godley B, Elkeeb A. Chronic traumatic giant macular hole repair with autologous platelets. Cureus. 2017;9(1):e955.
10. Lyu WJ, Ji LB, Xiao Y, Fan YB, Cai XH. Treatment of refractory giant macular hole by vitrectomy with internal limiting membrane transplantation and autologous blood. Int J Ophthalmol. 2018;11(5):818–22.
11. Raimundo M, Fonseca C, Silva R, Figueira J. Bilateral giant macular holes: a rare manifestation of Alport syndrome. Eur J Ophthalmol. 2019;29(1):NP13–6.
12. Hernandez-Da Mota SE. Posttraumatic giant macular hole. Case Rep Ophthalmol. 2011;2(2):283–6.
13. Vukkadala T, Mondal S, Azad SV, Kumar V. Giant macular hole in a case of multifocal choroiditis. Indian J Ophthalmol. 2020;68(9):1941–2.
14. Nagaoka K, Inoda S, Takahashi H, Arai Y, Inoue Y, Takizawa Y, Fujioka S, Kawashima H. A case of giant macular hole progression after rupture of a giant retinal pigment epithelial detachment. Case Rep Ophthalmol. 2019;10(2):195–9.
15. Ozkan B, Karabas VL. Surgical closure of giant traumatic macular hole with retinal graft. Eur J Ophthalmol. 2019;29(5):NP14–7.
16. Nelson DB, Grantham RL, Marcus DM. Traumatic giant macular hole. Retina. 2001;21(6):677–8.
17. Ch'ng SW, Elaraoud I, Karl D, Kalogeropoulos D, Lee R, Carreras E. A combination of surgical techniques to repair a giant traumatic macular hole. Case Rep Ophthalmol Med. 2018;2018:7595873.
18. Chen H, Jin Y, Shen L, Wang Y, Li Z, Fang X, Wang Z, Huang X, Wang Z, Ma Z. Traumatic macular hole study: a multicenter comparative study between immediate vitrectomy and six-month observation for spontaneous closure. Ann Transl Med. 2019;7(23):726.
19. Chen H, Chen W, Zheng K, Peng K, Xia H, Zhu L. Prediction of spontaneous closure of traumatic macular hole with spectral domain optical coherence tomography. Sci Rep. 2015;5:12343.
20. Yamashita T, Uemara A, Uchino E, Doi N, Ohba N. Spontaneous closure of traumatic macular hole. Am J Ophthalmol. 2002;133(2):230–5.
21. Liu J, Peng J, Zhang Q, Ma M, Zhang H, Zhao P. Etiologies, characteristics, and management of pediatric macular hole. Am J Ophthalmol. 2020;210:174–83.
22. Khurana RN, Wieland MR. Topical steroids for recurrent macular hole after pars plana vitrectomy. Ophthalmol Retina. 2018;2(6):636–7.
23. Kurz PA, Kurz DE. Macular hole closure and visual improvement with topical non-steroidal treatment. Archiv Ophthalmol (Chicago, IL: 1960). 2009;127(12):1687–8.
24. Marques RE, Sousa DC. Macular hole closure with topical carbonic anhydrase inhibitor. Ophthalmol Retina. 2019;3(4):304.
25. Halkiadakis I, Pantelia E, Giannakopoulos N, Koutsandrea C, Markomichelakis NN. Macular hole closure after peribulbar steroid injection. Am J Ophthalmol. 2003;136(6):1165–7.
26. Michalewska Z, Michalewski J, Adelman R, Nawrocki J. Inverted internal limiting membrane flap technique for large macular holes. Ophthalmology. 2010;117(10):2018–25.
27. Deshpande R, Narayanan R. Surgical repair of a giant idiopathic macular hole by inverted internal limiting membrane flap. BMJ Case Rep. 2015.
28. Morizane Y, Shiraga F, Kimura S, Hosokawa M, Shiode Y, Kawata T, Hosogi M, Shirakata Y, Okanouchi T. Autologous transplantation of the internal limiting membrane for refractory macular holes. Am J Ophthalmol. 2014;157(4):861–869.e861.
29. Shpak AA, Shkvorchenko DO, Krupina EA. Surgical treatment of macular holes with and without the use of autologous platelet-rich plasma. Int Ophthalmol. 2021.
30. Caporossi T, Pacini B, Bacherini D, Barca F, Faraldi F, Rizzo S. Human amniotic membrane plug to promote failed macular hole closure. Sci Rep. 2020;10(1):18264.
31. Tabandeh H. Vascularization and reperfusion of autologous retinal transplant for giant macular holes. JAMA Ophthalmol. 2020;138(3):305–9.

Posterior Polar Ocular Perforating Injury

11

Haixia Guo, Yuanyuan Liu, Wei Zhang, and Hua Yan

Abstract

Posterior pole perforating ocular injury (PPPOI) is a special type of POI in which the exit wound is located at the posterior pole, and the suturing of the small exit wound is virtually impossible due to its location and associated bad outcomes. The most severe complications caused by PPPOI are the direct damages to the macular and optic nerve, or the sequelae of the intraorbital foreign body, that may result in the ultimate irreversible visual impairment or blindness. The important issues in regard to PPPOI management include timing of vitrectomy and the disposal of exit wound and intraorbital foreign body. These issues are discussed in more detail based on our experience in this case-series report. The main controversies of PPPOI treatment appeared to be focused on whether to suture posterior pole exit wounds and the removal of retained intraorbital foreign body. In this chapter, we report a case-series study in order to better illustrate the clinical features, surgical techniques as well as the relative prognostic factors of PPPOI.

Keywords

Posterior pole perforating ocular injury Emergency · Vitrectomy · Foreign body Suturing

11.1 Introduction

Perforating ocular injury (POI) refers to the presence of an entrance and an exit wound, and both wounds were caused by the same object. Although the incidence of POI has been shown to be 0.2–2.9% in open globe injuries, the injury typically resulted in severe ocular complications and poor prognosis [1–3]. One option for the treatment of PPPOI was to place an autologous tenon capsule patch in the posterior exit wound. One common postoperative complication of that treatment was subretinal membrane proliferations which then result in traction with fixed retinal folds around the patch [4]. In an attempt to achieve better treatment outcomes of PPPOI, we explored new ways and surgical techniques to deal with problems associated with the posterior pole exit wound, timing of vitrectomy, and management of the intraorbital foreign body. Most of the previ-

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ous POI studies were case reports with no in-depth analysis of the clinical characteristics, treatment approaches, prognosis, and relative risk factors in PPPOI [5–9]. The main controversies of PPPOI treatment appeared to be focused on whether to suture posterior pole exit wounds and the removal of retained intraorbital foreign body. In this chapter, we report a case-series study in order to better illustrate the clinical features, surgical techniques as well as the relative prognostic factors of PPPOI.

11.2 Definition

Posterior pole perforating ocular injury (PPPOI) is a special type of POI in which the exit wound is located at the posterior pole, and the suturing of the small exit wound is virtually impossible due to its location and associated bad outcomes.

11.3 Case (Brief Case Report Based on Figs. 11.1, 11.2, 11.3, and 11.4)

In this retrospective, noncomparative, and consecutive case series study, 22 patients with PPPOI who underwent combined vitrectomies in Tianjin Medical University General Hospital and Xiamen Eye Center from November 2010 to December 2019 were enrolled. In all 22 patients, 20 (90.9%) were male and 2 (9.1%) were female. The age ranged from 15 to 63 years old with an average of 41.5 ± 12.1 years. Twenty-one patients presented in the emergency within 24 h, and only one patient delayed 5 days after injury due to misdiagnosis in the local hospital. All patients had different levels of proliferative vitreoretinopathy (PVR) after PPPOI which included grade B in 8 eyes and grade C in 14 eyes. The follow-up period ranged from 1 month to 30 months with a mean of 6.7 ± 6.6 months.

The diagnosis and classification were performed according to Birmingham Eye Trauma Terminology System and Ocular Trauma Classification Group [10, 11]. Informed consent forms were obtained from all patients, and the approval of the Ethics Committee of Tianjin Medical University General Hospital and Xiamen Eye Center was carried out.

Clinical data including reasons of PPPOI, visual acuity, intraocular pressure (IOP), location and size of the entrance and exit wounds, concomitant damages of intraocular tissues, treatment, outcomes, and postoperative complications were collected from the Eye Injury Register of Tianjin Medical University General Hospital and Xiamen Eye Center in patients with PPPOI.

11.3.1 Case 1

Patient 1 with long iron nail penetrating the eye after posterior pole perforating ocular injury. (A, B) A 44-year-old man was injured by an iron nail on the right eye with the vision of hand movement, and the long iron nail penetrated the eye from the cornea to orbit confirmed by CT. (C) The slight exudates in the anterior chamber was observed 2 days after emergency surgery for suturing corneal wound. (D) A large amount of vitreous hemorrhage was observed by ocular B-scan ultrasonography. (E) The delayed vitrectomy was performed 10 days after primary surgery, and an exit wound was found proximity to fovea with hemorrhage overlying and sclera exposure during vitrectomy. (F, G) The injured cornea healed well, the scleral scar exposed at the posterior wound, and the optic nerve was atrophy 5 months after vitrectomy (white arrow). The final vision was finger counting at the last follow-up.

11.3.2 Case 2

Patient 2 with final retained intraorbital foreign body after posterior pole perforating ocular injury. (A) A 43-year-old man was injured by an iron dust on left eye during mechanical work with the vision of light perception. Slit-lamp examination revealed a 3 mm full-thickness irregular corneal laceration inferotemporal with iris incarcerated in the corneal wound. (B) The emergency orbital CT demonstrated a metallic foreign body in the orbit of the left eye. (C) The corneal wound healed well with posterior synechia of the iris 10 days after the emergency sur-

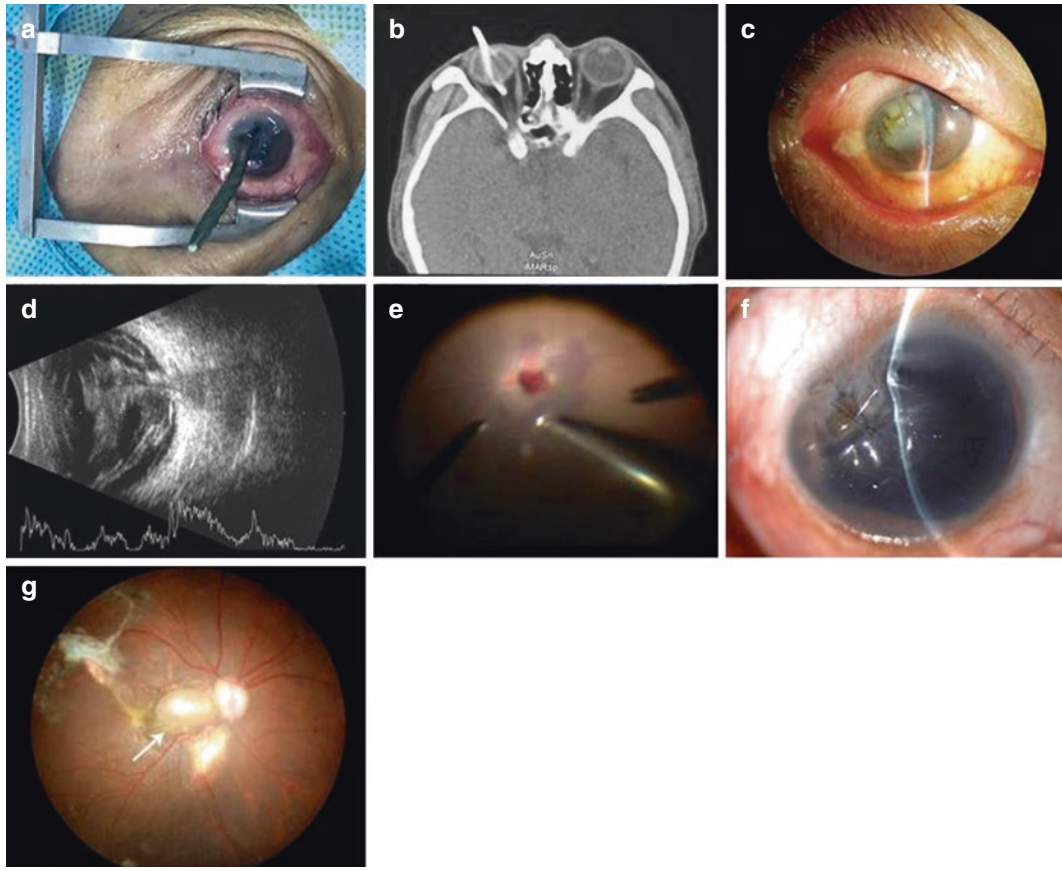

Fig. 11.1 (**a**, **b**) A 44-year-old man was injured by an iron nail on the right eye with the vision of hand movement, and the long iron nail penetrated the eye from cornea to orbit confirmed by CT. (**c**) The slight exudates in the anterior chamber was observed 2 days after emergency surgery for suturing corneal wound. (**d**) A large amount of vitreous hemorrhage was observed by ocular B-scan ultrasonography. (**e**) The delayed vitrectomy was performed 10 days after primary surgery, and an exit wound was found proximity to fovea with hemorrhage overlying and sclera exposure during vitrectomy. (**f**, **g**) The injured cornea healed well, the scleral scar exposed at the posterior wound, and the optic nerve was atrophy 5 months after vitrectomy (white arrow)

gery. (D) The dense vitreous hemorrhage was shown by an ocular B-scan examination. (E) Delayed vitrectomy was performed 11 days after primary surgery, and a self-sealed exit wound was observed inferotemporal to the fovea with hemorrhage overlying and partial sclera exposure during vitrectomy. (F, G, H, I) The injured cornea healed smoothly, the retina attached and the posterior wound healed with the scleral scar exposed (white arrow), the vitreous cavity was clear, and the foreign body lodged in the orbit without infection confirmed by orbital CT 5 months after vitrectomy. The final best-corrected visual acuity (BCVA) was 0.4.

11.3.3 Case 3

Patient 3 with final retained intraorbital foreign body after posterior pole perforating ocular injury. (A) A 23-year-old man was injured by an iron dust on right eye with the vision of no light perception. Slit-lamp examination revealed an 8 mm full-thickness irregular corneal laceration from 3:00 directly extending to 7:00, and was sutured closely in emergency surgery. (B) The emergency orbital CT demonstrated a metallic foreign body in the orbit of the right eye. (C) The dense vitreous hemorrhage was seen by ocular B-scan examination 12 days after repair-

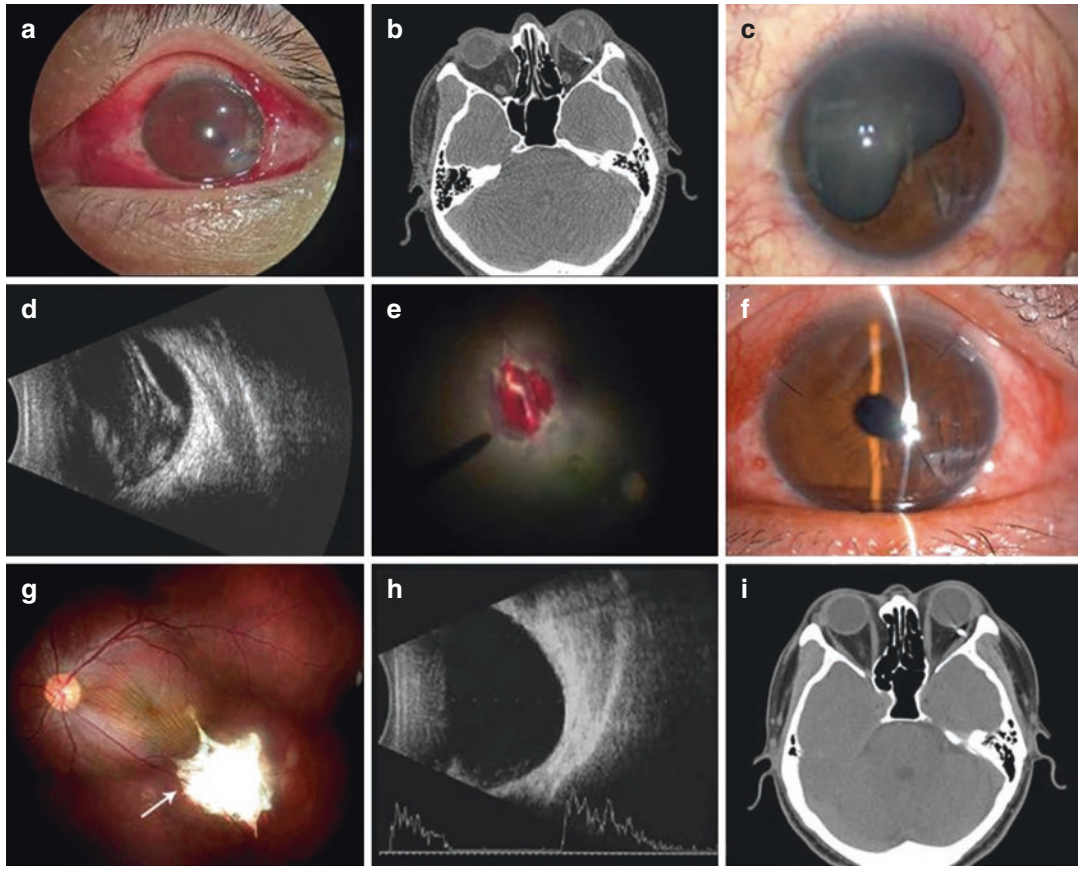

Fig. 11.2 (**a**) A 43-year-old man injured by an iron dust on left eye during mechanical work with the vision of light perception. Slit-lamp examination revealed a 3 mm full-thickness irregular corneal laceration inferotemporal with iris incarcerated in the corneal wound. (**b**) The emergency orbital CT demonstrated a metallic foreign body in the orbit of the left eye. (**c**) The corneal wound healed well with posterior synechia of iris 10 days after the emergency surgery. (**d**) The dense vitreous hemorrhage was shown by ocular B-scan examination. (**e**) Delayed vitrectomy was performed 11 days after primary surgery, and a self-sealed exit wound was observed inferotemporal to the fovea with hemorrhage overlying and partial sclera exposure during vitrectomy. (**f–i**) The injured cornea healed smoothly, the retina attached and the posterior wound healed with the scleral scar exposed (white arrow), the vitreous cavity was clear, and the foreign body lodged in the orbit without infection confirmed by orbital CT 5 months after vitrectomy

ing surgery. (D) Delayed vitrectomy was performed 13 days after primary surgery, and a self-sealed exit wound was observed proximity to fovea with hemorrhage covering and partial sclera exposure during vitrectomy. (E, F, G) The cornea was clear with neovascularization growing into the scar, the retina attached and the scleral scar exposed with the posterior wound healed (white arrow), and the retained orbital foreign body was stable without infection 30 months after vitrectomy. The final BCVA improved to 0.1.

11.3.4 Case 4

Patient 4 with an iron foreign body penetrating the eye after posterior pole perforating ocular injury. (A) A 26-year-old woman was injured by an iron foreign body in her right eye for 8 h on the right eye with the vision of no light perception. And the iron foreign body penetrated the eye from cornea to orbit confirmed by CT. (B) Slit-lamp examination revealed a 6 mm full-thickness irregular corneal laceration from 4:00 directly extending to 6:00, and was

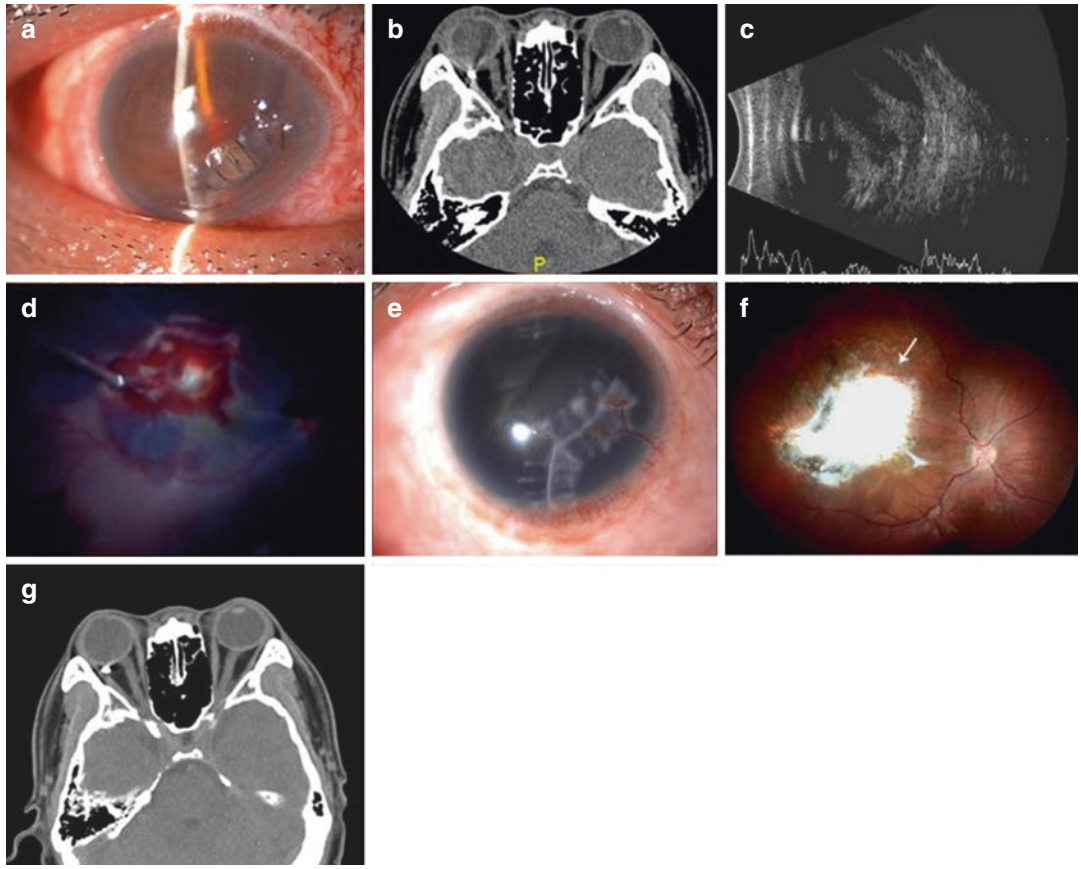

Fig. 11.3 (**a**) A 23-year-old man was injured by an iron dust on right eye with the vision of no light perception. Slit-lamp examination revealed an 8 mm full-thickness irregular corneal laceration from 3:00 directly extending to 7:00, and was sutured closely in emergency surgery. (**b**) The emergency orbital CT demonstrated a metallic foreign body in the orbit of the right eye. (**c**) The dense vitreous hemorrhage was seen by ocular B-scan examination 12 days after repairing surgery. (**d**) Delayed vitrectomy was performed 13 days after primary surgery, and a self-sealed exit wound was observed proximity to fovea with hemorrhage covering and partial sclera exposure during vitrectomy. (**e**–**g**) The cornea was clear with neovascularization growing into the scar, the retina attached and the scleral scar exposed with the posterior wound healed (white arrow), and the retained orbital foreign body was stable without infection 30 months after vitrectomy

sutured closely after emergency surgery. (C) The dense vitreous hemorrhage was shown by ocular fundus examination. (D) The vitreous hemorrhage was cleared 2 weeks after a delayed vitrectomy was observed by ocular B-scan ultrasonography. (E) The foreign body was removal confirmed by CT. The final BCVA improved to 0.4. (F) The fundus image obtained 2 months after removement of the FB. The retina was attached and fibrous membrane tied to the injured retina.

11.4 Important Signs, Examinations, Diagnosis, Surgical Procedures, or Postoperative Treatment for Complications

11.4.1 Pre- and Post-operative Examinations and Diagnosis

The diagnosis and classification were performed according to Birmingham Eye Trauma

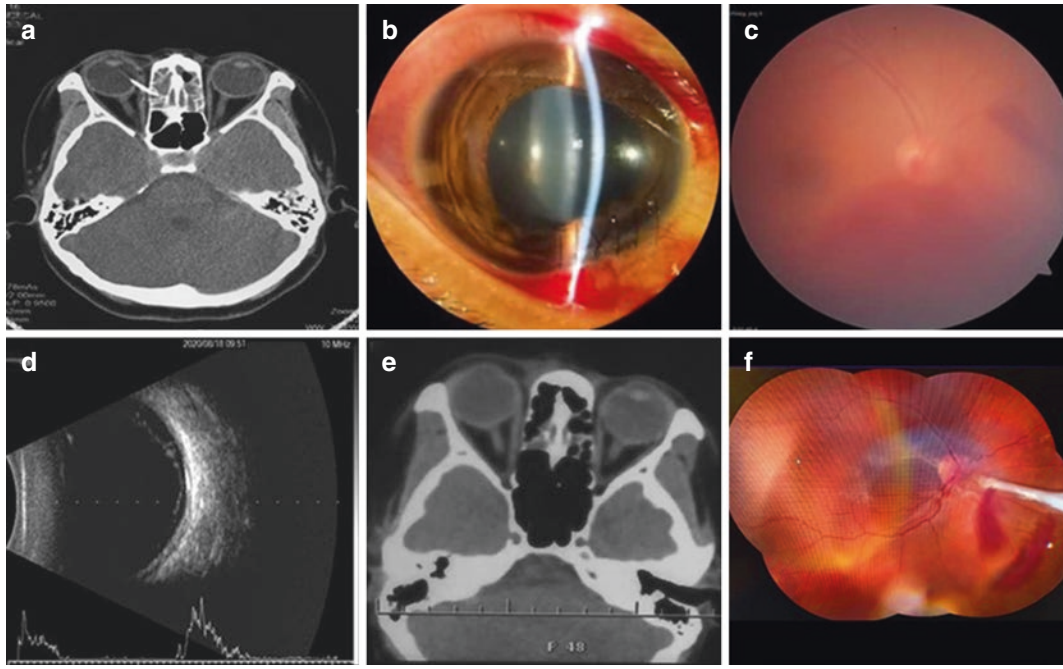

Fig. 11.4 (**a**) A 26-year-old woman was injured by an iron foreign body in her right eye for 8 hours on right eye with the vision of no light perception. And the iron foreign body penetrated the eye from cornea to orbit confirmed by CT. (**b**) Slitlamp examination revealed an 6 mm full-thickness irregular corneal laceration from 4:00 directly extending to 6:00, and was sutured closely after emergency surgery. (**c**) The dense vitreous hemorrhage was shown by ocular fundus examination. (**d**) The vitreous hemorrhage was cleared two weeks after a delayed vitrectomy observed by ocular B-scan ultrasonography. (**e**) The foreign body was removal confirmed by CT

Terminology System and Ocular Trauma Classification Group [10, 11]. The pre- and postoperative examinations mainly included visual acuity, slit-lamp examination, ocular fundus, and IOP. Ocular computerized tomography (CT) scanning was performed in all eyes for determining the foreign body before repairing surgeries in an emergency. Ocular B-scan ultrasonography was carried out to identify the changes of the posterior segment after the entrance wound sutured.

11.4.2 Surgical Procedures

11.4.2.1 Emergency Repairing Surgery

The emergency repairing surgery was performed under local anesthesia by retrobulbar and peribulbar injections of 2% lidocaine (Tianjin Jinyao Pharmaceutical Company, Tianjin, China) in all PPPOI patients. In cases of uveal prolapse, an attempt was made to reposit the uveal tissue after irrigation with balanced salt solution. The globe was then closed and secured with nylon suture. The orbital foreign bodies were removed by an electromagnet in three eyes just after the wound closure, and the foreign bodies across the eyewall were directly removed by forceps in two eyes (Fig. 11.1). Prophylactic intravitreal injection of vancomycin (Eli Lilly Italia S.P.A, Italy) 1 mg/0.1 ml was administered at the end of the repairing surgery to prevent the occurrence of endophthalmitis. Systemic antibiotics was given for 3 days postoperatively, and antibiotic combined with corticosteroid eye drops were used three times a day for 1 week postoperatively. The postoperative ocular examinations were performed routinely every day for 1 week.

11.4.2.2 Timing of Vitrectomy

The average interval time from primary repairing surgery to delayed vitrectomy was 9.8 ± 5.3

days with a range from 1 to 21 days. Only one patient received early vitreoretinal surgery 1 day after repairing surgery for traumatic endophthalmitis, and two eyes for suspicious intraocular foreign bodies.

11.4.2.3 Vitrectomy

All 23-gauge vitrectomies were performed under local anesthesia. Anterior segment reconstruction was performed by removal of papillary membranes, lenticular remnants and condensed anterior vitreous. After the vitreous hemorrhage had been excised, the retinal tissue and posterior wound were exposed, and 0.1 ml triamcinolone acetonide (TA) (40 mg/ml, Kungming Jida pharmaceutical company, Kung Ming, China) was injected into eyes to clearly visualizing the tiny residual vitreous gel and proliferative membranes [12, 13]. The hemorrhage and proliferation on the surface of the posterior wound were dissected carefully and completely by a vitrectomy cutter. In eyes with the retina incarceration or retinal rugosity at the exit wound, prophylactic retinectomy was performed to avoid the postoperative PVR and tractional retinal detachment [14, 15]. Laser photocoagulation was not applied routinely if the retina around the posterior wound self-sealed and attached well; otherwise, it was conducted after air-fluid exchange. In patients with concomitant retinal tears besides the posterior wound, laser was performed simultaneously. Intravitreal reinjection of 0.1 ml TA (40 mg/ml) was performed to prophylactic inhibition of proliferation at the end of the surgery. Silicone oil was instilled in eyes with retinal detachment. The orbital foreign bodies were taken out by electromagnet in four eyes intraoperatively. Patients with silicone oil filling were instructed to keep face-down position for 2 weeks. In all patients, 18 eyes (81.8%) filled with silicone oil, 2 (9.1%) filled with C3F8, 2 (9.1%) with the balanced salt solution according to the retinal status. The antibiotic and corticosteroid eye drops were used three times a day for 1 week postoperatively. Follow-up was performed daily during the first postoperative week, then every week for 1 month, and every 3 months up to 1 year after surgery. Follow-up visits then were performed every 6–12 months. Silicone oil removal was usually performed around 3 months postoperatively, and the intraocular lens (IOL) implantation was considered simultaneously if best-corrected visual acuity (BCVA) was better than 0.1 [16].

For the 19 patients complicated with traumatic cataract, 15 eyes underwent lensectomy, 4 eyes underwent phacoemulsification simultaneously during vitrectomy, and 2 eyes performed secondary IOL implantation after 5–6 months. Laser photocoagulation was applied in 11 eyes around the posterior wound due to the local retinal detachment among which 2 eyes had prophylactic retinectomy.

11.4.3 Characteristics of Wounds in Posterior Pole Perforating Ocular Injury

The entrance wounds were located on the cornea in 10 eyes (45.5%), on the sclera in 5 eyes (22.7%), on the limbus in 2 eyes (9.1%), and across the cornea and sclera in 5 eyes (22.7%). The average length of the entrance wound was 4.7 ± 1.3 mm with a range from 3 to 8 mm. Iris laceration was observed in four eyes (18.2%).

The detailed information of the exit wounds was documented during the vitrectomy. After removing the posterior vitreous cortex, the posterior wounds appeared covered with a large amount of blood and little fibrous membranes. When the hemorrhage and proliferative membrane around the irregular retinal wound were carefully cleared away, the irregular exit wound presented with a scleral scar covered. The unsutured posterior wound sealed very well without any leakages except in two eyes with early vitrectomies (Figs. 11.1, 11.2, and 11.3). The average length of the posterior wound was 2.5 ± 0.7 papilla diameter (PD) with a range of 1–4 PD. The posterior wounds located at the fovea in 4 eyes (18.2%), proximity to the fovea in 8 eyes (36.4%), and others in 10 eyes (45.5%).

The posterior wound leakage was only found in two eyes who underwent early vitrectomy 1 day after primary surgeries for removing the sus-

picious intraocular foreign bodies, and the immediate air-fluid exchange was conducted to prevent further leakage and waited for the delayed vitrectomy.

11.4.4 Visual Acuity

The BCVA ranged from no light perception (NLP) to 0.12 in all eyes after PPPOI. It was 0.12 in one eye, 0.06 in one eye, finger counting (FC) in two eyes, hand movement (HM) in six eyes, light perception (LP) in eight eyes, and NLP in four eyes. Postoperatively, the final BCVA ranged from NLP to 0.6. The vision improved in 14 of 22 eyes (63.6%), remained stable in 5 eyes (22.7%), and decreased in 3 eyes (13.6%). In 19 eyes with traumatic cataracts, only 2 eyes received secondary IOL implantation 5–6 months after vitrectomy, and the BCVA improved from 0.06 to 0.6 and from HM to 0.4 respectively with IOLs well-centered at the last visit.

11.4.5 Management of Intraorbital Foreign Bodies

Of the 18 eyes with intraorbital foreign bodies, the foreign bodies were extracted by an electromagnet in 3 eyes (16.7%) during primary surgery, and in 4 eyes (22.2%) during delayed vitrectomy, and retained in 11 eyes (61.1%) for hard to be removed.

11.4.6 Anatomic Outcomes

The irregular corneal and scleral wounds healed well. All eyes (100%) had a final retinal attachment. Four eyes (18.2%) had recurrent retinal detachments which underwent successful repeat vitrectomies. Scar tissue with the flat edge of the attached retina exhibited around the posterior wound in all eyes (100%) (Figs. 11.1, 11.2, and 11.3). The posterior wounds healed in all cases.

11.4.7 Intraocular Pressure

The mean postoperative IOP was 12.0 ± 3.7 mmHg with a range from 7.1 to 20 mmHg at the final follow-up. There was no hypotony or elevated IOP.

11.4.8 Comparisons of Factors Related to the Final Vision

Fisher's exact test was used to analyze the factors related to a postoperative vision which included age, presenting vision, iris laceration, hyphema, the anterior wound site and size, the posterior wound site, traumatic cataract, PVR, timing of vitrectomy, and situation of foreign bodies. The result illustrated that the posterior wound located in the fovea ($P = 0.0000$) and PVR at Grade C ($P = 0.0001$) were the related factors to final poor vision.

11.4.9 Postoperative Complications

The postoperative complications mainly included secondary glaucoma and optic nerve atrophy in one eye (4.5%), and recurrent retinal detachment in four eyes (18.2%) which were required repeat vitrectomies. There was no incidence of infection in retained intraorbital foreign body cases. No cases of endophthalmitis and sympathetic ophthalmia were noted.

11.5 Personal Experience or Matters Need Attention

The most severe complications caused by PPPOI are the direct damages to the macular and optic nerve, or the sequelae of the intraorbital foreign body, that may result in the ultimate irreversible visual impairment or blindness. The important issues in regard to PPPOI management include timing of vitrectomy and the disposal of exit wound and intraorbital foreign body. These issues are discussed in more detail based on our experience in this case-series report.

11.5.1 Timing of Vitrectomy

The purpose of primary surgery is to restore the structural integrity of the globe as early as possible and to prevent endophthalmitis and further prolapse of intraocular contents in treating POI. We had performed all emergency surgeries within 24 h except one delayed surgery for 5 days due to the misdiagnosis of outside referring hospital. Vitrectomy plays a crucial role in treating POI. Although the optimal timing of vitrectomy remained controversial for the treatment of ocular trauma, most of the surgeons advocated an interval time of 7–14 days to prevent severe intraocular hemorrhage, inflammatory reaction, and overwhelmed PVR formation [17, 18]. Therefore, the factors such as exit wound healing and avoiding PVR formation should be considered when PPPOI was treated with vitrectomy. In our study, the mean interval time between primary surgery and delayed vitrectomy was 9.8 ± 5.3 days. All 23-gauge vitrectomies were performed under local anesthesia, due to the posterior wound healed around 1 week after primary surgery. The prompt vitrectomy has resulted in better clinical outcomes of exit wound healing and less severe PVR formation.

11.5.2 Dealing with the Scleral Wound

Is the suturing of a small exit wound necessary in PPPOI? Usually, the wound of the eyewall should be sutured in time to prevent endophthalmitis or sympathetic ophthalmia in open globe injures [19, 20]. However, suturing the exit wound is a technical challenge in PPPOI due to the unique location at the posterior pole of the eye. The improper management of the exit wound may result in secondary devastating sequels such as traction damage of the optic nerve, more prolapse of intraocular contents, or even rapid collapse of the globe. It is often necessary to temporarily dissect rectus muscles and overstretch the globe to suture the exit wound which will then cause possible intraocular hemorrhage and prolapse of intraocular contents. Since most of the posterior wound is small, it is possible to heal without suturing by pinning of the orbital tissue. Therefore, we recommend that suturing of the small exit wound is not necessary during emergency surgery and vitrectomy. In our study, the average size of the exit wound was 2.5 ± 0.7 PD and was not sutured in all eyes without any evidence of postoperative hypotony.

The posterior wound healed very well without any leakages in non-sutured eyes around 1 week after primary surgery. We observed the exit retinal wound sealed spontaneously with the sclera exposed and retina attached during vitrectomy. The possible mechanism of exit wound healing was the effect of pinning from intraorbital tissue on the sclera side. The blood clot and inflammatory reaction at the posterior wound easily allow for the tissue adhesion of retina, RPE, and sclera by associated factors of platelet-derived growth factor (PDGF), transforming growth factor (TGF), insulin-like growth factor (IGF), vascular endothelial growth factor (VEGF) released by the activated platelet [21].

11.5.3 A Special Paragraph to Discuss the Specific Challenges

Vitrectomy for PPPOI is a great challenge in consideration of the potential leakage from the exit wound, disposal of exit retinal wound and possible intraoperative bleeding, and use of tamponade and laser photocoagulation.

The intraoperative IOP should be controlled normally during and at the end of vitrectomy so as to avoid the wound dehiscence and leakages of intraocular fluid or tamponades from the posterior wound. Yonekawa [22] suggested to keep IOP < 30 mmHg during POI vitrectomy. Kuhn [23] considered only about a half or two-thirds silicone oil tamponade that the eye would usually require is sufficient in order to manage the dehiscence of exit wound temporarily and timely suspending surgery is always needed at this time.

The complete posterior vitreous detachment (PVD) during vitrectomy is indispensable for the cause of the vitreous remnants could potentially

provide a scaffold for fibroblastic proliferation in open globe injuries [24]. However, the vacuum of vitrectomy should be controlled very well to avoid the excessive traction at the retinal wound that makes the healed retina reopened during PVD. Sufficient shaving of the hemorrhage and epiretinal proliferation should be conducted from the peripheral to the center around the exit retinal wound area until the sclera and the edge of retina tear are exposed.

The laser photocoagulation was unnecessary in PPPOI patients with already attached retina around the exit wound during vitrectomy; otherwise, it should be applied. The reason is that the superfluous laser exacerbates intraocular cellular proliferation around the exit retinal wound that could induce possible traction or damage to the retina. In the present study, 11 eyes (50%) appeared local retinal detachment or elevated retinal margins around the exit retinal wounds, and endolaser were applied. However, the posterior wounds showed scar tissue with flat retina edge and without leakage and hypotony in eyes without laser photocoagulation at the final visit. As we know from previous studies, laser photocoagulations at the attached exit retina site are controversial [25–27].

TA was used in all eyes in our study for preventing post-traumatic PVR. Usually, intravitreal TA injection during vitrectomy can assist the completed PVD, hemostasis, and staining the tiny remaining vitreous and proliferative membrane on the retina, which is helpful to facilitate the vitrectomy procedure [13]. With the use of TA during vitrectomy, the successful rate of retina attachment was 81.8% after one vitrectomy procedure in this study.

Traumatic PVR is a crucial cause for visual loss of the severely injured eye and also leads to the anatomical loss of the eyeball. In PPPOI patients with retina incarceration or retinal rugosity at the exit wound, prophylactic chorioretinectomy was performed to prevent the development of PVR that could result in tractional retinal detachment [14, 15]. There were two eyes that received retinectomy at the exit retinal wound in our study, and the outcomes were favorable with the retina attached.

The intraorbital foreign body is not always removed due to its special position in some of the POI patients. Fulcher [28] established a simplified but comprehensive protocol, when complications of strabismus, infection, or fistula formation arise, the surgery should be performed. The severity of intraorbital foreign body complications is highly depending on the location, material, and size. Therefore, long-term follow-up of the complications relative to intraorbital foreign body retaining is needed. Foreign bodies like metal or glass are well tolerated, and it may be left in situ if not causing any abnormal symptoms or signs [29]. However, organic matters like wood and vegetable are poorly tolerated and liable to elicit fungal infection which should be removed as soon as possible [28, 30–34]. In this series of cases, 11 eyes without removal of the intraorbital iron foreign body received close follow-up, and there was no endophthalmitis, orbital abscess, or orbital cellulitis finally.

Investigations on the risk factors associated with poor visual outcomes in PPPOI is of great importance which can predict the prognosis and provide some suggestion on the management of PPPOI. Our results indicated that the risk factor of poor postoperative vision is the site of exit retinal wound and PVR though this is not a study on a large sample. That means the closer the posterior wound is located to the fovea, the worse vision will be. In our study, four eyes (18.2%) had a retinal wound at fovea with the final vision of NLP. So far, few studies focus on the prognosis of PPPOI. Marcus's study concluded that both sites of entrance and exit wounds were the best predictors of functional and anatomic success in their patients [35].

PPPOI usually resulted in the worst prognosis due to damage of the macular and optic nerve [36]. By using our innovative surgical technique such as non-suturing of a small posterior scleral wound and without laser photocoagulation at the attached retinal wound, we achieved an encouraging outcome of 63.6% eyes with improved vision and 36.4% eyes with BCVA 0.1 or better. There was no endophthalmitis, sympathetic ophthalmia, hypotony or phthisis eventually. These outcomes compare favorably to the previous

reports [6, 7, 35]. In this present study, we also performed the treatment of four eyes with NLP, and final vision was 0.1 in one eye, and hand movement in one eye.

To our knowledge, there are few previous studies on detailed analysis of the clinical features, surgical interventions and prognosis of PPPOI in the Chinese population. Although our study is a small sample size case-series report and limited because of the heterogeneity of surgical management options, we draw a limited conclusion that combined vitrectomy is a safe and effective method in PPPOI without suturing the small exit wound. Further studies on a larger scale with longer follow-ups are warranted in order to confirm our findings.

References

1. Beshay N, Keay L, Dunn H, Kamalden TA, Hoskin AK, Watson SL. The epidemiology of Open Globe Injuries presenting to a tertiary referral eye hospital in Australia. Injury. 2017;48(7):1348–54.
2. AlDahash F, Mousa A, Gikandi PW, Abu El-Asrar AM. Pediatric open-globe injury in a university-based tertiary hospital. Eur J Ophthalmol. 2020;30(2):269–74.
3. Batur M, Seven E, Esmer O, Akaltun MN, Yasar T, Cinal A. Epidemiology of adult open globe injury. J Craniofac Surg. 2016;27(7):1636–41.
4. Ma J, Zhang Y, Moe MC, Zhu TP, Yao K. Transocular removal of a retrobulbar foreign body and internal patch of the posterior exit wound with autologous tenon capsule. Arch Ophthalmol. 2012;130(4):493–6.
5. Ding X, Liu Z, Lin Y, Yang Y. Perforating ocular fishhook trauma: a case report. Clin Exp Optom. 2018;101(2):297–8.
6. Cui Y, Li Z, Wang Y, Shi L. Removal of an intraorbital metallic foreign body following double-penetrating ocular injury: a case report. Medicine (Baltimore). 2018;97(51):e13790.
7. Chen KJ, Sun MH, Hou CH, Chen TL. Retained large nail with perforating injury of the eye. Graefes Arch Clin Exp Ophthalmol. 2008;246(2):213–5.
8. Žiak P, Mojžiš P, Halička J, Piñero DP. Bilateral perforating eye injury with metallic foreign bodies caused by tire explosion: case report. Trauma Case Rep. 2017;11:20–2.
9. Chee YE, Kanoff JM, Eliott D. Remarkable visual recovery after severe open globe injury. Am J Ophthalmol Case Rep. 2016;3:34–5.
10. Kuhn F, Morris R, Witherspoon CD. Birmingham Eye Trauma Terminology (BETT): terminology and classification of mechanical eye injuries. Ophthalmol Clin North Am. 2002;15(2):139-v.
11. Pieramici DJ, Sternberg P Jr, Aaberg TM Sr, et al. A system for classifying mechanical injuries of the eye (globe). The Ocular Trauma Classification Group. Am J Ophthalmol. 1997;123(6):820–31.
12. Yan H, Cui J, Zhang J, Chen S, Xu Y. Penetrating keratoplasty combined with vitreoretinal surgery for severe ocular injury with blood-stained cornea and no light perception. Ophthalmologica. 2006;220(3):186–9.
13. Zhou Y, You C, Wang T, et al. Anastalsis of triamcinolone acetonide during vitrectomy in proliferative diabetic retinopathy. Chin J Exp Ophthalmol. 2017;35(5):439–42.
14. Kuhn F, Schrader W. Prophylactic chorioretinectomy for eye injuries with high proliferative-vitreoretinopathy risk. Clin Anat. 2018;31(1):28–38.
15. Ferreira N, Monteiro S, Meireles A, Kuhn F. Outcome of vitrectomy and chorioretinectomy in perforating eye injuries. Ophthalmic Res. 2015;53(4):200–6.
16. He T, You C, Chen S, Meng X, Liu Y, Yan H. Secondary sulcus-fixed foldable IOL implantation with 25-G infusion in patients with previous PPV after open-globe injury. Eur J Ophthalmol. 2017;27(6):786–90.
17. Ung C, Stryjewski TP, Eliott D. Indications, findings, and outcomes of pars plana vitrectomy after open globe injury. Ophthalmol Retina. 2020;4(2):216–23.
18. Yu H, Li J, Yu Y, et al. Optimal timing of vitrectomy for severe mechanical ocular trauma: a retrospective observational study. Sci Rep. 2019;9(1):18016.
19. Zheng L, Tan J, Liu R, et al. The impact of primary treatment on post-traumatic endophthalmitis in children with open globe injuries: a study in China. Int J Environ Res Public Health. 2019;16(16):2956.
20. Chaudhry IA, Shamsi FA, Al-Harthi E, Al-Theeb A, Elzaridi E, Riley FC. Incidence and visual outcome of endophthalmitis associated with intraocular foreign bodies. Graefes Arch Clin Exp Ophthalmol. 2008;246(2):181–6.
21. Opneja A, Kapoor S, Stavrou EX. Contribution of platelets, the coagulation and fibrinolytic systems to cutaneous wound healing. Thromb Res. 2019;179:56–63.
22. Yonekawa Y, Chodosh J, Eliott D. Surgical techniques in the management of perforating injuries of the globe. Int Ophthalmol Clin. 2013;53(4):127–37.
23. Kuhn F. Perforating injuries. Ocular traumatology. New York: Springer; 2008. p. 391–403.
24. Alfaro DV, Tran VT, Runyan T, Chong LP, Ryan SJ, Liggett PE. Vitrectomy for perforating eye injuries from shotgun pellets. Am J Ophthalmol. 1992;114(1):81–5.
25. Iqbal M, Charteris DG, Cooling RJ, Mackintosh GI. Conservative management of double penetrating ocular injuries. Eye (Lond). 2000;14(Pt 2):249–51.
26. Abdullatif AM, Macky TA, Abdullatif MM, et al. Intravitreal decorin preventing proliferative vitreoretinopathy in perforating injuries: a pilot study. Graefes Arch Clin Exp Ophthalmol. 2018;256(12):2473–81.

27. Mohamed AA. Vitrectomy in double-perforation gunshot injury. Clin Ophthalmol. 2013;7:2219–24.
28. Fulcher TP, McNab AA, Sullivan TJ. Clinical features and management of intraorbital foreign bodies. Ophthalmology. 2002;109(3):494–500.
29. Ho VH, Wilson MW, Fleming JC, Haik BG. Retained intraorbital metallic foreign bodies. Ophthalmic Plast Reconstr Surg. 2004;20(3):232–6.
30. Al-Mujaini A, Al-Senawi R, Ganesh A, Al-Zuhaibi S, Al-Dhuhli H. Intraorbital foreign body: clinical presentation, radiological appearance and management. Sultan Qaboos Univ Med J. 2008;8(1):69–74.
31. Green BF, Kraft SP, Carter KD, Buncic JR, Nerad JA, Armstrong D. Intraorbital wood. Detection by magnetic resonance imaging. Ophthalmology. 1990;97(5):608–11.
32. Cartwright MJ, Kurumety UR, Frueh BR. Intraorbital wood foreign body. Ophthalmic Plast Reconstr Surg. 1995;11(1):44–8.
33. Sullivan TJ, Patel BC, Aylward GW, Wright JE. Anaerobic orbital abscess secondary to intraorbital wood. Aust N Z J Ophthalmol. 1993;21(1):49–52.
34. Nasr AM, Haik BG, Fleming JC, Al-Hussain HM, Karcioglu ZA. Penetrating orbital injury with organic foreign bodies. Ophthalmology. 1999;106(3):523–32.
35. Colyer MH, Chun DW, Bower KS, Dick JS, Weichel ED. Perforating globe injuries during operation Iraqi Freedom. Ophthalmology. 2008;115(11):2087–93.
36. Feng K, Shen L, Pang X, et al. Case-control study of risk factors for no light perception after open-globe injury: eye injury vitrectomy study. Retina. 2011;31(10):1988–96.

Giant Intraocular Foreign Body

12

S. Natarajan, Astha Jain, and Sneha Makhija

Abstract

IOFBs are an important cause of blindness and visual morbidity especially in the working age group. Metallic foreign bodies are the most common. Ocular imaging lays an important role while evaluating a case of IOFB. The imaging modalities commonly used are B-scan ultrasonography (USG), X-ray, and computed tomography. Three port pars plana vitrectomy (PPV) with removal of IOFB through limbal or scleral incision is the most common approach. Silicone oil tamponade is used in cases with retinal detachment, proliferative vitreoretinopathy, or endophthalmitis.

Keywords

IOFB · Vitrectomy · PVR

12.1 Introduction

Intraocular foreign bodies (IOFBs) occur in 18–41%of open trauma [1]. IOFBs are an important cause of blindness and visual morbidity especially in the working age group [2]. Due to variations in the clinical presentation, associated complications and outcomes, management of IOFB presents a major challenge to the ophthalmologist. The injury to the eye could be due to mechanical effect, associated infection, or specific reaction [3]. IOFB can be chips of iron or steel, stone, glass, lead pellets, copper, spicules of wood, etc. Of these, metallic IOFBs are most common followed by organic material and nonmetallic material [1]. Visual prognosis is dependent on several factors such as age, length of wound, time between injury and repair, and complications such as relative afferent pupillary defect (RAPD), retinal detachment and endophthalmitis.

12.2 Case Report

A 34-year-old male presented with the history of penetrating ocular trauma with gun pellet in the right eye. He had undergone primary corneal tear repair with iris reposition and anterior chamber wash locally in his right eye. He was currently using topical antibiotics, lubricants, cycloplegics, and systemic antibiotics.

On examination, right eye vision was hand movement close to face and left eye was 6/6 on Snellen's chart. On slit lamp examination, corneal sutures were in place and rest of the cornea appeared clear. Anterior chamber was well

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formed and showed +2 cell reaction, posterior synechiae was noted from 9 to 11 o' clock. A traumatic cataract with pigments on the anterior lens capsule was noted. Posterior segment was not visible in the right eye due to the traumatic cataract (Fig. 12.1). The ocular examination of the other eye was within normal limits.

B-scan ultrasonography of the right eye showed multiple hyperechoic reflections in the vitreous cavity suggestive of vitreous hemorrhage (Fig. 12.2). Computed tomography scan of the orbit revealed a metallic foreign body in the right eye near the optic nerve head (Fig. 12.3). Flash electroretinogram showed right-sided marked diffuse reduction in photoreceptor function and pattern ERG findings were suggestive of reduced function of right macular photoreceptors (Fig. 12.4). The patient was underwent phacoemulsification with posterior chamber intraocular lens implantation with 23 Gauge pars plana vitrectomy with IOFB removal with silicone oil injection with endolaser under local anesthesia. Extremely guarded visual prognosis and need for possible multiple surgeries were explained to the patient. IOFB was removed through a superior pars plane sclerotomy using a basket type IOFB forcep.

On first postoperative day, hand movement vision was present in the right eye. Intraocular lens was seen in situ. Retina was attached with an oil filled vitreous cavity. At 1 month follow-up, the vision in the right eye had improved to finger counting at 1 m, the cornea was clear, intraocular lens was in place, and the retina was attached (Figs. 12.5 and 12.6). No emulsification of silicone oil was noted. The patient was advised close follow-up.

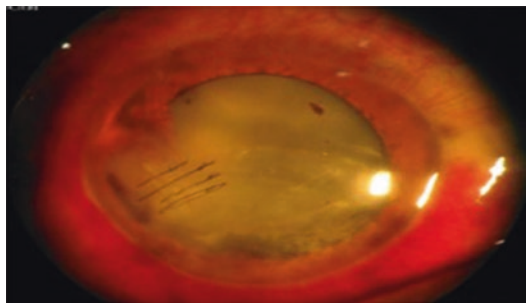


Fig. 12.1 Anterior segment photograph showing sutured corneal entry wound with posterior synechiae and traumatic cataract

Fig. 12.2 B-scan ultrasonography suggestive of vitreous hemorrhage

12.3 Preoperative Evaluation

12.3.1 History

A detailed history is important while evaluating a patient with IOFB. Details about the time from injury to presentation, mechanism of injury which would help in identifying the type of IOFB, presence of visual symptoms, and any prior treatment should be obtained. Assessment of any concomitant, potentially life threatening condition is also crucial. For medicolegal purposes, it is also important to note if the injury

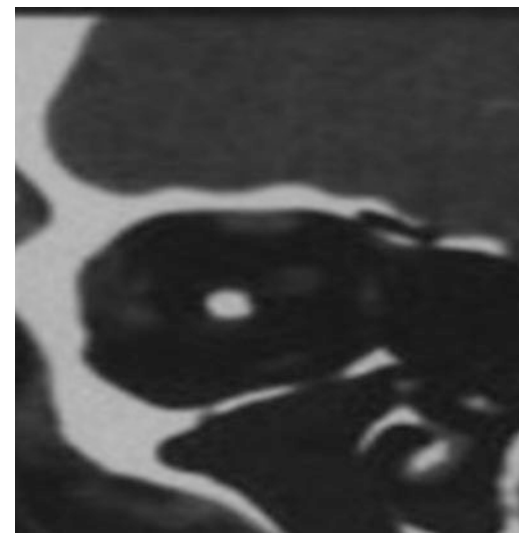
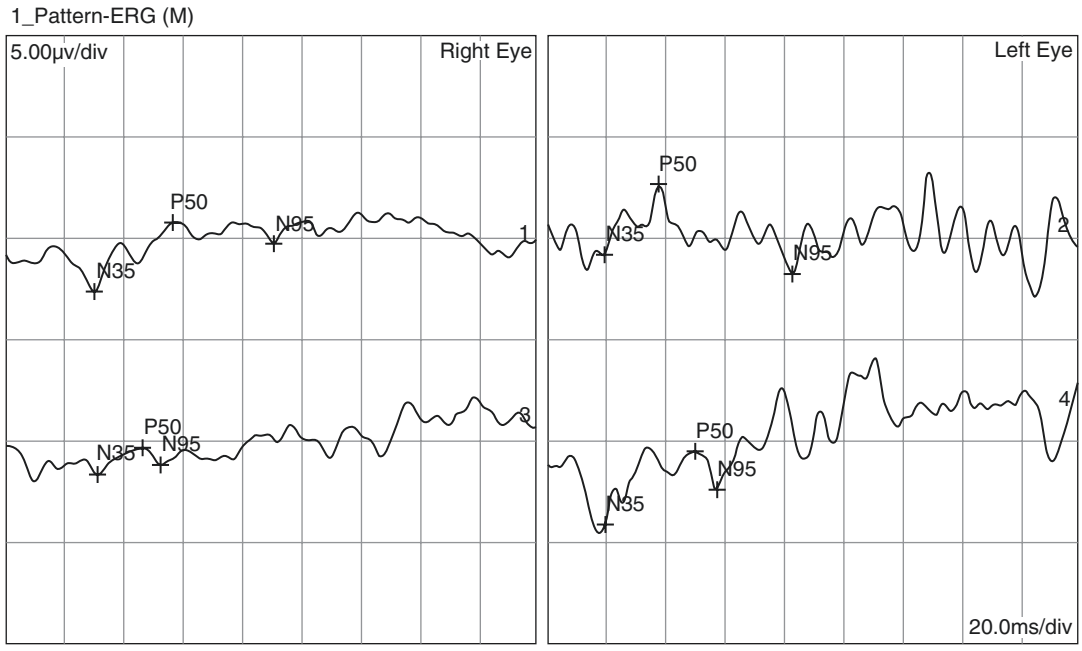


Fig. 12.3 CT scan of right orbit showing metallic intraocular foreign body



Normals	25-45	40-60	85-105	1.00µV-8.00µ	2.00µV-10.00µ
Channel	**N35 [ms]**	**P50 [ms]**	**N95 [ms]**	**N35-P50**	**P50-N95**
1 R-1 48 min	29.9	56.7	90.9	3.42µV	1.06µV (!)
3 R-1 48 min	31.0	46.5	52.5 (!)	1.29µV	805nV (!)
2 L-2 48 min	20.1 (!)	38.4 (!)	83.8 (!)	3.46µV	4.45µV
4 L-2 48 min	20.1 (!)	50.7	58.1 (!)	3.63µV	1.90µV (!)

Fig. 12.4 Pattern ERG showing reduced P50 and N95 responses as compared to the left eye

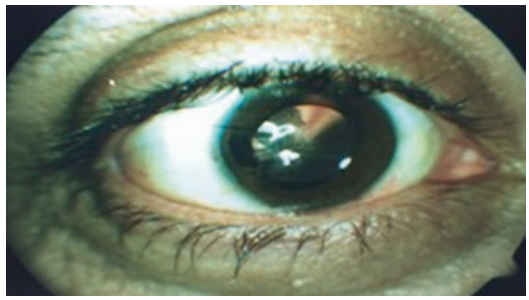


Fig. 12.5 Anterior segment photograph on 1 month post-operative follow-up

occurred in the workplace and if protective eyewear was used at the time of injury.

12.3.2 Examination

After proper history taking, documentation of visual acuity and RAPD should be done. Intraocular pressure measurement is usually deferred in cases of open globe injuries. Examination of the periocular area must be done to look for any laceration, superficial FB, orbital fractures, etc. Slit lamp evaluation of the anterior segment should then be performed. Any laceration of the conjunctiva, cornea, or sclera should be noted. Anterior chamber should be evaluated for presence of hypopyon, hyphema, fibrin, cells, or lens material. Presence of peaked pupil is suggestive of an open globe injury. Lens should be examined for zonular dialysis, cataractous changes, and breach in the lens capsule. Fundus evaluation should be done to look for any pathology such as vitreous hemorrhage, retinal tear, retinal detachment, choroidal detachment, choroidal rupture, or presence of posterior exit wound. IOFB can be located in the vitreous, ciliary body, retina, choroid, or the posterior sclera and hence these areas must be carefully examined. However, scleral depression should be avoided if globe rupture is suspected to avoid

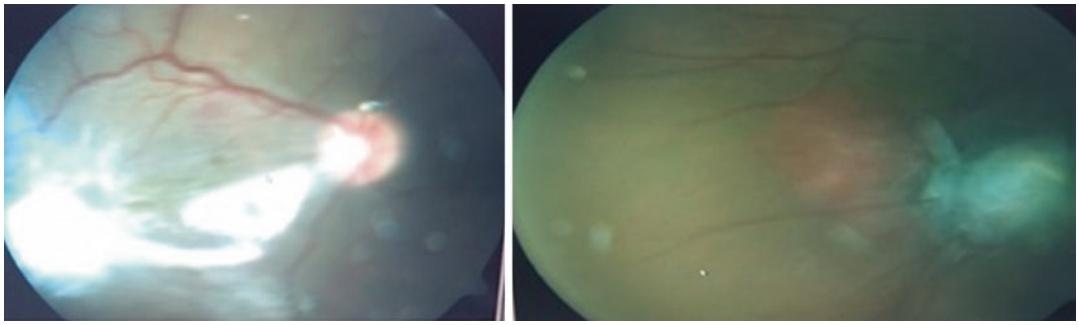


Fig. 12.6 Posterior segment photograph on 1 month postoperative follow-up

expulsion of intraocular contents. Presence of media opacities such as hyphema, cataract, and vitreous hemorrhage can obscure the view of the fundus. Imaging modalities are needed in such cases to detect the presence of IOFB.

12.3.3 Imaging

Ocular imaging lays an important role while evaluating a case of IOFB. The imaging modalities commonly used are B-scan ultrasonography (USG), X-ray, and computed tomography (CT). The modality of imaging used to appropriately visualize the foreign body depends on its composition.

Plain X-ray can be used to identify several IOFB materials such as metal, glass, and slate. However they do not provide the exact localization of the IOFB. Therefore, non-contrast CT with thin cuts (1 mm) and axial, coronal, and sagittal views is the preferred imaging modality. It provides the exact localization of the IOFB and also provides information about other trauma sequale such as orbital fracture, retrobulbar hemorrhage, and any intracranial injury. CT scan can identify most IOFBs well. Wooden and plastic foreign bodies are poorly identified [1]. Wooden IOFBs are hypodense and may be mistaken for air or fat [4]. Magnetic resonance imaging should be avoided in cases of open globe injury due to the concern for a ferromagnetic IOFB.

12.4 Preoperative Care

Appropriate preoperative management is required to prevent spread of infection. Tetanus immunization history should be obtained, and tetanus toxoid or tetanus immune globulin should be administered if necessary. Broad-spectrum systemic and frequent topical antibiotics should be started. Oral levofloxacin is known to have adequate ocular concentration and hence can be used in such cases [5].While awaiting surgery a rigid ocular shield (fox shield) should be placed over the injured eye to prevent further damage. IOFBs should be removed as soon as possible (within 24 h) to reduce the risk of endophthalmitis and PVR. However, studies from Iraq have shown that primary closure of wound with systemic antibiotic and delayed removal of IOFB can result in similar visual outcomes [6].

12.5 Surgical Management

The first step in the management is the closure of the primary wound. Corneal wounds are easily identified and closed with 10-0 nylon suture. Scleral wounds can be concealed by the overlying conjunctiva. Presence of dense subconjunctival hemorrhage may point towards underlying scleral tear. Exploration is needed during surgery to identify the area of scleral tear.

Three port pars plana vitrectomy (PPV) with removal of IOFB through limbal or scleral incision

is the most common approach. 20G or 23G PPV can be performed. However port sites may need to be enlarged in small gauge surgeries as both the foreign body forceps and intraocular magnets require 19G ports. An encircling band may be used to decrease the risk of postoperative PVR [7].

Complete vitreous removal is the first step to prevent any retinal traction during IOFB removal. Triamcinolone assisted PVD induction can be performed. Vitreous around the IOFB and port site vitrectomy should be meticulously performed. Only when all the vitreous is removed, should one attempt to remove the IOFB. Perfluorocarbon liquid can be injected over the macula to prevent any iatrogenic damage.

Cases with coexisting lenticular damage or cases with IOFB lodged in the pars plana may require a pars plana lensectomy. Such cases can have the IOFB removed through a limbal incision. In cases where lens is preserved, IOFB can be removed by enlarging an existing sclerotomy. IOFB removal should be attempted after creating the wound for removal.

Metallic IOFB can be removed by intraocular magnet. Non-magnetic foreign bodies can be removed by forceps. Attempt should be made to grasp and align the IOFB in a way that its thinnest part is removed through the sclerotomy. A thorough peripheral retinal examination is essential after removal of IOFB to look for any tears. Laser should be performed at the site of the IOFB and any other retinal tear. Gas or oil tamponade can be used.

12.6 Postoperative Complications

12.6.1 Endophthalmitis

Vitreous culture should be performed if endophthalmitis is noted before or during surgery. Broad-spectrum intravitreal antibiotics such as vancomycin and ceftazidime should be used. In case of an organic IOFB, antifungals may be added. Silicone oil tamponade is preferred in patients with endophthalmitis. Prophylactic intravitreal antibiotics in the absence of endophthalmitis at the time of surgery may be considered as they have shown to decrease the rate of traumatic endophthalmitis [8].

12.6.2 Retinal Detachment

Retinal detachment is one of the serious complications of IOFB removal surgery. Risk factors for postoperative RD are endophthalmitis and IOFB larger than 4 mm in size [9]. Patients with RD at the time of IOFB removal surgery are also at an increased risk of postoperative RD due to increased chances of iatrogenic retinal break [1]. Silicone oil tamponade is preferred in such cases because of increased risk of PVR.

12.6.3 Proliferative Vitreoretinopathy

Proliferative vitreoretinopathy is one of the most common causes of secondary retinal detachment. Risk factors for PVR include size of IOFBs, size and number of retinal tears, and associated vitreous hemorrhage or choroidal detachment [1].

12.6.4 Sympathetic Ophthalmia

Sympathetic ophthalmia (SO) is a granulomatous panuveitis which occurs days to months after penetrating ocular injury or surgery in one eye. The incidence of SO is 0.2–0.5% after penetrating injury and 0.01% after intraocular surgery [10, 11]. Current treatment with steroids and immunosuppressive agents allow control of disease and retention of good visual acuity in the fellow eye [12]. Due to small risk and effective available treatment for SO, attempt should be made to defer enucleation and plan surgical repair after appropriate patient counseling, even in eyes with no light perception.

12.7 Personal Experience

The timing of surgery is important. Removal of IOFB should be undertaken at the earliest possible. It is also important to decide the site of removal of the IOFB. If lens is being spared, the sclerotomy may need to be enlarged to remove the IOFB. However in case of large and irregular IOFB, lens may need to be sacrificed to remove the IOFB through the limbal incision. Silicone oil is the tamponade of choice in IOFBs associated with retinal detachment.

12.8 Specific Challenges

Removal of large IOFB should be targeted in a way to minimize damage to other intraocular structures. Damage to retina due to slippage of large irregular IOFB can be prevented by use of PFCL. Management of concurrent retinal damage is also challenging. Retinectomy may be required at times due to presence of PVR. Any iatrogenic break should be identified and treated.

References

1. Loporchio D, Mukkamala L, Gorukanti K, Zarbin M, Langer P, Bhagat N. Intraocular foreign bodies: a review. Surv Ophthalmol. 2016;61:582–96.
2. Nicoara SD, Irimescu I, Calinici T, Cristian C. Intraocular foreign bodies extracted by pars plana vitrectomy: clinical characteristics, management, outcomes and prognostic factors. BMC Ophthalmol. 2015;15:151.
3. Pandey AN. Ocular foreign bodies: a review. J Clin Exp Ophthalmol. 2017;8:2.
4. Pinto A, Brunese L, Daniele S, et al. Role of computed tomography in the assessment of intraorbital foreign bodies. Semin Ultrasound CT MR. 2012;33:392–39.
5. Sakamoto H, Sakamoto M, Hata Y, et al. Aqueous and vitreous penetration of levofloxacin after topical and/or oral administration. Eur J Ophthalmol. 2007;17(3):372–6.
6. Colyer MH, Weber ED, Weichel ED, et al. Delayed intraocular foreign body removal without endophthalmitis during Operations Iraqi Freedom and Enduring Freedom. Ophthalmology. 2007;114(8):1439–47.
7. Azad RV, Kumar N, Sharma YR, Vohra R. Role of prophylactic scleral buckling in the management of retained intraocular foreign bodies. Clin Experiment Ophthalmol. 2004;32(1):58–61.
8. Soheilian M, Rafati N, Mohebbi MR, et al. Prophylaxis of acute posttraumatic bacterial endophthalmitis: a multicenter, randomized clinical trial of intraocular antibiotic injection, report 2. Arch Ophthalmol. 2007;125:460–5.
9. El-Asrar AM, Al-Amro SA, Khan NM, Kangave D. Retinal detachment after posterior segment intraocular foreign body injuries. Int Ophthalmol. 1998;22(6):369–75.
10. Makley TA, Azar A. Sympathetic ophthalmia: a long-term follow-up. Arch Ophthalmol. 1978;96:257–62.
11. Marak GE. Recent advances in sympathetic ophthalmia. Surv Ophthalmol. 1979;24:141–56.
12. Arevalo JF, Garcia RA, Al-Dhibi HA, Sanchez JG, Suarez-Tata L. Update on sympathetic ophthalmia. Middle East Afr J Ophthalmol. 2012;19(1):13–21. https://doi.org/10.4103/0974-9233.92111.

Orbital Foreign Bodies

13

Jayanta Kumar Das and Gangadhara Sundar

Abstract

Orbital foreign bodies are uncommon, can be vision threatening, cause serious consequences and often poses a great challenge not only to diagnose but for management as well. Although they may occasionally be missed, they are frequently diagnosed based on clinical suspicion and appropriate imaging. A conceptual approach to clinical examination, appropriate imaging and interpretation, determining the timing and threshold to intervene, and techniques of removal are discussed in the chapter below.

Keywords

Orbit foreign bodies · Orbital trauma Orbitotomy · Orbital foreign bodies · Metallic foreign bodies · Non-metallic foreign bodies Organic foreign bodies

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13.1 Introduction

Any foreign or particulate material trapped within the orbital region is referred to as the orbital foreign body. Orbital injuries in general and foreign bodies in particular are an important cause of ophthalmic morbidity, especially among the young population, the significance of which is sometimes poorly recognised.

The wound of entry can be either through the eyelids, through the globe, or sometimes, in rare cases as in blast injuries, can be through the orbital walls [1]**.** The presence of an orbital foreign body can either be "overt" where the wound of entry is obvious and the history is suggestive, or "latent," especially with self-sealing wounds and unwitnessed injuries. Foreign bodies may either be completely embedded within the orbit, partially extruding or less commonly penetrating into the adjacent intracranial cavity or paranasal sinuses, labeled as transorbital foreign bodies.

The vast majority of affected patients are males owing to their occupation, mode of transportation, and high-risk behavior—assaults, alcohol consumption, etc. High-velocity injuries are generally devastating visually, especially in the presence of open globe injuries, traumatic optic neuropathy or associated intracranial injuries at presentation.

It should be remembered that patients with globe ruptures, severe relative afferent pupillary

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defect (RAPD), poor or no light perception at presentation, delayed presentations with infections, etc., may signify a poorer final visual prognosis and should be attended to immediately.

13.2 Classification

Orbital Foreign bodies can be classified according to the material, location, nature and severity of impact [2, 3].

Material: Based on type, it can be broadly divided into Metallic and Non-Metallic.

1. Metallic foreign bodies may be ferromagnetic (iron, steel, etc.) or non-ferromagnetic (copper, lead, etc.). They may arise from industrial injuries, striking hammer on chisel, assaults or pellets and splinters related to grenade or bomb blasts.

2. Non-metallic foreign bodies may be further divided into Organic or Inorganic FBs.

(a) Organic FBs may be vegetative matter (plant, wood, etc.) or rarely animal substrate.
(b) Inorganic foreign bodies may be made of plastic (spectacle lenses, toys, etc.) and glass (e.g., windshield, bottles etc.).

Location: Based on the location, they may be classified into Intraorbital and Transorbital foreign bodies.

1. Intraorbital FBs may be either penetrating and perforating.
   (a) Penetrating FBs—some part of the FB will be outside the septum with the other end embedded within the orbit.
   (b) Embedded FBs—the whole of the FB will be inside the orbital boundary. These may be Occult or Latent.
2. Transorbital FBs extended beyond the bony orbit and lodged partially within the cranium or paranasal sinuses. These are commonly associated with bony orbital disruption. They can be termed as.
   (a) Transorbital cranial foreign bodies [4]
   (b) Transorbital sinus foreign bodies

Nature and Severity of Impact: According to the nature of the impact, clinical consequences and ease of access, orbital foreign bodies can be divided into simple and complex.

Isolated, well-delineated, and anteriorly located FBs, which may be directly and easily accessed and extracted with minimal consequences, may be considered Simple foreign bodies (Fig. 13.1). Multiple, contaminated and organic foreign bodies that may be missed, easily fragmented with a high risk of residual material, especially if lodged deeply within the orbit against vital structures in the orbit, and the brain may be considered Complex foreign bodies (Fig. 13.2).

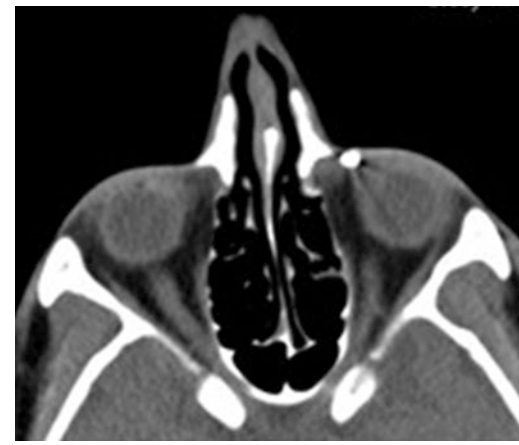

Fig. 13.1 CT scan axial view showing radio-opaque metallic foreign body in the left anterior and inferomedial orbit

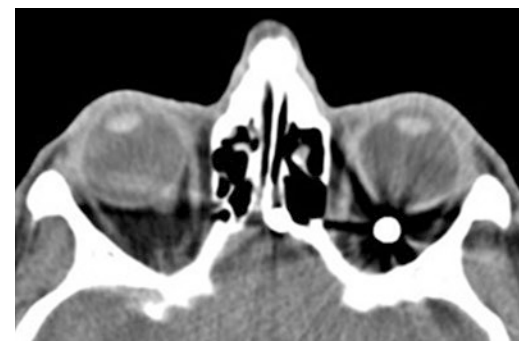

Fig. 13.2 CT scan axial view showing pellet foreign body lodged in the retrobulbar space abutting the left optic nerve

High-velocity and blast injuries are most destructive as apart from orbital involvement, the patient might suffer facial injuries including fractures, extensive soft tissue damage, cervical spine with airway injuries or intracranial injuries.

13.3 Etiopathogenesis

Sources of injury predisposing to orbital foreign bodies include road traffic accidents, industrial accidents, assaults, blast injuries, pellet injuries from accidental or intentional release from weapons such as rifles and guns, which may be encountered in regions of social unrest or border wars. In urban environments and high-income countries, most injuries are industrial, road traffic accidents or assault-related. Affected patients often have a definite history of trauma, and studies have shown that more than 75% of the patients are young working males [4–7]. Domestic accidents and school injuries are often associated with plastic or wooden orbital foreign bodies, where the history may be unreliable. In rural areas, especially in low-income countries, organic orbital FBs are common as manual agriculture is prevalent.

In law and order disturbed regions and in regions with border conflicts or warzones, FBs encountered maybe pellets (lead, rubber bullets, etc.) or related to a grenade or bomb blasts. Penetrating orbital injuries with retained FB is more common in people involved in military services. In such situations, multiple foreign bodies should be suspected as they are commonly encountered.

13.4 Clinical Presentation

Most patients present with a history of injury to the face and periorbital region. Findings at clinical presentation depend on the mode of injury, size and velocity of impact and the type of foreign body. A detailed history of the nature of the injury, the geographical location and circumstances, the severity of the injury, including objects involved at the site of impact, both from the patients and witnesses, should be obtained and documented [5].

Patients may present with variable pain, swelling, visual loss, double vision or in late presentations, even severe infection. Not infrequently in rural accidents and following severe trauma, organic orbital foreign bodies may be discovered late in the convalescence period, especially when the patient is unconscious at presentation or in unwitnessed injuries. In situations where the patient is unable to recall the nature of the injury, an orbital injury with the retained foreign body often presents a confusing and critical clinical picture, especially when the initial entry wound was small and self-sealing or when a foreign body had been partially removed earlier [6, 7].

Depending on the duration and nature of the injury, clinical signs include subconjunctival hemorrhage, chemosis, orbital hematoma, ocular dysmotility, proptosis, and varying visual acuity.

High-velocity injuries are most destructive, as in such cases, apart from orbital involvement, the patient might suffer facial injuries including facial fractures, extensive soft tissue damage, cervical injuries with airway obstruction, or even intracranial injuries (Fig. 13.3). In unconscious and critically ill patients, the diagnosis and management maybe even more challenging, as the orbits and the facial injuries may get attention only after life-saving interventions.

Rarely atypical presentations of orbital or intracranial foreign bodies include secondary consequences presenting as orbital cellulitis or even an orbital abscess. Occasionally, a traumatic Carotico-cavernous fistula with an acute presentation of proptosis, erythema, etc., may be mistaken for underlying orbital cellulitis and mismanaged, as shown below (Fig. 13.4).

13.5 Evaluation

Assessment of life-threatening situations, associated polytrauma, head, neck, and intracranial injuries should be performed first. A suspected open globe should be shielded (not patched) from further injury. Patients should be given adequate analgesia or even sedation as indicated to facili-

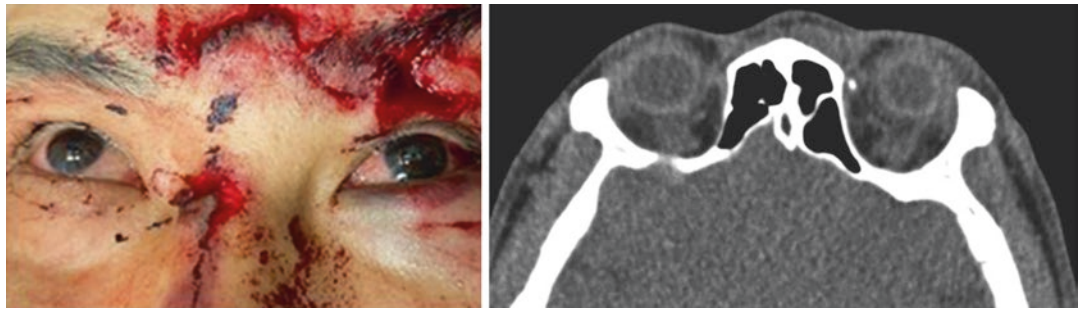

Fig. 13.3 Young patient following motorcycle accident with multiple periorbital lacerations and embedded orbital foreign body seen on axial CT scan

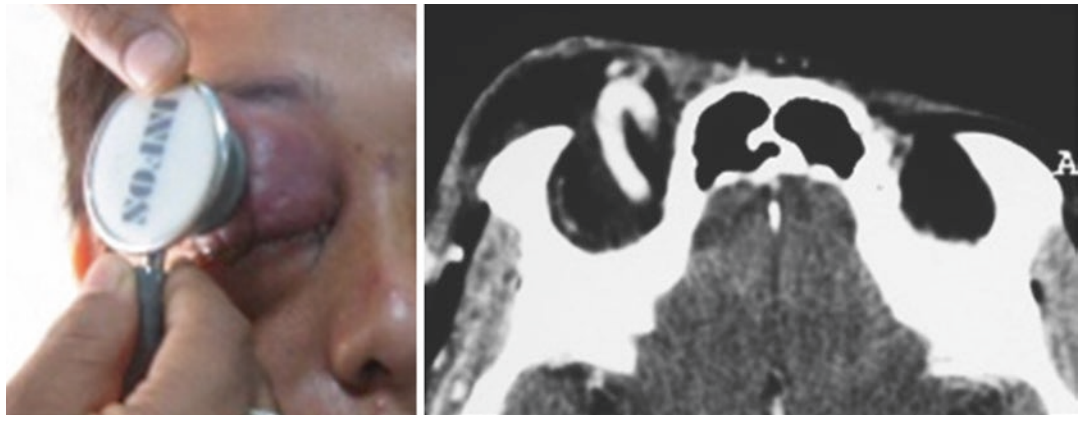

Fig. 13.4 A high flow carotid-cavernous fistula post-trauma managed as orbital cellulitis following orbital trauma. CT scan showing dilated right superior ophthalmic vein

tate a better clinical examination and prevent further injury from a traumatic examination. Evaluation of the orbital injury includes obtaining a detailed history, performing a meticulous examination with a high suspicion for foreign body presence complemented by appropriate imaging studies. History should also include the time, location, nature, and circumstances, emergency interventions, if any, and interval changes from initial injury to the time of presentation.

As part of a general head and neck examination, a comparison to the unaffected contralateral face and orbit should be made. A quick and focused ophthalmic examination including crude visual acuity, inspection for intactness of the globe and pupillary evaluation should be performed atraumatically. Periorbital soft tissue assessment for eyelid and canalicular lacerations, subcutaneous emphysema, foreign bodies, orbital rim step off, or tenderness and periorbital hyper or hypoesthesia should be looked for. A palpable thrill, if present, is suspicious for high flow carotid-cavernous fistula. Gentle finger insinuation may also be considered once globe injury and orbital compartment syndrome has been ruled out, to feel for embedded foreign bodies.

The rest of the ophthalmic examination should include assessment of globe movement limitation, gaze-evoked amaurosis [8], anterior segment and dilated posterior segment examination. In patients with visual loss and intact globe, a detailed posterior segment examination, including for the presence of vitreous hemorrhage, retinal detachment, choroidal rupture or signs of optic neuropathy, should be looked for and documented. Where globe injuries are present, appropriate documentation using the BETT terminology system with the calculation

of the Ocular Trauma Score (OTS) [9, 10] should be performed. It should be remembered that an essential part of the initial assessment is maintaining an accurate electronic medical record with good photographic documentation as well.

13.6 Investigations

13.6.1 Imaging

Radiological investigations play a very important and often decisive role to avoid misdiagnosis of a retained intraorbital or intracranial FB [11], particularly if the history is unreliable or injury is unwitnessed [12]. This is true, especially in children and patients who are unconscious or intoxicated at presentation.

In general, plain X-ray and ultrasonography have a limited role in the diagnosis and management of orbital FB owing to low sensitivity and limited localisation. Plain X-rays may, however, serve as a screening tool in the emergency room not only to recognize gross facial skeletal disruption but also to identify potential radiopaque foreign bodies prior to further Magnetic Resonance neuroimaging MRI (Fig. 13.5).

Non-contrast CT scan remains the imaging modality of choice for studying the bony skeleton and the orbital, facial and intracranial soft tissues in trauma [13]. In addition to detectingandcharacterizing orbital and orbitofacial fractures, it helps detect the presence of foreign bodies and also aids in characterizing and localizing them, including extension into the paranasal or intracranial spaces [7, 14], and thus guide management. Ideally, CT imaging should be performed with the Imaging Guidance Study (IGS) protocol with sub-mm fine cuts, which can be viewed in axial, coronal, and sagittal planes (Fig. 13.6a–c). The radiodensity of foreign bodies often provides reliable clues on the nature of the material by assessment of the Hounsfield Units (HU), shown in Table 13.1. Cone beam CT scans (CBCT) may also be considered in special situations, partly to diagnose, monitor, and assess for residual radiopaque foreign bodies, which may also minimize cost and radiation exposure [15].

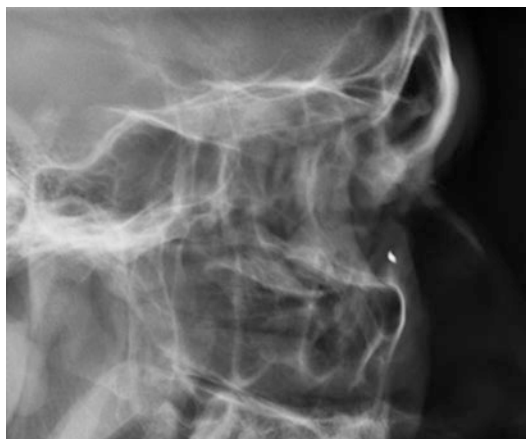

Fig. 13.5 Plain x-ray lateral view demonstrating radiopaque foreign body of inferior orbit

A CT Angiogram of the head and neck region may also be performed as an emergency screening tool in cases of severe head and neck injuries to provide preliminary bony, soft tissue and vascular anatomy of the patient, especially in the unconscious patient suspected to have an intracranial bleed.

Likewise, when diagnosing and planning management of orbital and orbitofacial fractures, the presence of intraocular or intraorbital foreign bodies plays a key role in prioritization, timing, and approach to removal of such foreign bodies. A useful mnemonic "ABCDEFGH" used in the management of such orbitofacial fractures is shown in Table 13.2 where "F" stands for Intraocular or Orbital Foreign Body [16].

Although CT scans provide quick, cost-effective, and very useful information about bones and soft tissues, MRI provides more accurate information about the soft tissues, including the brain, optic nerve-sheath complex and in detecting radiolucent torganic foreign bodies invisible to CT scans (Fig. 13.7a–c) guiding re-exploration and foreign body removal (Fig. 13.7d, e). In addition to the detection of embedded organic foreign bodies [17, 18], it is also useful in detecting plastic and "unleaded" glass foreign bodies. It should be remembered that MRI is contraindicated in acute situations, especially when ferromagnetic metallic FB has not been ruled out along with its

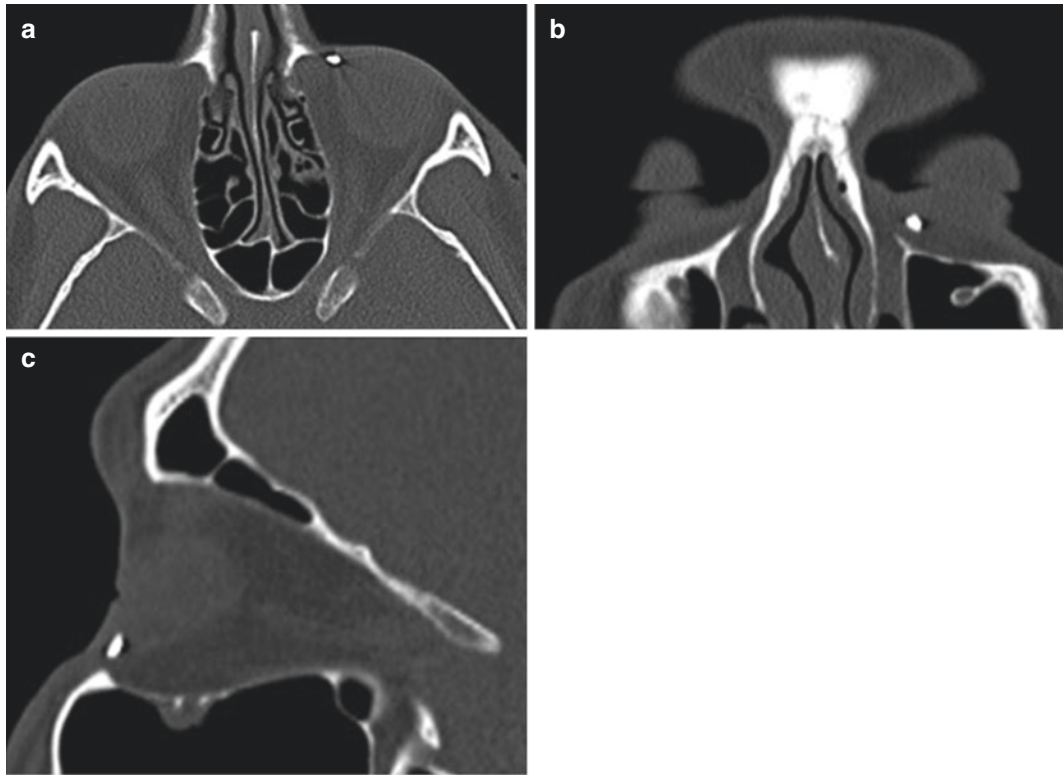

Fig. 13.6 (**a, b, c**) CT scan bone windows: Axial, Coronal, and Sagittal bone windows aiding in three-dimensional localization of left anterior orbital radiopaque foreign body

Table 13.1 Hounsfield Units of various orbital soft tissue structures and foreign bodies

Material	Hounsfield unit (HU)
Metal	4000
Glass	2407
Wood	60
Stone	1876
Acrylic resin	193
Graphite	742
Tooth	1881
Bone	700 to 3000
Muscle	71
Blood	45 to 65
Hematoma	40 to 90
Plastic	112 to 133
Air	−932 to −1000
Water	0

limitation of movement artefacts in an agitated patient.

In summary, imaging not only helps to detect FBs but also aids localization and its proximity to radiologic landmarks and vital structures within the orbit and the brain.

13.6.2 Perimetry

Visual field testing when possible is often useful for baseline documentation, assessment, and severity of intraocular/optic nerve injury. Apart from confrontational visual fields and red-dot perimetry, automated perimetry, where possible, should be performed to assess the severity of optic nerve and /or intracranial injury in alert and cooperative patients.

13.6.3 Electrophysiological Tests

Visual evoke potential (VEP) recordings provide information about the status of the optic nerve. It is very helpful to detect early optic nerve injury

Table 13.2 Patient modifiers regarding the impact of foreign bodies in orbitofacial trauma management

Patient modifiers in orbital and orbitofacial trauma		
Age	Infant, child, young adult, elderly	Urgency, the timing of intervention
Bilateral or Unilateral	Prioritizing unilateral or bilateral repairs	Need for simple techniques vs. advanced technology, e.g., treatment planning, intraoperative navigation, etc.
Complex or Simple orbital fractures	Oculoplastic or multidisciplinary approach	Simple vs. multidisciplinary, multiple incisional approaches, complex implants
Displaced or non-displaced fractures	Threshold for intervention	
Entrapment or not	Orbital soft tissues (EOM-IMS Complex), globe, etc.	Urgency of intervention, larger incisions
Foreign body presence	Intraocular, Intraorbital or both	Antibiotics, localize and plan early removal
Globe and/or optic nerve injuries	Management closed or open globe injury, prevent additional damage	Address after life-threatening injuries before specific orbital interventions
High-risk patient or not	Low-risk patients: young healthy, adults or children without intracranial injuries/ polytrauma High-risk patients: elderly, comorbidities, intracranial injuries/polytrauma	Multispecialty approaches with life preservation and if indicated delayed orbital intervention.

as even in cases of normal visual acuity, the VEP may be altered. Likewise, when the clinical examination is normal, but patient complains of profound visual loss, electroretinography (ERG) along with VEP may be useful to differentiate organic from non-organic causes of visual loss.

13.7 Management

Management of the patient with traumatized orbit and foreign body depends on the severity of the injury, visual morbidity and the general fitness of the patient. It may thus range from conservative management to complex surgery [19]. Initial management of trauma is the stabilization of systemic conditions and proper control of bleeding. Broad-spectrum antibiotics, systemic anti-inflammatory agents and analgesics play a very important role in the management of orbital trauma with a retained foreign body. Corticosteroids may be used judiciously when indicated once infections have been ruled out.

Indications and timing of removal of orbital foreign bodies depend on the nature of the injury, type of foreign body (material, size, and location), as well as other associated conditions related to the injury.

Most small inorganic and inert orbital foreign bodies may be left alone if inaccessible without significant subsequent morbidity. Anteriorly located inorganic foreign bodies may be explored and easily removed with good preoperative localization (Fig. 13.8a–c). Posteriorly located foreign bodies, especially against vital structures, e.g., optic nerve, superior orbital fissure, optic canal, may either be observed or, if indicated, be removed with meticulous atraumatic approach with good preoperative and intraoperative localization. Intraconal lesions may be accessed by disinsertion of the corresponding extraocular muscle(s) (Fig. 13.9). In the case of firearm and blast injury, apart from removing the foreign bodies, the tract containing gun powder and debris should also be removed where possible as they may cause severe postoperative inflammation (Fig. 13.10).

Retained wooden orbital foreign bodies often shows a tendency to fragment during attempted removal, especially in case of old injury (Fig. 13.11). To overcome all intraoperative difficulties of FB removal along with residual

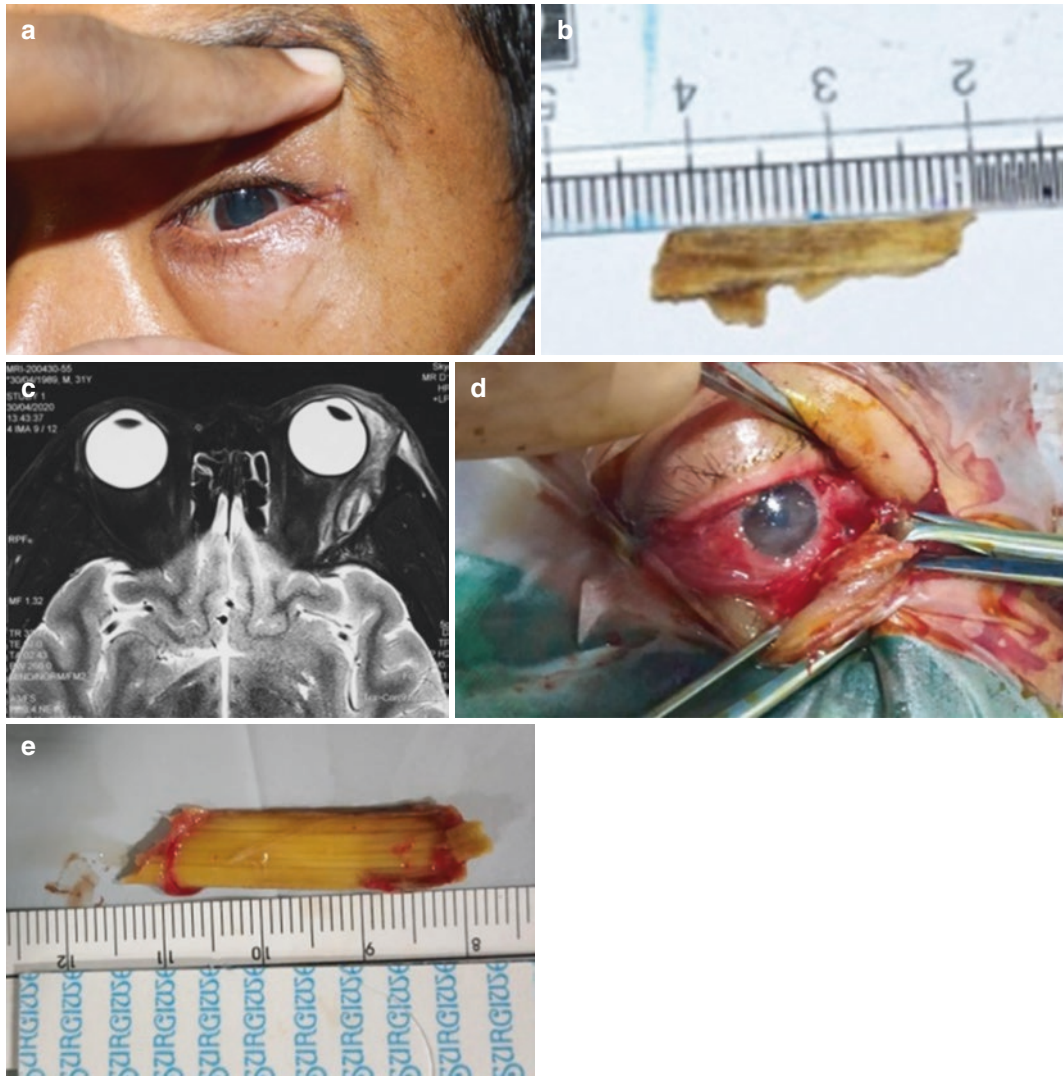

Fig. 13.7 Young male with previous orbital exploration with foreign body removal (**a, b**). MRI for recurrent lower eyelid abscess showed a residual foreign body against the left lateral rectus (**c**), which was extracted with a transconjunctival orbitotomy (**d, e**)

debris, a special technique is to use a gauze piece as a fishing net. The saline-soaked gauze piece with dilute adrenaline is passed along the tract to extract the foreign body along with the debris minimizing soft tissue trauma while ensuring complete removal of the foreign body along with particulate material. Complete removal is confirmed by a clean gauze coming out of the FB tract, which can be verified by endoscopy along the tract. After the removal of FB, the specimen is sent for microbial assessment of culture and sensitivity from the surface of the foreign body, which can guide further antimicrobial therapy.

An unusual but very serious type of injury causing orbital foreign bodies are blast injuries. These may occur from landmines, quarry blasts, riots and crowd control situations from firecracker injuries as well. More often than not, multiple foreign bodies are present despite maximal removal of foreign bodies.

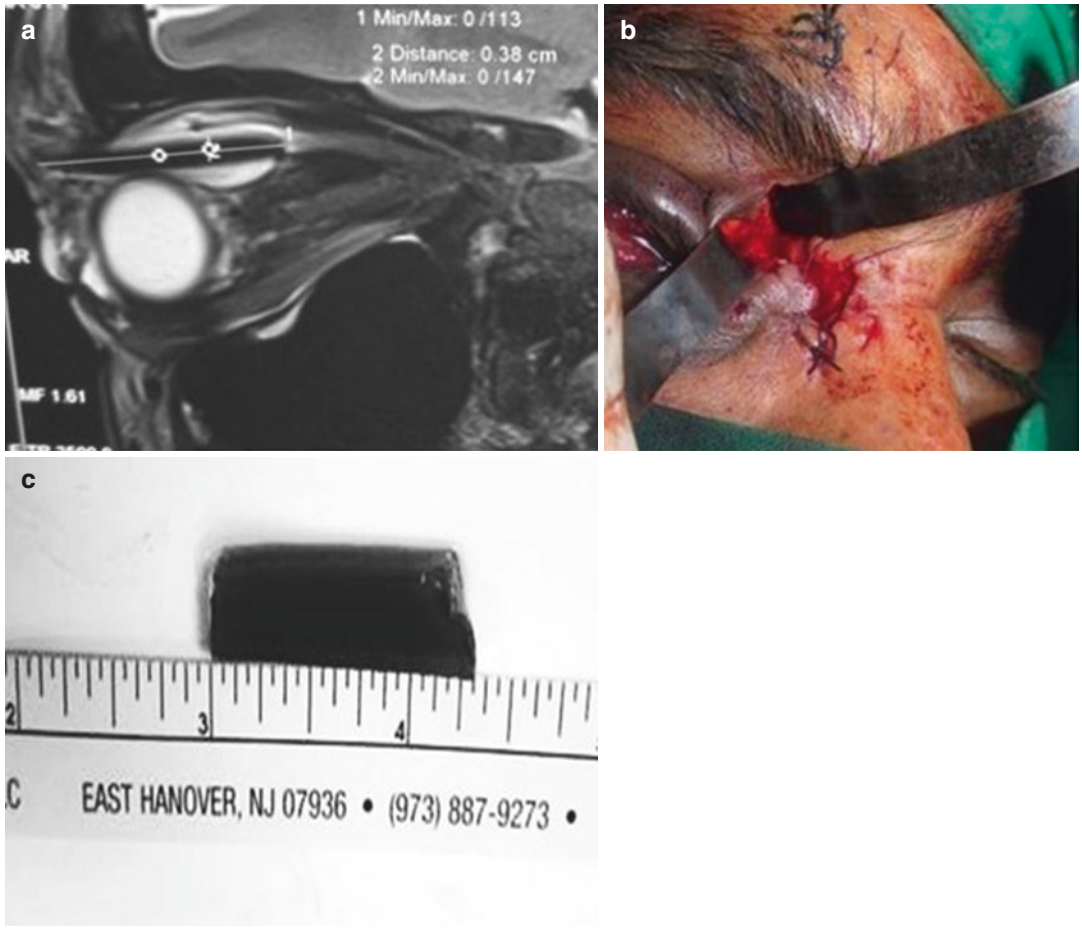

Fig. 13.8 (**a–c**) Anteriorly palpable linear glass foreign body visualized on MRI removed through an anterior orbitotomy approach

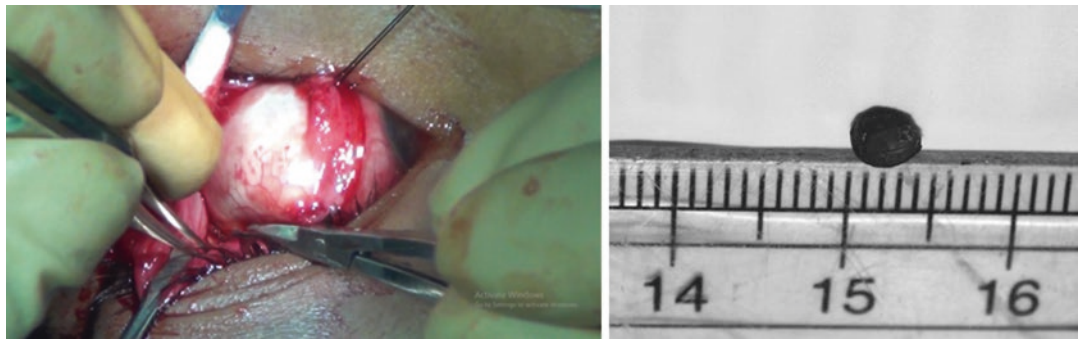

Fig. 13.9 Orbital foreign body removed after globe injury repair followed by orbital exploration with disinsertion of the lateral rectus

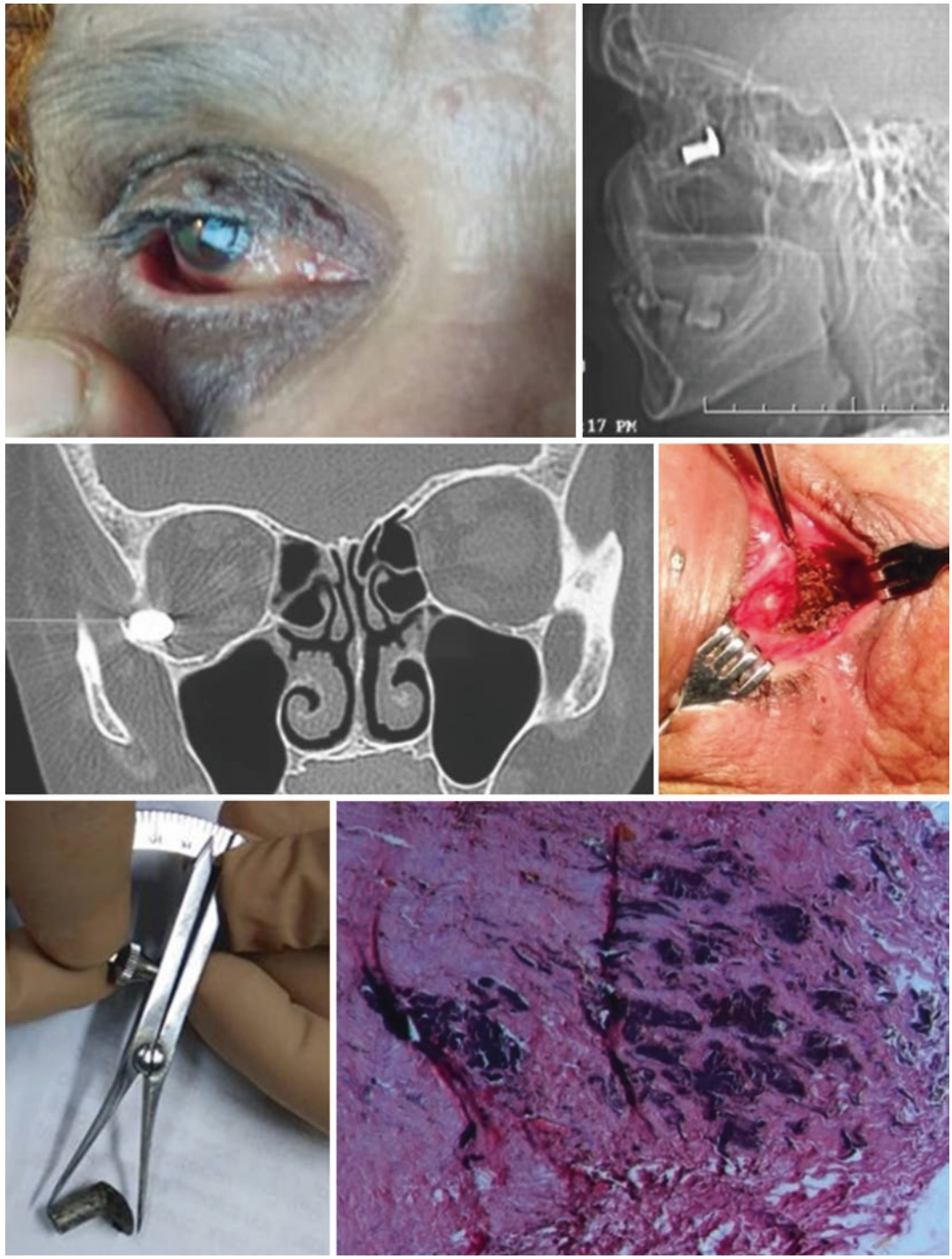

Fig. 13.10 Late presentation as chronic inflammation in an elderly old bomb blast injury victim. Plain x-ray followed by CT scan demonstrating the lodged radiopaque foreign body in the lateral wall of the orbit. Transconjunctival orbitotomy with foreign body removal including the tract showing siderotic tissue changes on Perl's stain

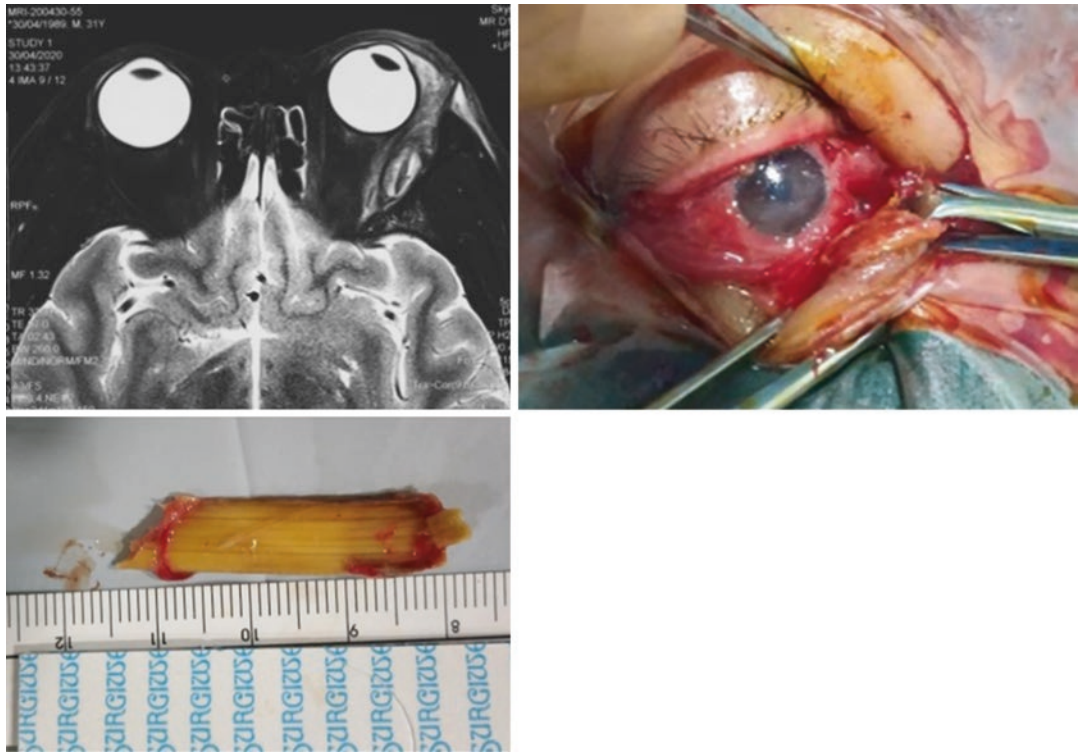

Fig. 13.11 Recurrent draining left orbital wound as a sentinel sign of retained residual or incompletely removed the organic foreign body

13.8 Complications

Despite advances in the recent era, sometimes a proper diagnosis is challenging, and patients may land up with poor outcomes due to missed foreign bodies, chronic infections and inflammations with residual fibrosis and globe limitation causing intractable diplopia and visual loss. The final outcome depends on various factors like initial visual acuity, the involvement of the optic nerve, location of the FB, the time elapsed between the injuries and reporting time and the expertise of management.

13.9 Prevention

To avoid blast and grenade injury, reforms of law and order situation along with the socio-economic development in that disturbed areas is probably the best way to prevent. Regarding orbital injury following road traffic accidents, the best possible prevention is the strict obedience of traffic rules and by introducing compulsory seat belt legislation. Airbags have gained extensive popularity in the recent era for the prevention of orbital trauma as well as morbidity and mortality of victims. Protective shatter-proof eye goggles and face shields go a long way in preventing shrapnel and projectile foreign bodies of the eyes and orbit.

13.10 Conclusion

A proper history, clinical examination with suspicion of foreign bodies, imaging followed by the appropriate threshold, timing and technique of foreign body extraction protecting the globe and other vital structures go a long way in the optimal management of afflicted patients.

References

1. Garg A, Moreno JMR, Shukla B, et al. In: Salam RA, El Toukhy E, editors. Chapter 21 Clinical diagnosis of ocular trauma. St. Louis: Jaypee Brothers Medical Publishers; 2009. p. 108–24.
2. Shukla B. New classification of ocular foreign bodies. Chin J Traumatol. 2016;19(6):319–21.
3. Rao R, Honavar SG. 2007 Chapter 17: orbital foreign bodies, pp. 246-257, in Aditi Swarup; Albert Y Wu Orbital fractures, principles, concepts and management. Sundar G. Imaging Science Today LLC: New York., ISBN–13: 978-0997781922
4. Balasubramanian C, Kaliaperumal C, Jadun CK, Dias PS. Transorbital intracranial penetrating injury-an anatomical classification. Surg Neurol. 2009;71(2):238–40. https://doi.org/10.1016/j.surneu.2007.07.050. Epub 2008 Mar 4
5. Fulcher TP, McNab AA, Sullivan TJ. Clinical features and management of intraorbital foreign bodies. Ophthalmology. 2002;109(3):494–500.
6. Shinder R, Gutman J, Gunasekera CD, Connor M, Nakra T. Occult orbital organic foreign body. Ophthal Plast Reconstr Surg. 2011;27:463–4.
7. Baranwal VK, Gupta RP, Johri S, et al. A case of orbito-cranial foreign body. Med J Armed Forces India. 2014;72(1):82–4.
8. Danesh-Meyer HV, Savino PJ, Bilyk JR, Sergott RC, Kubis K. Gaze-evoked amaurosis produced by intraorbital buckshot pellet. Ophthalmology. 2001 Jan;108(1):201–6. https://doi.org/10.1016/s0161-6420(00)00433-4.
9. Kuhn F, Pieramici DJ. Ocular trauma, principles and practice. New York: Thieme; 2002.
10. Shah M, Sundar G, Shah S. Ocular trauma score revisited – making sense of it all. Lat Am J Ophthalmol. 2019;2:4.
11. Gilmour DF, Ramaesh K, Fleck BW. Transorbital intra-cranial air gun injury. Eur J Ophthalmol. 2003 Apr;13(3):320–3. https://doi.org/10.1177/112067210301300314.
12. Lustrin ES, Brown JH, Novelline R, Weber AL. Radiologic assessment of trauma and foreign bodies of the eye and orbit. Neuroimaging Clin N Am. 1996 Feb;6(1):219–37.
13. Almousa R, Amrith S, Mani AH, Liang S, Sundar G. Radiological signs of periorbital trauma – the Singapore experience. Orbit. 2010;29(6):307–12. https://doi.org/10.3109/01676830.2010.510235. Epub 2010 Oct 18
14. Mohapatra SD, Das JK, Bhattacharjee H. An unusual case of orbito-cranial wooden foreign body with amazing outcome: a case report. Indian J Ophthalmol. 2020;68(1):219–21.
15. Kaviani F, Rashid RJ, Shahmoradi Z, Gholamian M. Detection of foreign bodies by spiral computed tomography and cone beam computed tomography in maxillofacial regions. J Dent Res Dent Clin Dent Prospect. 2014;8(3):166–71.
16. Sundar G. Chapter 3: Practical classification of orbital & orbitofacial fractures. In: Swarup A, Wu AY, editors. Orbital fractures, principles, concepts and management. New York: Imaging Science Today, LLC; 2018. p. 28–36. ISBN – 13:978-0997781922.
17. Shelsta HN, Bilyk JR, Rubin PA, Penne RB, Carrasco JR. Wooden intraorbital foreign body injuries: clinical characteristics and outcomes of 23 patients. Ophthalmic Plast Reconstr Surg. 2010;26(4):238–44.
18. Nasr AM, Haik BG, Fleming JC, Al-Hussain HM, Karcioglu ZA. Penetrating orbital injury with organic foreign body. Ophthalmology. 1999;106:523–32.
19. Dolar Bilge A, Yilmaz H, Yazici B, Naqadan F. Intraorbital foreign bodies: clinical features and outcomes of surgical removal. Ulus Travma Acil Cerrahi Derg. 2016;22(5):432–6.

Traumatic Glaucoma

14

Zhiliang Wang, Xin Che, Jing Jiang, and Yiwen Qian

Abstract

Glaucoma resulting from trauma is termed as traumatic glaucoma. Traumatic glaucoma is a brunch of secondary glaucoma, so it should be called traumatic secondary glaucoma. Traumatic secondary glaucoma can be caused by penetrating, blunt, chemical, or other injuries of eye. Unlike primary glaucoma, the pathogenesis of traumatic secondary glaucoma is always complicated and the treatment is difficult. Traumatic glaucoma can be classified in different ways, but generally it is sorted by etiology. This chapter is about to introduce traumatic glaucoma induced by intraocular hemorrhage, lens dislocation, rupture of lens capsule, malignant glaucoma, neovascular, angle recession, cornea perforation, iris coloboma, and uveitis.

Keywords

Blunt trauma · Post-surgery · Intraocular hemorrhage · Lens causes · Angle lesion · Cornea perforation

Supplementary Information The online version of this chapter (https://doi.org/10.1007/978-981-16-5340-7_14) contains supplementary material, which is available to authorized users.

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14.1 Introduction

Glaucoma resulting from trauma is termed as traumatic glaucoma. Traumatic glaucoma is a brunch of secondary glaucoma, so it should be called traumatic secondary glaucoma. Traumatic secondary glaucoma can be caused by penetrating, blunt, chemical, or other injuries of eye. Unlike primary glaucoma, the pathogenesis of traumatic secondary glaucoma is always complicated and the treatment is difficult. Traumatic glaucoma can be classified in different ways, but generally it is sorted by etiology. This chapter is about to introduce traumatic glaucoma induced by intraocular hemorrhage, lens dislocation, rupture of lens capsule, malignant glaucoma, neovascular, angle recession, cornea perforation, iris coloboma, and uveitis.

14.2 Glaucoma Secondary to Traumatic Intraocular Hemorrhage

Anterior chamber hemorrhage is the leading cause of traumatic ocular hypertension.

14.2.1 Hyphema

14.2.1.1 Etiology

Hyphema usually occurs after ocular blunt trauma with blood mainly coming from iris. The

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intraocular pressure elevates due to obstruction of angle by blood cells, so it is a type of secondary open-angle glaucoma.

14.2.1.2 Clinical Manifestation

Hyphema often manifests as massive bleeding in the anterior chamber (Fig. 14.1) and a significant rise of IOP. In general, the IOP will not reach 30 mmHg when the blood level is under half of the anterior chamber, but the risk of glaucoma increases when rehemorrhage occurs in 20% of patients 5d after injury because of: a) severe iris damage and loss of ciliary body; b) blood in the posterior chamber and vitreous flowing into the anterior chamber.

14.2.2 Hemosiderin Glaucoma

14.2.2.1 Etiology

Repeated bleeding in the anterior chamber, vitreous, and retina. Usually caused by hemosiderin leading to sclerosis and obstruction of the trabecular meshwork, or iron deposits caused by the long-term retention of iron.

14.2.2.2 Clinical Manifestations

This type of glaucoma progress slowly. Brown iron particles are visible on the corneal parenchyma, lens, and retina (Fig. 14.2).

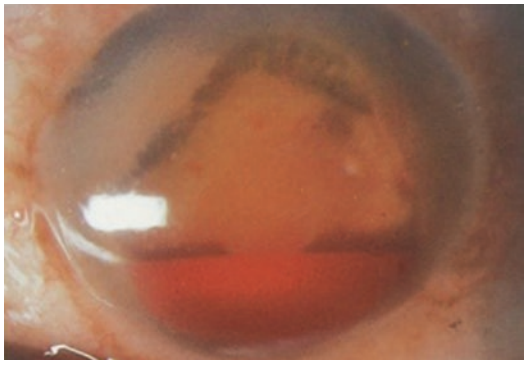

Fig. 14.1 Blunt injury, hyphema 1/3

14.2.3 Hemolytic Glaucoma

14.2.3.1 Etiology

Hemolytic glaucoma is usually caused by vitreous hemorrhage which comes from eyeball perforation, contusion, and foreign body injury. When it occurs, the erythrocytes enter the vitreous body and erupted under the pressure of oxygen and carbon dioxide. The hemoglobin is swallowed by macrophages and entering the anterior chamber blocking the trabeculae, resulting in obstruction of the outflow of aqueous humor and a sudden increase in IOP.

14.2.3.2 Clinical Manifestations

It usually occurs about 1 week after vitreous hemorrhage (Fig. 14.3). Patients suddenly feel headache, eye pain, and sudden increase in IOP. Pigments and macrophages can be seen in anterior chamber angle.

14.2.4 Ghost-cell Glaucoma

14.2.4.1 Etiology

Ghost-cell glaucoma is an uncommon condition that occurs in association with intraocular hemorrhage. In this entity, erythrocytes degenerate in the vitreous, migrate forward to the anterior chamber through a disrupted anterior hyaloid

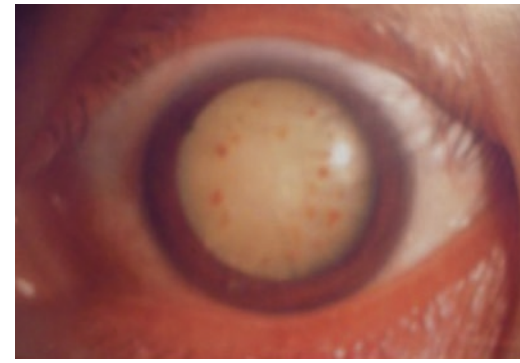

Fig. 14.2 Intraocular iron foreign body. Six months after trauma, brown iron particles were deposited in the angle, the posterior surface of the cornea and the anterior surface of the lens, lens opacity

face, and then obstruct the trabecular meshwork and cause an increase in IOP.

The RBCs in the vitreous degenerate to tan-colored spheres (ghost cells), which appear empty except for clumps of denatured hemoglobin called Heinz bodies. The ghost cells are more rigid than are normal RBCs and thus are less able to pass through the trabecular meshwork.

14.2.4.2 Clinical Manifestations

IOP depends on the number of ghost cells in the anterior chamber. Numerous brown particles can be seen in the anterior chamber. Similar to hemolytic glaucoma, but the content of aqueous humor is different.

14.2.5 Treatment

Glaucoma caused by intraocular hemorrhage is open-angle glaucoma. The principle is to remove intraocular hemorrhage, degenerated blood cells, and inflammatory cell components by anterior chamber irrigation or vitrectomy. Drugs can help lower the IOP.

14.2.5.1 Drug Treatment

β-Receptor blockers, α-receptor agonists, carbonic anhydrase inhibitors, hypertonic agents can be used for lowering IOP. Miotics are not recommended. Mydriatic should be used for preventing posterior synechiae.

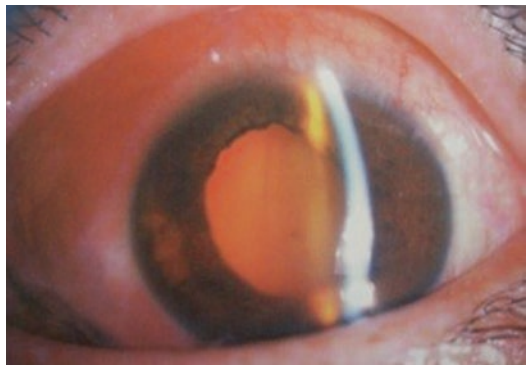

Fig. 14.3 Ocular contusion, vitreous hemorrhage for 1 week, hemocyte / Denatured hemoglobin in anterior chamber

14.2.5.2 Anterior Chamber Irrigation

Anterior chamber irrigation can be used to wash out the blood of hyphema, also the inflammatory cell components such as hemosiderin, ghost cells, and macrophages.

Vitrectomy

For patients whose IOP cannot be well-controlled through medication and anterior chamber irrigation, especially those with vitreous hemorrhage, vitrectomy is necessary.

14.2.5.3 Glaucoma Drainage Surgery

For patients with long-term intraocular hemorrhage caused by trabecular meshwork degeneration and complete obstruction of the angle outflow tract, glaucoma drainage surgery is required to control IOP.

14.3 Secondary Glaucoma by Lens Dislocation

14.3.1 Etiology

1. Dislocated lens in the anterior chamber (Fig. 14.4), causing open-angle glaucoma due to pupillary block by the synechia of lens and anterior iris.
2. Dislocated lens into the vitreous cavity (Fig. 14.5) makes vitreous body obstructing the anterior chamber.
3. Dislocated lens incarcerated in pupil (Fig. 14.6), causing angle-closure glaucoma

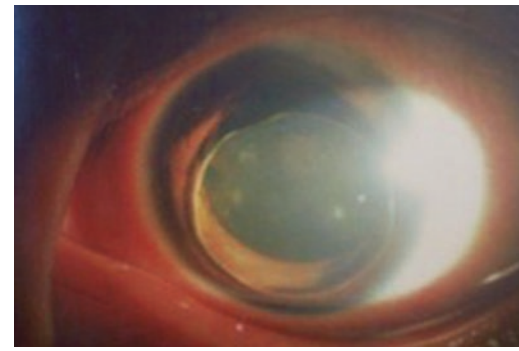

Fig. 14.4 Dislocated lens in the anterior chamber

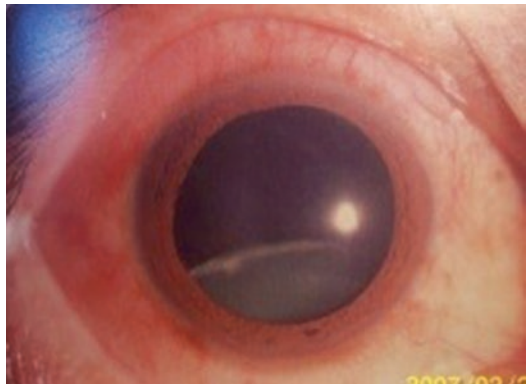

Fig. 14.5 Dislocated lens in the vitreous cavity

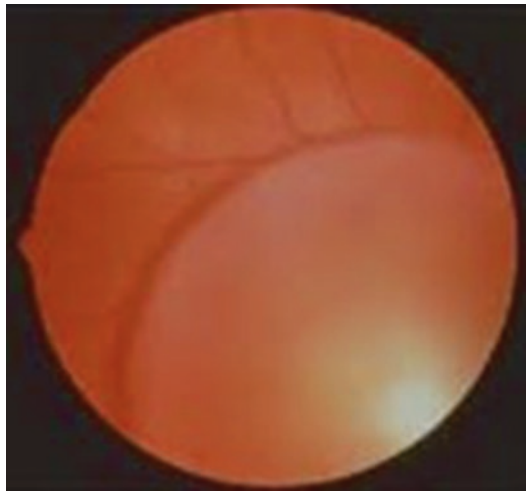

Fig. 14.6 Lens subluxation

due to elevated pressure of the posterior chamber.
4. Dislocated lens hitting ciliary body, causing excessive secretion of aqueous humor and secretory glaucoma.

14.3.2 Clinical Manifestation

1. Visual loss is the main symptom and iridodonesis is the typical sign. Complete lens dislocation into anterior chamber usually shows as deepened anterior chamber retroposed iris, rising IOP by vitreous body incarcerated in pupil. Narrowed or closed angle will be seen when the pupillary block occurs, which is similar to primary angle-closure glaucoma (PACG) in acute stage.
2. Repeated IOP trace is needed for diagnosis of secretory glaucoma, showing as high IOP, normal outflow facility, and vast aqueous fluid generation.

14.3.3 Treatment

1. Mydriasis should be performed rather than myosis.
2. IOP-lowering medication. Local use of beta-blocker, alpha receptor agonist, systemic use of hypertonic agent such as mannitol, and oral administration of carbonic anhydrase inhibitor are all optional according to the IOP level and clinical manifestations.
3. Surgery. The principle of surgery is to rebuild the aqueous humor drainage from the posterior chamber to the anterior chamber, by removing the dislocated lens and vitreous body obstructed in the anterior chamber. Based on our experience, IOL fixation to sclera without suturing (Fig. 14.7) is recommended.

14.3.4 Surgical Procedures (Video 14.1)

A 23-gauge vitrectomy is performed under retrobulbar anesthesia.

The conjunctiva is cut open 3.0 mm at 2 o'clock and 8 o'clock.

A 2.8 mm clear corneal incision (CCI) is made at 11 o'clock.

Two 3.0 mm long limbal parallel scleral tunnels (counterclockwise) of approximately 50% scleral thickness are created 2 mm from the limbus using a disposable syringe needle and dissected 1.0 mm from 1 side at the beginning of the scleral tunnels.

Two clockwise scleral incisions (besides the tunnel) are made using a 23-gauge needle at an angle 30 degrees above the scleral. The 2 incisions are 180 degrees from each other (usually at 2 o'clock and 8 o'clock).

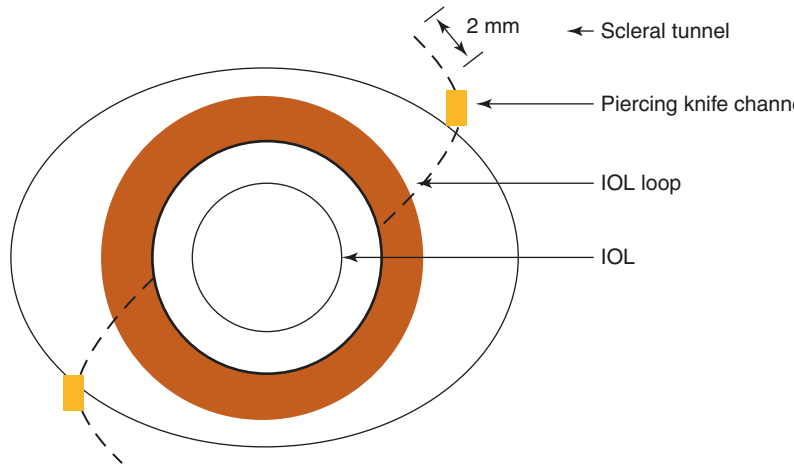

Fig. 14.7 Pattern diagram of IOL fixation to sclera without suturing

3-Piece IOL is inserted into the anterior chamber.

The leading haptic is held with a forceps and then pulled out of the eye through the scleral incisions.

The trailing haptic is held with a forceps and both haptics are externalized onto the sclera.

The haptics are inserted approximately 2.0 mm into the scleral tunnel; the IOL is centered.

At the completion of surgery, the infusion cannula is removed, and all sclerotomy sites are inspected for wound leakage.

14.4 Glaucoma Secondary to Traumatic Rupture of the Lens Capsule

14.4.1 Lens Allergic Glaucoma

14.4.1.1 Etiology

Lens injury may cause allergic reaction to lens substance (protein) and fibrin exudates block the angle of the chamber. Or the fibrous exudative membrane forms membrane closure to exhibit the posterior aqueous humor entering into the anterior chamber, resulting in intraocular hypertension.

14.4.1.2 Clinical Manifestation

Inflammation can occur within hours or days, or as late as a few months. Uveitis can be mild or severe, with a large number of hyphema and lens fragments in the anterior chamber. When clinical signs are suspected to be allergic inflammation or severe uveitis, diagnosis of anterior chamber aspiration and diagnostic vitreous body should be performed.

14.4.2 Lens Particle Glaucoma

14.4.2.1 Etiology

It may be due to particles (phagocytosis of lens protein by macrophages), or the lens cortex and capsule fragments block the angle.

14.4.2.2 Clinical Manifestation

There are white lens cortex and transparent capsule fragments circulating in aqueous humor, which can also deposit on the corneal endothelium with aqueous flare and hyphema.

14.4.3 Phacolytic Glaucoma

14.4.3.1 Etiology

Secondary open-angle glaucoma is caused by soluble crystalline blocking the angle after trauma.

14.4.3.2 Clinical Manifestation

Red eye, pain, corneal edema, progressive increase of intraocular pressure, aqueous flare, large transparent cells (macrophages) phenomenon, small particles circulating in aqueous humor.

14.4.3.3 Treatment

On the basis of hormone and antiglaucoma drugs/IOP-lowering medicine, surgical treatment should be performed as soon as possible to remove damaged cortex and nucleus.

14.5 Traumatic Malignant Glaucoma

14.5.1 Etiology

1. Post-glaucoma, cataract, and other intraocular surgery, ciliary body swells and adheres with lens, causes ciliary block. As a result, aqueous humor flows into the vitreous cavity, and increases the pressure, which further pushes forward the lens-iris diaphragm, making the anterior chamber become shallow, and IOP rises sharply (Figs. 14.8 and 14.9).
2. Ciliary block glaucoma is secondary to ocular chemical injury, contusion, and perforation. Ciliary body adheres to lens due to inflammatory edema and spasm.
3. Vitreous hemorrhage, opacity, and liquefaction of traumatic vitreous are one of the conditions for aqueous humor to accumulate in vitreous body.
4. Traumatic vitreous hemorrhage, turbidity, or liquefaction is one of the conditions of aqueous humor accumulation in vitreous.

14.5.2 Clinical Manifestation

1. Abnormal anatomical structures of the affected eye: Short axis, shallow anterior chamber, large lens thickness, loose suspensory ligament, and relatively anterior lens position.
2. Persistent high intraocular pressure(IOP) is an important sign. Mydriasis will increase rather than decrease the IOP. So it is called adverse glaucoma.
3. Obvious iris bombe and closed angle.

14.5.3 Treatment

1. The treatment of ocular trauma according to the different causes.
2. Treatment of malignant glaucoma:
   (a) IOP-lowering drugs: Atropine: dilate the pupil and prevent adhesion; hypertonic agents, carbonic anhydrase inhibitors: reduce IOP; corticosteroids: reduce inflammatory reaction. Surgery is necessary.
   (b) Laser iridotomy: promote aqueous humor drainage into the anterior chamber, but it does

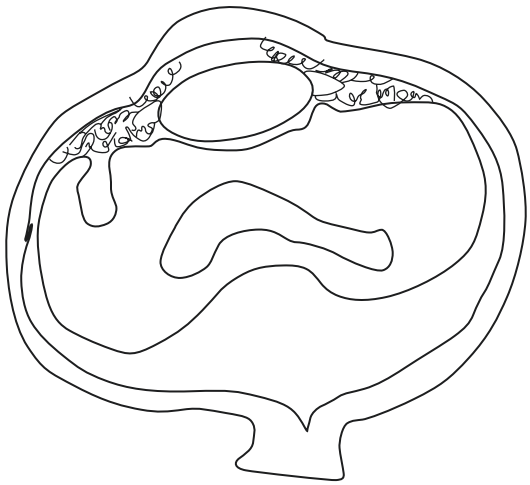

Fig. 14.8 Pattern diagram of malignant glaucoma

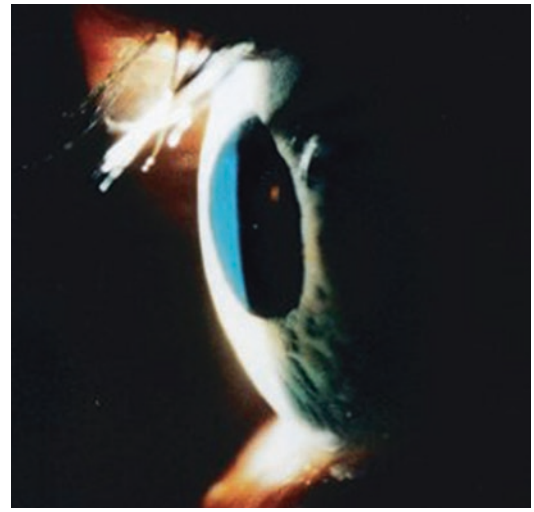

Fig. 14.9 Disappear of anterior chamber

not relieve the pressure of lens-iris diaphragm forward. Moreover, iridotomy is easy to be blocked by inflammatory exudates.

3. Anterior vitrectomy

The principle of treatment is to reverse the aqueous humor drainage from vitreous body, lens suspension ligament, and iris terminal. Reverse the lens-iris diaphragm to anatomical position to make the posterior aqueous humor can enter the anterior chamber, and reopen the angle.

4. Glaucoma drainage

For patients with complete angle occlusion caused by malignant glaucoma, glaucoma drainage surgery should be performed on the basis of anterior vitrectomy to control IOP.

We improved the success rate of anterior vitrectomy significantly through local minimal invasive vitrectomy.

14.5.4 Case Presentation

The patient was treated for "eye pain with visual loss for 2 months after trabeculectomy in the both eyes."

14.5.5 Ophthalmic Examination

VOD: 0.15, VOS: LP TOD:30 mmHg, TOS:27 mmHg, Ocular axis: R 20.04 mm, L: 20.06 mm.

Anterior chamber disappeared, the pupil was irregular, the lens was opacity, optic papilla was pale in both eyes, C / D = 0.8 (Figs. 14.10, 14.11, 14.12, and 14.13).

14.5.6 Surgical Procedure (Video 14.2)

1. Partial PPV

After the routine disinfection and retrobulbar anesthesia, a 23-gauge vitrector was inserted into the vitreous cavity via a self-healing incision about 3.5 mm after limbus on the same median of preexisting peripheral iridectomy. No infusion or illumination was needed. Then partial vitrectomy was performed until approximately 0.5 ml vitreous was removed from the anterior vitreous cavity just behind the position of iridectomy.

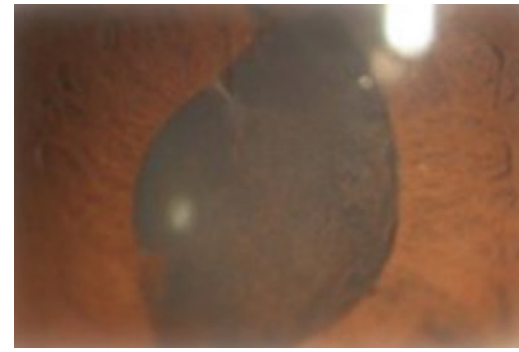

Fig. 14.10 Malignant glaucoma: pupilloplegia, anterior synechia (right eye)

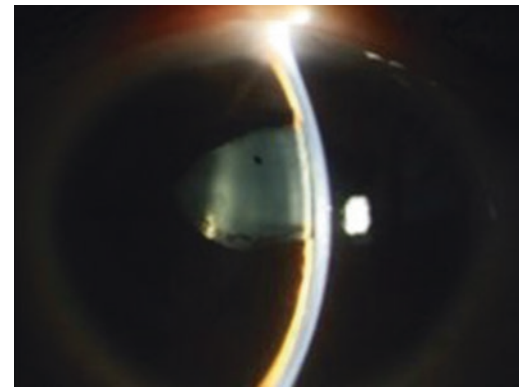

Fig. 14.11 Anterior chamber disappears (left eye)

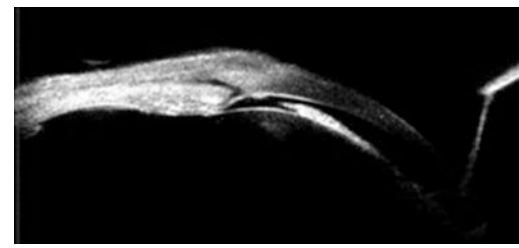

Fig. 14.12 UBM shows that anterior chamber is very shallow, the lens-iris diaphragm moves forward, iris bombe, the anterior synechia, the angle closed (the right eye, the left eye)

2. Phacoemulsification and IOL implantation
3. Zonulohyaloidectomy and posterior capsulectomy

The partial vitrectomy was performed again to confirm that vitreous behind the peripheral iridectomy was cutted. Then the tip of the cutter was introduced carefully through this iridectomy incision forward the anterior chamber to achieve a zonulohyaloidectomy. Finally, in the central zone, the posterior capsular of about 4 mm in diameter was cut with the help of vitrector to prevent the occurrence of posterior capsular opacity.

In all eyes, we realized the significant deepening of the anterior chamber by the flow of aqueous fluid forward. Dexamethasone 2 mg was given by subconjunctival injections and a topical treatment of tropicamide was applied for 2 weeks.

14.6 Traumatic Neovascular Glaucoma

14.6.1 Etiology

Be common in diabetic retinopathy (DR), retinal vein occlusion (RVO), Eales disease, and other retinal ischemic diseases. Some cases were caused by trauma. Neovascularization promotes the formation of peripheral anterior synechia (PAS) and pulls the iris to the cornea. Sometimes the neovascularization membrane covers and blocks the angle (Fig. 14.13).

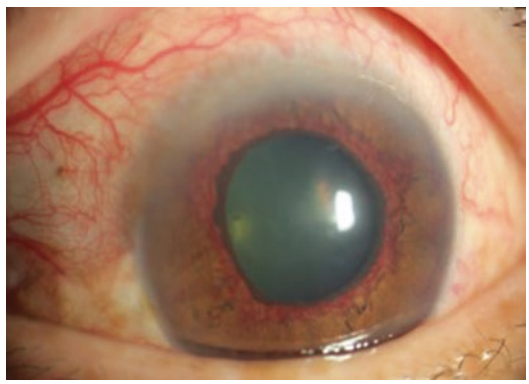

Fig. 14.13 Whole iris neovascularization

14.6.2 Clinical Manifestations

1. Usually a late complication of trauma, but maybe advanced when traumatic uveitis is severe.
2. Secondary to severe vascular injury, recurrent fundus hemorrhage and uncontrollable traumatic uveitis, or serious lens injury and residual cortex.
3. The main basis of diagnosis is the history of ocular trauma, persistent intraocular hypertension, and rubeosis of iris. Gonioscopy showed angle adhesion and vascular membrane formation.

14.6.3 Treatment

1. IOP-lowering medication
2. Intravitreal injection of anti-VEGF drugs: promote the regression of NV, reduce intraocular pressure (IOP), and create conditions for subsequent surgery.
3. Glaucoma drainage surgery (Fig. 14.14): due to severe inflammatory reaction, trabeculectomy, which may cause scar adhesion is not recommended. The short-term effect of glaucoma valve implantation is better. Cyclocryosis is one of the effective methods at present in recent years, but it causes damage.
4. Treatment of disease causes: Vitrectomy or panretinal photocoagulation (PRP) and other

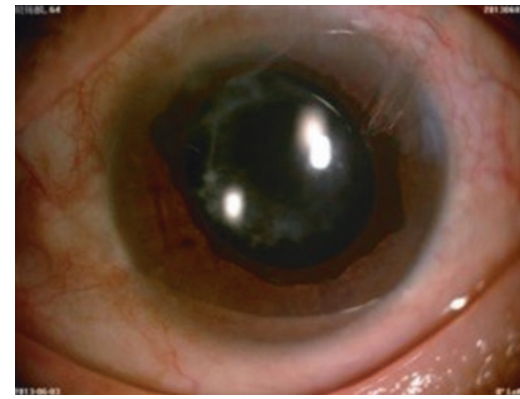

Fig. 14.14 Neovascularization disappeared postoperative, Valve pipe is visible

treatments to reduce the ischemic state of intraocular tissue.

14.7 Glaucoma Secondary to Traumatic Angle Recession

Angle recession is quite often(81%–93%) in ocular contusion. Incidence of angle recession glaucoma accounts for 2%–10% of the total.

14.7.1 Etiology

Traumatic angle recession is mainly caused by ocular contusion, with the separation between the circular and longitudinal muscles of the ciliary body (Fig. 14.15). While the longitudinal muscle is still attached to the scleral process, so the angle of the chamber is widened and deepened.

14.7.2 Clinical Manifestation

Gonioscopy showed the ciliary body band (CBB) widened. The peripheral anterior chamber was deepened, and the terminal of iris moved backward.

14.7.3 Treatment

(1) IOP-lowering.
(2) Glaucoma drainage surgery: trabeculectomy is the first choice; mitomycin C can be used to reduce the scar formation of filter valve.

14.8 Low IOP Glaucoma Secondary to Traumatic Cyclodialysis

14.8.1 Etiology

Due to the detachment of the whole ciliary body from the sclera after ocular contusion (Fig. 14.16). The adverse flow of aqueous humor into the superior ciliary cavity leads to ciliary body detachment, secondary low intraocular pressure glaucoma, macular edema, and other complications.

14.8.2 Clinical Manifestations

Pupil deformation, peripheral anterior chamber deepening, intraocular pressure lower than normal, retinal edema and thickening, macular edema can cause visual loss. Gonioscopy shows a gap between iris and sclera, which is distinguished from ciliary body detachment.

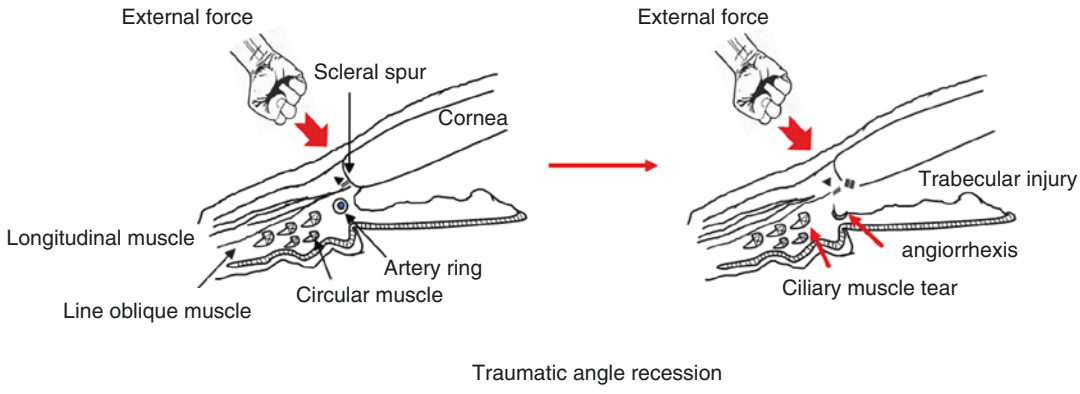

Fig. 14.15 Pattern diagram of traumatic angle recession

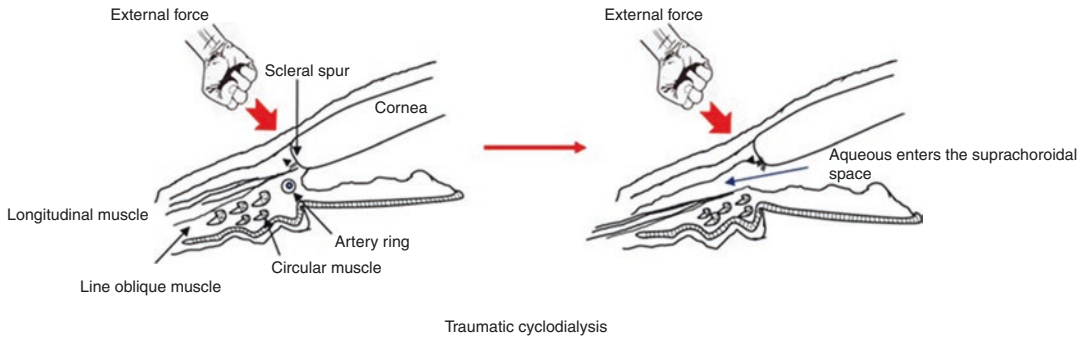

Fig. 14.16 Pattern diagram of traumatic cyclodialysis

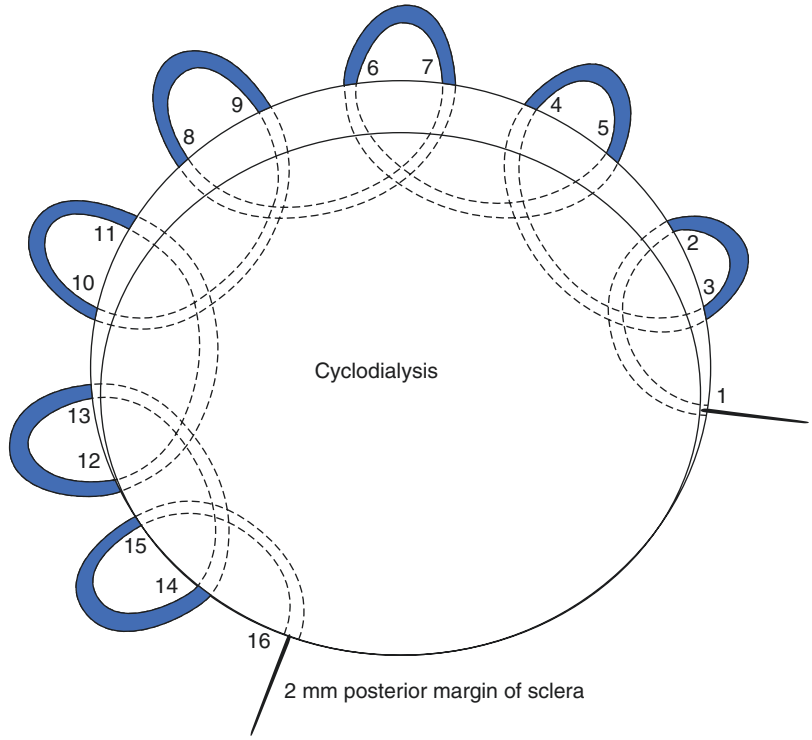

Fig. 14.17 Pattern diagram of the suture of cyclodialysis

14.8.3 Treatment

Local therapy can be applied to patients with mild ciliary body detachment and without significant visual loss. Surgery is necessary if macular edema happens (Fig. 14.17).

14.8.3.1 Medication

Corticosteroid eye drops, especially dexamethasone can help increase IOP and reduce intraocular inflammation. Mydriasis also helps to increase IOP.

14.8.4 Surgical Procedure (Video 14.3)

The surgery is performed under retrobulbar anesthesia.

After preoperative marking of the location of the cleft at the limbus, the conjunctiva is opened at the limbus with this peritomy extending 30° beyond both ends of the cyclodialysis cleft.

The infusion cannula is first set to maintain the intraocular pressure.

Phacoemulsification is performed under low flow rate (infusion bottle height 30 cm).

A 13-mm Morcher Type 1 L CTR, a modified Cionni type CTR with 1 eyelet, is then inserted into the capsular bag.

A foldable posterior chamber intraocular lens (IOL) (AR40e, AMO) is implanted in the capsular bag through the corneal tunnel.

Then a three-port pars plana vitrectomy is performed.

After removal of the vitreous body, one of the two long curved needles of a double-armed 10/0 polypropylene thread is pierced into the eye through the sclera 2 mm from the limbus and passing from the inside of the eye through the ciliary body and sclera to the outside of the globe.

The continuous and cerclage sutures are parallel to the limbus and we do every 3 sutures/quadrant.

After all the cyclodialysis clefts are repaired, the sutures on both ends of the cleft are fixed on the sclera.

The knots of the suture are placed on the surface of the sclera and are covered by conjunctiva. The length of the thread ends is longer than 3 mm so that the thread ends are located tensionless under the conjunctiva without great risk of piercing through the conjunctiva.

After re-fixation of the ciliary body, the retina is carefully checked and to address a retinal detachment if present.

At the completion of surgery, the infusion cannula is removed.

The conjunctival incisions are closed with 8–0 absorbable sutures.

14.9 Glaucoma Secondary to Corneal Perforation

14.9.1 Etiology

Corneal perforation with serious iritis is prone to peripheral anterior synechia (PAS) (Figs. 14.18 and 14.19). Iris prolapse caused by corneal perforation can cause adhesive corneal leukoplakia if not handled properly, causing secondary angle closure glaucoma.

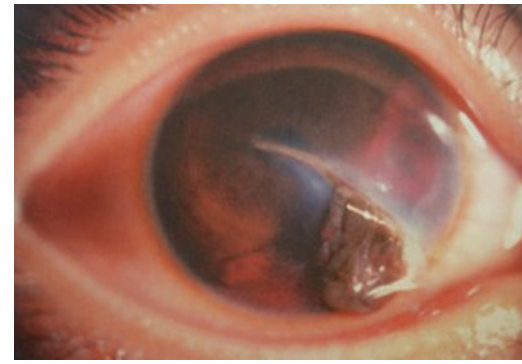

Fig. 14.18 Sharp injury, iris prolapses, and incarcerates into the cornea

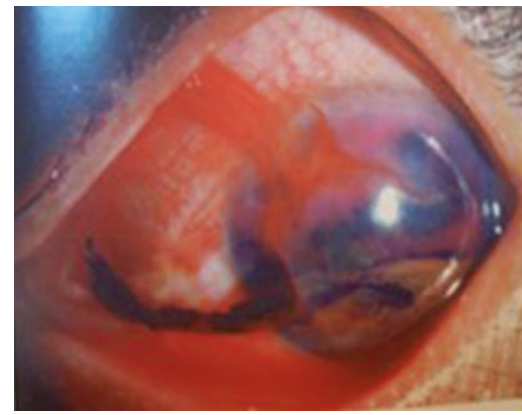

Fig. 14.19 Sharp injury, hyphema

14.9.2 Clinical Manifestation

1. Be similar to the symptoms of acute angle closure glaucoma attack.
2. Intraocular hypertension; corneal leukoplakia, corneal scar will affect the results of IOP.
3. PAS and anterior chamber angle occlusion.

14.9.3 Treatment

(1) IOP-lowering medications.
(2) Partial iridectomy or filtering operation was selected according to the synechia.

14.10 Glaucoma Secondary to Traumatic Iris Defect

14.10.1 Etiology

Blunt injury, sharp injury, iridectomy, and excessively broken iris are the main reasons. When the iridodialysis is over 180 degrees, iris may be curled and sinks into the angle and lead to Iris tissue necrosis, decomposition, and absorption (Figs. 14.20 and 14.21).

14.10.2 Pathogenesis

The residual iris in the aqueous humor, which lacks of elasticity and tension, is easy to adhere to trabeculae.

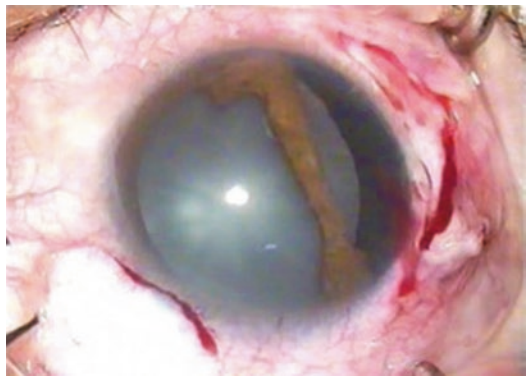

Fig. 14.20 Iridodialysis occurred after blunt trauma

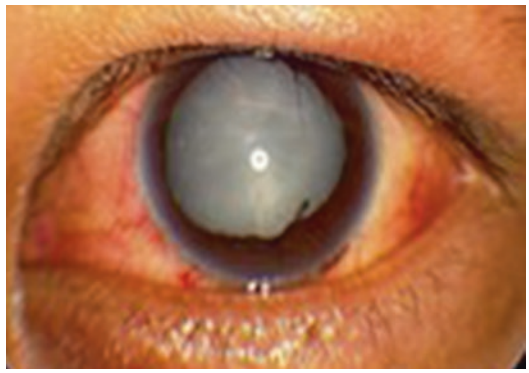

Fig. 14.21 Iris atrophy occurred one month after blunt trauma

14.10.3 Clinical Manifestation

Acute angle-closure glaucoma can be secondary to iris defect. The patient had a sudden onset of headache, eye pain, nausea, and vomiting. Conjunctival swelling or mixed congestion, corneal edema, stubborn ocular hypertension.

14.10.4 Treatment

1. IOP-lowering medications.
2. Glaucoma drainage surgery such as goniotomy and trabeculectomy.

Male, 27-year old, left eye pain and blurred vision for 1 month after the blunt ocular trauma(Figs. 14.22 and 14.23).

Ophthalmic examination: VOS: 0.4, TOS: 36 mmHg.

14.10.5 Treatment

Surgery: Repair of iris injury; suture the broken pupillary sphincter.

Iridodialysis: Sewing machine suture of iris at the posterior border of corneal limbus (Figs. 14.24 and 14.25).

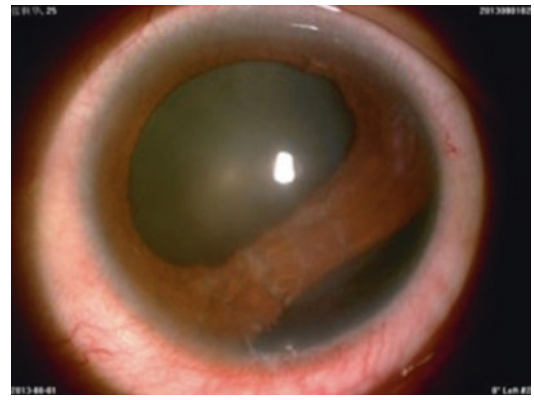

Fig. 14.22 Iridodialysis, pupillary deformation, and lens opacity

14.10.6 Case Presentation

Male, 27-year old, severe eye pain with blurred vision, 3 days after blunt trauma.

Examination: VOS: HM/BE, TOS: 36 mmHg, mixed congestion, whole iridodialysis (Fig. 14.26).

Diagnosis: Glaucoma and cataract after trauma.

14.10.7 Surgical Procedure (Video 14.4)

The surgery is performed under retrobulbar anesthesia.

A single 23-gauge port for infusion cannula to maintain IOP.

Cutting the conjunctiva along the limbus.

Making 0.5 mm sclera incision every 15° about 1.75 mm–2 mm behind the limbus.

Viscoelasticity into the anterior chamber.

Fixation of the defected iris through the sclera with 8–0 absorbable sutures (Vicryl) (suture impaction).

Cataract surgery

At the completion of surgery, the infusion cannula is removed.

The conjunctival incisions are closed with 8–0 absorbable sutures.

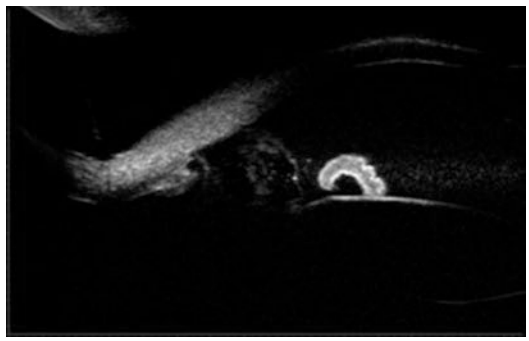

Fig. 14.23 UBM: Anterior chamber exudates, iris curling iris detachment

14.11 Glaucoma Secondary to Traumatic Uveitis

14.11.1 Etiology

Ocular contusion, perforating injury, and chemical injury can cause severe uveitis with serious exudates, resulting in a series of iridocyclitis, such as posterior synechia, Peripheral anterior synechia (PAS), pupillary occlusion, and iris bombé (Figs. 14.27, 14.28, and 14.29).

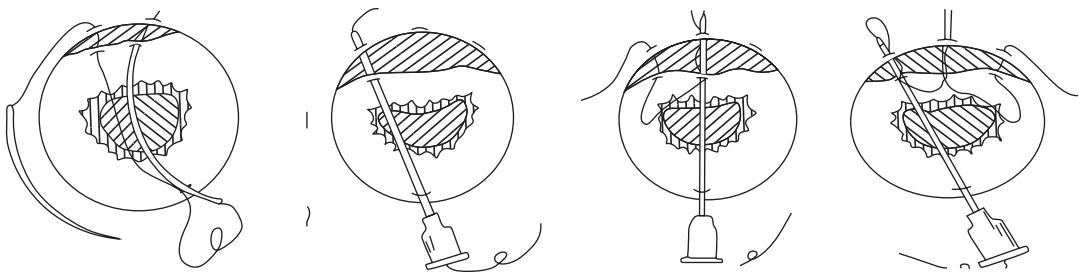

Fig. 14.24 Pattern diagram of sewing machine suture

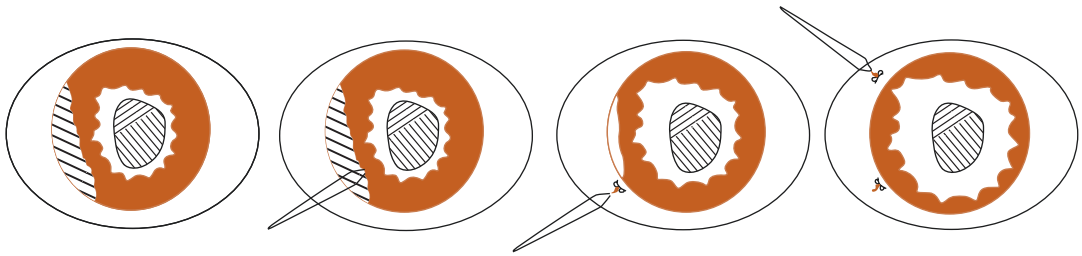

Fig. 14.25 Our experience of suture impaction

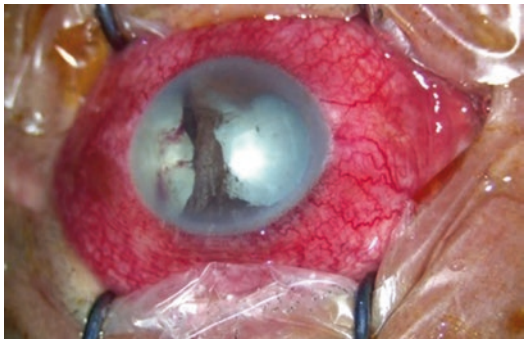

Fig. 14.26 Iridodialysis and cataract after blunt trauma

14.11.2 Occurs In

1. Intraocular infection due to trauma.
2. The disappearance of anterior chamber, delayed formation of anterior chamber, and wound leakage caused by trauma, lead to PAS and secondary angle closure glaucoma.
3. Traumatic lens rupture and residual cortex after lens extraction.
4. Chemical ocular trauma.

14.11.3 Clinical Manifestation

An acute eye pain, headache, blurred vision based on ocular trauma. Conjunctival congestion, corneal edema, shallow anterior chamber, iris bombé, PAS, intraocular hypertension.

14.11.4 Treatment

1. Medications: In accordance with uveitis.
2. Surgery: Treatment of primary causes.

14.12 Glaucoma Secondary to Intraocular Foreign Body

Intraocular foreign bodies, including iron, copper, zinc, and other metals, cause oxidation reaction in the eye, and damage of the angle of the eye leads to open-angle glaucoma (Fig. 14.30).

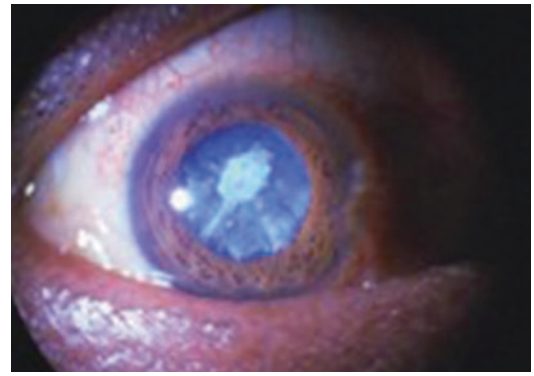

Fig. 14.27 About 2 months after blunt injury, lens opacity

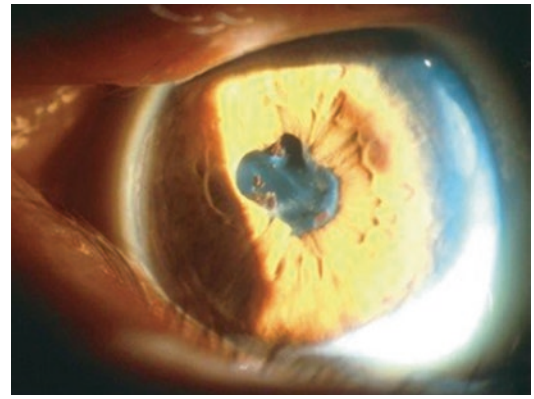

Fig. 14.28 About 1 month after blunt injury, PAS, iris nodules

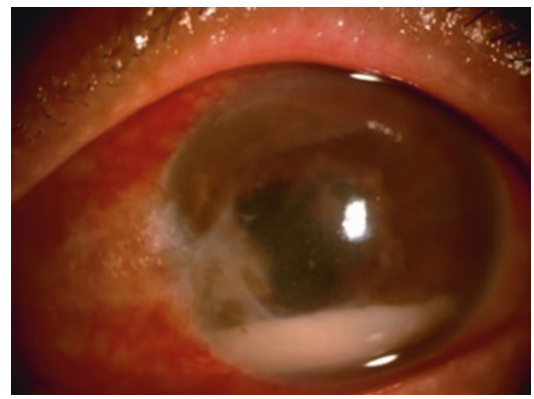

Fig. 14.29 About 3 days after sharp injury, hypopyon

Foreign body should be removed immediately if it damages the visual function.

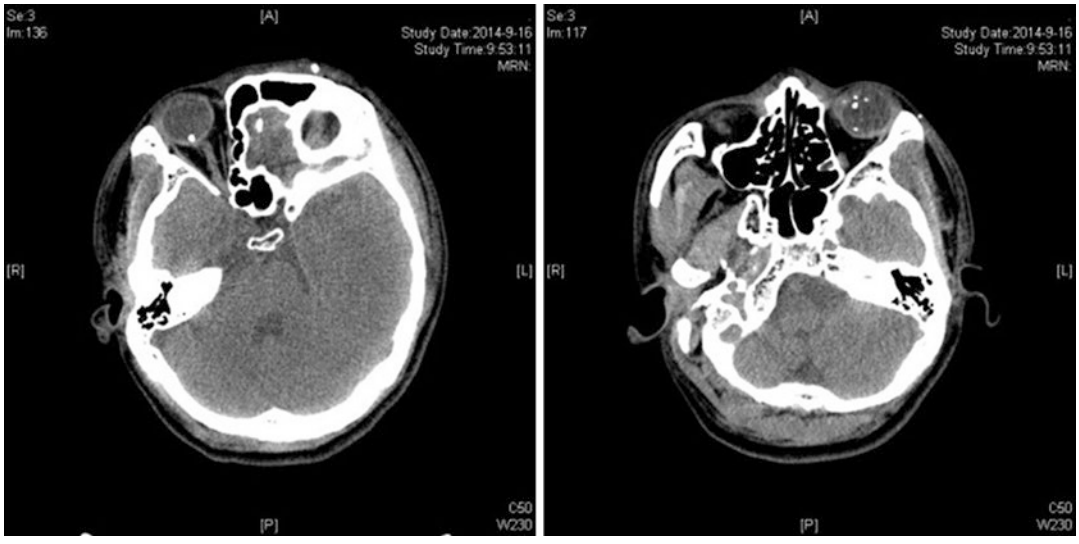

Fig. 14.30 Brain MRI shows: Hyper-reflective in the eyeball of both eyes

Traumatic Endophthalmitis

15

S. Natarajan, Astha Jain, and Sneha Makhija

Abstract

Post traumatic endophthalmitis is a serious complication of open globe injures. It carries a poorer prognosis compared to postoperative endophthalmitis due to the presence of more virulent organisms and delay in diagnosis and treatment. The diagnosis of endophthalmitis is clinical and is supported by the smear and culture of the vitreous and/or aqueous fluid. The threshold for vitrectomy in cases of posttraumatic endophthalmitis is low. Vitrectomy should be performed if there is no response to intravitreal antibiotics within 24 h or if associated with complications like retinal detachment or intraocular foreign body.

Keywords

Endophthalmitis · Intravitreal antibiotics
Vitrectomy

15.1 Introduction

Post traumatic endophthalmitis is a serious complication of open globe injures. The reported incidence of endophthalmitis following open globe injuries ranges from 0 to 12% [1–3]. Risk factors for endophthalmitis in cases of open globe injury include delayed primary globe repair, presence of intraocular foreign body (IOFB), large wound size, prolapse of ocular tissue, lens rupture which gives the microorganisms direct access to the vitreous cavity, and trauma in rural settings due to higher chances of soil contamination [4]. Post traumatic endophthalmitis are associated with poorer visual outcome when compared to postoperative endophthalmitis which could possibly be due to presence of more virulent organisms and delay in diagnosis and treatment [5].

15.2 Case Report

A 24-year-old male presented with history of injury to the right eye by a stone prior to presentation with complain of diminution of vison in the eye and whitish appearance of the front of the eye. He gave history of a primary corneal tear repair that was done locally 3 days after the injury. On examination, visual acuity of the right eye was perception of light and left eye was 6/6 on Snellen's chart. Extraocular movements of

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https://doi.org/10.1007/978-981-16-5340-7_15

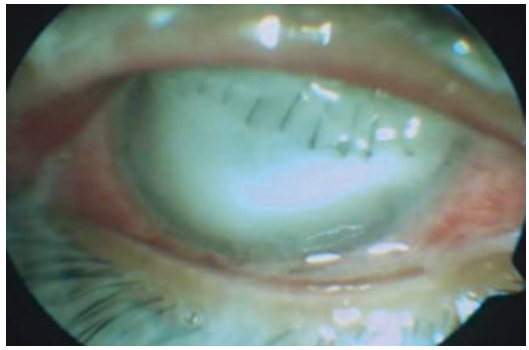

Fig. 15.1 Anterior segment photograph of a case of traumatic endophthalmitis showing total corneal abscess with corneal sutures in place, conjunctival congestion, and chemosis

both eyes were normal. Severe congestion and lid edema were present in the right eye. Slit lamp examination of anterior segment revealed a total corneal abscess, corneal sutures in place, and conjunctival congestion (Fig. 15.1). Lens, iris, and posterior segment details were not visible. The findings of the other eye were unremarkable. Right eye B-scan ultrasound sonography revealed multiple hyperechoic shadows in the vitreous cavity suggestive of vitreous exudates, lens echo was noted, and choroidal thickening was present suggestive of endophthalmitis. The patient was started on systemic and topical antibiotic and cycloplegic eye drops.

He underwent right eye penetrating keratoplasty with 23G pars plana vitrectomy with intravitreal antibiotic injections. Local anaesthesia in the form of peribulbar anaesthesia was given in the right eye. A Flieringa ring was sutured in place to stabilize the eye. Corneal button was excised, lens was expulsed, and a temporary keratoprosthesis was sutured in place for better visualization of the posterior segment (Fig. 15.2). 3 23 Gauge ports were put and vitrectomy was done to remove the exudative material from the posterior chamber. The ports and temporary KPRO were removed. A large full thickness graft was sutured using 10–0 nylon sutures. Intravitreal vancomycin, ceftazidime, and voriconazole were injected and the eye was patched. The corneal button and vitreous tap were sent for culture and sensitivity.

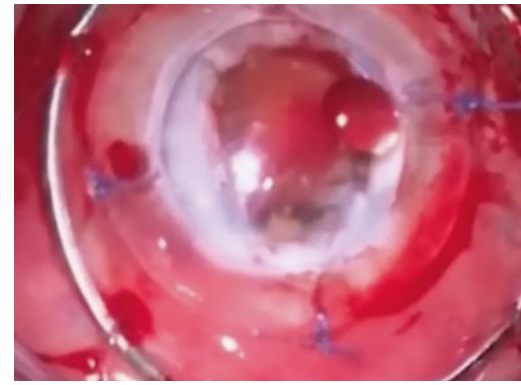

Fig. 15.2 Intraoperative photograph showing use of a Flieringa ring for stabilization and support and a temporary keratoprosthesis for better posterior segment visualization

Post operatively an edematous corneal graft was noted. The edema precluded a definite posterior segment evaluation. However, the retina appeared attached. The KOH smear of the samples sent to microbiology department showed fungal filaments so the patient was started on topical natamycin and voriconazole eye drops and itraconazole tablet. In 48 h, the vitreous tap showed no growth on culture; however, the corneal button showed growth of some black coloured fungus. He was started on fortified amphotericin B eye drops and was given intravitreal amphotericin B injection for the same. On 2 weeks follow-up, perception of light was present in the right eye. On anterior the edema in the corneal graft had decreased and corneal sutures were in place. No exudates or hyphema was noted in the anterior chamber. However, vascularization was noted in the angles. Posterior segment showed retina attached. The patient was started on topical steroid drops to prevent graft rejection.

15.3 Microbiology

Identification of the causative organism forms an important part of the management. Positive culture depends on the method of sample collection, previous medical therapy, and the method of inoculation and culture medium used. In a large

retrospective series of post traumatic endophthalmitis, only 38.1% cases were found to be culture positive [6]. Smear test with Gram and Giemsa staining helps in identifying the causative organism even when cultures are negative. Mixed bacterial infection is commonly seen. Both Gram positive and Gram negative organisms can be seen. Coagulase negative Staphylococcus species followed by Gram negative bacilli like pseudomonas are the most commonly found organisms. IOFBs are found to be positively associated with *S. epidermidis* [7]. Bacillus infections are fulminant and can be associated with a ring abscess and are found particularly in the setting of IOFB. Fungal infections are less common and usually seen in cases of trauma with vegetative matter or open globe injuries with soil contamination. Aspergillus is the most common species followed by yeast like organisms [8].

15.4 Antibiotic Susceptibility

Intravitreal vancomycin and ceftazidime have been commonly used to provide a broad spectrum coverage of both Gram positive and Gram negative organisms. However increasing resistance to antibiotics is being reported. Common use of topical fluoroquinolone drops has led to increased resistance among conjunctival flora specially coagulase negative staphylococcus [9].

While a 20 year retrospective data between 1990 and 2009 showed that both Gram positive and Gram negative organisms were highly susceptible to ceftazidime [6], a recent 15 year review of post traumatic endophthalmitis conducted between 2004 and 2019 found the susceptibility of Gram negative bacilli to ceftazidime decrease over time [10]. According to this study, Gram positive organisms showed greatest susceptibility to vancomycin followed by fluoroquinolones. Gram negative organisms were most susceptible to levofloxacin followed by meropenem. Susceptibility of both Gram positive and negative organisms to trimethoprim-sulfamethoxazole (TMP-SMX) was shown to increase over time. Another recent study from India found increasing resistance of pseudomonas to ceftazidime, fluoroquinolones, and amikacin [10]. Intravitreal imipenem 50 microgram/0.1 ml has been used in cases of ceftazidime resistance Gram negative endophthalmitis [11].

15.5 Diagnosis

The diagnosis of endophthalmitis is clinical and is supported by the smear and culture of the vitreous and/or aqueous fluid. Ocular signs of post traumatic endophthalmitis are similar to that of any other infectious endophthalmitis and include anterior chamber cells and flare, hypopyon and vitreous exudates. Aqueous tap can be obtained through a paracentesis using a 25-27G needle. 0.1 ml of aqueous is sufficient for microbiological examination. Vitreous sample can be obtained by aspiration through a needle or by vitreous biopsy as a part of the therapeutic vitrectomy. 0.2–0.3 ml of sample can be collected. Sometimes a "dry tap" may be obtained because of difficulty in aspirating the gel. It is important to collect an undiluted sample of the vitreous at the start of the vitrectomy to be sent for culture. Vitreous diluted by the irrigating solution can also be collected as "washings" and sent for culture.

15.6 Treatment

15.6.1 Prophylaxis

Oral and intravenous antibiotics have been used in cases of open globe injuries to prevent the onset of endophthalmitis. Oral ciprofloxacin has been found to be as effective as intravenous cefazolin or gentamicin [12]. Prophylaxis with intravenous cefazolin and oral ciprofloxacin or oral ciprofloxacin and oral cefuroxime for 3 days from admission was found to be equally effective in one of the studies [13]. Use of intracameral or intravitreal antibiotic reduces the risk of endophthalmitis in open globe injuries [14]. Intraocular gentamicin and clindamycin were found to be effective in preventing post-traumatic bacterial endophthalmitis in cases with retained IOFB [15]. Broad spectrum intra-

vitreal antibiotics like vancomycin and ceftazidime provide coverage against both Gram negative and Gram positive organisms including methicillin resistant *Staphylococcus aureus* [16] and have shown to reduce the risk of posttraumatic endophthalmitis [17]. Definitive antibiotics can be started after getting the culture and sensitivity reports.

15.6.2 Vitrectomy

The threshold for vitrectomy in cases of posttraumatic endophthalmitis should to be low. In cases with visual acuity better than hand motion with adequate visualization of retina, intravitreal antibiotics can be tried. However, if there is no improvement within 24 h, or if associated with complication such as retinal detachment, immediate vitrectomy should be performed.

23G transconjunctival vitrectomy can be performed. A vitreous biopsy should be performed before starting the infusion cannula to collect an undiluted ample of vitreous. An anterior chamber infusion line can be used in cases where there is no visibility of the posterior segment. Corneal epithelial scraping may be required in cases with corneal edema. Exudates and fibrin are removed from the anterior chamber. It can be done using microforceps or by doing a limbal vitrectomy. Care must be taken to avoid damage to iris tissue. Synechiae may have to be released to allow pupillary dilatation. In cases of traumatic cataract, lensectomy may have to be performed. A thorough core vitrectomy followed by detachment of posterior vitreous and shaving of vitreous skirt should be performed. Intravitreal antibiotics can be used depending on the vitreous culture report if available or a broad spectrum antibiotic covering both Gram positive and Gram negative organisms such as vancomycin with ceftazidime can be used. Intravitreal dexamethasone can be used if fungal infection is not suspected. Although intravitreal steroids help in early reduction of inflammation, it has no effect on the final visual outcome [18]. Silicone oil is found to have bactericidal and fungistatic properties [19]. Therefore, silicone oil tamponade is the preferred tamponading agent especially in severe cases, cases with retinal breaks or large areas of retinal necrosis, or cases associated with retinal detachment. Endoscopic vitrectomy can be used in cases of poor posterior segment visualization due to anterior segment opacification [20, 21].

15.6.3 Personal Experience

Risk factors for endophthalmitis such as delayed wound closure, contaminated wound, presence of IOFB should be kept in mind while dealing with a case of open globe injury. With slightest of suspicion, intravitreal antibiotics should be given at the time of primary repair. In cases of confirmed endophthalmitis, vitrectomy should be done within 24 h of diagnosis.

15.6.4 Specific Challenge

Presence of large corneal tears or corneal abscess may make visualization of the posterior segment difficult. Hence one may have to do an open sky vitrectomy or use a temporary keratoprosthesis or do an endoscopic vitrectomy.

References

1. Colyer MH, Weber ED, Weichel ED, Dick JS, Bower KS, Ward TP, et al. Delayed intraocular foreign body removal without endophthalmitis during operations Iraqi freedom and enduring freedom. Ophthalmology. 2007;114(8):1439–47.
2. Andreoli CM, Andreoli MT, Kloek CE, Ahuero AE, Vawas D, Durand ML. Low rate of endophthalmitis in a large series of open globe injuries. Am J Ophthalmol. 2009;147(4):601–8.
3. 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.
4. Dehghani AR, Rezaei L, Salam H, Mohammadi Z, Mahboubi M. Post traumatic endophthalmitis: incidence and risk factors. Glob J Health Sci. 2014;6(6):68–72. Published 2014 Jun 30. https://doi.org/10.5539/gjhs.v6n6p68.

5. Ahmed Y, Schimel A, Pathengay A, et al. Endophthalmitis following open-globe injuries. Eye. 2012;26:212–7. https://doi.org/10.1038/eye.2011.313.
6. 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. Published 2014 Mar 25. https://doi.org/10.1186/1471-2415-14-34.
7. Al-Omran AM, Abboud EB, Abu El-Asrar AM. Microbiologic spectrum and visual outcome of posttraumatic endophthalmitis. Retina. 2007;27(2):236–42. https://doi.org/10.1097/01.iae.0000225072.68265.ee.
8. Ramakrishnan R, Bharathi MJ, Shivkumar C, Mittal S, Meenakshi R, Khadeer MA, Avasthi A. Microbiological profile of culture-proven cases of exogenous and endogenous endophthalmitis: a 10-year retrospective study. Eye (Lond). 2009;23(4):945–56. https://doi.org/10.1038/eye.2008.197.
9. Milder E, Vander J, Shah C, Garg S. Changes in antibiotic resistance patterns of conjunctival flora due to repeated use of topical antibiotics after intravitreal injection. Ophthalmology. 2012;119(7):1420–4. https://doi.org/10.1016/j.ophtha.2012.01.016.
10. Liu C, Ji J, Wang Z, Chen H, Cao W, Sun X. Microbiological isolates and antibiotic susceptibilities in cases of posttraumatic Endophthalmitis: a 15-year review. J Ophthalmol. 2020;2020:5053923. Published 2020 Apr 29. https://doi.org/10.1155/2020/5053923.
11. Dave VP, Pathengay A, Nishant K, et al. Clinical presentations, risk factors and outcomes of ceftazidime-resistant gram-negative endophthalmitis. Clin Exp Ophthalmol. 2017;45(3):254–60. https://doi.org/10.1111/ceo.12833.
12. Dehghani A, Rafiemanzelat AM, Ghaderi K, Pourazizi M, Feizi A. Post-traumatic endophthalmitis prophylaxis with oral ciprofloxacin in comparison to intravenous cephazolin/gentamicin. J Res Med Sci. 2018;23:98. Published 2018 Nov 28. https://doi.org/10.4103/jrms.JRMS_384_18.
13. Du Toit N, Mustak S, Cook C. Randomised controlled trial of prophylactic antibiotic treatment for the prevention of endophthalmitis after open globe injury at Groote Schuur Hospital. Br J Ophthalmol. 2017;101:862–7.
14. Thevi T, Abas AL. Role of intravitreal/intracameral antibiotics to prevent traumatic endophthalmitis – meta-analysis. Indian J Ophthalmol. 2017;65:920–5.
15. Soheilian M, Rafati N, Mohebbi MR, 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. https://doi.org/10.1001/archopht.125.4.460.
16. Meredith TA. Antimicrobial pharmacokinetics in endophthalmitis treatment: studies of ceftazidime. Trans Am Ophthalmol Soc. 1993;91:653–99.
17. 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.
18. Das T, Jalali S, Gothwal V, et al. Intravitreal dexamethasone in exogenous bacterial endophthalmitis: result of a prospective randomized study. Br J Ophthalmol. 1999;83:1050–5.
19. Dave VP, Joseph J, Jayabhasker P, et al. Does ophthalmic-grade silicone oil possess antimicrobial properties? J Ophthalmic Inflamm Infect. 2019;9:20. https://doi.org/10.1186/s12348-019-0187-6.
20. Pan Q, Liu Y, Wang R, et al. Treatment of *Bacillus cereus* endophthalmitis with endoscopy-assisted vitrectomy. Medicine. 2017;96(50):e8701. https://doi.org/10.1097/md.0000000000008701.
21. Wong SC, Lee TC, Heier JS. 23-Gauge endoscopic vitrectomy. Dev Ophthalmol. 2014;54:108–19.

Permanent Silicone Oil Tamponade

16

Xixuan Ke and Haoyu Chen

Abstract

Although long-term tamponade of silicone oil may have several complications, permanent silicone oil tamponade may be needed in some eyes with severe ocular trauma. Removal of silicone oil in these eyes would lead to phthisis bulbi. Proliferative vitreoretinopathy and persistent hypotony are two major reasons requiring permanent silicone oil tamponade in severe ocular trauma. The complications of silicone oil tamponade include migration of silicone oil to the anterior chamber and subretinal space, band keratopathy, silicone oil emulsification, secondary glaucoma, and optic nerve atrophy. Several strategies should be adopted during silicone oil tamponade to prevent the complications. The patients should be followed up and potential complications should be managed. This chapter discusses the definition, pathogenesis, clinical manifestations, examination, and management of permanent silicone oil tamponade.

Keywords

Permanent silicone oil tamponade
Proliferative vitreoretinopathy · Persistent hypotony · Complication

16.1 Introduction

Severe ocular trauma involving the posterior segment usually complicates with retinal detachment and proliferative vitreoretinopathy (PVR). There may also be damage to the choroid and ciliary body, which aggravating posterior and anterior vitreoretinal proliferation. Furthermore, the damage of the ciliary body may lead to reduced secretion of aqueous humor and hypotony.

Vitrectomy can remove vitreous hemorrhage and proliferation, reattach the retina, and seal retinal break. Silicone oil tamponade is usually required in addition to vitrectomy for these cases. Although the advance of vitreoretinal surgery has significantly improved the outcome of complicated ocular trauma, there is still a significant proportion of patients requiring permanent silicone oil tamponade, which is also called silicone oil-dependent eye.

Long-term silicone oil tamponade may have several complications, such as migration of silicone oil to the anterior chamber, subretinal space, band keratopathy, silicone oil emul-

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sification, secondary glaucoma, and optic nerve atrophy. Therefore, the prevention and management of these complications are important.

16.2 Definition

There was no well-established definition for permanent silicone oil tamponade in the literature. There were some articles reporting the long-term results of silicone oil tamponade, which were more than 12 months [1–3]. But permanent silicone oil tamponade means that the silicone oil is left inside the eye without a plan of removal. Removal of silicone oil in these eyes would lead to phthisis bulbi, evisceration, or enucleation. Silicone oil-dependent eye is defined as one of the following: 1. The eyes whose retina cannot be reattached even when silicone oil tamponade and need numbers of reoperations; 2. The eyes whose retina is reattached, but the intraocular pressure is lower than five mmHg last for more than three months [4].

16.3 Pathogenesis and Mechanisms

1. PVR is the leading cause of the retinal re-detachment and requirement of permanent silicone oil tamponade.

Severe ocular trauma involving the posterior segment usually complicates with retinal detachment and PVR. Several preoperative independent risk factors have been found were associated with PVR post-trauma, such as delayed vitrectomy for more than two weeks from injury, severe vitreous hemorrhage, total retinal detachment, the types of injuries (open globe injuries have a higher risk than closed globe injuries) [5]. In Blackford's study, injuries caused by an IOFB, macular hemorrhage, or unsuccessful repair were significant risk factors for PVR development [6]. In Han's report, Zone 3 Injury, Zone 3 retinal laceration, massive vitreous hemorrhage, retinal disorder, the timing of vitrectomy, and type of injury were identified as the risk factors for PVR [7].

The mechanism of PVR in ocular injury is a proliferative reaction to the injury. The injuries induce inflammation, activate cellular proliferation, and form fibrovascular membranes, which would heal the wound caused by the injury. However, the inflammation after an ocular injury is usually excessive and interferes with physiologic wound healing. Therefore, the severer the injury, and severer the PVR. The zone 3 injury and other injuries involving the retina, RPE, and choroid would have more severe PVR because the healing process would take place on these tissues and induce proliferative membrane on these tissues. Furthermore, the choroid is rich with blood vessels, and more inflammatory cells will be recruited when the choroid is injured.

Besides the ocular injury condition, surgical intervention is also an important factor of PVR in ocular injury. As mentioned above, delayed surgical intervention is a risk factor for PVR. Because the cellular proliferative reaction takes time, early intervention, removal of the intraocular hemorrhage, and vitreous, inflammatory cells can prevent PVR or reduce its severity. Furthermore, surgical procedures can also affect postoperative PVR. Retinotomy and excessive retinal cryotherapy could lead to extensive area pigment epithelium exposure and promote retinal pigment epithelial cells' diffusion into the vitreous cavity. Weller et al. [8] found that the quality of the primary surgical handling of retinal detachment is the most important prognostic factor for subsequent PVR development. Complete removal of vitreous and relieve retinal traction is the key to reducing the occurrence of PVR.

The proliferation and contraction of the fibral tissue not only take place at the vitreoretinal interface but also inside and beneath the retina. Contraction of these proliferative membranes mechanically tracts the neurosensory retina from RPE. Furthermore, traction may also cause new retinal breaks, which is also a significant cause of retinal re-detachment.

In some cases, PVR is too severe that the traction cannot be removed entirely by vitreoretinal surgery. The injury caused by the operation

induced further proliferation and led to the recurrence of retinal detachment. In these cases, several procedures have been tried to reattach the retina, but there may be early or late failure or retina re-detachment after silicone oil removal. These cases require permanent tamponade of silicone oil.

2. Hypotony is another important cause of permanent silicone oil tamponade.

The intraocular pressure is maintained by the balance of aqueous humor secretion and absorption. Severe and persistent hypotony may lead to phthisis bulbi and silicone oil is required to maintain the intraocular pressure. There may be several mechanisms of hypotony in ocular injury.

First, in severe ocular injury, the ciliary body's function of aqueous humor secretion may be severely impaired, caused by direct and indirect mechanical damage, long-term detachment of the ciliary body, and anterior PVR. Zone 2 injury would directly damage the ciliary body. The indirect mechanical impact would also cause damage to the ciliary epithelium.

Second, detachment of the ciliary body and choroid from the sclera would increase the outflow of aqueous humor. Ciliary detachment may be a result of mechanical contusion, cyclodialysis, and traction of anterior PVR. Mechanical compact can result in the ciliary body and choroidal detachment by itself or cyclodialysis cleft. Cyclodialysis results from the separation of the longitudinal ciliary muscle fibers from the scleral spur, which creates an abnormal pathway for aqueous humor drainage that may lead to ocular hypotony [9]. Anterior PVR is an important cause of hypotony. There may develop some fibral membrane at the ciliary body and cause traction force on the ciliary body. The traction prevents the reattachment of the ciliary body [10]. Furthermore, hypotony, ciliary and choroidal detachment, and intraocular inflammation can aggregate each other and develop a vicious circle.

Third, in some severe cases, the scleral wound may not be closed completely. The leakage from the inadvertent fistula would also cause hypotony [11, 12].

Persistent hypotony may lead to choroidal fold, hypotonic maculopathy, intraocular inflammation, and corneal edema. The eyeball with hypotony looks smaller compared to the contralateral eye.

For persistent hypotonic eyes, it is important to identify the cause of hypotony and used medicine or surgery to treat the causes, such as corticosteroid, cycloplegia, repairing cyclodialysis cleft and remove anterior PVR membrane. However, in severe cases, silicone oil is required to maintain the intraocular pressure even after several procedures have been tried.

16.4 Symptoms and Signs

1. Anterior segment

   Silicone oil may migrate to the anterior chamber and cause corneal endothelial damage, including band keratopathy, corneal decompensation, and degenerative changes. Patients often present with foreign body sensation, irritation, and tearing. There may be chronic anterior uveitis. Posterior synechia, seclusion of the pupil, and complicated cataract are common clinical features in permanent silicone oil tamponade eyes. Silicone oil emulsification is another common feature of long-term silicone oil tamponade. It usually presents as some small white droplet at the superior anterior chamber, as an inverse pseudohypopyon. There may be secondary glaucoma due to the blockage of trabecular meshwork by the emulsified silicone oil.
2. Posterior segment

   The commonly used silicone oil has a lower density than water, and its floating effect has better oppression on posterior and superior breaks. Therefore, most retinal detachment is located at inferior in oil tamponade eyes, with mild bulge and slowly progressive. Few cases of retinal detachment happened at the superior or posterior pole. The reopening of the original retinal tear, the formation of new tears, and PVR are the leading causes of retinal detachment in silicone oil tamponade eyes. Fundus examination can reveal small white crystal

particles formed by the emulsification of silicone oil on the surface of the retina, the oil–liquid interface when the inferior vitreous cavity is insufficiently tamponade with silicone oil, proliferative membrane and strand at the base of vitreous, annular contraction and tract the retina forward, a gray-white hyperplastic membrane forms on the surface of the retina, rigid retina, and even total retinal detachment.

16.5 Examinations

Wide-field scanning laser ophthalmoscopy can demonstrate the posterior segment if the optical media is clear. It has the advantage of viewing the peripheral retina even when the pupil is small. Optical coherence tomography (OCT) can show the high-resolution cross-sectional structure of the retina. It may demonstrate preretinal proliferation, retinal detachment, subretinal membrane, and other disorders, especially those located at the macula.

Although ultrasound B scan images can be disturbed by silicone oil, it still can show whether there is retinal detachment, choroidal detachment, or PVR. Especially, it is useful in the presence of severe refractive medium opacity or tiny pupil. Ultrasound biomicroscope (UBM) can be used to examine the anterior segment and peripheral fundus to know the severity of anterior PVR and/or ciliary body detachment.

16.6 Management of Complication in Permanent Silicone Oil Tamponade

Since the silicone oil tamponade is required permanently, there may be various complications. Management of these complications would help to improve the symptoms and even visual acuity.

1. Anterior chamber migration of silicone oil

   Silicone oil is lighter than water. Therefore, silicone oil may migrate to the anterior chamber when the patients sleep in the supine position. It usually happens in aphakic eyes with a ruptured or absent capsule. In these eyes, the vitreous cavity is connected directly with the anterior chamber. If there is an iris, a six o'clock peripheral iridectomy should be performed, allowing the aqueous humor circuit to the anterior chamber via the iridectomy and pushing the silicone oil back with a prone position. However, in the cases with iris defect, sutures with a grid pattern may provide a barrier for silicone oil [13].

   Silicone oil migration to the anterior chamber may also happen in phakic eyes, pseudophakic eyes, or aphakic eyes with an intact capsule because there is zonular dialysis. For phakic and pseudophakic eyes, an operation to remove the silicone oil from the anterior chamber is needed. Hyaluronate sodium can help the removal safely. While for aphakic eyes with an intact capsule, laser capsulectomy and six o'clock iridectomy would be able to push the silicone oil back.
2. Silicone oil emulsification

   Emulsification is common in long-term silicone oil tamponade. It does not need management if it is mild, and the intraocular pressure is normal. However, in severe cases, the patients may feel vision decrease in a supine position when the emulsified silicone oil droplet moves to the central anterior chamber and block the visual axis. The patients may have secondary glaucoma. In these cases, removal of silicone oil and reinstallation of new silicone oil are needed.
3. Retinal re-detachment and subretinal migration of silicone oil

Different operations would be chosen according to the condition of retinal detachment. If there is a shallow detachment located in the peripheral retina without the involvement of the macula, laser photocoagulation can be applied to the posterior edge of the detachment zone. If there is macula detachment, reoperation can be performed. Anterior or posterior PVR is often seen in retinal detachment in oil tamponade eyes. Scleral encircling buckle could relieve the rigid

retina and traction. After removing oil, carefully check the vitreous base and pars plana by compression scleral and clear residue vitreous. The preretinal or subretinal proliferative membrane must be peeled. If all the above procedures still cannot reattach the retina, retinectomy should be considered. Hocaoglu and associates [14] report the outcomes of patients with retinal detachment following penetrating injury underwent combined 360-degree retinotomy, anterior flap retinectomy, and radial retinotomy for the management of advanced proliferative vitreoretinopathy, and the complete retinal reattachment rate was 75% after 51.5 months follow-up.

4. Hypotony

Some patients may still have hypotony even in the presence of silicone oil tamponade. It is usually due to anterior PVR, cyclodialysis cleft, posterior PVR and retinal re-detachment, or scleral fistula. Identify the cause of ocular hypotony and treat the cause can improve this condition. It was reported that endoscopy-assisted vitrectomy could dissect the ciliary body membrane in anterior PVR and improve hypotony [15]. Cyclodialysis cleft can be repaired using laser, direct cyclopexy, scleral buckling, or sulcus capsular tension ring or three-piece intraocular lens [9].

16.7 Case of Retinal Detachment in Silicone Oil Tamponade Eye

A 38-year-old man presented to the ophthalmology emergency department with pain and redness in his left eye after being wounded by an iron block. The best-corrected visual acuity was hand motion, and the intraocular pressure was 7 mmHg in his left eye. On slit-lamp biomicroscope examination of the left eye, there was nasal conjunctiva hyperemia and massive dark red subconjunctival hemorrhage. The cornea was transparent. There was Tyndall (+), cell (+) in the anterior chamber. There is posterior capsule opacity at the lens. The vitreous hemorrhage was +++, and the retina cannot be visualized (Fig. 16.1a). His right eye uncorrected visual acuity was 1.0, and intraocular pressure was 12 mmHg. Computed tomography at orbit indicated a metal foreign body retained in the vitreous cavity of the left eye. The patient was diagnosed with open globe injury with intraocular foreign body, traumatic cataract, and vitreous hemorrhage in the left eye based on the history and examinations.

After exclusion of intracranial injury, an emergent operation was performed: corneal laceration sutured, lens aspiration, vitrectomy to remove vitreous hemorrhage, removal of the intraocular foreign body through the limbal incision, the retinal wound was photocoagulation by laser and vitreous tamponade with silicone oil after gas-liquid exchange. Prone positioning was instructed, and topical steroid and antibiotic drops were used during postoperative follow-up. Ultra-wild-angle fundus photography shows retinal attached, minor preretinal hemorrhage at the posterior pole, superonasal gray-white lesion surrounded by laser spot when the patient was discharged three days postoperation (Fig. 16.1b). Five months later, the patient corrected visual acuity improved to 0.5, and intraocular pressure was 11 mmHg. Slit-lamp examination of the left eye revealed transparent cornea and quiet anterior chamber with aphakia, dilated funduscopic revealed attached retina, a small piece of white proliferative membrane locates at the inferior equatorial retina, and there were pigmented laser spots at the superonasal retina (Fig. 16.1c). Silicone oil was removed, proliferative membrane peeled, and the intraocular lens was fixed in the ciliary sulcus.

Two weeks later, the patient presented with sudden visual acuity loss in the left eye. The best-corrected visual acuity was HM in the left eye. Anterior segment examination was unremarkable. Fundus examination showed almost total retinal detachment, and the inferior retina was elevated highly (Fig. 16.1d). The patient underwent the third operation. During the operation, a new retinal break was found at the inferior retina, and then the retina was flattened with perfluorocarbon liquids. Laser photocoagulation was applied to the retinal break, and a direct

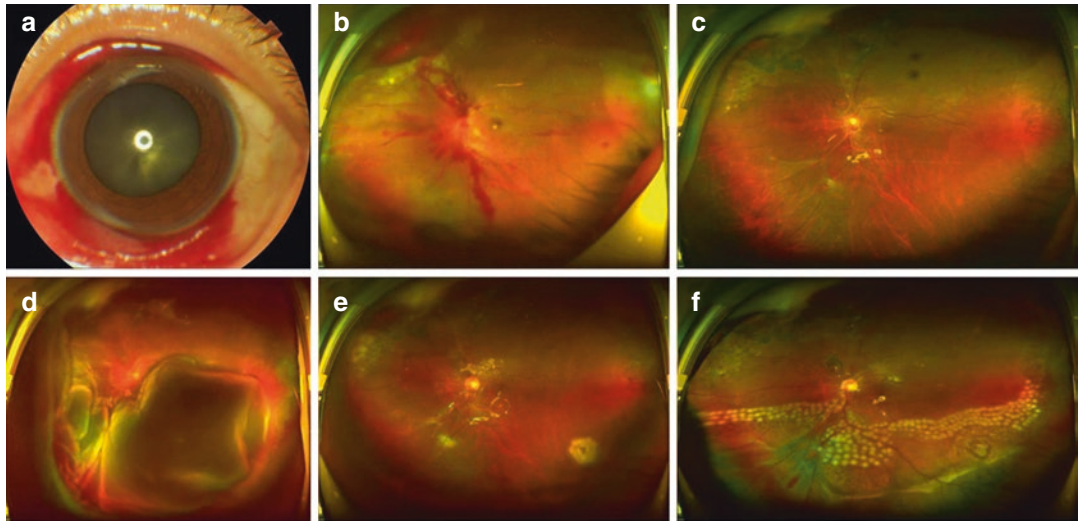

Fig. 16.1 (**a**) Photograph of the anterior segment after perforating injury with an intraocular body; (**b**) Ultra-wide-angle photograph seven days after the first surgery tamponade with silicone oil shows attached retina with mild preretinal hemorrhage; (**c**) The retina was flat before removal of silicone oil; (**d**) Retinal detachment two weeks after the silicone oil removal; (**e**) The retina was reattached after the second time silicone oil tamponade; (**f**) Complementary laser photocoagulation applied to prevent inferior retinal detachment from progressing

perfluorocarbon-silicone oil exchange was conducted (Fig. 16.1e). Topical eye drops and prone positioning were required. Three months later, the nasal-inferior retinal detached with preretinal and subretinal membrane proliferation. Complementary laser photocoagulation was applied to prevent further detachment of the retina. The retina remained stable six months after the photocoagulation was performed, and the best-corrected visual acuity was 0.3 (Fig. 16.1f).

16.8 Personal Experience

Silicone oil tamponade has a better effect on the superior retina but less effect on the inferior retina. And there is usually some space filled with aqueous humor but not silicone oil at the inferior vitreous cavity. And this space has a high concentration of inflammatory factors and cells. Therefore, the inferior retina is at higher risk of PVR. In this case's management, a timely intervention with laser photocoagulation would prevent the progression of inferior retinal detachment to the fovea and keep good visual acuity in the presence of silicone oil permanent tamponade.

16.9 Specific Challenges

The challenges for permanent silicone oil eyes are silicone oil-associated complications, such as keratopathy, glaucoma, and emulsification. Although several methods can be tried to manage these complications, some complications are intractable, and the patients may need to receive many operations.

Therefore, seeking or inventing better vitreous substitutes has been conducted decades ago. Foldable capsular vitreous body (FCVB) is a promising substitute. It is a capsule implanted in the vitreous cavity, and the silicone oil is injected into the capsule. Therefore, the silicone oil would not migrate to the anterior chamber and damage the corneal endothelium. There are some publications reporting the results of FCVB in the management of silicone oil-dependent eyes [16, 17]. But more studies with a larger sample size and longer time follow-up are needed to investigate the effectiveness and safety of FCVB.

FCVB can avoid silicone oil-related complications, but it cannot solve persistent hypotony

caused by ciliary body dysfunction. There is an insufficient aqueous secretion of the ciliary body caused by direct trauma or anterior PVR. Even after silicone oil tamponade or artificial vitreous implantation, some eyeballs would gradually develop into phthisis bulbi.

References

1. Abu-Yaghi NE, Abu Gharbieh YA, Al-Amer AM, AlRyalat SAS, Nawaiseh MB, Darweesh MJ, Alkukhun LR, Abed AM, Saleh OA, Ababneh OH. Characteristics, fates and complications of long-term silicone oil tamponade after pars plana vitrectomy. BMC Ophthalmol. 2020;20(1):336.
2. Morphis G, Irigoyen C, Eleuteri A, Stappler T, Pearce I, Heimann H. Retrospective review of 50 eyes with long-term silicone oil tamponade for more than 12 months. Graefes Arch Clin Exp Ophthalmol. 2012;250(5):645–52.
3. Li M, Tang J, Jia Z, Yao Y, Jin E, Wang Z, Hu J, Sun G, Yin H, Liang J, et al. Long-term follow-up of primary silicone oil tamponade for retinal detachment secondary to macular hole in highly myopic eyes: a prognostic factor analysis. Eye (Lond). 2020;35(2):625–31.
4. Dong F, Dai R, Jia Y. [Clinical features of silicone oil dependent eyes]. [Zhonghua yan ke za zhi]. Chin J Ophthalmol. 2008;44(11):998–1001.
5. Feng K, Hu Y, Wang C, Shen L, Pang X, Jiang Y, Nie H, Wang Z, Ma Z. Risk factors, anatomical, and visual outcomes of injured eyes with proliferative vitreoretinopathy: eye injury vitrectomy study. Retina (Philadelphia, Pa). 2013;33(8):1512–8.
6. Blackford BG, Justin GA, Baker KM, Brooks DI, Wang HH, Ryan DS, Weichel ED, Colyer MH. Proliferative vitreoretinopathy after combat ocular trauma in operation Iraqi freedom and operation enduring freedom: 2001-2011. Ophthalmic Surg Lasers Imaging Retina. 2020;51(10):556–63.
7. Han L, Jia J, Fan Y, Yang L, Yue Z, Zhang W, Liu F, Kang H, Huo T, Han S, et al. The vitrectomy timing individualization system for ocular trauma (VTISOT). Sci Rep. 2019;9(1):12612.
8. Weller M, Wiedemann P, Heimann K. Proliferative vitreoretinopathy--is it anything more than wound healing at the wrong place? Int Ophthalmol. 1990;14(2):105–17.
9. Gonzalez-Martin-Moro J, Contreras-Martin I, Munoz-Negrete FJ, Gomez-Sanz F, Zarallo-Gallardo J. Cyclodialysis: an update. Int Ophthalmol. 2017;37(2):441–57.
10. Lewis H, Verdaguer JI. Surgical treatment for chronic hypotony and anterior proliferative vitreoretinopathy. Am J Ophthalmol. 1996;122(2):228–35.
11. Peiris TJ, Khouri AS. Cat-scratch penetrating globe injury with inadvertent fistula and hypotony. Can J Ophthalmol. 2017;52(1):e5–7.
12. Zhao R, Lin L, Zeng J, Duan F, Yang Y, Su SB, Lou B, Lin X. Development of a rabbit model of persistent hypotony without ciliary body injury. Exp Eye Res. 2020;190:107858.
13. Yuksel K, Pekel G, Alagoz N, Alagoz C, Baz O, Yazc AT. Silicone oil barrier sutures in Aphakic eyes with Iris defects. Retina. 2016;36(6):1222–6.
14. Hocaoglu M, Karacorlu M, Muslubas I, Ozdemir H, Arf S, Uysal O. Peripheral 360 degree retinotomy, anterior flap retinectomy, and radial retinotomy in the management of complex retinal detachment. Am J Ophthalmol. 2016;163:115–121.e111.
15. Lee GD, Goldberg RA, Heier JS. Endoscopy-assisted vitrectomy and membrane dissection of anterior proliferative vitreoretinopathy for chronic hypotony after previous retinal detachment repair. Retina. 2016;36(6):1058–63.
16. Zhang X, Tian X, Zhang B, Guo L, Li X, Jia Y. Study on the effectiveness and safety of foldable capsular vitreous body implantation. BMC Ophthalmol. 2019;19(1):260.
17. Lin X, Wang Z, Jiang Z, Long C, Liu Y, Wang P, Jin C, Yi C, Gao Q. Preliminary efficacy and safety of a silicone oil-filled foldable capsular vitreous body in the treatment of severe retinal detachment. Retina (Philadelphia, Pa). 2012;32(4):729–41.

Traumatic Optic Neuropathy

17

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Abstract

Traumatic optic neuropathy is a rare pathology caused by an acute injury of the optic nerve secondary to trauma. The diagnosis is mainly clinically based on history and ophthalmic signs. Images sometimes show a direct lesion but most frequently the mechanism is indirect and affects the intracanalicular portion of the nerve. Incidence is higher in young males. Different treatments have been proposed including corticosteroids and surgery but the data in the literature to date has not shown any treatment to be superior to observation. Moreover, 40–60% of TON have spontaneous recovery, excluding severe cases.

Keywords

Trauma · Direct, indirect mechanism
Multifactorial · Spontaneous recovery

17.1 Introduction

Traumatic optic neuropathy (TON) refers to an acute injury of the optic nerve secondary to trauma. The diagnosis of TON is made clinically, based on history and ophthalmic signs. Like other optic neuropathies, patients with TON may have decreased central visual acuity (VA), decreased color vision, an afferent pupillary defect, and/or visual field (VF) defects.

17.2 Definition

TON is an optic nerve (ON) injury as a result of trauma, which results in total or partial loss of function, which may be transient or permanent. Trauma can lead to direct or indirect aggression to the nerve, the latter being the most common.

It may be due **to severe, moderate, or even mild head trauma**. The gravity varies according to different series; **43 to 56% have a severe visual loss,** but we must keep in mind that mild and/or spontaneously improved cases are often not reported. A vast majority are unilateral, but it can be bilateral if it involved major trauma. Since it involves head trauma, management should be carried out by a multidisciplinary team that includes emergency room doctors, head and neck surgeons, neurosurgeons, and ophthalmologists. Ophthalmological assessment should be carried out at the first possible opportunity, after stabilization of vital functions [1, 2].

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17.3 Incidence: Etiology

Is a rare pathology; it occurs in 1 to 5% of closed traumas. Its highest incidence is in young males: in *the International optic nerve trauma study,* the average age of involvement was 34 ± 18 years and 85% were male.

The most common cause is accident with motor vehicles or bicycles (49%), falls (27%), and aggression (13%). In pediatric series, the most common cause are falls (50%) followed by traffic accidents (40%). The most common clinical form is indirect form.

17.4 Classification

In accordance with the mechanism of action:

- **Direct:** There is a significant anatomical alteration of the optic nerve, for example, by a projectile that penetrates orbit at high speed, or as a result of avulsion of the optic nerve, which usually occurs with severe visual loss.
- **Indirect:** Forces that occur during the blow are transmitted from a distant site to the ON, which occurs in 0.5 to 2.5% of all closed head trauma, without any manifest damage to the structures of the surrounding tissues. The deforming tension transmitted to the skull of closed trauma is concentrated in the optic channel region.

According to the site of the injury:

- Head of the ON
- Intraorbital
- Intracanalicular
- Intracranial

The **intracanalicular portion is** the most likely to be injured in closed trauma, as the upper half of the nerve in the canal is strongly attached to the periosteum by the fusion of the nerve with the nerve sheath, and therefore has very reduced mobility making it especially susceptible to trauma. Direct nerve damage or ischemic damage due to vascular injuries may occur. Imaging studies, particularly computed tomography, show variable results—up to 50% according to some series—in relation to detecting fractures in the optical channel that are difficult to highlight. Sometimes it is possible to detect small bone fragments that can completely or partially section the nerve, or compress it.

The **intracranial part of** the ON begins at the optic channel output and reaches the chiasma. The sickle dural fold is very close and also fused to the nerve sheath, so injury may occur at this level. It is the second most common location of TON. The chiasmatic region is the third in frequency of involvement.

In accordance with the commitment of Fundus Oculi - FO-:

- **Previous** neuropathy: Lesions prior to the entry of the central artery of the retina into the ON, about 1 cm from the back of the eyeball, cause alterations in the FO: hemorrhage or edema, due to arterial or venous compromise. Optical nerve avulsion may also be observed in severe trauma.
- **Subsequent** neuropathy: FO is normal which makes diagnosis difficult. If the trauma causes definitive damage, partial, or total paleness will become apparent late, within a variable period of 3 to 6 weeks.

17.5 Pathophysiology

In direct **TON** the mechanism is evident and neuroimaging usually shows direct damage to the optic nerve or compression by a bone fragment or hematoma [3].

In indirect **TON** the mechanism is usually **multifactorial** with a combination of mechanical and vascular events. The biomechanical response of cranial content to trauma is an important component of the mechanisms that are generated. Holographic studies with laser interferometry of the orbital cavity have shown that biomechanical forces triggered by trauma are transmitted through the wall of the orbital cavity and produce maximum deformity at the vertex level. Elastic deformation of sphenoids is manifested by the

appearance of canal fractures. The complex arrangement of collagen fibers in the ON pods and their firm bonding to the canal allow stress forces to be transmitted to the nerve, with a longitudinal orientation. When intense, they cause stretching of nerve fibers. In addition, there is compression and rupture of the nutrient vessels in their journey through the scissor pattern of the collagen fibers of the dura mater, when shearing forces are generated, resulting in the appearance of microinfarctions and microhemorrhages with edema; this, in turn, further compromises the blood supply in an inextensible space such as the bone walls of the canal [4].

Primary damage **occurs to** axons in ganglion cells, which are irreversibly injured with consequent cell degeneration. Secondary mechanisms of edema, **especially significant ischemia** within the narrow optic channel are also triggered. This creates a compartment syndrome and a vicious circle between the two mechanisms involving multiple metabolic pathways—release of free radicals, alteration of calcium metabolism, alteration of vasoregulatory mechanisms—leading to death by apoptosis of affected ganglion cells. Postulated treatments aim to try to curb this secondary damage.

Diffuse **axonal damage should also** be considered as an underlying mechanism in indirect TON. Axon deformations occur in brain white tissue tracts that damage the axonal cytoskeleton resulting in impaired axoplasmic transport.

On the other hand, ophthalmologic controls should be maintained because visual decrease may not occur immediately to trauma but over time, due to the **development of intraorbital hematomas** or in the sheath of the optic nerve. Orbital hemorrhage results in compartment syndrome with increased orbital pressure that compromises the circulation of the ON. Orbital **emphysema,** after fractures, is usually a benign condition. However, cases have been reported in which a valve mechanism is generated, with a large increase of intraorbital pressure and ON compression, although it is an extremely rare form of TON.

The least common variant of TON is **ON avulsion.** It usually occurs after closed-balloon trauma and can be total or partial. Three likely production mechanisms have been proposed:

- Sharp increase in intraocular pressure from traumatic compression that disinserts the ON. It behaves similarly to an eye rupture at the level of an anatomically weak site in the eye wall.
- Increased orbital pressure with balloon protrusion, resulting in a stretching of the nerve, until it is avulsed from its scleral insertions.
- Extreme rotations of the globe with disruption of the laminar region.

17.6 History

Clinical **history is, of course, fundamental.** The type of trauma, the time of evolution, whether or not there was loss of consciousness, and the duration of the trauma should be recorded. If there was a visual loss, if it was perceived immediately or deferred. If the patient has received any treatment or surgery.

In mild cases, the patient may have difficulty referring trauma as he/she does not link it to late visual damage. In severe cases, particularly loss of consciousness, it is difficult to obtain both a history and the patient's examination.

A complete **ophthalmological examination should always be performed for signs and symptoms that** may accompany TON: edema—bruising—exophthalmos—obvious wounds or fractures—commitment to motility—and, except contraindication, FO with dilation to rule out, for example, retinal detachment, vitreous hemorrhage, intraocular foreign body, choroid ruptures, or crystalline dislocation. A history consistent with TON would be vision loss after blunt or penetrating trauma that could not be explained by slit lamp or dilated fundus findings.

In **physical examination,** it is essential to feel the orbital rim to look for signs of fractures. When orbital compartment syndrome is suspected, the existence of resistance to eye backpulsion and intraocular pressure should be explored which, if elevated, point to such diagno-

sis. The ocular globe and its annexes should be examined for concomitant eye disturbances.

1. **Decreased Visual Acuity:** The compromise is variable and ranges from non-light perception to minimum or imperceptible loss. Up to 40–60% of patients have a severe loss. Direct TON with very small VA has a worse prognosis. In the case of indirect injuries, recovery may be greater, and the initial VA has proven to be the most important prognostic factor: the higher the initial VA the greater the chance of improvement.
2. **Afferent pupillary defect**: Pupil evaluation is critical and should always be recorded in clinic history, particularly in cases with normal FO where the patient refers to marked visual disorder. On the other hand, in a patient with severe head trauma the onset of uni- or bilateral mydriasis implies the possibility of brain herniation with life risk. In bilateral cases, we can find diminished or abolished pupillary reflexes.
3. **Alteration in chromatic vision**: The blue–yellow axis can be affected with good VA and respected VF **or** red–green axis, when there is great visual affectation and damage of the central VF. Diffuse defects without a defined axis may also occur, and when the VA is severely affected, it is not possible to evaluate color vision correctly [5].
4. **Variable campimetric defects**: Lower hemianopsia (by involvement of upper pial vessels in the optic canal) and central dense scotomas are common.
5. **Fundus oculi:** The compromise is variable depending on the location of the insult. It is noteworthy again that a normal FO does not rule out TON as paleness takes time to become apparent.

17.7 Complementary Examinations

- **Visual field.**
- **OCT**: Allows follow-up injuries. When there is FO damage, it will show the thickness increasing of the RFNL and/or macula by edema and bleeding and its subsequent evolution toward normalization or atrophy.
- In indirect TON, defects will not become evident for 3 or 4 weeks, when a reduction in thickness in the peripapilar RFNL can be seen. The loss will stabilize at 20 weeks [6].
- **Orbital ultrasound** has limited utility restricted to anterior lesions.
- **Visual Evoked Potentials (VEPs)** are generally of limited utility because of the impossibility of mobilization in most cases. In patients with amaurosis and normal FO, highly altered or non-recordable VEPs allow to establish the extent of the damage; the possibility of visual recovery is highly unlikely.
- **NEUROIMAGES**: They are fundamental.
- **Computed Tomography** (**CT**) allows better detection of cranial fractures, and in particular, those that affect the optic channel. It is the most accessible and easiest study to perform even in severe traumas with loss of consciousness. It also allows to discard metallic foreign bodies, emphysema, bone fragments and, although with less definition, hematomas.
- **Magnetic Resonance Imaging (MRI)** allows for better assessment of brain matter, (presence of edema or stroke), as well as its prognosis when performing diffusion and perfusion techniques; it also detects signs of intracranial hypertension syndrome in particular cerebrospinal fluid in the optic nerve sheath.

17.8 Differential Diagnosis

- Posterior ischemic optic neuropathy
- Optic neuritis
- Nonorganic vision loss
- Pre-/intra-/subretinal hemorrhage
- Choroidal rupture
- Commotio retinae

17.9 Treatment

It remains controversial and targets the recovery of secondary axonal damage mechanisms. The

main current options are corticosteroids and surgical decompression, both non-risk-free and without clear evidence in trials.

Moreover, 40–60% of TON have **spontaneous recovery,** excluding severe cases [7–9].

17.10 Corticosteroids

There is no standardized dose. It is based on small retrospective series, anecdotal cases, and extrapolations from studies on brain and spinal trauma.

The **doses** used may be moderate (60–100 mg oral prednisone), high (1 g/day intravenous methylprednisolone), or megadoses (30 mg/kg IV followed by 5.4 mg/kg/h for 24 h) [10–12].

The **NASCIS 2** (National Acute Spinal Cord Injury Study) compared placebo, naloxone, and methylprednisolone, and only showed some superiority of the latter if administered within the first 8 h after trauma: significant improvement in spinal cord motor and sensory function. However, the design of this study showed numerous gaps that detract from its conclusions [13–15].

The **CRASH** (Corticosteroid Randomization After Significant Head Injury) study, which used steroid megadoses, concluded that they should not be routinely used as the treated group had higher mortality than untreated [16, 17].

17.11 Surgical Treatment

Decompressive optical channel surgery is the most commonly used procedure. It can be performed with endoscopic techniques—less invasive—or extracranial. One of the drawbacks is that the general condition of the patient sometimes forces to differ surgery so it could lose effectiveness. Some of the risks of the procedure are CSF leaks and meningitis; both have decreased with the use of endoscopic techniques: they allow good visualization of the orbital vertex and facilitate a faster recovery with better aesthetic results. It is discussed whether it should be performed after 48 h of corticosteroid, so there is less edema, or immediately, without studies allowing a conclusion to be drawn [18, 19].

Some authors recommend it when bone fragments exist in the optical channel, while others argue that their presence already implies that there may have been direct, difficult-to-recover axonal injury, even if the fragment is removed. The IONTS study endorses this view.

The **fenestration of ON** is another reported surgical procedure. Indicated in hematomas of the optic sheath, in N2 portion. A correct preoperative MRI evaluation is vital in these cases, which requires the exact location of the hematoma, because there is no evidence of its success in interstitial hematoma.

In patients with depressed lateral wall fractures or orbital subperiostic hematomas that compress the ON, the best option seems to be **to perform an Orbitotomy,** which allows the reduction of the fracture or the evacuation of the hematoma respectively. On the other hand, if an orbital hemorrhage is demonstrated that compromises the function of the nerve, the immediate realization of **a Cantotomy that allows** the expansion of the orbital content is a priority. If this does not provide sufficient relief, **Decompressive Orbital Surgery should be performed.**

The **IONTS** (International Optic Nerve Trauma Study 1999), designed specifically to evaluate the treatment of TON, compared observation to decompressive surgery and corticosteroids, but its methodological defects were numerous. The end result was that there was no significant difference between the visual recovery of the three groups. That is, between treating and not treating independently of the type of treatment.

TONTT (Traumatic Optic Neuropathy Treatment Trial 2017), compares the use of **erythropoietin** (EPO) vs corticosteroid treatment vs observation. There were no significant differences in the final VA between the 3 groups and, it should be noted, that the 3 groups showed significant improvement. Risk factors for poorer recovery were the initial VA of Light Perception and Late Treatment (after 3 days) [20–23].

From the evidence presented, we conclude that no therapy of those employed **to date in TON can be considered superior to the rest, even compared to mere observation** of the

patient. The decision of using or not steroids will depend on the evaluation of each particular case, with the most widespread pattern being 1 g of methylprednisolone for 3 days followed by oral prednisone in the following days. As well as the possibility of combining medical and surgical treatment, which is suggested in patients who initially have marked VA commitment.

A recent study "Surgical Decompression or Corticosteroid Treatment of Indirect Traumatic Optic Neuropathy: A Randomized Controlled Trial" (Ann Plast Surg 2020) would confirm this trend. In this trial, all patients (30 enlisted patients) were treated: 12 with surgery and 18 with corticoids, without finding significant differences in outcome between the two groups except in cases of initial VA of Light Perception or Amaurosis that would benefit primarily from surgery. Other recent studies with neuroprotective factors such as amniotic membrane derivatives have shown promising results, but do not yet have clinical application and are still in the experimental stage.

17.11.1 Prognosis

- It is poor in **direct lesions** where there is partial or total damage from the optic nerve.
- In **indirect lesions,** a percentage of spontaneous recovery—40 to 60%—is observed. It does not appear to be any significant difference to date between treating and not treating, although it will depend on individual consideration in each case. For example, the presence of large edema justifies the use of corticosteroids, or an important orbital hematoma, surgery.

References

1. Miller NR, Newman NJ. Walsh & Hoyt's clinical neuro-ophthalmology, vol. 1. 6th ed. Baltimore: Lippincott Williams & Wilkins; 2004. p. 431–46.
2. Steinsapir KD, Goldberg RA. Traumatic optic neuropathy. Surv Ophthalmol. 1994;38:487–518.
3. Lee A, Brazis P. Neuro-ophthalmology an evidence-based approach, vol. 4. 2nd ed. New York: Thieme; 2019. p. 120–1.
4. Singman E, Daphalapurkar N, White H, Nguyen T, Panghat L, Chang J, McCulley T. Indirect traumatic optic neuropathy. Mil Med Res. 2016;3:2.
5. Hart WM. Acquired dyschromatopsias. Surv Ophthalmol. 1987;32:10–31.
6. Kanamori A, et al. Longitudinal study of retinal nerve fiber layer thickness and ganglion cell complex in traumatic optic neuropathy. Arch Ophthalmol. 2012;130(8):1067–9.
7. Chaon BC, Lee MS. Is there treatment for traumatic optic neuropathy? Curr Opin Ophthalmol. 2015;26:445–9.
8. Gabriela GA, Dine Kimberly KRS, Larry B, RGC SK. Neuroprotection following optic nerve trauma mediated by Intranasal delivery of Amniom cell Secretome. Invest Ophthalmol Vis Sci. 2018 May;59(6):2470–7.
9. Wu N, Yin ZQ, Wang Y. Traumatic optic neuropathy therapy: an update of clinical and experimental studies. J Int Med Res. 2008;36(5):883–9.
10. Steinsapir KD, et al. Treatment of traumatic optic neuropathy with high-dose corticosteroid. J Neuroophthalmol. 2006;26:65–7.
11. Yu-Wai-Man P, Griffiths PG. Steroids for traumatic optic neuropathy. Cochrane Database Syst Rev. 2013;6:CD006032.
12. Lai L, Liao H-T, Chen C-T. Risk factors analysis for the outcome of indirect traumatic optic neuropathy with steroid pulse TherapyI. Ann Plast Surg. 2016;76(Suppl 1):S60–7.
13. Bracken MB, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the second national acute spinal cord injury study. N Engl J Med. 1990;322:1405–11.
14. Liu Z, Yang Y, He L, Pang M, Luo C, Liu B, Rong L. High-dose methylprednisolone for acute traumatic spinal cord injury: a meta-analysis. Neurology. 2019;93(9):e841–50.
15. Nesathurai SJ. Steroids and spinal cord injury: revisiting the NASCIS 2 and NASCIS 3 trials. Trauma. 1998;45(6):1088–93.
16. Edwards P, Arango M, Balica L, et al. Final results of MRC CRASH: a randomised placebo-controlled trial of intravenous corticosteroid in adults with head injury-outcomes at 6 months. Lancet. 2005;365:1957–9.
17. Edwards P, Arango M, Balica L, Cottingham R, El-Sayed H, Farrell B, Fernandes J, Gogichaisvili T, Golden N, Hartzenberg B, Husain M, Ulloa MI, Jerbi Z, Khamis H, Komolafe E, Laloë V, Lomas G, Ludwig S, Mazairac G, de los Angeles Muñoz Sanchéz M, Nasi L, Olldashi F, Plunkett P, Roberts I, Sandercock P, Shakur H, Soler C, Stocker R, Svoboda P, Trenkler S, Venkataramana NK, Wasserberg J, Yates D, Yutthakasemsunt S, CRASH Trial Collaborators.

Final results of MRC CRASH, a randomised placebo-controlled trial of intravenous corticosteroid in adults with head injury-outcomes at 6 months. Lancet. 2005;365(9475):1957–9.
18. Yu-Wai-Man P, Griffiths PG. Surgery for traumatic optic neuropathy. Cochrane Database Syst Rev. 2013;6:CD005024.
19. Chen HH, et al. Surgical Decompression or corticosteroid treatment of indirect traumatic optic neuropathy: a randomized controlled trial. Ann Plast Surg. 2020;84(1S Suppl 1):S80–3.
20. Levin LA, et al. The treatment of traumatic optic neuropathy: the international optic nerve trauma study. Ophthalmology. 1999;106:1268–77.
21. Entezari M, Esmaeili M, Yaseri M. A pilot study of the effect of intravenous erythropoietin on improvement of visual function in patients with recent indirect traumatic optic neuropathy. Graefes Arch Clin Exp Ophthalmol. 2014;252(8):1309.
22. Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy: international optic nerve trauma study. Ophthalmology. 1999;106(7):1268–77.
23. Kashkouli MB, Yousefi S, Nojomi M, Sanjari MS, Pakdel F, Entezari M, Etezad-Razavi M, Razeghinejad MR, Esmaeli M, Shafiee M, Bagheri M. Traumatic optic neuropathy treatment trial (TONNT): open label, phase3, multicenter, semiexperimental trial. Graefes Arch Clin Exp Ophthalmol. 2018 Jan;256(1):209–18.

Complex Orbital Fractures

18

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Abstract

Complex orbital fractures are those that involve multiple orbital walls, often involve the adjacent facial skeleton (orbitofacial fractures), often with severe visual, structural and esthetic consequences. Management of such fractures not only depends on the type of the fracture but also upon the various patient modifiers—designated by the 'ABCDEFGH' mnemonic detailed below. The following fractures: combined orbital floor-medial wall fractures, zygomatico-maxillary complex fractures, naso-orbito-ethmoidal fractures, Le Fort fractures, Cranio-orbital and cranioorbitofacial fractures, panfacial fractures, revision orbital fractures, and finally orbital fractures with globe injuries, optic neuropathy or foreign bodies, are discussed in detail in the chapter below.

Keywords

Orbital fractures · Orbitofacial fractures Blow out fractures · Complex orbital fractures · Patient modifiers · ABCDEFGH classification · Computed tomography (CT scan) · Magnetic resonance imaging (MRI) Zygomatico-maxillary complex fracture (ZMC) · Naso-orbito-ethmoidal fractures (NOE) · Le Fort fractures · Panfacial fractures

18.1 Introduction

Orbital fractures are common, seen in children, young adults, middle-aged, and the elderly [1]. While ophthalmologists commonly encounter blowout fractures, more commonly they occur in association with midfacial and other facial trauma. Most pure orbital blow out and blow in fractures are managed by ophthalmologists which may be either managed conservatively or by surgical intervention, with standard practice guidelines of surgical approach, implant design, and placement. However, there is a large group of orbital fractures that are considered complex—either from a clinical and surgical decision-making perspective, surgical approach, implant design and placement, intraoperative confirmation, monitoring, and managing postoperative outcomes including morbidity, if any.

Shown below is a practical classification of Orbital & Orbitofacial Fractures of use to the ophthalmologist and the orbital surgeon (Table 18.1) [2] and approximate frequency in our institution.

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Table 18.1 Practical classification of orbit and orbitofacial fractures

"Simple"/Pure orbital fracture	"Complex" orbitofacial fracture
1. Linear fractures 2. Blow out fractures a. 1 wall fractures (21%) i. Floor blow out fractures ii. Medial wall blow out fractures iii. Roof blow out fractures iv. Trapdoor fracture b. 2 wall fractures (10%) v. Floor and medial wall blow out fractures 3. Blow in fractures.	1. Zygomatico-maxillary complex fractures (ZMC) Type I-III (18%) 2. Naso-orbito-ethmoidal fractures (NOE) Type I-III (3%). 3. Cranio-orbital fractures (10%) 4. Orbitofacial fractures Le Fort II fracture (5%) Le Fort III fracture (14%) 5. Cranio-orbitofacial fracture (14%) 6. Panfacial fracture (5%).

Table 18.2 Patient modifiers in orbital and orbitofacial fractures

Patient factor	Definition	Clinical implication
Age	Infant, child, young adult, elderly	Urgency, timing of intervention
Bilateral or Unilateral	Prioritizing unilateral or bilateral repairs	Need for simple techniques vs. advanced technology, e.g., treatment planning, intraoperative navigation, etc.
Complex or Simple orbital fractures	Oculoplastic or multidisciplinary approach	Simple vs. multidisciplinary, multiple incisional approaches, complex implants
Displaced or non-displaced fractures	Threshold for intervention	
Entrapment or not	Orbital soft tissues (EOM-IMS Complex), globe etc.	Urgency of intervention, larger incisions
Foreign body present or not	Intraocular, Intraorbital or both	Antibiotics, localize and plan early removal
Globe and/or optic nerve injuries	Management closed or open globe injury, prevent additional damage	Address after life-threatening injuries before specific orbital interventions
High-risk patient or not	Low-risk patients: young healthy, adults or children without intracranial injuries/ polytrauma High-risk patients: elderly, comorbidities, intracranial injuries/polytrauma	Multispecialty approaches with life preservation and if indicated delayed orbital intervention.

In addition, patient-specific factors shown in Table 18.2 remembered by the mnemonic ABCDEFGH, help formulate decision making regarding the threshold for intervention, timing of intervention including delaying surgery to address underlying globe and optic nerve injuries, coexisting orbital foreign bodies, involving additional surgical subspecialties and considerations regarding additional risks from anesthesia and surgery in medically high-risk patients.

Not infrequently, in addition to the comprehensive classification system shown above, there are several other clinical scenarios where clinical diagnosis and surgical management are fraught with pitfalls resulting in delays, intraoperative complications, and postoperative morbidity with suboptimal outcomes. These complex orbital fracture scenarios are listed in Table 18.3.

A brief overview of the nature of the complexity of fracture and principles of management of each of the complex orbital fracture scenarios is described below with illustrative examples.

18.2 Combined Orbital Floor: Medial Wall Fractures

Indications for repair: Significant displacement of orbital contents from disruption of the inferomedial orbital strut with or without other orbital wall fractures with resultant enophthalmos,

globe/orbital displacement, and motility disorders.

Principles of management: Ensure inferior orbital rim is intact or well reconstructed. A wide exposure to the orbital floor with a swinging eyelid approach (inferior transconjunctival incision with lateral canthotomy and inferior cantholysis) is important for good exposure of the orbital floor with visualization of the posterior ledge.

Table 18.3 Complex orbital fracture scenarios

	Type of complex orbital & orbitofacial fracture
1.	Combined orbital floor—medial wall fractures
2.	Zygomatico-maxillary Complex Fracture
3.	Naso-orbito-ethmoidal fracture
4.	Le Fort II & Le Fort III Fractures
5.	Cranio-orbital & Cranio-orbitofacial fracture
6.	Panfacial Fractures
7.	Orbital fractures with Globe injuries—Open and Closed Globe injuries
8.	Orbital fractures with traumatic optic neuropathy
9.	Orbital fractures with foreign body
10.	Revision Orbital fractures

Beginners are advised to extend the incision medially with a retrocaruncular incision preserving the Horner-Duverney muscle posterior to the lacrimal sac. The inferior oblique muscle may be elevated subperiosteally to obtain wide and continuous exposure. The orbital contents are then reduced into the orbital cavity. A prebent prefabricated anatomic plate based on the Angle of Inferomedial Orbital Strut (AIOS) (Fig. 18.1a) made either of anodized MatrixORBITAL® preformed titanium plates (Fig. 18.1b, c) or a composite titanium—porous polyethylene (TITAN®) implant, is then guided along the floor and medial wall. It is placed over the posterior ledge along the floor and medial wall, confirmed either by direct visualization or with intraoperative navigation with repeat forced duction test, prior to closure. In select patients, a prebent bioresorbable plate may be used as well, with good bony healing in 18–24 months (Fig. 18.2a, b). A postoperative scan is often recommended to ensure good positioning of the orbital implant as not infrequently these complex fractures are poorly reconstructed (Fig. 18.3).

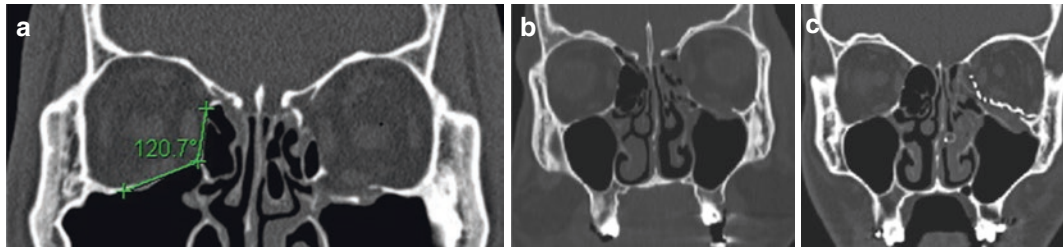

Fig. 18.1 (**a–c**) Preoperative and postoperative CT Scan showing left orbital combined floor-medial wall fracture with a prebent prefabricated anatomical titanium implant

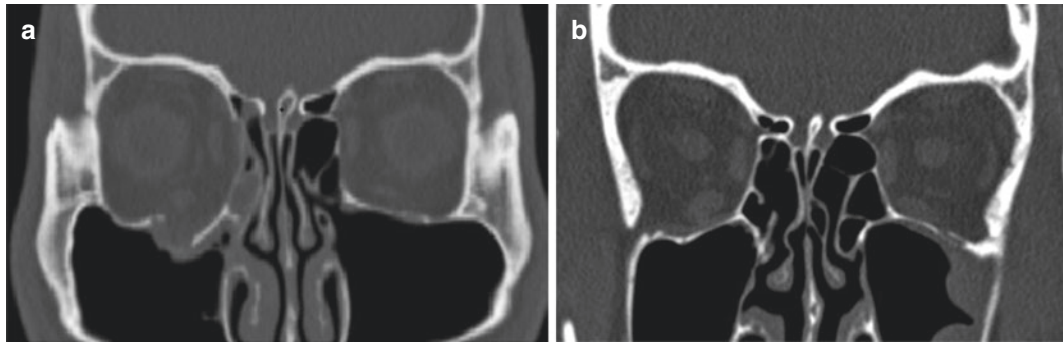

Fig. 18.2 (**a, b**) Preoperative and late postoperative coronal CT scan of right orbital floor-medial wall fracture repaired with bioresorbable implant (RapidSorb®) demonstrating complete bone healing and implant resorption

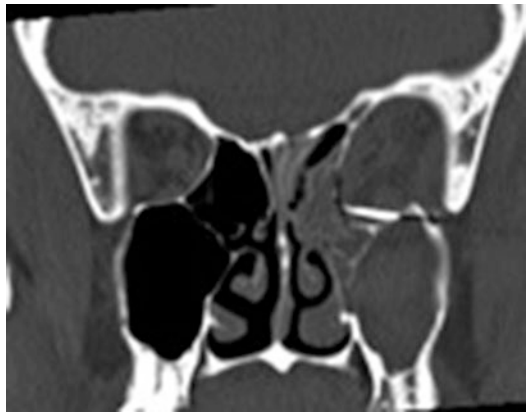

Fig. 18.3 Poorly reconstructed orbital floor—medial wall fracture

18.3 Zygomatico-maxillary Complex (ZMC) Fractures

These are the second most common midfacial fractures, commonly from falls and assaults. Zingg [3] classified these fractures similar to Manson's low, mid, and high impact injuries into Type I: single suture disarticulation with minimal displacement and easy repair , Type II: multiple site disarticulation with more challenging repair (Fig. 18.4a, b) and Type III: comminuted displaced fractures from high impact injuries, which requires multiple and wide exposure with 3 point fixation along the frontozygomatic suture, the zygomaticomaxillary buttress, the inferior orbital rim (Fig. 18.4c, d) and when necessary the zygomatic arch. Associated displaced fractures of the orbital floor and medial wall may also need to be reconstructed. A good landmark of accurate orbital reduction with reconstruction is the alignment of the zygomatico-sphenoid suture of the lateral wall of the orbit (Fig. 18.5).

18.4 Naso-orbito-ethmoid (NOE) Fractures

These are uncommon yet highly challenging fractures to manage, typically arising from high-velocity injuries to the central midfacial skeleton. It may be unilateral or bilateral and may be associated with other midfacial and skull base fractures. The Manson–Markowitz classification [4] is as follows: Type I—single fragment displacement with intact medial canthal tendon, Type II—multiple comminuted butterfly segment with medial canthal tendon, and Type III—severely comminuted and displaced medial butterfly fragment commonly with avulsion or detachment of the medial canthal tendon.

Indications for repair: All displaced NOE fractures unilateral or bilateral especially in young healthy patients should be repaired as early as possible, after ruling out and managing CSF leaks and underlying brain/globe injuries.

Principles of management: Adequate exposure is usually obtained using a coronal approach and when necessary bilateral transoral facial degloving approach. Fractured segments are reduced and either fixated using a miniplates (Type I NOE fracture) (Fig. 18.6a, b) or using transnasal intercanthal wiring with mild overcorrection placing the medial canthus posteriorly and superiorly [5]. The nasolacrimal ducts may also be intubated in suspected fractures at risk of nasolacrimal duct obstructions from postoperative manipulations.

18.5 Le Fort II and III Fractures

These are common orbitofacial fractures that involve the mid-face. Le Fort II fractures involve the maxilla, the medial wall, and floor of the orbit (pyramidal fractures) and Le Fort III fractures, the lateral wall of the orbit as well (craniofacial dysjunction) [6]. Frequently asymmetrical, they may be associated with other facial fractures such as NOE, craniofacial or panfacial fractures.

Principles of management: After stabilization of life and vision, displaced fractures often will require open reduction and internal fixation through a buccal, transconjunctival, and occasionally a coronal approach with degloving of the facial skeleton for better exposure and reduction of the fracture fragments (Figs. 18.7a, b and 18.8a, b). The nasolacrimal drainage system may require prophylactic stenting if involved.

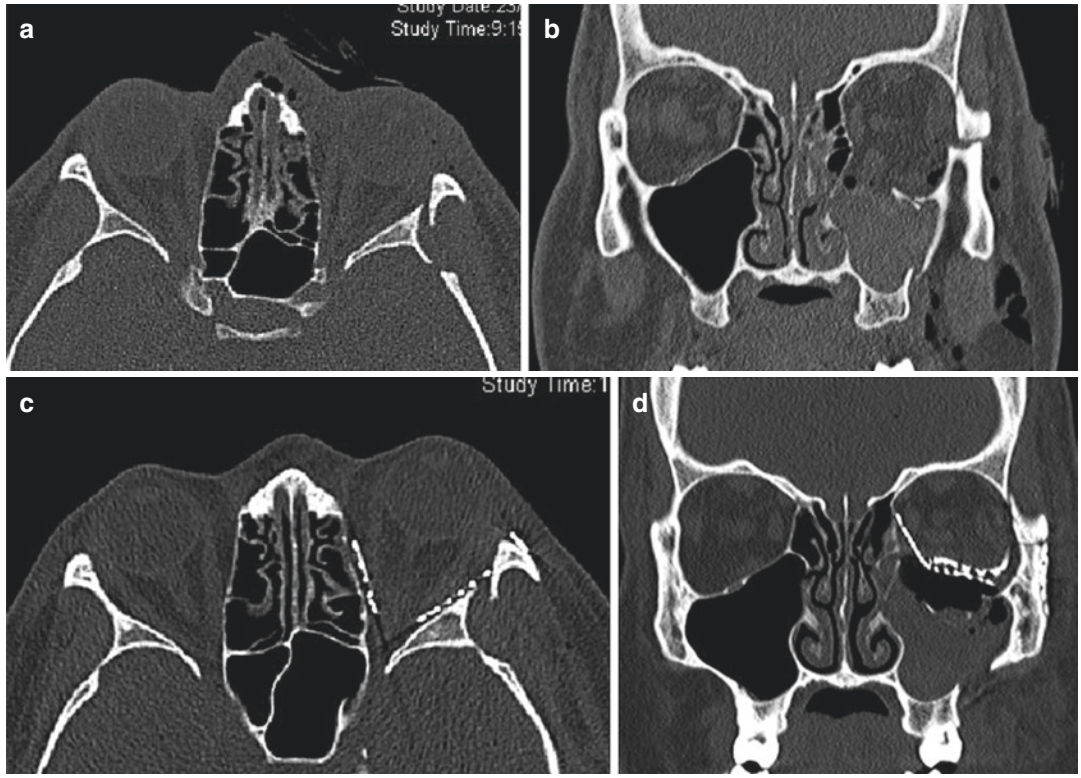

Fig. 18.4 (**a, b**) Preoperative axial and coronal CT scan showing left orbito-zygomatic complex fracture. (**c, d**) Postoperative axial and coronal CT scan showing reconstructed left ZMC fracture

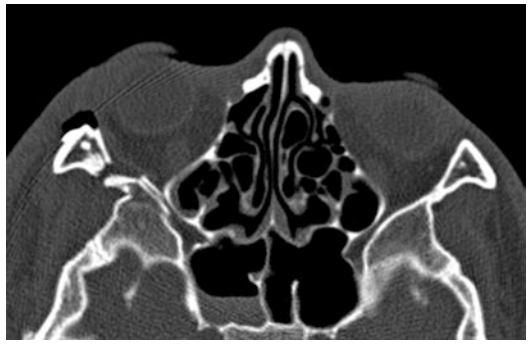

Fig. 18.5 Poorly reconstructed right lateral orbital wall in zygomatico-maxillary complex fracture

18.6 Cranio-orbital and Cranio-orbitofacial Fractures

These are from high impact injuries which may be seen in children, young adults, and the elderly from falls or direct frontal injuries. The orbital roof which forms the skull base adjacent to the anterior cranial fossa may be involved, sometimes along with the temporal bone and with pneumocephalus, orbital tissue prolapse into the anterior cranial fossa, dural tears with CSF leak with increased risk of meningitis and occasionally herniation of intracranial contents [7]. An increased risk of traumatic optic neuropathy is also encountered.

Principles of management: This will often require co-management with a Neurosurgeon with the urgent repair of dural tears/CSF leak through a craniotomy approach. Open fractures must be immediately addressed to minimize the risk of meningitis. Orbital and midfacial fractures may be repaired primarily or secondarily after stabilizing the patient. Isolated orbital roof fractures may be repaired either through a transcranial approach or an eyelid crease approach if minimally displaced and the dura is intact (Fig. 18.9a–d).

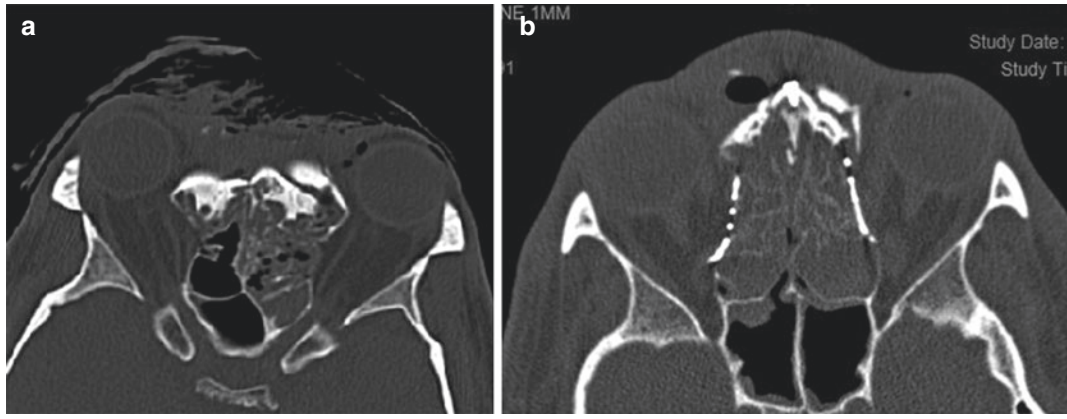

Fig. 18.6 (**a, b**) Axial bone window CT scans showing a preoperative and well-reconstructed postoperative bilateral naso-orbito-ethmoidal fracture

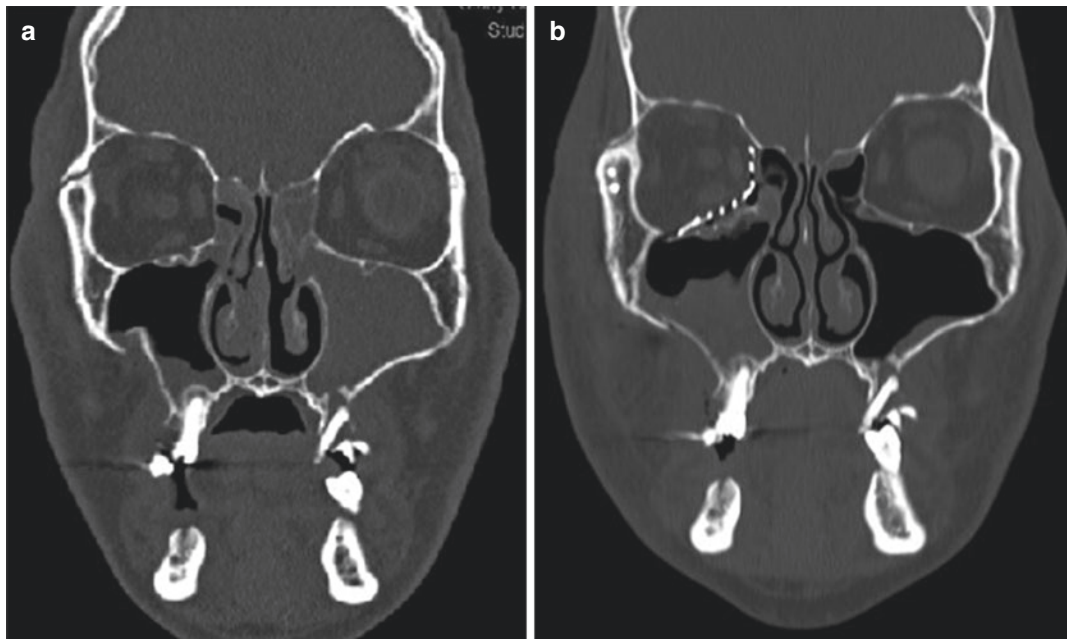

Fig. 18.7 (**a, b**) Le Fort II fracture: Preoperative and postoperative coronal CT scans

18.7 Panfacial Fractures

These are fractures that involve the upper, mid, and lower face thereby requiring not only airway stabilization and good primary management of polytrauma if associated, but also a meticulous and detailed evaluation of the neurologic and ophthalmologic status before considering a repair. Depending on the complexity of the fractures with associated vital structure involvement, a single-stage or multiple-staged approach may be pursued.

Principles of management: Life and neurologic assessment, globe evaluation and stabilization, followed by fracture repair. Meticulous preoperative planning is essential. Fractured fragments are to be accurately reduced and fixated to the stable bone in a "top-to-bottom" or

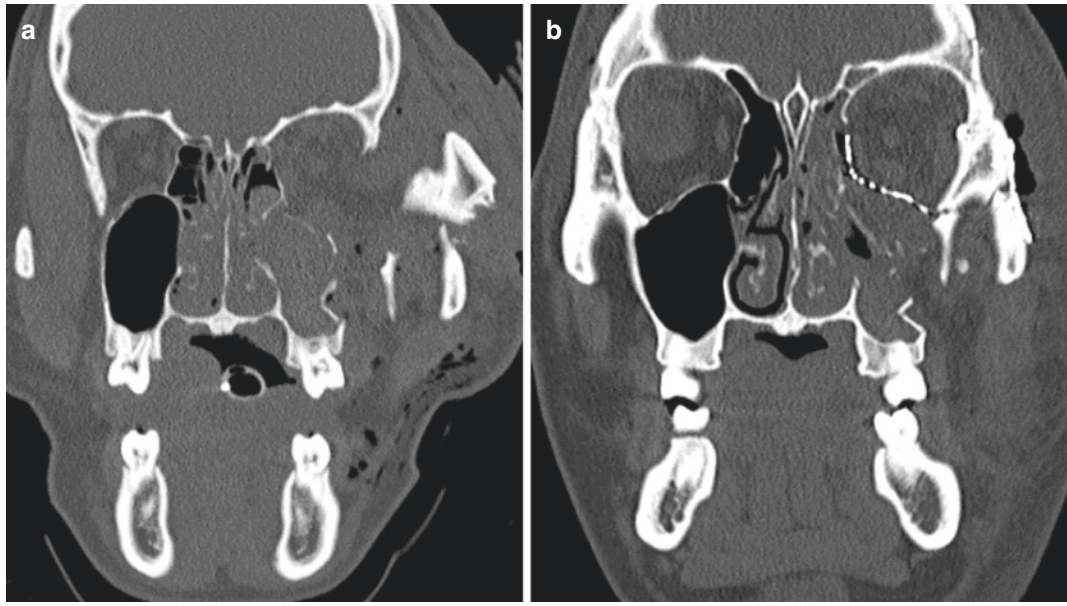

Fig. 18.8 (**a, b**) Le Fort III fracture: preoperative and postoperative coronal CT scan images

"bottom to-top" sequence—along with an outside–inside approach for the midface including the orbits (Fig. 18.10) [8]. Intraoperative navigation may be essential if fragments are severely comminuted and displaced [9]. Preoperative treatment planning may be performed based on the contralateral side for accuracy of reduction and placement of orbital implants.

18.8 Orbital Fractures with Globe Injuries

A high proportion of isolated orbital fractures and orbitofacial fractures are associated with globe injuries [10]. However, the risk of open globe injuries is relatively low, owing to the "shock absorber effect" and the buckling of the orbital walls minimizing globe dehiscence. It is only after globe injury management and stabilization should orbital reconstruction be performed.

Principles of management: Close collaboration between the ophthalmologist and orbital/craniomaxillofacial surgeon is important to plan the sequence, timing and also ensure globes are adequately protected during the orbital repair. Secondary interventions to the globe may also be necessary after orbital and facial fractures have been repaired. In extreme situations where the globe is irreparable or becomes phthisical, a secondary ocular enucleation with orbital implant and customized prosthesis may be necessary for structural, esthetic, and psychosocial rehabilitation (Fig. 18.11a–d).

18.9 Orbital Fractures with Traumatic Optic Neuropathy

While any orbital fracture may be associated with globe and/or optic nerve injury, typically high-impact injuries especially to the medial wall and orbital roof have a higher incidence, especially in adults. Preoperative ophthalmic examination including the pupils and visual fields is therefore essential before embarking on orbital and facial fracture repairs. This has both medical and medicolegal consequences.

Principles of management: Intravenous corticosteroids may be considered despite the lack of strong clinical evidence if no contraindica-

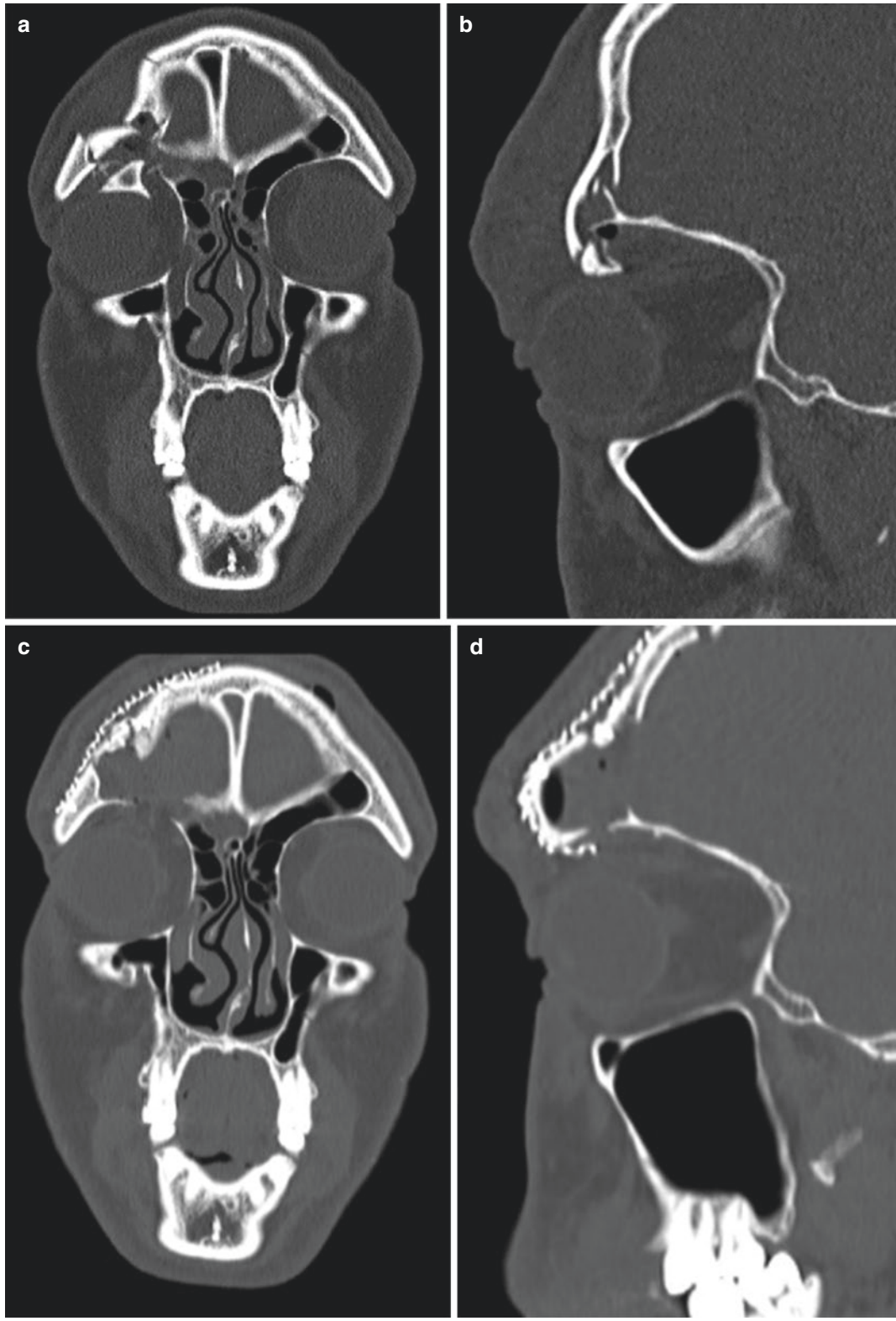

Fig. 18.9 (**a, b**) Coronal and sagittal bone window CT scan showing comminuted displaced right cranio-orbital fracture. (**c, d**). Coronal and sagittal bone window CT scan showing well-reconstructed right cranio-orbital fracture

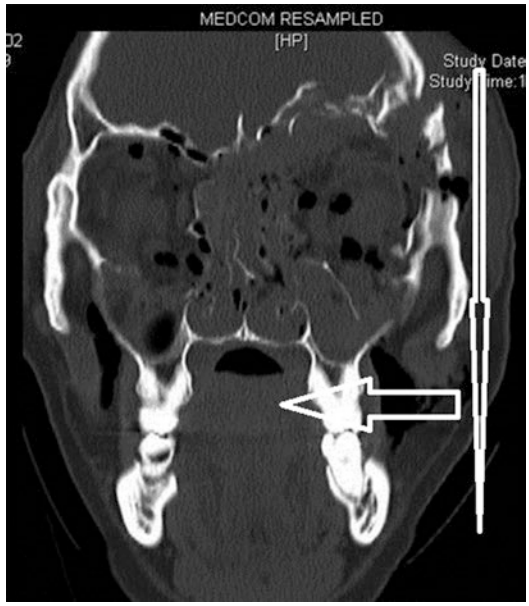

Fig. 18.10 Panfacial fracture demonstrating principle of reconstruction sequence

tions such as intracranial injuries or concurrent infections are present. Orbital fracture reconstruction should be delayed until the definitive visual prognosis is known and well understood by the patient. In partially sighted patients, a conservative approach or less aggressive orbital dissection and reconstruction may be recommended.

18.10 Orbital Fractures with Foreign Bodies

Penetrating and blast injuries of the eye and orbit may be associated with foreign bodies [11]. These may be classified into overt and latent foreign bodies and may also be labeled as radiooopaque and radiolucent foreign bodies. Rarely organic foreign bodies may be missed by routine CT scans and may be detected only on MRI. It

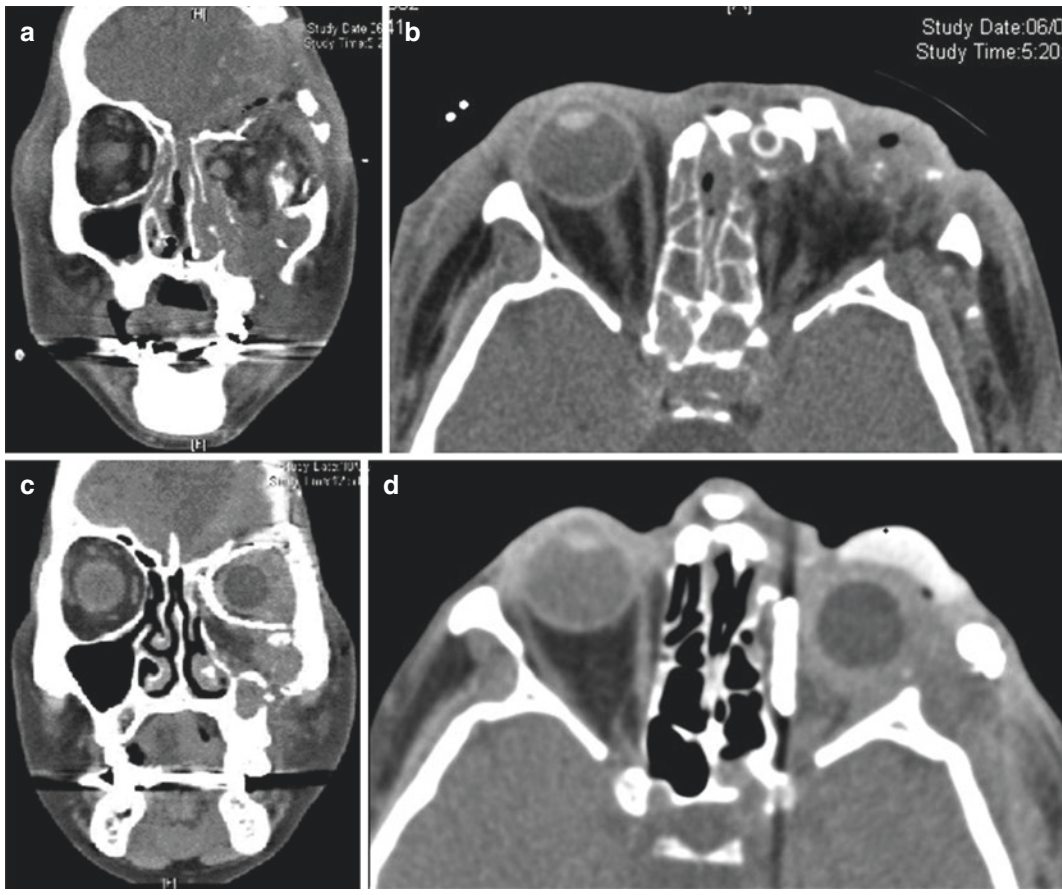

Fig. 18.11 (**a, b**) Cranioorbitofacial fracture with severe globe injury. (**c, d**) Post reconstruction of left orbitofacial skeleton and anophthalmic socket reconstruction

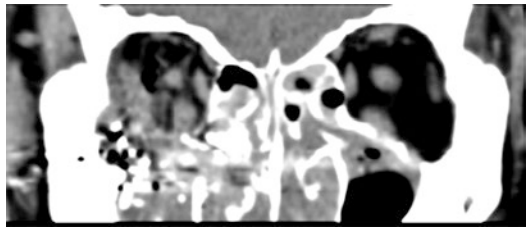

Fig. 18.12 Gunshot wound to the face with multiple pellet foreign bodies and right orbital floor fracture

should be remembered that large or high-velocity foreign bodies may involve the paranasal sinuses and/or intracranial cavity as well and thus should be appropriately managed (Fig. 18.12).

Principles of management: As there is a high incidence of visual morbidity and infection from intraocular and orbital foreign bodies, prophylactic antibiotics should be followed by foreign body extraction as early as possible. While large, contaminated, and vegetative foreign bodies should be mandatorily removed, fine particulate or small inert foreign bodies especially near vital structures at the orbital apex may be conservatively managed. Orbital reconstruction may be delayed when an underlying orbital infection is suspected.

18.11 Revision Orbital Fractures

Several factors play a role in the poor management of orbital fractures. These include extreme complexity from multiple orbital wall involvement, severely comminuted and displaced midfacial and panfacial fracture not providing stable bone for fixation, lack of availability of dedicated orbital-anatomical or patient-specific implants, poor patient health precluding meticulous and staged repair, absence of an orbital surgeon as part of the craniomaxillofacial team and finally limited or poor training and experience of the surgeon [12]. It should be remembered that revision surgeries are often riskier in terms of visual loss, induced diplopia with residual soft tissue deformities, which may require additional revision or enhancement procedures over time.

Principles of management; This involves a detailed soft tissue assessment and study of the deformed orbitofacial skeleton along with the implant position either against vital structures or within the adjacent spaces and cavities. A multidisciplinary approach should be adopted with good preoperative treatment planning, atraumatic removaling of existing implants, good reduction of orbital soft tissues followed by identification of bony landmarks. Orbital rim reconstruction should be performed first followed by orbital wall reconstruction with optimal placement of prebent anatomical orbital implants or patient-specific implants. Wide incisions, with adequate intraoperative visualization and where possible intraoperative navigation and even intraoperative radiologic imaging go a long way in ensuring the best possible reconstruction with the least postoperative morbidity (Fig. 18.13a–d).

A detailed overview of all the complex orbital and orbitofacial fractures including their definition, challenges in management, surgical approaches, implant considerations, the role of ancillary technologies including potential pitfalls and complications is summarized in Table 18.4.

18.12 Summary

Orbital fracture reconstruction and secondary management can be challenging, yet rewarding. Determination of complexity of orbital reconstruction, building multidisciplinary relationships, detailed preoperative evaluation, meticulous surgical planning and execution along with realistic informed consent go a long way in the optimal management of these challenging orbital fractures.

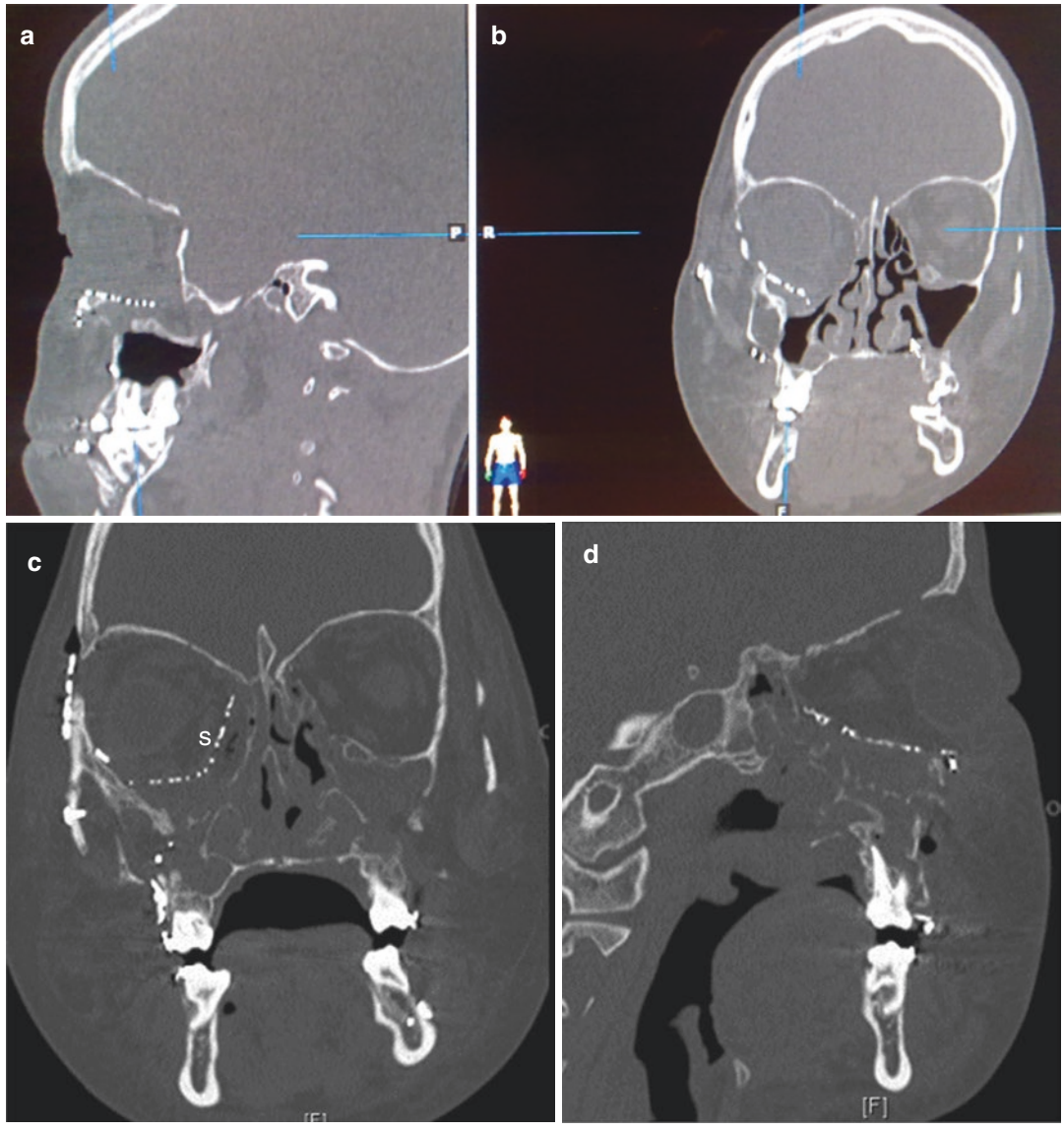

Fig. 18.13 (**a, b**) Comminuted displaced Le Fort III fracture referred post initial repair with a poor reconstruction of the orbit and the face (**c, d**) Post revision orbitofacial fracture repair under navigation guidance with titanium mesh implants and miniplates

Table 18.4 Complex orbital and orbitofacial fractures—an overview

	Definition	Challenges	Principles of management	Timing of intervention	Surgical approaches	Implant considerations	Specialties involved	Technologies involved	Consequences of suboptimal repair	Pitfalls and complications
Combined Orbital floor-medial wall fracture	Comminuted fracture of orbital floor and medial wall with disruption of the inferomedial orbital strut	Exposure to medial wall and floor Reduction of orbital contents Reconstruction of both medial wall and orbital floor with inferomedial orbital strut	Exposure Reduction of orbital contents Intraoperative verification Prebending implants Placement of implant Intraoperative verification	Within 2 weeks	Swinging eyelid approach with retrocaruncular incision with / without inferior oblique disinsertion Swinging eyelid approach alone	Prebent prefabricated anatomic plates Patient-specific implants Permanent or bioresorbable implants	Ophthalmologist/ Orbital surgeon	Patient-specific implant Prebent prefabricated customized implant Intraoperative navigation Intraoperative imaging	Enophthalmos Implant in ethmoid/ maxillary sinus	Undercorrection—common Incomplete or no repair of one wall Motility disorders with globe dystopia
Zygomatico-maxillary complex fractures	Disruption and displacement of one or more of the 4–5 articulations of the Zygoma	Incomplete reduction with comminuted, significantly displaced and late fractures	Preoperative analysis and treatment planning Wide multiple incisional approaches Adequate reduction with intraoperative verification Internal fixation Orbital floor reconstruction	Within 1 week in adults, earlier in children	Coronal approach (comminuted displaced orbitofacial or cranioor-bitofacial fractures) Upper blepharoplasty incision Swinging eyelid approach Transoral approach	Titanium implants for orbital rims Permanent or bioresorbable implants for orbital wall reconstruction	Orbital surgeon Craniomaxillofacial Surgeon	Intraoperative navigation Intraoperative imaging	Lateral canthal dystopia, enophthalmos, malar flattening, etc. Inadequate floor- medial wall reconstruction	Malar hypoplasia, lateral canthal dystopia, pseudoeno-phthalmos, pseudoproptosis, Difficult reduction with late reconstructions

Naso-orbito-ethmoidal fractures	Unialteral or bilateral variable disruption of medial canthal tendon with/ without underlying bone(butterfly) fragment with resultant telecanthus	Detection (missed diagnosis) Inadequate reduction Poor exposure Late intervention Associated lacriaml drainage system injuries	Early intervention Adequate exposure (coronal incision) Miniplate fixation Intercanthal wiring Secure fixation Overcorrection by default Consider bone grafting to nasal bridge Consider nasolacrimal duct stenting or primary DCR	Early (3–5 days if possible)	Coronal approach* Transoral approach Transcon-junctival approach	Miniplates Stainless Steel wires with or without barb	Orbital Surgeon Craniomaxillofacial Surgeon	Stainless steel wire Miniplates	Unilateral or bilateral telecanthus Nasolacrimal duct obstruction with chronic dacryocystitis	Undercorrection Residual telecanthus Chronic dacryocystitis CSF leak & meningitis
Le Fort Fractures Types II & III	Midfacial fractures involving the maxilla, the medial wall, floor (Le Fort II) and lateral wall of the orbit (Le Fort III)	Requires maxilla buttress, medial wall, floor and lateral wall fixation Variably associated with other fractures including NOE fractures	Adequate exposure Open reduction, Internal fixation Orbital reconstruction	1–2 weeks	Transoral, Transcon-junctival approaches	Miniplates for midfacial buttress Sheet or anatomical implants for orbital reconstruction	Orbital surgeon Craniomaxillofacial surgeon	Endoscopy Intraoperative navigation	Midfacial flattening Suboptimal orbital reconstruction	Midfacial flattening Enophthalmos Chronic dacryocystitis
Cranio-orbital and Cranioor-bitofacial fractures	Skull base fractures with orbital roof and midfacial fractures	Neurosurgical injury, CSF leak	Combined neurosurgical and orbital / orbitofacial approaches	Within 1 week	Coronal approach Transcon-junctival approach Transoral approach	Permanent or bioresorbable implants	Orbital surgeon Craniomaxillofacial Surgeon Neurosurgeon	Intraoperative navigation	CSF leak, meningitis Orbital deformities Diplopia	Meningitis, Growing fracture (children), Delayed fracture repair Diplopia, enophthalmos, etc.

(continued)

Table 18.4 (continued)

	Definition	Challenges	Principles of management	Timing of intervention	Surgical approaches	Implant considerations	Specialties involved	Technologies involved	Consequences of suboptimal repair	Pitfalls and complications
Panfacial fractures	Upper (frontoorbtial), midfacial/ orbital, and lower (Mandible) facial fractures	Complex reconstruction Staged approaches/ Multiple surgeries Multiple disciplines	Stable to unstable bone Superior to inferior fixation Lateral to medial fixation Craniofacial repair followed by internal orbital repair	1–2 weeks	Coronal approach Transcon-junctival approach Transoral approach	Titanium mesh implants Titanium miniplates Orbital implants (permanent & Bioresorbable)	Neurosurgeon Orbital surgeon Craniomaxillofacial surgeon	Intraoperative navigation Intraoperative or postoperative imaging	CSF leak, meningitis Orbital deformities Midfacial deformities Lower facial deformities	Cranioorbitofacial deformity
Revision Orbital fractures	Previously operated orbital fractures with residual deformity, misplaced implants, postoperative complications	Scarring of orbital and facial tissues Difficulty removing implants Additional orbital/soft tissue trauma Increased risk of diplopia, visual loss, additional procedures, etc	Preoperative analysis and counseling Undo previous surgery—implants and soft tissue Reduction with intraoperative verification without tissue damage Implant placement with verification	Earlier the better	As indicated, in general, larger incisions than usual surgeries	As above	Orbital surgeon Craniomaxillofacial surgeon	Patient-specific implants Intraoperative navigation	Orbital deformity, diplopia, enophthalmos, chronic dacryocystitis, etc.	Globe, EOM, intermuscular septum damage Inability to completely reduce orbital tissues Residual enophthalmos, diplopia, etc
Orbital fractures with significant Open or Closed Globe injury	Any orbital fracture with Open globe injury or severely contused closed globe injury	Potential delay in orbital fracture repair until globe injury recovery	Ensure best visual outcome/globe protection before orbital wall fracture repair	Ideally within 2 weeks after intraocular status is stabilized	Routine approaches with minimal globe manipulation Adopt subciliary and non-transcon-junctival approaches if necessary	Routine implants—permanent or bioresorbable	Dependent on the type of fracture	Routine techniques, navigation/ PSI for complex or multiple wall fractures	Residual enophthalmos, motility disorder with/ without diplopia	Iatrogenic globe injury from aggressive orbital fracture repair

Orbital fractures with Traumatic Optic Neuropathy	Any orbital fracture with subnormal visual acuity with RAPD from TON	Ensuring good correction without causing additional optic nerve injury/ visual loss	Ensure visual status optimization (medical and medicolegal) before orbital fracture repair	Within 2 weeks if stable	Routine approaches with minimal optic nerve/orbital apex manipulation	Routine implants, avoid apex of orbit reconstruction	Depending on type of fracture Ophthalmologist to stand by	Intraoperative VEP Intraoperative imaging	Complete visual loss—intragenic in addition to preexisting injury (Second optic nerve trauma)	Additional optic nerve injury Suboptimal orbital fracture repair Difficulty with late repairs
Orbital fractures with Orbital Foreign bodies	Any orbital fracture with intraorbital foreign body	Rule out intracranial injuries/foreign body Foreign body removal concurrent or before orbital wall reconstruction Single vs multistage procedure Orbital infections	Address and remove intraorbital foreign body first Aggressive antibiotics/ antifungals (vegetative FB) as indicated Consider primary vs. secondary repair as indicated	After ensuring complete foreign body removal and ruling our infections	Routine approaches, through foreign body tract if present and accessible	Routine implants, metallic porous implants preferably to non-porous sheet implants	Dependent on fracture	Intraoperative imaging Intraoperative navigation	Residual foreign bodies Orbital acute or chronic infection/ inflammation risk MRI contraindication with metallic residual foreign bodies	Incomplete foreign body removal (multiple, vegetative materials) Orbital infections/ inflammations Delay in orbital fracture repair

References

1. Runci M, De Ponte FS, Falzea R, Bramanti E, Lauritano F, Cervino G, Famà F, Calvo A, Crimi S, Rapisarda S, Cicciù M. Facial and orbital fractures: a fifteen years retrospective evaluation of north East Sicily treated patients. Open Dent J. 2017;11:546–56. https://doi.org/10.2174/1874210601711010546.
2. Sundar G. Practical classification of orbital & orbitofacial fractures. In: Swarup A, Wu A, editors. Orbital fractures – principles, concepts & management. New York: Imaging Science Today LLC; 2020. ISBN-13: 978-0997781922.
3. Zingg M, Laedrach K, Chen J, Chowdhury K, Vuillemin T, Sutter F, Raveh J. Classification and treatment of zygomatic fractures: a review of 1,025 cases. J Oral Maxillofac Surg. 1992 Aug;50(8):778–90. https://doi.org/10.1016/0278-2391(92)90266-3.
4. Markowitz BL, Manson PN, Sargent L, et al. Management of the medial canthal tendon in nasoethmoid orbital fractures: the importance of the central fragment in classification and treatment. Plast Reconstr surg. 1991;87:843–53.
5. Rosenberger E, Kriet JD, Humphrey C. Management of nasoethmoid fractures. Curr Opin Otolaryngol Head Neck Surg. 2013 Aug;21(4):410–6. https://doi.org/10.1097/MOO.0b013e3283631936.
6. Manson PN, Hooper JE, Su CT. Structural pillars of the facial skeleton: an approach to the management of Le Fort fractures. Plast Reconstr Surg. 1980;66(1):54–62.
7. Righi S, Boffano P, Guglielmi V, Rossi P, Martorina M. Diagnosis and imaging of orbital roof fractures: a review of the current literature. Oral Maxillofac Surg. 2015;19(1):1–4. https://doi.org/10.1007/s10006-015-0482-9. Epub 2015 Jan 14
8. Attoya KA, Mirvis SE. Radiological evaluation of the craniofacial skeleton. In: Dorafshar AH, Rodriguez ED, Manson PN, editors. Facial trauma surgery – from primary repair to reconstruction. Edinburgh: Elsevier; 2020. ISBN: 978-0-323-49755-8.
9. Udhay P, Bhattacharjee K, Ananthanarayanan P, Sundar G. Computer-assisted navigation in orbitofacial surgery. Indian J Ophthalmol. 2019;67:995–1003.
10. Ross M, El-Haddad C, Deschênes J. Ocular injury in orbital fractures at a level I trauma center. Can J Ophthalmol. 2017;52(5):499–502. https://doi.org/10.1016/j.jcjo.2017.01.013. Epub 2017 Apr 29
11. Faverani LP, Capelari MM, Ramalho-Ferreira G, Gomes-Filho JC, Fabris AL, Marzola C, Toledo GL, Toledo-Filho JL. Ocular reconstruction after zygomatic complex fracture with retention of a foreign body. J Craniofac Surg. 2011 Jul;22(4):1394–7. https://doi.org/10.1097/SCS.0b013e31821cc2e2.
12. Kim JS, Lee BW, Scawn RL, Korn BS, Kikkawa DO. Secondary orbital reconstruction in patients with prior orbital fracture repair. Ophthalmic Plast Reconstr Surg. 2016;32(6):447–51. https://doi.org/10.1097/IOP.0000000000000591.

Traumatic Globe Luxation

19

Wei Zhang, Yanming Huang, Haibo Li, Yuanyuan Liu, and Hua Yan

Abstract

Increased orbital pressure such as trauma, hemorrhage, and tumors drives the eyeball forward and out of the palpebral fissure. Highly prominent eyeballs and blepharospasm are more likely to occur, which is called Globe Luxation, also known as Globe Luxation (luxation of the eyeball) from orbital cavity. Traumatic eyeball luxation is often caused by sudden orbital shocks, violent beats, or a large foreign body acting between the eyeball and the orbit, and the sudden reflex movement of the head in the opposite direction, which causes the eyeball to protrude toward the orbital position. According to the degree of eyeball luxation, it can be divided into semi-luxation and total luxation. Traumatic eyeball luxations are relatively rare in clinical practice, most of which are severe and have a poor prognosis. This chapter aims to explore the pathogenesis, clinical manifestations, and management principles of traumatic Globe Luxation.

Keywords

Traumatic Globe Luxation · Pathogenesis
Emergency · Suturing · Risk factors

19.1 Introduction

Luxation of the eyeball is caused by a variety of reasons that increase the orbital pressure, which drives the eyeball forward and out of the palpebral fissure. The most common cause of globe luxation is a variety of ocular trauma [1]. Other rare causes include thoracic trauma, orbital cavernous hemangioma, intraorbital venous hemangioma, spontaneous bleeding, hemophilia, Engelmann Disease (abnormal backbone development), etc., [2–5]. Traumatic luxation of the eyeball is relatively rare in clinical practice. It is often caused by sudden violent shocks, violent beats, or a large foreign body acting between the eyeball and the orbit, and the sudden reflex movement of the head in the opposite direction, which causes the air entering into the gap of orbital tissues or the orbital fracture site, and the orbital

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pressure suddenly increases, causing the muscles, nerves, and other tissues suddenly break or lose tension, so that the eyeball protrudes to the orbital orifice.

According to the degree of luxation of the eyeball, it can be divided into sub-luxation of the eyeball and total luxation of the eyeball. Sub-luxation of the eyeball refers to the part of the eyeball caught outside the palpebral fissure, and the equator of the eyeball is located outside the vertical line of the palpebral fissure. Total globe luxation means that the eyeball detaches from the orbital orifice, and can even enter the maxillary sinus. The patient may have the history of trauma and no history of primary disease (referring to hemophilia, orbital tumors, or hyperthyroidism, etc.), traumatic ocular luxation can be easily differentiated from diseases such as enlarged eyeballs, Graves ophthalmopathy, and eyelid retraction [6–9].

19.2 Definition

Traumatic luxation of the eyeball is relatively rare in clinical practice. It is often caused by sudden violent shocks, violent beats, or a large foreign body acting between the eyeball and the orbit, and the sudden reflex movement of the head in the opposite direction, which causes the air entering into the gap of orbital tissues or the orbital fracture site, and the orbital pressure suddenly increases, causing the muscles, nerves, and other tissues suddenly break or lose tension, so that the eyeball protrudes to the orbital orifice.

19.3 Case (Brief Case Report Based on the Figures)

A 75-year-old male was brought to the emergency department of our institute, with history of loss of sight for 2 h after being hit by a cart in the right eye. Two hours before admission, the right eye was injured by the hand of a trolley. He immediately suffered severe pain in the right eye, with bleeding, and tears. The vision suddenly dropped to invisible things. No coma, dizziness, headache, nausea, vomiting, etc. Physical examination: the right eye had no light perception, multiple lacerations on the upper and lower eyelids, the eyeball protruded out of the orbit (Fig. 19.1). Computerized tomography (CT SCAN) imaging showed the laceration of optic nerve and hemorrhage behind the eyeball (Fig. 19.2).

After admission, the examination was completed. Under local anesthesia, we performed the right eye sclera debridement suture, extraocular muscle reduction and anastomosis, eyeball reinstatement, eyelid debridement sutured, intraoperative exploration. All four rectus muscles were cut off at 5 mm away from the end of the muscle posteriorly. The rectus muscles were fixed and sutured to the corresponding deep orbital rim. Postoperative anti-inflammatory and infection prevention treatments were performed. Then his condition was getting better (Fig. 19.3).

One day post trauma, the right eye had no perception of light, the cornea was hazy and avascular. The conjunctiva re-epithelized (Fig. 19.4a). The dense vitreous hemorrhage and retinal detachment were shown by ocular B-scan examination (Fig. 19.4b).

One month post trauma, the right eye had no perception of light, the cornea continued to be avascular and hazy (Fig. 19.5a). The dense vitreous hemorrhage and retinal detachment were also shown by ocular B-scan examination (Fig. 19.5b).

The right eye was atrophy three months after trauma (Fig. 19.6a). The right eye was atrophy

Fig. 19.1 A 75-year-old male was brought to the emergency department of our institute, with history of loss of sight for 2 h after being hit by a cart in the right eye

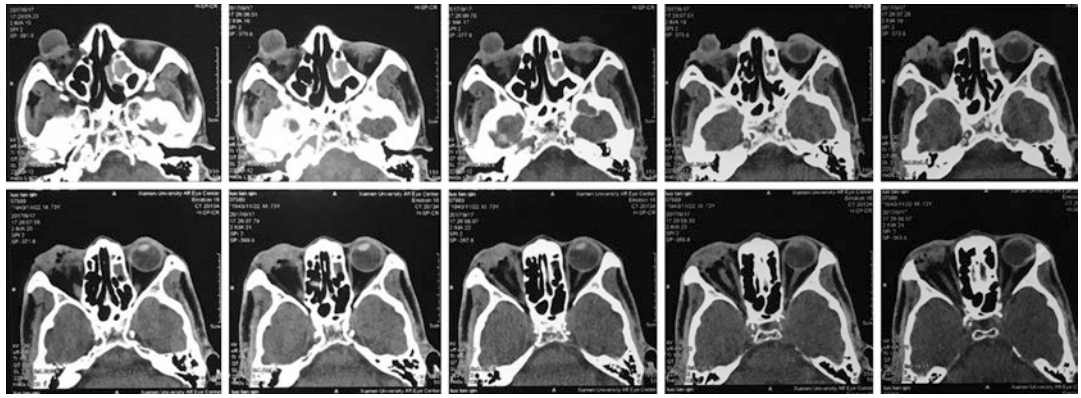

Fig. 19.2 CT scan showed the laceration of optic nerve and hemorrhage behind the eyeball

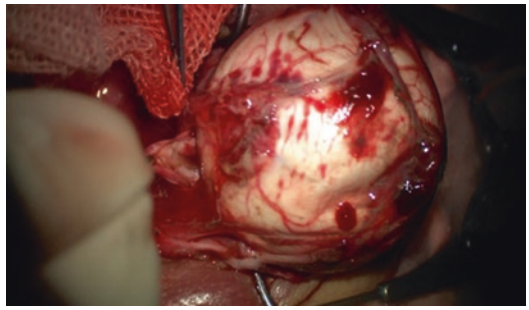

Fig. 19.3 Under local anesthesia, we performed the right eye sclera debridement suture, extraocular muscle reduction and anastomosis, eyeball reinstatement, eyelid debridement sutured, intraoperative exploration. All four rectus muscles were cut off at 5 mm away from the end of the muscle posteriorly. The rectus muscles were fixed and sutured to the corresponding deep orbital rim

was also shown by ocular B-scan examination (Fig. 19.6b).

19.4 Important Signs, Examinations, Diagnosis, Surgical Procedures, or Postoperative Treatment for Complications

19.4.1 Traumatic Eyeball Luxation Examinations and Diagnosis

The treatment principle of eyeball luxation actively deals with the primary disease (such as orbital tumor, frontal sinus tumor, hemophilia,

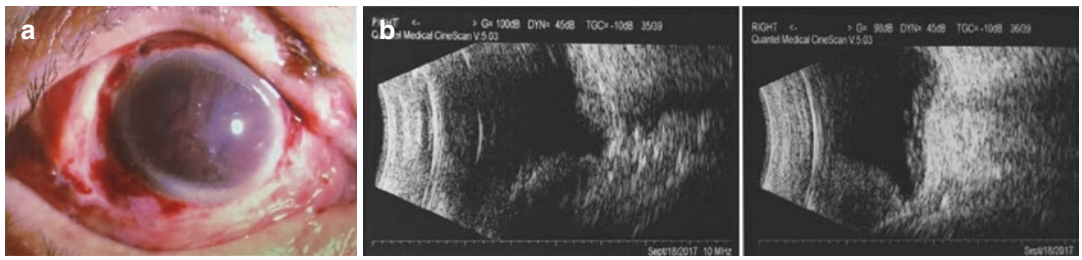

Fig. 19.4 One day post trauma, the right eye had no perception of light, the cornea was hazy and avascular. The conjunctiva re-epithelized (**a**). The dense vitreous hemorrhage and retinal detachment were shown by ocular B-scan examination (**b**)

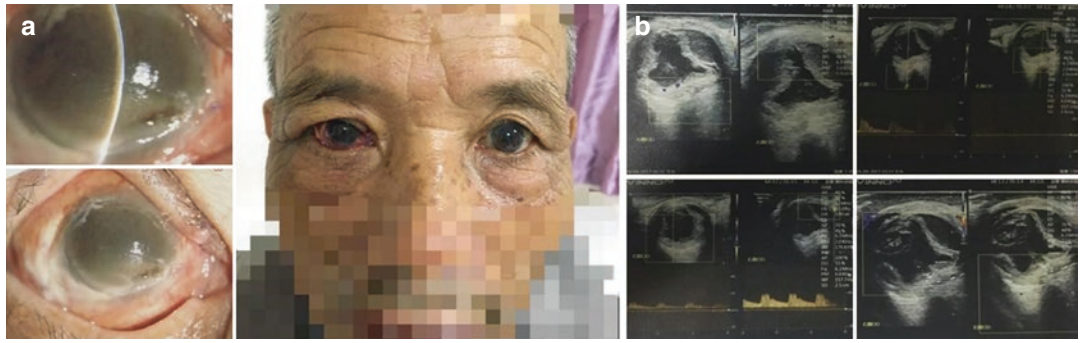

Fig. 19.5 One month post trauma, the right eye had no perception of light, the cornea continued to be avascular and hazy (**a**). The dense vitreous hemorrhage and retinal detachment were also shown by ocular B-scan examination (**b**)

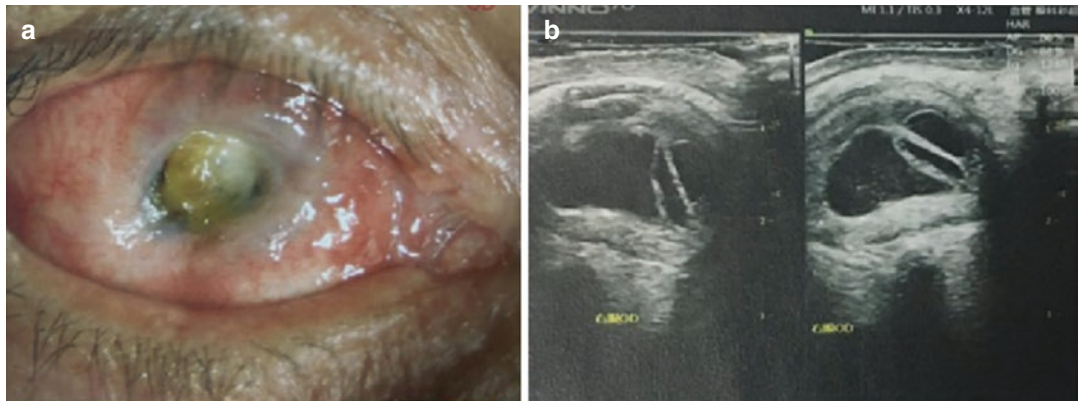

Fig. 19.6 The right eye was atrophy three months after trauma (**a**). The right eye was atrophy was also shown by ocular B-scan examination (**b**)

etc.) [10–12]. For traumatic eyeball luxation, the first is to restore the integrity of the eyeball and actively deal with the scleral laceration that may be combined. The second is to restore the eyeball and make it anatomically reset to reduce tissue edema and damage to the optic nerve. The last is to restore visual function and appearance (such as treatment of strabismus, orbital wall fractures, eyelid deformities, broken lacrimal ducts).

Traumatic luxation of the eyeball is due to the eyeball is highly protruding and the eyeball loses the protective barrier of the eyelid [13–16]. It is necessary to carefully wash and remove foreign bodies, and pay attention to protect the cornea. Globe luxation can be combined with scleral lacerations, and severe scleral lacerations can even be difficult to suture. When the scleral laceration is hidden, the wound is small or located behind, it may be diagnosed by CT or B-ultrasound. Patients with massive hemorrhage under the bulbar conjunctiva, even if the intraocular pressure is normal. Scleral laceration can be combined with intraocular hemorrhage and blood clots in the wound, and is wrapped by the bulbar conjunctiva and eyeball fascia, resulting in possible post-injury intraocular pressure not low or even high. Scleral wound exploration should also be carried out while still receiving the eyeball to confirm the integrity of the eyeball and avoid misdiagnosis.

19.4.2 Surgical Procedures for Traumatic Eyeball Luxation

The method of eyeball restoring can firstly use hypertonic agents (such as mannitol) and hemo-

static agents to reduce intraorbital pressure [17–20]. Partially dislocated eyeballs can be restored by conservative methods; if conservative treatment fails, no compression method or lateral canthal incision method can be used to accommodate eyeballs. Luxation of the eyeball can accompany a fracture of the orbital wall. Subluxation of the eyeball combined with orbital wall fracture, all received the eyeballs restored surgically and the prognosis was poor (no light perception after the operation). Orbital wall fracture could be treated with medication to restore the eyeball, and the temporal light perception could be preserved after the operation, indicating that the luxation of the eyeball combined with the orbital wall fracture may be more severe and the prognosis worse.

Luxation of the eyeball can also be combined with extraocular muscle injury, skin laceration, and canaliculus injured. During the operation, the stump of the extraocular muscles should be found as much as possible and anatomically reduced. If the medial stump is not detected, the muscle can be fixed to the inside of the eyeball fascia sac as far back as possible, so that the eyeball can reach the upright position. Lacrimal canaliculus rupture can decide whether to perform a one-stage anastomosis depending on whether the patient's general condition is stable or not.

An important issue of globe luxation is whether the eyeball is removed. For luxations of the eyeball caused by non-traumatic factors, such as orbital tumors and frontal sinus tumors, a certain degree of vision can be restored after repositioning the eyeballs [20–23]. Therefore, the primary disease should be actively treated. For the total luxation of the eyeball caused by traumatic factors, serious ocular muscle and optic nerve damage will inevitably occur, and even optic nerve avulsion and partial or complete ocular muscle rupture will occur. Therefore, even if the eyeball is successfully repositioned, the visual function and movement of the injured eye will often suffer difficulties, which is also consistent with the pathological examination of the dislocated eyeball. For sub-luxation of the eyeball caused by trauma, the ocular muscles and optic nerve may be damaged to varying degrees, and may also be combined with orbital wall fractures and scleral lacerations.

Ocular sub-luxation was a kind of severe ocular trauma, and combined with orbital wall. In the literature, a considerable proportion of patients can recover useful vision [24–27], the visual acuity of some injured eyes after surgery can reach 1.0 or 1.5, which shows that traumatic luxation of the eyeball does not mean optic nerve avulsion and amaurosis. In addition, the eyeball may have no light perception even for those without scleral laceration. Preserving the eyeball is of great significance to the patient's appearance and psychology, even if to the development of the orbital curettage of children [28–30]. Therefore, the eyeball cannot be easily removed, and the eyeball should be repositioned as much as possible. If the dislocated eyeball has no light perception and is associated with serious infection, the structure of the eyeball is severely damaged, and more than three eye muscles are completely broken. Even if the injured eye is surgically repositioned, it is inevitable that the anterior segment of the eyeball will be ischemia. Therefore, it is inevitable that the dislocated eyeball will be removed.

19.5 Personal Experience or Matters Need Attention

Traumatic globe luxations are mostly caused by violent violence on the orbital wall. It spreads rapidly into the orbital area, and the orbital pressure suddenly rises. This series of mixed mechanical makes the eyeball suddenly undergo a high-speed movement in the opposite direction [30–33]. The muscles, nerves, intraorbital ligaments, fascia, and other tissues which restrict the eyeball movement suddenly rupture or loss of tension, causing the eyeball to protrude toward the orbital. This may be similar to physics hydraulic transmission mechanism: applying a force to an object, using Pascal's original Reason to make this force larger, so as to play the effect of lifting heavy objects. Traumatic globe luxations are often accompanied by serious damage to other tissues of the eye, such as orbital wall fractures, skin splits injury, tear duct injury,

extraocular muscle injury, scleral laceration, optic nerve injury or even broken, and so on. Most traumatic eyeballs with severe damage to the extraocular muscles and optic nerve luxation, even if the reposition of the eyeball is successful, the visual function and movement of the injured eye are often affected and suffer irreversible damage.

The primary problem in the treatment of traumatic luxation of the eyeball is whether the eyeball needs to be removed [34–36]. We believe that the treatment should first protect the cornea and try to accommodate the eyeball, that is preserve the appearance and psychology of the eyeball to the patient, especially for children. The development of the orbit of children is of great significance, so do not remove the eye easily. If the dislocated eyeball has no light perception, combined with severe infection, the eyeball structure is severe damaged, it is inevitable to remove the eyeball. In addition, surgery is required as soon as possible. In summary, the treatment of traumatic luxation of the eyeball should first restore the eyeball. Orthopedics actively deal with scleral lacerations that may merge and try to accommodate the eyeballs to make them anatomical reduction, and then the restoration of visual function and appearance.

References

1. Beshay N, Keay L, Dunn H, Kamalden TA, Hoskin AK, Watson SL. The epidemiology of open globe injuries presenting to a tertiary referral eye hospital in Australia. Injury. 2017;48(7):1348–54.
2. AlDahash F, Mousa A, Gikandi PW, Abu El-Asrar AM. Pediatric open-globe injury in a university-based tertiary hospital. Eur J Ophthalmol. 2020;30(2):269–74.
3. Batur M, Seven E, Esmer O, Akaltun MN, Yasar T, Cinal A. Epidemiology of adult open globe injury. J Craniofac Surg. 2016;27(7):1636–41.
4. Ma J, Zhang Y, Moe MC, Zhu TP, Yao K. Transocular removal of a retrobulbar foreign body and internal patch of the posterior exit wound with autologous tenon capsule. Arch Ophthalmol. 2012;130(4):493–6.
5. Ding X, Liu Z, Lin Y, Yang Y. Perforating ocular fishhook trauma: a case report. Clin Exp Optom. 2018;101(2):297–8.
6. Cui Y, Li Z, Wang Y, Shi L. Removal of an intraorbital metallic foreign body following double-penetrating ocular injury: a case report. Medicine (Baltimore). 2018;97(51):e13790.
7. Chen KJ, Sun MH, Hou CH, Chen TL. Retained large nail with perforating injury of the eye. Graefes Arch Clin Exp Ophthalmol. 2008;246(2):213–5.
8. Žiak P, Mojžiš P, Halička J, Piñero DP. Bilateral perforating eye injury with metallic foreign bodies caused by tire explosion: case report. Trauma Case Rep. 2017;11:20–2.
9. Chee YE, Kanoff JM, Eliott D. Remarkable visual recovery after severe open globe injury. Am J Ophthalmol Case Rep. 2016;3:34–5.
10. Kuhn F, Morris R, Witherspoon CD. Birmingham eye trauma terminology (BETT): terminology and classification of mechanical eye injuries. Ophthalmol Clin N Am. 2002;15(2):139-v.
11. Pieramici DJ, Sternberg P Jr, Aaberg TM Sr, et al. A system for classifying mechanical injuries of the eye (globe). The ocular trauma classification group. Am J Ophthalmol. 1997;123(6):820–31.
12. Yan H, Cui J, Zhang J, Chen S, Xu Y. Penetrating keratoplasty combined with vitreoretinal surgery for severe ocular injury with blood-stained cornea and no light perception. Ophthalmologica. 2006;220(3):186–9.
13. Zhou Y, You C, Wang T, et al. Anastalsis of triamcinolone acetonide during vitrectomy in proliferative diabetic retinopathy. Chin J Exp Ophthalmol. 2017;35(5):439–42.
14. Kuhn F, Schrader W. Prophylactic chorioretinectomy for eye injuries with high proliferative-vitreoretinopathy risk. Clin Anat. 2018;31(1):28–38.
15. Ferreira N, Monteiro S, Meireles A, Kuhn F. Outcome of vitrectomy and chorioretinectomy in perforating eye injuries. Ophthalmic Res. 2015;53(4):200–6.
16. He T, You C, Chen S, Meng X, Liu Y, Yan H. Secondary sulcus-fixed foldable IOL implantation with 25-G infusion in patients with previous PPV after open-globe injury. Eur J Ophthalmol. 2017;27(6):786–90.
17. Ung C, Stryjewski TP, Eliott D. Indications, findings, and outcomes of pars Plana vitrectomy after open globe injury. Ophthalmol Retina. 2020;4(2):216–23.
18. Yu H, Li J, Yu Y, et al. Optimal timing of vitrectomy for severe mechanical ocular trauma: a retrospective observational study. Sci Rep. 2019;9(1):18016.
19. Zheng L, Tan J, Liu R, et al. The impact of primary treatment on post-traumatic endophthalmitis in children with open globe injuries: a study in China. Int J Environ Res Public Health. 2019;16(16):2956.
20. Chaudhry IA, Shamsi FA, Al-Harthi E, Al-Theeb A, Elzaridi E, Riley FC. Incidence and visual outcome of endophthalmitis associated with intraocular foreign bodies. Graefes Arch Clin Exp Ophthalmol. 2008;246(2):181–6.
21. Opneja A, Kapoor S, Stavrou EX. Contribution of platelets, the coagulation and fibrinolytic systems to cutaneous wound healing. Thromb Res. 2019;179:56–63.

22. Yonekawa Y, Chodosh J, Eliott D. Surgical techniques in the management of perforating injuries of the globe. Int Ophthalmol Clin. 2013;53(4):127–37.
23. Kuhn F. Perforating injuries. Ocular traumatology. New York: Springer; 2008. p. 391–403.
24. Alfaro DV, Tran VT, Runyan T, Chong LP, Ryan SJ, Liggett PE. Vitrectomy for perforating eye injuries from shotgun pellets. Am J Ophthalmol. 1992;114(1):81–5.
25. Iqbal M, Charteris DG, Cooling RJ, Mackintosh GI. Conservative management of double penetrating ocular injuries. Eye (Lond). 2000;14(Pt 2):249–51.
26. Abdullatif AM, Macky TA, Abdullatif MM, et al. Intravitreal decorin preventing proliferative vitreoretinopathy in perforating injuries: a pilot study. Graefes Arch Clin Exp Ophthalmol. 2018;256(12):2473–81.
27. Mohamed AA. Vitrectomy in double-perforation gunshot injury. Clin Ophthalmol. 2013;7:2219–24.
28. Fulcher TP, McNab AA, Sullivan TJ. Clinical features and management of intraorbital foreign bodies. Ophthalmology. 2002;109(3):494–500.
29. Ho VH, Wilson MW, Fleming JC, Haik BG. Retained intraorbital metallic foreign bodies. Ophthalmic Plast Reconstr Surg. 2004;20(3):232–6.
30. Al-Mujaini A, Al-Senawi R, Ganesh A, Al-Zuhaibi S, Al-Dhuhli H. Intraorbital foreign body: clinical presentation, radiological appearance and management. Sultan Qaboos Univ Med J. 2008;8(1):69–74.
31. Green BF, Kraft SP, Carter KD, Buncic JR, Nerad JA, Armstrong D. Intraorbital wood. Detection by magnetic resonance imaging. Ophthalmology. 1990;97(5):608–11.
32. Cartwright MJ, Kurumety UR, Frueh BR. Intraorbital wood foreign body. Ophthalmic Plast Reconstr Surg. 1995;11(1):44–8.
33. Sullivan TJ, Patel BC, Aylward GW, Wright JE. Anaerobic orbital abscess secondary to intraorbital wood. Aust N Z J Ophthalmol. 1993;21(1):49–52.
34. Nasr AM, Haik BG, Fleming JC, Al-Hussain HM, Karcioglu ZA. Penetrating orbital injury with organic foreign bodies. Ophthalmology. 1999;106(3):523–32.
35. Colyer MH, Chun DW, Bower KS, Dick JS, Weichel ED. Perforating globe injuries during operation Iraqi freedom. Ophthalmology. 2008;115(11):2087–93.
36. Feng K, Shen L, Pang X, et al. Case-control study of risk factors for no light perception after open-globe injury: eye injury vitrectomy study. Retina. 2011;31(10):1988–96.

Complicated Blepharal Trauma

20

Juan Ye and Jiajun Xie

Abstract

As the key function of an eyelid is to protect the eye from drying out and from injury, blepharal involvement is common in ophthalmic trauma. Complicated blepharal trauma poses frustrating challenges not only to diagnosis but for management as well. Some injuries are insidious and can be missed if not thoroughly examined. Adaptation of general-principle is important for optimal treatment in unique cases [1]. This chapter includes the essentials of approaches of evaluation and treatment of marginal eyelid laceration, canalicular lacerations, eyelid foreign bodies, eyelid burns, and traumatic ptosis. Special attention is addressed toward avoidance of complications.

Keywords

Eyelid laceration · Canalicular laceration
Eyelid foreign body · Eyelid burn
Traumatic ptosis

20.1 Full-thickness Eyelid Laceration

20.1.1 Introduction

Full-thickness eyelid lacerations are most commonly associated with trauma to the entire orbital area. Since the skin of the eyelid has little underlying subcutaneous fat, it is susceptible to contracture if not well treated after a traumatic injury. Thus, well management in the acute phase is essential to anatomical and functional restoration of a full-thickness eyelid laceration.

20.1.2 Definition

Eyelid lacerations is the commonest blepharal trauma due to blink reflex as a protective mechanism. The extent of eyelid laceration can vary greatly. Scar formation in the sub-acute phase during the healing process can lead to lagophthalmos, eyelid malposition, and blepharon deformities. The key to satisfactory repair of a full-thickness eyelid laceration is precise reapproximation of the eyelid margin.

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20.1.3 Important Signs, Examinations, Diagnosis, Surgical Procedures and Skills, or Postoperative Treatment for Complications

The extent of eyelid laceration is variable from minor to major. Trauma history is important to help to estimate the existence of globe and orbit injuries. The involvement of the lacrimal system must be evaluated, especially when the medial eyelid is affected. CT scanning is required if other injuries or foreign bodies are suspected.

Local anesthesia generally offers satisfactory effect unless special cases with poor patient cooperation, such as children, or patient who is unconscious. Because of the sufficient blood supply, the eyelid tissue is usually allowed to be preserved even if it seems devitalized. Thus, tissue debridement is not recommended in eyelid laceration. Copious irrigation and meticulous closure of the wound are promptly required.

The key to satisfactory repair of a full-thickness eyelid laceration is precise reapproximation of the tarsal plate [2]. The laceration of the tarsal plate itself is usually linear. Expose the tarsal plate with skin hooks to retracted the anterior lamella, then apposed the tarsal plate with interrupted 5-0 Vicryl sutures without passing full-thickness through the posterior surface of the eyelid. The placement of the lid margin suture is performed next. Skin and orbicularis are closed with 6-0 sutures. Make sure the lid margin is slightly eversion after the closure of the wound to allow scar contracture during the healing process.

Tetanus immunization must be up to date. Antibiotic ointment is instilled and a patch may be applied. The skin and marginal sutures can be removed at 7–10 days. Skin scars formed during the process can be managed almost after 6 months when they are stable.

20.1.4 Case 1

A 28-year-old female came to the oculoplastic clinic with the chief complaint of improving her appearance of the lower eyelid in the left eye. She had an eyelid laceration in a traffic accident one year ago and underwent wound management immediately after the trauma. Figure 20.1 shows a cicatricial contracture in her left lower eyelid. The cicatricial contracture in the left eye was released, and the morphology of the lower eyelid was restored. She was satisfied with the results of the reconstruction surgery (Fig. 20.2).

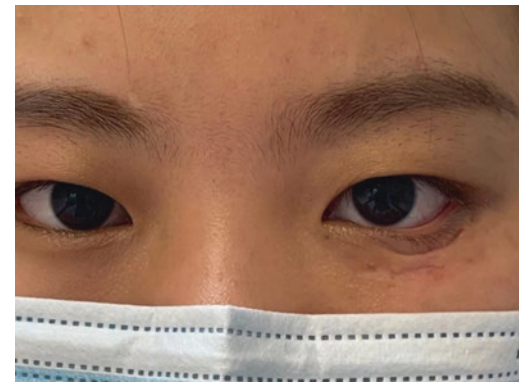

Fig. 20.1 The cicatricial contracture in the lower eyelid of the left eye one year after an eyelid laceration and surgical suture. A transverse notch is along the lid margin which caused lower lid ectropion

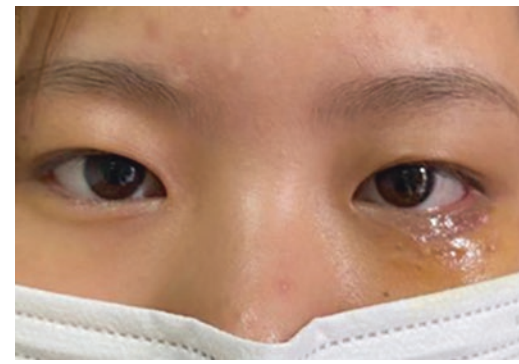

Fig. 20.2 The cicatricial contracture was released, and the lower eyelid of the left eye was reconstructed in a repair surgery, with satisfactory results

20.2 Canalicular Laceration

20.2.1 Introduction

Injury of the lacrimal drainage system, usually the canaliculus, is often encountered with medial eyelid trauma. Since the dense fibrous tissue of

the tarsal plate is significantly stronger than the medial canthal tendon, any avulsing force placed along the lid margin is more likely to tear the medial soft tissue and result in damage to the lacrimal canaliculus [3].

20.2.2 Definition

Any cut or tear in the medial portion of the upper or lower eyelid must be evaluated for a canalicular laceration, no matter how superficial or minor it appears. Probing of the canalicular system when canalicular laceration is suspected.

20.2.3 Case 2

A 42-year-old female reported with a history of trauma to her right eye by a stick 2 h back. Figure 20.3 Shows a 0.8 cm laceration in the medial portion of the lower eyelid, with the lower canaliculus and medial canthus ligament involved. Visual acuity in the right eye was 20/20. No specific finding of the eyeball was observed after the slit-lamp examination. A bicanalicular silicone tubing intubation was performed to repair the lower canaliculus (Figs. 20.4 and 20.5). The medial canthus tendon was adequately reapproximated, and laceration was interrupted sutured with 6-0 Vicryl. In this case, the tubing is left in the lacrimal system for 3 months.

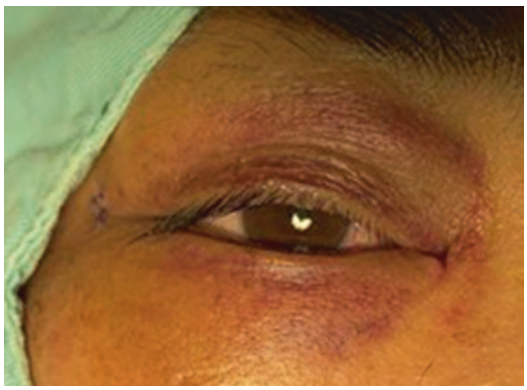

Fig. 20.3 Canalicular laceration. Any cut or tear in the medial portion of the upper or lower eyelid should be presumed to involve the lacrimal canaliculi. In this case, the laceration involved the lower canaliculi although the cut was minor

20.2.4 Important Signs, Examinations, Diagnosis, Surgical Procedures and Skills, or Postoperative Treatment for Complications

The medial eyelid is the weakest area of the eyelid. Any medial eyelid laceration should be presumed to involve the lacrimal canaliculi, no matter how superficial or minor it appears. Both upper and lower lacrimal canaliculi can be

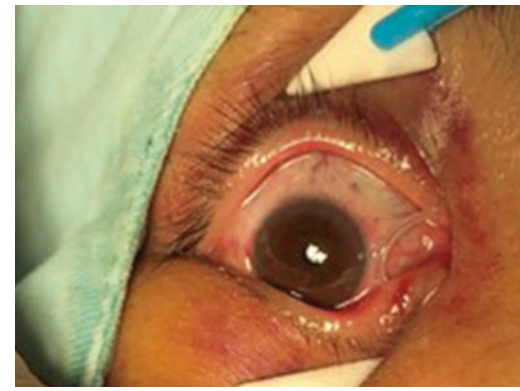

Fig. 20.4 A bicanalicular silicone tubing intubation was performed to repair the lower canaliculus. The ends of the silicone tubing in the nose were tied to form a continuous loop

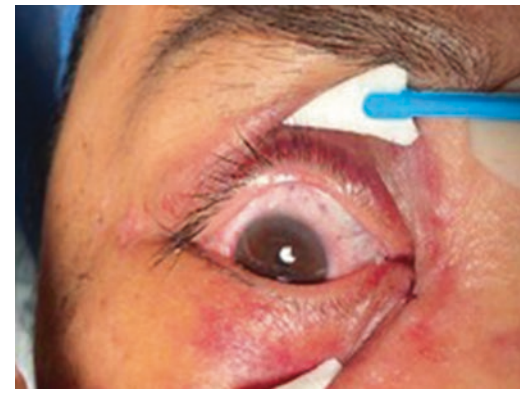

Fig. 20.5 The tension of the silicone tubing was adjusted with care, avoiding subsequent punctal erosion

involved, since trauma history varied from blunt force, dog bites, to sharp objects. Clear trauma history and probing of the lacrimal system help to make the diagnosis.

Primary surgical repair with anastomosis of the canalicular ends and intubation of the lacrimal system is preferred. The key to satisfactory repair of a canalicular laceration is to identify the distal (medial) cut end of the canaliculus. The farther the laceration is medially from the lacrimal puncta, the more difficult it is to locate the distal cut end. Loupes or an operating microscope are usually required.

Depending on the severity of the injury and the patient's cooperation, general or nerve block anesthesia is preferred. A well-performed infraorbital nerve blockage prevents patient discomfort during the operation and reduces the need for local anesthetic, which may result in tissue swelling and distal end of the canaliculus being obscured.

With dry operative field and sufficient exposure, the distal cut end of the canaliculus is usually possible to be identified. A bicanalicular silicone tubing intubation is preferred in our experience. The silicone tubing serves to align the cut ends and maintain the lumen during the healing phase. The silicone tubing was first passed through the punctum and proximal segment of the injured canaliculus, then introduced through the identified distal cut end and advanced until it abuts the medial wall of the lacrimal sac. Then similarly incubate the silicone tubing through the opposing canaliculus. Once the laceration has been bridged, special attention should be paid to exactly reapproximate the medial canthal tendon structures when dealing with the deeper and more medial laceration. Closure of the canaliculus is then carried out using 8-0 Vicryl interrupted sutures without penetrating the canalicular epithelium. After reformation of the canaliculus and the medial canthal tendon structures, the overlying skin orbicularis can be closed with interrupted fine sutures. The ends of the silicone tubing in the nose are tied to form a continuous loop, taking care to adjust the tension to avoid subsequent punctal erosion.

The silicone tubing is usually left in place for 6 weeks to 6 months, depending on the severity and the individual practition.

20.3 Eyelid Foreign Body

20.3.1 Introduction

Foreign bodies of the eyelid can have a diverse range of clinical presentations, depending on the size and feature of the objects, as well as the position of the affected area. Careful examination and appropriate treatment are essential to reduce complications and avoid secondary injury.

20.3.2 Definition

Foreign bodies of the eyelid are usually followed with blepharal trauma, most of which is obvious while sometimes is latent. The foreign bodies can be variable, including stone, metal, wood, and rarely, other objects [4]. Detailed present history, ocular examination, and CT or ultrasound scan all can be of diagnostic usefulness.

20.3.3 Case 3

A 43-year-old male presented with the chief complaint of trauma to his left eye by a fishhook occurring approximately 2 h back. To avoid secondary injury, the artificial bait was tied and reinforced to his forehead with adhesive tape during transferring. The eyeball was luckily not involved (Fig. 20.6). After meticulous evaluation, the bait was retrieved with forceps, and the wound was irrigated and closed under local anesthesia (Fig. 20.6).

20.3.4 Case 4

A 34-year-old female showed blepharon deformity with a pink scar in the left upper eyelid. She had a traffic accident and underwent immediate debridement and suturing operation 3 months

Fig. 20.6 The artificial bait was present on the patient's left eyelid. The band and shank were outside the eyelid, while the point was not visible. After retracting the upper eyelid, the eyelid was found not perforated. Visual acuity in the left eye was 20/20. No specific finding of the eyeball was observed after the slit-lamp examination

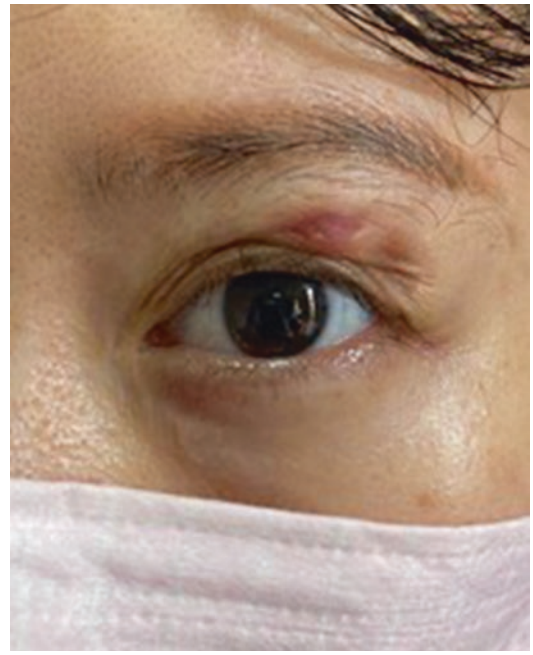

Fig. 20.7 A patient who had a history of trauma and debridement operation showed blepharon deformity with a pink scar in the left upper eyelid. A firm lump with a size of 4 mm*6 mm could be felt inferior to the left brow

prior to her visit. At the first examination, we felt a firm lump inferior to the left brow (Fig. 20.7). Further CT scan also revealed a positive retained foreign body in the left upper eyelid (Fig. 20.8). The patient did not show ptosis at that time. The mass was removed via a skin incision under local anesthesia. Six months after the removal of the glass, the patient did not show any complications, including ptosis (Fig. 20.9).

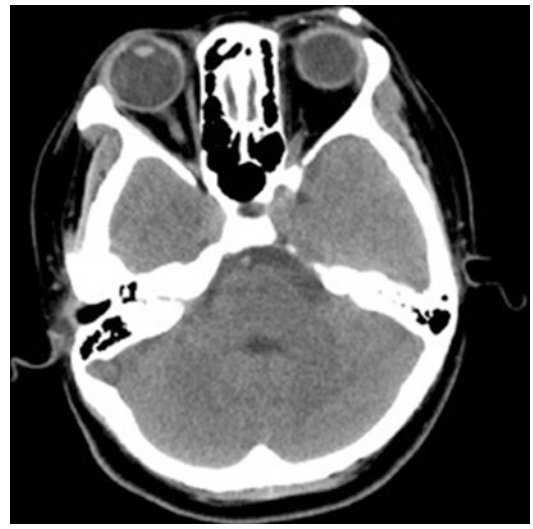

Fig. 20.8 Further CT scan confirmed a positive retained foreign body in the left upper eyelid, with a clear edge and no artifacts

20.3.5 Important Signs, Examinations, Diagnosis, Surgical Procedures and Skills, or Postoperative Treatment for Complications

According to the slit-lamp examination and auxiliary examination, combined with the ocular trauma history, the general diagnosis of eyelid foreign body is not difficult. However, a foreign body can be insidious in some cases. It is of the

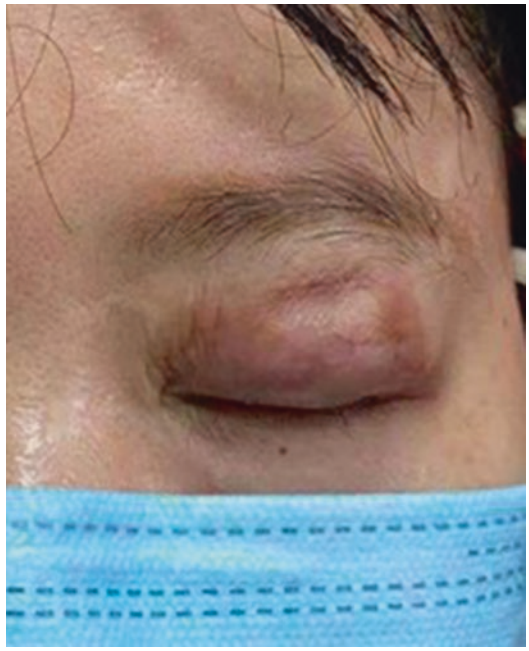

Fig. 20.9 After incising the skin, releasing the scar, and sufficiently isolating, a piece of glass was found without the fibrous capsule or granulation tissue above the orbital septum. The patient did not show any complications one week post the operation, including ptosis

most importance to not missing concealed or small foreign bodies. Retained foreign bodies following ocular injury may lead to serious complications, including inflammation or infection.

Foreign body located in the subconjunctival space is rare while most likely to be missed, for example, rigid gas permeable (RGP) contact lens had been reported to migrate into subconjunctival space superior to the upper tarsus after blunt blepharon trauma [4]. Special attention should be paid in cases of penetrated eyelid trauma. Take extra effort to carefully inspect the corresponding site on the eyeball to the perforation site on the eyelid to exclude any foreign body on the cornea, conjunctiva, or sclera. In cases where foreign bodies are suspected, imaging methods such as CT and ultrasonography should additionally be performed to identify the size, property, and location.

Local anesthesia usually provides satisfactory patient cooperation in cases of superficial eyelid surgery. A prompt, appropriate primary surgical intervention is crucial for a good outcome with fewer complications including blepharon deformities and traumatic ptosis. The technique should be carefully chosen by taking into account the type, the depth, and the relationship between the foreign body and related ocular tissue. In case 2, we used an eyelid plate to protect the eyeball and a back-out technique to remove the fishhook after enlarging the entry wound to avoid secondary injury during the operation. In cases with deep and contaminated wounds, copious irrigation is the most effective treatment to decrease the risk of infection. Tetanus immunization must be up to date.

20.4 Eyelid Burns

20.4.1 Introduction

Eyelid burns are usually associated with burns over a large percentage of the body unless they are electrical or chemical. Compared to the initial ocular surface injury, sustained eyelids injury can be more challenging, which leads to secondary complications such as lagophthalmos, exposure to keratopathy, and trichiasis. Appropriate early and sustained management is vital to improve the prognosis.

20.4.2 Definition

Eyelid burns are generally associated with other facial or body burns unless the etiology is electrical or chemical. Facial burns are commonly caused by thermal trauma with ocular involvement. Burns of the eyelid vary in depth and severity. All burns take days to weeks for full tissue death and necrosis. With time, the eyelids will scar, resulting in poor closure and more corneal exposure. Reconstruction can be very difficult because of poor vascularization and lack of normal tissue.

20.4.3 Case 5

A 55-year-old male presented with right upper lid burns, total corneal epithelial cell loss with stromal edema, and early corneal neovascularization

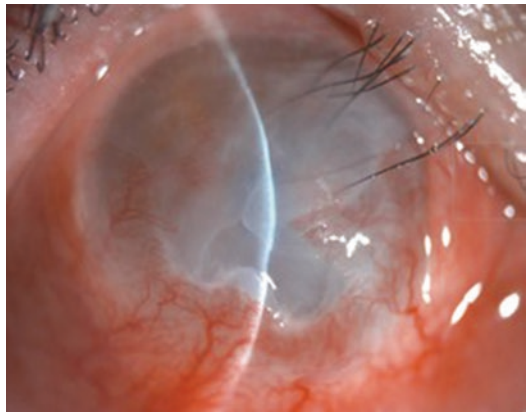

Fig. 20.10 A 55-year-old male presented with right upper lid burns, total corneal epithelial cell loss with stromal edema, and early corneal neovascularization 13 days after molten aluminum burns to his right eye and lids. There was no view of the anterior chamber. Visual acuity in the right eye was counting fingers

13 days after molten aluminum burns to his right eye and lids (Fig. 20.10). There was no view of the anterior chamber. Visual acuity in the right eye was counting fingers. Besides medicine, an amniotic membrane graft and lateral tarsorrhaphy were performed to encourage healing of the epithelial defect and prevent further injury caused by secondary trichiasis. Surgical intervention for trichiasis of the upper eyelid is required when scar formation stabilized.

20.4.4 Important Signs, Examinations, Diagnosis, Surgical Procedures and Skills, or Postoperative Treatment for Complications

20.4.4.1 Evaluation

Most ocular complications are secondary to the development of eyelid deformities, which are potentially preventable with adequate and prompt initial treatment [5]. The depth and extent of injury should be assessed as soon as possible for patients with facial burns. The depth of burn depends on the intensity and duration of exposure, and the thickness of the epidermis and dermis. Since periocular skin is thin with no subcutaneous fat, burns can be deeper in these areas. It takes weeks for the total amount of tissue necrosis to manifest with electrical burns, while less time with thermal burns.

Special attention should be addressed to exclude any eyelid, ocular, intraocular, or intraorbital foreign body especially in cases of explosion injury. Bell's phenomenon should be documented as a protective mechanism to help to prevent corneal epithelial defect in the presence of lagophthalmos.

20.4.4.2 Initial Treatment

Head elevation may be helpful in preventing further swelling around the eyelids. The basic principles of wound management for eyelid burns are assessment, cleansing, and protection followed by re-surfacing for deeper burns. Singed or scorched eyelashes are recommended to be removed with fine scissors to relieve symptoms of ocular surface discomfort as well as conjunctival hyperemia. The eyelids should be cleaned of any debris as soon as possible. Foreign bodies should be removed with copious irrigation and cotton swabs, including careful sweeping of conjunctival fornix, if any.

Burn patients often have reduced tear production, blink reflex, and eyelid mobility or excursion. Thus, the first concern is to protect the cornea with lubrication during the initial phase. Prompt prophylactic lubrication appears to have a beneficial effect in preventing exposure to keratitis. Topical steroids should be avoided due to the risk of secondary infection.

In cases of developing epithelial defect and poor closure or poor Bell's phenomenon, a temporary suture tarsorrhaphy is preferred to improve eyelid closure and allow examination of the eye as well as topical installation. A bolster is used to support the eyelid and reduce the tension. If ectropion persists for months due to persistent eyelid shrinkage, a surgical tarsorrhaphy would need to remain the lid margin in place until scars mature for definitive ectropion repair.

In severely damaged eyelids where no viable tissue exists, the masquerade procedure can be carried out to close the eye until further reconstruction can be accomplished. All necrotic tis-

sues are excised, a conjunctival flap is mobilized and sutured together to cover the ocular surface. A skin graft is then applied to cover the entire eyelid area, leaving a small gap nasally and temporarily for tear drainage. The flap is divided horizontally approximately 1–3 months later to create new functioning lids.

20.4.4.3 Complications

As the burns heal, cicatricial changes become more prominent. Common complications include trichiasis, eyelid deformities, palpebral aperture stenosis. Severe burns may require multiple surgeries and staging skin grafts for eyelids and ocular surface reconstruction.

20.5 Traumatic Ptosis

20.5.1 Introduction

Traumatic ptosis cases can result from a broad range of insults including direct muscular injury, mass effect from scars or foreign bodies, and cranial nerve damage. Appropriate intervention and timing according to the mechanism of ptosis will often contribute to a better prognosis.

20.5.2 Definition

A decreased height of the upper lid after an injury is considered as a "traumatic ptosis," which is the second most common relative incidence of blepharoptosis subtypes. Based on the mechanism of injury and ophthalmic findings, traumatic ptosis can be divided into subcategories as traumatic aponeurotic ptosis, traumatic myogenic ptosis, traumatic mechanical ptosis, traumatic neurogenic ptosis, and traumatic mixed mechanism ptosis. According to the mechanism and severity of the injury, traumatic ptosis may be transient or permanent, the degree of which may range from mild to severe.

20.5.3 Important Signs, Examinations, Diagnosis, Surgical Procedures and Skills, or Postoperative Treatment for Complications

Traumatic aponeurosis ptosis is caused by dehiscence of the levator aponeurosis from the tarsal without severe injury of the levator, when the upper lid is pulled, stretched or lacerated. The degree of ptosis is usually mild, with fine levator function [6].

Traumatic myogenic ptosis is caused by direct injury to the levator and/or Müller's muscle when a transverse laceration occurs in the upper lid. The degree of ptosis is mild to severe, with poor levator function.

Traumatic mechanical ptosis often results from scar tissue that either restricts lid excursion or creates a mass effect that weighs the lid down after an injury.

Traumatic neurogenic ptosis can occur secondary to third nerve injury, superior orbital fissure syndrome, or traumatic facial nerve palsy, which may be accompanied by symptoms such as oculomotor defect and abnormality of the pupils.

Traumatic mixed mechanism ptosis exists for that a dominant subtype cannot be identified, which mostly is a combination of myogenic and mechanical when the upper lid is lacerating traversed with direct levator injury and skin cicatricial tethered.

The prognosis and management of traumatic ptosis depend on the underlying mechanism of injury [7]. Since partial spontaneous improvement was appreciable in most cases of traumatic ptosis except for the neurogenic subgroup, adequate time should be allowed for improvement prior to surgical intervention. It is recommended to evaluate and operate under local anesthesia to obtain better control of the muscular function with the patient's cooperation. The observation

period without surgical intervention is usually 6 months in traumatic aponeurotic ptosis cases for spontaneous improvement, as well as in traumatic myogenic, mechanical and mixed mechanism ptosis cases for scar tissue stabilization. In traumatic neurogenic cases, surgery can be performed if no progressive improvement is observed, otherwise wait until 6 months. Levator advancement or frontalis sling is recommended to improve lid height in traumatic ptosis cases depending on levator function and eyeball protection mechanism, including oculomotor and Bell's phenomenon. Special attention should be addressed to some accompanying symptoms, for example, unbearable double vision caused by extraocular muscle injury should be treated prior to ptosis surgery.

20.6 A Special Paragraph to Discuss the Specific Challenges

The first concern when dealing with blepharal trauma is to perform a complete eye examination to rule out associated intraocular trauma. CT scanning is required to identify foreign bodies or fractures. If the medial eyelid is affected, the lacrimal system should be inspected for evidence of involvement. Because of the good blood supply, the eyelid and periocular tissue need not be debrided even apparently devitalized, as it will usually survive with appropriate management. Make sure of the status of the patient's tetanus immunity.

In some complicated blepharal cases, multiple and staged surgeries may be required for eyelid morphological and functional reconstruction.

References

1. Tse D. Trauma. In Color Atlas of Oculoplastic Surgery. 2nd ed. Philadelphia, PA: Wolters Kluwer; 2011. p. 22–8.
2. Beadles KA, Lessner AM. Management of traumatic eyelid lacerations. Semin Ophthalmol. 2009;9(3):145–51.
3. Robert BP. Eyelid Trauma. In: Color atlas & synophsis of clinical ophthalmology. 2nd ed, Chapter 4. Philadelphia, PA: Wolters Kluwer; 2012. p. 48–55.
4. Kang H, Takahashi Y, Kakizaki H. Migration of rigid gas permeable contact lens into the upper eyelid after trauma: a case report. BMC Ophthalmol. 2016;16(1):1–3.
5. FRCOphth RM, FRCS IS, and FRCS BD. The Management of Eyelid Burns. Survey of Ophthalmology. 2009;54(3):356–71.
6. Jacobs SM, Tyring AJ, Amadi AJ. Traumatic ptosis: evaluation of etiology, management and prognosis. J Ophthalmic Vis Res. 2018;13(4):447–52.
7. Boyle NS, Chang EL. Traumatic blepharoptosis. In: Evaluation and management of blepharoptosis. 3rd ed, Chapter 9. New York: Springer; 2011. p. 129–40.

Severe Ocular Chemical Injury

21

Weiyun Shi and Ting Wang

Abstract

Ocular chemical burns, usually caused by acids or alkalis, are serious emergencies in the eyes. The burns can bring about acute loss of vision and damage to the eye and may eventually result in severe visual and facial defects. The concentration of the chemical, the exposure area, and the duration before treatment are the key factors relating to the severity of the injury and the prognosis. In this chapter, the authors discussed different kinds of chemical injuries, especially alkali burns and acid burns, as well as their damage to the eyes and the possible treatment measures. In addition, fives classic cases are presented with brief descriptions, images, and management measures, aiming to provide a guide to the future diagnosis and treatment of ocular chemical injuries.

Keywords

Ocular chemical injury · Acid · Alkalis
Diagnosis and treatment · Keratoplasty

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21.1 Introduction

Ocular chemical injuries are true ophthalmic emergencies that can cause permanent corneal and intraocular damage leading to visual impairment or even blindness [1, 2] (Figs. 21.1 and 21.2). The list of responsible chemicals is very extensive including industrial and domestic cleaning agents, cement, plaster, fertilizers, lime, and fireworks [2]. Acids and alkalis are the agents most commonly associated with significant chemical eye injuries and the severity of the injury depends on a number of factors including (1) concentration and pH of the solution, (2) the extent of ocular surface exposure, and (3) the duration of ocular exposure before treatment is instigated [3].

Common causes of alkali injury to the eye include: cement, ammonia, lye, lime, potassium

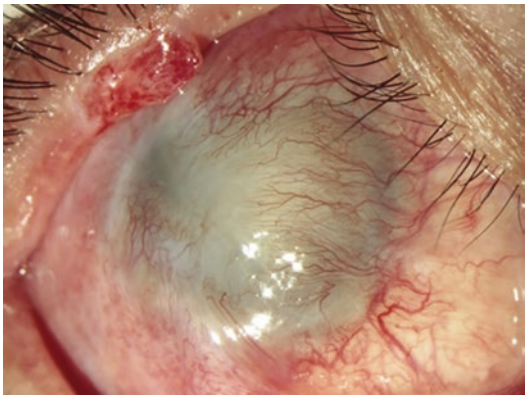

Fig. 21.1 Blindness caused by severe acid burns

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https://doi.org/10.1007/978-981-16-5340-7_21

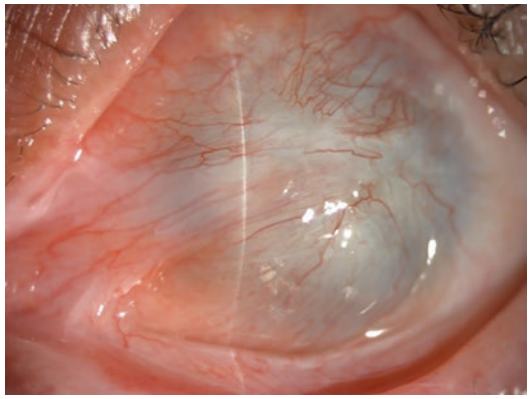

Fig. 21.2 Blindness caused by severe alkali burns

hydroxide, and magnesium hydroxide [4]. Stronger alkalis are associated with more rapid penetration and thus, higher pH agents such as ammonia and lye typically cause the most severe ocular injuries. pH changes of aqueous humor are observed within a few seconds after contact with ammonium hydroxide, and within 3–5 min after injury by sodium hydroxide [5, 6]. Fireworks may contain magnesium hydroxide, thus related ocular injuries are both chemical and thermal in nature.

Chemical injuries to the eye are associated with a wide range of acids, most commonly sulfuric, sulfurous, hydrofluoric, acetic, chromic, and hydrochloric acids [2]. The strength of an acid depends on its ability to lose a proton; strong acids ionize completely in an aqueous solution. The ability of a chemical to penetrate the eye influences the severity of the subsequent ocular injury. Alkalis characteristically penetrate the eye more rapidly than acids [2, 5] and typically cause the most severe chemical injuries. However, very strong acids may penetrate as rapidly as alkalis and studies have shown no clinically significant differences in clinical course, and prognosis between severe acid and alkali burns [7].

21.2 Definition

21.2.1 Types of Chemical Injuries

1. Acids: sulfuric acid, hydrochloric acid, nitric acid, phosphoric acid, etc.
2. Alkalis: (1) alkali metals: lithium, sodium, potassium, rubidium, cesium, and francium. (2) alkaline earth metals: beryllium, magnesium, calcium, strontium, barium, and radium. (3) Ammonia: ammonium hydroxide, ammonium chloride, etc.
3. Nonmetallic corrosive agents, such as phosphorus and its chemical compounds.

21.2.2 Factors Influencing the Severity of Eye Chemical Injuries

The severity and prognosis of ocular chemical injuries are related to many factors, mainly including the nature of the chemical substance, the area of the injured region, and the prompt and appropriate first aid treatment.

1. The time and range of chemical substances in the eye: Generally speaking, the longer the chemical substances become stuck in the eye, the greater the damages they will cause to the eye. The injured area will be larger and the damages to ocular tissues will be worse.
2. Types of chemical substances and their properties: The penetration of acids and alkalis into the eye tissue is different because of their different chemical properties, so the degrees of ocular damages and prognosis are not the same. Alkali burns are far more harmful than acid burns.
3. The severity is also related to the concentration of chemicals and the resistance of different parts of the eye to acid–base substances.

21.2.3 The Main Pathological Lesions in the Late Stage of Anterior Segment Chemical Injuries

1. Conjunctival damage: The damage of the conjunctiva is mainly the massive destruction of stem cells and goblet cells in the limbal of the cornea, causing the normal conjunctival epithelium to be replaced by fibroblasts with

abnormal hyperplasia. The conjunctiva loses its characteristics as normal translucent smooth mucosal tissue and is replaced by a large amount of hypertrophic fibrous connective tissue without normal conjunctival function. The loss of conjunctival goblet cells results in the destruction of the tear film. For example, when the area of necrosis in the conjunctiva exceeds half of the limbal stem cells, the regeneration of corneal epithelium is slow, and the conjunctivalization of the cornea may occur, which is characterized by the opacity of the repaired epithelium and the presence of a large number of goblet cells and corneal pannus. In this case, abnormal goblet cells of the cornea can cause instability of the cornea, leading to abnormal conjunctiva of the ocular surface, even if the tear volume is normal.

2. Corneal damage: The corneal epithelium can suffer the same damage as conjunctival epithelial tissue. The corneal epithelium, after contact with alkaline substances, will be destroyed and come off, so the area of the epithelial defects can directly reflect the location and area of corneal burns. The burn damages the corneal stroma lamellar collagen tissue and the abnormal immune response intensifies the destruction. Eventually, the epithelium is replaced by fibrous scar tissues. The loss of a large number of endothelial cells leads to a decline in the normal physiological functions, resulting in persistent corneal edema and new blood vessels ingrowth. In severe cases, the cornea may be completely vascularized and covered by tunica vasculosa tissue.
3. Destruction of tear film: Due to the destruction of conjunctival goblet cells, accessory lacrimal glands (Krause's glands and Wolfring's glands) and the corneal epithelium, the formation of the tear film is difficult. Under the fornix conjunctiva, there are accessory lacrimal glands with tear secretion function. Some or all the lacrimal gland openings are damaged due to adhesion and scar formation; therefore, the amount of tear secretion is significantly reduced and the eye is substantially dry, which is manifested as Schirmer tear secretion less than normal, unsuccessful formation of the tear film, abnormal tear film rupture time (BUT) (<10 s) and positive for rose bengal staining. Severe dry eye presents with corneal epithelium keratosis and dry and wrinkly conjunctiva.
4. Corneal vascularization: In the eyes suffered from mild and moderate alkaline burns, new vessels grow into part or all the cornea; in eyes with severe alkaline burns, except for corneal vascularization, the cornea is covered by hyperplastic fibrous tunica vasculosa tissues. There are two theories about the mechanism of neovascularization: (1) Leukocyte mediated theory: It is believed that leukocyte infiltration occurs before neovascularization. (2) Neovascular growth factor theory: Neovascular growth factor is a general term for growth factors that stimulate neovascularization, including fibroblast growth factor (FGF), platelet-derived growth factor (PDGF), transforming growth factor (TGF), etc. The mechanism of corneal neovascularization is still under intensive study.
5. Symblepharon: In cases with mild burns, only partial symblepharon occurs, while in cases with serious burns, partial or complete adhesion of the upper and lower eyelids and the eyeball may appear, leading to the complete fixation of the eyeball and symblepharon or even palpebral fissure closure.
6. Other intraocular complications: Secondary glaucoma is a serious complication. As the destruction of the angle tissue may be characterized by increased intraocular pressure, and most doctors only pay attention to abnormal external changes and ignore the intraocular pressure measurement, the patient may lose the chance of regaining vision due to optic atrophy even the time for anterior segment reconstruction arrives. Complicated cataract is also a complication, which can be treated with extracapsular cataract extraction (ECCE) and implantation of an intraocular lens during penetrating keratoplasty (PKP) or after PKP. In view of the fact that when corneal edema and opacity and neovascularization occur, it is impossible to determine the ocular

condition of many patients before surgery, so whether to implant an IOL can only be determined during the surgery. As long as the iris is essentially healthy, intraoperative efforts for IOL implantation will be made.

21.3 Alkali Burn

Alkali burns are the most common but intractable chemical injuries. Understanding the histopathological process of alkali burns is very important for first aid and treatment after injuries.

21.3.1 Pathological Process

Alkali is fat-soluble. When it comes into contact with eye tissue, there are three aspects of damages: (1) Causing rapid coagulation of tissue protein and cell necrosis, and through the effect of dehydration, causing an imbalance of fluid inside and outside the cell and then accelerating cell necrosis. (2) Playing a role in saponification with the lipids in tissue, thereby destroying the structure of the cell membrane. Alkali saponification creates a softened or liquefied environment in tissues, resulting in continuous alkali diffusion to the surrounding and deep tissues and damaging the adjacent or intraocular tissues. (3) Causing the thrombosis of normal blood vessels in the eye and tissue ischemia, leading to the insufficient nutrition of the corneal tissue and accelerating the destruction of the tissue while hindering the repair.

21.3.2 PH Value and Damage Degree

When the pH value exceeds 11, the matrix mucopolysaccharides are rapidly destroyed and the collagen fibers swell. In NH3 burns, the pH value in the anterior aqueous humor increases within 3 to 5 min after the burn, so the anterior chamber flushing should be performed as early as possible to be more effective.

21.3.3 Clinical Stage and Grade of Alkaline Chemical Injury of Anterior Segment

At present, there is no unified staging standard at home and abroad. Some concepts in the stands are vague and inconsistent with the concepts in clinical treatment. We recommend that alkaline chemical injuries be divided into 3 stages and 4 degrees, and the injury time, injury condition, and treatment principles should be combined for unified consideration.

1. Acute stage (early stage): In terms of pathological damage, it means within 1 week after the injury. The symptoms are acute tissue necrosis and aseptic inflammatory exudation.

Clinical manifestations: The early stage of burns, also known as the acute phase, refers to the first week after burns. Due to the effect on lipids saponification, alkali destroys the cell membrane of corneal layers, the ciliary body, and the trabecular meshwork cell membrane. Limbal and conjunctival ischemia also occur. Corneal edema, foggy opacity, pale conjunctiva, or anterior chamber with a large number of fibrinoid exudation are common in injured eyes and some severe cases even get cataracts.

After severe burns, IOP will rise, possibly because of (1) the elevated prostaglandins in aqueous humor, (2) collagenous shrinkage of the cornea and surrounding sclera due to burns, and (3) obstruction or atresia of the drainage system of aqueous humor veins. Therefore, in the acute stage of burns, doctors should pay attention to the detection of IOP, and take measures to lower IOP.

2. Repair and injury coexistence stage (middle stage): From the point of view of pathological damage, this stage is a coexistence period of inflammatory cell infiltration and tissue proliferation and repair, but these processes are accompanied by further damage to visual function. It is generally considered to be between 2nd and 6th weeks after injury. The

main clinical manifestations are the replacement of epithelial cells by fibrous tissue, the growth of new blood vessels into the cornea, the formation of tunicae vasculosa, the destruction of tear film and the resulting changes, corneal autolysis and perforation due to autoimmune reactions and increased collagenase, secondary bacterial infections, and other intraocular complications.

The procedure and duration of these lesions depend on the severity of the alkaline chemical injury and whether the treatment during the acute stage is appropriate. The treatment in this period is still focused on inflammation control and appropriate surgical treatment, mainly symptomatic management and treatment of complications, such as inhibition of collagenase activity, proper glucocorticoid application, prevention of bacterial infection, prevention of excessive symblepharon and secondary glaucoma.

Clinical manifestations: This period is characterized by the coexistence of tissue necrosis and repair. 1st and 2nd degree burns: The corneal and conjunctival epithelium is repaired and new blood vessels begin to proliferate. In these cases, the stroma often remains transparent. Burns often involve the iris and cause iritis. If there is anterior chamber empyema or injury, the ciliary body is often affected. 3rd and 4th degree burns: The corneal epithelium cannot be regenerated and the corneal stroma continues to be edema and turbid. The necrotic area of the corneal endothelium is replaced by the fibrocyte membrane. As the limbal stem cells are damaged, the corneal epithelium in the severely injured area cannot be repaired, resulting in the abnormal release of collagenase and metalloproteins, leading to a corneal stromal ulcer. In addition, fibrinogen is activated, causing the dissolution of the anterior corneal lamina fibers.

3. Stable stage (late stage): From the pathological point of view, after the above-mentioned tissue destruction and tissue reactive repair, the tissue trauma and repair process have been relatively stable, but leaving sequelae of alkaline injury. This stage is about the 6th to 12th weeks after injury. The coming of the stable stage depends on the severity of the injury and whether the conditions and methods of the treatment in the early and middle stages are appropriate. Surgical treatment is the main method in this period. (Since conventional surgical procedures have not made much progress after nearly a hundred years of development, we have first performed reconstruction engineering operations including tear film, conjunctiva, cornea, and even the anterior segment of the eye.)

Clinical manifestations: Late-stage refers to burns after 3 months. 1st and 2nd degree burns: The burned area is completely healed without complications. 3rd- and 4th -degree burns: Except for persistent tear film abnormality, secondary glaucoma and cataract may occur. The main manifestations are continuous non-healing of corneal epithelium, corneal edema and turbidity, continuous expansion of corneal ulcers, and corneal stromal neovascularization. At a later stage, eyeball adhesions, corneal neovascularization, and corneal pannus covering the entire burned area may appear. Corneal ulcers may progress and lead to autolysis and corneal perforation is not uncommon.

The selection of surgical treatment for the anterior segment after alkaline injury at the later stage is based on the severity of the lesion. The principles of surgical selection are the same, except that the contents and complexity of the operation are different. Therefore, it is extremely important to grade the severity of lesions at this stage, as it is the basis for selecting surgical methods.

Reference conditions for “mild”: (1) Conjunctival limbus fibrosis $\leq 1/2$ circumference. (2) Corneal neovascularization membranous tissue (or pseudopterygium tissue) invading cornea $\leq 1/2$ corneal tissue. The thickness of the transparent area of the cornea is basically normal under a slit lamp and the iris and pupil are faintly visible. (3) Schirmer test is in the normal range;

BUT is normal or abnormal; the rose bengal staining is partially positive or negative. (4) No intraocular complications.

Reference conditions for "moderate": (1) the range of conjunctival fibrosis >1/2 circumference or accompanied by a small-scale symblepharon. (2) The cornea is completely vascularized or covered by thinner vascular membranous tissue, but the outline of the corneal limbus can be seen, and the thickness of the cornea cannot be completely determined under a slit lamp. Ultrasonic Pachymeter measures cornea and the thickness is not uniform. Some parts are thinner than the normal thickness while some parts or all the cornea are thicker than the normal corneal thickness. (3) Schirmer test is roughly in the normal range; BUT is abnormal; the rose bengal staining is positive. (4) Concurrent cataracts or secondary glaucoma may occur and there are possibilities that no intraocular complications appear.

Reference conditions for "severe": (1) all conjunctiva fibrosis, with obvious symblepharon or upper and lower eyelid closure. (2) The cornea is completely covered by vascular membranous tissues, and the corneal limbal contour cannot be identified, or the corneal perforation has formed adhesive scars or/and accompanies corneal fistula, so the slit lamp cannot examine the intraocular tissues. The corneal thickness can be measured by ultrasonography. (3) Schirmer tear secretion test <5 mm; BUT is abnormal or no tear film appears. The rose bengal staining is strongly positive. (4) Concurrent cataracts and secondary glaucoma are possible intraocular complications. Some patients have inaccurate light perception, abnormal light projection, abnormal VEP and ERG during visual function measurement.

21.4 Acid Burn

Since acid is widely used in daily life, acid burns are not uncommon.

Pathological process: As the acid is water-soluble, it is not easy to penetrate the lipid-rich corneal epithelium, and its damage is generally limited to the exposed corneal epithelial tissues. Strong acid with very low pH often destroys the corneal epithelium and penetrates into the corneal stroma, leading to irreversible degeneration of the corneal stroma and protein precipitation and then the formation of a barrier, which can prevent acid from penetrating deeper into the eye. Due to tissue coagulation, tissue edema and decomposition are relatively light, so the boundaries of the injured tissues are obvious. Acid burns are milder than alkaline burns and cause fewer complications.

Clinical manifestations: Acid burns and alkali burns have the same clinical stages and degrees, but the severity and complications of acid burns are less than those of alkali burns. Sulfuric acid burns are the most common. In addition to the acid damage to the eyes, sulfuric acid produces a lot of heat when it meets with water, so sulfuric acid burns are also accompanied by thermal burns. Therefore, sulfuric acid burns are very serious.

Hydrochloric acid is a weak acid with a commonly used concentration of 32% ~ 38%. It has poor penetrating power and often leaves a yellow turbid area on the burned cornea.

Hypochlorous acid is often used in cleaning solutions. Long-term exposure to the acid may cause chronic blepharitis. Hydrofluoric acid is the only acid that can cause severe burns. Although it is a weak acid, it has a strong penetrating ability and can dissolve cell membranes and then enter the tissues of the eye. Therefore, understanding the characteristics of various acids is conducive to clinical symptomatic treatment.

21.5 Treatment of Alkali Burns

21.5.1 Drug Therapy

21.5.1.1 Emergency Treatment

External eye flushing: Emergency management after chemical burns should be down within 3 min after the injury. The eye should be rinsed as soon as possible, which is effective for diluting the harmful chemicals in the eye. When flushing, if possible, surface anesthesia can be used for the conjunctival sac and then the eyelids are opened

to take foreign bodies out. Experimental studies have found that if the ocular surface is rinsed briefly after alkali burns, the pH value can only decline for a period of time and then rise again. Therefore, to be effective, eye flushing should last for more than 30 min, until the pH is neutralized. Eyelid retractor or eyelid opener is conducive to flushing. Flushing solution with 0.01 ~ 0.05%EDTA can be used for calcium hydrochloride (carbon) burns.

Anterior chamber puncture: Alkali penetrates into the anterior chamber within 3 min, but the condition will be stable within 1 ~ 3 h. Therefore, the anterior chamber puncture performed one day after the burn has no clinical significance.

Bulbar conjunctival incision: In the early stage, incision of the bulbar conjunctiva radially before flushing can dilute the alkaline liquid penetrating into the conjunctiva.

Application of heparin: Chemical burns cause conjunctival vascular embolism and atresia. Heparin has a certain curative effect on dissolving limbal thrombus and restoring blood circulation. The author often instills a solution of 1000–2000 units/ml frequently until the limbus blood vessels dilate and even bloody tears come out.

21.5.1.2 Application of Soft Contact Lens

Soft contact lens can promote the repair of exposed ulcer surface and epithelium, but it reduces the oxygen supply to the cornea and increases the chance of infection. Now, it is seldom used.

21.5.1.3 Glucocorticoid

Glucocorticoid has obvious dual characters, that is, glucocorticoid has a good effect on anti-inflammatory, inhibiting capillary proliferation and reducing the hard ring of tissues, but it can also stimulate the activity of collagen and increase the dissolution of corneal tissues. Improper use of glucocorticoid can easily lead to corneal perforation. Therefore, physicians must understand the pathological process of chemical burns, and choose the appropriate time of medication to give full play to the advantages of glucocorticoids and reduce the occurrence of complications. The author's medication principle is: if there is no obvious corneal ulcer within 1 week after the burn, glucocorticoid should be applied locally and systemically, and the dosage should be reduced or stopped depending on the situation after 1 week, and then non-steroidal drugs should be used instead.

21.5.1.4 Collagen Preparations

0.2% EDTA is a commonly used collagen preparation at present, which may prevent the dissolution of the corneal lamina.

21.5.1.5 Infection Prevention

Local and systemic combined antibiotic application can prevent infection.

21.5.2 Surgical Treatment

Amniotic membrane transplantation is currently the most effective method for treating chemical burns. Early amniotic membrane transplantation is effective for all degrees of burns.

Surgical treatments of late complications include limbal stem cell transplantation, eyeball adhesion separation and formation, lamellar keratoplasty, penetrating keratoplasty, penetrating keratoplasty combined with stem cell transplantation and whole cornea transplantation combined with limbal transplantation.

21.6 Case 1

A 46-year-old man presented to our hospital one hour after the low-concentration alkaline solution entered the right eye. He has rinsed the eye with plenty of water after being injured. Examination results: FC/10 cm visual acuity, corneal edema and turbidity, epithelial defect (Fig. 21.3). After flushing again, amniotic membrane transplantation was carried out immediately (Fig. 21.4). Two weeks later, the stitches and the amniotic membrane were removed. The turbidity disappeared and corneal transparency was restored, but nebula can be seen (Fig. 21.5). Visual acuity was 0.8. The burn was mild and timely measures have been taken, so the burn only affected the corneal epithelium.

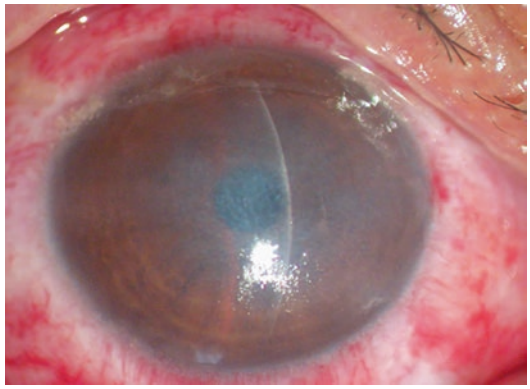

Fig. 21.3 The corneal edema is cloudy and the epithelium is defective

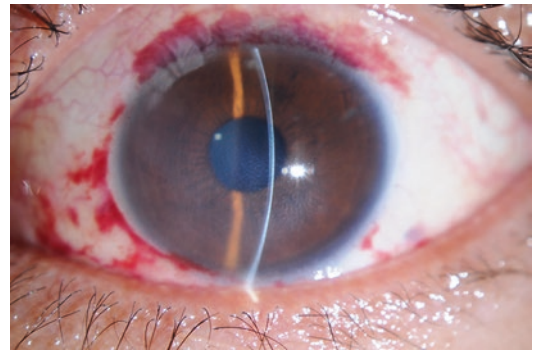

Fig. 21.5 After removing the amniotic membrane, the nebula can be seen

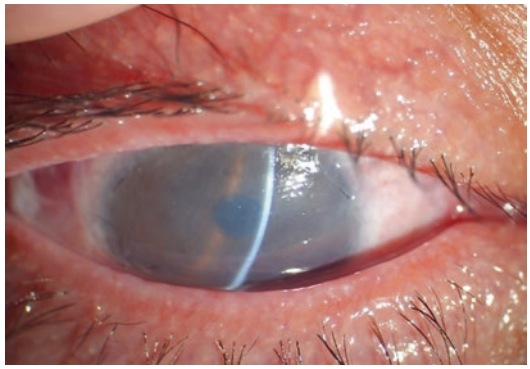

Fig. 21.4 The first day after amniotic membrane transplantation

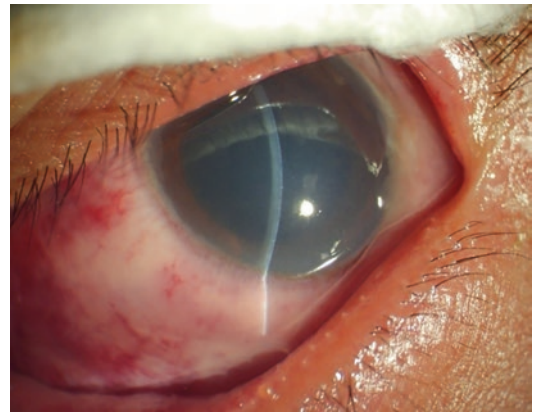

Fig. 21.6 Acute alkali burn of the left eye

21.7 Case 2

A 43-year-old man presented to our hospital one hour after the low-concentration alkaline solution entered the left eye. He has rinsed the eye with plenty of water after being injured. Examination results: FC/10 cm visual acuity, corneal edema, epithelial defect (Fig. 21.6). After flushing again, amniotic membrane transplantation was carried out immediately. One week after the operation, the amniotic membrane was still attached, but the corneal epithelium was not healed (Fig. 21.7). Amniotic membrane transplantation was performed again (Fig. 21.8). One week later, the amniotic membrane around the suture began to dissolve. Fluorescein staining was performed, but no corneal staining area was seen, indicating

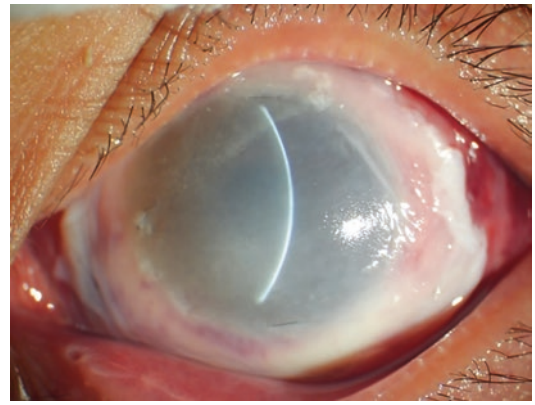

Fig. 21.7 One week after amniotic membrane transplantation

that the corneal epithelium has healed (Fig. 21.9). After 2 weeks, the sutures and the amniotic membrane were removed. The cornea was almost

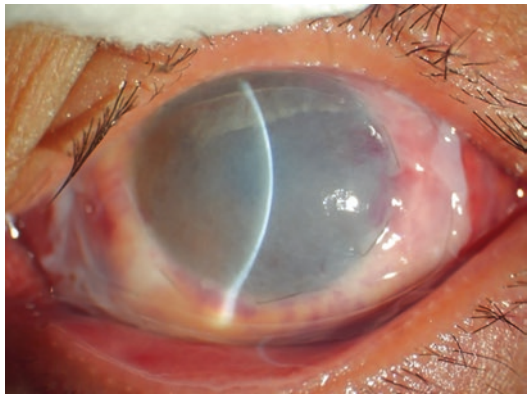

Fig. 21.8 Amniotic membrane transplantation was performed again as the corneal epithelium was not healed

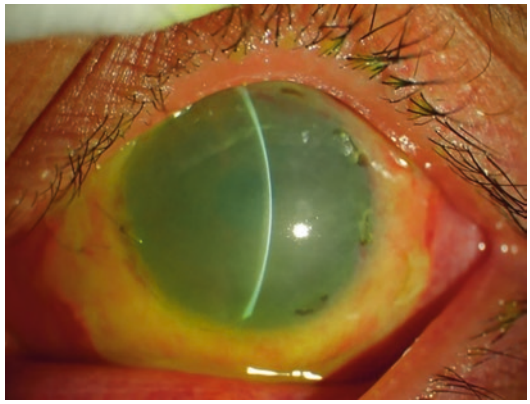

Fig. 21.9 Seven days after the second transplantation, the amniotic membrane around the suture began to dissolve. Fluorescein staining was performed, but no corneal staining area was seen, indicating that the corneal epithelium has healed

transparent and the opacity disappeared one month after the operation (Fig. 21.10).

21.8 Case 3

A 56-year-old man came to our hospital one day after a strong alkaline solution burned his eyes. Examination results: intraocular pressure 35 mmHg, all conjunctival ischemia, necrosis, total corneal opacity and edema, porcelain white, and intraocular loss of vision (Fig. 21.11). After admission, three times of amniotic membrane transplantations were performed to save the eyeball. Because the conjunctiva was almost necrotic and the stem cells were severely damaged, the healing is not satisfying. Five months later, eyeball adhesions appeared (Fig. 21.12).

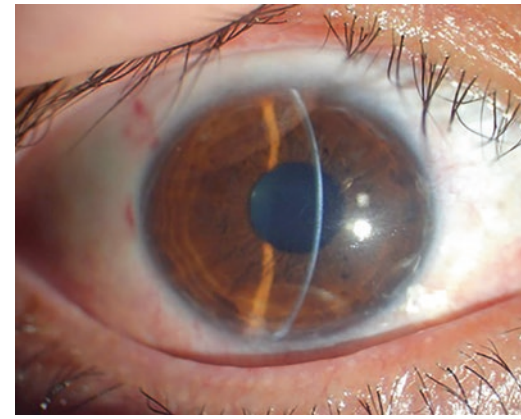

Fig. 21.10 The cornea was almost transparent one month after the operation

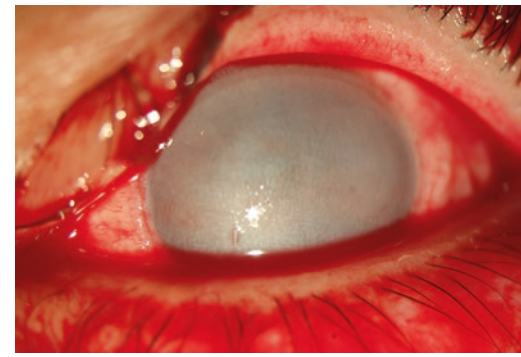

Fig. 21.11 All conjunctival ischemia, necrosis, total corneal opacity and edema, porcelain white, and intraocular loss of vision

21.9 Case 4

A 53-year-old male patient presented half a year after acid burns. He has lost sight. Examination results: visual acuity HM/BE, a large number of pannus in the whole cornea, the anterior chamber of the eye cannot be clearly seen (Fig. 21.13). After the first transplantation of amniotic membrane cultured stem cells, the eyeball adhesion was cured (Fig. 21.14). After full lamellar keratoplasty (with corneoscleral limbus) combined

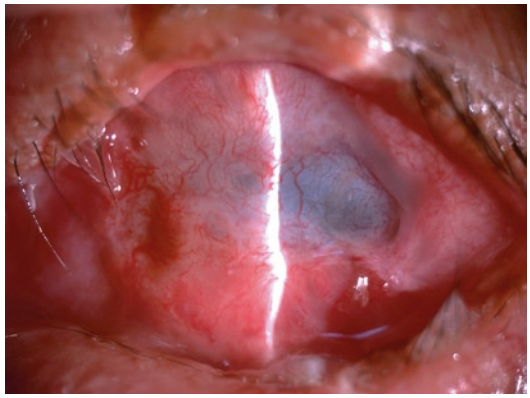

Fig. 21.12 Symblepharon and a large number of pseudopigium on the cornea

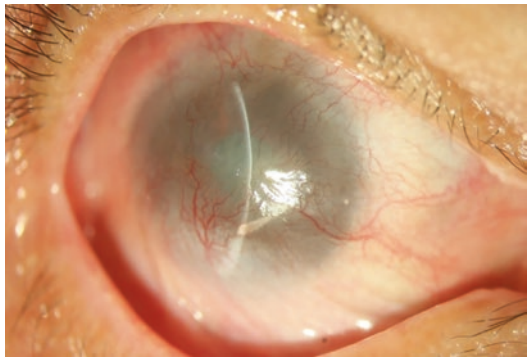

Fig. 21.13 A large number of pannus in the whole cornea

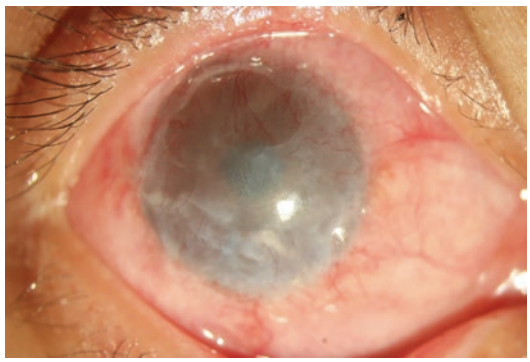

Fig. 21.14 After the first transplantation of amniotic membrane cultured stem cells, the eyeball adhesion was cured

with partial tarsorrhaphy, the corneal graft and bed were transparent and the pupil was visible (Fig. 21.15). The vision returned to 0.5.

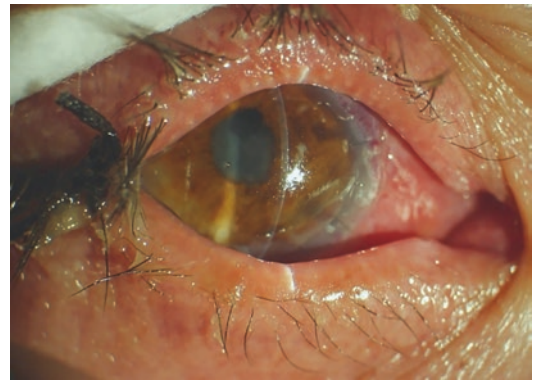

Fig. 21.15 After full lamellar keratoplasty combined with partial tarsorrhaphy, the corneal graft and bed were transparent

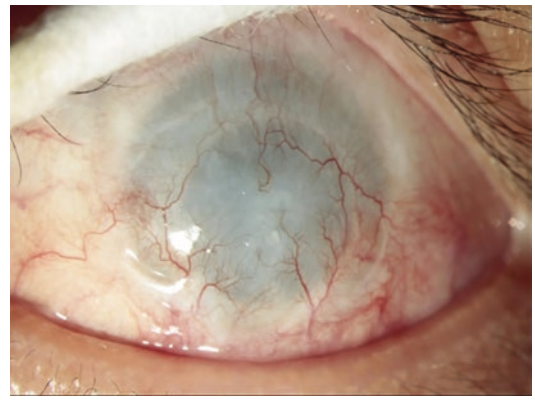

Fig. 21.16 Corneal neovascularization, corneal white opacity, pupil about 7 mm, lens opacity

21.10 Case 5

A 28-year-old man presented half a year after acid burns. He has lost sight. Examination results: visual acuity HM/BE, intraocular pressure 18 mmHg, corneal neovascularization, white cornea opacity, pupil about 7 mm in the eye can be seen, and lens opacity (Fig. 21.16). One month after surgical removal of the cataract and transplantation with keratoprosthesis (Guangdong Brilliant Vision Biotechnology Co., Ltd.), the patient's vision recovered to 0.5 (Fig. 21.17). Three months after surgery, the patient's vision recovered to 0.8 (Fig. 21.18). The ocular surface fluorescein sodium staining is shown in Fig. 21.19.

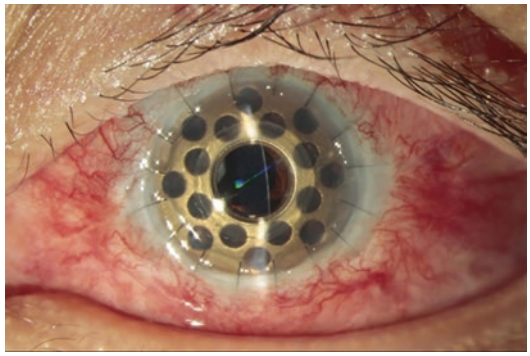

Fig. 21.17 One month after surgical removal of cataract and artificial corneal transplantation, the patient's vision recovered to 0.5

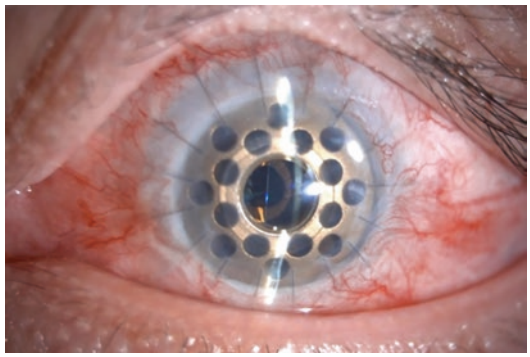

Fig. 21.18 Three months after surgery, the patient's vision recovered to 0.8

21.11 Personal Experiences or Matters Need Attention

A double continuous suture is recommended for amniotic membrane transplantation, which has the advantage of less ocular surface line junction and alleviation of the patient's irritation symptoms after operation. The amniotic membrane tissue closely adheres to the ocular surface, conducive to epithelial growth after surgery. Regarding the question of whether to open the bulbar conjunctiva when the amniotic membrane is covered, the authors think that it depends on the condition of the patient's eye injury during the operation. If extensive ischemic necrosis of the anterior segment of the eye is detected, the bulbar conjunctiva can be opened, after which, the surgeon can massage the blood vessels of the

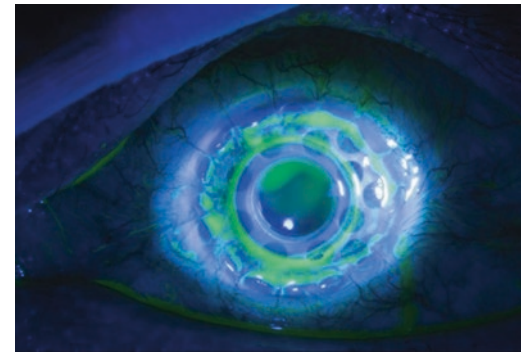

Fig. 21.19 The ocular surface fluorescein sodium staining results 3 months after the operation

conjunctiva and sclera that are blocked by microthrombi to restore the blood supply to the front of the eye, and then use conjunctival tissue to cover the surface of the cornea and sclera and cover the conjunctiva on the surface of the amniotic membrane. If the anterior segment of the eye is not severely ischemia, it is not necessary to open the bulbar conjunctiva [8].

If the chemical injury is about to cause corneal perforation, corneal transplantation is imperative. After the operation, attention should be paid to the abnormality of tears. Once the epithelial healing is delayed, measures should be taken as soon as possible. In the stable chemical scarring stage, it is recommended to detect tears. The tears guarantee the healing of epithelium after transplantation; otherwise, the corneal graft will easily melt.

References

1. McGhee CNJ, Crawford AZ, Meyer JJ, Patel DV. Chemical and thermal injuries of the eye. In: Mannis MJ, Holland EJ, editors. Cornea. St. Louis: Mosby; 2017. p. 1106–19.
2. Wagoner MD. Chemical injuries of the eye: current concepts in pathophysiology and therapy. Surv Ophthalmol. 1997;41:275–313.
3. Merle H, Gerard M, Schrage N. Ocular burns. J Fr Ophtalmol. 2008;31:723–34.
4. Kuckelkorn R, Makropoulos W, Kottek A, Reim M. Retrospective study of severe alkali burns of the eyes. Klin Monatsbl Augenheilkd. 1993;203:397–402.
5. Kuckelkorn R, Schrage N, Keller G, Redbrake C. Emergency treatment of chemical and thermal eye burns. Acta Ophthalmol Scand. 2002;80:4–10.

6. Paterson CA, Pfister RR, Levinson RA. Aqueous humor pH changes after experimental alkali burns. Am J Ophthalmol. 1975;79:414–9.
7. Kuckelkorn R, Kottek A, Reim M. Intraocular complications after severe chemical burns--incidence and surgical treatment. Klin Monatsbl Augenheilkd. 1994;205:86–92.
8. Weiyun S. Surgery of the cornea. Beijing: People's Medical Publishing House Co; 2012. p. 112–29.

Ocular Laser Burns

22

Ugur Acar and Gungor Sobaci

Abstract

The frequency of laser-induced eye injuries has increased in recent years with the increase in laser product sales and laser cosmetic applications. Unfortunately, laser devices have become more powerful and cheaper, incorrectly labeled, and can be easily purchased on the Internet or in the marketplace.

In this chapter, we focused on prevention and management of the laser-related injuries from laser sources used in daily life. Also, pathophysiology, clinical manifestations, differential diagnosis, and treatment of laser-induced eye injuries, especially on the posterior segment, were included, and sample cases were given.

Keywords

Laser · Laser product · Complicated · Injury
Retina · Eye · Prevention · Treatment

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22.1 Introduction

Advances in laser technology and decreased production costs have caused an increase in laser devices to enter daily life such as cosmetics, medicine, safety, communications, industry, and entertainment [1]. Many low-cost laser products, especially laser pointers, are available in the consumer market and even on the internet [1, 2].

Ophthalmology has been on the frontline in the use of laser for therapeutic purposes. Ophthalmologists have been familiar with the laser-related ocular injuries since the use of Ruby laser in the late sixties and argon sources in the seventies [3]. Several ocular complications and/or inadvertent damage to the eye, mild to severe, leading to an asymptomatic burn to blindness, have been documented elsewhere [3]. This chapter excludes laser-related injuries from ophthalmic use of medical-grade laser(s) by the laser surgeon. In this study, we focused on prevention and management of laser-related injuries from laser sources used in daily life. In this context, ocular injuries from non-ophthalmic use rather than ophthalmic use were included; periocular use of laser, however, it was mentioned due mostly to social-media-driven misuse of laser for cosmesis.

Non-ophthalmic use of laser-related eye injury in the literature was first reported by Mensah et al. in 1998 [4]. At that time, documented laser complications were usually related to the anterior

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segment such as punctate corneal epitheliopathy and most of them did not cause permanent vision loss [4, 5]. However, recent reports of laser-induced ocular injuries have become more frequent, especially due to high-energy laser pointers and most of them have caused visual impairment with both anterior and posterior segment injuries [1, 2, 6–24]. It is well-known that the eye is one of the most vulnerable organs to laser radiation due to the eyelid skin is thin, and also iris and retinal pigment epithelium have some pigment-rich chromophores that can absorb laser energy.

22.2 Definition

The International Commission on Non-Ionizing Radiation Protection (ICNIRP), an independent organization of scientific experts, has set guidelines on laser radiation exposure limits [25]. These exposure limits provide a scientific basis for the hazard classification of laser devices by the International Electrotechnical Commission (IEC) based on accessible radiation characteristics [26]. Laser sources that are highly monochromatic with a very well-defined color have the ability to emit beams with extremely high energy with a very little deflection as a common feature [7]. The term laser is an acronym for light amplification by stimulated emission of radiation. The hazard level of laser is based on the exposure level, wavelength, emission duration, and angular subtense of the laser source. At the present time, lasers are classified into four classes according to the European standard (Table 22.1) [26]. Class 1 is safe for eye if the device is used according to manufacturer recommendations. Since class 2 cannot pass through the skin it cannot cause a damage to the eye if the person closes the eyes with the palpebral reflex. Class III can be dangerous to the eye, because the person can be directly exposed to radiation above the safety threshold level, until the eyelid is closed. And lastly, class IV exhibits risks for the eyes even if the eye is not directly exposed to the laser beam. There are several subclasses in this classification related to the operating conditions of the laser system (1 M,

Table 22.1 Laser classification according to the European standard [26]

Laser Class	Hazard or potential for injury	Typical output power (P)	Typical use
1	Safe under reasonably foreseeable conditions	P < 0.4 mW	Scanner checkouts, DVD players
1 M	Hazardous to the eye when using telescopic optical instruments (otherwise as in class 1)	P < 0.4 mW; but the beam diameter is greater than 7 mm	
2	Direct intrabeam viewing must be avoided—retinal injury is possible at intrabeam viewing times exceeding 0.25 s	P < 1 mW	Laser pointers, laser spirit levels
2 M	Hazardous to the eye when using telescopic optical instruments (otherwise as in class 2)	P < 1 mW; but the beam diameter is greater than 7 mm	
3A	Hazardous to the eye only when using telescopic optical instruments	P < 5 mW; but the beam diameter is greater than 7 mm and the power density is related to the same pupil diameter as in class 2 lasers	
3R	Hazardous to the eye	P < 5 mW	Show and projection lasers, material processing lasers
3B	Always hazardous to the eye	P < 500 mW	
4	Always hazardous to the eye and skin	P > 500 mW	

2 M, 3R, and 3B). Optical radiation power depends on two factors: Emission wavelength and pulse duration. The maximum limits of laser beam are the following: 0.4 mW for class I; 1 mW for class 2; 5 mW for class 3R; and 500 mW for class 3B. Values above 500 mW correspond to class 4. The United Kingdom uses a similar classification of laser products which consists of 8 categories (Class 1, 1C, 1 M, 2, 2 M, 3R, 3B, and 4) [27]. Food and Drug Administration (FDA) categorizes laser products into 6 (Class I, IIa, II, IIIa, IIIb, and IV) according to the power output and hazard potential (Table 22.2) [28].

It is remarkable that cosmetic laser procedures such as laser-assisted hair removal, CO_2 laser facial resurfacing, and selective photothermolysis are the second most common cause of ocular injury after laser pointers in the literature [29–42]. Lasers have been used extensively for cosmetic purposes for many years as an office procedure and after approving laser-assisted hair removal by the FDA, number of use has been increasing rapidly. However, these lasers operate at high emission levels and they are in the class 3R and 4 categories. There are only a few reports on retinal complications due to laser epilation in the literature, mostly anterior segment complications such as corneal burns, iris injuries, pupillary distortion, anterior uveitis, and cataract have been reported [43].

Table 22.2 Laser classification according to the FDA [28]

Laser Class	Laser product hazard	Product examples
I	Nonhazardous; hazard increases if viewed with optical aids, including magnifiers, binoculars, or telescopes	Laser printers, CD/DVD players
II, IIa	Hazard increases when viewed directly for long periods or with optical aids	Barcode scanners
IIa	Depending on power and beam area, can be momentarily hazardous when directly viewed or when staring directly at the beam with an unaided eye	Laser pointers
IIIb	Immediate skin hazard from direct beam and immediate eye hazard when viewed directly	Laser light show projectors, industrial/ research lasers
IV	Immediate skin hazard and eye hazard from exposure to either the direct or reflected beam; may also present a fire hazard	Laser light show projectors, industrial/ research lasers, lasers used in LASIK eye surgery

The frequency of laser-induced eye injuries has increased in recent years with increase in laser product sales and laser cosmetic applications. Unfortunately, laser devices have become more powerful and cheaper, incorrectly labelled, and can be easily purchased on the Internet or in the marketplace [8]. According to the results obtained from the data of 19,765 Canadians, Qutob et al. determined that 48.1% of them use or have been exposed to a laser product in the previous 12 months [1]. This rate is really surprisingly high. The authors also found that the majority of the reported injuries were to the eyes, and the remaining were to the skin. These injuries were caused by laser cosmetic treatments or laser pointers, and approximately 75% of injuries occurred while someone else was using the device [1]. The researcher who use, or those in manufacturing laser devices, and soldiers who use laser weapons or devices for monitoring are open to laser-induced ocular injuries.

Since most of the events would not be published in the journals, estimates of the eye injuries likely represent a small percentage of the total number of laser injuries that have occurred. The exact prevalence of laser-induced eye injuries cannot be detected due to several reasons; a probable high number of unreported injuries, presence of asymptomatic or unnoticed cases, hiding the complaints of children who have laser-related eye injury from fear.

Laser effects on tissue are classified into three matters as follows: photocoagulation, photodisruption, and photoablation. The absorption of laser light by the target tissue and denaturing the protein with the heat generated is photocoagulation (e.g., retinal photocoagulation with argon laser). In photodisruption, laser light creates a great temperature rise and causes tissue rupture or perforation by producing an acoustic shock

wave (e.g., posterior capsulotomy with Nd:YAG laser). In photoablation, laser lights break the chemical bonds that hold tissue together, and vaporize the tissue without temperature rise (e.g., flap creation in refractive surgery with excimer laser). In general, short-wavelength (visible) lasers usually produce photocoagulation, long-wavelength (infrared, near-infrared) lasers produce either photodisruption or photocoagulation, whereas ultraviolet wavelength lasers produce photoablation.

Even ophthalmic use was considered, each of the mechanisms of action applied by the laser damages the intraocular tissues with the same mechanism, if used in the unstandardized protocol. In general, factors affecting eye injuries can be divided into 3 categories; laser-related factors, ocular-related factors, and protective factors (Table 22.3) [23, 36]. Lasers with an output power over 5 mW can easily cause an irreversible retinal damage despite the blink and aversion reflexes. It is obvious that a person who has a large pupil in the dark condition and does not wear protective glasses will suffer more damage than a person who has a small pupil in bright condition and wears proper protective glasses even if they are exposed to the same laser pointer for an equal duration. The most striking example of this situation is the misuse of night vision goggles and laser devices used for military purposes in a dark environment [44].

Currently, there are numerous types of laser, including ruby laser with 694 nm wavelength, alexandrite laser with 755 nm, and 800 nm wavelengths, Nd:YAG laser with 1064 nm wavelength, diode laser with 800 nm wavelength, and intense pulse light laser between 590 and 1200 nm wavelength [34]. Laser light in the visible to the near-infrared spectrum (400 nm–1400 nm wavelength) is the hazard region for the retina, whereas in the ultraviolet (290 nm–400 nm wavelength) or far-infrared spectrum (1400 nm–10,600 nm wavelength) is known as the hazard region for the cornea and lens [36]. Robertson et al. found that green laser pointers that have 490–575 nm wavelength are more damaging to the retina than red laser pointer which has 635–750 nm wavelength [45, 46].

Table 22.3 Factors affecting eye injuries

Laser-related factors	Ocular-related factors	Protective factors
Wavelength of the radiation Energy level of the beam Exposure time Pulse duration Distance from the source	Pupil size Degree of retinal pigmentation Refractive status Lens status Eye color	Blink reflex Use of protective eyewear Aversion responses

Evaluation of microstructural changes after recreational use laser maculopathy was associated with visible dot-blot-like subretinal or sub-internal limiting membrane hemorrhage and protruding foveal edema in Muslubas et al.'s series [47]. In optical coherence tomography (OCT) evaluation, however, full-thickness macular hole without a fluid-cuff or swelling of the surrounding retina may be presenting sign after Nd:YAG laser [48–50].

22.3 Cases

Case 1: A 28-year-old soldier who is a howitzer operator applied to our clinic with complaints of low vision and floaters in his right eye [44]. In his anamnesis; he stated that he accidentally looked at the emitted beam of the howitzer 6 weeks ago. The best-corrected visual acuity (BCVA) of his right eye was 0.1 with Snellen chart. The anterior segment of the eye and vitreous was normal. There was hemorrhage and edema in the macula in his right eye (Fig. 22.1a). Hyperfloresans compatible with window defect was detected in the macula by fundus fluorescein angiography (FFA) (Fig. 22.1b). The patient was only followed up without medication. After the fourth month, the BCVA increased to 0.5 in the right eye and the hemorrhage was completely resolved, but central scotoma was detected in the computerized vision field test. The window defect was still ongoing in FFA.

Case 2: A 22-year-old soldier applied to our clinic with the complaint of decreased vision in his right eye [44]. He stated that he looked at the light emitted by the telemeter for 30 seconds

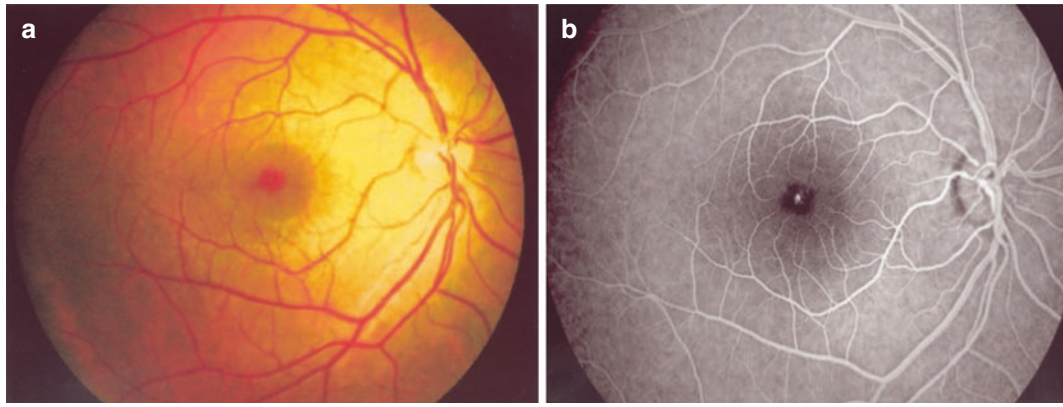

Fig. 22.1 (**a**) There was hemorrhage and edema in the macula in his right eye. (**b**) Hyperfloresans compatible with window defect was detected in the macula by FFA

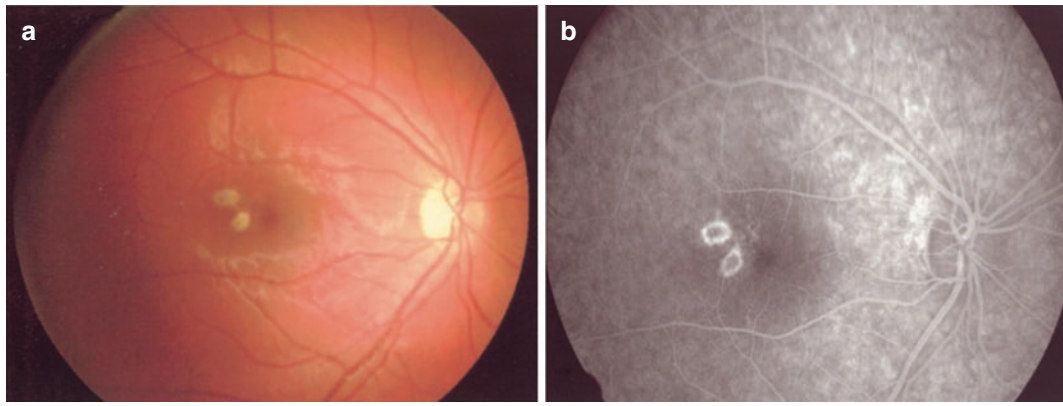

Fig. 22.2 (**a**) There were two well-circumscribed subretinal exudation yellow round lesions in the temporal of the macula. (**b**) In FFA, these lesions appeared as hypofluorescence areas surrounded by a hyperfluorescent border

3 weeks ago. The BCVA of his right eye was 0.2 with the Snellen chart. The anterior segment of the eye and vitreous was normal. There were two well-circumscribed subretinal exudation yellow round lesions in the temporal of the macula (Fig. 22.2a). In FFA, these lesions appeared as hypofluorescence areas surrounded by a hyperfluorescent border (Fig. 22.2b). The patient was given 80 mg/day prednisolone peroral for 5 days and the drug was used in a tapered way for 15 days and ceased. After the third month, the BCVA decreased to 0.1 in the right eye. There was hyperpigmentation in the form of scarring in the lesion areas of the macula and it appeared to be degenerated. A central scotoma was detected in the computerized vision field test.

22.4 Important Signs, Examinations, Diagnosis, and Management of Ocular Laser Injuries

Patients who underwent laser-related eye injuries can present to the eye clinics with different complaints according to the location and severity of injuries. Since the radiation with visible wavelength is not absorbed by the anterior segment of the eye, they do not cause the symptoms such as hyperemia, irritation, burning, stinging, or foreign body sensation; however, they may cause some symptoms related to posterior segment complications such as a sudden and sharp ocular pain, blurred vision, scotoma, or a black shadow.

When laser exposure is mentioned in the medical history, it will not be difficult to diagnose by an ophthalmologist. However, the diagnosis may be difficult or even missed, especially in children and delayed cases. It should not be forgotten that; laser exposure may result in a wide variety of clinical spectrum from asymptomatic to devastating sight-threatening complications such as corneal scar, anterior uveitis, cataract, retinal burn, macular hole, or choroidal neovascularization.

Anterior and posterior segment examination should be done in detail, and laser pointer exposure or cosmetic laser application should be questioned even if the patient does not say so. The most common location of laser-related posterior segment injuries is fovea because of the nature of ocular fixation. The fundoscopic view of laser injury varies widely from the normal or minimal pigmentary change to macular hole or atrophic changes depending on the severity of the injury and the time elapsed. Advance OCT devices are very helpful to ophthalmologists to diagnose laser damage. Classically, in OCT imaging, a well-circumscribed and localized oval hyperreflective alteration extending from the retinal pigment epithelium surface into the retina and damage in outer retinal layers under the fovea are observed [47]. However, OCT findings can vary from only a discrete break in the ellipsoid zone to greater disruption in this layer to a column of increased reflectivity within the outer nuclear layer.

In differential diagnosis of laser-related ocular injury, the history and the ocular findings are of special consideration. Especially in children, laser pointer-related macular damage can be misdiagnosed or confused with other conditions, including macular dystrophies, achromatopsia, macular telangiectasia and solar retinopathy [14, 51]. De Silva et al. recommend using the near-infrared autofluorescence (NIR-AF; with 787 nm wavelength) mode for improving the accurate diagnosis of retinal laser injuries [51]. Because the authors demonstrated that NIR-AF imaging obtains typical sharply demarcated lesions with increased signal at the center, and a reduced signal around the edge as a characteristic appearance in all patients exposed to lasers. They also showed that the other imaging methods including color fundus photography, blue light AF (B-AF with 488 nm wavelength), and OCT were less consistent or specific for laser-related retinal injuries. Conventional B-AF signal that is mainly derived from lipofuscin provides an important clue about the health of the retinal pigment epithelium, whereas the NIR-AF signal is mainly derived from melanin and melanolipofuscin in the retinal pigment epithelium and the choroid [51–53]. It is difficult to differentiate between malingering and occult maculopathy from the laser, especially in medicolegal conditions. In these circumstances, further evaluations with microperimetry and mfERG should be considered to disclose laser injury itself. All of these imaging modalities may also assist ophthalmologists in follow-up examinations and in determining prognosis.

Treatment options in laser-induced eye injuries would vary depending on the severity and the location of the developed complications. Superficial and mild lesions to the cornea can be treated with topical antibiotics and artificial tear, whereas contact lens application or patching may be added for deeper and more severe abrasions. Amniotic membrane transplantation and topical and oral ascorbic acid therapy may also be required in cases of corneal and conjunctival burns. In cases where the corneal endothelium is mildly affected, topical corticosteroid therapy is sufficient, but in cases where it is severely affected, keratoplasty may be required. In cases with iritis and anterior uveitis topical corticosteroid and cycloplegic drugs should be used, intraocular pressure must be followed closely.

Medical therapy for laser-related retinal injury is mainly limited to corticosteroid treatment. The aim is to minimize the cellular inflammatory response to injury [23]. The clinical course of laser-related retinal injury is usually characterized by a sudden visual acuity decrease because of foveal damage, followed by an increase in visual acuity thanks to spontaneous healing of damage within several weeks [6, 23]. However, no improvements may be seen in patients who have severe and extensive damage, and may result in long-term or permanent visual impairment. Although there are studies in the literature show-

ing that systemic corticosteroid therapy had beneficial effects such as enhancement in photoreceptor survival, rapid reestablishment of retinal layers, and decrease in vitreal accumulation of protein and prostaglandin E_2 in experimental animal models with laser-induced retinal injury [54–56], there are no randomized controlled clinical studies in this regard. And, there is no consensus on the dose and duration of treatment, either. Since, the commonly favorable and spontaneous healing course of laser-related retinal injuries, it is difficult to judge whether corticosteroid or other treatment is effective, or not [6]. The close observation of the patient with laser-related retinal injuries seems to be the most common treatment option.

Vitreoretinal surgery can be performed in patients with nonhealing vitreous hemorrhage, subretinal or subhyaloid hemorrhage, macular hole, and epiretinal membrane as a surgical option. Indeed, most of the vitreoretinal hemorrhages are expected to resolve spontaneously within a relatively short time, from 2 weeks to a few months [23]. However, successful anatomical and functional results can be obtained when macular surgery with internal limiting membrane peeling was performed in the early period [47]. Intravitreal anti-vascular endothelial growth factor administration is recommended not only for the developed choroidal neovascularization secondary to Bruch's membrane damage [24] but also acute macular edema [47].

It is obvious that the final visual acuity is dependent on the retinal lesion location and size.

22.5 Recommendations

Laser devices should have a warning sign on them.

Additional warning labels or information have to be included when the laser class is 2 and above.

It should not be left somewhere in the reach of children. Its batteries should be removed when not in use. Parents should be informed enough about this issue.

Uncontrolled sales via the Internet and consumer market should absolutely be prohibited.

No laser pointers of any class are made available to children. Even low-energy laser pointers should not be sold to children as a toy.

Serious sanctions should be imposed for those who do not follow the rules.

Both the practitioner and the patient should wear protective glasses that have specific protective features to the laser wavelength used during all cosmetic laser treatments.

Nobody other than the patient and the ophthalmologist should be present in the operating room during ophthalmic laser procedures such as Nd-YAG laser capsulotomy, argon laser photocoagulation. In case of necessity, the accompanying person must wear protective glasses and her/his eyes must be closed.

The patient should be referred to an ophthalmologist immediately without hesitation, especially unconsciously or drunk exposed to the recreational laser, or child abuse with a laser pointer.

22.6 In Brief

In today's world, many low-cost laser products, such as laser pointers, laser lights, and laser cosmetic devices are used more than ever. History of laser injury to the macula is of special concern due only in differential diagnosis, but also consequences of the macular injury Observation is sufficient in most cases. Currently, symptomatic treatment is preferred and applied on the patient basis; if surgery is needed, the natural course of the disease should be considered.

References

1. Qutob SS, O'Brien M, Feder K, McNamee J, Guay M, Than J. Prevalence of laser beam exposure and associated injuries. Health Rep. 2019;30(1):3–9.
2. Torp-Pedersen T, Welinder L, Justesen B, Christensen UC, Solborg Bjerrum S, La Cour M, Saunte JP. Laser pointer maculopathy – on the rise? Acta Ophthalmol. 2018;96(7):749–54.
3. Blumenkranz MS. The evolution of laser therapy in ophthalmology: a perspective on the interactions between photons, patients, physicians, and physicists:

the LXX Edward Jackson Memorial Lecture. Am J Ophthalmol. 2014;158(1):12–25.e1.
4. Mensah E, Vafidis G, Marshall J. Laser pointers: the facts, media hype, and hysteria. Lancet. 1998;351(9111):1291.
5. Sethi CS, Grey RH, Hart CD. Laser pointers revisited: a survey of 14 patients attending casualty at the Bristol Eye Hospital. Br J Ophthalmol. 1999;83(10):1164–7.
6. Birtel J, Harmening WM, Krohne TU, Holz FG, Charbel Issa P, Herrmann P. Retinal injury following laser pointer exposure. Dtsch Arztebl Int. 2017;114(49):831–7.
7. Alda J, Gómez Sanz F, González M-MJ. Laser pointer maculopathy. A new public health problem? Arch Soc Esp Oftalmol. 2017;92(1):1–3.
8. Chen X, Dajani OAW, Alibhai AY, Duker JS, Baumal CR. Long-term visual recovery in bilateral handheld laser pointer-induced maculopathy. Retin Cases Brief Rep. 2021;15(5):536–9. Online ahead of print
9. Linton E, Walkden A, Steeples LR, Bhargava A, Williams C, Bailey C, Quhill FM, Kelly SP. Retinal burns from laser pointers: a risk in children with behavioural problems. Eye (Lond). 2019;33(3):492–504.
10. Mtanes K, Mimouni M, Zayit-Soudry S. Laser pointer-induced maculopathy: more Than meets the eye. J Pediatr Ophthalmol Strabismus. 2018;55(5):312–8.
11. Rabiolo A, Sacconi R, Giuffrè C, Corbelli E, Carnevali A, Querques L, Sarraf D, Freund KB, Sadda S, Bandello F, Querques G. Self-inflicted laser handheld laser-induced maculopathy: a novel ocular manifestation of factitious disorder. Retin Cases Brief Rep. 2018;12(Suppl 1):S46–50.
12. Al-Amry MA, Alsulaiman SM, Ghazi NG. Anterior segment injury by a high-power handheld blue laser device. Ocul Immunol Inflamm. 2018;26(8):1174–6.
13. Mehta N, Tsui E, Ranka M, Dedania V, Lee GD, Modi Y. Inadvertent self-induced macular laser injury in an 8-year-old girl. J AAPOS. 2018;22(5):397–8.
14. Zhang L, Zheng A, Nie H, Bhavsar KV, Xu Y, Sliney DH, Trokel SL, Tsang SH. Laser-induced photic injury phenocopies macular dystrophy. Ophthalmic Genet. 2016;37(1):59–67.
15. Palakkamanil MM, Fielden MP. Effects of malicious ocular laser exposure in commercial airline pilots. Can J Ophthalmol. 2015;50(6):429–32.
16. Thanos S, Böhm MR, Meyer Zu Hörste M, Schmidt PF. Retinal damage induced by mirror-reflected light from a laser pointer. BMJ Case Rep. 2015;2015:bcr2015210311.
17. Scollo P, Herath G, Lobo A. Retinal injury by industrial laser burn. Occup Med (Lond). 2014;64(3):220–2.
18. Lim ME, Suelzer J, Moorthy RS, Vemuri G. Thermal macular injury from a 154 mW green laser pointer. J AAPOS. 2014;18(6):612–4.
19. Lally DR, Duker JS. Foveal injury from a red laser pointer. JAMA Ophthalmol. 2014;132(3):297.
20. Dirani A, Chelala E, Fadlallah A, Antonios R, Cherfan G. Bilateral macular injury from a green laser pointer. Clin Ophthalmol. 2013;7:2127–30.
21. Boosten K, Van Ginderdeuren R, Spileers W, Stalmans I, Wirix M, Van Calster J, Stalmans P. Laser-induced retinal injury following a recreational laser show: two case reports and a clinicopathological study. Bull Soc Belge Ophtalmol. 2011;317:11–6.
22. Tsuzuki A, Tsuzuki S, Fujii C, Kubo E, Akagi Y. A case of ocular injury from industrial laser burns. Jpn J Ophthalmol. 2004;48(2):173–5.
23. Barkana Y, Belkin M. Laser eye injuries. Surv Ophthalmol. 2000;44(6):459–78.
24. Xu K, Chin EK, Quiram PA, Davies JB, Parke DW 3rd, Almeida DR. Retinal injury secondary to laser pointers in pediatric patients. Pediatrics. 2016;138(4):e20161188.
25. International Commission on Non-Ionizing Radiation Protection. ICNIRP guidelines on limits of exposure to laser radiation of wavelengths between 180 nm and 1,000 μm. Health Phys. 2013;105(3):271–95.
26. International Electrotechnical Commission. IEC 60825–1, Safety of laser products – Part 1: Equipment classification and requirements, Ed 3.0, 2014–05.
27. British standard BS EN 60825–1:2014. Safety of laser products. Equipment classification and requirements. British Standards Institution. 2014. https://www.thenbs.com/PublicationIndex/documents/details?Pub=BSI&DocID=320928.
28. Performance standards for light-emitting products. U.S. Food and Drug Administration Code of Federal Regulations, Title 21, Chapter I, Subchapter J, Part 1040. Washington, DC: U.S. Government Publishing Office Bookstore. 2016. http://www.ecfr.gov/cgi-bin/text-idx?SID=c656f500beebfa4cf6a442a1f8f13677&mc=true&node=pt21.8.1040&rgn=div5.
29. Balyen L. Inadvertent macular burns and consecutive psychological depression secondary to Alexandrite laser epilation: a case report. Saudi J Ophthalmol. 2019;33(1):105–8.
30. Al Taleb RM, Alsharif HM, Younis AS, Alsulaiman SM, Abouammoh MA. Adherence to optical safety guidelines for laser-assisted hair removal. Photodermatol Photoimmunol Photomed. 2019;35(5):313–7.
31. Huang A, Phillips A, Adar T, Hui A. Ocular injury in cosmetic laser treatments of the face. J Clin Aesthet Dermatol. 2018;11(2):15–8.
32. Van Gemert MJC, Bloemen PR, Wang WY, van der Geld CWM, Nuijts RMMA, Hortoglu H, Wolkerstorfer A, de Bruin DM, van Leeuwen TG, Neumann HAM, Jager MJ. Periocular CO_2 laser resurfacing: severe ocular complications from multiple unintentional laser impacts on the protective metal eye shields. Lasers Surg Med. 2018;50(10):980–6.
33. Gulmez Sevim D, Oner AO, Unlu M, Mirza GE. Ocular complications after cosmetic periocular diode laser application to the eyelids. J Cosmet Laser Ther. 2018;20(7–8):447–8.
34. Asiri MS, Alharbi M, Alkadi T, Abouammoh M, Al-Amry M, AL Zahrani Y, Alsulaiman SM. Ocular injuries secondary to alexandrite laser-assisted hair removal. Can J Ophthalmol. 2017;52(2):e71–5.

35. Wang R, Wykoff CC, Christie L, Croft DE, Major JC Jr, Fish RH, Brown DM. Choroidal neovascularization secondary to alexandrite laser exposure. Retin Cases Brief Rep. 2016;10(3):244–8.
36. Shum JW, Iu LP, Cheung DN, Wong IY. A case of accidental ocular injury from cosmetic laser burn. Retin Cases Brief Rep. 2016;10(2):115–20.
37. Gunes A, Yasar C, Tok L, Tok O. Two cases of anterior uveitis after laser eyebrow epilation. Cornea. 2015;34:101–2.
38. Anaya-Alaminos R, Muñoz-Ávila JI, González-Gallardo MC, Mora-Horna ER, García-Serrano JL, Ramírez-Garrido MV. Accidental foveal photocoagulation secondary to alexandrite laser. Eur J Ophthalmol. 2014;24(5):808–10.
39. Lin CC, Tseng PC, Chen CC, Woung LC, Liou SW. Iritis and pupillary distortion after periorbital cosmetic alexandrite laser. Graefes Arch Clin Exp Ophthalmol. 2011;249(5):783–5.
40. Hammes S, Augustin A, Raulin C, Ockenfels HM, Fischer E. Pupil damage after periorbital laser treatment of a port-wine stain. Arch Dermatol. 2007;143(3):392–4.
41. Lin LT, Liang CM, Chiang SY, Yang HM, Chang CJ. Traumatic macular hole secondary to a Q-switch Alexandrite laser. Retina. 2005;25(5):662–5.
42. Blanco G, Soparkar CN, Jordan DR, Patrinely JR. The ocular complications of periocular laser surgery. Curr Opin Ophthalmol. 1999;10(4):264–9.
43. United States Food and Drug Administration. Radiation-emitting products. Washington, DC: FDA; 2018.
44. Durukan AH, Bayer A, Sobaci G, Bayraktar MZ, Akar Y. Macular Injury Due To Military Devices Which Use Laser. Ret-Vit. 2003;11(3):284–9.
45. Robertson DM, McLaren JW, Salomao DR, Link TP. Retinopathy from a green laser pointer: a clinicopathologic study. Arch Ophthalmol. 2005;123(5):629–33.
46. Robertson DM, Lim TH, Salomao DR, Link TP, Rowe RL, McLaren JW. Laser pointers and the human eye: a clinicopathologic study. Arch Ophthalmol. 2000;118(12):1686–91.
47. Muslubas IS, Hocaoglu M, Arf S, Ozdemir H, Karacorlu M. Macular burns from nonmedical lasers. Turk J Ophthalmol. 2016;46:138–43.
48. Chuang LH, Lai CC, Yang KJ, Chen TL, Ku WC. A traumatic macular hole secondary to a high-energy Nd:YAG laser. Ophthalmic Surg Lasers. 2001;32(1):73–6.
49. Menchini U, Virgili G, Giacomelli G, Cappelli S, Giansanti F. Mechanism of spontaneous closure of traumatic macular hole: OCT study of one case. Retina. 2003;1:104–6.
50. Sou R, Kusaka S, Ohji M, Gomi F, Ikuno Y, Tano Y. Optical coherence tomographic evaluation of a surgically treated traumatic macular hole secondary to Nd:YAG laser injury. Am J Ophthalmol. 2003;135(4):537–9.
51. De Silva SR, Neffendorf JE, Birtel J, Herrmann P, Downes SM, Patel CK, Hildebrand GD, Gliem M, Charbel IP. Improved diagnosis of retinal laser injuries using near-infrared autofluorescence. Am J Ophthalmol. 2019;208:87–93.
52. Delori FC, Dorey CK, Staurenghi G, Arend O, Goger DG, Weiter JJ. In vivo fluorescence of the ocular fundus exhibits retinal pigment epithelium lipofuscin characteristics. Invest Ophthalmol Vis Sci. 1995;36(3):718–29.
53. Keilhauer CN, Delori FC. Near-infrared autofluorescence imaging of the fundus: visualization of ocular melanin. Invest Ophthalmol Vis Sci. 2006;47(8):3556–64.
54. Brown J, Hacker H, Schuschereba ST, Zwick H, Lund DJ, Stuck BE. Steroidal and nonsteroidal anti-inflammatory medications can improve photoreceptor survival after laser retinal photocoagulation. Ophthalmology. 2007;114:1876–83.
55. Lam TT, Takahashi K, Fu J, Tso MO. Methylprednisolone therapy in laser injury of the retina. Graefes Arch Clin Exp Ophthalmol. 1993;231:729–36.
56. Naveh N, Weissman C. Corticosteroid treatment of laser retinal damage affects prostaglandin E_2 response. Invest Ophthalmol Vis Sci. 1990;31:9–13.

Pediatric Ocular Trauma

23

Hai Lu

Abstract

Children are vulnerable to trauma, due to their immature physical and psychological status. Ocular trauma is one of the most important causes of unilateral blindness in children. The clinical features of pediatric ocular trauma differ in many aspects from adult trauma. In this chapter, we focus on the clinical features of pediatric ocular trauma, the management, and the differences between pediatric and adult ocular trauma.

Keywords

Ocular trauma · Vitreous surgery · Pediatric diseases · Eye · Prevention · Treatment

23.1 Introduction

Children are vulnerable to trauma, due to their immature physical and psychological status. Ocular trauma is one of the most important causes of unilateral blindness in children. The clinical features of pediatric ocular trauma differ in many aspects from adult trauma.

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1. Eyeballs of children develop with age since birth. The ophthalmic structure and biological function may be profoundly influenced by trauma. Therefore, the process of visual development may be altered permanently.
2. Difficulties of office examination of children following ocular trauma: The medical history taking either from the parents or from children themselves can be hard and inaccurate. Besides, most children especially younger kids will not cooperate with physical examinations and other special equipment examinations due to pain and fear.
3. Ocular trauma to the eyeball may cause negative influences on ocular development. The changes of axial length caused by either trauma or surgical managements like encircling procedure may somehow influence the development of the eyeball and orbit.
4. The biological reaction of ocular tissue to either trauma or surgery may be more serious compared with those of adult patients, which is one of the factors of poor prognosis.

23.2 Epidemiology of Pediatric Ocular Trauma

The classification of pediatric ocular trauma is generally the same as that of adult [1]. Most cases have been caused predominantly by mechanical injuries [2–8]. The epidemiology of pediatric

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trauma is relatively less, although there have been reports published [9, 10]. An epidemiology study in Maryland America 1990 revealed that 15.2% of pediatric trauma patients were below 15 years old [11], whereas 22% of the hospitalized patients were below 15 years old in a Scotland study [12]. Approximately 17% of subjects with open globe eye injuries were children. The social-economic conditions of a family are one of the most important risk factors of eye trauma in children. As shown in some of the published data, children under the age of 5 years in families of low social income were more vulnerable to eye trauma in their family or living surroundings [13].

23.3 Clinical Evaluation and Management of Pediatric Eye Trauma

The history taking of trauma can be hard and inaccurate in children, especially in those of low ages. The patient's visit to the hospital is usually accompanied by the parents. The children are mostly incapable of describing the incidence of trauma in detail, or they may deliberately hide the truth duo the pain and fear, which may cause difficulties in making correct diagnoses and management.

Physical examination of the traumatized eye is not easy because of the pain and fear, so it is important for doctors to figure out ways to calm down the anxieties of both the patients and their parents.

Evaluation of visual function is the first thing to do at the primary visit. If the patients are unable to cooperate with visual acuity reading, other means like inducing reactions to flashlight or special objects can also be helpful for judgment.

The eye examination is generally the same as that of adults, from external to internal, from anterior to posterior segment. For those children who are constantly crying and struggling, sedation or general anesthesia may be needed to facilitate both physical and other equipment examinations, like B-scan and CT.

Checking of external eye should include the superficial skin damages, presence of foreign bodies, rupture of lacrimal duct, intrusion or extrusion of eyeball and the eye movements. Early presence of eye movement problems may indicate possible orbital wall fragmentation and incarceration of the eyeball. In cases of a large foreign body in orbit, the possibility of intruding into the carinal cavity should be excluded before removal.

To avoid excessive fear of patients, the primary eyeball evaluation should be painless and non-contact. A flashlight can be used to determine the pupil reflex and anterior injury. Sedation or even general anesthesia are usually needed for more detailed inspection of eyeball or equipment examinations like B-scan or CT. In cases with open globe injury, intraocular pressure should be felt with fingertips gently not to trigger the tissue loss.

Any evidence of conjunctiva and cornea break may imply the possibility of eyeball integrity problem. Subconjunctival hemorrhage may obscure the underlying rupture. CT scan is recommended when younger patients are unable to cooperate with the conventional eye examination. A deep laceration of the cornea may cause leakage of aqueous humor and hypotony, which can be clarified with fluorescein staining. It is important to pay attention to possible presence of intracorneal foreign bodies, especially when the foreign bodies are transparent like glass or plastics.

Anterior chamber hemorrhage is common in close globe trauma. Minor hemorrhage can be observed for spontaneous resolution if the intraocular pressure is normal or controlled with medication. Massive and clotted hemorrhage in the anterior chamber may cause secondary glaucoma and cornea blood staining if left untreated, therefore surgical removal with anterior chamber irrigation is indicated.

The anterior chamber should also be checked carefully to exclude the presence of foreign bodies, especially those that rest in the lower anterior

chamber angle. Once been found, surgical removal is needed by any necessary means.

Any defects of iris should be evaluated for related visual malfunctions like photophobia and blurred vision. Surgical repair to its utmost anatomy and histology is always recommended.

23.4 Emergency Management of Trauma

Following the primary office examination, emergency treatments should be done as soon as possible. A further detailed examination is still needed during the surgery to exclude the underlying or posterior rupture of eyeball. Any rupture or laceration that causes a leak should be sutured. Intraocular pressure should be maintained to its normal level. General anesthesia is usually indicated, especially in patients of lower age.

23.5 Managements of Pediatric Traumatic Cataract

Traumatic cataract is common both in penetrating and in non-penetrating injury. When vision is impaired, amblyopia may happen if cataract removal is delayed in children. A detailed evaluation of lens status should be done to determine the value of surgery. For those patients with partial lens opacity beyond the visual axis, lens sparing may retain the accommodation. Surgery is only indicated in a significant cataract that causes vision reduction or lens capsule rupture. The surgery varies from simply ECCE or phaco to lensectomy combined with vitrectomy. Cataract in low-age patients can be removed with a vitreous cutter of small gauge. It is still controversial whether intraocular lens should be implanted simultaneously during pediatric traumatic cataract surgery. Due to the uncertainty of biometry and the postoperative reactions of trauma cases, simultaneous intraocular lens implants should be careful. There have been a number of reports of postoperative complications like iris synechia, secondary glaucoma, and posterior capsule opacity. Improper lens implant in children may also increase the risk of posterior segment complications like retinal detachment and traction. Postoperative optical correction is needed with or without intraocular lens implant to avoid possible amblyopia.

23.6 Timing of Secondary Vitreous Surgery

For those patients with injuries like retinal detachment, vitreous hemorrhage and intraocular foreign bodies, modern minimal invasive vitreous surgery of either 25G or 27G is the most important surgery of choice.

The timing of secondary vitrectomy is still controversial. Early vitrectomy or even emergency vitrectomy may be difficult due to the hemorrhage or the low pressure caused by leaking, while delayed surgery weeks or months later may be more challenging because of severe retinal detachment and proliferative vitreoretinopathy (PVR). In patients with evidence of retinal detachment, early surgical intervention is recommended. A timely repair of retinal detachment has been reported to be correlated with a better visual recovery.

23.7 The Features of Pediatric Vitrectomy

Due to the anatomic and biochemical characteristics of children, vitrectomy in a traumatized pediatric eye can be challenging. Pediatric vitrectomy is not a mini version of adult vitrectomy. It differs in many aspects from conventional adult vitrectomy like anatomy, biochemistry, and postoperative proliferation. The vitreous is more intense and firmly attached to the retina, causing it difficult to produce posterior vitreous detachment

(PVD). PVR may develop and progress aggressively in children following trauma. Severe PVR is the most important cause of poor visual prognosis with or without vitrectomy. Pediatric retinal detachment with minimal or moderate PVR may be repaired with buckle surgery or combined with vitrectomy.

The entry locations of trocars should be based on different diseases and the anatomy of different ages. A well-controlled intraocular pressure is important with a setting by the equipment during the surgery, for a prolonged elevation of IOP may induce permanent visual damage.

In severe pediatric cases with retinal detachment, a complete clean vitrectomy of both core and peripheral vitreous is crucial, but challenging. Due to the firm adhesion of vitreous and retina, especially at the vitreous base, making a perfect posterior vitreous detachment (PVD) can be difficult and special attention should be paid to avoid iatrogenic retinal complications. In cases with retinal breaks, laser photocoagulation is recommended over cryotherapy which has a tendency to induce more aggressive PVR.

For those pediatric patients with severe peripheral traction, combined buckle surgery and vitrectomy may still have a role, but there are few issues to be considered following the procedure, like the postoperative elongation of eyeball and the related refraction changes.

Tamponade of vitreous cavity is important for retinal reattachment following vitrectomy. Although there is a tendency to use gas over silicone oil in adult vitrectomy, silicone oil is still a major choice in pediatric trauma patients, due to children's inability to keep face down position and more aggressive postoperative PVR. Some severe cases with persistent traumatic hypotony may even need long-term silicone oil tamponade to maintain the intraocular pressure.

Besides, the postoperative posture, visual rehabilitation should be always bear in mind to reduce the danger of amblyopia. Visual correction is always recommended in patients with lens removal and silicone oil tamponade while vision remains useful. In severe cases with traumatic phthisis bulbi, ocular plasty is needed for cosmetic and psychological purposes.

23.8 Prevention of Pediatric Trauma

The treatment of pediatric ocular trauma is far more difficult than adult patients, and the prognosis is generally poor than an adult. So, the prevention of pediatric ocular trauma is even more important [14]. The epidemiology of trauma in children has shown different features of different ages. Injuries of children under 3 years age are frequently related to nursing behaviors and happened mostly in housing environments. Children between 3 and 7 years old are more vulnerable to trauma in their playgrounds, while school-age children often got injured in schools and sports grounds. There has been an increase of eye trauma in children from traffic accidents in China during the last decades. By understanding the epidemiology, measures should be taken in accordance, and law enforcement of children's protection against dangerous toys and behaviors are far more valuable.

References

1. Knhn F, Morris R, Witherspoon CE, et al. A standardized classification of ocular trauma terminology. Ophthalmology. 1996;103:240–3.
2. Ariturk N, Sahin M, Oge I, et al. The evaluation of ocular trauma in children between ages 0–12. Turk J Pediatr. 1999;41:43–52.
3. Chan T, O'Keefe M, Bowell R, et al. Childhood penetrating eye injuries. Ir Med J. 1995;88:168–70.
4. Desai P, MacEwen C, Baines P, et al. Incidence of cases of ocular trauma admitted to hospital and incidence of blinding outcome. Br J Ophthalmol. 1996;80:592–6.
5. Farr A, Hairston R, Humanyun M, et al. Open globe injuries in children: a retrospective analysis. J Pediatr Ophthalmol Strabismus. 2001;38:72–7.
6. LaRoche G, McIntyre L, Schertzer R. Epidemiology of severe eye injuries in childhood. Ophthalmology. 1989;95:1603–70.
7. MacEwen C, Baines P, Desai P. Eye injuries in children: the current picture. Br J Ophthalmol. 1999;83:933–6.

8. Nelson L, Wilson T, Jeffers J. Eye injuries in childhood: demography, etiology, and prevention. Pediatrics. 1989;84:438–41.
9. Rapoport I, Romem M, Kinek M, et al. Eye injuries in children in Israel. A nationwide collaborative study. Arch Ophthalmol. 1990;108:376–9.
10. Smith G, Knapp J, Barnett T, et al. The rockets' red glare, the bombs bursting in air: fireworks-related injuries to children. Pediatrics. 1996;98:1–9.
11. Strahlman E, Elman M, Daub E, et al. Causes of pediatric eye injuries. A population-based study. Arch Ophthalmol. 1990;108:603–6.
12. Vasnaik A, Vasu U, Battu R, et al. Mechanical eye(globe) injuries in children. J Pediatr Ophthalmol Strabismus. 2002;39:5–10.
13. Jandeck C, Kellner U, Bornfeld N, et al. Open globe injuries in children. Graefes Arch Clin Exp Ophthalmol. 2002;238:420–6.
14. Moreira CJ, Debert-Ribeiro M, Belfort RJ. Epidemiological study of eye injuries in Brazilian children. Arch Ophthalmol. 1988;106:781–4.