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INSTRUCTION TO AUTHORS
Off-Label Prescriptions in Ophthalmic Practice

In Ophthalmic practice, off-label prescription of approved drugs include intravitreal injection of antibiotics, use of mitomycin-C and 5 Fluorouracil, periocular injection and intraocular administration of steroids, use of immumosuppressives for uveitis, use of photodynamic therapy for non AMD lesions, intravitreal injection of Bevacizumab (Avastin), and use of tissue plasminogen activator. Off –Label prescribing also known as “unapproved use” is the physicians practice of prescribing a drug or a medical device for a purpose different from one of the indications for which the product is approved by the Food and Drug Administration (FDA).

The FDA encourages the off-label use of drugs with the implied commitment to the profession to do the necessary clinical research to gain approved labeling for the new indication. The fact that the drug is already approved bypasses the regulatory maze necessary for a physician to try these agents. While the manufacturer cannot advertise the off-label use of a drug they have the responsibility to file an Investigational New Drug Application (IND application) with the FDA. The ophthalmic community has a tremendous responsibility to demonstrate safety and efficacy of off-label drugs by producing adequate supporting data via peer reviewed publication. The treating physician should document in the chart details of the decision-making process including previous treatment and diagnostic studies, dose and lot number of the drug as well as the discharge and follow up instructions. Use a drug specific informed consent (eg:- for Avastin; a drug specific informed consent can be downloaded from www.omic.com) and discuss the off-label status of the drug, making sure that the patient has adequate time to take a decision before signing the informed consent form. Discuss clearly the off-label status, the attendant risks as well as why the FDA approved options have not been considered. Failure to provide or at least discuss off-label therapy, if it is the standard of care will definitely make you liable to a malpractice suite. A situation akin to being “between the devil and the deep sea” …………Damned if you do! And Damned if you don’t !

Extension of the use of off-label drugs in pediatric practice before its safety and efficacy has been tested may result in unwanted toxic side effects in children. Development of pediatric indications for potentially beneficial off-label drugs has led to investigations and publishing of several studies to establish safe dosing guidelines for many off-label drugs that are now considered ‘standard-of-care’ in infants and children. Publication of well documented small case series in peer reviewed journals indicate that high quality evidence can and does exist beyond federally sanctioned approval and may be used to deliver safe and effective drug as well as expunge those that may be dangerous from the market.
Use of intravitreal triamcinolone acetonide and anti VEGF, a form of anti angiogenic therapy is on the horizon for aggressive posterior retinopathy of prematurity (AP–ROP) which develops in profoundly immature neonates. The rationale of the therapy with off-label drugs is the accepted fact that VEGF promotes retinal vascularisation. The BLOCK-ROP study, a phase I trial is underway to study these challenges. Let us wait for conclusive results before experimenting on the vulnerable target population.

Dr. Meena Chakrabarti  MS DO DNB
Editor
Traumatic Optic Neuropathy [TON] - A Review

Dr. Renuka Srinivasan MS, Dr. Chaitra S. MS

Traumatic Optic Neuropathy [TON] is a form of optic neuropathy typically caused by indirect optic nerve injury resulting in dramatic impairment of visual function accompanied by an ipsilateral relative afferent pupillary defect (Marcus-Gunn pupil).

Incidence

Traumatic optic neuropathy in the United States occurs in 0.5-5 % of patients presenting with closed head trauma and in 2.5 % of patients presenting with midfacial fracture. International rates of traumatic optic neuropathy vary from country to country. Rates depend on the occurrence of causative events, such as nonfatal motor vehicle accidents and assault.

It is generally presumed that penetrating injuries of the orbit are associated with direct optic nerve injury. The incidence of TON after craniofacial trauma has been reported to be 0.5-1.5 % in older series. However, recent surveys report a higher figure; 2-5 %. This increased incidence may represent the diagnosis of subtle forms of TON closer to the time of injury. These types of cases appear to represent a smaller percentage of cases in the earlier studies on TON and may have been missed at that time. Males represent the majority afflicted with TON, comprising 60-95 % of cases. In one series focused on children, 40 % of the cases were female.

Although the degree of visual loss after indirect traumatic optic neuropathy may be quite variable, approximately 50 % of patients are left with “light perception” vision, making traumatic optic neuropathy a significant cause of permanent visual loss.

There may be associated fractures in the form of anterior orbital fracture, posterior orbital fracture, orbital blow-in or blow-out fracture.

Etiology

Traumatic optic neuropathy is associated with high momentum deceleration injuries and midfacial trauma. Loss of consciousness is associated with TON in 40-72 % of cases. Motor vehicle and bicycle accidents are the most frequent causes, accounting for 17-63 % of cases depending on the series. Other causes include impact over the frontal region by falling debris, assault, stab wounds, gun shot wounds, seemingly trivial injuries, and endoscopic sinus surgery.

Relevant Anatomy

The human orbit is pyramidal, with its base oriented anteriorly and its apex oriented posteriorly. The orbital walls converge posteriorly near the superior orbital fissure and optic foramen. The optic canal is separated from the superior orbital fissure by the optic strut of the sphenoid. The sphenoid body and lesser wing comprise the bony walls of the canal. In the adult human, the optic canal is approximately 6.5 mm in diameter and 8-10 mm in length. Although the canal is elliptical in true cross section, its walls can be described by redefining it as a base-up triangle. The canal's roof separates the optic nerve from the sub frontal space of the anterior cranial fossa.
The optic strut bridges the sphenoid lateral wing and body and comprises the optic canal’s lateral wall. This wall separates the canal from the superior orbital fissure and its contained neurovascular structures. The canal’s lateral and medial walls separate it from the sphenoid sinus and, in approximately 12% of patients, the Onodi cells of the posterior ethmoidal sinus. Structures that pass through the optic canal include the optic nerve axons, their supportive glia, the ophthalmic artery, and branches of the carotid sympathetic plexus of the autonomic nervous system.

The optic nerve is 3-4 mm in diameter and measures 35-50 mm from the retina to the optic chiasm. The nerve is composed of intraocular (~1 mm), intraorbital (20-30 mm), intracanalicular (5-11 mm), and intracranial (3-16 mm) segments. The axons comprising the nerve have their origin in the nerve fiber layer of the retina and extend beyond the chiasm and optic tracts before synapsing within the lateral geniculate body. The topographic organization of the axons, as arranged by the retina, is preserved within the optic nerve. Except for its intraocular segment, the axons of the optic nerve are myelinated. Because the optic nerve is a white matter tract of the CNS, oligodendrocytes comprise two thirds of glia and are responsible for the production of myelin. Astrocytes are also present within the glial septa and provide nutritional support to the axons.

Throughout its intraorbital and intracanalicular course, the optic nerve remains surrounded by pia, arachnoid, and dura mater (optic nerve sheath). Within the optic canal, the dura remains fused to the sphenoid periosteum and, thus, is the only segment of the nerve or its sheath that is tightly fixed in space. At the posterior foramen of the optic canal, the optic nerve sheath reflects away from the nerve to fuse with the dura lining the calvaria. Therefore, the intracranial optic nerve lies within the subarachnoidal space.

Pial branches of the internal carotid, anterior cerebral and anterior communicating arteries perfuse the intracranial optic nerve. Small pial branches from the ophthalmic artery supply the intracanalicular optic nerve. The intraorbital optic nerve is supplied by perforating branches derived from the ophthalmic artery. The arterial circle of Zinn-Haller supplies the intraocular optic nerve with contributions from the posterior ciliary arteries, the pial arterial network, and the peripapillary choroidal vasculature.

**Classification**

Optic nerve injuries can be divided into direct or indirect injuries based on type of injury.

**Indirect Optic nerve injury**

Closed head trauma leads to indirect optic nerve injury which can be classified anatomically as

1. **Anterior**: the central retinal artery enters and the central retinal vein exits the optic nerve 8-12 mm posterior to the insertion of the nerve into the globe. Injuries anterior to this site are termed anterior.

2. **Posterior**: the injury is posterior to site of entry of the central retinal artery and exit of central retinal vein.

Anterior injuries disturb the retinal circulation while posterior injuries are associated with normal retinal circulation. Typically in the latter, no immediate change is seen on fundus examination. The disc appears normal for 3-5 wks after which it becomes pale as descending optic atrophy sets in.

**Direct optic nerve injury**

Direct optic nerve injuries result from objects that penetrate the orbit and impinge on the optic nerve causing optic neuropathy by partial or complete transection of the optic nerve sheath. Hemorrhages within and around the nerve may also occur.

Gunshot wounds, sharpnel, stab wounds etc are commonly reported etiological agents.

Unlike indirect optic nerve injuries, direct injuries lead to immediate changes in the fundus which can simulate central retinal artery occlusion, central retinal vein occlusion or anterior ischemic optic neuropathy. These can be detected on ophthalmoscopic examination.

**Pathogenesis**

Direct and indirect injuries both cause mechanical and ischemic damage to the optic nerve. Sometimes the
ocular injuries may be so subtle that there may be no external evidence \(^1\). Generally, direct injuries have a prognosis that is worse than indirect TON \(^1, 2\). Two mechanisms, primary and secondary, operate resulting in such damage to the optic nerve.

**Primary: Shearing Injury**

Traumatic optic neuropathy, in its most common form, is an indirect event that occurs during or shortly after blunt trauma to the superior orbital rim, lateral orbital rim, frontal area, or cranium. The most widely held belief maintains that compression forces from the trauma are transmitted via the orbital bones to the orbital apex and optic canal \(^1, 2, 3, 4\). Laser interferometry studies demonstrate the same \(^2\). Elastic deformation of the sphenoid then allows transfer of the shearing force of deceleration to the intracanalicular segment of the optic nerve. Contusion of the intracanalicular optic nerve axons and pial microvasculature produces localized optic nerve ischemia and edema. Edema of ischemic axons results in further neural compression within the fixed-diameter bony optic canal. A positive feedback loop is precipitated which triggers development of an intracanalicular compartment syndrome \(^1, 2, 3, 4, 5\).

Focal axonal abnormality is induced, characterized by impaired axonal transport. The nerve is functionally separated into a proximal and distal fragment, within 6-24 hours after injury. The distal portion undergoes Wallerian degeneration while the proximal portion i.e. cell body undergoes apoptosis \(^4\).

Tearing injuries of the microvasculature accompany the shearing injury to the axons leading to hemorrhage in the optic nerve and its sheaths.

Thus, primary mechanisms cause permanent damage to optic nerve axons at the moment of impact \(^2, 3, 4\).

**Secondary**

Owing to disturbances of cellular homeostasis adjacent to areas of irreversible optic nerve damage, diverse and interrelated mechanisms operate which lead to loss of axons which had survived the original insult \(^4\). These mechanisms are:

1. Ischemia and reperfusion injury – partial ischemia develops due to cessation of blood flow. However reperfusion of these transiently ischemic regions leads to peroxidation of cell membrane lipids leading to generation of oxygen free radicals which cause tissue damage \(^3, 4\).
2. Bradykinin: This is activated following trauma, and it leads to release of arachidonic acid from neurons. The prostaglandins derived from arachidonic acid metabolism, free radicals and lipid peroxides lead to edema in the optic canal, which further aggravates the ischemia \(^4\).
3. Calcium ions: Following optic nerve ischemia, calcium ions enter the intracellular compartment through voltage and receptor gated channels. Increased intracellular concentration of calcium ions acts as a metabolic toxin and leads to cell death \(^3, 4\).
4. Cell mediated mechanisms: Polymorphonuclear (PMN) cells predominate in the first two days following trauma, which are then replaced by macrophages in 5-7 days. While PMNs lead to immediate damage, macrophages cause delayed tissue damage, demyelination and gliosis \(^4\).

**Clinical Assessment**

The diagnosis of traumatic optic neuropathy is clinical. Following midfacial and cranial trauma, a high index of suspicion for a traumatic optic neuropathy is to be entertained \(^1, 2, 3, 4\). Patients with traumatic optic neuropathy typically experience sudden, severe, unilateral vision loss following blunt injury to the head or face. The condition may manifest immediately or within hours or days following the trauma. Occasionally, the vision loss may be insidious, and in some cases the patient may be unaware of any visual deficit until it is detected by routine examination \(^1, 2\).

Unfortunately there are a number of limiting factors to the assessment of patients with suspected optic nerve damage, such as presence of injuries to other major organ systems, patient’s level of consciousness, ability and willingness to cooperate with the examination. Attempt should be made to obtain as much information as is possible \(^3\).

Most commonly, patients are males and in their teens or twenties. The history is varied, and often includes a
blow to the head severe enough to induce loss of consciousness or high-speed penetration of the globe by foreign material. Or the trauma may seem trivial, and the patient neurologically intact. In some cases, there is obvious evidence of injury to the orbit such as periorbital or ocular hemorrhage, ecchymosis or lacerations, in other cases there may be no evidence at all.

**History**

A comprehensive history should be obtained from the patient if stable, or from relatives, friends or eye witnesses to the injury. It is also essential to determine if the patient had any visual deficits before the trauma due to concomitant ocular disease. A medical, drug and drug allergy history should also be obtained if possible.

**Examination**

A comprehensive ophthalmic examination should be performed on all patients in whom traumatic optic neuropathy is suspected and should include the following assessments:

1. **Visual Acuity**: should be determined ideally using a Snellen’s chart or hand held near vision cards. The incidence of no light perception vision following TON varies significantly with most studies of 15 or more cases reporting an incidence that ranges from 22 % to 78 %\(^1\). About 20 % may have acuity better than 20/200. However a high index of suspicion is to be maintained to avoid missing more subtle cases of visual loss. It should be kept in mind that in less than 10 % of cases a delayed visual loss develops due to secondary optic nerve injury. Hence visual acuity should be assessed again after 24 hrs\(^5\).

2. **Relative afferent pupillary defect [RAPD]**: RAPD is elicited with the swinging flashlight test. Light that shines into a normal eye stimulates the pupil of that eye to constrict and also stimulates the pupil of the other eye to constrict consensually. There is less pupillomotor stimulation reaching the brainstem when the light shines into the eye with optic nerve injury compared to the uninjured side, so the pupillary response is diminished. This relative afferent pupillary deficit is the basis for the swinging flashlight test.\(^1,2,3,4\) This is the most important clinical sign in an unresponsive patient. Test can be very useful for detecting unilateral optic nerve injury in an unresponsive patient and in the absence of fundus findings. Deficits greater than 2.1 log units when measured with neutral density filters are predictive of poor visual prognosis\(^1\). A relative afferent pupillary defect may not be present if TON is present bilaterally. However its presence doesn’t necessarily imply little or no vision in that eye. Only when the direct reflex is absent, but consensual reflex is retained, it can be deduced that there is no light perception in that eye\(^2\).

3. **Colour vision**: The patient is asked to look at a red object, one eye at a time. The object may be perceived as black, brown, or a faded red by the affected eye\(^2,3\).

4. **Visual Fields**: Though there is no pathognomonic field defect that is diagnostic of optic nerve trauma\(^1\), fields should be assessed in an awake and cooperative patient as it provides rough information about possible location of optic nerve damage. Visual field loss following partial avulsion of the optic nerve from the globe tends to correspond with the lesion. Altitudinal defects with macular and upper field sparing, nerve fiber bundles defects, generalized constriction and depression, as well as central and paracentral scotomas are also reported\(^1\). In the absence of formal visual fields, bedside confrontational visual fields are useful in patient assessment. The patient is asked to detect hand movements or light in various regions of the visual field.

5. **Ophthalmoscopy**: Ophthalmoscopy is performed with the aid of a short-acting mydriatic agent on all stable patients. The retinal and choroidal circulation, optic nerve head morphology are evaluated. The presence of ring-shaped hemorrhage adjacent to the optic nerve head indicates a partial or complete avulsion of the optic nerve head. Anterior optic neuropathies produce disturbances in circulation leading to arterial and venous obstruction and disc swelling. Hemorrhages in the optic nerve sheath posterior to origin of central retinal vessels may spare the circulation but produce disc swelling. Bilateral disc swelling
suggests papilledema. Optic atrophy in the setting of acute head trauma with evidence of optic neuropathy indicates that some disturbance of the optic nerve was present before the trauma, and was not caused by it. Finally, damage to distal optic nerve in the orbit, optic canal, or intracranial cavity does not cause any change in appearance for about 3-5 weeks. 

6. Ocular adnexa: Examination may reveal orbital rim and wall fractures, orbital edema, proptosis or enophthalmos, or extra ocular muscle dysfunction. Signs of penetrating injuries, such as protruding foreign bodies, extruding orbital contents, or conjunctival laceration, may range from obvious to subtle.

7. Intraocular pressure: Tonometry should be performed in intact globes. Increased intraocular pressure may accompany an orbital hematoma, diffuse orbital hemorrhage, orbital emphysema, or soft tissue edema.

**Visual Evoked Potential (VEP)**

Due to difficulty in neuro-ophthalmological testing on visual pathway functioning in severely injured patients or even during craniomaxillofacial reconstruction, VEP and electroretinogram (ERG) are believed to be reliable electrophysiological methods to collect distinct information whether the visual pathway function is intact, pathological but still present or absent. VEP is also a diagnostic consideration in patients who have suspected bilateral optic nerve injury. Logistically, a neurophysiological evaluation may be complicated by the inability to transport the patient to the neurophysiology laboratory or to perform a bedside VEP. Visual recovery is unlikely when VEP results are not recordable. In unilateral cases of traumatic optic neuropathy, flash VEP amplitude ratio (affected side/normal side) greater than 0.5 appears predictive of a favourable, long-term visual outcome.

The electrophysiological evaluation along with the multiplanar CT is important for the immediate identification of optic nerve trauma. The results of this evaluation will provide diagnostic information on whether surgical intervention and/or conservative therapy are required to prevent secondary optic nerve damage.

**Imaging**

In polytraumatized patients with loss of consciousness, CT scan with clinical exploration is the most important method for the assessment of traumatic optic neuropathy in the acute emergency setting. It may reveal specific pathology compromising the optic nerve, including optic nerve sheath hematoma, fractures involving the greater or lesser wing of the sphenoid, subperiosteal hematoma, hemorrhage affecting the orbital apex, ethmoid or sphenoid sinus, and pneumoencephalus. Fractures through the optic canal can be best depicted with thin-section CT scanning (e.g., 1.5-mm cuts with 1-mm intervals). While CT scanning is clearly superior to magnetic resonance imaging (MRI) in delineating fractures of bone, MRI is superior to CT scanning for soft tissue. Often both CT and MRI are required to evaluate a given clinical situation. Magnetic resonance imaging should be deferred until a metallic orbital or intraocular foreign body has been ruled out by CT scan or conventional x-ray. Finally, CT scanning is critical for surgical planning if optic canal decompression is contemplated.

Although OCT has for the most part been used to evaluate RNFL thickness, recent software improvements have made it possible to measure macular thickness as well. The efficacy of macular thickness measurements in documenting progressive neural damage is being evaluated in a longitudinal study using OCT.

No study has yet evaluated the role of macular thickness measurements in conditions associated with acute ganglion cell loss, such as optic nerve trauma. Previous studies have documented peripapillary RNFL thickness reduction after indirect traumatic optic neuropathy.

**Management of TON: The Controversies**

The management of a patient with TON is essentially by a multi-disciplinary approach involving the ophthalmologist, physician, neuro-surgeon, and an otorhinolaryngologist. Several controversies exist concerning the management of TON. The optimum management protocol is yet to be elucidated as there is paucity of prospective large-scale clinical trials. Most widely accepted contemporary treatments for traumatic optic neuropathy include observation, steroids, and surgical decompression.
Since 1990 there have been at least 16 studies of TON with at least 10 patients per series for a total of 715 patients\textsuperscript{1}. It is difficult to compare one study with another in any meaningful way because of marked variation in how patients were collected and examined, time lag between injury and presentation, follow-up, dose of corticosteroid employed, concomitant optic canal decompression, and how patient details have been summarized and presented\textsuperscript{1}. The timing and type of decompression procedure and selected use and optimal dosing of perioperative corticosteroids have also been widely reported but have not been validated by controlled outcome trials.

The International Optic Nerve Trauma Study was organized to help clarify the value of treatment of TON. Initially, a pilot study was organized to assess recruitment feasibility for a randomized, double-blind study.

76 investigators from 16 countries were involved in the study. They were asked to complete and submit standardized data forms to the data coordination center, on all patients who were examined during the study period. There was neither a predetermined sample size, nor protocol for examination, steroid treatment, surgical management and follow-up schedule. The form was to contain history of injury, examination findings, results of CT scan if performed, description of the management [which was to be based on investigator's customary practice], and surgical complications if any. Six months from the date of injury, a second data form was to be completed providing details of all follow-up vision examinations\textsuperscript{5}.

Patients with indirect optic neuropathy only, who had a vision assessment within 3 days of injury, were to be included in the study. Among the 206 patients for whom data forms were submitted, only 133 patients qualified for inclusion in the study. Since recruitment was insufficient, the study was converted to a comparative, nonrandomized interventional study. Patients received steroid treatment alone, surgery with or without steroids, or no treatment. There was no uniformity to the administered corticosteroid treatments. The study failed to find benefit for either corticosteroid therapy or optic canal decompression. The study has limitations, but it represents the largest, most unbiased study of TON to date\textsuperscript{5}.

The Study was organized with a strong bias that treatment for TON is beneficial. All but nine out of 133 patients enrolled in the International Optic Nerve Trauma Study received surgery or corticosteroids within 7 days of treatment. The authors stated that their results and the literature on TON provide "sufficient evidence to conclude that neither corticosteroids nor optic canal surgery should be considered the standard of care for patients with traumatic optic neuropathy." They further suggest that it is appropriate to make individualized treatment decisions for a particular patient\textsuperscript{1,5}.

1. Medical

The mainstay of medical management of TON is the use of mega dose steroids, the use of which was extrapolated from the National Acute Spinal Cord Injury Study 2 [NASCIS II], a multicenter clinical trial that evaluated patients with acute spinal cord injury. In this study, patients were treated with placebo, methylprednisolone [MP], or naloxone\textsuperscript{3,4,5,7,8}. Pharmacologically, high-dose or mega-dose methylprednisolone therapy is associated with stabilization of the microvascular circulation and calcium homeostasis\textsuperscript{4}. The study showed that methylprednisolone (30 mg/kg loading dose, followed by 5.4 mg/kg/h for 24 h) started within 8 hours of injury was associated with a significant improvement in both motor and sensory function compared to patients treated with a placebo\textsuperscript{5}. Whether methylprednisolone therapy is similarly effective in the treatment of traumatic optic neuropathy is unproven\textsuperscript{1,4,5}. Moreover, analysis of both the NASCIS II and the more recent NASCIS III has raised questions regarding the statistical assumptions made by these studies. The benefit seen in NASCIS II may be a statistical artifact\textsuperscript{1}.

2. Surgical

Surgical decompression of the optic canal and the intracanalicular optic nerve sheath has been advocated for the management of indirect traumatic optic neuropathy. However there is no consensus on the role as well as the optimum timing of the surgical intervention. As it is thought that increased intracanalicular pressure causes vascular compromise with ischemia and interruption of neuro-feedback mechanisms leading to blindness, optic nerve decompression theoretically relieves annular
strangulation and reestablishes nerve function. Based on this theory, approaches to the optic nerve have been devised since traumatic optic neuropathy was first diagnosed. These procedures are complimentary to steroids, which reduce inflammation and edema and are widely used to treat traumatic visual loss. Based on this study, approaches to the optic nerve have been devised since traumatic optic neuropathy was first diagnosed. These procedures are complimentary to steroids, which reduce inflammation and edema and are widely used to treat traumatic visual loss. Based on this study, approaches to the optic nerve have been devised since traumatic optic neuropathy was first diagnosed. These procedures are complimentary to steroids, which reduce inflammation and edema and are widely used to treat traumatic visual loss.

Criteria for adequate surgical decompression:

1. Removal of at least 50% of the circumference of the osseous canal.
2. Removal of bone along the entire length of the canal.
3. Total longitudinal incision of the dural sheath including the annulus of Zinn.

Various surgical approaches for decompression of the optic canal include trans-frontal craniotomy, extra-nasal trans-ethmoidal, trans-nasal trans-ethmoidal, lateral facial, sublabial and endoscopic approaches. The following is a brief description of the most commonly used approaches:

**Extra-nasal trans-ethmoidal**

This extra-cranial approach is the most popular approach and avoids the necessity of lifting the frontal lobe of the brain. However, since the optic nerve and the carotid artery are closely associated in the sphenoid sinus, damage to the carotid artery can occur during surgery.

**Endoscopic**

The endoscopic approach is based on the fact that the optic canals almost always protrude into the sphenoid sinus wall. The procedure is performed using 0° and 30° nasal telescopes with the patients under general anesthesia. After ethmoidectomy and sphenoidotomy, the bulge caused by the internal carotid artery and optic nerve is identified in the lateral wall of the sphenoidal sinus. The medial wall of the optic canal is thinned out with a micro-drill and removed with a microcurette. The annulus of Zinn and the optic nerve sheath are not incised.

The endoscopic technique should not be used in rare cases of conchal pneumatization. Optic nerve decompression with minimal morbidity and marked recovery of vision following the procedure has been claimed.

**Tran nasal trans-sphenoidal**

All cases are operated under general anesthesia with hypotension and with 15° head end elevation. The nasal cavity is decongested using xylocaine with adrenaline in a concentration of 1:1, 00,000. The middle turbinate is medialized and bulla opened, ground lamella is then entered, posterior ethmoidal and sphenoid sinus are entered sequentially. The sinus ostium is widened in the inferomedial direction and lateral wall bone is removed in the region of the optic canal in whole of the length of canal. The nerve is decompressed in the whole segment of the canal and then medicated pack is kept in nasal cavity.

**TON in children**

The vision loss caused by TON can be partial or complete, temporary or permanent. The management protocol is better defined in the adult population but its safety and efficacy is not yet established in the pediatric population. Early intervention has been recommended based on the difference in anatomy of the optic canal in adults and children. In adults, the optic canal is approximately 6.5 mm in diameter and 8–10 mm in length with the optic nerve having a diameter of 3–4 mm with a volume of around 1 ml. Structures that pass through the optic canal include the optic nerve axons, their supportive glia, the opthalmic artery, and branches of the carotid sympathetic plexus of the autonomic nervous system. Gupta et al opined that, in children, as the optic nerve canal is smaller, a lesser volume is available for the nerve to expand, thus early visual impairment could occur following trauma. The neuronal degeneration hence will occur if intervention not carried out early during the course of injury. Thus, they recommended early intervention in cases of traumatic optic neuropathy in children rather than waiting for spontaneous recovery and also opined that combined use of surgery and steroids might help around 80% children to regain vision.

The surgical technique used by Gupta et al was trans-nasal trans-sphenoidal optic nerve decompression, which was claimed to be minimally invasive and provided better surgical visualization, reduced hospitalization and avoided the morbidity associated with intracranial approaches.
Surgical treatment of optic nerve sheath hematoma and orbital hemorrhage

An optic nerve sheath hematoma can be evacuated by a medial or lateral orbitotomy depending on the location of the hematoma\textsuperscript{1, 4}.

In cases of orbital hemorrhage, a lateral canthotomy and cantholysis is done to permit expansion of orbital contents. This is followed by orbital imaging to look for subperiosteal hemorrhage or any other causes of visual loss. In case there is no visual improvement, an orbital decompression is to be considered. Systemic steroids are not used in this setting.

Some untreated cases of TON have been observed to improve even when the initial vision is no light perception. This rate of spontaneous visual improvement means that patients with TON may show improvement irrespective of whether treatment is instituted or not. The challenge comes in demonstrating that a particular intervention causes a rate of visual improvement different from the spontaneous rate of visual improvement. Due to the study variations mentioned earlier, older studies that were more observational and less interventional are unsuitable as controls for the more recent interventional studies\textsuperscript{1}.

Thus, a practical approach to management of TON can be put forth as follows\textsuperscript{1-11}:

1. In absence of any contraindications, the patient should be treated with systemic steroids: Methylprednisolone 30mg/kg as loading dose, 5.4mg/kg/hr maintenance thereafter for 48 hrs.
2. Failure to improve dictates a rapid taper and discontinuation.
3. Patients who improve can be switched over to a gradual tapering dose.
4. If the patient relapses when corticosteroids are discontinued, surgical decompression is to be considered.
5. In general, patients with visual acuity of 20/40 or worse can be taken up for surgical decompression.
6. Unconscious patients should not undergo decompressive surgery unless it is incidental to another operative procedure.
7. Two pronged approach with steroids and early surgical intervention may be considered in children.

Follow-Up

Recovery of visual function following traumatic optic neuropathy can be objectively defined by using serial assessment of multiple visual function parameters (e.g., visual acuity, visual field, quantification of afferent pupillary defect, assessment of abnormal color vision). Daily follow-up evaluations must be performed during the acute phase following trauma, immediately after surgical therapy, and during the period of mega-dose corticosteroid therapy. Less frequent examinations (i.e., every 4-7 days) are warranted during the intermediate period following trauma, surgery, or discontinuation of steroid therapy. Long-term observation is appropriate at a point 3 months or more from the date of injury to document the final level of visual function.

Prognosis

On the basis of several studies, the following four variables were considered to be poor prognostic factors for recovery of visual function\textsuperscript{1, 4, 10, 12}.

1. Presence of blood within the posterior ethmoidal cells
2. Age over 40 years
3. Loss of consciousness associated with traumatic optic neuropathy, and

Apart from these, posterior orbital fractures were found to be associated with a worse visual outcome than anterior fractures\textsuperscript{6}.

Recovery documented at the first follow up visit after treatment was significantly associated with recovery at the last follow up visit. These four negative prognostic signs in patients affected by traumatic optic neuropathy may be useful in predicting the visual outcome in patients developing visual loss after head trauma and in deciding on the need for surgical treatment. However recovery of vision can occur with or without treatment.
Conclusion

Traumatic Optic Neuropathy is a rare though important cause of visual morbidity in patients with closed head injuries and maxillofacial trauma. A high index of clinical suspicion, thorough clinical examination and radiological investigations performed at the earliest, aid in arriving at the diagnosis. Management is by a multidisciplinary approach, the options being observation, mega-dose corticosteroids [Methylprednisolone] or surgical decompression of the optic nerve. A consensus is yet to be established on the optimum management protocol for this condition, owing to the lack of large scale clinical trials. The prognosis is variable, though generally poor.

References

Risk Factors and Treatment Outcome After Laser Photocoagulation for Retinopathy of Prematurity

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Abstracts

Objective: To determine the risk factors and treatment outcome of threshold and prethreshold type A ROP in pre term babies during 2002-2007

Method: Retrospective chart analysis of pre term babies in two major neonatology centers.

Results: 116 eyes of 59 babies with threshold and prethreshold type A ROP were treated with laser using indirect ophthalmoscopic delivery. Favourable outcome was seen in 97 eyes (83.6%). 7 eyes progressed to blindness in spite of treatment. 4 eyes developed Stage 4 ROP with retinal detachment and underwent scleral buckling. All of them had attached retina after 1 year. 8 eyes had developed falciform retinal fold involving the macula.

Conclusions: ROP screening is essential in the present scenario and timely intervention can prevent blindness in a substantial proportion of patients with prethreshold ROP.

Key Words: Retinopathy of prematurity (ROP), Threshold, Prethreshold, Laser photocoagulation, retinal detachment, falciform fold, scleral buckling. ETROP, ICROP

Introduction

Retinopathy of Prematurity (ROP) is fast becoming a major cause of preventable blindness in the new born. The increase in the magnitude is due to advances in the neonatal care as well as increased awareness. In Kerala also the incidence of ROP is increasing. There are no specific data from this part of the world regarding the incidence and pattern of ROP. In the past decade there is increase in the survival of micro premature, extremely low birth weight babies. 50 % of very low birth weight (VLBW) survivors require special attention in academics and learning. 30 % of survivors among VLBW babies have some neuro sensory problems like ROP. So the economic burden of this problem can be immense. Here we present an interventional retrospective case series of ROP with special emphasis on the structural outcome after laser photocoagulation.

Patients and Methods

This was a retrospective interventional study, which was conducted between 2002-2007. It included 2 major
neonatology centers in Cochin. All the 3000 babies born preterm with a birth weight of 1500 grams or less were screened by AM and LP in the neonatal intensive care units. If the babies were discharged, they were examined weekly in out-patient units. The first examination was done after 4 weeks of birth or at 34 weeks post conception age whichever was earlier. Pupils were dilated by using specially prepared dilating drops containing Tropicamide with 2% Phenyl Ephrine. One drop was applied every 5 minutes for 3 times and indirect ophthalmoscopy was done with the help of Alphonso speculum and topical paracaine. If the baby showed any evidence of threshold, prethreshold or progressing plus disease, MG and AG were informed. Those approaching threshold or prethreshold were given laser photocoagulation using Laser Indirect Ophthalmoscopic delivery system. Diode laser was used with a power of 200-500 mW. A gray white burn was the end point (Flow chart 1). The entire avascular retina was treated using confluent burns. Following laser the baby was reviewed after 1 week. Additional laser was given in cases not responding to laser. Babies were followed up till retina was stable. There after examination was done at 3 months, 6 months, 1 year and then every year.

Flow Chart 1 Demonstrating the screening and treatment schedules

<table>
<thead>
<tr>
<th>Screening by AM &amp; LP 3000 babies during 2002-2007</th>
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<tr>
<td>Referred Threshold or prethreshold</td>
</tr>
<tr>
<td>Laser for 116 eyes of 59 babies</td>
</tr>
</tbody>
</table>

Primary outcome measure was structural outcome. It was divided into favourable and unfavorable outcomes. Unfavorable outcome was defined as per ETROP guidelines as (1) posterior retinal fold involving macula. (2) Retinal detachment involving macula. (3) Fibrous tissue obscuring the view of posterior pole. If there was complete or partial retinal detachment during the follow up visit, scleral buckling was done under general anaesthesia for stage IV, A and B ROP as per ICROP classification. Encircling band (# 240) was placed at equator by making a scleral tunnel in the 4 quadrants. Band was tied in the superotemporal quadrant. Subretinal fluid drainage was not attempted unless there was a rhegmatogenous element. For drainage, diode endolaser probe was used to perforate choroid after making a scleral pocket with choroidal knuckle. The child was followed up every month for 3 months and then every 3 months. Band cutting was done after 1 year to facilitate normal growth of the eye.

Statistical analysis was done using SPSS version 11.0 (Chicago INC).

Results

A total of nearly 3000 babies were screened by AM and LP. Of these 116 eyes of 59 babies fulfilled the criteria of threshold or prethreshold ROP and were included in the study.

Laser treatment was given in (1) Zone I and Zone II stage 3 ROP with 5 contiguous or 8 cumulative clock hours involvement with plus disease. (threshold as per the cryo ROP guideline). (2) Type I ROP defined by ETROP as Zone I any stage with plus, Zone I stage 3 without plus and Zone II stage 2 or 3 with plus.

Laser photocoagulation was performed under topical anaesthesia in NICU using with 28D lens and with vectis for scleral depression and Alphonso speculum. If the end of vascularisation was seen along with optic disc in a single field it was Zone I. If there was involvement of nasal periphery it was Zone II and others were Zone III. Plus disease was defined as vascular dilatation and tortuosity in at least 2 quadrants as defined by ETROP study. Signs of regression included lessening of plus and regression of vascular fronds with fibrous tissue.

116 eyes of 59 babies received treatment for both threshold and type A prethreshold as per ETROP were treated depending on the stage at presentation. The birth weight of babies ranged from 720 gram to 1900 grams with a mean weight of 1160 grams. The minimum follow up was 3 months and a maximum follow up was 66 Months (Mean 9.1 months). Gestational age was between 25 weeks to 34 weeks.

At the last visit, favourable outcome was seen in 97 eyes (83.6%). 4 eyes developed stage 4 retinal
detachment and underwent scleral buckling surgery under general anaesthesia followed by band cutting after 1 year. They had attached retina at the last follow up. 8 cases had falciform fold at the macula. This usually results from reattached tractional retinal detachment. 7 eyes progressed to stage 5 ROP. One patient underwent lensectomy vitrectomy surgery, but failed to reattach the retina. The following chart shows the results.

Statistical analysis showed the outcome to be significant (Wilcoxon signed rank test p 0.001)

**Discussion**

Despite major advances in the management of severe retinopathy of prematurity (ROP), retinal detachment and reduced visual acuity from ROP continue to be a major disability occurring in preterm infants and is one of the most common causes of severe visual impairment in childhood. The Multicenter Trial of Cryotherapy for Retinopathy of Prematurity (CRYO-ROP), the largest prospective trial of retinal ablative therapy for ROP, showed that 44.4 % of eyes with a history of severe ROP that were treated with cryotherapy had a visual acuity of 20/200 or worse when children were tested at age 10 years. In children whose treated eye had a visual acuity better than 20/200, only 45.4 % had a visual acuity of 20/40 or better. As a consequence, those involved in the care of infants with ROP have endeavored to find more effective approaches to treatment. One clinical trial, the Supplemental Therapeutic Oxygen for Prethreshold Retinopathy of Prematurity (STOP-ROP) study, showed no significant benefit to the use of supplemental oxygen therapy offered at a defined prethreshold point in the disease course. Another clinical trial, the Light Reduction in Retinopathy of Prematurity (LIGHT-ROP) study showed no benefit to preterm infants from a reduction in light exposure from birth to postmenstrual age 32 weeks.

In the CRYO-ROP study, peripheral retinal ablation was performed when the ocular findings indicated a risk of approximately 50 % for retinal detachment. This degree of severity was termed the threshold for treatment of ROP and was defined as at least 5 contiguous or 8 cumulative sectors (clock hours) of stage 3 ROP in zone I or II in the presence of plus disease (a degree of dilatation and tortuosity of the posterior retinal blood vessels meeting or exceeding that of a standard photograph). During the past several years, the timing for treatment of ROP have been questioned, with some physicians advocating earlier treatment and others recommending later initiation of treatment. A concern with earlier treatment is the expected increase in surgical intervention in eyes with ROP that would otherwise regress spontaneously. This concern has led to efforts to identify treatment selection criteria that will result in earlier treatment only in those eyes at highest risk for developing threshold ROP or an unfavorable visual or structural outcome in the absence of treatment.

In 1999, the National Eye Institute, Bethesda funded a cooperative agreement to study early treatment for ROP (Early Treatment for Retinopathy of Prematurity (ETROP) study). In the study, eyes of infants were randomized to early peripheral retinal ablation or standard (conventional) management if they developed prethreshold ROP and if RM-ROP2, a risk analysis program based on natural history data from the CRYO-ROP study, indicated a high risk of an unfavorable outcome. Prethreshold ROP was defined as zone I, any stage ROP that was less than threshold; zone II, stage 2 ROP with plus disease (Dilatation and tortuosity of posterior pole retinal vessels in at least 2 quadrants, meeting or exceeding that of a standard photograph); zone II, stage 3 ROP without plus disease; or zone II, stage 3 ROP with plus disease but fewer than 5 contiguous or 8 cumulative clock hours. The results of ETROP have set some new guidelines for the management of ROP. Treating at type A prethreshold
stage can reduce the unfavorable outcome from 15.6% in the conventional group to 9.1%. Our results are closer to the conventional group (Table: 1)

<table>
<thead>
<tr>
<th></th>
<th>Cryo ROP 1 year</th>
<th>Cryo ROP 10 years</th>
<th>ETROP Conventional treatment (Threshold)</th>
<th>ETROP Prethreshold treatment</th>
<th>Our Study</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25.7 %</td>
<td>27.2 %</td>
<td>15.6 %</td>
<td>9.1 %</td>
<td>16.4 %</td>
</tr>
</tbody>
</table>

### Conclusion

Screening and timely intervention in ROP can reduce the unfavourable outcome and blindness significantly.

### References


Glaucine Masqueraders – Our Clinical Experience – Has OCT Made Diagnosis Easier?

Prof. Meenakshi Dhar MS, Dr. Abhijeet S. Khake, Dr. Gopal S. Pillai MD DNB, Dr. H. Sujithra DO, Dr. S. Jisha, MS, Dr. Deepa PA.

Abstract

Conditions mimicking glaucoma with field changes or optic nerve head changes similar to glaucoma can puzzle the clinician. We present a series of 17 patients with optic nerve head pit, optic nerve head drusen, ONH coloboma, ocular ischemic syndrome, compressive optic neuropathy, and ocular hypertensives with high cup disc ratio in myopic optic discs which appear glaucomatous but with no field loss. OCT helped us to differentiate some of these. The clinical presentation, visual field analysis, OCT picture and also features clinching the diagnosis in these patients is discussed in detail.

Keywords: Optic pit, compressive optic neuropathy, Ocular ischaemic syndrome, OCT

Introduction

The diagnosis of primary open angle glaucoma (POAG) implies a life long disease with irreversible visual loss for which regular medication and followup is needed for the rest of the life. The visual loss is painless and progressive, and thus may go undetected as it does not involve the central vision initially. With early detection techniques we can halt and prevent visual loss due to glaucoma by appropriate and timely management. At the same time it must be differentiated from other causes which present similarly but with subtle differences in clinical presentation where both the prognosis and management differ.

The ‘glaucoma masqueraders’ mimick glaucoma in one or more ways. They may cause a painless visual loss, that involves the periphery earlier and more severely than central vision involvement, or some may have high intraocular pressure (IOP) e.g. Compressive optic neuropathy (CON), Ocular hypertension (OHT). Others may have optic nerve head (ONH) changes similar to glaucomatous optic neuropathy (e.g. Optic pit and isolated ONH coloboma). Still others may have visual field defects simulating or mimicking glaucoma. Superior arcuate scotoma may be seen in optic pit, anterior ischemic optic neuropathy (AION), compressive optic neuropathy. Glaucoma field loss affects the nasal quadrant initially and may mask an unusual visual field presentation of a pituitary tumor. Differentiating a case of normotensive glaucoma from OHT can be a diagnostic dilemma.
Materials and Methods

This is a nonrandomised, noncomparative, institution based, clinical observational case series study of 17 cases selected from the out-patient department from December 2004 to 2007 June.

Those patients who had at least 1-2 features of POAG, either a high IOP or an enlarged cup, or an arcuate scotoma or had a suspicious small disc with pallor were included.

All patients underwent a complete glaucoma evaluation including applanation tonometry, pachymetry, gonioscopy, automated perimetry with 30-2 on Humphrey, and a dilated stereoscopic optic nerve head evaluation.

OCT was done for all patients using Fast RNFL, Fast optic nerve head scan and Macular protocol on the Stratus OCT Version 4 machine.

Observations

The fundus findings, the HFA visual field, and OCT findings were noted and correlated for all the patients included in the study.

Table 1: Conditions mimicking glaucoma

<table>
<thead>
<tr>
<th>Condition</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optic pit</td>
<td>6</td>
</tr>
<tr>
<td>Optic Coloboma</td>
<td>1</td>
</tr>
<tr>
<td>Ocular Ischemic Syndrome (OIS)</td>
<td>2</td>
</tr>
<tr>
<td>Compressive Optic Neuropathy (CON)</td>
<td>3</td>
</tr>
<tr>
<td>AION</td>
<td>1</td>
</tr>
<tr>
<td>Orbital apex syndrome</td>
<td>1</td>
</tr>
<tr>
<td>Myopia and OHT</td>
<td>1</td>
</tr>
<tr>
<td>Optic nerve head drusen</td>
<td>1</td>
</tr>
<tr>
<td>Optic neuritis/ Angle closure glaucoma</td>
<td>1</td>
</tr>
</tbody>
</table>

Of the 17 patients (Table 1), 6 had optic nerve head pit-3 unilateral and 3 bilateral. All the 6 patients with optic pit had an arcuate scotoma (Fig. 1b)

Fig. 1. (a) Fundus photo of a case of Optic Pit

Fig. 1. (b) HFA 30-2 of a case of Optic Pit
with normal IOP which did not progress in the 12 months of followup. Central vision was decreased in 3 patients and three had bilateral pits. Pits were seen on the temporal aspect of the disc. The optic nerve head showed characteristic features of an optic pit (Fig. 1a). On OCT the pits could be seen on the temporal aspect of the cup on a horizontal line scan. There was RNFL loss around the disc (Fig. 1c, d, e) and normal optic nerve head parameters were found in all of our patients. One case has cystoid changes and schisis cavity formation in the peripapillary retina. The serous macular detachment on OCT communicated with the optic pit.

Two, had Ocular Ischemic syndrome (OIS) and three had Compressive optic neuropathy. One each had optic nerve head drusen, Optic nerve coloboma, AION, orbital apex syndrome, misdiagnosed optic neuritis.

The patient with Optic nerve head coloboma had an isolated coloboma with no anterior coloboma (Fig. 2a). The patient had a superior arcuate scotoma (Fig. 2c) with a suspicious looking disc and a normal intraocular pressure. There was a white excavation in the disc which was decentered inferiorly, with a thin inferior rim and a normal superior rim (Fig. 2a and b). OCT in this case showed inferior retinal fibre layer (RNFL) thinning (Fig. 2d) and signs of a connection

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Right Eye</th>
<th>Left Eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient 1</td>
<td>Optic pit</td>
<td>6/6</td>
</tr>
<tr>
<td>Patient 2</td>
<td>Optic pit</td>
<td>5/60 ph</td>
</tr>
<tr>
<td>Patient 3</td>
<td>Optic pit</td>
<td>6/6</td>
</tr>
<tr>
<td>Patient 4</td>
<td>Optic pit</td>
<td>5/6-60.6/36</td>
</tr>
<tr>
<td>Patient 5</td>
<td>Optic pit</td>
<td>6/6</td>
</tr>
<tr>
<td>Patient 6</td>
<td>Optic pit</td>
<td>6/6</td>
</tr>
<tr>
<td>Patient 7</td>
<td>Optic pit</td>
<td>6/6</td>
</tr>
</tbody>
</table>

Table 2: BCVA of patients of Optic Pit

Fig. 1. (c,d,e) OCT of a case of Optic Pit showing (c)–RNFL, (d)–ONH, (e)–line scan[clockwise]
between the perineural space and the inner retinal layers on the temporal optic disc border (Fig. 2e), as well as schisis-like changes extending from the disc to the macula, with cystoid degeneration and two lamellar holes. There was increased retinal nerve fibre layer thickness and reduced macular thickness.

The patient with Optic nerve head drusen had bilateral visible superotemporal drusen that lead to bilateral inferior arcuate scotoma. The drusen unless seen stereoscopically could be mistaken for notching of the cup. On stereoscopic examination it presented as a raised lesion. On OCT both eyes showed a superotemporal thinning on RNFL scan, and all parameters on ONH analysis were raised. (Fig. 3 a, b and c).

The two patients with Ocular Ischaemic syndrome had a high CD ratio, with a deep cup and extensive field loss with normal IOP’s. On OCT patient had RNFL loss, and decreased ONH parameters like VIRA, HIRW and rim area$^2$

History and Carotid Doppler helped clinch the diagnosis of Carotid artery stenosis (Fig. 4 a and b).

Three patients with Compressive optic neuropathy (Fig. 5) in thyroid exophthalmopathy and one with
Orbital apex syndrome presented with raised IOP, arcuate scotomas with ONH pallor associated with proptosis. The patients were managed on systemic steroids – (Methylprednisolone in severe cases) along with IOP lowering agents that decrease aqueous production.

On OCT in our patients all ONH parameters were decreased with inferior RNFL thinning with corresponding field changes (Fig. 5b & c). One had a double arcuate scotoma inferiorly more than superiorly (Fig. 5d).

The AION patient [Fig. 6a, b & c] had a small pale disc with a small cup, indicating a resolved neuropathy with resorption of the peripapillary oedema.

The field defect was an arcuate scotoma and IOP was normal. In the acute phase the gross elevation of the disc was clearly evident on OCT and RNFL was normal.

Also one had high intraocular pressure, and high cup disc ratio in a myopic optic discs which appeared glaucomatous. This patient had no field loss even on followup and normal OCT parameters. This patient had ocular hypertension with a borderline thick cornea, open angles, no field defects and normal OCT. The patient is on follow-up regularly and medication has not been started for last 1 year (Fig. 7).

Incomplete and partial Binasal hemianopia was seen in one patient. It appeared like glaucomatous optic neuropathy as the CD ratio was high. The IOP was normal, and had been mistakenly treated as bilateral POAG. After pituitary surgery on the adenoma, the field
in this patient neither RNFL loss nor ONH abnormalities were seen on OCT.

**Discussion**

**Optic Pit** The visual field defects in an optic pit is a superior arcuate scotoma similar to glaucoma, but it is nonprogressive, and management does not include an IOP lowering agent. The danger to visual loss is due to neurosensory detachment or retinoschisis. It is only the discerning trained eye of a clinician, that picks up the optic pit on stereoscopic fundus examination rather than diagnose it as optic cupping. OCT has made the diagnosis easier, as on OCT the pit is clearly visible on the temporal aspect of the cup on a horizontal line scan, and so is the associated neurosensory detachment. RNFL and optic nerve head parameters are normal. Optic pit is one condition where OCT helps in differentiating it from glaucoma. According to literature 15 % have bilateral disease, while three out of six of our patients had bilateral disease. The cystoid changes and schisis cavity formation in the peripapillary retina can be seen. The serous macular detachment on OCT can be seen communicating with the optic pit.
Optic Nerve head coloboma when isolated, i.e. with no anterior or retinal coloboma, presents with a superior arcuate scotoma in a suspicious looking disc in a patient with normal intraocular pressure. It is caused by the failure of complete closure of the proximal end of the embryonic fissure. It is characterized by a white excavation in the disc which is decentered inferiorly. The inferior rim is usually thin or absent whereas the superior rim is relatively normal.

OCT showed signs of a connection between the perineural space and the inner retinal layers on the temporal optic disc border, as well as schisis-like changes extending from the disc to the macula, with cystoid degeneration and two lamellar holes in their nasal portion. There is

Fig. 4. (a) Fundus findings in a case of Ocular Ischemic Syndrome (OIS)

Fig. 4. (b) OCT findings in OIS
increased retinal nerve fibre layer thickness and reduced macular thickness.

**Optic Nerve Head Drusen (OND)**

Optic disc drusen occurs in about 1% of the population and are found more frequently in Caucasians. 75% are bilateral. Inherited or sporadic, it is a form of calcific degeneration in some of the axons of the optic nerve.

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**Fig. 5. (a) Fundus findings in compressive optic neuropathy (CON)**

**Fig. 5. (b) OCT findings in thyroid ophthalmopathy causing CON**

**Fig. 5. (c) Corresponding field defects in the same patient**
Visual acuity is often not affected, but the visual fields of these patients can be abnormal and deteriorate over time. The drusen may be buried or evident. The disc picture shows an elevated disc that may be confused with papilledema (Fig. 3a). The lesion may be progressive. Buried drusens are less symptomatic. There is no existing treatment for optic nerve head drusen. Proper diagnosis and patient education is the best-available modality of care. Patients need to be aware of potential complications which, while rare, can affect vision. Visual-field testing can aid in monitoring for subtle changes in vision.

OCT has shown a thinning of peripapillary retinal nerve fiber layer. The lowest values were found in the superior and inferior quadrants. Calcification, picked up both on USG B scan and CT scan, is the characteristic feature for diagnosis for ONH drusen.

Visual field defects are uncommon in eyes with buried OND. Eyes with buried OND may have focal RNFL defects but have normal average RNFL thickness.

Coexistence of ONH drusen and POAG in eyes can be most effectively picked up by OCT earliest where the disc may appear elevated on examination but RNFL thinning may be picked up even prior to appearance of visual field defects.
In Ocular Ischemic syndrome OCT did not provide any corroborative evidence, rather, the OCT changes were indistinguishable from glaucoma. The clinical picture is hard to distinguish from normotensive glaucoma. The ONH picture was confusing and history and Carotid Doppler helped clinch the diagnosis of carotid artery stenosis.

Compressive optic neuropathy in thyroid exophthalmopathy or Orbital apex syndrome can present with raised IOP, arcuate scotomas with ONH pallor in a patient of proptosis with or without thyroid disease. The treatment is with systemic steroids, methylprednisolone in severe cases along with IOP lowering agents that decrease aqueous production. Episceral venous pressure is raised in these cases, thus medication that acts on the outflow mechanism may not work.

On OCT in our patient with CON all ONH parameters were decreased with inferior RNFL thinning. He had a double arcuate scotoma inferiorly more than superiorly (Fig. 9). Eyes with CON had significantly larger rim area and smaller cup parameters but similar RNFL thickness compared with controls on Heidelberg Tomograph but no study has been reported using OCT. However, caution has been advised by experts while interpreting the parameters obtained from the eyes with CON. One patient with bilateral field defects & severe proptosis was found to have improvement in visual fields and ONH parameters after radiotherapy.

AION patients have a small pale disc with a small cup especially in a longstanding one where the associated peripapillary edema has subsided.

The field defect can be an arcuate scotoma and IOP is normal. In the acute phase the gross elevation of the disc is clearly evident on OCT and RNFL is normal. Disc topography of eyes with AION was quantitatively
characterized by small and shallow cupping and a relatively large rim area compared to eyes with OAG matched for age and VFD. In eyes with AION, significant correlation with VFD was found only for the RNFL thickness evaluated with SLP but not for the HRT II parameters.

**Conclusion**

OCT can help in confirming the diagnosis in certain cases, and prevent overdiagnosis of POAG in clinical settings similar to glaucoma. Thus, patients of optic nerve head pit, coloboma, drusen, CON, AION, Ocular Ischemic Syndrome and ocular hypertensives can be managed better. OCT has the advantage vis a vis GDX that apart from the glaucoma evaluation, the retina can be simultaneously evaluated. CON and OIS have indistinguishable findings from glaucoma on OCT. The OCT helps in distinguishing optic pit, ONH coloboma, ONH drusen and AION.

**References**


Intravitreal Bevacizumab (IVB) In The Management of Recalcitrant Neovascular Glaucoma (NVG)

Dr. Sonia Rani John DNB, Dr. Meena Chakrabarti MS DO DNB, Dr. Arup Chakrabarti MS DO, Dr. Valsa Stephen MS DO DNB

Abstract

Aim: To evaluate the efficacy of intravitreal Bevacizumab (IVB) in refractory neovascular glaucoma.

Materials and Methods: Prospective non randomized interventional study. 15 consecutive patients with neovascular glaucoma, with refractory symptomatic elevation of intraocular pressure and pronounced anterior segment congestion received a single intravitreal injection of Bevacizumab (Avastin 1.25 mg / 0.05 ml) along with continuation of the maximal tolerated medical therapy. Follow up examinations were carried out daily for the first seven days post-injection, and thereafter at weekly intervals for 3 months and at fortnightly intervals for 6 months.

Results: Marked regression of neovascularisation and relief of symptoms within 48 hours was observed after intravitreal Bevacizumab injection. Substantial IOP reduction in 60 % of patients (from 52 mm Hg ± 7 mm Hg to 28 mm Hg ± 5 mm Hg) was observed within 2 weeks of intravitreal Bevacizumab injection. 40 % of patients required additional procedures like cyclocryotherapy and anterior retinal cryoablation for IOP control. Presence of peripheral anterior synechiae involving ≥ 2 quadrants on gonioscopy (6.6 %) was the single predictor for recurrence of elevated IOP on follow-up.

Conclusion: Rapid regression of iris and angle neovascularisation, observed after a single intravitreal injection of Bevacizumab (IVB) signifies its role as an adjunct in neovascular glaucoma management. Symptomatic relief in 100 % of the study population and IOP control in 86.6 % of subjects was maintained at 6 months follow up.

Introduction

Bevacizumab (Avastin); a recombinant monoclonal antibody directed against vascular endothelial growth factor (VEGF) was initially described for the treatment of colorectal cancer. VEGF has been implicated as the major anti angiogenic stimulus responsible for the formation of new vessels in almost all vasculopathies of the retina, and, drugs that inhibit the bioactivity of VEGF represents a new paradigm in the treatment of these diseases. The labelled indication of Avastin is for...
the treatment of colon cancer. Its use in the eye is therefore “off-label”. Intravitreal injection of Avastin has become an accepted practice due to “the scientific basis for the treatment, the overwhelming efficacy reported from the closely related drug Lucentis, the apparent short term safety, and the affordable low cost of the drug 1,2,3”.

There are several consecutive case series on the efficacy of off-label use of intravitreal Bevacizumab in the management of age-related macular degeneration 4, cystoid macular oedema 5, macular oedema following central retinal vein occlusion 6, proliferative diabetic retinopathy 7 and recalcitrant neovascular glaucoma 8,11 published in literature. These studies suggest that IVB (Intravitreal Bevacizumab) effectively reduces neovascular activity and vascular permeability in ocular tissues on short term follow up with no severe ocular or systemic side effects.

The present study aims to evaluate the efficacy of intravitreal Bevacizumab injection in the management of recalcitrant neovascular glaucoma refractory to maximal medical therapy and to formulate a comprehensive strategy for management of refractory neovascular glaucoma.

**Materials and Methods**

This study was designed as a prospective nonrandomized interventional study which recruited 15 consecutive patients with symptomatic neovascular glaucoma, unresponsive to maximally tolerated antiglaucoma therapy for at least a period of 3 months or more prior to enrolment into the study. After counseling on the safety, efficacy and off-label use of intravitreal Bevacizumab, an informed consent was obtained. Old records, if available, were examined, to detect the ocular diagnosis before development of NVG. A detailed clinical evaluation was made and documented which included the best corrected visual acuity, presence or absence of light perception, accuracy of projection, tension by applanation tonometry, slitlamp examination, gonioscopy, dilated fundus examination if there was enough view or B Scan ultrasonography. The patients were instructed to use gatifloxacin eye drops 4 times daily and to continue the antiglaucoma medications as well as topical steroids and cycloplegic agents.

Intravitreal injection of Bevacizumab (1.25 mg / 0.05 ml) was administered in the operation theatre under sterile aseptic condition after anesthetising the conjunctival sac using xylocaine jelly. Post-injection, the patients were discharged after 2 hours with advice to continue the topical medications as per the preoperative instructions. Visual acuity, applanation tonometry, slit lamp evaluation and fundus examination were performed at each post-injection follow up visit scheduled on a daily basis for the first seven days, at weekly intervals for 3 months and thereafter at fortnightly intervals for a period of 6 months. During this period, additional or adjuvant therapy in the form of panretinal photocoagulation or anterior retinal cryoablation was initiated depending on the ability to visualize the posterior segment of the eye. If the intraocular pressure was not controlled and the media haze did not permit adequate visualization of the posterior segment for panretinal laser photocoagulation, an anterior retinal cryoablation was performed for 360° under retrobulbar anesthesia after taking an informed consent. The IOP was strictly monitored during the period of the study and depending on the stability of intraocular pressure control, the patients were slowly weaned off the topical antiglaucoma medication to the minimum number required. The frequency of instillation of the topical steroid medication was also slowly tapered at monthly intervals and stopped by the third month post-injection.

All the 15 patients complied with the post-injection follow up and management schedule and hence there were no drop-outs during the study period.

**Results**

The patients were of the age group ranging from 45-75 yrs (Mean age = 60 years); 9 were females and 6 males giving a M:F ratio of 3:2. Associated systemic conditions included hypertension alone in 6 patients (40 %), hypertension and diabetes in 5 patients (33.3 %); and diabetes alone in 4 patients (26.7 %). No patient gave a history of treatment for coronary artery disease or cerebrovascular accident nor did any of the patient have newly detected cardiovascular or thromboembolic risk factors during the physical assessment prior to administration of intravitreal Bevacizumab.
Preoperative visual acuity ranged from 6/60 to light perception, and accurate projection was present in only one of the 6 patients with PL vision. Intraocular pressure measured with the Keeler Noncontact ‘Pulsair’ tonometer ranged from 52 mm Hg to 70 mm Hg. Slitlamp biomicroscopic evaluation findings are given in Table 1.

Gonioscopic grading of the angle was performed in 11 patients excluding those who had epithelial abrasions and were on bandage contact lens. Details of the gonioscopic findings are given in Table 2.

Fundus visualization was possible using the indirect ophthalmoscope in 9 patients excluding those with corneal opacity, vascularisation and severely oedematous cornea. In these 6 patients an indirect ophthalmoscopic fundus evaluation of the fellow eye was performed. Associated posterior segment conditions responsible for the ocular neovascularisation included: 1) Proliferative diabetic retinopathy in 8 eyes (60 %) (2) Ischemic central retinal vein occlusion in 4 eyes (26.7 %), (3) Recurrent retinal detachment, post scleral buckling, vitrectomy-gas status in 2 eyes (12 %) and (4) Recalcitrant end stage primary open angle glaucoma in 1 eye (6.6 %). B Scan ultrasonography was performed in 6 patients with significant media haze which precluded fundus visualization. The other eye examination showed no abnormality in 3 eyes, proliferative diabetic retinopathy in 1 eye, disc changes of POAG in 1 eye and features of hypertensive retinopathy Grade II in 1 eye. B Scan findings included vitreous hemorrhage (2) tractional retinal detachment at the posterior pole (2) total retinal detachment with proliferative vitreoretinopathy changes (1) and normal posterior segment (1). Table 3 lists the pre injection diagnosis in all the 15 eyes with recalcitrant neovascular glaucoma.

All the patients were on a combination of antiglaucoma medications, topical steroids, and mydriatic-cycloplegics for the preceeding 3 months and unresponsive to this therapy. The antiglaucoma medications included Glucomol 0.5 % twice daily, Dorzox eye drops thrice daily, Alphagan-P eye drops twice daily, Prednisolone acetate eye drops 4 times daily, and atropine sulphate 1 % ointment twice daily.

All patients received a single intravitreal injection of Bevacizumab 1.25 mg / 0.05 ml under sterile aseptic precautions in the operation theatre. Following the procedure the patients followed the prescribed follow up evaluation scheduled daily for the first week, weekly for 3 months and fortnightly for 3 months. The postoperative IOP changes, and slitlamp biomicroscopic findings are given in Table:4

Panretinal photocoagulation was initiated between 4\textsuperscript{th} and 8\textsuperscript{th} week post injection depending on the degree of corneal clearing obtained for fundus visualization. 9 eyes (60 %) underwent panretinal laser photo-coagulation in 2 sittings at an interval of 2 days. 5 (33.3 %) patients in whom fundus visualization was not possible underwent anterior retinal cryoablation.

<table>
<thead>
<tr>
<th>Sl.No</th>
<th>Clinical Finding</th>
<th>No. of Eyes</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Not performed</td>
<td>4</td>
<td>26.7 %</td>
</tr>
<tr>
<td>2.</td>
<td>PAS</td>
<td>15</td>
<td>100 %</td>
</tr>
<tr>
<td></td>
<td>a. Scattered</td>
<td>6</td>
<td>40 %</td>
</tr>
<tr>
<td></td>
<td>b. 1 quadrant</td>
<td>3</td>
<td>20 %</td>
</tr>
<tr>
<td></td>
<td>c. 2 quadrants</td>
<td>1</td>
<td>6.6 %</td>
</tr>
<tr>
<td></td>
<td>d. &gt; 2 quadrants</td>
<td>1</td>
<td>6.6 %</td>
</tr>
<tr>
<td>3.</td>
<td>Angle NV</td>
<td>15</td>
<td>100 %</td>
</tr>
<tr>
<td>4.</td>
<td>Blood in the angle</td>
<td>3</td>
<td>20 %</td>
</tr>
</tbody>
</table>

Table 2. Gonioscopic Findings

Table 1. Slitlamp Biomicroscopic Evaluation

<table>
<thead>
<tr>
<th>Clinical Finding</th>
<th>No. of Patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Circumcorneal congestion</td>
<td>15</td>
<td>100 %</td>
</tr>
<tr>
<td>2. Dilated Episcleral vessels</td>
<td>15</td>
<td>100 %</td>
</tr>
<tr>
<td>3. Large Epithelial bullae</td>
<td>9</td>
<td>60 %</td>
</tr>
<tr>
<td>4. Microcystic Epithelial bedewing</td>
<td>6</td>
<td>40 %</td>
</tr>
<tr>
<td>5. Corneal Vascularisation</td>
<td>6</td>
<td>40 %</td>
</tr>
<tr>
<td>6. Corneal Opacification</td>
<td>6</td>
<td>40 %</td>
</tr>
<tr>
<td>7. Neovascularisation Iris (NVI)</td>
<td>15</td>
<td>100 %</td>
</tr>
<tr>
<td>8. Ectropion Uveae</td>
<td>8</td>
<td>53.3 %</td>
</tr>
<tr>
<td>9. Cataract</td>
<td>9</td>
<td>60 %</td>
</tr>
<tr>
<td>10. Peripheral Anterior Synechia (PAS)</td>
<td>15</td>
<td>100 %</td>
</tr>
<tr>
<td>11. Hyphema</td>
<td>3</td>
<td>20 %</td>
</tr>
<tr>
<td>12. Plano.T.Bandage CL wear</td>
<td>4</td>
<td>26.7 %</td>
</tr>
</tbody>
</table>
eyes were however asymptomatic and hence a decision to keep the patient on follow up was taken.

Discussion

Vascular Endothelial Growth Factor (VEGF) antagonists represent a genuine breakthrough in the treatment of retinal and subretinal neovascularisation. Bevacizumab (Avastin) is an Ig G1 antibody that binds VEGF-A, has a size of 148 KDa, with lesser retinal penetration (than Ranibizumab), longer vitreous (5-6 days) and serum half lives (21 days) raising the possibility of both local and systemic overdosages on repeated administration. Systemic absorption of anti VEGF will inhibit important physiological functions of VEGF such as wound healing, formation of collateral circulation in myocardial and peripheral vascular ischaemia. Hence there is a definite but small risk of coronary artery disease (MI) or a thromboembolic episode following Avastin administration. However Avastin has now been used in > 10,000 patients world wide with few documented complications.

In this prospective non randomized interventional trial on 15 eyes with recalcitrant and symptomatic
neovascular glaucoma while on maximally tolerated medical therapy, our aim was to assess the safety and efficacy of a single intravitreal injection of Avastin as an adjunct to medical therapy. We also aimed to formulate treatment algorithms to manage neovascular glaucoma.

Previous studies \(^{2,7,8,11}\) have demonstrated the safety and efficacy of intravitreal Bevacizumab in causing regression of ocular neovascularisation. Kahook MY et al.\(^8\) and Davidorf F H\(^{11}\) et al have, in 2006, proved the efficacy of intravitreal Avastin injection as an adjunct in the management of neovascular glaucoma. In these studies rapid regression of iris neovascularisation and clearing of corneal oedema occurred within 48 hours giving symptomatic relief to the patient along with short term IOP control. In our study adjunctive measures were used (additional PRP, ARC, cyclocryotherapy) to quieten the neovascular process and maintain the IOP at 50% of pre treatment IOP with values ranging from 16-28 mm Hg within 2 months and this effect was maintained at 6 months. Refractory IOP elevation was seen in 2 eyes (6.6%) with extensive PAS formation.

Thus IVB as an adjuvant in the management of neovascular glaucoma may offer a more scientific

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**Table 4. Post - injection Follow up Findings**

<table>
<thead>
<tr>
<th>SL. No.</th>
<th>CASE NO</th>
<th>Pre R&lt;sub&gt;x&lt;/sub&gt; IOP</th>
<th>Post R&lt;sub&gt;x&lt;/sub&gt; IOP</th>
<th>Post R&lt;sub&gt;x&lt;/sub&gt; AC Reaction</th>
<th>Post R&lt;sub&gt;x&lt;/sub&gt; CORNEAL ODEMA</th>
<th>Post R&lt;sub&gt;x&lt;/sub&gt; IOP</th>
<th>Addl Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>PDR / Scattered PAS</td>
<td>56</td>
<td>Regression 1wk</td>
<td>Controlled 4 wks</td>
<td>Clearing 4 wks</td>
<td>1 m 3 m 6 m</td>
<td>PRP</td>
</tr>
<tr>
<td>2.</td>
<td>PDR / Scattered PAS</td>
<td>52</td>
<td>,,</td>
<td>,,</td>
<td>,,</td>
<td>22 26 24</td>
<td>PRP</td>
</tr>
<tr>
<td>3.</td>
<td>PDR / Scattered PAS</td>
<td>64</td>
<td>,,</td>
<td>,,</td>
<td>,,</td>
<td>32 30 28</td>
<td>PRP</td>
</tr>
<tr>
<td>4.</td>
<td>PDR / Scattered PAS</td>
<td>70</td>
<td>,,</td>
<td>,,</td>
<td>Persisted 2 m</td>
<td>56 40 32</td>
<td>Cyclo + ARC</td>
</tr>
<tr>
<td>5.</td>
<td>PDR / Scattered PAS</td>
<td>58</td>
<td>,,</td>
<td>,,</td>
<td>Cleared 2 wks</td>
<td>40 32 34</td>
<td>PRP</td>
</tr>
<tr>
<td>6.</td>
<td>PDR / Scattered PAS</td>
<td>60</td>
<td>,,</td>
<td>,,</td>
<td>Cleared 4 wks</td>
<td>18 20 20</td>
<td>PRP</td>
</tr>
<tr>
<td>7.</td>
<td>PDR / Scattered PAS</td>
<td>66</td>
<td>,,</td>
<td>,,</td>
<td>,,</td>
<td>20 22 22</td>
<td>PRP</td>
</tr>
<tr>
<td>8.</td>
<td>PDR / PAS = 2q</td>
<td>70</td>
<td>,,</td>
<td>,,</td>
<td>Persisted 2 m</td>
<td>42 38 32</td>
<td>ARC</td>
</tr>
<tr>
<td>9.</td>
<td>I-CRVO</td>
<td>70</td>
<td>,,</td>
<td>,,</td>
<td>Persisted 2 m</td>
<td>52 56 32</td>
<td>ARC + Cyclocryo</td>
</tr>
<tr>
<td>10.</td>
<td>I-CRVO / PAS</td>
<td>52</td>
<td>,,</td>
<td>,,</td>
<td>Cleared 4 wks</td>
<td>18 2 24</td>
<td>PRP</td>
</tr>
<tr>
<td>11.</td>
<td>I-CRVO</td>
<td>54</td>
<td>,,</td>
<td>,,</td>
<td>Cleared 4 wks</td>
<td>16 18 20</td>
<td>PRP</td>
</tr>
<tr>
<td>12.</td>
<td>I-CRVO</td>
<td>66</td>
<td>,,</td>
<td>,,</td>
<td>Persisted 2 m</td>
<td>40 22 20</td>
<td>PRP</td>
</tr>
<tr>
<td>13.</td>
<td>POAG</td>
<td>62</td>
<td>,,</td>
<td>,,</td>
<td>Cleared 4 wks</td>
<td>16 20 20</td>
<td>-</td>
</tr>
<tr>
<td>14.</td>
<td>(Post OP Recurrent RD / PAS = 1q)</td>
<td>70</td>
<td>,,</td>
<td>,,</td>
<td>Persisted 2 m</td>
<td>48 30 28</td>
<td>ARC</td>
</tr>
<tr>
<td>15.</td>
<td>Recurrent RD/ PAS = 1q</td>
<td>70</td>
<td>,,</td>
<td>,,</td>
<td>Persisted 2 m</td>
<td>62 50 40</td>
<td>ARC</td>
</tr>
</tbody>
</table>
rationale for the treatment of the causative neovascular trigger, might prevent further PAS formation and extension of secondary angle closure. It also facilitates early initiation of PRP, further dampening the neovascular trigger. Prospective randomized trials are required to validate the efficacy of IVB alone as monotherapy and its use in conjunction with maximal antiglaucoma medications, ARC, CPC and cyclocryotherapy. Long term results, chances of recurrence and the options to manage them can only be answered by a prospective trial in a larger series with longer follow up data.

References

Diagnosis of Pre-perimetric Glaucoma using Optical Coherence Tomography

Dr. S J. Saikumar MS, Dr. Saphy Jose, Dr. Savita Bhat MS, Dr. Mahesh G. MS, FRCS, Dr. A. Giridhar MS

Abstract

Objective: To study the efficacy of Optical Coherence Tomography in pre perimetric glaucoma.

Methods: 76 glaucoma suspects with normal or borderline white on white visual fields underwent RNFL analysis with Optical Coherence Tomography (Stratus OCT version 4).

Outcome measure: Borderline or definite RNFL thinning in the inferior or superior quadrants was taken as the main outcome measure.

Results: Six out of seventy six glaucoma suspects showed RNFL changes in OCT suggestive of glaucoma giving the test a specificity of 92 %. All six patients had cup disc ratio of 0.6 or more and five out of six patients had IOP of 18 or more and positive family history of glaucoma.

Conclusion: Optical Coherence Tomography is an useful tool for diagnosis of pre perimetry glaucoma especially in the presence of other risk factors like raised IOP, enlarged cup and family history of glaucoma.

Introduction

Glaucoma is an optic neuropathy characterized by a specific and progressive injury to the optic nerve and retinal nerve fiber layer. Because the injury due to glaucoma is irreversible, early detection and prevention of glaucomatous RNFL loss is of vital importance. Examination of the optic nerve head and its surrounding nerve fiber layer is considered essential in both detection and monitoring of glaucoma. Damage to the RNFL has been shown to precede visual field loss. Upto 30-50 % nerve fibre loss might have occurred before the first detectable field defect. Hence, objective methods of measuring these structures will aid ophthalmologists in making an accurate diagnosis.

Optical Coherence Tomography (OCT) which was first described in 1991, is a high resolution cross sectional imaging technique that allows accurate measurement of the retinal nerve fiber layer. With the ability to quantify the thickness of the RNFL with a resolution of 8 to 10 microns, clinicians potentially have a more objective tool in helping to diagnose glaucoma much earlier than visual fields. However, white on white perimetry is still considered the gold standard for diagnosis of glaucoma. AAO Preferred Practice Patterns still mentions that the diagnosis of glaucoma is based on appearance of the optic disc and standard achromatic perimetry. In this current study glaucoma suspects who had normal or...
borderline visual fields were subjected to RNFL analysis using Optical Coherence Tomography.

**Materials and Method**

The study was designed as a retrospective observational case series which included glaucoma suspects attending a tertiary care glaucoma centre.

**Study Population:** 103 glaucoma suspects who underwent RNFL analysis on Optical Coherence Tomography were initially enrolled, out of which 76 eyes of 76 patients fulfilled all criteria and were finally included in the study. Only one eye per person was included in the study. If one eye had abnormal fields and the other eye normal, the normal eye was included in the study. If both eyes were normal or both were borderline, the eye with the better PSD value on HFA was selected and assigned to the study.

Complete ophthalmic examination including visual acuity, intraocular pressure, gonioscopy and optic disc evaluation was performed. All patients initially underwent either 30-2 full threshold or SITA - standard white on white perimetry. The time gap between perimetry and OCT was not more than two weeks. Patients with spherical refractive errors of more than 6D, astigmatism more than 3 Dioptres, visual acuity worse than 6/12 were excluded from the study. Other exclusion criteria were secondary causes of raised IOP, abnormal disc appearance such as tilted discs and suspected neurological diseases which are likely to affect the field testing. Those patients with extensive peripapillary atrophy were also excluded from the study.

**Definition of glaucoma suspect:** For the purpose of this study a glaucoma suspect was defined as the presence of an abnormal disc appearance consistent with glaucoma as determined by an experienced clinician. Disc asymmetry of 0.2 or more was also included in the definition of glaucoma suspect.

**Observation procedures:** All selected patients had a reliable visual field on Humphrey Visual Field Analyzer. Reliable fields were defined as fixation loss rate of less than 33 % and false positive and false negative error rates of less than 20 %. Normal and borderline definitions were based on the glaucoma hemi field test.

**OCT measurements:** The test was performed after dilation of the pupil to at least 5 millimetre. Three measurements were performed for each eye. Only measurements with signal strengths of six or more were accepted as reliable for this study.

**Outcome Measures:** Definite RNFL thinning (shaded as red) or borderline RNFL thinning (shaded as yellow) in either the inferior or superior portion of the disc was taken as the main outcome measure. Charts with only temporal or nasal thinning were excluded from the study.

**Statistical Analysis:** The Fisher exact test was used to assess the statistical significance of glaucomatous changes in OCT in the presence of other risk factors like raised IOP, family history of glaucoma and enlarged cup-disc ratio. Odds ratio was also calculated for the above mentioned risk factors.

**Results**

After all exclusions, 76 eyes of 76 patients were enrolled in the study, of which 51 were males and 25 were females. The age group distribution is shown in Figure 1.

Fig. 1. Age group distribution of the study population

A total of 6 patients had abnormal OCT among the 76 patients enrolled in the study. 15 patients had a positive family history of glaucoma in either their first degree or second-degree relative and out of these 5 had abnormal OCT. Only one person with abnormal OCT had no family history of glaucoma. The difference between the positive family history group and the negative family history group was statistically significant (p value 0.001, Fisher exact test). The odds of having an abnormal OCT was 36 times in persons having a positive family history of glaucoma.

A total of 47 persons had a CD ratio of 0.6 or more, out of which 17 had CDR of more than 0.8 (Figure 2). 5 out of 6 OCT positive persons had a CDR of 0.8 or more, and one had a CDR of 0.7. When a CDR of 0.6 was taken as the cut-off, the chance for having an
abnormal OCT was not statistically significant (p value 0.0768, Fisher exact test). But when CDR of 0.8 was taken as the cut-off, the chances of having an abnormal OCT was statistically significant (p value 0.0017, Fisher exact test). Persons with a CDR of 0.8 or more are 22 times more likely to have an abnormal OCT, compared to those having a CDR of 0.7 or less.

The mean IOP of the 76 eyes enrolled in the study was 16.29. 26 patients had an IOP of 18 or more, out of which only 7 eyes had an IOP of 22 mm Hg or more (Figure 3). 5 out of the 6 patients who had an abnormal OCT had IOP more than 18 mm Hg. With 18 mm Hg as the cut-off, the chance of having an abnormal OCT was statistically significant (p value 0.0161, Fisher exact test). The odds of having an abnormal OCT was 11 times more in persons with an IOP of more than 18 when compared to those with IOP of 17 or less.

OCT in glaucoma is a very effective tool in identifying the true negatives. It shows that 70 out of 76 eyes was proven as not having glaucomatous damage. This gives the test a specificity of 92 %.

4 eyes had inferior thinning and 2 had superior thinning. No biarculate thinning was seen in any of the six cases. 5 eyes out of 6 had definite thinning of RNFL (shaded as red) and I had borderline thinning (shaded as yellow).

In the 70 persons with normal OCT, mean RNFL thickness was 119.3 and 124 in the superior and inferior quadrants respectively. Compared to this, the same values were 79 and 75.33 respectively in the 6 patients with abnormal OCT. (Table 1)

<table>
<thead>
<tr>
<th></th>
<th>Mean superior RNFL thickness (in microns)</th>
<th>Mean inferior RNFL thickness (in microns)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with normal OCT</td>
<td>119.3</td>
<td>124</td>
</tr>
<tr>
<td>Patients with abnormal OCT</td>
<td>79</td>
<td>75.33</td>
</tr>
</tbody>
</table>

**Discussion**

We have studied 76 eyes of 76 patients who were clinically found to have some suspicion of Glaucoma. The numbers were slightly smaller compared to a few other studies. Ours was an observational study and there was no control group, which were used in other studies. In the study by Kanamori et al normal population, glaucoma suspects, ocular Hypertensives and glaucoma patients were included in the study and compared. We were only studying the ability of the OCT in picking up Pre perimetric glaucoma in suspected cases.

The role of family history has been highlighted in many studies although the incidence of MYOC mutation among Indian primary open angle glaucoma patients has been found to be lower compared to certain other parts of the world. In our study, in glaucoma suspects with normal fields, those with positive family history of glaucoma are 36 times more likely to have an abnormality in OCT when compared to those with no family history of glaucoma.

Cup – disc ratio has been used to define glaucoma suspects in many other studies. Lalezary et al have
used stereo photographs to classify the study population into normal and glaucoma suspects. In our study the classification has been done by an experienced glaucoma specialist by using 78 D lens biomicroscopy.

In our study, the specificity was a healthy 92 %, which means the OCT is a good tool to rule out disease. Sensitivity was not calculated in our study since this requires a normal age-matched control group. Budenz et al showed a sensitivity of 84 % and a specificity of 98 % in their study on perimetric glaucomas.

As expected the mean RNFL thickness in the inferior and superior quadrant was significantly lower in the abnormal OCT group compared to the normal OCT group. Kanamori et al have studied the temporal and nasal thickness also along with the superior and inferior. We felt that many errors may crop up if the nasal and temporal thickness was included. Also the inferior and superior poles were the first affected in early glaucoma. The normal OCT showed the characteristic double-hump pattern which is in agreement with other human and histologic studies.

Self assessment of this study – STARD guidelines

Standards for Reporting of Diagnostic Accuracy (STARD) is an internationally accepted method of assessing a study conducted on a diagnostic equipment. A recent report on the quality of reporting of diagnostic accuracy was conducted with 30 published articles. The STARD guideline consists of 25 points which are to be fulfilled for a good diagnostic study. In this meta analysis only 26 % of the papers reported more than half of the STARD items. Our study has reported 11 out of the 25 STARD items, and this compares well with many published articles on diagnostic accuracy of OCT in diagnosis of early glaucoma.

Conclusion

Optical Coherence Tomography is a useful tool for pre perimetric diagnosis of glaucoma. The usefulness of this tool increases in the presence of other risk factors like family history of glaucoma, large cup disc ratio and high intra ocular pressure. The specificity of this tool in our study was 92 % which makes it a good tool to rule out those who do not have glaucoma. Comparative studies with the other imaging tools like HRT and GDx are needed in our population to determine which is the best tool for diagnosis of early glaucoma.

References

Pediatric Open Globe Injuries- Prognostic Factors And Visual Outcome

Dr. Meena C.K. MS, Dr. Elizabeth Joseph MS, Dr. Thomas Cherian MS

Abstract

Aim: A retrospective review of open globe injuries in children

Method: Medical records of 100 children with open globe injury were reviewed. The anatomic and functional status of the eyes before and following the repair, the details of all surgical procedures and final visual outcome assessed

Results: Male to female ratio was 3:2. More than 20% of cases needed a secondary procedure. Final visual outcome was based on type of wound and the involvement of the posterior segment. Mean follow up was 6 months

Conclusion: Prognosis following open globe injury in children is dependent on the initial damage and timely management

Introduction

Ocular injuries are a major cause of monocular blindness in children worldwide. When compared to the adult population children suffer a higher percentage of open globe injuries comprising 19% - 58.3% of all cases of ocular trauma. Ocular injuries in children are a matter of great concern due to a variety of reasons. Delay in presentation is one of them as a child may not recognize or even verbalize a history of ocular trauma. Open globe injuries involve a full thickness disruption of the eye ball. Management of open globe injuries pose great dilemma in children as the surgical repair in a child’s eye is much more difficult and challenging than the management of an adult trauma. Over and above, repeated examinations under general anesthesia, prolonged periods of visual deprivations which can be amblyopiogenic and visual rehabilitation are some of the many factors making a childhood open globe injury a difficult entity to deal with.

Materials and Methods

Retrospective review of the hospital records of children who were admitted to our tertiary eye care facility with trauma from December 2005 to January 2007 was done. 100 consecutive patients who met the inclusion criteria were selected. The inclusion criteria were age between 1 and 14 yrs, full thickness disruption of the globe with subsequent repair of the injury, minimum follow up of 3 months. Cases which had undergone repair elsewhere before admission were excluded. The data collected include patients demographic profile, cause of trauma, circumstances and time of injury,
extent of the open globe injury in terms of location and type according to the International Ocular Trauma Classification. Vision and slit lamp examination at presentation were recorded in children who could cooperate. In children who would not co-operate a detailed clinical assessment under general anesthesia was done prior to any intervention. Records were reviewed for the presence of uveal tissue prolapse, lens disruption, vitreous prolapse, signs of infection, posterior segment abnormalities and retained intraocular foreign bodies. Details of the primary and subsequent surgical procedures were noted. Visual acuity at the final visit and the various visual rehabilitation measures taken were noted.

All patients underwent primary repair within 12 hrs of presentation under general anesthesia. All Corneal wounds were repaired using 10-0 nylon monofilament and scleral wounds were repaired using 6-0 vicryl. Lens matter if existing in the anterior chamber was aspirated and any anterior segment foreign bodies were removed during the primary procedure. Vitreous in the wound were tackled with an automated vitrector.

All the patients received prophylactic systemic antibiotics. Topical antibiotics and cyclopiegics were started post operatively. Topical steroids were started in 24 hrs and in cases with severe cellular reaction systemic steroids were added.

Children who had an uneventful post operative period were discharged the 2nd postoperative day with advise to follow up regularly. Patients with a minimum of 3 months follow up were included in the study. The details of all subsequent procedures if any performed were recorded and the best corrected visual acuity and a detailed clinical assessment on the last follow up were recorded. Age appropriate charts with Snellen equivalent were used in small children.

**Results**

100 consecutive cases of open globe injuries in the age group 1-14 were included. 60 were males and 40 females making a 3:2 male to female ratio (Fig. 1). Age ranged from 1-14 yrs (mean-7.8 yrs). Children in the preschool age group (<4 yrs) comprised of 33% of the cases (Fig 2).

In 5 cases the cause was unknown. Kitchen knives (15) and broom stick (16) were the commonest causes of trauma. Kitchen knife injury was commonest in the preschool age group (10 of the 33). Pen and pencil tips accounted for most of the injuries in the school going group (13). Other objects included stone in 12 cases, cable wires, scissors, thorn, cracker burst injury in 2 cases and a single case of gun pellet injury (Table 1). Right eye was involved in 56 cases.

35 cases presented 24 hrs after the trauma. Of this 8 cases presented after 48 hrs of the insult. 10 of the 35 case with late presentation had a visual acuity of <6/60 on last follow up. 77 cases had only corneal
involvement; rest of the 25 cases had scleral involvement of which 15 were corneoscleral. Details of the zone of injury are given in fig 3. 68 of the total cases had associated findings like uveal tissue prolapse, lens disruption, hyphaema, exudates or vitreous in the wound.

Presenting visual acuity could be assessed in 71 patients, rest being too young or not cooperative. Visual acuity ranged from no PL to 6/6 on the Snellen chart. However on last follow up only 6 children were not cooperative for visual assessment.46 of the 71 patients had a presenting visual acuity of 6/60 or less. Final visual acuity of 21 of these 46 patients were <6/60 on last follow up. 15 of these 21 patients required more than one surgical procedure. The details of the subsequent interventions are given in table (2). 9 of the 21 cases had scleral involvement. 9 of the 21 cases had a delay in presentation, in 5 cases more than 24 hrs and in 4 cases more than 48 hours. The causes for reduced visual acuity in these patients are shown in table (3). 33 cases needed further interventions following the primary procedure. Of this 2 patients were not willing for the secondary procedures.

Poor structural and functional outcome were analyzed with independent variables like presenting visual acuity, delay in presentation, need for subsequent procedures and zone of trauma. Factors associated with poor visual outcome are shown in table (4).

### Table 1. Instrument of ocular injury

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knife</td>
<td>15</td>
</tr>
<tr>
<td>Stick(bamboo,broom)</td>
<td>16</td>
</tr>
<tr>
<td>Stone</td>
<td>11</td>
</tr>
<tr>
<td>Pen</td>
<td>9</td>
</tr>
<tr>
<td>Bangle</td>
<td>1</td>
</tr>
<tr>
<td>Pencil</td>
<td>9</td>
</tr>
<tr>
<td>Metal wire</td>
<td>10</td>
</tr>
<tr>
<td>Wooden piece</td>
<td>6</td>
</tr>
<tr>
<td>Thorn</td>
<td>2</td>
</tr>
<tr>
<td>Cracker burst</td>
<td>2</td>
</tr>
<tr>
<td>Iron nail</td>
<td>2</td>
</tr>
<tr>
<td>Coconut shell</td>
<td>1</td>
</tr>
<tr>
<td>Spectacle glass</td>
<td>4</td>
</tr>
<tr>
<td>Gun pellet</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>5</td>
</tr>
<tr>
<td>Cycle wheel spoke</td>
<td>1</td>
</tr>
<tr>
<td>Compass</td>
<td>2</td>
</tr>
<tr>
<td>Glass</td>
<td>2</td>
</tr>
<tr>
<td>Soda bottle cap</td>
<td>1</td>
</tr>
</tbody>
</table>

### Table 2. Subsequent Procedures

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retinal detachment surgery</td>
<td>6</td>
</tr>
<tr>
<td>Cataract extraction</td>
<td>15</td>
</tr>
<tr>
<td>Parsplana vitrectomy with intravitreal antibiotics</td>
<td>9</td>
</tr>
<tr>
<td>Anterior vitrectomy with pupilloplasty</td>
<td>1</td>
</tr>
</tbody>
</table>

### Table 3. Causes of reduced vision

<table>
<thead>
<tr>
<th>Condition</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retinal detachment(surgery done)</td>
<td>5</td>
</tr>
<tr>
<td>Central corneal scar</td>
<td>2</td>
</tr>
<tr>
<td>Phthysical</td>
<td>5</td>
</tr>
<tr>
<td>Endophthalmitis</td>
<td>5</td>
</tr>
<tr>
<td>Macular scar</td>
<td>1</td>
</tr>
<tr>
<td>Traumatic cataract (not willing for surgery)</td>
<td>1</td>
</tr>
<tr>
<td>Amblyopia</td>
<td>2</td>
</tr>
</tbody>
</table>

### Table 4. Factors correlating with reduced visual outcome

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>Final visual acuity &lt;6/60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presenting visual acuity &lt;6/60 (n=46)</td>
<td>21</td>
</tr>
<tr>
<td>Zone 1 injury (83)</td>
<td>10</td>
</tr>
<tr>
<td>Zone 2 injury(15)</td>
<td>9</td>
</tr>
<tr>
<td>Zone 3 injury(n=2)</td>
<td>2</td>
</tr>
<tr>
<td>Subsequent procedures (n=33)</td>
<td>18</td>
</tr>
</tbody>
</table>

### Statistical analysis

Multiple regression analysis showed the necessity for subsequent procedures and the zone of trauma to be significant risk factors and predictors of the final outcome. The delay in presentation as compared to the final outcome was not significant (Table 5).
Linear regression analysis of the presenting and final visual acuity was statistically significant with a p value of .01.

Discussion

Pediatric open globe injury though a very important cause of childhood blindness is not a well discussed or studied entity. Many of the injuries in children are preventable which can be assessed from the nature of injury the children sustain. Better awareness and improved safety procedures can avoid most of these injuries. The incidence of ocular trauma has changed little over time. Seidelmann 8 recorded the overall incidence of eye trauma in children below the age of 15 years to be 21.5 %. Holland 9 noted that out of 2,309 hospitalized patients of ocular trauma 20 % were children.

There was a preponderance of boys in our study (60 %) which is similar in other studies 12-13. This disparity in sex may be due to the aggressive behavior of boys.

In our series more severe injuries occurred in children more than 4 years of age that is in the school going age group. Prior studies have shown that majority of injuries occur in children less than 8 years of age.

Some studies have reported that visual acuity at presentation is a strong prognostic factor of final visual acuity14-15. Our study showed a similar correlation. This could be assessed as most of the severe traumas occurred in children more than 4 years of age where vision assessment was possible.

Many studies have shown delayed presentation to be an important predictor of final outcome 16-17. We were unable to find this correlation with significance possibly due to the lesser number of late presenters and greater awareness among the parents in our population.

We have found the zone of injury a very important predictor of both functional and structural outcome. Zone 2 injuries even small scleral tears with corresponding retinal tears predisposed to retinal detachment. Though posterior segment surgeries were performed in 15 cases only 4 of them had any significant improvement in vision.

Conclusion

Our study concludes that most of the childhood traumas are preventable. School going age group are found to be more vulnerable to such traumas. Parents and schoolteachers therefore need proper counseling in this regard. Over and above the most important factor is the awareness of children. Children who have reached the age to comprehend advice from others need to be made aware of the dangers of playing with sharp objects and should be counseled on proper utilization of common articles which can be quite dangerous in certain circumstances. Since the zone of injury and severity of the initial wound were the many predictors of outcome, prevention is the only way out of this sight threatening entity.

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17) Jesse R.Hill et al Evaluation of open globe injuries of children in the last 12years. Retina
NSAIDs: Emerging Trend

A. Chandra 1 MD, Rahul P. 2 MD, V.K. Raju 3 MD FRCS FACS

Non-steroidal anti-inflammatory drugs (NSAIDs) have been used for decades to relieve pain and fever. These agents block the cyclo-oxygenase pathway which converts arachidonic acid into endogenous prostaglandins, which are the most potent mediator of inflammation. Hence if prostaglandin production is inhibited, there is less inflammation. Unlike steroids, NSAIDs do not have a cyclopentanoperhydro phenanthrene skeleton.

Non-steroidal anti-inflammatory drugs have been used in ophthalmology as a topical agent for more than three decades. Topical Indomethacin was first used as a pre-operative drug to decrease post-operative inflammation after cataract surgery. It was formulated as a sesame seed oil solution, but due to local irritation and low efficacy its use was discontinued. Newer drugs which had increased penetration into the anterior chamber and more efficacy, slowly replaced the old ones. There are presently four classes of topical NSAIDs – indoles, phenylacetic acids, phenylalkanoic acids and arylacetic acids. Indomethacin is an indole, Diclofenac and Bromfenac are phenylacetic acids; Flurbiprofen, Suprofen and Ketorolac are phenylalkanoic acids and Nepafenac is a pro-drug arylacetic acid. On topical application, the NSAIDs penetrate the eye and have an aqueous humor level adequate to inhibit prostaglandin synthesis.1,2 Topical NSAIDs are commonly used in the management and prevention of ocular inflammation and cystoid macular edema related to intraocular surgery. Other common uses are maintenance of mydriasis during cataract surgery,3,4 reduction of discomfort after surgery and laser5 or in allergic conjunctivitis. Many clinical studies have also suggested a role of topical NSAIDs for the treatment of inflamed pinguecula, pterygia,6 strabismus,7 glaucoma,8 refractive errors,9 and corneal abrasions.10 Persistent inflammation in the anterior chamber leads to cystoids macular edema (CME), which is the most common cause of decrease in visual acuity after uncomplicated cataract surgery. Topical NSAID decrease the incidence of CME by decreasing the postoperative inflammation.11,12 Concurrent administration of corticosteroids and NSAIDs may prove a synergistic activity resulting in more rapid resolution of symptomatic CME.13 In patients with established CME, NSAIDs are more effective than steroids alone.14 It is evident that corticosteroids have a greater anti-inflammatory effect in suppressing AC cells and flare following cataract surgery. However the complementary role of NSAIDs in preventing CME has become increasingly clear from published studies and clinical practice. A recent study even showed improvement in quality of vision after concomitant NSAID application.15

The issue of preventing rather than treating CME is much more important. Some of the risk factors for the development of CME can be classified as follows-

A) Preoperative: diabetes, uveitis, prior history of CME, retinal vein occlusion (branch vein occlusion, central retinal vein occlusion, diabetic macular edema, macular degeneration)

B) Intra-operative: iris trauma, retained lens material, posterior capsule rupture, vitreous loss, sulcus and anterior chamber IOL implantation

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C) Post-operative: sudden discontinuation of medication, idiopathic increased intraocular inflammation, trauma

D) Other conditions: epiretinal membrane, retinitis pigmentosa, use of prostaglandin analogues just before surgery etc.

There are various reports of CME associated with latanoprost after uncomplicated cataract surgery. It is therefore not advisable to use latanoprost during pre and postoperative periods. If at all, they must be well covered with the judicious use of NSAIDs.

Diclofenac and Ketorolac are primarily used to decrease the pain and inflammation after ocular surgery, while Flurbiprofen and Suprofen are primarily used to inhibit postoperative miosis. Ketorolac also decreases itching caused by seasonal allergic conjunctivitis. It is the only NSAID which is available as a preservative free solution.

Bromfenac and Nepafenac are the recent NSAIDs. Bromfenac is much more active against COX-2 than COX-1 unlike Ketorolac.

A study was conducted to measure the relative potency of different NSAIDs with regards to the different molecules of cyclo-oxygenase enzyme i.e. COX-1 and COX-2. This is measured by IC50, which is the drug concentration required to inhibit the enzyme by 50%.

As the IC50 decreases, the potency of the drug increases. Inhibition of COX-2 is responsible for the anti-inflammatory action of these agents.

<table>
<thead>
<tr>
<th></th>
<th>IC50 COX-1</th>
<th>IC50 COX-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bromfenac</td>
<td>0.53uM</td>
<td>0.023uM</td>
</tr>
<tr>
<td>Diclofenac</td>
<td>0.95uM</td>
<td>0.085uM</td>
</tr>
<tr>
<td>Amfenac</td>
<td>0.25uM</td>
<td>0.150uM</td>
</tr>
</tbody>
</table>

Nepafenac is hydrolysed to form Amfenac. Bromfenac is 3.7 times more potent than Diclofenac and 6.5 times than Amfenac. Hence it is a stronger inhibitor of COX-2 enzyme as compared to other ocular NSAIDs.

A study conducted to measure the penetration of NSAIDs in ocular tissues detected the presence of Bromfenac even after 24 hours. The Bromfenac ocular concentrations were present for longest time period in comparison to Nepafenac. This accounts for the bid dosing of Bromfenac and possibly the earlier efficacy seen in reduction of pain.

The resolution of pain after cataract surgery was rapid in patients that received Bromfenac therapy. This was demonstrated by 2 US clinical trials where 93.3% of the patients receiving Bromfenac treatment were pain free as opposed to 63.7% of the patients receiving placebo treatment.

Less than 2% adverse effects are seen with Bromfenac. 1.4% of the patients who received this drug developed CME, while 4.7% in the placebo group developed CME.

Bromfenac also has a potential use in the management of allergic conjunctivitis. It is comparable to Pemirolast when used for allergic conjunctivitis.

Nepafenac is a new topical NSAID pro-drug which is hydrolysed by intraocular tissue to Amfenac, which is a potent anti-inflammatory agent. It has been approved by the US Food and Drug Administration (FDA) for the treatment of pain and inflammation after cataract surgery. It is the only NSAID which can reach the posterior segment and inhibit prostaglandin synthesis. Therefore this drug appears promising in reducing prostaglandin mediated vascular leakage such as cystoid macular edema. Nepafenac also has anti-VEGF properties and decreases neovascularisation.

Kern et al. demonstrated that topical application of this drug may inhibit progression of diabetic retinopathy.

The dosing schedule of Bromfenac is twice daily; Nepafenac is thrice daily and for all other NSAIDs is four times a day. There is a general consensus among ophthalmologists that NSAIDs should be started 1-3 days before surgery to decrease postoperative inflammation and the incidence of CME. Post-operatively it should be continued for about 4-6 weeks. For high risk patients it should be continued for three months or more. Common side effects include transient burning, stinging and hyperemia of the conjunctiva. Corneal complications like superficial punctuate keratitis, sub epithelial infiltrates, stromal infiltrates and persistent epithelial defects after topical NSAID use are uncommon. There have been few reports of corneal melt with topical Bromfenac, Diclofenac and Ketorolac drops. In addition, allergies and hypersensitivity reactions have been reported with all the NSAIDs.

Ophthalmologists must be aware of the conditions which predispose to corneal melting. Some of these are severe keratoconjunctivitis sicca, persistent
epithelial defect, and neuropathic keratopathy. Ocular cicatrical pemphigoid, Steven Johnson Syndrome and chemical burns. The mechanism of melting is unclear, probably multifocal. Recent studies have further defined the role of matrix metalloproteinases (MMPs) in the pathogenesis of corneal ulcerative keratolysis associated with the topical use of Diclofenac. Histopathologic examination of the corneal button which was perforated by the use of topical NSAIDs showed an increased level of MMP-8 in the corneal epithelium. Corneal melt may also occur as a result of increased amounts of precursor metabolized by the lipo-oxygenase pathway, resulting in an increased amount of lipo-oxygenase products, like leukotriene B4. Also, frequent instillation of Ketorolac and Diclofenac leads to significantly decrease in normal corneal sensation which resembles neurotrophic keratitis and therefore may trigger corneal melting.

Today, several NSAIDs are commercially available: Diclofenac, Flurbiprofen, Ketorolac, Bromfenac and Nepafenac. At present the ophthalmologists have to make a decision between the more efficacious topical Nepafenac. At present the ophthalmologists have to make a decision between the more efficacious topical Nepafenac. At present the ophthalmologists have to make a decision between the more efficacious topical Nepafenac. At present the ophthalmologists have to make a decision between the more efficacious topical Nepafenac. At present the ophthalmologists have to make a decision between the more efficacious topical Nepafenac. At present the ophthalmologists have to make a decision between the more efficacious topical Nepafenac. At present the ophthalmologists have to make a decision between the more efficacious topical Nepafenac.

Reference

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A-Scans: A Practical Review

Dr. Ashley Thomas Jacob MS DNB FRCOphth FICO

An A-Scan is an integral part of any ophthalmic facility that has an operating room. This instrument is undoubtedly the most important diagnostic equipment that an ophthalmic surgeon possesses.

The beginning ophthalmic surgeon is usually pre-occupied with purchase of the best Autorefractometer, Slitlamp, laser, etc that he forgets the one single instrument that may turn out to be the cornerstone in a successful cataract surgery practice. A good A-scan consistently gives the surgeon outstanding accuracy in IOL power calculation and enhances good visual outcomes in a patient. Owning a good A-Scan might be the fine line between mediocre results and excellent outcomes.

**Sonomed**

PacScan 300 series: Model 300 A  
Country of Origin: USA  
Cost in INR: 150,000 (with printer)  
Distributor in India: Suraj Hi-Tech Pvt. Ltd, Kochi

**General attributes**

- Touch screen operation, LCD Monitor, Archiving software  
- Direct contact / Immersion scan modes  
- Formulae: Binkhorst, Regression-II, Theoretic/T, Holladay, Hoffer-Q, Haigis

**Probes**

- Direct contact probe for hand held, immersion & slit lamp mounted application  
- Soft Touch probe for minimizing corneal compression whilst performing scan

**Special Features**

- Post refractive IOL formulae:  
  1. Latkany Myopic regression  
  2. Aramberri Double-K  
- Clinical Accuracy : +/- 0.1mm  
- Electrical Accuracy : +/- 0.0484mm  
- Also Available  
  A-Scan with pachymeter, AB Scan

**Summary**

User friendly, Helpful in post lasik cases, largely accurate, robust reputation, Good Service backup

**OTI – OPKO Technologies**

**OTI Scan Model A-2000**

Country of Origin: Canada  
Cost in INR: 100,000 (presently an upgrade with B-scan)
Distributor in India: Biomedix Optotechnik & Devices, Bangalore

**General attributes**

Laptop or PC based, connect with USB cable, Archiving software

Direct contact / Immersion scan modes

Formulae: Binkhorst, Regression-II, Theoretic/T, Holladay, Hoffer-Q, Haigis

**Probes**

Direct contact probe

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**DGH**

Model: DGH 5000e

Country of Origin: USA

Cost in INR: 160,000 (with printer)

**General attributes**

Direct contact / Immersion scan modes

Formulae: Binkhorst, Regression-II, Theoretic/T, Holladay, Hoffer-Q, Haigis

**Probes**

Direct contact probe . Immersion shell upgrade available

---

**Special Features**

1. Built in algorithms for Silicone oil filled eyes, Acrylic / PMMA/ Silicone pseudophakos, dense cataracts. Automatically selects the best algorithm
2. Post-measurement optimization of A-Scan gain & TGC
3. Instant capture feature to prevent compression of soft eyes
4. Laser or inkjet print possible, with multiple IOL formulations

Clinical Accuracy : +/- 0.1mm

Electrical Accuracy : +/- 0.01mm

**Also Available**

AB Scan

**Summary**

User friendly, being PC or Laptop based has advantages and disadvantages, Good Service backup. Presently available only as AB Scan

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**Special Features**

1. Corneal compression detection software

Clinical Accuracy : +/- 0.1mm

Electrical Accuracy : NA

**Also Available**

A-Scan with pachymeter

**Summary**

User friendly, robust design, portable in camp situation

---

**TOMEY**

Model: AL 100 biometer

Country of Origin: Japan
Cost in INR: 180,000 (with printer)
Distributor in India: My Healthskape, Mumbai

**General attributes**

- Touch screen operation, LCD Monitor, Archiving software
- Direct contact / Immersion scan modes
- Formulae: Binkhorst, Regression-II, Theoretic/T, Holladay, Hoffer-Q, Haigis

**Probes**

Direct contact probe for hand held, immersion

**Special Features**

- Clinical Accuracy : +/- 0.1mm
- Electrical Accuracy : +/- 0.01mm

**Also Available**

A-Scan with pachymeter, AB Scan

**Summary**

User friendly, largely accurate, good reputation, Good Service backup

**BIOMEDIX**

Model: Echorule 2
Country of Origin: India
Cost in INR: 150,000 (with printer)
Manufacturer: Biomedix Optotechnik & Devices, Bangalore

**ZEISS**

Model: IOL Master, Optical coherence biometry
Country of Origin: Germany
Cost in INR: 800,000
Distributed in India by Zeiss India

**General attributes**

- Archiving software
- Direct contact / Immersion scan modes
- Formulae: Binkhorst, Regression-II, Theoretic/T, Holladay, Hoffer-Q, Haigis

**Probes**

Direct contact probe for hand held, immersion & slit lamp mounted application

**Special Features**

- Clinical Accuracy : NA
- Electrical Accuracy : NA

**Summary**

Indian make, good service back up
Probes
Non contact coherence method

Also Available
Hagis –L formula for myopic lasik/PRK

Summary
The future is here. Higher accuracy and consistency required. Service is a concern in some areas.

Conclusion
Whilst reviewing the various models I was convinced that the ideal surgical center would like to possess two ultrasound A-Scans (especially if the surgeon has a refractive practice also). One AB-Scan (to enable posterior evaluation), and another A-scan (alone).

Tomey, Sonomed and OTI have combination AB-Scans. However, monetary considerations would tilt the scales in favor of purchasing one ultrasound A-Scan that would deliver consistent results in normal population and is supported by good service personnel.

I am convinced that both Sonomed and OTI score in this respect. But OTI presently bundles the A-scan in combination with its AB-Scan and is not available as A-Scan alone in India.

If the surgeon has a demanding camp schedule and requires a robust system for transportation to camp sites, then DGH or Echorule2 would fulfill that requirement.

IOL master may not offer consistent results in a vast majority of the patients in the Indian context. However, if brand image is a factor, it would be a good investment.

Special Features
1. Formulae for calculating corneal power after refractive surgery
2. Measurement possible in staphylomas, silicone filled eyes
3. Automatic left-right recognition
4. IOL calculations for Angle supported and Iris fixated lenses

Clinical Accuracy : 0.1mm
Electrical Accuracy : NA
A Modification for the Streak Retinoscope

Dr. Mathew Joseph  MS

Introduction
The addition of a + 1.50 D Spherical Lens to the viewing hole of a streak Retinoscope relaxes the accommodation of the examiner and so makes Retinoscopy less tiring, especially for presbyopes.

Discussion
It is a common experience for those who use the Streak Retinoscope for refractions to develop symptoms of asthenopia, and head aches. This is especially true for those who are presbyopic and those who do a large number of cases daily.

The Optical principle
Retinoscopy is done at a distance of 2/3 of a metre, i.e. 66 cm, between the eyes of the examiner and the patient.

It is very important to bear in mind that the following calculations are for an examiner who is an emmetrope, or, in the case of an ametrope, one who is wearing his/her full distance correction.

To calculate the accommodative power required to focus an intermediate point within the range of accommodation, the formula $A = V - R$ is used, where

$A$ is the accommodative power required in dioptres.

$V$ is the dioptric value of the intermediate point.

$R$ is the dioptric value of the far point.

The far point $R$ is infinity for an emmetrope, and the reciprocal of infinity is zero.

For $V$ the intermediate distance of 66 cm - that is 0.66m, the dioptric value is 1.50 D (Dioptre being 1/the focal length expressed in metres, i.e. $1/0.66m=1.50D$).

Therefore, $A$ the accommodative power the examiner needs to exert to focus at the distance of 66cm is 1.50D.

This means that if a 1.50D sph lens is placed in front of the examiner’s eye, his/her accommodation for that distance is completely relaxed. It also follows that the patient’s eye is at exactly the principal focus of the 1.50D lens, i.e. at its focal length of 66cm. Apart from relieving the examiner’s accommodative effort, the retinoscopy shadow is seen clearly and so facilitates the exact neutralization point.

The small magnification provided by the lens helps when the subject’s pupil is small and when the glow is dull due to either media opacities or low charge in the batteries. These last two points should be helpful even to the non-presbyopic retinoscopist.

Fig. 1. A + 1.50 D Spherical lens is stuck over the viewing hole of the retinoscope

Ozanam Eye Centre, Benziger Hospital, Kollam 691 001.
In the illustration, the lens is seen stuck with adhesive (Fig. 1) over the viewing hole of the retinoscope. One can request an optician to grind the lens to the requisite size. It need not be as small as in the illustration. It is desirable that the optical centre of the lens be marked before grinding and is well centred over the viewing aperture.

References

Humour in Ophthalmology

Acronyms in Ophthalmology

RRV

Acronym is defined by any standard dictionary as ‘a word made up of first letters of a compound name’. It is derived from the Greek root ‘acro’ meaning ‘tip’ or ‘point’. So we join the tips of different words and make up another. Apart from innocuous ones like UNICEF, UNESCO, WHO, DME, SNDP, UPA, NDA etc., acronyms are most widely used by us, doctors. It is the secret weapon of medical men (and women, of course) one can say. It is a secret language, anyway.

As one enters the medical college, one is bombarded with TC, DC and ESR in Physiology and mysterious things like FTM in Biochemistry. (Anatomy is relatively free of acronyms, having instead, regal sounding names like Gluteus maximus and funny ones like Medulla oblongata). As one progresses through the clinical classes, one gets familiar with entities like PUO, AML, TAO, PPBS etc. By the time you finish those five and a half years, you are an expert ‘acronymist’.

Each specialty has its own array of them and they remain a mystery to the laity as well as colleagues in other specialties. And as soon as you manage to decipher one, they change it into something more obscure. The good old IHD of my student days is now a more politically correct CAD.

The first acronym I met as a senior house surgeon in Ophthalmology was ‘SPK’. Then a hyphenated A-R pupil to be followed by CSR, RP, RD, CRVO and a plethora of others. (By the way, one doesn’t come across SPK now a days. May be it has metamorphosed into a more exact EBPLUO [Extra Bowman’s punctuate lesions of unknown origin] or some such). I think among Ophthalmologists the Vitreoretinal specialists use the maximum and maximally complicated acronyms. Who else can complicate diabetic retinopathy into BRDM, NPDR, PDR etc. with PD and PVD thrown in for good measure? But for me the one that takes the cake is APMPPE. Wow! It sounds like the initials of a political leader from Andhra Pradesh, doesn’t it?

Not that others are far behind. Even humble General Ophthalmologists do use things like ICCE and ECCE, not to mention SICS. Recently someone mentioned TICS (T for tiny). But I take my hat off to my friend who routinely performs what he calls “LICEX”. He coined that term from Limited Incision Cataract Extraction. How can one call a three or three and a half millimeter incision as small or even tiny when people are trying out sub-one millimeter incisions, he asks. LICEX has the added advantage that it sounds suspiciously like LASER.

It seems he operated on the wife of a colleague and the husband, being a medical man asked him if he used laser for cataract extraction. He coolly replied. “No! No! I use the NMNR Technology.” Of course, the word ‘technology’ satisfied the doctor husband; but what he didn’t know that NMNR stood for non-mechanical nucleus removal, meaning he did a bi-/tri-section of the nucleus to get it out through a three millimeter section.

That convinced me that acronym was the most useful thing to come out since they invented the wheel.

So friends, S.Y.A.I.N.I.I.H. (See You All In the Next Issue, I Hope)
Local Anaesthesia In Ophthalmic Surgery

Dr. Charles K. Skariah MS

Local anaesthesia is currently preferred for many ophthalmic surgeries as it is associated with reduced morbidity and mortality, early patient mobilization, improved patient satisfaction and reduced hospital stay when compared with general anaesthesia. 90 % of all ophthalmic surgeries can be done under local anaesthesia. Local anaesthesia can be administered topically or by orbital injections.

A) Topical anaesthesia :- It is becoming increasingly popular for phacoemulsification surgery although many other procedures may also be performed topically. Procedures that can be done under topical anaesthesia in a suitable patient :-

- Application of surgical scrubs Povidone 2.5%-10%
- Tonometry
- Excision of superficial conjunctival lesions such as cysts and naevi
- Removal of foreign bodies
- Removal of sutures
- Debridement of corneal epithelium
- Corneal scrapings / biopsy in infective keratitis
- Removal of pterygia and Conjunctival autografts
- Refractive surgery
- ECCE and phacoemulsification

Although topical anaesthesia is an extremely simple technique, it may add to the complexities of surgery as operating conditions may be more challenging and it demands understanding and increased cooperation from the patient. The demands on the surgeon and the patient limit the use of topical anaesthesia to relatively short procedures. It achieves anaesthesia of cornea, conjunctiva and anterior sclera. It does not anaesthetize eye lids, posterior sclera, extraocular muscles and intraocular structures.

Pre-assessment :- Careful patient selection is essential if topical anaesthesia is to be used safely and effectively. Patient need to be co-operative and not unduly anxious. A good surgeon-patient relationship and communication facilitates the procedure. Patient must be able to lie still and be comfortable in the supine position.

Perioperative care :- Patient should be counselled well and consent obtained for surgery under topical anaesthesia. Communication with the patient while operating is a surgical skill that has to be acquired. Application of local anaesthetic drops should start 20-30 minutes before surgery. Different regimes are described but in general two or three drops are instilled every five minutes. Sufficient absorption should occur over this period to render the surface of the eye anaesthetized. As the cornea is avascular, once absorbed the local anaesthetic remains for approximately half an hour. Additional drops can be given at any stage during the operation if discomfort is experienced. The brightness of the microscope light source should be lowered to reduce photophobia and limit patient distress.

- Topical anaesthetic application alone produces detectable levels of local anaesthetic agent in the anterior chamber and provides good analgesia. However certain manoeuvres such as iris manipulation, IOP fluctuations and insertion of intraocular lens can be uncomfortable. Any discomfort is better tolerated if
the patient is fully informed preoperatively. Because visual function is maintained during surgery, the patient may be more aware of the operative procedures and some patients find this stressful and often request sedation. Short acting intravenous drugs such as midazolam are popular choices although premedication with oral benzodiazepines may be just as effective.

**Choice of topical drops**: Amides preferred over ester preparations. There is no consensus on which topical local anaesthetic eye drop provides the best analgesia. Tetracaine 0.50%, Amethocaine, Proparacaine 0.50%, Lignocaine 1-4%, and Bupivacaine 0.50-0.75% have all been used successfully. Availability may determine the clinician’s choice but it is essential that the preparation is preservative free. Small sponge soaked with the drops can be kept in the inferior and superior fornix or a ring saturated with drops can placed in the paralimbal region to maintain corneal clarity.

**Intra cameral injection**: To improve analgesia, the local anaesthetic can be injected intraoperatively into the anterior chamber of the eye. The ‘intracameral’ injection produces superior analgesia improving comfort and co-operation. The drug should be preservative free as Benzalkonium chloride is toxic to corneal endothelium. 0.5 ml of lignocaine 1% is the most popular solution used. There is no difference in anaesthetic effect between bupivacaine 0.50% and lidocaine 1% but the possibility of damage to corneal endothelium occurs with bupivacaine. Anaesthetic solution is washed out by injection of viscoelastic material after 15-30 seconds.

**Anaesthetic result**: Patient satisfaction with intraoperative analgesia after topical anaesthesia appears comparable to that of orbital injection techniques. Surgically related complication rates under topical anaesthesia are similar to conventional orbital blocks. However without akinesia an inexperienced surgeon may experience some difficulty with capsulorhexis and phacoemulsification. On the other hand the lack of akinesia may be used by the surgeon to his advantage as the patient can be asked to consciously fix or alter their gaze during the operation.

- In the event the patient becomes distressed or the procedure becomes complicated or lengthened, a subtenon block can be administered to provide a retrobulbar analgesia with akinesia.

**Post operative care**: Topical anaesthesia does not cause unwanted postoperative ptosis, or diplopia, nor does it affect the secretion of tears. As the protective mechanism of the eye is preserved it is not essential that the eye be patched. The rapid return of visual function facilitates an early discharge from the hospital.

**B) Orbital injections**

**Advantages**:
- Attains anaesthesia of conjunctiva, sclera, cornea, intraocular structures and extraocular structures
- Extraocular movements are greatly eliminated
- Superior rectus suture can be used
- No restriction to the extent of intraocular manipulation

**Disadvantages**:
- Pain & anxiety of injection
- Post operative patching of the eye is necessary

<table>
<thead>
<tr>
<th>Advantages of Topical Anaesthesia</th>
<th>Disadvantages of Topical Anaesthesia</th>
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<tbody>
<tr>
<td>1. Absence of pain of injection</td>
<td>1. No intraocular anaesthesia</td>
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<td>2. No complications of injection</td>
<td>2. Iris manipulation painful</td>
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<td>3. No temporary visual loss</td>
<td>3. Discomfort on IOL insertion &amp; IOP fluctuation</td>
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<td>4. No need to patch after surgery</td>
<td>4. Anxiety due to full awareness of surgery</td>
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<td>5. Day care procedure</td>
<td>5. No akinesia</td>
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<td>6. No lignocaine toxicity</td>
<td>6. SR suture painful / oculocardiac reflex</td>
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<td>7. No ptosis</td>
<td>7. Photophobia due to microscope light</td>
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<td>8. If on anti coagulants can be contd</td>
<td>8. Difficulty to manage complications</td>
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<td>9. No diplopia</td>
<td>9. suited for short procedures only</td>
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<td>10. No systemic side effects</td>
<td>10. SPK, corneal haze</td>
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<td>11. Needs very co-operative patient</td>
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<td></td>
<td>12. Aware of pressure and discomfort of speculum</td>
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<td>13. May need sedation</td>
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Complications like retrobulbar haemorrhage, Globe perforation, and optic nerve injury

**Anaesthetic agent:** A mixture of 5ml of 0.5% bupivacaine plus 5ml of 2% xylocaine with 1,200,000 adrenaline and 75 units of hyaluronidase (one vial / ampoule is 150 units). One ampoule of hyaluronidase can be mixed in two bottles of xylocaine. Lignocaine provides early action. Bupivacaine prolongs the efficacy. Hyaluronidase permits diffusion into orbit. Adrenaline decreases the absorption and there by prolongs the duration of anaesthesia and reduce bleeding during surgery and also prevents surge in plasma levels. Adrenaline is contraindicated in patients with hypertension, cardiovascular disease and cerebrovascular disease.

**Routes of administration**

1) Peribulbar injection
2) Parabulbar injection
3) Retrobulbar injection

Peribulbar injection: Also known as extraconal anaesthesia as the tip of the needle is outside the muscle cone. This technique is as effective as retrobulbar block and has almost replaced Retrobulbar block (Fig. 1a & b).

Procedure- The patient in supine position is asked to look straight ahead in primary position. 24-25mm long 23-26 gauge needle can be used. The needle can be inserted either trans conjunctivally or trans cutaneously at the junction of middle 2/3rd and lateral 1/3rd of lower lid. The needle is held perpendicular to the orbital margin and advanced adjacent and parallel to orbital floor for about 2.5 cm. After gentle aspiration of syringe to alleviate possible entry of needle into a blood vessel, 5ml of anaesthetic agent is injected into the lateral adipose tissue of the orbit. Injected solution diffuses into the superior, nasal and inferior compartments of the posterior orbit while inferior sulcus of the lower lid is kept compressed by the surgeon’s fingers. Filling up of the superior lid furrow and drooping of the upper lid are early signs of the block coming into action.

Ocular compression is applied for few minutes. Then just medial to medial canthus same needle is inserted to a depth of 2.5 cm and a further 3 ml is injected. Alternatively second injection may be given just inferomedial to supra orbital notch but is likely to cause more complications. Anaesthesia & analgesia begin in 5 minutes and is maximum in 15 minutes time. Supplementary injections may be necessary inferiorly for persisting inferolateral movements and superiorly to block superior and medial movements.

**Do’s and Do not’s & Tips for a good block**

- Use primary gaze position or down & out
- Avoid medial gaze as it rotates the optic nerve to injection site
- Bevel of the needle towards the globe
- Align the needle tangential to the globe
- Use fine sharp needle
- Select an avascular site
- Needle inserted perpendicular to orbital margin and parallel to floor of orbit
- Have a sound anatomic knowledge
- Avoid extraocular muscles
- If sclera is touched eyeball turns downwards
- Always maintain verbal contact with patient
- Measure axial length and extra caution applied in long myopic eyes

**Parabulbar anaesthesia / sub tenons block / pin point anaesthesia**

Anaesthetic agent is injected into posterior subtenon space bathing the nerves and muscles within the cone. A mixture of 2.5 ml of Xylocaine 2% and 2.5 ml of bupivacaine 0.5% is flushed into the sub tenon space.
by a blunt cannula after dissecting the conjunctiva under topical anaesthesia half way between inferior limbus and fornix. All four quadrants can be used but the infero nasal quadrant is preferred. This quadrant allows good fluid distribution superiorly while avoiding the area for surgery and damage to the vortex veins. Short ciliary nerves are blocked as they pass from the sub tenon space to the globe causing loss of sensation.

**Advantages & disadvantages :-** Parabulbar anaesthesia completely avoids vascular and optic nerve injury. It requires only low volumes of the drug. It provides better anaesthesia to iris and anterior segment. But it causes more postoperative morbidity in the form of chemosis and subconjunctival haemorrhage.

**Complications :-**

**Retrobulbar haemorrhage** – More common in very old patients with fragile vessels, patients with vascular and haematological disease, coagulation failure, and those on drug therapy with aspirin, NSAIDS, steroids and anti coagulants. Retrobulbar haemorrhage manifests as ecchymoses, periorbital haemorrhage, subconjunctival haemorrhage, proptosis and raised IOP. Proptosis, visual acuity, pupils, pain, IOP and arterial patency needs to be monitored in the acute phase of severe orbital haemorrhage. Surgery has to be postponed.

**Globe perforation** - Perforation is seen in myopic eyes longer than 26 mm especially those with posterior staphyloma. Poor patient co-operation, jerky movements of head, eye and face can cause perforation

**Optic nerve damage** - The tip of the needle may pierce the optic nerve and cause optic nerve damage leading to optic atrophy (Fig. 4).

**Vascular occlusion**

**Myotoxicity** - Extra ocular muscle palsies causing diplopia and ptosis

**Combined retro peribulbar block :-**

Multiple communications between extraconal and intraconal compartments have been demonstrated by CT studies using radio contrast material. Thus the injected material diffuses from one to the other. This division into extraconal, intraconal components is artificial, because the globe, extraocular muscles and the septal compartments function as a unit. and there are no anatomically discrete divisions. A combined intraconal and extraconal block may be effective when repeated peribulbar fails to take effect.
Facial nerve block:-
Orbicularis spasm affecting intraocular surgery can be alleviated by blocking the facial nerve. Facial nerve may be blocked at various sites

**Van Lint block**- Terminal branches to orbicularis muscle are blocked at the lateral orbital margin and along the superior and inferior orbital margin.

**O’Brien block**- facial nerve is blocked as it crosses the condyle of the mandible 2mm below the tragus of the ear.

**Atkinson block** – Facial nerve blocked midway between stylomastoid foramen and orbicularis oculi.

**Spaeth block** – nerve is blocked before it divides after emerging from the stylomastoid foramen.

**Systemic complications of local anaesthetic agents**
Systemic complications may be due to overdose or accidental intravascular injection or due to allergy to the drug or the preservative. Accidental injection can occur into CSF through the duramater around the optic nerve.

**Safe dose of Local anesthetic**
1. Xylocaine 2% without adrenaline – 3mg / kg (maximum 10 ml)
2. Xylocaine with adrenaline - 7 mg / kg (maximum 20 ml)
3. Bupivacaine with or without adrenaline - 2mg / kg (maximum 25 ml)

**How to minimize toxic effects**
1. Low volume at the rate of 1ml /10 second
2. Low concentration of the drug
3. Mix different LA drugs like Xylocaine & bupivacaine
4. Use adrenaline as adjuvant if not contraindicated
5. Increase threshold to local anesthetics by premedicating with diazepam or midazolam

**Signs and symptoms of toxicity**
There is no strict correlation between blood levels and toxic symptoms

**Management of Systemic complication**
1. IV cannula should be placed in all patients for any possible emergency
2. Ventillatory support with oxygen – Ambu bag, Boyle’s apparatus or ventilator
3. Ringer lactate drip if BP is low
4. Thiopentone 50-100mg, Diazepam 5-10 mg or Midolam 1-2 mg if convulsions are present

Short acting muscle relaxant scoline 1-2 mg /kg to secure the airway by endo tracheal tube

Vasopressive adrenergic agents – mephentine, adrenaline, steroids if the BP is low.

Monitor- pulse rate, BP, pulse oxymetry, ECG for rate, rhythm, ST-T changes & arrhythmias

(Midazolam is five times more potent than diazepam. Has fast onset and short action, fast and complete recovery with no hang over and amnesia. Usual dose is 0.03 – 0.05 mg / kg .The usual dose administered is 1-2 mg IV. May be repeated after half hour. In extremes of age the safe dose is 1mg.)

Oculocardiac reflex – Oculo cardiac reflex is a complication which can occur during block or surgery due to traction on extraocular muscles or pressure on the globe. It may manifest as bradycardia, bigeminy, ectopic beats, nodal rhythm, a.v block, arrhythmias, or periods of asystole. It most commonly occurs during
muscle surgery, RD surgery and enucleation. Oculo cardiac reflex may occur after retrobulbar block or retrobulbar haemorrhage. Retrobulbar block may block the reflex. But it itself has the risk to induce it.

Management- No treatment needed if reflex manifests as bradycardia or infrequent ectopic beats with the BP remaining stable. Cessation of surgery is indicated if arrhythmias become significant. Surgery may be resumed after a brief pause as oculocardiac reflex fatigue easily. Usually there is little or no activity after a brief pause in surgical stimuli. In severe cases oculocardiac reflex is managed with anticholinergic drugs like atropine or glycopyrolate. Prolonged tachy dysarrythmias may result with large doses of atropine. So caution should be exercised in giving large dose of atropine in the management of oculocardiac reflex.

Anaesthetic support in OT
Qualified anaesthesiologist should be available in all ophthalmic theatres to meet any emergency during block or surgery. Full anaesthetic back up including emergency medicine tray, Oxygen, pulse oxymeter, cardiac monitor, Boyles apparatus, defibrillator and ventilator should be made available in the OT. Operating in an OT with out full life support measures will be suicidal in these days of consumerism. The ophthalmologist should be trained in intubation and other resuscitative techniques.

Conclusion
Although ophtalmic surgery under local anaesthesia is a relatively safe technique compared to general anesthesia, one should undertake surgery only with the assistance and support of the anaesthesiologist. Atleast the anaesthetist should be available on call. It is mandatory to equip the theatre with life support measures and life saving anaesthetic equipments. It is preferable that all ophthalmic surgeons undergo training in emergency resuscitative measures.
Management of Pterygium – A Brief Review

Dr. V.K. Raju 1 MD FRCS FACS, Dr. Abhishek Chandra 2 MS, Dr. Rahul Doctor MS

Pterygium is defined as a fibrovascular growth originating from subconjunctival tissue and encroaching the cornea. Histopathologically, it shows signs of elastotic degeneration.

Typically a pterygium appears as a fleshy mass over the nasal cornea. If not treated it may encroach the entire pupilary axis and thus cause a significant decrease in the visual acuity. The contractile forces of pterygium on peripheral horizontal cornea, leads to significant flattening of the horizontal meridian (with the rule astigmatism), which is proportional to the size of the pterygium. 1,2,3

Pterygium was first described by Susruta (India), the world’s first surgeon ophthalmologist before 1000 A.D. 4,5 In Susruta Samhita he describes:

“With the patient recumbent on an operation table, the pterygium is loosened and disturbed by sprinkling powdered salt into the eye. With the patient looking laterally, a sharp hook is used to secure the growth at its loosened upturned part, and is held up with a toothed forceps, or a threaded needle is to be passed from below the part which would be held up with the thread. The pterygium is then scratched with a sharp round –topped instrument. The root of the pterygium should be pushed as under from the black outline (cornea) of the eye to the medial canthus and then excised and removed”.

The next recorded study is of Celsus (Rome 50 A.D.) where he passes a needle and thread beneath the pterygium and with a sawing motion separates the tissue 4. It was then described by Vegabhatt (India-300 A.D.), Paul Aegineta (Greek – 7th century), Al Rhazes (Arabia – 932 A.D.), Avicenna (Greek 980-1036 A.D.) and Chakradatta (India -1060 A.D.). In the nineteenth century Scarpa, Travers, Desmarres, Knapp, Klein, Prince, Boeckman, Wright, Hobby, Alt, Mackenzie and others have all suggested various methods for the treatment of pterygium.

For more than thirty centuries, man has tried to conquer this little growth. But it is still a challenge for ophthalmologists. It has been incised, removed, split, transplanted, excised, cauterized, galvanized, heated, inverted, dissected, rotated, coagulated, repositioned, irradiated, excimer lasered, stripped and grafted; but it still remains an enigma for ophthalmologists. Despite the best techniques in the hands of the greatest surgeons there have been recurrences and when they recur they are much more aggressive. Various theories have been suggested for its etiology: ultra-violet light 6,7, genetic 8,9, allergic 10, windy environment, immunological 11 and infection 12,13.

Histopathology and Histochemistry

On histopathological examination pterygium shows features of fibrovascular proliferation and elastotic degeneration 14. It has been proven in almost all the studies and therefore we do not need to send the pterygium tissue for histopathological examination. On immunohistochemistry with antibodies, the markers for smooth muscle cells are positive while it is negative for nerve sheath marker 15. The presence of these myofibroblasts causes with the rule astigmatism in patients with pterygium. These myofibroblasts can
originate from the conjunctiva or migrate from the normal fibro-adipose tissue as hypothesized by Tseng et al.

Matrix Metalloproteinases present in primary or secondary pterygium are similar to that found in Tenon’s capsule and are not implicated in the genesis of recurrent pterygium. Primary and recurrent pterygium do not show any difference in MMP-9 expression. The recurrence maybe due to the incomplete excision associated with fibroblast proliferation and production of MMP under the influence of inflammatory cytokines.

Management

A variety of surgical techniques are being used currently for the management of pterygium. Bare sclera technique has been used since times immemorial but it has a very high recurrence rate. The use of thiopeta, radiation, steroid drops, lamellar keratoplasty, excimer laser are no more used in routine practice.

The use of autologous conjunctival graft for the management of pterygium has been for more than fifty years now and even today it is the mainstay in the treatment of pterygium.

Naib K. was one of the first to introduce this technique. Starck T. et al described the technique in detail. Various studies have shown the recurrence rate with this technique from 1%–10%2,23,24. Limbal – conjunctival autograft transplantation, a modification of this technique has been utilized by many who feel that the primary cause of pterygium is hypofunctioning of limbal stem cells. There have been various reports that suggest that amniotic membrane has a comparable result in the management of pterygium27,28. The adjunct use of Mitomycin C has been shown to decrease the recurrence. The key to pterygium still lies in almost total removal of the fibro-vascular growth and large conjunctival autograft. Amniotic membrane and Mitomycin C serves important adjunct for recurrent pterygium but conjunctival autograft still remains the most accessible modality for its management. In patients with multiple recurrences a combined use of all the above modalities will give the best results.

Complications

Complications of pterygium include irritation, redness, diplopia, distortion/decrease in vision and scarring of the conjunctiva, cornea and medial rectus muscle.

Postoperative complications include infection, reaction to suture material, diplopia and scarring. Retinal detachment, vitreous hemorrhage and perforation of the globe though rare can occur.

The most common complication of pterygium surgery is recurrence. Simple Excision is associated with 50% - 80% recurrence rate. Although not usually vision threatening, a recurrent pterygium is disfiguring, uncomfortable and more difficult to treat. Without effective adjunctive therapy, there is a high risk of recurrence after repeated excisions. There is extensive data to support that the use of amniotic membrane transplantation in reducing the recurrence rates in both primary and recurrent pterygia. The surgery consists of thorough removal of abnormal tissue, restoring the matrix in the excision area through the use of amniotic membrane which provides a new basement membrane for rapid epithelialization. To further reduce inflammation, a subconjunctival injection of corticosteroids may be considered.

The main advantage of using amniotic membranes is its ability to restore large excised areas (double head or recurrent pterygia) where a conjunctival graft is not possible or in cases in which the conjunctiva is already scarred from previous surgery or has to be conserved for possible glaucoma surgery.

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Ocular Surface Disorders - Current Concepts and Management

Dr. Jayasree Menon P. MS, Dr. C.V. Anthrayose MS, Dr. Alex Joseph MS

Ocular Surface Disorders

Ocular surface disorders are a group of disorders of diverse pathogenesis in which, the disease results in failure of mechanisms that maintain a healthy ocular surface. (Fig. 1)

Ocular Surface

The ocular surface refers to the mucosal lining between upper and lower eyelids. The functional unit is comprised of tear film, conjunctival epithelium, limbal epithelium, corneal epithelium, lacrimal gland and meibomian gland (Fig. 2).

The 2 components essential for maintaining ocular surface health are

1. Healthy ocular surface epithelium and
2. Normal, stable precocular tear film

These act as a vicious cycle.

Ocular Surface Epithelium

This includes corneal, limbal and conjunctival epithelium. Of these, most important are the limbal stem cells since these maintain the dynamic equilibrium (Fig 3).

Fig. 1. OSD with unhealthy ocular surface

Fig. 2. Lacrimal gland and drainage apparatus

Fig. 3. Ocular surface epithelium

Fig. 4. XYZ Hypothesis of Thoft and Friend

The cells at the limbus are the proliferative cells. These include the SC (Stem Cells) which have the unique
property of self renewal and the TAC (Transiently Amplifying Cells) that are derived from mitotic division of stem cells and amplify by undergoing a few rounds of cell division. Cells in the non proliferative compartment are the PMC (Post Mitotic Cells) which are in the different stages of maturation by differentiation into the TDC (Terminally Differentiated Cells) 3.

This follows the limbal stem cell X,Y,Z hypothesis proposed by Thoft & Friend 4 in 1983 (Fig. 4).

**Ocular Surface Defence**

The 2 factors essential for the ocular surface defence 3 are

(1) Normal, adequate and stable tear film and

(2) Normally functioning hydrodynamic factors

The tear film consists of mucin layer, aqueous layer and lipid layer (Fig. 5).

Hydrodynamic factors include periodic, adequate and complete lid blinking to distribute an even tear film over the ocular surface and proper tear clearance to ensure adequate turnover and refreshment 3. Thus, the eyelids, the external adnexal glands and the ocular surface epithelium all play a major role in maintaining a normal tear film and ocular surface (Fig. 6).

Two neuronal reflex arcs function in this process. For both the arcs, the 1st branch of trigeminal nerve controls the ocular sensitivity as the afferent sensory input and the parasympathetic branch and the motor branch of the facial nerve are the afferent output 3 (Fig. 7).

These 2 components are interrelated. An alteration in the quantity or quality of any of the elements of the tear film can lead to an unstable tear film and secondary changes in the epithelium. Vice versa, primary changes of the ocular surface epithelium as part of ocular surface failure can lead to a secondary dry eye. Thus, an intimate relationship exists between the two and any change in this can lead to the occurrence of various ocular surface disorders.

**Ocular Surface Failure - Etiopathogenesis**

2 major types of ocular surface failure have been identified based on the epithelial phenotype in impression cytology 5.

(1) With intact limbal stem cells

(2) With limbal stem cell deficiency

**With intact limbal stem cells**

Here, the normal nonkeratinized ocular surface epithelium undergoes squamous metaplasia into keratinized epithelium 5,6. This is also associated with loss of goblet cells and mucin expression. This altered epithelial differentiation renders the ocular surface epithelium non wettable. This leads to an unstable tear film, the hallmark of various dry eye disorders.

This is usually due to poor ocular surface defense and dry eye may be secondary to 7,8,9:

(a) Aqueous tear deficiency

   Idiopathic – Age related, Hormonal
   Hyponutritional – Vit.A deficiency
Sensory denervation – After surgery or keratitis
Collagen vascular diseases – Rheumatoid arthritis, Wegeners, SLE
Sjogren’s syndrome and other autoimmune disorders
Drugs – Oral Contraceptives, Antidepressants, Antihistamines, Beta blockers
Lacrimal gland infiltration – Amyloidosis, Sarcoidosis, Tumors
Lacrimal gland fibrosis – Radiation
Contact lens use

(b) Lipid tear layer deficiency
Blepharitis
Acne rosacea
Contact lens

(c) Mucin tear layer deficiency
Vit.A deficiency
Trachoma
Mucocutaneous disorders
Contact lens
Conjunctival scarring – Ocular pemphigoid, Steven Johnson Syndrome, Chemical burns
Topical medications

(d) Increased tear film evaporation
Lagophthalmos
Ectropion
Computer vision syndrome
Lid retraction
Exophthalmos
Defective sensation

(e) Delayed tear clearance
Obstruction of tear outflow
Ineffective lacrimal pump

With limbal stem cell deficiency
Here, the normal corneal epithelium is replaced by conjunctival epithelium. The salient features here are conjunctivalisation, vascularisation, chronic inflammation, poor epithelial integrity manifested as an irregular surface, recurrent erosion and persistent ulcer, destruction of basement membrane and fibrous ingrowth. Conjunctivalization is the hallmark of limbal stem cell deficiency. Conditions with limbal stem cell deficiency can be classified in terms of the following.

(a) Primary limbal stem cell deficiency
These patients exhibit a gradual loss of limbal stem cell function over time. Seen in association with Peripheral keratitis
Pterygium/pseudopterygium
Aniridia
Neurotrophic keratopathy

(b) Secondary limbal stem cell deficiency
These patients have a clear pathogenic cause that is responsible for destruction of limbal stem cells.
Chemical / Thermal burns
Steven Johnson Syndrome
Ocular rosacea
Ocular pemphigoid
Contact lens wear
Multiple surgeries/Cryotherapies
Antimetabolite (5 FU) toxicity

Etiology – Common factors
Aging
Hormonal - Post menopausal females
Excessive computer use – reduced blinking
Excessive use of contact lens
Eye surgeries, injuries
Drugs
Keratoconjunctivitis sicca

Classification

The Madrid Triple Classification of Dry Eye
Dry eye classified depending upon 3 factors – Etiopathogenesis, Anatomo-pathologic and Severity

The features are:

A. Classification according to etiopathogenesis

The etiologic factors are divided into 10 groups:

1. Age related – With aging, all cellular structures of body undergo a progressive apoptosis including lacrimal glands. The lacrimal secretion begins to diminish from the age of 30 years and becomes insufficient for the necessities by 60 years.

2. Hormonal – Lacrimal secretion is affected by some endocrine gland activity, the most important of which are androgens, estrogens and prolactin. Aqueous and lipid secretions are the most affected.
3. Pharmacologic – Systemic- Antidepressants (Fluoxetine, Imipramine), Anxiolytics (Bromazepam, Diazepam, Clorazepate), Antiparkinsons (Bipireid, Benztropine), Diuretics (Chlorthalidone, Frusemide), Antihypertensives (Clonidine, Chlorothiazide), Anticholinergics (Atropine, Metoclopramide), Antihistaminics (Dexchlorpheniramine, Cetrizine), Antiarrhythmics (Disopyramide, Mexiletine)

Topical – Preservatives (Benzalkonium chloride, Thiomersal, Chlorobutanol, EDTA), Anasthetics (Tetracaine, Proparacaine, Lidocaine)

4. Immunopathic – Autoimmune disorders –

(1) Primary Sjogren’s syndrome – those preferentially affecting glands – where vasculitis by immune complex deposits, pseudolymphomas and lymphomas are sometimes associated.

(2) Secondary Sjogren’s syndrome which includes Rheumatoid Arthritis, Systemic Lupus erythematosis, Dermatomyositis, Scleroderma etc.

(3) Autoimmune attack of other tissues and secondary destruction of glands as in Steven Johnsons Syndrome, Ocular pemphigoid etc.

(4) Affecting other tissues – Thyroid and adrenal insufficiency


6. Dysgenetic – Genetic and congenital diseases that affect one or several types of dacyro glands – Aqueoserous (Alacrima, Dysplasia ectodermica anhidrotica), Lipid (Blepharophimosis syndrome, Keratopathy- ichthyosis- deafness syndrome, First branchial arc syndrome), Mucin (Aniridia, Bietti syndrome), Ocular surface epithelium (Meemann dystrophy, Cogan microcystic dystrophy)

7. Inflammatory/Infectious – Dacryoadenitis, Blepharitis, Trachoma, H.simplex, H.zoster

8. Traumatic – Surgical, Chemical, Radiation induced, Accidental

9. Neurologic – Lacrimal secretion is very dependent on nervous stimulation.

10. Tantalic – These patients despite having enough tears, have a dry ocular surface. There are 3 types of tantalic dry eyes:

(1) Lid-eye incongruency – Lid cannot create, maintain and reshape the tear film onto the ocular surface as in lid palsy, ectropion, lagophthalmos, lid coloboma, exophthalmos, local protrusion of pterygium or dermoid cyst etc.

(2) Epitheliopathic – Epithelial dystrophies, limbal deficiency, Corneal conjunctivalisation, Endothelial decompensation etc. makes corneal epithelium less wettable.

(3) Evaporation – Environmental conditions like hot dry climates, excessive air conditioning, open car window etc.

Most of the dry eye conditions are multifactorial

B. Classification according to damaged glands and tissues

The affected parts of the lacrimal basin may be summarized in this histopathologic classification with the acronym ALMEN;

A – Aqueoserous deficiency
L – Lipid deficiency
M – Mucin deficiency
E – Epithelial deficiency
N – Nondacryologic exocrine deficiencies

C. Classification according to severity

In the initial Madrid classification severity of dry eye was divided into 5 grades:
Subclinical – Symptoms only when overexposure
Mild – Habitual symptoms
Moderate – Symptoms plus reversible signs
Severe – Symptoms plus permanent signs
Disabling – All of the above plus visual incapacity

**Recent Triple Classification of dry eye for practical clinical use**

In 8th congress of the International Society of Dacryology and Dry eye (Madrid, April, 2005), the previous Triple classification of dry eye approved in the XIV congress of European Society of Ophthalmology (Madrid, June, 2003) was modified.

Here, classification according to etiopathogenesis and affected glands and tissues are retained. Classification depending on severity was modified into 3 grades for practical use

1. **Grade 1 or Mild – Symptoms without slit lamp signs**
2. **Grade 2 or Moderate – Symptoms with reversible signs**
3. **Grade 3 or Severe – Symptoms with permanent signs.**

**Goals of Therapy**

Major goals include:

1. Supplementation of a deficient tear film
2. Preservation of the available tear by re-establishment of lid motility with normal lid-corneal congruity.
3. Supplementation of limbal tissue containing epithelial stem cells for the management of epithelial disease of cornea.
4. Improvement or supplementation of a basement membrane substrate.
5. Restoration of clear visual axis.

**Treatment Guidelines**

**A. Medical**

Along with tear substitutes, it is important to treat co-existing lid disease like blepharitis, trichiasis and meibomian gland dysfunction as well as to preserve the available tear by punctual occlusion.

**1. Tear substitutes and Lubricants**

Lubricants act as a physical means of protecting already compromised ocular surface from desiccation and irritation, but the preservatives like benzalkonium chloride may counteract the benefit. Hence preservative free lubricants are preferable although more expensive.

**Guide to various types of therapy for specific tear film abnormalities**

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<td>Excimer Photo Therapeutic Keratoplasty</td>
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**2. Nutritional Supplements**

Boerner et al found 98% patients reported improvement in the symptoms with omega 3 supplementation. Omega 3 fatty acids produces anti-inflammatory eicosanoid acid which suppress inflammation by blocking the gene expression of pro inflammatory cytokines.

**3. Tear Stimulants (Secretagogues)**

(a) **Diquafosol tetradosium**

This molecule acts as a uridine nucleotide analogue that acts as a agonist of P2Y2 receptor present on ocular surface. Diquafosol increases water transport via chloride channel activation and enhances nonglandular secretion of tear fluid. The drug is under clinical trial.

(b) **Pilocarpine**

Pilocarpine is a cholinergic parasympathomimetic that binds to muscarinic M3 receptors and stimulates salivary and lacrimal glands.

(c) **Cevimeline**

Cevimeline is an acetylcholine analogue and has high...
affinity for muscarinic M3 receptors of salivary and lacrimal glands.

(d) Eledoisin
Eledoisin is an endecapeptide which when applied locally has secretostimulant effect.

(e) Mucinous stimulators
Bromhexine and N-acetylcysteine are stimulants of mucin production. Topical medicines such as geranyl farnesylacetate and hydroxyeicosatetraenoic acids have been introduced which improve the epithelium and stimulate the goblet cells.

B. Suppression of Inflammation

(1) Corticosteroids
Topical corticosteroids are beneficial for ocular surface defects associated with intense inflammation as in Sjogrens syndrome, Pemphigoid, Steven Johnsons Syndrome. The anti-inflammatory effect of corticosteroids are mediated through stabilization of the cytoplasmic and lysosomal membranes, thereby reducing the release of inflammatory mediators and inhibiting chemotaxis. However, careful monitoring of these patients is essential to watch out for steroid related complications.

(2) Non steroidal anti-inflammatory agents
Antiinflammatory agents like salicylates, indomethacin, flurbiprofen and progestational steroids reduce inflammation without suppressing wound repair.

(3) Immunosuppressives
Immunosuppressants like antimetabolites have been effective in treating ocular surface disorders of autoimmune origin like rheumatoid arthritis, Systemic Lupus Erythematosus and ocular cicatricial pemphigoid

C. Limitation of Tissue Destruction

1. Tissue Adhesives
Tissue adhesives like isobutyl cyanoacrylate have been used as an adjuvant in the management of corneal ulcers and small perforations. It gives structural support and can arrest further stromal loss. Early application of tissue adhesive in the management of stromal melts, can postpone or reduce the need for keratoplasty or conjunctival flaps.

2. Mechanical protection of corneal epithelium
Methods:
(a) Taping of lids
(b) Tarsorraphy
(c) Botulinum induced ptosis
(d) Therapeutic soft contact lens
This is useful to protect the loosely adherent remaining transient amplifying cells or regenerating epithelium from the action of blinking eyelids has significantly improved the management of persisting epithelial defects. Soft contact lenses are undesirable in dry eye patients because of the high risk of infection. Only silicone provides adequate oxygen transmission for continuous wear, however Omafilcon A (proclear), a novel biomimetic, 59 % water content hydrogel soft contact lenses for daily wear has been found to give better comfort.

3. Promotion of epithelial wound healing and differentiation

(a) Topical autologous serum
Topical autologous serum not only moistens the ocular surface, but also provides necessary tear proteins such as epidermal growth factor (EGF), vitamin A, transforming growth factor-B (TGF-B), fibronectin and other cytokines. The effect of EGF is primarily on epithelial wound healing whereas fibronectin appears to be involved in stromal healing.

(b) Topical retinoids
These are essential for epithelial growth and differentiation. All trans retinoic acid 0.05 % in Vaseline BD has shown to increase the epithelial healing rate. Retinol palmitate ophthalmic solution has also shown an increase in goblet cells and non keratinized cells.

(c) Topical trisodium citrate 10 % and sodium ascorbate 10 %
These have been found to reduce the incidence of ulceration and perforation in the immediate treatment of alkali burns.

(d) Topical Cyclosporin A 0.05 % & 0.1 %
Its efficacy may be due to an immunomodulatory and anti-inflammatory effect on the ocular surface, thus
facilitating ocular surface healing. This has been used in severe forms of dry eye where long term use of corticosteroids are required.

D. Preservation of natural tears

(a) Punctal occlusion

Punctal and canalicular closure increases mainly the aqueous component of natural tears but also has secondary beneficial effects on goblet cell density, tear film stability, and tear osmolality. This also increases the retention of artificial tears.

(1) Thermal occlusion

(i) The hot cautery method utilizes the direct transmission of heat from a hot probe to produce a controlled burn injury to the punctual opening. It is important to treat not only the surface of punctum, but also to insert the tip of cautery gently into the proximal lumen to achieve a more effective and permanent closure.

(ii) Diathermy utilizes 455 kHz to 100 mHz radiofrequency energy to heat the tissues in the area of punctual opening and proximal lumen. A fine needle electrode is introduced into the canaliculus through the punctum and the electromagnetic current is activated until the surrounding tissues blanch and contract.

(iii) Argon laser photocoagulation has a shorter duration of effect compared to thermal cautery. Here, the punctual opening is first encircled with laser spots and then additional spots are delivered into the punctum itself.

(b) Punctal obstruction

Lacrimal punctum and canaliculi may be occluded temporarily or permanently with tissue glue or implanted foreign bodies. Temporary obstructive procedures are useful in assessing the beneficial effects of lacrimal obstruction prior to restoring to permanent occlusion.

(i) Glue

Cyanoacrylate tissue adhesive or the more recent fibrin surgical glue may be applied to the punctal opening or proximal canaliculus using 25-27 G canula or needle. Occlusion with glue lasts for only several days to week since the epithelial cells lining the lumen slough during the natural cell turnover cycle.

(ii) Absorbable implants

Collagen implants are the widely used absorbable implants. They degrade over 3-7 days, although total degradation takes up to 14 days. Catgut (2-0) or chromic catgut (4-0) sutures are alternative absorbable implants.

(iii) Non absorbable implants and plugs

Non absorbable implant materials include polyethylene, silicone and acrylic (Fig. 8).

Newer one is hydrophobic acrylic which is heat responsive and its physical dimensions undergo transition at temperatures above 320 °C. No sizing of punctal opening is required because one plug size fits all puncta before heat activation.

(iv) Surgical procedures

These are indicated in multiple punctal occlusion failures. Methods include

(i) Punctal hot cautery and suturing with nylon.

(ii) Vertical canaliculus sutured with a single 8-0 polyglactin full thickness eyelid mattress suture.

(iii) Surgical laceration of horizontal canaliculus medial to the punctum on the eyelid margin, thermal cauterization of the exposed canalicular and punctal surfaces and suture closure of both the canaliculus and punctum.

(iv) Medial tarsorraphy

(v) Bulbar conjunctival autograft from one of the fornices can be sutured as a patch over the punctal orifice after surrounding epithelial tissue is excised.
(vi) Translocation of punctal orifice anteriorly to eyelash line.

(vii) Cisternoplasty- Creating an additional skinfold at the outer angle of the eye which acts as a natural reservoir for the tears.

A. Surgical

There are many surgical approaches for treating ocular surface disorders. It is important to control inflammation before surgery, correct the precipitating problem and give prophylaxis for postoperative inflammation. Methods to restore the ocular surface epithelium include conjunctival transplantation and limbal stem cell transplantation. Amniotic membrane transplantation has been used to restore the stromal environment by replacing basement membrane for epithelial cells and stromal matrix for mesenchymal cells. Other strategies to improve basement membrane include anterior stromal puncture, excimer photo therapeutic keratectomy and lamellar or penetrating corneal grafting.

Limbal Stem Cell Deficiency

Limbal stem cells being the source of newly generated corneal epithelial cells, any injury to them can cause severe derangement of the ocular surface especially the corneal surface leading to limbal stem cell deficiency which is a serious threat to vision. The definitive treatment of limbal stem cell deficiency would be to replace those abnormal limbal stem cells with healthy one.

Algorithm of Limbal Stem Cell Deficiency Management

![Algorithm of Limbal Stem Cell Deficiency Management](image)

{LSCD-Limbal stem cell deficiency, CLAU-Conjunctival limbal autograft, KLAL-Keratolimbal allograft, Lr-CLAL-Living related conjunctival allograft, Cu-LAU-Cultured limbal autograft, Cu-LAL-Cultured limbal allograft}

Preparation of bed

Under peribulbar anesthesia, 360° peritomy is performed 3-4mm from limbus with removal of abnormal epithelium, pannus and symblephara and bleeding points are cauterized.

Amniotic Membrane Transplantation

Clinical properties of amniotic membrane
1. Aids tissue epithelialisation
2. Reduces inflammation
3. Reduces vascularisation
4. Reduces scarring
5. Diminishes pain
6. Protects against infection

Indications for Amniotic Membrane Transplantation

1. Reconstruction of conjunctival surface
   (a) After resection of extensive lesions (tumours, scars)
   (b) Symblepharon reconstruction
2. Reconstruction of corneal surface
   (a) Persistent epithelial defects
   (b) Partial limbal stem cell deficiency
   (c) Total limbal stem cell deficiency (prior to limbal transplantation)

Other uses of Amniotic Membrane
1. Cultivation of limbal stem cells
2. Carrier for cultivated limbal stem cell transplantation

Limitations of Amniotic Membrane
1. Absolute deficit of limbal stem cells
2. Severe stromal necrosis
3. Severe neurotrophic changes
4. Severe ischaemia
5. Absence of tear film

Surgical technique

After preparation of the bed, a processed and preserved amniotic membrane in Dulbecco’s modified Eagles medium and glycerol at -80 °C is spread over the ocular
surface with the epithelial side up. The amniotic membrane is sutured with 6-8 circumferential 10-0’ nylon monofilament interrupted sutures at the limbus and with 8-0’ polyglactin sutures in the periphery with the conjunctival edge.

**Conjunctival Limbal Autograft (CLAU)**

**Indications**
1. Partial / Unilateral Limbal Stem Cell Deficiency
2. Reconstruction of ocular surface following pterygium excision, excision of large tumours, symblepharon

**Complication**
Iatrogenic donor site Limbal Stem Cell Deficiency

**Surgical technique**
Donor tissue can be obtained from the same eye (ipsilateral CLAU) or from the other eye (contralateral CLAU). After peritomy, a non-contiguous 6 clock hours (3 superiorly & 3 inferiorly) of donor tissue is harvested, the size being 4+4 mm conjunctiva with the limbus including 1mm of superficial clear corneal stroma. These should be placed with the epithelial side up and the limbal area of the donor near the limbus which are secured with 2 circumferential sutures 10-0’ nylon monofilament and conjunctival part with 8-0’ polyglactin.

**Cadaveric Keratolimbal Allograft (KLAL)**

Being an allogenic tissue, immunosuppressants are mandatory to prevent immunological rejection.

**Indications**
1. Bilateral Limbal Stem Cell Deficiency
2. Total Limbal Stem Cell Deficiency in one-eyed patients
3. Total Limbal Stem Cell Deficiency where live related donor or cultivated limbal stem cells are not available

**Advantage**
1. Easy availability
2. Repeatability

**Surgical technique**
Cadaver donor tissue obtained from young patients below 50 yrs within 72 hrs of death. Either multiple limbal lenticules of partial thickness or an annular rim of peripheral cornea and limbus of 1/3-1/2 thickness dissected and secured to the limbus with 10-0’ nylon monofilament. Simultaneous AMT or PKP from the same donor can also be performed after KLAL.

**Live- related conjunctival limbal allograft (Lr-CLAL)**

**Indications**
1. Bilateral total Limbal Stem Cell Deficiency
2. Total Limbal Stem Cell Deficiency in one-eyed patients
3. Severe Ocular Surface Disorders as in Steven Johnsons Syndrome, Ocular cicatricial pemphigoid, severe chemical burns

**Surgical technique**
The related donor usually 1st degree relative should be screened for potential blood borne infectious diseases including Hepatitis B and C and HIV 1 & 2. HLA typing is performed preoperatively to find the best match. Technique is similar to CLAL and not more than 6 clock hours should be harvested.

**Post operative regimen**
Topical corticosteroid 3-4 times daily.
Topical antibiotics till the epithelium has healed (1-3 wks).
Maintenace- Tapered dose of topical corticosteroid and lubricants.
Systemic Prednisolone 1mg/kg/day, with a slow taper over 3-6 mths.
Systemic immunosuppression with oral Cyclosporine A or FK 506.

**Cultivated Limbal Epithelial Transplantation**

It is the most recent and promising technique of limbal stem cell transplantation. It can be an autograft (ipsilateral or contralateral) or allograft from a live related donor.
Indications

Autograft: Unilateral Limbal Stem Cell Deficiency
Bilateral Limbal Stem Cell Deficiency with partial
Limbal Stem Cell Deficiency in one eye
Allograft: Bilateral Limbal Stem Cell Deficiency
Total Limbal Stem Cell Deficiency in one eyed patient

Surgical technique

Limbal biopsy

2×2 mm limbal tissue with 1mm into clear corneal stromal tissue at the limbus is excised and transported in human corneal epithelial medium to the tissue culture laboratory.

Cultivation of epithelia

The shredded limbal tissue bits are explanted over the central 10mm of a 3×4 cm, de-epithelialized, preserved human amniotic membrane which is the most widely used substrate for cultivation of limbal stem cells. The cells are cultured using human corneal epithelial cell medium with 10 % foetal bovine serum or autologous serum. The growth is monitored daily and medium changed in 2 days. The culture is maintained for 10-15 days, by which time a confluent monolayer of limbal epithelial cells are grown.

Transplantation

After preparing the bed, the human amniotic membrane with the monolayer of cultivated limbal epithelial cells is transplanted on the recipient cornea.

Post operative immunosuppression is required for allogenic transplants.

Other modalities

 Conjunctival flap

In conditions such as neurotrophic keratitis and sterile stromal ulceration as in chemical burns, a conjunctival flap supplies the necessary vascularity to reverse ischaemia related complications.

Buccal Mucous Membrane Transplantation

Used in eyelid position abnormalities caused by cicatrisation as in SJS, OCP, bilateral fornix reconstruction.

Keratoprosthesis

Artificial corneas are recommended in heavily scarred and vascularised corneas and in severely blind dry eyes. Osteo-odonto keratoprosthesis is a recent development in this field.

Phototherapeutic keratectomy (PTK)

Excimer laser PTK is beneficial in treating recurrent corneal erosions and persistent corneal epithelial defects by improving the basement membrane.

Guide to Dry Eye Management

First step

- Mild cases : Lubricants and artificial tear supplements- eyedrops and gels
- Moderate- Severe cases : Lubricating ointments
- Severe : Patch with lubricating ointments
  Artificial tear inserts
  Topical steroids

Intermediate step

- Temporary punctual occlusion with collagen or silicone
- External tarsorrhaphy
- Botulinum toxin induced ptosis

Final step

Very severe cases :

- Cyanoacrylate glue tissue adhesive for closure of perforation/ descemetocele
- Corneal or corneoscleral patch/ conjunctival flap for impending/ frank perforation
- Lateral tarsorrhaphy in facial nerve palsy, trigeminal nerve lesions or severe exophthalmos
- Amniotic membrane graft
- Limbal stem cell transplantation

References

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Bacterial Keratitis

Dr. K.V. Raju MS

Introduction
Microbial keratitis is due to proliferation of any of the microorganisms on the cornea. Bacterial Keratitis is the most common cause of suppurative corneal ulceration. It is the duty of the clinicians to identify both the systemic and local risk factors and to find out the aetiology of each case. Laboratory investigations have to be considered in selected cases and treatment plan may be implemented accordingly. The therapeutic plan has to be changed if there is no response. The outcome depends on the proper treatment and the virulence of the organism. Surgery should be considered if medical therapy fails to stop the progress of the ulcer or vision is lost due to scarring and opacity.

Risk Factors
Cornea is normally protected by the natural defense mechanisms. They are eye lids, tear film, corneal epithelium, and normal ocular flora. Eye lids protect the cornea by providing a physical barrier and by resurfacing the pre-corneal tear film due to blink. Normal amount of tear secretion is 1 to 2 microlitres per minute and it protects the cornea due to the presence of enzymes like lysozyme, bactoferrin, betalysins and ceruloplasmin. Intact corneal epithelium is another normal defense barrier. Only few organisms like Coryne bacterium diphtheriae, Nisseria gonorrhoeae, Haemophilus aegyptius and Listeria monocytogenes can invade the normal corneal epithelium. If epithelium is destroyed due to injury, contact lens wear or any surgery, there is possibility for bacterial infections. Inappropriate use of topical antibiotics may change the normal conjunctival flora, and may cause opportunistic organisms to attack the cornea.

Local risk factors for keratitis are bullous corneal oedema from corneal injury or surgery, absence of corneal sensation from herpetic corneal infection or topical anaesthesia abuse and local immunosuppression from prolonged use of topical steroids. Other factors are use of contaminated water for eye wash, and, contaminated eye drops. Various lid problems like lagophthalmos, entropion, ectropion, trichiasis, dry eye, and contact lens wear are other local risk factors for bacterial keratitis. All type of contact lenses like hard, gas permeable, soft, disposable, and cosmetic lenses may predispose for microbial keratitis. Aphakes, patients with a corneal transplant, and patients wearing a bandage contact lens for chronic keratopathy are at high risk for bacterial keratitis. Ocular pemphigoid, Steven-Johnson syndrome, atopic keratoconjunctivitis, chemical injuries, and Vitamin-A deficiency may produce ocular surface disorders and hence can cause bacterial keratitis. After keratoplasty, and LASIK, there is possibility for bacterial corneal infection. (Fig 1-8 explain the various risk factors for corneal ulcer)

Aetiologic Agents
Bacterial keratitis may be due to any organism, and from the clinical features the organism cannot be identified, but we can keep a strong suspicion based on various clinical presentations. In an uncompromised cornea, the microorganism may be Pseudomonas aeroginosa, Streptococcus pneumoniae, Moraxella, Beta Haemolytic Streptococcus, and Klebsiella pneumoniae.
Fig 1: Acute conjunctivitis

Fig 2: Spring catarrh (palpaberal type)

Fig 3: Spring catarrh (Bulbar type)

Fig 4: Blepharitis

Fig 5: Chronic Dacryocystitis

Fig 6: Hordeolum Internum lower lid

Fig 7: Corneal Abrasion

Fig 8: Lagophthalmos
In a compromised cornea, the organisms which can be suspected are Staphylococcus aureus, Streptococcus epidermidis, Alpha Haemolytic Streptococci, Beta Haemolytic Streptococci, Proteus, Enterobacter Aerogenes, Escherichia and Nocardia. If a patient wearing contact lens develops keratitis, the suspected organisms are Pseudomonas aeruginosa, Staphylococcus aureus, Staphylococcus epidermidis, E. coli, Klebsiella, and Proteus.

**Clinical Examination**

Detailed history and clinical examination is necessary for proper diagnosis. Most important is the history of any injury and any previous surgery, or chronic use of any topical steroids and antibiotics. History of unilateral watering and discharge is a clue to chronic Dacryocystitis. Contact lens wear has to be enquired and if present, further details like what type of lens and duration of the wear has to be enquired. History for symptoms of dry eye also has to be evaluated. Examination should not be restricted to the eye only and systemic orientation should be there. Basic illness like diabetes and infectious diseases or malignancy has to be suspected. Incidence of CP angle tumour presenting with intractable corneal ulcer has been reported.

Ocular examination should include visual acuity, torch light examination and slit lamp biomicroscopy. Visual acuity testing may be difficult due to photophobia, chemosis and oedema of lids, but it has to recorded for further evaluation of prognosis. Vision and posterior segment examination in the unaffected eye also has to be assessed. Torch light examination is for evaluation of the eye lids, conjunctiva, sclera, lacrimal sac, ulcer proper, anterior chamber, iris, pupil and lens. Position of the ulcer on the cornea, and condition of the surrounding area also has to be examined. Using slit lamp look for the depth, width, floor, and margin of the ulcer. By retroillumination, presence of oedema and the endothelial aspect of the ulcer has to be evaluated. Presence of microperforation, small hypopyon, and signs of early iritis also has to be looked for. Condition of the surrounding cornea is examined for corneal oedema and satellite lesions.

**Clinical Presentation**

Clinical presentation and symptoms are variable and they depend on virulence of the organism, duration of infection, pre-existing corneal conditions, immune status of the patient, previous use of antibiotics or corticosteroids. If the virulence of organism is more and the immune status of the patients is poor, the clinical signs will be severe and acute. Another factor which decide the clinical presentation is contact lens wear. Infection associated with contact lenses are multifocal. Epithelial and stromal infiltration is more diffuse. Usual clinical signs are conjunctival congestion, chemosis, lid oedema, decreased vision, pain, tearing, photophobia, and purulent discharge. Conjunctival reaction will be close to the limbus. Corneal epithelium becomes ulcerated and there will be infiltration which may be grayish white and necrotic. Infiltration and oedema of cornea may be seen away from the main lesion. Stromal abscess may be seen with deep infiltration with relatively clear stroma and intact epithelium. Anterior chamber reaction may lead to hypopyon, and fibrin plaques may be seen on the endothelium. The ulcer is considered to be severe, if the lesion progresses rapidly, has an infiltration dimension larger than 6 mm, involve deeper than one-third of corneal thickness and presents with impending perforation, or has scleral involvement. The organisms that produce severe and rapidly progressive ulcers include S. Aureus, S. Pneumoniae, Beta Haemolytic Streptococcus and P. Aeruginosa. Less severe and slowly progressive ulcers are usually caused by organisms such as coagulase negative Staphylococcus, S. Viridans, Actinomyces, Nocardia, Moraxella, and Serratia.

Hypopyon is due to outpouring of fibrin and polymorpho nuclear leukocytes from the vessels of iris and ciliary body. Usually hypopyon is sterile as long as Descemet's...
membrane is intact. Hypopyon may be there in any of the bacterial infections but it will be severe and more common in infection with S. Pneumoniae and Pseudomonas.

**Specific Bacterial Ulcers**

From the clinical presentation we can suspect the presence of some organisms, but it has to be confirmed by laboratory investigations.

**Ring Ulcer**

It is severe form of ulcer seen around the cornea. It may be due to haematogenous spread or penetrating injury. The organisms like Pseudomonas, Streptococcus, Listeria, and Proteus has to be suspected.

**Infectious Crystalline Keratopathy**

The organisms responsible for this condition are S.Pneumoniae, S. Epidermidis, Haemophilus, Peptostreptococcus, and Candida. The stromal opacity is crystalline in appearance, resembling a snowflake, and not associated with cellular infiltrate or other sings of inflammation. Infection usually occurs within a corneal graft in the midstroma with clear stroma superficial to it. The epithelium will be intact.

**Staphylococci**

They produce acute purulent infection. Severe pain, photophobia, and decreased vision are seen in early cases. There is tendency to spread towards the centre of cornea. The edge of the ulcer may be undermined and covered by over hanging tissue.

**Streptococci**

Ulcer due to S. Pneumoniae occurs after corneal trauma, chronic dacryocystitis, or filtering bleb infection. The ulcer will be acute, purulent, and rapidly progressive with deep stromal abscess. Anterior chamber reaction will be severe with marked hypopyon and retrocorneal fibrin coagulation. Perforation secondary to ulcer is common.

**Nocardia**

Nocardia asteroides produce indolent ulcer after minor trauma with exposure to contaminated soil. Ulcer usually waxes and wanes. The characteristic features of Nocardia keratitis include raised, superficial pin head like infiltrates in a wreath like configuration. Keratitis may simulate mycotic infection.

**Non Tuberculous Mycobacteria**

They produce slowly progressive keratitis usually after removal of corneal foreign body, trauma, or following corneal surgery particularly after LASIK. The symptoms are delayed in onset and severe ocular pain develops 2 to 8 weeks after exposure to the organism. Lesion can be solitary or multifocal with variable anterior chamber reaction. Lack of response to conventional antibiotic therapy is usually a clue to the diagnosis.

**Pseudomonas Aeroginosa**

It causes a rapidly spreading ulcer which can extend to twice its size in 24 hours and perforation can occur within 2-5 days. Ulcer will be either central or paracentral. Dense stromal infiltration and necrosis are characteristic. There will be oedema of surrounding cornea, posterior corneal stromal folds, endothelial plaques and hypopyon. Diffuse epithelial graying or a “ground glass” appearance is noted in the nonulcerated area of the cornea. A copious mucopurulent discharge with a greenish colour is seen. Early desemetocele formation, melting and perforation may occur. Ulcer may spread to sclera and in those cases prognosis will be bad.

**Moraxella**

They cause corneal ulcer in compromised hosts, particularly alcoholics, diabetics, malnourished and other debilitated patients. Ulcer is indolent paracentral or peripheral, usually oval in shape and localized with an undermined necrotic edge. It progresses deep into stroma and in untreated cases may perforate. Hypopyon may be present and the anterior segment is highly inflammed.

**Neisseria**

N. Gonorrhoea and N. Meningitides can invade the cornea and conjunctiva through an intact epithelium and cause an ulcer. They are dangerous especially in newborns because they may lead to perforation. Marked purulent discharge, congestion and chemosis usually are present.

**Laboratory Investigations**

Laboratory investigations include corneal scraping for culture and sensitivity. Majority of cases will resolve
with empirical therapy and managed with out smears or cultures. Cultures are indicated in severe cases with corneal infiltrate that is large and extends to middle stroma, is chronic in nature or unresponsive to broad spectrum antibiotic therapy. It is also indicated to exclude the presence of fungus, amoeba, and myobacteria. Cultures are useful for the modification of the therapy in patients with poor clinical response to empirical treatment.

**Culture**

Scraping for corneal material is performed after putting local anaesthetic drops. Proparacaine hydrochloride 0.5% is preferred because of it’s minimal inhibitory effect on microorganisms. Specimen is collected using a heat sterilized spatula (Kimura spatula), No. 15 Bard Parker blade, large gauge disposable needle or sterile cotton swab. If deep stroma is involved a small trephine can be used to get adequate specimen. Multiple samples has to be collected from the advancing margins of the ulcer. Specimen should be directly inoculated into the culture media. Blood agar, chocolate agar and thyoglycollate broth are the standard medias used for bacterial keratitis.

**Stains**

By examining the stained smears of corneal scraping various organisms can be identified. Gram stain is the one routinely used and it can confirm the presence of microorganisms with a sensitivity of 55-79 %. It can also distinguish bacteria from fungi. Giemsa stain also is useful to distinguish bacteria, fungi and Acanthamoeba. Chlamydia inclusion bodies can be identified with Giemsa stain. Cabolfusin, or Ziehl-Neilsen acid first stains are for identification of Mycobacterium, Actinomyces, or Nocardia.

**Other Diagnostic Methods**

**Corneal Biopsy**

It is indicated in partially treated or unresponsive corneal ulcers or if culture negative for more than one occasion. It is also indicated if the infiltrate is in the mid or deep stroma with normal overlying tissue. Under topical anaesthesia, a small trephine or blade is used to excise a small piece of stromal tissue. The specimen should be at least 1-2 mm in diameter, and the edge of the ulcer should be included. One portion may be taken for biopsy and another for culture. In cases of deep corneal abscess with overlying clear cornea the biopsy should be taken from below a lamellar flap.

Impression cytology, polymerase chain reaction (PCR), and confocal microscopy are other rare diagnostic tools. Confocal microscopy is a new and non invasive procedure in which four dimensional view of internal structures are possible at cellular level.

**Specific Therapeutic Agents**

**Cephalosporins**

Cephalosporines contains betalactum ring that is necessary for bactericidal activity. Cefazolin with an excellent activity against grampositive organisms and minimal toxicity after topical administration is a widely used first generation cephalosporin.

**Glycopeptides**

Vancomycin is a glycopeptide antibiotic with activity against penicillin resistant Staphylococci. It is active against gram positive organisms.

**Aminoglycosides**

They have bactericidal action against aerobic and gram negative bacilli. Tobramycin, gentamycin and amikacin are the commonly available aminoglycosides. They are active against pseudomonas.

**Macrolides**

They are Erythromycin, azithromycin, clarithromycin and roxithromycin. They have broad spectrum of activity against gram positive and gram negative organisms.

**Fluoroquinolones**

The second and third generation fluoroquinolones ciprofloxacin, ofloxacin and levofloxacin are commercially available for ophthalmic use. Now the fourth generation fluoroquinolones like gatifloxacin and moxifloxacin also are available. They are active against aerobic gram negative and gram positive bacteria.

**Strategies for Initial Management**

Antibiotics can be used as either drops or ointments. Subconjunctival injection has to be considered if the ulcer is severe and extends to sclera. Systemic antibiotics are not necessary in routine cases. It has to
be considered if the infection spread to intraocular structures. Soft contact lenses soaked in antibiotics may sometimes be useful. For severe ulcers a loading dose every 5 to 15 minutes for first hour followed by application every 15 minutes to 1 hour can be considered. For less severe cases dose can be decided in each case. For selection of antibiotics any of the following approach can be considered.

**Culture Guided Approach**

Corneal scraping for staining and microbiological culture are performed in all cases and treatment will be started only after that. This option can be implemented only where cornea sub specialities are functioning and they deal with only referred patients. Those patients may have severe presentation caused by unusual organisms. Major inconvenience of this approach is cost. If the patient is on treatment with antibiotics it should be stopped for 12 to 24 hours before taking the specimen for culture.

**Empirical approach**

Treatment is started based on pre-existing culture and sensitivity data without obtaining corneal specimens from the patient. Broad spectrum antibiotics has to be started. Either single drug or fortified preparations can be considered.

**Case based approach**

In selected severe cases only culture and sensitivity is tested. In routine cases treatment will be started with broad spectrum antibiotics. Culture will be taken for ulcers affecting visual axis, large and deep ulcers, and those ulcers developed after trauma with contaminated vegetable materials. For small and peripheral ulcers treatment is started without any investigations. This approach is more practical in the management of corneal ulcer.

**Selection of Antibiotic and Initial Management**

Based on any of those approach the antibiotics can be selected. The frequency of instillation of the medicines has to decided based on the severity of each case. For more severe ulcers topical preparations should be given every 15 to 30 minutes and then hourly for 6 hours for adequate loading dose. After the loading therapy frequent and regular administration is necessary to reach a sustained therapeutic level. If the ulcer is responding the dose can be gradually tapered to reduce the medication induced complications. Prolonged intensive therapy may induce epithelial toxicity and delay corneal healing. Subconjunctival infection has to be considered in those ulcers spreading to sclera and cases of impending perforation. Systemic antibiotics are indicated if there is either intraocular or scleral spread of infections. In severe cases with impending perforation or frank perforation also systemic antibiotics has to be considered. Cycloplegic agents should be used to reduce pain, congestion and to decrease the formation of synechiae. Single drug therapy with either third or fourth generation fluoroquinolone is as effective as combinations therapy. Fortified preparations can be considered if there is no positive response with commercially available preparations.

**Modification of therapy**

The clinical response after 48 hours has to be evaluated, and modification of therapy if necessary should be done. Signs of improvement are reduction of pain, consolidation and sharp demarcation of the ulcer, decrease in density of the infiltration, reduction of stromal oedema, reduction in the anterior chamber inflammation and re-epithelialization. If there is no positive response therapeutic regimen may be modified based on the results of culture and sensitivity. If there is signs of improvement with initial therapy it need not be modified based solely on culture and sensitivity results.

**Follow up**

After one week the response has to be evaluated and if the ulcer completely heals, medication can be discontinued. If the ulcer is still progressive the medication should be stopped at least for 24 hours and microbiological work up may be repeated. Special staining or culture media or corneal biopsy may be required in those cases. Non infectious causes or rare organisms like mycobacteria, Nocardia, or Acanthamoeba should be suspected. If there is no response with antibiotics other reasons like drug toxicity, and possibility of local and systemic risk factors has to be considered. Non healing ulcers sometimes can be improved by debridement of necrotic corneal stroma, frequent lubrication and temporary tarsorraphy.
Adjunctive therapy

Additional treatment is necessary if there is impending or frank perforation or if there is signs of endophthalmitis. Impending perforation is suspected if there is thinning of the cornea and descematocele. Frank perforation is suspected if the anterior chamber depth is suddenly reduced and a black discolouration is seen at the base of the ulcer. Black discolouration may be due to incarceration, iris or pigments, in the base of the ulcer. Sudden relief of severe pain is the clinical symptom of frank perforation. Pressure bandage, cyanoacrylate tissue adhesives, or bandage contact lens application are the modifications to be introduced at this stage.

Surgical Management

Conjunctival flap

This is to save the eye ball and is indicated in non-healing corneal ulcers. In peripheral ulcers flap can be placed without compromising vision. Flap can bring blood vessel to the infected area promoting healing and provide a stable surface covering.

Penetrating keratoplasty

If the ulcer is not responding to any type treatment this surgical option is indicated. It is also indicated in ulcers involving limbus with scleritis, impending and frank perforation. Intensive antibiotic administration for 48 hours before surgery will minimize the risk of recurrence and endophthalmitis.

Summary and Conclusions

One of the major cause for corneal blindness is contributed by Microbial keratitis. The incidence of corneal blindness can be reduced if the disease is identified and managed early. Evaluation of each case should start with history of any trauma, and mode of onset. All the system has to be closely examined to exclude or establish the presence of any systemic risk factors. Then search for any local risk factors and closely observe the ulcer for a clinical diagnosis. At this stage the surgeon should built a clinical conclusion that he is dealing with Bacterial, Fungal, Viral or Parasitic keratitis. Laboratory confirmation is not required in all cases but in severe and selected cases specimen is collected for further investigations. Based on clinical evaluation a broad spectrum topical antibiotics has to be started, which could either be one commercially available or fortified. If there is no positive response to treatment, modification of the regimen should be considered. Surgical intervention is the ultimate way out in non-healing ulcers.

References

Bioethics of Off-Label Prescriptions

Dr. Meena Chakrabarti MS

Off–Label prescribing also known as “unapproved use” is the physicians practice of prescribing a drug or a medical device for a purpose different from one of the indications for which the product is approved by the Food and Drug Administration (FDA). This practice is widespread and it is estimated that nearly 60% of drug prescriptions per year are for off-label indications. Some common off-label drug uses include 1) treating a disease other than the one for which the drug is approved for, 2) giving a drug at a different dose or frequency than it’s been approved for and 3) using a drug to treat children when its approval is limited to adults.

Real concerns about unexpected safety issue of drugs surfaced for the first time in 1962 closely following the “Thalidomide tragedy” where use of this drug, approved as a sedative, and marketed by Chemie Greunthral resulted in the birth of several thousand malformed babies to mothers who had taken this drug for hyper emesis during the first trimester of pregnancy. Amendments were made in the drug laws which made it mandatory for all new drugs to pass through ever so stringent testing for drug toxicity, safety and efficacy before they are even taken up for their first use in clinical trial on humans.

In Ophthalmic practice, off-label prescription of approved drugs include 1) Intravitreal injection of antibiotics, 2) Use of mitomycin-C and 5 Fluorouracil, 3) Periocular injection and intraocular administration of steroids, 4) Use of immunosuppressive for uveitis, 5) Use of photodynamic therapy for non AMD lesions, 6) Intravitreal injection of Bevacizumab (avastin), and 7) use of tissue plasminogen activator.

The IND (Investigational New Drug Application) for clinical trial stipulates restriction of the trials for specific age groups leaving out pediatric (below 12 years), women in the reproductive phase (17-45 years), lactating mothers, and, even geriatric patients (60 years and above). Consequently when the drug goes for approval its use will be initially restricted to patients belonging to the class and type on which clinical trials have been completed and the drug was found to be safe and effective for the indication for which the drug was tested.

What is good for the goose may not be good for the gosling! Extension of its use to the pediatric and other population would be possible only after the drug has been tested for such population in specially conducted trials which in many cases may take a couple of years more after the first approval. In practice until use in these population is approved, no claim can be made on the safety and efficacy of the drug in such population. Development of pediatric indications for potentially beneficial off-label drugs has led to investigations and publishing of several studies to establish safe dosing guidelines for many off-label drugs that are now considered ‘standard-of-care’ in infants and children. Publication of well documented small case series in peer reviewed journals indicate that high quality evidence can and does exist beyond federally sanctioned approval and may be used to deliver safe and effective drug as well as expunge those that may be dangerous from the market.

All these parameters change when the same drug is administrated for a new indication for which no data has been generated. Thus the treatment schedules, the dosages, and duration of treatment in off-label use could

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be very different from the approved schedules and such ad-hoc approaches could result in serious adverse reaction or may have even fatal outcomes. Testing for a new indication calls for fresh clinical trials and sometimes even repetition of toxicology studies to ensure that the doses used for the new indication does not result in toxic manifestations and adverse drug reactions.

As a corollary to this, such use cannot be indicated in labels and product literature, but also cannot be promoted directly or indirectly by the manufacturer or the distributor, to the medical profession.

From a legal and ethical standpoint, off-label use represents a delicate balance between the regulatory objective of protecting patients from unsafe or ineffective drugs and medical devices on the one hand, and the prerogative of physicians to use their professional judgment in treating patients on the other.

The practice of off-label drug prescriptions raises a number of legal and ethical issues. To state a few: 1) Is off-label prescription a form of a human experimentation? 2) Could a failure to prescribe off-label medications leave the physician vulnerable to a malpractice suite? 3) Does the physician have a duty to inform the patient that the product is being prescribed off-label? 4) In prescribing a drug for an unapproved use, does the physician act as ‘learned intermediary’, thereby relieving the drug manufacturer of liability for resulting patient harm? 5) How does the FDA regulate off-label prescribing? 6) Can a manufacturer promote a product for an off-label use?

In the world of law, where matters are rarely clear cut, there are few certainties about off-label use. First it does not violate FDA law. The agency itself acknowledges this and its centre for drug evaluation states that “Neither the FDA nor the federal government regulate the practice of medicine. Any approved product may be used by a licensed practitioner for uses other than those stated in the product label”.

The pace of medical discovery runs ahead of the FDA’s regulatory machinery and hence the off-label use of some drugs is frequently considered as ‘state-of-the-art’ treatment and in some circumstances an off-label use of a particular drug or device may even define the standard of care.

However this doesn’t mean that a physician should feel free to use a product off-label in the same way that he or she might employ the product for one of its approved indication. The physician lacks information on its use, dosage, and route of administration. Furthermore the safety and efficacy for the unapproved use may not have been established by adequate and well controlled clinical trials. Clearly, anecdotal data does not take the place of such investigations and the fact that the product has been proven safe and efficacious for one use does not mean that it is safe and efficacious for any other. Given the risk of liability for using a product for an unapproved purpose, physicians should do so only when they are convinced that the unapproved status of use is outweighed by the potential benefit to the patient. Often this will be obvious- for example, when there are no other available products, such as is the case with many drugs prescribed for children, which until recently were rarely tested in that population.

Is off-label use a form of human experimentation? A clear cut distinction rarely exist between research and therapy and the main key for this distinction is the physician’s intent. If the intent is primarily to benefit the patient the intervention is therapy. If the intervention is solely to test a hypothesis and obtain generalisable knowledge, the intervention is an experiment. Yet in employing a product for an unapproved use the physician often has both objectives in mind. She hopes to benefit her patients, and also hopes to find out whether this intervention can help similar patients. The distinction becomes even more blurred if the physician has a strong financial interest in the success of the intervention.

Should manufacturers be permitted to promote goods for unapproved uses? Off-label promotion makes more information available to the physicians, enabling them to make better treatment recommendations for patients. It allows manufacturers to avoid or postpone the cost of obtaining FDA approval so that they can make products available more quickly and invest more in research and development. Off-label promotion especially benefits patients with orphan diseases, who often must rely on off-label uses for treatment. On the other hand it undercuts the FDA’s ability to ensure safety and efficacy and removes incentives for manufacturers to conduct studies on
safety and efficacy and encourages them to seek FDA approval for the narrowest, most “easy-to-support” indications.

The doctrine of informed consent obliges physician to provide patients with material information about proposed treatments, alternatives the potential risk and expected benefits of each. An informed consent will safeguard both physician and patients especially when the proposed line of therapy involves off-label use of medicine or device. The ophthalmic community has a tremendous responsibility to demonstrate safety and efficacy of off-label drugs by providing adequate supporting data via peer reviewed publication. The treating physician should document in the chart details of the decision-making process including previous treatment and diagnostic studies, dose and lot number of the drug, as well as the discharge and follow up instructions. Use a drug specific informed consent (e.g. for Avastin; a drug specific informed consent can be downloaded from www.omic.com) and discuss the off-label status of the drug, making sure that the patient has adequate time to take a decision before signing the informed consent form. Discuss clearly the off-label status, the attendant risks as well as why the FDA approved options have not been considered. Failure to provide or at least discuss off-label therapy if it is the standard of care may make the physician liable to a malpractice suite. —— Damned if you do! Damned if you do not!

The question of reimbursement! Some insurance companies will not cover the cost of a drug – most often if it is a costly drug and if it is prescribed for off-label use. The doctor can prove through the use of peer reviewed medical studies or other reliable information that the off-label application is appropriate – that should bolster the case in getting reimbursement.

FDA approved drugs used for indications other than what is indicated on the official label may be covered under Medicare if the carrier determines the use to be the medically accepted, taking into consideration authoritative medical literature and or accepted standard of medical practices.

Clearly off-label use of drugs by individual physicians is legal and leads to new therapeutic advances. A clever clinician putting together inferences from pathophysiology of diseases and the known pharmacological properties of approved agents may accumulate data on efficacy and toxicity in new settings.

The FDA encourages the off-label use of drugs with the implied commitment to the profession to do the necessary clinical research to gain approved labeling for the new indication. The fact that the drug is already approved bypasses the regulatory maze necessary for a physician to try these agents. While the manufacturer cannot advertise the off-label use of a drug they have the responsibility to file an IND application with the FDA.

Individual physicians interacting with their patients should give thought to the benefits of off-label drug use based on pathophysiology and therapeutic logic and side effects in that individual patient. The physician does not require an IND application or review by the Institutional review board, however records should be maintained with the goal of accumulating information that could form the basis of pilot studies that ultimately should lead to expanded indications for the already approved drug. Many of the off-label use of a drug are described and reported as case reports which alert other physician to the possibilities of using these drugs in other patients. With appropriate transparency to the medical community and the patient, these types of small prospective studies will hopefully pave the way for more effective therapies for diseases that has been therapeutically problematic to date.

Use of intravitreal triamcinolone acetonide and anti VEGF, a form of anti angiogenic therapy is on the horizon for aggressive posterior retinopathy of prematurity (AP –ROP) which develops in profoundly immature neonates. The rationale of the therapy with off-label drugs, is the accepted fact that VEGF promotes retinal vascularisation. The BLOCK-ROP study, a phase I trial is underway to study their challenges. Let us wait for conclusive results before experimenting on the vulnerable target population of precious immature neonates in the interim period, and even the AAO advocates exercise of caution in the use of Anti VEGF drugs outside a clinical trial in premature neonates.

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Dirofilaria is a filarial worm of carnivorous animals. It affects the ocular and periocular tissues. Zoonotic dirofilaria occurs widely throughout European, African, Middle eastern and Asian countries. Cases from India have been reported. Man is a dead end for the parasite and it causes severe inflammatory reaction in the host. It has been reported in the subcutaneous tissue, subconjunctival space, in the lacrimal gland and as an intraocular parasite. This is the first report of dirofilaria in an extraocular muscle.

Case Report

A 24 year young lady presented with swelling in right eye since 5 months. There was no complaint of pain redness in right eye. She consulted a local doctor who diagnosed it as a scleral nodule and started topical antibiotics and steroids, but there was no change in size of nodule. She had no complaint of double vision. Ocular examination revealed minimal congestion nasally. There was a 0.5 cm grey white nodule, 1 mm medial to nasal limbus at 3 o’ clock position which was firm in consistency and attached to sclera. There was no sign of uveitis. Rest of the ocular examination was normal. Visual acuity was 6/6 in both eyes. Intraocular pressure was normal in both eyes. All haematological tests were normal except a raised ESR.

The nodule was excised under local anaesthesia. After separating the nodule from the overlying conjunctiva the cyst was found attached to the medial rectus muscle and was separated by blunt dissection and removed enbloc. The patient was put on oral systemic steroids for a week with oral serratiopeptidase to decrease inflammation. Patient did not have any other foci with the worm. She has been followed up for 2 years, has a visual acuity of 6/6 with no recurrence locally or elsewhere and no recurrent ocular swelling.

Histopathology showed a cyst containing a worm identified as Dirofilaria with surrounding granulomatous inflammation.

Pathological Findings

The identification of dirofilaria is based on the microscopic features of individual parasite including a thick laminated cuticle with external longitudinal ridges and the presence of well developed circumferential musculature interrupted by 2 lateral cords. The number of reproductive tubes and their content (egg, microfilaria) help to determine the sex of the parasite and the reproductive state of female worm. [Fig 1, 2,3]

Discussion

Ocular dirofilariasis is a form of subcutaneous dirofilaria caused by Dirofilaria repens. The infection is transmitted to humans accidentally, by insect vector like mosquito. Humans are the dead end for the parasite. In human infection, parasite development is impaired and microfilariae are not produced. The most common involvement in the eye is in the conjunctiva.
and periorbital region. Subconjunctival involvement is common. Other ocular involvement includes inflammatory reaction, uveitis and glaucoma. Periocular Dirofilariasis presents as inflammatory painful mass lesion. Patient presents with inflamed subcutaneous nodules that are painful, erythematous and sometimes migratory. The diagnosis is confirmed by studying the morphology of the worm after their removal. These have thick laminated cuticle, broad elongated ends and large muscle cells. Length of the female worm varies from 8 to 13 cm and males from 4 to 4.8 cm. The recommended treatment is surgical removal of the mass including the worm. It is important to identify the nematode as *Dirofilaria* to avoid treatment with antihelminthic agents.

*Dirofilaria* is a zoonotic infection by filarial nematode of genus *Dirofilaria*. Humans are aberrant/accidental hosts for *Dirofilaria*. In humans the worms usually die before maturing provoking a focal granulomatous reaction in subcutaneous tissue or small pulmonary infarcts. There are 2 subgenera – *Dirofilaria dirofilaria* and *Dirofilaria nochtiella* based on absence or presence of external longitudinal cuticular ridges (Nochtiella). Approximately 20 species of *Dirofilaria* are in subgenus Nochtiella but only *D. repens*, *D. tenius* and *D. striata* infect humans. Four species of *D. Nochtiella* were once called *D. conjunctivae*, *D. tenius*, *D. ursi*, *D. subdermata* and *D. striata*. Useful characteristics for differentiating between *Dirofilaria* species are the size and features of the body wall. *i.e.*, thickness of cuticle, its structure, ridges, lateral chords and number and type of muscle cells.

Adult female is the definitive host – 230-310 mm long and 1-2 mm in diameter and are longer than males (120-190 mm). Females have 2 sets of tubular reproductive organs. All *Dirofilaria* that infect human have a multilayered striated cuticle and prominent lateral cords. Only immature worms lodge in human pulmonary vessels. These worms are 100-350 mm in diameter and are partially degenerated in tissue section. Mosquitoes deposit larvae in human subcutaneous tissue which may provoke lesions. Some larvae migrate to heart and die. Dead worms produce infarcts when they lodge in pulmonary vessels. Worms localise most frequently in tissue of orbit, scrotum, breast, arm or leg.

Worm infestation in extraocular muscle (EOM) have been reported extensively in literature. Most innumerable are those of myocysticercosis, Fig. 1. Transverse section of immature female *Dirofilaria Noch* demonstrating central uteris outer cuticle with intervening muscle. Fig. 2. Section of the worm along with surrounding granulomatous inflammation. Fig. 3. Section showing high power view of the same.
many of them being from India. Cysticercosis can be picked up on neuroimaging and responds to oral steroids and albendazole, requiring surgical intervention only rarely. Sparaginosis (Tapeworm) in EOM have been reported, as also the hydatid cyst. References

Unexpected complications can sometimes be associated with even the most innocuous surgical procedure. We present a “nightmare” of complications following the commonly performed “I and C”: Incision and curettage of a chalazion under subcutaneous infiltration of a local anesthetic.

A 60 year old healthy lady with no systemic illness underwent a chalazion incision and curettage. Xylocaine intradermal challenge was performed and the local anaesthetic 2% xylocaine with adrenaline infiltrated after confirming absence of any reaction at test site on forearm.

48 hours later, the patient presented with painful lid oedema, erythema and induration with black eschar at the injection site. Ocular examination was normal. The intradermal xylocaine challenge site showed erythema, induration, vesicles and pruritis.

Culture and Sensitivity of the slough was positive for coagulase positive staphylococcus aureus (Table 1).

Table 1. Culture and sensitivity results

<table>
<thead>
<tr>
<th>SENSITIVE (S)</th>
<th>RESISTANT (R)</th>
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<tbody>
<tr>
<td>1. Clarithromycin S+</td>
<td>1. Methicillin</td>
</tr>
<tr>
<td>2. Erythromycin S+</td>
<td>2. Ampicillin</td>
</tr>
<tr>
<td>3. Gentamycin S+</td>
<td>3. Amoxycillin</td>
</tr>
<tr>
<td>5. Nitrofurantoin S+</td>
<td>5. Norflox S++</td>
</tr>
<tr>
<td>7. Cefotaxime S+</td>
<td>7. Linezolid S+++</td>
</tr>
<tr>
<td>9. Ciplox S++</td>
<td>9. Linezolid S+++</td>
</tr>
</tbody>
</table>

Conservative treatment with local saline Compresses, Dipgenta cream, topical oflox eye drops and ointment, Systemic Linid 600 mg twice daily X 14 days and oral antiallergic medications were prescribed and brought dramatic relief. The eschar sloughed leaving a large full thickness lid defect with infiltrated yellow sloughing margins which healed slowly over a period of 1 month leaving behind a full thickness lid defect well hidden by the lid crease causing a small area of scleral exposure.
The patient refused advice of a minor surgical procedure for closure of lid defect.

**Discussion**

Lidocaine Hydrochloride is the favoured anaesthetic agent in outpatient surgical procedures. Adverse reaction to lidocaine are uncommon and most reactions are Type I immediate hypersensitivity reactions.

Performing with other injectable anaesthetics. (a) Esters-benzoacaine 5%, tetracaine hydrochloride 1%, procaine hydrochloride 2.5%, benoxinate hydrochloride and 2.5%, cocaine hydrochloride. (b) Amides-Lidocaine, prilocaine hydrochloride, bupivacaine hydrochloride, mepivacaine, etidocaine hydrochloride). If the patient develops a positive patch test for any of these local anesthetics an intradermal challenge should follow.

A positive skin testing may be seen in < 10% of patients and the presence of a negative skin testing does not rule out allergy!

Methicillin-Resistant Staphylococcus Aureus (MRSA) is a nosocomial pathogen almost exclusively confined to individuals with risk factors (recent surgery or hospitalization, residence in a long-term care facility, presence of a percutaneous device or indwelling catheter, or recent dialysis) and are known as Hospital-Associated MRSA. In the 1980s MRSA infections were reported in persons who lacked traditional MRSA-risk factors. These infections appeared to be acquired in the community and are now known as Community Associated MRSA (CA-MRSA).

The most common clinical manifestation of CA-MRSA are skin and soft tissue infections (SSTIs) such as abscesses, cellulitis, and less commonly necrotizing pneumonia, osteomyelitis and septicaemia.

**Antimicrobial Therapeutic Considerations:**

- HA-MRSA and CA-MRSA are sensitive to Daptomycin, Linezolid, Quinupristin, Dalfopristin, Minocycline and Vancomycin
- Daptomycin is useful to treat persistent MRSA bacteremias and MRSA treatment failures with other drugs.

**What did we learn from, this “NIGHTMARE” ?**

We learnt the following facts:

- That a negative skin test is not a foolproof indication against allergy. Skin test positivity is seen only in < 10% of patients following intradermal challenge with preservative – free lidocaine.
- Type IV Hypersensitivity does occur with lidocaine although it has been rarely reported.
• A history of prior exposure to an injectable anaesthetic is very important.

• A history of drug allergy is to be taken seriously and in such patients it is wise to do a patch test for injectable medications which will be used during surgical procedure. The patient should be counselled on the necessity to maintain a list of “Safe Medications” for use after patch testing.

When Type IV delayed hypersensitivity presents at the surgical site, erythematous vesicular crusting lesions will be present at the skin test site also.

• A word of caution: Delayed hypersensitivity reaction should be included in the presurgical counselling and in the informed consent!

References


A Case of Conjunctival & Corneal Intraepithelial Neoplasia (CCIN): Carcinoma in situ of the Conjunctiva & Cornea

Dr. Mohammed Haneef MS, Dr. Manoj Venugopal MS, Dr. Mallika O.U. MS, Dr. Mini P.A. MS, Dr. Padmasree K.M. MS, Dr. Anuja Sathar MS

Introduction

When the cytological features of malignancy are present but the malignant cells are confined to epithelium without invasion across the basement membrane, it is called as carcinoma in situ or intraepithelial neoplasia. CCIN is an uncommon, benign, slowly progressive unilateral disease with low malignant potential. Risk factors are ultraviolet light exposure, HPV infection and Acquired Immunodeficiency Syndrome.

Case Report

A 78 year old man presented with a raised gelatinous growth at the superior limbus of left eye of 28 years duration which was slowly progressive in nature. As it was asymptomatic the patient did not seek any medical advice. (Fig 1)

On Examination

There was a raised pinkish white gelatinous mass with tufts of vessels on its surface, at the superior limbus extending 4 mm on its conjunctival as well as the corneal side for more than 180 degrees of the limbus (Fig 2)

Initial Management

Initially 2mm x2mm of the growth was excised (Fig 3) and sent for histopathological examination: HPE revealed epithelial dysplastic changes. One month later, the entire lesion was excised with adjoining 4 mm of normal conjunctiva while on the corneal side it was completely shaved off. HPE showed strips of squamous epithelial tissue with severe dysplasia and carcinoma.
in situ changes with attempted epithelial pearl formation.

![Image](image1.png)

**Subsequent management**

After receiving the HPE report the patient was put on topical Mitomycin C 0.2 mg/ml eye drops twice daily for 15 days.

**Follow up**

The patient was closely followed up for any recurrence. No recurrence was noted till date with 8 months of follow up (Fig 4).

![Image](image2.png)

**Discussion**

Carcinoma in situ is rare in the eye compared to other parts of the body.

It may present as Leukoplakia, Papilloma, or as complication of Pterygium or Pinguecula. Impression cytology may be useful in diagnosis.

As the basal membrane of the epithelium remains intact and the sub epithelial tissue is not invaded, simple shaving off the lesion is sufficient and there is no need for deep dissection. Only infrequently the lesion becomes invasive.

If the CCIN is localized, excision with cryotherapy is curative while the diffuse form CCIN is difficult to treat as the borders of the lesion are poorly defined, making a complete excision impossible. Recurrence rate would be high, and may be treated with other modalities like topical mitomycin, 5-fluorouracil & interferon alpha-2b as alternatives or as adjuncts to surgery.

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A 71 yr old male presented to the department of Ophthalmology, Medical College Hospital, Calicut with swelling of the left lower eyelid of 6 months, progressive since 1 month. He had no pain, visual disturbance or constitutional symptoms.

On examination, he had generalized lymphadenopathy with preauricular, submandibular, cervical and inguinal nodes (Fig. 2). These nodes were 1-2 cm in size, firm, discreet and non tender. Systemic examination was within normal limits (Fig. 3).

Ocular examination revealed a non tender swelling in the left lowerlid 0.5 cm x 1.0 cm, with the skin and lower palpebral conjunctiva freely mobile over it (Fig. 2). The pupillary light reflex was normal in both eyes and there was incipient lenticular opacities which accounted for a visual acuity of 6/24. Colour vision, fields and fundus examination were within normal limits in both eyes.

The patient underwent routine lab workup. Complete blood counts and peripheral smear were within normal limits.

Bone marrow trephine smear showed scattered erythroid, myeloid haemopoeitic cells with normal maturation (Fig. 4).

FNAC of lid swelling was performed; Papanicoloau stain revealed sheets of uniform immature lymphoid cells with irregular chromatin clumps, irregular nuclear border with a cell size larger than mature lymphocytes. (Fig. 5 and 6).

Endonasal biopsy from a bulge in the nasopharynx showed sheets of uniform immature lymphoid cells (Fig 7 & 8). Both FNAC and biopsy confirmed low grade Non Hodgkins lymphoma (NHL).

Chest X-ray and X ray PNS were within normal limits. Based on a diagnosis of lowgrade NHL, a management option of chemotherapy was given (CHOP regimen was completed in 5 cycles).

Discussion

Among all systemic lymphomas, – ocular adnexal lymphomas constitute a small fraction only. Most of these tumors are NHL – B cell type.

Ocular Adenexal lymphoma constitute 6-8 % of orbital tumours and 6 % of extra nodal lymphoma. Ocular adnexal lymphoma is considered to be primary if it involves the ocular adnexa alone and secondary if it is accompanied by lymphoma of identical type at another site. The frequency of involvement of adnexa are as follows. Conjunctiva 20-33 %, Orbit 46-74 %, and eye lid 5-20 %.

The classification of lymphoproliferative disorders has been a major problem in diagnostic pathology. But with the development of of immunohistochemical analysis clonality can be identified from the CD molecules these cells express. There are various classifications of
Fig. 1. Picture showing lower eye lid swelling

Fig. 2. Cervical lymphnode enlargement

Fig. 3. Chest X ray - Normal

Fig. 4. Bone marrow: Normal maturation of erythroid and myeloid series

Fig. 5. Lid swelling - Low power view (HPE)

Fig. 6. Lid swelling - High power view (HPE)

Fig. 7. Endonasal biopsy - Low power view (HPE)

Fig. 8. Endonasal biopsy - High power view (HPE)
lymphoma which are quite extensive and includes Revised European American Lymphoma (REAL) classification, Rappaport classification and NIH working formulation classification. Ocular Adnexal Lymphoma (OAL) exhibits a limited spectrum with majority being Non Hodgkins B cell. Types of OALs based on the Immunophenotypic analysis are Extra Nodal Marginal Zone Lymphoma (EMZL ), Follicular Mantle cell, Lymphoplasmacytic (LPL) and Diffuse Large B Cell Lymphoma (DLBCL).

The diagnosis of OAL is made by the clinical and imaging studies followed by the combination of histopathologic, immunophenotypic and molecular genetic studies.

Management of OAL is a multidisciplinary approach first requiring a comprehensive staging evaluation. A complete blood count, hepatic enzyme levels, LDH, Chest Radiography, CT scan of abdomen and chest and Bone marrow studies are often required to stage the disease prior to the commencement of treatment. If conjunctival or eyelid OAL is identified a CT/MRI of the orbit is indicated for staging.

The factors that determine the prognosis in OAL are disease stage at presentation, type of lymphoma, site of disease, increased expression of tumor cells and age of the patients. Among the common OAL types, EMZL Follicular and LPL are considered low grade where as DLBCL and Mantle cell lymphoma are high grade.

Treatment of OAL is dependent on specific tumor type and its staging.

Surgery can be useful in certain cases of lymphoma and is appropriate for lesions localized to conjunctiva and orbit.

Radiation is the most frequently used modality for treating OAL in localized disease.

Chemotherapy is recommended in patients with Stage II or greater disease and this includes the use of standard regimens for systemic lymphoma including Cyclophosphamide, Doxorubicin, Vincristine and Prednisolone and Chlorambucil.

Cases which are seen in outpatient department such as the one discussed, where eyelid involvement is present, may mimic benign tumours like chalazion and have to be taken care of, evaluated, and, properly managed.

References

Terriens Marginal Degeneration –
A Varied Presentation

Dr. R. Nirupama Balaji DO, Dr. K.S. Chandrakanth DO DNB, Dr. Sheeja Vishwanathan MS, Dr. Ramakrishnan MS, Dr. Tresa Mathew MS, Dr. Venkitachalam MS

Introduction

Terriens marginal degeneration is an uncommon disease of the peripheral cornea, occurring at any age, 75% being males. This condition may be bilateral or unilateral. Lesions begin usually superonasally rarely inferiorly with development of fine, white sub epithelial, peripheral opacities that spare the limbus. The opacities coalase and this is followed by corneal thinning, typically with a sloping central edge and a fairly steep peripheral edge to the resultant furrow.

The epithelium is typically intact with yellowish white lipid deposits in the centre of the gutter with associated vascularisation. Etiology is unknown, although inflammatory, degenerative, and immune mediation have been proposed.

Case Report

A 22 year male patient presented to our hospital with an accidental detection of visual defect in the left eye while attending an eye camp. He had never used glasses earlier. His visual acuity was checked and was found to be 6/6 right eye and 6/12 left eye. He underwent a complete cycloplegic refraction and his final correction was found to be 6/6 with -1.00 Dioptre cylinder x 105° axis. The patient underwent a thorough slit lamp examination which revealed a unilateral, inferior circumlinear corneal thinning with intact epithelium, without neovascularisation, minimal lipid deposition and a clear area existing between the lesion and limbus. (Fig. 1a and b)

There was no epithelial defect noted by fluorescein staining. The patient underwent a corneal topography (Fig. 2) which clearly showed the area of inferior thinning with steepening 90° opposite to the central point of thinning. This is typical of Terriens. This has given rise to the high oblique astigmatism in this patient.

Fig. 1. (a) Classic presentation of Terriens marginal degeneration (b) Atypical presentation showing unilateral inferior circumlinear corneal thinning
A diagnosis of unilateral Terriens marginal degeneration was made based on the presence of inferior lesion, absence of vascularisation and unilaterality. Our patient had subtle variation compared to a regular Terriens. The patient's fundus was normal.

**Discussion**

In Terriens degeneration when the thinning is restricted to the superior or inferior area of the peripheral cornea, there is relative steepening approximately 90° away from the mid point of the thinned area resulting in astigmatism characteristic of this disorder. Histologically epithelium may be normal, thickened, and the thinned Bowmans layer and the lamellae may be split or fibrillated (Fig. 3).

Stroma is thinned. Whether or not there is vascularisation, the inflammation depends on the form of the disease. Lipid is found consistently. Though a lot of etiologies have been proposed, levels of circulating immune complexes are not elevated in patients with Terriens. In our patient the right eye was absolutely normal. Marginal corneal degeneration, dellen, collagen vascular diseases, sclerokeratitis, staphylococal marginal keratitis etc have been proposed as differential diagnosis. This patient was prescribed glasses and advised rigid contact lenses for his left eye, and advised regular followups to rule out early development of
lesion in the right eye. Usually pseudo pterygia occur in Terriens degeneration in 20% of cases\textsuperscript{1,13}. Very rarely extreme thinning occur when reconstructive surgery is indicated. A full thickness or lamellar corneo scleral graft (often hand fashioned to fit the defect) may be necessary\textsuperscript{7,11,12}. The progressive increase in against the rule astigmatism or oblique astigmatism with advanced disease can be arrested up to 20 yrs by grafting. Severe astigmatism can also be treated by a crescentric shaped excision of the gutter with suturing of the healthier margins. Corneal thinning may progress sometimes, despite intact epithelium, to the point at which a deep corneal break leads to hydrops or even frank perforation in about 15% of cases\textsuperscript{2,4,14}. In these cases keratoplasty either a full thickness or deep lamellar sector is necessary\textsuperscript{14}. So though an uncommon clinical entity we should keep Terriens degeneration in mind with its possible variations, and treat the patient accordingly with regular followups to see the progression of the disease.

References

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Misadventures in the Anterior Chamber

Dr. Sonia Rani John DNB, Dr. Arup Chakrabarti MS DO, Dr. Valsa T. Stephen MS DO DNB, Dr. Meena Chakrabarti MS DO DNB

The Multiflex anterior chamber intraocular lens has a stable track record and is the first surgical option that comes to our mind while dealing with a patient having a compromised capsulozonular integrity. However in the presence of endothelial cell decompensation, compromised anterior chamber angles or glaucoma, AC IOL should be used with caution. Hence a thorough preoperative assessment of the corneal endothelial cell count, anatomy of the anterior chamber angles and measurement of intraocular pressure is mandatory preoperatively. It is also necessary to measure the limbal – white – white distance pre or peroperatively and ensure that an appropriately sized AC IOL is implanted.

Complications of malpositioned IOLs in the anterior chamber include uveitis, glaucoma, hyphaema, cystoid macular oedema. Thus the majority of these eyes are irritable requiring explantation for corneal decompensation, chronic intraocular inflammation or cystoid macular oedema.

In this photoessay we present four situations where we managed “Mis - adventures in Anterior Chamber”. (Fig. 1). We hope to stress the importance of a proper surgical technique, and the need to maintain a library of common powers of ACIOLs of appropriate size.

Situation I: Oversized anterior chamber intraocular lens

65 year old female patient presented with aching pain, tenderness on touching the globe, blurring of vision, photophobia and haloes of 2 months duration. She had undergone a complicated cataract surgery with an AC IOL implant 4 months prior to onset of symptoms. Ocular examination revealed a visual acuity of 6/36, an oversized AC IOL in the anterior chamber, applanation tonometry reading of 28 mm in left eye and fundus showing cystoid macular oedema (Fig. 2).

The patient underwent AC IOL exchange for an appropriately sized ACIOL. She was asymptomatic throughout the followup period of 12 months, regained a vision of 6/12, with resolution of cystoid macular oedema, and good IOP control with a single medication. (Fig. 3).

Situation II: AC IOL implant with haptic in section in an 65 year old lady resulted in an irritable eye due to localized corneal oedema superiorly. Resurgery to dial the IOL and orient it horizontally resulted in a quiet eye (Fig. 4).

Situation III: PC IOL in Anterior Chamber

Young 35 year old lady who presented with a visual acuity of 6/6, had a PC IOL implanted in the anterior...
chamber, a history of recurrent uveitis and recalcitrant glaucoma. (Fig. 5) IOL explantation with a trial of bandage contact lens was advised

Situation IV: Flipped AC IOL

72 year old lady with cirrhosis liver and portal hypertension was referred for recurrent intraocular inflammation and secondary glaucoma in her left eye. Ocular evaluation revealed the presence of chronic uveitis, secondary glaucoma and inflammatory deposits and cocoon membranes on the IOL and angle of anterior chamber as well as a flipped AC IOL implant. B scan ultrasonography showed plenty of vitreous opacities, a partial retinal detachment with another dislocated IOL in the vitreous cavity.(Fig 6. a & b).

The patient underwent explantation of both AC and PC IOL implants along with retinal detachment repair and silicone oil tamponade under general anaesthesia. She developed sterile postoperative reaction with hypopyon which resolved with conservative treatment. (Fig 7.) Silicone oil removal was performed after 6 months leaving a stable visual acuity of 5/60.

Discussion

Although surgical intervention is definitive, conservative observation and pharmacologic therapy should always be considered as long as signs and symptoms of intraocular inflammation are absent. Surgical treatment of malpositioned and dislocated IOL does remain an important and challenging clinical problem. The course of action must reflect the type and location of the lens, the age of the patient, the symptoms, visual acuity, corneal endothelial health, the presence and severity of intraocular inflammation and the status of the fellow eye. When surgical intervention is under consideration a decision must be made with regard to the timing of
surgery, the approach and the disposition of the intraocular lens.

Kline et al\textsuperscript{3} found corneal edema, cystoid macular edema and uveitis as the most common causes for explantation of IOL. Poorest visual outcomes were seen in patients with UGH syndrome.

Complications\textsuperscript{4,5,6} encountered following lens exchange include retinal detachment, glaucoma and corneal decompensation. Surgical goals included correction of malposition, resolution of corneal edema, uveitis, elevated IOP or cystoid macular edema. Explantation of an IOL is always challenging, yet armed with knowledgeable preparation and meticulous operative technique, the results may be satisfying to both the patient and the surgeon.

**Conclusion**

Though multiplex anterior chamber IOLs have a very good track record, proper surgical technique, correct sizing of the IOL and proper orientation in the anterior chamber is mandatory for a satisfactory postoperative outcome. Posterior chamber IOLs should never be implanted in the anterior chamber.

**References**

Management of Incomplete Silicone Oil Fill

Dr. A. Giridhar 1 MS, Dr. G. Mahesh 1 MS, Dr. Sunil Neelakantan 2 MS, Dr. Thomas Cherian 3 MS, Dr. Gopal S. Pillai 4 MS, Dr. Manoj 5 DNB, FRCS, Dr Meena Chakrabarti 6 MS DO DNB

67 year old male patient was referred for management of a phakic subtotal retinal detachment in his only eye. He was hypertensive and was diagnosed to have primary open angle glaucoma in his right eye five years back. He had lost vision in his left eye following a road traffic accident. Details of the injury sustained seven years back in the road traffic accident were not available. He had been on Glucomol 0.5 % eye drops for the past 5 years in both his eyes and was quite compliant to therapy. He also had incipient cataract and a visual acuity of 6/18 in his left eye when he developed sudden onset of defective vision.

Ocular examination revealed a visual acuity of hand movements in the right eye with inaccurate projection, and no light projection in the left eye. Anterior segment evaluation in the right eye was unremarkable except for incipient lens opacity and an IOP of 20 mm Hg. The left eye showed evidence of chronic uveitis with extensive posterior synechiae and partially absorbed cataract. Fundus examination of right eye revealed a subtotal macula off retinal detachment with an inferior and temporal giant retinal tear of 180° with a mobile inverted posterior flap and no evidence of PVR. He underwent scleral buckling with pars plana vitrectomy, a thorough base excision, injection of PFCL to unfold the posterior flap, and 360° endolaser barrage followed by a PFCL-Silicone oil exchange. He had an uneventful post operative period and was discharged on the third post operative day with a stable attached retina, and controlled IOP on a 2 drug regimen including Timolol 0.5 % and topical dorzolamide eye drops twice daily. He regained a stable vision of counting fingers at 4 metres, and adequate IOP control. Fundus examination showed an attached posterior pole, incomplete silicone fill, and there was difficulty in confirming whether the tear was closed and well positioned on the buckle. (Fig 1.) By the end of 6 months postoperatively, his vision had dropped to counting fingers at 1 metre and his cataract had progressed significantly.

Fig. 1. Postoperative Fundus photograph showing an incomplete silicone fill
My question to the panelists is on the further management of this one-eyed patient.

**Dr. A. Giridhar**

From the detailed case history, we have a one-eyed patient with a recurrent retinal detachment, significant cataract in the only useful eye. It appears that the cause for the recurrent retinal detachment could be slippage of the posterior flap which could be due to an incomplete silicone oil fill. As far as further management is concerned the patient can undergo cataract extraction, inferior iridectomy, silicone oil removal, re-attach the retina again, further endo laser and re-inject again with silicone oil. If the retina is taut inferiorly the procedure will have to be combined with retinectomy and use of heavy silicone oil tamponade. I feel with this management it should be possible to achieve re-attachment.

**Dr. Mahesh G.**

The objective of any retinal detachment surgery is complete anatomical re-attachment of retina. The colour photograph of the fundus of right eye shows inferior retina is elevated and there is incomplete silicon oil fill. Also cataract is significant. In this situation I prefer to take him for re-surgery. In cases with giant retinal tears the visualization of peripheral retina is extremely essential and so it is prudent to remove the cataract without implanting the lens. Inferior peripheral iridectomy need to be done and oil removed. If there is re-detachment, revision of the surgery by removing the membrane has to be done. Relaxing retinectomy may be required in the presence of taut retina. Subsequent steps include putting perfluorocarbon liquid till it covers the anterior edge of the retina and a thorough endo laser barrage. If PFCL goes under the retina it indicates incompletely relieved traction and the need for further retinectomy. Silicone oil with PFCL exchange can be done carefully watching for falling back of the flap. Patient is advised prone position for 2 weeks postoperatively. Silicone oil can be removed after 3 months if the retina stabilizes. This has to be considered because the patient has glaucoma also. If there is inflammation postoperatively the cover of steroids may be given. A possibility of inflammation due to sympathetic ophthalmia due to the injury to the other eye must also be kept in mind. Intra ocular pressure has to be frequently monitored postoperatively.

**Dr. N. Sunil**

It is indeed a difficult case to manage. The initial site of GRT has not been mentioned.

The tear seen in the fundus picture appears to be inferior in location and appears elevated and the posterior eges are rolled out, suggesting PVR changes. Phacoemulsification with IOL implantation, preferably through a superior section has to be performed, coupled with posterior segment intervention. I would prefer a large optic PMMA IOL, care being taken to avoid a PC rent and get a complete cortical wash. The tunnel wound has to be sutured.

I would start the posterior segment intervention with a wide bore infusion cannula, connected to a syringe filled with silicone oil. With the light probe in one sclerotomy, I would inject PFCL over the posterior pole. Meticulous removal of the membranes to release the PVR and mobilise the folds will have to be done. More PFCL will have to be then injected slowly, to flatten the
retina. Care has to be taken to monitor the IOP, keeping a watch on the retinal artery pulsations. Intermittently, the PFCL cannula has to be removed to release silicone oil and control the IOP.

Additional Laser barrage has to be then performed all around.

This will have to be followed by PFCL-Silicone oil exchange.

Anterior chamber has to be checked for any shallowing, before suturing the sclerotomies. It has to be reformed well, and the IOP should be normalised by releasing silicone oil, if needed.

Post operatively a course of systemic steroids, in tapering doses along with topical steroids, Antibiotics, Cycloplegics, Antiglaucoma medications have to be given.

Postoperative examination should include regular IOP checking and slit lamp examination to see for inflammation, and AC depth.

Silicone oil will have to be retained as long as possible in the eye.

Dr. Thomas Cherian MS

At present, this patient has an incompletely oil filled eye, with open retinal breaks and a cataract. This, if left alone, will progress to a retinal detachment in the near future. The option would be to reoperate on him - a cataract surgery with intraocular lens implant, silicone oil exchange, with endolaser and trimming of any rolled retinal edges, if found difficult to unfold. The laser should be not just to the edge of the tear, but he requires 5 – 6 rows of laser for a strong retinopexy.

Once the retina in the right eye is found to be stable, I would suggest that he undergoes an enucleation with implant and a custom made prosthesis for the left eye, since leaving this unattended, could mean, keeping a potential ‘exciting eye’ and inviting trouble in future.

Gopal S Pillai

Analyzing the information of this patient, he had an inferior 180 degree GRT with inverted posterior flap, but no PVR changes. He underwent the standard surgical procedures involving scleral buckling, pars plana vitrectomy, vitreous base dissection, PFCL, endolaser and PFCL silicone oil exchange. Post operatively the retina was flat and attached, his visual acuity was 4/60 and IOP well maintained.

**Comments on initial treatment already done:**

1. **Buckle placement**

In patients with glaucoma, it is always fruitful to use buckles of lesser size and extent and also to leave some healthy conjunctiva without peritomy to aid in a later trabeculectomy if needed. So I would have used a style 276 or smaller buckle, and may have limited the buckle to inferior 180 degrees giving limited peritomy in the superior quadrant. However it is important in this case to give a higher buckle indent than is normal on account of inferior GRT and its possible foreshortening. To increase the height of the buckle, I would have made the eye hypotonic before I tied the buckle sutures, so that the IOP may not rise post operatively. The sutures used with style 276(7 mm) would be passed at a distance of less than 10 mm between each other to have a higher indent.

2. **Management of lens**

Being an inferior GRT with inverted flap, we realize that silicone oil is not going to tamponade the tear and keep the GRT closed. More over you are forced to give a slight silicone oil underfill on account of his glaucomatous state. We cannot afford an overfill, not even a near total fill in the presence of buckle, extensive laser and glaucoma as immediate post operative pressures may shoot up very much. Hence there is great importance in performing vitreous base dissection, especially inferiorly and I would have been more than happy to remove the lens in the first surgery. The removal of lens will allow a more thorough vitrectomy and vitreous base dissection, without any fear of lens touch, especially in the presence of cataract. Inferior vitreous base dissection in the presence of lens in situ may be difficult. So my surgical steps in this person would also have involved a phacoemulsification after the buckling procedure, but efore entering the eye for a vitrectomy.

3. **Cryo and laser pexy**

It would be very important in this case to provide more than adequate laser and cryopexy to the inferior retina as the silicone oil cannot tamponade the inferior tear. I may laser the entire inferior retina and cryo the
periphery under the buckle before doing the PFCL silicone oil exchange.

4 Choice of silicone oil

Since he is a 67 year old person and the average life expectancy of an Indian is 64, the choice of silicone oil is important. In such a one eyed case, we would not like to go in for a silicone oil removal if we were not confident of 100% attachment post silicone oil removal. In such a case, silicone oil with 5000 centistokes can be used as the chances of emulsification and therefore glaucoma are very low when compared to a 1000 centistoke oil.

5 Postoperative positioning

Strict prone with Trendelenberg positioning to tamponade the inferior retina till the laser and cryo induced chorioretinal adhesions become permanent.

Comments on the post operative picture shown

Though the cataract has progressed significantly, the picture shows clearly, the high inferior and posterior buckle indent very clearly. A visual acuity of 1/60 is not corroborating with the cataract. However the best vision possible in this person may be 4/60 which he reached during his initial postoperative days. It also shows the inferior tears apposed against the buckle, but with little laser around them.

This shows that the retina posterior to the buckle is very well attached. If it were to detach, the silicone oil wouldnt have prevented the detachment. At 6 months, if we see such a buckle indent and attached posterior retina in an RD with inferior tear, for all practical purposes, we take it that the retina is well attached. If the inferior meniscus of the silicone oil bubble is causing visualisation problems, we can position the patient and avoid the inferior meniscus to do a detailed examination.

Comments on further course of action

The further course of action in this person depends on multiple factors

Life style and activities of daily living Systemic illnesses and prognosis for life

Conservative approach

Already being a none eyed patient, we will usually follow a very conservative approach in his management. If there are added systemic diseases which may reduce the life expectancy or if his life style and activities that he need are limited, we may still wait and observe.

Risks for Observation:

Development of denser cataract which will reduce vision and also reduce our visualization of retina

Emulsification of oil which may increase the IOP in an already predisposed patient.

Perisilicone oil membranes which may contract and cause redetachment.

If dense cataract develop which precludes retinal examination, we can do a phacoemulsification and IOL implantation without disturbing the oil. This will help us assess retina well and laser some critical areas better before we plan for an eventual silicone oil removal.

Early silicone oil emulsification induced glaucoma can be controlled medically, but sooner or later, he may require silicone oil removal or graded diode cyclophocoagulation without removal depending on his visual acuity and acceptance to take risks. More and more people around the world are moving forward to shunt procedures as primary management of silicone oil induced glaucomas. I do not have any personal experience with this technique.

Aggressive approach

A more aggressive approach in some patients, especially younger, with higher visual needs is simultaneous cataract extraction with silicone oil removal at this stage. If the retina redetaches, one can always go inside and reattach the retina with silicone oil. In this patient, as the GRT seems to be attached, such a procedure may have good results.

However resorting to such a procedure requires a clear understanding and willingness from the patients' side to accept any untoward redetachment that may occur with this. In the unlikely event of redetachment, we may reattach the retina and fill it with 5000 centistoke oil.

If during silicone oil removal, it is felt that there is residual traction in the retina and it may cause post
operative redetachment, it may be better to sever the tractions and do silicone oil exchange with 5000 centistoke oil, then and there. If 1000 centistoke oil is used, early emulsification can ensue.

Above all, it may be necessary to pray sometimes for such a patient as divine intervention works with some other illunderstood mechanisms.

Dr. Manoj S.

The patient definitely requires cataract surgery at this stage. The cataract surgery of choice will be a small incision cataract surgery- either manual SICS or phacoemulsification. The cataract wound will be preferably placed at 12,0 clock so as it does not hinder with the temporal sclerotomy ports for vitrectomy considering the fact that it is an already operated eye with areas of scleral thinning temporally. I would also prefer a scleral wound for better stability.

I would like to assess the state of the posterior segment intraoperatively after the cataract removal by indirect ophthalmoscopy. The rest of the surgery will depend on the state of the posterior segment. The questions that will be there in my mind will be

1- should I remove the oil ?
2- how should I manage the residual RD/ elevated GRT edge if any ?
3- what should I use for retinal tamponade- gas or oil ?
4- should I implant an IOL ?

If there is an evidence of silicon oil emulsification and the retina appears definitely attached then I would proceed with oil removal and also plan an IOL implantation the same sitting.

If there is evidence of retinal detachment then I would perform a silicone oil removal followed by membrane peeling at the region of the detachment if there is evidence of PVR changes. A relaxing retinotomy if needed will be performed to relax the stiff retina. Then I would use PFCL to flatten the retinal detachment and perform barrage laser augmentation. Membrane peeling under PFCL may be helpful if it is difficult otherwise. I would perform a fluid air exchange and air-silicone oil exchange if slippage is not expected otherwise a PFCL oil exchange will be done. I would also implant an IOL in the same sitting.

If the edge of the GRT is elevated and there is no evidence of retinal detachment, then evidence of PVR changes if any at the edge of the GRT is noted. After silicone oil removal these membranes if present have to be peeled or a relaxing retinotomy made. A fluid air exchange will be done followed by endolaser barrage augmentation. As this patient is one eyed I would decide to use silicone oil tamponade inorder to help early mobility. I would also implant an IOL in the same sitting.

Regarding the glaucoma the degree of optic nerve damage- cupping needs to be assessed. If the cupping has not progressed and the IOP is under control with 2 medications then being one eyed he may best be followed up periodically. If there is evidence of advanced glaucomatous damage or uncontrolled IOP he may require an early trabeculectomy after a trial of 3 drug regime and this could be planned along with the silicone oil removal. Field analysis or OCT evaluation of the glaucomatous status may not be feasible in this patient.

The need for multiple surgeries, frequent follow up and the guarded visual prognosis will be explained to the patient.

Discussion

This vulnerable one-eyed patient with primary open angle glaucoma, repaired GRT with incomplete silicone oil fill, and progression of cataract to a visually significant opacity posed several problems regarding further management. Definitely a cataract surgery with IOL implantation was indicated. The density of the lens opacity prevented a detailed fundus evaluation and a firm decision regarding retinal stability, the safety and feasibility of silicone oil removal in combination with cataract surgery. Since there was no evidence of silicone oil emulsification and a definite doubt about the peripheral retinal stability, we decided to perform a phacoemulsification with implantation of a rigid intraocular lens alone postponing the silicone oil removal to a later date preferably after reinforcing the peripheral retinal barrage postoperatively when the media became clear after the cataract extraction. (Fig:2).

We went ahead with the phacoemulsification, and before IOL implantation indirect ophthalmology was
performed. Retina appeared attached and stable with the break sitting comfortably on the buckle indentation. However the retinopexy appeared inadequate. Hence IOL was implanted and the patient advised to report after a month for an LIO barrage for 360° in 6-8 rows.

Since the intraocular pressure was under control with medical therapy the patient was advised to continue the same postoperatively. The patient regained a vision of 6/60 in the second postoperative week. By the end of the 4th postoperative week the vision dropped to 2/60 due to posterior capsular opacity for which yag laser capsulotomy was performed. (Fig 3)

A 360° barrage laser retinopexy under silicone oil was performed using the LIO delivery system. Silicone oil removal was performed 6weeks after laser reinforcement without any untoward effects. The patient at present has a stable retina, controlled IOP on a two drug regimen, and, a visual acuity of 6/24. The importance of regular IOP checking and compliance to therapy and followup has been explained.
Comparison of Ultrasound Biomicroscopic Parameters After Laser Iridotomy In Eyes With Primary Angle Closure And Primary Angle Closure Glaucoma

T Dada, S Mohan, R Sihota, R Gupta, V Gupta and RM Pandey
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Primary angle-closure glaucoma (PAGG) is the major form of glaucoma in the Asian population and an important cause for blindness worldwide. The development of primary angle closure (PAC) into PACG can be prevented by performing a laser peripheral iridotomy (LPI). However, there has been no accurate and objective documentation of the changes in anterior chamber angle morphology induced by an LPI in various subtypes of PACG.

The aim of this study, which was conducted at RP center AIIMS was to find out changes in anterior segment morphology after laser peripheral iridotomy in primary angle closure and primary angle closure glaucoma using ultrasound biomicroscopy (UBM).

Ninety-three eyes of 93 patients underwent anterior segment evaluation including gonioscopy, disc evaluation with +90D lens, applanation intraocular pressure, and standard achromatic perimetry. A single trained ophthalmologist took the UBM images pre-and post-LPI. Trabecular-Iris Angle (TIA), the Angle-Opening Distance (AOD 250/500) and the Central Anterior Chamber depth (ACD) were measured.

The superior TIA widened from mean of 7.54 ± 3.15 to 15.66 ± 6.69° and inferior TIA increased from mean of 9.0 ± 4.7 to 15.9 ± 3.7 after LPI in PAC.

In PACG the mean superior angle changed from 4.55 ± 2.5 to 6.12 ± 3.9 and inferior angle increased from 4.75 ± 2.0 to 7.9 ± 3.7. The mean ACD increased from 2.19 ± 0.36 to 2.30 ± 0.36 mm in PAC group with no significant change seen in the PACG group 1.79 ± 0.32 vs. 1.82 ± 0.33 mm, (P = 0.13).

According to the authors this study is the first one to demonstrate clearly that there is widening of the anterior chamber angle and deepening of the anterior chamber after LPI in eyes with PAC but there is no significant change in any of the anterior segment parameters in eyes with PACG.

In conclusion, this ultrasound biomicroscopic study establishes that LPI opens the narrow angle recess and deepens the anterior chamber in eyes with PAC but is not effective in altering the anterior segment morphology in eyes with PACG.
Archipelago Keratitis: A Clinical Variant of Recurrent Herpetic Keratitis?


Herpes simplex keratitis (HSK) is the leading infectious cause of unilateral corneal blindness in industrialized countries. During the primary infection, HSK is the result of viral replication, and it takes on a dendritic aspect in 50% of cases.

In this study which was designed as a case series study, a series of 6 patients with an unusual form of superficial keratitis were analysed at Department of Ophthalmology, Foundation Ophthalmologique A.deRoth-schild and Bichat Hospital, Paris, France.

Here the authors describe a series of 6 patients with keratitis consisting of foci of epithelial erosions associated with subepithelial nummular inflammatory infiltrates and disposed in a radial, centripetal, archipelago like pattern originating from limbus. All the patients had a past history of herpetic epithelial keratitis, herpetic vesicles on the ipsilateral lid or both.

Polymerase chain reaction – based screening for herpes simplex virus 1 and 2 in corneal scrapings demonstrated positive results in 2 patients. In vivo corneal confocal microscopy revealed focal areas of hyperreflective epithelial cells and hyperreflective subepithelial dendritic structures overlying activated keratocytes. All the patients improved with oral valacyclovir treatment followed by topical steroid therapy.

According to the authors, Archipelago Keratitis may arise from centripetal progression of marginal HSK. This clinical pattern may be the result of the migration of limbal stem cells across the HSV marginal ulcer, resulting in HSV dissemination toward the center of the cornea.

To conclude Archipelago Keratitis may be a new clinical variant of herpetic keratitis, reflecting herpetic dissemination from limbus to the center of the cornea.

Sterile Endophthalmitis after Intravitreal Triamcinolone: A Possible Association with Uveitis


Intravitreal injections of Triamcinolone (IVTA) to treat macular pathologic features have increased exponentially in frequency in the last few years.

The forms of endophthalmitis described to occur after IVTA are infectious endophthalmitis, sterile endophthalmitis, and pseudo endophthalmitis. The latter is not really endophthalmitis and results when triamcinolone particles migrate into the anterior chamber to masquerade as a hypopyon and is more likely to occur in pseudophakic or aphakic patients or patients with peripheal iridectomy.

Normally triamcinolone - crystalline, milky liquid, remains in the vitreous for a few days after injection as a discrete white cloud with little or no reaction in the vitreous.
surrounding vitreous. However, sterile endophthalmitis may occur in rare instance. Sterile endophthalmitis is believed to result from an inflammatory reaction to either triamcinolone or more likely to its vehicle. Kenalog-40 the commercial form of triamcinolone contains 0.99 % benzyl alcohol, 0.75 % carboxymethylcellulose sodium, and 0.04 % polysorbate 80 in the suspension and the presence of these chemicals may serve as a potential stimulus for an inflammatory reaction with the eye.

Factors supporting sterile endophthalmitis include earlier presentation, lack of pain, and relative rapid recovery of vision with good prognosis.

The purpose of this study was to report an association between uveitis and sterile endophthalmitis after intravitreal triamcinolone acetonide injections.

A retrospective analysis of all patients receiving intravitreal triamcinolone injection at the Cole Eye Institute, Ohio US from January 2006 to September 2006 was carried out to evaluate for the occurrence of bacterial or sterile endophthalmitis. Indication for treatment, ocular history, best-corrected Snellen visual acuity and clinical findings were recorded from clinical charts before injection and at last follow-up.

A total of 310 eyes received intravitreal triamcinolone injection for various causes. There were no cases of culture-positive infectious endophthalmitis. There were six cases (1.9 %) of sterile endophthalmitis. Of these cases, four had prior history of uveitis, whereas only 20 out of the 310 cases had a prior history of uveitis. All six patients sought treatment within three days of injection and all recovered rapidly. Presenting visual acuity was either counting fingers or hand movements. Median best-corrected visual acuity before injection was 20/100 whereas median final visual acuity was 20/80.

Reported risk factors for developing sterile endophthalmitis after IVTA injection are pseudophakia with impaired posterior capsule, diagnosis of CME resulting from Irvine-Gass syndrome, and prior vitrectomy.

This study suggests that prior history of uveitis may be an additional risk factor for developing sterile endophthalmitis after IVTA injection. Four (67 %) of six patients with sterile endophthalmitis had history of uveitis, whereas only 20 (6.4 %) of 310 total patients who received IVTA had history of uveitis. Thus, in patients with a history of uveitis, there was an incidence rate of 20 % (four of 20) compared with 0.68% (two of 290) for other causes.

In conclusion, an additional risk factor for developing sterile endophthalmitis after IVTA injection is prior history of uveitis. Such patients should be advised about the increased risk and caution should be exercised before injecting these patients.

Reviewed by Dr Alex Baby DO, DNB. Little Flower Hospital and Research Centre, Angamaly.
Minimally Invasive Cataract Surgery (Bimanual Phaco/MICS)

Edited by: Ashok Garg MS Ph.D, I Howard Fine MD, David F Chang, Hiroshi Tsuneoka MD.
Published by: Jaypee Brothers, New Delhi, First – 2007.
Price: 595/-

Phacoemulsification techniques have advanced at an astounding rate in the last decade. The most recent breakthrough is still further reduction in the size of incision from standard 3.2 mm to below 1 mm and implanting the foldable IOLs through the same incision. This technique known as Minimally Invasive Micro-incision Cataract Surgery (MICS) is really worthwhile technique to deliver quality vision and ensure rapid rehabilitation of the patient.

Under several banners, including microincision cataract surgery, microphaco, and phakonit a global consortium of surgeons and industry leaders have developed the capability to remove a cataract through two or three 1.0 to 2.0 millimeter incisions. These techniques, while continuously evolving, are now robust enough to teach to every ophthalmic surgeon.

This book Step by Step Minimally Invasive Cataract Surgery (Bimanual Phaco/MICS) written by internationally renowned Dr. Ashok Garg and his co-editors summarize the current art and science of minimally invasive cataract surgery. Accompanied by two interactive CD ROMs, this book provides the interested surgeon a quality base of knowledge to expand their cataract surgery and lens implantation skills.

Ophthalmic Surgery

Edited by: Sandeep Saxena MS MAMS
Published by: Jaypee Brothers, New Delhi, First Edition - 2007.
Price: 395/-

No ophthalmologist can be an expert in all facets of ophthalmic surgery. In this challenging intellectual environment, an atlas bringing together eminent authors from multiple subspecialties is welcome.

Dr. Saxena along with his co-authors have produced a book, which is an important contribution to contemporary ophthalmic education, which will be a welcome addition to the library of ophthalmologists around the world.

Ophthalmic Surgery provides a comprehensive mini atlas in the form of black and white and colored figures complemented with text and several surgical videos. This atlas attempts to update the state of art in ophthalmic surgery in a lucid, authoritative and well-illustrated manner.

The value of this book lies in the quality and expertise of the text chapters and surgical video contributors from around the globe. This book is intended for the experienced ophthalmologists, postgraduates as well
as those in training. It is hoped that this atlas will be sufficiently comprehensive to aid the ophthalmic surgeon in performing competent surgery. This pragmatic book provides an understanding of surgical techniques so important to the everyday practice of ophthalmology.

Phacoemulsification

Edited by: Sunita Agarwal MS, Athiya Agarwal MD, Amar Agarwal MS FRCS
Published by: Jaypee Brothers, New Delhi, First Edition – 2007.
Price: - 495/-

This mini atlas on Phacoemulsification offers a concise, well illustrated and demonstrative approach to intricacies of phacoemulication. With so many photographs and illustrations, this mini atlas is an easily accessible and navigable data source.

It starts with a section on basics of phaco and the simple, yet very helpful concept of gas forced infusion popularised by Sunita Agarwal few years ago, and goes through other steps of the phaco technique. A section on tough cases like subluxated nuclei then follows.

Complications and nightmares are faced even by the most the experienced surgeon, and to these, a special section is dedicated.

This book is a small book so that it can be kept in the operation theatre or carried in the hand. When one is starting a tough case the book can be read easily so that the difficulty of the case can be easily conquered. There are lots of figures and a DVD-ROM also in the book so that one can understand the concepts easily.

Biaxial Lens Surgery

Edited by: Arturo Perez-Arteaga MD
Published by: Jaypee Brothers, New Delhi, First Edition – 2008.
Price: - 595/-

Many books are now worldwide in the medical market about the topic of biaxial lens surgery with many names (e.g. MICS, Phakonit and Bimanual). Most of them are divided into chapters according to the surgical steps and some others according to the surgical techniques employed by different surgeons: hence the reader grasps different ideas and there is no definite sequence of steps in his mind to achieve a good enough learning curve. This is the cause of the loss of strength, charm and good feeling about biaxial surgery.

This book is an attempt to take the novice surgeon in a sequential teaching-learning process where the surgeon is able to go one step after another converting in a safe way and feeling all the advantages of each new step he is doing in the biaxial technique. The structure of this sequence allows the surgeon to go back to the technique he is accustomed to perform at any time he wants, needs to or feels afraid to go further. In this mode each surgeon can choose the speed of converting according to their capacities, abilities, cases, patients and feelings.

As you will see when you travel through the different chapters and DVD – ROM of this book, that the purpose of the authors is to guide you step by step, provide solid theoretical information, guiding you to the best way of getting the benefits of using a new surgical technique avoiding the most common pitfalls or mistakes to get a successful outcome.

In the first section, the authors have presented a review of history, current concepts and technology involved in cataract surgery and after in the particular field of biaxial cataract surgery.

In the second section, the core of this book, the authors are taking the learning surgeon in a sequential teaching-
learning process where the surgeon is able to go one step after another converting in a safe way and feeling all the advantages of each new step he is doing in the biaxial technique.

In the third section the authors show different situations and complications that can be more easily solved with biaxial techniques, rather than coaxial.

Small Incision Cataract Surgery

Edited by: Anita Panda MD MRC, Tanuj Dada MD.
Published by: Jaypee Brothers, New Delhi, First Edition – 2007
Price: - 395/-

Manual small incision cataract extraction has become the state of art surgery for management of age-related cataracts in many developing countries.

Although large textbooks have been written on manual small incision cataract surgery, it is difficult for the trainee surgeon to decipher useful information from these large texts. This book has been conceived to provide the reader with a video-assisted instruction course on “how to do a manual small incision cataract surgery”, covering each step of the surgical procedure. Each surgical detail has been explained in the text and added as a video-clip to enable the reader to actually perform the maneuver during surgery.

Salient features of the book are:-

- Video-assisted instruction course on basics of manual small incision cataract surgery (SICS)
- Covers each step of the surgery including incisions, capsulotomies, hydroprocedures, nuclear delivery methods, cortical aspiration, IOL insertion and wound closure
- Each surgical step explained with a video clip
- Includes surgical instrumentation required for SICS
- Provides practical tips on management of complications
- Well-illustrated with over 70 color photographs and 2 interactive CD-ROMs
- A must for residents and practicing ophthalmologists

(Dr. C. V. Andrews Kakkanatt, JMMC Thrissur)
STATE CONFERENCES

“CME on OSD”
6th April 2008
Regional Institute of Ophthalmology, Trivandrum
Dr. Chithra Raghavan
Ph: 09387207227

CME on “What is possible in Ophthalmology with the latest in Technology “
20th April 2008
Vasan Eye Care Hospital, Kochi
Dr. Anil B. Das
Ph: 09895780923

Chaithanya Academic 2008 CME on Retina
27th April 2008
Chaithanya Eye Hospital, Trivandrum
Dr. Unnikrishnan Nair
Ph: 0471-2447183.

CME on Retina
4th May 2008
Al-Salama Eye Hospital, Perinthalmanna
Dr. Mohammed Swadique
Mob: 9895225511

CME by Palakkad Ophthalmic Club
18th May 2008
Palakkad Ophthalmic Club, Palakkad
Dr. Rajesh Radhakrishnan

“Eye – A systematic Approach”
26th May 2008
Comtrust Eye Hospital, Tellicherry
Dr. Sreeni Edakhlon
Ph: 2721620, 2723793

INTERNATIONAL CONFERENCES

ASCRS
ASCRS Symposium on Cataract, IOL and Refractive Surgery
April 4-9, 2008

ASCOA
ASCOA Congress on Ophthalmic Practice Management and Clinical & Surgical Staff Program
April 4-8, 2008
Chicago 2008
McCormick Place, West Building
American Society of Cataract and Refractive Surgery
American Society of Ophthalmic Administrators.

“Complications and Difficulties in Cataract Surgery”
2nd June 2008
AIMS Kochi
Dr. Gopal S Pillai
Mob: 9447391266

CME on Low Visual Aids and Contact Lens
15th June 2008
Little Flower Hospital, Angamaly
Dr. Alex Baby, Organising Secretary
Mob: 94470 13856

CME 2008
6th July 2008
Chakrabarti Eye Care Centre, Trivandrum
Dr. Arup Chakrabarti.
Mob: 9946410540
28th June-2nd July 2008

**WOC 2008. World Ophthalmic Congress**

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Symposium of Asia Pacific Academy of Ophthalmology
XII Congress of Chinese Ophthalmic Society.
XX Hong Kong Ophthalmological Symposium
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Web: www.woc2008hongkong.org

**XXVI Congress of the European Society of Cataract and Refractive Surgeons**

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Email: escrs@escrs.org
Web: www.escrs.org

2008 SEAGIG and AACGC Joint Congress

September 25-27, 2008
Venue: Seoul, Korea
Organised by Organising Committee of SEAGIG 2008 and Seoul AACGC 2008
Web: www.seagig-aacgc.org

2008 Joint Meeting of the American Academy of Ophthalmology and the European Society of Ophthalmology (SOE)

November 8-11, 2008
Venue: Georgia World Congress Centre, Atlanta Georgia
Web: www.aao.org.
Friedrich Wilhelm Ernst Albrecht von Graefe, Ophthalmologist Extraordinaire

Prof. Padmaja Krishnan, Kozhikode

Friedrich Wilhelm Ernst Albrecht von Graefe, the Father of modern Ophthalmology, was born in Berlin on 22nd May 1828 to Karl Ferdinand von Graefe, a surgeon in the Prussian army. The family was rich and he had a happy childhood despite being orphaned at the age of 12.

In the autumn of 1843, not yet 16, von Graefe joined the University of Berlin to study Philosophy, Logic, Natural Sciences and Anatomy. He was a brilliant student and his teachers, Virchow, Schlemm, von Brücke, Du Bouis-Reymond, and Johannes Müller, expected much of him. Four years later, in 1847, he graduated in Medicine with honours, but could not decide on his specialization. So beginning in December 1847, von Graefe travelled through Europe to work with the leading specialists of that time. He was so impressed by Ferdinand Arlt in Prague that he chose Ophthalmology as his career. He then went to Paris to be with Sichel, Desmarres, Louis and Claude Bernard and to Vienna to observe the Jaegers- father and son.


Von Graefe got back to Berlin in 1850, just 22 years old, and enriched by his experience, founded his own clinic on 1st Nov 1850. He welcomed his first patients with a famous advertisement that ran in all the Berlin papers for six weeks, stating that he would treat poor patients free of charge. Though he had only three small rooms and little equipment, he soon had a surprisingly busy practice.

That same year Hermann Ludwig Ferdinand von Helmholtz had announced his great invention, the ophthalmoscope, and von Graefe was the first to use it routinely. He was thus able to describe retrobulbar neuritis as a condition where “the observer sees nothing and the patient very little”.

von Graefe : stamp issued by West Germany in 1978 on his 150th birth anniversary
Von Graefe’s first surgical cases were a cataract extraction and an optical iridectomy in an organ grinder. He did these in two rooms he rented from a tailor. Despite the operations going well, the patient of cataract developed delirium tremens on the evening of surgery, fell out of his bed and attacked the organ grinder with his fists!

The great care he took of his patients and his genius as a scientist gave his clinic a great reputation, not just among his patients, but also among his colleagues all over the world. As he grew rich he treated many patients for free.

With his immense reserves of energy, von Graefe was able to carry on a punishing work schedule and yet devote time to his physiological and other scientific studies. He noted that many patients had visual impairment without organic lesions of the eye, diagnosed sudden visual loss due to retinal artery embolism, studied and classified glaucomas, introducing the term “simple” glaucoma into literature. He searched for ways to properly examine the eyes, pioneered the design and use of instruments for clinical perimetry, and in 1862 developed one of the very first tonometers for ophthalmic practice.

His thesis ‘On the action of the ocular muscles’ submitted to the University of Berlin in 1852 made him a qualified teacher. Shortly after, his lecture on the surgical correction of squint caused a sensation. He was an excellent teacher and postgraduates from all parts of the world attended his clinics. Karl Weber commented “One was spell-bound in his clinic, as if in a magic place. The multitude of new facts and viewpoints never heard before, the fascinating presentation and glowing enthusiasm acted like a revelation”.

In 1854, von Graefe, aged 26, founded the journal Archiv für Ophthalmologie. In its first issue of January 1854, 400 of the 480 pages were authored by him and some articles had the nature of a medium-sized monograph. By publication of volume 16, he had contributed 2500 pages. Because of his great reputation, he was able to recruit Arlt and Donders as co-editors. The journal was later renamed Albrecht von Graefe’s Archiv für Ophthalmologie. It is still published by Springer under the title of Graefe’s Archive for Clinical and Experimental Ophthalmology.

He was appointed Associate Professor of Ophthalmology in Berlin in 1857- the first German professor of diseases of the eye, and became a full professor in 1866. In 1864, von Graefe modified the standard cataract operation. By using a thin pointed sword-like knife which he created himself, he altered the conventionally used semicircular corneal incision to one under a conjunctival flap. This procedure improved the success rate of cataract surgery, reduced infection and was much appreciated by von Graefe’s peers; it was then used by countless ophthalmic surgeons all over the world and well into the 20th century.

**Bottom of Form**

He maintained lively scientific and friendly associations with the grand men of Ophthalmology who, with him, laid the foundation of modern ophthalmology: Hermann von Helmholtz, the genial physicist and optician, Ferdinand von Arlt, the great clinician and surgeon who caused von Graefe to devote himself to Ophthalmology, Cornelius Donders, the founder of the new doctrine of refraction, Eduard von Jaeger, the skilled ophthalmoscopist, William Bowman, the anatomist, and Friedrich Horner. Among the students who flocked to him from all over the world, were Argyll Robertson and Theodore Billroth who were impressed by his “great personal kindness and his great humility”.

In personal appearance von Graefe was tall, slender and elegant, with a handsome face, long dark hair and a full beard. He was modest in his lifestyle; his greatest pleasure was entertaining his friends to dinner at home as his massive workload left time for little else.

In 1861 he developed tuberculous pleurisy, but his condition went into remission and the following year he married Anna Gräfin Knuth. The couple had five children through their eight years of marriage, two of whom died.

His pulmonary tuberculosis reactivated, and on 20th July 1870, he died in Berlin, aged only 42.

His contributions to ophthalmology were multiple and in his short career he performed over 10,000 eye operations. His name continues to be remembered for the von Graefe sign in exophthalmic goitre and the von Graefe cataract knife. He was undoubtedly the most important ophthalmologist of the 19th century.
CRVO: A Review On Management Modalities

CENTRAL RETINAL VEIN OCCLUSION

- Unilateral Visual Loss: extensive IR H/ages, Oedema
- 34% with CRVO: Ischaemic
- 45% - 85% NVI & NVG in I-CRVO
- 5% NVI & NVG in NI-CRVO
- 50% Associated with Systemic HT
- 40% Associated with Glaucoma
[CRVO STUDY GROUP: ARCH OPHTHALMOL 1993; 111: 1087-1095]

- CRVO: Common Visually Disabling Disease
- CRVO: Unfavourable Natural History
- CRVO: No Effective Proven Therapy to Date
- CRVO: No Significant Publishable Activity in literature
- CRVO: Multiple Aetiopathogenesis: Vascular, Haemorheological, dynamic, mechanical
[CRVO STUDY GROUP: Natural History & Clinical Management of CRVO
[ARCH OPHTHALMOL 1997; 115 : 486-491]

PAN RETINAL PHOTOCOAGULATION

- NV Complications of CRVO—Reduced

CRVO STUDY GROUP REPORT

Evaluating Grid Pattern PHC for Macular Oedema in CRVO
- Reduction in Amount of Macular Oedema
- No Visual Improvement

LASER INDUCED CHORIORETINAL ANASTAMOSIS

- Successful in 30% of Cases
- Associated with Clinical Improvement
- Frequent Complications
  - *Retinal H/ages *BRVO *Fibrosis
  - *TRD *VH *CNVM

- Intentional Complete Interruption of Retinal Vein after Vitrectomy - Might Improve the Rate of Successful Chorio Retinal Anastamosis
  - PPV – Cutting off the Affected Retinal Vein.
  - Making Small Incisions on Either Side of Vein.
  - Interruption thro Full Thickness Bruchs’ Membrane.
  - Successful C - R Venous Anastamosis.

ERBIUM – YAG LASER INDUCED CHORIO RETINAL VENOUS ANASTAMOSIS FOR MANAGEMENT OF ISCHAEMIC CRVO
- PPV ; PVD ; 4 Erbium : YAG Laser Induced C - R Anastamosis (1 / quadrant)
- Resolution of h/ages, ME, Visual Improvement
- Successful C - R Anastamosis
- No NV Complications

Correlation of Increase VEGF with NV & ↑ Permeability in I-CRVO
Boyd SR; Zachary I, Chakravarthy U et al [Arch Ophthalmo 2002 Dec 120 (12) : 1644-50]
- VEGF Levels in Aqueous ˜ Onset , Persistence Regression of NVI, Extent of CNP; Vasc Permeability
- Anti VEGF Therapy in Early Stage Therapeutically Beneficial

ROLE OF ISOVOLEMIC HAEMODILUTION IN CRVO
- Confirmed Benefit
  - Haemorheological Parameters
  - Retinal Circulation Time
  - Final V/A
- Benefit of Early Tt. (within 1st Two Weeks)

RADIAL OPTIC NEUROTOMY FOR CRVO: Retrospective Pilot Study of 11 Consecutive Cases
E. Mitchell ;Opremcak; Robert Bruce et al, RETINA 21; 408-415; 2001
Intra Vitreal ‘TA’ for Treatment of Macular Oedema in CRVO


LOGIC BEHIND RON

Hypothesis that CRVO is a Neurovascular Compression Syndrome (Scleral Outlet Compartment Syndrome)
Pressure within Confined Space of Scleral Outlet
Radial Cut in ON Opens Scleral Ring - Releases Compression
Incising Lamina Cribrosa— Radial Optic Lamino Neurotomy

Does It Really Work ? If Yes – How ?
Relieves Compression
↑ CRV Lumen Size
↑ Venous Blood Flow
Clearing of Venous Thrombosis

Mechanism of Action

Doesn’t Really Work– Hayreh et al (Retina , 2002 Dec 22 (6), 827)
Development of Opticociliary Shunt Vessels (Dev in 30% of I-CRVO) (Giufe et al BJO 1993, Dec 77 (12))
Radial Cuts May Induce Enough Trauma to Stimulate OCV Growth

Treatment of CRVO by Vitrectomy with Lysis of Vitreopapillary & Epipapillary Tissue & TPA Injection and Photocoagulation


Chronic CRVO Associated with VP & EP Traction

Super Selective Ophthalmic Artery Fibrinolytic Therapy for Treatment of CRVO

6/9 Eyes with Combined CRAO-CRVO Improved

Injection of tPA into BRV in Eyes with CRVO


CORTICOSTEROIDS IN CRVO : Mx

Dramatic Improvement in VA & Macular Oedema

ORAL STEROID

Shaikh & Blumenkranz : Retina 2001, 219 (2) 176-178

I / Vit. Inj of TA

JP, Kumar et al Arch Ophthalmol 2002 Sep 120 (9)

Role of Triamcinolone Acetonide in Recalcitrant Macular Oedema

Local Reduction of Inflammatory Mediators
Increased Diffusion by Modulation of Ca Channels
Decreased Levels of VEGF
Restoration of Blood Retinal Barrier

Intra Vitreal ‘TA’ for Treatment of Macular Oedema in CRVO


Anti Vegf Therapy in NI-CRVO

Trials with Bevacizumab and Ranibizumab
Dramatic regression of Macular Oedema

Anti Vegf Therapy in I-CRVO with Neovascular Sequelas

Regression of Neovascularisation
Recurrence / Relapse ??

Compiled by:

Dr. Meena Chakrabarti, Editor, KJO
GENERAL INSTRUCTIONS TO AUTHORS

The Kerala Journal of Ophthalmology (KJO) is a quarterly; peer reviewed one, devoted to dissemination of the latest in ophthalmology to the general ophthalmologists as well as to specialists in the various subspecialties of this discipline. It invites submission of original work dealing with clinical and laboratory materials.

Authors submitting materials to this journal are requested to adhere STRICTLY to the norms laid down below. The matter must be typed on one side of the paper. A margin of 1” must be left all around and the material must be double-spaced. A page should contain not more than 25 lines. Two copies of the text in paper and one copy in a CD must be submitted to the Editor and the corresponding author is advised to keep another copy with him. The corresponding author must give it in writing in his covering letter that the same matter will not be submitted elsewhere if accepted. He must also enclose the copyright transfer of his work to this journal. The papers sent will be subjected to peer review. The accepted manuscripts become the permanent property of this Journal. The author is informed that, if his work is returned to him for correction / clarification after peer review, he should effect the same and send the manuscript back to the Editor within one month. Each manuscript component mentioned here under must begin with a new page and the pages are to be numbered at the right tip corner starting from the Title page.

1. TITLE: The title of the work must be brief and precise. It should not exceed two lines and 40 characters (including comma, period) Author (s) full name (s) must be given along with his (their) degree and the affiliations. Corresponding author’s name, correct address (including e-mail and Fax, if available) and phone number must be mentioned at the bottom left hand corner of the first page.

2. ABSTRACT: The abstract is to be given in the beginning itself. It should not exceed 200 words. It must contain the aim, methodology, results and conclusion. For case report, summary / conclusion alone is to be given.

KEY WORDS (maximum five) in capitals are to be included at the end of Abstract.

3. INTRODUCTION: Describe the aim of the study, along with the hypotheses that were tested. Only necessary references are to be given.

4. METHOD: Give in detail the materials used and the methods employed. Describe the type of study. Pharmacological names only must be mentioned for the drugs used and, if proprietary name is used, then the manufacturers name must be given in parentheses. Except for standard, well-accepted abbreviations (Including SI Units), all others must be introduced in parentheses when the full term is used for the first time in the article.

5. RESULTS: Give only the results obtained by the study under discussion. State the statistics in the correct scientific form (P value, mean etc). Results based on assumptions must not be given. Indicate in the text the place where the tables have to be inserted.

6. DISCUSSION: The discussion should be to the point and relevant to the subject under discussion. This section can be combined with the previous one if the author desires. Avoid speculations. Use only standard abbreviations or the abbreviations already introduced.

7. ACKNOWLEDGEMENT: This is to be made only to those who were directly and scientifically involved with the preparation of the paper. Permitting authorities, technicians, photographers who assisted in the work need not be mentioned.
8. REFERENCES: The references should be given in numerical order in which they first appear in text and not in alphabetical order (Citation Order System). It should be numbered consecutively in the text. The references will not be checked by the Editor or by the Peer reviewer and hence the author is solely responsible for its completeness and the accuracy. Period should not be employed anywhere in the references. Personal communication, unpublished data and poster references, if mentioned, should be in the text itself and the source mentioned in parentheses. References should be in the following form:-

Journal reference: Author(s) full title, Journal name (as abbreviated in Index Medicus), volume number, pages and year. If there are more than three authors, then mention the first three authors and then ‘et al’.

Book reference: Authors(s) (& Editor, if any), title of book (and chapter), publisher, place of publication, page number (s) of the cited portion and year.

9. THE LEGEND: The legend for the illustrations (and tables, if necessary) must be given in a separate sheet of paper and should be typed double-spaced.

Illustrations: The photos and figures should be prepared in glossy prints with good contrast and of the size 6” x 4”. Only salient details should be included. On the back of the illustration, the figure number in text, title of the paper, the first author’s name and the top side (marked with an arrow) must be specified. Except for arrows, no text is to be on the photos. It is the duty of the author(s) to get the patient’s written permission when the subject is identifiable in the photo. Submit two sets of illustrations. Illustrations from other Journals and books are usually not accepted. If used, it rests with the author(s) to get the copy right permission from the original author / publisher and this permission letter must be sent to the Editor at the time of submitting the manuscript. For Histological figures the stain and magnification used should be noted e.g.: - H & E Stain x 70.

10. TABLE: It should be in double space. Each table must have an Arabic numeral (except for single table) and a title both in a single line. Each column in the table must have a short heading. If a table is large, then it must be continued in a second page, which also must have the table number and the title. Avoid vertical lines in the tables. Two sets must be submitted.

The manuscripts are to be sent to The Editor by Courier Mail or by Registered post. The corresponding author will receive communication from the Editor within two weeks of receiving the manuscript.

All manuscripts are subjected to editorial board review.

12. Other Categories of Manuscript

a) Original Articles should generally not exceed 3,000 words or 12 double – spaced pages.

b) Review Articles: can be on topics of relevance to clinical practice, research methodology, community ophthalmology or investigative work, of relevance to visual science. These articles should include up to date review of existing literature, and summarize the current status / preferred practice for that particular topic.

Brief reports are short communication of new instruments, new laboratory techniques or surgical techniques as well as interesting case reports with unique findings. These should not exceed 1000 words with a maximum of 2 illustrations. They should follow the format - introduction, case, and discussion. No more than 8 references should be cited. Each brief report must begin with a 75-100 word summary that highlights the significance of the articles.
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Welcome to KSOS

All new members to this community are welcome to use the KSOS website that is feature rich and informational. The website address is

www.ksos.in

How to register as in the website?

All members are requested to provide the webmaster of KSOS, their details such as
1. First Name     2. Last Name     3. email id

What the webmaster will do:

The webmaster will add these details into the member list. The system will generate the User Id and Password, which will then be emailed to you.

Once the members receive their user ids and passwords, they are requested to visit the website and log in using the Member Login area (top right of the website). Then use the “View My Account” link to see your account details. Please update your details in that section. You are also allowed to change your User Id and Password, to your liking. Please use names that you can always remember, for your user id. If you change your password, do memorise the new password.

Online facilities for the Members

KSOS members have access to very good online resources. All members can then take advantage of the advanced facilities provided such as

1. KSOS journals  Members alone can access the KSOS journals online. You have to login to access the journals.

2. Message Board  Members alone can feed messages into the message board for the rest of the KSOS members.

3. Discussion Forum  for members  All members are also requested to take part in the discussion forum (a new feature) in the website. For this, you are requested to register separately. The instructions for this have been given in the “News Update” section of the website.

4. Video Streaming has been added to the website

This feature has been added to the KSOS website, making the website even more vibrant. It features streaming video content of medical surgeries and procedures.

5. Useful links  The KSOS Website provides links to useful and important websites across the world.

6. Member search  KSOS members can access the contact details of any other member, using the website. All members are requested to visit the website and see the features in the website and get accustomed to the online system.