Attention DOS Member

**DOS Times is not published in the month of May & June each year**

www.dosonline.org
Editorial

My Dear Friends and Colleagues,

The first issue of the new DOS year. A new cover to give our DOS times a fresh look to entice you into reading and contributing.

A year as the secretary of this great society. A tumultuous year, a stormy, turbulent year with its fair share of controversies. And we are faced with a stark question? A dilemma that refuses to quietly lie on the floor, but stares us squarely in the face and makes us uneasy.

What is the role of a society?

Is it only about conferencing. If it is, we are doing fine with our monthly meets and our midterm and the grand gala annual and along with the journal and the dos times and the dost programme. We have all that it takes; if it is just that. But is it? In our hearts in your heart and mine we know that it isn’t and that is what makes us uneasy and guilty. How much of a consensus have we achieved and how much action have we taken on the burning issues facing the Ophthalmologists. The Issues of TPA’s and Ceilings and Day Care Centre and the coming Medical Accreditation Bill. “None”. We are a house as divided as ever and that makes us helpless and defenseless to be used and abused as pawns in the hands of powers that be. We spend so much time and money conferencing and we can’t spend a little time, energy and funds to save our profession and protect our turf.

A Perfect time to act never comes. It always will be late and so the time to act is now.

Ophthalmology beyond Cataract & Lasik.

Live Surgeries on Oculoplasty, Squint, Glaucoma, Retina, Cornea etc. One day before the midterm conference. This is under DOST programme, all are welcome to attend.

MidTerm Conference On 27th and 28th Nov.

Theme: Ophthalmology Techniques and Innovations.


Conference Website will be functional soon for online registrations etc.

Last year we tried to do something new and different. We put DOS on the facebook. Uploaded over a hundred videos from the DOST and the Conferences. The response is mixed, but we have preserved fantastic academic material from the finest minds in Ophthalmology for all of you to see and benefit. If you are still out of the loop, just send me a mail and I’ll send you the invite.

The festival season is here. A Happy Independence Day and Raksha Bandhan to all DOS Members.

Yours Truly.

Thanking you,

Dr Amit Khosla
Secretary,
Delhi Ophthalmological Society

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62nd ANNUAL CONFERENCE OF

Delhi Ophthalmological Society

15th to 17th April, 2011,
Friday, Saturday & Sunday
Ashok Hotel, Chanakyapuri,
New Delhi, India

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TRENDS IN OPHTHALMOLOGY

Please join us in New Delhi
INVITATION

Dear friends & colleagues,

Greetings from Delhi Ophthalmological Society. We wish to invite you to our 62nd Annual Conference from 15th to 17th April 2011 at Hotel Ashok, Delhi. Indian Ophthalmology is highly advanced and Ophthalmology in Delhi is vibrant and alive. Delhi has perhaps the maximum number of Ophthalmologists, the largest number of Ophthalmology training institutes and the largest number of Ophthalmology residents in the world. All subspecialities of Ophthalmology are highly developed and all surgeries-classic and recent advances are routinely performed.

The Delhi Ophthalmological Society is the Largest State Society in India with over 5000 members and The Annual Conference of our society is a 3 day celebration of Ophthalmology-live surgeries and wet labs, workshops, instruction courses and free papers showcasing original research work. Ophthalmologists of International repute participate in this conference. This year our theme is "Trends in Ophthalmology" and we will be abundantly pleased to welcome you to our conference and our city.

Delhi, the Capital city of India is a modern metropolis with all the world class facilities and a grand historical legacy. A brand new airport, comparable to the best in the world awaits your arrival. Wide roads and swank radiocabs provide excellent connectivity. The Delhi Metro Rail is fast, clean and punctual and there is a fast express link from the airport to the heart of the city. Delhi's air is clean and highly breathable. It is one of the greenest cities in the world. Your stay will be extremely comfortable. Our hotels are among the world's best and provide unmatched service. Indian cuisine is appreciated and duplicated the world over and Delhi Restaurants will provide you with a memorable culinary experience and the taste of India in Delhi.

The comfortable and Airconditioned Shopping Malls in and around Delhi always have a festive atmosphere and your shopping experience is bound to be incredible. In addition there are the traditional local markets like Karol Bagh and Chandni Chowk to provide you with the local flavour. Connaught Place in the heart of Delhi is a huge commercial centre built in a circular fashion. It was built by the British and is an icon for the city. There are number of theaters playing the classical and latest movies from Hollywood and Bollywood. The Golden Triangle Tour (Delhi-Agra-Jaipur-Delhi) offers you with an opportunity to witness the great Indian heritage.

I wish forward to welcome you to Delhi in the pleasant month of April.

Yours truly

Dr. Amit Khosia
Secretary, Delhi Ophthalmological Society

Highlights

- Live Surgery
- Instruction Courses
- Scientific Sessions
- Video Assisted Courses
- Video Stations
- Film Festival
- Meet the Masters
- Free Paper
- Trade Exhibition
- Early Bird Prizes
- Gala Dinner
- Yoga
- Meditation

Submit your registration, faculty form & abstract online for the Annual DOS Conference at www.dosonline.org
PHTHALMIC

Techniques and Innovations

27th & 28th November, 2010
India Habitat Centre, Lodhi Road,
New Delhi
Cataract surgery in India, presents at any given time, a linear panorama from ancient, through medieval to most modern techniques. Manual SICS fits in at all levels of development and is a very precise surgery, for primary to the most advanced level of patient care. Manual SICS has fewer complications, is an easy and NO additional cost option, and has arguably the same objective as phaco emulsification. This technique has a relatively hazard free, smoother and easier learning curve; rather, there is NO learning curve for one who is already into ECCE and implant surgery. There is not a single indication that one can think of, for Phaco or conventional ECCE surgery, which could be labeled as a contra-indication for Manual SICS. In addition to the superior approach, Manual SICS can be performed as a routine, through the temporal or supero-temporal, sclero-corneal or limbo-corneal approach as well, depending upon the astigmatic status or in patients having a trabeculectomy bleb or a deep set eye.

Manual SICS is a revolutionary technique that has brought the benefits of small incision, IOL implants and anastigmatism to the Primary and Secondary eye surgery centres of rural & semi-urban India, more so, because a phaco machine and all the paraphernalia that goes with its instrumentation, sterilization, repair and replacement are a costly luxury for small clinics. Learning and mastering Manual SICS is a MUST, even for a Phaco Surgeon, for times when he has to CONVERT, due to Phaco machine failure, Posterior capsular rupture, Zonular Dilation, Hard black or brown cataract, or having used too much phaco energy. It is also ideal for teaching institutes, tertiary centres in the Government sector, or wherever finance is a constraint. The charm of Manual SICS is its simplicity and good results.

(KPSM): Dr. K.P.S. Malik, MS, DOMS, MNAMS, FICS, Head of the Department of Ophthalmology & Additional Director General Health Services, Safdarjung Hospital, New Delhi.

(DB): Dr. Debasish Bhattacharya, MS, Founder Chairman & Senior Consultant of Disha Eye Hospitals, Barrackpore, Kolkata, West Bengal.

(PK): Dr. Philip Kuruvilla, MBBS, DOMS, Aradhana Eye Hospital, Fort, Trivandrum

(RG): Dr. Ruchi Goel, MS, DNB, FICS, Associate Professor, Guru Nanak Eye Centre, Maharaja Ranjit Singh Marg, New Delhi

(AK): Dr. Arun Kshetrapal, MS, Kshetrapal Eye Hospital and Lasik Laser Center, Kutchery Road, Ajmer, Rajasthan

(RSD): Dr. Ranjit S. Dhaliwal MD, DOMS, Eye Infirmary, Nabha, Punjab

(RSD): At the outset, any one of you are doing only Manual SICS? Or do you do both -Phaco & Manual SICS?? And when??

KPSM: I do both phaco and SICS. Phacoemulsification in institute hospital and manual SICS in my rural community work. I do SICS for all grade 4+ and suprahard cataracts for preserving the corneal endothelial cells.

DB: Both Phaco and Manual SICS.

Phaco as the surgery of choice and Manual SICS for people who cannot afford. Also if 3 of the 4 factors like suprahard cataract (age 70 years), small pupil, bad endothelium or weak zonules are present I would recommend SICS surgery.

PK: I used to do phaco and SICS 10 yrs ago; but I gave up phaco as I found SICS a better technique in my hands. I do only SICS now.

RG: I do both. In camps it is 100% SICS. In a tertiary care centre where we have best of the phaco machines, I do 98% phacoemulsification and perform SICS in patients with rock hard nucleus with compromised endothelium, extensive corneal opacity etc.

AK: I do both Manual SICS and Phacoemulsification. In cases of compromised media clarity (corneal degeneration/ corneal opacity) or in cases of non-dilating pupil, I prefer to do Manual SICS. I also do Manual SICS whenever the nucleus is very hard.

(RSD): Pre-op. in Manual SICS - a Hypo, Hyper or Normo-tensive globe? What is your preference, what do you have to say and how do you achieve it??

KPSM: I shall prefer a Normo-tensive eye. It is easy to make side ports, insert ACM and dissect tunnel in Normo-tensive or hypertensive state. If I found that the tension is low, I inject OVD in AC to achieve desired ocular tension.

AK: I do both Manual SICS and Phacoemulsification. In cases of compromised media clarity (corneal degeneration/ corneal opacity) or in cases of non-dilating pupil, I prefer to do Manual SICS. I also do Manual SICS whenever the nucleus is very hard.

DB: Normo-tensive globe. Gentle peribulbar block with pinky ball application.
PK: I prefer a hypo or normo tensive globe. I apply pressure after block with weight over the eye. Injecting Visco through a side port makes the globe tense enough before making the incision.

RG: Preoperative Normo-tensive or slightly hypertensive state in preferred. No pressure is used even if peribulbar block is given. If the eye appears to be soft then a side port is created and visco-elastic is injected before the tunnel dissection is started.

AK: I prefer a normotensive globe or a slightly hypotensive globe. If you have new and sharp blades a normotensive globe is good enough for making good incision. A normotensive or slightly hypotensive globe also facilitate manipulations in AC. Some times when the globe is hypertensive the Iris Lens diaphragm tend to bulge forward making capsulorrhexis difficult. Chances of posterior capsular rupture during cortical cleaning are more in a hypertensive globe. Hypotensive globe is achieved by placing a soft cloth bag filled with steel ball bearings (the total weight being around 150 to 200 gms) over the globe for 5 to 10 min before surgery.

(RSD): What is your preferred approach in Manual SICS – superior, temporal or supero-temporal? And why??

KPSM: Superior always. Since manual SICS requires conjunctival and scleral dissection, the redness so produced shall be covered by upper lid. Eye is cosmetically white and normal looking on day one. My incision are astigmatically neutral near neutral. Any pre-resisting/post-op astigmatism over 1.0D, I manage with incisional keratotomy. Temporal site is unsightly, unstable and prone to fish mouthing or infections.

DB: Superior with a slight right hand shift of the incision. The incision is covered by the lids and the right hand shift is for the ease of the operation. Should there be a pre-op astigmatism more than 1 Dcyl x90°, I would give temporal incision.

PK: Depends on the preop K reading. I make the incision in the steepest meridian. i.e, temporal, if it is against the rule astigmatism and vice versa. If it is a spherical cornea to begin with, I make a superior incision as surgically induced astigmatism is less than 0.5 D

RG: I usually practice superior approach, as it hides the area of conjunctival dissection under the lid providing better cosmesis and safety against infection. In case of astigmatism >ID I place my incision along the steep meridian.

AK: Normally I prefer to make incision on the steeper axis. In case both the meridians are equal and the difference between them is less than 0.75 diopter, I would prefer a superior or a supero-temporal site. I prefer this site as superior part is covered with the lid and this will hide the redness which may sometime occur at the site of incision in immediate post-operative period. However priority is to be given to the steeper meridian. I prefer temporal site if a filtration bleb is present superiorly.

4. What is your preferred placement of the incision in Manual SICS - Scleral, Clear Corneal, or Limbal? And why?? How far back, if scleral - 1, 1.5, and 2 mm from limbus blue white junction??

KPSM: Always scleral, nearly 2 mm from limbus at 12 O'clock both end curving upward in frown shape within astigmatically neutral funnel. The horizontal width of the incision should be 5 - 5.5mm.

DB: A scleral incision 1.5mm behind the limbus.

PK: I make the horizontal part of the incision at the blue white junction on the limbus

RG: My preferred external incision is scleral, 2mm from limbus, frown shaped, 5-5.5mm in size with backward extensions at its edges.

AK: I prefer a scleral incision which is placed about 1.5 to 2 mm behind the limbus. Scleral incision is likely to produce less astigmatism than an incision of same length at limbus or clear cornea. Furthermore the scleral incision will be covered by the conjunctiva at the end of the surgery and there is less chances of endophthalmitis as compared to clear corneal incision which is exposed and could also leak.

(RSD): What is your preferred type of the incision in Manual SICS - straight, straight with radial cuts, smile or frown? Why, and do you also prefer making scleral pockets??

KPSM: Frown with radial extensions because it holds up the tunnel like a hanging bridge supported by side strings. It won't slide down and won't produce against the rule astigmatism (ATR). Scleral pockets are a must in my surgery to accommodate any size of nucleus making external incision small (5-5.5 mm), more over it has been proven that more the area of the tunnel lesser the astigmatism.

DB: Frown incision with scleral pocket to accommodate the thickness of the Suprahard Cataract nucleus which will not mould.

PK: A straight 3 mm incision with radial cuts and scleral pockets.

RG: I make side pockets as any technique where nucleus is delivered as a whole or debulked at the exit side pocket dissection is a must to ensure engagement of nucleus within the tunnel. Side pockets are not required if the nucleus is divided within the anterior chamber like phaco section.

AK: I prefer a frown incision which is about 1.5 to 2 mm away from the limbus at the center and about 2.5 to 3 mm at the ends. I usually do not make scleral pocket but make my incision funnel shape i.e. inner opening is about 2 mm wider than the outer opening. This helps in the engaging of nucleus in the tunnel.
RG: Even the hardest of the nucleus can be delivered with PK: Even for a hard nucleus, I do not need more than 5 mm DB: The incisional size will depend on the size of the hard KPSM: Usually 5-5.5 mm. In exceptionally hard and very large (RSD): What are your preferred dimensions of the incision AK: I usually make temporal incision about 1 to 1.5 mm RG: My dimensions depend on nuclear hardness and not on PK: When I do a temporal incision, it is to correct a pre DB: 6.5mm frown scleral incision, 1.5mm behind the limbus for (6, 7, or 8 mm or more) to be used in case of a hard nucleus? And why?? KPSM: Usually 5-5.5 mm. In exceptionally hard and very large DB: The incisional size will depend on the size of the hard nucleus, scleral pockets will take care of the thickness. Generally, I would do 6.5mm external incision which would stretch and corneal internal incision is carried down to 3 or 9 o’clock. So we have at least an 8/8.5mm internal incision with a safe corneal valve that does not slip into the limbus. PK: Even for a hard nucleus, I do not need more than 5 mm incision- I mean the horizontal part – then back cuts are there on either ends. RG: Even the hardest of the nucleus can be delivered with external 5.5mm incision as once it is engaged in the tunnel, I debulk it at its exit by chopping of pieces from the presenting portion, push it back and re-engage the smaller diameter for exit from the tunnel. AK: I prefer to enlarge my incision up to 6.5 to 7.00 mm depending on the hardness and size of the nucleus. It is recommended to take out nucleus through an optimally sized tunnel rather than forcefully prolapsing it out through a small tunnel. If a big sized nucleus is prolapsed through a small tunnel you will need to raise intraocular pressure too high to push that hard nucleus through the small tunnel. Raising the intraocular pressure too high may be detrimental to the retinal circulation and to the disc especially in those who already have compromised disc circulation (glaucomatous disc). If a large sized nucleus is pushed out through a small tunnel, it will come out with a jerk and immediately after the prolapse of nucleus the iris may also prolapse out due to high intraocular pressure or the fluid can seep into the vitreous through the zonules causing vitreous bulge and shallow chamber. The size of the incision should be such that the nucleus can be prolapsed out gently. In some cases I even enlarge the tunnel to 8.00 mm and then later suture it for stability. (RSD): Do you make side port/s? If yes, one or two?? And what are the steps you perform, through the side ports?? KPSM: I always make 2 side ports, one at 11 o’clock and another at 7 o’clock in RE and at 5 o’clock in LE for ACM. I use 11 o’clock side port for capsulorrhexis, dialing of IOL in bag, for injecting visco-elastics and for olive tip cannula in bimanual irrigation aspiration (ACM and olive tipped cannula). DB: One side port on the right-hand side which is used mainly for sub incisional cortical cleaning and also for nuclear rotation and anterior chamber tightening. PK: 2 side ports. One for AC Maintainer . The other for subincisional cort wash if needed. RG: I make 2 side ports, one at 10 O’clock and other at 7 o’clock (right eye) and 5 o’clock (left eye). Through the 10 o’clock port, I perform CCC, inject and wash dye, inject visco, dial the IOL, perform cortical aspiration. The other port is used for ACM insertion. AK: I usually avoid making side ports. However if the chamber is shallow, I prefer to make a side port to perform capsulorrhexis. If the pupil is non-dilating I would prefer to make 2 side ports 180º apart for bimanual irrigation aspiration. (RSD): Do you use the AC Maintainer? Why?? Or why Not?? KPSM: Always use the AC maintainer. AC maintainer is life line for (i) keeping eye normo-tensive or hypertensive all the time, (ii) preserving the vascular dynamics of intraocular tissues (choroid and retina), (iii) preventing the post-op CME. I use visco-elastic in the ACM, which coats the endothelium and protects it always. ACM keeps chamber deep allowing you to manipulate nucleus without hurting the posterior capsule or corneal endothelium. DB: I don’t use an AC maintainer because I am used to the irrigating vectis technique. This seems to be more convenient because the vectis guides the hardest nucleus through a right sized incision where as an AC maintainer would require a bigger incision to possibly achieve this. PK: ACM for 100 % of cases. Keeps IOP + ve most of the time; aids in nuclear delivery; washes debris out of AC; aids in cortical wash. Etc etc. etc. RG: I use ACM because it allows nuclear delivery through a very small incision with minimum manipulation inside the anterior chamber.

I. The fluid continuously washes out the anterior chamber contents ensuring good visibility. It is especially useful in milky cataracts.
II. The anterior chamber is kept deep all the time and the need for repeated injection of viscoelastic is obviated.

III. Allows easy aspiration of 12 o'clock cortex keeping the posterior capsule safely behind.

AK: I have never used an AC maintainer. I learned doing SICS way back in 1995 and during that period AC maintainer was not available. So I developed my technique without AC maintainer and then never really felt the need to use it.

(RSD): What anaesthesia, do you use in Manual SICS? And why??

KPSM: Peribulbar by and large. In willing and cooperative patient I do SICS under topical or 1cc subconjunctival block at 12 O'clock.

DB: Peribulbar anaesthesia because the incision is slightly more traumatic than Phaco.

PK: Peribulbar; topical, if the patient insists on a non injection technique (after putting drops I painlessly give a subconjunctival injection of Xylocaine without telling the patient !)

RG: Topical (2% Xylocaine jelly) with subtenon in cooperative patient otherwise peribulbar.

AK: Usually prefer a peribulbar block. A cocktail of 3.5 ml Xylocaine and 1.5 ml of Bupivacaine along with hyaluronidase is injected along the inferior orbital margin at the junction of lateral 1/3rd and medial 2/3rd. I feel that with topical anaesthesia there is always some degree of patient discomfort. Whenever the patient is in discomfort, the surgeon is also in discomfort.

(KPSM): Capsulorrhexis for its inherent advantages of centered IOL, and safer irrigation and aspiration. I do make comma shaped cuts sometimes in rhexis margins if nucleus is very big.

DB: Big capsulorrhexis because it preserves the sulcus for the IOL placement in case of PC disturbance.

PK: Capsulorrhexis for its inherent advantages of centered IOL, safer irrigation and aspiration. It obviates the need for repeated injection of viscoelastic.

RG: Capsulorrhexis almost always as it ensures safety in rest of the manipulations. But the size of the rhexis should be large enough for nucleus to be prolapsed out of the bag otherwise one can land up doing an ICCE. Also a good hydrodissection is a must to avoid cortical entrapment especially at 12 O'clock.

AK: I always prefer a capsulorrhexis. The size of capsulorrhexis has to be adequate so the nucleus can be prolapsed out comfortably. Usually most of the nuclei can be prolapsed out through 5.5 mm capsulorrhexis. In case the nucleus is big, a slightly bigger capsulorrhexis will be required. However if at any stage I feel that the nucleus will not prolapse out through the capsulorrhexis I do not hesitate in giving relaxing incision in the capsulorrhexis margin. Prolapsing out a large sized nucleus through a small capsulorrhexis opening can be detrimental. It can lead to zonular dialysis or even intracapsular cataract extraction.

PK: I prefer Visco expression of the nucleus. Once the nucleus is in AC, I inject some viscoelastic between the nucleus and the endothelium. Then I inject viscoelastic underneath the nucleus to push the posterior capsule back. Now I take my cannula under the nucleus to six o'clock position and start injecting viscoelastic. At the same time I press on to the posterior lip of the tunnel.
With increasing pressure of viscoelastic inside the AC the nucleus is pushed towards the tunnel and finally out of the tunnel. Important point to be stressed here is that the posterior lip of the tunnel has to be pressed so that the nucleus gets engaged in the tunnel. There is no point in injecting viscoelastic until the nucleus is engaged in the tunnel. If the nucleus is not engaged properly in the tunnel the viscoelastic will simply come out of the tunnel. I do not prefer to use vectis or snare as I feel that they are bulky and they increase manipulation inside the AC and there is more chances of injury to structures of the anterior chamber. I sometimes make use of Fish Hook. I use a fish hook whenever I am not able to engage the nucleus properly into the tunnel due to faulty tunnel design or any other reasons. Fish hook is also very helpful whenever one is not able to build up pressure inside the AC for expulsion of nucleus. One must remember that all the techniques employ force to push the nucleus out of the AC through the tunnel by increasing the pressure inside the AC. It is only the fish hook which uses pull out technique and there is no rise in intraocular pressure during removal of nucleus.

(RSD): Would you like to cart wheel the nucleus into AC or flip it out? Why?? When??

KPSM: In deep AC, I generally will scoop out nuclear edge through 11 O’clock side port with the dialer and then cart wheel it out of bag. I would switch off the ACM and inject visco through the side port during this maneuver.

DB: Cart-wheel the nucleus into the AC after hydro procedures which is essential for irrigating vectis technique.

PK: I hydro dissect, one part of the nucleus pops into the AC, rotate it out of the rhexis with 2 dialers – ACM running all the time. If one pole of the periphery of the nucleus does not pop into the AC when I hydrodissect and hydrodelineate, it means that either the rhexis is too small or there are posterior synechiae and cortical capsular adherions : warnings to be extra careful.

RG: A hard nucleus needs to be cart wheeled out to avoid damage to posterior capsule and rhexis margin. Smaller ones tend to prolapse with hydrodissection itself.

AK: I usually like to flip out the nucleus through the capsulorrhexis opening. If the nucleus is hard or bigger then the capsulorrhexis margin I would like to cart wheel the nucleus because flipping a big or a hard nucleus through capsulorrhexis opening can lead to zonular dialysis. Prolapsing the nucleus by cart wheel method is gentle on the zonules.

(RSD): What should be sequence for the sites of injection of the Visco during Visco-expression of the nucleus? 3 O’clock, 6 O’clock, 9 O’clock, above it or below it? Why??

KPSM: My visco expression is visco through ACM. I also use visco in front and behind the lens when it has been delivered in to AC out of bag (My ACM is off this time).

DB: I don’t practice Visco expression. Usually it is at 6 o’ clock.

PK: I don’t visco expression to take out the main nucleus. But sometimes bits of nucleus remain in AC after the main chunk is pulled out by sandwich. Then I visco express by putting in more visco through the main wound.

RG: If the nucleus is hard, I instruct my assistant to inject the viscoelastic through the ACM. This helps in pushing the nucleus towards the tunnel and protecting the endothelium from injury at the same time. For this purpose I use only 2% methylcellulose. We need to ensure adequacy of the size of the internal incision before the visco is pushed through ACM. In extremely hypotonous eye or high myopes, viscoelastic facilitates engagement of the nucleus in the tunnel.

AK: I have answered this question along with question no. 12.

(RSD): Are there any steps in Manual SICS (Blumenthal Technique), during which AC Maintainer should be turned off? Why??

KPSM: As detailed earlier ACM is generally used while delivering the nucleus by visco pressure or hydro pressure. It is also on during bimanual irrigation and aspiration, CCC and nucleus prolapse in the AC.

DB: I don’t use AC maintainer but it should be turned off during expression of nucleus from the bag and maybe even during hydro dissection.
KPSM: I use only iris repositor or lens glide under the lens to give a little scleral pressure backwards for delivery of nucleus.

AK: Whenever I feel that there is slippage of anterior flap of tunnel over the posterior flap, I do not hesitate in taking sutures because even though such a tunnel may be water tight it will induce lot of astigmatism. It is always a good idea to take suture in large tunnel or whenever the tunnel is leaking. I usually use 10.0 nylon sutures in the figure of eight to secure the tunnel.

(RSD): Would you like to pull on the superior rectus bridle suture, to apply counter pressure along with scleral depression, to assist in the nucleus and epinucleus delivery, or apply counter pressure with another instrument or swab – stick?

KPSM: I use only iris repositor or lens glide under the lens to give a little scleral pressure backwards for delivery of nucleus.

AK: I do not use AC maintainer.

DB: I would prefer to hold the globe with a good fixation forceps for counter pressure and orient the globe slightly downwards.

PK: No.

RG: No I do not pull on anything while using Blumenthal technique. A bridle suture does facilitate nuclear delivery. In Blumenthal technique, the nucleus moves out by hydro/visco pressure generated through ACM and pressing the scleral lip guides it out.

AK: The only counter pressure I apply is on the posterior lip of the tunnel with my visco cannula while injecting the visco elastic into the anterior chamber while prolapsing out the nucleus. Sometimes (very rarely) I would request my assistant to pull on to the bridle suture when the nucleus has been engaged in the tunnel to assist prolapsing out of the nucleus.

(RSD): In smaller pupils, do you think ECCE offers any advantage over SICS?

KPSM: No. Small pupil have to be enlarged for nuclear delivery out of the bag irrespective of ECCE or SICS.

DB: I don't think so; small pupils can be much better managed in close chamber condition by pupillary stretch, visco tamponade or Iris hooks.

PK: No.

RG: No, in small pupil, SICS is perfectly safe and ECCE offers no added advantage.

AK: I prefer SICS even in small pupil and feel that ECCE offers no added advantage in smaller pupil.

(RSD): Any preferences for the type of Visco you would like to use? And why??

KPSM: I use cohesive visco for CCC in very hard hyper mature cataract otherwise cheaper dispersive visco are good enough for SICS. In fact dispersive visco adhere to back of cornea for longer period and give better protection than costly cohesive viscoselastics.

DB: This is a cost effective surgery

PK: 2 % methyl cellulose. But I stick to one or two companies which are ‘reliable’ (meaning – I have found that product OK by past experience.) I do not experiment on new companies. I have found no difference in results when I have used costlier visco elastics. I get a clear cornea day one and there is no iritis with methyl cellulose.

RG: Though a lot is being discussed about use of chondritin sulphate as the best visco dispersive agent, it is far too expensive to be used routinely 2% Methyl cellulose gives good results in most of the cases. In patients with shallow AC, compromised endothelium and extremely hard nucleus I use sodium hyaluronate.

I prefer to use HPMC. It is cheap, readily available and offers adequate endothelial protection and maintains the AC well during capsulorrhexis. If it is removed
meticulously after the surgery there are very rare chances of any post-operative uveitis.

(RSD): **What are the common complications do you encounter in SICS?**

KPSM: SICS procedure has been friendly to me for many years now. I misjudge size of rhesis vis a vis nucleus especially in hard cataract and produce zonular dehiscence sometimes. Most common complication I face in patients over 90 years, with brown black 10-12 mm nucleus is atonic iris which runs to tunnel first before the nucleus. As a result small to large iridodialysis takes place which has to be anchored back in tunnel lip. Judicious and well planned extension of tunnel can save us from these complications still maintaining the valvular tunnel.

DB: Zonular dialysis – the Rhesis is small, PC Rent – during cortical cleaning and a once in a while iridodialysis and descemet’s stripping.

PK: Rarely PC rent. If there is preop pathology, I get striate keratitis, flare up of iritis, etc.

RG: The worst thing to happen is a poorly constructed tunnel. Poor quality or blunt knives are the cause for this. If rarely there is a premature entry, iris chaffing & superior iridodialyses can occur.

AK: Common complications which I encounter are premature entry into the AC, Descemet’s membrane detachment and sometimes zonular dialysis.

(RSD): **Which is your preferred IOL, which you use as a routine in your SICS cases? And why??**

KPSM: I use foldable in most of SICS patients these days providing advantage of foldable material and design of lens, minimizing or delaying the posterior capsule opacification.

RG: I prefer using acrylic aspheric with square edge foldable IOLs due to the advantages they offer in regards to image quality and decreased PCO rates.
In this cosmetically oriented world, the inability to retain an ocular prosthesis remains a psychologically devastating problem for the patient. Contracted socket is defined as the shrinkage and shortening of all or a part of orbital tissues causing a decrease in depth of fornices and orbital volume ultimately leading to inability to retain prosthesis. Various techniques have been described to deepen the superior and inferior fornices using a combination of different grafts and conformers depending on the grade and the type of contraction. Guibor has classified clinically contracted socket into 4 morphological types namely, anophthalmic, ophthalmic, microphthalmic and hypoplastic. The hypoplastic socket, requires bony expansion in addition to soft tissue stretching. This may be achieved by using bone grafts or tissue expanders.

Factors leading to socket contracture

**Etiology related**
- Alkali burns
- Radiation therapy

**Surgery related**
- Fibrosis from the initial injury
- Poor surgical techniques during previous surgeries—enucleation/evisceration with extensive dissection of the orbital tissue
- Excessive sacrifice of the conjunctiva and tenons capsule

**Site related**
- Poor vascular supply
- Severe ischemic ocular disease in the past
- Cicatrizng conjunctival diseases
- Chronic inflammation and infection

**Implant and prosthesis related**
- Implant migration
- Implant exposure
- Not wearing a conformer/prosthesis
- Ill fitting prosthesis

Characteristics of a contracted socket

Whatever the initial insult is, the ultimate outcome is extensive loss of conjunctival surface area, deep cicatrix formation, atrophy of the orbital fat, fornix contraction and volume redistribution leading to post enucleation syndrome (superior sulcus depression, pseudoptosis of upper lid and ptosis of lower lid)

**Classification of contracted socket**

**Gopal Krishna classification**

**Grade 1:** The socket is characterized by a shallow lower fornix or shelving of the lower fornix. In this case, the lower fornix is converted into a downwards sloping shelf that pushes the lower lid down and out, preventing retention of an artificial eye. (Figure 1)

**Grade 2:** Loss of the upper and lower fornices

**Grade 3:** Loss of the upper, lower, medial, and lateral fornices

**Grade 4:** Loss of all fornices and reduction of the palpebral aperture in horizontal and vertical dimensions

**Grade 5:** In some cases, there is recurrence of contraction of the socket after repeated trials of reconstruction

**Another classification**

Mild includes grade I and II where only one fornix is involved and there is shortening of the posterior lamella of the lids.

Moderate includes grade III where both superior and inferior fornices are involved.

Severe comprises of cases in which all fornices are involved along with phimosis of palpebral aperture.

Malignant contracted socket is the most severe variety of contracted socket resulting from severe trauma or multiple surgeries.

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**Figure 1: Grade 1 contracted socket**
In the study conducted by Gopal Krishna, the main causes of Grade-I contracted sockets were physical injuries, endophthalmitis and retinoblastoma. In group-II, the main causes were physical injuries, endophthalmitis, panophthalmitis and retinoblastomas. In grade-III & IV, the main causes were chemical injuries and panophthalmitis and in grade-V it was chemical injuries.

**Management of a contracted socket**

The primary aim of management is to create a socket so as to maintain a prosthesis with a good cosmetic appearance. Excellent or even fair motility of the prosthesis cannot always be expected even with good operative results. Before commencing a definitive therapy, it is necessary to identify, classify and eliminate any precipitating factors leading to contracture.

The best treatment as always is prevention, which includes:

- Proper dissection at the time of initial procedure
- Preserving as much conjunctiva and tenon’s capsule as possible during enucleation
- Secured closure of all layers over the implant without tension or superior displacement of the inferior fornix
- Avoidance of ill-fitting or roughened prosthesis as it may cause a more rapid contracture, symblepharon formation and total abandonment of prosthesis
- Elimination of any source of chronic infection that may arise from lid margin, socket, canaliculi, lacrimal sac, chemical or thermal injury
- Identification of conjunctival cicatrizing diseases like pemphigoid, Stevens-Johnson syndrome
- Avoidance of oversized prosthesis so as to prevent migration of the implant into the inferior fornix and thereby obliteration of inferior cul-de-sac.

The conformer must be left in place until the graft is healed which usually requires at least 3 weeks. If the socket has undergone prior irradiation, chemical, or thermal injury, the conformer has to be left for a much longer time. It is important that the graft maintains good apposition to the underlying vascular bed. Major reconstructive surgery should be performed only if there has been no cicatrical activity for a minimum of 9 months.

**Examination of the contracted socket**

A good preoperative assessment is performed with emphasis on the following:

- Examination of the orbital area and eyelids for any abnormalities & eyelid closure.
- Tone of the orbicularis and tarsal sulci
- Prosthesis – fit, size and its appearance with respect to fellow eye
- Area of the socket and assessment of the fornices especially the inferior.
- Volume of the socket by noting the depth of the socket compared to the fellow eye.
- Presence of any chronic inflammation or infection
- Cicatrical bands and degree of contracture.
- Whether the socket is dry or wet as a dry fibrosed conjunctiva may indicate poor vascularity.
- Movements of the muscles are looked for.
- Associated bony contracture.

**Treatment of contracted socket**

The inciting cause is treated initially and if only the inferior fornix is short, the prosthesis is modified. In a more severe contracture, gradually increasing the size of the conformer or prosthesis may be useful. Once the ocularist achieves the best possible expansion or prosthesis, socket is evaluated for additional surgical procedures.

**General considerations before socket reconstruction**

- Pre informed consent and prognosis of the surgery should be well explained.
- Cases that need oral mucosa grafting should have mouth washes started at least 2 weeks prior to the surgery.
- Socket should be free of infection. (Figure 2)

**Mild socket contracture**

Mild socket contracture involves shortening of the posterior lamina of the lids resulting in entropion but the fornices are not significantly lost. The surgery is planned as follows:

**Correction of entropion-Weiss procedure**

Mild contracture is treated by a transverse blepharotomy with marginal rotation. (Figure 3) If significant lid laxity co-exists, the rotation may be combined with horizontal lid shortening. If these procedures do not correct the entropion, a scleral or cartilage graft may be used to lengthen the posterior lamina of the lid.

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**Morphological classification of contracted socket**

<table>
<thead>
<tr>
<th>Socket Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anophthalmic contracted sock</td>
<td>Most common seen after enucleation or evisceration</td>
</tr>
<tr>
<td>Ophthalmic contracted socket</td>
<td>Following chemical or irradiational injuries</td>
</tr>
<tr>
<td>Microphthalmic contracted sock</td>
<td>In association with microphthalmos and microcornea</td>
</tr>
<tr>
<td>Hypoplastic contracted socket</td>
<td>Congenital under development of bony socket</td>
</tr>
</tbody>
</table>

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Closed method of fornix repair

While there may be an adequate amount of conjunctival tissue, there may be a decrease or loss of the inferior fornix in some patients. One may try deepening of the fornix by pressing mattress sutures over the silastic stent. These sutures are kept for 2-3 weeks and then modified prosthesis may be attempted.

Open method of fornix repair

Loss of inferior conjunctival tissue leads to shallow inferior fornix which causes a spontaneous prolapse of the inferior aspect of the prosthesis, especially in upgaze, when the soft tissues of the orbit shift infero-anteriorly. These patients may also have an increased horizontal laxity of the lower eyelid. A lateral canthal tendon procedure along with a fornix reconstruction is performed.

Moderate and Severe contracted socket

In moderate and severe socket contractures, the socket is assessed as to whether it is moist or dry. Moist sockets are treated with mucous membrane graft and dry sockets require a split thickness skin graft.

Treatment of moist socket:

Partial-thickness mucous membrane grafts are more susceptible to shrinkage and contracture.

Full thickness mucous membrane contracts less and may be obtained with minimal postoperative complications at the donor site. However, mucosal contracture and submucosal scar formation increase with the size of the oral mucous membrane harvested and mucous membrane lacks the rigidity needed for grafting the palpebral surface.

A full thickness mucous membrane graft is obtained from:

- Oral mucosa of cheek and lips-most common
- Hard palate
- Prepuccial skin
- Skin of the labia

The graft should be 40-50% larger than anticipated to allow for subsequent contracture with healing. It is helpful to harvest the graft at the beginning of the procedure so that it can be soaked in antibiotic solution before use.

To obtain a oral mucosal graft from the lip the mucosal area is infiltrated with 2% lidocaine with epinephrine. A no. 15 bard-parker blade is used to incise the mucosa 3mm from its junction with the lip across its entire extent. The full thickness graft is then excised using blunt and cutting dissection with blunt tipped scissors. A graft measuring approx 6-8 cm x 2.5 cm should be obtained and submucosal tissue is trimmed from the graft. (Figure 4)

The socket is infiltrated with 2% lignocaine with epinephrine. The conjunctiva is incised across the entire horizontal width of the socket. (Figure 5) Extensive dissection is performed releasing conjunctiva from underlying diseased scar and connective tissue. The free mucous membrane graft is then sutured into place with
interrupted or running 6.0 → 6.0 vicryl sutures. (Figure 6) It is sewn to pre-existing conjunctiva posteriorly and to palpebral conjunctiva anteriorly. A conformer is inserted to maintain the graft against the underlying vascular bed. The conformer should be left till the graft is healed which usually takes three weeks. For placement of larger grafts, a silicone (240 retinal band) stent is positioned in the lowest aspect of the inferior fornix and is anchored to the adjacent periosteum.

Hard palate grafts (HPG) can also be used for socket reconstruction, because it has both a mucosal layer and a fibrous collagen matrix that can provide rigidity and resistance to contraction. Additionally, HPG is easily harvested, undergoes minimal shrinkage, has a short healing period, and has a low donor site morbidity rate.

While the sole use of HPG in socket reconstruction may be adequate in cases with less severe contracture or single fornical involvement, it is sometimes difficult to obtain sufficient graft material from the hard palate to recreate both the palpebral and bulbar surfaces of both upper and lower fornices. The use of a combined mucous membrane and HPG in socket reconstruction may be an alternative approach.

Amniotic membrane can also be used instead of mucosa. It has less patient morbidity, faster recovery and better fitting of prosthesis. No contracture is observed with amniotic membrane as against mucous membrane. It is cheap and easily available and has no significant complications associated with it. Treatment of Dry socket

The socket is lined with a split thickness skin graft in these cases. Using a dermatome a split thickness graft can be obtained from the inner aspect of thigh. The scar tissue is removed as much as possible. A pliable dental compound “Mizzy” is used to make an impression of the socket. The substance is soaked in hot saline solution until it becomes soft and is then used to make a mold of the orbital space. After the material has been allowed to cool and harden it is softened again and is slightly reduced in size. The skin graft is placed around the mold with the epithelial surface towards it and perforations are made in the graft. The edges of the graft are sewn together with absorbable sutures. Incisions made through the skin over eyebrow and the inferior orbital rim is carried up to the periosteum and two holes are drilled in the bony rim 5 mm apart superiorly and inferiorly. A no. 30 stainless wire is passed through the openings superiorly and inferiorly and through the mould and the wires are twisted over bolsters. (Figure 7) After 1 month graft is split open in area of palpebral fissure. The mold is kept for at least 4 months after which the wires are removed and finally a permanent prosthesis is prepared.

Dermis fat graft

Dermis fat graft is used to correct for the conjunctival and volume deficit in a contracted socket. It is an autologous transplant consisting of de-epithelialized epidermis with its adjacent subcutaneous fat tissue. Currently, it is the only autologous transplant used as orbital implant in ophthalmic plastic and reconstructive surgery.
The preferred donor site is the upper outer quadrant of gluteal region. This is not a weight bearing area and there is no risk of damaging the sciatic nerve. After disinfection of the region, the graft is harvested from an area 5 cm below the middle point of the line that joins the anterior iliac crest and ischial tuberosity. Before making the incision, a circle with a maximum diameter 25 mm is marked on the skin. The skin is then incised superficially with a no.15 blade, incision not extending into dermis. A deep incision is then made perpendicular to the surface, through the dermis and into subcutaneous fat. The graft is severed from donor site and preserved in a gauge pad moistened with isotonic saline. (Figure 8,9) The donor site is closed with interrupted 2-0 absorbable sutures placed through fat and subcutaneous tissue, skin is closed with 2-0 black silk mattress sutures.

The recipient site is prepared by performing a conjunctival incision from the caruncle horizontally through the centre to the lateral canthus. A few mm of conjunctiva is mobilized in a circular fashion by subconjunctival dissection. Then a space to accommodate the graft is created by blunt dissection, bands of scarred tissue are separated, the graft is implanted into the recipient area. If necessary the graft can also be trimmed to remove excess fat to allow the tissue to be transferred into anophthalmic socket bed with out undue pressure. At all times, gentle pressure is maintained on the graft, keeping the fat within the orbit. The margin of the conjunctiva is reapproximated to the surface of dermis with multiple 6,0 → 6-0 vicryl interrupted sutures. (Figure 10) At the end of the procedure, a clear plastic shell (conformer) is inserted into the socket behind the eyelids. Antibiotic and steroid eyedrops can be employed before a firm dressing of moist and dry pads is applied and left for 2-3 days.

Dermis-fat grafts have been reported to produce good cosmetic and functional results in sockets with minimal contracture and good orbital vascularity. These are useful when increased volume and surface area are desired. (Figure 11, 12)
Complications of dermis fat graft

- Loss of the transplant due to the necrosis from infection
- Development of deep subconjunctival cysts – caused by long standing exposure or chronic erosion of alloplastic implants with subsequent invasion of conjunctival epithelium which then cannot be excised completely.
- Spontaneous atrophy of the graft – if there is more than 5-10% volume loss, it is considered to be clinically evident atrophy
- Post operative hemorrhage can cause significant pressure behind the implant which can be a reason for loss of the transplant
- Pyogenic granuloma – it develops on the surface of the graft around the suture material.
- Central ulceration can develop in the graft
- Growth of fatty tissue can induce proptosis of the artificial eye
- Donor site wound dehiscence, prolonged wound healing, scar formation and contour deformity

Precautions during dermis fat transplant

- Ablate the epidermis completely to avoid the risk of smelly socket
- Discoid shape is a stable form with a low risk of central ulceration. Alternatively, a narrower banana shaped transplant has been described in which donor site is easier to close
- It is essential not to push the graft into socket with force
- Never insert a dermofat graft onto an alloplastic implant
- Circular grafts measuring 25 mm in diameter and about the same in depth is a suitable size for any primary procedure. However in secondary procedures, graft often needs to be smaller.
- Attach the conjunctival layer without tension onto the dermis

Temporalis muscle graft

Scarred, contracted sockets with obliterated fornices are particularly difficult to treat. Although autogenous dermis-fat orbital implantation is an effective means of orbital reconstruction, there is a 30% chance of atrophy of at least half of the graft volume when it is implanted in an avascular socket. Introducing a pedicle flap into the orbit as a vascular bed for an autogenous dermis fat
Temporalis muscle graft is supplied by superficial temporal artery, a branch of external carotid artery. Therefore, transposing a pedicle flap of temporalis muscle into the orbit reduces the chances of atrophy.

Under general anaesthesia, a 4 cm preauricular incision is extended superiorly behind the hairline over the anterior aspect of temporalis muscle, inferiorly it is curved anteriorly to the lateral palpebral canthal raphe, but it does not reach the lateral canthal angle. The superficial muscular aponeurotic system of face with its terminal branches of facial nerve is reflected as separate layer. Lateral orbital wall is exposed by retracting temporalis muscle. (Figure 13) A 35 mm wide pedicle of temporalis muscle and epicranium is developed and released with periosteal elevator and transposed into the orbit through a window in the lateral orbital wall. (Figure 14) The aponeurotic layer at donor site is closed with 5-0 chromic sutures and skin is sutured with 4-0 nylon.

The most common donor site complication is transient or permanent alopecia. Among other complications are blood loss, hematoma formation, slight depression over temporalis fossa.

**Horizontal palpebral fissure widening**

The switch flap technique classically described by Mustarde for the management of a blepharophimotic contracted socket in congenital anophthalmos can be used for upper eyelid reconstruction to widen the palpebral aperture. In this two staged procedure, in stage 1 horizontal expansion of the upper eyelid with a switch flap from lower eyelid hinged laterally is performed. A full thickness vertical incision is made in the upper eyelid at the junction of the middle and lateral thirds. The lower eyelid switch flap is accommodated in the upper lid defect and sutured in layers. Stage 2 is performed after 6 weeks. The switch flap is released and upper lid margin is reconstructed in layers. (Figure 15)
Management of recalcitrant cases

A socket that has undergone multiple unsuccessful operations and has excessive scar tissue is unlikely to benefit from further repair. For such sockets Dortzbach and Callahan have advocated exenteration of the eyelid and residual socket material to create a cavity into which a prosthesis is fitted.10

Optical methods to improve the appearance

- Spectacle prosthesis
- Tinted or smoked lenses
- Plus or minus lenses to magnify a microphthalmic socket or to minimize buphthalmic socket
- Prisms to change the apparent horizontal or vertical position of malpositioned prosthesis or socket

Conclusion

Contracted sockets offer difficult management problems for the ocularist and the oculoplastic surgeon, and is best approached as a team. Most importantly, the patient's needs and desires should be fully explored, since the lack of communication between all involved frequently results in failure and/or disappointment.

Cases with mild contractures or with minimal posterior laminar shortening are the ones best treated by surgery.

On the other hand cases with moderate to severe contracture are the most difficult hence should be pre informed about the prognosis of the surgery.

Another important aspect is pressure treatment of socket because if the ocular prosthesis remains out of the socket at any time up to one year after surgery it invariably results in a cicatricial contraction of the socket and inability to retain an ocular prosthesis. Capillary traction device and Lafuente pressure mask are devices used for application of pressure in the post graft period.11

References

Sebaceous gland carcinoma is also called as sebaceous cell carcinoma or sebaceous carcinoma or meibomian gland carcinoma. These have major predilection for ocular adnexa. 75% of the tumors are periocular.

Incidence

In contrast to most common eyelid tumors (basal cell carcinomas) these comprise 5% of eyelid malignancies in United States. Greater frequency of the tumor has been seen in Asians. Recent increase in the incidence may be due to increase awareness of the lesion and more accurate diagnosis. Indians are more commonly affected; 40-60% of malignant eyelid tumors in Indians are sebaceous carcinomas.

Risk factors

It is generally a disease of older individuals (mean age 57-72 years). 75% of the patients are females. Prior irradiation for germline retinoblastoma is a known risk factor. Sebaceous carcinomas are associated with Muir-Torre syndrome.

Site of origin

Majority of tumors (95%) involve meibomian glands in tarsal plate. Rarely Zeis gland and pilosebaceous units of caruncle are involved. Infrequently cases of intraepithelial carcinoma without intratarsal or intradermal component have been reported in the conjunctiva. Lacrimal gland can be involved secondarily. Tumors can be multicentric in nearly 20% of the cases.

Broad spectrum of clinical features

Two third of tumors are misdiagnosed clinically. Clinically these tumors are commonly misdiagnosed as chalazion or chronic blepharoconjunctivitis (Masquerade syndrome). Therefore clinicians should be advised to submit chronic or atypical chalazia to pathology and to biopsy chronic blepharoconjunctivitis that is unresponsive to therapy. Patients may present with diffuse pseudoinflammatory pattern such as diffuse unilateral thickening of eyelid, unresponsive conjunctivitis or keratoconjunctivitis or corneal pannus. Pedunculated or papillary lesions look like cutaneous horn or conjunctival papillomas.

Histopathology

50% of the cases are misdiagnosed histopathologically as squamous cell carcinoma, basal cell carcinoma and unspecified neoplasm. Intraepithelial pagetoid spread is a characteristic feature which is present in near about 50% of the cases.

Degree of differentiation

Varies from well to poorly differentiated areas. Well differentiated tumors contain cells with foamy, finely vacuolated cytoplasm and distinct cell borders usually in center of tumor nests (Figure 1a). Poorly differentiated areas show often scant cytoplasm with indistinct vacuoles, central comedo necrosis (Figure 1b) and pagetoid involvement of overlying skin/mucosa.

Histologic patterns

These include lobular, comedocarcinoma, papillary and mixed patterns.

Special stains: Sebaceous differentiation can be confirmed by fat stains such as oil red-O.

Immunohistochemistry

No reliable marker is present however p16 immunostain is considered helpful in recognizing intraepithelial spread of the disease.

Figure 1(a): Foamy cells represent well differentiated areas

Figure 1(b): Central comedo necrosis in an anaplastic variant
tumor. Anti-EMA, BRST-1, and Cam 5.2 may help distinguish poorly differentiated sebaceous carcinomas from other tumors when distinction can not be clearly made by light microscopy alone. Adipophilin can also distinguish sebaceous tumors from basal carcinomas and squamous lesions. Mismatch repair genes are mutated in sebaceous tumors associated with Muir-Torre syndrome. Stains for MSH-2, MLH-1 may be indicated if Muir-Torre syndrome is suspected.

**Modes of spread**

Characteristically show pagetoid spread. Diffuse Bowenoid spread may be seen as diffuse full thickness replacement of epithelium. Tumors may spread directly to orbit, sinuses and intracranially. Perineural, lymphatic and hematogenous metastasis has also been reported.

**Treatment**

Complete surgical removal is required. Supplemental cryotherapy and topical chemotherapy can be used. In cases with unresectable orbital invasion, orbital exenteration is needed. Radiotherapy may be used as a palliative measure in advanced cases.

**References**


Malignant Uveitis Masquerade Syndrome

1. Naginder Vashisht MD, 1 Prashant Naithani MD, 1 Subijoy Sinha MD, 1 Sumeet Khanduja MD, 1 Pradeep Venkatesh MD, 1 Satpal Garg MD

Choroidal metastases are recognized as the most common intraocular malignancy1,2. They are most common with breast cancer in females and lung cancer in males. The incidence of metastatic tumors as a cause of symptomatic disease has been reported to be 1% to 3%. Approximately, one third of these patients have no previous history of primary cancer at the time of diagnosis3,4,5. The primary site was discovered after complete oncologic evaluation in nearly half of the patients2.

We report here a case referred to our centre with history of progressive blurring of vision in left eye and headache since two months, which were the first signs of bronchogenic carcinoma.

Case Report

A 34 year old non smoker healthy male was referred to the uvea services of Dr. R.P. Centre for Ophthalmic Sciences with complaints of progressive dimunition of vision left eye and headache since two months. The patient had momentary improvement with systemic steroids (pulse & oral steroids) and no improvement with anti-tubercular (ATT) drugs, which he received elsewhere before he was referred to us. This patient in the absence of significant past medical history and systemic complaints underwent clinical examination, ocular and systemic imaging. On clinical examination, the best corrected Snellen's visual acuity (BCVA) was 6/18 in the right eye and hand motions close to face in the left eye. The BCVA reduced in both the eyes from 6/6 in right eye and 6/24 in left eye (noted elsewhere when patient presented for the first time two months back) to the present vision as mentioned above. Fundoscopy revealed amelanotic mass lesion with serous retinal detachment (RD) in the right eye and total serous retinal detachment in the left eye (Figures 1 & 2). There was absence of anterior chamber & retrolental cells on slit-lamp examination. The patient underwent fluorescein angiography (FFA) which showed hypofluorescence in early phases and late pinpoint leaks in right eye (Figure 1). The left eye showed generalized pooling of the dye due to total retinal detachment (Figure 2). The ultrasound B-scan revealed solid choroidal mass lesions in both the eyes with total exudative RD in the left eye and inferior exudative RD in the right eye. All these findings, absence of inflammation and past history of patient of no response on systemic steroids and ATT suggested uveal metastases from carcinoma of an unknown primary. All the laboratory tests were normal. The chest X-ray showed patchy consolidation involving the lingular segment and lower lobe of the left lung. The sputum tests for tuberculosis were negative for 3 consecutive days. A contrast enhanced computed tomography scanning of the chest demonstrated thick walled cavitary lesion with speculated margins in the left perihilar region in lingular segment with surrounding consolidation. Bronchoscopic biopsy showed the presence of intrabronchial tumour in the left lingula with complete obstruction and bronchoscopic aspirate revealed the presence of abnormal histology, consistent with adenocarcinoma of the lung. To further add on, the bronchoalveolar aspirate samples were negative for tuberculosis and fungus but raised suspicion of malignancy. The patient was then referred to the oncology department for further treatment.

Discussion

Intraocular metastases were at one time thought to be extremely rare, but it is now known that more than 10% of patients have intraocular metastatic foci1,2. However, the incidence of metastatic tumors as a cause of symptomatic disease has been reported to be less than 5%. Approximately, one third of these patients have no previous history of primary cancer at the time of diagnosis3,4,5. The primary site was discovered after complete oncologic evaluation in nearly half of the patients2. Bronchogenic carcinoma is the most common primary tumor detected in patients with unknown primary at the time of ocular diagnosis6.

The lung is the second most common primary site after breast for choroidal metastases and Soysal HG et al (2007) reported 7% incidence. Unlike our case the lung metastases to uvea are usually unilateral and unifocal. Another characteristic feature of lung metastases, similar to this case is the likelihood of uveal tumor presenting before the discovery of the lung cancer2,3. Stephens and Shields reviewed 70 cases of choroidal metastases and noted the presence of following symptoms in decreasing order of frequency: blurred vision, pain, photopsia, red eye, floaters and field defects. Exudative RD is commonly associated with uveal metastases and may occur in upto 90% of the patients. Multiple foci and bilaterality are important features of metastatic choroidal tumours. In 20%-40% of the cases, the lesions are bilateral. As in the case mentioned above, the patient was asymptomatic inspite of the lesions in the opposite eye, similarly in one series 56% of opposite eye lesions were detected while asymptomatic on follow-up examination5.

The diagnosis of uveal metastases (with unknown primary) is essentially based on thorough clinical workup supplemented by imaging studies. Many lesions may be confused with choroidal metastases for example primary choroidal melanomas, benign lesions such as choroidal haemangiona, choroidal osteoma and inflammatory granulomas and rarely inflammatory conditions like Harada's disease, posterior scleritis, choroidal tuberculosis and uveal effusion syndrome. However, in patients similar to our case (bilateral choroidal lesions with exudative RD) ruling out the aforesaid inflammatory conditions is of prime importance. But there were certain factors mentioned below which attributed to clinical dilemma for the primary clinician. These factors are:

Clinical history & systemic examination

The patient under study was a young, well-built, healthy & non-smoker male. The history of patient was also insignificant. These factors could have provided difficulty to arrive at the accurate diagnosis.

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2. Sir Ganga Ram Hospital, Rajender, Nagar, New Delhi

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The slit lamp examination of patients with such presentation is mandatory (see Figure 3). The slit lamp examination of our patient was missed at the initial presentation. Further though, choroidal metastases are usually non-inflammatory, some inflammation may be associated with them. The inflammation is usually mild, non-granulomatous and refractory to steroid treatment.

Ultrasonography is of extreme importance in evaluating the patients of choroidal lesions with serous RD (see Figure 3). It is of prime significance especially when pin-point leaks are noted on FFA as in our case. The ultrasound picture of the various entities is described below:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Ultrasound Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Choroidal metastases</td>
<td>B-scan: Echogenic subretinal mass with diffuse ill defined borders on B-scan with overlying RD (commonly associated) and moderate sound attenuation.</td>
</tr>
<tr>
<td></td>
<td>A-scan: Moderate to high internal reflectivity</td>
</tr>
<tr>
<td>Vogt-Koyanagi-Harada's (VKH) Syndrome</td>
<td>Diffuse low to medium reflective thickening of the choroid. Serous RD, mild vitreous opacities and choroidal detachment usually associated</td>
</tr>
<tr>
<td>Posterior Scleritis</td>
<td>Diffuse retinochoroidal thickening with episcleral edema. “T-sign” when seen in peripappillary region. Choroidal detachment and serous RD also associated</td>
</tr>
<tr>
<td>Ocular Tuberculosis</td>
<td>Solid elevated mass lesion with low to moderate internal reflectivity. Exudative RD may be associated.</td>
</tr>
</tbody>
</table>

**Figure 1:** It shows color photograph of the right eye of the patient at initial presentation and at our centre two months later. Note the progression of the flat amelanotic choroidal lesion to exudative RD with involvement of the macula. It also shows FFA done at our centre revealing early hypoflourescence followed by late pin-point leaks.

**Slit lamp examination**

The slit lamp examination of patients with such presentation is mandatory (see Figure 3). The slit lamp examination of our patient was missed at the initial presentation. Further though, choroidal metastases are usually non-inflammatory, some inflammation may be associated with them. The inflammation is usually mild, non-granulomatous and refractory to steroid treatment.

**A and B-scan ultrasonography**

Ultrasonography is of extreme importance in evaluating the patients of choroidal lesions with serous RD (see Figure 3). It is of prime significance especially when pin-point leaks are noted on FFA as in our case. The ultrasound picture of the various entities is described below:
The ultrasound evaluation of this patient was not done during the initial visit.

**Examination of the opposite eye**

Multiple foci and bilaterality are important features of metastatic choroidal tumours. In 20%-40% of the cases, the lesions are bilateral\(^1\). The exudative RD in left eye of this patient initially responded with pulse steroids nevertheless the choroidal lesions in the right eye increased in extent and size. This further confirms that the serous RD secondary to unknown primary are steroid resistant. Further to add on as our patient was asymptomatic inspite of the lesions in the opposite eye, similarly in one series 56% of opposite eye lesions were detected while asymptomatic on follow-up examination\(^6\). Thus a careful, meticulous and repeated examination of the opposite eye is warranted.

Thus, a through clinical examination (including history taking, ocular and systemic examination) along with ocular imaging and radiological workup is mandatory if a metastatic tumor is suspected. The use of systemic steroids and ATT in patients with atypical posterior uveitis (unless investigated) should be avoided. This through approach (see algorithm 1) aids in prompt diagnosis and accurate treatment which can significantly improve the quality of life of these patients.

**References**


Intraocular foreign bodies (IOFBs) are common problems in ocular injuries. Up to 40% of eyes with an open globe injury contain at least one IOFB. Retained IOFB represent true emergency and can cause severe vision loss by endophthalmitis, retinal detachment (RD) and metallosis.

As most of the IOFB injuries occur more frequently in young and productive member of the society, these injuries cause ocular morbidity and economical burden. Visual prognosis is best when the IOFB is removed during initial wound repair surgery or as soon as possible. Therefore detailed examination and timely management is very important for a favorable outcome.

Causes

Common causes of IOFBs include gun shot injury, explosive injury, use of machine tool, road traffic accident (RTA) and assault.

Majority of the IOFB are small, sharp projectiles produced by hammering metal or stone.

Up to 90% are metallic and 55 to 90% are magnetic.

Epidemiology in India

IOFB occur in 18-41% of the open globe injuries, mostly in young males in the age group 20-40 year in work place accident are most common cause of IOFB with 60-80% involving hammering metal or stone. In ocular trauma cases, IOFB is present in 14.4% eyes, primarily in the posterior segment.

Common composition of the IOFB in the descending order is Stone (28.8%), wood (24.3%), iron (16.5%), and chemicals (6.7%).

Ocular pathophysicsology

Foreign body entering the eye may cause damage in one of the following three ways

- Mechanical effect
- Introduction of infection
- By specific action (chemical or others) on intraocular tissues

Evaluation of a case of IOFB

- Thorough history and suspicion of its presence

History

- Circumstances of the trauma
- Exposure to hammering, drilling, grinding or explosion
- Time elapsed since injury
- Use of safety glasses

Highly suspicious cases

Above mentioned history and subtle ocular signs such as:

a) Self sealing corneal wound
b) Localized lenticular opacity
c) Mild intraocular IOP asymmetry
d) Iris hole, iris heterochromia, pupillary asymmetry

History solely can give clue in case of severely traumatized eye with hyphema, scleral tear, vitreous haemorrhage, RD and media opacity

Examination

Important information to document is

- External examination
- Base line visual acuity, pupillary examination, intraocular pressure (IOP)
- Slit lamp examination, assessment of media clarity
- Extent and localization of corneal wound, iris colour, lens status
- Size, shape, location, number, type, magnetic properties and entry path of IOFB
- Presence of retinal tear and detachment.

**Imaging**

- All eyes suspected of harboring IOFB should be properly imaged to rule out the presence of IOFB

**Ultrasound**

- It is a useful tool in localizing IOFBs, and it’s careful use is possible even if the globe is open
- Detects both radiolucent and radio-opaque IOFB
- Helps in determining the precise site of IOFB

- Ultrasound with A and B mode with low gain showing hyper reflective echoes on B-Scan and corresponding high spike on A mode is suggestive of IOFB
Method should be selected so as to cause minimal surgical trauma

Non magnetic IOFB
- Require vitrectomy
- Always pass the IOFB through the extraction wound in plane of smallest cross section and grasp it with the heaviest instrument possible as it become exposed
- This minimizes the risk of dropping of IOFB over macula
- Liquid Perfluorocarbon should be placed to damp on the impact and protect the retina.

Magnetic IOFB
- Magnets are commonly used
- Their ability to align ferromagnetic IOFBs in long axis of the magnetic field and deliver the small diameter of IOFB through sclerotomy; thus minimizing the surgical trauma
- Two types of magnets

Internal Rare earth magnet
- Smaller create more uni-directional magnetic field
- More controlled
- Generate less force
- Require vitrectomy and Intraocular forceps

External magnet
- Capable of producing great force
- Bulky, restricted to use outside the eye
- Provide less controlled extraction

Complications of magnetic extraction
- Impaction of IOFB into lens
- Traction on vitreous cavity, vitreous base and retina
- Intra-retinal and sub-retinal IOFBs should be removed through sclera cut down with T-shape uveal and scleral flap with magnetic extraction
- Diathermy should be applied to uveal bed before removal to decrease the chance of hemorrhage
- Endolaser photo- coagulation should be applied around the impaction (site retinal) (sub-retinal) to prevent RD
- Retinotomy can be made to remove sub-retinal IOFB if it is not possible to remove it through sclera cut down.

- Caution is needed when vitreous cavity is full of dot like echoes due to vitreous haemorrhage or vitreous debries - in such cases the hyper-reflective spike of IOFB persists even at low gain while the hyperreflective spike of vitreous haemorrhage and vitreous debries disappears.6
• In case of glass and organic material sound beam should be always perpendicular to it’s long axis to accurately indicate it’s size and density.\(^7\)
• False negative results are possible when IOFB is wooden, small size IOFB and IOFB adherent to the ocular coats or engulfed by the inflammation.
• Ultrasound can be utilized to determine magnetic properties of foreign bodies. One method described by Ossoining, utilizes the standardized A scan to detect small vertical movements of foreign body spike as pulsed magnet is slowly advanced towards the eye ;these slight ,pulsatile movement of FB spike indicate that it is metallic IOFB.

**Ultrasound biomicroscope**

• It may help when an IOFB is in the anterior segment.

**Plain x-ray**

• All high velocity injuries with the evidence or a strong suspicion of IOFB should undergo a plain X-ray of the orbit.
• Most of the metal fragments are clearly visible on plain X-ray, although some metals are relatively radiolucent (e.g. aluminium).\(^8\)
• Single anterior-posterior and lateral orbital X-ray is sufficient.
• Also useful in cases of stone IOFB.
• Identification of multiple IOFBs and those embedded in the ocular adnexa.
• Limbal metallic ring or contact lens may be used to localize the IOFB.
• X-ray have 60% false negative rate amongst in all cases of IOFB.\(^9\)

**CT-Scan**

• It is the imaging study of choice for IOFB localization.
• Advantages are:-
  a) Little need for patient’s cooperation.
  b) No manipulation of traumatized globe.
  c) Exact localization of the IOFB.
• Axial and coronal cuts of < 1.5 mm are advised.
• Small retained magnetic foreign bodies may lose their radio-density and may not be visible on plain X-ray and Ultrasound these are detected on CT scan.\(^10\)

**MRI**

• Only test capable of detecting small plastic and or wood IOFBs.\(^11\)
• Is not recommended for metallic IOFBs.

**ERG**

• Initially the B – wave shows supernormal response with loss of oscillatory potential in case of retained IOFB.
• As the condition progresses ERG deteriorates with reduction of B Wave, which ultimately become extinguished.
• Results should be interpreted in conjugation with ultrasonography because co-existing retinal detachment will also affect these tests.\(^12\)

**Management**

• Management of IOFB requires immediate closure of the globe and removal of IOFB.
• Delay in primary IOFB removal >24 hours produces four fold increase in the risk of endophthalmitis and severe vision loss.13

• Prompt removal before encapsulation of IOFB facilitates removal and prevents toxicity due to IOFB.

• Eyes should be protected with eye shield

• Intravenous broad spectrum antibiotic prophylaxis while awaiting surgery

• Tetanus prophylaxis

Surgical Management

• Appropriate method depends upon the location, size, type, number of IOFBs, associated condition like cataract, vitreous haemorrhage, retinal tear and retinal detachment.

Management of associated conditions

Endophthalmitis

Microbiological specimen should be collected when endophthalmitis is suspected and appropriate intra-vitreal injection should be given after surgical closure.

Retinal detachment

Should be managed with either sclera buckle placement or vitrectomy, lensectomy, photocoagulation and internal tamponade with either gas or silicone oil.

Retinal breaks are common with IOFB injuries; those associated with impaction site and those distant from the site. Demarcation photo-coagulation or cryotherapy is needed in such conditions.

Proliferative Vitreoretinopathy (PVR)

Risk is more with the presence of multiple retinal breaks, choroidal hemorrhage and multiple surgeries.

Mechanism of vision loss in such cases is mainly due to macular wrinkling, sub-retinal fibrosis and tractional retinal detachment (TRD).

Pars-plana vitrectomy with removal of posterior hyloid is thought to be a better way of preventing PVR.

Management of IOFBs at other sites

IOFB in the anterior chamber

Are ideally removed through secondary limbal wound after closure of primary corneal wound, as removal of IOFB through corneal laceration leads to more surgical trauma to the tissue and tissue loss.

Viscoelastics should be used to reduce the risk of iatrogenic damage to the corneal endothelium and the lens.

Intracameral antibiotic wash is always advisable after IOFB removal.

An intra-lenticular IOFB

Usually the IOFB is extracted first followed by the lens extraction and an intraocular lens (IOL) is implantation.

Devastating Complications

Metallosis

Siderosis

Develop from retained iron IOFB. Time of onset and degree of destruction depends upon the iron content and the location of the IOFB.

Signs includes reduced vision, iris heterochromia, dark brown deposits beneath the anterior capsule and cataract formation.

ERG showing initial supernormal response followed by progressive decrease in the b-wave

The prognosis of a posterior IOFB is worse than an anterior segment IOFB.

Chalcosis

Occurs with copper containing IOFBs; pure copper causes rapidly progressive severe purulent panophthalmitis culminating in phthisis bulbi. Therefore this needs prompt treatment.

“Prevention is always better than cure”

• Eye protection with protective eyewear, if appropriate (3 mm of polycarbonate) prevents virtually all injuries. This is especially recommended in risky activities (e.g., hammering).

• Public awareness.

• Educating factory workers regarding eye care safety procedures.

References

1. Thompson et. al Infectious endophthalmitis after penetrating injury with retained IOFB; Ophthalmology 100; 1468-1474; 1993
2. Thompson et. al Infectious endophthalmitis after penetrating injury with retained IOFB; Ophthalmology 100; 1468-1474; 1993) + (Armstrong ; review of IOFB ;Int. Ophthalmology 12;112-117, 1988)
3. B. Shukla ,S. Natrajan, Management of ocular trauma , chapter 23:page 236
7. Sandra Frazier Byren – ultrasound of the eye and orbit, chapter 4 page109)
8. Vitreosctal surgery of the injured eye D. Virgil Alfaro & peter liggett page 62 chapter 6)
10. Vitreosctal surgery of the injured eye D. Virgil Alfaro & peter liggett page 63 chapter 6)
12. Vitreosctal surgery of the injured eye D. Virgil Alfaro & peter liggett page 61 chapter 6)
13. Thompson JT, Parver et al Infectious endophthalmitis after retained IOFB;ophthalmology;100;1468-1474;1993)
Retained lens fragment is one of the most feared complications during cataract surgery (Figure 1a,1b) and usually results from posterior capsular rupture or zonular dehiscence. Originally referred to as “Lost lens syndrome,” it plagues unfortunate patients, cataract surgeons and vitreoretinal surgeons. Though the incidence is low (estimated at 0.3 to 1.1% in different studies), it is not an infrequently encountered situation considering the large number of cataract surgeries performed.

Surgeons in the learning phase of phacoemulsification are more likely to be confronted with this situation, though it can occur even in the most experienced of hands. Other common risk factors for vitreous loss and subsequent retained lens fragments include small pupil, brown cataract, hypermature cataract, traumatic cataract, subluxated lens, posterior polar cataract and pseudoexfoliation.

Though it can occur at any step of the surgery, most commonly a nucleus piece dislocates into the vitreous cavity during the fragmentation phase of phacoemulsification. The size of the displaced fragment may thus range from the entire nucleus (when it has fallen posteriorly during hydrodissection) to any fraction of it. The major complications attributed to retained lens fragments include rise in intraocular pressure (IOP), intraocular inflammation, cystoid macular edema (CME), corneal edema and retinal detachment. These may vary from patient to patient depending on the size of retained nuclear fragment, time since cataract surgery, the extent of intraocular manipulation during the initial surgery and individual’s inflammatory response.

What should be done by a Cataract Surgeon?

Falling lens fragments present an excellent time for the surgeon to recall the first principle of medical practice, primum non nucerum—“first, do no harm.” Some surgeons advocate attempts to remove the fragments with techniques such as posterior irrigation and assisted levitation. However, others caution against such actions because they exacerbate vitreous prolapse, create uncontrolled traction on the vitreous base, and increase the risk of retinal detachment. Any “fishing” manoeuvre to remove sunken fragments must not be undertaken.

The following steps are advocated:

- Good anterior vitrectomy (with or without the use of Triamcinolone Acetonide) must be performed, so as to free the anterior chamber and the wound of vitreous
- Lens matter and fragments should be removed from the anterior chamber and the capsular bag
- An Intraocular lens (IOL) may be placed in the anterior chamber, ciliary sulcus or capsular bag provided that it is stable enough and will not dislocate with indentation procedures (to remove trapped fragments and prolapsed vitreous) during future vitreoretinal intervention.
- The corneal/scleral incision should be sutured to promote wound integrity and prevent leakage during subsequent vitreoretinal surgery.
- The patient and his family should be promptly informed of the situation.
The patient should be referred for vitreoretinal consultation. Appropriate anti-inflammatory and anti-glaucoma medication must be instituted in the meantime.

**Indications for removal and timing of surgery**

Majority of the patients, who have large nuclear fragments with attendant raised IOP and/or uveitis will need surgical intervention to remove the fragments and break the cycle of progressive lens induced inflammation and glaucoma. With improving techniques of vitreo retinal surgery and better instrumentation, surgical removal of retained lens matter is now indicated in almost all patients, to expedite the process of visual recovery. Associated retinal detachment, retinal tears or endophthalmitis are themselves an urgent indication for surgery.

Unfortunately the optimal timing of pars plana vitrectomy is not well established. The general notion is that early surgery prevents late complications, though it has not been proven by any large clinical trials.

**Immediate Simultaneous Vitrectomy** is usually less technically challenging with the obvious advantage of avoidance of a second surgery. This is however not always possible as a large number of cataract surgeries are performed in outpatient surgical centres or set ups without immediate access to vitreoretinal surgeons.

**Delayed Vitrectomy** may be complicated by suboptimal visualisation because of corneal edema. It is prudent in these cases to wait for sufficient corneal clarity before the patient is taken up for removal of the retained fragments. This may be associated with raised IOP which must be controlled as well prior to surgery. Postoperative management in such cases involves frequent topical corticosteroids as well as the full gamut of antiglaucoma medications including beta blockers, topical and/or systemic carbonic anhydrase inhibitors and hyperosmotics. The role of systemic steroids is controversial.

**Surgical Techniques**

The surgical technique involves a three-port pars plana vitrectomy (PPV) with removal of as much as possible of the vitreous gel prior to the removal of the lens fragments. A posterior vitreous detachment must be induced if not present. Adequate initial vitrectomy avoids unintended vitreous traction during phacofragmentation. If the basal vitreous gel is not debulked prior to the lens fragmentation, small fragments of lens may become embedded in it. Retinal damage can occur when these fragments are being removed (Figure 2a).

While a very soft nucleus piece or the cortical lens matter can be removed by the vitrectomy cutter alone, this makes the cutter blunt and reduces its performance subsequently. Phacofragmentation is the procedure of choice in most cases. An intermittent pulsed ultrasound-mode, with reduced ultrasound power of 10-20% should be maintained to allow continuous occlusion of the suction port, thereby minimising the mechanical trauma to the retina from projectile particles. Notably, ultrasonic power should be applied only in the mid vitreous and away from the retinal surface, to avoid inadvertent damage to retina.

With the availability of modern phacofragmatomes most of the nuclear fragments can be eaten up in the vitreous cavity itself. An extremely hard nucleus however poses a problem for phacofragmentation. Lancing it with a needle or MVR knife and removal through the limbal route is an option in such cases. An IOL should not be placed during the initial surgery in cases where this manoeuvre may be anticipated; otherwise it must be removed before this can be done.

**Role of Perflorocarbon Liquids (PFCL):** Though most of the cases can be managed without PFCL, it can serve as a useful adjunct to the surgery in the following 2 ways:

- PFCL can be used for floatation of the entire nucleus and subsequent removal through the anterior route, following
adequate pars plana vitrectomy. Unsayingly the IOL poses a hindrance for the same and needs to be removed if present.

- PFCL can be used during phacofragmentation to protect the posterior pole from mechanical trauma caused by the flying pieces of nuclear fragments (Figure 2b).

Management of retinal detachment (RD) is like any other complex rhegmatogenous RD. IOL can be implanted if it has not been done previously.

**Outcome after surgery**

The visual results of managing cases of posteriorly dislocated or retained lens fragments are generally good. Published reports between indicate that about 44% to 71.3% achieve a final visual acuity of 20/40 or better, with a mean of 58.3%.7-10

Post-operative complications include cornea edema, glaucoma, persistent intraocular inflammation, and retinal detachment. The incidence of rhegmatogenous RD after uncomplicated phacoemulsification surgery is 1% to 1.5%,5 rises to 6.8% to 8.6% if an anterior vitrectomy is performed and escalates to 8% to 17% with disruption of the anterior hyaloid face and posterior dislocation of nuclear lens fragments.6 It is of critical importance to carefully evaluate the retina throughout the perioperative course in these patients including a meticulous intraoperative inspection of the retinal periphery at the conclusion of PPV.

**Conclusion**

Retained lens material after cataract surgery represents a potentially vision-threatening complication. Recent improvements in both phacoemulsification and PPV techniques as well as detection and treatment of surgical complications may have contributed to improved visual outcomes.

**References**


**Editorial comment for the article**

**Indications for vitrectomy**

(i) Nucleus Drop
(ii) Epinucleus Drop
(iii) Cortical matter Drop with vitritis / glaucoma
(iv) Vitreous in anterior chamber / corneal wound

**Timing:** within 2 weeks

**Urgent:** If nucleus drop associated with retinal tear / retinal detachment / endophthalmitis.

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**Monthly Clinical Meetings Calendar 2010-2011**

<table>
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<th>Event</th>
<th>Date</th>
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<tbody>
<tr>
<td><strong>Max Eye Hospital</strong></td>
<td>25th July, 2010 (Sunday)</td>
</tr>
<tr>
<td><strong>Sir Ganga Ram Hospital</strong></td>
<td>29th August, 2010 (Sunday)</td>
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<tr>
<td><strong>Army Hospital (R&amp;R)</strong></td>
<td>26th September, 2010 (Sunday)</td>
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<tr>
<td><strong>Centre for Sight</strong></td>
<td>31st October, 2010 (Sunday)</td>
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<tr>
<td><strong>Shroff Charity Eye Hospital</strong></td>
<td>21st November, 2010 (Sunday)</td>
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<tr>
<td><strong>Midterm Conference of DOS</strong></td>
<td>27th &amp; 28th November, 2009 (Saturday - Sunday)</td>
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<tr>
<td><strong>Bharti Eye Foundation</strong></td>
<td>26th December, 2010 (Sunday)</td>
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<tr>
<td><strong>Guru Nanak Eye Centre</strong></td>
<td>30th January, 2011 (Sunday)</td>
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<tr>
<td><strong>Safdarjung Hospital</strong></td>
<td>27th February, 2011 (Sunday)</td>
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<tr>
<td><strong>Centre for Sight</strong></td>
<td>31st October, 2010 (Sunday)</td>
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<tr>
<td><strong>Shroff Charity Eye Hospital</strong></td>
<td>21st November, 2010 (Sunday)</td>
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<tr>
<td><strong>Annual Conference of DOS</strong></td>
<td>15th to 17th April, 2011 (Sunday)</td>
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**First Author**

Vinod Kumar Aggarwal MS, DNB, MNAMS, FICO, FRCS
Retinoblastoma is the most common primary intraocular malignancy in children. The incidence is approximately 1 in 18,000 live births worldwide. Around 300 new cases of retinoblastoma are diagnosed every year in the United States. It is estimated that India has one of the highest number of children affected by retinoblastoma in the world, with around 1600 new cases being diagnosed every year.

Retinoblastoma is emerging as a national priority among pediatric cancers, as it is curable in nature and the prognosis for survival is excellent, if detected early. In view of this, the Indian Council of Medical Research has started a new project named National Retinoblastoma Registry. The aim of this registry is to study the magnitude of the problem of retinoblastoma in the country and to collect reliable data on children suffering from retinoblastoma. This is a hospital based registry and the sites which have been identified are New Delhi, Hyderabad, Chennai, Guwahati, Lucknow, Chandigarh, Ahmedabad, Bangalore, Kolkata, Madurai, and a Miscellaneous site. For Delhi, Dr. Rajendra Prasad Centre for Ophthalmic Sciences, AIIMS, has been designated as the nodal point. Each site has been entrusted with the responsibility of local networking with other hospitals in the city to register as many retinoblastoma cases as possible.

The objectives of the registry are as follows:

- To record information according to age, sex and residence of the patient
- To adopt a uniform staging system
- To uniformly study the histological type of the disease
- To record the treatment protocol used
- To record the remission status of the disease
- To record the role of heredity from history only and/or family history of cancer
- To determine mortality information from different regions of the country.

The functioning of the registry is as follows:

A Principal Investigator has been designated for each site. Each site has research staff which has been recruited to fill up a Proforma. The Proforma includes information on patient demographics, clinical data describing signs and symptoms associated with the tumor, and how the initial diagnosis was made. A uniform classification of the disease which is to be followed has been incorporated. The treatment received by the patient has been included. It also describes the number of chemotherapy cycles and the number of local therapy sittings received by the patient. Other information includes the pathological staging of the tumor and the remission status of the patient at completion of therapy. Information on survival, quality of life and the cause of mortality, if applicable, is also obtained.

**Patient Information Sheet and Consent Form**

Before recruitment, the patient's parent is read out aloud (if illiterate)/ made to read (if literate), a patient information sheet containing all the relevant information. If the parent is willing, he/she is made to sign a consent form made in simplified local language.

**Training Modules**

Training workshops have been held for the medical officers, social workers and data entry operators before the registry started functioning.

**Web Development**

All Principal Investigators can enter the data of the registry online from their respective sites with the help of a software web which is in place.

**Quality Assurance**

IARC quality control programme is used to validate the data. The internal quality control of their respective sites is done by the Principal Investigators. He/ She ensures proper filling up of the form as well as online entry of the data. Consistency checks for various parameters in the proforma are done. External quality control is being done by the Central Coordinating unit of ICMR by randomly picking up 5% of the recorded cases and reabstracting and comparing with the earlier data. The data is checked and subjected to analysis by a statistician.

In an effort to include as many cases as possible, all Regional...
Institutes of Ophthalmology located in different parts of the country are being involved with the Registry.

This project was started in April 2009, and to achieve the desired goals, the National Retinoblastoma Registry needs the support and co-operation of all ophthalmologists, who have diagnosed a child with retinoblastoma, to ensure registration of the case at a designated site nearest to their area of practice. If needed, Retinoblastoma (RB) proformas can be downloaded from the site www.icmr.nic.in/downloadforms.

Retinoblastoma helpline no. 011-26588562, 9810258631.

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**Delhi Ophthalmological Society**

**Monthly Clinical Meeting, August 2010**

**Venue:** Auditorium Sir Ganga Ram Hospital, Rajinder Nagar, New Delhi

**Date & Time:** Sunday 29th August 2010, 10:30 a.m.

**Annual General Body Meeting**

9:30 a.m.

**Tea:** 10:30 - 11:00 a.m.

**Scientific Meeting**

10:30-11:00 a.m. Onwards

**Clinical Cases:**

1. Management of severe post surgical eyelid retraction
   - Discussant: Dr. Shaloo Bageja
   - Dr. Swapna Parekh 10 min

2. TPA assisted treatment of Choroidal Neovascular Membrane
   - Discussant: Dr. S.N. Jha
   - Dr. Rohini Grover 10 min

**Clinical Talk:**

Correction of pre-existing astigmatism in Phacoemulsification
- Dr. Harbansh Lal 15 min

**Mini Symposium: Tips & Tricks in Ophthalmic Surgery**

**Chairman:** Dr. A.K. Grover

**(Video Assisted Programme)**

**Panelists:**
- Dr. H.K. Tewari
- Dr. Harbansh Lal
- Dr. S.N. Jha
- Dr. Amit Khosla
- Dr. Neeraj Manchanda
- Dr. Shaloo Bageja
- Dr. Ashima Chandra
- Dr. Tinku Bali
- Dr. Nidhi Tanwar
- Dr. Nagender Vashisht

**20 Early Bird Prizes**

**Lunch 1:00 P.M.**

Sponsored: M/s. Shrey Nutraceuticals & Herbals Pvt. Ltd. New Delhi
Prostaglandins, also known as autacoids, are a class of local hormones exerting multiple effects through several types of receptors. They have several favorable characteristics that render them as first choice drugs for various types of glaucomas, especially primary open angle glaucoma and chronic angle closure glaucoma (post laser iridotomy).

**Background**

In 1978, timolol maleate was introduced as the first topical anti-glaucoma medication. Thereafter in 1980s and early 90s several selective and non-selective beta-blockers were introduced into the market. Timolol became the gold standard against which all other drugs were compared for their IOP lowering ability. The non-selective alpha adrenergic agonist apraclonidine and the selective brimonidine were introduced in 1988 and 1996 respectively. Dorzolamide as the first topical carbonic anhydrase inhibitor became available for use in 1995. The effects of prostaglandins on eye were first reported in 1985 as tromethamine salt of PG F2 alpha which produced sustained IOP lowering for > 24 hours. It was however associated with severe conjunctival hyperemia and discomfort. Less polar substitutes like 17-phenyl PG F2 alpha had increased corneal penetration and significant IOP lowering with minimal discomfort and hyperemia. The more potent R-epimer of the above received US FDA approval in 1996 as latanoprost.

**Mechanism of action**

Prostaglandin analogs act primarily by increasing the uveoscleral outflow and also produce a variable increase in the trabecular outflow.

The various mechanisms proposed by which prostaglandins increase the uveoscleral outflow include the following:

- Remodelling of the extra cellular matrix of the ciliary muscle and the sclera causing changes in the permeability of these tissues and widening of the connective tissue spaces among ciliary muscle bundles. This is caused by dissolution of collagen 1 and 3 by alterations induced in the concentrations of matrix metalloproteinases and tissue inhibitor of matrix metalloproteinase.
- Ciliary muscle relaxation which also widens the connective tissue spaces. This is responsible for the initial fall in IOP with topical Prostaglandins.
- Changes in the shape of the ciliary muscle fibres caused by relocalisation of actin and vinculin in the muscle cells.

Prostaglandin analogs also produce a variable increase in the trabecular outflow by directly stimulating matrix metalloproteinase and neutral protease induced extra cellular matrix degradation.

**Individual Agents**

Latanoprost and Travoprost are ester prodrugs of 17-phenyl PGF2 alpha. These are converted by corneal hydrolases to their respective free acids in the corneal epithelium. The esterification makes them more lipid soluble and less polar thereby increasing their corneal permeability. The respective free acids then bind to specific PGF 2 receptors in the trabecular meshwork and the ciliary body in turn increasing the aqueous outflow through these routes. Latanoprost requires maintenance of cold chain prior to uncapping of the bottle and thereafter, has a limited shelf life of 44 days at room temperature. Travoprost has no such considerations. Travoprost is also available as a benzalkonium chloride preservative free travoprost 0.004% ophthalmic solution probably having a better side effect profile (Travoprost Z). Bimatoprost on the other hand is an amide prodrug of 17-phenyl PGF2 alpha and hence characterized as a prostamide. It is

<table>
<thead>
<tr>
<th>Table 1 Currently available Prostaglandin analogs:</th>
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<tr>
<td><strong>FP Receptor analogs, amides</strong></td>
</tr>
<tr>
<td>1. Latanoprost</td>
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<tr>
<td>2. Travoprost</td>
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<tr>
<td>3. Bimatoprost</td>
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<tr>
<td>4. Unoprostone</td>
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<tr>
<td>5. Tafluprost</td>
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<tr>
<td><strong>DP Receptor agonist</strong></td>
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<tr>
<td>1. AL-6598</td>
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<tr>
<td><strong>EP 2 Receptor agonist</strong></td>
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<tr>
<td>1. Butaprost</td>
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<tr>
<td>2. 8-iso PGE2</td>
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<tr>
<td>3. 17-phenyl trinor 8-iso PGE2</td>
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<tr>
<td><strong>EP4 Receptor agonist</strong></td>
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<tr>
<td>1. 3,7-dithia PGE1</td>
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</tbody>
</table>
converted to its free acid, Bimatoprost acid which is a potent stimulator of PGF2 receptor. It is also postulated that Bimatoprost may act through a novel prostamide receptor. Bimatoprost acid is 3 to 10 times as potent as latanoprost acid. In spite of this, the therapeutically used concentration of Bimatoprost is 6 times that of latanoprost. This is because of the slow conversion of bimatoprost to Bimatoprost acid.

The recommended dosing regimen for Latanoprost, Travoprost and Bimatoprost is once daily topical application preferably in the evening to reduce the early morning diurnal spike. Studies have shown that all the available classes of prostaglandins have excellent 24 hour IOP control despite once a day dosing making it extremely patient convenient. In fact, multiple dosing has been shown to reduce the IOP lowering effect.

Unoprostone is a docosanoid. It is a pulmonary metabolite of PGF2 alpha. It is much less potent compared to the previous three prostaglandin analogs with an IOP lowering effect of up to 18% from baseline. It has minimal effect on uveoscleral outflow in humans and mainly acts by increasing the trabecular outflow. It is available in concentrations of 0.12% and 0.15% for a twice daily (b.d.) dosing.

Tafluprost is a new difluoro prostaglandin analog undergoing clinical trials in Japan. Animal studies have shown it to act by increasing the uveoscleral outflow. In addition, it may also act by stimulating PGF receptor mediated endogenous prostaglandin production in turn acting via prostanooid EP3 receptor. It is approved in some countries (Denmark since April 2008 and Germany since May 2008) as a preservative free 0.0015% solution for once daily dosing.

In general, the reduction of IOP starts approximately 2 to 4 hours after the first administration of the prostaglandin analog with the peak effect reached in about 8 to 12 hours. Maximum IOP lowering effect is achieved 3 to 5 weeks from commencement of treatment.

Butaprost and other EP agonists are being investigated for their therapeutic potential in animal studies.

Indications

- Primary open angle glaucoma – Latanoprost, travoprost and Bimatoprost have all received the EMEA and FDA approval as first line agents for treatment of open angle glaucoma or ocular hypertension.
- Normal tension glaucoma
- Chronic angle closure glaucoma
- Pigment dispersion syndrome
- Exfoliation syndrome

There are limited reports of clinical experience of these drugs in other types of glaucomas.

Contraindications and cautious use:

- Patients allergic/sensitive to prostaglandins
- Pregnant or nursing mothers
- Children – responses may be inadequate in some
- Uveitic glaucomas
- Patients with iritis
- Patients with active or healed herpes simplex keratitis
- Immediate post operative period following any intraocular surgery
- Patients with risk factors for cystoid macular edema like aphakia, pseudophakia with torn posterior capsule, history of uveitis, retinal inflammation or vascular diseases.
- Patients should not administer these drugs while wearing contact lenses, but contact lenses can be reinserted 15 minutes following administration of the drugs.

Side effects

The well documented and more common local side effects with prostaglandin therapy include:

- Conjunctival hyperemia, burning and stinging.
• Elongation and darkening of eye lashes.
• Induced iris darkening.
• Periocular skin pigmentation.

The former two are less frequently seen with latanoprost compared with bimatoprost and travoprost. The mechanism for the latter two is increase in the melanin content of melanocytes due to increase in the size or the number of melanin granules in the cytoplasm of the melanocytes. Induced iris darkening is more commonly seen in hazel irises that have mixed colouring and is usually irreversible. On the other hand, periocular skin pigmentation involving mostly the eyelid skin is usually reversible. The above side effects are an important consideration prior to starting a patient on prostaglandin analogs for unilateral glaucomas.

The less common but more sight threatening complications include:
• Iris cysts
• Anterior uveitis
• Cystoid macular edema
• Reactivation of herpes simplex keratitis.

Due to their low therapeutic concentration and rapid systemic inactivation, topical prostaglandins have an excellent systemic safety profile. Dyspnea, asthma and exacerbation of asthma are the systemic complications that have been identified during the post marketing use of topical prostaglandin analogs in clinical practice. As they are voluntarily reported from a population of unknown size, estimates of their frequency cannot be made.

Pregnancy and Nursing Mothers

There are no adequate and well controlled studies on topical prostaglandin use in pregnant women. At present, they may be used during pregnancy only if potential benefit justifies the potential risk to fetus. Whether the drugs or their metabolites are excreted in breast milk is also not known.

Class Interswitch among Prostaglandins

Studies comparing the interclass switching among prostaglandins have shown good IOP control, feasibility and tolerability on class switch from latanoprost to bimatoprost and latanoprost to travoprost. Clinical experience with this class of drugs shows that often patients unresponsive or intolerant to one class of prostaglandins benefit from switching over to another class of prostaglandin analogs. The wash out time for most of the prostaglandin analogs varies from 4 to 6 weeks.

Drug interaction and drug additivity

Topical preparations containing thiomersal as preservative form a precipitate with latanoprost and hence must be instilled with a gap of at least 5 minutes between the two.

Clinical studies have demonstrated additive effects of prostaglandins with all other anti glaucoma medications including beta blockers, alpha adrenergic agonists, carbonic anhydrase inhibitors and cholinergic agonists.

As the non selective beta blockers and prostaglandin analogs may both be effective with od dosing, fixed drug combination of timolol 0.5% with each of the three frontline prostaglandin analogs are commercially available. These have been shown to have significantly more IOP lowering effect than either drug used alone. Also, the IOP reduction achieved is not significantly less than concomitant treatment with prostaglandin analog od and timolol bd.

Prostaglandin analogs HEAD to HEAD

A meta-analysis would be a reliable means of comparing the IOP lowering ability of the various ocular hypotensive medications. The results of the meta-analyses assessing the efficacy of the three frontline prostaglandin anlogs are summarized in table 4.

It is notable that three independently performed meta-analyses found the three prostaglandin analogs to have equivalent IOP lowering ability.

Adherence, Persistence, Endurance and Tolerability among Prostaglandin analogs

‘Adherence’ in simple terms refers to the extent to which the patient behaviour conforms to the health care provider’s prescription. Having recently replaced the term ‘compliance’ to highlight the patient’s responsibility and involvement in their care, it plays an

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<td>5⁵¹</td>
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<tr>
<td>7⁵³</td>
<td>PACG</td>
<td>9</td>
<td>Independent</td>
<td>Lat=Trav=Bimat</td>
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</table>

(Lat = Latanoprost; Trav = Travoprost; Bimat = Bimatoprost; POAG = Primary Open Angle Glaucoma; OH = Ocular Hypertension; PACG = Primary Angle Closure Glaucoma)
important role in chronic diseases such as glaucoma. ‘Persistence’ on the other hand, refers to continued following of treatment orders over a period of time such as refilling of prescriptions.

Studies have shown that both adherence and persistence to topical ocular hypotensive therapy are poor. Among the available agents, prostaglandins have been reported to have higher rates of persistence than other classes. Among the three major prostaglandin analogs, latanoprost users were reported to have greater odds of achieving medication possession and had more days covered in the first therapy year compared to travoprost and bimatoprost. Since compliance to medication is poor and patients often miss medications for a 24 hr period, a sustained action beyond 24 hrs is beneficial. A study on 24hr IOP control with travoprost found that the molecule had a sustained IOP lowering capability even after omission of one or two doses, especially at night time.

As regards tolerability, a recent meta-analysis comparing the three prostaglandin analogs reported a significantly lower conjunctival hyperemia with latanoprost and travoprost compared to bimatoprost. Moreover, in a recent study comparing the preservative free travoprost with the BAK-preserved prostaglandins found significantly better ocular surface disorder profile, decreased hyperemia and equal or better IOP control with the preservative free travoprost.

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<td>Ioprost 2.5</td>
<td>199</td>
<td>FDC</td>
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<tr>
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<td>Latochek 2.5</td>
<td>220</td>
<td>Indoco</td>
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<tr>
<td></td>
<td>Ioptame 2.5</td>
<td>264</td>
<td>Cadila</td>
</tr>
<tr>
<td></td>
<td>Latodrops 3.0</td>
<td>275</td>
<td>Intas</td>
</tr>
<tr>
<td></td>
<td>Latoprost 2.5</td>
<td>310</td>
<td>Sun</td>
</tr>
<tr>
<td></td>
<td>9 PM 2.5</td>
<td>343</td>
<td>Cipla</td>
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<tr>
<td></td>
<td>Xalatan 2.5</td>
<td>1142</td>
<td>Pfizer</td>
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<tr>
<td>Travoprost 0.004% Brands</td>
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<td>Micro</td>
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<td>Travatan 2.5</td>
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<tr>
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<td>Careprost 3.0</td>
<td>210</td>
<td>Sun</td>
</tr>
<tr>
<td></td>
<td>Intaprost 3.0</td>
<td>350</td>
<td>Intas</td>
</tr>
<tr>
<td></td>
<td>Lumigan 3.0</td>
<td>433</td>
<td>Allergan</td>
</tr>
<tr>
<td>Latanoprost 0.005% + Timolol 0.5% FDC Brands</td>
<td>Latochek – T 2.5</td>
<td>250</td>
<td>Indoco</td>
</tr>
<tr>
<td></td>
<td>Latocom 2.5</td>
<td>307</td>
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<td>Xalacom 2.5</td>
<td>1320</td>
<td>Pfizer</td>
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<td>Travocom 2.5</td>
<td>695</td>
<td>Alcon</td>
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<td>Careprost plus 3.0</td>
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<tr>
<td></td>
<td>Ganfort 3.0</td>
<td>445</td>
<td>Allergan</td>
</tr>
</tbody>
</table>
Generic Prostaglandins

There are a number of low cost prostaglandins now available in India (Table 5). There are hardly any published studies on the efficacy of these drugs. Narayanaswamy A et al18 from Sankara Netralaya, Chennai evaluated the efficacy and tolerability of Xalatan with generic latanoprost (Laptoprost) in subjects with primary open angle glaucoma (POAG) or ocular hypertension (OH) in 30 subjects. In subjects administered Xalatan, intraocular pressure (IOP) showed a greater decrease (P < 0.001) from 23.64 +/- 3.13 mmHg at baseline to 14.29 +/- 1.61 mmHg at week 12 (fall of 9.35 +/- 3.55 mmHg, 38.66% +/- 10.29) than that seen in the Laptoprost group (22.74 +/- 2.47 mmHg to 16.98 +/- 2.49 mmHg, fall of 5.76 +/- 1.41 mmHg; 25.42% +/- 5.98). In period 2 when subjects were crossed over to Xalatan from Laptoprost, there was a further fall from 16.98 +/- 2.49 mmHg to 16.09 +/- 1.49 at week 24 (fall of 0.89 +/- 1.59 mmHg; 4.3% +/- 8.76). However, when subjects were crossed over to Laptoprost from Xalatan, the IOP rose from 14.29 +/- 1.61 mmHg to 15.36 +/- 1.71 mmHg at week 24 (8.86% +/- 17.76). The authors concluded magnitude of IOP lowering in patients with POAG and OH with Xalatan and Laptoprost is different. The authors concluded magnitude of IOP lowering in patients with POAG and OH with Xalatan and Laptoprost was different and IOP lowering with Xalatan was higher than that with Laptoprost.

In the current economic scenario in our country, patients may be prescribed generic drugs but the practitioner should check the IOP lowering efficacy (% IOP reduction) on a case to case basis to determine the best fit for life long therapy.

Conclusion

As a class, prostaglandin analogs have several unique properties. These agents act by increasing the uveoscleral outflow which is different from that of other anti glaucoma drugs which either decrease aqueous production or increase the trabecular outflow. This novel mechanism of action empowers them to potentially lower the IOP below the episcleral venous pressure – a potential advantage in Normal Tension Glaucoma. Also this makes these agents, for glaucoma management. Expert Opin Investig Drugs 2001;10:721—31.


References


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Young male 30 yrs old reported in Emergency with history of road traffic accident. He was in the front seat of car and took the brunt of broken window pane glass on whole face, as he was not wearing seat belt.

At the time of admission, he was semi conscious, with multiple injuries on face and eye.

- Tetanus prophylaxis + topical and systemic antibiotics
- IV steroids for suspected traumatic optic neuropathy in left eye
- Detailed documentation with informed high risk consent
- Ultrasound B Scan of eyes after primary reconstructive surgery to evaluate posterior segment and decide further course of action

**Ocular Examination**

<table>
<thead>
<tr>
<th>Top</th>
<th>Right Eye (Figure 1)</th>
<th>Left Eye (Figure 2a &amp;b)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual Acuity</td>
<td>PL only, PR doubtful</td>
<td>PL, PR accurate, HM close to face</td>
</tr>
<tr>
<td>Eyelids</td>
<td>Edema++, ecchymosis</td>
<td>Edema, Lower lid full thickness laceration</td>
</tr>
<tr>
<td>Globe</td>
<td>Full thickness scleral perforation with uveal + vitreous prolapse</td>
<td>Full thickness scleral perforation with uveal + vitreous prolapse</td>
</tr>
<tr>
<td>Cornea</td>
<td>Nasal Perforation, Oedema</td>
<td>Clear</td>
</tr>
<tr>
<td>Ant chamber</td>
<td>Shallow, Hyphaema</td>
<td>Formed /Pupil sluggish reaction</td>
</tr>
<tr>
<td>Lens</td>
<td>No View</td>
<td>Clear</td>
</tr>
<tr>
<td>Fundus</td>
<td>No View</td>
<td>Disc normal, Macular Edema</td>
</tr>
</tbody>
</table>

**CT Scan – Orbit** (Figure 3): Multiple foreign bodies in superficial adnexa in right eye. Two large foreign bodies (glass) in lateral orbit of left eye.

**Plan**

- A combined reconstructive plastic and ophthalmic surgery was planned under general anesthesia.

**Peroperative**

**Right Eye** (Figure 4): Exploration of the wound was done to see the extent of corneoscleral perforation. Superficial vitrectomy + repositioning of choroidal tissue + corneo. scleral wound repair was done. Anterior chamber was washed and formed with saline. Multiple tiny glass foreign bodies removed from lids, conjunctiva and tenon's.

**Left Eye** (Figure 5a & b): Removal of glass foreign bodies from lateral orbit followed by superficial vitrectomy + repositioning of choroidal tissue + scleral wound repair was done. Lower lid full thickness repair was done.

**Postoperative Ultrasound** (Figure 6) – After primary repair, patient was undertaken for B Scan Ultrasound to evaluate posterior segment. There was vitreous haemorrhage but no retinal detachment.

**Postoperative -1 month**

**Right Eye** (Figure 7): The corneoscleral wound had healed well but there was no fundus glow due to Traumatic Cataract formation by this time. At the site of nasal corneal perforation, there was iris tissue loss with suspected zonular dialysis.

**Left Eye** (Figure 8): The lower lid was well apposed to globe with no ectropion. Scleral wound was also healing well. Posterior segment evaluation was normal.
Right Eye (Figure 9): Phacoemulsification with foldable intraocular lens with capsular tension ring support was done under local anesthesia.

Postoperative-2 months (Figure 10)

Right Eye: Vision restored to 6/18p with glasses, Pseudophakia, IOL stable, c/o glare due to mid dilated atonic pupil. Resolving vitreous haemorrhage - PVD, opacities/strands, No retinal breaks. Tinted soft contact lens trial given to avoid glare.

Left Eye: Vision restored to 6/6, normal anterior and posterior segment

Discussion

Severe perforating injuries often have a poor prognosis for both recovery of visual function and salvage of the eye. Initial surgery should be directed towards optimal repair of the perforating wound, correction of damage caused by the injury and prevention of secondary complications. Even severe injuries should undergo at least one full hearted attempt at repair as they may have a chance to restore some visual function.

Evaluation and surgical repair are best performed under general anesthesia due to advantages of akinesia, anesthesia, reduced intraocular and orbital pressure.

Few key points of surgical repair are listed as following:

Corneal/Scleral Perforation repair:

• Meticulous exploration of the wound to assess the extent of injury.
• Tissue adhesive with bandage contact lens can be used if corneal perforation is very small.
• Prolapsed iris may be preserved and reposited back if the look is viable. In case of old injuries or infection, it should be excised.
• Vitreous incarceration should be relieved by anterior vitrectomy to avoid risk of enophthamitis, chronic inflammation, cystoid macular edema and retinal detachment.
Figure 5(a): Perop-Large glass foreign bodies removal

Figure 5(b): Perop-left eye-after repair

Figure 6: B Scan Ultrasound-vit hage,no RD

Figure 7: RE-1 month post op-traumatic cataract with dialysis

Figure 8: LE-1 month post op

Figure 9: RE-Phacoemulsification-with CTR support-IOL implant in bag
10-monofilament is used to repair corneal perforations and the bite should pass through 2/3rd of corneal thickness. Large bites are taken (1.5 to 2mm) from edges so that the edematous edges are well approximated. The knots should be rotated and buried.

In a corneoscleral perforation, the first sutures placement is at limbus to realign.

Donor scleral graft should be arranged in OT in case the wound gape is large with loss of tissue

Scleral perforations are sutured with spatulated needle 8-0 silk or ethibond. Some use 6-0 vicryl also. Scleral sutures should be 75-90% deep, with entry and exit at least 1 mm from the wound edge. Start anterior and progress posterior, so the wound is more stable when you need to rotate the globe for posterior access.

Posterior scleral perforations should be handled by posterior segment surgeon as they are mostly accompanied by retinal trauma.

Avoid ointment at end of surgery as it can get entrapped in repaired lacerated corneal tissue and delay healing.

Full thickness eyelid laceration repair:

- If the margins are smooth they can be simple approximated. If the vertical cut is ragged or there is loss of tissue, excise in a pentagonal fashion.

- 6-0 vicryl or silk on spatulated needle is used to reapproximate the gray line to achieve correct approximation of the margin edges. Place two additional 6-0 silk suture anterior and posterior to initial grey line suture. This prevents any notch formation in the eyelid

- Keep the knot away from the eyeball because irritation or potentially an ulceration may result if the knot rubs on the conjunctiva or cornea.

- Use two 6-0 vicryl suture bites to reapproximate the tarsal plate and or orbicularis muscle in one or two layer.

- Close the skin with 6-0 silk or nylon

Overview:

- No matter how serious the ocular injuries look at presentation, always give a full hearted attempt for meticulous repair

- CT Scan vs MRI: CT Scan is the choice of radiography in ocular trauma-detailing of bony margins, globe integrity, muscles, optic nerve status, presence and location of foreign bodies, can all be detected on fine 1-2 mm axial and coronal cuts.

- Always explore under GA- advantages of akinesia, anesthesia, reduced intraocular and orbital pressure

- Role of steroids in traumatic optic neuropathy- IV steroids- methyl prednisolone-mega pulse therapy for 3 days-effectiveness is unproven, but may be given in the absence of contraindications.

- Visual & PSYCHOLOGICAL rehabilitation – Loss of vision, disfigurement, or opting for prosthesis can all be psychologically traumatic for the patient. Surgeon should not only help in visual rehabilitation but also give positive counseling and encouragement for him to cope up in his life.

Note: The patient was not wearing seat belt and hence sustained grievous injuries due to the sudden impact. Let this also be a reminder to all our colleagues that the use of seat belt in car is a must for everybody. Most of the injuries can be prevented by following the safe driving guidelines... The adage “an ounce of pre-vention is worth a pound of cure” is most apt for ocular trauma.
# Forthcoming Events: National

**October 2010**

**22-24 UTTARAKHAND**

NZOS & Uttara Eyecon 2010  
(Combined Annual conference of North Zone Ophthalmological Society and Uttarakhand State Ophthalmological Society)  
**Venue:** Hotel Park Plaza, Mall Road, Mussorie, Uttarakhand  
**Organizing Secretary:**  
Dr. B.K.Oli  
57, Haridwar Road, Dehradun-248001  
**Phone:** 0-99971-22222, 0-94123-19035  
**Email:** dr.bkoli@gmail.com

**November, 2010**

**12-14 NEW DELHI**

20th Annual Conference of Glaucoma Society of India & 5th International Congress on Glaucoma Surgery  
**Venue:** Le Meridien Hotel, Janpath, New Delhi & India Habitat Centre, Lodhi Road, New Delhi  
**Conference Secretariat:**  
Dr. Harsh Kumar  
D-8/8127, Vasant Kunj, New Delhi-70  
(M): 9810442537, Tel.: 91-11-4519910, 25513051, Fax: 91-11-26122053

**December 2010**

**2-4 MYSORE**

19th Annual Conference of Vitreo Retina Society - India 2010  
**Organizing Secretary:**  
Retina Institute of Karnataka  
#122, 5th Main Road  
(Next to Venlakh Hospital)  
Chamarajpet, Bangalore - 18  
**Ph.:** +91-80-22410106 / 536 (Hospital),  
**Fax:** +91-80-26607811  
**E-mail:** retinainstitute@sify.com

**January, 2011**

**28-31 NEW DELHI**

10th Annual Meeting of Uveitis Society of India  
**Venue:** Advanced Eye Centre, PGI Chandigarh  
**Contact Person & Address**  
Dr. Vishali Gupta  
ext: vishalisara@yahoo.co.in  
www: http://www.usi2010.in

# Forthcoming Events: International

**September, 2010**

**2-5 PARIS**

10th Euretina Congress  
**Venue:** Le Palais des Congrès, Porte Maillot, Paris, France  
**Euretina Secretariat**  
European Society of Retina Specialists  
Temple House, Temple Road, Blackrock, Co. Dublin, Ireland  
**Tel:** + 353 1 2100092  
**Fax:** + 353 1 2091112  
**Website:** www.euretina.org  
**Email:** euretina@euretina.org

**September, 2010**

**4-8 PARIS**

XXVIII Congress of the ESCRs  
**Venue:** Le Palais des Congrès, Porte Maillot, Paris, France  
**Organiser Secretary**  
ESCRS, Temple House, Temple Road, Blackrock, Co. Dublin, Ireland.  
Tel: +353 1 209 1100 Fax: +353 1 209 1112  
Email: maria.crowley@escrs.org  
Web: www.escrs.org | www.euretina.org
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**SPECIFICATION of DOS Times:**

- Size of Advertisement page: 7.5” x 10.25”
- Frequency of DOS Times: Monthly (10 issues in a year) (July – April)
- Frequency of Delhi Journal Ophthalmology: Monthly (4 issues in a year) (July – April)
- Model of Printing: Offset
- Advertisement Material: CDR open file with fonts & proof
- Mailing and Contact: Delhi Ophthalmological Society
  Room No. 2225, 2nd Floor, New Building
  Sir Ganga Ram Hospital,
  Rajinder Nagar, New Delhi - 110060.
  Ph. 011-65705229

**Email:** dosrecords@gmail.com, dostimes@airtelmail.in
(LIFE MEMBERSHIP FORM)

Name (In Block Letters) _______________________________________________________________________________________________
S/D/Wo ____________________________________________________________________________  Date of Birth ___________________
Qualifications ________________________________________________________________________  Registration No. ________________
Sub Speciality (if any) ________________________________________________________________________________________________

ADDRESS

Clinic/Hospital/Practice ________________________________________________________________________________________________
Phone _______________________
Residence ________________________________________________________________________________________________
Phone _______________________
Correspondence ________________________________________________________________________________________________
Phone _______________________
Email ________________________ Mobile No. ________________________

Proposed by
Dr. ________________________ Membership No. __________ Signature ________________________

Seconded by
Dr. ________________________ Membership No. __________ Signature ________________________

[Must submit a photocopy of the MBBS/MD/DO & State Medical Council / MCI Certificate for our records.]

I agree to become a life member of the Delhi Ophthalmological Society and shall abide by the Rules and Regulations of the Society.
(Please Note: Life membership fee Rs. 3100/- payable by DD for outstation members. Local Cheques acceptable, payable to Delhi Ophthalmological Society)

Please find enclosed Rs.___________ in words ____________________________________________________ by Cash
Cheque/DD No.____________________ Dated___________ Drawn on________________________

Signature of Applicant with Date

Three specimen signatures for I.D. Card.

FOR OFFICIAL USE ONLY

Dr. ________________________ has been admitted as Life Member of the Delhi Ophthalmological Society by the General Body in their meeting held on ________________________________
His/her membership No. is ________________. Fee received by Cash/Cheque/DD No.____________________ dated___________
drawn on ________________________________.

(Secretary DOS)
INSTRUCTIONS

1. The Society reserve all rights to accept or reject the application.

2. No reasons shall be given for any application rejected by the Society.

3. No application for membership will be accepted unless it is complete in all respects and accompanied by a Demand Draft of Rs. 3100/- in favour of “Delhi Ophthalmological Society” payable at New Delhi.

4. Every new member is entitled to receive Society's Bulletin (DOS Times) and Annual proceedings of the Society free.

5. Every new member will initially be admitted provisionally and shall be deemed to have become a full member only after formal ratification by the General Body and issue of Ratification order by the Society. Only then he or she will be eligible to vote, or apply for any Fellowship/Award, propose or contest for any election of the Society.

6. Application for the membership along with the Bank Draft for the membership fee should be addressed to Dr. Amit Khosla, Secretary, Delhi Ophthalmological Society, Room No. 2225, 2nd Floor, New Building, Sir Ganga Ram Hospital, Rajinder Nagar, New Delhi - 110 060

7. Licence Size Coloured Photograph is to be pasted on the form in the space provided and two Stamp/ Licence Size Coloured photographs are required to be sent along with this form for issue of Laminated Photo Identity Card (to be issued only after the Membership ratification).

8. Applications for 'Delhi Life Member' should either reside or practice in Delhi. The proof of residence may be in the form Passport/ Licence/Voters Identity Card/Ration Card/Electricity Bill/MTNL (Landline) Telephone Bill.
Diabetic Maculopathy

**Exudative Maculopathy / CSME**

- DME (Controlled Systemic Parameters) (on FFA)
- Ischemic Maculopathy
- Mixed

**Ischemic maculopathy (on OCT)**
- without edema: No treatment
- with edema: Follow the protocol of Exudative Maculopathy

**Focal Leak**
- > 500 μm away from FAZ
  - Focal Laser
  - Review x 3 months if leak persists
  - Follow same protocol

**Diffuse Leak (DDME)**
- < 500 μm away from FAZ
  - If FFA is combined with OCT, it would result in better monitoring
  - PST steroids / IVTA / IVA
  - followup x 4 weeks

**OCT**
- Spongiform Edema
- Cystoid, Edema
- Neurosensory Detachment

**IVTA / IVA**
- without traction
  - Macular grid ± IVTA / IVA
  - followup x 4 weeks

**with traction**
- Vitreo-macular traction syndrome
- Epi-retinal membrane

**IVA / IVTA**
- If vision < 6/18 then PPV
- If vision > 6/18 then followup / IVTA

*IVA*: Intravitreal anti VEGF Agents
*IVTA*: Intravitreal Triamcinolone Acetonide

**Laser Treatment is the only gold standard treatment available for diabetic macular edema**

1. Naginder Vashisht MD, 1. Amit Khosla MD, 1. Sanjeev Gupta MD,
2. Sir Ganga Ram Hospital, Rajender Nagar, New Delhi
3. Siri Fort Laser Eye Centre, New Delhi