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Focus Collagen Cross Linking Macular Hole Surgery Robotic Eye Surgery Ocular Blood Flow in Glaucoma





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Editorial

Dear Friends,

Keratoconus is a degenerative non inflammatory disease of the cornea with onset generally at puberty. It is progressive in 20% of cases and can be treated by lamellar or penetrating keratoplasty. Its incidence in the general population is reported to be about 1 in 2000.

The technique of corneal collagen cross-linking consists of photo polymerization of stromal fibers by the combined action of a photosensitizing substance (riboflavin or vitamin B2) and ultraviolet type A rays (UVA) from a solid-state UVA source. Photo polymerization increases the rigidity of corneal collagen and its resistance to keratectasia. Refractive results showed a reduction of about 2.5 D in the



mean spherical equivalent, topographically confirmed by the reduction in mean K. The aim was to slow or arrest progression to delay or avoid recourse to penetrating keratoplasty.

The basis for its use finds clinical and scientific support in the fact that young diabetic patients never have keratoconus; it predated the onset of diabetes and did not progress due to the natural cross-linking effect of glucose, which increases corneal resistance in these patients.

Collagen turnover is about 2 to 3 years. Cross-linking "freezes" stromal collagen, increasing the biomechanical stability of the cornea.

The method of corneal cross-linking using riboflavin and UV light is technically simple and less invasive than all other therapies proposed for keratoconus, and unlike other mini-invasive methods, such as intrastromal rings (INTACS) and excimer laser surgery, that does not block keratectasia but merely treat the refractive effects of the disease, it treats and prevents the underlying patho physiological mechanism.

Study	Ν	Age	Indication	Technique	Parameters	FU	Result	Side effects
Wollensak et al AJO2003	23	13-58 yrs	Mod. to severe keratoconus (k-48-72)	0.1% RF every 5min,UVA 3mW/cm ² at 1cm	BCVA VKG IOP Endothelial density	3-47 Mnth.	BCVA improved in 65%, K decreased in 70%	Transient stromal edema until 3days
Wollensak et al Ophthalmologe 2003	16		Progressive keratoconus (k 48-72)	Similar as above	Visual acuity corneal topography endothelial count	1-3yrs	Progression stopped in all eyes, BCVA and max k improved in 50%	none
Caporossi et al JCRS2006	10	31.4 yrs	Progressive keratoconus	3mW/cm² UVA at1cm	UCVA ,BCVA Topography Optical tomography Endothelial count, Pachymetry,IOP confocal	6mths	Improvement of 3.6 lines UCVA 1.6 lines BCVA k reduction by 2.1 in central cornea	
Wollensak et al (Current opinion Ophthalmol 2006)	60		Progressive keratoconus	0.1% RF-UVA 3mW/cm2	BCVA,VKG, IOP, Endothelial count, CP	3-5yrs	Progression stopped in all eyes 31 reversal of flattening by 2.87D	None
Mazotta et al (Cornea 2007))	10	18-60 yrs	Progressive keratoconus	0.1% RF-UV 3mW/cm ²	HRT-2 Confocal microscopy	6mths	1mth-decrease in keratocytes, 6mth-repopulation	none
Farhad et al (JCRS 2007)	10	27-43 yrs	Post LASIK keratectasia	0.1%riboflavin, UVA 3mW/cm ²	BCVA Topography Schiemplug imaging IOP	25mths	Decrease in max k in all signi- ficant decrease of 2D in 50%	Endothelial opacity in 1case

There are cases of haze formation reported in literature after collagen cross linking. Mazzotta et al (2007) studied two cases through in vivo confocal microscopy with stage III keratoconus that developed stromal haze after cross linking treatment and concluded that detection of reticular hypo-reflective microstriae by in vivo confocal microscopy , with or without Vogt's striae represent a relative contraindication to perform a riboflavin-UVA-induced corneal cross linking. In fact in these cases there is a high risk of development of late stromal scarring during the post operative course. Although the haze does not seem to effect the visual acuity, it is not possible to exclude this complication without longer follow-ups.

CONCLUSION

Cross linking treatment of keratoconus is a new method of treating keratoconus. At the present stage of knowledge, the treatment should only be performed in patients with documented progression of keratoconus (rapidly changing refraction and keratometry) in the preoperative months. With more long term experience, prophylactic treatment of keratoconus at an early stage might become possible. To avoid serious side effects it is mandatory in each patient to perform pre-operative pachymetry to exclude extended areas with less than 400um stromal thickness, and to check the UVA radiance before each treatment using a UVA-meter.

Thanking you,

Namrata Sharma

Secretary, Delhi Ophthalmological Society

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Collagen Cross Linking in Keratoconus



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Keratoconus is a degenerative, noninflammatory disease of the corneal stroma that is associated with decreased biomechanical strength of the tissue, probably caused by diminished intra- and interfibrillar cross-links of the collagen fibers. Usually the onset occurs at puberty. Incidence of keratoconus is 1/2000. It is progressive in 20% of cases and can be treated by lamellar or penetrating keratoplasty.

Recently, a new method has been developed for the treatment of progressive keratoconus, which currently is under clinical study: **Corneal collagen crosslinking with riboflavin / UVA.** The technique of corneal collagen cross-linking consists of photopolymerization of the stromal fibers by the combined action of a photosensitizing substance (riboflavin or vitamin B2) and UV light from a solid-state UVA source. Photopolymerization increases the rigidity of the corneal collagen and its resistance to keratectasia. Basically, cross-linking treatment markedly stiffens the cornea and increases the biomechanical strength by a factor of 4.5. To avoid potential irradiation damage to the corneal endothelium by UVA light, the technical parameters are set in a way that only the anterior 300 mm of the corneal stroma is treated.

We tried to find out how the above treatment could be optimally utilised for best results in patients of keratoconus. Dr. Gregor Wollensak (**GW**), introduced the technique for the first time. Dr. Farhad Hafezi (**FH**) has worked on collagen cross linking and published lot of work on it. Dr. Sudhank Bharti (**SB**), Dr. Mahipal S. Sachdev (**MSS**), Dr. S.P.S.Grewal (**SPSG**), Dr. J.S.Thind (**JST**), Dr. Rishi Mohan (**RM**) and Dr. Ajay Khanna (**AK**) have been using the technique in India for past few years. These leading ophthalmologists were asked for their opinion regarding the use of collagen crosslinking in the treatment of keratoconus.

GW: Dr. Gregor Wollensak, Department of Ophthalmology, Vivantes-Klinikum Neukölln, Berlin, Germany, FH: Dr. Farhad Hafezi, Associate Professor of Ophthalmology, University of Zurich, Editorial Board Member of the Journal of Refractive Surgery and the Iranian Journal of Ophthalmology. SB: Dr. Sudhank Bharti, Medical Director and Chief Consultant, Bharti Eye Foundation, Delhi, MSS: Dr. Mahipal S. Sachdev, Former Associate Professor at R.P. Centre, AIIMS, and now Chairman and Medical Director, Centre For Sight, Delhi. SPSG: Dr. SPS Grewal, Former Associate Professor, PGIMER and now Director, Grewal Eye Institute, Chandigarh, JST: Dr. J.S. Thind, Lasik and Phaco Surgeon, Jalandhar, RM: Dr. Rishi Mohan, Director, MM Eyetech Institute, Delhi, AK: Dr. Ajay Khanna, Director, Vitreo-retina and Refractive Surgery Dr. Om Parkash Eye institute, Amritsar.

Dr. Shubha Bansal (SB) DNB, who is working as an Senior Research Officer at Dr. Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi, interviewed them on the Collagen Cross Linking in Keratoconus.

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SB: What is your criteria of selection of patients?GW: The treatment should only be performed in patients with documented progression of keratoconus in the preoperative months.

- **FH:** We have the following inclusion criteria:
- minimal corneal thickness (without the epithelium) of 400 μm
- documented progression (topographies)
- cornea not scarred
- optical rehabilitation with contact lenses or glasses is possible
- prior treatment of mechanical underlying reasons, i.e. excessive eye rubbing
- **SB:** I would do Corneal Cross Linking in the following patients:



MS

We now use hypotonic Riboflavin (diluted in distilled water) in patients with thinner corneas. This causes hydration and swelling of the cornea and protects the corneal endothelium from UV-A radiation.

- Keratoconus & Pellucid Marginal Degeneration showing progress over a 6 months period on Topography.
- Post LASIK ectasia.
- MSS: I recommend collagen cross linking for all patients with Progressive keratoconus as I have found it to be a very safe and effective technique to halt the progression of disease. Though traditionally it has been recommended that progression should be documented on successive topographies over 6 months or more, I rely on clinical assessment and even a single topography showing a progressive steepening or a repeated change in glasses/ contact lenses is an indication for collagen cross linking. During the procedure 0.1% Riboflavin eye drops (Figure1) in Dextran solution are applied to the cornea for 30 minutes followed by exposure to UV-A light (365 nm) at 3mW/cm² to achieve cross-linkage of the corneal collagen fibres. This increases the bio-mechanical strength of the cornea by upto 300% and arrests the progression of keratoconus.
- **SPSG:** I would recommend collagen cross linking for a proven case of keratoconus between the age group 12 to 40 years. The thinnest corneal thickness should be more than 350 microns. It is strongly recomended in cases of keratoconus where Intacs of corneal graft is being planned.
- **JST:** Selection Criteria Post Refractive surgery Keratoconus, Progressive Keratoconus (Thinnest Cornea = / More than 400 microns), Pellucid marginal degeneration.
- **RM:** The major indication for Corneal Collagen CrossLinking is Progressive Keratoconus. All patients with classic parameters of keratoconus are candidates. Unfortunately, documentation of progression is not always available as many patients have inadequate old records and one has to take into account the history of visual loss & increase in astigmatism on the glasses prescriptions or history of rapid changes in the contact lenses.
- **AK:** I would do Corneal Cross Linking in the following patients:
- Progressive Keratoconus.
- Minimum corneal thickness 400 Microns or more (From 360 Microns to 400 Microns thickness, Corneal thickness is increased temporarily intra-operative with hydration using hypotonic solution).
- Steepest 'K' less than 65D.
- Progressive Iatrogenic Keratectasia.



Figure 1: Riboflavin/UV-A treatment in a patient with a double UV-A diode at 1 cm distance and the yellowish riboflavin on centrally abraded cornea.



Figure 2: Corneal scar in advanced keratoconus

SB: What is your exclusion criteria?

- **GW:** Preoperative pachymetry with less than 400mm stromal thickness.
- **FH:** Any condition that does not meet the above mentioned criteria.
- **SB:** Corneal thickness of less than 450 micron because a minimum thickness of 400 micron is safe so that the ultraviolet light does not cause any harm to Retina. In cases where the original thickness is less than 450 microns, after removal of epithelium the target thickness is 400 microns and should be achievable with hydration of cornea either with distilled water or with aqueous riboflavin solution.

now use hypotonic Riboflavin (diluted in distilled water) in patients with thinner corneas. This causes hydration and swelling of the cornea and protects the corneal endothelium from UV-A radiation, hence allowing the procedure to be carried out safely. Patients with significant apical scarring can also undergo collagen cross linking as it stabilizes the cornea without affecting the scarring thereby preventing or delaying the need for penetrating keratoplasty. I'm more aggressive in my approach and have successfully performed it in patients with advanced keratoconus with steeper and thinner corneas as well, as in any case we are strengthening the cornea and preventing further progression.

- **SPSG:** There are two main exclusion criteria:
 - 1. Extremes of Age(<12 years or >40 years).
 - 2. Corneal thickness less than 350 microns.
- JST: Exclusion Criteira Thinnest Cornea less than 400 microns
 - Diabetics
 - Pregnancy
 - Central Corneal Scarring
 - Active or Healed Viral (Herpetic) Keratitis
 - Maximum corneal curvature should not be more than 60 D.
- **RM:** Patients with a stable power, a pachymetry of less than 375 microns at the thinnest point, a poor endothelial cell count, forme fruste and hydrops (both acute and healed with scarring) are excluded. Pregnancy should be inquired into in women of child-bearing age and lactating mothers should be excluded. Special precautions are recommended for patients with severe vernal catarrh and features of stem cell deficiency in whom there are wound healing concerns and in diabetics.
- AK: Very early Keratoconus Non progressive.
- Very advanced Keratoconus with Corneal Opacity.
- Steepest 'K' more than 65D.
- Minimum corneal thickness less than 360 Microns on Pentacam (From 360 Microns to 400 Microns thickness, Corneal thickness is increased temporarily intra-operative with hydration using hypotonic solution).

SB: What is the cut off age for patients who can undergo the procedure?

- **FH:** There currently is no upper nor lower age limit for us. However, classic keratoconus almost never progresses after the age of 55 and only starts before puberty if excessive eye rubbing is the cause.
- On the other side, we have seen and treated patients as young as 11 years old (prepuberty) that had marked keratoconus due to eye rubbing and patients aged 60 and older with pellucidal marginal corneal degeneration, the latter NOT arresting at a certain age.



Figure 3: Two views of the collagen cross linking machine

- **SB:** 30 years because natural crosslinks in the cornea become strong enough to retard/stop progression of keratoconus after this age.
- **MSS:** Patients with progressive keratoconus can be taken up for collagen cross linking at any age. Younger patients are known to progress more and hence derive greater benefit from collagen cross linking while older patients usually have stable corneas and donot show much progression. However there is no contra-indication and collagen cross linking can be performed at any age.
- **SPSG:** Patients with age less than 12 years or greater than 40 years are not candidates for this procedure.
- **JST:** No cut off age as such but preferably below 50 years.
- **RM:** There is no prescribed cut-off age for this procedure. The youngest I have done is at age 10 years and the oldest is 34 (the other eye has had a PKP). The issue in the older patient is the possible spontaneous cessation of progression as the cornea naturally cross-links as we age.
- AK: There is no cut off age minimum or maximum. It can be done if patient meets criteria & Keratoconus or Keratectasia is progressing.

SB: How effective is the procedure in your settings – in terms of visual regain and time?

GW: Collagen crosslinking might become the standard therapy for progressive keratoconus in the future diminishing

significantly the need for corneal transplantation. Preoperative pachymetry and individual control of the ultraviolet A-irradiance before each treatment are mandatory.

- **FH:** When the inclusion criteria are respected, we can expect a regain of VA to the preoperative values at 10-12 weeks after surgery. Before, haze might lower VA for 1 or 2 lines when compared to the preop state.
- **SB:** I am extremely satisfied with the results in terms of visual recovery and cessation of progression of the disease. Most of my patients regain vision in a week's time. In cases where I have performed PRK with collagen cross linking , the results have been more heartening.
- **MSS:** We have found it extremely effective with improvement of vision by 1 to 3 lines in 30-40% patients and stabilization in the rest. The visual gain has remained stable over a 1 year follow up in all our patients. It also significantly reduces the Surface Asymmetry Index (SAI) and increases the glass/ contact lens tolerance of these patients.
- **SPSG:** Procedure is extremly effective in terms of stabilizing the keratoconus and protecting further deterioration of keratoconus. My clinical experience shows the safety of the procedure. Some patients do gain visual acuity by 1-2 snellen lines post collagen cross linking but improvement in vision is not the prime objective of this procedure. BCVA remained stable at 1 year followup. There were no significant changes observed in mean anterior and posterior corneal curvature (horizontal and vertical) central and apical corneal thickness.
- **JST:** Most of the patients show obligations with regression to tune of 0.50 D to 1.50 D in 2-3 months times.
- **RM:** The procedure is done for providing stability and not for visual gain. This must be emphasized to the patient who may have unrealistic expectations. The vision regains slowly; we refract the patient at around the $10^{th} 14^{th}$ day and provide glasses. A fresh topo-assisted contact lens trial is done at around 4 5 weeks. Some patients complain of a slight haze but the majority recover fully and actually gain in BCVA. At 6 months all patients have achieved or exceeded pre-op BCVA.
- **AK:** After about 3 months of collagen cross linking procedure, there is gain of 1-2 lines in visual acuity in few patients and decrease in steepening of 2-3 dioptres.

SB: How many times it can be repeated and how often?

- **GW:** Where recurrence of keratoconus is present, a second crosslinking procedure might be a choice.
- FH: Up to now, we never had to repeat a treatment. However, we know from earlier studies from literature that the turnover time of the corneal stroma is approx. 10-15 years. So, theoretically, there should be no more cross-linked stroma in a human cornea at 10 years after treatment and maybe, we will have to re-treat these patients after 10 years.

- Nowadays, the longest follow-ups available are 8 years, so we will know more in a few years
- Even if the human cornea will be entirely reorganized after 10 years these patients will also be 10 years older and we know that classical keratoconus progresses more slowly with age
- **SB:** I have not repeated Crosslinking in any eye.
- **MSS:** It can be repeated if required if progression is documented on corneal topography. However, in a 9 year follow up of patients after collagens cross linking, a re-treatment was not required. Theoretically the procedure can be done again without any sight threatening side effects.
- **SPSG:** We have not repeated treatment in any of our cases so far. The first treatment should stabilize the keratoconus for lifetime.
- **JST:** We have not repeated it yet, but can be repeated after 2-3 years if needed as collagen turn over in cornea is estimated to be between 2-3 years but only if disease is progressive.
- RM: The theoretical concern is that the crosslinked collagen may

We cover the eye with ofloxacin ointment and a therapeutic contact lens until the epithelium is healed, followed by fluorometholone eye drops twice daily for several days.

be replaced in time by defective collagen and the cone progression may re-start. Follow-up worldwide is in the range of 7 years and there are no reports of repeat procedures. In our series too there seems to be no cause for us to perform a re-do.

AK: It can be repeated but I have not found the need to repeat till date. After collagen cross linking, there is decrease in corneal thickness of about 20-40 Microns (as measured by Pentacam) in all the cases as the corneal lamellae become more compact after corneal collagen cross linking.

SB: What procedure do you follow for removal of corneal epithelium?

- **GW:** The central 7mm of the corneal epithelium are removed to allow better diffusion of riboflavin into the stroma.
- **FH:** We remove the epithelium completely
 - At the 3rd international congress on corneal cross-linking in Switzerland (organized by our institute, IROC, www.iroc.ch), an italian group (Baiocchi et al.) has presented very interesting data where the concentration of riboflavin in rabbit corneas was measured at 30 minutes after riboflavin application with and without prior abrasio.
 - Their results clearly demonstrated that the group with intact

epithelium had a 10-times lower riboflavin concentration when compared to the abrasio group.

- SB: In cases where I do only collagen cross linking, I remove epithelium in the cross pattern with 5 horizontal and 5 vertical line of 1-2 mm breadth of de-epithelisation. With PRK + collagen cross linking I remove epithelium with a hockey knife in a 9 mm diameter.
- **MSS:** The instrument I use to remove the epithelium is the hockey stick. I have also used alcohol to debride the epithelium in a couple of cases. A brush rotator can also be used for mechanical debridement.
- **SPSG:** We use hockey stick knife, to remove linear streaks of the epithelium with few strokes, leaving the centre epithelium intact.
- **JST:** I do not remove epithelium, but give scratch marks in epithelium in criss-cross fashion.
- **RM:** Various modes of epithelial removal including mechanical debridement are being used. We use a commercially available filter paper swab soaked in 70% isopropyl alcohol, cut into a disc of 8 mm diameter and placed for 15 secs on the center of the cornea. The epithelium just peels off with a moistened sponge and the cornea is irrigated well with BSS before starting the riboflavin instillation.
- **AK:** I usually remove epithelium manually with hockey stick knife, only partial interrupted epithelium removal as it serves the purpose as well as causes early healing of epithelium.
- SB: Your post operative period management pertaining to pain. Any special precautions for herpes?
- **FH:** We cover the eye with ofloxacin ointment and a therapeutic contact lens until the epithelium is healed, followed by fluorometholone eye drops twice daily for several days
- In cases of severe pain we hand out 1:5 diluted oxybuprocaine 0.4% eye drops, but only in cases where we can trust the patient's compliance that he/she will not abuse on these drops (no more than once per hour, danger of prolonged epithelial healing)
- We take no special precautions for herpes
- **SB:** I give Diclofenac 50 mg dispersible tablets on SOS basis. I also give 0.2% Xylocaine in lubricant every 2 hours for 2-3 days. A Vigamox impregnated Bandage CL is put at the end of procedure to be removed when slit lamp examination shows complete epithelization (Mostly 2-3 days).
- **MSS:** We routinely prescribe antibiotic-steroid combination, NSAIDs & lubricating eye drops. A BCL (bandage contact lens) is placed for patient comfort and removed after 3-5 days depending on the healing and response. Patients with HSV are considered high risk for refractive surgery even though cases have been done without recurrence or problems. I have not encountered a case of Herpes with keratoconus so far.

- **SPSG:** Pain is not an issue in the post operative period. I recommend liberal use of lubricating eye drops. Patient is followed up daily till the epithelial defect heals and bandage contact lens in removed.
- **JST:** After the treatment we patch the eye for 24 hours, Orally NSAID (Combiflam) three times in a day for 2 days. Antibiotic eye drops QID (Vigamox) for 7 days, NSAID eye drops TID for one week, and Lubricating eye drops. We are not doing collagen cross linking in cases where we suspect herpes.
- RM: The patient must be adequately counseled. Pain, discomfort, watering and foreign body sensation postoperatively are significant features. We now apply a bandage lens after the procedure is completed. Systemic ibuprofen & paracetamol are prescribed for 4 days. Lubricant drops 6 times daily help decrease the discomfort. We have not encountered any concern regards Herpes in our series.
- **AK:** I place bandage contact lens on cornea and prescribe postoperative Ketarolac Eye drops (4 times a day) with lubricating and antibiotic drops & oral pain killer to be taken, if needed.

We don't need any precautions for herpes in case of normal Keratoconus / Keratectasia patient.

- SB: Your clinical experience with the above procedure. (Number of cases and results)?
- **GW:** Dresden clinical study shows that in all treated 60 eyes the progression of keratoconus was at least stopped ('freezing'). In 31 eyes there also was a slight reversal and flattening of the keratoconus by up to 2.87 diopters. Best corrected visual acuity improved slightly by 1.4 lines. So far, over 150 keratoconus patients have received crosslinking treatment in Dresden.
- **FH:** We have now 5 years of clinical experience at IROC.
- Our preliminary study one-year follow up data confirm earlier results showing a stabilization of the keratoconus in all cases that met the inclusion criteria and slight increase in BSCVA of approximately one line.
- SB: Over a period of 1 year and 2 months, I have done 102 eyes
 97 eyes have achieved either improvement or stability in the disease. 5 eyes have shown progression over 6 months. An improvement is appreciable in a weeks time when astigmatism shows tremendous reduction and keeps on getting better for next 6 12 months.
- **MSS:** We have done 42 eyes and found the procedure to be highly effective. On an average the BCVA improved by 1.4 lines and an average regression of 2.8 D took place in the keratometry. There was stabilization of keratoconus in all patients. There was an overall flattening of the corneal contour with a reduction of Sim K astigmatism between 0.2- 6.9 D with the effects being most apparent in on the anterior surface (ABFS). On an average the astigmatism reduced between 1.0 to 7.0 D.

- **SPSG:** We have done collagen cross linking in about 150 eyes till date and all the eyes are doing well in terms of not only stability but also in maintaining good vision with help of contact lenses or glasses. Some cases have haze lasting for up to three weeks.
- **JST:** We have done over 100 eyes, in almost all cases progression has stopped and some of them improved from 0.50 D to 1.50 D.
- **RM:** My co-workers and I have performed over 140 procedures in the last 15 months. About 35 patients have completed 1 year follow-up. All treated eyes have achieved stabilization of cone progression. The other eye, in those who underwent one eye treatments, has progressed over the same period.

UCVA improved in 80%, BCVA has improved in over 2/3rd and contact lens tolerance in over 75%. Topographic flattening of the cone is observed in the majority with a reduction in corneal irregularities and decrease in corneal astigmatism. Some patients complain of visual haze till 4 months but this improves thereafter. No patient recorded a drop in BCVA.

- **AK:** Till now, I have treated 18 eyes in last 11 months and I am quite satisfied with the procedure and in all the patients, Keratoconus is stabilized but long term follow-up is needed.
- SB: Other indications in which you are doing collagen cross linking as a line of treatment?
- **FH:** We have successfully established and published CXL for three further indications:
- LASIK-induced keratectasia
- PMD (pellucidal marginal degeneration)
- acute corneal melting processes
- I will attach the corresponding papers to the Email.
- SB: As of now I am treating only conus and ectasias.
- **MSS:** I have used collagen cross linking in patients with Pellucid marginal degeneration and Post-LASIK keractesia and found good results. I've also used it in conjunction with Kera Rings for patients with advanced keratoconus and



along with Toric Implantable Contact Lens to correct myopic astigmatism and further improve vision after collagen cross linking.

- **SPSG:** We are doing collagen cross linking in cases having proven Post Lasik Ectasia and also in some patients with progressive pellucid margin degeneration.
- **RM:** Any progressive ectatic corneal condition can in theory be benefited by crosslinking. Though the majority of our cases are of progressive Keratoconus, we have performed the procedure successfully in many cases of post-LASIK keratectasia and some patients with Pellucid marginal degeneration.
- **AK:** Apart from progressive Keratoconus, other indication is progressive Iatrogenic Keratectasia.



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Macular Hole Surgery-Revisited

Atul Kumar MD, FAMS, Raj Vardhan Azad MD, FRCS, Yog Raj Sharma MD, Parijat Chandra MD

Acular holes are characterized by the absence of neurosensory retinal tissue at the fovea. Most are due to aging, trauma and high myopia¹ .Investigations using OCT and ultrasonography suggest that idiopathic macular holes are caused by tractional forces associated with perifoveal vitreous detachment (early PVD). Surgery involves pars plana vitrectomy, removal of the pre-foveal cortical vitreous, fluid-gas exchange, gas injection and postoperatively prone positioning. Recent studies have shown that the success rate is more than 90% in both senile and myopic macular holes, with the new surgical technique involving removal of the internal limiting membrane (Figure 1).²⁻³ For those who are less compliant to positioning, e.g. children, non-willing adults, obese, arthritis patients, besides patients who need to fly early in the post operation period, using silicone oil instead of gas may be desirable.^{4,5}

Macular hole size is a predictor for anatomical closure. Holes >400 μ m in aperture size on an OCT scan are more likely to close only when ILM is peeled.⁶

Classification of Macular Holes

Idiopathic macular holes occur primarily in the sixth through eighth decades of life, affect men more frequently than men, and appear at a younger age in myopic eyes. The observation that an idiopathic macular ole appears to be a complication of the earliest stage of age-related PVD helps explain the age and sex demographics of this condition, which are similar to those of PVD. The foll-owing description of the stages of macular hole formation and what OCT reveals at each stage is useful in interpreting biomicroscopic findings and making management decisions. (Figure 2-5)⁷



Figure1: ILM peel in an eye with idiopathic macular hole

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- A stage 0 or premacular hole state occurs when patients develop a perifoveal vitre-ous detachment, and only subtle changes in macular topography, such as loss of the foveal depression, can be seen. Patients usually have normal visual acuity and most do not develop advanced stages of macular holes.
- Patients with stage 1 macular holes (also known as impending macular holes) have visual symptoms that typically include central vision loss (with visual acuity typically measuring 20/25 to 20/60) and metamorphopsia. On biomicroscopy, there is loss of the foveal depression associated with a small yellow spot (stage IA) or yellow ring (stage IB) in the center of the fovea. OCT examination reveals that a stage IA hole is a foveal 'pseudocyst' or horizonal splitting associated with a vitreous detachment from the perifoveal retina but not from the foveal center. In stage IB holes, there is progression of the pseudocyst posteriorly to include a break in the outer foveal layer, the margins of which constitute the yellow ring seen clinically. As many as 50% of stage 1 holes resolve spontaneously following separation of the vitreofoveal adhesion and spontaneous relief of tractional forces (Figure2).
- A stage 2 macular hole represents the progression of a foveal pseudocyst to a full thickness dehiscence, as a tractional break develops in the "roof" (inner layer) of the pseudocyst. The small opening in the inner layer «400 11m diameter) may be either centrally or eccentrically located. Progression to stage 2 typically occurs over several weeks or months and usually involves a further decline in visual acuity. OCT demonstrates that the posterior hyaloid typically remains attached to the fo-veal center in stage 2 holes (Figure 3).
- A stage 3 macular hole is a fully developed hole (<400 um diameter), typically ac-companied by a rim of thickened and slightly elevated retina. Visual acuity may range from 20/40 to 5/200, but it is generally around 20/200. The posterior hyaloid remains attached to the optic disc, but it is detached from the



Figure 2: Line scan OCT showing stage I A hole



Figure 3: OCT of stage 2 hole showing an eccentric flap in the inner wall of the pseudocyst



Figure 4: Stage 3 full-thickness hole with elevation of adjacent retinal edges with detachment of the post hyaloid over the macula

macular region. An operculum may or may not be present, suspended on the posterior hyaloid overly-ing the hole (Figure 4).

• A stage 4 macular hole is a fully developed hole with a complete posterior vitreous detachment signified by a Weiss ring (Figure 5).

Fluorescein angiography in eyes with stage 2, 3, and 4 holes demonstrates a circular trans-mission defect as a result of the loss of xanthophyll at the site of the hole and because of RPE depigmentation and atrophy in the base of the hole. *However, the gold standard in the diagnosis of the various stages of macular holes is OCT examination.*^{7b}

Retrospective studies estimate the incidence of bilaterality in macular holes to be 1 %-25%, but this figure is difficult to establish from the current literature because of loss to follow-up or short follow-up. *Patients who present with a full-thickness macular hole in one eye and are symptomatic, with loss of foveal depression or stage lA abnormalities in the second eye, have a substantial risk of progressing to a stage 2 hole in the second eye.* Patients with a full-thickness macular hole in 1 eye and a normal retina with a vit-reomacular separation in the other eye are at minimal if any risk of developing a macular hole in the second eye. Finally, patients presenting with a full-thickness macular hole in 1 eye and a normal fellow eye with an attached posterior vitreous probably have an intermedicate risk of developing vitreomacular interface abnormalities in the second eye over their lifetimes.

Vital stains in Macular Hole Surgery

Vital stains have a promising role in vitreoretinal surgery as they enhance and display in relief, tissues such as ILM, posterior hyaloid, Vitreoschisis and fine ERM. ICG, a fluorescent dye used in choroidal angiography, was recognised for its potential in the operating room by cataract surgeons, who first used it to stain the anterior capsule to facilitate capsulorrhexis in difficult cases. Vitreoretinal surgeons, taking notice of their anterior segment colleagues' success, soon found that ICG similarly stained the internal limiting membrane (ILM) of the retina. This was important because peeling of the nearly invisible ILM from the retinal surface had recently been advocated in macular hole surgery, ⁸⁻¹¹ and the arrival of ICG made ILM peeling easier, faster, and less traumatic.



Figure 5: Stage 4 full-thickness hole with operculum and complete PVD

Indocyanine green is prepared for surgical use by reconstituting it with pure water as a solvent, before bringing it to its final concentration in balanced saline solution; it is the hypo-osmolarity of the solvent and the final solution is suspected of disrupting cellular elements of the neural retina. Reports are there of using 0.5 ml of 5 mg/ml ICG solution (approx 0.4mg/ml) which is relatively nontoxic. In contrast, infracyanine green, which uses 5% glucose solution as its solvent for an iso-osmotic final solution, offers comparable staining of the ILM while reducing the untoward osmotic effects. In previous in vitro studies by this group, infracyanine green in 5% glucose did not demonstrate cytotoxicity to cultured RPE cells, while ICG exposure led to significantly increased cell death.¹²⁻¹³ Similarly, in the same study ILM specimens excised with infracyanine green were not noted to include Muller cell footplates or other evidence of neural retinal disruption when examined by histopathology and electron microscopy.

Unlike the green dyes which selectively stain the acellular ILM but not overlying membranes and vitreous, trypan blue directly stains epiretinal membranes (ERMs), making it valuable in cases such as macular pucker removal. Whereas ICG can only indicate the presence of the ERM by its lack of green staining within an area of stained ILM (referred to as "negative staining"), trypan blue has an affinity for the cellular material composing epiretinal membranes (ERMs), providing visualisation and localization. Brilliant Blue dye has recently become available (Geuder) which also is effective for ILM staining

Some surgeries exploit the "complementarity" of these dyes in a double staining technique for macular pucker removal, first with trypan blue to peel the ERM, then with infracyanine green to peel the underlying ILM.

Triamcinolone Acetonide crystals with solvent removed, provide excellent identification and help in peeling ERM / ILM in macular hole surgery. They have the added advantage of not possessing the toxic properties of vital dyes.

One can envision in the future an array of vital stains e.g., a sort of surgical palette, with different intraoperative dyes or even noninvasive dyes having distinct affinities for specific membranes or cell types. Diabetic fibrovascular membranes, vitreous cortical hyaloid, proliferative vitreoretinopathy membranes, neurosensory retina, choroidal neovascular membranes, active tumor cells—all promising targets for vital stains.

Surgery for Macular Holes

Careful patient selection is critical to a successful outcome. The ideal candidate would be a patient with bilateral holes of relatively recent onset, with vision in his better eye less than or equal to 6/36. Patients with unilateral symptomatic holes with recently reduced vision to 6/24 or worse are also good candidates. Both the laser interferometer and the potential acuity meter have been found to be modestly accurate in predicting postoperative visual acuity.

Prospective randomized clinical trials have shown that surgical intervention in stage 2, 3 or 4 macular holes results in some visual benefit.¹⁴

The objectives for surgical repair of macular holes include relief of all tangential traction and retinal tamponade. Tangential traction is relieved by identification and removal of the cortical vitreous or posterior hyaloid and removal of fine epiretinal membranes around the hole. *Tamponade is provided by total gas-fluid exchange with air/* 20-25% SF6/12-14%C3F8 gas mixture and strict face-down positioning, often now for less than one week duration only...

Using active aspiration (150-250 mmHg), a silicone-tipped suction cannula/vitreous cutter, using suction only is used very effectively for PVD induction, added use of the hyaloid lifter helps in extending the PVD peripherally. The area immediately around the hole is avoided. In case the silicone tip is used, it is noted to flex once the cortical vitreous is engaged. This has been termed as "fishstrike sign" or "diving rod sign". Once engaged, a PVD can be created with continuous suction with anterior-posterior-tangential traction while moving the tip over the retinal surface. The dissection is carried from the area of initial detachment to adjacent attached areas in an attempt to complete the detachment from the posterior retina to the equatorial zone. The vitreous cortex or posterior hyaloid becomes visible as a translucent sheet, especially with oblique illumination. Occasionally, the disc attachments are so firm that the vitreous cutter (on suction only), tissue forceps or pick manipulation is required to complete the PVD in these areas. A 36-gauge subretinal pick can be useful in engaging the posterior hyaloid near the optic nerve and then pulling off the Weiss ring. Frequently, an operculum is detected as a glial fragment attached to the vitreous cortex. Once the vitreous is completely detached, vitrectomy is completed. If residual vitreous cortex is present, it appears as a gelatinous substance on the surface of the retina during completion of the fluid-air exchange. (which can be removed by performing airinfused vitrectomy).

Fifty percent of operated eyes have some degree of epiretinal membrane (ERM) proliferation. These ERMs, unlike typical ERMs, tend to be finer and more friable and at times are densely adherent to the retina. The ERMs may be present surrounding the hole or can involve only a few clock hours. A bent MVR blade is used to



Figure 6a: 23G vitrectomy showing insertion of trocar-cannula





Figure 6b,c: TA enhanced ILM peel in progress, in an eye with macular hole



hole on OCT

create an edge in the ERM, which is then grasped with end-opening tissue forceps and peeled.

ILM peeling is then performed using 0.15% trypan blue or triamcinolone acetonide crystals injected over the macula. Peeling as shown in the Figure 7b, c is carried out using the ILM forceps. It is preferable to peel the ILM across the hole to relieve all traction at the *edge of the hole.* (Figure 6a,b &c)

During this manoeuvre, it is common to create small hemorrhages around the hole. Damage to inner retina should be avoided, an early sign of which may be the development of fluffy whitish areas.

Prolonged intense illumination from the light pipe near the macula should also be avoided to prevent photo toxicity. A total fluid-air exchange is performed and effort is made to dehydrate the vitreous cavity. The shallow fluid in the base of the optic disc cup is aspirated repeatedly, with a soft-tipped cannula, until fluid no longer collects. Frequently, the edges appear to slide closer together, which is considered a good prognostic sign. A nonexpansile concentration of long-acting gas is exchanged for air. A shorter-duration gas 25% SF6 is often being preferred. Postoperatively, strict prone positioning is prescribed for a very short duration of 3-5 days only.⁵ (Figure 7a & 7b).

Intravitreal air as tamponade versus longer-acting gases

Advantages of air include less demanding positioning, patient willingness for surgical procedure, potentially reduced risk of "ulnar neuropathy, less risk of cataract formation and less risk of postoperative IOP rise. (Figure 8)

At the 1-week visit if the edges of the macular hole are flattened and imperceptible with flattening of the cuff of retinal detachement, anatomic success is assured. However, if the edges are still visible and the cuff elevated, anatomic failure is probable. Macular hole surgery is able to close full thickness macular holes in approximately 90% of cases.

The etiology of anatomic failure is uncertain. Possible causes are: Poor case selection (extremely large holes often with ragged edges as in traumatic holes are unlikely to close), Patient noncompliance in postoperative prone positioning or even *instillation of topical eye* drops frequently interferes with face-down positioning and may affect final hole closure, and recommendation includes using an antibiotic ointment only in the post-operative period.¹⁵ residual ERMs



Figure 7b: post ILM peel closed hole, at 1 month post operatively



Figure 8: Post operative closed macular *hole with residual gas bubble*

producing traction, and intrinsic retinal changes causing stiffness and preventing retinal reattachment. If failure is believed to be secondary to residual ERMs, reoperation has been successful. If noncompliance with postoperative prone positioning is thought to be the cause of failure, newly motivated patients can be given a second chance. Macular holes can reopen after initial surgical repair.¹⁶⁻¹⁷ The cells that may lead to the closure of an idiopathic macular hole may also contribute to its recurrence if the reparative process goes away. Anatomically unsuccessful closure of the hole correlates with small enlargements in the diameter of the macular hole and its surrounding subretinal cuff and with a slight decrease in visual acuity. However, visual acuity may improve substantially with reoperation after previously failed surgery.

Bovine and recombinant transforming growth factor (TGF-beta), autologous serum, autologous plasma, thrombin and fibrin, autologous platelet concentrate, have been used as adjunctive substances in macular hole surgery.¹⁸ It appears that closure of a full-thickness macular hole is associated with a limited healing response which may be encouraged by the use of adjunctive substances. [Glaser et al proposed using TGF-beta 2 as a pharmacologic adjuvant in surgery on macular holes to increase the anatomic success rate. Single application of TGF-beta 2 had a statistically significant beneficial effect. *Despite the initial promising* *results, a large prospective randomized study did not indicate any additional benefit of TGF-beta 2.*

Ligget et al proposed the use of human autologous serum. In a small pilot study, resolution of the surrounding fluid and flattening of macular hole occurred in all eyes, but a large randomized controlled study is yet to be reported. In another study, where macular hole surgery was done with autologous serum in one group and without autologous serum in the other group, the macular hole closure rates were identical.

Lamellar Macular Holes: Surgical treatment including vitrectomy, ERM-ILM-peeling and endotamponade appears to benefit patients with lamellar macular hole. Surgical treatment can close the lamellar macular hole and restore foveal architecture and improve functionally.¹⁹

Complications

The most common complication of a vitrectomy for macular hole is the occurrence or progression of nuclear sclerotic cataract. Nuclear sclerotic cataracts progress substantially after macular hole surgery with long-acting intraocular gas tamponade. Visual acuity often decreases 12 or more months after vitrectomy because of cataract progression Posterior segment complications have been noted in 23% of surgical cases. These include peripheral retinal breaks, rhegmatogenous retinal detachment from a peripheral retinal break, enlargement of the hole, late reopening of the hole, retinal pigment epithelium loss under the hole, phototoxicity and endophthalmitis. Iatrogenic retinal breaks tend to be in the inferior and temporal retina which establishes the need for greater intraoperative surveillance in these areas. Peripheral retinal tears may develop during stripping of cortical vitreous. Retinal pigment epitheliopathy after macular hole surgery may portend a guarded visual prognosis. This may be the result of individual patient sensitivity to manipulation, direct trauma or a prolonged exposure to the endoilluminator. Late reopening can complicate initially successful macular hole surgery. Reopening has been documented to occur between 2 and 22 months and it has been hypothesized that the growth of an ERM plays a part in at least some of the eyes. Repeat vitrectomy with gas injection may result in reclosure of the hole and improvement in vision. Some eyes develop increased intraocular pressure (IOP) after macular hole surgery. The increase occurs most frequently between 2 days and 2 weeks postoperatively.

Dense wedge-shaped temporal and/or inferior visual field deficits have been noted upon gas resorption. Proposed origins of this visual field defect include ischaemia of or direct trauma to the optic nerve head, posterior segment ischaemia and transient intraoperative/postoperative raised IOP.²⁰

Future horizons

The benefits of macular hole surgery are reasonably well established. The more exciting "instruments" are the pharmacologic agents. Chondroitinase-ABC, an agent that disinserts vitreous and/ or preretinal membranes from the neurosensory retina, has been developed. This holds tremendous promise and Phase I FDA-approved clinical trials are underway.

Shorter duration prone position or no positioning may gain importance as there is a greater patient compliance.

Use of newer non-toxic but effective staining agents e.g. Brilliant Blue may help in the overall success

Last but not the least, better surgical techniques, use of minimal gauge vitrectomy and improved instrumentation will help.

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at Hotel Ashok, Chanakya Puri, New Delhi

Optical Coherence Tomography Findings in Bietti's Crystalline Dystrophy

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Bietti's crystalline dystrophy (BCD) was first described by Bietti in 1937.^{1, 2} This rare retinal dystrophy with an autosomal recessive inheritance pattern is characteristically associated with crystalline deposits in the cornea, crystalline deposits in the retina, and progressive atrophy of the retina, retinal pigment epithelium, choriocapillaris, and choroid. Variants without the presence of corneal deposits have been described in literature from Asian countries.³ The CYP4V2 gene has been described to be the causative gene for BCD.^{4, 5} This gene has been thought to play a role in fatty acid and corticosteroid metabolism.⁴ BCD has been reported to be associated with abnormal lipid metabolism because of deficient lipid binding, elongation, or desaturation.⁶ Complex lipid inclusions in the conjunctiva, fibroblasts, and circulating lymphocytes have also been reported, supporting the evidence that BCD may result from a systemic abnormality of lipid metabolism.⁷

Before the introduction of optical coherence tomography (OCT), histopathology was the only means to identify the pathological changes in the retina in patients with BCD. The present case series attempts to correlate the reported clinico-histopathological findings in BCD and the in-vivo OCT observations made on the sample patients.

Methods

This study adheres to the tenets of the declaration of Helsinki. Five patients with BCD from retina clinic of our institution were included in this study. In this cross sectional observational case series, the patients underwent a complete eye examination including bestcorrected Snellen visual acuity, slit-lamp biomicroscopy, applanation tonometry, Goldmann perimetry, colour vision (Ishihara plate), electroretinogram, electrooculogram, fundus photography, fluorescein angiography, and optical coherence tomography (Stratus OCT, Carl Zeiss Meditec, Dublin, California, USA). The clinical fundus findings were staged into following four stages as described by Mataftsi A et al.⁸ In the early stage, crystals are seen in the posterior pole and midperiphery with patchy RPE changes. The progression of RPE atrophy and gradual disappearance of crystals from the posterior pole heralds the onset of intermediate stage. In the advanced stage, marked chorio-retinal atrophy involving the posterior pole and retroequatorial retina is seen. The end stage is characterized by complete chorioretinal atrophy and vascular attenuation of the chorio-capillaris. Crystals are extremely rare in the end stages. OCT was performed in all cases using line scan and fast macular scan protocol. OCT analysis of the macula was done to assess the distribution and density of the crystal and associated changes in various layers of the retina and retinal pigment epithelium in different stages of BCD.

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Results

The study population consisted of three females and two males. The age of the patients ranged from 28 to 50 years. All except one (case 2) patient were born of non-consanguineous marriages. The patients presented with nyctalopia (case 1-3, 5) and progressive diminution of vision (case 1-5). Medical and drug history were normal. Siblings of three patients (case 2, 3, and 4) were available for examination but none were affected by this disease. Clinical characteristics of these patients are summarized in table 1.

Optical Coherence Tomography findings

Optical Coherence Tomography parameters of these patients are summarized in table 2. Overall four stages of evolution were observed on OCT in our series of patients (ten eyes) with BCD.

Stage 1: (case 4: right eye) (Figure 1A-C)

In this stage multiple focal hyper-reflective dots are seen distributed uniformly from centre to periphery through out the retinal layers corresponding to fine, crystalline deposits at the macular area. However, the crystals are more abundant in the inner retinal layers. On OCT retina does not have the layers we ordinarily expect to differentiate. The IS/OS boundary is not seen and the outer nuclear layer is poorly discernable. Retinal pigment epithelial (RPE) layer looks more hyper-reflective than normal. Reflectivity of the crystals and RPE are very close. Retinal thickness as well as average retinal nerve fiber layer thickness remains within normal reference range. Normal foveal contour is preserved in this stage.

Stage 2: (case 1: left eye; case 4: left eye; case 5: right eye and left eye) (Figure 2A-C)

Focal hyper-reflective dots gradually become less in deeper retinal layers but are still present in the inner retinal layers. Hyperreflective dots are also seen less frequently or absent gradually towards the fovea. Like stage, the IS/OS boundary is not seen and the outer and inner nuclear layers are poorly discernable. The outer retina shows hyporeflective region subfoveally suggestive of photoreceptor layer attenuation. This attenuation seems to begin in the centre and evolves centrifugally. Retinal pigment epithelial layer persists as more hyper-reflective than usual as seen in stage 1. Retinal thickness and retinal nerve fiber layer thickness are still normal. Foveal depression becomes accentuated in this stage.

Stage 3: (case 1: right eye, case 2: right eye) (Figure 3A-C)

Focal hyper-reflective dots in the inner retinal layers disappear from the centre but can be elicited in the line scan through extra foveal region (Figure 4A-C). The retinal layers in this stage in the line scan are poorly discernable, and are not discrete. Retinal thinning takes place with accentuated foveal depression. Average retinal nerve fiber layer thickness also shows subnormal value.

Case no	Age /Sex	Eye	Snellen Visual acuity	Corneal crystals	Fundus	Visual fields	Colour vision	EOG/ERG ⁺	FFA [*] findings	Clinical Stage
1	28/ M	R/E*	20/120	No	Retinal crystals mostly outside the macula, RPE" degeneration and focal RPE hyperplasia, choroidal atrophy but normal disc and vessels.	Not done	Red-green deficiency	Arden's ration 1.15/ ERG subnormal	Areas of hypofluorescence in early phase with RPE and choriocapillaries atrophy	Advanced Stage
		L/E*	20/120	No	-do- ⁺⁺	Not done	Red-green deficiency	Arden's ration 1.10/ ERG subnormal	-op-	Advanced Stage
7	36/ M	R/E	20/80	No	Abundant crystals in the posterior pole and midperiphery, diffuse RPE degeneration, choroidal atrophy, normal disc and vessels.	-op-	Red-green deficiency	Arden's ratio 1.35/ ERG subnormal	-op-	Intermedi ate Stage
		L/E	20/200	No	-op-	Mild constriction, central scotoma	Red-green deficiency	Arden's ration 1.24/ ERG subnormal	-op-	Intermedi ate Stage
<i>w</i>	37/F	R/E	20/300	No	Few retinal crystals mostly outside the macula, patchy RPE hyperplasia, diffuse RPE and choroidal atrophy, mild disc pallor and arterial attenuation.	Moderate constriction, central scotoma.	Red-green deficiency	EOG not done/ ERG extinguished	Not done	End Stage
		L/E	20/300	No	-op-	Moderate constriction, central scotoma.	Red-green deficiency	EOG not done/ ERG extinguished	Not done	End Stage
4	36/F	R/E	20/30	No	Absent foveal reflex, abundant crystals in the posterior pole and midperiphery, minimal RPE changes, normal disc and vessels.	Mild constriction, paracentral scotoma.	Normal	Not done	Not done	Early Stage
		L/Ë	20/40	No	-do-	-op-	ked-green deficiency	Not done	Not done	Early Stage
Ŋ	50/F	R/E	20/40	No	-op-	Not done	Red-green deficiency	Not done	Areas of hypofluorescence in early phase with RPE and choriocapillaries atrophy	Early Stage
		L/E	20/40	No	-op-	Not done	Red-green deficiency	Not done	-op-	Early Stage
		· /E. 1.6					- 4			, ,

* R/E: right eye, L/E: left eye † EOG/ERG: electrooculogram/electroretinogram ‡ FFA: fundus fluorescein angiography ** RPE: retinal pigment epithelium †† -do-: same as above

Table 1: Clinical characteristics of the patients in present series.



crystals in the posterior pole with patchy retinal pigment epithelial (RPE) changes suggestive clinically of early stage of Bietti's crystalline dystrophy (BCD). **1B, C:** Horizontal and vertical 6 mm OCT line scans of same eye show multiple focal hyper-reflective dots distributed from centre to periphery through out the retinal layers but more abundant in the inner retinal layers. Retinal pigment epithelial (RPE) layer looks more hyper-reflective than usual. Foveal contour is well preserved.

Hyper-reflectivity of RPE-Bruch's membrane complex extends towards the underlying choroid.

Stage 4: (case 2: left eye; case 3: right eye and left eye)(Figure 5A-C)

Global retinal thinning and foveal atrophy is seen in this stage. Average retinal nerve fiber layer thickness also decreases markedly. Along with the absence of the photoreceptor and retinal pigment epithelial layers, thick hyper-reflective layer of choroidal vasculature, which seems to spread far behind it, is clearly evident on the serial OCT scans. This extended hyper-reflectivity is suggestive of sclerosis of the choroidal layer.

Symmetry between eyes

Although clinically all of our patients have symmetrical involvement in both eyes, OCT demonstrates asymmetrical involvement in two of them (case 2 & 4). In case 2, clinically an intermediate stage, right eye shows stage 2 OCT findings whereas marked retinal thinning and abrupt foveal atrophy without any progressive curve from periphery to centre suggestive of stage 4 is seen in the fellow left eye. The OCT of case 4, clinically staged as early type, shows only hyper-reflective dots in the retina without photoreceptor layer attenuation in the right eye suggestive of stage 1 whereas left eye shows decreased hyper-reflective dots in the deeper and



Figure 2: Fundus photographs and OCT images. **2A:** Fundus photograph of right eye of case 5 suggestive clinically of early stage of BCD. **2B, C:** Horizontal and vertical 6 mm OCT line scans of same eye show less hyper-reflective dots in deeper retinal layers. The outer retina shows poorly differentiated outer retinal layers and hyporeflective region suggestive of photoreceptor layer attenuation. Foveal depression is accentuated.



Figure 3: Fundus photographs and OCT images. 3A: Fundus photograph of right eye of case 2 shows refractile crystals in the posterior pole with diffuse RPE atrophy suggestive of intermediate stage of Bietti's crystalline dystrophy (BCD). 3B,C: Horizontal and vertical 6 mm OCT line scans of same eye show disappearance of hyper-reflective dots due to crystals in the retinal layers and photoreceptor layer attenuation.

central retina with photoreceptor layer attenuation suggesting stage 2 OCT findings.

Discussion

In this era, noninvasive OCT has evolved as the primary investigative modality for retinal disorders (particularly macular), as it gives vivid cross-sectional description of the retinal layers. Though OCT findings in crystalline retinopathies other than BCD have been described,⁹ to the best of our knowledge only single case reports describing OCT finding in BCD have been cited in literature till date.¹⁰ As far as we are aware; OCT staging of BCD has not been reported earlier.

Bietti's crystalline dystrophy is an uncommon cause of crystalline retinopathy that usually presents in the second or third decade of life and is characterized by a triad consisting of yellow glistening intra-retinal crystals in the posterior pole, tapeto-retinal degeneration associated with choroidal sclerosis and crystalline dystrophy of the cornea. However, no crystals were found in cornea in any of our patients. In this study by Wada et al, all subjects with Bietti's crystalline dystrophy had mutations in the CYP4V2 gene yet 3 of 6 subjects did not have corneal deposits.⁵ Other studies also support that corneal crystal is not a consistent feature of BCD particularly in Asian patients.^{3,11}

Age of the patients does not seem to correlate with the onset, progression and severity of the disease. In our series the youngest patient, a 28-year-old male (case 1) had an advanced stage while the 50 year-old female (Case 5) had an earlier stage of the disease.

On the basis of clinical fundus and angiographic findings, BCD can be seen in four different stages.8 In our study we also found nearly similar spectrum of findings on OCT but OCT staging does not always corroborate with clinical staging. The left eye of case 2 shows stage 4 OCT findings though clinically seem to be an intermediate stage (Figure 6). Similarly right eye of case 5 shows stage 2 OCT findings while clinically looks like early disease (Figure 2A-C).

Table 2: Optical coherence tomography (OCT) parameters of the eyes in present series.

Case no	Eye	CMT (μm)*	MV (μl)†	RNFLT (µm) [‡]	OCT Staging
1	right	219	6.13	89.65	Stage 2
	left	202	6.00	92.50	Stage 2
2	right	165	5.09	30.00	Stage 3
	left	153	4.01	26.00	Stage 4
3	right	92	4.02	21.02	Stage 4
	left	136	3.91	24.52	Stage 4
4	right	216	6.12	98.12	Stage 1
	left	197	6.00	95.36	Stage 2
5	right	213	6.57	90.70	Stage 2
	left	208	6.40	90.84	Stage 2
* CMT	': Central 1 Magularya	mm subfie	ld macula	r thickness	an protocol)

VIV: Macular volume (from 6 mm fast macular scan protocol) *[‡] RNFLT: Retinal nerve fiber layer thickness*

In BCD, crystals are reported to be seen at all levels of the retina on histopathology.¹² However in our patients, we found most of the hyperreflective crystals in the inner retinal layers. No shadowing from the crystals was evident indicating that the crystals may not prevent passage of light. Meyer et al¹⁰ attributed this fact to lower light absorption leading to more back scattering as well as scattering of light by crystals to this highly reflective retinal layers. Outer retinal hypo-reflectivity seen on OCT scan in stage 2 onwards is indicative of photoreceptors loss and correlates well with histopathological reports in the literature.¹³ The atrophy of the retinal layers, RPE and sclerosis of choriocapillaris results in increased reflectivity from the choroid.

Due to retinal atrophy and less contrast subtle macular changes are poorly discernable on clinical examination in patients with



Figure 4: Fundus photographs and OCT images. 4A: Fundus photograph of right eye of case 1 shows fewer crystals with marked chorioretinal atrophy in the posterior pole suggesting advanced Bietti's crystalline dystrophy. 4B: Infrared OCT fundus image of same eye showing scan location.
4C: OCT line scans from centre to periphery show absence of hyper-reflective dots in the centre but are present in abundant in the periphery (right side of the scan).



Figure 5: Fundus photographs and OCT images. **5A:** Fundus photograph of right eye of case 3 shows absence of crystals with complete chorioretinal atrophy in the posterior pole suggesting clinically of end stage BCD. **5B, C:** Horizontal and vertical 6 mm OCT line scans of same eye show retinal thinning and foveal atrophy. This extended hyper-reflectivity from RPE-Bruch's membrane complex is suggestive of sclerosis of the choriocapillaris. 2H: Macular thickness map shows global retinal thinning.



Figure 6: Fundus photographs and OCT images.
6A: Fundus photograph of left eye of case 2 shows intermediate stage Bietti's crystalline dystrophy. Macular hole is not clinically discernable. Black arrow represents the OCT scan location and direction.
6B: Horizontal OCT line scan of the same eye reveals retinal thinning with an atrophic macular hole.

BCD. OCT may be more useful in detecting such subtle pathological changes. For examples, in case 2, OCT revealed an atrophic macular hole in the left eye (Figure 6) and in case 5, mild epiretinal membrane formation was seen in left eye when both were not clearly evident on clinical examination. Macular hole formation in Bietti's dystrophy has been reported previously clinically and on histopathology.^{13, 14}

Mataftsi A et al⁸ reported sparing of macular island until later in the disease even when atrophy of the RPE and choriocapillaris complex involves the whole posterior pole. However, in our case series OCT demonstrate subfoveal photoreceptor attenuation in all eyes except one (case 4, right eye) which was an early stage disease with stage 1 OCT findings.

Though clinically BCD seems to be symmetric in all of our cases, the variation between the two eyes on OCT scans in some of our patients clearly indicates asymmetry in terms of onset and progression of the disease.

The present case series has the limitation of being a cross sectional one with fewer number of patients. We were also unable to document the changes on OCT at the retroequatorial or equatorial retina.

In conclusion, OCT features in difference stages of BCD show significant correlation with features reported on clinicohistopathological studies. It is also useful in detecting other changes such as retinal thinning and RNFL thinning, sub clinical macular hole or epiretinal membrane formation. The identification of these changes may help explain the cause for decreased visual acuity in patients with BCD. Prospective studies involving larger patients are required to evaluate the natural history of this disease.

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Keratoconus - Therapeutic Options

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K eratoconus, a non-inflammatory corneal ectasia, is characterized by progressive corneal thinning and apical protrusion¹. Typically, the patients present in early adulthood and visual symptoms result from irregular astigmatism and increasing myopia^{1, 2}. It is reported to have bilateral involvement in over 90% of patients, with asymmetric presentation. *Most keratoconus patients can be adequately corrected with spectacles or contact lenses*. However, in recent years there has been a rapid advancement in the therapeutic options for keratoconus management.

Spectacle Correction

In the early stages of keratoconus, the patient's refractive error can often be successfully managed with spectacle lenses. It is important to communicate to the patient that there is no evidence to support the theory that early contact lens intervention is of therapeutic benefit in preventing or lessening the progression of the disease. However, wearing contact lenses typically provides the patient with better visual acuity than can be obtained with glasses by neutralizing the regular and irregular refractive errors induced by the condition.

Contact lenses

As the condition progresses, spectacles may fail to provide the patient with a satisfactory degree of visual acuity, and most clinical practitioners will move to managing the condition with contact lenses. Contact lenses improve vision by means of tear fluid filling the gap between the irregular corneal surface and the smooth regular inner surface of the lens, thereby creating the effect of a smoother cornea. In the early months of 1888, a French ophthalmologist, *Eugene Kalt*, began work on a crude glass shell

designed to "compress the steep conical apex thereby correcting the condition." This was the first known application of a contact lens for the correction of keratoconus.

Traditionally, lenses for keratoconus have been the "hard" or rigid gas-permeable contact lens variety. For rigid gas permeable (RGP) lens fitting, most contact lens fitting methods use keratometry values in combination with the fluorescein pattern for selection of the back optic zone radius. For most patients with keratoconus, a *three point touch* contact lenses design is ideal, and is preferred over apical clearance and apical touch designs. The base curve should be steep enough to provide a slight central touch, shown by thinning of fluorescein, at the corneal apex and slight touch mid-peripherally at 3 and 9 o'clock along the horizontal meridian. This creates three points of lens touch along the horizontal meridian (Figure 2).

In mild to moderate keratoconus, the lens diameter selected is usually 7.5-8.5mm. A small size facilitataes tear exchange and allows a steeper fit to accommodate the cone. Central nipple cones do best with small diameter lenses. When the cone is displaced peripherally, as with oval and globus cones, one usually ends up fitting a larger, flatter lens.

Several specially designed contact lenses have been devloped to facilitate fitting in advanced, difficult to fit keratoconus cases. *Soper lenses* are one of the best known lenses. This is a bicurve design with a steep central curve to accommodate the cone and peripheral curve to align with the peripheral cornea. They are fitted by varying the sagittal depth which in tun is done by varying the diameter of the lenses. *Mcguire lenses* are modified soper lenses. They have central vaulting to minimise central bearing and peripheral cornea



Figure1: Eugene Kalt, and his glass shell contact



Figure 2: Fluorescein staining lens (from google.com) showing a three point touch pattern

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Figure 3: The SoftPerm lens design for keratoconus



Figure 4: Traditional and custom piggyback lens designs

bears the major pressure. *Nicone* and *Rose K* designs have also been developed. The Rose K lens design is a fully flexible lens that works well on early to advanced keratoconus patients. Complex lens geometry, combined with the enhanced material benefits of Boston ES^{m} , makes the Rose K lens the good fit enhancing patient comfort and visual acuity. Multiple parameters make fitting the Rose K lens possible for most keratoconic eyes.

Hybrid lenses have been developed which are hard in the centre and encompassed by a soft skirt (Figure 3). Soft or hybrid lenses



do not however prove effective for every patient. Some patients also find good vision correction and comfort with a *"piggyback" lens* combination, in which gas permeable rigid lenses are worn over soft lenses, both providing a degree of vision correction (Figure4).

Wave Custom Designed Contact Lens is a topography based designed contact lens. The corneal map is loaded into a lens designing software. This software is then used to design a lens specifically for that cornea.

Boston Scleral lenses Prosthetic device (BSLPD)

It is a fluid-ventilated gas-permeable contact lens that rests entirely on the sclera creating a fluid-filled space over the diseased cornea. They are sometimes prescribed for cases of advanced or very irregular keratoconus; these lenses cover a greater proportion of the surface of the eye and hence can offer improved stability and comfort.BSLPD has been worn with all day wearing comfort in many RGP lens intolerant patients.³

The larger size of the lenses may make them unappealing or uncomfortable to some, however their easier handling can find favour with patients with reduced dexterity, such as the elderly.High cost prohibits widespread usage.The Boston MiniScleral lens device (rests on peripheral cornea) has been recently developed for keratoconus patients.

Refractive surgery

Laser in situ keratomileusis (LASIK) or photorefractive keratectomy (PRK) is contraindicated in these patients because of a greater risk for scarring and excessive thinning leading to possible post-LASIK ectasia. Thorough topographic evaluation should be done to rule out keratoconus fruste or suspect before considering these procedures.

For similar reasons, radial keratotomy has also generally not been used for keratoconic patients. An Italian clinic has however reported some success with a modified asymmetric radial keratotomy procedure and mini-A.R.K, in which the incisions are confined to one sector of the eye⁴. The corneal thickness is first measured using a pachymeter, then the surgeon makes cuts to a depth of 70-80% of the measured thickness. The patient may initially experience photophobia and fluctuation of vision after radial keratotomy, just as with other forms of refractive surgery.



Unlike refractive procedures, phototherapeutic keratectomy (PTK) has been helpful for some selected keratoconus patients to reduce steepness of the cone and for nodular subepithelial scars in patients who have become contact lens intolerant. The resultant flattening of the cone makes contact lens fitting easier. The key to the safety of the procedure is that the very shallow ablation is not intended to have a refractive effect.⁵

Intrastromal corneal ring segments

A recent surgical alternative to corneal transplant is the insertion of intrastromal corneal ring segments. These inserts are designed to be placed at a depth of approximately two-thirds the corneal thickness and are surgically inserted through a small radial incision into a track created within the corneal stroma. The use of a femtosecond laser for Intacs channel creation seems as effective as mechanical dissection⁶. They are oriented horizontally in the cornea at 12 and 6 o'clock (Figure 6). They shorten the corneal arc length and have a net effect of flattening the central cornea (Figure5). The amount of flattening is determined by the insert's thickness. Rings are available in thicknesses of 0.250, 0.275, 0.300, 0.325 and 0.350 mm (recently 0.400 and 0.450 are also available). Intacs are indicated for contact lens intolerant patients with early keratoconus who have minimal central stromal scarring.

The two principal types of intrastromal rings available are known by the trade names of Intacs and Ferrara rings. Intacs are flatter and less centrally placed than the Ferrara rings. Intacs were first approved by the Food and Drug Administration for the treatment of keratoconus in July 2004. Ferrara rings await FDA approval for keratoconus.

Intacs implantation is being increasingly considered and shown effective in early keratoconus cases⁷.

Clinical studies on the effectiveness of intrastromal rings on keratoconus are in their early stages, and results have so far been generally encouraging, ^{8,9} though they have yet to enter into wide acceptance with all refractive surgeons. Potential complications of intrastromal rings include accidental penetration through to the anterior chamber when forming the channel, post-operative



infection of the cornea, and migration or extrusion of the segments. The rings offer a good chance of vision improvement even in otherwise hard to manage eyes and can always be a good option before taking up the patient for surgery.¹⁰

A development on the concept involves the injection of a transparent synthetic gel into a channel bored through the stroma. As the gel polymerises, it stiffens and takes on similar properties to the preformed rings.¹¹ The procedure has however not been put to much clinical use.

Contact lenses may be needed for keratoconus patients who have INTACS inserts and have a role in augmenting their vision. Contact lens tolerance was restored in over 80% of cases in a study.¹² Rigid gas-permeable or toric soft lenses can be used.

Corneal collagen crosslinking with riboflavin (C3-R)

Corneal collagen crosslinking with riboflavin (C3-R) is the name given to the treatment that combines the use of riboflavin(vitamin B2) with ultraviolet light for the treatment of keratoconus.

The riboflavin 0.1% eyedrops in 20% dextran are activated by approximately 30 minutes illumination with UV-A (370nm) light. This treatment is applied to deepithelised cornea. The currently used UVA radiant exposure of 5.4 mJ/cm2 and the corresponding irradiance of 3 mW/cm² is below the known damage thresholds of UVA for the corneal endothelium, lens, and retina¹³. C3-R augments the collagen cross-links within the stroma and so recovers some of the cornea's mechanical strength ¹⁴ (fig 7).

The treatment has been shown to slow or arrest the progression of keratoconus, and in some cases even reverses it, particularly when applied in combination with intracorneal ring segments¹⁵. *In these cases, C3-R treatments stabilize keratoconus from getting worse as well as help the Intacs reverse the keratoconus steepening that had already occurred up to the time of the treatment.* The need for penetrating keratoplasty might then be significantly reduced in keratoconus.Clinical trials are continuing, and to date relatively few procedures have been performed but the technique is definitely showing promise in treating early cases of the disease.^{16,17,18} It is being opted by an increasing number of practitioners.

Corneal transplant

Approximately 10% to 25% of cases of keratoconus will progress to a point where vision correction is no longer possible, thinning of the cornea becomes excessive, or scarring as a result of contact lens wear causes problems of its own, and a corneal transplantation becomes required^{1, 2}.



Figure 8: Pre and post-PK clinical photograph of a keratoconus patient



Penetrating keratoplasty(PK) has been the gold standard surgery for keratoconus patients with success rates of more than 90%¹⁹. In this procedure, the keratoconic cornea is prepared by removing the central area of the cornea, and a full-thickness corneal button is sutured in its place. Usually trephines between 8.0-8.5 mm are used. Fleischer's ring can be used as the limit of the conical cornea. Generally, the second eye is not grafted until the first eye is successfully rehabilitated. Depending on the criteria used to assess the success rate, this surgery is 90% to 95% successful. Most of these patients who are grafted for keratoconus are younger than the majority who are grafted for other reasons. Contact lenses are often required after this procedure for best visual rehabilitation. (Figure 8)

An alternative is *lamellar keratoplasty*, a partial corneal transplant. The cornea is removed to the depth of posterior stroma, and the donor button is sutured in place. This technique is technically difficult, and visual acuity is inferior to that obtained after penetrating keratoplasty. As a result, use of lamellar keratoplasty is largely confined to the treatment of large cones or keratoglobus when tectonic support is needed. This technique requires less recovery time, and poses less chance for corneal graft rejection or failure. Its disadvantages include vascularization and haziness of the graft. (Figure 9)

Lamellar keratoplasty has recently been almost replaced by an alternative highly rewarding procedure of *deep anterior lamellar keratoplasty* (DALK)^{20,21,22}. In DALK, the patient's corneal endothelium is retained, giving some additional structural integrity to the post-graft cornea. Because a graft rejection usually begins in the endothelium, the chance of a rejection episode is greatly reduced. DALK thus provides lower postoperative complications, faster postoperative recovery, fewer graft rejections and similar visual outcomes compared to PK. It is however a technically demanding procedure.

A rarely performed but once tried procedure, *thermokeratoplasty*²³ involved placing a hot ring (Holmium yag laser, 2100nm) along the base of the cone to heat and traumatize the cornea, resulting in a corneal scar which reduces the corneal curvature, and allows a flatter contact lens to be fitted. The disadvantages of the procedure were a transitory corneal haze, development of corneal scarring and the fact that it does not preclude future keratoplasty.

*Epikeratoplasty*²⁴is primarily suited for contact-lens-intolerant patients in whom scarring has not yet occurred. In this procedure, the central host epithelium is debrided, and the donor cornea is sewn over the keratoconic cornea. This is a rarely performed procedure today as the outcome is generally less favorable.

Phakic intraocular lens implantation is being considered for keratoconus patients. It can be used to correct high myopia and associated astigmatism of selected keratoconus patients. Anterior chamber phakic intraocular lens have also been combined with intacs with good results^{25,26}. The Intacs implantation is followed by toric phakic intraocular lens implantation to correct the residual myopic and astigmatic refractive error.

Thus to conclude, a number of treatments have been tried in keratoconus. Newer modalities like INTACS, C3R and newer contact lens designs are continually helping us achieve better visual rehabilitation and delay the penetrating keratoplasty which is the standard treatment for advanced cases. RGP contact lenses are the mainstay in the rehabilitation of keratoconus patients. This holds true even though the patient may have undergone other procedures like keratoplasty,INTACS and C3R.

Before subjecting the keratoconus patients to any surgical procedure, he should be given a trial of rigid gas permeable contact lenses. Only if they are intolerant to these lenses, they should be considered for any surgical procedure.

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Color Doppler in Evaluating Ocular Blood Flow in Glaucoma

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The pathogenesis of glaucomatous optic atrophy has remained a matter of controversy since the mid-19th century when two concepts were introduced in the same year. In 1858, Muller¹ proposed that elevated intraocular pressure (IOP) led to direct compression and death of the neurons (the mechanical theory), while von Jaeger¹ suggested that a vascular abnormality was the underlying cause of the optic atrophy (the vascular theory). Enough evidence now supports vascular insufficiency, perfusion deficits of optic nerve head, choroid, retina in the etiopathogenesis of glaucomatous optic neuropathy.¹

Autoregulation is a local regulatory mechanism wherein the blood flow to the tissues is adjusted according to its requirement at different times. Dysregulation implies a mismatch between tissue need and blood flow, which causes over- or underperfusion. Constant underperfusion leads to tissue damage, and atrophy whereas unstable perfusion (underperfusion followed by reperfusion) leads to oxidative stress.^{2,3} In glaucomatous states autoregulation becomes defective and unstable perfusion is the biggest culprit in causing glaucomatous optic neuropathy.⁴

Evidence for Vascular etiopathogenesis for glaucomatous optic neuropathy

Normal tension glaucoma (NTG) with possible associations of vasospastic angina, migraine headache, Raynaud's disease indicates that other factors might also be involved in the pathogenesis of glaucomatous optic neuropathy, either damaging the eye directly or rendering it more sensitive to IOP³. Elegant studies have demonstrated neuroretinal rim blood flow values to correspond with the regional visual field defect in NTG eyes.^{56,7}

Glaucomatous visual field loss typically reflects the horizontal meridian which is supposed to reflect the distribution of vascular arcades.

Ocular hypertension: Optic nerve head (ONH) autoregulation is intact in ocular hypertension but defective in open angle glaucoma.⁶, ⁸ Studies have demonstrated reduced rim perfusion in high-risk ocular hypertensives before manifestation of field defects.⁶ Highrisk ocular hypertensives have reduced blood flow when compared with low-risk ocular hypertensives / normals⁹

In asymmetric glaucoma patients, lesser blood flow has been documented in the more affected eye.¹⁰

Progressive glaucomatous damage can and does occur despite IOP control. Visual field deterioration in patients with high ophthalmic artery resistive index (diminished ocular blood flow) has been shown to be much higher that with those with a lower

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resistive index.¹¹ Decreased blood flow in the small retrobulbar vessels and presence of vascular risk factors has been incriminated in progressive optic nerve damage despite IOP remaining controlled.^{5,12,13,14}

Gluatamte mediated ganglion cell neurotoxicity is consistent with ischemic insult since glutamate accumulation is a primary response to cellular ischemia.¹⁵

Blood pressure:

- In NTG patients there is increased variability of nocturnal blood pressure, which translates into fluctuating ocular perfusion (in the absence of effective autoregulation), thereby causing ischaemic episodes at the optic nerve head.^{16,17,18}
- Low diastolic pressure has been documented to be a risk factor in open angle glaucoma in Baltimore and Barbados eye studies.^{19,20}

Types of dysregulation

Primary dysregulation leads to a unstable oxygen supply. Reperfusion injuries lead to diffusion of endothelin and metalloproteinases to the optic nerve head causing vasoconstriction, venous occlusion and result in splinter hemorrhages.²¹ Its role in primary open angle glaucoma and normotensive glaucoma is now unquestioned. ²² Primary dysregulation is most evident in racial and gender based vulnerability to normal-tension glaucoma, for example in Japanese and women respectively. ²¹

Secondary dysregulation is due to autoimmune diseases like rheumatoid arthritis, giant cell arteritis, systemic lupus erythematosus, multiple sclerosis, ulcerative colitis. ⁴ Such patients have a high level of circulating endothelin-1 (ET-1), which causes diminished blood flow to optic nerve head and choroid.

Techniques of blood flow imaging

Colour Doppler imaging (CDI)^{12, 23}

Color Doppler optical coherence tomography (CDOCT): Simultaneous cross-sectional imaging of tissue microstructure and velocimetry of blood flow in living tissues.^{24, 25}

Pulsatile ocular blood flow (POBF): A pulsatile waveform is created in the eye with each heart beat, which is measured with a pneumotonometer. POBF measures 70% of the total blood flow to the eye which is primarily posterior ciliary in origin. Central retinal artery only accounts for 10 % of the ocular blood flow. It calculates the blood flow based on amplitude of IOP variation.²⁶ POBF measures are inversely related to axial length. An average of 5 readings is advisable.^{27, 28, 29} Measure is positively correlated with CCT (increase of 0.38 mmHg in IOP/ 10 micrometer increase of CCT).³⁰



Heidelberg retina flowmeter (HRF)^{5, 6}

Scanning laser fluorescein and indocyanine green (ICG) angiography of the peripapillary choroid and the retinal circulation,

Scanning laser Doppler flowmetry (SLDF).³¹

Colour Doppler imaging (CDI)

Color Doppler Imaging (CDI)^{32,33} is one of the important technique for non-invasive assessment of retro bulbar circulation. It is a method of detecting changes in the frequency of sound reflected from flowing blood, allowing estimation of flow velocity. The technology of duplex scanning allows simultaneous B-mode imaging and pulse wave Doppler facility. A triplex display is highly desirable for the operator convenience. High frequency probes of 7.5-10 mHz frequency are suitable and linear transducers with medium sized footprint are preferable.

First of all, a B-scan image is obtained (Figure 1) and the globe, retro bulbar space and the optic nerve are identified. Colour Doppler is applied on the b-scan image to identify the desired vessel. (Figure 2) The vessel is next interrogated by placing a sample volume curser angled approx 60 to the flow, and a waveform is obtained.

Since the lumina of the vessels in the eye and orbit are too small to be imaged with conventional Duplex scans, Doppler spectra are obtained without precise localization and without knowledge of the Doppler angle. To facilitate localization and assessment of the Doppler angle, the two-dimensional flow information in color Doppler imaging (CDI) is encoded in color and superimposed on the gray-scale image (B-scan anatomic detail) of the structure.^{34, 35}

Detected motion is assigned a color usually red or blue. When blood is flowing towards the transducer color is depicted as red, and when it flows away from transducer it is depicted as blue. (Figure 3) and colour gain are adjusted so as to obtain complete filling of vessels without the presence of artifacts.

The hue and intensity color changes with change in Doppler shift frequency. Usually a bright colour is used to designate a high velocity flow. The color saturation in the image represents the average frequency (first moment average) from a spectral analysis performed at each sample site. These frequencies can be turned into velocities by solving the Doppler equation for velocity.³⁶

Doppler frequency shifts are received from a specific sample volume. This sample volume is placed over a vessel of interest, and the frequency shifts received can be assembled into a spectral wave form.

The velocities present in the sample volume follow the cardiac cycle, allowing measurements to be taken at the peak of systole (Peak systolic velocity {PSV}) and at the lowest point of diastole (end diastolic velocity {EDV}). Both of theses measurements are dependent on the angle subtended between the probe and the vessel, the Doppler angle. The Doppler formula used to compute the blood velocity takes this angle into consideration. Because the *PSV and EDV are both dependent on the Doppler angle*, they are both regarded, to a degree, as operator dependent. To relate the systolic and diastolic velocities to each other, a ratio, the *resistive index* (*Pourcelot's index*), *is used. This ratio is angle independent* and is





regarded as a good method to quantify the vascular resistance of the circulation. $^{\rm 37}$

OPP=2/3 (MAP-IOP)¹¹, where MAP= mean arterial pressure

Ocular perfusion pressure (OPP) in the sitting position is defined by the equation: This equation shows that in normotensive patients increased IOP leads to decreased ocular perfusion. $^{\rm 38}$





Figure 5: Central retinal artery imaged in the substance of optic nerve. Note the short wave forms at the bottom, indicating low flow

Technique

An Ultrasound probe is applied over superotemporal part of closed eye lids in supine position. Methyl cellulose coupling gel is employed. The patient is asked to fix his/her gaze toward the ceiling and restrict ocular movements The probe is pointed towards orbital apex by tilting it approximately 20-30° to saggital plane. The same transducer is applied on common carotid artery. A normal carotid waveform rules out carotid obstruction. It is advisable to obtain the waveform parameters from the ipsilateral internal carotid artery for reference values

The vessels imaged are:

- Ophthalmic artery (OA),
- Central retinal artery (CRA)
- Short and long posterior ciliary arteries. (SPCA) ²³

The parameters assessed are:

- Peak systolic velocity (PSV)
- End-diastolic velocity (EDV)
- Resistive index (RI)

Ophthalmic artery

The ophthalmic artery is imaged as it courses just lateral to the hypo reflective stripe representing the optic nerve, before it changes its course to cross the nerve in the posterior half of the retro bulbar space (Figure 4).

Central retinal artery

The central retinal artery and accompanying central retinal vein can be seen within the anterior 2mm of the optic nerve shadow. The CRA can be traced up to where it enters the optic nerve approximately 13 mm behind the optic disc.^{36,39,40} Branches of CRA supply surface nerve fibre layer (Figure 5).

Short posterior ciliary arteries

The short posterior ciliary arteries are imaged as colored pixels as they pierce the ocular wall around the optic nerve head in the posterior part. These arteries comprise the main source of blood supply to the optic nerve head and supply prelaminar and laminar regions from branches of their Zinn Haller circle (Figure 6).^{39,40,41}

The value of CDI as a diagnostic tool has been validated by various authors wherein decreased flow velocities and increased vascular resistance in both central retinal artery (CRA) and posterior ciliary arteries (PCA) have been documented in open angle and normotensive glaucoma patients.^{8,12,23,31}

In addition, either spontaneous or artificial elevation of IOP was associated with decreased flow velocity and increased resistive index in the CRA and PCA. Consistent control of IOP after trabeculectomy has shown an improvement in ocular blood flow by CDI.⁴²

Limitations of CDI

• If gains are too high or display threshold is to low, noise would overwhelm the image.



- Imaging is angle dependent
- Velocity of blood is measured at specific points, not the overall velocity, the latter would need vascular dimensions,
- Reliability and reproducibility of its measures are least for SPCA
 , which comprises the bulk of optic nerve head supply.⁴³
 Reliability and reproducibility is best for Ophthalmic artery.⁴⁴

Current Relevance of ocular blood flow measurement in glaucoma:

Diagnostic: Vascular evaluation of blood flow is an alternative method to study the optic nerve head status and function and could serve as a guide to monitor and assess therapy.

Therapeutic implications: Modalities or substances which improve ocular blood flow would have a definitive role in halting glaucoma progression independent of their effect on IOP.

- Aspirin by stabilizing microcirculatory flow aspirin improves optic nerve head perfusion.⁴⁵
- Ginkgo biloba by increasing ocular blood flow and platelet activating factor inhibitory activity it has been demonstrated to improve ocular blood flow. This drug is now being extensively studied for improving vascular perfusion in glaucomatous eyes.⁴⁶
- Calcium channel blockers also act by improving ocular perfusion.⁴⁷
- Unoprostone with a antiendothelin-1 effect, betaxolol with its calcium-channel blocker action, and carbonic anhydrase inhibitors all have been documented to increase the retinal circulation.⁴⁸ All these drugs are touted as neroprotective due to their effect on optic nerve head circulation.
- Systemic Blood pressure: Drop in nocturnal systemic blood pressure is to be avoided while treating coexisting hypertension in glaucoma patients, since it is particularly deleterious for the optic nerve head.¹⁷
- Trabeculectomy has been documented to improve ocular hemodynamics along with IOP control⁴²

That there is a flip side of this therapeutic effect of improved vascular perfusion is also evident. $^{\rm 49,\,50}$

The moot question which defies clarification is whether diminished blood flow is the cause of glaucomatous optic nerve damage or it occurs subsequently, the victim of a sick optic nerve (damaged due to apoptosis or mechanical effects of intraocular pressure). Or is it as Grieshaber suggests that mechanical and vascular theories are not mutually exclusive; on the contrary, a vascular dysregulation increases the susceptibility to intraocular pressure.⁴

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Orbital Cellulitis, A Clinical Emergency for Ophthalmologists

Sanjay Teotia MBBS, DOMS

Orbital cellulitis is defined as purulent inflammation of the Cellular tissue of the orbit. Orbital Cellulitis is one of the clinical emergencies that ophthalmologists encounter. Orbital Cellulitis is a condition mostly, affecting Children and young adults and causes inflammation & Distension of lids, fever, Peiorbital pain, redness, swelling, local rise of temperature, chemosis, Proptosis, painful or difficult eye movement, decreased vision & ophthalmoplegia.

Etiopathogenesis

Majority of instances of orbital cellulitis are secondary to sinusitis, most commonly involving ethmoid sinus or secondary to Dental infection, Dacryocystitis, Subacute bacterial endocarditis, Injury penetrating orbital septum, Oribtal foreign bodies. It has been estimated that 80% of Orbital cellulitis is secondary to paranasal sinus Infections. Most commonly ethmoid due to extreme thinness of the lamina papyracea, the plate of bone that separates the medial orbit from the ethmoid. In addition, blood flows freely between the ethmoidal and ophthalmic veins. For these reasons, direct contamination of orbit with septic material from the ethmoids is all too easy. There are multiple neurovascular foramina present in the orbit that are pathways for spread of infection.¹

The causative organisms are streptococcus pneumoniae S. aureus, Haemophilus Influenzae, staphylococci, Moraxella, Pseudomonas, Klebsiella, atypical mycobacteria, mycobacterium tuberculosis Mucormycosis and aspergillosis is typically found in diabeties and immunocompromised patients.^{2,3}

Orbital Cellulitis may be graded in terms of clinical severity. The mildest form is early inflammatory edema, with swollen lids and slight proptosis with actual polymorphonuclear cellular and bacterial infiltration of the orbit (True orbital cellulitis). Vision and ocular motility may begin to decrease if an abscess forms between the bony wall of orbit and the periorbita, the eye is usually deviated down and out. The abscess may rupture through the orbital septum and present in the eyelid. An abscess formation that begins in the orbit results in more marked proptosis, (Figure-1) chemosis, ophthalmoplegia and reduction in visions. If vision and motility become impaired in the contralateral eye, one must consider the true emergency of cavernous sinus thrombosis.

On local examination unilateral axial proptosis is seen which is later non-compressible. The lids are swollen, conjunctiva congested, (Figure-2) ocular movements are painful and decreased in all directions equally by these signs we can rule out the possibility of preseptal cellulites (Figure-3) where ocular movements are usually present. There is pupillary abnormalities and disc edema present although it is difficult to examine disc and pupil because of swollen eyelids and irritable state.

> C.H.C., Muradnagar, Ghaziabad (U.P.)

Investigation

A case of orbital cellulitis should be thoroughly investigated. A complete Hemogram with differential count, while Blood cell count is more increased in orbital cellulitis as compared to preseptal



Figure1: Orbital Abscess



Figure 2: Rt. Eye Showing Swollen Lid and Cnjunctiva Congested



Figure 3: Preseptal Cellulitis

Management of Oribtal Cellu	ılitis			
Medical Management	Broad Spectrum IV Antibiotics	IV Cefuroxime (0.75 – 1.5gm 8 hrly)Or Ceftriaxone (1 – 2 gm. / day)Or Combination of Vancomycin (15 mg/ kg. 12 hrly) with Tobramycin (1–1.5 mg./kg. 8 hrly).Or Amikacin (15 mg. / kg./day).		
	For Anaerobic Organisms	Metronidazole (10 mg/kg. in three divided dose)		
	IV antibiotics should be given till improvement of signs & symptoms of orbital cellulitis later on start oral antibiotics.			
Management of Orbital Cellulitis due to fungal infection	 i) Correction of systemic metab ii) I.V. Amphotericin – B 1 mg./l iii) Surgical debridement of necret 	olic disturbances. kg./day. Total dose 2 to 4 gms otic tissues.		
Surgical Management	 i) Drainage of Abscess ii) Restoration of normal sinus of iii) Subperiosteal Abscess can be 	drainage into nose. drained extraperiosteally without entering the orbit.		

celulitis, blood culture, gram staining and culture of any discharge must be done, orbital ultrasonography gives information about the pus pockets. C.T. Scan is more important. CT scan of orbit and Sinuses shows an abscess and is diagnostic. We can rule out sinusitis, intracranial pathology and orbital foreign body with the help of C.T. Scan⁵.

Complications

Orbital cellulitis is a serious condition. Any delay in diagnosis and treatment or Inappropriate treatment can lead to serious complications like meningitis or cavernous sinus thrombosis. The urgency of treating this condition is stressed because purulent (Septic) optic neuritis

can cause permanent loss of vision within several hours. Other complications of orbital cellulitis are exposure keretitis, Raised I.O.P., CRAO, CRVO, optic atrophy subperiosteal abscess, orbital abscess, decreased vision, brain abscess, bacteremia etc.^{4,6}

Management

Medical Management:- In preantibiotic era, approximately 17% of patients with this disease died from meningitis or cavernous sinus thrombosis, and approximately 20% of the cases terminated into blindness. The availability of antibiotics will do today's patient no good if the correct diagnosis is not made and if the appropriate drugs are not administered.⁴ Treatment of orbital cellulitis consists of administering broad spectrum intravenous antibiotics followed by oral antibiotics. Initial broad spectrum antibiotics effective against Gram +ve and Gram –ve anaerobes, later specific antibiotics according to culture I.V. cefuroxime (0.75 – 1.5 gm. 8 hrly) or ceftriaxone (1 – 2 gm/day) or combination of vancomycin (15 mg / kg. 12 hrly) with

Tobramycin (1-1.5 mg./kg. 8 hrly) or amikacin (15 mg./kg/day). It is also useful to cover for anaerobic organisms with metronidazole (10mg./kg. in three divided dose) especially in cases occuring after trauma. Total duration of treatment 10 days to 3 weeks depends on patient response (decrease in orbital congestive signs such as proptosis, gaze limitation, edema) Corneal lubricants should be given for exposure keratitis.^{5,8,10} Intravenous antibiotics should be continued till an afebrile period of at least 4 days and improvement of signs and symptoms of orbital cellulitis. Later on start oral antibiotics, antimicrobial therapy should be modified according to reports of culture & sensitivity. If patient has sinusitis than Antihistaminics and nasal decongestants are given which helps in sinus drainage.

Surgical Management:- Surgical Intervention is required in case of abscess, foreign body, worsening of proptosis despite 48 hrs. of intravenous antibiotics, progression of vision loss and worsening of ocular motility.

Surgical Procedure required drainage of abscess and restoration of normal sinus drainage into the nose. Subperiosteal abscess can be drained extraperiosteally without entering the orbit. Sub periosteal abscess drainage is not required always but in case of afferent pupillary defect, vision is decreased, cavernous sinus thrombosis, not responding to intravenous antibiotics, severe proptosis drainage is must.⁷

Management of orbital cellulitis due to fungal infection:- Orbital Cellulitis may be due to fungal infection particularly mucormycosis and aspergillosis. Mucormycosis is more common in diabetics and immunocompromised persons. Treatment of orbital fungal infection requires correction of systemic metabolic disturbances, Intravenous Amphotericin-B 1 mg./kg./day total dose 2 to 4 gm. and requires surgical debridement of necrotic tissues. The prognosis for both conditions are very serious with mortality rate reaching 43% for mucormycosis and 80% for aspergillosis.⁹

Despite the advent of more potent antimicrobials orbital cellulitis is still a potential killer because of delay in diagnosis, inadequate treatment of virulent and resistant strains otherwise results are satisfactory if cornea or optic nerve are not involved early.

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DKUSEN

Dr. Mohita Sharma for completing Fellowship of International Council of Ophthalmology (ICO) in "Vitreoretina" from University of Regensburg, Regensburg, Germany in July 2008.

MACULA

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Robotic Eye Surgery: da Vinci System

Subrata Mandal MD, FRCS, Prashant Naithani MD, FICO

undreds of robotic surgical systems worldwide are currently used for performing minimally invasive abdominal, urology, cardiothoracic, and orthopedic surgeries. However, robotic surgical systems have not yet been applied clinically to intraocular surgery. Intraocular ophthalmic surgery is unique, because the surgeon obtains a direct three-dimensional, high magnification view of the inner ocular structures through the cornea. In the robotic system, during the operation, the surgeon manipulates a set of controls known as the master. These are connected through a highperformance computer to the robot. The robot's limbs move in exactly the same way, except that the movements can be scaled down as much as a thousand times, thus eliminating hand tremor and reducing damage to the eye. The computer also creates a threedimensional view of the inside of the eye, which the surgeon can see wearing a virtual reality helmet and "feel" via a sensory feedback system which emulates the forces generated by cutting with a surgical tool. Robotic eye surgery may prove attractive if it can save time, reduce surgical complications, and open the door for more delicate surgical manipulations.

Studies are ongoing between researchers in the Jules Stein Eye Institute, Center for Advanced Surgical and Interventional Technology (CASIT), and the Henry Samueli School of Engineering and Applied Science to assess the feasibility of robotic intraocular surgery using existing robotic surgical systems, and to develop new technologies to support future robotic intraocular surgical efforts. The investigators have published two articles regarding the feasibility study and use of intraocular robotic surgery on porcine eyes with the da Vinci surgical system.^{1,2}

The da Vinci Surgical System (Intuitive Surgical, Sunnyvale, California, USA) incorporates three-dimensional stereoscopic vision with three robotic slave arms that can be equipped with instruments and have 7 degrees of freedom and wrist-like motions. The operating microscope has till date remained the preferred standard in ophthalmic centres throughout the world but this might change with the unique attributes of the da Vinci robot, which is suitable for minimally invasive surgery, make it an attractive option for performing intraocular microsurgery, a discipline that demands optimal visualization, minimalization of tremor, technical skills and precise surgical manipulations.

Ocular robotic surgery technique: After placing the prepared porcine globe in a human manikin head in the anatomical position, the operating room table was rotated 90° relative to the robotic cart. Visualization of the eye was achieved with a 0°, upward-facing, three-dimensional endoscope placed above the globe in the midline, mimicking the axis of standard ocular surgery using the operating microscope. The arm ports for the 8-mm robotic instrumentation were placed on either side of the globe at about 45° angles from the axis created by the mid-line position of the endoscope. The surgeon was seated at the surgical console, about 15 feet from the surgical table and robotic cart. The surgeon viewed the operative field via a three-dimensional image while his hands



Figure 1: Robot draped and positioned over the head and eye

held the master controls at a comfortable distance below the display. Each slave arm was equipped with sterile black diamond microforceps. (Figure 1)

Initially the investigators reported the repair of a corneal laceration in a porcine model.¹ Later on using modified robotic instruments, 25-gauge pars plana vitrectomy, intraocular foreign body removal, and anterior capsulorhexis have been reported with the da Vinci system on porcine eyes.² They assessed the surgical system's ability to provide the control, dexterity, maneuverability, and visualization necessary for intraocular surgery. They found that the control of the robotic wrist-like instruments allowed for full range of movement and the dexterity of the robotic arms was also excellent. However, controlling the robotic arms was not as intuitive as moving the wrist. A high stable point of rotation induced motion-related stress at the site of instrument insertion. Visualization of the external operative field during intraocular procedures required camera realignment, and absent retroillumination made anterior segment surgery difficult to perform. Other major drawbacks are the expense and slower speed of surgery than with standard ophthalmic microsurgical instruments.

Conclusions: The da Vinci Surgical System provides adequate dexterity for performing delicate intraocular manipulations. In the current design, the kinematics of the robotic arms is found to be insufficient for standard intraocular surgery. The system's endoscope do not did not yield the same detail acquired by an ophthalmic microscope. However these initial findings support the use of a surgical robot for ocular surgery and establish a foundation for further investigation of the feasibility and applicability of robotic systems in controlled human trials.

References

- 1. Tsirbas A, Mango C, Dutson E. Robotic ocular surgery. British Journal of Ophthalmology 2007;91:18-21.
- Bourla DH, Hubschman JP, Culjat M, Tsirbas A, Gupta A, Schwartz SD. Feasibility study of intraocular robotic surgery with the da Vinic Surgical System. Retina, 2008;28(1):154-8.

Dr. Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi

Bileteral Localised Orbital Castleman's Disease

Shreekant A Damgude MBBS, Anuj Mehta MS

Mehta MS ally in each thorax and

A 14 year old girl presented to Dept of Ophthalmology, Safdarjung Hospital with chief complaints of gradual onset painless progressive swellings of both upper eyelids of 2 years duration (Jan 2007). Swellings were noted simultaneously with their sizes being approximately similar bilaterally throught the progression. There was no history of fever , night–sweats, weight loss or weakness (Figure 1).

General and systemic examination revealed no significant findings. Ophthalmic examination revealed two swellings (4cm x 3cm each) placed deep in superolateral aspect of each orbit. They were globular, firm, nontender, non pulsatile, non- reducible with lobulated surface and well-defined margins , posterior extent being not palpable with no effects on valsalva or on beding forward, with no adherence to underlying bones, with no palpable thrill or auscultatory bruit or trans-illumination. Visual acuities were 6/6 for both eyes with parallel axes with no proptosis. Extraocular muscle movements were restricted superolaterally due to mass effects in both eyes. Fuduscopy was normal in both eyes.

Differential diagnosis

Lymphoproliferative disorder, Sarcoidosis, Mikulicz syndrome, amyloidosis, pseudotumour and tuberculosis was considered.

Invetigation

Hematological parameters showed elevated ESR. Serological reports of RA factor, VDRL, ANA, ELISA for HIV were negative. Mantoux test was negative.

USG orbit showed bilateral enlargement of lacrimal glands. Orbital CECT scans and MRI showed bilateral extraconal masses of about



Figure 1: Preoperative

Department of Ophthalmology Safdarjung Hospital, New Delhi 40-43 HU suggestive of soft tissue density superolaterally in each orbit. Brain CECT scans and MRI were normal. CECT thorax and USG abdomen were normal (Figure 2, 3 & 4).

Incisional biosy showed lymphoid follicle hyperplasias with vascular hyperplasias, enlarged follicular area, interfollicular excess hyaline tissue, lymphocytic infiltrate, crushed lymphocytes.

These histopathological findings were suggestive of hyaline vascular type of Castleman's disease (Localised unicentric variant) (Figure 5).

Discussion

Castleman's disease was first described by Dr Benjamin Castleman, Pathologist from Massachusetts General Hospital in 1954 as "Rare lymphoproliferative disorder of idiopathic origin consisting of nonneoplastic abnormal proliferation of lymph node follicles⁽¹⁾."

Clinical features range from asymptomatic discrete lymphadenopathy to severe systemic symptoms.





Figure 2: Preoperative CT scan



Figure 3: CECT bone window



Figure 4: T1W MRI, 1.5T, C

It is broadly classified as:

Hyaline Vascular (90%)

• Commonly unicentric

Plasma cell type

- Unicentric / Multicentric
- Fever, weight loss, skin rash
- Hemolytic anaemia
- Plasmablastic (POEMS) Polyneuropathy, Organomegaly, Endorinopathy, Monoclonal proteins, skin changes

Mixed

Incidence

30000 to 10000 cases in $US^{(1)}$

Casper C .The aetiology and management of Castleman disease at 50 years: translating pathophysiology to patient care. Br J Haematol. 2005 Apr;129(1):3-17.

Review

1 case/100 orbitotomies⁽²⁾

Snead M.P. Orbital lymphomas and Castleman's disease Eye (1993) 7,84-88

There have been just 7 case reports of Orbital Castleman's disease in literature only two of which were bilateral and unicentric. (Table)

According to literature surgical excision provides maximum cure in localised unicentric variety of Castleman's Disease^(3,4). Hence bilateral surgical excision (In toto) was done in this case through subbrow incision. Postoperatively oral steroids were given as a short course to relieve oedema rapidly. (Figure 6a,6b, 7)

Patient is under follow-up and no recurrence is found till 4 months postoperatively (Figure 8).

Other treatment modalities⁽¹⁾:

- Cytoreductive therapy
 - CHOP/CVAD regimen
- Radiotherapy⁽⁵⁾



Figure 5





Figure 6a: Bilobed, about 5cm x 3.5 cm in size, Arising from Lacrimal Gland, Yellowish in Colour, Smooth, Firm, Rubbery Consistency



Figure 6b: Bilobed, About 4.5cm x 3cm, Arising from Lacrimal gland, Yellowish in colour, Smooth, Firm, Rubbery Consistency

S. No.	Authors	Yr./Journal	Туре	B/L or U/L	Treatment	Recurrene
1	Gittinger J. W., et al	1989, Ophthalmol	multicentric	U/L	Radiotherapy	Good outcome
2	Snead M.P. et al	1993, Eye	Unicentric/HVV	U/L	Surgical Excison	No recur. till 5 yrs.
3	T. Kurukawa, et al	1999, AJO	multicentric	B/L	Corticosterod	Died d/t aspergillus, CMV
4	Park K.S. et al	2002,Acta Opht.Scand	Unicentric/ HVV	B/L	Surgical Excison	1 yr F/U – no recur.
5	Alhaya et al	2002, Acta Opht.Scand	Mixed/ unicentric	B/L	Corticosterod, Immuno- suppressive, RT	No recur. till 7 yrs.
6	Ide M et al	2003, Br J of Haemat	multicentric but HVV (unusual)	B/L	Rituximab (Anti- CD20 Antibody)	No recur. till 10 months
7	Inatani M. et al	2005, Jpn J Ophthal	Mixed multicentric	U/L	Rituximab (Anti- CD20 Antibody)	Turned into NHL





- Interferon alpha
- All-trans retinoic acid
- Thalidomide (Antiangiogenic)
- Anti-IL-6 monoclonal antibody
- Anti- IL-6 receptor antibody (Atlizumab)⁽⁶⁾
- Rituximab (Anti CD-20 antibody) (In CD-20 +ve patients) (7)
- Antiviral therapy in HHV-8 +ve cases

(Ganciclovir, foscarnet, cidofovir)

References

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- Nishimoto N. Humanized anti-interleukin-6 receptor antibody treatment of multicentric Castleman's disease.Blood.2005 Oct; 106(8): 2627-2632.
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Glaucoma Risk and the Consumption of Fruits and Vegetables Among Older Women in the Study of Osteoporotic Fractures

Coleman AL, Stone KL, Kodjebacheva G, Yu F, Pedula KL, Ensrud KE, Cauley JA, Hochberg MC, Topouzis F, Badala F, Mangione CM; Study of osteoporotic fractures research group.

Department of Ophthalmology and Jules Stein Eye Institute, Los Angeles, California; School of Public Health, Los Angeles, California.

Am J Ophthalmol. 2008 Mar 19 [Epub ahead of print]

PURPOSE

To explore the association between the consumption of fruits and vegetables and the presence of glaucoma.

DESIGN

Cross-sectional cohort study.

METHODS

In a sample of 1,155 women located in multiple centers in the United States, glaucoma specialists diagnosed glaucoma in at least one eye by assessing optic nerve head photographs and 76-point suprathreshold screening visual fields. Consumption of fruits and vegetables was assessed using the Block Food Frequency Questionnaire. The relationship between selected fruit and vegetable consumption and glaucoma was investigated using adjusted logistic regression models.

RESULTS

Among 1,155 women, 95 (8.2%) were diagnosed with glaucoma. In adjusted analysis, the odds of glaucoma risk were decreased by 69% (odds ratio [OR], 0.31; 95% confidence interval [CI], 0.11 to 0.91) in women who consumed at least one serving per month of green collards and kale compared with those who consumed fewer than one serving per month, by 64% (OR, 0.36; 95% CI, 0.17 to 0.77) in women who consumed more than two servings per week of carrots compared with those who consumed fewer than one serving per week, and by 47% (OR, 0.53; 95% CI, 0.29 to 0.97) in women who consumed at least one serving per week of canned or dried peaches compared with those who consumed fewer than one serving per month.

CONCLUSIONS

A higher intake of certain fruits and vegetables may be associated with a decreased risk of glaucoma. More studies are needed to investigate this relationship.

Long-term outcome of transscleral diode laser cyclophotocoagulation in refractory glaucoma

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Br J Ophthalmol. 2007 Dec;91(12):1631-5. Epub 2007 May 10.

BACKGROUND

Long-term outcome and complications of diode laser cyclophotocoagulation (DCPC) may be important, since eyes, once treated with DCPC, are less likely to be subjected to other types of interventions in the further follow-up. METHODS: Retrospective review of 131 eyes of 127 patients treated from 2000 through 2004. Success was defined as intraocular pressure (IOP) at last visit 6-21 mm Hg; hypotony: IOP </=5 mm Hg.

RESULTS

Mean follow-up (FU) was 30.1 (SD 16.7) months. Mean number of treatment sessions per eye was 1.54, 89% of the eyes having 1 or 2 sessions; overall re-treatment rate: 38.9%. Mean total laser energy delivered per eye: 133.9 (73.7) J; mean energy per treatment episode: 86.8 (22.0) J. Eyes with 3 or more treatments (11%) had a significantly larger proportion of post-traumatic glaucoma, and patients were significantly younger. All eyes had refractory glaucomas on maximal medication, neovascular glaucoma (NVG) representing the largest subgroup (61%). IOP decreased from 36.9 (10.7) mm Hg pretreatment to 15.3 (10.4) mm Hg at the end of FU. Success was noted in 69.5% (91 eyes), failure (non-response) in 13%. Hypotony occurred in 17.6% eyes, of which 74% had NVG. Hypotony developed after mean 19.3 (11.0) months, range 6 to 36; with 96% of these eyes having received only 1 or 2 treatments; delivered energy did not differ from that in the successful eyes.

CONCLUSIONS

DCPC is an efficient treatment for refractory glaucoma. Hypotony, the most common complication, may develop as late as 36 months post-treatment. Diagnostic category and age seem to influence the outcome stronger than laser protocol and delivered energy.

A Prospective Study of Early Intraocular Pressure Changes After a Single Intravitreal Triamcinolone Injection

Im L, Allingham RR, Singh I, Stinnett S, Fekrat S.

Department of Ophthalmology and Visual Sciences, University of Maryland, Baltimore, MD †Duke University Eye Center, Albert Eye Research Institute, Duke University Medical Center, Durham, NC

J Glaucoma. 2008 Mar;17(2):128-132.

PURPOSE

To prospectively monitor intraocular pressure (IOP) and gonioscopy changes within the first month after a single 4-mg intravitreal injection of triamcinolone acetonide (IVK) (Kenalog, Briston-Meyers Squibb, New York).

DESIGN

Prospective comparative interventional case series.

METHODS

A consecutive series of 28 eyes of 14 patients with no prior intravitreal injections or history of glaucoma were prospectively enrolled. After baseline evaluation in both eyes, including IOP, gonioscopy, and optic nerve evaluation, a single 4-mg IVK was given in a standard sterile fashion in the eye to be treated. Eyes received IVK for macular edema associated with retinal vein occlusions and in conjunction with photodynamic therapy for choroidal neovascularization secondary to age-related macular degeneration, ocular histoplasmosis, and high myopia. The fellow eye served as the control. After the injection, IOP and gonioscopy were repeated at 1, 2, and 4-week intervals in both eyes.

RESULTS

Of the 14 patients, the 5 women and 9 men had a mean age of 67.6 years. Mean baseline IOP of the treated and fellow control eyes were similar at 15.9 versus 16.6 mm Hg, respectively. The control eyes maintained a small IOP range (15.6 to 16.6 mm Hg) during the 1-month follow-up period. In the treated eyes, the mean maximum IOP was 54% above baseline during follow-up, compared with 11% for control eyes. Six of 14 (43%) treated eyes had IOP elevation to 24 mm Hg or higher with mean change of 8.6 mm Hg and a mean maximum IOP of 32.1 mm Hg. There was no correlation between IOP rise and age, sex, diagnosis, or optic nerve appearance. However, during the course of the study, 4 of 6 (67%) of the treated eyes that required topical drops for the IOP elevation had documented abnormal inferior angle changes characterized by pigmented particulate matter in the inferior angle not present at the baseline exam. The most frequent time point for an IOP elevation that required treatment was at 2-week postinjection. No eyes required surgical management of IOP during the course of this 4-week study.

CONCLUSIONS

We observed a significant IOP rise in eyes after a single intravitreal injection of 4 mg of triamcinolone within 1 month of injection. In this study, the most frequent time point that required IOP treatment was at 2-week postinjection, suggesting that early and frequent monitoring of IOP should be considered. Two-thirds of eyes that required medical control of IOP developed gonioscopy changes, characterized by particulate matter in the inferior angle, not present at baseline. Eyes that developed gonioscopic changes were 5 times more likely to be treated for IOP elevation than those without gonioscopic findings.





	Delhi Ophthalmological Society Monthly Clinical Meeting, July 2008							
	Dr. Rajendra Prasad Centre for Ophthalmic Sciences							
	Venue: Jawahar Lal Auditorium, All India Institute of Medical Sciences, Ansari Nagar, New Delhi -110029							
	Date and Time : 3 rd August, 2008 (Sunday)							
	First 40 Early Bird Prizes: 1GB pen drive							
	Tea Break: 10:30 AM – 10:55 AM Clinical Session : 11:00 AM - 12:00 Noon							
Cli	nical Cases:							
1.	An unusual case with medial canthal swelling	:	Rachna Meel	10 Mins				
2.	Pulsating ONH coloboma with detachment	:	Prashant Naithani	10 Mins				
Cli	nical Talk:							
Cu	rent status of retinal stem cell transplantation	:	Rajvardhan Azad	15 Mins				
Symposium: From Evolution to Revolution								
	Chairman: Prof. Supriyo Ghose Co-Chairman: Prof. Rajvardhan Azad							
1.	23 G Vitrectomy	:	Atul Kumar	10 Mins				
2.	Toric IOL Implantation	:	Jeewan S. Titiyal	10 Mins				
3.	Long-term prognosis of glaucoma in India	:	Ramanjit Sihota	10 Mins				
4.	AGV in Refractory Glaucoma - A Corneal Perspective	:	Anita Panda	10 Mins				
	To be followed by Lunch sponsored by	v M/s NRI Vision Care (M	ISTY OLO MO-4)					

Monthly Clinical Meet	ings Calendar 2008-2009
Dr. R.P. Centre for Ophthalmic Sciences	Midterm Conference of DOS
3 rd August, 2008 (Sunday)	November, 2008 (Saturday - Sunday)
Army Hospital (R&R)	Mohan Eye Institute
14 th September, 2008 (Sunday)	28 th December, 2008 (Sunday)
New Hospital/Institute	New Hospital/Institute
28 th September, 2008 (Sunday)	25 th January, 2009 (Sunday)
Sir Ganga Ram Hospital	Guru Nanak Eye Centre
26 th October, 2008 (Sunday)	22 nd February, 2009 (Sunday)
Centre for Sight	Venu Eye Institute
23 rd November, 2008 (Sunday)	15 th March, 2009 (Sunday)

Annual Conference of DOS 20th-22nd March, 2009 (Friday, Saturday & Sunday)

Annual General Body Meeting

The Annual General Body Meeting of Delhi Ophthalmological Society will be held on **Sunday the 14th September 2008 at 09.00 A.M.** in Monthly Clinical Meeting **Venue:** Army Hospital (R&R) Delhi Cantt, Dhaula Kuan, Delhi. All members are requested to attend.

> Namrata Sharma Secretary, DOS

Forthcoming Events : National

August 2008

21-24 MUMBAI

Eye Advance 2008 Contact Person & Address: Dr. Kaiki R. Mehta World Trade Centre, Mumbai The Mehta International Eye Institute Sea Side, 147, Shahid Bhagat Singh Road, Mumbai - 5 Tel.: 91- 22-22151303, Fax: 91-22-22150433 Email: admin@eyeadvance.com Website: eyeadvance.com

October 2008

2-5 CHENNAI

A National Board Post Graduate Program Contact Person & Address **Prof. Amar Agarwal** Dr. Agarwal's Eye Hospital 19, Catheral Road, Chennai-600086 Tel.: 91-44-28112811, Fax: 91-44-28115871 Email: dragarwal@vsnl.com Website: www.kalpavriksha.dragarwal.com

October 2008

2th

NEW DELHI

13th Dr. R.K. Seth Memorial Symposium on Glaucoma Contact Person & Address Dr. Abhishek B. Dagar Venu Eye Institute & Research Centre, 1/31, Sheikh Sarai Institutional Area, Phase II, New Delhi-110017 Tel.:- 011-29251155/56, 29250757, Fax - 01129252370 Email : training.venu@spectranet.com education@venueyeinstitute.org

3-4 NEW DELHI

National Workshop on Strabismus Contact Person & Address Prof. Pradeep Sharma Dr. Rohit Saxena, Assistant Professor Room No. 485, Dr. Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, Ansari Nagar, New Delhi - 110 029 Tel.: 011-26593185, Fax: 011-26588919, Email: workshoprpc@gmail.com

17-19 UJJAIN, MADHYA PRADESH

Nayan Kumbh'08

Annual Conference of M.P. State Ophthalmic Society For details contact : Dr. Arvind Bhatnagar, Chairman, Organizing Committee Celll : 98260 56021

18-20 NEW DELHI

Annual Conference of Strabismological Society of India Contact Person & Address Dr. Subash Dadeya Room No. 205 OPD Block, Guru Nanak Eye Centre, Maharaja Ranjit Singh Marg, Delhi-110 002 Tel.: 91-011-23234622 Extn.-292 Mobile: 981057899, 9868245792 E-mail: dadeyassi@gmail.com, dadeya86@hotmail.com

31st Oct. CHANDIGARH (U.T.)

2nd Nov.

18th Annual Conference of the Glaucoma Society of India Contact Person & Address Dr. S.S. Pandav Advanced Eye Centre, Postgraduate Institute of Medical Education & Research, Chandigarh (U.T.) Telefax: 0172-2747837, Email: sspandav@yahoo.com

December 2008

4-6 WEST BENGAL

XVII Annual Conference of Vitreoretinal Society of India Fort Radisson, RAICHAK, West Bengal 4th to 6th December, 2008 *Contact Person & Address* Secretary, VRSI Dr Ajit Babu Majji L V Prasad Eye Institute,, L V Prasad Marg, Banjara Hills, Hyderabad- 500 034, India E-mail: ajit@lvpei.org, Website: vrsi.in

February 2009

5-8 JAIPUR

AIOS Annual Conferences *Contact Person & Address Prof. (Dr.) P.K. Mathur* C-126, Moti Nagar, Bapunagar, Jaipur-302015 Ph.: 0141-2705972, 0141-2701030, (M) 0-9314614932 Fax: 0141-2705246, Email: pradeepmathur@hotmail.com

<u>March 2009</u>

20-22 NEW DELHI

Annual Conference of Delhi Ophthalmological Society Contact Person & Address Dr. Namrata Sharma Room No. 474, 4th Floor, Dr. Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, Ansari Nagar, New Delhi – 110029 Ph.: 011-65705229, Fax: 26588919, E-mail: dosonlin@vsnl.net, Website: www.dosonline.org

Forthcoming Events : International

July, 2008

20-25 COLORADO, UNITED STATES

Retinal Neurobiology and Visual Processing Snowmass Village, Colorado, United States *Contact:* Levin, Julie Phone: 301-634-7010, Fax: 301-634-7007 E-Mail: snowmass@faseb.org Website: http://src.faseb.org

31 July GEORGIA, UNITED STATES 3 AUG.

32nd Annual Meeting of the Christian Ophthalmology Society

Pine Mountain, Georgia, United States *Contact:* Cannon, M.D., Sterling Phone: 706-478-0764, Fax: 706-660-9191 E-Mail: COScallaway@yahoo.com

August 2008

10-15 RHODE ISLAND, UNITED STATES

Visual System Development Gordon Conference Newport, Rhode Island, United States *Contact:* Cagan, Ross Phone: 212-241-1427, E-Mail: ross.cagan@mssm.edu Website: grc.org/programs.aspx?year=2008&program=visual

22-24 NEW DELHI, INDIA

Biennial Meeting SAARC Academy

of Ophthalmology India Habitat Centre, Lodhi Road, New Delhi *Contact:* Dr. Namrata Sharma Room No. 474, 4th Floor, Dr. Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, Ansari Nagar, New Delhi - 110 029 Phone: 91-11-26593144, Fax: 91-11-26588919

25-29 MARYLAND, UNITED STATES

AFIP's Ophthalmic Pathology Course Bethesda, Maryland, United States *Contact:* Molina, TSgt. Oscar Phone: 202-782-2637, Fax: 202-782-5020 E-Mail: came@afip.osd.mil, Website: www.askafip.org

E-Mail: sao2008@gmail.com, Website: www.sao2008.org

September, 2008

13th WASHINGTON

JCAHPO Continuing Education Program Washington, District of Columbia, United States *Contact:* JCAHPO

Phone: 800-284-3937, Fax: 651-731-0410 E-Mail: jcahpo@jcahpo.org, Website: www.jcahpo.org

13-17 BERLIN, GERMANY

XXIV Meeting of the European Society of Cataract and Refractive Surgeons Contact: M Events Cross Media GmbH Heimstr. 5 a, 82152 Krailling, Germany Phone: +49 - (0) 89 - 43 56 96 58 Fax: +49 - (0) 89 - 43 56 96 59 E-mail: info@m-events.eu, Website: www.m-events.eu

24-27 GENOA, ITALY

XXVII Annual Congress of the European Society of Regional Anaesthesia (ESRA 2008) Genoa, Italy *Contact:* International, Kenes Phone: 41-22-908-0488 E-Mail: esra2008@kenes.com, Website: www.kenes.com/esra

25-27 PHILADELPHIA

ISOT 2008-11th Congress of the International Society of Ocular Toxicology Philadelphia, Pennsylvania, United States *Contact:* Peiffer, Robert Loews Hotel Philadelphia, 1200 Market Street, Philadelphia PA 19107 Phone: (215) 627-1200, Fax: (215) 231-7305

October, 2008

18-19 SWITZERLAND

Glaucoma Meeting Basel 2008 Basel, Switzerland *Contact:* Haunstein, Daniela Ph.: 41-61-2658718, Fax: 41-61-2658652 E-mail: info@glaucoma-meeting.ch Website: www.glaucoma-meeting.ch



Online Journal Available

Many New Journals at DOS Library

Dear DOS Members,

We are pleased to announce that DOS has subscribed to online access of the following 18 journals. We are also in the process of adding a few more journals. These journals can be accessed at the DOS library situated at 4th floor of Dr. R.P. Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi-110029. The timings are from 9.30 A.M. to 6.00 P.M. on week days and 9.30 A.M. - 2.00 P.M. on Saturday. The Library will remain closed on Gazetted Holidays. Members are requested to utilise the available facilities i.e. Computer with Video Editing & Conversion facility VHS to VCD, Journals Viewing, Books and Journals etc. The DOS members can get the full text articles of the current issues as well as many back issues of these subscribed journals.

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- British Journal of Ophthalmology
- Contemporary Ophthalmology
- Current Opinion in Ophthalmology
- International Ophthalmology Clinics
- Journal of Neuro-Ophthalmologica
- Journal of Refractive Surgery
- Ophthalmology Management
- RETINAL Cases & Brief Reports

- Acta Ophthalmologic Scandinavica
- Clinical & Experimental Ophthalmology
- Cornea
- Evidence-Based Ophthalmology
- Journal of Glaucoma
- Journal of Pediatric Ophthalmology & Strabismus
- Ophthalmic Surgery, Lasers and Imaging
- Retina
- Techniques in Ophthalmology

You are welcome to give any more suggestions for the improvement of the library facility and making the process simpler for us.

Looking forward to hearing from you and hope this facility would be of benefit to all of us.

Regards.

(**Dr. Namrata Sharma**) Secretary, DOS **(Dr. Vinay Garodia)** *Library Officer Incharge* Mob: 9811084552 Email: vinay@visitech.org, doslibrary@gmail.com

Delhi Ophthalmological Society

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INSTRUCTIONS

- 1. The Society reserve all rights to accepts 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 received 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. Namrata Sharma, Secretary, Delhi Ophthalmological Society, R.No. 474, 4th Floor, Dr. R.P. Centre for Ophthalmic Sciences, AIIMS, Ansari Nagar, New Delhi 110 029.
- 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).

Congratulations

- Dr. A.K. Grover Consultant, HOD, Department of Ophthalmology, Sir Ganga Ram Hospital, New Delhi was elected as Vice-President of the Asia Pacific Society of Oculoplastic and Reconstructive Surgery (APSOPRS) at the recently concluded meeting of the society at Seoul (Korea) 20-22 June, 2008 & also for being a chairperson in a session symposium on Oculoplastic Surgery at the World Congress of Ophthalmology in Hongkong (June 28th-2nd July, 2008).
- Dr. Sanjay Ahuja & Dr. Aparna Ahuja have jointly been awarded a cash prize of Rs. 20,000/- for their hindi language book on eyes titled "*Aankhein-Rog Aur Savdhaniya*" by the *Central Health Ministry, Govt. of India in April 2008*. For the same book, they had received another award in 2006 from the ICMR, New Delhi.

DOS Quiz

Anagram Time

Each of the following words is a jumbled ophthalmic or related term. There is, however, an extra letter in every set of letters. These extra letters will also form a six letter ophthalmic word when unjumbled.

So get cracking.

1. SCENTO		_
2. SURENDR		_
3. UACLAIM		_
4. ISAYSLIDE		_
5. TEXASDUNE		_
6. CHATTEDAMEN		_
Answers on page number 51	Saurabh Sawhney DO, DNB Ashima Aggarwal MS, DN Insight Eye Clinic, New Dell	<i>B</i> ni

Delhi Ophthalmological Society Fellowship for Partial Financial Assistance to Attend Conferences

Applications are invited for DOS Fellowship for partial financial assistance to attend conference(s).

Conferences

International: Two fellowships per year (two fellowships can be awarded at a time if committee feels that papers are very good)

- Maximum of Rs. 25,000/- per fellowship will be sanctioned
- National: Three fellowships per year (only for AIOS)
- Maximum of Rs. 5,000/- per fellowship will be sanctioned

Eligibility

- DOS Life Members (Delhi Members only)
- 75 or More DCRS Points
- Accepted paper for oral presentation, poster, video or instruction course.

Time since last DOS Fellowship

Preference will be given to member who has not attended conference in last three years. However if no applicant is found suitable the fellowship money will be passed on to next year. Members who has availed DOS fellowship once will not be eligible for next fellowship for a minimum period of three years.

Authorship

The fellowship will be given only to presenting author. Presenting author has to obtain certificate from all other co-authors that they are not attending the said conference or not applying for grant for the same conference. (Preference will be given to author where other authors are not attending the same conference). If there is repeatability of same author group in that case preference will be given to new author or new group of authors. Preference will also be given to presenter who is attending the conference for the first time.

Quality of Paper

The applicant has to submit abstract along with full text to the DOS Fellowship Committee. The committee will review the paper for its scientific and academic standard. The paper should be certified by the head of the department / institution, that the work has been carried out in the institution. In case of individual practitioner he or she should mention the place of study and give undertaking that work is genuine. The fellowship committee while scrutinizing the paper may seek further clarification from the applicant before satisfying itself about the quality and authenticity of the paper. Only Single best paper has to be submitted by the applicant for review (6 copies). Quality of the paper will carry 50% weightage while deciding the final points.

Poster and Video

The applicant will need to submit poster and video for review.

Credit to DOS

The presenter will acknowledge DOS partial financial assistance in the abstract book / proceedings.

The author will present his or her paper in the immediate next DOS conference and it will be published in DJO/DOS Times.

Points Awarded

•	is / thui ded	
) A	ge of the Applicant	Points
a)	< 35 years	10
b)	36 to 45 years	07
c)	45 years plus	05
2) Ty	pe of Presentation	
a)	Instructor/ Co-instructor of Course	12
b)	Free Paper (Oral) / Video	07
c)	Poster	05
3) In	stitutional Affiliation	
a)	Academic Institution	15
b)	Private Practitioner	20
4) D	CRS Rating in the immediate previous year	
a)	75-150	05
b)	> 150	08

c) < 75 not eligible for fellowship

Documents

- Proof for age. Date of Birth Certificate
- Original / attested copy of letter of acceptance of paper for oral presentation / video / poster or instruction course.
- Details of announcement of the conference
- Details of both International & National Conferences attended in previous three years.
- Copy of letter from other national or international agency / agencies committing to bear partial cost of conference if any.
- At least one original document should be provided, that is ticket, boarding pass or registration certificate along with attendance certificate of the conference.
- Fellowship Money will be reimbursed only after submission of all the required documents and verified by the committee.
- Undertaking from the applicant stating that above given information's are true.
- If found guilty the candidate is liable to be barred for future fellow-ships.

Application should reach **Secretary's office** and should be addressed to President, DOS before **31**st **July** and **31**st **January** for International Conference and before **30**th **September** for National Conference. The committee will meet thrice in a year in the month of August, October and February with in 2 weeks of last date of receipt of applications. The committee will reply within four week of last date of submission in yes/no to the applicant. No fellowship will be given retrospectively, that means prior sanction of executive will be necessary.

Dr. Namrata Sharma

Room No. 474, 4th Floor, Dr. Rajendra Prasad Centre for Ophthalmic Sciences All India Institute of Medical Sciences, Ansari Nagar, New Delhi – 110029 **Ph.:** 91-11-65705229, **Fax:** 91-11-26588919 **E-mail:** dosonlin@vsnl.net, **Website:** www.dosonline.org

Application Invited from Institutions for Holding the DOS Monthly Clinical Meetings

As per the DCRS ratings two institutions will be dropped from the monthly calender of 2008-2009. We request all the institutions/hospitals interested in holding the DOS monthly meeting to kindly see if they fulfill the criteria given below. They may apply to the Secretary's Office with details latest by 18th August, 2008.

No meeting is held in May and June. Meetings are usually held on the last Saturday of the month.

Criteria for selection of a place:

- (a) Seating capacity of 100-200 persons, preferably AC mini auditorium / hall definitely within the premises of the institutions.
- (b) Audio Visual facilities to be available

– moving mike	1 set
– multimedia projector	1 set
- double slide projectors	1 set

- (c) Institute should send the details of the meetings/CME etc., held at that institute in past 1 years to the DOS office
- (d) A sizeable staff in Ophthalmology who would be able to conduct the meeting themselves without any major outside participation as speakers/presenters.
 - Before the submission of application for holding the DOS clinical meeting, all the above mentioned criteria should be met.
 - These may be verified by President and Secretary.

– **Dr. Namrata Sharma**, Secretary, DOS

impor	tant and challengi	ng for the Ophthalmologist. Here, we d	discuss IOL power cal	lculations in some unusual case sc	enarios.	
s. No	Type of Eye	Considerations	Corneal Power (K) Measurement	AL Measurement	IOL Formula	Remarks
		The correct axial length (AL) measurement is fallacious as the		Preferably IOL Master	Holladay 2 SRK-T	Retinal Thickness Factor not incorporated by conventional A-
-	Highly Myopic	most posterior portion of the globe (Anatomic AL) may not correspond with the centre of		Or Immersion vector A/B-scan	Optimized Haigis	Scans Scans Scans And hot he mood (AT >
		fovea (Refractive AL).		Avoid Applanation A-Scan	Holladay 1	JANN II SHOUND HOU DE USEU (AL > 26mm).
7	Silicone Oil Filled	The apparent decrease in speed of ultrasound in the silicone filled eyes causes apparent increase in the AL resulting in hypermetropic errors		The IOL Master is ideal. Or Prior to IOL placement, remove the silicone oil. AL is then measured in the usual way and IOL power calculated	Holladay 2 is preferable as it compensates for the higher refractive index of silicone oil Or SRK-T	If the silicone oil is to remain in the eye for an extended period of time after cataract surgery, an adjustment in IOL power is to be made (usually about +3.0 to +3.50D) and silicone material lenses are to be avoided.
			Master K		Hoffer Q	The most important step in
20	Highly		Other options – Auto K	IOL Master	(AL 20 - 21.99mm)	calculating the IOL power is calculation of a parameter called
24	Hyperopic		Sim K Manual K	UI Immersion A-scan	Holladay 2 or optimized Haigis	effective lens position (ELP) previously referred to as the anterior chamber depth (ACD).
					(AL18.00-19.99mm)	
		Measurement of AT.		IOI. Master ideal	The Holladay 2	Optimise the lens constant for the type of lens being used and the type of formula being used
3b	Primary Piggyback	Calculation of the total capsular bag IOL Power Tunes of IOI and their		Immersion A scan is a reasonable alternative.	Or Hoffer Q	A high-powered lens with a negative form factor is recommended for
		placement		using Immersion biometry	Or	capsular bag placement. The residual lens is placed in the ciliary sulcus.
					Optimised Haigis	I his residual lens should be made up of a different material
4	Aphakic	Calculation of AL		Calculate AL using the aphakic mode of the	т ЧСS	A-constant to be noted in every type
		Power Calculation		biometer.	1-VNC	01 IUL (AUFUSF)

IOL Power Calculations in Unusual Cases

Cataract surgery is fast becoming a kind of refractive surgery. In an effort to achieve emmetropia and provide excellent quality of vision, calculation of IOL power becomes very

S. No	Type of Eye	Considerations	Corneal Power (K) Measurement	AL Measurement	IOL Formula	Remarks
υ	Previous Kerato- refractive surgery	Central corneal refractive power is the most important step. Traditional instruments for measuring K have limitations as they make assumptions that are not valid post- kerato-refractive surgery eyes They overestimate the central corneal power following myopic kerato-refractive surgery which results in a post-operative hyperopic surprise Incisional kerato-refractive surgery for myopia flattens both the anterior corneal radius and the posterior corneal radius whereas ablative kerato- refractive surgery for myopia flattens only the anterior corneal radius.	Post-RK Eyes Elevation data of the central 4.0mm from the Zeiss Humphrey Atlas topographer Or Hard contact lens over- refraction method Post Ablative Surgery Holladay clinical history method Or Or Hard Contact Lens method	Preferably IOL Master Or Immersion vector A/B-scan Or Or Immersion A-scan	Popular Formulas1. Clinical Histor1. Clinical Historis calculated brefractive surgivalues obtained2. Hard Contactsum of the conrefraction minmanifest refract3. Feiz and ManIOL power is fiundergone corcalculated usinmeasured just hadded the LASdivided by 0.7.4. Aramberri DoK is used to essurgery K is usedodified Malkand is helpful w6. Corneal Bypausing the post-target refractioequivalent7. On-line Calculation sopatients whereand the IOL po	 y method Postoperative corneal power / subtracting the change in manifest the corneal plane induced by the call procedure from the corneal power before refractive surgery Lens method K is calculated as the tact lens base curve, power, and overus the spherical equivalent of the ion without a contact lens. nis IOL power adjustment method: st determined as if the patient had not real refractive surgery. To this value is g pre-LASIK K values and the AL is efore cataract surgery. To this value is if the patient had not real refractive surgery. To this value is if the patient had not neal refractive surgery. To this value is if the patient had not real refractive surgery imate the ELP and the post-refractive surgery imate the POL power is calculated the IOL power is calculated in is set for the pre-LASIK spherical ators. Web sites like ascrs.org have fitwares for post-refractive surgery the relevant data needs to be filled in wer is calculated.
				+ Senior Specialist, Ophthal	Pankaj Lam * Lamba Ey mology Services, Employee Sta	<i>ba</i> DO, DNB, FRCS*, <i>Neeraj Varma</i> MS ⁺ e Clinic 14 A /26,WEA, Karol Bagh, New Delhi 5 te Insurance Hospital, Basaidarapur New Delhi-15

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