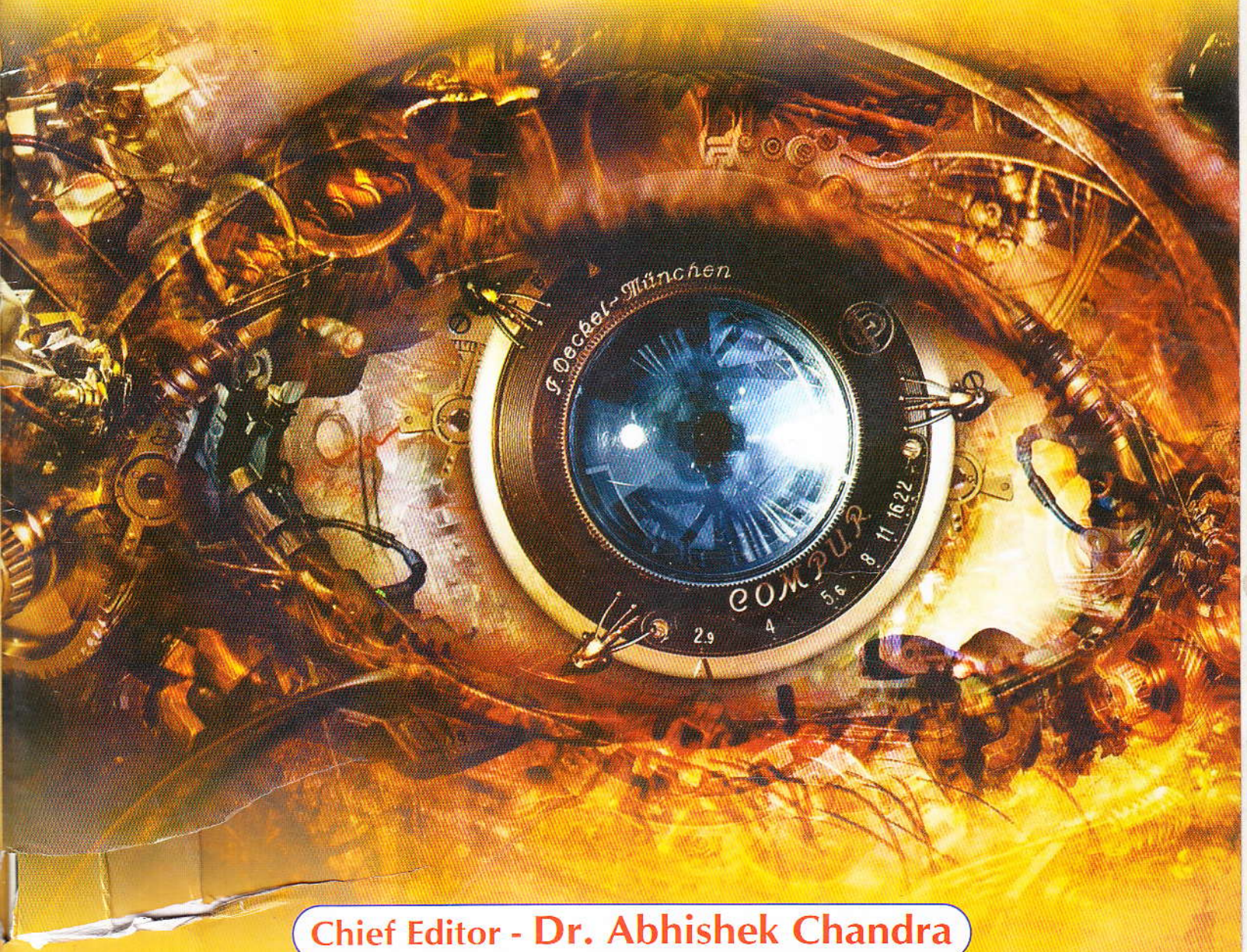
Vol.2, 2018

ISSN : 2250-1916

UP JOURNAL OF OPHTHALMOLOGY



Chief Editor - Dr. Abhishek Chandra

The Scientific Journal of U.P. Ophthalmological Society

U.P. JOURNAL OF OPHTHALMOLOGY

Editorial Board

Chief Editor

Dr. Abhishek Chandra

MS, DNB, MNAMS

Ex-Fellow, Univ. of Wisconsin, USA

Director, Chandra Eye Care

A-1, S.K.Gupta Colony, Lanka, Varanasi

Mobile: 7052525353

Email: upjoeditor@gmail.com

Web: chandraeyecare.com

Contents

From office of the President UPSOS ...

Dear Esteemed Member,

It has been wonderful to hold this office, for which you entrusted me 3 years back. I tried my best to nourish the society with whatever I have in my basket. Many thing I could do but a lot is left which I am sure incoming President will do. We implemented long awaited amended bye laws and renewal of Society Registration. Also started Zonal Meetings, in that sequence, we conduct one Zonal meeting on 13th May at Moradabad.

I think, start is a bit difficult but I am sure in this subsequent year all five Zonal meetings will be held.

We could adhere to our Society's year calendar which was made within 6 weeks after Jim Corbett annual meeting with Mid term meet at Sitapur, Zonal Meet, two Executive meetings at Sitapur and Kanpur respectively with Annual meet at Kanpur.

Tentative committees for Legal, Member's Family Welfare and Industry coordination has been planned and if it is given green signal, they may be taken up.

Dr. Abhishek Chandra has laid down the foundation for UPJO for getting it indexed. We the members of our esteemed Society will expect that now this Journal will be index in forthcoming tenure.

I personally and on Behalf of my family, I thank and will be indebted to whole executive and every esteemed individual member for belief in me and offer chance to serve this prestigious body.

Thanking you,

(RC Gupta)

President UPSOS

From the Editor's Desk...

Dear Friends,

I am indeed happy to bring forth the 2nd edition of UP Journal of Ophthalmology for the year 2018. This would be my last edition as Editor, UPJO.

Friends, when I began, my first aim was to make the publication of the Journal regular and send hard copy of the journal to all the members of the society. I wish to share my happiness with you that I could achieve this aim without asking for any funding from the UPSOS.

Being given the responsibility of Editor of the Journal was particularly challenging. The main challenge was to enhance the quality of publication and involve and motivate the members of UPSOS, especially the residents of the Medical Colleges, to contribute to the Journal. I have been trying to include articles covering a vast spectrum of issues, along with ensuring originality of research published. However, we have a long way to go...

I also express my thanks to the entire editorial team who helped me during the last three years. I would also like to thank various pharma companies especially Pharamatak and Optho Remedies, without whose support the regular publication of the Journal would not have been possible.

I also take this opportunity to congratulate Dr. Shalini Mohan for becoming the unopposed Editor of UP Journal of Ophthalmology for the next term and I am sure that UPJO would keep growing under her guidance.

I thank all the members of UPSOS for having posed their faith in me, and being very supportive all through my term. I hope that the Journal will continue to grow with respect to quality of research publication, contributions, as well as spread in terms of readership

(Abhishek Chandra)

Editor, UP Journal of Ophthalmology
Director, Chandra Eye Care
Varanasi

Emerging Ophthalmic trends that would affect the future around us

Dr. Malay Chaturvedi, Dr. Saurabh Gupta, Dr.Abhishek Dixit, Dr. Abhijaat Chaturvedi

Interesting developments are happening in the field of ophthalmology. We would like to know about them as very soon they would be floating in and around us-

- Stairway study data out comes: In a phase 2 trial, Faricimab has been compared with Q4 dosing of Ranibizumab. BCVA outcomes of patient treated with Q16 and Q12 week showed that Faricimab was comparable to Q4 week Ranibizumab. Similarly the outcomes were comparable in terms of CST and anatomical reduction in CNV size. They are no new safety signals identified for the disease. Outcomes of this phase 2 trial would open doors to global phase 3 trial of Faricimab for neovascular CNVM that would start next year.
- Iris registry analysis unveils important risk factors for blindness in Diabetic retinopathy: The blindness criteria identified was 20/200 or worse, on two consecutive visits more than 3 months apart or no improvement beyond 20/100 after the first reading of 20/200 or worse-

  Development of vitreous hemorrhage (hazard ratio 9.4 was the most significant risk factor. The other risk factors being development of glaucoma, Age related macular degeneration, RVO, diabetic macular edema and PDR.

  In glaucoma trends are fading away for the drops. The new devices that are likely to strike the market are:

- Bimatoprost implant erodible pallet injection for anterior chamber,
- A titanium device containing travoprost to be implanted in trabecular meshwork
- Travoprost XR- ENV 515 errodable implant.

  Other external devices likely to hit the market would include puntal plug containing a slow release prostaglandin analogue, and a Bimatoprost ring to be put in conjunctival cul de' sac.

- Topical treatment for retinal diseases: In an interesting contrast to glaucoma management where people are trending away from topical medication, retinal topical medication is being looked after as a viable alternative to intraocular implants. Such medication are using nano technology and are based on increase intra ocular permeability thus providing adequate drug concentration on site. Although still far away proponents of topical retinal medication are enthusiastic and would advocate this as patient friendly measure with ease of self medication.
- In an interesting presentation on removal of a subluxated Synchrony Accommodating IOL at least two of the panelist advocated use of anterior chamber implant. The other options included an iris claw IOL, a trans scleral fixation of PMMA IOL or a single piece foldable IOL.

DSEK & DMEK: Present and Future of Endothelial Keratoplasty

- Abhishek Chandra*, Kshama Dwiwedi**, Diksha Sareen*, Govind V. Khalkho*

In this decade with the advent of DALK and DSEK, penetrating keratoplasty is being replaced more and more with Lamellar keratoplasty as only the diseased part of the cornea is replaced giving better results with low complication rates.

Why there is need for lamellar keratoplasty

- PKP induces astigmatism in range of 3 to 7 diopters
- Decline in endothelial cell count leading to graft failure
- Allograft rejection and endothelial decompensation were the major concerns
- Postoperative discomforts and wound healing time more
- Wound strength in lamellar graft superior
- Non penetrating surgery, it reduces the risk of intraocular complications like glaucoma, cataract, CME, RD, endophthalmitis

Endothelial keratoplasty (EK) represents the selective replacement of dysfunctional endothelium with healthy donor endothelium and is the preferred treatment for any cornea which has diseased endothelium and relatively normal overlying corneal tissue. EK has evolved over the past 10 years from very difficult, time consuming procedure of Deep Lamellar Endothelial Keratoplasty (DLEK) to its current form of DSEK and DMEK.

It includes:

- Descemet's stripping endothelial keratoplasty(DSEK)
- Descemet's stripping automated endothelial keratoplasty(DSAEK)
- Descemet's membrane endothelial keratoplasty(DMEK)

History:

- Dr. Gerrit Melles described a posterior lamellar keratoplasty (PLK) years later in 1998, where only a select portion of the cornea was transplanted.
- PLK was originally performed by creating a deep stromal pocket originating at the limbus with a 9 millimeter (mm) incision and then manually dissecting out posterior stroma, DM, and endothelium using a specialized trephine or scissors
- Dr. Melles later revised the technique by folding the graft, which enabled the incision size to decrease to 5 mm.
- Dr. Mark Terry subsequently adopted the technique in 2001, adding modifications including the use of viscoelastic to stabilize the anterior chamber, and renamed the procedure as deep lamellar endothelial keratoplasty (DLEK).
- Dr. Melles then described a procedure where the DM and endothelium were stripped from the host cornea (descemetorhexis) and replaced with a donor button consisting of posterior stroma, DM, and endothelium.

*Chandra Eye Care, Lanka, Varanasi
**

- Dr. Francis Price was the first to publish clinical results of the technique, which he named Descemet's stripping endothelial keratoplasty (DSEK).
- Dr. Mark Gorovoy reported the use of a microkeratome for donor dissection in 2006 and coined the term Descemet stripping automated endothelial keratoplasty (DSAEK).
- In 2006, Dr. Melles developed another technique that he named Descemet's membrane endothelial keratoplasty (DMEK). DMEK involves only donor DM and endothelium being transplanted, in contrast to posterior stroma, DM, and endothelium used in DSAEK
- A modification of DMEK was described in 2009 where a rim of stroma was left at the periphery of the donor tissue.
- This was named Descemet's membrane automated endothelial keratoplasty (DMAEK)
- Another technique was described in 2010, called Descemet's membrane endothelial keratoplasty with a stromal rim (DMEK-S).
- The difference between DMEK-S and DMAEK is that DMEK-S donor tissue is prepared manually while DMAEK utilizes a microkeratome or femtosecond laser for the initial posterior lamellar dissection.
- In Descemet's stripping endothelial keratoplasty (DSEK), the patient's Descemet membrane is peeled off, using specially designed strippers and replaced with a partial thickness graft: a transplanted disc of Posterior Stroma, Descemet and Endothelium (20-30 % of the inner donor cornea)

Indications:

- Acquired Pseudophakic or Aphakic bullous keratopathy,
- Failed previous graft.
- Inherited Fuch's Endothelial Dystrophy and Iridocorneal Endothelial Syndrome.

Indian Scenario

Total no. of eyes being operated in India annually approximately 1 crore

Incidence of pseudophakic bullous keratopathy approximately 0.5%

This implies that 50,000 patients would require EK

Ideal time to perform DSEK :

- There is a significant relationship between Cataract Extraction to DSEK time and Best Spectacle Corrected Visual Acuity.
- Performing earlier (<6 M) DSEK for pseudophakic corneal edema appears to be associated with improved visual outcomes.

Surgical Technique

- This procedure, which takes approximately 45 min, is done under local or general anesthesia.
- First the endothelium and Descemet's membrane of the cornea is stripped away through a corneal incision.
- Then a circular disc is removed from the inner lining of a donor cornea.
- This thin layer is then transplanted into the recipient eye and attached to the posterior cornea of the recipient.

Donor tissue preparation:

- Corneoscleral buttons are excised from donor globes and stored by organ culture.
- Each globe is mounted on a purpose-designed holder and the anterior chamber is filled with air to create an air-endothelium interface
- With dissection spatulas, a manual stromal dissection is made at approximately 95% stromal depth using air-to-endothelium reflex to monitor dissection depth.
- Stromal dissection is extended up to limbus over 360 degrees
- After dissection is completed, a 16.0 mm corneoscleral rim is excised from each globe and the endothelium is evaluated with an inverted light microscope and stored in organ culture until time of transplantation.

Surgical technique:

- With a reverse Sinskey hook, a circular portion of Descemet membrane is scored and stripped from the posterior stroma so a descemetorrhexis is created and the central portion of Descemet membrane is removed from the eye.
- A temporal self-scaling 5.0 mm sclerocorneal incision is created with a crescent knife. After trephinating an 8.5 or 9.0 mm diameter DSEK-graft from the predissected corneoslceral rim, the tissue is folded over 60/40, like a taco, and stained with trypan blue.
- A plastic glide is carefully inserted through the temporal incision.
- Then, the graft is inserted into the anterior chamber of the recipient by sliding over the plastic glide using a 30-gauge bent needle.
- The glide is removed, and the DSEK graft is unfolded in the recipient anterior chamber with balanced salt solution and an air bubble and positioned against the posterior stroma of the host.
- The graft is unfolded over the recipient peripheral iris, taking care of touch between stromal surface of the graft and the underlying structures to avoid endothelial damage intraoperatively.
- After the DSEK graft is unfolded, the anterior chamber is completely filled with air.
- Dilating drops are used to prevent any pupillary block from air bubble.
- Once the donor disc is in final position with no interface fluid the surgeon removes the air in the anterior chamber and replaces it with BSS to pressurize the eye.
- An air bubble of approximately 8 to 9 mm is usually left in place to help further stabilize the donor disc position over the first 24 hours postoperatively.
- The air bubble pushes the graft in place until it heals in an appropriate position, giving time for the pumping action of endothelium to help the donor tissue bind to its new host
- The structure of the cornea remains intact.

DSEK Procedure Advantages Over PK:

- Less Invasive, smaller surgical incisions
- No corneal-graft sutures
- Faster visual recovery

- Less risk of sight threatening complications and less induced astigmatism
- Post-surgery stronger eye (less prone to injury)
- Less risk of immune rejection of the transplanted corneal tissue
- Shorter post-operative care

DSEK itself:

- Increases overall donor tissue availability, using the posterior layer of the donor cornea in one patient and the anterior lamellar graft in another patient.
- Faster to learn. DMEK Surgical technique may require more training, technically more challenging.

DSEK Procedure Challenges Over DMEK:

- Suboptimal visual acuity.
- Optical irregularities due to stromal layers being transplanted in DSEK.
- Slow visual rehabilitation.
- Interface problems, folds in the donor disk from maladaptation to the recipient stroma, decentration of the donor disk, and excess donor corneal thickness.

Overcome Complications

- Primary graft failure: a primary graft failure rate of 5.7%. Endothelial pump function has an important role in graft adhesion. In many cases, graft fails to adhere because the surgeon was too aggressive in handling it and damaged endothelial cells.
- Graft Rejection: Rejection can develop months or years after the transplant. Patients can be asymptomatic. When patients develops redness, blurry vision and light sensitivity the rejection is severe. To prevent rejection patients should be under a close follow-up care and kept on a prophylactic tapering steroid eye drops regimen.
- Over expected cell-loss: Assessing endothelial cell density (ECD) after DSEK, it is expected a median cell loss of 32% in the perioperative period. After that the ECD declines at a linear rate of approximately 110 cells/mm2 per year between 6 months and 10 years. Gradual reduction in endothelial cell density over time can lead to loss of clarity and require repeating the procedure.

Follow up care

- Use the slit lamp: to ensure that the graft is fully attached and to look for signs of rejection (scattered keratic precipitates, edema or conjunctival hyperemia).
- Check IOP: monitor for steroid-induced pressure spikes.
- Check the refraction after first month.
- Check the central corneal thickness: a graft that is getting thicker over time may be failing and a graft that gets thicker suddenly signals rejection.
- Watch for detachments: Anterior segment OCT can assess for graft detachments. If the graft is detached, it has to be reattached by rebubbling the anterior chamber. Since the graft has been in aqueous fluid, it often works well after reattachment.

Descemet's membrane endothelial keratoplasty (DMEK)

- Descemet's membrane endothelial keratoplasty (DMEK) is a partial thickness cornea transplant where the host Descemet membrane (DM) and endothelium are replaced by donor DM and endothelium. The Indications are same as those for DSEK.

ADVANTAGES:

- Reduction of interface haze
- Less incidence of graft dislocation
- Shorter visual recovery as total corneal thickness remains same
- Larger donor surface provides more viable endothelial cells
- Less strong graft-host apposition at interface allows easier removal of failed/rejected donor lenticule
- No costly instruments for donor lenticule preparation

DISADVANTAGES:

- Difficult and more traumatic manipulation of DM
- Higher endothelial cell loss rates with current techniques

SURGICAL STEPS:

A. Preparing recipient eye:

- Perform retrobulbar injection. DMEK can also be done under general or topical anesthesia.
- A Honan balloon is placed to add ocular pressure for 10- 15 minutes to reduce posterior pressure.
- Adjust calipers to 3.5 mm and mark clear corneal limbus at 12-o' clock position for main wound. Mark with marking pen
- Create 2-3 additional markings for paracentesis sites (1-o' clock, 6-o' clock, 11-o' clock adjust to surgeon preference)
- Using 1 mm diamond keratome, create the paracentesis sites indicated by the markings
- Fill the chamber with Healon
- Use 8.0 mm trephine to mark the central surface of the cornea
- Use the marking pen to create multiple spots along trephination mark
- Insert reverse Sinskey hook via paracentesis and score Descemet's membrane along 8.0 mm marking
- Refill the anterior chamber with Healon to pressurize the eye
- A keratome is used to make a 3.5 mm incision at the corneal limbus along the marking
- Insert reverse Sinskey hook through paracentesis or main wound to continue stripping of Descemet's membrane
- Remove host Descemet's membrane through main wound
- Utilize the phacoemulsification irrigation/aspiration device to remove all the Healon from the anterior chamber
- Observe the size of the pupil. Ensure the pupil in as small as possible

- Stroke the iris surface or use miochol or miostat in order to constrict larger pupils
- The IOP is left normal or slightly soft by using BSS injections

Preparing, injecting and positioning donor endothelium-Descemet's membrane

- Using tying forceps, remove prepared DMEK tissue from viewing chamber and use swab spears to remove excess fluid from scleral rim. Use caution to ensure graft does not displace from stroma. If graft displaces, use BSS and swab spears to encourage replacement.
- Place corneoscleral rim in empty shallow container
- Stain with VisionBlue by applying enough dye to cover the surface of the endothelium for 60 seconds
- Remove stain and gently rinse with BSS
- Ensure that the endothelium-Descemet's membrane is lying flat on the posterior stroma • If tissue is not laying flat, refloat with BSS and use spear sponges to draw the tissue toward the edges.Be careful to avoid touching the endothelium
- Mount and center tissue on vacuum block endothelial side up
- Apply suction by depressing syringe attached to vacuum block to secure tissue in place
- Obtain and slowly lower trephine punch onto vacuum block until trephine is resting on endothelium
- Gently apply pressure and tapping to cut donor Descemet's membrane and minimal stroma 360° around the edge of the graft. Do not perform complete trephination
- Optional: If S stamp is not used, obtain 1.0mm trephine and punch three holes along peripheral edge of graft in a manner that will allow distinguishing between the endothelial and epithelial views. If possible, position these marks at locations of larger tags or tears
- Use tying forceps to remove peripheral Descemet membrane and place in shallow container filled with BSS (For practice loading and unloading modified Jones tube).
- Be careful not to remove peripheral tissue too quickly as some of the graft may not have been cut. If areas are still attached, use diamond knife to hand cut or repeat trephine
- Apply BSS on top of graft to submerge endothelium
- Use tying forceps to gently lift the edge of the graft 180° from the marked hinge
- Slowly peel graft back toward hinge and lift out of BSS
- While holding tissue with forceps, fill corneoscleral button with VisionBlue
- Lower graft into stain and apply further stain on top to completely submerge tissue
- Allow staining for 3 minutes
- During this time, construct the insertion device
- Obtain 14 French gastric tubing and cut 1.5-2.0cm section with drape scissors
- Soak the inside with BSS
- Connect one end of tubing into Luer lock of 3cc syringe
- Attach the other end of tubing to the proximal tip of modified Jones tube

- Draw BSS into syringe via Jones tube and withdraw to ensure tight junctions
- Retain enough BSS to fill Jones tube
- Test the injection device by drawing peripheral segments of the graft set aside earlier. Practice loading and unloading into BSS to appreciate the amount of pressure required in doing so. Avoid aspirating air during this process
- Return attention to donor tissue submerged in VisionBlue
- Use spear sponges to remove VisionBlue. Use caution to prevent touching tissue
- Gently apply BSS onto corneoscleral rim to dilute VisionBlue and remove with spear sponges
- Repeat until blue graft is floating in almost clear solution
- Use tying forceps to carefully transfer corneoscleral rim to shallow chamber filled with BSS and float graft off of corneoscleral button and into the shallow dish
- Use forceps to remove corneoscleral rim
- Obtain assembled injecting device
- Submerge tip of Jones tube into BSS containing donor graft and situate bevel next to the end of the EDM
- Gently aspirate tissue into Jones tube keeping in mind the amount of pressure needed as tested prior
- Check orientation of EDM by observing the direction of curling edges. Edges should be curling upward
- Insert tip of modified Jones tube into main wound of recipient while maintaining correct orientation of graft. Tip of Jones tube should end on top of pupil
- Again, check orientation of graft ensuring that scrolls are facing upward. Rotate injector as necessary
- Slowly depress syringe plunger to inject graft into anterior chamber while removing injector even more slowly. Inject extra bursts of BSS to help orient the graft perpendicular to main wound and prevent efflux.
- Be careful that the graft does not eject from wound. Prevent this by allowing fluid to drain from main wound or paracentesis or use a cannula to close the main wound while withdrawing injector.
- Use 10-0 nylon to place one interrupted suture closing the main wound
- Again, check orientation of graft ensuring that the scrolls are facing upward while the graft is floating in the anterior chamber
- To manipulate the graft in the anterior chamber, utilize bursts of BSS if necessary to flip graft into correct orientation, center the EDM, or open a tightly scrolled Descemet roll
- Use the cannula to perform short swift taps to the external cornea to help center the graft and open the scroll
- Manipulating the graft is facilitated by obtaining a shallow anterior chamber. This can be done by using the index finger on the non-dominant hand and applying pressure about 5mm from the limbus
- After centering and fully unrolling the graft, introduce tip of cannula attached to 20% SF6 into the anterior chamber, posterior to the graft taking care never to touch the endothelium
- Once the tip is above the pupil, slowly inject gas to allow apposition of the graft to the posterior stroma allowing the edges to unfold and the center to touch stroma

- Fill the anterior chamber with gas
- Observe the entire margin of the graft evaluating for any folds and detachments
- Manipulation of the bubble, or bubble bumping, can help reduce folds and detachments
- Once all the edges are checked, perform sweep of entire surface of cornea with a barraquer spatula

Postoperative Management

- One sample medication regimen: Prednisolone acetate 1% should be used every two hours while awake for the first week, 4 times daily over the next 3 months, then slowly tapered and stopped at year
- Antibiotic drops should be used for 1 week after surgery.

Complications

- Graft detachment
- Damage to tissue during preparation or surgery
- Upside down grafts
- Epithelial defect or erosion (3.0%)
- Raised intraocular pressure (IOP) in as high as 12% of patients, with ~ 2.8% developing secondary glaucoma
- Descemet graft folds (1.9%)
- <1% risk of anterior synechiae, hypotony, pupillary block, subepithelial haze, and interface pigment deposits.
- Cystoid macular edema (CME): one study reported a high rate of CME of 12.5% in eyes with DMEK alone and 13.3% of eyes with DMEK and cataract extraction.

Surgical Outcomes:

- Visual acuity at 3 months: 63% with vision ≥ 20/25 and 26% ≥ 20/20.
- Visual acuity at 6 months: 79–94% with BCVA ≥ 20/40 and 22–47% ≥ 20/20.
- Multiple studies have reported that DMEK causes a mild hyperopic shift of < +0.50 D after 6–12 months follow-up. DSEK has been reported to have a hyperopic shift of around +1.00, due to the shape of the donor tissue.
- Postoperative refraction stabilizes at 3 months with no significant spherical equivalent change between 3 and 6 months postoperatively.
- Endothelial cell loss estimates following DMEK vary widely, from 32-40% at 3 months to 36-40% at 6 months.

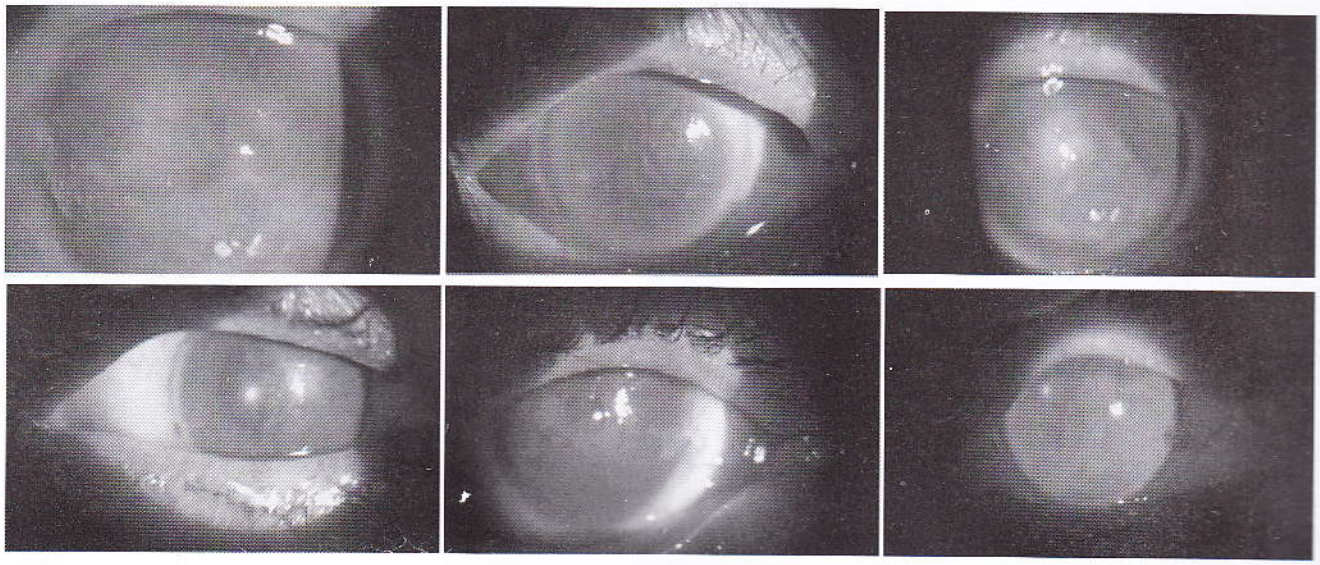
Our Experience with DSEK/DMEK

We have been doing DSEK regularly since 2008 in the Dept. of Ophthalmology, IMS, BHU and DMEK since 2016 at Chandra Eye Care, Lanka, Varanasi. The results of the Surgery are extremely gratifying with more than 90% success rate and more than 50% patients achieving BCVA of more than 20/30.

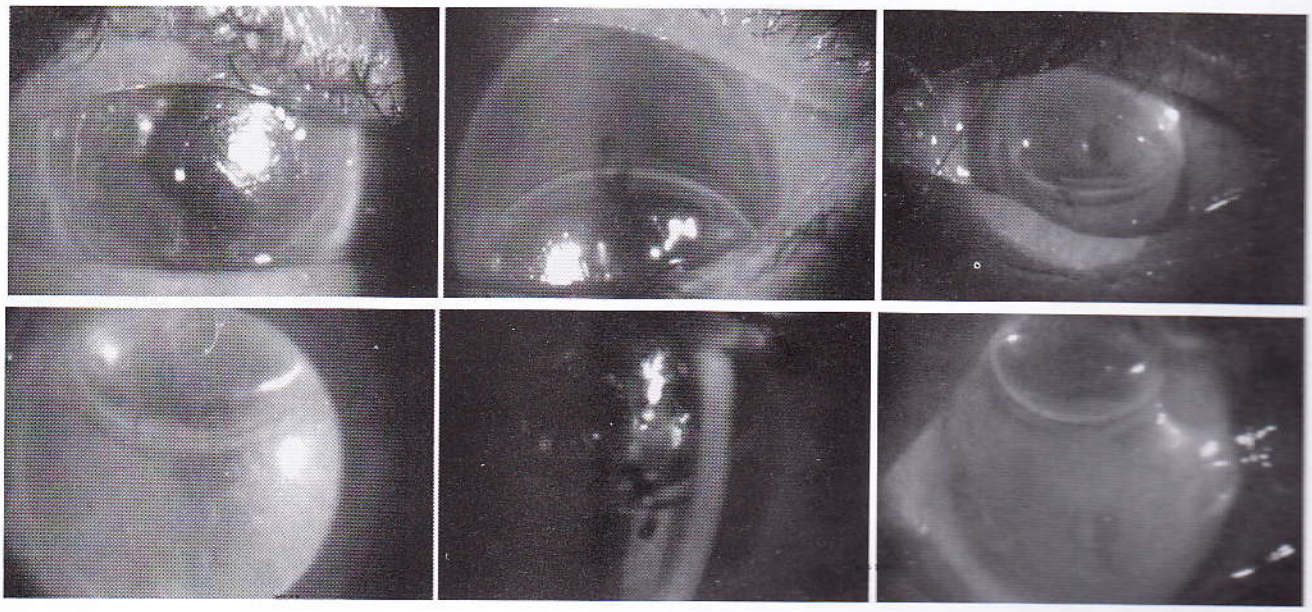
With DMEK the results are even better with 80% patients achieving BCVA of more than 20/30. Presently we are doing 6-8 cases of DMEK every month. Now we have almost completely shifted to DMEK from DSEK. However there are few indications where we still do DSEK. These are Aphakia, Vitreous in Anterior Chamber, Vitrectomised Eye, Large Peripheral Iridectomy in an operated eye.

The Donor criteria with DMEK is very stringent. The Donor age typically should be between 50 to 70 with endothelial cell count of more than 2700 per mm2. Now with the help of Sight Life and other Community Eye Bank it is relatively easier to get such tissues.

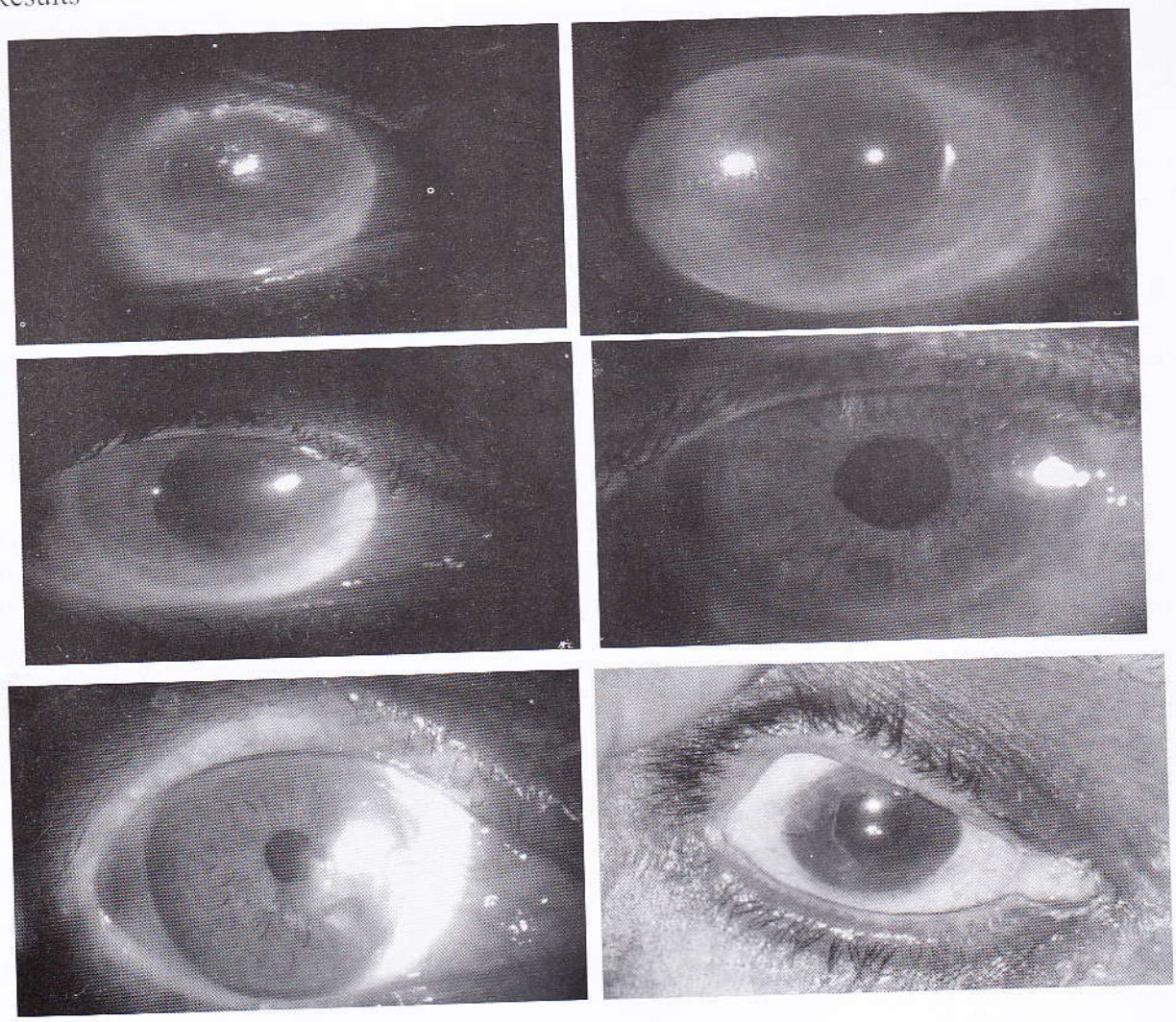
Pre-operative Phtographs



Stamped Corneas as seen post-operativel



Final Results



Conclusion:

DMEK is now a preferred technique in corneal endothelial dysfunction. The advantages of DMEK outweighs penetrating keratoplasty by early visual rehabitilation, low astigmatism, better ocular surface, absence of suture related complications, low incidence of graft rejections and minimal risk of wound rupture. The visual recovery time has now come down to 1 month in DMEK which was almost one year in Penetrating Keratoplasty.

NON NECROTOZING RETINITIS FOLLOWING VIRAL FEVER: A CASE SERIES

Dr. Shailendra Verma

Viral retinitis is considered as an important sight-threatening infectious disease of the retina which can occur in both immunocompetent and immunocompromized individuals.[1] Viral retinopathies can be necrotizing/non-necrotizing. Viral retinopathies include acute retinal necrosis syndrome, progressive outer retinal necrosis syndrome & CMV retinitis. Non-necrotizing retinopathies/NNHR has been reported after Chikungunya[2] & herpes infection[3,4]. Steroids and antivirals can be used for treatment of Viral retinitis. But role of Antiviral is uncertain[2]. In an effort to treat cases of non-necrotizing retinitis with suspected viral etiology, we tried the use of Steroids with or without antivirals & assess the improvement in terms of resolution of retinitis & visual recovery.

Aim

To report 6 cases of suspected Viral Retinitis presented at a Tertiary eye care centre.

Study Design

New cases & records of Retinitis cases treated at the Retina Clinic were reviewed. 10 eyes of 6 patients of non necrotizing Retinitis with the history of suspected viral fever were studied. All patients were either treated for viral fever or gave history suggestive of viral fever. History of occupation, complaints, any trauma/ infection, any treatment, any systemic disorder were noted from records. CBC, serum creatinine and blood urea, Mantoux test, chest x-ray and VDRL tests were done & findings are recorded along with detailed slit lamp examination & indirect ophthalmoscopy. Findings of Fundus Fluorescein Angiography and Optical Coherence Tomography were also recorded. Diagnosis of Viral Retinitis was made on the basis of history & clinical features. 2 cases were given Acyclovir along with Steroid & other cases were received only Steroid in tapering doses.

Results

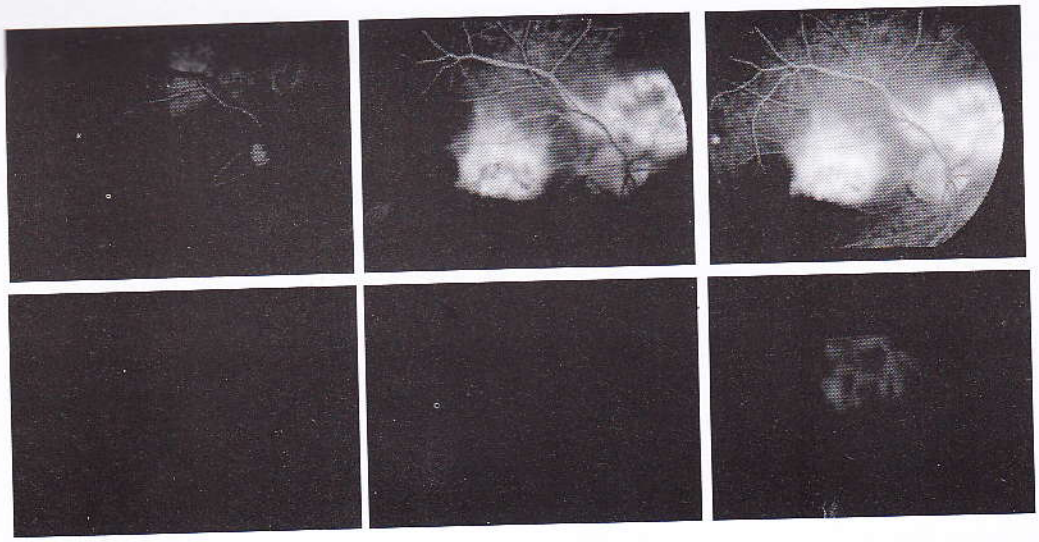
- Females patients: 2
- Male patients: 4
- Age range of patients: 14 - 45 years
- Retinitis: Bilateral in 4 cases (case 2,3,5,6) & Unilateral in 2 cases (case 1,4).

Fundus examination – On fundus exam following main features were found-

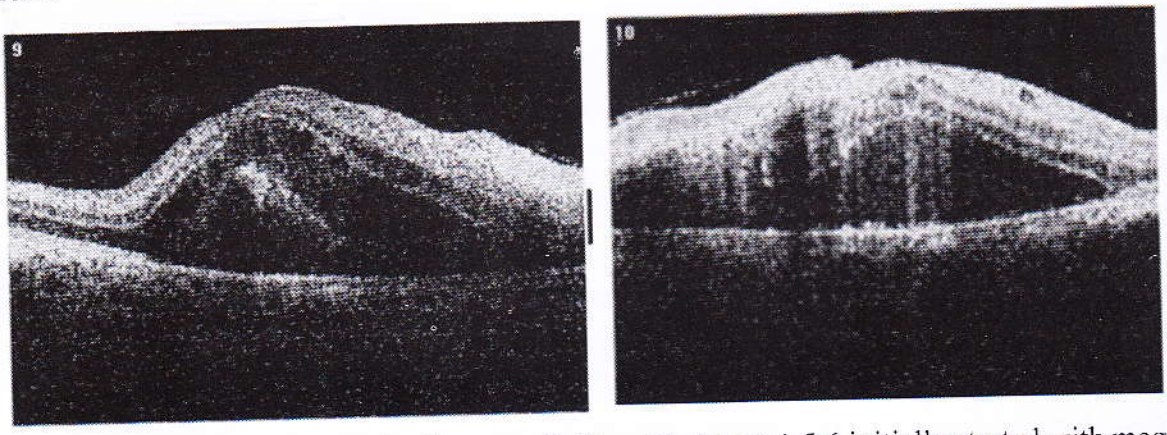
- AC cells, Vitritis
- Area of confluent retinal opacity suggestive of retinitis
- Hard Exudates (H. Ex), Cotton Wool Spots (CWS)
- Hemorrhages
- Exudative mac. detachment (EMD)
- Macular Oedema

FFA - early hypofluorescence and late hyperfluorescence seen.

Consultant - Vitreo Retina, ROP,& Uveitis, Contacare Eye Hospital, Kanpur.



- ***OCT*** - Increased reflectivity in the nerve fiber layer zone corresponding to the areas of retinitis with aftershadowing was observed.
- Fluid-filled spaces in the outer retina (case 3,4,5) with subfoveal serous detachment (case 1,2,3,4,5) also present.



- Four out of six patients- received only systemic Steroids (cases 1,5,6 initially started with megadose). Two cases (case 2,3) - received a combination of systemic Acyclovir and oral Steroids. All patients had improvement in visual acuity as well as resolution of retinitis with treatment. Two Pts are still under F/U with improvement.

Results – Case 1 -

Age - 45y,F

Duration – 1 mth

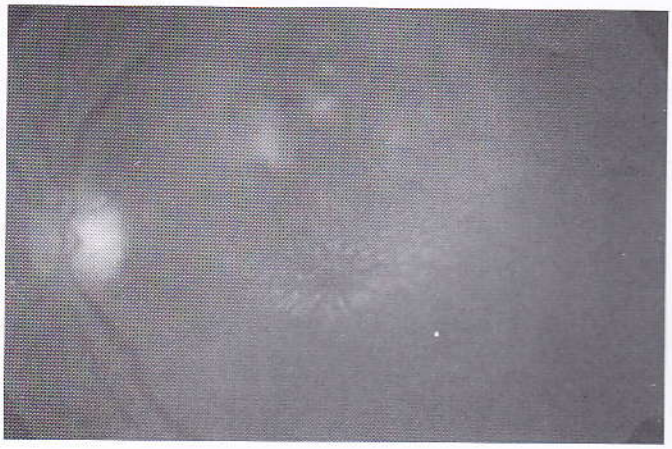
Involved eye - LE

Initial VA – 2 FFC

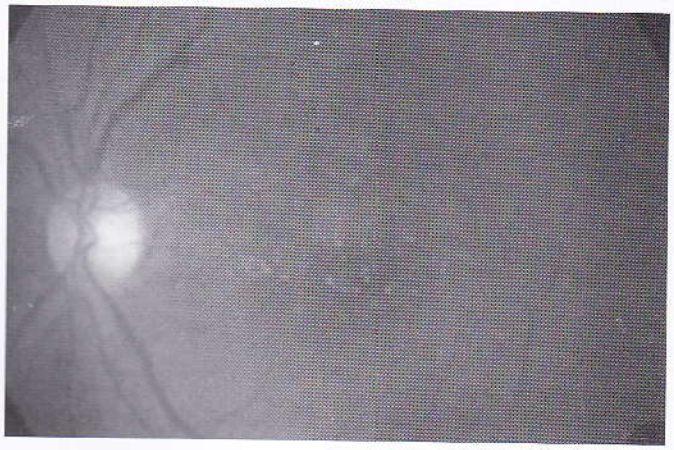
Final VA – 6/6 P

Reaction – Vit. +

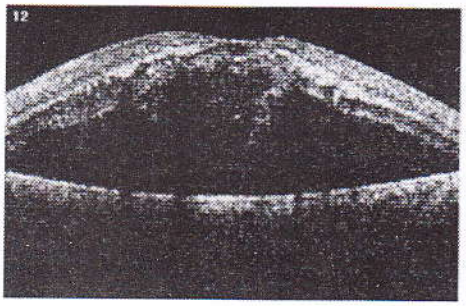
H/O fever

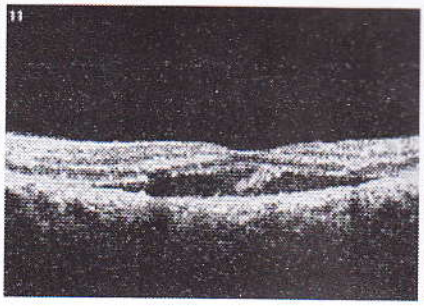


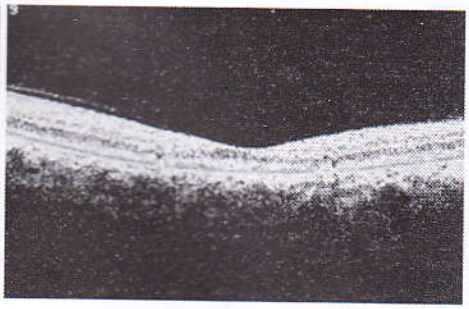
Initial – Pre Tt



After 1& ½ mth – Post Tt







Initial – Pre Tt

After 2 mths

After 2 & 1/2 mths– Post Tt

Results – Case 5 -

Age - 14 y, M

Duration – 17 days

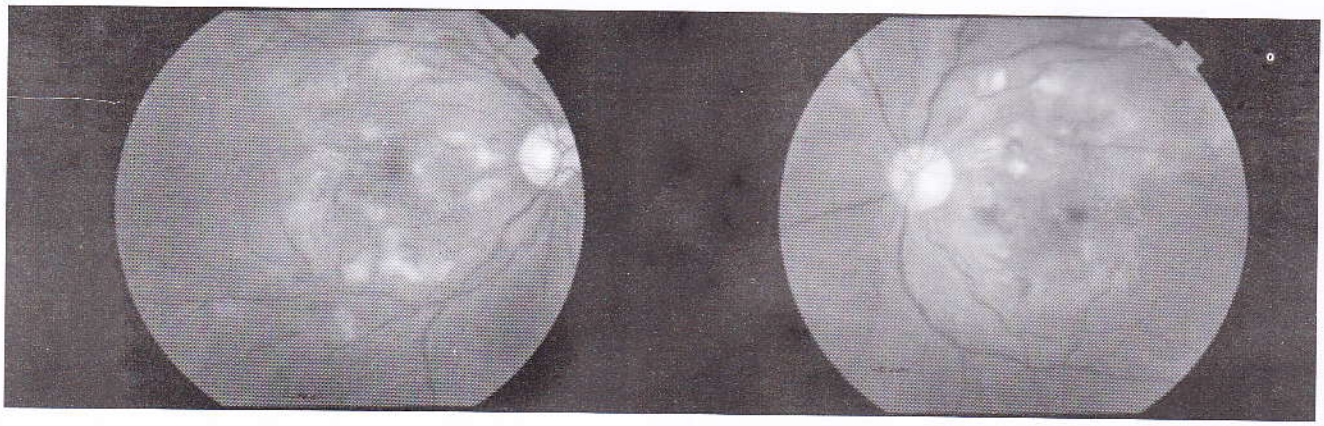
Involved eye - BE

Initial VA – 2 FFC,2 FFC

Final VA – 6/24, 6/12

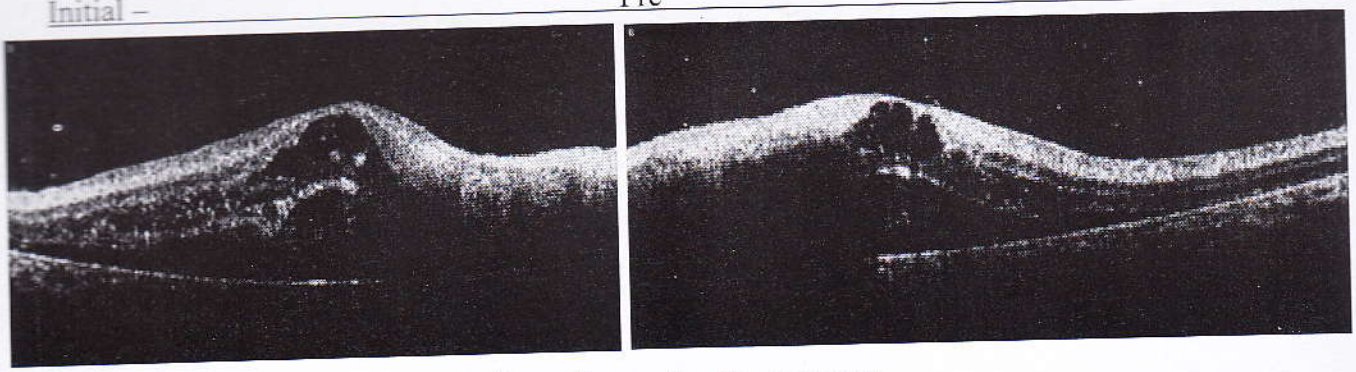
H/O fever

Initial – Pre Tt



After 1 & ½ mth - Post Tt

Initial – Pre Tt

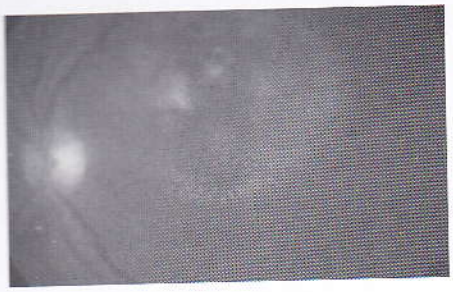


Case Report – Summary

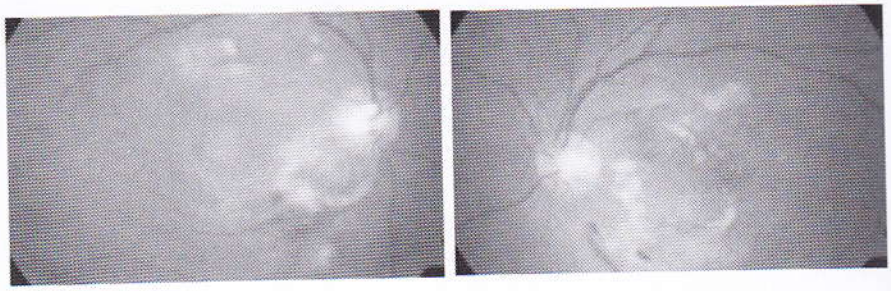
Case	Age(Yr)/ Sex	Compl aints	History & Systemi c Sympto ms	Inte rval betw een Feve r and Eye Sym pto ms	Vn RE	Vn LE	Ocula r Findi ngs	Clin ical Dia gno sis	Tt & Follow-up	Fina l Vn RE	Fina l Vn LE	Final Result
1	45y, F	DOV LE 2 wk,	Fever and skin rash 1& 1/2mth back, Tt for viral fever	1 mth	6/6	2 FF C	Cells occ, Vit+, CWS, H Ex, Retini tis,M E, EMD	LE Vira l Reti nitis	**IV Dexa** 100 mg in 150 ml of 5% dextrose x 3 d(Mega dose),F/B oral steroids (tapering dose over 3 mth),1 wk F/U	6/6	6/6 P	Resolve d
2	45y, M	DOV BE 25 days	Fever 3 mth back,jt pain. Tt for viral fever	2 mth 5 days	5/60	4/6 0	C+,Vi t+,C WS, H Ex, Hgs, Retini tis,E MD	BE Vira l Reti nitis	**T. Acyclovir** 800 mg 5t/d x 1wk then tapering doses, oral steroids (tapering dose over 2 mth), 1-2 wk F/U	6/6	6/9	Resolve d

3	38y, M	DOV BE 17days	Fever 1 mth & 25 days Back, jt pain, skin lesions	1 mth & 8 days	4/60	1/60	Vit+, CWS, H Ex,Retinitis, EMD, ME	BE Viral Retinitis	**T. Acyclovir** 800 mg 5t/d x 1wk then tapering doses, oral steroids (tapering dose over 2 mth), 1 -2 wk F/U	6/6 P	6/12	Resolved
4	45y, M	DOV RE 1 mth	Fever 2 mth Back, skin rash, jt pain	1 mth	1/60	6/6	C1/2+, CWS, H Ex,Retinitis, EMD, ME ,Hgs	RE Viral Retinitis	oral steroids (tapering dose over 2 mths), 1 wk F/U	6/60 after 1 wk	6/6	Resolving
5	14 y, M	DOV **BE** 9 days	Fever 26 days Back,skin lesions	17 days	2 FFC	2 FFC	Vitritis 2+, CWsS, Hgs, HEx, Retinitis, PSC	BE Viral Retinitis	**IV Dexa** 100 mg in 150 ml of 5% dextrose x 3 d,F/B oral steroids (tapering dose over 3 mth),1-3 wk F/U	6/24 (RPE changes)	6/12	Resolved
6	25y, F	DOV **BE** 17 days	Fever 1& 1/2mth back, Tt for viral fever	28 days	6/36	2 FFC	Vitritis 1+, CWS, Hgs, Hex,	BE Viral Retinitis	**IV Dexa** 150 mg in 150 ml of 5% dextrose x 3 d,F/B oral steroids (tapering dose over 3 mth),1-3 wk F/U.	6/24 after 2 wk	4/60	Resolving

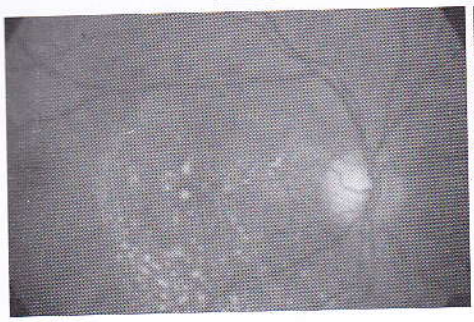
Case - 1



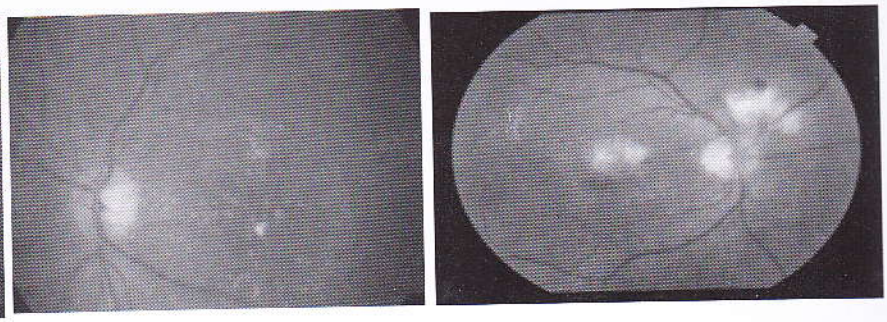
Case - 2



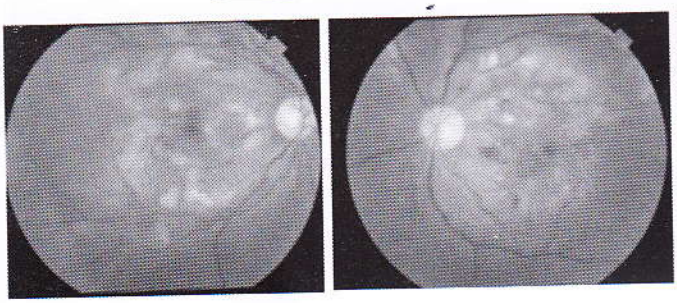
Case – 3



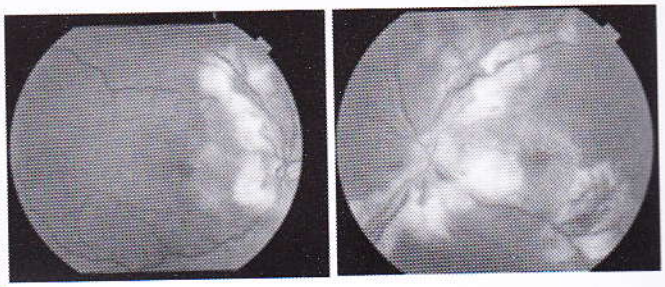
Case - 4



Case - 5



Case - 6



Discussion –

All patients had clinical features of Viral fever - with headache, nausea, vomiting, occasional rigors with chills, joint pains and maculopapular skin rashes in few cases. Patients presented at a range of 17 days-9 wks intervals after onset of fever. History, Associated systemic manifestations & ocular changes were favourable to make a diagnosis of viral fever associated retinitis in our cases. Retinitis responded well with both systemic acyclovir and corticosteroids with gradual resolution over 6 to 8 weeks period.

Not enough evidence in the literature to support the use & duration [3] of antiviral in non necrotizing viral retinitis cases, we used it in our 2 initial cases presented with posterior pole involvement in both eyes affecting vision. In a study by P Mahendradas et al [2] they reported similar kind of retinitis in chikungunya pts & treated successfully with systemic acyclovir and steroids.

Bodaghi B et al[3] also reported Nonnecrotizing retinopathies in herpetic patients & successfully treated with systemic acyclovir and steroids.

Although all cases showed resolution of retinitis with preservation of Vision, role of antiviral is still not clear, we feel that further studies are required to confirm the importance of antiviral in cases of non necrotizing viral retinitis.

Conclusion -

Visual recovery in this series of patients with suspected viral aetiology non necrotizing retinitis was good. The outcome did not differ whether patients were treated with or without Acyclovir. Nonnecrotizing retinitis is uncommon & relatively newer therefore Ophthalmologists need to be aware of these features.

References

1. Biswas, Rao NA. Diagnosis and management of ocular lesions in acquired immunodeficiency syndrome. Indian Ophthalmol 36:151-155,1988.
2. Padmamalini Mahendradas et al. Ocular Manifestations Associated with Chikungunya. Ophthalmology 2008;115:287–291
3. Bodaghi B, Rozenberg F, Cassoux N, Fardeau C, LeHoang P.Nonnecrotizing herpetic retinopathies masquerading as severe posterior uveitis.Ophthalmology. 2003 Sep; 110(9):1737-43.
4. Guex-Crosier Y, Rochat C, Herbort CP. Necrotizing herpetic retinopathies. A spectrum of herpes virus-induced diseases determined by the immune state of the host. Ocul Immunol Inflamm 1997;5:259–65.

CHILDHOOD BLINDNESS: CHALLENGES AHEAD

O.I. Haque, A. Waris

Childhood blindness is an important cause contributing to the burden of blindness globally. Worldwide, 19 million children are visually impaired, of whom 1.4 million are irreversibly blind and need visual rehabilitation interventions for a full psychological and personal development[1]. Visual impairment in childhood has implications in all areas of a child's development. It poses educational, occupational and social challenges, with affected children being at risk of behavioral, psychological and emotional difficulties, impaired self-esteem and poor social integration. The pattern of childhood blindness varies depending on the accessibility of affordable health care services as well as socio-cultural factors. A major cause of childhood blindness in one country can be insignificant in another, and even over a decade the causes of childhood blindness can change quite dramatically in the same country[2,3]. The variations in the major causes of blindness in pediatric age group from different parts of the world is determined by socioeconomic factors and the availability of primary eye care services. In developed countries, lesions of the retina, optic nerve and higher visual pathways predominate as the cause of blindness, whereas corneal scarring is the major cause in low-income countries. Retinopathy of prematurity is an important cause in middle-income countries.

Early work by Rahi et. Al. (1993) showed that retinal dystrophies and albinism are emerging causes of childhood blindness"[4]. Similarly, Dandona and Dandona (2003), observed that 50% of the blindness were due to causes that are currently not treatable or preventable, of which a major proportion was of congenital eye anomalies and retinal degeneration[5]. Likewise, Gogate et. al. (2007), found that congenital anomalies and retinal disorders together accounted for more than 50% of the cases of blindness, which was higher than in a similar study conducted 10 years ago[6]. Another study by Bhattacharjee et al. (2008) observed that retina and optic nerve are amongst the most affected anatomical sites of visual loss[7]. Again Ozturk et. al. (2016) in agreement to previous studies, observed that in severe visual impairment (SVI) or blindness, the most common anatomic site is retina[2]. Latest research by Prakash et. al. (2017), observed that optic nerve atrophy and retinal dystrophy are the emerging causes of blindness underlining the need for genetic counseling and low vision rehabilitation centers, along with a targeted approach for avoidable causes of blindness[8].

The prevalence of so-called 'non-treatable' causes of childhood blindness amongst all studies is found to be relatively increased due to the significant reduction in the frequency of preventable causes of visual impairment and blindness, underlying the need for trained professionals, newer diagnostic techniques and multi-disciplinary approach. The exact reason for the changing trend is difficult to ascertain, but increased health services might have a role to play. There might be regional differences in the trends depending on whether the study is conducted in a rural population in remote areas or an urban setup with good access to health care facilities.

While optical coherence tomography (OCT) is crucial for accurate diagnosis and detailed analysis of structural anomalies [9], Next-generation genetic sequencing are emerging as a vital tool for accurate diagnosis and patient-tailored therapy as mutations in approximately 250 genes have been linked to cause inherited retinal degenerations with a high degree of genetic heterogeneity. New techniques in next-generation sequencing are allowing the comprehensive analysis of all retinal disease genes thus changing the approach to the molecular diagnosis of inherited retinal dystrophies. These new sequencing tools are highly accurate with sensitivities of 97.9% and specificities of 100%.[10]

More recently, data suggest that the prevalence of functional low vision (corrected visual acuity in the better

Institute of Ophthalmology, JNMCH, AMU, Aligarh

eye ranging from <6/18 to, and including, light perception from untreatable causes) is approximately twice the prevalence of blindness: there are almost 3 million children worldwide who have the potential to benefit from low vision care. It is, therefore, essential that low vision services be part of eye care services for children at all levels of service delivery.[11]

Future Research

The U.S. Food and Drug Administration on December 19, 2017 approved Luxturna (voretigene neparvovec-rzyl), a new gene therapy, to treat children and adult patients with an inherited form of vision loss that may result in blindness. Luxturna is the first directly administered gene therapy approved in the U.S. that targets a disease caused by mutations in a specific gene. Luxturna is approved for the treatment of patients with confirmed balletic RPE65 mutation-associated retinal dystrophy that leads to vision loss and may cause complete blindness in certain patients.–[1214]

Retinal gene therapy clinical trials are underway for multiple genes including RPE65, ABCA4, CHM, RS1, MYO7A, CNGA3, CNGB3, ND4, and MERTK for which a molecular diagnosis may be beneficial for patients. Recent developments in genetic testing and gene therapy has now given new hopes for the diseases which were previously considered 'incurable'.

References

1. WHO | Visual impairment and blindness [Internet]. WHO [cited 2017 Sep 15];Available from: http://www.who.int/mediacentre/factsheets/fs282/en/
2. Ozturk T, Er D, Yaman A, Berk AT. Changing trends over the last decade in the aetiology of childhood blindness: a study from a tertiary referral centre. Br J Ophthalmol 2016;100(2):166–71.
3. Titiyal JS, Pal N, Murthy GVS, Gupta SK, Tandon R, Vajpayee RB, et al. Causes and temporal trends of blindness and severe visual impairment in children in schools for the blind in North India. Br J Ophthalmol 2003;87(8):941–5.
4. Rahi JS, Sripathi S, Gilbert CE, Foster A. Childhood blindness in India: Causes in 1318 blind school students in nine states. Eye 1995;9(5):545–50.
5. Dandona R, Dandona L. Childhood blindness in India: a population based perspective. Br J Ophthalmol 2003;87(3):263–5.
6. Gogate P, Deshpande M, Sudrik S, Taras S, Kishore H, Gilbert C. Changing pattern of childhood blindness in Maharashtra, India. Br J Ophthalmol 2007;91(1):8–12.
7. Bhattacharjee H, Das K, Borah RR, Guha K, Gogate P, Purukayastha S, et al. Causes of childhood blindness in the northeastern states of India. Indian J Ophthalmol 2008;56(6):495–9.
8. Prakash MV, Sivakumar S, Dayal A, Chitra A, Subramaniam S. Ocular morbidity patterns among children in schools for the blind in Chennai. Indian J Ophthalmol 2017;65(8):733.
9. Jeng-Miller KW, Cestari DM, Gaier ED. Congenital anomalies of the optic disc: insights from optical coherence tomography imaging. Curr Opin Ophthalmol 2017;
10. Recommendations for Genetic Testing of Inherited Eye Diseases - 2014 [Internet]. Am. Acad. Ophthalmol.2014 [cited 2018 Jan 12];Available from: https://www.aao.org/clinical-statement/recommendations-genetic-testing-of-inherited-eye-d
11. Pal N, Titiyal JS, Tandon R, Vajpayee RB, Gupta S, Murthy GVS. Need for optical and low vision services for children in schools for the blind in North India. Indian J Ophthalmol 2006;54(3):189–93.
12. HAFLER BP. CLINICAL PROGRESS IN INHERITED RETINAL DEGENERATIONS: GENE THERAPY CLINICAL TRIALS AND ADVANCES IN GENETIC SEQUENCING. Retina Phila Pa 2017;37(3):417–23.
13. Commissioner O of the. Press Announcements - FDA approves novel gene therapy to treat patients with a rare form of inherited vision loss [Internet]. [cited 2018 Jan 10];Available from: https://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm589467.htm
14. Sharif W, Sharif Z. Leber's congenital amaurosis and the role of gene therapy in congenital retinal disorders. Int J Ophthalmol 2017;10(3):480–4.

OCULAR BLOOD FLOW AND GLAUCOMA

Shalini Mohan, Rajnath Kushwaha, Komal Sah, Jayati, Ashok Kumar Verma, Surendra Singh Sachan; Mohit Khattri

Primary open angle glaucoma **(OAG)** is a multi-factorial optic neuropathy characterized by progressive retinal ganglion cell death and tissue remodeling of the optic nerve head (ONH). This is followed by visual-field defects corresponding to the damage of the neuroretinal rim (NRR). Prevalence of glaucoma is 0.7% in the 5th decade and it increases to 7.7% in subjects over 80 years of age.

Traditionally, diagnosis and treatment has been directed towards the control of increased intraocular pressure (IOP), which is the most important risk factor. However, all patients with glaucomatous ONH damage do not have elevated IOP, and glaucomatous neuropathy may progress even at IOP in the low teens, the so called Normal Tension Glaucoma (NTG). Some glaucoma patients continue to progress and subsequently develop irreversible loss of vision despite the medical lowering of IOP. For instance, in the early manifest glaucoma trial **(EMGT)** the disease progression rate in the treatment group was 45% as compared to 62% in the control arm. .

In the collaborative initial glaucoma treatment study **(CIGTS)** substantial visual field loss occurred in 10-13.5% of participants during 5 years of follow-up. Specifically, increased incidence of visual field deterioration occurred with older age (increased risk of VF loss by 40% every 10 years), race (nonwhites had a 50% increased risk relative to whites) and diabetes (59% increased risk relative to non-diabetic patients). Likewise, 20% of normal tension glaucoma (NTG) patients show continued visual field loss even after 5 years of IOP reduction treatment.

Therefore these facts doubt the pathophysiological concept of glaucoma based only on IOP, and compel us to contemplate to find other associated factors causing damage and subsequent progression of the disease.

Compromised ocular blood flow and deranged vascular autoregulation in the ONH is emerging as the most important factors in the various studies throughout the world. Mounting evidence suggests that glaucoma patients have reduced blood flow to the retina, choroid and optic nerve. Still it is not yet known whether the vascular component is consequent to increased IOP or the two risk factors are independently acting to affect the ONH damage in the long run. Some of the vascular risk factors which may be related to OAG pathogenesis include: aging, systemic blood pressure, nocturnal hypotension, ocular perfusion pressure, migraine, disk hemorrhage, diabetes and directly assessed reductions of ocular blood flow.

Pathophysiology of Various Factors that Influence the ONH Circulation

To understand the role of vascular insufficiency of the ONH in the pathogenesis of glaucoma, it is fundamental to understand the blood flow in the ONH in health and disease and the various factors that influence it.

To calculate the ONH blood flow, the following formula is used:

$$\text{Blood Flow} = \frac{\text{Perfusion pressure}}{\text{Vascular Resistance}}$$

*GSVM Medical College, Kanpur
*Regency Hospital

Perfusion pressure- When we deduct IOP from mean arterial blood pressure (MABP) then we get perfusion pressure.

- **Perfusion Pressure = MABP - IOP**
- **MABP = Diastolic BP + 1/3 (Systolic BP - Diastolic BP)**

Based on the above equation we can say that decrease in BP or increase in IOP reduces perfusion pressure.

The blood flow depends upon three parameters: (1) vascular resistance, (2) BP, and (3) IOP.

Vascular Resistance: It depends upon the state and caliber of the vessels feeding the ONH circulation and rheological properties of the blood which is influenced by a large variety of hematologic disorders, particularly those causing increased blood viscosity.

Arterial blood pressure: It is clear from the above equation that arterial blood pressure is major factor which can affect the perfusion pressure in the ONH. Nocturnal arterial hypotension is an important risk factor for the development of ONH ischemic disorders. Therefore it is important to record the night time BP as the daytime recording gives no information about the BP during sleep.

Intraocular pressure: There is an inverse relationship between IOP and perfusion pressure in the ONH. In persons with normal BP and autoregulation, a much greater rise in IOP would be required before the ONH blood flow is compromised. By contrast, in persons with arterial hypotension, defective autoregulation or other vascular risk factors, even "normal" IOP may interfere with the ONH blood flow (e.g., in normal tension glaucoma). This mechanism is important in the pathogenesis of glaucomatous optic neuropathy, particularly in normal tension glaucoma. A rise of IOP during sleep and concurrent development of nocturnal arterial hypotension may together constitute an important hidden risk factor for ONH ischemia in vulnerable subjects.

Evaluation of the ONH Circulation

The measurement of ocular perfusion is must as low perfusion pressure has been implicated as an important risk factor in the various studies. The measurement of the ocular circulation is done by OBF imaging techniques which are relatively complicated. These are transcranial Doppler, laser Doppler flowmetry, scanning laser Doppler flowmetry, magnetic resonance imaging, pulsatile ocular blood flow method (Figure 1), color Doppler ultrasound imaging (CDI) (Figure 2), fluorescein fundus angiography, confocal scanning laser fluorescein angiography, retinal photography oximetry and temperature measurement. These imaging techniques reveal the information which is not always valid scientifically and these methods have there own limitations. Another important aspect is that the measurements should be done throughout 24 hours so as to assess there variability.

We will describe two important techniques with which we are exposed to:

Pulsatile ocular blood flow:

This is the technique measured with Pneumotonometer (Figure 1) in which pulsatile waveform is created with each heart beat. Seventy percent of the total blood flow is measured by pneumotonometer. It is a well known fact that central retinal artery circulation accounts only 10% of the total ocular blood flow and rest of the circulation is formed by the posterior ciliary artery. Eighty five percent of the total ocular blood flow is made up of the ciliary circulation[24,25] and it is the main supply of the optic nerve head and therefore POBF has got importance in the glaucomatous damage. It utilizes the pressure-volume relationship as described by Langham and Toney, and Silver et al is relevant in investigating blood flow in choroidal circulation.

Color Doppler Imaging (CDI):

It detects changes in the frequency of sound reflected from flowing blood, allowing estimation of flow velocity. A simultaneous B-mode imaging and pulse wave Doppler facility is achieved with the help of high frequency probes of 7.5-10 mHz frequency. Colour doppler is applied on the B-scan image to identify the desired vessel which is next interrogated by placing a sample volume curser angled approx 60 to the flow, and a waveform is obtained. The vessels imaged are Ophthalmic artery (figure 3), Central retinal artery (figure 4), Short and long posterior ciliary arteries (figure 5).

Effect of Systemic Medications on Ocular Blood Flow

It has been found that certain drugs can affect OBF. These drugs are following:

1. **Antihypertensive medications:** It has been found that centrally acting calcium channel blockers nimodipine and lomerizine increase ocular blood flow whereas the peripherally acting like nifedipine do not cause. Other studies also suggest that angiotensin converting enzymes may lead to increase in the blood flow along with the decreasing IOP; although no study is done in the glaucoma patients.
2. **Estrogens:** It was found in Rotterdam eye study that the females on Hormone replacement therapy and post menopausal are at lesser risk of developing glaucoma. [32] These are also known to be neuroprotective.
3. **Nitric oxide:** It is a mediator of vasodilatory response to Bradykinin, histamine, acetylecholine, substance P, and insulin. Nitric oxide synthase reduces the choroidal and optic nerve head blood flow besides causing decrease in the choroidal blood flow while going into dark from light.
4. **Ginko biloba extract:** It has been showed as a neuroprotective for retinal ganglion cells in experimental studies. It has also been found to increase normal artery blood flow in normal subjects.

Topical Medications Affecting Ocular Blood Flow

Many existing medications are able to interact with vasculature, altering ocular blood flow, therefore, it is essential that current and future medications for glaucoma be evaluated for their effect on ocular circulation.

1. Effect of dorzolamide on augmentation of OBF in the ONH: Dorzolamide, a topical carbonic anhydrase inhibitor (CAI), was one of the first topical CAIs labeled by the FDA for the treatment of glaucoma. It decreases IOP by about 18% through a block of the carbonic anhydrase enzyme at the level of the ciliary body. In POAG patients timolol does not seem to alter the ocular hemodynamics, whereas dorzolamide increases the retinal microcirculation and ocular blood flow (OBF). Increased OBF with dorzolamide is possibly caused by a direct vasodilator effect of dorzolamide and not secondary to a decrease in IOP. The pre-systolic velocity of CRA in glaucomatous eyes and the end –diastolic velocity of the ophthalmic and central retinal artery significantly increases with dorzolamide. The minimal velocity of the central retinal vein showed significantly higher values after the instillation of dorzolamide.

2. Effect of Latanoprost: A study showed that Latanoprost increases ocular blood flow besides decreasing IOP in normal subjects.

3. Beta blockers (Timolol and Betaxolol) in various studies have found not to later the ocular blood flow. The long-term treatment with ophthalmic betaxolol improves ocular hemodynamics by lowering the resistivity index of the ophthalmic artery and results in an improvement in the visual fields of patients with

NTG. In view of this positive effect on blood flow and visual function, betaxolol is recommended in the management of patients with NTG.

4. Brimonidine does not alters the ocular hemodynamics.

5. Rho kinase Inhibitors: These are newer addition to medical management of glaucoma which increase the blood flow by relaxation of smooth muscles. These are still under trial phase.

References

1. Heijl A, Leske MC, Bengtsson B et al.; Early Manifest Glaucoma Trial Group. Reduction of intraocular pressure and glaucoma progression: results from the Early Manifest Glaucoma Trial. Arch Ophthalmol. 2002;120:1268-79

2. Lichter PR, Musch DC, Gillespie BW et al. Interin clinical outcome in the collaborative initial glaucoma treatment study comparing initial treatment randomized to medications or surgery. Ophthalmology 2001;108:1943-1953

3. Hayreh S. Blood supply of the optic nerve head and its role in optic atrophy, glaucoma and edema of the optic disc. Br J Ophthalmol 1969;53:721-48

4. Weinreb RN. Ocular blood flow in glaucoma. Can J Ophthalmol. 2008 ;43(3):281

5. Williamson TH, Harris A. Ocular blood flow measurement. *Br J Ophthalmol* . 1994;78:939-45

STEROID INDUCED GLAUCOMA

Reena Sharma, Asma Khan, Namita Sharma

Steroid induced glaucoma is a type of drug induced glaucoma which results from the use of steroids in any form.

Pathophysiology

The primary cause of IOP elevation in steroid induced glaucoma is increased outflow resistance. Increased responsiveness to steroids may be facilitated by upregulation of glucocorticoid receptors on trabecular meshwork cells.

1. Steroid causes stabilization of lysosomal membranes and accumulation of polymerized glycosaminoglycans (GAGs) in the trabecular meshwork, which become hydrated, producing "biologic edema" and increased the outflow resistance.
2. Steroids have been shown to increase the expression of extracellular matrix proteins like fibronectin, glycosamioglycans and elastin.
3. Steroids also suppress phagocyctic activity which may lead to increased deposition of material in the juxtacanalicular meshwork of eyes with steroid induced glaucoma.
4. Glucocorticoids alter the trabecular meshwork cell morphology by causing an increase in nuclear size and DNA content.
5. Blockage of intertrabecular spaces by white crystals after intravitreal triamcinolone injection(actual physical obstruction).
6. Patients with increased levels of endogenous corticosteroids (e.g., Cushing syndrome) can also develop increased IOP, which generally returns to normal when the corticosteroid-producing tumor or hyperplastic tissue is excised.[1]

Risk Factors

The risk of glaucoma with the use of steroids is dose and duration dependent. A higher than average risk for steroid glaucoma is found in patients with:

1. Known case of open angle glaucoma
2. Family history of glaucoma (first degree relative)
3. Very young age (age less than six years old) or an older age
4. Type 1 Diabetes Mellitus[4]
5. A history of previous steroid induced intraocular pressure (IOP) elevation
6. Connective tissue disease[5]
7. Penetrating keratoplasty, especially in eyes with Fuchs endothelial dystrophy or keratoconus
8. High myopia[6,7]

In this subset of patients, intraocular pressures should be monitored regularly. Care should be taken to avoid corticosteroids, if possible. If corticosteroids are indicated, the judicious use of an adequate potency and duration should be considered.

UP University of Medical Sciences and Research, Saifai, U.P.

Many studies independently reported that the normal population could be divided into 3 groups based on their response to the topical administration of dexamethasone and bethamethasone:

(1) High responders, 4–6% of the population, developed an intraocular pressure (IOP) above 31 mm Hg or a rise of more than 15 mm Hg above baseline;

(2) Moderate responders, approximately one third of the population, had IOPs between 20 and 31 mm Hg, or a pressure rise of 6–15 mm Hg;

(3) Nonresponders, the remaining two thirds, had pressure increases of less than 6 mm Hg and IOPs of less than 20 mm Hg

Routes of administration

1-Topical Ocular Preparations -IOP rise may occur with corticosteroid drops or ointment applied to the eye or with steroid preparations applied to the skin of the eyelids (Figure 1).

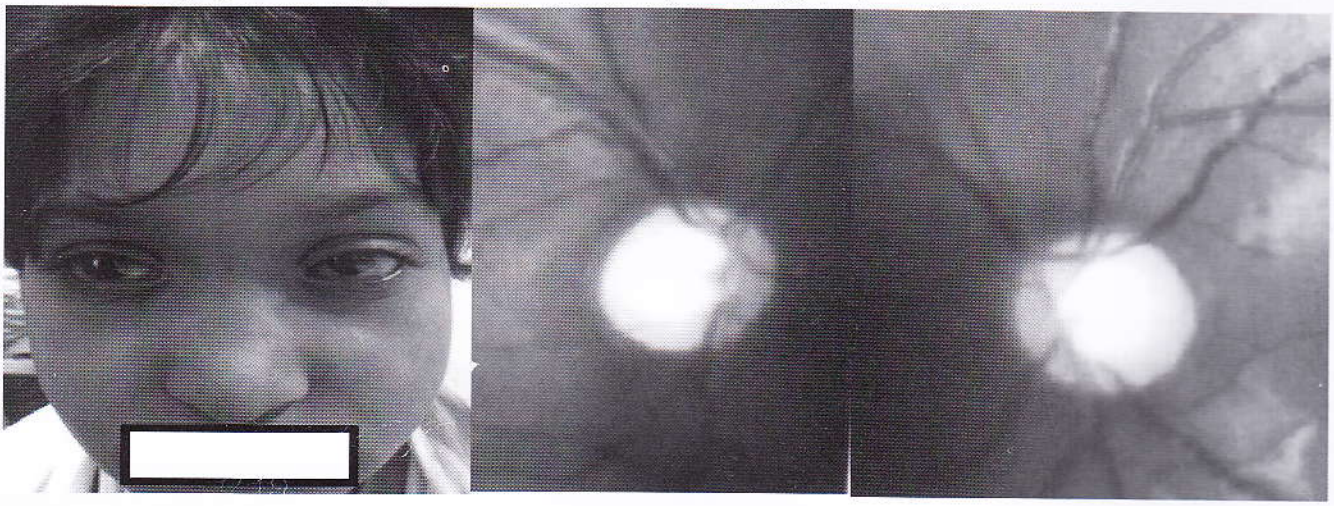


Figure 1- A child with Vernal keratoconjunctivitis OU, presented with glaucoma in both eyes following use of Dexamethasone eye drops for almost a year. She also had posterior subcapsular cataract.

The risk of IOP rise increases with duration of use and may be directly correlated to its anti-inflammatory effect. For example, dexamethasone and prednisolone increase the IOP more frequently than fluoromethalone, hydrocortisone, and rimexolone. Fluoromethalone has poor intraocular penetration with fewer tendencies towards IOP elevation.

According to study [8], inhaled fluticasone at the regular dose used over a short period (6-24 months) was not associated with a significant effect on CCT and IOP measured with noncontact devices in asthmatic children between 5 and 15 years, without a family history of glaucoma.

2. Periocular- This route of steroid delivery includes subconjunctival, sub-tenon's, or retrobulbar injections. Sometimes, it is necessary to excise the depot of steroids in order to control the intraocular pressures

3. Intravitreal IOP elevation develops in about half the patients that receive intravitreal triamcinolone, usually developing between two to four weeks after the injection Smithen *et al*[9] found that 40.4% of patients receiving triamcinolone show a pressure rise to greater than 24 mmHg over at an average of 100 days after treatment.

4. Dermatologic Steroid induced glaucoma may develop after application of steroid preparations applied to the skin of the eyelids. This elevation occurs most frequently with chronic use, such as in patients with atopic dermatitis.

5. Systemic Steroids by mouth (PO) can elevate the IOP as well. The elevation appears to be correlated to the patient's IOP response to topical steroids. Though not common, elevation of IOP has also been noted with the use of inhalational and nasal corticosteroids as well as after intra-articular steroid injections.

Treatment

1-Monitoring of IOP

A baseline measurement of IOP should be taken prior to commencement of corticosteroid therapy. Patients on topical therapy should then have their IOP measured again 2 weeks after initiation of treatment, then every 4 weeks for 2–3 months, then 6-monthly if therapy is to continue.

2-Cessation of corticosteroid treatment

It is ideal to ensure that the suspected corticosteroid is responsible for the glaucoma and if glaucoma is established (and especially if progressive), use of the corticosteroid should be stopped. The chronic corticosteroid response resolves in 1–4 weeks, whereas the rare acute response may resolve within a few days of steroid cessation.[10]

3-Alternative corticosteroid formulations

Topical treatments can be changed to preparations such as fluoromethalone 0.1% or rimexolone 1%, which are claimed to have less effect on IOP[11] or in certain situations to nonsteroidal anti-inflammatory drugs (NSAIDs).

4-Irreversible steroid-induced ocular hypertension/glaucoma

In about 3% of cases, and in particular when there is a family history of glaucoma and/or chronic use of steroid (at least 4 years), the ocular hypertensive response has been shown to be irreversible.[12,13] The management of such cases is no different from that for POAG.

Medical antiglaucomatous therapy

Beta-blockers Topical beta-blockers can be used to control corticosteroid-induced glaucoma, preferably following cessation of steroid therapy and are a popular first-line agent for the condition.

Prostaglandin analogues Concomitant latanoprost has been shown to be as effective as cessation of therapy in controlling the IOP rise associated with corticosteroids so can be useful if steroid treatment must be continued. However, latanoprost has been reported to induce uveitisand is relatively contraindicated in eyes with uveitic glaucoma.

Alpha agonists Brimonidine can be useful in many patients with steroid-induced glaucoma, although there have been reports of brimonidine-induced uveitis in a minority of patients.

Carbonic anhydrase inhibitors Over longer periods, the side effect profile of acetazolamide tends to make it poorly tolerated and it is contraindicated in certain patients, such as those with renal impairment. However, topical carbonic anhydrase inhibitors (dorzolamide and brinzolamide) are of use in the control of IOP due to corticosteroid-induced glaucoma.

Miotics Corticosteroid-induced glaucoma has appeared to be relatively refractory to miotics while the steroid has still been used. Furthermore, in addition to being less popular than more modern agents, miotics are also contraindicated in inflamed eyes (ie those that may require topical steroid therapy) since they can exacerbate the formation of posterior synechiae.

Filtration surgery

Trabeculectomy remains an effective treatment for glaucoma in those patients who have a persistently

raised IOP following cessation of corticosteroid therapy and are refractory to medical therapy.[14] However, as always, the adverse consequences of trabeculectomy or other forms of drainage surgery should be considered in relation to the potential benefits.

Future Therapies

Glucocorticoid receptor blockers have been proposed as useful potential therapeutic agents for treating corticosteroid-induced glaucoma like Anecortave acetate(an analog of cortisol acetate) and Mifepristone (RU 486-6, a peripheral progesterone blocker with antiglucocorticoid properties),which is still under research area.

REFERENCES

1- Epstein DL, Allingham RR, Schuman JS, eds. *Chandler and Grant's Glaucoma.* 4th ed. Baltimore: Williams & Wilkins; 1997.

2- Becker B, Hahn KA. Topical corticosteroids and heredity in primary open-angle glaucoma. *Am J Ophthalmol* 1964; **54**: 543–551.

3- Davies TG. Tonographic survey of the close relatives of patients with chronic simple glaucoma. *Br J Ophthalmol* 1968; **52**: 32–39.

4- Becker B. Diabetes mellitus and primary open-angle glaucoma. *Am J Ophthalmol* 1971; **71**: 1–16.

5- Gaston H, Absolon MJ, Thurtle OA, Sattar MA. Steroid responsiveness in connective tissue diseases. *Br J Ophthalmol* 1983; **67**: 487–490.

6- Podos SM, Becker B, Morton WR. High myopia and primary open-angle glaucoma. *Am J Ophthalmol* 1966; **62**: 1038–1043.

7- Spaeth GL. Traumatic hyphaema, angle recession, dexamethasone hypertension, and glaucoma. *Arch Ophthalmol* 1967; **78**: 714–721.

Miller D, Peczon JD, Whitworth CG. Corticosteroids and functions in the anterior segment of the eye. *Am J Ophthalmol* 1965; **59**: 31–34.

8- Middle East Afr J Ophthalmol. 2012 Jul-Sep;19(3):314-9. doi: 10.4103/0974-9233.97936.

9- Smithen LM, Ober MD, Maranan L, Spaide RF. Intravitreal triamcinolone acetonide and intraocular pressure. *Am J Ophthalmol* 2004; **138**: 740–743.

10- Weinreb RN, Polansky JR, Kramer SG, BaxterJD. Acute effects of dexamethasone on intraocular pressure in glaucoma. *Invest Ophthalmol Vis Sci* 1985; **26**(2): 170–175.

11- Cantrill HL, Palmberg, Zink HA, Waltman SR, Podos SM, Becker B. Comparison of *in vitro* potency of corticosteroids with ability to raise intraocular pressure. *Am J Ophthalmol* 1975; **79**: 1012–1017.

12- Francois J. Corticosteroid glaucoma. *Ann Ophthalmol* 1977; **9**: 1075–1080.

13- Espildora J, Vicuna P, Diaz E. Cortisone-induced glaucoma: a report on 44 affected eyes. *J Fr Ophthalmol* 1981; **4**: 503–508.

14- Thomas R, Jay JL. Raised intraocular pressure with topical steroids after trabeculectomy. *Grafes Arch Clin Exp Ophthalmol* 1988; **226**: 337–340.

POSTERIOR POLAR CATARACT

Mohmad Uzair, Summia Singh, Neelima Mehrotra

Abstract

Although cataract surgery in posterior polar cataract is associated with posterior capsular rent and nucleus drop, by taking precautions the rate of complication can be decreased. The aim of this article is to review the etiology, pathogenesis, clinical features, and management of posterior polar cataract.

1. Introduction

Posterior polar cataract (PPC) is a relatively uncommon form of congenital cataract accounting for around 0.5% to 2% of the total cataract. It presents a special challenge to cataract surgeon because of high likelihood of posterior capsular rent (PCR) and nucleus drop during surgery. Although incidence of posterior capsular dehiscence has been reported to be 26% - 36% [1] [2] in older studies, its incidence has been reduced to 7.1% - 16.4% [3] according to recent study.

Incidence—Incidence of PPC ranges from 3 to 5 in 1000 [4]. It is found to be bilateral in 65% - 80% of the cases [2] [5]. There is no sex predilection in general.

Morphology—Posterior polar cataract is a variant of developmental cataract. The lens may have a small opacity at birth. However, the cataractous changes usually occur later in life. It appears as a white dense opacity located in the posterior cortex, posterior capsule and the posterior subcapsular region. These cataracts have a different appearance compared with the routine posterior subcapsular axial opacity that occurs in young adults. There are onion-like concentric rings around the central opacity (bull's eye).

Inheritance and genetics—It has been recognized that ppc seems to follow an autosomal dominant inheritance pattern [6], although it is occasional and sporadic. Positive family history was found in 40% - 55% of the patients [1] [2]. Molecular genetic analyses have demonstrated that an autosomal-dominant posterior polar cataract is a genetically heterogenous disease [6]. The direct cause of the lenticular fiber malformation during lens development has not been well understood. There are five genes attributed to posterior polar cataract (CTTP) that have been identified. CTPP1 (OMIM 116600) has been mapped to 1p36 [6]. CTPP2 has been associated with *CRYAB* on 11q22-q22.3, and a Pro20Ser mutation and a deletion mutation (450delA) have also been highlighted [7]. The *CHMP4B* gene on chromosome 20p12-q12 is responsible for CTPP3 (OMIM 605387). Three mutations of *PITX3* gene on chromosome 10q25, 38G > A mutation, 17-bp insertion, and 650delG have been reported to cause CTPP4 (OMIM 610623) **[6]**.

2. Pathogenesis

The position of lens opacity is largely determined by the anatomy of the lens and the timing and nature of the insult that caused the abnormality by altering the embryogenesis. It has been suggested that posterior polar cataracts are caused by persistence of the hyaloid artery [5] or invasion of the lens by mesoblastic tissue. It appears that posterior polar cataract forms during embryonic life or early in infancy and usually becomes symptomatic 30 - 50 years later. The exact pathogenesis of posterior polar cataract is still unknown. However, it has been noted to occur as a result of genemutation [6]. A posterior polar cataract consists of dysplastic lens fibers, which, in their migration posteriorly from the lens equator, exhibit progressive lens opacity, increased degenerative changes, with the formation of a characteristic discoid posterior polar plaque-like cataract and the accumulation of extracellular material [8]. It seems that the high incidence of posterior capsule rupture during surgery for those patients might be because of two reasons. First, there might be tight adherence of the plaque to an otherwise normal capsule. Second, the posterior capsule itself underlying the plaque is exceptionally thin that ruptures to minimal trauma.

*Sri Ram Murti Smarak Institute of Medical Sciences, Bareilly

3. Classification

3.1. Duke and Elder's Classification [9]

Stationary—Circular well-defined opacity with central concentric ring on posterior capsule, giving a bull's eye appearance. It may or may not be associated with a satellite rosette lesion or nuclear sclerosis. The stationary type is compatible with good visual acuity.

Progressive—Characterized by radiating rider type of opacity in posterior cortex. Symptoms are more in this type of posterior polar cataract.

3.2. Singh's Classification [10]

Type 1: The posterior polar opacity is associated with posterior subcapsular cataract.

Type 2: Sharply defined round or oval opacity with ringed appearance like an onion with or without grayish spots at the edge.

Type 3: Sharply defined round or oval white opacity with dense white spots at the edge often associated with thin or absent posterior capsule. These dense white spots are a diagnostic sign (Daljit Singh Sign) of posterior capsule leakage with or without repair and extreme fragility, the incidence of this type in Indian adult cataract patient population was found to be about one in 300.

Type 4: Combination of the above 3 types with nuclear sclerosis.

Singh observed the possibility of conversion of posterior polar cataract from type two to type three over following patients who for many years were reluctant to have surgery. He recommended not to delay surgery unnecessarily in these cases.

3.3. Schroeder's Classification [11]

This classification grades posterior polar cataract in pediatric patients according to its effect in pupillary obstruction in the red reflex testing as follows:

Grade 1: A small opacity without any effect on the optical quality of the clear part of the lens.

Grade 2: A two-thirds obstruction without other effect.

Grade 3: The disc-like opacity in the posterior capsule is surrounded by an area of further optical distortion. Only the dilated pupil shows a clear red reflex surrounding this zone.

Grade 4: The opacity is totally occlusive; no sufficient red reflex is obtained by dilation of the pupil.

3.4. Clinical Features

Posterior polar cataract patients present with defective vision and glare.

3.5. Diagnosis

On slit-lamp examination the pathognomonic feature of PPC is bulls eye appearance. Examination of the anterior vitreous may reveal oil-like droplets or particles **[5]** and the presence of these should raise the possibility of pre-existing posterior capsular opening. It can be associated with other ocular features like microphthalmia, microcornea, anterior polar cataract **[12]**. In addition, it has been found to be associated with ectodermal dysplasia, Rothmund disease, scleroderma, incontinentia pigmenti, congenital dyskeratosis, congenital ichthyosis and psychosomatic disorders (**Figure 1** and **Figure 2**).

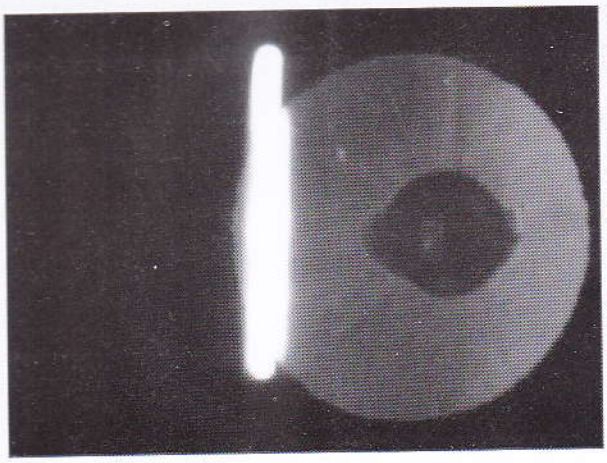


Figure 1: Bulls eye appearance of ppc in diffuse

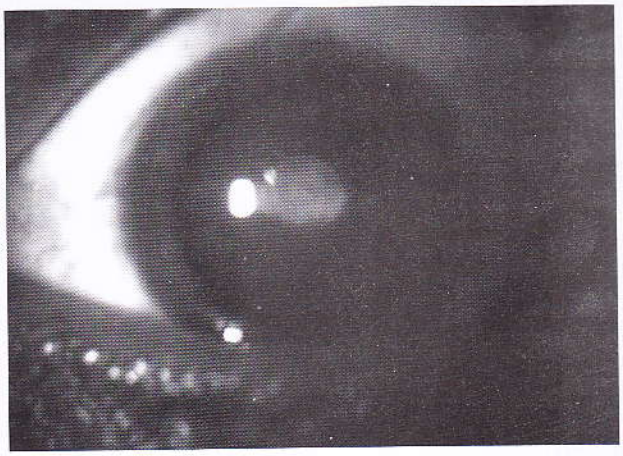


Figure 2: Appearance of PPC in retroillumination

3.6. Decision to Operate

Most of the surgeons opine that surgery should be delayed as long as possible and undertaken only when patients find difficulty in performing routine activities. However, when it is visually significant in childhood, it is considered amblyogenic, which warrants an early intervention in these cases.

Counselling- During the preoperative examination, the physician should inform the patient of the possibility of the nucleus' drop intraoperatively, posterior capsular rupture, a relatively long operative time, secondary posterior segment intervention, and a delayed visual recovery. In addition, the surgeon should discuss Nd:YAG capsulotomy for residual plaque [1] [2] and emphasize the possibility of preexisting amblyopia, especially in cases of unilateral posterior polar cataract. Because the understanding of posterior polar cataract, an autosomal dominant condition, genetic counseling for the parents in addition to screening of family members is important. Anaesthesia-Peribulbar anaesthesia with oculopressure to soften the globe decreases intraoperative positive pressure [2].

3.7. Surgical Techniques

While Osher *et. al.* [1] found no difference in the rate of posterior capsule rupture between phacoemulsification and extracapsular cataract extraction (ECCE), Das *et. al.* in a retrospective analysis concluded that phacoemulsification is preferred to ECCE in posterior polar cataract as they found higher rates of complications with ECCE [13]. However, they recommended ECCE in harder cataract and dense central plaques. They found out that posterior capsule rupture occurs most commonly during emulsification of the nucleus in phacoemulsification and during nucleus expression in ECCE cases. Vasavada and Singh [2] found that rupture occurs most commonly during epinucleus removal in phacoemulsification [1], while Osher *et. al.* found it to happen during removal of the posterior polar opacity or during cleaning of the posterior capsule after plaque removal [2].

4. Phacoemulsification

4.1. The Incision

Starting with the side port incision followed by the injection of viscoelastic material might be better than starting with the main incision. This will avoid the possible chamber collapse that might predispose to premature rupture of the capsule. The incision of the phacoemulsification surgery can be a usual coaxial one, whether corneal or scleral, or it can be microincision for bimanual technique.

4.2. Capsulorhexis

Ideally, the capsulorhexis should be no larger than 5 - 5.5 mm because a larger opening may not leave adequate support for sulcus fixated intraocular lens in case the posterior capsule is compromised. Although a size of 4 mm or less could be detrimental if the surgeon must prolapse the nucleus into the anterior chamber [2] [14].

4.3. Hydro Procedure

Selection of the proper hydroprocedure is one of the most important steps in the management of posterior polar cataract. Most of the authors considered the cortical-cleaving hydrodissection [14] as an absolute contraindication in posterior polar cataract cases, as it can lead to hydraulic rupture & can precipitate an instant nucleus drop. While Fine *et al.* [14] did hydrodissection in multiple quadrants with tiny amount of fluid without allowing the wave to transmit across the posterior capsule.

4.4. Hydrodelineation

Hydrodelineation is the most preferred hydro procedure. It helps to separate the endonucleus from the epinucleus & prevents directly disturbing the posterior capsule. It also creates a mechanical cushion of epinucleus which acts as a safeguard. It is worth mentioning that the surgeon should avoid vigorous decompression of the capsular bag after the delineation.

4.5. Inside-Out Hydrodelineation

Vasavada and Raj described a technique that was described for dense and posterior polar cataract called inside-out delineation [15]. In this technique, a trench is first sculpted and a right-angled cannula is used to subsequently direct fluid perpendicularly to the lens fibers in the desired plane through one wall of the trench. This would avoid the possibility of inadvertent subcapsular injection and overcome the difficulty of introducing cannula to a significant depth in a dense cataract.

4.6. Rotation

It should be avoided, as attempt to rotate the nucleus can lead to posterior capsule rupture [2].

4.7. Phacoemulsification Parameters

According to studies, slow motion phacoemulsification with low parameters should be used. The low vacuum and aspiration rates maintain a very stable chamber and the reduced infusion drives less fluid around the lens.

4.8. Nucleotomy Technique

Bimanual cracking and division of the nucleus involve outward movements and can distort the capsular bag. For nuclear sclerosis > −2 step by step chop in situ and lateral separation technique for chopping should be used. The resultant fragments are removed with a stop and chop and stuff technique [4]. For soft nucleus, the entire nucleus can be aspirated within the epinuclear shell. Lee and Lee [3] sculpted the nucleus in the shape of the Greek letter lambda "λ technique", then cracking along both arms and removing the distal central piece. The advantage of this is its gentleness in not stretching the capsule while removing the quadrants, especially the first one.

4.9. Epinucleus Removal

Removing the epinucleus is again the critical step. The central plaque should be uprooted last to prevent any epinuclear fragments from falling into the vitreous cavity. First the peripheral lower half of epinucleus should be stripped off, central area of epinucleus remains attached. Next, the peripheral upper epinucleus (subincisional) can be mobilized with gentle, focal, multiquadrant hydrodissection using a right-angled

cannula that faces right and left [15]. Some surgeons have suggested performing viscodissection of the epinucleus by injecting viscoelastic under the capsular edge to mobilize rim of epinucleus. If posterior plaque firmly adherent to capsule is uncovered it is left behind which can be dealt with ND:YAG laser capsulotomy post operatively. However, some surgeons may prefer primary posterior capsulorhexis [15] before lens implantation.

4.10. Posterior Capsular Dehiscence

Recognizing the early signs of posterior capsule rupture during cataract surgery and its early management can lead to good visual outcomes.

Signs of an early posterior capsular dehiscence or zonular dehiscence includes sudden deepening of the anterior chamber with momentarily expansion of pupil;

Sudden transient appearance of clear red reflex peripherally;

Newly apparent inability to rotate previously mobile nucleus;

Excessive lateral displacement of nucleus and partial descent of nucleus into anterior vitreous face.

Nucleus drop—Retained posterior nuclear fragments greatly increase the risk of postoperative complications and usually must be retrieved subsequently by posterior segment approach. The likelihood of a dropped nucleus increases the longer the posterior capsule rupture goes unrecognized. The detection allow the surgeon the opportunity to convert to a large incision or extra capsule cataract extraction. A brunescent nucleus may abruptly and rapidly sink through the liquefied vitreous without antecedent vitreous loss. However, if enough supporting formed vitreous is present the nucleus will descend only partially, allowing time for rescue manoeuveres. The

worst strategy for recovering a descending nucleus is to try to chase and spear the nucleus propelling the nucleus away. Attempting to phacoemulsify or aspirate it may snag vitreous in the tip, which could lead to giant retinal tears or a retinal detachment.

4.11. IOL Implantation

Before implanting the IOL, viscoelastic substance is injected in to the capsular bag to expand it adequately. But it is important not to over pressurize the eye. The IOL is carefully implanted, taking care to avoid contact with the posterior capsule during the implantation. Residual viscoelastic material is removed at the end. In case of PC rent, before placing the posterior chamber IOL in the ciliary sulcus, estimate the sulcus diameter by measuring the horizontal white-to-white corneal diameter. if necessary, an attempt can be made to convert the posterior defect into a posterior capsulorhexis [3], perform a central vitrectomy then implant the IOL in the ciliary sulcus and perform the optic capture in the bag (The Rhexis-Fixated Lens) [15]. In case of PC rent, after implanting pciol the viscoelastic should be removed.

Bimanual Phacoemulsification [14] [16]—Bimanual phaco is very good option for posterior polar cataracts to minimize the risk of complications allowing posterior polar cataract extraction to be performed more safely. The added advantages of bimanual phacoemulsification are

A controlled operating environment for slow motion phaco by virtue of its low fluidics;

Allowing withdrawal of phaco needle first while maintaining the anterior chamber with infusion from the separate irrigating chopper.

4.12. Modified Epinucleus Prechop for Dense Polar Cataract

For management of dense posterior polar cataracts, anterior epinucleus is first pre chopped in piecemeal in situ maneuver before mobilizing, segmenting, and emulsifying the dense endonucleus [17]. This is followed

by the removal of the posterior epinucleus and posterior polar plaque. Because chopper is repositioned at different meridians in the mid-periphery of the anterior epinucleus, it stops short of the central posterior epinucleus, thus avoiding extension of the crack toward the posterior polar plaque and posterior capsule.

Prognosis-Prognosis of cataract surgery is good if it is done carefully.

5. Conclusion

Though posterior polar cataract is a challenge for cataract surgeons, patience coupled with meticulous attention to the technique, which includes carefully sized capsulorhexis, avoidance of cortical cleaving hydrodissection, gentle hydrodelineation, atraumatic nucleus handling and tackling the central epinuclear plate in the last part of cortical clean up, ensures a safe surgery and favourable postoperative outcomes.

References

[1] Osher, R.H., Yu, B.C. and Koch, D.D. (1990) Posterior Polar Cataracts: A Predisposition to Intraoperative Posterior Capsular Rupture. *Journal of Cataract Refractive Surgery*, 16, 157-162. http://dx.doi.org/10.1016/S0886-3350(13)80724-9

[2] Vasavada, A.R. and Singh, R. (1999) Phacoemulsification with Posterior Polar Cataract. *Journal of Cataract Refractive Surgery*, 25, 238-245. http://dx.doi.org/10.1016/S0886-3350(99)80133-3

[3] Lee, M.W. and Lee, Y.C. (2003) Phacoemulsification of Posterior Polar Cataracts—A Surgical Challenge. *British Journal of Ophthalmology*, 87, 1426-1427. http://dx.doi.org/10.1136/bjo.87.11.1426-a

[4] Vogt, G., Horvath-Puho, E. and Czeizel, E. (2006) A Population-Based Case-Control Study of Isolated Congenital Cataract. *Orv Hetil*, 147, 1077-1084.

[5] (1924) Congenital Anomalies of the Lens as Seen with the Slit Lamp. *American Journal of Ophthalmology*, 7, 678-680.

http://dx.doi.org/10.1016/S0002-9394(24)90741-0

[6] Addison, P.K., Berry, V., Ionides, A.C., Francis, P.J., Bhattacharya, S.S. and Moore, A.T. (2005) Posterior Polar Cataract Is the Predominant Consequence of a Recurrent Mutation in the PITX3 Gene. *British Journal of Ophthalmology*, 89, 138-141. http://dx.doi.org/10.1136/bjo.2004.053413

[7] Berry, V., Francis, P., *et al.* (2001) Alpha-B Crystallin Gene (CRYAB) Mutation Causes Dominant Congenital Posterior Polar Cataract in Humans. *The American Journal of Human Genetics*, 69, 1141-1145. http://dx.doi.org/10.1086/324158

[8] Eshaghian, J. and Streeten, B.W. (1980) Human Posterior Subcapsular Cataract: An Ultrastructural Study of the Posterioly Migrating Cells. *Archives of Ophthalmology*, 98, 134-143. http://dx.doi.org/10.1001/archopht.1980.01020030136016

[9] Duke-Elder, S. (1964) Posterior Polar Cataract, System of Ophthalmology, Vol. iii, pt. 2: Normal and Abnormal Development, Congenital Deformities. MO, CV Mosby, St. Louis, 723-726.

[10] Masket, S. (1997) Consultation Section: Cataract Surgical Problem. *Journal of Cataract and Refractive Surgery*, 23, 819-824. http://dx.doi.org/10.1016/S0886-3350(97)80236-2

[11] Schroeder, H.W. (2005) The Management of Posterior Polar Cataract: The Role of Patching and Grading. *Strabismus*, 13, 153-156. http://dx.doi.org/10.1080/09273970500387753

[12] Harman, N.B. (1909) New Pidegrees of Cataract-Posterior Polar, Anterior Polar and Microphthalmia, and Lamellar. *Transactions of the Ophthalmological Societies of the United Kingdom*, 29, 296-306.

[13] Das, S., Khanna, R., Mohiuddin, S.M. and Ramamurthy, B. (2008) Surgical and Visual Outcomes for Posterior Polar Cataract. *British Journal of Ophthalmology*, 92, 1476-1478. http://dx.doi.org/10.1136/bjo.2007.129403

[14] Fine, I.H., Packer, M. and Hoffman, R.S. (2003) Management of Posterior Polar Cataract. *Journal of Cataract & Refractive Surgery*, 29, 16-19. http://dx.doi.org/10.1016/S0886-3350(02)01616-4

[15] Vasavada, A.R. and Raj, S.M. (2004) Inside-Out Delineation. *Journal of Cataract & Refractive Surgery*, 30, 1167-1169. http://dx.doi.org/10.1016/j.jcrs.2003.10.034

[16] Haripriya, A., Aravind, S., Vadi, K. and Natchiar, G. (2006) Bimanual Microphaco for Posterior Polar Cataracts. *Journal of Cataract & Refractive Surgery*, 32, 914-917. http://dx.doi.org/10.1016/j.jcrs.2006.02.049

[17] Lim, Z. and Goh, J. (2008) Modified Epinucleus Pre-Chop for the Dense Posterior Polar Cataract. *Ophthalmic Surgery, Lasers and Imaging Retina*, 39, 171-173. http://dx.doi.org/10.3928/15428877-20080301-11

STUDY OF ETIOLOGICAL FACTOR OF PEDIATRIC CATARACT IN GORAPKHPUR AND SURROUNDING REGION

Ram Kumar, Saraswati

Abstract

Cataract is the opacification of the eye lens. Based upon the age at appearance, cataract can be classified as congenital, infantile, juvenile, presenile and senile. Pediatric cataracts are responsible for more than 1 million childhood blindness.

Aims and Objectives:

1. To know the incidence of different varieties of pediatric cataract.
2. To know about the etiology of pediatric cataract in Western UP.

Material and Method:

This is a prospective observational study for 1 year with the sample size of 160 patients.

Result and conclusion:

It could be concluded that in pediatric age group, maximum percentage of patients had non traumatic type cataract, out of that idiopathic was found to be more common. Morphological total type cataract was also common. In traumatic cataract, most common cause included arrow - bow /wooden stick injury, followed by cracker and stone injury.

Introduction

Cataract is the opacification of the eye lens. Based upon the age at appearance, cataract can be classified as congenital, infantile, juvenile, presenile and senile. Pediatric cataracts are responsible for more than 1 million childhood blindness in Asia[1]. In developing countries like India, 7.4 - 15.3% of childhood blindness is due to cataract[2-4].The prevalence of cataract in children has been estimated between 1-15/10,000 children[5].

In eastern UP (Gorakhpur, Deoria, Maharanganj, Mau, Gazipur), the prevalence of pediatric cataract seems to be high as the number of children presenting with the problem is significant. Pediatric cataract is the leading cause of childhood blindness. Its high incidence in Eastern UP gives an opportunity to know about its prevalence as well as causative factors. This may provide glimpse into how to provide improved treatment, as also decrease the incidence of childhood blindness in India.

Aims and Objectives:

1. To know the incidence of different varieties of pediatric cataract.
2. To know about the etiology of pediatric cataract in Western UP.

Material and Method

This is a prospective observational study for 1 year (July 2017-July 2018) in Ophthalmology Department of BRD Medical College, Gorakhpur; including camps (school & rural), organized by the department.

Sample size -160

Data collection: Data was collected from patients coming to the Ophthalmology Department, Gorakhpur district hospitals including camps.

* Department of Ophthalmology, BRD Medical College, Gorakhpur

Inclusion criteria

Age: 0-15 years

Unilateral, bilateral & traumatic cataract.

A team of ophthalmologists and pediatricians attached to the centre examined all the patients preoperatively. For preoperative examination, the pupil was dilated using atropine eye drops. Children who did not cooperate were examined by giving them short-term general anesthesia under operating microscope. The type of cataract was determined using slit lamp bio-microscopy or operating microscope. To find out any other ocular pathology, vertical -horizontal corneal diameter, intraocular pressure, keratometry, axial length were measured and fundus status evaluated with indirect ophthalmoscope. Biochemical investigations such as urine sugar and blood glucose level were performed in all patients.8 TORCH test was done only in children less than one year of age with central nuclear or total cataract unilaterally or bilaterally and whose mothers revealed a history of illness accompanied by a rash during the pregnancy. The complete clinical details of the patient including cataract type and other ocular and non-ocular disorders and laboratory investigations were recorded. Information regarding proband cataract history, parental health history, prenatal and postnatal history, child's birth history, consanguinity, socioeconomic and demographic status were also noted. Informed consent was obtained from all the families included in this study. Clinical information of all patients was analyzed according to their etiology. The cases were divided into traumatic and non-traumatic. The traumatic patients were further classified based on the causes of trauma. The non-traumatic cases were classified into four groups based on the following considerations.

A-Hereditary: This group included cases with positive family history.

B. Secondary: This group-included case with any other ocular disease, metabolic –systemic diseases, and cataracts associated with known syndromes. The syndromic cataracts were determined based on the clinical observation. The metabolic disorders were confirmed by the proband's history and earlier diagnosis of referring pediatrician.

C. Rubella: This group-] included cases caused by diagnostically confirmed congenital rubella syndrome.

D. Undetermined: This group included idiopathic cases with no known cause.

Observation

Table- 1: Gender distribution in study

Gender	Frequency	Percentage
Male	100	62.5%
Female	60	37.5%

Table-2: Etiology of non traumatic cataract

Cause	Unilateral/%	Bilateral/%	total
Hereditary	1(.73%)	8(5.84%)	9(6.57%)
Rubella	1(.73%)	4(2.92%)	5(3.65%)
Secondary	2(1.46%)	18(13.14%)	20(14.60%)
Undetermined	33(24.09%)	70(51.09%)	103(75.18%)
Total	37(27.01%)	100(72.99)	137(100%)

Table-3: Etiology of cataract in children less than 1 year age

Causes	Unilateral/(%)	Bilateral (%)	Total
Non traumatic			
Hereditary	0	5(6.17%)	5(6.17%)
Rubella	1(1.23%)	2(2.46%)	3(3.70%)
Secondary	1(1.23%)	3(3.70%)	4(4.94%)
Undetermined	27(33.33%)	40(49.38%)	67(82.72%)
Traumatic	2(2.46%)	0	2(2.47%)
Total	31(38.27%)	50(61.73%)	81(100%)

Table-4: Morphological type of cataract

Cataract type	Number	Percentage
Total cataract	49	35.32%
Lamellar	29	21.05%
Nuclear	12	8.55%
Posterior subcapsular	6	4.6%
Posterior polar	3	1.90%
Mixed	36	26.0%
Blue dot cataract	1	1.30%
Sutural	1	0.65%
Total	137	100

Table-5: Etiology of traumatic cataract by age

Cause	<1	1-5	6-10	11-15	Total
Crackers	-	2	1	1	4(17.39%)
Wood stick	-	1	4	-	5(21.74%)
Finger	-	1	1	-	2(8.69%)
Chemical	1	-	-	-	1(4.34%)
Hair pins	1	-	-	-	1(4.34%)
Needle	-	1	-	-	1(4.34%)
Wire	-	1	-	-	1(4.34%)
Stone	-	1	1	2	4(17.39%)
Others	-	2	1	1	4(17.39%)
Total	2	9	8	4	23

Result and discussion

This is a prospective observational study for 1 year including 160 patients with 100 (62.5%) male and 60 (37.5%) female patients respectively. In our study we found that 160 patients presented with cataract out of that 137(85.62%) were non traumatic and 23(14.38%)were traumatic. It was found that 81 (50.62%) cataract patients were below 1 year of age and 79 (49.38%) were above 1 year of age.

On study of etiological factor of non-traumatic cataract, we found that maximum number of cataract occurred due to idiopathic causes 103(75.18%), followed by secondary cataract 20(14.60%), hereditary 9(6.57%) and rubella (3.65%) and among that unilateral cataract 37(27.01%) and bilateral cataract 100(72.99%). Similar results were reported by **Haargaard** *et. al.* Idiopathic cases showed a higher proportion of unilateral cataract and of additional ocular dysmorphology compared with cases of known etiology. The etiology was unknown in 87% of unilateral cases and in 50% of bilateral cases.[6]

On study of morphological of cataract in non-traumatic group we found that maximum percentage was 49(35.37%) of total type of cataract, followed by mixed 36(26.0%) and lamellar 29(21.05%). We found that traumatic cataract maximum times occurred due to arrow bow/wood stick (21.74%), cracker (17.39%) and stone (17.39%).

Conclusion

It could be concluded that in pediatric age group, maximum percentage of patients had non traumatic type cataract, out of that idiopathic was found to be more common. Morphological total type cataract was also common. In traumatic cataract, most common cause included arrow - bow /wooden stick injury, followed by cracker and stone injury.

Awareness programs for pregnant women forconcerning precautions during pregnancy andalso for keeping records of medications takenduring pregnancy might help in future etiological studies. School children must also be educated regarding factors, which can cause traumatic cataract, which might reduce the incidence of childhood cataract.

References

1. World Health Organisation. Prevention of childhood blindness. Geneva: WHO; 1992.

2. Gilbert CE, Canovas R, Hagan M, Rao S, Foster A. Causes of childhood blindness: results from West Africa, South India and Chile. Eye 1993;7:1848.

3. Dandona L, Williams JD, Williams BC, Rao GN. Population-based assessment of childhood blindness in souther n India. Arch Ophthalmol 1998;116:545-6.

4. Rahi JS, Sripathi S, Gilbert CE, Foster A. Childhood blindness in India: causes in 1318 blind school students in nine states . Eye 1995;9:545-50.

5. Foster A, Gilbert C, Rahi JS. Epidemiology of cataract in childhood: A global perspective. J Cataract Refract Surg. 1997;23:601-4.

6. **Haargaard *et. al.*** (*Ophthalmology 2004;111(12):2292-8.*

MYOPIA OF PREMATURITY: A MISSED OPPORTUNITY

S.A. Rizvi, N. Akhtar

Preterm babies are defined as babies born before 37 weeks of pregnancy. About 15 million preterm babies are born every year and 1 million children die each year due to complications related to preterm birth.[1] Globally, prematurity is the leading cause of mortality in children under the age of 5 years. Many babies who survive, face lifetime of disabilities like learning disability, visual and hearing problems. Due to recent advances in neonatal care, survival of preterm babies has improved drastically. However, this has also opened a Pandora's Box of challenges.

It is well documented that refractive disorders such as myopia, anisometropia and astigmatism, are common in preterm infants. World Health Organization (WHO) classified Myopia as one of the leading causes of blindness and visual impairment in the world today.[2] The prevalence of myopia varies, depending on age at examination, family history,[3,4] ethnicity,[3] and occupation.[5] Genetic factors, environmental factors, premature birth, and retinopathy of prematurity (ROP) are well known to be associated with the development of a specific form of myopia.

According to Chen et. al., "The most important predictors for refractive outcomes are optical components such as anterior chamber depth, lens thickness, and axial length. Gestational Age (GA), Birth Body Weight (BBW), and some postnatal diseases exert their effects on refractive status mainly through indirectly affecting optical components."[6] Many cross-sectional studies have revealed that premature babies as compared to full-term infants are more prone to development of myopia from an early age and may remain myopic later on in childhood and adolescence. This is known as myopia of prematurity (MOP),[7-12] and it can continue to increase up to the age of 2 years.[13,14]

Fielder and Quinn[15] classified myopia associated with premature birth into three types: (1) physiologic myopia; (2) myopia with preterm birth; and (3) myopia induced by ROP. The prevalence of these specific types of myopia varies and likely depends on the preterm birth, such as low birth body weight (BBW) and small gestational age (GA) at birth, the severity of ROP, and emmetropization in early infancy. The biometric components that have been shown to contribute to this refractive error include a shallower ACD,[16] increased lens power,[12] increased CC,[16] and a shorter overall AL than would be expected for the dioptric value of the eye.[16] Later on, reports of increased PSL are noted. It seems that the early effect of growth restriction associated with ROP is followed later by a deregulation of ocular growth within the posterior segment.

The most comprehensive prospective data came from the Early Treatment for Retinopathy of Prematurity (ETROP) study and Cryotherapy for Retinopathy of Prematurity (CRYO-ROP) study[14, 17]. These studies have found that preterm infants with severe ROP exhibit significant refractive errors at a higher frequency than those with mild ROP and without ROP. The risk of myopia at 12 months of age doubles with each increasing stage of the disease, birth weight of less than 751 gram leads to a threefold increase in the risk of developing myopia. In eyes randomized to no treatment overall incidence of myopia was 21% at 1 year, falling to 16% at 4.5 years of age. The incidence of myopia in eyes with stage 0 disease was found to be 10%; in eyes with spontaneously regressed ROP, 20%; and in eyes with severe ROP and sequelae, 80%. Fledelius also contributed much to our knowledge of about the association between myopia and ROP and its treatment [8,10,16]. The incidence of myopia ranges from 1% to 16% in eyes with stage 0 disease.[14,18,19] If mild ROP is present, this incidence ranges from 17% to 50%,[14,17] increasing in some publications up to 100% in eyes with stage 3 disease.[14] In contrast to MOP, however, myopia after severe ROP is relatively stable in early childhood .In summary children with laser-treated severe ROP had the highest prevalence of refractive errors during the first 2 years of life, indicating that such children should be monitored for at least 2 years.

*Institute of Ophthalmology, JNMCH, AMU, Aligarh

In conclusion, refractive errors especially myopia is common among preterm babies. They may or may not be associated with ROP and may hinder development of normal binocular vision. In order to prevent significant visual handicap, all preterm babies should have an ophthalmological examination at one year of age and long-term follow up should be insured.

References

1. Liu L, Oza S, Hogan D, Chu Y, Perin J, Zhu J, et al. Global, regional, and national causes of under-5 mortality in 2000-15: an updated systematic analysis with implications for the Sustainable Development Goals. Lancet. 2016;388(10063):3027-35.

2. PararajasegaramR. Vision 2020 : the right to sight: from strategies to action. *Am J Ophthalmol*. 1999;182:359–360.

3. ZadnikK, SatarianoWA, MuttiDO, SholtzRI, AdamsAJ. The effect of parental history of myopia on children's eye size. *JAMA*. 1994;2711:1323–1332.

4. LeeKE, KleinBE, KleinR, FineJP. Aggregation of refractive error and 5 year changes in refractive error among families in the Beaver Dam Eye Study. *Arch Ophthalmol*. 2001;119(11)1679–1685. [CrossRef] [PubMed]

5. WuSY, NemesureB, LeskeMC. Refractive errors in a black adult population: the Barbados Eye Study. *Invest Ophthalmol Vis Sci*. 1999;40(10)2179–2184. [PubMed]

6. Chen TC, Tsai TH, Shih YF, Yeh PT, Yang CH, Hu FC, et al. Longterm evaluation of refractive status and optical components in eyes of children born prematurely. Invest Ophthalmol Vis Sci 2010;51:6140e8.

7. O'ConnorA, StephensonT, JohnsonA, et al. Long term ophthalmic outcome of low birth weight children with and without retinopathy of prematurity. *Paediatrics*. 2002;109(1)12–18. [CrossRef]

8. FledeliusHC. Myopia of prematurity: changes during adolescence. *Doc Ophthalmol Proc Series*. 1981;28:63–69.

9. HungerfordJ, StewartA, HopeP. Ocular sequelae of preterm birth and their relation to ultrasound evidence of cerebral damage. *Br. J. Ophthalmol*. 1986; 70:463–468. [CrossRef] [PubMed]

10. FledeliusHC. Pre-term delivery and subsequent ocular development: a 7–10 year follow up of children screened for ROP 1982–4. *Acta Ophthalmol Scand*. 1996;74(3)297–300. [PubMed]

11. BirgeH. Myopia caused by prematurity. *Trans Am Ophthalmol Soc*. 1955.292–298.

12. Gordon A, Donzis P. Myopia associated with ROP. *Ophthalmology*. 1986;93:1593–1598. [CrossRef][PubMed]

13. Page J, Schneeweiss S, WhyteH, Harvey P. Ocular sequelae in premature infants. *Paediatrics*. 1993;92(6)787–790.

14. Quinn G, Dobson V, Repka MX, et al. The CROP-ROP Group. Development of myopia in infants with birthweights less than 1251 g. *Ophthalmology*. 1992;99(3)329–340. [CrossRef] [PubMed]

15. Fielder AR , Quinn GE. Myopia of prematurity: nature, nurture, or disease? Br J Ophthalmol 1997;81:2-3.

16. FledeliusHC. Pre-term delivery and subsequent ocular development: 7–10 year follow-up of children screened 1982–84 for ROP. 4. Oculometric and other metric considerations. *Acta Ophthalmol Scand*. 1996;74(3)301–305. [PubMed]

17. QuinnG, DobsonV, KivlinJ, et al. The Cryo-ROP Group. Prevalence of myopia between 3 months and 5 ½ years in premature infants with and without ROP. *Ophthalmology*. 1998;105(7)1292–1300.[CrossRef] [PubMed]

18. SchafferD, QuinnG, JohnsonL. Sequelae of arrested mild ROP. *Arch Ophthalmol*. 1984;102:373–376.[CrossRef] [PubMed]

19. DarlowB, ClemettRS, HorwoodLJ, MogridgeN. Prospective study of New Zealand infants with birth weight less than 1500g and screened fro ROP: visual outcome at age 7–8 years. *Br J Ophthalmol*. 1997;81:935–940. [CrossRef] [PubMed]

NATAMYCIN RESISTANT FUNGAL KERATITIS: A CASE REPORT

*Diksha Prakash, *Abhishek Chandra, *Govind V. Khalkho, **OPS Maurya

Introduction

Mycotic keratitis is a challenging condition in ophthalmological practice. Ocular trauma, particularly trauma caused by vegetative material, is reported as the most common predisposing risk factor [1, 2]. Treatment options are limited, and many cases require surgery to maintain corneal integrity [3, 4]. Overall, the most common causes of filamentous mycotic keratitis worldwide are the Aspergillus and Fusarium genera [5, 6]. In the Aspergillus group, Aspergillus fumigatus followed by Aspergillus flavus (section Flavi) are the most commonly encountered opportunistic pathogens. This is an interventional case report describing the course and treatment of natamycin resistant Aspergillus flavus keratitis in an elderly male.

Case report

A 62 year old male presented with chief complaints of pain, redness and decreased vision in his left eye for 7 days. On examination his right eye had vision of 6/18 and NS 2 cataract. His left eye had vision of CF 1m. There was diffuse conjunctival congestion along with central 6mm corneal infiltrate and overlying epithelial defect (Fig. 1 and 2). The posterior view was hazy.

A diagnosis of OS fungal keratitis was made and corneal scraping was sent for smear and culture-sensitivity. He was started on topical natamycin 5% eye drops half hourly along with atropine sulphate 1% eye drops and oral anti-inflammatory tablets for pain.

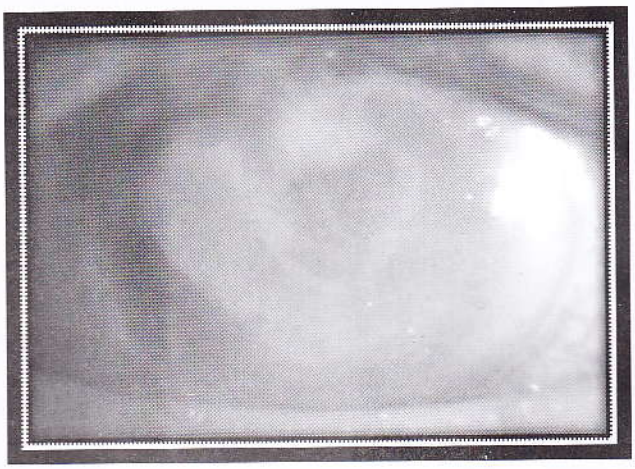


Figure 1

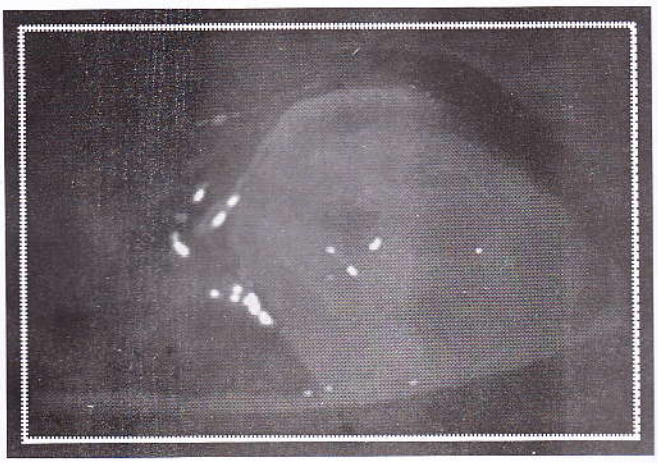


Figure 2

On the next follow up after 10 days, the corneal infiltrates had increased along with presence of hypoyon which suggested worsening of keratitis (Fig. 3). The culture report showed Aspergillus flavus which was susceptible to voriconazole and itraconazole. So voriconazole (10mg/ml) was started at half hourly interval and patient was called after a week.

After a week, patient showed symptomatic and clinical improvement. The corneal infiltrates had decreased and hypopyon was also reduced (Fig. 4). The same treatment was continued and patient was called again after one week.

Chandra Eye Care, Varanasi, U.P.
Department of Ophthalmology, Institute of Medical Sciences, BHU.

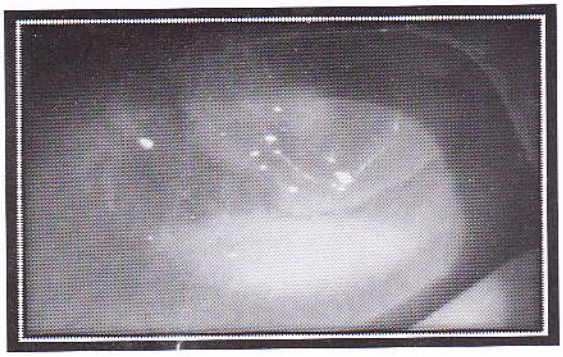


Figure 3

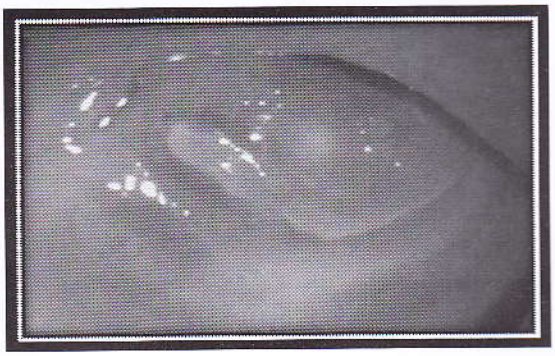


Figure 4

On the next follow up, the corneal ulcer had healed with scarring and hypopyon had completely resolved (Fig. 5). His vision was CFCF.

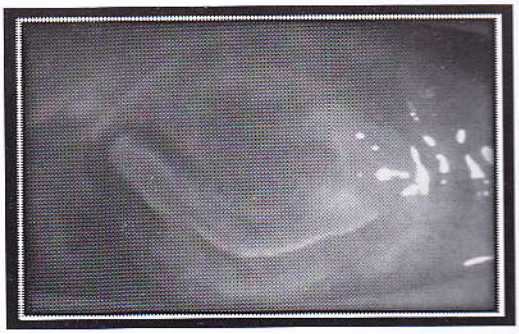


Figure 5

Discussion : The treatment of mycotic keratitis is challenging because of the poor corneal penetration of medications and the limited efficacy of the available drugs. Voriconazole is a second-generation triazole that shows good corneal penetration [7]. In our case, Natamycin was not efficacious so we used voriconazole after the culture-sensitivity report. Despite intensive antifungal treatment, perforation is not uncommon, so adjuvant treatments may be needed to prevent complications [8].

Conclusion : As natamycin 5% is the first line of management of fungal keratitis in our set up, resistance to this drug is of major concern. In all the cases of corneal ulcer, corneal scraping should be sent for culture and sensitivity as the culture report in our case helped us in saving the eye of our patient. Voriconazole is the drug of choice for treatment of Aspergillus spp. that show resistance to topical natamycin and amphotericin B. In our other case series of 24 patients, we observed that although A.fumigatus was susceptible to natamycin but A.flavus was highly resistant to natamycin and susceptible to voriconazole.

References

1. Manikandan P, Varga J, Kocsube S, et al. Epidemiology of Aspergillus keratitis at a tertiary care eye hospital in South India and antifungal susceptibilities of the causative agents. Mycoses. 2013; 56:26–33.
2. Thomas PA, Kaliamurthy J. Mycotic keratitis: epidemiology, diagnosis and management. Clin Microbiol Infect. 2013; 19:210–20.
3. Jurkuna U, Behlau I, Colby K. Fungal keratitis: changingpathogens and risk factors. Cornea. 2009; 28:638–43.
4. Chen WL, Wu CY, Hu FR, et al. Therapeutic penetratingkeratoplasty for microbial keratitis in Taiwan from 1987–2001. Am J Ophthalmol. 2004; 137:736–43.
5. Tanure MA, Cohen EJ, Sudesh S, et al. Spectrum of fungalkeratitis at Wills Eye Hospital, Philadelphia, Pennsylvania. Cornea. 2000; 19:307–12.
6. Thomas PA. Current perspectives on ophthalmic mycoses.Clin Microbiol Rev. 2003;16:730–97.
7. Johnson LB, Kauffman CA. Voriconazole: a new triazoleantifungal agent. Clin Infect Dis. 2003; 36:630–7.
8. Prajna NV, Mascarenhas J, Krishnan T, et al. Comparison ofnatamycin and voriconazole for the treatment of fungal keratitis. Arch Ophthalmol. 2010; 128:672–8.

KERATOPROSTHESIS

Anushri Agrawal,* Shri Kant**

Introduction

The basic idea of using an artificial cornea or performing keratoprosthesis to replace a damaged and opaque cornea is derived from the practice of placing a window in a house to be able to see out. This idea first occurred to the French doctor Guillaume Pellier de Quengsy, who published the feat in the times of the French Revolution (18th century) [1-3].

Keratoprosthesis is a surgical procedure where a diseased cornea is replaced with an artificial cornea. Traditionally, keratoprosthesis is recommended after a person has had a failure of one or more donor corneal transplants. While conventional cornea transplant uses donor tissue for transplant, an artificial cornea is used in the Keratoprosthesis procedure. The surgery is performed to restore vision in patients suffering from severely damaged cornea due to congenital birth defects, infections, injuries and burns.

Keratoprotheses are made of clear plastic with excellent tissue tolerance and optical properties. They vary in design, size and even the implantation techniques may differ across different treatment centers. The procedure is done by Ophthalmologists, often on an outpatient basis.

During the 19th century, there were scattered surgeons who attempted to follow on Quengsy's footsteps, but with equally disastrous outcomes (endophthalmitis, extrusion, and loss of the eye). Thus, it was not until the 1950's with the introduction of new materials, such as transparent non-toxic plastics, that some measure of success began to be reported[4-7]. The good results of these new designs are also attributed to the discovery of antibiotics and steroids, which have significantly improved the postoperative management.

Prosthetic corneas form the last resort for corneal blindness, especially in eyes with end-stage ocular surface disorders and in those at a high risk for conventional penetrating keratoplasty[8,9]The choice of keratoprosthesis (Kpro) depends on the underlying etiology, the anatomy of the ocular surface and the tear film status.

Types/Designs of Keratoprosthesis

Broadly speaking, keratoprostheses are categorized into the Type 1 and 2 Kpros based on the type of eye they cater to. Largely, eyes with normal lids, blink and tear meniscus without an underlying immunological etiology are considered as candidates for the Type 1 Kpro, the prototype of which is the Boston Type 1 Kpro. However, in eyes with severely dry or keratinized ocular surface with an underlying immunological disorder, associated with lid abnormalities, Type 2 Kpros are considered to be the surgical choice.

The design of a Kpro can be likened to some extent to that of an intraocular lens consisting of an optic and a haptic. The optic, which forms the central part of the Kpro responsible for viewing, in most types is a cylinder made of polymethyl methacrylate (PMMA) – creating an optically clear window. It is the haptic of the Kpro which determines the type of the prosthesis. It could be classified as:

- Biocompatible – usually a PMMA skirt with the corneal graft as in the Boston Type 1 and 2 Kpro.
- Biointegrated – as in the Dacron mesh that forms the skirt around the PMMA optic in the Pintucci Kpro.
- Biological – tooth or the bone that forms an autologous biological tissue that supports the optical cylinder in the osteoodonto and the osteo-Kpro, respectively.

The supporting cover tissue adds to the Kpro complex which is the bandage contact lens in the Type 1 Kpro that prevents the carrier graft desiccation. In Type 2 Kpros, the supporting cover is the skin in the Boston Type 2 and the buccal mucosa for the osteo and the osteo-odonto and Pintucci Kpros, respectively.

Indications

Kpros are performed for bilateral corneal blindness not amenable to conventional penetrating keratoplasty.

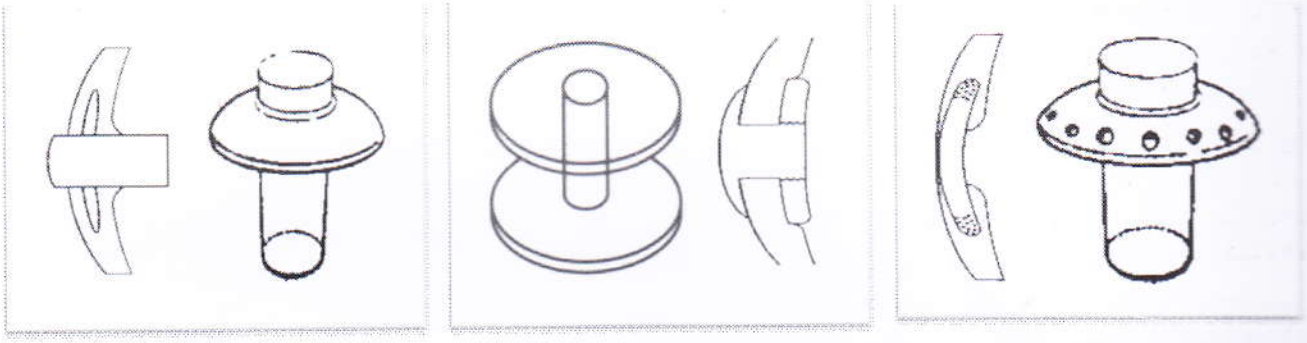
- Stevens – Johnson Syndrome
- Ocular Cicatricial Pemphigoid (Stages 3 & 4)
- Chemical Injury
- Trachoma (Stage C0 according to WHO)
- Vascularized corneas with complete stem cell loss and dryness.
- Multiple failed penetrating Keratoplasty/ Amniotic membrane or stem cell grafting.

Keratoprosthesis designs have primarily been variations of 3 main types.

First Type- PMMA stem with skirt embedded within the cornea.

Second Type- Transparent membrane with porous edges inserted into the cornea.

Third Type- PMMA 'collar button' with cornea between the plates



Boston Keratoprosthesis

The Boston Type I Keratoprosthesis is currently the most commonly used keratoprosthesis device in the US. It consists of a clear plastic polymethylmethacrylate (PMMA) optic and back plate sandwiched around a corneal graft and secured with a titanium locking ring. After the device is assembled, a partial-thickness trephination is performed on the host cornea. Full-thickness resection of the patient's cornea is then completed using curved corneal scissors. The keratoprosthesis is then secured to host tissue using interrupted or running sutures.

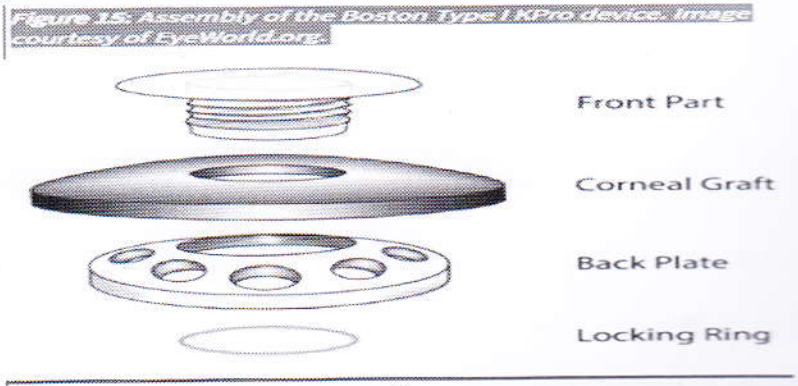
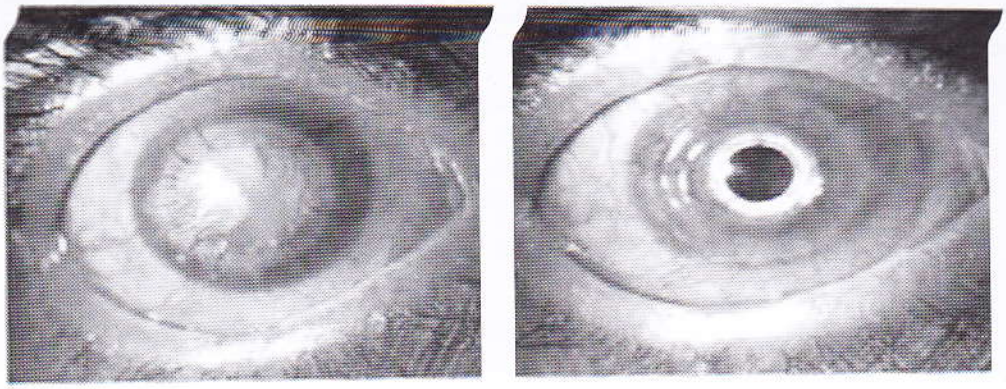


Figure 15: Assembly of the Boston type I KPro device. Image courtesy of Eyeworld.org.



Osteo-odonto Keratoprosthesis

The use of osteo-dental tissue for Keratoprosthesis was first described by Strampelli in the early 1960[10]. There is long term retention of the implant. The surgery is multi-staged and requires cross speciality experience consisting of:-

Stage I

- Preparation of globe with buccal mucous membrane graft
- Preparation of Osteoodonto acrylic lamina (OOAL)

A monocuspidate tooth is removed along with the adjacent maxillary bone and a thin section is cut from the tooth. An optical cylinder made up of PMMA is inserted through a hole made in the section. A pocket is created in the lower eyelid into which the entire prosthesis is inserted and left for 3 months. During this time soft tissue grafts to the bone to which the tooth is attached.

Stage II

Implantation of OOAL- Part of the oral mucosa is stripped off the cornea and sclera to make space for the final implantation of prosthesis. The prosthesis is detached from the eyelid pocket and implanted, with the optical cylinder protruding through a hole in the mucosa.

Chirila (AlphaCor) Prosthesis

It is the newest keratoprosthetic device. It was FDA-approved in August 2002 for patients at high risk for donor penetrating keratoplasty (PKP).

Design

The implant is a 7-mm diameter, one-piece, non-rigid synthetic cornea. It is composed of an outer skirt, that is an opaque, porous, high- water PHEMA (poly[2-hydroxyethyl methacrylate]), with a transparent central optic core of gel PHEMA.

Surgical Procedure

In Stage I, an intrastromal trephine is used to remove the central posterior corneal lamellae for insertion of the device. A corneal incision is made and dissection instruments are used to continue the corneal dissection throughout the circumference of the corneal graft, thereby creating an intralamellar pocket. An AlphaCor sizer, used to test the size and centration, is inserted into the intralamellar pocket followed by removal of the posterior disc via a 3.5 mm intrastromal trephine. After insertion of the device and closure of the limbal incision, the surface is often covered with a Gundersen conjunctival flap. If the Gundersen flap is inadequate

to cover the cornea an amniotic membrane graft may be required. In Stage II performed approximately 2 months after Stage I, the overlying conjunctiva created by the Gundersen flap is removed and trephination of the central 4 mm of the conjunctival flap and anterior corneal lamellae is done.

Pintucci Keratoprosthesis

The Pintucci KP can be implanted in thinned or perforated corneas, in corneas with stromal melting, and in eyes that have undergone several procedures including penetrating keratoplasty, other KP implantations, and glaucoma, cataract and vitreoretinal surgery.

Design

The supporting element of the Pintucci KP is made of a biointegrated Dacron fabric skirt that allows three-dimensional colonization by newly formed vascularized connective tissue. This fabric is soft and pliable, can be easily cut into the desired shape and sutured, and is chemically inert and not subject to resorption. The Dacron fabric support is fixed to the PMMA optical cylinder with a specific reliable method (international patent pending).

Keratoprosthesis still carries a somewhat greater burden post- operatively than standard keratoplasty. Successful outcome requires patient compliance, more frequent follow-up and more demands on physician time. However, in cases where further Keratoplasty appears futile, keratoprosthesis can be most rewarding.

Complications

Though the rate of success with Keratoprosthesis is high, in rare cases, certain serious complications could occur.

- Necrosis of tissue around the Keratoprosthesis (which if unchecked can lead to leak, infection, extrusion).
- Postoperative Uveitis –can lead to the following:
  - Retroprosthetic Membrane
  - Vitreous Opacities
  - Retinal Detachment
  - Macular Oedema
  - Epiretinal Membrane etc.
- Glaucoma – especially in Stevens Johnson Syndrome, pemphigoid, chemical burns.
- Infection – Endophthalmitis –now rare.

Glaucoma and extrusion of the implant are serious complications that could occur.

Sudden vitritis can cause a drastic reduction in vision. However, it is possible to treat this condition through antibiotics or by a minor laser surgery.

What next?

Keratoprosthesis is continuously evolving with newer generation materials that seek to improve treatment outcomes. The advances documented herein are only a small sample of the boiling point which the field of keratoprotheses has recently reached at. Current research is aimed at improving, on the one hand, the

anatomical results by using more biocompatible materials that provide better integration with the host tissue, and on the other hand, at providing optimal long-term and sustained visual acuity to our patients. However, post-operative complications remain the biggest challenge (mainly glaucoma, infection, and extrusion). Future designs will have to incorporate the use of newer materials that provide excellent optical properties, while at the same time become biointegrated with the ocular tissue. In short, the perfect keratoprothesis is yet to be discovered, although every day the goal gets closer and closer.

References

1. Mannis, M.J.; Dohlman, C.H. The Artificial Cornea: A Brief History. In *Corneal Transplantation: A History in Profiles*; Mannis, M.J., Mannis, A.A., Eds.; Hirschberg History Ophthalmology; Wayenborgh Press: Oostende, Belgium, 1999; pp. 321–335.
2. Chirila, T.V.; Hicks, C.R. The origins of the artificial cornea: Pellier de Quengsy and his contributions to the modern concept of keratoprosthesis. *Gesnerus* **1999**, *56*, 96–106.
3. Keeler, R.; Singh, A.D.; Dua, H.S. Guillaume Pellier de Quengsy: A bold eye surgeon. *Br. J. Ophthalmol.* **2014**, *98*, 576–578.
4. Franceschetti, A. Corneal grafting. *Trans. Ophthalmol. Soc. UK* **1949**, *69*, 17–35.
5. Györffy, I. Acrylic corneal implant in keratoplasty. *Am. J. Ophthalmol.* **1951**, *34*, 757–758.
6. Stone, W.J.; Herbert, E. Experimental study of plastic material and replacement of the cornea; A Preliminary report. *Am. J. Ophthalmol.* **1953**, *36*, 168–173.
7. Wünsche, G. Versuchezurtotalen Keratoplastie und zur cornea arteficialis. *ArztlicheForsch.* **1947**, *1*, 345–356.
8. Khan B, Dudenhoefer EJ, Dohlman CH. Keratoprosthesis: An update. CurrOpinOphthalmol2001;12:282-7.
9. Saeed H N, Shanbhag S, Chodosh J. The Boston keratoprosthesis. CurrOpinOphthalmol2017;28:390-6.
10. Strampelli B: Osteoodonto Keratoprosthesis, Ann Ottalmol Clin Ocul.1963; 89:1039.

YELLOW IOLs: DO WE REALLY NEED THEM?

Mohit Khattri, Shalini Mohan, Malay Chaturvedi, S.K. Sachan

We know that prolonged exposure to short wavelength light (blue light) is harmful. Also, the normal, clear crystalline lens of the eye turns yellow as we get older. Is it a natural, protective way against the blue, short wavelength light?

In the recent past, there has been an upsurge in the use and marketing of yellow tinted IOLs. What are the potential advantages and disadvantages of these IOLs? Ophthalmologists should be aware of both sides of the coin and be judicious in choice of IOL for their patients.

Potential advantages of Yellow IOLs

Yellow IOL is postulated to reduce the glare disability after IOL implantation (1). It is attributed to Rayleigh's Law, which essentially states that when shorter wavelengths are blocked, scatter is reduced, thereby reducing the glare.

Study by Yuan et. Al. (2) postulated that yellow IOLs are preferable to ordinary IOLs in preserving spatial contract sensitivity and cause lesser photophobia and cyanopsia. The blue light filtering IOLs are also more effective in protecting the Retinal Pigment Epithelium (3) and (4). Although these have been mainly in the animal studies and not supported in epidemiological studies in AMD pathogenesis. Marshall (5) advocated the protective effect of UV blocking IOLs against the proliferation of human uveal melanoma cell lines.

Potential disadvantages of Yellow IOLs

It has been postulated that blue light is beneficial in scotopic light conditions. In fact, blue light provides 35% scotopic sensitivity. This is due to the Purkinje Shift. Hence blocking of blue light might result in decreased mesopic contrast acuity and scotopic short wavelength sensitivity. Some studies also report alteration in colour perception with the use of yellow IOLs. However, these were statistically insignificant and have been reported as anecdotal (6).

Disturbed circadian rhythm is another potential drawback of the yellow IOLs. In the retinal ganglion, photosensitive cells are responsible for entrainment of the circadian clock for light-dark cycles. These photopigments, specially melanopsin and cryptochrome, are most active in the blue light spectrum upto 430 to 480nm and the concern is that these photopigments cannot be active if the blue light is blocked. Consequently, the light dark cycles get disrupted resulting in daytime sleepiness. This is because melatonin suppression reduces daytime sleepiness and 55% suppression of melatonin is due to blue light and yellow IOL blocking the blue light is postulated to increase the daytime sleepiness. Hence blockage of UV light with transmission of blue light may prove to be selectively more beneficial (7)

References

1. Hammond BR, Bernstein B, Dong J. The effect of the AcrySof Natural lens on glare disability and photostress. Am J Ophthalmol. 2009;148(2):272-276.
2. Yuan Z, Reinach P, Yuan J. Contrast sensitivity and colour vision with a yellow intraocular lens. Am J Ophthalmol. 2004;138:138-140
3. Kernt M, Neubaueryh AS, Liegl R, et al. Cytoprotective effects of a blue light filtering intraocular lens on human Retinal Pigment Epithelium by reducing phototoxic effects on vascular endothelial growth factor-a, Bax, and Bcl-2 expression. J Cataract Refract Surg. 2009;35 (2):354-362.
4. Nolan JM, O'Reilly P, Loughman J, et al. Augmentation of macular pigment following implantation of blue light filtering IOLs. Invest Ophthalmol Vis Sci. 2009;50(10):4777-4785.
5. Marshall JC, Gordon KD, Mc Cauley CS, et al. The effect of blue light exposure and use of intraocular lenses on human uveal melanoma cell lines. Melanoma Res. 2006;16(6):537-541.
6. Wirtitsch MG, Schmidinger G, Prskavec M, et al. Influence of blue light filtering IOL on colour perception and contrast sensitivity. Ophthalmology. 2009;116:39-45.
7. Asplund R, Lindblad BE, Sleep and sleepiness 1 and 9 months after cataract surgery. Arch Gerontol Geriatr.2004;38:69-75.

MEIBOMIAN GLAND DYSFUNCTION AND HYPERCHOLESTEROLEMIA- A REVIEW ARTICLE

*Priyanshi Awasthi, *Rohit Shahi, *O.P.S.Maurya, **S.K.Singh

What is meibomian gland dysfunction?

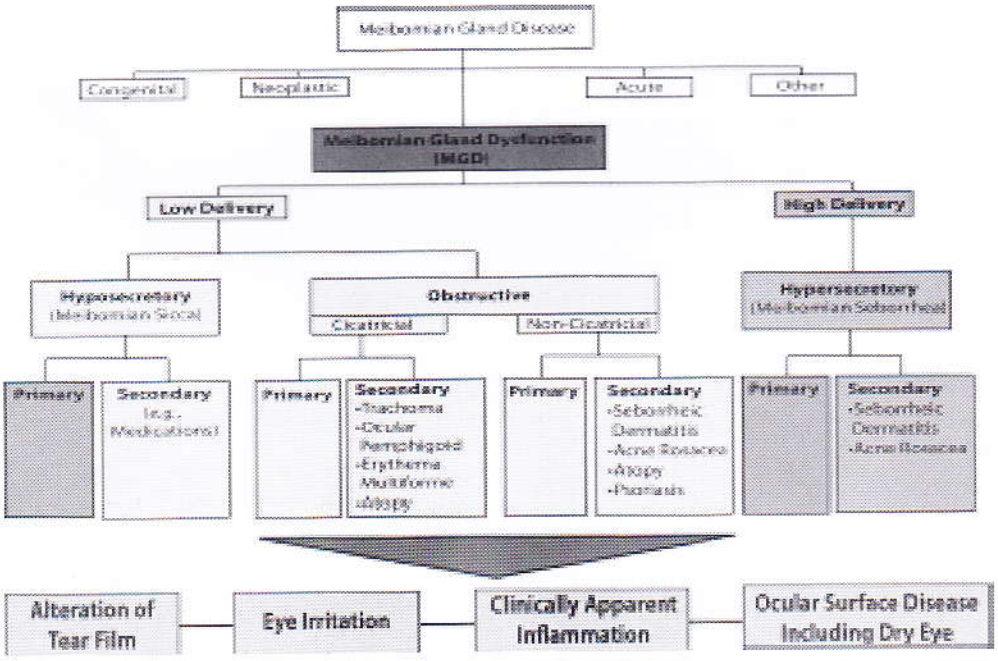
Meibomian gland dysfunction (MGD) is a chronic, diffuse abnormality of the meibomian glands, commonly characterized by terminal duct obstruction and/or qualitative/ quantitative changes in the glandular secretion. It may result in alteration of the tear film, symptoms of eye irritation, clinically apparent inflammation, and ocular surface disease.

Dysfunction-function of meibomian gland is disturbed. Diffuse-most of the glands involved. Obstruction of meibomian gland orifices and terminal ducts and qualitative or quantitative changes in gland secretion is the most prominent aspect of MGD. Subjective symptoms of eye irritation are included in the definition.

Meibomian gland disease is used to describe a broader range of meibomian gland disorders like neoplasia and congenital disease. Other terms such as meibomitis or meibomianitis describe a subset of disorders of MGD associated with inflammation of the meibomian glands. Although inflammation may be important in the classification and in the therapy of MGD, these are not sufficiently general, as inflammation is not always present.

Hypercholesterolemia (total cholesterol _200 mg/dl) is a significant risk factor for ischemic heart, cerebrovascular, and peripheral vascular disease. Increased cholesterol in the glandular secretion has been postulated to be necessary for the development of meibomian gland dysfunction (MGD), a common form of chronic blepharitis.

CLASSIFICATION OF MGD (TABLE 1)[16]



Department of Ophthalmology, Institute of Medical Sciences, BHU
Department of Endocrinology, Institute of Medical Sciences, BHU

Epidemiologic studies have firmly established abnormal lipid levels as significant risk factors for cardiovascular disease [1-3] and stroke [4-5]—some of the leading causes of mortality in the developed world [6-7].

Emerging studies [8] have linked increased cholesterol esters in meibomian secretions to patients with meibomian gland dysfunction (MGD). Meibum with higher cholesterol composition has a higher melting point [9], which is postulated to result in more viscous secretions that may then obstruct meibomian glands or alter the quality of posterior eyelid excreta. Furthermore, patients with moderate to severe MGD seem to have a higher prevalence of abnormal serumcholesterol levels versus the general public [10-12]. MGD is a common cause of ocular surface disease,[13-14] yet its impact on patients' overall health is often overlooked [15]. Eye care providers may be the first to detect systemic diseases such as cerebrovascular disease because of their initial ocular manifestations (e.g., amaurosis fugax & retinal vascular occlusion). Retinal vascular occlusions are just one example of how the ophthalmic exam could providemclues about the presence of systemic disease. A known risk factor for cardiovascular illness is dyslipidemia. This term encompasses several abnormalities in the serum lipid profile such as a total cholesterol C200 mg/dL, triglycerides C150 mg/dL, low-density lipoprotein (LDL) C130 mg/dL, or high-density lipoprotein (HDL) B40 mg/dL [10-12,15].

Pathophysiology of MGD

Meibomian gland dysfunction is caused primarily by terminal duct obstruction with thickened opaque meibum containing keratinized cell material. The obstruction, in turn, is due to hyperkeratinization of the ductal epithelium and increased meibum viscosity. The obstructive process is influenced by endogenous factors, such as age, sex, and hormonal disturbances, as well as by exogenous factors such as topical medication. Meibomian gland dysfunction is caused primarily by terminal duct obstruction with thickened opaque meibum containing keratinized cell material. The obstruction, in turn, is due to hyperkeratinization of the ductal epithelium and increased meibum viscosity. The obstructive process is influenced by endogenous factors, such as age, sex, and hormonal disturbances, as well as by exogenous factors such as topical medication. The obstruction may lead to intraglandular cystic dilatation, meibocyte atrophy, gland dropout, and low secretion, effects that do not typically involve inflammatory cells. The outcome of MGD is a reduced availability of meibum to the lid margin and tear film. The consequence of insufficient lipids may be increased evaporation, hyperosmolarity and instability of the tear film, increased bacterial growth on the lid margin, evaporative dry eye, and ocular surface inflammation and damage.

Table 2: Population based study providing estimates of prevalence of mgd[16]

Study	Participants	Ethnicity	Parameter	Prevalence (%)	Age (y)
Beijing Eye Study	1957	Mainland Chinese	Telangiectasia (asymptomatic) Telangiectasia (symptomatic of dry eye)	68 69.3	>40
Japanese study	113 pensioners	Japanese	Gland dropout, expressibility and nature of meibum secretion	61.9	>60
Shihpai Eye Study	1361	Taiwanese Chinese	Telangiectasia or meibomian gland orifice plugging	60.8	>65
Melbourne Visual Impairment Project	926	Caucasian	Tear break up time <1 SD (10 s) Tear break up time <1.5 SD (8 s)	19.9 8.6	40–97
Salisbury Eye Evaluation	2482	Caucasian	Meibomian gland plugging or collarettes (grades 2 and 3)	3.5	>65

Correlation with Altered Lipid Profile:

Patients with moderate to severe mgd have increased levels of total cholesterol compared to population controls. Increased cholesterol at the glandular secretion level has been studied and hypothesized to be a factor in meibomian gland dysfunction.[17,20] Because normal whole meibomian lipids have a melting point of 30°C to 34°C[18] and cholesterol has a melting point of 148°C,[18] increased concentration of cholesterol in meibomian lipid would increase the melting point of the meibomian lipid milieu, theoretically increasing viscosity and leading to plugging of the meibomian glands.[17] The composition of normal meibum secretions has been well studied and cholesterol content has been reported to be 1% to 2%[19] however, to our knowledge, meibum composition in patients with meibomian gland dysfunction has not been studied.

One study attempted to associate serum cholesterol and human tear fluid and found that cholesterol concentration in the tear film bore no correlation to serum cholesterol.[21] However, this research measured the concentration of cholesterol in the aggregate tear film. It did not measure this concentration in the lipid layer, in the meibum, or in the meibomian gland. Altered meibomian lipid concentration, including an increase in meibum cholesterol, has been shown to cause dry eye symptoms. In another study, patients on antiandrogen therapy were found to have an altered meibomian lipid composition with a notable increase in the quantity of cholesterol.[20] These patients experienced a subjective increase in dry eye symptoms, including light sensitivity, painful eyes, and blurred vision. Under biomicroscopy, the patients were found to have an increase in tear film debris, the presence of an abnormal tear film meniscus, irregular posterior lid margins, increased vital dye stain, conjunctival injection, and a significantly decreased tear break-up time, all signs of dry eye disease.[20] Furthermore, it has been shown that patients with Sjögren syndrome have increased cholesterol content in their tear film.

More studies, both prospective and at the basic science level, will be needed to examine the significance of this finding.

Stage	MGD Grade	Symptoms	Corneal Staining
1	+ (minimally altered expressibility and secretion quality)	None	None
2	++ (mildly altered expressibility and secretion quality)	Minimal to mild	None to limited
3	+++ (moderately altered expressibility and secretion quality)	Moderate	Mild to moderate; mainly peripheral
4	++++ (severely altered expressibility and secretion quality)	Marked	Marked; central in addition
"Plus" disease	Co-existing or accompanying disorders of the ocular surface and/or eyelids		

Diagnosis of MGD

Clinical examination:

1. Assessment of blink rate and intrtblink interval: normal is 14-18 timesper minute.

2. Examination of eyelid and its margfin:compromised eyelid margin like tylosis,entropion,ectropion,trichiasis,dydtichiasis can predispose to mgd.also surrounding skin features of rosacea like telangiectasia is an important precursor of mgd.the meibomian gland duct orifices are assessed whether open or capped.

3. Assessment of Tear Film: Normal meniscus height is more than 0.25mm.

4.Tear film break up time: On fluorescein staining normal time taken for tear film to break is more than 10 seconds. TBUT of >10 sec is suggestive of dry eye. The stability is largely dependent on lipid layer that is secreted by meibomian glands but it can also be altered in aqueous deficient dry eye. Therefore TBUT cannot differentiate between MGD AND DED .

5. Ocular Surface Staining: Cornea best assessed with fluorescein and conjunctiva is best assessed with lissamine green.the staining pattern has been given a clinical score that is useful for diagnosis and follow up management. Most commonly used scoring system is NEI(national eye institute)grading system.

6. Meibomian gland expression: Expressibiliy is assessed along with nature of meibum.

Investigations

These can be divided into-

1. Routine tests

2. Specialized tests

Routine Tests: These are done in a specific order to minimize the extent to which one test may influence the other.the recommended sequence is as follows:

Symptom questionnaire

Blink rate and blink interval-blink rate is number of blinks in one minute

Blink interval is calculated by 60/blink rate

Tear meniscus height: can be calculated with or without fluorescein instillation. Normal is between 0.2-0.3mm(1,2). TMH is measured as the distance between the darker edge of the lower eyelid and the top of reflex from tear strip.

Tear film osmolarity: Osmolarity is graded within ranges of mOsmls/l: normal-(275-300), mild-(303-310), moderate(320-335) and severe (350)

Instillation of fluorescein and measurement of TBUT and Ocular protection index: TBUT is defined as interval between last blink and the appearance of first randomly appearing black spot. It is affected in lipid layer deficiency .So it is a good indicator of dry eye in mgd even though it may be affected in aqueous deficiency dry eye. A TBUT of <10 seconds is considered abnormal.

Grading of corneal and conjunctival fluorescein staining: Various grading systems have been proposed like NEI(national eye institute)grading and Bijstervald grading. It has been reported that staining along upper and lower lid margins is more likely to be associated with MGD or some form of blephritis and central staining is more associated with aqueous deficient dry eye.[2]

Lid examination and expression

Meibography: This technique is developed solely for directly observing the morphology of meibomian glands in vivo.

Schirmer's test: This should be the final test to be done as it may affect other staining tests. A score of <5mm in 5 minutes is an indicator of severe dry eye.

Specialized Tests

Interferometry: This technique is used to analyse lipid layer. The most recent invention is Lipiview interferometer which uses white light interferometry to form a pattern that is termed interferogram.

Invivo confocal microscopy (ICVM): It is a contact procedure that has been evaluated for meibomian gland examination. It can be used to assess acinal density and diameter, secretion reflectivity and peri glandular inflammation in patients with MGD. It can be also be used to make the resistant demodex mites in the meibomian gland orifices.

Management of MGD

Management of MGD is based on the staging of the disease.

Stage 1

Inform patient about MGD, the potential impact of diet, and the effect of work/home environments on tear evaporation, and the possible drying effect of certain systemic medications. *Consider* eyelid hygiene including warming/expression.

Stage 2

Advise patient on improving ambient humidity; optimizing workstations and increasing dietary omega-3 fatty acids intake.

Institute eyelid hygiene with eyelid warming (a minimum of four minutes at 40-45 degrees celsius, once or twice daily) followed by moderate to firm massage and expression of MG secretions.

'Lipiflow' treatment- warms the internal surface of both lids and massages the lids.

Artificial lubricants, topical azithromycin.

Stage 3

Oral tetracycline derivatives should be considered along with above mentioned methods.

Stage 4

Anti-inflammatory therapy for dry eye along with above methods like - Cyclosporine A (has an immunomodulating effect on T lymphocytes) 5% N-acetylcysteine (has mucolytic, anti-collagenolytic, and antioxidant properties).

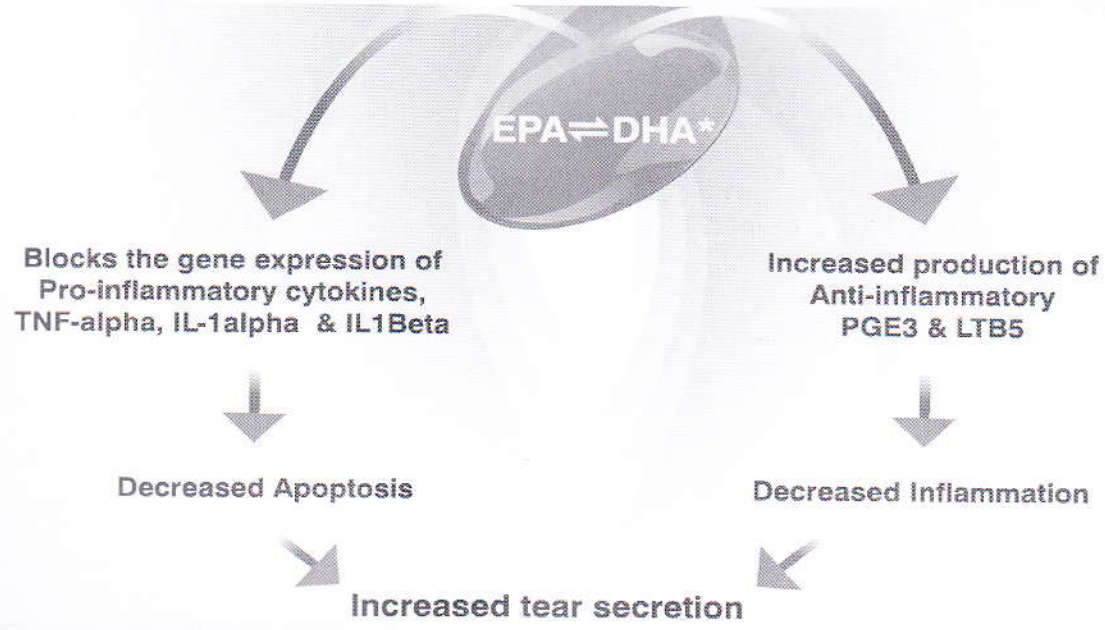
Steroids are used only for acute exacerbations or topically for the treatment of marginal hypersensitivity keratitis.

Probing mechanically opens and dilates the orifices and ducts of the meibomian glands, provided scarring has not caused irreversible damage. This facilitates a free flow of meibum.

Role of tetracycline:

1. Suppresses migration of leucocytes and inflammatory cytokines
2. Reduces production of nitric oxide and reactive oxygen species which play a role in the build up of inflammation.
3. Inhibition of matrix metalloproteinase .
4. Inhibition of phospholipase A2 which in turn inhibits bacterial endotoxins.
5. Reduces levels of irritative fatty acids and diglycerides by suppressing bacterial lipases.
6. Antimicrobial activity leads to reduction in number of vital bacteria.
7. Inhibition of keratinization

Role of omega 3 fatty acids:



References

1. Muntner P, Colantonio LD, Cushman M, Goff DC Jr, Howard G, Howard VJ, Kissela B, Levitan EB, Lloyd-Jones DM, Safford MM (2014) Validation of the atherosclerotic cardiovascular disease Pooled Cohort risk equations. JAMA 311(14):1406–1415

2. Wilson PW, D'Agostino RB, Levy D et al (1998) Prediction of coronary heart disease using risk factor categories. Circulation 97:1837–1847

3. Expert Panel on Detection (2001) Evaluation, and treatment of high blood cholesterol in adults. Executive summary of the third report of the National Cholesterol Education Program

(NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA 285:2486–2497

4. Mok V, Kim JS (2015) Prevention and management of cerebral small vessel disease. J Stroke 17(2):111–122

5. Larsson SC,A ° kesson A, Wolk A (2015) Primary prevention of stroke by a healthy lifestyle in a high-risk group. Neurology 84(22):2224–2228

6. Mathers CD, Boerma T (2009) Ma Fat D. Global and regional causes of death. Br Med Bull 92:7–32

7. Mathers CD, Ma Fat D, Inoue M, Rao C, Lopez AD (2005) Counting the dead and what they died from: an assessment of the global status of cause of death data. Bull World

Health Organ 83:171–177 Int Ophthalmol (2016) 36:469–476 475

8. Shine WE, McCulley JP (1991) The role of cholesterol in chronic blepharitis. InvestOphthalmolVis Sci 32:2272–2280

9. Shine WE, McCulley JP (2003) Polar lipids in human meibomian gland secretions. Curr Eye Res 26:89–94

10. Dao AH, Spindle JD, Harp BA et al (2010) Association of dyslipidemia in moderate to severe meibomian gland dysfunction. Am J Ophthalmol 150:371–375

11. Pinna A, Blasetti F, Zinellu A, Carru C, Solinas G (2013) Meibomian gland dysfunction and hypercholesterolemia.Ophthalmology 120(12):2385–2389

12. Bukhari AA (2013) Associations between the grade of meibomian gland dysfunction and dyslipidemia. Ophthal Plast Reconstr Surg 29(2):101–103

13. Bron AJ, Tomlinson A, Foulks GN, Pepose JS, Baudouin C, Geerling G, Nichols KK, Lemp MA (2014) Rethinking dry eye disease: a perspective on clinical implications. Ocul Surf 12(2 Suppl):S1–S31

14. Rege A, Kulkarni V, Puthran N, Khandgave T (2013) A clinical study of subtype-based prevalence of dry eye. J Clin Diagn Res. 7(10):2207–2210

15. Pinna A, Piccinini P, Carta F (2007) Effect of oral linoleic and gamma-linolenic acid on meibomian gland dysfunction. Cornea 26:260–264

The International Workshop on Meibomian Gland Dysfunction: Executive SummaryKelly K. 16 Nichols,1 Gary N. Foulks,2 Anthony J. Bron,3 Ben J. Glasgow,4,5 Murat Dogru,6Kazuo Tsubota,6 Michael A. Lemp,7 and David A. Sullivan8

17 Driver PJ, Lemp MA. Major review: meibomian gland dysfunction. Surv Ophthalmol 1996;40(5):343.

18. Butovich IA, Millar TJ, Ham BM. Understanding and analyzing meibomian lipids—a review. Curr Eye Res 2008; 33(5):405– 420.

19. Butovich IA. The meibomian puzzle: combining pieces together. Prog Retin Eye Res 2009;28(6):483–498.

20. Krenzer KL, Dana MR, Ullman MD, et al. Effect of androgen deficiency on the human meibomian gland and ocular surface. Clin Endocrinol Metab 2000;85(12):4874 – 4882.

21. Morozova RP, Zhaboiedov HD, Kirieiev VV, Nikolenko IA. The normal tear fluid and decreased tearing in patients with Sjogren's disease and Sjogren's syndrome II. The lipid content. Ukr Biokhim Zh 1996;68(2):82–87.

AN INSIGHT INTO LOW VISION AIDS

-Jayati Pandey, R.N Kushawaha, R.C Gupta, Parul Singh, Dr. Anuj Kushawaha

In India according to Person with Disability Act, 1995 as well as under the National Programme for Control of Blindness (NPCB), Blindness refers to condition where person suffers from any of the following condition namely:

- Total absence of sight ; or
- Visual acuity not exceeding 6/60 or 20/200(Snellen) in better eye even with correction lenses; or
- Limitation of field of vision subtending angle of 20 degree or worse.[1]

The Persons with Disabilities Act, 1995 also recognizes low vision as a category of disability and defines it as follows:

"Person with low vision" means a person with impairment of visual functioning even after treatment or standard refractive correction but who uses or is potentially capable of using vision for the planning or execution of a task with appropriate assistive device".[2]

The WHO working definition of Low Vision (WHO, 1992) is as follows:

"A person with low vision is one who has impairment of visual functioning even after treatment, and/ or standard refractive correction, and has a visual acuity of less than 6/18 to light perception or a visual field of less than 10 degrees from the point of fixation, but who uses, or is potentially able to use vision for the planning and/or execution of a task".[3]

The points emphasized are that in a significantly reduced vision, visual performance is affected but that there still is vision that can be used. This last point is very important: if there is usable vision, training to use that vision might be possible. In addition, this person is not labelled blind.

The estimated number of people visually impaired in the world is 285 million, 39 million blind and 246 million having low vision; 65 % of people visually impaired and 82% of all blind are 50 years and older.[4]

India projects a higher number of blind people at international forums because of its definition. India currently has around 8 million blind people against 39 million globally -- which makes India second highest in world's blind population.[5]

People with low vision can remain independent and continue to lead fulfilling lives with the help of low vision aids (LVA). Some low vision aids work by making things bigger and brighter while others work by allowing patients to rely more on audio and hearing. People with low vision can also reorganize their environment at work and at home to make daily life easier for them.

Many Low vision aids are available which can be prescribed to the patients according to the patient's visual status, mental status, need, occupation and working and residential environment.

Optical LVA for distant viewing such as hand-held/spectacle mounted telescopes, spectacle model telescope, and bioptic telescope can be used for distant tasks such as recognizing faces, reading blackboard, street signs, recognizing vehicles etc. However spherical aberrations, reduced field of vision, decrease depth perception and cost can be limiting factor.

GSVM Medical College Kanpur

Bioptic Telescope

SEE TV (Spectacle Model Telescope)

CONTRAST ENHANCEMENT

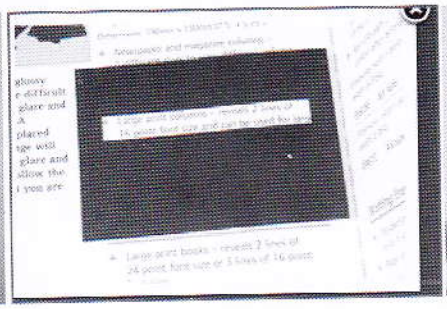
Poor Contrast

Better Contrast

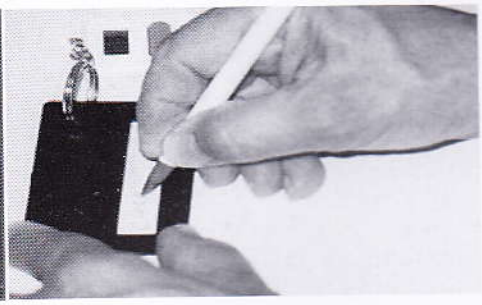
Optical LVA for near vision such as bifocal spectacles, hand-held/stand magnifiers, notex (for currency identification) make daily household activities such as reading easier for patients. Non-optical aids such as approach magnification, lighting, contrast enhancement (typoscope) are other devices used for reading. Letter writers, fibre-tipped pens and signature guides can be used to write and sign in straight line. Hand held magnifiers are cumbersome, as they are not hands free like their counterparts, making it difficult for patients with tremors and while doing tasks that require use of hands, like writing, etc.

HAND-HELD MAGNIFIER

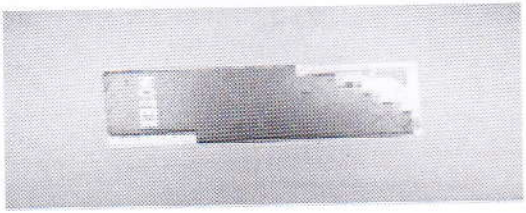
TYPOSCOPE



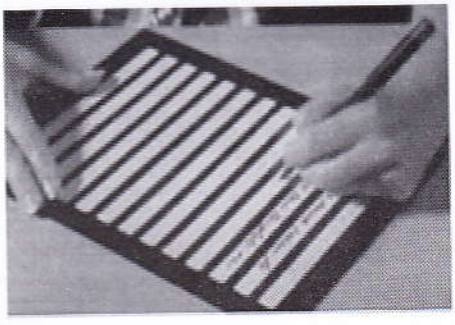
SIGNATURE GUIDE



Notex - Currency Identification

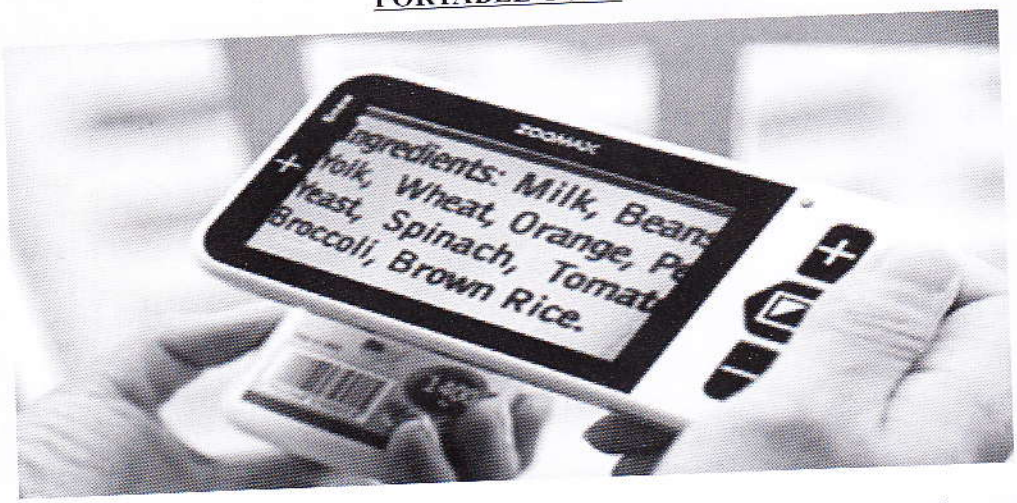


Letter Writer - to write in straight line



Recent advances in digital image processing provide promising methods for maximizing the residual vision of the visually impaired. To name a few devices such as talking watches, GPS, e-readers, computer software (JAWS, MAGIC), smartphones and tablets have made the world easy for them. Smartphones and tablets offer a range of apps and built-in functions to help people with low vision: iRead, iLoupe, Tap Magnify, iCan See and iMagnify use the device's camera and light source to magnify and illuminate text. [6] Electronic video magnifiers like closed-circuit TVs (CCTVs) provide distortion free viewing, a large field of view and a very large range of magnification levels.

PORTABLE CCTV



The approach in managing a patient with low vision should not be that 'Nothing can be done for your disease', but rather, 'since not much can be done medically or surgically for your disease, you will be evaluated for enhancement in the vision with the help of visual aids'.

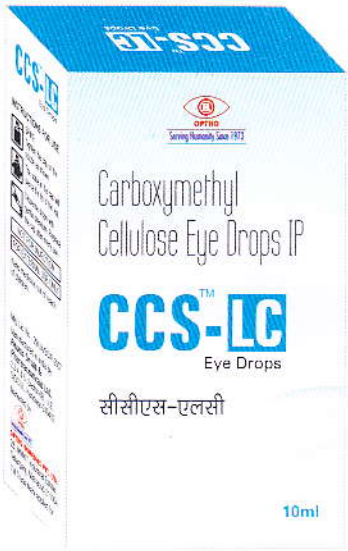
REFERENCES

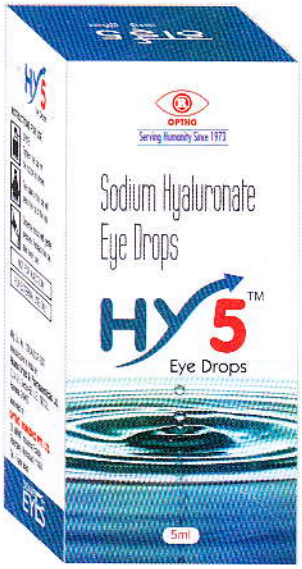
1. Bachani, (Dr.) D.; and Limburg, (Dr.) H. (1996): National Programme for Control of Blindness - Course Material for Training District Programme Management, New Delhi: Ophthalmic Section, Directorate General of Health Services, Ministry of Health & Family Welfare, P. 169
2. The Persons with Disabilities (Equal Opportunities, Protection of Rights and Full Participation) Act.1995. Available from: http://newsonair.nic.in/PWD_Act.pdf
3. World Health Organisation International Statistical Classification of Diseases and Related Health Problems 10th revision Current version Version for 2003 Chapter VII H54 Blindness and low vision. http://www.who.int/classification/icd/en
4. World Health Organization, Global Data on Visual Impairments 2010, 2012
5. Vision 2020 India News Letter September 2007. Article on Internet.www.vision2020india.org
6. Sundaram Natarajan, (Dr.): Low vision aids-a boon. Indian J Ophthalmol. 2013 May; 61(5): 191–192.

Size: 3m x 2.5m


Cipla
Eye Drops
Flogel Ultra
Polyethylene Glycol 400 0.4%, Propylene Glycol 0.3%
Smart working Gel
Eye hydration at work
• Forms a protective gel matrix and restores ocular surface[1]
• Greater lubricity compared to other artificial tears[2]
• Extended relief in dry eye symptoms[3]
Cipla
Polyethylene Glycol 400 And Propylene Glycol Eye Drops
Flogel Ultra
Eye Drops

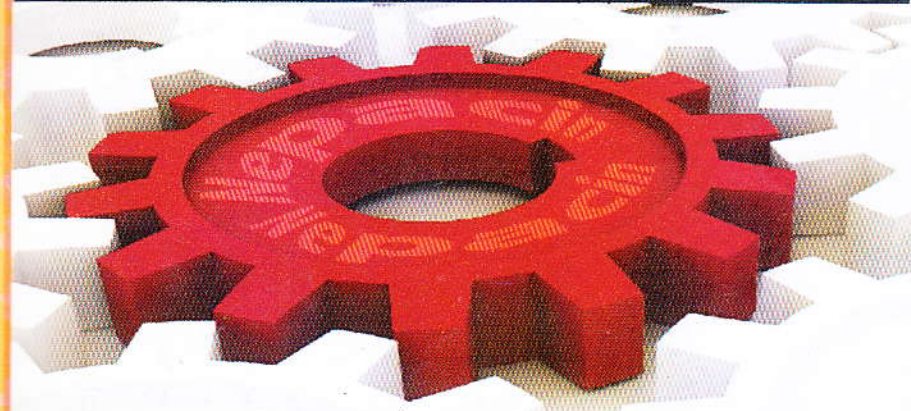
With Best Compliments From Optho Remedies





In ocular pain & inflammation

Augentus Pharma
A Division of Optho Remedies



OUTSTANDING *Performance*

Nepacin

Eye Drops

Nepafenac	0.1% w/v
Benzalkonium Chloride Sol.	0.01% v/v
Sterile Aqueous Vehicle	q.s.

Reversing the Damage of Dry Eyes in a Soothing way.....

HYTAK

EYE DROPS

Sodium Hyaluronate	B.P.	1.8 mg
Sodium Perborate	B.P.	0.028% w/v
As Preservative		

THERAPEUTIC INDICATION

Dry eye and ocular surface damage, due to diseases such as:

Superficial Keratitis
Sjogren's Syndrome
Primary Dry Eye Syndrome
Post Eye Surgery- Cataract Removal or Lasik.

PHARMTAK
Ophthalmics (I) Pvt. Ltd.

50-52, The Discovery,Borivali (E),
Mumbai – 400066 (Maharashtra)
EMAIL ID: info.pharmtak@gmail.com
Visit Us @ www.pharmtakindia.com
Toll-Free: 1800-532-9700