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“Beyond The IOL”: Will the Cataract Surgeon Be Put Out of a Job?

Implanting IOLs has become a way of life for many cataract surgeons – a routine uncomplicated operation that dramatically restores vision and improves the quality of life of millions of patients worldwide. However, a change in the wind by way of preliminary results of research on “dissolving cataracts”, and “growing your own lens” are in the pipeline and may become a cause for cataract surgeons to panic! How close are we to a world beyond the IOL……… where cataracts can be dissolved and the lens replaced with a new one grown from stem cells? 1

Way back in the December of 2003, the British newspaper The Daily Mail splashed in its headlines, the discovery by Russian Scientist of “eye drops that dissolved cataract” in a matter of few weeks. This revolutionary treatment gained popularity in Europe after being featured in a television show. Eltros Gmbh Schweiz, the Swiss manufacturer claimed that this could mean an end to cataract surgery ! Their product called Ethos Endymion “Bright Eyes” 2 used a special derivative of the naturally occurring neuropeptide L- Carnosine also called N- Acetyl Carnosine (NAC) which is a super antioxidant and antiglycating agent. Clinical trials carried out claimed efficacy in dissolving 100 % of primary senile immature cataract and 80 % of mature cataracts over a 6 month period with sustainable results 24 months later. The product was advertised as “Bright Eyes: a simple inexpensive course of eye drops to dissolve cataracts as opposed to invasive surgery” and was marketed at 100 Swiss Francs/ box of 5 vials each containing 2 ml of the drops.

N- Acetyl Carnosine (NAC) 2 the chemical component of this eye drop was later stripped of its magical cataract dissolving powers. Interest in eye drops to cure cataract was reawakened in mid 2007 when Dr. Randall. J. Olson3 of Moran Eye Centre, University of Utah presented evidence that a formulation of the heavy metal chelator EDTA had the power to dissolve cataract. The phase I and phase II FDA trials showed positive results. The topical treatment appeared to ‘clear’ multi lamellar bodies found 10 times more frequently in cataract than in normal lenses.

The results of the phase III trials will definitely tell us whether a permeabilised formulation of EDTA is here to stay or will follow the same disastrous journey of N- Acetyl Carnosine.

Results of the several new studies have shown that you may be able to “grow your own lens” after cataract surgery. The possibility of lens re-growth is by no means restricted to newts, other amphibians and fish - mammals including man have been known to regrow lens tissue after cataract removal. Regrowth as we all know definitely occurs – only rarely is clear lens tissue regenerated. More frequently the new lens fibres are chaotically organized and opaque and serves no useful visual purpose.
For the past two decades Dr. Gwon from the University of California has been experimenting with various methods of encouraging regrowth of clear lens fibers across the capsular bag. Inflating the capsular bag after phacoemulsification with air or any other material, to keep the walls of the capsule taut and to prevent them from sticking together is the first step. Beyond that Dr. Gwon’s research has focused in providing a suitable scaffold to entrain fiber-cell growth encouraging regular parallel growth characteristics of a clear lens. Most effective results were obtained when hyaluronic acid was used as scaffold.

Using this scaffold Dr. Gwon has successfully re-grown a clear lens in rabbits at least a couple of times! However there are plenty of hurdles to overcome. The rate of regrowth is frustratingly slow and may be expected to be even slower in elderly patients. The second technical difficulty involves removing the hyaluronic acid scaffold after a few weeks. Researc is underway using hyaluronidase - a technically demanding procedure - to dissolve the scaffold!!

So the logical conclusion to this is to ponder whether the time has comes for the cataract surgeon to panic? When will this “world beyond IOL” put cataract surgeons out of jobs? There are promising evidences that powerful topical application may block cataractogenesis and dissolve existing cataracts. Acceptance of this mode of therapy by ophthalmologists will depend on the result of phase III trial. This may be years ahead and will be applicable to only a few types of cataracts.

Lens regrowth will be at least a decade before realization and will still require an accomplished cataract surgeon to perform the phacoemulsification and may be it will turn out to be a more interesting job than the standard phacoemulsification with IOL implantation.

References
1. Nick Lane PhD, Beyond the IOL Eurotimes, Vol 12 Issue 9, Sept 2007.
2. Bright eyes drops for pets and for people; www.google.com

Dr. Meena Chakrabarti MS DO DNB
Editor
Amblyopia - Current Trends in Management

Dr. Ramesh Murthy MD FRCS

Introduction
The detection and management of amblyopia lies at the heart of pediatric ophthalmology. As John T Flynn noted in the 17th annual Frank Costenbader lecture “it is to amblyopia to which my heart returns” 1. Till recently, literature on amblyopia consisted predominantly of retrospective reviews. In 1997, the pediatric eye disease investigator group (PEDIG) was formed to conduct clinical research in eye disorders affecting children. The studies were conducted through simple protocols with limited data collection and implemented by both university and community based pediatric eye care practitioners as part of their routine practice. Amblyopia is the most common cause of monocular visual impairment in children and young and middle aged adults and hence PEDIG has laid emphasis on studies of treatment modalities of amblyopia, the Amblyopia Treatment Studies.

Definition
Amblyopia has been defined as corrected visual acuity of >20/40 or >1 line difference in corrected visual acuity between the 2 eyes. This is not a result of any organic problem of the eye or the visual pathway 2.

Prevalence
The prevalence of amblyopia in the age group 4 years has been reported to be 1.07 % in the population screened at infancy, to 2.57 % in the population that has not been screened at infancy 3. The optimal timing to screen amblyopia has been a matter of debate. In the Avon longitudinal study of parents and children, an assessment of the effectiveness of early treatment of amblyopia was performed. It was noted that intensive screening protocol at a young age resulted in better amblyopic eye acuity at 7.5 years of age in those who underwent screening 4. A Cochrane review on the value of programmes for screening showed that the lack of data from randomized trials makes it difficult to analyse the impact of existing screening programmes on the prevalence of amblyopia. This does not imply that vision screening is not beneficial, but that the intervention has not been tested in robust trials 5.

Neuronal Basis of Amblyopia
The visual processing system is susceptible to the influences of abnormal environmental factors. The seat of amblyopia is not the retina but the striate and the extrastriate cortex. The effects on the lateral geniculate nucleus are minimal 6. The cells of the layer 1,4,6 from the contralateral and 2,3,5 from the ipsilateral eye can be affected if monocular deprivation occurs early. There were two sets of synaptic inputs and with experience one pathway from the open eye takes over preempting the territory of the closed eye.

In the visual cortex, the principal abnormality is at the level of the primary visual cortex (striate cortex, V1, Brodmann’s area 17) 6. Normally eye inputs to the area IV C are divided equally between the eyes. The deprived eye shows a marked shrinkage of its input stripes (ocular dominance columns) and a corresponding expansion of the nondeprived eye 7. Radioactive aminoacids and axonal transport from eye to cortex.
shows a marked shrinkage of the input stripes (ocular dominance columns) and a corresponding expansion of the non deprived eye. In addition to the differential retraction of terminals, the result is produced by sprouting of axonal terminals. Changes in the area V1 are qualitatively related to the depth of amblyopia. Changes in V1 typically predict smaller deficit than measured behaviourally. Qualitatively abnormalities in the eye dominance columns and the spatial properties of visual cortex neurons were related on a case by case basis to the depth of amblyopia. Quantitative analysis suggests that these abnormalities alone do not explain the full range of visual deficits in amblyopia and there may be unknown abnormalities in the extrastriate cortex \(^8\). The suggested mechanisms of visual loss in amblyopia include abnormal neural response properties, poor synchronization of neuronal responses, abnormal topographic representation of topographic receptive fields and undersampling of visual space \(^8\).

At the molecular level, synaptogenesis determines the loss of vision and improvement in amblyopia \(^1\). If performed early in the critical period, patching the good eye can lead to a complete switch in fixation preference \(^1\). The geniculate innervation of layer IVC reverses and the initially deprived eye can take over much of the lower part of layer IVC, but fails to reverse the domination of the other eye in the upper part of layer IVC. The critical period is different for different cell types. Even after prolonged patching the loss of binocular cells may not reverse and there may be permanent loss of stereopsis. The risk associated with unilateral patching or alternate patching is the loss of stereopsis and not fusion. Levodopa can help in the improvement of visual acuity and pattern VEP amplitudes \(^10\). Cytidine 5’ diphosphocholine also improves visual acuity, contrast sensitivity and visual evoked potentials of amblyopic subjects. Functional MRI a relatively new modality may reveal changes which can be used to assess the effect of amblyopia treatment in humans.

**Recent Concepts in Management**

**Atropine versus patching**

This was studied in the first PEDIG trial (amblyopia treatment study 1). The objective was to compare patching of the sound eye with atropine instillation. In this prospective randomized multicenter clinical trial, moderate amblyopes (20/40 to 20/100) in the age group 3 to 7 years were included. 419 patients with an average age of 5.3 years were enrolled. 96% completed examination at 6 months. The atropine group received 1 drop of 1% atropine in the sound eye daily. If by 4 months, acuity had not reached 20/30 or improved by 3 lines from baseline, distance correction was removed to augment atropine effect. Patching group was prescribed minimum 6 hours patching daily, if by 4 months they had not reached 20/30 or improved by 3 lines from baseline, full time patching was prescribed. The mean visual acuity in the amblyopic eye at enrolment was 20/63 with a mean difference of 4.4 lines between eyes. Visual acuity improved 3.16 lines in the patching group and 2.84 lines in the atropine group. Improvement initially was faster in the patching group, but at 6 months difference in acuity between the 2 groups was clinically insignificant. At 6 months, 79% in patching group and 74% in the atropine group had acuity 20/30 or better and/or improvement from baseline by 3 lines. The conclusions were both treatments were well tolerated, though atropine had more tolerability and patching works faster than atropine \(^11\).

A 2 year follow up of the same study group to look at the long term follow up revealed that the improvement was 3.6 lines in the atropine group and 3.7 lines in the patching group. In moderate amblyopia, atropine and patching both showed moderate improvement. The amblyopic eye acuity was also noted to be 2 lines less than the worse eye. This study gave concrete evidence about the well known aspects of atropine and patching \(^12\).

**Part time occlusion**

Patching regimens used to be divided into full time patch for days depending on the age. Compliance and the risk of occlusion amblyopia was an issue with these regimens. The ATS 2A (amblyopia treatment study 2A) compared 6 hours versus full time patching for severe amblyopia (20/100 to 20/400) in children 3 to 7 years old. In this prospective study 175 patients were enrolled with an average age of 4.8 years. The severe amblyopic patients were randomized to 6 hours or full time (all but 1 waking hour daily patching). The mean acuity at enrolment was 20/160, with a mean difference in acuity between the eyes of 7.8 lines. The 4 months follow up
was completed by 90% patients. Mean improvement in the visual acuity was 4.8 lines in the 6 hour group and 4.7 lines in the full time group. At 4 months there was no difference in acuity between the groups. 86% of the patients in the 6 hour group and 82% in the full time group had improvement in acuity of more than 3 lines from baseline. This study formed the basis for a paradigm shift from full time daily patching routines to part time occlusion.13

The amblyopia treatment study (ATS 2B), compared 2 hours versus 6 hours of daily patching for moderate amblyopia in children aged 3 to 7 years old. In this prospective study 189 patients were enrolled with a mean visual acuity of 20/63 and randomized to 2 or 6 hours of patching. Visual acuity improved in both groups and at 4 months there was no difference between the 2 groups. 62% of patients in each group had a visual acuity 20/30 or better and or improvement of 3 lines. It was noted that prescribing greater hours of patching did not seem to have a significant beneficial effect in the first 4 months of treatment. It was also noted that the hours of patching did not affect the rate of improvement 14-17.

**Recurrence of amblyopia after discontinuation of treatment**

The amblyopia treatment study 2C was undertaken to study the recurrence of amblyopia after discontinuation of treatment. In this prospective trial, 156 children less than 8 years of age, who received continuous treatment for amblyopia for the previous 3 months (prescribed at least 2 hours of daily patching or at least 1 drop of atropine per week) and who had improved at least 3 log MAR levels of treatment. Follow up was performed at 52 weeks to assess the recurrence of amblyopia defined as 2 or more log MAR line reduction of visual acuity from enrolment, confirmed by a second examination or restarting of treatment due to a 2 or more log MAR level reduction of visual acuity. Approximately one fourth of the children were noted to have a recurrence of amblyopia in the first year post treatment. This is similar in the patching and atropine group. In patients with intense patching (6-8 hours per day), recurrence was more common when the treatment was not reduced prior to cessation, than when treatment was reduced to 2 hours per day prior to cessation 18.

**Older children**

The amblyopia treatment study 3, ATS 3, evaluated the effectiveness of treatment in children aged 7 to 17 years. In this prospective randomized clinical trial, 507 patients were studied with a visual acuity ranging from 20/40 to 20/400. The optimal optical correction was given followed by randomization to treatment group (2-6 hours per day of prescribed patching combined with near visual activities for all patients plus atropine sulphate for children aged 7 to 12 years) or optical correction alone. With treatment 53% responded and with optical correction 25% responded. For patients aged 7 to 12 years, 2 to 6 hours of patching with near visual activities and atropine improves the visual acuity even if the amblyopia has been previously treated. For patients aged 13 to 17 years, 2 to 6 hours of patching per day with near visual activities may improve when amblyopia has not been previously treated, but is of little benefit if amblyopia was previously treated with patching 19.

**Optical correction**

A study was conducted to evaluate the results of 2 hours of daily patching for amblyopia in children aged 3 to 7 years old. There were 2 phases (1) spectacle phase in which maximum improvement with spectacles was noted and the (2) randomized trial comparing a group using patching treatment and spectacles with a control group using spectacle correction alone. In the spectacle phase 84 children with anisometropia were enrolled and in the randomized trial there were 180 children. In the randomized trial group, children were assigned to either 2 hours of daily patching with 1 hour of near visual activities or spectacles alone. 96% patients were seen after 5 weeks. With optical correction alone amblyopia improved in 77% of the patients and resolved in 27%. In the randomized group, visual acuity improvement from baseline to best measured visual acuity at any visit averaged 2.2 lines in the patching group and 1.3 lines in the control group. Following treatment with spectacles, 2 hours of daily patching combined with 1 hour of near visual activities modestly improves moderate to severe amblyopia in children 3 to 7 years old 20-21.

In another study on the treatment of bilateral refractive amblyopia in children 3 to less than 10 years of age,
113 children with previously untreated bilateral refractive amblyopia were treated with optimal spectacle correction. Bilateral refractive amblyopia was defined as 20/40 to 20/400 best corrected binocular visual acuity in the presence of 4 diopters or more hypermetropia by spherical equivalent, 2 diopters or more of astigmatism or both in each eye. There was improvement in visual acuity by 3.4 lines in the group with visual acuity 20/40 to 20/80 and 6.3 lines in the group 20/100 to 20/320. The probability of a binocular visual acuity of 20/25 or better was 74 % at 52 weeks. The conclusion of this study was treatment of bilateral refractive amblyopia with spectacle correction improves binocular visual acuity in children 3 to less than 10 years of age 22.

**Role of near activities**

A study was designed to determine whether children randomized to near or non-near activities would perform prescribed activities and to obtain a preliminary estimate of the effect of near versus non near activities on amblyopic eye visual acuity when combined with 2 hours of daily patching. In this study 64 children in the age group of 3 to < 7 years were randomly assigned to receive either 2 hours of daily patching with near activities or 2 hours of daily patching without near activities. Parents completed daily calendars for 4 weeks. After 4 weeks, there was greater improvement in the amblyopic eye visual acuity in those assigned to near visual activities. Children patched and instructed to perform near activities for amblyopia spent more time performing those near activities compared to children not instructed. Performing near activities while patching may be beneficial in treating amblyopia 23.

**Dose response relationship**

In this study the dose-response relationship for amblyopia therapy was studied. There were 3 distinct phases – baseline: to measure visual status, refractive adaptation: an 18 week period of spectacle wear with 6 weekly measurements of logMAR visual acuity, occlusion: participants prescribed 6 hours of patching per day. Patching was monitored using a dose response monitor attached to the patch. The average concordance with patching was 48 %. Increasing the dose rate beyond 2 hours per day hastened the response but did not improve outcome. More than 80 % of the improvement occurred within 6 weeks. Treatment outcome was significantly better for children < 4 years of age than those older than 6 years 24.

The PEDIG studies suggested radical changes in the management protocols for amblyopia. It is interesting that another study was conducted to see the effect of the randomized trial of patching regimens for treatment of moderate amblyopia on pediatric ophthalmologists. Two questionnaires were mailed one in 2003 and one in 2006 to 560 members of the American Association for Pediatric Ophthalmology and Strabismus. Of the 107 responses (20 %) received, it was noted that 55 % of the respondents had decreased their prescribed patching regimens as compared to 28 % in 2003. There was no significant increase in the prescription of near visual activities or only a 2 hour patching regimen 25.

**Conclusions**

These studies conducted by PEDIG put forth new ideas and concepts in the management of amblyopia based on prospective trials. The conclusions of the various studies have been summarized below.

- Patching works faster than atropine, however at 6 months the improvement is the same with patching and atropine in moderate amblyopes.
- Improvement with full time and 6 hours patching similar in severe amblyopes.
- In moderate amblyopes 2 hours patching gives similar results to 6 hours of patching.
- About 25 % of the children have a recurrence of amblyopia in the first year post treatment.
- Recurrence is less common when patching was tapered to 2 hours per day before stopping.
- In patients 7 to 12 years of age, visual acuity improves with treatment even if amblyopia has been previously treated; in the 13 to 17 year age group, there may be little benefit if the amblyopia has been previously treated.
- Refractive correction improves visual acuity in untreated anisometropic amblyopia
- Most cases of moderate amblyopia (20/40 to 20/100) resolve.
- Performing near activities while patching may be beneficial in treating amblyopia.
Bilateral refractive amblyopia can be treated with spectacle correction with remarkable improvement of visual acuity.

Maximum improvement with therapy occurs in the first 6 weeks.

Younger the child better the outcome.

References

Is UBM Useful For Zonular Integrity?

Dr. Meenakshi Dhar MS, Dr. Abhijeet S. Khake, Dr. H. Sujithra DO, Dr. Niranjan Pehere

Abstract

We conducted a study of ultrasound biomicroscopy (UBM) for 50 patients with mature senile cataract, pseudoexfoliation, trauma, or those with clinical suspicion of zonular dehiscence (n=5). 10 had clinical zonular dialysis with phacodonesis / iridodonesis which was confirmed by UBM. Three patients were noted to have zonular dehiscence on UBM. Areas of zonular dehiscence were mapped on UBM and the UBM findings are described. Surgery could be planned accordingly and complications prevented. Sensitivity for preoperative detection of zonular loss is high with UBM. Surgical surprises can thus be avoided and better visual outcomes ensured.

Key words Zonular dehiscence, subluxation, ultrasound biomicroscopy

Introduction

One of the challenging situations in cataract scenario with an unpredictable outcome despite advances in cataract surgery, even in the hands of well trained phacosurgeons is zonular dehiscence. Such situations can be more enigmatic especially in hard cataracts as these pupils usually fail to dilate.

Ultrasound biomicroscopy (UBM) has come as a boon for visualizing structures in the angle of anterior chamber and the ciliary body area. The location of the ciliary processes and the integrity of the zonules can now be assessed, thus enabling preoperative detection of subtle degrees of subluxation. Occult zonular defects detected preoperatively, can be managed more effectively. Preventive measures can be taken to ensure maximum bag stability. These include, avoiding pressure or massage after the peribulbar block, giving I/V Mannitol to decrease vitreous up-thrust. Extra care needs to be taken, and only low fluid phaco maneuvers are carried out, and abrupt shallowing or deepening of the anterior chamber are to be avoided.

With UBM one gets images of cross sections of the intact anterior globe at microscopic resolution. It uses a high frequency ultrasound transducers (50 MHz), with a resolution of 20 μm, and structures upto a depth of 4 mm can be seen.

Occult zonular defects identified preoperatively enabled modification of surgical technique to ensure improved outcomes.

Materials and Methods

We selected patients coming for cataract evaluation and management to the Out Patient Clinic of the Ophthalmology Department at Amrita Institute of Medical Sciences between Oct. 2006 and Oct. 2007. Consecutive cataract patients with mature cataract or pseudoexfoliation syndrome or those with history of trauma were included in the study.
All patients underwent a comprehensive ophthalmic evaluation including vision assessment, refraction, slit lamp biomicroscopy, fundus examination (unless the cataract was mature) keratometry, and biometry for IOL calculation.

In addition, a UBM was done for these patients in an undilated pupil using the UBM machine from Appasamy with the 35 MHz and 50 MHz probe, a scan angle of 10-30 degrees. The probe was aligned radially along the 12 clock hours placed perpendicularly over the cornea so as to include the angle of anterior chamber, iris, the ciliary body, zonules and peripheral lens edge.

Scanning was performed under topical anesthesia using a 20 mm eye cup which was inserted between the lids and filled with saline/Ringer solution, with the patient in a supine position in standardized room lighting conditions.

Initial scan was done over the central anterior chamber to observe for any tilt of the bright reflective line of the anterior lens capsule. Scanning was then done over the iris root for 360° to assess zonules directly.

Radial scanning was performed with probe oscillations parallel to the zonules & with the focal plane of the transducer at the depth of the zonules.

To prevent a false impression of zonular defect, the long axis of the transducer was kept perpendicular to the zonular fibres, helped by asking the patient to position the eye accordingly.

In view of the familiarity with discussing the anterior segment in terms of clock hours, we recorded zonular defects in terms of clock hours as well. Beginning radial scanning at 12 o’clock position, we carried it out in clockwise fashion till 6 o’clock position with the probe marker toward the limbal side.

Then the marker was located toward the corneal side from 6 to 12 o’clock position for each eye.

All these patients subsequently underwent cataract surgery and the UBM findings were correlated with the intraoperative observations.

In those with zonular dehiscence detected clinically or with UBM a capsular tension ring was inserted during the procedure. The visual and surgical outcomes were analysed.

Observations

Of the 50 patients undergoing UBM, zonular dehiscence was seen in 13 patients. Positioning of the probe was very important and it had a long learning curve with fibre orientation being perpendicular to the probe. The oscillations of the UBM scan had strict radial orientation towards the limbus.

Seven patients had clinical zonular dialysis in one eye, and the other eye was normal.

Area of zonular absence as seen by UBM was less than/ = 1 quadrant in 5 cases, 3 at 1 site only & 2 at two sites. The other 2 cases had almost 180° dehiscence.

Table 1: Clinical details

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<thead>
<tr>
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<tbody>
<tr>
<td>Coloboma</td>
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<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Trauma</td>
<td>4</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Pseudoexfoliation</td>
<td>19</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>MSC/ HMSC [Fig.1]</td>
<td>33</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Homocystinuria [fig2]</td>
<td>1</td>
<td>1</td>
<td>1</td>
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<tr>
<td>Marfan’s Syndrome[Fig.3]</td>
<td>1</td>
<td>1</td>
<td>1</td>
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</table>

3 patients had anterior coloboma in both the eyes with both iris and lenticular coloboma. There was absence of zonules on UBM in the colobomatous areas, and the remaining zonules were found to be firm and normal. Extent of zonular absence was noted in clock hours for each of the eyes. In all colobomas, the zonular absence was in an area larger than the iris coloboma of the same eye. The maximum extent of coloboma was 270° (n=1) and minimum<90°(n=2). Ultrasound biomicroscopy of the lens “Coloboma” revealed a greatly increased sphericity of the lens and the deficiency of zonules in the “colobomatous” area.

Capsular tension ring was used effectively in these patients increasing capsular bag stability during both phacoemulsification and IOL placement. The CTR was inserted by hand over hand method.

One patient gave a history of unilateral blunt trauma with history of a coconut falling on her a few years ago. She was found to have subluxation clinically with iridodonesis, shallow anterior chamber superiorly and a pupil that did not dilate. This was
confirmed on UBM. Zonules were missing for 180°, the ciliary body was flattened and increased lenticular sphericity was noted.

Of the 19 patients with pseudoexfoliation [PXE], two had iridodonesis, of which one had phacodonesis as well. On UBM, 4 patients with PXE had zonular dehiscence evidenced by absence of zonules and localized increase in lenticular sphericity.

Occult zonular loss was detected in 3 patients on UBM. In 2 others with mature/hypermature cataract with iridodonesis the zonular dialysis was confirmed with UBM. There were either missing zonules [n=2], or/and zonules were stretched [n=2] (fig. 3 and 4), and/or ciliary body flattening [n=2] was observed. The lenticular sphericity was increased in the area of zonular defects [n=3] (fig. 1). Other effects seen were pupillary block, angle crowding and direct iridal irritation. Angle of anterior chamber was increased [Fig. 5] in a patient with posterior subluxation.

Fig. 1. Clumping of ciliary body processes and zonules in the right half of the field, increase in anteroposterior diameter of the lens -Indication of subtle subluxation in a mature cataract

Fig. 2. UBM of a homocystenuria patient with B/L subluxation, managed conservatively with once daily instillation of Pilocarpine. The UBM shows deepening of AC with widening of the angle

Fig 3. Increase in anterior chamber depth, stretched zonules and lens flattened in patient of Marfan’s syndrome with subluxation on UBM

Fig 4. UBM of a patient with stretched zonules

Fig 5. Large angle of anterior chamber 51deg.on UBM in a patient with post subluxation
One patient with homocystinuria, had bilateral high myopia with subluxation and mildly raised IOP. It was managed conservatively. Patient had best corrected visual acuity (BCVA) of 6/9 in both eyes. She was on Pilocarpine 2 % twice daily as the zonular dialysis was >180 degrees in both the eyes. The extent of zonular defect found on UBM was higher than that seen clinically in a dilated pupil. The anterior chamber on UBM was found deep with iris falling backward with no support. No zonules or lens was seen in this area with flattening of ciliary body [Fig.4].

Discussion

Patients more prone to zonular dehiscence and subluxation are, those with pseudoexfoliation and hypermature cataracts in the senile age group. Also an unsuspecting, mild blunt trauma, might cause zonular dehiscence, an event the patient may have well forgotten about. The UBM can today come to the rescue for the unsuspecting surgeon, and help in the diagnosis of zonular defects. The surgeon needs to have a high degree of suspicion. Certain periorperative maneuvers can enhance the degree of subluxation, increasing the risk of dreaded complications like nucleus drop or vitreous prolapse. The safe placement of the PCIOL was impossible in these cases, until the capsular tension ring started being used for subluxated lenses almost a decade ago.

Certain steps taken during surgery can ensure a favourable outcome. These include gentle pressure if peribular is given, Preferably giving I/V Mannitol to minimize vitreous upthurst, and gentle steady manipulation of globe. The incision is to be made away from site of zonular defect and if possible opposite to it. The initiation of rhexis is difficult in subluxated lenses and this step may be an important subtle indicator of an undetected zonular weakness. The rhexis should be initiated in an area of intact zonules so that counter traction is avoided. It should be small and central, with slow careful hydrodissection at multiple sites. The use of capsular tension rings has of course revolutionized the outcome, combined with the availability of dispersive and cohesive viscoelastics. At all steps sudden shallowing of the anterior chamber as well as deepening are to be avoided. Phaco chop is the preferred method with adequate power and not a high flow rate.

One unique feature was the deposition of granular material on the zonules in PXE. This has been noted earlier, and depending on the extent has been classified into mild, moderate and severe cases. There are diagnostic criteria proposed by the Japanese for the early detection of PXE, based on the changes found in the zonules by UBM.

The preoperative diagnosis of the zonular defect can help in judging the appropriate timing of intervention. In the presence of large zonular defect, it is better not to wait till cataract is more significant in a patient with extensive zonular damage.

Conclusion

Ultrasound biomicroscopy is a sensitive and accurate method of assessing zonular dehiscence, although there is a long learning curve in mastering the technique. The orientation of the probe is important, otherwise false negative results may be high, i.e. the zonular defect may be missed. UBM can image zonules in patients suspected to have zonular dehiscence, allowing a prepared approach to careful surgery for these patients. The CTR insertion ensures good stability both for the procedure of the cataract surgery as well as for the long term placement of the intraocular lens in the bag.

Bibliography

Clinical Study of Fungal Corneal Ulcer

Dr. K.V. Raju MS, Dr. M.S. Vijayalakshmi MS, Dr. Lakshmi J.

Abstract

Aim: To study the epidemiology of fungal corneal ulcers in relation to age, sex, mode of injury, clinical presentation, etiology and the response to treatment with topical and systemic antifungal medications.

Procedure: 30 patients who were found positive for KOH and / or fungal culture during a period of one year attending the Ophthalmology outpatients department, Medical College, Kozhikode were studied.

Introduction

Mycotic keratitis is an important ophthalmologic problem causing preventable visual disability. During the last 4 decades there have been reports from different parts of the world about the increasing incidence of this entity. Depending on the characteristics of the population and the geographic areas, there is variation in the distribution of the causative organisms.

Some of the factors that have been held responsible for this increasing incidence of fungal keratitis include the wide spread use of broad spectrum antibiotics and steroids, the frequent and sometimes prolonged use of contact lenses and the growing number of corneal surgeries being performed. Steroid is a double-edged weapon that controls the inflammation but increases the susceptibility of the individual to the microorganisms.

In countries like India with primarily Agrarian population, trauma is very common. In addition, favorable tropical environment and unhygienic practices like putting herbal medicines in the eyes make mycotic keratitis very common. Mycotic keratitis is a serious ocular infection, which requires urgent diagnosis and appropriate treatment. Blindness due to mycotic keratitis can be prevented by early intervention. Heightened awareness of this problem among the ophthalmologists and medical microbiologists has contributed to the increasing recognition of the disease.

The aim of this study was to find out the epidemiology, clinical presentations and outcome of treatment for mycotic keratitis in a large teaching hospital subserving the northern districts of this state (Kerala).

Inclusion Criteria

All the patients with corneal ulcers attending the Ophthalmology out patient department of Kozhikode, Medical College, during the period of the study were subjected to KOH stain and fungal cultures of their corneal scraping. Those patients who were found to be KOH positive or fungal culture positive or both were taken up for the present study.

Exclusion Criteria

Ulcers with picture of viral origin, ulcers from which scrapings cannot be taken due to perforation and uncooperative children were excluded from the study.
Materials and Methods

A detailed history regarding the disease with special reference to the mode of injury, patient’s age, sex and occupation were noted. Any history of ocular or systemic illness was noted. All patients were subjected to detailed clinical examination. The scrapings were sent for KOH mount to detect fungal filaments. Fungal culture was done in Saboraud’s dextrose agar medium and bacterial culture in blood agar. Subcultures were done in necessary cases with the assistance from the Department of Microbiology. After the material was sent to microbiology, patients were put on broad spectrum antibiotics and if there was a history suggestive of fungal infection Natamycin was added. If the KOH mount was positive for fungus, patients were put on topical Natamycin 5 % at two hourly interval. If the patients were found to be positive for culture, Tab.Ketoconazole 200 mg twice daily was started for 2-3 weeks. The response to treatment was assessed daily by slit lamp examination till the ulcer started to heal and the patients showed symptomatic improvement. The patients were then followed up weekly for a period of two months and in some cases for extended periods.

Results and Discussions

Out of the 100 cases, the incidence of mycotic keratitis was 30 % in this study.

Age Distribution

The agewise distribution of keratitis showed that the incidence is highest in 31-40 years age group (30 %) which closely followed by 41-50 years age group. This could be due to the fact that they are physically active and working out doors and prone to injury. Our study tallies with the peak age group detected by Choudhary et al (1999-2001) (37 %).

Sex incidence

The present study showed a male preponderance with 23 males (77 %) and 7(23 %) females. This could be due to their outdoor work and increased chance of ocular trauma.

The study by Bharathi et al showed male preponderance of 65.05 % and Choudhary et al it was 68 %.

Incidence in relation to occupation

In the present study agricultural workers showed the highest incidence (40 %) followed by manual labourers (33 %). In the study by Bharathi et al 64.75 % fungal corneal ulcer was seen in farmers.

Incidence of predisposing factors

Of the 30 cases 22 patients (72.6 %) had history of trauma. In the study by Srinivasan M et al in 1997 trauma as a predisposing factor was seen in 65.4 %.

Ocular trauma, most commonly was with leaf and thorn 8 (26.7 %) followed by injury with stick 4 cases (13.3 %), with coconut shell 1 case (3.3 %). 43.3 % cases injury was with vegetable matter. This tallies with the 52.8 % reported by Srinivasan M et al.

Systemic predisposing factors were present in 42.9 %. Among this the commonest was diabetes mellitus 5(16.7 %) 3 patients were anaemic (10 %) 3 were alcoholic (10 %). Tuberculosis and leprosy 1 case each. Ocular predisposing factors were present in 16.7 % in this study.

Incidence of previous treatment

Of the 30 cases, only 8 had history of previous treatment. Twenty-two patients had no previous treatment. 16.7 % were on antibiotics 6.7 % were on native medicines and one patient was on steroids. In the study by Choudhary et al topical corticosteroids had been prescribed to 21 % of patients at the onset of symptoms.

Clinical features

Of the 30 cases, right eye was involved in 19 cases and LE in 11 cases. There was not a single bilateral case.

Almost all the ulcers had textbook description of dull, gray-raised surface. Some had a viral like appearance to start with. In the study by Bharathi et al 75.43 % had dry thick and raised corneal surfaces. 20 % of patients presented with an ulcer size of more than 5 mm. 70 % had a size of 2-5 mm and 10 % of less than 2 mm. Majority of patients had an ulcer depth of ¼ - ½ (43.3 %) 9 patients had hypopyon (29.7 %). In our study most of the hypopyon were 2 mm or less.
at the time of presentation. Satellite lesions were seen in 10 %. Dendritic pattern was seen in 3.3 %. Corneal abscess was seen in 1 case (3.3 %). In one case there was corneal perforation (3.3 %).

**Fungal incidence**

Of the 30 cases studied the most common fungi isolated was fusarium species in 13 cases (42.9 %). This was followed by Aspergillus species 6 cases (19.8 %). (table 1). This is comparable to the study by Sreenivasan M et al (Fusarium 47.1 % and Aspergillus 16.1 %). In this study 10 % KOH mount was positive in 27 cases (90 %).

<table>
<thead>
<tr>
<th>Organisms</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Fusarium</td>
<td>42.9</td>
</tr>
<tr>
<td>Aspergillus</td>
<td>19.8</td>
</tr>
<tr>
<td>Curvularia</td>
<td>6.66</td>
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<tr>
<td>Pencillium</td>
<td>3.33</td>
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</table>

**Treatment response**

Natamycin and ketoconazole were the drugs used in this study. Small superficial ulcers showed good response. Only one case of fungal corneal ulcer perforated. This case had corneal abscess, hypopyon and perforated and had previous treatment with steroids.

**Conclusion**

Prevalence was found to be more common in male rural dwelling agriculturists. Fusarium is the commonest species identified. 10 % KOH mount is a very sensitive, simple and rapid test in detecting fungal filaments. 5 % Natamycin is the drug of choice for most of the filamentous fungi. Small superficial ulcers, early diagnosis and appropriate treatment showed faster healing.

**References**

5. Gopinathan V et al. The epidemiological feature and laboratory results of fungal keratitis, a 10yr review at a referral eye care centre in S. India, Cornea, 2002; 21: 555-559.
Primary Vitrectomy for Pseudophakic Rhegmatogenous Retinal Detachment Uncomplicated by Proliferative Vitreoretinopathy (PVR)

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

Introduction

The primary aim of a retinal reattachment procedure is to close all responsible retinal breaks and relieve any vitreoretinal traction. This can be achieved by an external scleral buckle, placed after localization and retinopexy of the break, to mechanically close it. The same effect can also be achieved by pars plana vitrectomy.

Since its introduction in 1970 by Dr. Robert Machemer, pars plana vitrectomy has played an important role in the management of complicated retinal detachments. To date several indications have been identified:

1. presence of various grades of proliferative vitreoretinopathy (PVR)
2. Giant retinal tear
3. complex arrangement of retinal breaks such as large posterior tears
4. macular holes
5. multiple breaks at different levels
6. presence of media opacities

However the precise role of vitrectomy in the management of uncomplicated rhegmatogenous retinal detachment is still controversial.

Pseudophakic Retinal Detachments \(^1,^2\) have presented unique and difficult problems to vitreoretinal surgeons since it was initially described by Tasman and Annesely in 1960. The clinical features of retinal detachment after cataract surgery \(^3,^4\) are different from those in phakic eyes: (1) the responsible tears are small located anteriorly at the insertion of vitreous base. (2) the detachments are more extensive and macular involvement is very common. (3) signs of PVR are common. (4) the incidence of ‘no break found’ is more common as there is difficulty in visualizing the ora due to the glare and reflexes produced by the IOL, the presence of posterior capsular opacification as well as poor pupillary dilatation. Significant predispositions include intraoperative posterior capsular rent with vitreous disturbances, Yag laser capsulotomy, myopia with axial length > 25 mm, h/o retinal detachment in the fellow eye or a family history of retinal detachment \(^5,^6,^7,^8,^9\).

The basic surgical principles of scleral buckling and vitrectomy applies to pseudophakic retinal detachment (RD) also. However they present certain unique difficulties during surgery. The greatest problems \(^10,^11\) are

1. Difficult visualization of periphery and hence a higher incidence of “no break found” necessitating larger buckle placement, more extensive cryopexy both of which stimulates a greater degree of post operative inflammation and PVR formation.
The location of the IOL presents problem during scleral buckling and vitrectomy. Anterior movement of the anterior chamber intraocular lens (caused by hypotony) during subretinal fluid drainage or during fluid-air exchange can damage the cornea or press against the angle causing hyphema intraoperatively.

Presence of PC rent causes problems for visualization after fluid-air exchange due to moisture condensation on the IOL intraoperatively. So also silicone IOL-silicone oil interaction leaves behind a firmly adherent layer of silicone oil, impairing intraoperative visualization as well as the postoperative visual recovery.

Previous reports on the management of retinal detachment has shown a poor rate of anatomic success in the presence of trans-sclerally sutured posterior chamber intraocular lens and have speculated the existence of certain risk factors which were associated with poor prognosis for surgical repair. These includes presence of vitreous in front of the intraocular lens, poor pupillary dilatation, difficulty in visualization during fluid-air exchange due to air gushing into anterior chamber and persistent tractional forces at the vitreous base due to the haptic of the IOL or the fixation sutures.

In this paper we analyze the results of primary vitrectomy in 50 eyes with uncomplicated pseudophakic rhegmatogenous retinal detachment.

Materials and Methods

We performed a prospective study on the efficacy of primary pars plana vitrectomy in the management of uncomplicated pseudophakic rhegmatogenous retinal detachment in 50 consecutive patients attending our tertiary care referral centre between 2004 and 2007.

The patient’s age ranged from 7 yrs – 64 yrs, the M: F ratio was 2:1. All patients had a detachment of less than a month due to superior breaks and the macula was off in all the cases. There was no evidence of PVR. All patients were pseudophakic and included 36 eyes with posterior chamber intraocular lens implants, 11 eyes with anterior chamber intraocular lens and 3 eyes with transclerally sutured posterior chamber intraocular lens implants.

The surgical procedure included a conventional pars plana vitrectomy. Any vitreous traction present around the retinal tear was relieved. Indirect ophthalmoscopy was then performed to rule out any other preexisting or iatrogenic retinal tears. Fluid-air exchange with simultaneous endodrainage was performed either through the retinal tear if it was posterior or through a drainage retinotomy. Once pneumohydraulic reattachment was achieved, retinopexy was performed either by trans-scleral cryopexy or LIO laser barrage. Long acting tamponade was achieved by a non-expansile mixture of air and C₃F₈ gas. The patients were advised to maintain a face down position for at least 12 hours – 16 hours daily for 3 weeks postoperatively. The duration of hospital stay was 3 days. The patients were monitored for any postoperative reaction and rise of intraocular pressure.

Follow up examination was performed every month for the first 3 months and then at 2 monthly intervals for the next 6 months. The duration of follow up varied from 6 months-48 months.

Results

The patients were of the age group ranging from 7 years to 54 years (Mean-37 years). The male:female ratio was 2:1. All patients had a detachment of less than a month due to superior breaks and the macula was off in all cases. The preoperative fundus findings were tabulated in Table 1.

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<tbody>
<tr>
<td>PCIOL: 36, AC IOL: 11, SF IOL:3</td>
<td>15 eyes (30 %)</td>
<td>10 eyes (20 %)</td>
<td>12 eyes (24 %)</td>
<td>4 eyes (8 %)</td>
<td>9 eyes (18 %)</td>
<td>6 eyes (12 %)</td>
<td>3 eyes (6 %)</td>
<td>50 eyes (100 %)</td>
<td>9 eyes (18 %)</td>
<td>9 eyes (18 %)</td>
<td>5 eyes (10 %)</td>
<td>14 eyes (28 %)</td>
<td>5 eyes (10 %)</td>
<td>3 eyes (6 %)</td>
<td></td>
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</table>
The study population included 36 eyes with PC IOL implants (72%); 11 eyes with AC IOL implants (22%); and 3 eyes with trans-sclerally sutured PC IOL (6%) implants. Preoperative hypotony (30%); significant anterior chamber reaction (20%); vitreous in anterior chamber (6%); poor pupillary dilatation (24%); malpositioned IOLs (8%), PCO (18%) and presence of vitreous haze (10%) made intraoperative visualization difficult. Preoperative topical and systemic steroid administration to control the inflammation and combat hypotony was successful in achieving a quiet eye in 20% cases. Use of flexible iris retractors, sphincterotomies, posterior capsulotomy and repositioning of the IOL into the bag or sulcus was performed intraoperatively to achieve adequate pupillary space for visualization of fundus periphery and to perform a thorough base excision. 18% of patients had choroidal detachments associated with hypotony and total retinal detachment. Choroidal detachments were drained before placement of infusion canula. A break responsible for the detachments could not be identified in 18% of patients either pre or intraoperatively.

Table 2. Intraoperative problems and complications

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Problem</th>
<th>Incidence (%)</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Corneal Odema</td>
<td>12 eyes (24%)</td>
<td>Debridement</td>
</tr>
<tr>
<td>2</td>
<td>Intraoperative hyphema</td>
<td>2 eyes (4%)</td>
<td>Viscoelastic inj into AC</td>
</tr>
<tr>
<td>3</td>
<td>Pupillary Miosis</td>
<td>14 eyes (28%)</td>
<td>Iris Retractors, adding Adrenalin to infusion fluid</td>
</tr>
<tr>
<td>4</td>
<td>Displacement of IOL</td>
<td>2 eyes (4%)</td>
<td>Conservative (1); Repositioning (1)</td>
</tr>
<tr>
<td>5</td>
<td>Shallowing of AC</td>
<td>4 eyes (8%)</td>
<td>Reformed spontaneously with prone positioning Capsulotomy and drainage</td>
</tr>
<tr>
<td>6</td>
<td>Blood in Capsular bag</td>
<td>1 eye (2%)</td>
<td>Clear with flute needle</td>
</tr>
<tr>
<td>7</td>
<td>Air in AC during fluid air exchange.</td>
<td>3 eyes (6%)</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Moisture condensation on IOL</td>
<td>6 eyes (12%)</td>
<td>Scleral depression assisted base excision</td>
</tr>
<tr>
<td>9</td>
<td>Difficulty in base excision</td>
<td>4 eyes (8%)</td>
<td></td>
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</tbody>
</table>

Development of corneal odema intraoperatively and pupillary miosis were the two major intraoperative difficulties experienced during surgery. Epithelial debridement was necessary in 24% of eyes to clear the visual axis for adequate visualization during surgery. Pupillary miosis intraoperatively was countered using iris retractors or by adding adrenalin to the infusion fluid in 28% of cases. Intraoperative hyphema was caused by fluctuations in the intraocular pressure intraoperatively in 4%. Injection of viscoelastic into the anterior chamber cleared the pupillary area. Shallowing of the anterior chamber after fluid-air exchange was observed in 8% of eyes. In these 4 patients, the chamber reformed spontaneously when prone position was maintained postoperatively. Minimal displacement of the PC IOL which occurred intraoperatively in one patient was managed conservatively. Collection of blood within the capsular bag intraoperatively impaired visualization in 2% of eyes necessitating central capsulectomy for drainage. Bubbling of air into the anterior chamber during fluid-air exchange occurred in all the 3 patients with sclerally sutured PC IOLs impairing visualization. Intraoperative moisture condensation in 12% was cleared by flute needle. Moisture condensation on the posterior surface of the IOL in 6 eyes (12%) with pre-existing posterior capsular rent was managed by gently sweeping the posterior surface of the IOL with a soft tipped aspiration needle.

The patients were followed up for 12 months. The anatomic reattachment rate was 96% with a single procedure. In 2 eyes (4%) recurrent retinal detachment with advanced proliferative vitreoretinopathy and gross hypotony necessitated retvitrectomy with silicone oil tamponade. 80% of the patients achieved a visual acuity > 6/12 to 6/60. Poor visual recovery in 10 eyes (20%) was attributed to (1) Epiretinal membrane formation 2 eyes (4%); (2) Macular hole 2% (4) persistent cystoid macular edema 4% (5) persistence of SRF in macular area detected by OCT in 10%.

We analyzed factors which predicted poor visual recovery and redetachment. These included preoperative anterior chamber reaction, choroidal detachment, older patients and longer duration of detachment. There was no significant difference in the reattachment rates with the different IOL types.
Discussion

The basic surgical principles of scleral buckling and pars plana vitrectomy continue to apply to pseudophakic retinal detachment, but there are certain problems unique to pseudophakia. The greatest problem in repairing a pseudophakic retinal detachment is the difficulty in visualizing the peripheral retina. Retinal breaks cannot be found in as many as 20% of patients due to small miotic pupils, difficulty in viewing through edge of IOL, perilenticular membranes and posterior capsular opacification.

Visualization tends to be more difficult in iris fixated and AC IOLs. Because of uncertain visualization, there may be a tendency towards greater use of cryotherapy which is associated with increased inflammation and postoperative PVR. The location of the IOL, whether in anterior chamber, iris plane or in the posterior chamber presents problems during scleral buckling and vitrectomy.

1. An anterior chamber lens, during scleral depression may be forced against the angle causing bleeding.

2. Mobility of iris fixated lenses can cause corneal damage from anterior displacement at time of subretinal fluid drainage when the eye is hypotonous.

3. An intravitreal gas bubble will also displace an iris fixated lens anteriorly. Prior injection of air/healon into the anterior chamber will prevent this.

4. Postoperative pupillary block and choroidal detachment also causes anterior displacement of IOL against cornea requiring immediate repositioning to prevent irreparable corneal damage.

5. Dislocation of lens into vitreous can occur with posterior chamber intraocular lenses. Posterior chamber intraocular lenses pose the fewest difficulties during scleral buckling and vitreous surgery.

6. Intraoperative moisture condensation on the posterior surface of IOL during fluid-air exchange may impair visualization of posterior segment in eyes with preexisting posterior capsular rents.

7. Interaction between silicone oil and silicone foldable IOL in the presence of PC rent causes impaired visualization for surgery.

8. In our series of 3 cases of pseudophakic retinal detachment associated with trans-sclerally sutured posterior IOLS, the clinical features and problems faced during surgery did not differ from eyes with conventional posterior chamber intraocular lenses. All patients had well dilated pupils. Absence of posterior capsular opacification and perilenticular cocoon membranes made visualization of retinal periphery easy. Intraoperative problems such as IOL dislocation, significant IOL decentration, vitreous haemorrhage, hyphema and pseudophakic corneal touch were not experienced while managing these patients. The only intraoperative problem was difficulty in visualization during fluid-air exchange as air gushed into the anterior chamber. Similar problems are experienced in eyes with anterior chamber IOLs or posterior chamber IOLs with posterior capsular rent or following YAG capsulotomy.

IOL explantation for intraoperative decentration or dislocation of the IOL or to permit visualization for vitrectomy was not necessary in any patient. Adequate anterior vitreous base dissection was possible in all the eyes which permitted intraoperative retinal flattening.

An anatomic success rate of 93.0% (88.8-93%) for pseudophakic retinal detachment cases have been reported by Schepens in 1991. The PC IOL group has a significantly greater prevalence of good postoperative visual acuity compared to AC IOL groups which have the worst visual prognosis. The cause of poor visual recovery after retinal detachment repair in patients with AC IOL is attributed to the increased occurrence of postoperative corneal oedema. Patients having an AC IOL implant have a higher degree of occurrence of breakdown of BAB (blood-aqueous barrier) before treatment of retinal detachment. Therefore the persistent corneal dysfunction in the presence of an AC IOL may trigger the postoperative corneal oedema and explain the poorest visual acuity outcomes.

The statistically significant poor prognostic indicators for reattachment are.
1. Presence of AC IOL (2) Eyes with AC reaction (3)
Presence of macula off RD (4) Preoperative PVR (5)
Older age etc

The reasons for failure include the development of proliferative vitreoretinopathy, failure to close an existing break or development of a new break. Although the anatomic reattachment rate is comparable to that in phakic eyes, the visual results are not so good. The postoperative visual recovery is worst with AC lenses due to the presence of corneal decompensation and higher incidence of cystoid macular edema. The other causes of poor visual recovery after successful anatomic reattachment are macular degeneration, cystoid macular edema, epimacular membrane proliferation, macular pucker and photoreceptor dysfunction.

Pars plana vitrectomy has become accepted as the treatment of choice for certain complex retinal detachments. The commonest indications are difficult breaks and PVR. In simple retinal detachments, external scleral buckling procedures are still preferred. The role of primary vitrectomy in the management of these cases is still controversial.

Scleral buckling procedures can be complicated by a variety of intraoperative and postoperative complications which can impair the final visual result. A combination of factors may result in intrusion, extrusion or infection of the scleral buckle. Mobility problems may be induced by the presence of bulky scleral buckle or due to disinsertion or rupture of the extraocular muscle during surgery. External drainage of SRF may result in subretinal haemorrhage, retinal incarceration or a retinal break. Encircling elements may reduce the blood flow as shown by colour doppler studies and results in anterior segment ischaemia. Refractive changes are a rule after scleral buckling procedure and may be especially problematic if a significant degree of anisometropia has been induced in a previously emmetropic eye. Buckling elements may result in distortion of macula and reduction of macular function. Further buckling procedures may be associated with postoperative CME which may account for delayed return of vision.

Vitrectomy offers certain advantages over scleral buckling in that it affords a direct approach to vitreous traction. Internal drainage of SRF with simultaneous FAE allows pneumohydraulic retinal reattachment. Vitreous opacities are removed at the time of vitrectomy and postoperative mobility is less. It should also be recognized that there are potentially hazardous complications of this procedure like progression of nuclear sclerotic cataract, glaucoma, iatrogenic tears and detachment and vitreous haemorrhage, etc.

Primary pars plana vitrectomy offers an alternative to scleral buckling procedures in the management of selected cases of primary rhegmatogenous retinal detachment. A larger series with longer follow up is needed before the efficacy of this procedure is established.

References

12 K.U. Bartz-Schmidt,B Kirchhof Primary Vitrectomy for pseudophakie retinal detachment Br.J. Ophthamal .1996 April :80(4); 346-349
15. D.Singh,Y Sharma,N.Pal Scleral buckling versus primary Vitrectomy Ophthalmol Vol 113(7) 1246-1247

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**Humour in Ophthalmology**

**Political Correctness (Or Euphemism?)**

Recently I happened to come across a slim volume titled ‘Politically Correct Fairy Tales’. The first fairy tale was named “The Melanotically Impoverished Person of Royal Descent and Seven Vertically Challenged People”. Lost, aren’t you? It was the story of Snow White and the Seven Dwarfs.

The word ‘political correctness’ will not be found in any dictionary printed before the nineties. But once it came into vogue, it is there with a vengeance. Nowadays anything one say may be interpreted on its political correctness.

As we are all aware there are no blind people any more. They are all ‘visually handicapped’ or still better, ‘visually challenged’. Even the word handicapped is considered improper. ‘Differently abled’ is the current correct term, it seems. Sometimes we tend to forget and blurt out the outmoded word and cause wrinkles to pop up on the foreheads of discerning listeners.

Even though it has got disadvantages for people over forty (who are used to speaking or writing in a straight forward manner), it has some advantages too. This comes in handy while explaining the (unfavourable) outcomes of your treatment, surgical or medical. The *aqueous percolation through the artificial intralamellar pathway is suboptimal*, you can say about a failed bleb. These ‘suboptimal’ or ‘deficiently optimal’ results can be there in any treatment. The more obfuscating the word/ phrase is, the better. The same is true for prognostication too. You can be fully honest, yet incomprehensible. And the patient/ by-standers will be impressed into the bargain.

Another advantage is that all of us who called ourselves as ‘General Ophthalmologists’ can now use the much more impressive sounding ‘Comprehensive Ophthalmologist’.

Some time back, in a hospital attached to a Central Government Institution, one of those pompous Central Secretaries came for a visit. The Ophthalmologist was conducting the weekly medical board when a visit was paid to the OPD. “Ah…m..m. You check the fields of all the crane operators, don’t you?” asked the Secretariat Mandarin. “Of course sir, we do digital perimetry”. The answer satisfied him, especially the ‘digital’ part. I am sure that he did not know that ‘digital’ can allude to your fingers too. Another example of political correctness saving the day!
Clinical and Virological Study of Conjunctivitis during the Epidemic in Calicut October - December 2006

Dr. Sheeja Viswanath MS, Dr. K S Chandrakanth DO DNB, Prof. (Dr). R. Vijayan MS, Prof. (Dr). Venkitachalam MS
Dr. Nirupama Balaji DO DNB, Dr. Tresa Mathew MS, Dr. Ramakrishnan MS

Abstract

9 % of blindness in India is due to corneal diseases. With effective antimicrobials, and improved nutrition infective keratitis of non-viral origin and vitamin deficiency diseases are on the line of decline whereas viral keratitis tends to become more prevalent. Our aim was to analyse the clinical and virological aspects and to evaluate the efficacy of topical Acyclovir in preventing the development of keratitis. 55 patients were selected for virus isolation using human amnion and human lung cancer cells (AV- 3 and A549). Adenovirus type 8 was isolated from 40 %. All the cases including those having keratitis responded well with topical regimen. Our study highlighted the need for early and prompt institution of topical Acyclovir in preventing the development corneal complications and subsequent blindness.

Introduction

According to WHO, corneal diseases are the 2nd most common cause of blindness in the world today 1. A recent national survey conducted by the Govt. of India (1991-2001) estimated that the corneal lesions are responsible for 9 % of all blindness in our country 2. Majority of the causes of blinding corneal pathology are avoidable or preventable or treatable. Previously the corneal blindness was predominantly due to malnutrition, bacterial and fungal ulcers, followed by trauma. In the present era with improved nutrition and due to the advent of effective antimicrobials infective keratitis of non viral origin are on the decline whereas viral keratitis has become more prevalent.

A variety of viruses can be responsible for conjunctival infection. Adenovirus is the most common and is highly contagious during the first 2 weeks of infection. It has the tendency to occur in epidemics. Ocular adenoviral infections are characterized by highly distressing local symptoms. It can occur with corneal involvement within 4-5 days after the onset of symptoms. The corneal lesions range from diffuse fine superficial punctate keratitis to epithelial defects and finally to sub epithelial nummular opacities which can last long, even for years 3. These nummular opacities can impair visual function significantly and can cause glare. Currently no specific antiviral therapy is available to shorten the course of infection or to improve distressful clinical symptom, to stop viral replication and to avoid the development of corneal opacities 3. Research is ongoing for topical agents that have anti viral activity. One drug
that holds promise in this area is cidofovir. In 1996 Gordon et al first reported the clinical efficacy and safety of cidofovir in the treatment of the patient with proven acute keratoconjunctivitis. To the best of our knowledge no study was reported regarding the efficacy of topical Acyclovir in the management of acute phase of viral conjunctivitis.

**Aim of Study**

The purpose of our study was to analyze the clinical and virological aspects and also to evaluate the efficacy of topical 3% Acyclovir eye ointment in the treatment of acute phase of viral conjunctivitis and also in the prevention of development of corneal opacities in the late phase.

**Materials and Methods**

A total of 55 patients with clinical features of acute viral conjunctivitis were studied at Malabar Eye Hospital during the Epidemic in October -December 2006.

The diagnosis was made by clinical examination and confirmed by viral culture using human aminon cells and human lung cancer cells (AV-3 and A549). For virus isolation, conjunctival swabs taken from lower fornix were sent to Ooty and the results were analyzed. Only the acute cases of suspected viral conjunctivitis during the epidemic were included in the study. Conjunctival hyperemia petechial and sub conjunctival hemorrhages, involvement of pre auricular lymphnode, coryza, the infiltration of cornea and the response to treatment were evaluated in all patients. The efficacy of treatment was studied in 2 main groups.

First group included patients coming after treatment elsewhere with antibiotic drops alone and without much relief, 2nd group coming primarily to us with typical signs and symptoms of viral conjunctivitis. The patients seen primarily by us were treated in 3 groups of 10 each. The division was based on the severity of clinical features as mild or severe. Family members of culture positive patients who developed mild symptoms of conjunctivitis with minimal congestion, chemosis, follicles and petechial hemorrhages in the upper tarsal conjunctiva with or without involvement of pre auricular lymphnode were considered as mild. The remaining 20 cases with severe form of conjunctivitis were randomly divided into 2 treatment groups of 10 each (B1 & B2). Patients in group A (mild) were treated with antibiotic drops alone; group B1 (severe) with antibiotic-Acyclovir combination and group B2 with Acyclovir steroid antibiotic combination to the involved eye (the regime is given in table number II). All the patients treated outside were considered as severe and started on Type 3 regime.

Duration of treatment was for 21 days. Patients were followed up on 3rd, 7th, 14th, 21st and 30th day if required. At each visit, follicular reaction, conjunctival congestion and corneal involvement were documented.

**Observations and Discussion**

**Fig. 1. Age Incidence**

Total no: of pts.: 55

Maximum Age Incidence was between 20-40 yrs (40%) followed by 40-60 yrs (27%). Similar observations were made by Norn M.S. who found that 59% cases were adults.

**Fig. 2. Sex Incidence**

Total no: of pts.: 55

Out of 55 patients 29 (52.72%) were males and 26 (47.28%) were females. Gunderson (1938) in his study reported that both sexes were equally affected.
Total no of pts: 55

In 33 (62 %) cases, the disease was unilateral and 22 (40 %) cases bilateral. Khuran. A.K. et al. 7 reported 35 % bilaterality in 1984.

Table 1: Clinical Features

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>No: of Patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye ache</td>
<td>55</td>
<td>100 %</td>
</tr>
<tr>
<td>Foreign body sensation</td>
<td>55</td>
<td>100 %</td>
</tr>
<tr>
<td>Watering and discharge</td>
<td>55</td>
<td>100 %</td>
</tr>
<tr>
<td>Fever &amp; Throat pain</td>
<td>10</td>
<td>20 %</td>
</tr>
</tbody>
</table>

**SIGNS**

| Pre auricular lymphadenitis     | 24              | 43.63 %    |
| Conjunctival congestion        | 55              | 100 %      |
| Conjunctival Follicles          | 55              | 100 %      |
| Sub conjunctival hemorrhage    | 10              | 10.90 %    |
| Petechial hemorrhage           | 35              | 63.63 %    |
| Corneal involvement            | 12              | 10.8 %     |
| Uveitis                         | 0               | 0          |

Eye ache, foreign body sensation, watering and discharge were present in all cases. All patients had follicular type of conjunctival congestion (Table 1), 10 patients (20 %) had fever and more often ocular symptoms started 2-3 days after the onset of coryza (Fig. 4).

**Duration of Symptoms** (Interval between the onset of symptoms and initial visit) Fig 4.

Table 2. Results of Virological Study:

<table>
<thead>
<tr>
<th>Virus Isolation</th>
<th>13 samples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adeno virus Type</td>
<td>Type 08</td>
</tr>
<tr>
<td>Total no of specimen’s collected</td>
<td>55 samples</td>
</tr>
<tr>
<td>Investigated for virus isolation</td>
<td>32 samples</td>
</tr>
<tr>
<td>Contaminated</td>
<td>23 samples</td>
</tr>
<tr>
<td>Isolation Rate</td>
<td>40 %</td>
</tr>
</tbody>
</table>

Viral culture was +ve in 40 % cases and was found to be Adenovirus type 8. Out of 55 patients the sample of 23 patients were contaminated probably due to delay in sending the sample.

Type 8 Adenoviral conjunctivitis is a highly contagious disease. It is necessary to diagnose the disease on time for its proper management and all necessary steps should be taken to prevent transmission. According to many authors the treatment for viral conjunctivitis is only supportive. Other than Cidofovir which is found to be effective against the Type 5 Andeno viral conjunctivitis in rabbit model 12, no other antiviral drug is found effective against other strains of Adenovirus.
Table No: 3

<table>
<thead>
<tr>
<th>Regimen Group of patients to whom the treatment was instituted</th>
<th>Combination of medicines to the affected eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 Mild (A)</td>
<td>Antibiotic drops QID alone to the affected eye</td>
</tr>
<tr>
<td>Type 2 (B1)</td>
<td>Topical Acyclovir (3 % e/o 5 times daily) + antibiotic drops QID to the affected eye</td>
</tr>
<tr>
<td>Type 3 B2</td>
<td>Topical Acyclovir (3% e/o) with steroid antibiotic drops to the affected eye</td>
</tr>
</tbody>
</table>

In our study Acyclovir has demonstrated a significant antiviral activity in the early as well as late phase of Type 8 Adenoviral conjunctivitis. Moreover, the incidence of corneal infiltrate was found to be nil with the early application of Acyclovir.

Response to Treatment

Group 1 (consists of 25 patients treated outside with antibiotic drops alone.)- Started on type 3 regime.

Group 2 include 30 patients - Primarily treated here.

A1-10 Pts. with mild C F -Type I Regime
B 1–10 Pts. Treated- Type 2 Regime.
B2 –10 Pts severe-Type 3 Regime.

With the application of antibiotic drops alone, the patients in group 2 A responded well and recovered completely from all symptoms within one week. This might be due to the high immune response of the individual or due to the low virulence of the organism. Even though all the patients in group 2 B1 and B2 responded well with topical regimen, the recovery was found to be faster in group 2 B2. This might be due to the use of Acyclovir, which might be preventing the virus multiplication in the early phase. The applied steroids also play an important role by accelerating the subjective improvement of clinical features by suppressing the polymorphonuclear leukocyte migration and capillary permeability.

In group 1, the patients who did not have keratitis, the symptoms markedly improved within 4 days and the clinical features completely resolved within 7 days, with the topical Acyclovir antibiotic steroid combination. In those patients who had Keratitis the corneal lesions resolved completely without any sequelae within the first 2 weeks of treatment. This might be because the lesions were not deep enough to disrupt the Bowmans membrane to involve the stroma. The use of Acyclovir along with the steroids might have prevented the progression of the natural course of the disease and accelerate the improvement of symptoms.

Conclusion

The inferences from our study are:

1. Early institution of topical Acyclovir can shorten the course of Adenoviral infection.
2. The use of topical Acyclovir with steroids enhances the efficacy of treatment.
3. Topical Acyclovir prevents the development of corneal infiltration when applied in the very early phase of Type 8 Adenoviral conjunctivitis.
4. The administration of antibiotic drops alone may not be safe in all cases.

Table 4

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>3 DAYS</th>
<th>6th day</th>
<th>12th Day</th>
<th>21st Day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Gp1 A</td>
<td>Gp2 A</td>
<td>Gp1 A</td>
<td>Gp2 A</td>
</tr>
<tr>
<td>Eye ache</td>
<td>25</td>
<td>6</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>Fb sensation</td>
<td>25</td>
<td>8</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>Water discharge</td>
<td>25</td>
<td>8</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>CC</td>
<td>25</td>
<td>10</td>
<td>25</td>
<td>0</td>
</tr>
<tr>
<td>Follicles</td>
<td>25</td>
<td>10</td>
<td>25</td>
<td>0</td>
</tr>
<tr>
<td>Keratitis</td>
<td>12</td>
<td>0</td>
<td>12</td>
<td>0</td>
</tr>
</tbody>
</table>
5. Even in cases of Keratitis the administration of topical Acyclovir with steroid prevents the escalation of lesions into the deeper layers of the cornea there by reducing the chances of developing corneal opacities.

References
11. Gorden YJ, Romanowski E, Araullo – Cruz T, De Clercq E. Pretreatment with topical 0.1% (S hydroxyl – 2 – phosphonylmethoxypropyl) cytosine inhibits adenovirus type 5 replication in the new rabbit ocular model. Cornea. 1992;11:529-533

OPHTHALMIC HISTORY

Gerhard Rudolph Edmund Meyer- Schwickerath [1920-1992]

Prof. Padmaja Krishnan MS
(With lasers being the “in” thing in Ophthalmology today, here is the man who paved the way…..)

The power of sunlight to damage the retina was known from ancient times.

“People may injure their bodily eyes by observing and gazing on the sun during an eclipse, unless they take the precaution of only looking at the image reflected in the water or some similar medium”

-Socrates, quoted by Plato in ‘Phaedo’

Galileo injured an eye while looking at the sun with his newly invented refracting telescope, as did the father of photoagulation, Gerhard Rudolf Edmund Meyer-Schickerath, while experimenting with the production of radiant energy.

Meyer-Schickerath, the German ophthalmologist, was born in July 1920 at Wuppertal-Elberfeld, Germany. He left school in 1937 at the age of 17 and decided to become a doctor. This was against the family tradition of studying law as he did not fancy practising Law under the Nazi regime.

When the world war broke out, he joined up as a paramedic, but was sent back from the frontline after he sustained a knee injury. He was thus able to study Medicine, graduated in 1945, took up Ophthalmology and completed his Fellowship from Munster, Hamburg.

In 1952, he moved to the University of Bonn and in 1959 became Professor and Director of the University Eye Department in Essen, where he continued to work until his retirement in 1985.

Meyer-Schickerath conceived the idea of therapeutic photo-coagulation when he was just 25 years old while trying to develop a diathermy machine to treat retinal detachment. He had been seeing a lot of patients, including one of his students, with macular burns from watching the...
ROP Screening: Analysis of Factors Predictive of Poor Visual Outcome and Neurodevelopment in Preterms

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Studies analyzing the morbidity and long term neurological outcome in extremely low birth weight infants (ELBW = < 1000gms and GA ≥ 34 weeks) have shown that 20.6% of the babies develop major neurological sequlae. ROP affected 26.6%, while 14.8% of the children developed intraventricular haemorrhage and 14.1% developed periventricular leucomalacia.

Health professionals and parents of preterm infants fear the development of bronchopulmonary dysplasia and retinopathy of prematurity because these neonatal morbidities are risk factors for development of neurosensory impairment, low psychomotor development index scores on the Bayley scales of infant development II, and neurotic mental development. Ultrasonographic signs of brain injury such as periventricular and intraventricular haemorrhage, periventricular leucomalacia, ventriculomegaly also increase the risks of mental and motor impairments. Severe ROP is significantly associated with neurosensory developmental impairment in children.

Preterm birth, perinatal morbidities, retinopathy of prematurity are all associated with neurological damage and visual impairment. Visual impairment can range from blindness due to ROP or cortical visual impairment, which can be identified at an early age, to subtle deficits related to preterm birth only identified at a later age. Visual function deficits are not limited to visual acuity but can affect contrast sensitivity, field of vision and colour vision. Strabismus (17.8%) has been reported to occur in children who survive the morbidities of preterm birth. Esotropia and pseudoxotropia due to macular ectopia related cicatricial retinopathy of prematurity has been described. Logistic regression analysis showed an increased risk of strabismus in children with cicatrical regressed retinopathy, refractive error, family history of strabismus, poor neuro developmental outcome in particular impaired locomotor skills and hand eye coordination. Cryotherapy for ROP can cause acute inflammation and necrosis of muscle fibers which improves overtime and does not result in structural changes on long term followup. Therefore occurrence of strabismus in patients with ROP is considered to be attributable to reasons other than injury to EOM.

Ophthalmological followup of preterm infants showed severe visual impairment in 2.5%, pointing to the urgent need to follow-up all preterm babies screened for ROP. The rate of myopia was 33.3% in babies with ROP compared to 3.7% in babies with no ROP. Studies have shown that eyes with spontaneously regressed subthreshold stage II ROP were associated with better normal outcomes and lesser myopia than the treated groups.

There is a paucity of data from India on predictors of visual and neurological outcome in preterm babies.

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<33 weeks gestation. The aim of this study is to analyse the various risk factors that act as predictors of poor visual and neurological outcome in very preterm babies and to provide anticipatory guidelines to the parents. This study also highlights the need for long term followup in extremely preterm babies who survive the perinatal morbidities associated with preterm birth and tries to define a model for developing a regional data base.

Materials and Methods

This study was designed as a prospective nonrandomized screening and observational trial with active intervention in the management of ROP as and when required. The study population included 130 in-born and out-born premature babies <33 weeks admitted at the tertiary care NICU of a multispeciality hospital from May 2004 to May 2007. The birth weight, gestational age, multiple oxygen exposure, neonatal illness including respiratory distress syndrome, surfactant therapy, neonatal jaundice, sepsis, shock, hypoglycemia, necrotising enterocolitis were noted. All babies were screened for ROP performing indirect ophthalmoscopy under full pupillary mydriasis at the NICU or at our tertiary care retina clinic. Mydriasis was achieved by instillation of 1:1 dilution of tropicacyl with phenylephrine. The zone of involvement, the stage of the disease, presence or absence of PLUS or RUSH disease was noted. The fundus appearance was classified into three (1) Immature peripheral retinal vasculature (2) prethreshold ROP (3) threshold ROP as in the ICROP study 11. Prethreshold ROP was defined as zone-I involvement in any stage of disease, zone II stage II, plus disease, zone II involvement with a stage less than threshold ROP. Threshold ROP was defined as stage 3 disease in zone I or II with 5 contiguous or 8 total clock hours of involvement with plus disease. The babies were divided into two groups, those with threshold ROP and those without threshold ROP and the birth weight, gestational age and various risk factors compared between the two groups.

The protocol for screening employed by us was as follows. The initial screening was performed before 31 weeks post conceptional age or within 4 weeks of chronological age. The follow up schedule was based on the fundus findings and stage of ROP. Babies whose fundus did not reveal any evidence of ROP were screened every 2 weeks, while those showing features of ROP were screened weekly for any progression of disease. Babies with prethreshold ROP were screened daily. The end point of screening was when the baby achieved a post-conceptional age of 45 weeks or normal intraretinal vascularisation was observed. Babies with threshold ROP underwent cryo or laser ablation of peripheral retina according to the cryo ROP study guidelines 12 within 48 hours of diagnosis and were followed up daily for a week and monthly thereafter for 1 year. After discharge from the NICU, all the babies were reviewed monthly by performing indirect ophthalmoscopy and their refraction was assessed after atropinisation at 6 months after birth. Assessment for ocular motility disorders and binocular visual function was carried out at age of 9 months or earlier if the parents complained of noticing squinting in the child. Corrective spectacle for refractive error and surgery for correction of squint was performed between 1-1½ years. After one year the followup visits were scheduled every 3 monthly for 12 months and thereafter every 6 monthly to age of 3 years. At each followup visit, an assessment of the baby’s neurodevelopment was made by the neonatologists at the tertiary care multispeciality hospital. OAE (Oto acoustic emission) to assess hearing was performed before discharge and also at 3 months after discharge. Those babies with suspected hearing loss were subjected to Brainstem evoked response audiometry (BERA) as a definitive indication of hearing loss. Assessment for neurosensory development was performed by the Denver Developmental screening test (DDST) and the Development assessment scale for Indian infants (DASII) at ages 3, 6, 9 and 12 months. A thorough neurological workup was also performed at age 1, 3, 6, 9, 12 months and the Amiel Tison test for hypertony and spasticity was assessed at each evaluation.

Results

130 consecutive preterm babies with gestational age <33 weeks were screened for ROP and assessed for perinatal morbidities and neurosensory development. This study group constituted the inpatients of the tertiary care NICU of a multi-speciality hospital in Trivandrum and included both in-born and out-born
babies. The mean age was 30.30 weeks (Range 25-32 weeks) and the mean birth weight was 1367.9 gms (730-2400 gm).

Thus out of 130 preterm infants of gestational age <33 weeks who were screened, 30 % had immature vasculature, 10 % prethreshold and 11.7 % (14 babies) had threshold ROP (Table1). Out of these 14 babies, 3 were twins and one baby belonged to a quadruplet pregnancy. The 14 babies with threshold ROP were further analysed with respect to the mean gestational age and birth weight. Analysis of this data showed that these were ELBW babies (extremely low birth weight babies) with a mean gestational age of 28.25 weeks (range 25 weeks-31 weeks) and birth weight of 1048.57 gm (850-1200 gm). Of these 64.3 % babies had a gestational age ≤ 28 weeks, 85.7 % were ≤ 30 weeks while 14.4 % were > 30 weeks. The difference between birth weight and gestational age in the pre terms and those with threshold ROP with significant statistically (TABLE :2)

Preterm babies with threshold ROP underwent cryotherapy to the peripheral retina (2 patients) under short general anaesthesia or peripheral retinal laser ablation to the ischemic retina anterior to the ridge under topical anaesthesia (12 patients). Following treatment the babies were reviewed daily and the fundus periphery assessed for skip lesions. 6 eyes required additional laser after 2 days. In 2 eyes of two patients the retinopathy progressed to stage 4b for which scleral buckling was performed. The post operative outcomes in both was poor. Both eyes became phthisical (7.14 %). Thus there was 7.14 % severe visual loss in this group. In patients with ROP; 3 patients

<table>
<thead>
<tr>
<th>Table 1. Analysis of Fundus findings</th>
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<tbody>
<tr>
<td>Fundus Findings</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>Immature Vasculature</td>
</tr>
<tr>
<td>Demarcation Line</td>
</tr>
<tr>
<td>Prethreshold</td>
</tr>
<tr>
<td>Threshold</td>
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<td>Total</td>
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<table>
<thead>
<tr>
<th>Table 2. Comparing Gestional Age and Birth Weight in Preterms with ROP</th>
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</thead>
<tbody>
<tr>
<td>Preterm</td>
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<tr>
<td>Mean Gestational Age (Range)</td>
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<td>Mean Birth Weight</td>
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</tbody>
</table>

Table 4. Showing Ocular Findings on Followup in Patients with threshold ROP

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Babies/Eye</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refractive Errors</td>
<td>12 Eyes</td>
<td>42.85%</td>
</tr>
<tr>
<td>Myopia</td>
<td>8</td>
<td>66.67%</td>
</tr>
<tr>
<td>Hypermetropia</td>
<td>2</td>
<td>16.67%</td>
</tr>
<tr>
<td>Astigmatism</td>
<td>2</td>
<td>16.67%</td>
</tr>
<tr>
<td>Severe Visual Loss</td>
<td>2</td>
<td>7.14%</td>
</tr>
<tr>
<td>Esotropia</td>
<td>3</td>
<td>14.28%</td>
</tr>
<tr>
<td>Disc + Macular Drag</td>
<td>4</td>
<td>28.57%</td>
</tr>
</tbody>
</table>

Table 5. showing characteristics of threshold ROP babies with strabismus

<table>
<thead>
<tr>
<th>Strabismus ( Esotropia)</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cryotherapy</td>
<td>2</td>
</tr>
<tr>
<td>Myopia</td>
<td>5</td>
</tr>
<tr>
<td>Astigmatism</td>
<td>1</td>
</tr>
<tr>
<td>Macular and Disc Drag</td>
<td>3</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>2</td>
</tr>
<tr>
<td>Neurodevelopmental Delay</td>
<td>2</td>
</tr>
</tbody>
</table>

(21.4 %) had bilateral neovascularisation of iris and tunica vasculosa lentis which regressed after laser ablation. Two of these patients later developed stage 4b ROP in one eye.

Analysis of followup data at one and a half years of age showed esotropia in 3 patients (21.4 %); refractive error in 28.57 % with 87.5 % being myopia (≥ 3.00D Sph) and 12.5 % hypermetropia (≥ +2.00D sph). 4 babies had disc and macular drag (28.57 %) out of which 2 had undergone cryo and the other laser. All the 3 patients with esotropia had macular drag and nystagmus was present in two. Two patients who had undergone cryotherapy were myopic and had esotropia. Two of the three babies had mild neurodevelopmental
delay. Two more preterm babies who did not have ROP during screening developed esotropia on follow up. In one the accommodative component was predominant. Thus our series had a total of 5 babies (3.8 %) with strabismus. In the group with no ROP the incidence of strabismus was 1.7 % only.

Associated perinatal morbidity in our children in whom threshold ROP was detected during screening are given in TABLE-6 comparing them to babies without threshold ROP. Thus it can be seen that respiratory distress and multiple apnoeas and sepsis are more common in the group with threshold ROP while necrotising enterocolitis shows marginal increase in incidence. Respiratory distress, multiple apnoeas, and sepsis were found to be statistically significant. Though ventilation was statistically significant the incidence in those with ROP was less compared to the other group.

Four babies (28.57 %) showed mild neuro developmental delay- three in mental and motor development and one in language delay. There was no morbidity.

The neurodevelopmental followup was carried out in the neurodevelopmental clinic at KIMS Hospital in 63.3 % of babies. An abnormal neurosonogram was seen in 10 % of the 130 babies screened. 8 babies died before discharge and all of them had abnormal neurosonogram and intraventricular haemorrhage > grade 2.

Failed hearing screening (OAE) and definitive BERA was seen in 1 out of 92 babies screened (1.1 %). Abnormal neurological examination (tone/reflexes) and DASII motor score < 70 at 9 months of corrected age was seen in 3 out of the 64 screened (4.7 %) DASII mental score of < 70 at 9 months corrected age was seen in 2/64 screened (3.1 %). Correlating the NDD with gestational age showed that major NDD or death occurred in 42 % of preterm babies with gestational age ≤ 28 weeks in comparison to 9 % of babies with gestational age > 28 weeks (P=0.001) Table:7.

Thus the bad outcome determinants for major neurodevelopmental delay were

1) Gestational age ≤ 28 weeks
2) Need for ventilation
3) Abnormal (> Gr 2 IVH) neurosonogram
4) Shock
5) sPDA
6) Recurrent apnoeas

Regarding visual outcome the bad outcome determinants were extreme prematurity < 28 weeks, history of respiratory distress and multiple apnoeas, sepsis, birth weight < 1200 gm; tunica vasculosa lentis, and need for repeat lasers with poor response to treatment. Cryo treated babies had a higher incidence of myopia ≥ 3.00D Sph; disc and macular drag, esotropia and nystagmus. Strabismus was associated with higher risk of neurodevelopmental delay.

<table>
<thead>
<tr>
<th>Sl. No.</th>
<th>Risks</th>
<th>Threshold ROP</th>
<th>No Threshold ROP</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Babies</td>
<td>Percentage</td>
<td>No. of Babies</td>
</tr>
<tr>
<td>1</td>
<td>Ventilation and Oxygen Admin</td>
<td>10</td>
<td>71.43%</td>
<td>85</td>
</tr>
<tr>
<td>2</td>
<td>Respiratory Distress and multiple ap noeas</td>
<td>10</td>
<td>71.43%</td>
<td>19</td>
</tr>
<tr>
<td>3</td>
<td>Neonatal Jaundice</td>
<td>7</td>
<td>50%</td>
<td>92</td>
</tr>
<tr>
<td>4</td>
<td>Sepsis</td>
<td>8</td>
<td>57.14%</td>
<td>36</td>
</tr>
<tr>
<td>5</td>
<td>PDA</td>
<td>2</td>
<td>14.28%</td>
<td>21</td>
</tr>
<tr>
<td>6</td>
<td>NNEC</td>
<td>3</td>
<td>21.43%</td>
<td>19</td>
</tr>
<tr>
<td>7</td>
<td>Shock</td>
<td>2</td>
<td>14.28%</td>
<td>388</td>
</tr>
<tr>
<td>8</td>
<td>Hypoglycemia</td>
<td>2</td>
<td>14.28%</td>
<td>20</td>
</tr>
</tbody>
</table>
Discussion

There is paucity of data from India on the long term visual and neurological outcome of very low birth weight infants. This study aimed to

1. Analyse various factors that act as predictors of poor visual outcome and neurodevelopmental delay in extremely low birth weight premies ≤ 1250 gms.

2. To provide anticipatory guidelines to parents and help them plan rehabilitation of their precious child.

3. To provide a model for developing a regional data base.

In our study the incidence of threshold ROP was 13.3 %. This is similar to a study by Theng JT et al 13 (14.2 %). The mean gestational age was 28.28 weeks with a range of 25 – 31 weeks and mean birth weight was 1048.57 gms (850-1200 gm). A study in India showed that the mean birth weight of babies undergoing laser or surgery was 1254.5g (range 710 to 2000) and the mean period of gestation was 29.6 weeks 14, while another had 1554g (range 850 to 2290) and 31.75 weeks (28-34) as the mean birth weight and gestational age 15. Many other studies in India have shown threshold ROP occurring in babies > 1250 gm and 1500 g 16,17. There are also various reports of ROP seen in larger, bigger babies in Asia 20,21. However in our study, no babies were >1200g. This is similar to studies conducted in the West 18,19 which report no incidence in babies > 1250g +/- 100g. According to Vedantham 22, the difference between developed and developing countries might reflect the failure of very small infants to thrive and the quality of neonatal care in developing countries. Thus the discrepancy in our study compared to other Indian studies may be due to the fact that our study was in a tertiary referral multispeciality hospital with state of art NICU facilities where only the most high risk babies were referred and lower birth weight and gestational age babies survived.

In our study, 2 eyes (7.14 %) progressed to stage 4b and became blind in spite of treatment. Both had undergone laser therapy. Both had severe tunica vasculosa lentis at the time of laser. Gnanaraj L et al 23 also reports a similar incidence of 10 % severe visual loss in ROP treated eyes.

On follow up the incidence of strabismus was 21.4 % in the threshold ROP group and 1.7 % in the no threshold ROP similar to the study by Theng et al 13. All were esotropic. Refractive error was seen in 28.57 % with 66.67 % being due to myopia > -3.00 D sphere. Premature babies with ROP have been reported to have higher rates of myopia and strabismus especially in the cryotreated group 13,24. Strabismus is found to develop predominantly in the treated group and is frequently associated with neurological damage and anisometropia 24. Here two of the three patients with strabismus had mild neurodevelopmental delay. All had macular ectopia (5 eyes were myopic while one was mixed astigmatic). Two of them had been cryotreated supporting the hypothesis that cryo leads to increased incidence of strabismus and myopia. However according to Yu YS et al 8 only acute inflammation occurs to the muscle fibre during cryotherapy and does not result in long term structural change. Therefore strabismus is attributable to other reasons. According to Pennefather PM et al, increased risk of strabismus is seen with cicatrical retinopathy of prematurity, refractive error, family history of strabismus and poor neurodevelopmental outcome 25,26.

Table 8. Neurodevelopment Follow-Up

- Assessment Performed 63.3%.
- Abnormal Neurosonogram (>Gr II IVH) : 10%
- DASII Motor Score < 60:4.7% (Cerebral Palsy)
- DASII Mental Score < 70:3.1% (Mental Retardation)
- Defective BERA : 1.1%

Fig. 1. Bar diagram showing gestational age distribution in babies with and without threshold ROP.
On analysis of the perinatal risk factors, respiratory distress and multiple apnoeas, sepsis (0.03) were found to be significant risk factors for the development of threshold ROP. The occurrence of ROP correlates with more supplemental oxygen and the administration of CPAP according to Wagner RS 26. In India, Rekha et al 27 reported that duration of oxygen therapy and anemia were independent factors predicting the development of ROP. Dutta et al 28 reported administration of packed cell and double volume exchange transfusion as risk factors for development of threshold ROP. Venekar et al 29 found “out born”, RDS and exchange transfusion as independent risk factors for severe ROP in babies > 1250 gms.

Neurodevelopmental delay in the preterm babies came to 4.7% (cerebral palsy) and 3.1% (mental retardation). However in the threshold ROP group, three babies (21.4%) showed mild developmental delay while one baby showed language delay (total 28.57%). Severe ROP has been found to be significantly associated with neurosensory development impairment in children 3. In our study the bad outcome determinants for neurodevelopment and visual outcome were similar with respect to gestational age, need to ventilate and recurrent apnoeas. Thus there appears to be correlation between the development of the two.

Thus to conclude, there is an urgent need to followup these preterm babies who survive the perinatal morbidity especially with respect to visual and neurodevelopment and behavioral development. Awareness of the various risk factors and its significance on the long term development of the baby will help guide the parents to plan their child's future. A proper screening and early treatment of Retinopathy of Prematurity, correction of associated refractive errors, surgical correction of squint, training of parents and the child to use low visual aids will go a long way in preventing amblyopia, and help in the visual rehabilitation and optimum neurodevelopment of the visually impaired child.

The limitations of this study are its small sample size. Maternal factors and antenatal factors predisposing to the development of threshold ROP were not studied. The major bias is that the results presented here are the results from a single NICU which is a tertiary referral centre and hence high risk babies are referred here. Therefore this cannot be extrapolated as a population based data base. Pooling together available data, longer followup in larger series of preterm babies will help form a definitive data base from our country.

References

13. Theng J.T, Wong TY, Ling Y: Refractive errors and strabismus in premature Asian babies with or without


Fungal Keratitis

Dr. N. Bindu MS

Fungal infections of the cornea constitute an important eye problem in outdoor workers in tropical & subtropical countries. Favourable humid climate and large agricultural & manual labourer population makes fungal keratitis common here. 30-50 % of culture proved suppurative keratitis of non viral origin is constituted by fungal keratitis.

Globally there has been an increase in number of reported cases of fungal keratitis. Increasing use of antibiotics, injudicious use of steroids, increasing laboratory capability for recovering fungi from infected corneas and an increased awareness all have contributed to this.

Fungi causing human keratitis

A. Hyaline Filamentous fungi
   Fusarium ( F.solani,Foxyssporum)
   Aspergillus (A.fumigatus,A.flavus)
   Scedosporium
   Pencillium(Pspinulosum,Pcetrinum)
   Acremonium(Cephalosporium)
   Paecilomyces

B. Dematiaceous Filamentous fungi
   Curvularia(C.lunata,C.geniulata)
   Bipolaris
   Exserohilum
   Cladosporium
   Coelomycetes

C. Yeast like fungi
   Candida (C.albicans)

Aetiology

Fungal ulcer affects young healthy adults (21-60 yrs) who live in rural areas - agriculture being their main occupation. Males are commonly affected as they do most outdoor works than females. Majority of patients are immunocompetent without any external eye disease.

Epidemiology

Aspergillus species is the most common organism responsible for fungal keratitis world wide.

India

<table>
<thead>
<tr>
<th>Organism</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspergillus</td>
<td>27-64 %</td>
</tr>
<tr>
<td>Fusarium</td>
<td>6-32 %</td>
</tr>
<tr>
<td>Pencillium</td>
<td>2-29 %</td>
</tr>
</tbody>
</table>

In northern India, Nepal & coastal Karnataka Aspergillus is found to be more frequently involved. In Southern India, Fusarium is reported as the leading etiologic agent. Candida is very rare in India as causative organism.

Predisposing factors

- Local
- Systemic

Local

1. Trauma- injury to the cornea with vegetable matter or organic matter is reported in 55-65 % of fungal keratitis
2. Contact lenses- In industrialised countries contact lens wear has been identified as a risk factor (29 %).
Patients wearing any type of contact lens can get fungal keratitis

3. Iatrogenic – following cataract surgery, refractive surgery, LASIK, penetrating keratoplasty

4. Topical steroid use - 4-30 % has been reported in various studies. Steroid use tend to activate and increase virulence of fungi

5. Other factors - corneal surface disorders, dry eye, bullous keratopathy, exposure keratitis, allergic conjunctivitis are associated with the development of mycotic keratitis

**Systemic factors**-

- Diabetes - 5%
- Malnutrition - 1%
- Alcoholism – rare
- HIV – rare

Chronically ill & intensive care unit patients are prone to develop candida infection.

**Clinical features**

**Symptoms** – may not present as acutely as with other form of microbial keratitis. Usually signs of inflammation is minimal and absence of lid edema is a common feature.

**Signs** – greyish white/yellowish white infiltrates

Ulcer base filled with soft, creamy, raised infiltrates

Feathery borders or hyphate edges – in 70 %

Hypopyon – solid hypopyon with convex upper border-in 55 %

Satellite lesions - 10 %

Demataceeous fungi keratitis shows black/brown pigmented surface which is dry, rough, leathery and difficult to scrape (fig 1, fig 2, fig 3)

Other features

- Immune ring
- Posterior corneal abscess
- Endothelial plaque

Rarely fusarium lead to endophthalitis.

Depending on aetiological agent each case may vary.

Fusarium species usually produce severe keratitis leading to perforation, deep extension and malignant glaucoma

Aspergillus & curvularia produce less severe ulceration and are more amenable to therapy.

**Microbiological investigations**

Microbiological investigatons should always be performed where fungal infections of cornea is suspected, as it is not possible to distinguish between bacterial and fungal keratitis on clinical findings alone. It includes the following

- Smears
- Staining
- Fungal culture
- PCR
- Confocal microscopy

**Direct microscopic evaluation**-

Most valuable and rapid diagnostic tool for detection of fungal elements. Corneal scrapings are obtained by scraping the base and edges of the ulcer with sterile blade or spatula several times. Smears should be made as thin as possible.

**Stains used & their sensitivity**

<table>
<thead>
<tr>
<th>Stain</th>
<th>Sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram stain</td>
<td>45-73 %</td>
</tr>
<tr>
<td>Giemsa stain</td>
<td>66 %</td>
</tr>
</tbody>
</table>
Lactophenol cotton blue 70-80 %
Grocott's methanamine silver 89 %
Calcoflour white 80-90 %
KOH 10 % wet mount 80-90 %

Calcoflour white
Fungal hyphae and yeast cells are delineated against dark background seen even in thicker preparations also. Acanthamoeba cysts and P. carini can also be detected, but it requires UV microscope.

Culture
Fungal growth usually occurs within 3-4 days (48-72 hrs) but may require incubation upto 4-6 weeks.

Sabo0raud’s dextrose agar is used frequently. It is kept at room temperature. Initial growth occurs within 72hrs in 83% and within 1 week in 97 % cultures. (fig 7).

Gram stain-
Stains yeast cell and fungal hyphae equally well. It can identify bacteria also.

Lactophenol Cotton Blue (fig 6)-detect all common ocular fungi. It is commercially available and has long shelf life.

Blood agar also yields good positivity
Thioglycollate broth and brain heart infusion are not necessary routinely.

Corneal biopsy
Done if smears and cultures are negative in highly suspicious cases. Done under local anaesthesia using 2-3 mm trephine. Corneal button obtained from therapeutic PKP can also be used. Histopathology reveals the presence of fungal elements (fig. 8).
**Suggested lab workup**
KOH wet mount with or without stain
Gram staining
LCB
Culture in SDA and blood agar

**Other methods**
Impression debridement
Cellulose acetate filter paper is applied on to the ulcerated part, stained and examined.

**PCR**
Is an effective method of diagnosing fungal keratitis because it offers increased sensitivity and significant reduction in time (4 hrs) to establish diagnosis.

**Confocal microscopy**
Is a non-invasive invivo examination technique. Can reveal hyphal elements and yeast forms. It is more sensitive than culture.

**Medical Therapy**
Therapy of fungal keratitis is unsatisfactory. Antifungal agents available are mostly fungistatic and requires prolonged therapy.
Response to therapy is slow and the result is better in non severe ulcers

**Drugs available for treatment**

<table>
<thead>
<tr>
<th>Class</th>
<th>Drug</th>
<th>Topical %</th>
<th>Oral mg/d</th>
<th>Parenteral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polyenes</td>
<td>Amphotericin</td>
<td>1-2</td>
<td>200-600</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nystatin</td>
<td>1-2</td>
<td>200-400</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Natamycin</td>
<td>1-2</td>
<td>600-1200</td>
<td></td>
</tr>
<tr>
<td>Azoles</td>
<td>Imidazoles</td>
<td>1-2</td>
<td>60-100</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Clotrimazole</td>
<td>1-2</td>
<td>100-400</td>
<td>2mg/ml</td>
</tr>
<tr>
<td></td>
<td>Miconazole</td>
<td>1-2</td>
<td>200-400</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Econazole</td>
<td>1-2</td>
<td>200-400</td>
<td></td>
</tr>
<tr>
<td>Azoles</td>
<td>Triazoles</td>
<td>1-2</td>
<td>100-400</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fluconazole</td>
<td>1-2</td>
<td>200-400</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Itraconazole</td>
<td>1-2</td>
<td>200-400</td>
<td></td>
</tr>
<tr>
<td>Pyramidines</td>
<td>5-Flurocytosine</td>
<td>1-2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Natamycin –**
Available as 5% suspension (Natamet, Nata, Elmycin)
It is the drug of choice in filamentous fungi. It is effective against Fusarium, Aspergillus, Curvularia and Candida

**Amphotericine B –**
Available as systemic preparation (fungizeone)
Topical drops is prepared by diluting with dextrose or distilled water to a concentration of 0.15-0.5 %. Other routes of administration are
Subconjunctival -10 micrgrams
Intracameral – 5-7 micrograms
Intravitreal – 10 micrograms
Intravenous -0.1 mg/kg bodyweight.
It covers Candida and Aspergillus but is not effective against Fusarium. It is used as first line of therapy in Candida.

**Azoles**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Topical %</th>
<th>Oral mg/d</th>
<th>Parenteral</th>
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<tbody>
<tr>
<td>Ketoconazole</td>
<td>1-2</td>
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<td>Miconazole</td>
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<td>200-400</td>
<td>600-1200</td>
</tr>
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<td>Econazole</td>
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<td></td>
</tr>
<tr>
<td>Clotrimazole</td>
<td>1-2</td>
<td>60-100</td>
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</tr>
<tr>
<td>Fluconazole</td>
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<td>100-400</td>
<td>2mg/ml</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>1-2</td>
<td>200-400</td>
<td></td>
</tr>
</tbody>
</table>

Available commercially as Auroclot, nistin- (clotrimazole), Aurozole (econazole)

**Treatment protocol**

**Specific antifungal therapy**
Depend on availability of the drugs and the results of lab study and severity of ulcer
If hyphae are seen – natamycin or amphotericin topically
If yeast/pseudohyphae seen- amphotericin/ fluconazole/ flucytosine

**Dosage** – topical eye drops hourly during day and 2 hourly at night
In deep keratitis / endophthalmitis / scleritis / after PKP oral ketoconazole /itraconazole /fluconazole given
Miconazole is the drug of choice in paecilomyces
Amphotericin and imidazole are antagonistic.
Treatment should be maintained for 6-12 weeks


Antimicrobial activity of antifungals based on published reports.

<table>
<thead>
<tr>
<th>drug</th>
<th>Aspergillus</th>
<th>Candida</th>
<th>Csporium</th>
<th>Cladosp</th>
<th>Curvulari</th>
<th>Fusarium</th>
<th>Paecilio</th>
<th>Pencillium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amph-B</td>
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<td>S</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Nystatin</td>
<td>S</td>
<td>S</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Natamycin</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td></td>
<td>S</td>
<td>R</td>
<td></td>
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</tr>
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<td>Clotrimazole</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>I</td>
<td>S</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Miconazole</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>I</td>
<td>I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eonazole</td>
<td>S</td>
<td>I</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td></td>
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</tr>
<tr>
<td>Ketoconazol</td>
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<td>Fluconazole</td>
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<tr>
<td>Flucytosine</td>
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<td>S</td>
<td>R</td>
<td>R</td>
<td>i</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

S-susceptible, I-variable, R-resistant

**Surgical therapy**

**a) Frequent corneal debridement-**

Mechanical debridement of corneal epithelium helps in debulking fungi and enhances drug penetration.

It is done with spatula/blade using slitlamp under local anaesthesia every 24-48 hrs

**b) Therapeutic keratoplasty**

It is the ideal method to treat nonhealing fungal keratitis threatening perforation. Structural integrity and eradication of sepsis is achieved in 80-90 % of cases.

Lens should be left undisturbed as far as possible to prevent posterior extension.

Topical antifungal therapy is to be continued. In addition systemic antifungals to be given for 6-8weeks.

Use of post operative steroids topically is controversial

**Other surgical modalities**

- Cyanoacrylate glue
- Conjunctival flap
- Amniotic membrane transplantation

**Conclusion**

Diagnosis and treatment of fungal keratitis can be quite challenging.

Microbiological investigation is essential for correct diagnosis and treatment.

Prolonged medical treatment and prompt timing of surgical intervention are required to increase chances of cure.

Emphasis on prevention and early diagnosis of this potentially blinding disease.

**References**

6. A Panda, MDeb, P Sony , MS Pathengey Are we looking for only fungus in clinical keratomycosis? DOS Aug 2002: Vol 8, 15-7
8. George Alexandrakis Keratitis, Fungal E-Medicine may 27 2005
9. CORNEA-Krachner Chapter 98 -Fungal Keratitis
11. Eye Rounds, org-case 59-Fusarium-Fungal Keratitis, webeye, ophth.uiowa.edu/eyeforum/cases/59-Fusarium-Fungal keratitis-ReNu-moistureLoc.htm
Vital Dyes For Chromovitrectomy: Colours for the Vitreoretinal Surgeon!!!

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

Chromovitrectomy refers to the application of vital dyes during retinal surgery to visualize preretinal tissues and membranes. Chromovitrectomy arises from the difficulty in visualizing and removing the several thin and transparent tissues in the vitreo-retinal interface including the internal limiting membrane (ILM), epiretinal membrane (ERM) and the vitreous. These tissues are involved in the pathogenesis of several macular disorders including macular holes and diabetic macular oedema. Surgical manipulation of these poorly visualized tissues have been shown to induce gliosis, iatrogenic chorioretinopathy and phototoxity. Staining of these tissues with vital dyes may improve their visibility, enhance the ability to peel them as well as ensure complete removal of all tissues, which may lead to a better visual result postoperatively with a lesser recurrence rate.

An ideal vital dye for chromovitrectomy should have the “ability to selectively stain” the internal limiting membrane and the epiretinal membrane, leaving the retina unstained. It should provide adequate colour difference between the stained ILM/ERM and the normal retina. Other favorable characteristics are 1) rapid elimination from vitreous cavity 2) photochemical stability 3) solubility in balanced salt solution 4) absence of toxicity and 5) an adequate light absorption profile.

The vitreoretinal interface staining agents have been used since 2000. They can be classified into three generations depending on the time of introduction.

First Generation: (2000): Indocyanine Green (ICG)

Second Generation: (2003): Infracyanine Green (IFCG), Trypan Blue (TB) and Triamcinolone acetonide (TA)


Table 1 gives the comparison of staining characteristics and structures of various dyes used for chromovitrectomy.

Indocyanine Green (ICG)

ICG is a tricarbocyanine anionic dye with a molecular formula of C_{43}H_{47}N_{2}NaO_{6}S_{2} and a molecular weight of 775 Daltons. This green dye has amphiphilic properties and hence interacts biochemically with different human tissues. ICG demonstrates greatest affinity to the extracellular matrix components of the ILM, thereby exhibiting an ability to selectively stain the ILM.

Chromovitrectomy using ICG for dye assisted peeling of ILM gained acceptance for the management of macular holes. Its use was later extended to improve visualisation of the glial ERM, proliferative membranes of proliferative diabetic retinopathy (PDR) and proliferative vitreoretinopathy (PVR).

Controversial reports on the toxic effects of ICG have been published. These included Muller cell, RPE damage; visual field defects and optic atrophy.
Histopathology of the peeled ILM after ICG assisted ILM peel during chromovitrectomy revealed the presence of cellular structures on and under the ILM\textsuperscript{19,20}. Detection of the presence of retinal elements (plasma membrane of Mullers cells, myofibrocytes, astrocytes) on the peeled ILM raised the issue of retinal damage during peeling. Animal studies and in-vitro experiments indicated a dose dependent ICG mediated toxicity to retinal elements (Mullers cells, ganglion cells, photoreceptors and RPE) \textsuperscript{11, 21-24}. The hypotonic ICG solution has been shown to cause osmotic damage to the retinal cells.

Exposure to ICG causes damage to the photoreceptors and RPE cells leading to apoptosis or necrosis due to light induced damage \textsuperscript{25-27}. Subretinal injection of ICG in rabbit models may result in RPE damage even in concentration as low as 0.5 mg/ml.

There is paucity of published data comparing the effect of ILM peeling with and without the use of ICG.

Majority of studies (Table 2) used ICG in higher concentration and this factor could be responsible for the toxicity. We suggest three safety measures when using ICG for ILM peeling.

1. Perform a fast surgical procedure in order to minimize the duration of contact with RPE cells and to minimize the ICG exposure to light from endo illuminator.
2. Use ICG concentrations lower than 0.5 mg/ml to minimize the risk of RPE damage and possible retinal toxicity.
3. Avoid ICG injection direct through the macular hole by any method to control ILM staining (slow injection, use of the 20 gauge, prototype painting brush called vitreo retinal internal limiting membrane color enhancer (VINCE) \textsuperscript{31}, or by use of perfluorocarbons over the macular hole etc).

**InfraCyanine Green (IfCG)**

IfCG possesses two well recognized pharmacological differences from ICG, which vouches for its safe profile in chromovitrectomy \textsuperscript{32,35}.

1. IfCG is produced in a synthesis mode without sodium iodine. High dose of topical or intraocular iodine can induce severe corneal and retinal damage.
2. The presence of sodium iodine in the ICG solution requires dilution in water resulting in a hypotonic

<table>
<thead>
<tr>
<th>Molecular formula</th>
<th>SF</th>
<th>ICG</th>
<th>IfCG</th>
<th>TB</th>
<th>TA</th>
<th>PB</th>
<th>BrB</th>
<th>FMA</th>
<th>BBG</th>
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<tbody>
<tr>
<td>C_{6}H_{10}Na_{5}O_{5}</td>
<td>C_{6}H_{17}N_{2}NaO_{3}S_{2}</td>
<td>C_{6}H_{24}Na_{4}O_{4}S_{3}</td>
<td>C_{24}H_{31}O_{4}</td>
<td>C_{27}H_{33}N_{2}O_{4}S_{2}</td>
<td>C_{6}H_{11}Br_{4}O_{4}S_{2}</td>
<td>C_{6}H_{12}O_{4}S_{2}Na</td>
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<td></td>
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<td>Molecular weight (Daltons)</td>
<td>376</td>
<td>774</td>
<td>774</td>
<td>961</td>
<td>434</td>
<td>582</td>
<td>670</td>
<td>418</td>
<td>854</td>
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<tr>
<td>Chemical Group</td>
<td>Xanthene</td>
<td>Tricarbo cyanine</td>
<td>Tricarbo cyanine</td>
<td>Diazot</td>
<td>Long-acting Steroid</td>
<td>Triyl methane</td>
<td>Triyl methane</td>
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<td>Color</td>
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<td>Dark Green</td>
<td>Dark Green</td>
<td>Dark blue</td>
<td>White</td>
<td>Blue</td>
<td>Dark blue</td>
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<td>Blue</td>
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<tr>
<td>Affinity to ILM</td>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Moderate</td>
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<td>High</td>
</tr>
<tr>
<td>Affinity to ERM</td>
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<td>Low</td>
<td>High</td>
<td>Low</td>
<td>Moderate</td>
<td>moderate</td>
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<tr>
<td>Affinity to vitreous</td>
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<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>High</td>
<td>Moderate</td>
<td>Unknown</td>
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<tr>
<td>Toxicity to RPE</td>
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<td>Little</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Little</td>
<td>Unknown</td>
<td>Little</td>
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<td>Toxicity to neuroretina</td>
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<td>Moderate</td>
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<td>Little</td>
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<td>Little</td>
<td>Unknown</td>
</tr>
</tbody>
</table>
solution of 248-275 m mol/kg. The iodine free IfCG is dissolved in 5% glucose solvent generating an isoosmotic solution of 294 – 314 m mol/kg. IfCG in concentrations of 0.5 mg/ml selectively stains the ILM just like ICG and has a much safer profile for intravitreal use.

**Trypan Blue**

The anionic hydrophilic azo dye trypan blue has a chemical formula C_{34}H_{24}N_{6}O_{14}S_{4} and a molecular weight of 960 Daltons.

This vital stain traverses the cell membrane in dead cells thereby staining the dead tissues blue. In the 1990s Trypan blue was first used intraocularly to stain the anterior lens capsule to facilitate capsulorrhexis for cataract surgery. The use of trypan blue in chromovitrectomy is limited to the staining of ERM. It stains the ILM minimally sometimes necessitating repeated application to facilitate visualization. Trypan blue staining of ERM helps mark out its entire extent thereby ensuring complete removal.

Clinical studies have clearly demonstrated that Trypan blue exerts little or no toxic effects on the retina. Experimental data, however, disclosed evidence of retinal toxicity following trypan blue staining. Luke et al. reported irreversible damage to the retina after exposure to trypan blue in a bovine model. In contrast to this report Jin et al. and Narayanan et al. showed that damage to the rodent neurosensory cells was dose dependent and the toxicity could be eliminated at lower doses. A new indication for the use of trypan blue was to stain the edges of an open retinal tear on subretinal administration facilitating its identification during vitrectomy.

**Triamcinolone Acetonide (TA)**

Triamcinolone Acetonide (TA) is a synthetic insoluble corticosteroid, with a chemical formula C_{24}H_{31}FO_{6} and a molecular weight of 434 daltons. The white steroid suspension has been used for chromovitrectomy since 2003 to visualize the transparent vitreous gel and the posterior vitreous cortex. Guo et al. compared the effectiveness of four biostains (triamcinolone acetonide, indocyanine green, trypan blue and sodium fluorescein) in delineating the vitreous and reported the best visibility and contrast following use of triamcinolone acetonide. In addition triamcinolone crystals get deposited on the ERM and ILM making their identification and peeling easier. The safety of triamcinolone acetonide to the retina has been demonstrated by several in vivo and in vitro studies. Marison et al. demonstrated that the vehicle (benzyl alcohol) can induce toxicity to the retina in rabbit models and hence the use of preservative free triamcinolone acetonide is recommended.

**Patent Blue**

Patent Blue is a hydrophilic anionic triylmethane dye with a chemical formula of C_{27}H_{31}N_{2}NaO_{6}S_{2} and a molecular weight of 582 Daltons. This dye has been used to stain the anterior capsule during cataract surgery in a concentration of 0.24% . Patent blue exhibit minimal systemic toxicity, carcinogenicity and
mutagenicity. Preliminary clinical data demonstrates a moderate affinity of patent blue to ERM and vitreous and a poor affinity to the ILM. Toxicity studies revealed conflicting reports on retinal toxicity of patent blue. Luke et al demonstrated that patent blue exhibited mild and reversible retinal toxicity, whereas Westermeier et al showed that RPE cells exposed in vitro to patent blue showed no toxicity. Analysis of available data indicate a safer profile for patent blue in comparison to trypan blue, however the exact safe dosage of patent blue for intravitreal injection remains unclear.

**Brilliant Blue G**

Brilliant blue G, also known as Coomassie or acid blue, is a blue biostain with a chemical formula $C_{47}H_{48}N_{3}S_{2}O_{7}Na$ and a molecular weight of 854 Daltons. Human and animal studies on the use of BBG for chromovitrectomy and anterior lens capsular staining were published in 2006. These study results indicate a safe clinical profile for both capsular staining and chromovitrectomy. Absence of corneal endothelial cell damage, no significant retinal pathological changes on light and electron microscopy, no reduction in ERG waves, and no clinical evidence of long term toxicity, were the hallmark results of these studies.

The remarkable affinity to the ILM and absence of toxicity makes it a first real alternative to ICG and IfCG.

**Bromophenol Blue (BrB)**

(Bromophenolsulfonaphthalein) has a molecular weight of 670 Daltons and a chemical formula $C_{19}H_{10}Br_{4}O_{5}S$. In cataract surgery BrB represents an appropriate biostain at a concentration of 1.2 % to stain the anterior lens capsule intensively facilitating easy removal.

Preclinical experiments on six novel vital dyes for chromovitrectomy (Light green yellowish, E68, BrB, Chicagoblu, Rhodamine, Rhodulin blau- basic) showed that BrB stained the ERM and ILM better, and was free of toxicity at concentration of 1.2 % and 0.02 % Similar reports have been obtained from histopathological studies. At a higher concentration of 1 % and 2 % enhanced ILM colouring was possible. Further human clinical data on its safety profile and defining its dosage and indications in chromovitrectomy is awaited.

**Sodium Fluorescein**

Sodium Fluorescein is a hydrophilic xanthene dye with a chemical formula $C_{20}H_{10}Na_{2}O_{5}$ and a molecular weight of 376 Daltons. Abrams And coworkers in 1978 demonstrated the efficacy of intravitreally injected sodium fluorescein in staining the vitreous thereby aiding its complete removal. The toxicity of sodium fluorescein to the retina has not been reported.

**Fluoromethalone Acetate (FMA)**

Fluoromethalone Acetate (FMA) is a synthetic fluorinated glucocorticosteroid with an empirical formula $C_{24}H_{31}FO_{5}$ and a molecular weight of 418 Daltons. Hata et al performed pre-clinical investigations of flurometholone acetate as a potential new adjuvant during vitreous surgery. They found neither reduction in ERG or histological changes following the use of Fluoromethalone acetate and concluded that FMA could be used as an alternative to TA during chromovitrectomy.

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**Table 3. The techniques used for staining the ILM.**

<table>
<thead>
<tr>
<th>SL. No</th>
<th>Technique</th>
<th>Procedure</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Air-filled Technique</td>
<td>FAE to remove fluid from vitreous cavity before dye injection.</td>
<td>Concentrates dye at posterior pole.</td>
<td>Higher retinal toxicity.</td>
</tr>
<tr>
<td>2</td>
<td>Fluid-filled technique</td>
<td>Inj of dye intravitreously into the BSS/RL filled eye.</td>
<td>Immediate dye washout</td>
<td>Less staining as dye is washed out rapidly.</td>
</tr>
<tr>
<td>3</td>
<td>VINE (Vitreoretinal Internal limiting membrane color enhancer.)</td>
<td>Painting brush constructed of a silicone tube connected to a 20 G metal cannula, Diluted dye in silicone cartridge.</td>
<td>Selective staining</td>
<td>Not patented.</td>
</tr>
</tbody>
</table>

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Key issues
1. Chromovitrectomy improved the visualisation of preretinal structures in vitreoretinal surgery.
2. Intravitreal injection of dyes appear to be the most feasible approach to stain the vitreous and preretinal tissues.
3. Lower dye concentration and shorter exposure time can limit side effects.
4. Various technique are available to stain ILM (Table 3) 
   Newer vital dyes exhibiting selective staining of preretal tissue, and no retinal staining are less toxic to the retina.

References


Ultrasound Evaluation of the Posterior Segment of the Eye – A Ready Reckoner

Dr. Mahesh G. MS DO DNB FRCSEd., Dr. A. Giridhar MS, Dr. Ramkumar DO, Dr. Alpesh Rajput DO

Ultrasound Principles

Ultrasound is an acoustic wave that consists of an oscillation of particles in a medium. Ophthalmic ultrasound uses 8-10 MHz probes (1 MHz is 1000000 cycles per second). Ultrasound used in other medical specialties uses 1-5 MHz. When the frequency decreases the wave length increases and penetration increases. Longer wavelength also reduces the resolution. UBM uses higher frequencies like 50-100MHz resulting in less penetration and better resolution. Ultrasound wave is propagated as a longitudinal wave of alternating compressions and rarefactions of molecules. This wave can be refracted and reflected as light. It is the reflected wave or echo that is utilized in ultrasound evaluation. It depends on the acoustic impedence of the media and the difference in acoustic impedence at an interface called acoustic interface. Also the angle of incidence determines the amount of echo returning. Perpendicular incident waves produce maximum echoes.

Ophthalmic ultrasound uses a pulse echo system which is a piezo electric element which undergoes mechanical vibration when stimulated by electrical energy producing a longitudinal ultrasound wave. The parts of ultrasound system include a pulser, transducer, receiver and a display screen. Amplification plays an important role in ophthalmic ultrasound. It determines the ability of the system to display range of echo intensities. This dynamic range is displayed in units of decibels.

Terminologies

Linear amplification- is on a small range and can display minor differences in echo strength between two echo sources but the range of intensities that can be displayed is limited.

Logarithmic amplification- Large dynamic range can be displayed but the small differences between two echo signals cannot be displayed.

S amplification – developed by Ossoinig – Combines the wide range of logarithmic amplifiers and great sensitivity of linear amplifiers

Gain measured in decibels represents relative units of ultrasound intensity. By adjusting the gain, amplification of echo signals displayed in the screen can be changed. It is just like adjusting the volume of radio where we can control the signals received by the radio. The higher the gain the greater the ability of the machine to detect weaker signals. When gain is reduced only stronger echoes will be displayed.

Time gain compensation (TGC): To enhance weaker signals from deeper tissues. Allows selective amplification of weaker distant echoes compared to stronger nearer echoes.

Standardized echography: Combined use of standardized A scan and contact B scan developed by Ossoinig.

Indications for Ultrasound Examination

1. Posterior segment evaluation in the presence of
Few classic ultrasound findings

Fig. 1. 10 MHz ophthalmic ultrasound B/A scan machine with probes and display units

Fig. 2. Point like echoes in vitreous haemorrhage, membrane like lesion in retinal detachment and mass like lesion

Fig. 3. To differentiate RD and PVD. RD has 100% reflective membranous echo with attachment to optic disc and reduced after movement. PVD has variable spike height with good after movements and if complete no attachment at the disc.

Fig. 4 (a & b) Shifting fluid in exudative retinal detachment. The figure on the left shows membranous echo inserting at the disc with high reflectivity and good after movements. To the left is the same retinal detachment in sitting position showing shifting fluid. There is significant choroidal thickening and T sign.

Fig. 5. Dislocated cataractous crystalline lens. Lens capsule is not intact. Point like and membrane like echoes are present in the vitreous cavity. There will be mobility of the lesion on eye movements.

Ultrasound findings in Diabetic retinopathy

Fig. 6. Plenty of point like echoes in the vitreous cavity suggestive of vitreous haemorrhage

Fig. 7. Vitreous haemorrhage with incomplete posterior vitreous detachment. There are multiple point like echoes in the gel.

Fig. 8. Vitreous haemorrhage with schisis cavity inside and complete PVD. The after movements will be very good in presence of complete PVD.
Fig. 9. Membranous echo attached to the disc which is incomplete PVD. There are point like echoes beneath the membrane suggestive of subhyaloid haemorrhage. Also there are multiple echoes in the pre-papillary area due to adherent fibrous proliferation and peripapillary tractional retinal detachment.

Fig. 10. Membranous echo inserting at the disc with moderate after movements. This is PVD. There is point like echoes beneath this layer suggestive of sub vitreal or subhyaloid haemorrhage.

Fig. 11. Membranous echo in the lower part with plenty of point echoes beneath suggestive of Incomplete PVD of lower part with sub vitreal haemorrhage. There is intragel haemorrhage also.

Fig. 12. Two membranous attachment pulling the retina in a tent like fashion. This is tractional retinal detachment. There is no PVD between the TRD along the arcades. This is called table top TRD.

Fig. 13. Two membranous attachment pulling the retina in a tent like fashion. There is PVD between the TRD.

Fig. 14. Multiple membranous lesions attaching to the disc area with vitreoschisis. There is subvitreal haemorrhage as well as subretinal haemorrhage. In the lower part there is retinal detachment.

**Ultrasound findings in Trauma**

Fig. 15. High reflective (100% spike height) intraocular foreign body with shadowing behind. Low gain examination will help in the better delineation.

Fig. 16. High reflective point like echo with shadowing suggestive of radio opaque retained intraocular foreign body. Also there is a shallow retinal detachment seen as membranous high reflective echo with not much after movements.
opaque ocular media like corneal opacity, hyphema, cataract or vitreous haemorrhage
2. In clear ocular media – Tumors, choroidal detachment, optic disc anomalies like drusen
3. Intraocular foreign body

**Examination Techniques** - It is usually done with the eye lid closed and other eye kept open fixing at a target. Coupling medium like methylcellulose is applied on the B-scan probe. In case of trauma or recent ocular surgery, probe has to be cleaned before use.

**B-scan Probe Orientation:**
1. Transverse scan – The Probe is kept at the limbus with the axis of marker circumferential at limbus. The area of the marker is displayed in the upper part of the screen. This can be horizontal, vertical and or oblique transverse scans.
2. Longitudinal scan – The marker is perpendicular to the limbus.
3. Axial Scan - Is done with the patient fixing in primary gaze and probe centered in the cornea. It displays lens and optic nerve in the center of the echogram. This is useful for evaluation of macula.

**Basic B-scan screening protocol**
1. Transverse scan of 4 major quadrants at high gain.
2. Longitudinal scan in 4 major meridians
3. Axial scan.
After using high gain to detect vitreous opacities and gross fundus lesions low gain with improved resolution is used to detect flatter fundus elevations and to detect the topography of large lesions.

**Special examination techniques**

1. **Topography** - Location, extension and shape. 
   Lesion types can be point like, membrane like, band like and mass like.

2. **Quantitative** – Reflectivity, internal structure and sound attenuation.

**Quantitative Echography type-I**

*Reflectivity* – Spike height in A-scan (0-100%) and signal brightness in B-scan.

*Internal structure* – Architecture inside a mass like lesion – regular and irregular.

*Sound attenuation* - When sound energy is scattered, reflected or absorbed. On A-scan decrease in the spike height is called angle Kappa which is determined by drawing a line through peaks or lesion spikes. The steeper the angle, the greater the sound attenuation.

**Quantitative Echography type II**

To differentiate retinal detachment from vitreous membrane.

3. **Kinetic** – After movements and vascularity.

Kinetic Echography is used to dynamically assess the motion of or within the lesion. This includes 1. After movement on stopping the eye movement suddenly 2. Vascularity which is fast spontaneous motion best seen in standardized A-scan with eye steady 3. Convection movements are slow, spontaneous movements seen in longstanding intraocular haemorrhage or cholesterol debris.

2. **Anterior segment evaluation** using immersion techniques with scleral shells is mostly replaced by ultrasound biomicroscopy.

**Reference**

   (Ultrasound pictures from Giridhar eye Institute Archives. Authors have no financial interest in any product or machine shown)
Visual Restoration Using Keratoprosthesis Surgery

Dr. Srinivas K. Rao DO DNB FRCSEd

(The author wishes to acknowledge Prof Giancarlo Falcinelli and Dr Johnny Falcinelli for teaching him keratoprosthesis surgery and for the illustrations appearing in this article).

The term ocular surface refers to a complex unit that is comprised by many structures acting in an integrated and coordinated fashion to achieve comfort and excellent vision for the patient. A healthy tear film is vital for a healthy ocular surface. They provide lubrication, nutrition, trophic health factors, mechanical drainage conduits to remove trapped debris, and contain antimicrobial defense systems. A healthy lid and blink mechanism helps to facilitate the proper spread of tears and also in maintaining the drainage dynamics. Diseases that interfere with these factors will result in damage to the ocular structures as well.

The physical ocular surface is constituted by the conjunctival and corneal epithelial compartments and their distinctness is supported and maintained by the presence of a healthy limbus. Since many diseases can affect one or more or sometimes all of these structures, the commonest example being chemical burns of the ocular surface, the reconstruction of these damaged eyes is a complex process and requires attention to the many alterations that are present. In eyes with end-stage disease, resulting in a skin-like appearance of the ocular surface with extensive disorganization of the lid-eye relationships, a keratoprosthesis offers hope of visual rehabilitation.

If normal tear function is subnormal the use of newer tear substitutes that are either unpreserved or contain preservatives that are less toxic to the surface, tear preservation using punctual plugs or surgical options, and restoring tear secretion by transfer of the parotid duct or transplantation of the submandibular salivary gland may be needed. The use of secretagogues like oral pilocarpine has shown some promise in early trials, despite the presence of significant side effects.

Cicatrization of the conjunctiva can result in tethering of the lids to the globe and restriction of movement of both the lids and the globe. The principles of surgery include the thorough release of all adhesions of the conjunctiva to the globe and excision of the fibrotic scar tissues, restoring the normal anatomy in the region. In order to prevent a recurrence of the condition, it is important that sufficient regenerative potential is present in the conjunctival tissues. In this context, it is important during surgery to use a substrate that will serve to separate the raw tissues of the globe and the lids and will also promote conjunctival epithelial healing, while at the same time reducing the scarring response of the ocular tissues. The ideal material that provides these functions is amniotic membrane. If however, it appears that the extensive conjunctival loss has resulted in little potential for regeneration, it may be important to use a conjunctival substitute to reconstruct the surface. Traditionally, oral mucosa has been used and provides acceptable results, despite the anatomical differences compared to the normal conjunctiva.

In the context of corneal surface reconstruction, one of the important problems that often confront a surgeon

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A. Stage I

The affected eye

The extracted tooth

Osteodental lamina prepared from tooth

PMMA cylinder glued to osteodental lamina

Osteodental lamina – cylinder complex implanted in subcutaneous pocket

Pannus removal from affected eye

Excised cheek mucosal flap

Mucosa sutured to ocular surface
B. Stage II

Osteodental lamina – cylinder complex removed from pocket

Mucosal flap retracted from ocular surface

Central cornea trephined

Iris removed completely

Cryoextraction of lens

Limited anterior vitrectomy

Lamina implanted in eye

Lamina covered by mucosal flap with central opening for cylinder
is the presence of a persistent epithelial defect (PED). By definition this is a corneal ulcer that has persisted for 2 weeks despite adequate therapy. Such a persistent breach in the corneal epithelium has the potential to progress, often rapidly, to serious complications such as corneal infection, vascularization, thinning and even perforation. For the purpose of this discussion it is assumed that the persistent ulcer is not due to corneal infection – as the treatment approach of this entity is beyond the scope of this article. If an infection has been ruled out, it may then be necessary to identify the actual cause of the PED to allow a systematic approach to the diagnosis and management. The concept of the XYZ hypothesis is useful in addressing this problem.

The XYZ hypothesis proposed by Richard Thoft postulates that the health of the corneal epithelial surface is maintained if the relationship $X + Y = Z$ is present. The term $X$ refers to the centripetal migration of epithelial cells from the limbus into the corneal epithelial pool, while $Y$ represents the upward epithelial migration from the basal layers of the epithelium. The combination of these two should match the factor $Z$ which represents loss form the surface. Any imbalance in this relationship can affect the health and integrity of the corneal epithelial surface.

If the problem is with the factor $X$ – meaning that adequate corneal epithelial cells are not being produced by the limbus, it is necessary to pay attention to restoring the limbal stem cell population in these eyes. On the other hand the problem may be with the factor $Y$ – the cells produced at the limbus may have difficulty in migrating across the corneal surface, in adhering to the underlying structures, and therefore unable to further multiply and provide the upward growth phase. If this is identified as a factor, then efforts to resolve the underlying issues can be undertaken. Depending on the underlying condition, one or more of the following options may be considered -

- Pressure patching / contact lens
- Diluted topical steroids
- Debridement / Superficial keratectomy
- Corneal glue
- Anterior stromal puncture
- Amniotic membrane patch graft

The use of autoserum tears, proposed by Tsubota, can be an useful adjunct in this situation. The rationale for their use is that the serum is likely to contain many of the trophic factors that are present in normal tears and the use of these factors as topical drops can help healing in such eyes.

Thus, ocular surface problems have many manifestations and causes and careful attention must be paid to the examination of such eyes, in order to determine the causative factors. A planned, often staged surgical approach must then be considered to ensure that these factors are tackled appropriately. When such an approach is followed, adequate restoration of ocular surface function is possible in most instances. However, in a subset of eyes with end-stage disease, where the surface appears keratinized, the hostile surface environment and total lack of an ocular surface defense, results in very poor survival of any transplanted biological tissues. In such eyes, the only hope for restoration of visual function is the use of prosthetic devices and the use of the modified osteo-odontokeratoprosthesis (MOOKP) is described. This technique uses a composite bone-tooth lamina to help anchor a polymethyl methacrylate cylinder to the cornea. Originally pioneered by Prof Strampelli, it has evolved to its present form due to the interest and expertise of Prof Giancarlo Falcinelli from Italy.
The complex surgical procedure is performed in two stages. In the first stage, a canine tooth is harvested from the mouth of the patient after X-ray screening has determined that the tooth has a healthy and viable root structure. A surgical motorized saw is used to excise the canine root encased in alveolar bone from the jaw. The lamina is fashioned by sawing through the root of the tooth in a longitudinal fashion to expose the dentine and the root canal. The pulp in the root canal is scraped off and a hole is drilled in the widest part of the root – to a size of 3 to 4 mm depending on the width of the root at that point. An appropriate sized plastic cylinder of appropriate power (determined from the axial length of the eye to be operated) is then glued to the hole using dental cement. A subcutaneous pocket is created in the tissues of the cheek and the lamina-cylinder complex is placed and the pocket is sutured closed after installing antibiotic powder. In the eye, the symblephara are released, and scar tissue is excised as described earlier. A superficial keratectomy including the Bowman’s layer is performed to expose the bare corneal stroma after which a full-thickness circular piece of cheek mucosa about 4 mm in diameter is placed over the cornea and sutured to sclera, also covering the muscle insertions.

Stage II is performed 2 to 3 months later to allow time for a connective tissue cover to develop around the lamina implanted in the cheek. If required the integrity of the lamina can be checked by performing a spiral computed tomographic evaluation. During the second stage surgery, the lamina is retrieved from the subcutaneous location and excess connective tissue is removed from the two ends of the optic cylinder, and trimmed over the rest of the lamina. The mucosal graft on the ocular surface is incised superiorly and reflected from the superior sclera and cornea, in a downward direction. The inferior attachment of the mucosal graft is left undisturbed to ensure that the blood supply is retained.

A Flieringa ring is sutured in place and a 3mm opening is created in the center of the cornea. Three radial incisions are made in the cornea extending till the limbus. The iris is torn at the root and removed and hypotensive anesthesia is used to control the ooze. Constant irrigation with balanced salt solution also helps wash the blood away and prevents a large clot from forming in the anterior chamber. The lens is then cryoextracted and the corneal radial cuts are sutured closed. A limited anterior vitrectomy is performed and the lamina is then placed over the cornea, such that the posterior part of the optic cylinder is in the anterior chamber – entering through the central corneal opening. The lamina is sutured into position using the connective tissue covering and episcleral bites. At the conclusion of suturing, indirect ophthalmoscopy is performed to ensure that there is a good view of the disc and posterior pole. If this is not seen, a cylinder tilt may be responsible and sutures need to be adjusted to straighten the cylinder. Any bleeding into the vitreous cavity can also interfere with the visualization. After the cylinder and lamina are in satisfactory position, the mucosal flap is replaced and a small opening is created over the optic cylinder to allow the anterior portion of the cylinder to protrude through the mucosa. The superior edge of the mucosal flap is sutured in place and this completes the operation.
Haemato-oncological Disorders - Ocular Manifestations

Dr. Natasha Radhakrishnan MS DNB MRC Ophth, Dr. Lakshmi Nisha Menon DO, Dr. Gopal S Pillai MD DNB FRCS, Dr. Anuradha Rao MS

Ocular manifestations of hematological malignancies are protean. They often form the first inkling the patient and the physician get of these life threatening conditions. The findings can range from retinal, preretal and vitreous haemorrhages, exudates, vascular occlusions, infiltrations of retina and optic nerve head, orbit and conjunctiva. Here are some of these conditions and their ocular findings.

**Aplastic Anemia**

- Subhyaloid haemorrhage with macular star right eye
- Vitreous bleed with subhyaloid hemorrhage

**Erythroid marrow hyperplasia**

- Roths spots and retinal haemorrhages

**Non Hodgkins Lymphoma**

- Salmon patch in conjunctiva

**Multiple Myeloma**

- Multiple retinal haemorrhages with vitreous haemorrhage

**Lymphoblastic Leukaemia**

- Lymphoid infiltration of the retina

_Amritha Institute of Medical Sciences & Research Centre, Edappally, Cochin 682 026_
Aplastic anemia

Chronic myeloid leukaemia

Macular subhyaloid haemorrhage

Retinal haemorrhages left eye

After YAG hyaloidotomy

Haemorrhages, vascular sheathing and exudates

MEDICAL RETINA FELLOWSHIP AT CHAKRABARTI EYE CARE CENTRE TRIVANDRUM

Application invited for one year medical retina fellowship starting in August 2008. This fellowship will provide comprehensive training in diagnosis and management of common medical retinal conditions. Training in interpretation of fluorescein angiography, optical coherence tomography, laser and cryo as well as an exposure to surgical retina is planned as part of this fellowship. The candidate should have MS/DO/DNB degree from a recognized institution. Apply with full CV, contact details with two references to

Dr. Meena Chakrabarti
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Managing a Cosmetic Blemish

Dr. Ani Sreedhar 1 MS, Dr. Gangadhara Sundar 2 MS, Dr. Santosh Honavar 3 MS, Dr. K.R Satish 4 MS, Dr. Shantha Amrith 2 MS, Dr. Shruthi Tara 5 MS, Dr. Venkatesh Prabhakaran 6 MS

22 yr old female upon facing social stigma was referred for Enucleation / Evisceration for a disfiguring right eye with no perception of light. She gave no significant history of note and the blemish was attributed as a sequel to an old uncertain childhood trauma, with no surgical interventions in the past.

The affected right eye was congested (Fig. 1) with anterior and 360 degrees ciliary staphyloma with barely visible sclera superiorly, laterally and inferiorly (Fig 2). She also had a high lid crease in that eye and an entropion of the lower eye lid (Fig 3), which was thought to be secondary to the severe staphyloma.

Anterior segment and fundus examination of the left eye was unremarkable.

B scan ultrasonography and CT scan of the orbit did not reveal any intraocular lesion. The bony orbits were of the same size on both the sides. CT guided axial length measurement of both the globes were done. Right eye measured upto 26 mm and the left eye 22 mm.

Experts in oculoplasty were consulted and their opinions were sought for the following queries which most often arise prior to performing an evisceration or an enucleation procedure.

1. Would you prefer Enucleation or Evisceration in this young patient?
2. What would be the implant of choice?
3. What would be the appropriate size of the implant?
4. If you consider enucleation, what would be the wrapping material of choice?
5. If you are thinking in terms of evisceration, what modifications would you prefer in the surgical technique to accommodate a large size implant?
6. If you consider using a porous implant, will you keep pegging in mind, if so when?
7. How would you manage the entropion?

Dr. Ani Sreedhar

This case of ciliary staphyloma, which even though not painful can produce uncontrollable bleeding on slight trauma. So, I would do an evisceration with implant. An evisceration is always preferable to enucleation as it produces lesser trauma to the orbit and imparts more movement to the implant.

But, I would first do an ultrasound to confirm if there is enough space for an implant. A silicon implant would do well in an evisceration and a custom made artificial eye can give satisfactory movements within the conversational range.

Dr. Gangadhara Sundar

While the controversy continues, when cosmesis and motility of the prosthesis is the primary goal, an evisceration may be preferable to enucleation, although the potential possibility of sympathetic ophthalmia should be discussed with the patient and hence need for prompt attention with any symptoms in the left eye. Having said that, the slight increased incidence of wound dehiscence and exposure (porous)/extrusion
Although it has been shown that there is no significant difference in implant/socket motility and thus prosthesis motility whether a porous or non-porous implant is placed when pegging is not performed, if a pegging may be considered in the future, it is advisable to place a porous implant (porous polypropylene, synthetic or natural hydroxyapatite, ceramic). Thereby the first surgery has laid the foundation for future pegging, if/when indicated.

Sarah Kaltreider has shown that the appropriate size implant in symmetrical orbits is about 2 mm smaller than the contralateral eye. Thus in this case a 20 mm implant could be easily considered. Moreover compared to the standard evisceration patient, given the enlarged scleral volume/area, this should be easily accommodated and allow imbrication of the scleral lips as the first layer of closure. If a porous implant is placed, posterior scleral windows may be created to facilitate early vascularisation.

If enucleation is performed, choices include autologous material like fascia lata or temporalis fascia, allogenic donor material like banked sclera, allogenic material like bovine pericardium, or
synthetic material like vicryl mesh. Mersilene mesh has been shown to have a significant incidence of exposure and infection. When cost is a factor, donor sclera is readily available from certified tissue/eye banks and has been properly screened for Hepatitis B and C and HIV and has been preserved in alcohol and if there is no contraindication, it may be used. Alternatively, one of the other materials may also be considered, each with its own limitation.

In this patient, given the staphyloma, there should be adequate sclera (although thinned out) to cover the implant with imbrication of the anterior lips. When inadequate sclera is present (as is often the case in most blind/painful eyes, posterior radial sclerotomies or myoscleral flap techniques may be considered.

In general, motility should be better with the use of customized prosthesis and thus satisfactory in most patients. Only when the patient desires additional motility and is willing to accept potential complications such as exposure, infection and extrusion of the sleeve pegging may be considered, usually 4-6 months later when adequate vascularisation has been confirmed by orbital imaging (MRI with Gadolinium).

Entropion is probably acquired from the prominent eye stretching of the lower eyelid and causing relative movement of the anterior lamella over the posterior. Often, this resolves upon reduction of size of the globe/enucleation/evisceration and may not need specific measures unless persistent.

**Dr. Santosh Honavar**

I would prefer evisceration because of its obvious advantages over enucleation. Although one may be concerned about the thin anterior sclera, it would still be of useful tectonic quality. Posterior sclera is generally good. In the worst case scenario that the posterior sclera is also thin, the suggested modification in surgery would be to create posterior sclerotomy and place the implant posterior to the sclera.

PMMA is my choice. It is time tested, inexpensive and has minimal complications. The functional outcome in evisceration is not implant dependant unless pegging is planned. Generally, when an implant is placed intrasclerally without any relaxing incision, 16-18 mm would be optimal. However, larger implants can be used and posterior orbital volume replacement can be accomplished by providing relaxing incisions. In a situation of staphyloma, often there is a component of orbital fat atrophy and an implant even up to 22 mm may be required to adequately replace volume. I assess the correction intraoperatively. My end point would be planar matching of the apex of the conformer with the apex of the contralateral cornea at the end of the surgery. There will be enough room for a good prosthesis once the surgical edema settles down.

If enucleation is performed, I wouldn’t wrap PMMA and use myoconjunctival technique for optimal implant and prosthesis motility. I would use scleral cap for porous polyethylene and HA.

Sclerotomy by whatever technique – radial mid-posterior sclerotomy in oblique meridians in between the recti, circumferential equatorial sclerotomy, posterior circum-papillary sclerorotomy with radial relaxing incisions, clover leaf sclerotomy, four flap sclerotomy etc. can be done to accommodate large size implants.

Personally, I would discourage pegging because of its long-term complications.

I think entropion here is secondary to large staphyloma and consequent horizontal eyelid laxity. I have also seen that the lateral canthus is often lax. I would allow the eyelids to settle down for 6-8 weeks following evisceration and may consider horizontal eyelid shortening (sometimes for the upper eyelid as well) with lateral canthal refixation.

**Dr. K.R Satish**

It is indeed unfortunate that this cosmetic blemish not only sightless but is truly unsightly. The reason for presentation itself tells that this young lady is highly motivated to look better. It is important to give her a detailed account of the realistic expectations of the proposed surgery. That makes the surgeon’s job to be very diligent and well planned out.

I prefer to perform an enucleation of the right eye under GA. I feel that it gives us a controlled procedure in comparison to evisceration.
I am more inclined to use Medpor (biocompatible porous polyethylene) implants for a variety of oculoplastic procedures. My choice for this patient is the Quasiintegrated Medpor Quad Motility implant (Fig. 4) of medium size which can be implanted without wrapping. It has four mamillations on its anterior surface which helps to give corresponding elevations on the conjunctival surface. This will help to effectively lock in the posterior surface of the prosthesis later, thereby doing away with a separate pegging procedure.

The entropion should settle down spontaneously with removal of the staphylomatous eye.

Dr. Shantha Amrith

I would do an enucleation, as the sclera is very thinned out anteriorly and therefore it will restrict the size of the implant that you can use in an evisceration despite the modifications such as cutting the sclera around the optic nerve and giving relief cuts.

I would use Medpor implant (Fig. 5).

I will do an A scan for the good eye to determine the axial length. Axial length – 2 mm will be the size that I would like to use for the implant. However, I will check the size introperatively to confirm using the sizers available (Fig. 6).

Alloderm or vicryl mesh depending on the availability can be used to wrap the implant. We can also use the sclera of the same enucleated eye apart from the cadaver sclera.

I don’t peg normally, so I won’t be doing it here. A large eyeball pushing the lid could be the cause of entropion and that should settle spontaneously after enucleation.

Dr. Venkatesh Prabhakaran

I would prefer enucleation to avoid the remote risk of sympathetic ophthalmia in this case. When faced with a choice between enucleation and evisceration for blind eyes, I take into consideration previous history of trauma, previous surgeries, especially vitreo-retinal procedures, and B-scan findings (if fundus is not visible). The issue of sympathetic ophthalmia following evisceration is controversial as there are very few case reports but I believe it is a real risk especially in the situations cited above and would prefer enucleation for these cases.

Concerning the issue of cosmesis and motility following enucleation or evisceration, I do not think there is any real difference (although ocularists believe that evisceration results in better motility) if proper surgical techniques are employed this issue usually does not influence my choice of procedure.

For most patients I use a PMMA ball with a myo conjunctival enucleation technique with excellent results. Though polyethylene (Medpor) or bioceramic (aluminium oxide) wrapped in vicryl mesh tend to provide the best post-operative motility (as the muscles are sutured to the implant), most Indian patients are unable to afford these implants. In any case, if pegging is not employed, there is no significant difference in motility between PMMA and the integrated (porous) implants. I personally do not use hydroxyapatite since exposure occurs in about 5 % of patients and treatment is difficult.

Either 20 or 22 mm implant would be my choice. If sizing balls are available, the size can be checked per-operatively. I usually first try 22 mm and if it is too large, use 20 mm. I very rarely use a smaller size.

As I mentioned previously, I use PMMA implant with a myo-conjunctival technique. So I do not use a wrapping material. If I was using an integrated (porous) implant I prefer Vicryl mesh.

My standard evisceration procedure is a modified split-sclera technique wherein I make two oblique cuts in the sclera at 5 and 11 o’clock to extend posterior to the equator. I then make a circumferential incision around the optic nerve head so as to detach the sclera from the optic nerve. Once this is done any size implant may be used and I use a 20 or 22 mm implant in most cases. The implant is placed in the orbital fat and the sclera is then closed in front of the implant in a double-breasted fashion and then the Tenon’s capsule and conjunctiva are closed separately.

I have no experience with pegging porous implants. I would do lower lid retractor plication with a lateral wedge excision (even if it is a purely spastic entropion some form of lateral lid tightening is usually required, especially since laxity may be exacerbated following enucleation).
Enucleation vs. Evisceration, the procedure of choice has always been a topic of debate.

As ophthalmologists, we are all aware of the absolute indications and contraindications for enucleation and evisceration which gives us no room for any doubts regarding the procedure.

However, managing patients in the grey zone like, painful blind eye, blind disfigured eye, pre phthisical eye and chronic endophthalmitis is always challenging. Whatever be the procedure of choice, the ultimate goal is to remove the eye, replace adequate volume and restore cosmesis.

This young lady was highly motivated for some form of cosmetic correction as she was subjected to severe emotional trauma.

We decided to eviscerate, as the patient’s main concern was cosmesis and so, motility would be an issue. Evisceration has obvious advantages over enucleation which includes better motility and relative ease of the procedure.

Risks and benefits of the procedure, including that of general anesthesia, the theoretical chances of sympathetic ophthalmitis and implant exposure were discussed in detail with the patient.

Considering the socio-economic status of the patient and uncomfortable with pegging, we preferred using PMMA implant of size 20 mm. This was calculated based on the measurement of the axial length of the opposite eye minus 2 mm (22 mm-2 mm). Another alternative to calculate the volume would be by using the formula 4/3 r³.

The disadvantage of a standard evisceration technique is the difficulty in accommodating a large size implant to prevent superior sulcus deformity at a later date. To overcome this, myoscleral flap technique was used, this gave us enough area to house the implant and the scleral flaps were closed without tension. The tenon’s and the conjunctiva were closed in layers. This is important to prevent the possible chances of implant exposure.

After evisceration, entropion of the lower lid settled spontaneously. As she also had a high lid crease she has been warned about the possibility of a ptosis. She is currently on conformer and waiting for her customized prosthesis which can be fitted 4-6 weeks after the initial procedure.

In conclusion, the procedure of choice is based on the clinical judgment, surgeon’s comfort level, patient’s choice and individualization of the implant for optimal volume replacement and aesthetic results, stressing a multi-factorial approach in managing such patients.

References

Mucormycosis - Genuinely Sight Threatening and Life Threatening

Dr. Anuradha S. Rao MS, Dr. Lakshmi Nisha Menon MS, Dr. Indudharan, Dr. Lakshmi C., Dr. Jisha Vimoj

Introduction

Mucormycosis is a well described but often misdiagnosed and eventually mismanaged complex disease which appears unseemingly benign but is fatally malignant in its behaviour. The nidus of infection is always hidden in the sinus but the manifestation is invariably orbital and hence the need for the ophthalmologist to be aware of this entity.

It is usually described that the infection is most commonly seen in uncontrolled diabetes as acidosis and hyperglycemia provide a rich source of nutrients for growth: but any immunocompromised state can precipitate the disease.

Here are a few cases with varied presentations of mucormycosis.

1) A 36 year old male presented with acute onset proptosis of the right eye of 7 days following an episode of viral fever. On examination his visual acuity in both eyes was 6/6. Except for the minimal axial proptosis of 22 mm in the right eye, extraocular movements were full, anterior segment and fundus of both eyes were normal. On further examination he had diffuse fullness on the right side of face and infraorbital anaesthesia.

Blood investigations revealed leucopenia

ENT examination was done and sinus endoscopy revealed frank sinusitis with slough in the paranasal sinuses. No organism was identified.

The patient rapidly deteriorated with restriction of extraocular movements and acute loss of vision in the right eye within a few days of presentation. His vision dropped to no perception of light. There was a relative afferent papillary defect in the right eye but fundus was normal. A clinical diagnosis of orbital apex syndrome was made.

MRI showed enhancing lesion involving the right anterior orbit and right sphenoidal sinus and extending to the cavernous sinus (Fig. 1)

MRI after endoscopy and sinus lavage with Amphotericin –B and debridement of mucor (Fig 2.)

Fig. 1. MRI showing involvement of right anterior orbit, right sphenoidal sinus with extension to the cavernous sinus

Fig. 2. MRI after endoscopy and sinus lavage
Within 24 hours, the patient developed a central retinal artery occlusion which gave us a strong suspicion of Orbital Mucormycosis.

On repeat endoscopy, black tarry slough were seen in the sinuses which was sent for biopsy that revealed mucor. Extensive orbital debridement, medial wall decompression & sinus irrigation with lyophilized Amphotericin B was done.

But he developed meningitis and altered sensorium. Disease had spread to the other eye with periorbital edema & proptosis suggestive of Cavernous sinus Thrombosis. Patient ultimately succumbed.

2) A 53 year old female, a known diabetic (uncontrolled) presented with moderate proptosis, complete ptosis, and decrease in vision in the left eye, one week following tooth extraction.

On examination, visual acuity was perception of light. There was complete ptosis, with total ophthalmoplegia and lower motor neuron facial nerve palsy. There was no reaction to light, both to direct and consensual in the left eye. On examination fundus showed a pale retina with cherry red spot. Clinically our first suspicion was mucormycosis.

Endoscopy revealed the presence of black necrotic slough in the paranasal sinuses confirmed by MRI.

3) 58 year old, a well controlled Diabetic with past history of pulmonary tuberculosis came with sudden loss of vision in the right eye of 2 weeks duration with proptosis (22 mm) and total ophthalmoplegia (Fig. 4a) with numbness on the right side of face. Vision in the right eye was Perception of light and in the left eye was 6/6.

Total ophthalmoplegia was present on the right side. Patient was diagnosed to have Orbital apex syndrome and was treated with steroids. He showed no improvement but developed an ophthalmic artery occlusion. Sinus endoscopy showed tar smeared brawny sinus and fungal growth of rhizopus. In the Right eye limited exentration was done but it did not help. Eventually he presented with cavernous sinus thrombosis and died due to pneumonia (flare up of pulmonary tuberculosis) MRI showed mucor involving the sphenoidal sinus. (Fig. 4 b-c).

3) 60 year old female, a known diabetic (uncontrolled) presented with right eye periorbital oedema and sinusitis. On examination her visual acuity was 6/6 in both eyes There was right infraorbital anaesthesia. In consultation with ENT, an endoscopy was done which showed black necrotic areas in the maxillary sinus (Fig. 5). These areas were completely debrided and was sent for histopathological examination which showed broad non-septate hyphae (Fig. 6a).

Patient was immediately treated with Amphotericin – B injection and showed dramatic improvement (Fig. 6b).
Discussion

The clinical presentation of rhinoorbital mucormycosis can be acutely fulminant or indolent. Disease often presents as cold orbital cellulites with ophthalmoplegia and cranial nerve dysfunction. Sudden loss of vision can be due to toxic optic neuritis, central retinal artery occlusion or cavernous sinus thrombosis. A characteristic black eschar, representing tissue necrosis can be seen on skin, nasal mucosa which is always very late. Vascular invasion and occlusion are hallmark features of mucor but again signs that occur too late in the course of this disease.

Early clinical suspicion is mandatory to save the vision. In our experience, any immunosuppressed individual, not necessarily a diabetic (age no bar), presenting with orbital & periorbital edema, chronic sinusitis, infra orbital anesthesia is a Mucor suspect. One need not necessarily wait for tissue biopsy to demonstrate broad, non septate hyphae but can definitely start on empirical therapy with Amphotericin B than to wait for the hyphae growth.

Pterygopalatine fossa is considered the main reservoir for rhinocerebral mucormycosis and extension into the orbit and facial tissues usually follows this route. After proliferation in the nasal cavity, mucor reaches the pterygopalatine fossa, inferior orbital fissure, retroglobal space of the orbit resulting in ocular signs. Facial soft tissues, palate and inferotemporal fossa can be infected through the connecting pathways from pterygopalatine fossa. So the most definite method of treatment of mucor infection is debridement of pterygopalatine fossa.

In early cases just a sinus lavage with Amphotericin –B and good sinus debridement is sufficient for moderate to severe proptosis. Orbital decompression is helpful if lesion is medially situated. Exenteration is done in only severe cases when vision is lost and there is threat to the other eye.

Antifungal therapy without exenteration is currently recommended for initial management of patients with rhinoorbital mucormycosis. Amphotericin-B is the agent of choice given daily and increased gradually to reach a cumulative dose of 2 to 4 grams. Treatment must be continued for weeks to months.

To reduce the nephrotoxicity of conventional amphotericin –B, lipid formulations have been introduced. Lipid formulations allow a significant increase in therapeutic index with decreased toxicities.

Repeated debridement without exenteration and local delivery of amphotericin B have been used in patients in whom the disease was diagnosed early.

The overall mortality is 50-70 % or higher with cerebral involvement. Prognosis depends on early diagnosis and treatment as well as resolution of the underlying metabolic disorder. Exentration is unnecessary, however repeated surgical debridement may be necessary. The optimum duration of treatment depends on therapeutic response.

Orbital Mucormycosis should be differentiated from allergic fungal sinusitis which is a Type-1 hypersensitivity response to inhalation of fungal spores. This is usually seen in immunocompetent patient with history of allergic asthma/chronic sinusitis. Tissue diagnosis shows allergic mucin, Charcoat Leyden crystals (eosinophilic granules) and fungal hyphae (Fig. 7). But there is no fungal tissue invasion and good response to steroids.
MRI shows erosion of frontal bone as the mass erodes into the bone and endoscopy shows the paranasal sinuses filled with greenish material consistent with the allergic mucin.

Points to Remember……..

- Mucormycosis is not seen in diabetics alone but can occur in any immunosuppressed individual.
- Its ocular presentations can vary from loss of vision to proptosis, ophthalmoplegia, infraorbital anaesthesia …..
- One must suspect Mucormycosis if there is any evidence of sinusitis with the above clinical features
- Sinus Endoscopy & MRI are two most important investigative parameters that can help in detecting mucormycosis
- If the clinical features and above findings strongly correlate, it is always better to err towards mucormycosis
- A peep into the pterygopalatine fossa during endoscopy will be of help as it is commonly the reservoir of infection
- Lyophilized Amphotericin –B is better than conventional Amphotericin –B
- Exenteration is always reserved as the last choice

References

Drug Induced Retinopathies

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Case 1

A 40 year old lady with endometrial carcinoma on tamoxifen presented with decreased visual acuity in both eyes of 1 year duration. On examination her best corrected visual acuity in both eyes was 6/12 N8. Anterior segment examination and intraocular pressure of both eyes were normal. Dilated fundus examination revealed golden intraretinal perifoveolar crystals in both eyes (Fig. 1). Optical coherence tomography showed hyperreflective intra retinal deposits at the macula (Fig. 2). Based on the clinical background and the typical findings a diagnosis of tamoxifen crystalline maculopathy was made.

Tamoxifen retinopathy

Tamoxifen is an estrogen antagonist which competitively inhibits estrogen binding to its receptors. Estrogen receptors are present in the retina. Tamoxifen is used as adjuvant therapy of estrogen dependent breast carcinoma after initial curative surgery and it has been reported to decrease incidence of contralateral breast cancer by 35-50 %. The US FDA has approved Tamoxifen to reduce the incidence of breast cancer in women at high risk. Hence an increase in preventive tamoxifen therapy is expected.

Patients may present asymptotically or with decreased visual acuity and color vision. Retinal toxicity with Tamoxifen therapy is dose related. Ocular findings are generally associated with a cumulative dose of 100 g. Even with current low dose regimens(20 mg/day) for more than 2 years subtle cases of crystalline deposition have been reported.

Pathology: Tamoxifen is cationic and cause drug polar lipid complexes which accumulate in lysosomes or may induce retinal injury through complex molecular interactions resulting in oxidative damage. It may act as an antagonist of glutamate transporters in retinal pigment epithelial cells. Tamoxifen leads to an increase in glutamate that intern leads to axonal degeneration observed histopathologically and the crystalline deposits correspond to the degenerative products observed clinically. Muller cell impairment may follow retinal neuron injury and generate atrophy and formation of an intraretinal foveolar cyst. The crystals have a predilection for macula but is also found in the periphery. The predilection of crystalline deposits on the macula in part may relate to its greater blood supply.

Diagnosis is based on clinical history and characteristic findings on retinal examination. Ophthalmoscopy demonstrates golden intra retinal crystalline deposits that may be associated with pigmentary changes of macula and macular edema in severe cases. The intraretinal crystals cluster within the perifoveal macular region and may have an annular distribution with variable density. Crystals appear to be confined to nerve fiber layer and inner plexiform layer.

In a study by Gorin et al of 303 women with breast cancer including women never on Tamoxifen (85), those on Tamoxifen for 4.8 years and then been off the drug for 2.7 years (140) and women on the drug continuously for an average of 7.8 years (78), there were no cases of vision threatening ocular toxicity. Intra retinal crystals (OR 3.58 P=0.178) and posterior capsular opacities (OR 4.03, P= 0.034) were more frequent in the tamoxifen group.
In a study of 63 patients treated with 20mg/day for 35 months 4 were observed to have decreased visual acuity, bilateral macular edema, retinal yellow-white dots, and corneal opacities. These toxicities were reversible after cessation of therapy 5.

Fundus fluorescein angiography (FFA) may show leakage and features suggestive of cystoid macular oedema. FFA leakage can be found even when the Optical Coherence Tomography (OCT) findings of cystoid macular oedema are absent. OCT shows multiple hyper-reflective inner retinal deposits or cystoid macular oedema5. OCT can also reveal foveolar cystoid space with focal disruption of photoreceptor line without increased macular thickening or edema 4.

A decreased photopic and scotopic a and b wave amplitude is noted on ERG (electroretinogram) testing.

The differential diagnosis of Tamoxifen Retinopathy includes idiopathic juxta foveal telangiectasia, cuticular drusen and the other peculiar crystalline retinopathy such as Beitti crystalline tapeto retinal dystrophy, canthaxanthine maculopathy, cystinosis and hyperoxaluria 7. Determination of the level of retinal crystalline deposits and a thorough review of the medical history and medication are essential in distinguishing Tamoxifen retinopathy from other diseases causing refractive retinal lesions 7.

Management: Even with current low dose therapies, yearly ophthalmic evaluation with retinal examination is recommended. Ancillary testing with formal visual field and color analysis have not been established as reliable screening tools. If retinal crystal deposits are observed a fluorescein angiogram should be obtained. Discontinuation should be considered at the first sign of retinal deposits 2. Cessation is strongly recommended if numerous deposits are present or macular edema is noted 2. OCT analysis is helpful in determining the presence and severity of macular edema as well as following its resolution. Crystalline deposits may persist even after cessation of treatment. Visual function and macular edema can improve if maculopathy is not severe.

Prompt reporting of symptoms and yearly ophthalmic examinations are mandatory in patients on tamoxifen to detect toxic effects while these are still reversible.
**Case 2**

A 63 year old male presented with 8 months history of central scotoma in the left eye. On examination his best corrected visual acuity in the right eye was 6/6,N6 and in the left eye was 6/18, N18. Anterior segment examination and intraocular pressure in both the eyes were normal. Dilated fundus examination revealed concentric ring of retinal pigment epithelial atrophy in both eyes (Fig. 3). Digital fluorescein fundus angiography showed two rings of hyperfluorescence with an area of hypofluorescence in between and in the centre (Fig. 4). OCT of the right eye was normal and that of left eye showed foveal thinning (Fig. 5).

**Chloroquine/Hydroxychloroquine Retinopathy**

Chloroquine was first used as an anti malarial drug in World War-II. Currently it is prescribed for the treatment of amoebiasis, rheumatoid arthritis, systemic lupus erythematos and for prophylaxis against malaria. Retinal toxicity with degeneration of retinal pigment epithelium and neuro sensory retina as a result of long term daily use of chloroquine are well described. However most cases of retinopathy have developed when a higher than currently recommended (250 mg/day or 3.5 mg/kg/day) dose was used. A daily dose exceeding 250 mg with a total cumulative dose between 100 and 300 gm is customarily needed to produce toxicity. The mean daily dose (MDD), lean body weight adjusted daily dose (LBWDD) and keratopathy are risk factors associated with chloroquine retinopathy. 

A para central scotoma may be the earliest manifestations of retinal toxicity and can precede the development of any ophthalmoscopic or ERG abnormality. Sub macular pigment stippling with loss of foveal light reflex usually appears on fundus examination before the development of classic bull's eye maculopathy in which a ring of de-pigmentation is surrounded by an area of hyper pigmentation centered on the fovea. Visual acuity decreases when the retinal pigment abnormalities involve the center of the fovea. The peripheral retina can display pigment mottling which in severe cases develop the appearance of primary tapeto retinal degeneration with narrow retinal vessels, optic disc pallor and eventual blindness.

After cessation of chloroquine treatment early subtle macular changes can revert to normal. Although far advanced cases may progress despite discontinuation of the drug most patients remain stable with long term follow up. Chloroquine is very slowly excreted from the body. This prolonged presence may account for the onset of chloroquine retinopathy seen up to seven years or longer after discontinuation. Fluorescein fundus angiography can be helpful in revealing early pigment abnormalities at the macula. There is minimal evidence of damage to the choriocapillaris on fluorescein angiography in the areas of pigment disturbance.

The ERG (electroretinography) and EOG(electro oculogram) may be abnormal early, although the EOG is sometimes supernormal initially. Histopathologic sections demonstrate loss of RPE pigmentation with an accumulation of pigment-laden cells in the outer retinal layers with damage and reduction of photoreceptors. Electron microscopic studies reveal more widespread damage to the retina, especially the ganglion cell layer.

The mechanism of chloroquine-mediated retinal toxicity is unknown. Like the phenothiazines, chloroquine is bound by melanin and concentrated in the RPE and uveal tissues. Possible explanations include inhibition of critical enzymes and interference with the metabolic function of the RPE and photoreceptors.

Use of static perimetry through the vertical meridian with a red test object may be the best method to detect an early paracentral scotoma. These changes usually occur before visible retinal abnormalities and therefore should be performed on follow up examinations. The red Amsler grid is also useful in detecting an early paracentral scotoma and may be substituted for static perimetry.

**Hydroxychloroquine:** Only a few cases of toxicity have been well documented involving decreased visual acuity, paracentral scotoma and bull’s eye maculopathy.

In a retrospective case record study of 1207 patients who took hydroxy chloroquine the incidence of definite toxicity was absent in patients treated with < 6.5 mg/kg/day. They recommended annual screening in patients who take >6.5 mg/kg/day for more than 10 years continuously. In those who take <6.5 mg/kg/day routine screening is not indicated if renal function is normal.
In another study the incidence of hydroxychloroquin related retinopathy in 400 patients treated with recommended dose of the drug for a mean period of 8.7 years was 0.5% \(^{10}\). They recommended annual screening after 6 years treatment. There are case reports of progression of retinopathy even after discontinuation of HCQ \(^{11}\). Screening for chloroquine retinopathy can be improved by using a sensitive color test. Disturbance of the Tritan axis appears to occur first. A normal test result on computerized color testing (Arden) virtually excludes any retinopathy by chloroquine/hydroxychloroquine. The EOG is of little diagnostic value \(^{12}\). Multifocal pattern electroretinogram (mf PERG) can detect early changes in ganglion cells (reduction in PERG and mf PERG responses) and hence useful in detecting early signs of toxicity \(^{13}\). Functional testing of retina with mf ERG (multifocal electroretinogram) shows locally decreased retinal function. Characteristic mf ERG (increased R1/R2 ratio) abnormalities are detected in 50% of patients with a cumulative dose of >1250 g of hydroxychloroquine \(^{14}\). Significant abnormalities were seen with cumulative doses as low as 400 g.

Ocular screening is generally recommended annually in patients who have taken the drug even in recommended doses after 6-10 years.

Reference

Self Inflicted Bilateral Rupture Globe Resulting in Total Permanent Loss of Vision - A Case Report

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Self Inflicted Eye Injuries are a rare but important group of ophthalmic condition. The disease spectrum can vary from simple conjunctivitis to severe trans orbital penetrating trauma. While the act of self enucleation is rare other self inflicted eye injuries may be more common.

We report a case of a young man, an engineer by profession suffering from schizophrenia, who attended our hospital after injuring both his eyes with his own thumb and blinding himself.

Self inflicted eye injuries are a recognized ophthalmic problem in adults with psychological disorder and drug addicts. Self injurious behavior is not a new phenomenon to the human existence, but it is only of late that we began to talk about it. Self inflicted wounds pose a diagnostic dilemma to the examining clinician and forensic specialist. This group of conditions require special attention and insight as they may be due to ulterior motives and have a medico legal significance.

Case History

A 36 year old unmarried male engineer presented to us in the emergency department with history of self inflicted eye injuries and profuse bleeding from both eyes (Fig. 1). He was brought by his father and he gave history of psychiatric illness.

Fig. 1. At the time of admission with self inflicted eye injuries in both eyes

On examination

Scleral rupture extending from 10 o'clock to 2 o'clock position, in the ciliary region concentric to limbus with extensive uveal and vitreous prolapse were noted in both eyes. Right eye appeared to be more damaged. A/B scan showed retinal detachment with vitreous haemorrhage both eyes (Fig. 2 a & b).

General and systemic examination of the patient was within normal limits. Patient was calm, cooperative and well oriented to time and place.

According to the patient he had impulsively ruptured both his eyes with his finger and this act was in response to a sudden urge to escape from his disturbing hallucinations.

We managed the case in consultation with our psychiatry department. As per the psychiatrist this was a case of paranoid schizophrenia with suicidal tendencies.

The purpose of this case report is to describe the circumstances and phenomenology of patients who remove or pierce their eyes or orbits during psychotic illness.
Discussion

Self inflicted eye injuries are a rare but important group of ophthalmic conditions that require close co-operation between different medical specialties to provide the best care to the disturbed patients. The most dramatic and disturbing cases involve deliberate self injury, self mutilation, destruction or alteration of body tissue without conscious suicidal intent which occur in a variety of psychiatric diseases. Majority of self mutilation involves eye enucleation, amputation of limbs or genitals. These self inflicted injuries are followed by relief from anxiety when completed or by frustration when injury was prevented.

Psychiatric theories of pathogenesis for self inflicted behavior include religious and sexual ideation, symbolism, guilt and displacement. Biological theories include disorders of serotonergic, dopaminergic and opiate neuro transmitters. Clinical characteristics of self mutilators include acute or chronic psychosis, drug induced psychosis, other psychiatric conditions and certain organic states. Management of self inflicted eye injury requires close co-operation between the ophthalmologist and psychiatrist as well as other medical specialists to ensure quick resuscitation of the patient, prompt diagnosis and treatment of any injuries and treatment of underlying behavior that led to the injuries.

Eye injuries assume importance as they are vision threatening and of great medico legal significance. One must rule out all possible organic causes before making the diagnosis of self injury. Once concluded however, prompt intervention by a multi –pronged approach of cooperation between doctors can help the patient.

References

7. GH Psychiatry Vol 22 issue 3, pages 215-216
An Unsolicited Guest: Ocular Larva Migrans by Angiostrongylus Cantonensis

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Though eosinophilic meningoencephalitis caused by the nematode, Angiostrongylus cantonensis (Rat Lung Worm) is common, ocular larva migrans has been rarely reported. Here, we present a case of ocular larvae migrans in a patient on treatment for eosinophilic meningitis.

Case History

A 41 year old female gynaecologist who was on treatment for meningitis for one and a half months with no relief of symptoms presented in our OPD with complaints of seeing a floater in the left eye of 3 days duration. She had been admitted with fever, headache and vomiting, and was not responding to treatment. She had a previous history of migraine. All investigations for the cause of meningitis were negative. Cerebro spinal fluid studies for bacteria, mycobacterium, fungi and cultures were negative. HSV PCR was normal. Mantoux test being positive (12 mm) anti tuberculous treatment was started empirically but with no improvement in symptoms. MRI brain was initially normal, but repeat MRI showed ring enhancing lesions in the left cerebellar hemisphere and posterior parietal grey white junction with multiple tiny hemorrhages. Repeat cerebro spinal fluid study showed elevated proteins and 10 % eosinophil like cells with negative TB DNA PCR; cryptococcal antigen or malignant cells. Blood showed increasing peripheral eosinophilia of upto 27 %. She was extensively evaluated for possible occult malignancy all tests of which were normal. With a provisional diagnosis of eosinophilic meningitis, she was started on treatment with IV methyl prednisolone for 3 days followed by a short oral course of prednisolone and 21 days of DEC (Diethyl Carbamazine) but with only transient improvement. At the time of presentation she was on Amphotericin and Rifampicin for amoeboid meningitis which had been empirically diagnosed on seeing amoeboid cells in the cerebro spinal fluid.

On presentation at our outpatient department, the patient’s general condition was poor. There were no focal neurological signs. Kernig’s sign was positive. Her best corrected visual acuity as 6/6 , N6 in the right eye and 6/12 in the left eye. Left anterior chamber showed signs of mild anterior uveitis with 2+cells and flare. Fundus showed vitritis with a dead worm 12 mm long floating in the mid-vitreous cavity (Fig. 1 a & b).

She underwent triamcinolone assisted pars plana vitrectomy by a combined 20 guage and 23 guage technique under local anesthesia. The dead worm was removed in toto by grasping it with an intraocular forceps. Few crystals of triamcinolone acetonide were left behind to ensure that there was no postoperative flare up of the intraocular inflammation (Fig. 2). She was started on a course of albendazole and systemic steroids.

The worm was identified to be an adult male angiostrongylus cantonensis which was later confirmed by getting an opinion from the Centre of Disease Control, Atlanta (Fig. 3).
Discussion

Angiostrongylus, Baylisascariasis and gnathostomiasis are the three predominant parasitic infections associated with eosinophilic meningitis. The causative agents are helminthic parasites, the multicellular metazoans or “worms,” which are typically associated with blood eosinophilia. In contrast, eosinophilia is not commonly seen with the cellular protozoan parasites.

Angiostrongylus cantonensis, the rat lungworm, is the most common cause of eosinophilic meningitis and ocular diseases in South East Asia, the Pacific Islands and Cuba. Hawaii is the only endemic site in the United States. It was first described in 1945 in Taiwan from a boy with suspected meningitis. Human infections are caused by eating infected snails, slugs, or other mollusc intermediate hosts, or other members of the food chain that have acquired the infective larvae by eating these hosts. The larvae migrate to the brain, producing an eosinophilic meningitis. The incubation period is usually about 20 days but can be up to 47 days. The main symptom is severe headache. Other symptoms include
convulsions, vomiting, facial paralysis, neckstiffness and fever. It is accompanied by moderate eosinophilia in cerebrospinal fluid and blood. Eye involvement is by visual impairment, ocular pain, keratitis and retinal oedema. Retinal detachment and intraocular haemorrhage can occur. Paresthesias and ocular palsies are common. There is no specific treatment, but mebendazole has been used. The prognosis is usually favourable, the infection being self-limiting. However fatalities have been reported.

Angiostrongylus cantonensis is a nematode, which utilizes the rat as the definitive final host and the mollusc as the intermediate host. The habitat of the adult worm is in the branches of the pulmonary artery of the rat. The gravid female worm lays eggs into the bloodstream which are carried to the smaller blood vessels of the lungs where they lodge as emboli. The eggs become embryonated and hatch to form the first stage larvae. The first stage larvae migrate through the alveolar walls of the lung and travel up the bronchus and trachea and it is swallowed by the rat into its alimentary canal. The first stage larva is eliminated in the faecal matter of the rat and contaminates fresh or seawater where it can survive for a period of 3 days to 6 days. The intermediate host includes the terrestrial mollusc or land mollusc such as the snail or slug. The first stage larva enters the digestive canal of the intermediate host by piercing the cuticle, moults twice and becomes a larger 3rd stage larva. The third stage larva can survive in the body of the snail for a period 12 months.

Human infection occurs by ingestion of raw vegetables containing the 3rd stage larvae, by ingesting the tissues of improperly cooked infected intermediate host such as the amphibiun snail, the carriers or the paratenic hosts such as the snails, prawns and pigs. Infection can also occur by drinking water contaminated with the infective larvae. The third stage larvae after entering the alimentary canal of man follows the same route as in the rat and reaches the brain. They are unable to proceed further and die there, inciting an inflammatory reaction in the brain and meninges. The eosinophilic response in the cerebro spinal fluid of the infected human beings has been suggested to be due to the metabolic products left behind by the parasite or resulting from death of the parasite in the central nervous system.

Presumptive diagnosis can be made on the basis of the patients symptoms ie fever, meningitis, ocular involvement and severe headache. Eosinophilis in cerebrospinal fluid in more than 95% and peripheral blood in 2/3rd cases is also suggestive. Larvae or young adults can be recovered in the cerebrospinal fluid. ELISA can confirm the diagnosis. Western blot analysis for antibody to 31 kd antigen of Angiostrongylus cantonensis can be done.

Treatment is usually supportive; however levamisole, albendazole, thiabendazole(25 ml/kg 3 times daily for 3 days) or ivermectin can be tried. Symptomatic treatment with analgesics or corticosteriods may be necessary. One study suggested that a 2 week course of prednisolone (60 mg/day) could effectively relieve the headache. Neurological symptoms usually recover completely within 3-6 weeks. The parasite dies and the patient recovers spontaneously, usually without sequelae. The mortality rate is less than 1%. A single infection does not confer permanent immunity and recurrences have been reported.

References
Medical Malpractice law is emerging as a very important area of law. Doctors being sued for negligence on their part was said to be a common occurrence in the western countries. With the era of globalization and increased awareness of people, it is now becoming common in India also. In this context, it is necessary to know the existing law, with respect to Malpractice.

There are various ways of approaching the problem of medical negligence. There are various laws under which the complainant can approach the court (which means that following are the ways in which a Medical Practitioner can be taken to task).

A. Consumer Protection Act

The Consumer Protection Act is a social welfare legislation which was enacted in 1986 in pursuance of the constitutional guarantee to develop and promote the social and economic status of the citizens of India. The main purpose of the Act is to protect the consumers from exploitation by unfair trade practice of the traders, who are supposed to discharge services to the consumers; So far, no such speedy remedy was available to consumers in the Civil Courts. The Act gains much importance because most of the citizens of India earlier had no forum wherein they could get quick remedies against unscrupulous traders and providers of services.

The question of application of the Consumer Protection Act to the medical profession with a view to provide a forum for patients to approach, in order to address their grievances has been a very controversial area. There have been arguments and counter arguments in this regard. The matter was finally settled by the Supreme Court in Indian Medical Associations Vs V.P Shanta case. The Supreme Court in this case ruled out that medical services would come within the purview of the Consumer Protection Act. Hence it becomes important to see as to what would fall under the Consumer Protection Act and the remedies available to the patient and the defenses available to a doctor.

The main issues that have to be dealt with are:

1) Meaning of the term services under S.2(1) (o), Consumer Protection Act and the inclusion of medical services within this term.

2) Meaning of the term consumer.

Before going into what actions would bring a doctor under the Consumer Protection Act, let us examine the judgment of the Supreme Court in IMA Vs V.P Shanta case. The court ruled that medical practitioners, though belonging to the Medical Profession are not immune from a claim of damages on the grounds of negligence. Hence, if a person avails of the services of a doctor, he would become a user of the service and he would have a remedy against the doctor if the doctor has been deficient in his service.

The Court further ruled that the Act permitted the consumer himself / herself, or a group of consumers or a consumer organization to file a complaint in the consumer forum. With respect to government hospitals, the case made a distinction between hospitals where the patients have to pay a consideration for the services availed and ruled out that government doctors would not be covered under the Act, since they do not take any consideration for the services provided by them.

A complaint may be filed by a patient against the doctor for deficiency of services under the Consumer Protection Act if the following conditions are fulfilled.
1) The services should be hired or availed of or agreed to be hired or availed by the patient.

2) The services should have been rendered or agreed to be rendered by the doctor to the patient.

3) The services of the doctor should have been hired or availed of or agreed to be hired or availed of for a consideration.

4) The services of a doctor so hired or availed of or agreed to be availed or hired suffers from a deficiency in service.

5) The services should not have been rendered free of charge or under a contract of personal service.

**Contract of Services and Contract for Service:**

The issue that comes up is whether the service that is rendered by a medical practitioner is a contract for service or a contract of service. If it is the latter, it will not come within the purview of the Consumer Protection Act.

Every contract of service implies same relationship of master and servant and involves an obligation to obey orders in the work to be performed and as to its mode and manner of performance. The court in *Ready Mixed Concrete (South East) Vs Minister of Pensions and National Insurance*, ruled that for a contract of service to exist, the following three conditions are to be met:

1) The servant agrees that in consideration of a wage or other remuneration, he will provide his work and skill in the performance of some service for his master,

2) He agrees that he will subject himself sufficiently to the orders of the master, implying that he is under the master’s control,

3) The Provision of the contract are consistent with its being a contract of service.

The Supreme Court in *Dharangadhara Chemical Works Limited Vs State of Saurashtra case*, held that the primary test for determining if a contract was a contract of service was the existence of the right in the master to supervise and control the work done by the servant not only in the manner of directing what work the servant is to do, but also the manner in which he shall do the work. The correct method of approach as per the court is, having regard to the nature of the work, there was a due control and supervision of the employer.

With reference to the Consumer Protection Act, the *National Commission in Cosmopolitan Hospitals Vs Vasantha Nair case* held that a contract of personal service involves the master-servant relationship which is wholly different from a medical doctor-patient relationship. The Court ruled that it will be totally wrong to call services rendered by a medical doctor to his patients as personal service, coming within the exempted category of service mentioned in S.2 (1) (o), Consumer Protection Act.

**Liability of Doctor under Consumer Protection Act**

A doctor, as mentioned earlier will be liable for action under Consumer Protection Act if the service rendered by him is deficient. The issue is what amount is the deficiency in service. The basic principle involved here to ascertain liability is similar to that under Law of Torts. The doctor, by virtue of his qualification is expected to possess a certain amount of skill and is expected to treat the patient in a manner, in which a similarly placed doctor would. The doctor is required to take due care and caution in exercising his skill. If this has been done, the doctor has sufficiently discharged his duties. He is not required to guarantee complete success. The doctor is expected to exercise a reasonable degree of skill and knowledge. He is not expected to cure all his patients. If a patient dies or is injured due to lack of skill on the part of the doctor, the doctor will be liable to pay damages under the act. The law does not require highest degree of competence from the doctor. He is answerable when he falls below the standard of a reasonably competent medical person or he departs from a normal course.

In *Bolam Vs Friern Hospital Management Committee*, the plaintiff, who was suffering from mental illness, was advised by a consultant attached to the defendant's hospital to undergo electro convulsive therapy. He signed a consent form to the treatment, but was not warned of the risk of fracture involved. There was evidence that the risk of fracture was very low, of the order of one in ten thousand. On the second
occasion, when the plaintiff was being administered therapy, he however sustained fractures. No relaxant drugs nor manual control were used, but a male nurse stood on each side of the treatment couch, throughout the treatment. The use of relaxant drugs would admittedly have excluded the risk of fracture. Among those skilled in the profession and experienced in this form of therapy, however, there were two bodies of opinion. One favored the use of relaxant drugs or manual control as a general practice, and the others thinking that the use of these drugs were attended by mortality risks, confined the use of the relaxant drugs to the case where there were particular reasons for the use. The plaintiff’s case was not such a case. Similarly there were two bodies of opinion on the question whether if the relaxant drugs were not used, manual control should have been used. So also, there were two bodies of opinion whether the patient ought to be warned of the risk of fracture. The jury in this case returned a verdict for the defendants and held that they were not negligent.

However wrong diagnosis itself does not amount to negligence. It has been held that diagnosis is nothing but forming an opinion on examination of the suffering, and from such an examination, the opinion is formed as to the disease from which the patient is suffering. The opinion formed or diagnosed may vary from one medical expert to another. Only on the basis of the diagnosis, is treatment given. Such an opinion cannot amount to negligence or deficiency in service on the part of such a professional.

Liability of Government Hospital

As per the settled law, a government hospital cannot be brought within the purview of the Act. There have been arguments to bring a service within the purview of the Act. There have been arguments that the tax paid by the tax payer is used to pay salaries of the doctors working in government hospitals, but this argument has not been accepted by the Courts.

The Supreme Court in Indian Medical Association Vs VP Shanta, ruling on the medical service under the Consumer Protection Act has held as follows:

1) Service rendered to a patient by a medical practitioner (except where he renders service free of charges to every patient or under a contract of personal service), by way of consultation, diagnosis and treatment, both medicinal and surgical, would fall within the ambit of “service” under Section (2) of the Consumer Protection Act.

2) The fact that medical practitioners belong to the Medical profession and are subject to disciplinary rules of the Medical Council of India or the State Councils would not exclude the service rendered by them from the ambit of the Act.

3) A contract of personal service has to be distinguished from a contract for personal service.

4) The services rendered by a medical officer to his employer under the contract of employment would be outside the purview of service as defined under S.2(1)(o) of the Act.

5) Service rendered free of charge by a medical practitioner attached to a hospital/nursing home where such a service is rendered free of charge to everybody, would not be “service” under S.2(1)(o) of the Act. The payment of a token fee would not alter the position. (Even if incidental expenses are incurred, it does not make the complainant a consumer).

6) Service rendered at a non-governmental hospital/Nursing home with no charge whatsoever is outside the purview of S.2(1)(o).

7) Services rendered at a non-governmental hospital/Nursing Home where charges are required to be paid by the persons availing of such services falls within the purview of the expression “service” defined in the Act.

8) Services rendered in a hospital, where the persons who can afford to pay are made to pay and treatment is provided free charge to other people, comes within the purview of expression “service” defined in the Act.

9) Service rendered by a Governmental hospital free of charge does not come within the purview of the Act.

10) Service rendered by the Governmental hospital, where payment is taken from some people would fall within the definition of the term “service”.
11) Service rendered by a medical practitioner cannot be regarded as free of charge and hence, outside the purview of the term “service” if the charges are borne by an insurance company.

12) Similarly, where as part of the conditions of service, the employer bears the expenses of medical treatment of an employee and his family members dependent on him, the service rendered to such an employee, and his family members by a medical practitioner or a hospital would not be free of charge and would constitute “service” under the Act.

B. Law of Torts:

In law of torts negligence has two meanings.

Firstly, an independent tort and secondly mode of committing other torts such as trespass, nuisance etc. In the latter sense, negligence is carelessness. The willful wrong doer is he who desires to do harm; the negligent wrongdoer is who does not sufficiently desire to avoid doing it. Negligence and wrongful intent are mutually exclusive states of mind.

(a) Tortious Liability:

Liability of medical profession falls mainly under three heads. Namely Criminal, Tortious, Contractual. The thrust of tortious liability is mainly in compensating the victim for the injury and loss suffered by him. It is in the nature of a civil proceeding and a civil court has to be approached to seek the remedy.

(b) Negligence:

Can be defined as the breach of a duty caused by omission to do something which a reasonable man, guided by those considerations which ordinarily regulate the conduct of human affairs would do, or doing something that a prudent and reasonable man would not do. The definition involves three constituents of the negligence.

1) Legal duties to exercise due care on the part of the party complained of towards the party complaining of the former’s conduct within the scope of duty.

2) Breach of the said duty;

3) Consequential damage:

Firstly, there must be “foresee ability of harm”, secondly relationship of proximity between the plaintiff and the defendant and thirdly that is fair, just and reasonable that a duty of care is imposed as a matter of policy.

These features are axiomatic in a doctor-patient relationship.

The standard of care is determined by the Court of law on the basis of standard of care expected from a reasonable man under the same circumstances. The Court will be concerned to decide as a matter of fact whether a practitioner has fallen below the ordinary skill of an ordinary practitioner exercising and professing to have the particular skill in issue.

C. Constitutional Remedy:

a) Article 21 of the constitution – Art.21 of the constitution declares that no person shall be deprived of his life or personal liberty except according to procedure established by Law.

b) Article 226 in High Court. – Power of High Courts to issue certain writs like habeas corpus etc.

c) Article 32 in the Supreme Court-

Which deals with humanism; which puts human interest before anything else

d) Under IPC - Stringent Remedy:

S 336: Whoever does an act rashly or negligently as to endanger human life or the personal safety of others shall be liable to be punished; action can be taken even if no harm follows.

S 337: deals with causing hurt to a person by doing an act so rashly or negligently so as to endanger the person’s life.

S 338: Causing grievous hurt to a person by doing a rash or negligent act.

S 304-A: Causing death by negligence. Whoever causes the death of any person by doing any rash or negligent act not amounting to culpable homicide; shall be punished with imprisonment of either description for a term which may extend to 2 yrs, or fine or with both.

But the good news is, in the year 2004 (4th of August 2004 to be precise ) Supreme Court of India (from
Punjab & Harayana High Court) Justice Y.K. Sabharwal & Justice D.M. Dharmadhikari gave the landmark judgement in the case (Dr. Suresh Gupta- Appellant Versus Government of N.C.T of Delhi & Aurangabad – respondents). It was stated that, every mishap or misfortune in the hospital or clinic of a doctor is not a gross act of negligence to try him for an offence of culpable negligence. It can be termed ‘criminal’ only when the medical man exhibits a gross lack of competence or inaction and wanton indifference to his patient’s safety and which is found to have arisen from gross negligence or recklessness.

On behalf of the doctor, learned counsel referred to section 80 & section 88 of the IPC to contend that in various kinds of medical treatment and surgical operation, likelihood of an accident or misfortune leading to death cannot be ruled out. A patient willingly takes such a risk. This is part of doctor-patient relationship and mutual trust between them.

Section 80 & 88 read as under:

**Sec 80:** Accident in doing a lawful act: nothing is an offence which is done by accident or misfortune and without any criminal intention or knowledge in doing of a lawful act in a lawful manner by lawful means and with proper care and caution.

**Sec 88:** Act not intended to cause death, done by consent in good faith for person’s benefit: nothing which is not intended to cause death is an offence by reason of any harm which it may cause, or be intended by the doer to cause, or be known by the doer to cause or be known by the doer to be likely to cause, to any person for whose benefit it is done in good faith and who has given a consent, whether express or implied, to suffer that harm, or to take the risk of that harm.

Although medical community provides good care to patients most of the time, in Western Countries 40% of doctors are being sued. In our country, fortunately the figure is less than 5%, which is good news. But, in ophthalmology with the newer & newer treatment modalities coming in, ophthalmologists getting sued is also going to increase.

For many doctors, the legal system is an uncharted territory. They have not dealt extensively with the legal process and therefore do not have an in-depth knowledge of the way the system works when facing a suit. Knowing the nature of the game, its rules & strategies can help a doctor avoid losing his or her professional confidence and self esteem to the litigation process and its outcome. A malpractice suit is just business to many lawyers and judges. To a judge with 70 cases on the docket or a lawyer who has been defending malpractice cases for some years, a case is simply part of their jobs. The litigation process is a legal analysis, not a medical work-up. The medical mind works on a different premise than the legal one. It emphasizes independent judgment, the ‘correct’ answer, and scientific evidence which establishes the ‘truth’. In contrast, the legal mind focuses on what is in dispute, either legally or factually. Generally, there is no correct answer or established truth, only disputed facts & differing legal interpretations. The judge interprets the law; jury interprets the facts; and jury determines what is ‘true’.

Many doctors do not know what to do when they are sued by their patients. The idea behind this article is to create an awareness among ophthalmologists on the litigational process and also to make the doctor practice the best medicine he/she can in a compassionate & caring way. Remember that most of our patients understand us; love and trust us. That is why they want us to be their doctor. Many of them won’t sue anyone no matter what happens, as long as you treat them with understanding, respect and provide best of care to them.
Incidence of Endophthalmitis after 20 - and 25 - Gauge Vitrectomy

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Ophthalmology is a continually evolving surgical subspeciality. Just like the cataract surgery techniques that have advanced dramatically in last few decades, the retinal surgery has also undergone an equal advancement in techniques and surgical instrumentations. This has broadened dramatically the spectrum of diseases that could be treated effectively with vitrectomy. It has also resulted in improved postoperative visual functions. In particular, 25 gauge vitrectomy has improved significantly the operative process for surgeons and patients. 25 gauge surgeries permit the use of smaller wounds, which may allow patients to recover more quickly and theoretically may avoid the discomfort sometimes associated with sutures.

The purpose of this study was to assess the incidence rate of endophthalmitis after 25 gauge pars plana vitrectomy and to compare it with the endophthalmitis rate after 20 gauge pars plana vitrectomy. It was designed as a retrospective, interventional, comparative cohort study.

Participants consisted of 8601 consecutive pars plana vitrectomy surgery patients. Surgeries performed at a single institution (Wills Eye Retina Service) between January 1, 2004, and September 1, 2006, were reviewed. Endophthalmitis developed in 1 of 5498 eyes after 20 gauge viterctomy (0.018 %) and in 7 of 3103 eyes after 25 gauge viterctomy cases (0.23 %; P=0.004). Median final visual acuity was counting fingers or hand movements (range, 20/50-no light perception), with comparable results between 20 gauge and 25 gauge endophthalmitis cases.

The authors try to figure out the possible causes for the high incidence of endophthalmitis following 25 gauge vitrectomy surgeries.

According to them the simple fact that 25 gauge wounds are not sutured at the end of the case may contribute to the higher endophthalmitis rates. Ultrasound biomicroscopy studies have demonstrated that it takes up to 2 weeks for complete 25 gauge wound closure to occur.

Another possible explanation for the higher rate of infection relates to the amount of vitreous removed in a standard 25 gauge case compared with a 20 gauge case. Typically, a larger vitreous skirt is left in a 25 gauge case. The extra vitreous may facilitate bacterial adherence, resulting in a potentially larger bacterial load and endophthalmitis.

Lower infusion rates are a feature of 25 gauge vitrectomy, which also may contribute to increased rates of endophthalmitis.

A final potential cause for the increased incidence of 25-gauge endophthalmitis is the substance filling the vitreous cavity at the conclusion of the case. A common feature between all the cases of endophthalmitis reported in this study is that all had a fluid-filled vitreous cavity at the end of the surgery; none had a silicone oil, gas, or air-filled vitreous cavity. It is possible that an air or gas-filled vitreous cavity allows superior wound integrity.

The results of the current study suggest that surgeons should make changes in the current 25 gauge
vitrectomy techniques to reduce the endophthalmitis rate. Potential modifications include altering the wound construction and closure. Second, wounds can be beveled to improve the self-sealing nature of the wound. Third, at the end of the case surgeons can spend a few extra moments checking for wound leaks at various intraocular pressures, in much the same way that cataract surgeons inspect their clear corneal incisions.

Although results reported here suggest a potential difference in this dreaded postoperative complication, the results must be kept in perspective and need to be validated.

If these findings are replicated, the authors hope that further research will attempt to identify causes of the increased risk.

Comparative Clinical Trial of Topical Anaesthetic Agents for Cataract Surgery with Phacoemulsification: Lidocaine 2 % drops, Levobupivacaine 0.75 % drops, and Ropivacaine 1 % drops


Topical anaesthesia for cataract surgery is now a widely accepted and well-established technique in phacoemulsification surgeries as an alternative to retrobulbar and peri bulbar blocks. Because it is less invasive, and eliminates the complications from needle and systemic toxicity, topical anaesthesia has gained popularity among surgeons.

Unpreserved lidocaine is the most frequently used and safest agent in topical anaesthesia. It is short acting and it may be associated with intraoperative/postoperative pain and discomfort. Ropivacaine is a monoamidine local anaesthetic agent with a long acting effect and a great margin of safety. Levobupivacaine the S isomer of bupivacaine, is less cardio toxic than racemic bupivacaine.

Purpose of this study which was conducted at department of Ophthalmology, Baskent University School of Medicine, Ankara, Turkey was to assess the safety and efficacy of topical lidocaine, levobupivacaine, and ropivacaine in cataract surgery with phacoemulsification. 105 patients scheduled for cataract surgery with topical anaesthesia were randomly allocated into 3 groups of 35 patients each to receive eye drops of lidocaine 2 %, levobupivacaine 0.75 %, or ropivacaine 1 % every 5 minutes starting 30 minutes before surgery.

The exclusion criteria were as follows: axial length >26 mm or <22 mm, hypermature cataract, pseudoexfoliation syndrome, iris-lens synechiae, previous use of miotics and/or small pupil, nystagmus, reported allergy to topical anaesthetics, unwillingness to receive topical anaesthesia, and/or poor patient cooperation, i.e., those with dementia or hearing impairment.

No systemic sedatives were given to the patients preoperatively or postoperatively.

Hemodynamic variables including the noninvasive blood pressure (NIBP) value, the results of an electrocardiogram (ECG), and heart rate (HR) were recorded every 5 minutes until the completion of surgery. To assess the pain score, a 10-point scale VPS (verbal pain score) was used. Patients were asked to
evaluate and grade the level of their pain and discomfort during surgery, at the end of the procedure, and 1 h and 24 h after surgery. The patient’s pain score, the level of patient and surgeon satisfaction (from 0 to 10), the duration of surgery, the need for supplemental anaesthesia, and surgical complications were recorded.

An ophthalmologist who was blind to which anaesthetic agents were used performed clinical evaluations of every patient’s VPS score. Patients were discharged 1 h after the procedure following VPS evaluations and these pain scores were repeated at 24 h postoperatively.

According to the results, there was no significant difference in duration of surgery and demographic variables among the groups. At the intraoperative period, end of surgery, and postoperative first hour the mean VPS in the lidocaine group was significantly higher than the others (P<0.01), but no significant difference was found between the levobupivacaine and ropivacaine groups. At incision and 24 h after surgery, it was not significantly different among the groups. Surgeon and patient satisfaction scores were significantly better in the levobupivacaine and ropivacaine groups than in the lidocaine group (P<0.01).

To conclude, topical anaesthesia with levobupivacaine and ropivacaine were safe, feasible and more effective than lidocaine in cataract surgery. Levobupivacaine and ropivacaine provided sufficient and long-lasting analgesia without the need of supplemental anaesthesia for each patient.

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Long-term Efficacy and Visual Acuity following Transscleral Diode Laser Photocoagulation in Cases of Refractory and Non-refractory Glaucoma

E. Ansari, J. Gandheswar
Eye 2007, 21:936-934

Transscleral diode laser cyclophotocoagulation (TSCP) plays an important role in the paradigm of glaucoma treatment especially for uncontrolled or refractory cases with poor visual acuity. Recently it has gained good attention as a modality for treatment of glaucoma cases with good vision and even as a primary surgical treatment in some situation, thereby establishing a broader role for TSCP.

The aim of this study, which was done at Eye department, Royal Glamorgan Hospital UK was to evaluate the long-term efficacy and safety of TSCP for a range of glaucoma conditions with particular emphasis on the long-term preservation of VA in those with ambulatory vision (6/36 or better).

The study was designed as a single center retrospective study where a single practitioner performed all the TSCP procedures. Treatment success was defined as IOP reduction of greater than 30%, with or without topical antiglaucoma medications.

Indications of treatment were inadequate control of IOP despite maximum tolerated medical therapy, allergy to antiglaucoma medications / inability to tolerate medication, patients unwilling to have drainage surgery, and painful, blind eye.

TSCP was performed with OcuLight SLx semiconductor diode 810 nm laser and contact probe G (360 degree, except at 3 and 9 o’clock).

Postoperatively patients were treated with topical Dexamethasone and were reviewed at regular intervals.

74 eyes underwent treatment over a period of 4-30 months. NVG accounted for 54% of patients followed by POAG (31%), CACG and secondary glaucoma (15%). Mean age was 76 years. Each patient received
an average of 30 burns and mean laser power was 2069 mw. Duration of laser was 2 seconds in all the cases. All together, mean IOP was reduced by 43 % from 40.3 to 21.1 mmHg at final index visit. Of all patients, 58 % had a reduction in glaucoma medications and all patients discontinued oral Azetozolomide. More than 30 % reduction was noticed in 75.7 % of NVG cases and in 91.3 % of POAG cases. Complications like ptithisis bulbi and hyphema were noticed in 13 % of patients.

To conclude TSCP can be used safely and successfully in seeing eye, therefore extending the role of TSCP in glaucoma management.

eclipse of July 10, 1945. He noticed that the retinal scars caused by this exposure to intense sunlight were similar to those he was trying to induce by applying heat to the surface of the eye.

“The idea came during a night when I could not sleep. I was afraid I would forget the idea, so I made a note of only two words: ‘light’ and ‘coagulation’, Meyer-Schwickerath was to write about his revolutionary invention.

Over the next several years, Meyer-Schwickerath carried out extensive experiments in an effort to perfect a technique of using light to coagulate retinal tissue, without damaging unacceptably large areas of the retina. He found that wavelengths of light between 400 and 900 nanometers could pass through to the retina without losing energy through absorption or scattering by proteins in the cornea and lens. When this light energy was absorbed by the adjacent pigment epithelium it raised the temperature of the normally transparent retina and turned it white. The area then began reflecting rather than absorbing the light, and coagulation stopped.

He first tried using natural sunlight and developed a complex system of mirrors, lenses and a heliostat, which kept the sun in the optical axis of his instrument despite the earth’s rotation. Apart from the fact that the patient had to be brought to the roof of the Hamburg hospital for treatment, this technique was weather dependent and there was often not enough early morning light in Hamburg.

He therefore turned to other sources of light. At first he tried a high intensity carbon arc lamp but this was far from perfect. The lamp smoked and dropped soot all over the place. Every seven minutes, this hot machine had to be unplugged and opened up to replace the carbon rod.

When he moved to Essen in 1952 he took his invention along and continued to work on his idea. The powerful xenon arc lamp had already been developed in the U.S for cinematography. Meyer-Schwickerath worked with Dr. Hans Littmann of Carl Zeiss Laboratories to develop the very first photocoagulator in 1956. It was a heavy, bulky instrument with a 50,000-watt xenon bulb as illumination and a large projection like an elephant’s trunk that hung over the patient’s face!

Xenon Arc Photocoagulator

However by 1961 quite a few of these were in use all over the world. The xenon photocoagulator was in time replaced by the first ophthalmic lasers but it was Meyer-Schwickerath’s invention of photocoagulation that revolutionised the treatment of retinal holes including those at the macula and of diabetic retinopathy.

In 1959, he published a book on the uses of photocoagulation.

Apart from photocoagulation, his electronic flash photographs of the retina laid the foundation for retinal angiography.

He was an inspiring professor, a gifted lecturer and teacher. In 1985, he retired, having received many laurels including the Gonin and Graefe medals, an honorary member of many ophthalmologic societies internationally. He passed away in 1992.
Irregular astigmatism has been increasingly recognized as an important corneal disease due to the rapid rise in the popularity of LASIK in recent years and a concurrent rise in the number of patients who have had keratorefractive complications. Among LASIK complications, irregular astigmatism is perhaps the most difficult to treat.

The first of its kind, Irregular Astigmatism : Diagnosis and Treatment synthesizes our knowledge and understanding of irregular astigmatism and addresses
There are generally 2 types of irregular astigmatism. The first type occurs on a stable cornea. Example of these include irregular astigmatism due to decentred LASIK treatment, deep lamellar keratitis (DLK), uneven excimer laser ablation or tissue absorption, small optical zone, central island, extreme flat or steep corneas etc. The treatment of irregular astigmatism in these stable corneas aims at correcting the local irregularity on the corneal surface.

The second type of irregular astigmatism arises from an unstable corneal structure. The best example of this type of irregular astigmatism is keratectasia, in which there is weakening of the entire cornea resulting in an anterior protrusion of the cornea resulting in irregular astigmatism. Treatment of irregular astigmatism arising from unstable corneas focuses on the underlying cause of the problem, namely corneal structural weakness, by increasing the corneal strength.

The book begins with Section I which is entitled “Optics, Etiology and Clinical Presentation of Irregular Cornea.” It describes the history and optics of regular and irregular astigmatism. Section II is entitled “Management of the Irregular Cornea.” It comprehensively reviews the current state-of-the-art technologies for the treatment of irregular astigmatism.

With the maturation of keratorefractive technology in recent years, effective treatment for iatrogenic irregular astigmatism is quickly developing. “Irregular Astigmatism: Diagnosis and Treatment” provides everything refractive surgeons, ophthalmologists and optometrists need to know about this important topic.
CME Programmes

STATE CONFERENCES

CME 2008
20th July 2008
Chakrabarti Eye Care Centre, Trivandrum
Venue: Hotel Residency Towers, Trivandrum
Dr. Arup Chakrabarti
Mob: 9946410 540

RIO Alumini CME
27th July 2008
Dr. Rajeevan. P
Mob: 9995356276

AUGUST OPHTHALMOLOGICA
3rd August 2008
Dr. Mahesh G
Mob: 9388467893.

CME on Glaucoma
24th August
Medical College, Calicut
Dr. Bindu N
Ph: 0495 2420772

TACOPSIA 2008
21st September
Dr. Rani Menon
Mob: 9447 284008

NATIONAL CONFERENCES

Indian Intraocular Implant and Refractive Surgery Convention
July 12-13, 2008
Venue: Hotel Taj Coromandel, Chennai
Ph: 914428116233
Email: dragarwal@vsnl.com
Website: http://www.iirsi 2008.com

Vision 2008- 56th TNOA CONFERENCE/ 3rd OASIS
8th, 9th, 10th August 2008
Venue: Regional Institute of Ophthalmology and Govt. Ophthalmic Hospital, Egmore, Chennai
Ph: 91-44-42616625
Mob: 9789984119

IXth Biennial Conference of SAARC Academy of Ophthalmology and IIIrd Indian AMD Congress
22nd- 24th August 2008
Venue: India Habitat Centre, New Delhi
Dr. Namrata Sharma.
Ph: +91-11-26593144
email: sao2008@gmail.com
website: www.sao2008.org

The VIIth International Congress on Advance in Ophthalmology
Eye Advance 2008
22nd- 24th August 2008
International Academy for Advances in Ophthalmology
Venue: World Trade Centre, Cuffe Ponade, Mumbai
email: admin@eyeadvance.com

XVII Annual Conference of Glaucoma Society of India
31st Oct- 2nd Nov 2008
Advanced Eye Centre, Post Graduate Institute of Medical Education and Research, Chandigarh
Dr. S.S. Pandav.
Ph: 0172-2756112, 2747837
email: sspandav@yahoo.com
INTERNATIONAL CONFERENCES

**Argentine Ophthalmological Society Symposium of India Ophthalmological Society**
July 26\(^{th}\) – 29\(^{th}\) 2008.
Buenos Aires- Sheraton Hotel and Convention Centre.

**XXVI Congress of the European Society of Cataract and Refractive Surgeons**
13- 17\(^{th}\) September
Berlin 2008
Tel: +3531 209 110 Fax: +3531209 112
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.
THE CLINICAL SPECTRUM OF WHITE DOT SYNDROME

**INTRODUCTION**
- Broad Spectrum of an Underlying Disease Entity
- Underlying Histopathological Lesion
  - Microgranuloma
  - Presumed Infectious Aetiology
- Acute Onset
- Minimal / No Permanent Long Term Sequelae

**MULTIPLE EVANESCENT WHITE DOT SYNDROME (MEWDS)**
- U/L
- Sudden Onset
- RAPD
- Discrete Dots at RPE Level
- Grainy Macula
- Mild Inflammation
- Occasional Sheathing
- Rarely Reccurs
- Visual Recovery Good
- FFA: Hyper with Late Staining
- ICG: Hypofluorescent Spots
- ERG: a wave & ERP: Reverses with Recovery
- Minimal RPE Perturbation

**MULTIFOCAL CHOROIDITIS & PAN UVEITIS (MFC)**
- B/L; Long Chronic Course
- 28-33 yrs, Moderately Myopic
- Significant Vitritis & Anterior Uveitis
- 50-300 μm Lesions; all over; Punched out; Pigment at Margin
- Mild Disc Oedema
- A/C Symptomatic Blind Spot Enlargement
- CME 14% - 41%
- CNVM: 30%

**SPECTRUM OF WHITE DOT SYNDROMES**
- Multiple Evanescent White Dot Syndrome (MEWDS)
- Multifocal Choroiditis and Panuveitis (MFC)
- Punctate Inner Choroidopathy (PIC)
- Acute Retinal Pigment Epitheliitis
- Acute Posterior Multifocal Pigment Epitheliopathy (APMPPE)
- Subretinal Fibrosis and Uveitis Syndromes
- Acute Zonal Occult Outer Retinopathy (AZOOR)

**MEWDS: DIFFERENTIAL DIAGNOSIS**
- APMPPE: B/L; Larger; Initial Hypofluo; RPE changes
- Multifocal Choroiditis: Severe Inflammation; Long Duration CME; Considerable RPE Perturbation
- Birdshot Retinochoroidopathy: Larger Lesion; Recurrences Vasculitis, CME, Progressive RPE Alterations

**MULTIFOCAL CHOROIDITIS & PAN UVEITIS (MFC): THERAPY**
- PDT to CNVM (Spaide et al: Retinal 22 2002)
MULTIFOCAL ACUTE POSTERIOR PLACOID PIGMENT EPITHELIOPATHY (APMPPE)

- Sudden Onset
- Moderate Visual Blurring
- Photopsia
- Young < 30 years
- B/L: ? Delayed onset in 2nd eye
- AS & Vit Inflamm+
- Cream Colored Plaques of (RPE Level) Varying Sizes
- Multiple Crops (Old & Fresh)

APMPPE PATHOGENESIS:
- Choroidal Vasculitis - Ischaemia (Variable Sized Lesions)
- Secondary RPE Alterations
- Photoreceptor Inv / Slow Recovery of Sensitive Measures of PR Func
- FFA
- Hypofluoresence (Blocked Choroidal Fluorescence)
- Staining of Cream Coloured Lesions
- RPE Transmission Defects in Healed Lesions

APMPPE SYSTEMIC ASSOCIATIONS: Viral Prodrome
- Episcleritis
- B/L CRVO
- Cerebral Vasculitis
- Wagners Granulomatosis
- Erythema Nodosum
- Neuro Sensory Deafness
- HLA – A 29 associated
- Steroids ; Immunosuppressives; iV Inj of Steroid
- Guarded Prognosis: Worsening in 52%
- Deterioration of Retinal Func ; Vasc Alteration as sequelae

ACUTE RETINAL PIGMENT EPITHELIITIS (ARPE)

- Acute Vision Drop; Metamorphopsia
- Young; UL/BL presentation
- Subtle RPE Alterations; Deep Grey spots & yellow halo
- Macular Pigmentary Clumps; CSR
- FFA: Hypo with Ring of Hyper; Lacy Hyper
- Abnormal EOG: Wide Spread RPE Inv
- Spontaneous Resolutions / No Therapy necessary
- Good Visual return

PUNCTATE INNER CHOROIDOPATHY (PIC)

- ? Variant of MFC
- A/C Onset Blurred Vision, Flashes, Paracentral Scotomas
- B/L; Usually seen in moderately Myopic Women
- 100 – 300 micron; Deep Punched Out, Cylindrical Lesions
- Absence of Vitreous Inflammation
- CME: Less Common
- Risk of CNVM

BIRDSHOT RETINOCHOROIDOPATHY

- Women , 3rd - 4th Decade
- DV; Floaters; Poor Night Vision; Defective Colour Discrimination
- Quiet AC; Vit Rn; Vasculitis
- CME (4 %) ; CNVM : Moderate Risk
- Abnormal ERG & EOG
- HLA – A 29 associated
- Steroids ; Immunosuppressives; iV Inj of Steroid
- Guarded Prognosis: Worsening in 52%
- Deterioration of Retinal Func ; Vasc Alteration as sequelae

Compiled by
Dr. Meena Chakrabarti MS DO DNB
Editor

ACUTE ZONAL OCCULT OCULAR RETINOPATHY

- Photopsia; 77% Young + U/L (69%)
- Scotomata (on VF Charts) .
- No Evidence of Inflammation
- Normal Fundus 82% .
- Healing : RPE Atrophy; Vasc Attenuation
- Abnormal ERG Persists
- Legal Blindness 18%
- VA 20/40 (68%)

SUBRETINAL FIBROSIS & UVEITIS

- Rare Entity ; B/L
- Progressive Gross Visual Deterioration
- Moderate AC Rn; Chronic Vit Inflammation
- Glotic / Fibrotic Yellowish White S/R Lesions
- Coalesce to Form Sheets of Fibrotic tissue
- Markedly Subnormal ERG & EOG
- FFA: Blocked Choroidal Fluoresence.
- Staining of Fibrous Lesions
- Role of Steroids, Cytotoxic Agents –Doubtful
- DD : Sarcoid ; Histoplasma, Syphilis, TB etc

AZOOR COMPLEX (GASS et al)

- MEWDS
- A/c Idiopathic Blind Spot Enlargement
- MFC
- A/C Macular Neuro Retinopathy
- PIC
- A/C Anular Outer Retinopathy
AZOOR

SUMMARY

- Range of Disorders
- (Mild Requiring no Treatment to Potentially Blinding Entities)
- ? Spectrum of Same Underlying Disease
- ? Infectious Aetiology
- Necessary to Clinically Differentiate
- Can Predict Visual Sequale to Patient
- Underlying Mechanism Speculated

.......... Only time will tell ..........
GENERAL INSTRUCTIONS TO AUTHORS

The Kerala Journal of Ophthalmology (KJO) is a quarterly, peer-reviewed, journal devoted to the 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.

11. 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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