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Changing Trends in the Management of Ocular Trauma

One of the most challenging issues in ophthalmology is the management of an injured eye. This is so because 1. Many a times eye injury may be associated with other life threatening injuries which means multi disciplinary approach may be needed 2. Most of the times it comes in odd hours of the day or night when the doctor may not be reachable 3. Usually eye injuries happen in working class people who are socio economically backwards 4. Always good patient counseling is mandatory in this situation. This can avoid unwanted situations in emergency department 5. Eye injuries happen in young people who are in their productive life. This will have economic implications. 6. Medico legal aspects

Gone are the days when a badly injured eye was considered as a gone case. With the current technical advancements most of the badly injured eyes may attain ambulatory vision. The main problem in management of ocular injuries is that there is no standardized methodology even now while approaching a case. Even the terminologies used by different doctors may differ. Ocular trauma society has come up with standardized nomenclature and grading system [1]

Terminologies
Eye wall – Sclera and cornea
Closed globe injury – No full thickness wound of the eye wall
Open globe injury- Full thickness wound of the eye wall
Contusion – No full thickness wound of eye wall
Lamellar laceration – Partial thickness wound of the eye wall
Rupture – Full thickness wound of the eye wall caused by a blunt object
Laceration – Full thickness wound of the eye wall caused by a sharp object
Penetrating injury – Has an entrance wound / retained intraocular foreign body
Perforating injury – Has entrance and exit wounds

When a patient presents in the emergency department with eye injury initial assessment and grading can help in giving the prognosis and also in medico legal aspects. Ocular trauma score is made for this purpose. Initial visual acuity is checked and if it is >/- 20/40 a score of 100 is given. From this depending on the ocular findings points are subtracted to get the final score (Table 1). For example if the patient has 20/200 vision with perforating injury and afferent papillary defect raw score sum will be 90-14-10=66. If you see table 2 this is OTS 3 and he has 44% chance of getting final vision of 20/40 or better.

Birmingham Eye Trauma Terminology System (BETTS)
The double-framed boxes show the diagnoses that are commonly used in clinical practice
A standardized terminology and approach will help in the maintenance of eye trauma registry for future research. This is extremely important in current scenario with lot of medico legal allegations.

This issue of KJO is a special issue or a collector’s item. We do not have many text books or literature in ocular trauma. Ocular emergencies are encountered by most of the ophthalmologists. This issue is covering most of the aspects of management of eye injuries. This involves not only primary repair but also second stage management and rehabilitation. Articles are contributed by experts in the field. Topics include primary repair, imaging, treatment of infections, management of optic neuropathy, special settings such as paediatric trauma and poly trauma, management of glaucoma and hyphaema and medico legal aspects. I am sure that this collection will help each and every one of us. Also the regular features will be continued.

I hope you will have a happy and useful time reading this issue and also look forward to hear from you regarding this issue.

Mahesh G

References:

<table>
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<th>Table 1. Computational method for deriving the OTS score</th>
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<tr>
<td><strong>Initial Visual Factor</strong></td>
</tr>
<tr>
<td>----------------------------------------------------------</td>
</tr>
<tr>
<td>A. Initial visual acuity category</td>
</tr>
<tr>
<td>NLP = 60</td>
</tr>
<tr>
<td>LP to HM = 70</td>
</tr>
<tr>
<td>1/200 to 19/200 = 80</td>
</tr>
<tr>
<td>20/200 to 20/50 = 90</td>
</tr>
<tr>
<td>≥20/40 = 100</td>
</tr>
<tr>
<td>B. Globe rupture</td>
</tr>
<tr>
<td>-23</td>
</tr>
<tr>
<td>C. Endophthalmitis</td>
</tr>
<tr>
<td>-17</td>
</tr>
<tr>
<td>D. Perforating injury</td>
</tr>
<tr>
<td>-14</td>
</tr>
<tr>
<td>E. Retinal detachment</td>
</tr>
<tr>
<td>-11</td>
</tr>
<tr>
<td>F. Afferent pupillary defect (Marcus Gunn pupil)</td>
</tr>
<tr>
<td>-10</td>
</tr>
</tbody>
</table>

**Raw score sum = sum of raw points**

<table>
<thead>
<tr>
<th>Table 2. Estimated probability of follow-up visual acuity category by the OTS Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Raw Score Sum</strong></td>
</tr>
<tr>
<td>-------------------</td>
</tr>
<tr>
<td>0-44</td>
</tr>
<tr>
<td>45-65</td>
</tr>
<tr>
<td>66-80</td>
</tr>
<tr>
<td>81-91</td>
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<tr>
<td>92-100</td>
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</table>
Open Globe Injuries-Primary Repair of Corneoscleral Injuries

Biju John MS, FRCS. Chithra Raghavan DNB.

Introduction
Open Globe Injury by definition is a full thickness defect in the cornea and or sclera. The care of such patients call for an approach which should be systematic and methodical, but at the same time gives enough opportunity or challenges (depending upon how you perceive it) for deviating from the well trodden path. Each case can be unique and calls for creativity and flexibility from the part of the surgeon. The need for a standardized terminology of the eye injury types has led to the now widely accepted classification designed by the Ocular Trauma Group based on the “Birmingham Eye Trauma Terminology”. The classification is dealt elsewhere in this journal but in a nutshell is given below in the following table¹,²

Table 1: Classification of Open Globe Injuries

<table>
<thead>
<tr>
<th>Type</th>
<th>Grade (Visual acuity)</th>
<th>Pupil</th>
<th>Zone</th>
<th>History</th>
</tr>
</thead>
</table>
| A. Rupture    | ≥ 20/40               | Positive Relative APD in injured eye | I. Cornea and Limbus   | First priority in any case of trauma ocular or non ocular is of course, the systemic status. So a general assessment of the injuries, recording of the vital signs etc are carried out first, usually in a triage area if available. Once the general condition is found to be stable, and other major organ injuries are taken care of, ophthalmic history and evaluation begins. History should be given the importance it deserves. A properly elicited history tell us a lot about the type of injury-open or closed globe, nature of the penetrating material, and the setting (work; home etc). A good history should lead the physician to make risk assessments for the possibility of globe perforation; occult rupture; posterior rupture; intraocular foreign bodies (IOFB),chemical exposure; endophthalmitis etc and fine tune the evaluation keeping in mind these clues. However one warning regarding history in Ocular Trauma- In our experience this is one condition (is it the only one?) where the patient might sometimes try to intentionally mislead the examiner away from the actual cause or nature of the injury. The devoted house wife who wouldn’t admit to the husband’s hand (literally or the hand might have held something) in the injury, the seemingly innocent child trying to escape the wrath of the grown ups or protect the involved friend or sibling- There are lots of examples. One case which we still vividly remember is the case of a 10 year boy with a clean cut laceration on the cornea due to a hit from a rubber ball. It was with great difficulty and round about questioning that we could extract from him that the rubber ball had rebounded from a heap of broken glass pieces and everything ended well with an active search for a foreign body revealing a small glass piece in the bottom of the anterior chamber. Ophthalmic Evaluation A complete and thorough ocular examination, keeping in mind the clues obtained from the history is the next vital cog in the trauma management wheel. This should include flash light examination; Slit lamp examination and fundus examination whenever possible. See Table No. 2 for some of the relevant findings to be looked for with the slit lamp. Poor presenting visual acuity and relative afferent pupillary defect are the most significant prognostic factors that can be detected on presentation. Signs such as diffuse chemosis; massive subconjunctival haemorrhage; asymmetric deepening of anterior chamber; Low intraocular pressure; “uveal show” under the conjunctiva; hemorrhagic choroid detachment etc especially in combination should make one think of and actively search for a scleral rupture which may be trying to escape detection owing to the intact conjunctiva or chemosis above it or due to its posterior location or location beneath the muscle insertion. If the initial examination still fails to exclude a rupture or a hidden full thickness scleral wound and even an iota of suspicion remains, then don’t hesitate to do an exploration in the OT after doing the necessary peritomy. In case of children this of course means general anesthesia.

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Imaging in Ocular Trauma

CT Scan is the imaging modality of choice in the acute setting of an Ophthalmic trauma were we are suspecting an Open globe injury. This can uncover or give corroborative evidence in cases of Occult globe rupture; detect IOFBs; give an idea of the orbital pathologies like retro bulbar hemorrhage; Orbital wall fractures. The B Scan Ultrasound is also very useful and is definitely a better test than CT to evaluate posterior segment structures, but it’s use in Open Globe injury on presentation is limited due to the necessity of contact of the probe with the cornea or lid. However following the primary repair it definitely has a role and can even identify some non metallic foreign bodies sometimes missed in the CT Scan like glass or wood. In fact not doing it in spite of having the facility may have serious medico legal implications. Magnetic Resonance imaging even though can be more sensitive and accurate than CT, is severely limited by the fact that it can’t be used when we are suspecting a metallic foreign body. But consider a situation where the patient is pregnant and the possibility of a magnetic foreign body is not very high. MRI may be the answer.

When state of the art imaging modalities like the above cannot be employed due to non availability or due to economic reasons then the Plain film Radiograph is still valuable.

Table: 2 Relevant findings to be looked for during Evaluation with Slit Lamp

<table>
<thead>
<tr>
<th>Structure/Layer</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conjunctia (manipulate conjunctival wound edges gently to look for underlying pathology)</td>
<td>Chemosis; Hemorrhagic chemosis; Foreign bodies sequestered in redundant folds and fornices</td>
</tr>
<tr>
<td>Subconjunctival space</td>
<td>Foreign bodies; Expelled Lens/Intraocular Lens; Prolapsed Uvea/Retina/Vitreous</td>
</tr>
<tr>
<td>Cornea</td>
<td>Epithelial defects; Flaps and any foreign material under the flaps; Siedel’s test if necessary to confirm wound leak or full thickness nature of laceration; Foreign body –part of which is in the Anterior chamber-Remove only in OT</td>
</tr>
<tr>
<td>Sclera</td>
<td>Not so obvious full thickness wounds; Occult rupture; Posterior rupture</td>
</tr>
<tr>
<td>Anterior Chamber</td>
<td>Cells; flare; fibrin; hypopyon; hyphema; IOFB; Shallowing – uniform/Irregular</td>
</tr>
<tr>
<td>Iris and angle</td>
<td>Sphincter tears; Iridodialysis; Iris holes; Iridodonesis; foreign bodies</td>
</tr>
<tr>
<td>Lens</td>
<td>Subluxation; dislocation; Anterior capsule or Posterior capsule defects ;Cataract formation; Leakage of Lens material into AC;Intralenticular foreign body</td>
</tr>
<tr>
<td>Ophthalmoscopy (Defer dilated examination in cases with iris prolapse till primary repair is over.)</td>
<td>Hemorrhages in various locations/layers; choroidal ruptures; Retinal breaks; IOFB ; Macular edema etc</td>
</tr>
</tbody>
</table>

Documentation
Accurate and methodical documentation of all the relevant details in the history and evaluation is extremely important for clinical and medico legal reasons. What you wrote or did not write in the case record might turn out to be more important in the court than what you did or did not do.

Initial Management and Planning the Surgery
Surgical repair of a severely traumatized eye is a complex procedure involving expertise, skill as well as proper infrastructure. If for any reason an ophthalmic surgeon say in a general ophthalmology set up feels that he/she cannot address all treatable lesions that require emergency management, it is usually preferable to refer the patient prophylactic systemic and topical antibiotics with broad spectrum coverage has to be started.

Timing of Intervention in Open globe injuries
The risk of endophthalmitis does not significantly increase in the first 24 or 36 hours (43) after the injury. So if appropriate equipment , staff or anesthetist is not available immediately, it is justifiable or even advantageous to wait for a few hours. However if the same is not an issue then it can be done immediately. In our Institute we make sure that the primary repair is done within 12 hours. Interventions like lens removal
if necessary, IOFB s without much risk of endophthalmitis; repair of retinal detachment etc can be done as a second planned procedure.

**Anesthesia**

General Anesthesia is usually preferred, as you can avoid retro bulbar/peribulbar injections which can induce or aggravate prolapse of intraocular tissues with a lot of undesirable consequences. However systemic risks in some patients; and a lot of practical considerations combine together to result in local anesthesia via the peribulbar or retro bulbar route, still being widely used in many centers. Topical Anesthesia supplemented by IV sedation can also be used in selected cases (7).

**Primary Repair of Injuries Involving Cornea**

**Self Sealed Corneal Wounds**

The first question that we generally ask ourselves in these types of injuries is of course whether suturing can be avoided. We call them self sealed corneal wounds which on slit lamp examination appears exactly as described by the term with the full thickness wound appearing as an irregular line with good anterior chamber depth , no iris in between the wound lips. Well whatever be the line of management you choose always think about and rule out the presence of an intraocular foreign body. As far as primary repair is concerned conservative management with only prophylactic antibiotics and no surgical repair is enough for very small self sealed corneal wounds of 2 mm or less, provided there is no other intraocular tissue at the wound, no other ocular structures are involved , no foreign material is present in the wound and Seidel’s test is negative. In such cases where it has been decided not to suture, it is always better to place a bandage contact Lens over the cornea and to leave it there for 2-3 wks. Intraocular tissue at the wound, no other ocular structures involved , no foreign material is present in the wound and Seidel’s test is negative. In such cases where it has been decided not to suture, it is always better to place a bandage contact Lens over the cornea and to leave it there for 2-3 wks.

Conservative management with only prophylactic antibiotics and no surgical repair is enough for very small self sealed corneal wounds of 2 mm or less, provided there is no other intraocular tissue at the wound, no other ocular structures are involved , no foreign material is present in the wound and Seidel’s test is negative. In such cases where it has been decided not to suture, it is always better to place a bandage contact Lens over the cornea and to leave it there for 2-3 wks. Topical Antibiotics and steroids can be continued.

For Larger Self Sealed Lacerations- There are 2 options.
1. Routine surgical repair
2. Cyanoacrylate Tissue glue with Bandage contact Lens.

The surgeon can select one of these based on his experience, expertise and comfort zone. Suturing may result in more astigmatism and also brings into the picture a 2nd intervention even though minor in the form of a suture removal later.

Whenever you are selecting conservative management or gluing over surgical repair, Ask yourself this question?
1.Is there a risk of the wound reopening especially once the patient returns home and engages in his routine work? Eg: inadvertent rubbing, accidental bumping of the eye.
2. Children are not good candidates for this line(non surgical) of management.

**Tip:** An approximate idea of the strength of the seal can be obtained by applying some pressure on the sclera with one finger through the lids while observing under slit lamp and looking for any wound gape or shallowing of anterior chamber (a sort of simulation of the forces that can act on the wound once the patient is out of the hospital). Double check with a Siedel’s test.

**Corneal Lacerations with Flaps**

When the wound is not full thickness or only approaching full thickness in a very small area so that it is well sealed, the aim should be to see that whether the flap can be kept well apposed in its correct anatomical location without sutures, as suture in such case may only induce additional astigmatism and may not be of any benefit in the wound healing. So if it is not displaced a bandage soft contact Lens is all that is required.

However if the flap is displaced, it has to be repositioned and secured with sutures. In such cases make sure that sutures are partial thickness through the surrounding stroma. The tightness and number of sutures should be just enough to hold the flap in place.

Some cases which present late (after 24 hours), may have epithelial growth underneath the flap and this has to be debrided off before suturing. Any way in a displaced flap it is a good idea to irrigate the bed and undersurface of the flap to clean off any debris, foreign body particles etc.

**Full thickness Non Self Sealing Corneal Wounds**

These require surgical repair in the operating room with 10-0 or 11-0 nylon sutures. The principles of suturing are dealt with separately. A small piece of advice to the young surgeons- Having done a good job in suturing – Please do not forget the finishing touches-viz burying your knots. When you do that then you might suddenly realize that many of your suture ends are quite long and requires trimming. If you don’t do this then you will get a few patients who cannot even open their eyes in the convalescent period due to the intense suture irritation and watering. Before long you can even see large papillae in the upper tarsal conjunctiva.

**Principles of Surgical repair of Corneal Lacerations**

Our aim here should be the restoration of the optically clear, smooth surface and curvature of the cornea as even small irregularities in the curvature can lead to significant visual disability.

The surgeon should already have a plan in mind of the surgical steps going to be done and in what order. The plan is of course based on the initial evaluation findings. So during the initial slit lamp examination itself the surgeon should be on the look out for the normal anatomical land marks and
other features that will aid in apposing the edges of the wound correctly and restoring the displaced tissues to their correct anatomical location. The idea is that apposition of the edges of the laceration with properly placed sutures should first happen at these landmarks. Once this is done correctly the chances of incorrect apposition of the remainder of the laceration are minimal and suturing can proceed smoothly.

Such landmarks which can be found in a corneal laceration are illustrated in Figure 1 below.
1. Limbus
2. Stellate Edges
3. Pigmentation Lines in the Epithelium
4. Sharp angles of the laceration

It is very unusual to get a corneal laceration without at least one of the above. Irrespective of whether he/she has made a plan taking into consideration all these in the initial evaluation, a reassessment should be done on the table after cleaning off debris if any from the wound edges and making the wound lips free of any extraneous tissue including iris (by repositioning/abscising as the case may be), vitreous etc. Based on this a final strategy on how to proceed with the wound repair should be made and executed.

**Basic Suturing Techniques**

**Suturing techniques**

**Interrupted Sutures**

The most preferred suturing method to appose the wound edges is interrupted suturing placed with 10-0 or 11-0 nylon with spatulated needle. Ideally a round suture loop should be placed in a single plane so that the 2 edges will be having a layer to layer apposition. Suture passes should be approximately 1.5 to 2 mm total in length i.e. 0.75 to 1 mm on either side. In case of edematous or macerated wound edges slightly longer passes may be required to incorporate healthy tissue by the suture. Equal amount of tissue should be incorporated on each side of the wound. The depth of the sutures should be 85-90% of full thickness, which would mean that the needle passes over the Descemet's membrane. Full thickness corneal lacerations generally have one of the following 2 anatomical configurations.
1. A more or less Vertical (perpendicular) laceration
2. An oblique (shelled or beveled) laceration

The 2 types require 2 slightly different approaches to suturing so as to facilitate correct wound edge apposition without any overriding or distortion.

In vertical lacerations the suture entry and exit sites should be equidistant from the wound margins so that the corneal suture is centered over the wound (See Fig 2). However we don't have to worry whether it is centered with respect to the anterior aspect of the wound or posterior aspect of the wound as both would be in the same perpendicular line. If any of the limbs of the suture is longer than the other, the wound edge on that side will override the other edge when tightened.

In beveled or shelved lacerations a slightly different approach is required. Here if you are taking the bites equidistant from the anterior aspect of the wound margin, there will be wound overriding and tissue distortion. To prevent this care should be taken to ensure that the suture is centered on the posterior aspect of the wound margin. This means that the suture entry and exit sites will be displaced with respect to the anterior aspect of the laceration but will be equidistant with respect to the posterior aspect. (See Fig 3)
Figure 3: Suturing of a shelved laceration. The distance from the anterior margin of the wound to the suture entry site (A) is not equal to that from the same point to the suture exit site (B). But what matters here is the distance from the entry and exit sites to the posterior margin of the wound (C & D), which is equal (C=D). (Adapted from 101 Pearls in Refractive, Cataract, and Corneal Surgery second edition Edited by Samir A. Melki and Dimitri T. Azar)

Note that ideal depth through which the suture should pass is 90% (14) which should take it just over the Descemet’s membrane.

An important factor to be taken into account during suturing of corneal lacerations is the issue of edematous wound edges frequently seen when the time gap between the injury and time of suturing is more than a few hours. This results in 2 problems

1. Because of the corneal edema being localized, you may end up with 2 opposing edges differing significantly in thickness. How to ensure layer to layer approximation (that is our aim) of the edges in such a situation?

2. Once the corneal edema decreases in the immediate post op period aided significantly by the topical anti-inflammatory drops that we invariably give, some of the sutures which we thought were of the correct tensile strength and producing good apposition are suddenly found wanting in doing the job they were entrusted with-viz holding the wound edges in close approximation.

A solution suggested by some authors for the first problem is to employ full thickness box suturing (15) (See Figure 4) as when the full thickness of the corneal edges are incorporated in the suture loop, there is anyway going to be good approximation -almost layer to layer irrespective of whether there is thickness difference or not. To achieve the same approximation with a partial thickness suture in such a situation will require considerable skills from the surgeon. However there is the risk, even though minimal of the suture acting as a conduit, enabling microorganisms or epithelial cells to enter the eye (15). Personally we try to avoid full thickness sutures as far as possible. The 2nd issue has to be kept in mind when the sutures are tightened and that allowance has to be given.

Care must be taken to see that the sutures are always placed at right angles to the wound edge to avoid wound slippage (See fig 5).

Tightening of the suture will cause compression of the tissues, but if correctly done there will not be any eversion or inversion of the edges. The tissues on either side of the laceration will be well apposed without any displacement and the chances of slippage later are minimal. Of course the suture tightening will cause some surface distortion. But this will get corrected later once the wound has healed and the sutures are removed, provided no anatomic distortion occurs during the wound healing. Once the flattening effect of the sutures is relieved by their removal the corneal curvature will return back to normal. On the other hand if wound slippage or misalignment has occurred at the time of suturing or during the immediate post operative period, both due to incorrect suture placement, well then the distortion that you
get will be permanent and no amount of suture removal or suture adjustment is going to correct that.

**Figure 5A and B.** Sutures should be placed at right angles to the wound at the point of the suture/wound intersection. Sutures at acute angles will cause wound slippage with tightening as illustrated (3) (Taken from Text book of Ocular Trauma Principles and Practice-Ferenc Kuhn; Dante j Pieramici)

**Running sutures**

Employing these may make the whole procedure faster. We use these for some lacerations with clean cut straight edges (e.g.: some knife wounds). They provide a continuous and uniform zone of compression (with some adjustment of tension before tying) and results in good wound apposition in ideal conditions. However the disadvantages are as follows

- Large zone of compression and thus excessive flattening of the cornea
- Misalignment of wound edges due to suture induced edge slippage
- Straightening of curvilinear incisions
- Rippling of corneal surface if sutures are not placed in the same depth or full thickness

So the continuous or running sutures can be employed in selected cases with good benefits.

**Rowsey-Hay’s Technique of Corneal Suturing and its importance**

Restoring the previous corneal curvature by your suturing-well that is attempting to recreate the exquisite work of the creator. You might be found severely wanting there. Our aim should be rather to provide the patient with a central cornea with uniform spherical contour rather than trying very hard at recreating the previous contour. This is facilitated by the Rowsey-Hay’s technique of corneal wound closure illustrated in Figure 6

Some sort of a qualitative keratometry can be utilized to ensure that the central corneal curvature is uniform. An easily available instrument in the OT for this will be a Fleringa ring. If that is not available a hinge spring of a safety pin may suffice. After the initial suturing, the surgeon can hold this ring over the cornea and utilizing the coaxial illumination, its reflection from the epithelial surface is examined. The ideal reflection should be a round circle. If there is some astigmatism induced, the ring appears oval or distorted. In such case the surgeon can then tighten or loosen some sutures keeping in mind the Rowsey-Hay principle so as to get an ideal reflection. The problem of course is that we hadn’t applied any adjustable sutures here. So what to do now? No other way than to remove and reapply some sutures. The other way of course is to apply these sutures initially in a manner in which they can be adjusted later i.e. by employing some sort of slip knots (e.g.: sliding clove hitch or sliding half hitch).

**Special Situations**

**Loose fragments**

Full thickness corneal wounds with a lot of loose fragments often displaced in impossible angles, can be really tough to repair. However all these fragments have to be patiently and meticulously repositioned into their normal anatomic position. Having done this the next big problem is how to
keep them there? As a first step try to place sutures through the edges of the fragments. If it works, good. If not then consider the following additional options
1. Oversewing
2. Using Bandage contact Lens as a splint
3. Glue

Stellate Wounds
Next to loss of tissue, this is the most difficult problem in corneal wound repair. They may require a combination of sutures and tissue adhesive, and sometimes, a patch graft for a proper closure. A purse string technique has been proposed by Eisner. (8) In some cases, the additional use of cyanoacrylic glue can be helpful.

Injuries to LASIK Flaps
The corneal flaps created during LASIK are vulnerable to traumatic dehiscence and dislocation, even years after the procedure. A partially displaced flap can be managed with a bandage contact Lens while a completely displaced flap should be repositioned and secured with sutures. It is always better to get the help of a refractive surgeon in these cases.

Loss of Tissue
This situation is extremely rare and in most cases the missing piece of the puzzle (tissue) will be there—mostly turned down as a flap into the anterior chamber or just displaced from its normal site. So before allowing the alarm bells to ring, do a careful examination of the full thickness of each of the wound edges, if necessary with the help of a blunt instrument like an iris repositor to detect and reposition such corneal fragments or displaced flaps. Once that is done all that is remaining will be to apply a few well placed sutures.

However wounds with loss of tissue can occur in a few cases of trauma with high speed missiles as in gunshot wounds or explosions. If the defect is small, the area can be closed with tight sutures even though it can result in significant amounts of tissue distortion and wound tension and may later cause significant irregular astigmatism. However when such tissue loss exceeds 5 mm in diameter a corneal patch graft is usually required. Full-thickness patch graft is technically easier to perform but requires a donor cornea. A lamellar patch graft is effective and may be performed with a corneal auto graft or donor sclera. These grafts are often located outside of the visual axis; therefore, graft clarity may not be essential for good postoperative vision.

Some general considerations while repairing corneal wounds
1. Handling of tissue should be kept to minimum. Repeated attempts at grasping the wound edges can lead to maceration especially as the tissue might be edematous and fragile. The surgeon will be making his job more and more difficult by doing this. The spatulated needle point of the 10-0 should be prevented from touching any other tissue other than the wound edge as it can easily get blunt and would make suturing more difficult, thereby forcing the surgeon to do just what he should not be doing i.e. grasping and regrasing the tissue. Many a time maintaining the anterior chamber with a tight air bubble lets you do the suturing without needing to grasp the wound edges with forceps or by just fixing the globe gently by grasping at the perilimbal episcleral tissue. An exquisitely sharp spatulated tip for the suture needle is absolutely necessary for this.
2. Options to be considered when wound leak persists in spite of suturing
   i. Bandage Contact Lenses
   ii. Tissue adhesives
   iii. Patch graft
3. Avoid putting sutures in the visual axis as far as possible. If unavoidable make sure that the suture limbs are kept very short.

Scleral and Corneoscleral Injuries
The main goals in the management here include
1. Restoration of integrity of the globe
2. Avoidance of Further injury to Ocular tissues
3. Prevention of Corneal scarring and astigmatism

Very small scleral defects as in a puncture wound by a sharp thin wire or the common broom stick, without uveal prolapse can be managed conservatively with appropriate antibiotic therapy. However most of the larger scleral wounds require surgical repair.

Unlike the purely corneal injuries, scleral wounds especially ruptures can sometimes be missed since they can be hidden by the intact conjunctiva and/or large subconjunctival hematoma. So there has to be a high index of suspicion in cases with suspicious signs and typical history (see evaluation). Any lingering doubts are to be settled by a globe exploration under the appropriate anesthesia. If necessary a 360 degree peritomy is made as in RD surgery so as to retract the conjunctiva and provide good exposure of the sclera. Special attention is to be given to the areas of muscle insertions and the areas in between them as this is one of the most common sites for a rupture.

General Principles of Closure of Scleral Wounds
Full thickness scleral wounds are generally apposed with interrupted sutures with “8-0” or “9-0” Silk or Nylon. A needle with a spatulated end is to be used.

1. Ensure proper exposure and visibility of the wound edges. A common mistake by many of the beginners is involvement of the tenon’s or even the conjunctiva in the suture supposed to approximate the scleral wound edges. Worse there might be only tenons and no sclera. Best way to ensure this doesn’t happen is to clear the tenons and conjunctiva completely off the wound edges before passing the suture.
2. Involvement of any prolapsed or prolapsing tissue (uvea, retina or vitreous) in the suture is also to be diligently avoided. So it follows that any such tissue must be gently reposited back or abscessed away as the case may be before passing the suture. Vitreous of course should be amputated at the scleral surface. When doing this any unnecessary traction on the vitreous should be avoided. The wound edges may be raised gently with forceps while passing suture bites so as to keep any uveal tissue from being impaled by the suture needle. Using viscoelastics to reposition uveal tissue in a scleral laceration may not be a good idea as the viscoelastics may enter the sub retinal space from where it is only poorly absorbed resulting in excessive inflammation and increased risk of Retinal detachment.

3. Scleral wounds are generally closed from anterior to posterior direction. Just as in the cornea here also suturing should begin from a recognizable landmark. If the Limbus is involved then without doubt that is where the first suture should go. Otherwise we start from a recognizable landmark like the apex of the laceration.

4. Large wounds extending posteriorly, it is better to employ a “Close as You Go” strategy as illustrated in Figure 7. Also if you leave the last tied suture ends long, these ends can be used to exert some traction on the globe so as to rotate it anteriorly so as to expose more of the posterior portion of the wound.

5. But even with this method there is a limit to the posterior limit up to which you can go in a wound extending posterior to the equator. In such cases some portions of the posterior most portions of the wound might have to be left unsutured. It is better to do so than to cause additional damage and further tissue prolapse with extreme manipulations.

6. Following the same principles an isolated posterior scleral wound near the optic nerve may be best left alone unsutured, trusting the surrounding orbital tissue to give good tamponade as the wound heals.

7. In cases where the scleral wound extends through or under an extra ocular muscle, an assistant can retract the muscle gently using a muscle hook to aid in exposure. If more exposure is needed especially if the laceration is under the insertion of the muscle, the same may need to be temporarily disinserted so as to allow the suturing. Following the closure of the scleral defect the muscle may be reinserted.

**Corneoscleral Wounds**

We just need to combine the principles of corneal and scleral wound repair here. Commonsense dictates that the major landmark here is the Limbus and so that is the area to be apposed first. Next continue with repair of the corneal aspect followed by the scleral aspect adhering to the principles already discussed. See Figure 8 for illustration

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**Figures:**

Figure 7: The “Close as You Go” technique for Exploration and Primary Closure of a Scleral Wound. Here the visible anterior portion of the wound is exposed and sutured first and having reached the visible end a little more of the wound is exposed by opening the conjunctiva and Tenons there and sutures are applied to this exposed area. Having finished this more of the wound is progressively exposed and sutured. This ensures that the still to be sutured, open part of the laceration is supported by the periorbita and this will prevent the intraocular contents from prolapsing out. (Taken from Text book of Ocular Trauma Principles and Practice-Ferenc Kuhn; Dante J Pieramici)

Figure 8: Left side fig shows the anatomic Land marks identified in the corneoscleral wound. These are Limbus (1) and the angles of the wound (2, 3). So first suture at (1), then corneal part and then scleral part. (Taken from Text book of Ocular Trauma Principles and Practice-Ferenc Kuhn; Dante J Pieramici)

**Special Situations**

**Scleral Defects Not repairable by suturing alone**

This can occur in severe trauma to the globe or even not so severe trauma involving areas already thinned out or weakened by previous infections; inflammations or degenerations (e.g. staphyloma in high myopia). In such cases repair using a scleral patch graft is indicated. Depending upon the size of the defect and need for structural support, a variety of materials can be used for grafting as follows
Other alternatives when autologous tissue is needed and sufficient sclera is not available are Fascia Lata, Periosteum from anterior tibial crest or split –thickness dermal graft. However this would mean multiple incision sites and general anesthesia and the patient should be able to tolerate this. Details of the techniques of harvesting the graft materials and the actual grafting are beyond the scope of this article and the reader is advised to refer standard text books for the same. Some general principles to be noted during preparation of the recipient bed are

i. Any frankly necrotic or infected appearing tissue has to be removed and the devitalized or irregular margins trimmed using scissors or a sharp blade to form a smooth circular recipient bed. Be careful not to cause any further injury to the underlying uveal tissue.

ii. Another approach is to do a partial thickness trephination and use a sharp scissors or blade to remove the rest of the tissue in the bed. A suction trephine will be better suited for trephining in these hypotonous globes.

**Controversies**

1. **Reconstruction Vs Enucleating in No PL Eyes**
   The current thinking is that even if the globe appears very badly damaged and vision is NO PL, every effort should be made to preserve the globe in the emergency management setting. In such cases secondary enucleating can be considered if later the eye despite all the reconstructive attempts remain No PL and the risk of sympathetic ophthalmitis is perceived to be higher than usual. The decision can then be taken after discussion with other colleagues and also the patients and relatives. The psychological trauma inflicted on the patient by an enucleation in the acute period can be tremendous and is best avoided.

2. **Timing of Primary repair- Immediate Vs Delayed**
   This has already been discussed. The gist of the matter is that several studies have proved that the risk of endophthalmitis in open globe injuries does not significantly increase in the first 24 or even 36 hours. So if a few hours delay can improve the quality of the surgery and patient care, then it is better that way. However where the risk of infection is high as in large uveal prolapse; certain types of intra ocular foreign bodies such a delay may not be prudent.

3. **Primary Wound Closure Vs Comprehensive Reconstruction**
   In the former the primary management is limited to a proper wound toilette; management of the prolapsed intraocular tissue (repositioning or abscessing); Removal of any obvious anterior chamber and wound lip foreign bodies and apposition of the wound by suitable sutures. The advantages are that things are kept very simple requiring only average skill and expertise. A thorough evaluation can be carried out in the post operative period including B scan; CT scan and even electrophysiology and further management involving complicated procedures like vitreo -Retinal procedures can be planned and executed in consultation with other colleagues or even other centers.

On the other hand a truly comprehensive management involves tackling the other co existing pathologies like cataract, Posterior segment IOFB, Posteriorly dislocated Lens or IOL, Retinal detachment Vitreous hemorrhage etc and depends on the availability of ophthalmologists who are fully trained to work in the anterior as well as the posterior segment of the eye or a comprehensive team of Anterior and Posterior Segment surgeons along with the proper infrastructure and technical support.

So even though the first is definitely more practical and also the only option in many centers, the latter approach has some advantages some of which are as follows

1) Comprehensive management is less expensive.
2) Comprehensive management offers potential prevention of endophthalmitis by removing the inoculated media (eg an Intravitreal non metallic foreign body)
3) Comprehensive management offers the potential reduction of post-injury inflammation and the prevention of scar tissue formation, such as proliferative vitreoretinopathy (PVR), again by removing stimulating factors (cytokines) present in the vitreous cavity.
4) Comprehensive management offers earlier visual rehabilitation.

**Table 3**

<table>
<thead>
<tr>
<th>Type of Defect</th>
<th>Materials that can be used for Grafting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small defects with little need for structural support</td>
<td>Conjunctiva/Tenon’s capsule</td>
</tr>
<tr>
<td>Moderately sized defects requiring some structural support</td>
<td>Tarso-Conjunctival Flap</td>
</tr>
<tr>
<td>Small to Large defects requiring support</td>
<td>Autologous Sclera (stored in glycerin, alcohol or frozen in antibiotic solutions)</td>
</tr>
<tr>
<td>Large defects requiring structural support</td>
<td>Homologous Sclera</td>
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</tbody>
</table>
Research is still going on in this field and may ultimately settle this question in favour of one of the approaches.

4. **Role of Prophylactic Cryotherapy in Corneoscleral Injuries**

Prophylactic cryo in a setting like this was done by a few surgeons in the hope that it might prevent a retinal detachment. However this might be counterproductive as it may trigger increased intraocular inflammation and fibrosis thereby increasing the chance of a detachment. If retinal view is there a barrage laser should be considered.

5. **Role of Prophylactic Scleral Buckle**

Despite numerous studies, there are no substantial data to indicate whether a scleral buckle might decrease the subsequent risk of retinal detachment or even reduce the need for secondary surgical intervention. However, if the scleral and retinal laceration extends posterior to the ora serrata, if the peripheral retina cannot be visualized, and in case of retinal incarceration, a prophylactic scleral buckle should be considered.

**Conclusion**

The principles of wound repair hitherto dealt with should be treated not as strict rules, but as guidelines. Trauma surgery is one area which needs a lot of innovations and thinking on the feet and modifying our techniques as per the situation. No set of rules or guidelines, however comprehensive cannot cover the entire panorama of wounds that is going to be thrown up at a trauma surgeon. But to the inquisitive and adventurous surgical minds these journeys along untried paths can be exhilarating.

**References**

3. Ocular Trauma Principles and Practice-Ferenc Kuhn, Dante J Pieramici.
Injury to ocular adnexa is a commonly encountered by ophthalmologists in day to day practice. So it is very important to know about basic rules of eyelid repair so that both cosmetic appearance and physiological function could be preserved.

**Basic Considerations**

1. Careful history about mode and time of injury is very important. Mode of injury may tell you about possibility of deep penetration into the orbit or any intraocular foreign bodies. History of alcohol use, any medico legal issues are worth mentioning in history. Also ask about any previous systemic illness and timing of last oral feed to plan for general anaesthesia.
2. Assess general condition of patient to rule out any multi system trauma especially any head injury which may be a priority to attend.
3. Record visual acuity of each eye.
4. Thorough examination of eye to rule out any globe injury/occult perforation. Any RAPD should be specifically looked for.
5. Appropriate radiological investigations to rule out any intraocular foreign bodies, injury to optic nerve. CT scan is preferable if any magnetic intraocular foreign bodies are suspected. MRI is the preferred choice in suspected optic nerve injury.
6. Counseling is very important to build up rapport with patient and relatives. Extensive trauma may need multistage repair about which should be explained in detail to patient and bystanders to rule out future discontentment.
7. Take pre-operative photographs to document extent of injury which may be important to explain to patient and from medico legal point of view.

**Anaesthesia:** Majority of lid lacerations can be repaired under local anaesthesia. Facial block can be supplemented. However extensive injuries which need considerable time for reconstruction should preferably be under taken under general anaesthesia. Injection of anesthetic mixture may distort the local anatomy and prevent correct approximation of wound edges.

**Wound toileting** is very important. Irrigate the wound thoroughly of all debris. Foreign bodies should be picked up with forceps.

**Supportive Treatment:** Systemic antibiotics should be started. Intravenous antibiotics are preferable for severely contaminated wounds. Tetanus toxoid and or Tetoglob can be given to non-immunized patients. It is not required for children who have keen vaccinated earlier including booster dose. Systemic steroids can be given to reduce edema and inflammation if there is no contra indication.

**Classification and Management**

Good vascular supply of fascial tissues rarely necessitates extensive debridement as these tissues heal very well. Following are the most common type of eyelid trauma encountered in daily practice and their management.

A. **Eyelid margin repair**

Eyelid margin lacerations require precise suture placement and critical suture tension to avoid notching of eyelid margin which can disrupt tear pump function post-operatively (Figure 1).

![Figure 1 Lid notching following eyelid repair](image1)

It can be accomplished by 2 or 3 sutures for alignment through lash line, meibomian gland plane and gray line and keep ends long to be incorporated in subsequent skin sutures. (Figure 2)

![Figure 2. Eyelid margin repair. A The eyelid margin is aligned with 6.0 vicryl tarsal sutures and the lash line, gray line, mucocutaneous junction with 6.0 silk sutures. B Tarsal sutures tied and cut, eyelid margin sutures are tied and left long. C Skin is closed with interrupted 6.0 silk sutures and long ends of eyelid margin sutures are incorporated into skin sutures to avoid corneal abrasion](image2)
6.0 silk is an ideal suture for eyelid margin and skin closure. Closure should result in moderate eyelid margin eversion of well approximated wound edges. Tarsus is sutured with absorbable 6.0 vicryl sutures. Tarsal sutures should never extend full thickness through conjunctival surface especially in upper lid to avoid corneal epithelial disruption. One must protect globe with lid guard well lubricated with antibiotic ointment to prevent inadvertent needle point injury while repair.

B. Lacerations not involving the lid margin
Superficial eyelid lacerations involving just the skin and orbicularis muscle require only skin sutures. To avoid cosmetically unacceptable scar, consider basic principles of repair like conservative debridement of wound, use of small caliber sutures, eversion of wound edges and early suture removal. As a rule, conjunctiva and orbital septum are not sutured while any lid repair is done. Conjunctiva will be in good approximation if levator and tarsus are correctly re-approximated. Any orbital fat visible indicates violation of orbital septum and need levator exploration. (Figure 3a, 3b)

Orbital septum should never be reattached as this may result in restrictive ptosis or lagophthalmos. Septal attachments between orbital rim and levator should remain completely open as contraction of septum during healing phase is uncontrolled and may lead to restriction of levator muscle. Leaving septum open also allows easy egress of orbital haemorrhage and may prevent orbital injury due to post-operative swelling and bleeding.

C. Eyelid Avulsion and Canthal Injuries
It is usually the result of horizontal traction on eyelid which causes avulsion at lid’s weakest points medial or lateral canthal tendon. Avulsion at medial canthal area demands evaluation of lacrimal drainage system. Canaliculal injury should be specifically looked for and repaired as discussed later. Medial canthal tendon (MCT) avulsion will cause rounding of medial canthal tendon and acquired telecanthus.

Treatment of MCT avulsion depends on nature of avulsion. If upper or lower limb is avulsed but posterior attachment of the tendon is intact, avulsed limb may be sutured to its stump or to the periosteum overlying anterior lacrimal crest. Posterior portion of MCT attached to posterior lacrimal crest needs special attention as this portion mainly gives stability to medial canthus. Inability to realize this can lead to medial canthus deformity and improper apposition of eyelid margin to globe. (Figure 4)

If entire tendon including posterior part is avulsed, avulsed tendon has to be reattached to post lacrimal crest. If there is associated nasoorbital fracture, transnasal wiring or plating is needed after reduction of fracture. Non-absorbable 6.0 or 4.0 prolene sutures are the choice for canthal injuries. Absorbing sutures do not have the tensile strength for this type of trauma repair.

Lateral canthal tendon has to be reattached to lateral orbital rim using non-absorbable prolene sutures. Sharp contour of lateral canthal angle has to be kept in mind while reconstruction.

Avulsion injuries accompanied by tissue loss need reconstructive surgery at a later date. The initial goal is to stabilize eyelid and protect the eyeball. Tarsorrhaphy can also be considered till patient gets access to oculoplastic surgeon.

Figure 3a: Eyelid laceration with orbital fat prolapse

Figure 3b: Four months postoperative appearance

Figure 4: Improper apposition of lid margin to globe due to failure to fixate posterior portion of medial canthal tendon. Mini monoka stent can be seen in situ in lower canaliculus.
trained in reconstructive procedures.

D. Canalicular laceration
All canalicular lacerations need to be repaired whether upper or lower. One should have a high index of suspicion for any injury involving medial canthus. It is not uncommon to see such injuries even with a trivial trauma (blouse hook injury to child while breast feeding).

Surgical Technique
Repair has to be undertaken under operating microscope preferably under general anaesthesia. Identification and retrieval of proximal end of canaliculus is a real challenge. (Figure 5)

Gentle traction at edges of wound with cotton applicator stick under high magnification will help. If it is difficult to identify, gentle irrigation of fluid or air injection through uninjured canalicular system can help in identification. Diluted fluorescein can also be injected to visualize canaliculus. Dyes like methylene blue can cause staining of tissues so better to be avoided. Use of pigtail probe has a high incidence of damage to uninjured canalicular system especially in inexperienced hands. If proximal end cannot be retrieved, eyelid should be closed without further manipulation.

After identification of cut ends, canaliculus has to be stented using either mononcanalicular or bicanalicular (Crawford) stents. Minimonoka monocanalicular stents are now available with excellent post-operative results. It has self retaining cap which sits at punctum giving it excellent stability and avoids extrusion or displacement of stent. (Figure 6).

It also has advantage of not disturbing uninjured canaliculus (Figure 7).

Only disadvantage is the high cost of stent. If nothing is available, Angiocath I/V cannula 22 gauze can be used to stent canaliculus. (Figure 8a, 8b, 8c).

Figure 7 Mini monoka stent in situ upper canaliculus
Figure 8a Bicanalicular laceration in a young boy following trauma with a sharp object
Figure 8b Postoperative 5th Day, lower lid mini monoka stent in situ and in upper lid Angiocath 22 gauge IV cannula used to intubate canaliculus.
After stenting, cut ends of canaliculus have to be sutured. One should not pass sutures through lumen of canaliculus, only pericanalicular tissues need to be approximated using absorbable vicryl sutures. Ideally stent should remain in place for 2 months. Patient should be instructed not to rub eye or pull the tube in medial canthus.

Traumatic Ptosis
It may be seen both with lid lacerations and with contusion injuries. Traumatic Ptosis following contusion usually improves almost fully spontaneously. So it is advisable to observe till 6 months before undertaking any repair. Exception to this rule may be young children in whom little early intervention can be considered to prevent deprivation amblyopia. Laceration of levator muscle or its aponeurosis need exploration and repair during primary surgery itself.

Dog Bites
More common in children. Initial management involves copious irrigation and cleaning of wound. Immunoglobulins can be injected locally around wound. Rabies and tetanus prophylaxis should be observed. Systemic antibiotics are also recommended. Definite repair should be undertaken only after 24-48 hours to allow for local Immunoglobulins to act. Early intervention without any rabies prophylaxis can lead to spread of rabies virus causing more damage.

Summary
Repairing eyelid and canicular injuries need patience and skill on part of the surgeon. The foremost rule is to never hurry up a closure. Maximal possible reconstruction should be done during primary repair itself. Repairing these tissues is rarely emergency and often one can wait for careful intervention performed by some one intimately familiar with anatomy and reconstruction of these structures. Improper primary repair can have significant and lasting visual and cosmetic side effects.

References:
1. American Academy of ophthalmology: Orbit, Eyelids and lacrimal system, Section7, 2007-2008
2. Ocular trauma principles and practice. Ferenc Kuhn, Dante J Pieramici. Mosby publications
Traumatic optic neuropathy is a rare but devastating cause of partial or complete visual loss caused by deformational forces that injure the optic nerve. Injuries can be broadly classified as direct or indirect. Direct injuries usually involve direct anatomical disruption of the optic nerve caused by injuries to the head, face and orbit due to projectiles. Indirect injuries result from deforming forces applied to the bony orbit, or by motion of the globe, where the optic nerve absorbs energy concentrated at the orbital apex. These injuries are seen in the absence of open wounds but with a positive history of blunt frontal trauma. Incidence of traumatic optic neuropathy in facial trauma is 0.7 to 5 percent. Causes of traumatic optic neuropathy can be as varied as motor injuries, falls, falling debris, assaults trivial causes such as weightlifting as well as following endoscopic sinus surgery.

**Relevant Anatomy**

The optic nerve is the neural conduit linking inputs received from the retina to the brain (consisting of approximately 1.2 million axons) originating from the retinal ganglion cells. The nerve also contains oligodendrocytes which provide axonal myelination; microglia which are immunocompetent phagocytic cells and modulate apoptosis; and astrocytes. The nerve is enclosed within all three meningeal coats - a perineural sheath from the pia mater which also contains the blood vessels, an intermediate sheath from the arachnoid, and an outer sheath from the dura mater, which is also connected with the peristeum as it passes through the optic foramen. Optic nerve fenestration involves incision of the outer two coats surrounding the optic nerve.

The total length is about 50 mm in length and is anatomically divided into:

- **Intracocular** (about 1 mm)
- **Intraorbital** (20-30 mm)
- **Intracanalicular** (5-11 mm)
- **Intracranial** (3-16 mm)

The optic nerve travels superomedially and passes through the annulus of Zinn to the entrance of the optic canal at the optic ring, which is located medial to the superior orbital fissure. The canal lies within the lesser wing of the sphenoid bone and is approximately 9 mm in length. The intraorbital portion of the nerve extends 18 mm from the posterior aspect of the globe to the orbital apex. The nerve measures between 20 and 30 mm, and, therefore, takes a sinuous course, allowing for a range of movements by the eye. However, as the nerve enters the optic foramen its dural sheath becomes continuous with that lining the orbit and the optic foramen, rendering it immobile. This portion of the nerve is the focus of forces encountered in head trauma and is the most common site of optic nerve injury.

The intracranial portion of the optic nerve may suffer trauma when it is displaced superiorly against the sharp edge of the falciform dural fold. In addition, the anterior clinoid process lies lateral to the nerve, and, when fractured, it can crush the nerve.

**Presentation, Examination and Diagnosis**

Traumatic optic neuropathy is a clinical diagnosis and it usually follows head trauma with or without a history of loss of consciousness. Patients of traumatic optic neuropathy usually present with reduced visual acuity which may be as profound as only light perception or even no light perception. In patients with relatively better visual acuity on presentation, loss of colour vision is a common finding.

Check list for examining a patient with suspected Traumatic Optic Neuropathy:

- **Vision Check**
- **Colour vision** (if vision permits)
- **Pupillary evaluation**
- **Check for globe rupture, IOFB, fracture**
- **Fundus examination**
- **Neuroimaging** (in case of suspected fracture)
- **VEP**
- **ERG**

Patients who have suffered extensive head trauma must be neurologically examined and an ophthalmological examination should be performed only after stabilizing the patient. The eye and the adnexa must be examined looking specifically for globe rupture, foreign bodies and fractures. Pupillary reflexes should be evaluated. The presence of an afferent papillary defect (APD) indicates the possibility of unilateral traumatic optic neuropathy. Ocular motility and visual fields should be evaluated, as possible. Fundus examination typically reveals a disc of normal appearance unless an avulsion or an anterior optic neuropathy is present. Further more, a ring of hemorrhage at the site of injury is indicative of partial or complete avulsion of the optic nerve head. Optic atrophy ensues usually after a period of 2 weeks to 3 months. In comatose uninjured patients, the visual evoked potentials may be needed in diagnosing traumatic optic neuropathy, especially when pupillary evaluation is not helpful, as in bilateral traumatic optic neuropathy.

In unilateral traumatic optic neuropathy, flash VEP amplitudes that are at least 50% of the normal eye are critical for a good visual outcome. An absent VEP response indicates that visual loss is complete, and recovery of vision may be unlikely, although reports have shown that patients with persistently negative VEPs may also show visual improvement.
absent electroretinogram (ERG) is associated with a poor potential for visual recovery\(^6\). A CT scan of the brain with fine cuts (axial sections of 1 to 1.5 mm) through the orbits should be sought. Coronal images are necessary to evaluate the optic canal properly and rule out a fracture. In patients with traumatic optic neuropathy, orbital fractures especially canal fractures have been associated with poorer visual acuity and a poor prognosis\(^7\). MRI of the orbit may reveal focal edema of the optic nerve or optic nerve sheath enhancement with gadolinium.

**Pathophysiology of traumatic optic neuropathy**

It is believed that damage in traumatic optic neuropathy is caused by a primary and secondary mechanism of injury. While the optic nerve can be injured anywhere along its course, the most common site of injury is the intracanalicular part followed by the intracranial portion. Blunt trauma to the frontal bone result in forces being transmitted to the fixed intracanalicular segment of the optic nerve which can result in a fracture of the optic canal. As mentioned earlier, the tight adherence of the optic nerve’s dural sheath to the periosteum within the optic canal causes the optic nerve to be vulnerable to the impact of skull injuries. Haemorrhage either within the optic nerve sheath or in the orbital cavity can cause loss of optic nerve function. Mechanical forces are considered to be the primary mechanism of injury. These forces cause lacerations, partial or complete avulsion of the retrobulbar nerve, contusion necrosis, and disruption of the nerve’s vascular supply, resulting in hemorrhages, and thereby cause permanent damage.

Once the vascularity of the optic nerve is disturbed, the secondary mechanisms of injury come into play. Oedema sets in soon after and this in turn further compromise the vascular supply by causing a rise in intraluminal pressure. Secondary mechanisms that have been studied include numerous pathways for the generation of free radicals and arachidonic acids, lipid peroxidation, production of inflammatory mediators such as bradykinin, loss of calcium homeostasis with disruption of cellular function, glutamate-induced excitotoxicity, cell-mediated inflammation, and inititation of neuronal apoptosis\(^2\). Most treatment modalities revolve around limiting the secondary injury with the hope of rescuing those axons which have survived the initial trauma. Direct traumatic optic neuropathy is less common because the laxity of the intraorbital optic nerve allows for both absorption and deflection of the penetrating object. The resilience of the dura to penetration also offers further protection.

**Treatment of traumatic optic neuropathy**

The management of traumatic optic neuropathy remains controversial owing to a general lack of understanding of the pathophysiology involved and uncertainty about clinical results after therapeutic intervention.

Three major therapeutic options for traumatic optic neuropathy exist:

- Corticosteroids
- Surgical optic nerve decompression
- Combination of the two.

**Steroids in Traumatic Optic Neuropathy:**

The use of systemic corticosteroids in traumatic optic neuropathy is currently thought to be the best form of treatment as opposed to none at all. It is hypothesized that that pathologic free radical reactions are initiated following major central nervous system trauma and that very high doses of corticosteroid functioned as antioxidants to inhibit free radical damage\(^11\). Steroids as a form of therapy for traumatic optic neuropathy has been accepted after the results of National Acute Spinal Cord Injury Study (NASCIS-2) which showed positive results when systemic corticosteroids were used in patients of acute spinal cord trauma\(^12\). NASCIS 2 was a multicenter, randomized, double-blind, placebo-controlled study involving patients with acute spinal cord injury, patients. These patients were randomly assigned to receive placebo, naloxone, or methylprednisolone within 12 hours of spinal injury. Intravenous methylprednisolone was given as an initial dose of 30 mg/kg followed by a continuous infusion of 5.4 mg/kg/h\(^12\). When compared with placebo, treatment with methylprednisolone within 8 hours of injury resulted in a significant improvement in motor and sensory function.

However, whether the results of NASCIS can be extrapolated to justify the use of systemic corticosteroids in traumatic optic neuropathy needs to be further investigated. The International Optic Nerve Trauma Study, in which visual outcomes were compared with patients following observation alone, high dose steroids given within 7 days of the injury, and optic canal decompression with or without corticosteroids and performed within 7 days of the injury; showed no clear benefit for either corticosteroids or optic canal decompression in patients of traumatic optic neuropathy\(^13\).

Other recent studies have also shown that there is no difference in visual acuity improvement between intravenous high-dose corticosteroids and placebo in treatment of recent traumatic optic neuropathy\(^14\). Most recently, the Corticosteroid Randomization After Significant Head Injury (CRASH) trial, a large randomized, placebo-controlled study, evaluated the effect of early administration of 48 hours infusion of methylprednisolone on the risk of death and disability after head injury. The investigators found a small but statistically significant increase in the risk of death within 2 weeks after head injury in the group allocated to corticosteroids compared with placebo\(^15\). Furthermore, recently A Cochrane systematic review in 2007 critically analyzed the available evidence for the role of systemic steroids in traumatic optic neuropathy and showed that there were no convincing data of additional benefits of
steroids over observation alone16. The spontaneous improvement seen in many patients makes it difficult to assess the efficacy of any treatment method. Thus the use of corticosteroids in traumatic optic neuropathy continues to be a controversy.

**Surgical Management of Traumatic Optic Neuropathy**

Optic canal decompression surgery has a limited role in the management of traumatic optic neuropathy. This treatment is based on the hypothesis that swelling in the optic canal may lead to a compartment syndrome. The increasing edema would decrease tissue perfusion to cause more postinjury ischemia to the optic nerve. This decompression procedure is believed to decrease edematous pressure in the optic canal to reverse ischemia and axonal conduction block, which can result in irreversible axonal degeneration. A variety of approaches have been described, including transfrontal craniotomy, and transtethmoidal, transantral ethmoidal, sphenoethmoidal, and endoscopic decompression. The surgical approach should be based on the location of the pathology as visualized by CT scanning. The goal of optic nerve decompression is to provide surgical relief of pressure on the intracanalicular segment of the optic nerve2.

Nerve sheath hemorrhage occurs rarely and the images must be looked at together by an experienced neuroradiologist and clinician to avoid calling peri-sheath blood an intrasheath hemorrhage. If intrasheath blood is convincingly and clinician to avoid calling peri-sheath blood an intrasheath hemorrhage. If intrasheath blood is convincingly demonstrated, optic nerve sheath fenestration is indicated17.

Although significant advances have been made in the understanding of neuronal damage and repair, there is still no consensus among practitioners regarding the protocol for managing traumatic optic neuropathy. The diagnosis of traumatic optic neuropathy remains a clinical one. Clinicians must use their discretion to decide with the patient or family whether the implementation of medical or surgical intervention outweighs the risks2.

**Recent advances and current research:**

It has been shown that the innate adaptive T-cell mediated immune response directed against self-antigens located at the site of damage can be neuroprotective after optic nerve or injury. By augmenting this response in individuals who spontaneously manifest it and by inducing this autoimmune response in those incapable of manifesting it, optimal neurological functional recovery was attained18. Research on neurotrophic and neurotropic agents such as monosialogangliosides and neurotrophic growth factors has had promising results19. Gene transfer of anti-inflammatory cytokines may help prevent neurodegeneration20. Also, a new family of corticosteroids known as lazarooids or 21-aminosteroids provides the free radical–inhibiting properties of corticosteroids without their glucocorticoid activity21.

**References:**


Ocular trauma is the most common cause of monocular blindness in children. The visual loss that occurs in ocular trauma in childhood is not only due to damage caused by the trauma itself but also due to resultant amblyopia after surgery. In children, trauma is the second most common indication for ocular surgery after strabismus. Both open and closed globe injuries along with orbital and adnexal trauma have been described.

Most paediatric ocular trauma reported from the west was sustained either at home, in motor vehicle accidents or due to fireworks. In the 11-15 year age group, boys with ocular trauma outnumbered girls. Orbital fractures were commonly seen with midfacial injuries. Even facial lacerations that seemed trivial on initial examination were associated with severe ocular trauma, hence the importance of careful evaluation of the globe in midfacial injuries.

In a large series reported from India, 80% of children sustained injuries in the rural setup. Outdoor sports including homemade bow and arrow caused ocular injuries in about 50%, household items like kitchen knives, door handles, pencil tip, and wire in 15%, fireworks in 10%. Closed globe injuries have even been reported with carbonated beverages and detergents. In injuries associated with fireworks, the eyeball is the most common body part involved, both in direct injury and in injuries to bystanders.

Serious (Gr IV) chemical burns and loss of vision in the eye have been caused by wall plastering material and the concentrated Ca(OH)2/CaCO3 used to spice up betel leaf eaten at home, falling into the eye. Babies being breast fed have sometimes shown lid lacerations due to the hook of the mothers clothing.

**Evaluation Of The Child With Ocular Trauma**

Most children with eye or adnexal trauma will present to the paediatrician or emergency medicine department. The first contact physician should

1. Elicit a history quickly, determine the extent of injury
2. If open globe injury is suspected a protective shield or eye pad should be applied and the child referred to a hospital with trauma unit.
3. A booster dose of tetanus toxoid and instruction to the parent to keep the child nil orally till a proper ophthalmological evaluation is done.
4. In cases of a chemical substance like wall plaster, toilet cleaner, UPS battery acid falling into the eye, thorough wash of the injured eye with normal saline should be done and the child referred to a higher centre. This timely treatment reduces the damage to the eye drastically.

Evaluation of the ocular trauma in a hospital setup-

1. Elicit a proper history. An older child may/may not offer a proper history in order to cover up for a forbidden activity (e.g. fireworks etc) and the parent may not always know the correct cause for the injury in a younger child. Reluctance to give history or stating a trivial cause for a major injury should lead one to suspect child abuse, lack of supervision or neglect on the part of parent or caregiver.
2. The exact agent of injury (stick, wire, ball) if known is useful to assess the type of injury expected. The approximate timing of sustaining injury alerts the ophthalmologist to the possibility of endophthalmitis, specially in injuries with vegetable foreign bodies like wood etc. Again rural setting injuries have a higher incidence of infection. The identity of chemical agent is very important in chemical injuries as immediate treatment is required to contain the extent of damage.
3. History of previous ocular surgeries, previous ocular injuries, the visual acuity of both eyes prior to injury and the history of systemic conditions like bleeding disorders is important from the point of view of surgery and the visual rehabilitation of the child.

**Examination Of The Child With Ocular Trauma**

After ensuring that the child’s vital signs are normal, attempt must be made to

1. Elicit all the external injuries including facial injuries and lid lacerations causing proptosis/endophthalmitis or ecchymosis. An older child may allow the examiner to record vision (unaided and pinhole) with a vision chart or counting fingers. Gentle retraction of the lids must be done even if lid edema is present, to look for open globe injuries and pupillary reactions for detecting traumatic optic neuropathy.
2. If the child is small and resists any attempt at examination, it is preferable to examine the extent of injury under general anaesthesia and proceed with the repair in the same sitting. Till then placing a sterile pad and bandage over the injured eye is advisable. Use of unsupervised sedation by the ophthalmologist to determine the extent of injury is not advisable as some sedatives specially midazolam is known to cause respiratory depression and in the absence of emergency resuscitation kit, a child with a minor eye injury may land up in the ICU with a severe hypoxic brain damage.
3. If the first examination reveals or suggests a serious open globe injury needing surgical intervention, then the child is admitted and kept nil orally. Intravenous antibiotics are started and the child is posted for primary surgical exploration.
or repair under general anaesthesia at the earliest available opportunity. Appropriate radiographic investigations, ideally a CT scan of brain and orbit must be ordered.

4. Animal bites should be cleaned thoroughly, antirabies immunisation administered and systemic antibiotic coverage given. Amoxycillin clavulanate is appropriate coverage, but in penicillin allergic patients azithromycin with clindamycin or septran offers a equivalent coverage.

**Eyelid And Adnexal Trauma**

Eyelid injury can be isolated or in conjunction with other ocular trauma. Upper eyelid injuries extending deep into the forehead should be explored and if any underlying fracture is suspected, the help of a neurosurgeon should be sought. The repair of eyelid injuries alone can be delayed up to 36-48 hours as this allows the oedema to subside and allows better identification of anatomical landmarks. Medial canthal tears can involve the canaliculus and this should be identified and repaired, if necessary around a silicon tube. In children it is preferable to use absorbable sutures, plain catgut or vicryl to avoid another exposure to anaesthesia for removal of the sutures. If used, superficial non absorbable sutures are removed on 5th or 6th day (earlier than in an adult) to prevent scarring.

**Orbital Trauma**

Orbital fractures are common in children suffering head and face trauma. The type of fracture, mechanism of injury and timing of possible treatment differ from that of adults. The most common orbital fracture in childhood is of the orbital roof, which is rarely fractured in adults. Indications for surgical repair include severe restriction of ocular motility, diplopia, persistent pain, nausea and vomiting. These are less likely to resolve in a child than in an adult.

Timing of surgical repair- Most studies recommend surgical intervention within 5-7 days of the injury in children versus 2 weeks or longer in adults. A study by Matteini et al recommends a classification system of orbital fracture in children based on the clinical, radiographic and prognostic criteria (table-I). They suggest that the surgical timing of these fractures relates to the anatomical location, CSF leak or penetrating wounds, patients age, muscle entrapment or signs of ischemia or compression of optic nerve. While type-V fracture needs surgical treatment within a few hours, type-IV should be treated within 24 hours, type III within a few days.

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>Fracture of orbital rim</td>
</tr>
<tr>
<td>Type II</td>
<td>Fracture of orbital wall with no functional impairment</td>
</tr>
<tr>
<td>Type IIIa</td>
<td>Fracture of orbital wall with diplopia, adults</td>
</tr>
<tr>
<td>Type IIIb</td>
<td>Fracture of orbital wall with diplopia, children</td>
</tr>
<tr>
<td>Type IV</td>
<td>Fracture with open wound, penetrating object, CSF leakage.</td>
</tr>
<tr>
<td>Type V</td>
<td>Fracture of orbital apex, fracture causing compression of globe, ischemia of optic nerve.</td>
</tr>
</tbody>
</table>

Adapted from Matteini et al. [14]

**Ocular Trauma-Anterior Segment**

Anterior segment injuries in children can range from minor abrasion to open globe injuries with extrusion of intraocular contents and severe chemical burns with 3600 limbal ischemia. An area of subconjunctival hemorrhage, chemosis or a small break in the eyelid skin may be the only surface manifestation of scleral perforation by a sharp pointed object such as a knife, scissor or blade. A peaked pupil may be the only evident sign of a small corneal or limbal perforation and if a retained intraocular foreign body is suspected, a CT scan of the orbit should be done.

Corneoscleral wounds are repaired on the same principle as adults. These wounds heal relatively early in children, hence sutures should correspondingly be removed early in children. Fibrin clots form quickly in the anterior chamber and these can often simulate the appearance of fluffy cataractous lens cortex. Lens removal therefore, should be avoided at the time of primary repair to avoid rendering the eye unnecessarily aphakic, thus risking amblyopia. Even if lens cortex is exposed, postponing cataract surgery for a week or two until severe post traumatic inflammation subsides may result in a smoother post operative recovery with reduced risks of complications.

The management of traumatic hyphema in children can be difficult, but most studies recommend adequate inpatient management to prevent rise of IOP and to prevent secondary hemorrhage and corneal blood staining. Patching however is avoided to prevent amblyopia. Medical management with corticosteroids and cyclopregics with IOP lowering drugs is recommended. Aspirin containing drugs are to be avoided. Systemic use of EACA has shown to reduce the rate of rebleeding in traumatic hyphema in children. This has been confirmed by a study done by Pieramice et al. Early(3-4 days) surgical evacuation of a total hyphema has been advocated in children because of difficulty in measuring IOP and detecting early corneal staining. Children with sickle cell anemia tend to have complications...
of hyphaema like blood staining at lower IOP. Other causes of spontaneous hyphaema like intraocular tumours, juvenile xanthogranuloma and bleeding disorders should be ruled out by doing blood counts, bleeding parameters and a CT scan.

**Ocular Trauma - Posterior Segment**

Posterior segment injuries including retinal detachment, vitreous haemorrhage, endophthalmitis, optic nerve avulsion have a devastating effect on the vision of the injured eye in patients of all age groups but the possibility of amblyopia in children below 8-9 years increases the visual morbidity in them. Retinal detachment in children with ocular trauma is caused more by compression/decompression of globe with lower sclera rigidity than the penetrating or perforating injury itself. Post traumatic endophthalmitis is a poor prognostic indicator for final visual outcome in children with ocular trauma. Delay in treatment due to an increase in lag period in children with ocular trauma from rural areas in our country has increased the risk of endophthalmitis causing poor visual recovery. Prophylactic antibiotics has been shown to decrease the rate of endophthalmitis after sustaining ocular trauma in children with risk factors.

Cranial nerve palsies including 3rd, 4th and 6th nerve are often found in conjunction with ocular and head injuries in children.

Traumatic optic neuropathy may be the only ophthalmic injury sustained after a closed head injury. The absence of history of visual loss in children and the difficulty in recording visual acuity at presentation and eliciting a Relative Afferent Pupillary Defect in swinging flash light test in an uncooperative child often makes diagnosis of traumatic optic neuropathy difficult in children. Forces transmitted through the thin skull bones of children cause injury to the canaliculal (fixed) part of the optic nerve in children. The visual outcome with any form of treatment of traumatic optic neuropathy -high dose of corticosteroids, optic nerve decompression is the same as that for adults.

**Prevention And Conclusion**

As children are very curious by nature and often exhibit naïve behaviour, even activities which would otherwise be considered harmless can cause ocular trauma in children. Children tend to play unsupervised with dangerous toys like guns and with fireworks without taking adequate protective measures. The prognosis in traumatized eyes in children being guarded, prevention is critical in reducing the ocular morbidity in children. Successful preventive measures depend on a vigilant awareness of activities and situations in which the children could get injured. Most ocular injuries of children occurring in predictable situations are preventable. The National society to prevent blindness estimated that the frequency and severity of at least 90% of all eye injuries could be prevented. Inadequate adult supervision is an important contributing factor in most cases of childhood eye injuries. Ophthalmologists should educate parents and children about the kinds of hazards that can result in needless eye injuries and encourage them to take preventive measures. All one eyed individuals should mandatorily wear protective eyewear. Since a high percentage of eye injuries occur at home and are caused by sharp objects, special safety standards should be applied with regard to dangerous furniture or toys. Lectures in schools and special programmes on the subject of eye injury via television and other media may assist in achieving this goal. Such educative attempts will definitely bring down the incidence of eye injury in children.

**References:**

Introduction:
Motor vehicle accidents, fall from height, assault, industrial accidents especially blast injuries are the most common causes of poly-trauma associated with ocular and orbital injuries. Many of these patients also present with head injuries (closed or open), therefore in addition to the ocular and adnexal injuries, neuroophthalmic involvement due to head injury often cause loss of vision. Injury to the anterior and posterior visual pathways, the pupillomotor pathways, the cranial nerves and the supranuclear and internuclear gaze pathways can all occur in a patient of polytrauma. [1]

Demographics
Most polytrauma patients are males with 4:1 preponderance over females. The most susceptible age group a young adult between 20 to 40 years. The most common causes of polytrauma in Indian population are motor vehicle accidents (especially two wheeler riders without helmets), industrial accidents, followed by assault. The incidence of sports associated injuries is less in Indian population than that quoted in western literature. [2]

Evaluation of Patients
Polytrauma patients with injury to the visual system often have concomitant neurologic, orthopaedic and internal organ injuries some of which may be life threatening. As with all trauma patients, initial assessment and management must emphasize airway management, control of haemorrhage, hemodynamic stabilization and neurologic assessment. Airway management may require anterior positioning of mandible or placement of nasopharyngeal, oropharyngeal device. Brisk bleeding may accompany injury to the craniofacial region due to the rich vascular supply. Intracranial injuries often accompany craniofacial trauma, hence patient’s level of consciousness should be assessed early to establish a baseline. Once airway management, hemodynamic stabilization is achieved and level of consciousness is determined, a detailed history and physical examination are conducted.

A multidisciplinary approach with cooperation of neurosurgery, general and trauma surgery, otolaryngology and facial trauma surgeons is imperative for optimizing prompt evaluation and treatment of patients. The appropriate time for ophthalmologic consultation should be as early as possible once the patient’s vital signs stabilize. The presence of ocular injuries may influence the timing of repair of craniofacial injuries, and also because early treatment of trauma related visual loss within 6-8 hours of injury has been shown to be beneficial to the patient.

Obtaining history and conducting thorough ophthalmologic examination of the polytraumatised patient with altered sensorium often presents a challenge to the examiner. Visual acuity and colour vision testing may be difficult if the patient has decreased level of consciousness or is heavily sedated. However, the assessment of gross visual acuity even light perception may be prognostic in the light of neuroophthalmic findings. If a open globe injury is evident or suspicion is high, immediate surgical repair of the globe under general anaesthesia is indicated and should take precedence over other nonlife saving interventions. In the absence of open globe injuries, adnexal injuries like lid and forehead lacerations, fracture of bones of the forehead and orbital rim fractures should be carefully looked for. Assessment of pupillary reactions both to direct and consensual to rule out afferent pupillary defect suggesting a traumatic optic neuropathy is urgent and very important especially in a patient in whom vision cannot be recorded due to altered sensorium. Presence of traumatic mydriasis, miosis, changes in size and shape of pupils due to coexisting cerebral edema, injury to the midbrain, pons may confuse the examiner but every attempt must be made to rule out TON. Failure of the dilated pupil to constrict with 1% pilocarpine suggests a structural injury to the iris. Traumatic miosis that increases in dim light and is associated with narrowing of palpebral fissure suggests a Horner’s syndrome. The above findings with neck pain should alert the examiner to rule out carotid – cavernous fistula caused by trauma. Ocular movements should be assessed whenever possible. Presence of ptosis, vertical and horizontal diplopia, movement restriction and gaze palsy suggests a cranial nerve palsy and can help locate the site of injury. Unilateral exophthalmos with orbital bruising, raised intra ocular pressure and extra ocular muscle palsy are findings characteristic of traumatic carotid – cavernous fistula. Oculomotor nerve palsy occurring after trauma is usually associated with decreased level of consciousness and severe head injury. Absent corneal sensation suggests involvement of trigeminal nerve while lagophthalmos indicates facial nerve injury.

Fundus examination must be done in a patient with visual dysfunction following trauma. However if the patient has altered level of consciousness the ophthalmologist must...
consult the attending house surgeon before dilating the pupil for fundus examination. The time and type of drug used to dilate the pupils must be documented. It is advisable to use short acting mydriatics in these patients.

Traumatic optic neuropathy is the most important cause for visual loss (often irreversible) in poly trauma. \[^{[1]}\]

**Traumatic optic Neuropathy**

Traumatic optic neuropathy (TON) is loss of visual function which is associated with trauma. The vision loss can manifest as sub normal visual acuity, visual field loss or colour vision dysfunction accompanied by ipsilateral Relative afferent pupillary defect (RAPD). TON is uncommon but important sequel of closed head injury. It occurs in 1.6% of head trauma cases and in 2.5% of patients with mid facial fractures.\[^{[2,3]}\]

**TON is classically separated into two types.**

1. Direct optic nerve injuries: result from orbital or cerebral trauma that transgress the normal tissue planes and disrupt the anatomic and functional integrity of optic nerve like optic nerve avulsion and transection of optic nerve.
2. Indirect optic nerve injuries: is caused by forces transmitted at a distance from the optic nerve. The anatomy and function of optic nerve is compromised by the energy absorbed by the nerve at the moment of impact.
3. A subset of injuries can cause TON due to diffuse orbital haemorrhage, retro bulbar haemorrhage/hematoma, optic nerve sheath hematoma or orbital emphysema.

Trauma induced damage to the optic nerve can occur anywhere along its course.

1. Optic disc trauma
2. Anterior optic neuropathy: within 10mm posterior to the globe has ophthalmoscopic features of CRAO, CRVO or AION.
3. Posterior optic neuropathy: from 10mm posterior to the globe, the intracanalicular part and intra cranial part. The fundus picture is normal for 3 – 6 weeks following which a temporal pallor of disc is seen.

The major focus of this discussion will be on indirect posterior optic nerve injuries. The intracanalicular part is the commonest site accounting for 81% followed by the intra cranial part (54%).

**Mechanism of Injuries:**

The most common form occurs during or shortly after a blunt trauma to the superior orbital rim, frontal area or cranium. The compression forces from the trauma are transmitted via the orbital bones to the orbital apex or the optic canal. The firm attachment of the dural sheath of the optic nerve in the optic canal makes it particularly susceptible to acceleration – deceleration injuries. In addition the vascular supply to this portion is subject to disruption from shear injury or from compression when the nerve swells up within the confines of the optic canal.

**Type of Injury:**

The injury to the optic nerve is a combination of both mechanical and ischemic damage. Injury may be – primary injuries due to haemorrhage into the nerve or its sheath, lacerations and contusions of the axons. Secondary injuries occur due to vascular obstruction or an ischemic damage initiated by shear injury with actual vascular disruption.

When there has been a mid facial or cranial trauma a high index of suspicion of optic nerve dysfunction must be kept in mind. Visual loss is typically immediate and severe with 24 – 86% of patients having no perception of light in a reported case series. There may be associated loss of consciousness (40 – 72% of cases).\[^{[2,3]}\]

In some cases, there may be prominent orbital edema and haemorrhage where as in there may be little external evidence of injury.

**Clinical Evaluation:**

Finding of decreased visual acuity with RAPD in the absence of intra ocular pathology, suggests a posterior optic nerve injury. Determining the time of visual loss relative to the injury is a strong prognostic indication (Immediate visual loss – poor prognosis, than a gradual visual loss). Red desaturation test is a sensitive test for optic nerve dysfunction.

The presence of RAPD must be quantified using neutral density filters (0.3 – 3 log units). This helps in evaluating the severity of visual loss and also in monitoring the recovery.

**Investigations:**

Visual fields: There are no pathognomonic visual field defects. It may be altitudinal, central scotomas or hemianopias (intra cranial)

VEP is especially useful in unresponsive patients and in cases of unilateral optic nerve injury, but may not be possible for logistic reasons

In unilateral TON, a flash VEP amplitude ratio (affected/normal side) greater than 0.5 appears to be predictive of favorable long term visual outcome.

Imaging of the orbits and brain must be done for detecting fractures of optic canal, bony fragment impinging on optic nerve, orbital and intra sheath hematomas. The fracture of sphenoid bone shows the severity of the trauma. \[^{[4]}\]

The treatment of TON is controversial due to lack of prospective, randomized, controlled studies. But it has been proven that visual improvement following treatment was significantly better than the recovery with no treatment (86% compared to 20 – 40%).

The corner stone of treatment is early use of high dose I.V. Methyl Prednisone. The rationale for this is based on lab
studies and on the National Acute Spinal Card injury study II, which showed that early use of steroids within 8 hours can reduce edema and tissue damage.[5]

Several mechanisms have been proposed to explain the neuroprotective effect of intravenous methyl prednisolone.

- Inhibition of oxygen free radical induced lipid peroxidation. This is dose dependent.
- Other effects which are less well understood include – support of energy metabolism, prevention of post traumatic ischemia, reversal of intra cellular calcium accumulation etc.

There are some factors which may indicate poor visual outcome as shown by some studies. These include, presence of blood in the posterior ethmoidal air cells, patients older than 40 years, loss of consciousness, failure of recovery after 48 hours, Flash VEP amplitude ratio less than 0.5 and APD more than 2.1 log units when measured with neutral density filters.

These predictive factors of visual outcome important for counselling patients regarding treatment and also for decision making regarding surgical decompression.

A meta-analysis of TON.[2] showed that recovery is related to the severity of initial injury and devised a grading system based on Visual acuity, the locations and type of fracture

Grade I – VA > 20/200 and without a posterior orbital fracture

Grade II – VA – 20/200 – light perception

Grade III – No light Perception or with a non displaced posterior orbital fracture

Grade IV – No light Perception and a displaced posterior orbital fracture.

This grading is useful for comparing studies and treatment protocols.

**Treatment Protocol:**[5,6]

Establish the diagnosis of TON based on reduction of visual acuity and presence of RAPD (quantitative RAPD using neutral density filters).

Rule out contraindications to high steroids such as pre existing infectious diseases, peptic ulcer disease, uncontrolled diabetes mellitus, pneumocephalus etc.

Institute high dose I.V. Methyl prednisolone (Optimal time to start is within 8 hours of injury)

Loading dose of 30mg/Kg body weight infused over 30 minutes.

Maintenance dose of 5.4mg/Kg / hour for 48 hours.

Monitor therapeutic response with serial visual acuity and RAPD measurements.

After 48 hours of I.V. Methyl Prednisolone.

If visual acuity and RAPD improves, change to oral prednisolone and taper rapidly over 15 days.

If visual acuity and RAPD worsens during oral steroid therapy, re instituted high dose I.V. M.P and consider surgical decompression.

Immediate surgical decompression of optic canal is considered in the following situations:

- Bony fragments impinging on the optic nerve
- Orbital and intra sheath hematomas

**Surgical Decompression:**[7, 8]

Procedure of choice is Transethmoidal – sphenoidal optic canal decompression. No correlation was found between outcome and timing of surgery, but preferably within one week. Surgery is not done in unconscious patients.

Decompression of optic canal consists of

- Removal of at least 50% of the circumference of osseous canal
- Removal of bone along the entire length
- Longitudinal incision of the dural sheath, including the annulus of Zinn.

As there are no prospective, randomized, controlled studies, the IONT study was proposed, (which initially started as a prospective study but later got converted into an observation study). The study concluded that there was insufficient evidence that either mega dose steroids or surgical decompression could be considered the standard of care[9].

**Newer drugs in the experimental stage:**[9]

The role of I.V. Mannitol should be considered, as brain edema causes loss of auto regulation of blood flow resulting in poor perfusion pressure to the optic nerve.

The goal in treatment TON must be early recognition and appropriate intervention.

Other possible neuro-ophthalmic findings in patients with poly trauma (specially head injury)[1]

Bilateral pinpoint pupils – narcotic intoxication, pontine haemorrhage, parasympathomimetic drug ingestion

**Cranial nerve palsies**

- 3rd nerve injury – Ptosis with anisocoria
- 3rd nerve with contralateral tremor – red nucleus injury
- 3rd nerve with contralateral hemiparesis – fasciculus injury
- 3rd nerve with contralateral ataxia – cerebellar injury
- 4th nerve injury – vertical diplopia with tilting of images
- 6th nerve injury – horizontal diplopia
- Multiple cranial nerve injury – cavernous sinus injury, Caroticocavernous fistula ,Cranial base fracture , Brain stem injury
- Horizontal gaze palsy – frontal lobe, pontine injury
References:
I. Introduction
Hyphaema (blood in the anterior chamber) can occur after blunt or lacerating trauma, or after intraocular surgery. Hyphaema can occur spontaneously in conditions such as rubeosis iridis (e.g. Associated with diabetic retinopathy, central retinal vein occlusion, carotid occlusive disease or chronic retinal detachment), vascular tufts at the pupillary margin, juvenile xanthogranuloma, iris melanoma, myotonic dystrophy, keratouveitis (e.g. Herpes zoster), leukemia, hemophilia, thrombocytopenia or Von Willebrand disease. Hyphaema also occurs in association with the use of substances that alter platelet or thrombin function (e.g. Ethanol, aspirin, warfarin).

II. Mechanisms of Haemorrhage and Blood Resorption
Blunt injury is associated with anterior-posterior compression of the globe and simultaneous equatorial globe expansion. Equatorial expansion induces stress on anterior chamber angle structures, which may lead to rupture of iris stromal and/or ciliary body vessels with subsequent haemorrhage. Secondary haemorrhage also termed re-bleeding, may be due to clot lysis and retraction from traumatized vessels. Lacerating injury can also be associated with direct damage to blood vessels and hypotony both of which can precipitate hyphaema. Hyphaema after intraocular surgery can be due to granulation tissue in the wound margin or due to damaged uveal vessels (e.g. from surgical trauma or IOL induced uveal trauma).

In conditions such as rubeosis iridis, juvenile xanthogranuloma, iris melanoma, iris leiomyosarcoma, myotonic dystrophy, and iris vascular tufts, iris vessel fragility itself may predispose to hyphaema. Minor trauma can precipitate hyphaema in this sitting. Duke-Elder proposed that hyphaema absorption might occur through the anterior part of the iris. Several groups have shown that erythrocytes leave the anterior chamber via the trabecular meshwork as relatively intact, undamaged cells. Uncomplicated hyphaema usually clear within approximately one week.

III. Epidemiology
The mean annual incidence of hyphaema is approximately 17 per 100,000 population. The peak incidence is between ages 10-20 years. The average age of patient is less than 25 years. The majority (80%) of hyphaema patients are males probably because most cases develop after trauma.

The causes for traumatic hyphaema are slightly different in developed and developing countries. In one study, sporting injuries accounted for 39.2% of all cases, criminal assault 20.7%, accidents in home 17.8% and accidents at work 9.9%. In another study in the 0-10 year age group 75% of eye injuries occurred at home. Injuries occurring at school were next, occurring in 20.1% of all cases. Work related injuries accounted for the 6.5% of all cases, and they occurred mostly in those aged over 20 years. Injuries that occurred on road side or street (7.6%) resulted from assaults and road traffic accidents and occurred mostly in the 21-30 years of age group. Sports related injuries that caused hyphaema were infrequent, and occurred in 1.9% in all cases.

IV. Clinical Examination and grading of hyphaema
At the baseline examination, a detailed history should be taken about the specific circumstances under which trauma took place and also a general medical history about other diseases (anemia, blood disorders, medications used, and liver or kidney disease).

The eye examination included a detailed examination at the slit lamp, intraocular pressure (IOP) measurement, fundus examination when it was possible (clear ocular media) with a +78 Diopter lens or with the indirect ophthalmoscope. There is no use of the three-mirror Goldmann or any other type goniolens during the initial examination in order to avoid any pressure on the globe and a secondary haemorrhage. Gonioscopy is performed after absorption of hyphaema to check for the presence of a possible angle recession. In certain cases where the patient underwent CT scan, MRI of the orbit or a B-ultrasonograph special care should be taken not to exert pressure on the globe.

Total hyphaema with iridodialysis
Because Read and Goldberg found that corneal blood staining was more likely to occur in patients with a total hyphaema associated with intraocular pressure ≥25 mmHg and ≥6 days duration, these investigators recommend that one manage such eyes surgically by day 6 if the hyphaema does not resolve below 50%.

The earliest sign of corneal blood staining is a straw yellow discoloration of the deep stroma, which should be distinguished from the light reflected off the surface of the blood clot in the anterior chamber. One clue to the presence of blood staining versus reflected light is the presence of greater stromal discoloration centrally than peripherally. Early signs of corneal blood staining include the presence of tiny yellowish granules in the posterior third of the corneal stroma or blurring of the fibrillary appearance of the corneal stroma. Crouch and Crouch believed that these easy biomicroscopic signs precede gross blood staining by 24-36 hours, and they suggest that clot evacuation at this stage can prevent gross staining with corneal clearing in 4-6 months. As indicated above, even if the intraocular pressure is normal, it is important to perform a daily slit lamp examination to detect corneal blood staining. The opacity usually clears from the periphery towards the center, and the process can require 2 or 3 years. The blood product protoporphyrin has been identified by Gottsch and coworkers as a phototoxic compound in the anterior chamber of patients with hyphaema and has been demonstrated to photosensitize the endothelium experimentally. Endothelial cell decompensation or degeneration is the earliest event in the pathogenesis of corneal blood staining. Mechanical disruption of the endothelium may play a role in the pathogenesis of endothelial decompensation, but photosensitization of the endothelium by hemoglobin-derived porphyrins in the presence of ambient light may also disrupt endothelial function. For this reason, patching the eyes with longstanding hyphaema may reduce the chance of corneal blood staining.

Pathologically endothelial degeneration and eosinophilic deposits distributed throughout the stroma characterize corneal blood staining. Ultrastructural studies reveal that hemoglobin tends to be extra cellular between collagen fibrils and hemosiderin tends to be in the keratocyte cytoplasm. Messmer et al posited the following mechanism for corneal blood staining. First, hemoglobin is released from the erythrocytes in the anterior chamber, diffuses across Descemet's membrane, and aggregate focally within the membrane as well as within the stromal lamellar. Second, the keratocyte phagocytose and metabolize hemoglobin, producing intracellular hemosiderin. Excess intracellular hemosiderin/hemoglobin induces keratocyte necrosis, with attendant decreased cellularity of the posterior stroma. Third, released hemosiderin is phagocytosed by keratocyte in the anterior stroma. Most clearing occurred from periphery towards the center, and the demarcation between cleared and stained corneal stroma was abrupt both clinically
and histopathologically. Corneal blood staining clears by diffusion.

B. Increased intraocular pressure
Approximately one third of all hyphaema patients exhibit increased intraocular pressure. In the setting of traumatic hyphaema intraocular pressure may be elevated due to the following:
1. Occlusion of the trabecular meshwork by the clot, inflammatory cells, or erythrocyte debris or
2. Pupillary block secondary to a collar button shaped clot involving both the anterior and posterior chambers.

In general larger the hyphaema volume, the greater the likelihood of increased intraocular pressure secondary haemorrhage is often associated with increased intraocular pressure. In the setting of a total hyphaema, a normal or low intraocular pressure should alert one to the possibility of a ruptured globe. An initial period of elevated intraocular pressure can be followed, however, by a period of normal or low intraocular pressure even in the absence of a ruptured globe, provided that a secondary haemorrhage does not occur. This period of temporarily reduced pressure may be due to decreased aqueous humor production and may play a role in predisposing patients to secondary hyphaema, particularly as the normal process of clot lysis proceeds.

The incidence of late-onset glaucoma in eyes with a history of traumatic hyphaema ranges from 0-20%. Glaucoma developing days to years after the initiating injury can arise from damage to the trabecular meshwork (often associated with angle recession), descemetization and fibrosis of the trabecular meshwork, sclerosis of the trabecular endothelium or peripheral anterior synechiae formation leading to secondary angle closure glaucoma. The incidence of angle recession after eye trauma ranges from 20-94%. The possibility of developing glaucoma in an eye with angle recession appears to be related to the extent of angle recession. The greater the circumferential extent of angle recession, the greater the chance of subsequently developing glaucoma, particularly if more than 180ºof the anterior chamber angle is involved. If extensive, posterior synechiae which can form as a result of inflammation also can cause secondary angle closure glaucoma. Ghost-cell glaucoma, causes by dehaemoglobinized erythrocyte diffusion from the vitreous cavity in to the anterior chamber weeks to months after a vitreous haemorrhage, can be associated with a khaki coloured hyphaema and is another cause of late onset intraocular pressure elevation after trauma.

Elevated intraocular pressure is routinely managed medically with topical beta adrenergic antagonists or alpha-2 adrenergic agonists. If these medications are inadequate, topical or systemic carbonic anhydrase inhibitors are added. If these measures are ineffective isosorbide, oral glycerin or intravenous mannitol is administered. Pilocarpine is not recommended for these reasons. First, Pilocarpine may increase vascular permeability and promote fibrin deposition in an already inflamed eye. Second, the possibility of iridolenticular adhesions and seclusio pupillae may be greater with a miotic pupil. Third, fundus examination is impaired. Prostaglandins usually are not employed in this setting because of a presumed increase in the inflammatory response.

C. Peripheral Anterior Synechiae Persistence of the hyphaema for more than one week can result in the formation of peripheral anterior synechiae (PAS). The incidence of PAS increased with size and duration of visible hyphaema greater than 8 days. Posterior synechiae also can form presumably synechiae formation is the result of inflammation or clot organization.

D. Optic Atrophy
In the setting of traumatic hyphaema, optic atrophy tends to occur as a result of elevated intraocular pressure or due to optic nerve contusion. In a prospective study, Read and Goldberg found that 6% eyes had optic atrophy characterized by pallor without glaucomatous cupping. In 4% eyes transient IOP elevation was noted, and optic atrophy without cupping was attributed to this pressure elevation. In 2% eyes, no period of elevated intraocular pressure was detected. The latter cases may represent traumatic optic neuropathy secondary to short posterior ciliary artery damage caused by optic nerve contusion. The rest of optic atrophy related to elevated intraocular pressure appears to be greater if the pressure is allowed to remain at 50 mmHg or more for 5 days or 35 mmHg or more for 7 days, in otherwise healthy individuals. In these eyes optic nerve head cupping does not develop with optic nerve atrophy as it does in chronic glaucoma patients. Patients with sickle cell disease/trait can develop optic atrophy with smaller intraocular pressure elevations.

E. Secondary haemorrhage
Secondary haemorrhage is said to be present if the size of the hyphaema increases or if a layer of fresh blood is noted over the older darker clot in the anterior chamber, or if dispersed erythrocytes appear over the clot after the blood has settled. Total and near total hyphaema, which often appear dark red, may become bright red at the clot periphery as the clot dissolves. This change in colour is due to clot lysis and this should be distinguished from secondary haemorrhage. Rebleeding can cause a substantial increase in the size of hyphaema. For this reason rebleeding can be associated with complications such as increased intraocular pressure, corneal blood staining, optic atrophy, and peripheral anterior synechiae. Considering the relatively high incidence of surgical intervention for complications of rebleeding, the
risks of surgery (including general anaesthesia) may justify the use of a treatment that significantly reduces the incidence of rebleeding. Although some studies report a greater likelihood of secondary haemorrhage with larger hyphaema, others report no clear relationship between the initial size of the hyphaema and the incidence of secondary haemorrhage. Thus, one should consider the use of medications to reduce the likelihood of rebleeding regardless of hyphaema size.

F. Accommodative impairment

In one study 7% patients had reading disability requiring asymmetric spectacle correction of greater than 2.5 Diopter. Thus evaluation of accommodative amplitude may important when following these patients.

VI The Prognosis of traumatic hyphaema

The prognosis of the traumatic hyphaema depends on the hyphaema height its colour, the reoccurrence of the haemorrhage, the time that takes for the anterior chamber to clear the blood, and mostly on the IOP rise and the corneal blood staining. Patients with predisposing factors are associated with a higher risk of complications and should be more closely followed.

Regarding the blood colour as prognostic indicator, light red colour is indicative of a continuous circulation of the aqueous humor and of an efficient oxygen supply in the anterior chamber. On the contrary, dark red or black colour of the blood (due to the transformation of hemoglobin in methemoglobin) shows the discontinuation of the aqueous humor circulation and the lack of oxygen in the anterior chamber, which means the prognosis of hyphaema must be made very cautiously.

VII. Medical Management to prevent rebleeding

A. Pharmacologic Therapy:

1. Antifibrinolytic drugs: In most studies antifibrinolytic agents (i.e. tranexamic acid and E-aminocaproic acid) significantly lower the rate of rebleeding after traumatic hyphaema and also may delay clot resorption. E-aminocaproic acid is a water soluble antifibrinolytic agent that resemble amino acid lysine. Amicar competitively inhibits fibrin clot digestion by occupying plasmin lysine binding site. Also, E-aminocaproic acid competitively inhibits activating substances in plasma that convert plasminogen to plasmin, perhaps by binding to plasminogen and preventing its binding to fibrin, even after activation to plasmin. Tranexamic acid also resembles lysine and is similar to E-aminocaproic acid in mechanism of action. Dosage is 50 mg/kg up to max. 30 gm/daily for 5 days in 4 hourly divided doses. The dose of Amicar must be adjusted for patients with renal failure. Amicar can precipitate renal colic in patients with renal failure and even mild cases of hemophilia. Active intra vascular clotting and known allergy to E-aminocaproic acid are contraindicators to the use of Amicar. Relative contraindications include a history or predisposition to thrombosis, haematuria of upper renal tract origin renal failure and hemophilia. Adverse effects of Amicar are nausea, vomiting or diarrhea. Other side effects includes pruritis muscle cramps, rash nasal stuffiness, arrhythmia and confusional states.

2. Corticosteroids: Trauma induced breakdown of the blood ocular barrier might enhance the diffusion of some plasma proteins into the anterior chamber including plasminogen, thus increasing the risk of secondary haemorrhage. By stabilizing the blood ocular barrier and by directly inhibiting fibrinolysis corticosteroids might reduce the risk of secondary haemorrhage. Systemic prednisolone appears to be as effective as systemic amicar. Both antifibrinolytics and corticosteroids use are not associated with a statistically significant benefit on final usual outcome despite of the fact that both decrease the rate of rebleeding.

3. Conjugated estrogens: E.g. Premarin can increase the prothrombin concentration and decrease antithrombin activity. It can reduce likelihood of secondary haemorrhage.

4. Mydriatic and Miotic agents: Rakusin found no significant difference in the incidence of rebleeding, in the final visual acuity in the rate of clot absorption, or in the incidence of complications, regardless of whether the patient was using a mydriatic, miotic, neither or both. Because patients with traumatic hyphaema commonly have iridocyclitis, it is preferable to prescribe a cycloplegic agent (atropine 1% once per day) to relieve photophobia and to prevent formation of posterior synechiae. Atropine is preferred because once a day dosing reduces the amount of ocular manipulation needed which may reduce the chance of secondary haemorrhage.

5. Aspirin: It seems prudent to avoid aspirin and nonsteroidal anti-inflammatory analgesic medications in this setting.

In summary, there is strong evidence that systemic as well as topical medications including corticosteroids, E-aminocaproic acid and tranexamic acid decrease the risk of rebleeding among patients with traumatic hyphaema. Recommended treatment regime include Topical corticosteroid (e.g. Prednisolone acetate 1% QID) to reduce intraocular inflammation, long acting cycloplegic (e.g. Atropine 1%). In addition either systemic Prednisolone or a systemic amicas is used in most cases. Acetaminophen or codeine is used as analgesics and is better to avoid aspirin and NSAIDs.

B. Bed rest versus Ambulation Management

For most patients, it appears that there is no clear advantage to prescribe bed rest instead of quiet ambulation as long as the environment can be controlled. Children may represent
a subset of patients in whom bed rest may be preferable to ambulation in a hospital setting, if there is a question of control.

C. Effect of eye Patching:
It is recommended that patients with hyphaema wear a metal or hard plastic shield at all times (including sleep) to prevent further trauma to the eye. Gottsch et al suggested that patients with long standing hyphaema who may have prolonged light exposure might be at risk for developing endothelial dysfunction and corneal blood staining. Patching of these patients affected eyes may be prudent.

D. Outpatient hyphaema management:
One should consider outpatient management only if the parents and child were likely to comply with medical recommendations and keep follow up appointments. Similar considerations were to be given to patients with time delay before presentation, penetrating ocular injuries, markedly elevated intraocular pressure and monocular status.

VIII. Surgical Management
Rakusin found that the surgically treated cohort had a higher proportion with absorption in one week. The medically treated cohort had better final visual acuity and a lower incidence of complications.

Read and Goldberg and Deutsch et al developed the followed empirical criteria for surgical intervention, Hyphaema evacuation is recommended in the following cases:
1. A patient has sickle cell disease or trait and if the mean intraocular pressure is greater than 24 mmHg over the first 24 hours or if the intraocular pressure spikes repeatedly over 30 mmHg.
2. In non-sickling patients if the intraocular pressure is greater than 60 mmHg for 2 days (to prevent optic atrophy)
3. The intraocular pressure is greater than 25 mmHg with a total hyphaema for 5 days (to prevent corneal blood staining)
4. There is microscopic blood staining
5. The hyphaema fails to resolve to less than 50% of anterior chamber volume by 8 days (to prevent peripheral anterior synechiae formation)

The surgical approach used depends on the clinical setting and to some degree the training of the surgeon. There are various surgical methods to evacuate hyphaema few are described below:

To lower intraocular pressure quietly, an anterior chamber paracentesis can be performed at the slit lamp under topical anaesthesia if the patient can co-operate. With a sterile lid speculum in place and after sterilizing the ocular surface with topical povidone iodine, a 0.5 inch 30 gauge needle attached to a tuberculin syringe is introduced at the limbus. While the surgeon holds the syringe in place the assistant aspirate the bloody aqueous humor slowly. This approach will not be effective if most of the anterior chamber is filled with clot.

Definitive clot evacuation is done in the operating room. A clear corneal incision is created near the limbus just superior to the horizontal meridian on the side of the surgeon’s non-dominant hand. A bent 23 gauge needle is introduced through this incision for balanced salt solution infusion. The infusion pressure is adjusted to 30-40 mmHg. The vitrectomy probe is introduced into the anterior chamber through a second clear corneal incision near the limbus just superior to the horizontal meridian on the opposite side. The vitrectomy probes cutting port is occluded with the clot. Working at low suction (~50 mmHg) to avoid anterior chamber collapse, the surgeon first attempts to aspirate the liquefied blood. Solid clot is engaged with the probe, drawn centrally and excised by activating the cutting action of the probe. The cutting port is not directed towards the crystalline lens, particularly, while the cutting suction mode is activated. The clot that adheres firmly to the iris is left behind. Clear corneal incisions parallel to the iris are preferred to avoid contact between the intraocular instruments and the iris crystalline lens.

At the end of the procedure, the limbal incisions are closed with 10-0 nylon suture. A peripheral iridectomy is performed if there is a concern regarding the development of pupillary block post-operatively or if the iris prolapses and cannot be repotted.

Other methods include irrigation and if necessary, aspiration of the anterior chamber blood with simcoe irrigation aspiration probe leaving any firmly adherent clot in the eye. Compared to the scleral tunnel incisions, clear corneal incisions seem less likely to predispose to contact between the instruments and the crystalline lens, particularly if a collar button clot with Pupillary block is present, and the iris-lens diaphragm is displaced anteriorly.

If the intraocular pressure is elevated primarily because of dispersed red blood cells, one can irrigate the anterior chamber.

Another surgical technique used was an ab externo corneal section of 90 under a limbus based flap with preplaced sutures. The clot was removed by a combination of spontaneous extrusion and gently manual expression with a muscle hook. Residual clot was removed with week sponges and the corneoscleral section was irrigated with balanced salt solution.

Viscoelastic evacuation of traumatic hyphaema was described early. The viscoelastic properties of Healonid are used to separate the hyphaema from other ocular tissues and to extrude it through a small corneal incision. Healonid maintains a deep anterior chamber and a stable intraocular pressure. It also protects the lens, cornea, and iris and allows clear observation.

A 1 mm stab incision is made with a keratome shaped
Role of Trabeculectomy

Trabeculectomy is done uncommonly. Trabeculectomy can be an effective intervention, particularly in patients with total hyphaema, very high intraocular pressure, and particular susceptibility to intraocular pressure-induced damage (e.g., pre-existing glaucomatous optic atrophy, sickle cell disease/trait). Also if corneal blood staining severely compromises the view of the anterior chamber a trabeculectomy may offer the safest approach to management. A standard technique is used. It may be appropriate to make the ostium some what larger than normal if the retained clot is large.

Special Situations

Sickle cell haemoglobinopathy

Patients with sickle cell disease or trait have a higher incidence of increased intraocular pressure optic nerve atrophy and secondary haemorrhage is the setting of traumatic hyphaema compared to non-sickle all patients. Fibrinolysis may be enhanced in patients with sickle cell trait which could predispose to secondary haemorrhage. In the setting of sickle cell disease or trait the size of the hyphaema may not be a reliable indicator of the subsequent clinical course. For example, there is a poor correlation between hyphaema size and the ease with which intraocular pressure is controlled. Sickled erythrocytes are less able to pass through the outflow channels of the trabecular meshwork than are normal erythrocytes which are analogous to the inability of “ghost erythrocytes” to pass through the outflow channels. Increased intraocular pressure is poorly tolerated in patients with sickle cell disease as evidenced by the fact that central retinal artery occlusion has followed the formation of small hyphaema in young individuals with sickling haemoglobinopathy. Flow in the central retinal artery of sickle cell patients may be impaired significantly at intraocular pressures greater than 40 mmHg.

These observations have led Goldberg to suggest avoiding medical treatments that promote sickling when managing patients with sickle cell disease/trait and hyphaema. Repeated or excessive dosages of hyperosmotic/diuretic agents (e.g., glycerin, isosorbide, and mannitol) should be avoided, as they may cause haemoconcentration and increased blood viscosity in the ocular microvasculature. Systemic carbonic anhydrase inhibitors not only promote haemoconcentration but also induce systemic acidosis, which is known to exacerbate erythrocyte sickling. Besides lowering the aqueous humor PH, acetazolamide increases the concentration of ascorbic acid in the aqueous humor and ascorbate may exacerbate the sickling process itself possibly by acting as a reducing agent. Methazolamide creates less systemic acidosis than acetazolamide and hence it is recommended in patients with traumatic hyphaema and sickle cell haemoglobinopathy. The following protocol is recommended in patients with sickle cell disease/trait. Use timolol as the primary topical agent. Add topical brimonidine or apraclonidine if an additional agent is needed. If a further agent is necessary, use topical Dorzolamide before using methazolamide. A hyperosmotic should be used infrequently, for example, only once every 24 hours and as last resort to avoid surgery. Surgical evacuation of hyphaema should be considered at lower intraocular pressures than proposed in the management of hyphaema in the non-sickle patient. 1Deutsc et al have suggested that one consider surgical intervention if the intraocular pressure averages more than 24 mmHg over any consecutive 24 hours period despite
maximum tolerated medical therapy. In addition, if the intraocular pressure increases transiently and repeatedly above 30 mmHg surgery should be considered.

**Clotting disorders:**
It is recommended that in the presence of even minor signs of intraocular haemorrhage the patient should be admitted to the hospital and the deficient clotting factor (or cryo precipitate) should be infused regularly during the high risk period for secondary haemorrhage (i.e., first 5-7 days after the injury). If the patient must undergo surgery to evacuate hyphaema, it may worthwhile to provide replacement therapy sufficient to restore levels of clotting factor levels to 100% of normal during the procedure. Patients with even mild cases of hemophilia may be at increased risk for acute renal failure if treated with E-aminocaproic acid.

**Management of Hyphaema in Children**
Hospital admission for children are recommended if there are concurrent injuries mandating admission; if the hyphaema is large (e.g.: ≥ 50% of anterior chamber volume); if the intraocular pressure is elevated; if the patient has sickle cell disease/trait or clotting diathesis, if there is a time delay before presentation; or if there is concern regarding medication delivery, compliance with activity restrictions, ability to return for follow up, or safety of the home environment. One hyphaema complication unique to the paediatric population is the development of amblyopia, which can occur as a result of corneal blood staining.

The use of Amicar or prednisone is recommended routinely in children with hyphaema. Both the apparent efficacy of systemic and topical steroids in reducing the incidence of secondary haemorrhage and the commonly accepted value of steroids in attenuating the sequelae of intraocular inflammation (which often accompanies ocular trauma) lead to prefer the use of systemic steroids with or without topical steroids, rather than Amicas, in managing children with traumatic hyphaema, independent of whether they are managed as inpatients or outpatients.

**Recommendations**

**Step 1:** Obtain complete history. Ocular (Fuchs, glaucoma, amblyopia etc) systemic, medications and details of injury

**Step 2:** Identify and treat associated injuries and conditions along with complete eye examination

**Step 3:** Medical management. Topical corticosteroid, cycloplegic, IOP lowering agent, analgesic antiemetic agents, rigid shield and patch

**Step 4:** Systemic medication to reduce secondary haemorrhage (Amicar/prednisone./Clotting factors/platelets in case of haemastological abnormalities)

**Step 5:** Daily slit lamp examination. Visual acuity, hyphaema height, IOP, cornea

**Step 6:** Discharge the patient if IOP is satisfactory and hyphaema <1/2 of AC. Also depends on patient compliance

**Step 7:** Surgery if risk of corneal blood stain, risk of optic atrophy or synechiae formation (See above)

**References**
1. Introduction
Traumatic endophthalmitis is a major cause of visual failure following open globe injuries and seemingly small injuries without obvious intraocular damage. Non-surgical trauma is involved in 25% of cases of endophthalmitis \(^1\) and 2% to 7% of all penetrating injuries result in culture proven endophthalmitis \(^2, 3\). In spite of recent advances, overall prognosis remains poor. This is primarily due to infection with organisms of high virulence and delay in treatment. It is also observed that endophthalmitis is more commonly associated with cases involving intraocular foreign body (IOFB) than with cases without IOFB \(^2\). This article describes the microbiology, diagnosis, treatment and prognosis of endophthalmitis.

2. Microbiology
Traumatic endophthalmitis is unique in having high incidence of Bacillus species in particular B. cereus and associated with IOFB (Table 1). This organism produces enzymes and exotoxins that may result in loss of the eye over a short period of time. Clostridia are uncommon after trauma and may be associated with constitutional signs and symptoms. Fungal endophthalmitis is invariably caused by filamentous fungi. Fusarium solaneae and similarly virulent species are associated with the worst prognosis. The microbiology of paediatric post-traumatic endophthalmitis differs from adult disease, with streptococcal species as the most common infecting organism \(^17\).

3. Diagnosis
Traumatic endophthalmitis is often difficult to diagnose as the signs of the disease could be masked by the consequences of trauma. The mean interval between injury and onset of clinically detectable endophthalmitis is found to vary depending on the causative organism. The mean interval varies from as low as 4 days for bacterial cases to as high as 57 days for fungal cases \(^16\). Brinton et al. studied the injury-to-treatment interval \(^2\) in a set of patients. In the case of failure in treatment, the mean injury-to-treatment interval was observed to be 3 days. The mean injury-to-treatment interval was higher in the case of successful treatment and was observed to be 8 days. This implies that prognosis is better with organisms of lower virulence, as in these cases endophthalmitis may develop more slowly and remain treatable for a longer time.

Clinical features of traumatic endophthalmitis include pain, proptosis, eyelid swelling, echymosis, elevated intraocular pressure, corneal edema, corneal ring abscess, anterior chamber reaction, hypopyon, lens damage, intraocular gas bubbles, vitreous exudates and retinal periphlebitis \(^12\). The presence of intraocular gas bubbles can be attributed to clostridia or B. cereus \(^18\).

<table>
<thead>
<tr>
<th></th>
<th>Traumatic (%)</th>
<th>Postoperative (%)</th>
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<tbody>
<tr>
<td>S.epidermidis</td>
<td>24</td>
<td>S.epidermidis</td>
</tr>
<tr>
<td>Bacillus species</td>
<td>22</td>
<td>S.aureus</td>
</tr>
<tr>
<td>Streptococcus species</td>
<td>13</td>
<td>Gram negative Organisms</td>
</tr>
<tr>
<td>Gram negative Organisms</td>
<td>11</td>
<td>Streptococcus species</td>
</tr>
<tr>
<td>Mixed flora</td>
<td>10</td>
<td>Fungi</td>
</tr>
<tr>
<td>S.aureus</td>
<td>8</td>
<td>Misc.gram-positive organisms</td>
</tr>
<tr>
<td>Fungi</td>
<td>8</td>
<td>Anaerobes</td>
</tr>
<tr>
<td>Anaerobes</td>
<td>3</td>
<td>Mixed flora</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>Bacillus species</td>
</tr>
</tbody>
</table>

Table 1. Causative Organisms of traumatic and postoperative endophthalmitis (%)
Ultrasound B scan examination is absolutely necessary to rule out IOFB, vitreous involvement and retinal detachment, in all cases where visualization of the posterior segment is obscured. A CT scan may also be necessary in cases of penetrating trauma where IOFB is suspected, to facilitate IOFB removal along with primary repair.

Diagnosis ultimately rests with identification of infectious organisms in the eye by appropriate cultures and stains. Vitreous and aqueous samples must be obtained in all suspect cases before therapy. Samples must be examined by gram stain. However, it is important to keep in mind that a negative result does not mean non-treatment, because a gram stain is positive only in 60% of culture positive cases. KOH mount and calcoflour mount of sample is also recommended for fungus. Aspirate should be promptly plated on blood agar, chocolate agar and thioglycolate broth and incubated at 37 degrees. A separate sabouraud’s plate for fungi and cooked meat medium should be incubated anaerobically. If one uses vitrectomy cassette fluid, concentrating the sample with membrane filtering system improves organism yield significantly. Approximately 64% of eyes with clinical endophthalmitis will display a positive culture. Repeat cultures may be performed 48-72 hours after initial therapy especially if initial results turn negative. Identification of organism using polymerase chain reaction (PCR) technique is useful, especially those from whom ocular samples prove to be culture-negative.

4. Treatment

(1) Prophylaxis

The routine use of intravenous, periocular and topical antibiotics is indicated in virtually all cases of penetrating trauma, unlike in post-operative endophthalmitis. The breakdown of blood/ocular barrier by inflammatory processes in the setting of trauma improves antibiotic penetration and the actual intracocular drug level may even reach therapeutic levels. For gram-positive coverage, many isolates including S. epidermidis are uniformly sensitive to vancomycin and aminoglycosides (amikacin, gentamycin) are useful for gram-negative coverage. Ciprofloxacin may be an alternative in case of resistance.

Are intravitreal antibiotics necessary in all cases of globe rupture? Clinical experience suggests that most ocular lacerations without IOFB do not develop endophthalmitis. However, since such trauma is usually closely followed up, such an intervention can be attempted with the onset of definite signs and symptoms.

On the other hand, the presence of IOFB even without signs and symptoms merits intravitreal therapy, as there is a higher risk for endophthalmitis with organisms as virulent as B. cereus.

(2) Active management

Vancomycin and amikacin/cefazidime may be considered along with dexamethasone intravitreally as first line drugs against bacterial pathogens. For organisms like B. cereus, clindamycin with amikacin/cefazidime is the most effective regimen. Anti-fungal therapy is usually initiated only after a positive culture or smear is obtained. Amphotericin B or voriconazole (if resistance) are the drugs used in such cases. A summary of the treatment is given in Tables 2, 3 and 4.

a. Vitrectomy

The advantages of vitrectomy include opportunity to obtain vitreous sample, clear ocular media, remove intraocular inflammatory and toxic products and facilitate greater drug diffusion. Vitrectomy is done as a primary procedure when endophthalmitis is associated with IOFB or retinal detachment. Driebe et al. reported 94% success rate with recovery of 20/400 vision with intraocular antibiotics only compared to a 50% success rate in the vitrectomy with antibiotics group. However, there was selection bias in this study, with vitrectomy being reserved for worst cases. Puliafito et al. found better visual results when vitrectomy was performed within first 24 hours than later. Raichand et al. recommended vitrectomy if eye was worse 24 to 48 hours after intravitreal antibiotic therapy. Pflugfelder et al. favored vitrectomy in association with intravitreal therapy for fungal cases. Forster showed that less virulent organisms like S. epidermidis responded well to intravitreal drugs alone, in comparison to more virulent organisms which ultimately needed vitrectomy. Disadvantages of vitrectomy include rapid clearance of intravitreal drugs, retinal breaks and retinal detachment. Nelson et al. noted a 21% incidence of retinal detachment in cases treated with vitrectomy.

b. Corticosteroids

Prophylaxis with steroids should be avoided as it may mask clinical signs and symptoms. A single intravitreal steroid injection may be considered as part of initial therapy especially for cases with severe inflammation. As a rule steroids are omitted in early management of traumatic fungal endophthalmitis but may be used in the setting of improved clinical course at a later stage.

5. Prognosis

The prognosis remains dismal in spite of recent advances. Factors include a delay in diagnosis, virulent spectrum of organisms and extent of associated injury. Presence of IOFB, retinal detachment and retinal toxicity in the setting of multiple intravitreal injections indicate poorer outcomes.
**Recommended drug therapy in suspected traumatic endophthalmitis**

<table>
<thead>
<tr>
<th>Intravitreal administration</th>
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<tbody>
<tr>
<td><strong>Foreign body absent</strong></td>
<td>Vancomycin 1 mg in 0.1ml and amikacin 400 microgram/ceftazidime 2.25 mg in 0.1 ml</td>
</tr>
<tr>
<td><strong>Foreign body present</strong></td>
<td>Clindamycin 250 microgram in 0.1ml and amikacin 400 microgram/ceftazidime 2.25 mg in 0.1 ml</td>
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</table>

Dexamethasone 200 to 400 microgram may be considered for all cases with severe inflammation, but strict guidelines for intravitreal steroid use have not been determined.

<table>
<thead>
<tr>
<th>Subconjunctival administration</th>
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</tr>
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<tbody>
<tr>
<td><strong>Foreign body absent</strong></td>
<td>Vancomycin 25 mg and gentamicin 20 mg</td>
</tr>
<tr>
<td><strong>Foreign body present</strong></td>
<td>Clindamycin 34 mg and gentamicin 20 mg</td>
</tr>
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<table>
<thead>
<tr>
<th>Topical administration</th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Foreign body absent</strong></td>
<td>Vancomycin 25 mg/ml drops administered q4h alternated with fortified gentamicin 14 mg/ml drops q4h / Cefazolin 133 mg/ml / ceftazidime 50 mg/ml</td>
</tr>
<tr>
<td><strong>Foreign body present</strong></td>
<td>Clindamycin 20 mg/ml drops q4h alternated with fortified gentamicin 14 mg/ml drops q4h / ceftazidime 50 mg/ml</td>
</tr>
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In cases with severe inflammation, prednisolone acetate 1% drops may be added after initiation of antibiotic therapy.

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<tr>
<th>Systemic administration</th>
<th></th>
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</thead>
<tbody>
<tr>
<td><strong>Foreign body absent</strong></td>
<td>Vancomycin and aminoglycosides / ceftazidime</td>
</tr>
<tr>
<td><strong>Foreign body present</strong></td>
<td>Clindamycin and aminoglycosides / ceftazidime</td>
</tr>
</tbody>
</table>

In cases with severe inflammation, prednisone may be given orally after initiation of antibiotic therapy.

**Recommended drug therapy for traumatic fungal endophthalmitis**

<table>
<thead>
<tr>
<th>Intravitreal administration +</th>
<th>Amphotericin B 5 microgram in 0.1 ml.</th>
</tr>
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<tbody>
<tr>
<td>(Voriconazole 50 microgram in 0.1 ml [20] may be used in addition or given as a subsequent injection after failure of treatment with amphotericin B)</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Subconjunctival administration +</th>
<th>Miconazole 5-10 mg or amphotericin B 1 mg</th>
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<table>
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<tr>
<th>Topical administration +</th>
<th>Natamycin 5% or amphotericin B 0.15% or miconazole 1% drop q1h or Voriconazole 0.01 mg/cc</th>
</tr>
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<table>
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<tr>
<th>Systemic administration $</th>
<th>Ketoconazole 400-600 mg daily by oral administration. Voriconazole maybe tried in select cases</th>
</tr>
</thead>
</table>

These recommendations are derived partly from the article by Pflugfelder et al, *to which the reader is referred.

*Therapy for fungi is administered if the vitreous or aqueous specimen displays fungal elements on fresh smear or a positive fungal culture is obtained.

+Intravitreal injections are not repeated except in cases with a positive repeat culture or smear.

+Pflugfelder et al *reserve adjunctive subconjunctival and topical therapy for cases with significant anterior segment involvement.

$ Ketoconazole therapy appears to be well tolerated and may be used orally as sensitivities indicate. Systemic therapy with amphotericin B has a high incidence of toxicity, and its use in fungal endophthalmitis may be questioned. If intravenous therapy with amphotericin B / voriconazole is selected, careful attention to systemic toxicity is essential.
Guidelines for the management of suspected traumatic endophthalmitis

1. Prompt collection of vitreous and aqueous specimens for culture, stain, and smear in all suspected cases.
2. Evaluation for intraocular foreign bodies by CT, X ray, and ultrasound where indicated.
3. Systemic, periocular, and topical antibiotic prophylaxis in all cases of globe laceration, rupture or penetration. These eyes may be carefully followed in a hospital setting without intravitreal drug therapy if a foreign body is not involved and signs of endophthalmitis are absent.
4. Intravitreal antibiotic therapy (in addition to systemic, periocular, and topical therapy) in all cases of suspected traumatic endophthalmitis and all cases of intraocular foreign body or soil-related injury.
5. Vitrectomy (often dictated by management of specific trauma such as intraocular foreign body) offered to cases of suspected traumatic endophthalmitis that display loss of red reflex, severe inflammation, or intraocular gas. Limited vitrectomy is preferred to minimize the risk of iatrogenic retinal detachment.
6. Consideration of intravitreal steroid therapy for cases with severe inflammation.
7. Repeat culture of vitreous and aqueous in 48 to 72 hours in cases with a positive initial culture and clinical deterioration following initial therapy.
8. Avoidance of repeat intravitreal injections, except in cases with a positive repeat culture or stain.
9. PCR in culture negative cases.

Table 4: Guidelines for the management of traumatic endophthalmitis

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References

4. Affeldt JC et al, Microbial endophthalmitis resulting from ocular trauma, Ophthalmology.1987; 94: 407-413
The peripheral vitreoretinal interface is the final "ring of tissue" damaged in blunt trauma. This is the one area most difficult to examine in the immediate post-traumatic period. Examination of this area is particularly important in that undetected pathologic changes can have the most devastating effects on vision in a relatively shorter period of time. The impact of blunt injury affects the sclera, choroid, vitreous, retina and the optic nerve, the manifestations of which is dealt with in detail in this article.

**Mechanism of Blunt Injury**

The four phases of blunt injury are compression, decompression, overshooting and oscillations. Anterior – posterior compression of the globe at the cornea results in equatorial expansion and shortening of the globe along the visual axis. The anterior- posterior diameter of the globe decreases by as much as 41% and the equatorial diameter increases up to 128% of normal. The driving mechanism for pathological changes in this area is the equatorial elongation, creating shearing forces between the extensible ocular wall and the much less extensible vitreous. (Figure 1)

**Scleral rupture**

Direct rupture of sclera is uncommon and occurs at the site of impact. Indirect rupture occurs remote from site of impact in an area of scleral weakness. The sclera is thinnest at the insertion of the rectus muscles (Figure 2), where it measures 0.3mm. It is thickest at the posterior pole (1mm) and about 0.4 to 0.5mm at the equator. It is at the muscle insertions and the equator that a rupture is most likely to occur. At the clinical limbus, the thickness is reduced to approximately 50% and can infrequently result in rupture especially in operated eyes. Likewise, although thick in the area of insertion of the optic nerve, tissue density is least at the margin of lamina cribrosa and can result in tears or avulsions. Pathological processes and iatrogenically induced weakness can predispose the sclera to rupture.

![Figure 1](image1.png)

![Figure 2](image2.png)

![Figure 3](image3.png)

Scleral rupture (Figure 3) is often occult mainly because ophthalmic examination may be limited by ocular media opacities, including hyphema, cataract and vitreous hemorrhage. Signs of occult rupture are visual acuity of perception of light or no PL, reduced ductions, ocular hypotony, hyphema, severe chemosis, abnormally deep or shallow anterior chamber, afferent pupillary defect, vitreous hemorrhage and a large bulous subconjunctival hemorrhage. The only definitive way to rule out scleral rupture is careful controlled exploration of the globe. Eyes with scleral rupture have a high incidence of microbial contamination and are often severely injured internally. Early primary repair becomes the most important step in management.
Choroidal Rupture

This can occur directly at the site of impact or indirectly in a contre-coup fashion. Choroidal ruptures occurring anterior to the equator are usually direct injuries while posterior choroidal ruptures are indirect injuries.

The classic posterior choroidal rupture (Figure 4) is a tear in the choriocapillaries and overlying Bruch’s membrane that typically forms concentric to the optic nerve. These breaks are caused by sudden horizontal expansion occurring as a result of anteroposterior compression. These may be associated with intra or subretinal hemorrhage or in some cases may be subtle identified only on angiogram. These ruptures generally heal with scarring and retinal pigment epithelial hyperplasia. Formation of choroidal neovascular membrane is a known late complication.

Direct ruptures form at the site of contusion impact from compression necrosis and tend to be anterior, oriented parallel to ora serrata, most commonly in the inferotemporal quadrant.

When choroidal rupture is accompanied by retinal rupture it is termed ‘retinitis sclopeteria’. This classically results from high velocity missile accidents. Though not strictly a result of blunt injury, this is caused by concussive forces transferred to the globe. Shock waves are transmitted from the object to adjacent globe with resultant hemorrhagic rupture of the choroid and the retina leading to necrosis and fibrous proliferation.

Commotio Retinae

Commotio retinae or Berlin’s edema is a post traumatic retinal condition that may manifest even after mild blunt trauma. It may be observed in the retinal periphery and in the posterior pole. Sipperley and coworkers showed histologically that the clinical retinal changes were a result of structural changes, with disruption of photoreceptor outer segments. (Figure 5 a &b)

Commotio retinae is characterized by a whitish gray retinal appearance observed several hours after blunt trauma. It may be accompanied by intraretinal hemorrhage or even choroidal rupture. Visual acuity may be normal or profoundly decreased to 20/400. As the white retinal changes resolve visual acuity improves unless severe retinal pigment epithelial damage, choroidal rupture or severe intraretinal hemorrhage has occurred. No effective treatment for commotion retinae is known.

Hypotonic Maculopathy

Manifests as disc edema, choroidal folds and in severe cases choroidal detachment. (Figure 6)

Post traumatic retinal breaks and dialysis

Post traumatic retinal holes result from contusion necrosis due to the direct result of coup or contre-coup forces especially if they are situated posteriorly. Post traumatic macular holes also are known to occur due to contusion necrosis resulting in macular cyst formation. Subsequent rupture of the cyst leads to macular hole formation.

Traumatic retinal breaks occur in the periphery when the vitreous is violently shifted away from the retina. These occur in areas of strong vitreoretinal adhesion. The vitreous base, lattice degeneration, old chorioretinal scars and retinal blood vessels are the most common sites of retinal tearing. Superonasal is the most common site.

Retinal dialysis (Figure 7) followed by late retinal detachment can be one of the most devastating visual consequences of blunt injury to the posterior segment. This refers to a break occurring at the ora serrata, whose anterior edge is at the ora serrata and posterior edge is attached to the vitreous base. Trauma is the cause of about 20% of dialysis. This is commonest in the inferior temporal quadrant but is also known to occur in the superior quadrants and a supernasal dialysis is classically secondary to trauma. Retinal dialysis occurs when the vitreous base is avulsed. The retina and vitreous are tightly adherent at the vitreous base, and as the vitreous base is avulsed into the vitreous cavity the retina follows creating a tearing at or near vitreous base. Management of retinal dialysis depends on the presence or absence of retinal detachment.
Traumatic retinal detachment

Traumatic retinal detachments account for 10-19% of all phakic retinal detachments. This is associated with inferior temporal dialysis in 31%, superior nasal dialysis in 22%, giant retinal tears in 11% and lattice associated tears in 8%.

Vitreous changes in blunt trauma

The vitreous can be injured in blunt trauma by disinsertion or hemorrhage. Disinsertion occurs at the vitreous base, optic nerve, retinal vessels, lattice degeneration or scars. Commonest changes are avulsion of the vitreous base (Figure 8) with associated retinal dialysis, posterior vitreous detachment with retinal tear and vitreous hemorrhage. The avulsed vitreous base has the appearance of a hammock or ribbon suspended loosely through the vitreous cavity. Vitreous haemorrhage commonly occurs due to torn retinal blood vessels. Pigment in the vitreous is an indication for careful search for retinal tear or dialysis.

Optic nerve injury

The optic nerve is rather resilient to injury from blunt trauma. Injury generally occurs following severe concussive force with concurrent multisystem trauma and brain stem injury. The common causes include motor vehicle or bicycle accidents, falls or physical assault. This has been rarely reported following minor injury. Blunt trauma results in indirect injury to the optic nerve. These kinds of injuries occur when the force of impact is transmitted to the nerve through the bones or by motion of the globe.

Optic nerve injuries can be divided into anterior or posterior types. Anterior indirect injuries involve the intraocular portion. Posterior indirect injuries damage the intracanalicular portion. The intraorbital portion is involved less frequently because this portion of the nerve is not tethered to the globe or the orbit, allowing movement when forces are applied. Anterior injuries are rare and result when the globe suddenly is rotated or displaced anteriorly, such as when the eyes are accidently poked. The most accepted theory suggests that blunt force to the globe causes extreme rotation and sudden strain on the nerve. These forces result in tears or avulsion of the anterior optic nerve at the margin of lamina cribrosa. Ophthalmoscopically this is seen as a defect in the optic disc with or without massive hypotony. Visual loss is usually profound and permanent. A permanent pit like defect is seen in the centre of the optic nerve later with fibrogial proliferation. (Figure 9) Prelaminar injuries are less severe usually and presents with disc swelling and peripapillary hemorrhages. Later this results in varying severity of optic atrophy.
**Introduction**

Trauma related glaucomas are a mixed bag of conditions that raise the intraocular pressure (IOP) and hence compromise the optic nerve function. When we talk about trauma we tend to think about blunt trauma and penetrating trauma (as well as chemical, electrical and radiation traumas) \(^1\,^2\). Most of the secondary glaucomas are a result of blunt trauma. It can occur acutely or more often delayed as in angle recession.

**Blunt Trauma and Glaucoma**

Blunt frontal trauma causes the corneal apex to indent and consequently, the limbal ring gets stretched. The peripheral cornea is pushed outward and the iris root rotates backwards. The zonules are stretched pushing the lens (lens iris diaphragm) backwards. When this happens violently enough that would tear tissues to detach from their attachment at the limbus – leading to iridodialysis, trabecular meshwork tears, angle recession, cyclodialysis, zonulolysis (Figure 1). This would first open up blood vessels and cause a hyphaema.

**Hyphaema**

Hyphaema is presence of blood in the anterior chamber. It occurs after trauma due to a tear into a blood vessel bounding the anterior chamber – most often in the angle recess. Hence one should always suspect angle recession in any case of traumatic hyphaema.

Hyphaema causes a rise in intraocular pressure via a few mechanisms. Primarily it causes an increase in resistance to aqueous outflow by the blood cells (predominantly RBCs) blocking the trabecular meshwork. Viscosity of aqueous is also marginally increased by the blood proteins. To add to this there is inflammation due to the trauma itself causing a trabeculitis and swelling of the trabecular meshwork reducing the pore spaces in the same. If the clot covers the pupil then it will occlude the same and cause pupillary block glaucoma as well. If the situation continues long enough then peripheral anterior synechiae can form due to pupillary block over a wide area or due to the inflammation per se in limited areas. Both can compromise aqueous outflow over long term.

At the initial setting the IOP may be normal, low or high. This is due to associated changes. There maybe a cyclodialysis cleft draining out the aqueous. Alternatively the ciliary body may temporarily shut down in the context of inflammation. There may be a retinal detachment. (Of course a globe rupture or penetration has to be ruled out and managed). The IOP is the net effect of inflow vs. outflow. Hence when the inflow exceeds out flow the IOP will rise. So IOP should be monitored at least daily in the acute setting.

A specific situation warrants mention. Normally the RBCs would wriggle out through the trabecular meshwork. If they become stiff (not pliable) they get stuck in the meshwork. This happens in sickle cell disease. When deoxygenation occurs, the RBCs sickle because the PH decreases. The sickled cells clog the meshwork more as they are stiff and the cells are now more tapered and can wedge better. This causes the IOP to rise fast and thereby reduce the retinal blood flow. Slowing of blood in the retinal circulation causes intravascular sickling, as the cells get deoxygenated more, leading to obstruction of small vessels and even a central retinal artery occlusion. Thus in sickle cell disease and trait patients hyphaema causes a rise in IOP more often and the damage to retina and optic nerve are more profound. Additionally acetazolamide which is the first choice drug to be used in hyphaema related IOP rise worsens sickling and is contraindicated in these patients. Hyperosmotics like mannitol cause haemoconcentration and this causes sickling as well. (So even though sickle cell disease is rare in our patients it is still prudent to order a thick peripheral blood smear or a sickle preparation with your clinical pathologist in all cases of hyphaema.)
Rebleeds
By the 2nd to 4th day the clot retracts and this can reopen the bleeding site causing an increase in the hyphaema. Thus these patients need at least daily examinations to look for the same. Earlier management of hyphaemas included epsilon aminocaproic acid as an antifibrinolytic agent to combat this. The side effect and cost profile did not match the risk involved and not many favour this anymore.

Management of IOP in Hyphaema
All hyphaema patients need treatment of the inflammation with topical steroids and cycloplegics (Earlier pilocarpine was advocated to increase surface area of iris for absorption of blood. But the added inflammation due to pilocarpine has prompted its withdrawal from treatment regimes). The IOP is to be treated when elevated medically. One prefers aqueous humor suppressants in this situation viz., acetazolamide oral, topical β-blockers and topical dorzolamide. In sickle cell disease methozolamide systemically and dorzolamide topically are probably safer than acetazolamide which is contraindicated. Hyperosmotics like mannitol are also effective. Prostaglandin analogues and α-agonists are not well tolerated by inflamed eyes (though not absolutely contraindicated)

Surgical intervention is required in hyphaema in certain situations. If there is an eight ball hyphaema or a total blood clot filling the anterior chamber or if the clot is causing an obvious pupillary block, then the glaucoma is an angle closure glaucoma. If the pupil has been given enough time and cycloplegic medications to dilate and this situation persists, then it is unlikely to respond only to medical management. A peripheral iridectomy is warranted. Laser iridotomies are too small and a surgical iridectomy is easier in the presence of a blood clot and can be done to relieve the pupillary block. Surgical intervention is usually indicated on or after the fourth day. Overall, indications for surgical intervention are outlined below:

- Microscopic corneal blood staining (at any time)
- Total hyphaema with intraocular pressures of 50 mm Hg or more for 4 days (to prevent optic atrophy)
- Total hyphaemas or hyphaemas filling greater than 75% of the anterior chamber present for 6 days with pressures of 25 mm Hg or more (to prevent corneal blood staining)
- Hyphaemas filling greater than 50% of the anterior chamber retained longer than 8-9 days (to prevent peripheral anterior synechiae)
- In patients with sickle cell trait or sickle cell disease who have hyphaemas of any size that are associated with intraocular pressures of greater than 35 mm Hg for more than 24 hours

A host of surgical options are described. The ideal day for surgery is probably day 4 when the clot retracts. The intervention can be anterior chamber washout with BSS, mechanised hyphaemectomy with a vitrectomy probe, a routine trabeculectomy, etc. Blind pulling on the clot is to be discouraged as one may cause a rebleed from the injured vessel in the least and may cause inadvertent tissue injury by pulling off iris, descemets, lens capsule, etc.

Trabecular meshwork tears
If one does a gentle gonioscopy in the first few days after trauma one may see a trabecular meshwork tear (Figure 2). This is a disinsertion of the trabeculum from the Schwalbe’s line. Initially this leads to a lowering of IOP as it works like a trabeculotomy – opening up the Schlem’s canal directly to the anterior chamber. Later on a glass membrane forms over it and the tear gets obscured. Now the IOP rises as that part of the angle is non-functional. If the initial tear was not deep enough to cause a recession picture the late picture could be akin to a POAG. Medical control of IOP is done and trabeculectomy is an option only if medical management fails.

Figure 2: Trabecular tears (black arrow in Photograph and white arrow in the UBM picture) are disinsertion of trabecular meshwork from Schwalbe’s line. Can initially work like trabeculotomy with low IOP but later glass membrane grows over and IOP rise.

Lens related glaucomas in Trauma
Trauma can cause subluxation or dislocation of the lens. It can disrupt the capsule and allow lens matter to travel into the anterior chamber. The contusion itself can cause lens swelling or intumesce of the lens. All these can affect the IOP.

A subluxated or dislocated lens can cause pupillary block glaucoma. This can either be by the lens moving forward or by a knuckle of vitreous blocking the pupil. If the lens is clear and reasonably supported by zonules a laser PI can be attempted as a first option. If there is significant cataract or if lens edge is in the pupillary area lens extraction is indicated.

A subluxated lens can move forward and physically push the peripheral iris forward and close the angle. Lens intumesce also does the same. These situations cause phacomorphic glaucoma like picture. Again lens extraction would cure the glaucoma.

If the lens capsule be torn, then the lens matter can move
into the anterior chamber. The fragments of lens fibres would block the trabecular meshwork causing a lens particle glaucoma. All these situations warrant lens extraction – preferably extra capsular, if not possible then intra capsular. Preoperatively an indentation gonioscopy is needed as there could be a compromised angle contraindicating an anterior chamber IOL (in case the lens is removed intra capsularly). In general, we prefer an in the capsular bag IOL or a scleraly fixated IOL over the use of ACIOL in cases with significant trauma.

**Angle recession glaucoma**

Angle recession glaucoma is a post traumatic secondary open angle glaucoma. This is often under diagnosed as the rise in IOP is often delayed and by then history of injury is often forgotten.

Angle recession is sequelae of blunt trauma and is characterised by cleavage between the circular and longitudinal muscle planes of the ciliary body (Fig 3). For this to occur, the cleavage has to go through the aqueous drainage channels in the angle causing variable changes in outflow resistance. The histologic basis for this theory has been described by Wolf and Zimmerman.

The initial IOP after trauma may be low, normal or high. This could be due to ciliary body shut down as part of inflammation following the trauma. Later on this aqueous inflow picks up over a few weeks and IOP may rise. Healing can cause a glass membrane to form across the angle as an extension of the descemet’s membrane and cause an increase in outflow resistance. Trabecular fibrosis also contributes to rise in IOP 1. Hence the rise in IOP can be delayed by weeks to years in these patients.

On gonioscopy one sees a widened angle recess with an irregularly widened ciliary body band. Scleral spur appears abnormally white 1. One may also see torn iris processes on gonioscopy. Even in the absence of a history of trauma one may see telltale signs of an old hyphema in the form of black pigment balls (hemosiderin). Iris pigment looks more brown and not black in the angle (Fig 4). These last for very long time – years in fact.

**Closure of a Cyclodialysis cleft**

A cyclodialysis cleft causes a hypotony. But over a period of time these can spontaneously close (or need surgical closure to improve vision). At this point the outflow resistance goes up and the IOP rises. Essentially this is also due to angle damage from trauma (recession) and needs management like recession. The cleft closure only affects the timing of the rise in IOP.

Identification of cyclodialysis is by indentation during gonioscopy. In Figure 5 the first gonioscopic picture looks normal and on indentation we see the cleft open up.
Ghost Cell Glaucoma
Trauma can cause a vitreous hemorrhage. The blood degenerates over 3 weeks or so in the vitreous. The blood cells lose hemoglobin and become stiff spherical skeletons of the cells called Heinz bodies or ghost cells. These cells if they migrate into the anterior chamber would block the pores of the trabecular meshwork. Unlike RBCs in a hyphaema, which are pliable and can wriggle out of the trabecular meshwork, these stiff cells get stuck. This increases the outflow resistance and the IOP rises with even a few cells.

For migration of ghost cells often there has to be a breach in the anterior vitreous face and the zonular diaphragm (often present in the setting of trauma). Clinically Ghost cells appear as tan (light brown or khaki) coloured cells in the anterior chamber. These can settle down as a tan coloured hypopyon. If blood cells in different stages of degeneration are there they get layered giving us the candy stripe sign in this hypopyon. The setting looks like a uveitis but keratic precipitates are conspicuous by their absence. In the vitreous we see old vitreous hemorrhage.

Management would be by anterior chamber washout. The diagnosis can be confirmed by centrifuging the washout and looking for Heinz bodies under phase contrast microscopy 11 or using a wet mount preparation with methyl violet stain 12 (Fig 6). Invariably this washout will need to be repeated in a few weeks, if the reservoir of altered blood in the vitreous is not removed. So a pars plana vitrectomy is also indicated. Alternatively anterior chamber washout with trabeculectomy can be done in phakic eyes 12.

Penetrating Trauma
Penetrating trauma causes the anterior chamber to stay shallow in the setting of an inflamed eye. This causes peripheral anterior synechiae to form 1, 2. This leads to an angle closure glaucoma. Medical management is opted for first but often a trabeculectomy is required.

Lens trauma can occur as well and is dealt with as described earlier in this treatise.

Fibrous ingrowth and epithelial downgrowth (Fig 7) can occur 1, 2, 13. These cause intractable glaucoma and aggressive surgery to remove the offending cell layers are described. A mitomycin augmented trabeculectomy or a seton is also indicated in this setting.

Figure 5: Cyclodialysis cleft opens up on indentation during gonioscopy (Black arrows). On UBM we see disinsertion of ciliary body from the scleral spur (white arrow) and a shallow ciliochoridal detachment behind it.

Figure 6: Altered blood cells (Heinz bodies) seen with wet mount preparation with methyl violet stain

Figure 7: the arrow shows an epithelial down growth
Retained metallic foreign bodies can cause siderotic glaucoma. Elemental iron causes degeneration of the trabecular meshwork. Localization and removal of the foreign body solves the problem in its initial stages. Sympathetic ophthalmia can also be associated with glaucoma.

**Schwartz syndrome**

Retinal detachment can cause glaucoma if the rod outer segments migrate into the anterior chamber and block the trabecular meshwork. The anterior chamber seems to have cells (actually the photoceptor outer segments) and can resemble a uveitic glaucoma. On careful dilated indirect ophthalmoscopy, one may find a shallow peripheral rhegmatogenous retinal detachment. Surgical closure of the offending retinal break would relieve this glaucoma.

Occasionally one may get a curious iris retraction syndrome. Here there is a seclutio pupilae due to 360 degree posterior synechiae. The anterior chamber is shallow when the IOP is high and on administering an aqueous suppressant (acetazolamide) the anterior chamber dramatically deepens to a concave iris and hypotony. Here again it is a combination of retinal detachment and secondary angle closure that is the culprit. The carbonic anhydrase inhibitors stimulate the retinal pigment epithelial pump and hence reverse the aqueous flow from anterior to posterior via the retinal break. Here both the angle closure and the retinal detachment need to be addressed to resolve the situation.

**Chemical Injury**

Chemical injury causes a bimodal increase in IOP. Initial insult causes the sclera to shrink causing a transient elevation of IOP in about 10 minutes to pressures in the range of 50mmHg. Later the ciliary body shutdown would lower the IOP. In about 1-2 hours the trabecular inflammation causes a more sustained rise in IOP. In this acute setting the status of the cornea does not allow objective IOP measurement and we are often left with presumptive treatment (aided by finger tonometry only). The treatment is only medical with aqueous suppressants alone.

In the intermediate and late stages of evolution of the chemical injury, IOP rises due to presence of peripheral anterior synechiae and trabecular fibrosis due to sustained inflammation. The lens may also become intumescent and worsen the situation. Again treatment is medical and if inadequate one can opt for graded cyclodestruction (trabeculectomy is not an option as almost all conjunctiva would be scarred down). By graded cyclodestruction we mean 90 degrees at a time, either cyclophotocoagulation or cyclocryotherapy. Further cyclodestruction is embarked on only after a period of at least 3 weeks, to assess the full effect of previous therapy. This is usually done prior to keratoplasty in these patients.

**Miscellaneous**

Exposure to penetrating radiation as part of treatment or otherwise leads to radiation retinopathy and as a consequence neovascular glaucoma can occur.

Electrical injury or lightning strike can cause iris pigment dispersion causing a transient rise in IOP that needs no treatment.

Periocular trauma can cause a caroticocavernous fistula, leading to elevated episcleral venous pressure and hence a secondary glaucoma.

Technically rise in IOP due to a retro bulbar hemorrhage and orbital emphysema also is secondary glaucomas. These often need only conservative management but if there is spontaneous central retinal artery pulsation or a central retinal artery occlusion, we need to decompress the orbit. This again needs a graded approach monitoring the fundus till retinal blood flow is established (without spontaneous arterial pulsations). A lateral canthotomy cantholysis → cantholysis cantholysis → 360 degree peritomy bony orbitotomy (as described in good old Duke Elder) can be attempted. These procedures allow for propostsis of the eye to decompress the orbit and hence post operatively one should look out for and manage the consequent exposure keratopathy.

**Epilogue**

Significant blunt trauma usually causes a hyphaema. Glaucma can appear immediately, weeks later or even years later. So every patient with hyphaema needs IOP monitoring and at least one gonioscopy to rule in/out angle recession. In this setting the patient needs to be monitored for life.

**References**


**Ultrasonography in Ocular Trauma**

**Jyotiprakash Vyas MS. Mahesh G MS FRCSEd**

**Introduction**
Ultrasound imaging can be employed to characterize accurately the internal ocular anatomy. In the hands of an experienced echographer, ultrasound is reliable in detecting retinal detachment, posterior vitreous separation, vitreous hemorrhage and opacities, choroidal detachment (can differentiate between serous and hemorrhagic), areas of vitreoretinal adhesion, choroidal and scleral ruptures, vitreous incarceration, and intraocular foreign bodies.

**Advantages**
- Unlike MRI and CT, ultrasound provides real-time images of the eye and orbit.
- The relatively high frequency of the sound waves (10 MHz) affords outstanding resolution (0.1 to 0.01 mm), an ideal choice to image intraocular structures.
- Multiple cross-sectional and radial cuts of the eye can be rapidly obtained at the bedside or in the operating room.
- Serial echography permits following the clinical course of various conditions (e.g., choroidal detachment resolution, membrane and retinal detachment development).
- Ultrasonography is less expensive than radiological studies.

**Disadvantages**
- Because ultrasound requires direct contact with the eyelids and/or globe, it should not be used in eyes with a high risk of extrusion of intraocular contents (e.g., large wound, uncooperative patient). In these cases, echography can be performed in the operating room after the globe has been closed and the patient is under general anesthesia.
- Training and skill are required.
- It is not useful in diagnosing orbital fractures.

**Pitfalls**
- False-negative results are possible on echography if the IOFB is small, wooden, or of vegetable matter.
- False-positive result may be found in the presence of gas bubbles.
- B scan tends to overestimate the size of the IOFB and should not be used for measuring purposes.
- For finding and localizing small, nonmetallic IOFBs in the anterior segment, ultrasound biomicroscopy is superior to CT, MRI, and contact B-scan ultrasonography.
- It is limited in its ability, however, to distinguish between different materials and cannot be used if the globe has an open wound.

**Timing of USG Scan**
- In close globe injury--- If no view of the fundus is afforded, gentle B scan at presentation may be done.
- If Occult Globe rupture is suspected--- Gentle B scan at presentation may be done over closed eye lid without applying pressure.
- In Perforating/penetrating trauma---- Only after primary repair has been done, never before that.

**Parameters to be noted in a B scan**
- Topography- location, extent and shape (borders and contour). Echoes can be point like, membrane like, band like, or mass like.
- Quantitative – reflectivity estimate and measurement (spike height on A-scan and signal brightness on B-scan), internal structure, sound attenuation, absorption and shadowing.
- Kinetic- mobility, after movements and vascularity.

**Basic probe positions include**
- Transverse (Lateral. Here circumferential slice of opposite fundus is seen).
- Longitudinal (Anteroposterior. To see membrane insertion, mass borders etc).
- Axial (To find relation to landmarks).

**Gain**
- High gain is used to see vitreous opacities and gross lesions.
- Low gain is used to see flat fundus elevations.

**Ultrasound in Trauma**

**What to look for?**
- IOFB.
- To assess the posterior segment in closed globe injury with mediahaze (Dislocated lens, vitreous haemorrhage, RD, CD).
- In open globe injuries to detect occult perforation and to rule out IOFB, RD, CD, Vitreous track, Vitreous haemorrhage.
- Iatrogenic globe injuries. e.g. needle perforation.

**Note**
- Should be done over the closed eyelid.
- Probe should be cleaned before use.
- Gentle.

Blunt trauma to the eye can produce marked distortion of the globe resulting in more severe damage than maybe clinically apparent.
Sequelae of Blunt Trauma

<table>
<thead>
<tr>
<th>Anterior Segment</th>
<th>Posterior Segment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyphema</td>
<td>Vitreous Hemorrhage</td>
</tr>
<tr>
<td>Cataract</td>
<td>PVD</td>
</tr>
<tr>
<td>Subluxation of lens/IOL</td>
<td>Retinal tear</td>
</tr>
<tr>
<td>ALC/PC rupture</td>
<td>Retinal detachment</td>
</tr>
<tr>
<td>Corneoscleral rupture</td>
<td>Edema of retinochoroid layer</td>
</tr>
<tr>
<td>Cyclodialysis cleft</td>
<td>Hemorrhagic Choroidal Detachment</td>
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<tr>
<td></td>
<td>Posterior Scleral Rupture</td>
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Sequelae of Acute Penetrating Trauma

<table>
<thead>
<tr>
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<tbody>
<tr>
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<td>ALC/PC rupture</td>
<td>Retinal detachment</td>
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<tr>
<td>Shallow AC</td>
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<td>Ciliary body detachment</td>
<td>Scleral fold</td>
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<td>Iridodialysis</td>
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</table>

Intraocular foreign body (IOFB)

IOFB following trauma are readily picked up by USG. Even if a FB has been detected by the CT it may be unable to indicate whether it lies just within or outside the globe. Metallic FBs produce a very echo-dense signal that persists even at low gain settings. In addition, there is usually marked shadowing of the ocular and orbital structures just posterior to the foreign body (although FB < 0.5mm in diameter or like a metallic wire may not produce obvious shadowing even if they are very echo dense). The FB shows a very high reflectivity on A scan regardless of sound beam direction. The echographic detection of a FB can also be facilitated if it has produced a hemorrhagic track within the eye. For foreign bodies in the anterior chamber an immersion technique maybe necessary. This can only be done after the entrance wound has been sutured or has healed. Examination through the lids with a soft stand off technique may also be necessary if the immersion technique cannot be performed. Now ultrasound biomicroscopy is used for imaging anterior segment in a better way.

Case examples:

Case 1 Vitreous hemorrhage

A transverse scan of the eye showing multiple point like echoes in the vitreous cavity suggestive of a vitreous hemorrhage and a membrane like lesion which shows a moderate spike on corresponding A scan suggestive of partial PVD

Case 2 Posteriorly Dislocated Lens

A transverse B scan of the eye showing a lanceolate lesion in the mid to posterior vitreous cavity suggestive of a posteriorly dislocated natural lens. The corresponding A scan shows a highly intense signal at the interface of the surfaces of the lens.

Case 3 Vitreous Hemorrhage with Hemorrhagic Choroidal Detachment

A transverse scan of the eye showing multiple pin point echoes in the vitreous cavity suggestive of vitreous hemorrhage. Another dome shaped lesion inferiorly with echoes within the substance of the lesion suggestive of hemorrhagic choroidal detachment.

Case 4 Vitreous Incarceration with vitreous hemorrhage
A transverse scan of the eye showing a broad membrane like lesion extending across the mid vitreous cavity attaching to the ocular wall at one end suggestive of vitreous incarceration. In addition it also shows vitreous hemorrhage

Case 5 Vitreous haemorrhage with Serous Choroidal Detachment

Dome shaped echo with clear central area suggestive of serous choroidal detachment inferiorly with point like areas in vitreous cavity suggestive of vitreous haemorrhage

Case 6 Reverberation echoes due to air bubble

Case 7 Retinal detachment with PVD

There is a high reflective membranous echo with not much after movements attached to disc. This is a retinal detachment. Inside that there is another low reflective membranous echo with good after movements which is a complete PVD

Case 8 Occult scleral rupture

There is distortion of ocular coats with discontinuity at the upper part suggestive of rupture. Hype echoic areas outside the globe is haemorrhage. Also there is point like echoes inside the eye suggestive of vitreous haemorrhage

Case 9 Occult Scleral rupture

Longitudinal section through the superior rectus insertion showing discontinuity of ocular coats suggestive of rupture

Case 10 Retained metallic intraocular foreign body

High reflective (100%) echo with shadowing behind
Case 11 IOFB with RD

High reflective (100%) echo with shadowing behind suggestive of metallic foreign body. Also there is a high reflective membranous foreign body with no after movements. This is RD

Role of serial ultrasound in trauma

Case 12

Left- Point like echoes in vitreous cavity suggestive of vitreous haemorrhage. Also there is membranous echo going towards primary repair site. This is vitreous incarceration. After 2 weeks (right) new high reflective membranes have appeared. This is RD

Case 13

Left – Dome shaped lesion with some echoes inside (Kissing choroidals or scalloped appearance). After 2 weeks there is reduction in choroidal detachment

Ocular ultrasound has more than 90% sensitivity and specificity in the diagnosis of ocular trauma cases

References

**Medico Legal Aspects of Ocular Trauma**

**K. V. Raju, MS**

**Introduction**

Basic nature of mechanical injuries is either accidental suicidal or homicidal so they are always associated with legal problems. Many such cases may end up in consumer court or in criminal court. Therefore a doctor who has examined and treated such patients will be called to give evidence as expert witness. Negligence suit may be filed against the doctor if he fails to carry out the medico legal duties in an accepted way. Every doctor must be familiar with the procedure of examination and recording of data.

**Important aspects**

During examination note the type, location, direction, dimensions and presence or absence of foreign bodies in the wound. Measurement should be accurate (as far as possible) and terms like ‘about’ ‘approximately’ must the avoided. If possible take photographs or draw simple sketches of the injury. Next important medico legal duty is to fill up the ‘Accident cum wound certificate’ carefully. Certificate format must be readily available in all hospitals, where there is permission to treat medico legal cases. It is to be prepared in duplicate by taking a carbon copy. Original of this wound certificate must be submitted to the police as a confidential report. Duplicate (Carbon copy) must be kept under safe custody either by the doctor or the institution. After that doctor has to decide about the procedure to be performed for each case. Before doing any procedure an informed written consent has to be signed.

**Consent:** It is defined as “Voluntary agreement, compliance, or permission for specified act or purpose”. The Indian contract Act Section 13, states “two or more persons are said to consent when they agree up on the same thing in the same sense”. Medical practitioners should sign consent before examination and treatment. If they treat or operate without consent will be considered as an internal interference with the patient’s body without legal sanction. This in turn is equivalent to assault for which patient can legally claim for damage. Consent is to be obtained from conscious, mentally sound adults, or from the parent of a child who is less than 12 years of age. Consent is not necessary if the patient is in coma and need emergency treatment. A medico legal case referred by a court of law for examination also doesn’t need consent.

**Police intimation:** A government doctor must intimate all wounded cases, irrespective of the manner and nature of the injury to the nearest police station. A private doctor can abstain from such intimation if the patient does not give consent for the same.

If a medical practitioner complete all those processes like writing ‘wound certificate’ make a ‘written consent’ with the patient for examination and treatment, and ‘if the matter is intimated to police’, his medicolegal duties can be considered as satisfactory. Then he can effectively execute the treatment utilizing his basic knowledge in the concerned branch of medicine. If he fails to perform those medicolegal duties, a negligence suit may be filed against him in a court of law.

**Professional negligence**

“Negligence can be defined as doing something which a prudent and reasonable man would not do, or the omission to do something which a reasonable man would do. Negligence may be either due to lack of knowledge and skill or failure to provide due care during a procedure. Every medical practitioner should have basic reasonable knowledge but it doesn’t mean that he should posses the highest degree of skill and knowledge. No doctor is expected to posses all currently available medical knowledge, and need not apply all known diagnostic and therapeutic technique.

Elements negligence: There are three cardinal elements in negligence.

1. Duty of care
2. Failure to exercise duty of care (Dereliction)
3. Causation of damage to the patient on account of dereliction.

**Duty of care:** A doctor charged with negligence must be under a ‘duty of care’ to the person complaining of negligence. If the doctor was not under duty of care, he can not be charged for negligence even though there is damage to the complaining party. In an emergency care if at all patients is unconscious, if the doctor attend the case it will be considered as duty of care. Duty of care doesn’t exist while attending an injured person on road side following accident. No duty of care exists in medicolegal examination for issuing medical certificate for disability and drunkenness.

**Dereliction of duty:** It is defined as failure of a doctor to honour his duty that is owed to his patient. Such breach of duty may be an act of commissioner an act of omission. Example for commission intravitreal injection in normal eye for the affected other eye. Example for omission is failure to give antitetanus prophylaxis when it is indicated.

**Damage:** It is the injury or disability suffered by the patient due to dereliction of duty. However negligent a doctor might
be, a patient cannot sue him for negligence if no damage has occurred. It should be proved that the breach of duty was the real cause of damage. Example for damage is enucleation of an eye for a retained intraocular foreign body, which was not diagnosed and treated in the initial stage.

**Preventive measures against Negligence**

Prevention is better than cure. If all the medical practitioners function as per the following guidelines the excessive number of negligence suits can be reduced.

1. Do not criticize another doctor: One of the most common cause of negligence arise out of criticism from another doctor, without knowing full facts. Some times patients go from doctor to doctor asking for an opinion. In most of such cases patients wish to have some psychological reassurance. A casual bad remark by another doctor can create problems by shaking the patient’s confidence in the doctor whom he has originally consulted.

2. Employ qualified staff and associate with good partners: Doctors will be responsible for the act of commission or omission of his assistants, non-technical staff, and partners in the course of treating a patient.

3. Update the knowledge: Doctor must be exposed to the latest developments in the concerned field by attending local, national and international conferences.

4. Keep accurate and complete records: An accurate case record should contain history, present illness, physical examination, investigations, impression about the case, treatment adopted, any cross consultations and any refused from the side of patient for any investigations and treatment.

5. Valid consent: Consent must be obtained before starting an examination, diagnostic or investigative procedure or treatment. For the consent to be valid it must be informed, with patient being informed of relevant facts regarding procedure.

6. Ensure reasonable skill and care: Doctor should use reasonable skill and care in both diagnosis and treatment. When in doubt, it is good to take a second opinion. A doctor must ensure that all the instruments used by him are maintained well and sterilized.

7. Guard against therapeutic hazards: Before starting treatment doctor should enquire about the past history of any adverse drug reactions. All the facilities to manage a case of drug reaction must be available.

**Conclusion**

Even though the primary duty of a doctor is patient care, the medicolegal aspects associated with all cases of trauma should get adequate care before the execution of treatment. Record keeping must be accurate, perfect and safe because surgeon may forgot the facts in the due course, but the records will remain as such which can be effectively utilized in a court of law. Your patients may die, but the records will never die, it will speak in its own. Patients must be treated, and the surgeon must be legally protected, for that every body should follow the guidelines mentioned above.

**References**

2. Gupta BD. The Indian Medical Council (Professional conduct, Etiquette and ethics) Regulations 2002.
4. Forensic medicine and toxicology V.V. Pillay.
Surgical and Visual Outcome Following Repair of Retinal Detachment with Giant Retinal Tear

Thomas Cherian MS, Reesha K.R MS, T.P. Ittyerah MS

Abstract

**Aim:** To evaluate the surgical and visual outcome following repair of Retinal Detachment (RD) with Giant Retinal Tear (GRT)

**Materials and methods:** Eleven cases of RD with GRT were surgically managed between July 2007 and June 2008. Belt Buckling + Pars plana vitrectomy with endolaser + silicone oil injection was done in all cases; perfluoro carbon liquid was used to unroll the GRT. Mean follow up was 6 months following the final surgical procedure.

**Results:** All cases had oil removal after 4 months; retina remained attached after 3 months in 9, 1 case needed oil exchange, 1 needed reinjection of oil. 4 cases underwent cataract extraction with IOL along with oil removal. 9 cases had 6/36 or better vision; the causes of poor vision in the remaining 2 were epiretinal membrane and macular hole.

**Conclusion:** Surgical repair of RD with GRT gives good results, the cause of poor visual outcome is a macular pathology.

Introduction

Giant retinal tears (GRT) are retinal breaks that extent over 90 degrees or more in circumference. Progress in vitreoretinal surgical techniques during the past 25 years has greatly improved the anatomic and visual results after surgery for retinal detachment caused by giant retinal tears. Because of the difficulty of unfolding the retinal flap and keeping it attached, the prognosis was traditionally poor. In particular, the intraoperative use of perfluorocarbon liquids has greatly facilitated the manipulation of the retina and the preservation of the lens.

However, recurrent detachments caused by various factors (e.g., extension of preexisting tear, formation of new retinal tears and reopening of tear from proliferative vitreoretinopathy (PVR) still continue to be a challenge.

Encircling scleral buckling has been traditionally used to reduce the risk of such complications. The scleral buckle is thought to reduce early and late traction within the vitreous base, thus decreasing the risk of recurrent retinal detachment. Nevertheless, by inducing a distortion of shape of the eye, scleral buckle may enhance the risk of slippage of retina posteriorly. In addition, scleral buckling induces secondary axial lengthening.

Until now, successful repair of GRTs without scleral buckling has been described, but pre operative PVR has remained an indication for additional scleral buckling as part of repair.

Hoffman et al, for example, treated a small series of six eyes with preoperative photocoagulation of the attached peripheral retina from the ora serrata to the equator. They performed a vitrectomy, an intraoperative transcleral light cryotherapy to the choroidal bed of the tear and to the retina at the tear’s ends, and a C3 F8. Tamponade without placement of scleral buckle. The day after vitrectomy, photocoagulation was applied to the anterior flap of the GRT and the previously detached retina.

Materials and Methods

This study included eleven eyes of eleven patients with GRT operated at Little Flower Hospital Angamaly between July 2007 and June 2008. Nine were males and two were females with a mean age of 44 years (range 15-68 yrs).

Pre operative assessment

The following preoperative clinical characteristics of patients were collected for statistical analysis: age, sex, preoperative trauma, the number of detached quadrants of retina, whether the central area of macula was involved in the detachment, and presence of proliferative vitreoretinopathy. Pre and post operative best corrected visual acuity was measured. Using slit lamp examination the following preoperative variables were noted: the lens status, and whether the patient was phakic, pseudophakic, or aphakic. Fundoscopy was performed using binocular indirect ophthalmoscopy. Fundus drawings were made of the retinal detachment in clock hours.

Surgical technique

Scleral buckling (Belt buckling) was done in all eyes with a #240 silicone band. A standard three port pars plana vitrectomy was performed. The vitreous was cut and aspirated meticulously. The infusion bottle height was lowered and perfluoro-n-octane was then injected slowly to produce a single bubble completely filling the vitreous cavity using a 20 gauge blunt or silicone tipped cannula, placed above the optic disc to unroll the folded retina. Endolaser was then applied around the tears and to the basal retina for 360° in all eyes. PFCL was not used in one eye in which only break was there and retina was not detached. Lensctomy was performed if the lens was subluxated. Then PFO was exchanged directly with silicone oil. Silicone oil 1000 centistokes was used for exchange. Retinal reattachment, recurrent retinal detachment, final visual outcome and complications were assessed.
Statistical analysis

Statistical analysis was performed using SPSS software. Snellen visual acuities were converted to a logarithmic scale (log MAR, i.e. the logarithm of the minimum angle of resolution). Comparisons between preoperative and postoperative visual acuities were made using the paired sample t test.

Results

Eleven eyes of 11 patients were included; nine males and two females with age ranging from 15 to 68 (mean 44 yrs). Three eyes were highly myopic (> 6 D), three had history of trauma. 6 eyes were phakic, 2 were aphakic, 2 were pseudophakic with PC IOL, one had dislocated lens.

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Myopic/Trauma</th>
<th>eye</th>
<th>Lens Status</th>
<th>Quadrant</th>
<th>BCVA</th>
<th>Post op retina</th>
<th>VA after SOR</th>
<th>Cat. Surgery with SOR</th>
<th>3 months after SOR</th>
<th>Resurg</th>
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<td>68</td>
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</table>

The upper temporal quadrant was involved in 9 patients, lower temporal quadrant in 7 patients, followed by upper nasal (5) and lower nasal quadrant (2). Retina was not detached in one patient.

Pre operative vision was 6/24 or better only in 2 patients, whereas post operatively 6 patients attained vision better than 6/24.

One patient who developed bullous keratopathy later underwent penetrating keratoplasty.
1. Best corrected visual acuity 3 months (t test)

**P value and statistical significance:**
The two-tailed P value equals 0.0165
By conventional criteria, this difference is considered to be statistically significant.

**Confidence interval:**
The mean of pre op minus post op 3 months equals 0.6400
95% confidence interval of this difference: From 0.1298 to 1.1502

**Intermediate values used in calculations:**
t = 2.6166
df = 20
standard error of difference = 0.245

<table>
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</tr>
<tr>
<td>SD</td>
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<td>0.4100</td>
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</tbody>
</table>

2. Best corrected visual acuity post silicone oil removal (t test)

**P value and statistical significance:**
The two-tailed P value equals 0.0016
By conventional criteria, this difference is considered to be very statistically significant.

**Confidence interval:**
The mean of pre op minus post SOR equals 0.9100
95% confidence interval of this difference: From 0.3900 to 1.4300

**Intermediate values used in calculations:**
t = 3.6504
df = 20
standard error of difference = 0.249

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**Discussion**

Our series of 11 patients is comparable to previously published series in terms of patient demographics (with the usual male predominance), and predisposing factors. In our study perfluorocarbon liquids were used which serve as useful intraoperative tool during vitreoretinal surgery to unfold the detached retina in GRT cases to provide counter traction and retinal stabilization during membrane peeling. In the other published study reporting prognostic factors associated with visual acuity outcomes after management of giant retinal tears using intraoperative using perfluoroperhydrophenanthrene (Vitreon), the factors significantly associated with a poor visual outcome included hypotony, macular detachment, history of cataract extraction, poor preoperative visual acuity, giant retinal tear greater than 180 degrees, and a higher grade of proliferative vitreoretinopathy. In this study with PFCL a post operative vision of 6/36 or better was achieved in 82% patients. In 92% patients retina remained attached. In the study using Vitreon, all the eyes were followed up to 6 months. 91% of retina remained attached at final follow up, and 65% showed improvement in visual acuity.

**References**

Abstract

Introduction
Organ transplantations i.e. liver transplantation, renal transplantations are done more frequently nowadays. Primary systemic disease and or postoperative immunosuppression can affect the eye.

Aim
To find the common ocular complication after organ (Liver & Kidney) transplantation.

Materials and methods
Case records of all patients who underwent organ transplantation both liver and renal transplantation in our institute from January 2004 was reviewed.

Results
Common ocular complications following organ transplantations are cataract, lid infections, central serous retinopathy, endogenous endophthalmitis and viral retinitis.

Early complications are endogenous endophthalmitis, lid infection, and cataract.

Viral infections occur after long-term immunosuppression.

Conclusion
Immunosuppression induced ocular complications are resistant to treatment and have very high ocular morbidity. Patients who are undergoing organ transplantation require thorough ophthalmic evaluation before transplantation and regular follow-up after surgery so that ocular complications can be diagnosed and treated promptly.

Introduction:
Organ transplantation is an established therapeutic modality for the treatment end stage renal liver disease. Solid organ transplantation has evolved dramatically in recent years with the advent of several potent immunosuppressive agents i.e. Cyclosporine-A, tacrolimus, mufocenolate mofetil. Organs are now transplanted with great success.

Organ transplantation may result in significant ophthalmic complications because of the underlying disease process and or long term immunosuppression.

Aim of study
To find the common ocular complication after organ (Liver & Kidney) transplantation.

Sujithra H, Anuradha S Rao, Gopal S Pillai, Natasha R

Materials and methods
It is a retrospective study. Case records of all patients who underwent organ transplantation both liver and renal transplantation in our institute from January 2004 was reviewed. All renal transplant patients underwent thorough preoperative ophtalmic evaluation. Only symptomatic patients after liver transplant had ophthalmic evaluation.

Results
Case record of 203 renal transplant patients and 35 liver transplant patients were reviewed. Age of patients who underwent renal transplant ranges from 14 to 60 years (Table-1). Maximum number of patients was in the age group of 20 to 40. 155 were male and 48 females. 181 patients had related donor transplant, 17 spousal donors and 5 cadaver transplant. In liver transplant patient age range from 7 to 69 and maximum number patients were in the age group of 41 to 60 (Table-2). 28 were males and 7 were females. 19 patients had related donor transplant, 12 spousal donor and 4 cadaver transplant. Number of patients who had ocular complications is shown in Table-3. Renal transplant patients who had ocular complications, their donor, systemic complication and systemic immunosuppressive agents are detailed in Table-4. Details of liver transplant patients are shown in Table-5.
Table 3

<table>
<thead>
<tr>
<th>Diagnosis</th>
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<th>Liver transplant</th>
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<td>Lid infection</td>
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CSCR: Central serous Chorioretinopathy. RPE: Retinal Pigment Epithelium.

Table 4

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<th>No</th>
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<td>Cyclosporine toxicity after 8 months</td>
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CSCR: Central serous chorioretinopathy, CSA: cyclosporine A, AZA: azathioprine, pred- prednisolone, MMF- mycophenolate mofetil, IL2- interlukin two blocker
Discussion:

Cataract

Corticosteroid cataract is common complication after transplantation. Cataract surgery in transplant recipients is similar to cataract surgery in the general population and has an excellent outcome. In Renal transplant patients systemic nonsteroidal anti inflammatory drugs and systemic antiglaucoma medications are avoided. Hence precaution should be taken to prevent postoperative IOP spike and intraoperative manipulation should be minimal to reduce postoperative inflammatory reaction. In Liver transplant patients bleeding time & clotting time (PT/INR) should be monitored before surgery. It is preferable to do cataract surgery under topical anesthesia so that periocular hemorrhage due to blocks can be avoided.

Central serous chorioretinopathy

CSCR is commonly seen after solid organ transplantation. Pathogenesis of CSCR after transplantation is associated with high doses of steroids, emotional stress and systemic hypertension. It has also been suggested there may be retinal pigment epithelial changes caused by choriorapillary ischemia leading serous retinal detachment. Visual prognoses in these patients are generally good. Treatment with Photocoagulation is needed in visually significant lesion, as stopping or reducing steroid is often not possible because of possibility of graft rejection. In this study four renal transplant patients had CSCR. Two patient undergone laser treatmetns the other two were managed conservatively. One liver transplant patient had CSR preoperatively. He was advised to avoid massive dose of steroid.

Cyclosporine Toxicity

Cyclosporine is a potent immunosuppressive agent. It is nephrotoxic, causes endothelial cell injury, thrombotic occlusion of microcirculation and neurological side effects i.e seizures, cortical blindness, increased intra cranial pressures, encephalopathy and cerebellar and spinal syndromes. Cyclosporine induced retinal toxic blindness has been reported. One patient in our study had multiple retinal pigment epithelial changes which may be due to cyclosporine toxicity. One of our post renal transplant patient who developed CSR also had Cyclosporine toxicity reported.

Scleritis

Two of post renal transplant patients developed scleritis at one month and three months post operative period. They were treated with topical steroids without changing systemic immunosuppressive agents. The donor for these patients being spousal and cadaveric donors respectively. Both these patients were treated with IL 2 blockers and MMF for one of them. The complications like episcleritis and scleritis and conjunctivitis are more common with OKT3 but there are no known ocular complications with MMF and IL2 blockers.

Tacrolimus Toxicity

Tacrolimus is a relatively new immunosuppressive agent and now commonly being used in liver and renal transplantations. Preclinical toxicity studies in rats showed that Tacrolimus might cause cataract due to an accumulation of sorbitol in the lens secondary to the diabetogenic effect of the drug. Nine patients developed within six months of organ transplantation. In the post Liver transplant patients 2 patients developed cataract within six months and both of them were on MMF and Tacrolimus and in post Renal transplant patients 7 patients within six months cataract of which 3 patients were on IL2 blockers and MMF. Tacrolimus may be the cause for early cataract in these patients.

Ocular infections

Ocular infection is major problems after organ transplantation. However ocular infections are relatively uncommon when compared to systemic infections. In our study three patients had lid infection and four patients had conjunctivitis. One liver transplant patient had endogenous fungal endophthalmitis (aspergillus) within three months of transplantation.
Conjunctivitis was managed with topical antibiotics. Lid infections were managed with topical and systemic antibiotics. Fungal endophthalmitis was managed with intravitreal and intravenous amphotericin B.

Common ocular infections reported are lid infections, conjunctivitis, dacryocystitis, metastatic endophthalmitis and viral infections [CMV retinitis, herpes zoster]. Fungal infections occur early after transplantation. In addition to Candida & aspergillus. Disseminated fusarium infections with secondary fungal endophthalmitis has been reported. Transplant recipients are at risk of metastatic fungal intraocular infection as a result of their immunosuppression and because of large number of invasive vascular procedures performed i.e. intravenous catheters. Viral infections occur after long-term immunosuppression.4,5

This retrospective study underestimates the incidence of ocular complication as only symptomatic patients came for ophthalmic evaluation.

Conclusion

Ocular involvement in transplant patients is often overlooked because of other grave systemic manifestation. Immunosuppression induced ocular complications are resistant to treatment and has very high ocular morbidity.

Patients who are undergoing organ transplantation require thorough ophthalmic evaluation before transplantation and regular follow-up after surgery so that ocular complications can be diagnosed and treated promptly.

References

Case of post traumatic cyclodialysis cleft refractory to treatment

Koteshwar Rao. MS, Mahesh G. MS. FRCSED

45 year old man presented with a history of loss of vision in his left eye following trauma with a wooden stick a month ago. He was treated with topical steroids by a local doctor. There was no positive systemic history. He has never used any glasses or took treatment for any ocular problem. On examination his best corrected visual acuity was 6/6, N6 in right eye and 2/60, <N36 in left eye. Left eye showed sluggishly reacting pupil with phacodonesis. Applanation tonometry showed intraocular pressure to be 10mmHg in the right eye and 0 mm Hg in the left eye respectively. Gonioscopy showed open angles with evidence of angle recession. On fundus examination the details could not be elicited because of dense vitreous hemorrhage. B-scan examination of left eye showed point like echoes in the vitreous cavity suggestive of vitreous hemorrhage with partial posterior vitreous detachment and minimal choroidal thickening.

Considering non resolving vitreous haemorrhage he underwent parsplana vitrectomy. Intraoperatively retina was attached and there were no retinal breaks or dialysis. He was put on topical steroid and antibiotic therapy and with an atropine eye drops. Post operatively fundus examination showed striae at the macula (Figure 1).

Optical coherence tomography showed mild spongiform oedema at the macula with serous macular detachment. (Figure 2)

Cryotherapy was performed to the area of cyclodialysis cleft. In spite of meticulous surgical intervention and cryopexy and medical management the intraocular pressure raised only up to 2mm Hg. UBM showed persisting cyclodialysis cleft. Visual acuity deteriorated to 3/60 due to secondary macular changes as a result of hypotonic maculopathy.

Discussion:
Aqueous humor formed by the ciliary body at approximately 2.5µl/min and with 1% turnover every minute. Most of the aqueous passes out through the trabecular meshwork as a result of pressure gradient and uveoscleral outflow. The equatorial stretch due to the indirect forces can separate the ciliary body from the sclera spur where this creating an abnormal communication between the anterior chamber and the suprachoroidal space resulting in hypotony i.e. 6mm Hg or <10% of normal IOP. Cyclodialysis is the disinsertion of longitudinal ciliary muscles from scleral spur resulting in a cleft which connects anterior chamber with suprachoroidal space. Blunt trauma can cause the transient lowering IOP and can develop hypotony many years after the initial injury. Angle recession can be seen in good gonioscopic examination.
Cleft does not bear directly on the degree of hypotony. It may be microscopic in size can be called as occult which can be missed easily. In this case the flare in the hypotonic eye confirms the normal production of aqueous from the ciliary body. Clefts can close spontaneously with dramatic rise in IOP. Ultrasound biomicroscopy (UBM) provides the best means for diagnosing cyclodialysis clefts. The management of cyclodialysis clefts requires a step-wise approach. Initially, it is of particular importance to identify the full extent and location of the cleft as in some cases more than one cleft may be present requiring a variety of nonsurgical and surgical interventions. Nonincisional interventions include the application of various lasers and cryotherapy in the vicinity of the cleft. The traditional approach of direct cyclopexy has more recently been complemented by recent reports of employing modified external plombage procedures, vitrectomy and gas assisted endotamponade. There are insufficient studies formally evaluating these techniques to be able to assess their safety and efficacy. In this case non surgical intervention by cryotherapy was not successful in closing the cyclodialysis cleft.

**Conclusion:**

Post traumatic cyclodialysis cleft can result in profound visual loss even after intervention due to failure of cleft to obliterate.

**References:**

Klaus Heimann is considered as the father of modern trauma management in the vitrectomy era. He was born in Witten, Germany on 22 June 1935. After attending medical school in Marburg, Vienna, and Munich, he received ophthalmological training in Marburg between 1962 and 1966. He moved from Marburg University to the prestigious University Eye Hospital of Cologne in 1966. At a very young age, he established his scientific and clinical basis in vitreoretinal diseases. He was educated by Edward N.D. Norton and Robert Machemer. Later he became Head of the Department of Vitreoretinal Services in University Eye Hospital, Cologne.

Prof. Klaus Heimann was internationally renowned for his surgical expertise in repairing eyes that had suffered perforating injuries or had retained foreign particles. He proved the value of vitrectomy and silicone for reconstructing the injured eye and subsequently explored the use of Daunomycin to inhibit the postoperative proliferation. His numerous research projects included functional morphology of the choroids, hereditary vitreoretinopathies, traumatology of the retina, pathophysiology of proliferative vitreoretinopathies (PVR), surgical strategies for the treatment of PVR, options for vitreous replacement, silicone oil application in complicated retinal detachment, pathophysiology and therapy of ARMD, pigment cell transplantation of retina, immunopathology of pigment epithelium, retinal apoptosis, retinal neuroprosthesis and intraocular visual aids.

Prof. Klaus Heimann served as president of the club Jules Gonin, the German ophthalmology society, and the German retinology society and as a member of numerous national and international societies. He published numerous chapters on vitreoretinal surgery in textbooks and handbooks of ophthalmology. He was a recipient of numerous international honors and awards.

He was a superb surgeon, an innovator and an editor. He possessed a lively sense of humor, common sense and honesty. Outside ophthalmology, he had an outstanding and knowledgeable expertise in classical music. He is married to Susanne and had three children.

He died on 25 June 1999 after recurrence of lymphoma.
Early predictors of traumatic glaucoma after closed globe injury - Trabecular pigmentation, widened angles and higher baseline intraocular pressure.

Ramanjit Sihota, Sunil Kumar, Viney Gupta, Tanuj Dada, Seema Kashyap, Rajpal Insan, Geetha Srinivasan.
3.4% cases develop glaucoma after ocular contusion within 6 months. This increases to 10% during 10 years after trauma. The aim of this study was to evaluate the eyes prospectively to find the risk factors leading to post traumatic glaucoma. Eyes that had sustained with closed globe injury were enrolled. Objective was to analysis clinically and by using ultrasound biomicroscopy the factors that can predict occurrence of chronic traumatic glaucoma during the six months follow-up. 121 eyes with closed globe injuries were enrolled. 92 patients fulfilled the criteria and were used for analysis. Of this, patients underwent gonioscopy and ultrasound biomicroscopy after 4 weeks of injury and they were followed up for six months. Eyes with elevated intraocular pressure more than 21 mmHg and requiring glaucoma therapy for at least three months after closed globe injury were diagnosed as traumatic glaucoma. The clinical and UBM findings in patients with glaucoma and without glaucoma were compared. Demographic profile was similar in both groups. 40 of 92 patients had persistent glaucoma for at least three months; remaining 52 patients did not have an IOP rise of more than 21 mmHg. Visual acuity less than 6/60 at initial examination, elevated mean IOP at presentation, hyphema and trabecular pigmentation more than grade 3 were more in the traumatic glaucoma group. These were of statistically significant. Relative risk of developing chronic glaucoma after closed globe injuries was 20.8 with dark trabecular pigmentation 7.5 with 360° angle recession, 6.9 with hyphema, 3.5 with lens displacement and cataract, and 2.5 with visual acuity less than 6/60. In UBM large angle opening distance at 250 microns, angle recession area, and a wider distance from sclera spur to iris root predicted chronic glaucoma. On the other hand, presence of cyclodialysis was found to protect against development of glaucoma. In conclusion, the clinical signs of increased pigmentation of angle, elevated baseline IOP, hyphema, lens displacement and angle recession of more than 180° were significantly likely to have chronic glaucoma. In UBM a wider angle and absence of cyclodialysis were likely to have traumatic glaucoma. One drawback of this study was lack of longer follow-up. This is important because glaucoma after blunt trauma has one peak less than one year and in peak after many years. In spite of this drawback, the study gives some insight into the factors predisposing to traumatic glaucoma. This may be useful in clinical evaluation.

Characteristics and outcome of work related open globe injuries

Justin M Kanoff, Angela V Turalba, Michael T Andreoli, Christopher M Andreoli.
This study was done to evaluate the characteristics and outcome of patients treated for open globe injuries sustained at work and to compare this to the patients injured outside work. A retrospective chart review of 812 consecutive patients with open globe injuries were analyzed. There were a total of 146 patients who sustained work related injuries. 98% of these people were men and average age was 35.8 years. Initial vision was worse than 20/200 in 49.3%, 56.8% had penetrating injury, 27.3% had retained IOFB and 14.4% were blunt injuries. Mechanism of injury was nails in 28.1% and other projectile in 32%. Compared to the control population, the patients injured at work were significantly likely to be male (P = 0.0001). Also younger age group were injured more frequently compared to the control group (P = 0.0016). After the initial evaluation and surgical repair the average follow-up was 281 days. The final visual acuity was 20/40 or better in 63.9%. The study demonstrates that occupational open globe injuries have a better prognosis than the non work related rupture globe. Workers are significantly more likely to have useful vision after surgical repair. Occupational injuries are more likely to be due to sharp or penetrating mechanism when compared to blunt rupture in non-work related open globe injuries. Also occupational injuries were less likely to be associated with retinal detachment. This study showed there was no significant difference in the initial time to presentation between occupational injuries and control group of non occupational open globe injuries. However, it is important to note that 17% of workers took more than 12 hours to present for medical evaluation and 10% presented more than 24 hours after injury. This has to be tackled in occupational eye injuries. Also the article highlights the need for use of safety glasses or other protective equipments. This study also shows a double peak in the time of injury during work day with most injuries occurring either before lunch or near the end of the day. In conclusion, work related injuries can cause significant morbidity in young population. Based on average patient follow-up and final visual acuity, those injured at work is slightly better than those with open globe injuries.
injuries sustained outside the work. There is an increased incidence of retained IOFB in work population emphasizing the importance of use of eye protection at work place.

Management of ocular trauma in emergency (MOTE) trial: A pilot randomized double blinded trial comparing topical amethocaine with saline in the outpatient management of corneal trauma.


Corneal abrasion with or without retained foreign body and welding flash burns are the commonest minor ocular trauma requiring outpatient management. In these patients, pain may be a problem till the cornea heals. It is unclear whether local anaesthetic drops can be safely used for topical anaesthesia in patients with minor corneal injury who are discharged from emergency department. This study was done to assess whether topical 0.4% Amethocaine is useful in this scenario. It was a pilot randomized double blind trial comparing Amethocaine with topical normal saline. 47 patients were randomized to two groups. 22 received Amethocaine and 25 received normal saline placebo after discharging from the emergency department. The outcome measures include healing of the cornea as well as subjective assessment of pain. Patients were reviewed after 48 hours.

There was significant drop out of patients after two days in this study. The primary outcome was the healing of corneal defect. Potential delay in healing was the main concern with the use of outpatient topical anaesthesia. Complete re-epithelialisation was defined as absence of fluorescein staining at two weeks. Telephonic interview was done to find secondary outcome measures which included use of oral analgesia, unscheduled medical review, visual problem or satisfaction with treatment. Result of the study showed that persisting corneal defect was slightly more in the Amethocaine group than Saline group after 48 hours. None of the patients were given any cycloplegic drugs or eye patching to relieve pain. There was no statistically significant difference between the two groups in corneal healing due to the small number. However, secondary outcome did not differ between the two groups at two weeks. Amethocaine was effective in reducing ocular pain in the first 48 hours. Although topical anaesthesia has been found to be safe and effective in the treatment of post keratectomy pain, there are no published literature on topical anaesthesia used after any uncomplicated minor trauma. The major drawback in this study was significant drop out rate and the small number. This study concludes with topical Amethocaine was an effective method to relieve ocular pain in the first 48 hours but definitely not safe due to the delay in wound healing. Another disadvantage of this study is that it is not comparing non-steroidal and inflammatory drops or cycloplegic or artificial tears which can also alleviate pain.
Corneal Transplantation

Editor: Rasik B Vajpayee
Associate Editors: Namrata Sharma, Geoffrey C Tabin, Hugh R Taylor
Published by: Jaypee – Highlights Medical Publishers, INC
Second Edition -2010
Pages 366

The first edition of “Corneal Transplantation” was published in year 2002 and techniques of corneal grafting surgery have undergone almost revolutionary changes since then. There have been many significant and exciting developments in the technical aspects of corneal transplantation as we have been progressively refining the relatively cumbersome use of penetrating keratoplasty to treat all types of corneal disease to the use of much more elegant and precise ‘Customized component’ corneal transplantation surgery. These customized lamellar corneal transplantation surgeries aim to replace only the diseased part of the cornea by selective transplantation of the appropriate corresponding healthy donor lenticule. Surgical techniques like ‘Big Bubble’ DALK, Microkeratome assisted ALTK and DSAEK have found favor with corneal surgeons over penetrating keratoplasty for corneal disorders that effect only specific layers of cornea.

The present edition of the outstanding book has aimed to include all these developments and like the first edition, this version too has been designed as a practical guide elucidating the many and varied aspects of modern corneal transplantation surgery. A conscious effort has been made to keep the format very simple and easy to follow by using a straightforward ‘How to do’ kind of approach.

The rapidly evolving techniques for endothelial transplantation, new effective methods of deep anterior lamellar keratoplasty, application of femtosecond laser techniques, limbal stem cell and buccal mucosal transplantations, keratoprosthesis, prosthetic contact lenses, modern eye bank techniques and organizations, etc. are explained lucidly and in a practical manner in this new edition. The renownedness of the multiple authors is a guarantee that this text will respond to a world wild need and it should be highly successful. This book has 6 sections with 50 chapters contributed by 69 authors.

This new edition includes a detailed description of all new techniques of lamellar corneal transplantation surgeries including some very innovative techniques like “Tuck in” lamellar keratoplasty, Suture less DSAEK Triple surgery, DMEK and “ Double Bubble” Deep Anterior lamellar keratoplasty. It also explains the various acronyms that seem to have populated modern corneal surgery. The book carries a very wide range of and sound practical advice based on the experience of the world’s leader in this field who have described their surgical techniques and other aspects of corneal transplantation surgery in a lucid and well structured manner.

A DVD of all these surgical techniques has been provided to help beginners to acquaint themselves with the state – of- the art techniques in corneal grafting surgery. This ‘User Friendly’ book would be able accomplish its main objective of simplifying and spreading the knowledge of various aspects of contemporary corneal transplantation surgery.

This book provides a comprehensive knowledge and information about the developments in corneal grafting surgery which has undergone a revolution in the recent times. World renowned keratoplasty surgeons from various developed and developing countries have come together to address complex issues in corneal transplantation in a simple and effective style. These stalwarts enlighten the readers with their experience in their surgical skills in a lucid and a practical manner. This book has full color text with colored photographs for better understanding of the text and the DVD video clips which are accompanied by real time surgeries and animations help to understand the surgical steps of different techniques.

Surgical and Medical Management of pterygium

Editors: Ashok Garg, Essam EL Tourkhy, Belquiz A Nassaralla, Sunil Moreker
Published By: Jaypee Brothers Medical Publishers (p) Ltd. St. Louis (USA)
First Edition 2009
Pages 145

Pterygium is an important clinical entity which has assumed an importance in the last one decade, as the patient is more conscious to cosmetic appearance. In the recent times, there has been advances in the surgical and medical management of Pterygium with better visual acuity and cosmetic appearance.

Patient with pterygium is a common problem in Ophthalmology. Regrowth of pterygium is not rare and reoperations are more complex and difficult. Although it is a challenging surgical problem even for the specialists of
ocular surface disease, ophthalmic plastic and reconstructive surgery and anterior segment surgery, the comprehensive ophthalmologists face and manage the condition frequently. Studies on new surgical techniques with or without flaps and grafts, treatment regimens with adjunctive chemicals bring more effective solutions.

The book Surgical and medical Management of Pterygium written by Dr. Ashok Garg and co-editors, assemble a lot of information in the field with great ambition, diligent work and effort, and shared it with other colleagues.

The tremendous work to bring this book in life, obviously necessitates a wide collaboration among ophthalmologist. However, this hard work may not be so hard for Dr. Ashok Garg, who had a wide circuit of national and international co-editors who enthusiastically contribute to ophthalmic literature. And this great enthusiasm and dedication to Ophthalmology and to the spreading of ophthalmic knowledge brought together all of these information, experience and innovations in his new book that helped ophthalmologists, ophthalmology residents, physicians and related health workers and most important of all, for the patients we all value highly.

The present book has been prepared with the aim of providing latest information about pterygium, pathophysiology, differential diagnosis and various treatment modalities in a comprehensive and easy-to-read format. Twenty-five chapters of this book provide complete insight into various parameters of pterygium. A special chapter has been added to provide details about recent advances in this field. Accompanying video DVD shows various surgical procedures of pterygium management by the international masters of this field.

This book shall help all the keen ophthalmologist worldwide to help them in decision making and sharpen their surgical skills for the better management of pterygium patients.
Identify?

Send your answers to: maheshgopalakrishnan@yahoo.com

Exciting prizes to be won

Last issues’ spot diagnosis answer is
“Late phase of normal Indocyanine green angiogram”.

Correct answer was send by only one person. Dr. Veena Viswam

Collect your prize during the KSOS Drishti 2010 in Kochi
There are many different types of needles available in ophthalmology surgery and they can be grouped into four main types according to the point configuration (i.e. the shape of the point):

a. **cutting**  
b. **reverse cutting**  
c. **taper point**  
d. **spatula**

### Needle Anatomy

<table>
<thead>
<tr>
<th>Point of configuration</th>
<th>Cutting</th>
<th>Reverse cutting</th>
<th>Taper point</th>
<th>Spatula</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagrams</strong></td>
<td><img src="image1.png" alt="Diagram" /></td>
<td><img src="image2.png" alt="Diagram" /></td>
<td><img src="image3.png" alt="Diagram" /></td>
<td><img src="image4.png" alt="Diagram" /></td>
</tr>
</tbody>
</table>
| **Configurations**     | Triangular with cutting edge at the top.  
|                        | Cuts at tips and edges of the needle. | Triangular with cutting edge at the bottom.  
|                        | Cuts at tips and edges of needle. | Round and taper to a point.  
|                        | Cuts at the tip only. | 4 or 6 sided with cutting edges on the side.  
|                        | Cuts at tip and sides parallel to the tissue plane. |
| **Properties**         | Suture canal extends superficial to path of needle tip.  
|                        | May pull out tissue during needle passage. | Suture canal extends deep to path of needle tip.  
|                        | Ideal for oculoplastic surgery as the needle allows easy passage through epidermis.  
|                        | Accidental perforation may occur with partial thickness suture such as rectus slera fixation. | Atraumatic  
|                        | Produces the smallest hole of all needles.  
|                        | Useful in iris repair. | Allow needle to split the tissue plane and avoid accidental perforation.  
|                        | Allow the needle to stay in the tissue plane.  
|                        | Most commonly used needle in the anterior segment such as squint operation. |

Double-armed prolene sutures with straight needles are also available for use in Scleral fixation of IOL.
There are three parts to a suture needles:
1. **Swage** (connection point for the suture)
2. **Body**
3. **Point**

All needles are made of stainless steel

A suture needle has 5 geometries:
1. **Length** - distance of the circumference from the swage to the point
2. **Chord length** - distance of the straight line from the swage to the point (which determines the width of the bite)
3. **Radius** - length of the line from the center of the circle
4. **Needle diameter** - measured in mils (1/1000 of an inch) and 1 mil is about 25 um, a smaller diameter needle required less force and cause less trauma during passage through the tissue
5. **Bicurve** - two radii on a needle, the radius near the point is usually shorter than the radius of the body near the swage

### Absorbable Sutures

<table>
<thead>
<tr>
<th>Materials</th>
<th>Polyglactin 910 (eg. Vicryl, Ethicon)</th>
<th>Polyglycolic acid (eg. Dexon)</th>
<th>Plain gut</th>
<th>Chromic gut</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Types</strong></td>
<td>Polyglactin 910 is made of glycolic acid and lactic acid. Coated Vicryl:polyglactin 910 coated with polyglactin 370 + calcium stearate (make the surface smoother and reduces tissue drag). Available as braided or monofilaments.</td>
<td>Dexon S: braided polyglycolic acid without coating. Dexon plus with surface lubricant Poloxamer 188.</td>
<td>Prepared from the mucosa of sheep or sub-mucosa of beef intestines.</td>
<td>Plain gut tanned with chromic salt.</td>
</tr>
<tr>
<td><strong>Tissue reaction</strong></td>
<td>Mild</td>
<td>Mild</td>
<td>Marked</td>
<td>Moderate</td>
</tr>
<tr>
<td><strong>Tensile strength</strong></td>
<td>2-3 weeks</td>
<td>2-3 weeks</td>
<td>1 week</td>
<td>2-3 weeks</td>
</tr>
<tr>
<td><strong>Other characteristics</strong></td>
<td>Hydrolytic degradation</td>
<td>Hydrolytic degradation</td>
<td>Enzymatic degradation</td>
<td>Enzymatic degradation</td>
</tr>
<tr>
<td><strong>Indications</strong></td>
<td>Commonly used in strabismus surgery and to close scleral ports after vitrectomy. Available as double armed sutures (needle at both ends).</td>
<td>Seldom used in anterior segment surgery.</td>
<td>Seldom used in anterior segment surgery.</td>
<td>Seldom used in anterior segment surgery. Used in lid surgery such as Weiss's procedure to encourage fibrosis.</td>
</tr>
</tbody>
</table>

Note: Hydrolytic degradation elicits less reaction than enzymatic degradation

**Advantages of monofilament over multifilament**:
1. Monofilament provides easy passage through the tissue whereas multifilament tend to provide tissue drag
2. The smooth surface of monofilament does not support bacterial growth whereas multifilament provide a nidus for infection.
<table>
<thead>
<tr>
<th>Materials</th>
<th>Polyamide (Nylon)</th>
<th>Silk</th>
<th>Polypropylene (eg. prolene)</th>
<th>Polyester (eg. Mersilene)</th>
</tr>
</thead>
</table>
| Types        | Monofilaments     | Types of fine silk sutures:  
Virgin silk \textit{(fibrin coated by sericin)}  
Braided silk \textit{(degummed ie sericin is removed)}  
Both are multifilaments | Monofilaments also available as multifilaments. | Monofilaments |
| Tissue reaction | Minimal | Moderate | Minimal | Minimal |
| Tensile strength | High, losing 10 to 15% of strength per year. | Moderate, lasting 3 to 6 months. | High, maintain strength for over 2 years. | High, permanent. |
| Other characteristics | Mild elasticity.  
Stiff suture ends which must be buried to avoid irritation. | Inelastic  
Suture ends soft and therefore well-tolerated. | Most elastic suture materials.  
Stiff suture ends and irritate if not buried. | Strongest monofilament and less elasticity than other monofilaments. |
| Indications  | Most widely suture for corneal wound sutures. Also used for scleral flap closure in trabeculectomy. | Rarely used to close corneal wound sutures but often used as stay sutures and bridle sutures. | Used mainly in iris repair and scleral fixation of intraocular lens. Also used in oculoplastic work and lacrimal drainage surgery and scleral repair. | Preferred by some surgeons for its low elasticity and high tensile strength. Used in lid surgery and keratoplasty (continuous suturing) and scleral repair. |