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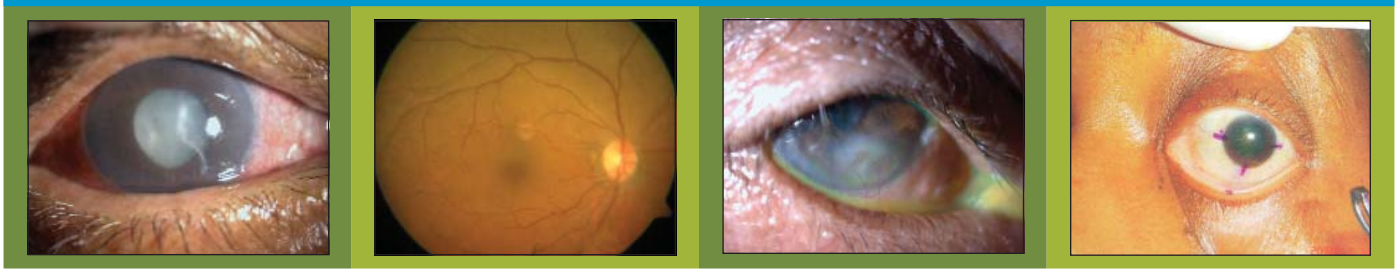

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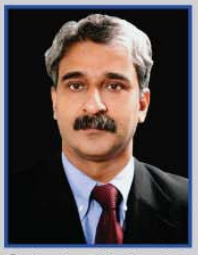

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Editorial

My Dear Friends and Colleagues,

Welcome to the DOS midterm conference on 27th and 28th nov at India Habitat Centre, and the DOST programme on 26th nov at Army R&R institute.

Live Surgery At DOST

26 th is a day of live surgery on all Ophthalmology except Phaco and Refractives. The mentors and the masters, will all be there, showing and teaching their techniques and sharing their secrets. It is a day of true blue Ophthalmology and all hard core Ophthalmologists are welcome to attend. Even though the course is primarily focussed on Residents, but all lovers of ***"Sport Ophthalmology"*** are invited to play! And as an additional incentive, the attendees will enjoy a dinner with the teachers and peers. Hard work through out the day and some well earned relaxed evening! The curtain raiser for the conference.

The Mid Term

27th and 28th are the two main conference days. The first day as ordained and expected, will be devoted and dedicated to Phaco. Our servant and our master! Our scotch and our bread! Live surgery by the maestro's. It is almost like a rock show! or is it BigBoss! It will sure have its heartstopping moments and everyone is assured a rollicking good time. Nothin beats our very own reality show!

28th is the day of serious teaching and learning. ***"Techniques and Innovations"*** are our two buzz words and we wish to focus on them. On our cross hairs and for your benefit. New Ideas and Innovative techniques. The latest and the best Ophthamic epicurian recipies served piping hot.

Fellowship

And the evening of 27th. Relaxed and entertaining, Dinner and Fellowship and a wonderful november airconditioned weather specially arranged for you, with our complements.

Skill Transfer Workshops

Recently we have concluded a highly satisfying and extremely well attended skill transfer workshop on squint at Dr R P Eye Centre. The highlight of this workshop was the live WebCasting. Even today it is available on our Website for your perusal, if you dezire.

The next skill transfer will be on Oculoplasty. And it will be at GNEC on 19th dec. All DOS members are invited to take advantage of this knowledge sharing and enhance their Ophthalmic Skills.

See you in the midterm.

Yours Truly.

Thanking you,

Dr Amit Khosla
Secretary,
Delhi Ophthalmological Society

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MIDTERM CONFERENCE

Delhi Ophthalmological Society

PHTHALMIC

Techniques and Innovations

27th & 28th November, 2010
India Habitat Centre, Lodhi Road,
New Delhi

www.dosonline.org

Non Healing Corneal Ulcer

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Very often, we find our selves in a position when we are unable to treat a patient of corneal. Inspite of use of the best available antibiotics and combination 'cocktail' therapy the ulcer seems to increase in severity making us helpless. Often such patients are then referred to tertiary care eye centers for opinions of cornea experts. So let us learn from some of the well known cornea experts how a case of Non-Healing corneal ulcer is approached. Let us learn a few tips and tricks which will help understand the pathology and its appropriate management strategy.

We may start our discussion with the fact that all patients of corneal ulcers must undergo detailed evaluation with clinical history, study of predisposing risk factors, slit lamp and detailed ocular examination. All patients of corneal ulcer should undergo corneal scraping of the ulcer edges and base for Gram stain, KOH wet mount, bacterial and fungal cultures. Approach to patients may be tailored as per clinical conditions and experience of the treating ophthalmologist.

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Dr. Prakashchand Agarwal (PA): MD, FRCS, Assistant Professor, People College of Medical Sciences and Research Centre, Bhopal, Madhya Pradesh.

PA: How would you define a "non healing corneal ulcer"?

NVP: When the ulcer does not show any signs of improvement or shows worsening despite appropriate and adequate therapy, I would label it as a non healing ulcer.

NS: An ulcer not responding to conventional medical therapy should be labeled as a "non healing corneal ulcer".

UM: A non-healing ulcer is a corneal ulceration that is not responding to anti-microbial treatment for a period of three to four weeks.

PP: While the definition of a 'Non-healing corneal ulcer' and

RA I would consider the corneal ulcer to be non healing if despite the appropriate treatment based on microbiological investigations the ulcer size increases over ensuing 48 hrs, esp. the maximum size in two dimensions or there is fresh appearance of hypopyon or increase in the size of existing hypopyon. Important to differentiate between non infective and infective pathology.

PA: After how many days of treatment would you call an ulcer as "non healing"?

NVP: It really depends on the nature of the virulence of the infecting organism. For example, drug resistant pseudomonas can cause a rapidly progressive ulcer (non healing) in a matter of days, while organisms such as Nocardia can linger on for days without healing.

NS: In cases of bacterial keratitis, the ulcer should be labeled as "non healing" if it fails to stabilize or show improvement after 48 to 72 hours of effective antimicrobial therapy and in cases of fungal keratitis if it fails to respond in 5-7 days.

UM: An ulcer not responding to treatment for a period of three to four weeks would be classified as 'non-healing'.

PP: Is contained in the phrase itself, what remains ambiguous is the time period before which a corneal ulcer can be labeled 'non-healing'. Generally, if a corneal ulcer does not respond to conventional treatment within 4 to 6 weeks, it is termed 'non-healing' even though this period is arbitrarily chosen.

RA: It is important to follow the ulcer closely to begin with. Initially, even no further progression in the ulcer dimensions itself highlights responsiveness of ulcer to treatment. Any increase in dimensions, non localizing of margins of ulcer, fuzzy ill-defined borders, increase in slough are the indicators for non responsive ulcer to treatment. When the patient is followed on daily basis, worsening of above parameters in 48-72 hrs indicate ulcer not responding to the treatment instituted and need for revised diagnosis or revised medication.

PA: What various microbiological media are used to send corneal scrapings in such a case?

NVP: The first step is to re-culture the ulcer using standard microbiological media (Blood agar, Potato dextrose agar) and to reconfirm/identify the original diagnosis. For example, fungal corneal ulcers are known to get contaminated with Pseudomonas, when treated with Natamycin suspension. If no organism is isolated, then enriched media such as the following are used.

I. Chocolate agar, thioglycolate broth-(for isolating fastidious bacteria)

II. Sabourauds dextrose agar and Brain heart infusion broth (for fungi and Nocardia.)

III. Nonnutrient agar with E Coli overlay is especially useful if there is a high suspicion for Acanthamoeba.

NS: Apart from a routine Gram staining and a KOH wet mount preparation, special stains include the use of Ziehl-Neelsen acid-fast stain for identification of suspected Mycobacteria, Actinomycetes, or Nocardia. The culture examination should include the chocolate agar for the growth of Hemophilus, Neisseria and Moraxella, Lowenstein Jensen media for suspected mycobacteria, non nutrient agar with E.Coli overlay for the isolation of Acanthamoeba organisms in addition to the blood agar and Sabouraud's agar done routinely in cases with corneal ulcer. Pre-reduced Anaerobically Sterilized Media (PRAS) is an ideal medium for isolation of the anaerobic bacteria.

UM: A case of non-healing ulcer may not always be due to an infecting agent that can be cultured. Before resorting to a microbiological work-up, the following need to be done:

1. Take a careful, detailed history. Information on immunodeficiency status, previous infections and contact lens use, may all provide diagnostic clues to the causative infectious agent.

2. Test corneal sensation. Before you place any drops in the eye, be sure to check corneal sensation. Oftentimes, decreased corneal sensation may indicate an underlying neurotrophic ulcer or herpetic ulcer.

3. Examine the ocular adnexae. Look for sac infections and lid abnormalities like entropion and lagophthalmos Look carefully at the fornix and evert the upper lid to look for a foreign body underneath the lid?

Ulcers with infiltration should be cultured. The infiltration should be subjected to Gram's stain, Giemsa stain and KOH 10% mount routinely. ZN stain for mycobacterium and nocardia, may be indicated in a non-healing ulcers. Acanthamoeba is better seen with calcoflour white stain which requires a fluorescent microscope, but can be picked up on KOH mount too. Microscopy using Gram's staining method and potassium hydroxide (KOH) preparation is simple and quick to perform and often gives useful information.

The material is also inoculated on various solid and liquid media that facilitate the growth of bacteria, fungi, and canthamoeba. These include fresh blood agar, chocolate agar, Saburaud's dextrose agar (SDA), non-nutrient agar with an overlay of Escherichia coli, thioglycolate broth and brain heart infusion broth. LJ slope inoculation may be indicated when suspecting mycobacterium infection.

RA: It is preferable to use blood agar, nutrient agar, chocolate agar, Mcconkey medium for suspected bacterial corneal ulcers, SDA for suspected fungal ulcers, e. coli laden blood agar for suspected acanthamoeba cases.

PA: What according to your experience has been the organism identified in most cases on detailed investigations?

NVP: Usually, it is fungal keratitis especially those caused by Aspergillus species, resistant to conventional antifungal therapy .Secondary bacterial infection especially with Pseudomonas in cases of fungal ulcers is a likely cause too.

NS: Various unusual and emerging microorganisms like Nocardia species, Actinomyces species, Microspodia, mycobacteria have been identified in many of the cases being managed as non healing infectious keratitis.

UM: In my experience, the leading cause of non-healing ulcers is neurotrophic ulceration, commonly secondary to herpetic infection.

RA: Commonly identified organisms have been S. aureus, coagulse negative staph, pnemococcus, aspergillus, curvularia.

PA: Can you give few atypical organisms one should look for in such cases?

NVP: Atypical mycobacteria, acanthamoeba, nocardia, mixed fungal and bacteria infections

NS: Mycobacteria, Nocardia species, Actinomyces species, Listeria monocytogenes, anaerobic bacteria like Clostridium species, protozoan infections like microspodiosis.

UM: Atypical presentations may be seen in herpetic disease, where the diagnosis is essentially clinical. Viral cultures and PCR studies may be ordered, if available. The other uncommon causes are Mycobacterium, Nocardia and Acanthamoeba in our scenario. Microsporidia is emerging as a new organism causing keratitis from some parts of the country.

PP: In addition to the routine stains (i.e. Gram's stain, KOH/ Calcoflour) the other special staining techniques that are useful in these cases include:

a) Zeihl-Neelson's stain for Acid-fast-bacteria (Mycobacteria)

b) Modified Zeihl-Neelson's stain for Nocardia and Microsporidia.

c) Immunoflorescent stain for detection of viruses.

Special culture media like the L-J medium or Bactec medium are useful for the isolation of Mycobacterium tuberculosis.

Corneal scraping specimen can also be inoculated into tissue culture cell lines for isolation of viruses like Adenovirus, HSV and VZV.

PCR based DNA sequencing tests are now possible for detection of bacteria or fungi that are difficult to culture as also for the detection of viruses. These molecular techniques have proved invaluable in the detection of Acanthamoeba, Microsporidia and other fastidious organisms.

RA: Nocardia, atypical mycobacteria, acanthamoeba, acetinobacter

PA: Which ocular investigations should be done in cases of non healing ulcer?

NVP: Repeat scraping for microbiological diagnosis

I. Reexamine the ocular adnexa to rule out dacryocystitis(to perform syringing),

II. Rule out ocular surface disease,

III. Check corneal sensations

IV. Corneal biopsy is indicated in deep stromal keratitis unresponsive to therapy.

VI. Anterior chamber tap for PCR analysis, if facilities are available..

NS: A complete microbiological work up including the use of special stains and culture on special media should be done in these cases. Herpes simplex keratitis is relatively underdiagnosed in our country. It is important to note corneal sensation to rule out viral keratitis. Mixed or polymicrobial infections must also be considered in the differential diagnosis of non healing corneal ulcer. Additionally, the cases should be investigated for dry eye and definite measures should be taken for the management of the same.

UM: Apart from the microbiological investigations, corneal sensations should be noted, examination after everting the lids and syringing of the sac should be performed in all cases. Corneal biopsy may be indicated where routine cultures have not yielded any organism and where the infiltration is deep.

PP: Investigations in case of non-healing ulcer could.

RA: Look for associated dry eye, check for raised IOP, entropion, trichiatic cilia, foreign body, coexistent use of topical steroids, presence of suture or exposed suture knot.

PA: What systemic investigation should be done in such cases?

NVP: We routinely do screening for diabetic status .In a known diabetic, glycosylated hemoglobin is more reflective of glycemic control. Assessment of the nutritional status of the patient and looking for any systemic disease that may impair normal healing response eg: autoimmune diseases,malignancy, HIV is helpful in some cases.

NS: Certain systemic diseases like diabetes mellitus, acquired immunodeficiency syndrome and Sjogren's syndrome may act as risk factors for the occurrence of microbial keratitis. Moraxella lacunata ulcers are usually associated with alcoholics, diabetes and debilitated patients. Cases with immunodeficiency syndrome and advanced malignancies are prone to develop Candida keratitis. Pseudomonas keratitis occurs more frequently in cases of burn patients and comatose patients.some occupations may render a person susceptible to developing microbial keratitis e.g. Listeria monocytogenes keratitis is known to occur especially in cases of animal handlers. Evaluation of these factors should be considered especially in cases with non responding corneal ulcers.

UM: Blood sugar measurement, examination to rule out Hansen's disease, immunocompromised states like HIV may need to be investigated, clinical and laboratory investigations to rule out collagen vascular diseases like Rheumatoid arthritis may be indicated. Malnutrition in children and Malabsorption syndrome in adults can lead to Vit A deficiency- not too uncommon in our country.

RA: Investigate for diabetes, HIV, tuberculosis, autoimmune disorder.

PA: Is there a role of test for HIV, HBsAg, HCV or similar systemic diseases?

NVP: HIV testing is indicated if there is high index of suspicion or in cases of Herpes Zoster ophthalmicus in young patients. HBsAg or HCV testing do not contribute in these situations.

NS: HIV testing should be done for young patients especially with a history of spontaneous onset keratitis without any preceding history of trauma or other risk factors. Although the incidence of infectious keratitis has been found to be the same in cases with HIV, these patients have been shown to have increased severity of infections. HBsAg and HCV testing should be conducted to find out the underlying cause in cases with peripheral ulcerative keratitis.

UM: Yes, the possibility of an immunocompromised status must be kept in mind in a case of corneal ulceration that is refractory to conventional therapy.

PP: Be considered under the following headings.

1. To Reconfirm the causative organism in case of an infected corneal ulcer.

- Repeat scraping for smear and culture
- PCR
- Corneal biopsy

2. To rule out reservoir of infection eg infected lacrimal sac, lid margin disease, infected sinus etc.,

3. To investigate possible factors which interfere with would healing eg. Dry eye, lid malfunction, limbal stem cell deficiency, damage to corneal nerves etc.,

4. To rule out systemic disease associated with delayed would healing eg., Leprosy, rheumatoid arthritis, HIV infection etc.,

RA: Minimal.

PA: How would one differentiate between infective and non-infective cause?

NVP: Infective and noninfective keratitis may overlap each other. However, the edge of the ulcer may point to the aetiology. Usually, a non infectious ulcer has a punched out margin.

NS: Non infectious keratitis generally presents with an epithelial defect, whereas in infectious cases, the epithelial defect is associated with stromal infiltrates. However, a corneal scraping should be done in all cases of corneal ulcer. Thus, microbiological evidence remains the mainstay to distinguish between infective and non infective keratitis.

UM: An infective etiology is usually more acute and painful. Epitheilial defect with slough and discharge is more common in an infective case. Infective etiologies are more central while the non-infective causes are closer to the limbus.

PP: A non-infective non-healing ulcer may begin as a persistent epithelial defect. This typically has rounded edges with no evidence of corneal infiltrates. However, several hydrolytic enzymes including Collagenases and proteases contributes to the development of corneal ulceration and sterile melting of the cornea. Clinically, this leads to corneal thinning, descemetocoele and ultimately corneal perforation. Infected ulcers on the other hand, produce a more severely inflamed eye, with significantly more signs like lid edema, conjunctival congestion and chemosis. The corneal infiltrates are more dense, their pattern depending on the etiological agent. The anterior chamber also reflects the intense inflammatory response and it is not uncommon to see a hypopyon. However, sometimes in picture may by confusing. A non-infective ulcer could also have super-added infection. An infected ulcer on the other hand, could have become 'sterile' in response to antimicrobial treatment and yet, there may be a delay in re-epithelialization. Even after successful elimination of active microbial infection, the effect of microbial toxins may persist for longer, and continue to cause proteolytic degradation of stromal collagen and extracellular matrix components.

RA: Duration of ulcer, rate of progress of ulcer size, infiltration, presence of slough, hypopyon are some important parameters to differentiate. Recurrent episodes of watering, photophobia, corneal events go in favour of viral aetiology. Long standing ulcer not responding to conventional anti bacterials and anti fungals with progressive thinning are usually non infective with immunologic aetiology .Peripheral location of ulcer and lack of slough or infiltration are also in favour of noninfective aetiology. Obvious presence of deformities or RA or collagen vascular disorder favour non infective pathology. Presence of severe photophobia and single or multiple corneal infiltrates in children in our set up are usually due to hypersensitivity to tuberculoprotein. They usually have large necrotizing mantoux reactions. A word of caution when dealing with neurotrophic ulcers which are un necessarily treated with topical antibiotics, esp post zoster or post hemiparetic or hemiplegic attack. Here simple tarsorraphy does wonders.

PA: Are there any pointers for systemic autoimmune etiology?

NVP: Peripheral ulcerative keratitis with adjoining scleritis, bilateral disease, sterile corneal melt, and necrotizing scleritis are suggestive of autoimmune etiology.

NS: Underlying systemic autoimmune disorders should be considered in cases of peripheral ulcerative keratitis. The investigations generally performed in such cases include HBsAg, HCV, c-ANCA, p-ANCA, rheumatoid factor.

UM: Detailed history of systemic disease like joint pains, dry mouth, skin lesions should be asked specifically. Fellow eye should be examined for dry eye features and old corneal scarring. Autoimmune conditions may cause ulceration in the form of peripheral ulcerative keratitis with or with out scleral involvement.

PP: A detailed history could often point towards a systemic autoimmune etiology.

RA: Relentlessly progressive ulcer with significant corneal thinning, dry eye, impending perforation should alert one to investigate for systemic pathology.

PA: Is there a role of empirical antiviral or antifungal therapy in cases of non healing corneal ulcer on antibacterial therapy?

NVP: If there is a history of previous viral keratitis, and even if the patient is presenting with a suppurative keratitis now, I would add topical antiviral therapy along with the antibacterials. All effort must be put to isolate the causative organism and treat accordingly.

NS: Antiviral therapy should be considered in cases with dendritic ulcer, disciform keratitis, endothelitis. A history of previous episodes and diminished corneal sensations are indicators of a viral etiology. Antifungal therapy should only be started after a microbiological evidence in the form of either a KOH positivity for fungal hyphae and fungal growth obtained in culture. The study done by Dahlgreen et al(Am J Ophthalmol. 2007 Jun;143(6):940-944.) showed that the proportion of correctly predicted. fungal infections was 38%. So it was concluded that clinical features could not replace laboratory investigations in cases of keratomycosis.

UM: Diagnosis of Herpetic keratitis is essentially clinical. Treatment of viral keratitis may be initiated on clinical suspicion. Antifungal treatment should ideally be started only after proving presence of fungi microbiologically.

PP: No, there is no role for empirical antiviral or anti-fungal therapy in patients on antibacterial therapy. The toxicity of many of these drugs could be harmful.

RA: Not really, associated dense hypopyon deep infiltrates or endo exudated where scraping doesnot yield good material could be the basis for anti fungals. Antivirals if there is ulcer in graft and indication for surgery was macula/ macula leucomatous corneal opacity, as tere is fair chance of recurrence of viral ulcers in graft and epithelial and stromal keratitis when present together may also have slough.

PA: Role of Vitamin C/ Multivitamin/ Doxycycline/ any other drug therapy in such cases?

NVP: Vitamins usually are not a part of our therapeutic regimen, unless the patient shows clinical signs of such deficiency. If there is associated corneal melting or meibomian gland disease, then oral doxycycline can be used.

NS: Vitamin C and Doxycycline have anticollagenolytic properties. We generally start these drugs in cases with stromal lysis and thinning to prevent imminent perforation.

UM: I have found Doxycycline to be extremely effective in preventing corneal perforation and melting in sterile ulcers and use it routinely.

PP: Ascorbic acid is required for collagen synthesis and has been found to be low in eye with chemical injuries. Topical application of freshly prepared 10% ascorbate has been found useful in reducing stromal ulceration in such patients.

Sodium citrate is believed to inhibit polymorphonuclear cell migration and thus reduces inflammation. It also has an inhibitory action on collagenase. Likewise, the tetracycline family of antibiotics also has some anti-collagenolytic activity all of these could be useful supplements to try to reduce stromal melting.

RA: Don't believe in the role of vitamins in ulcer healing except when there is present accompanying vitamin A deficiency and supplemental vitamin A is helpful and therapeutic. Doxycycline has a role if there is present accompanying meibomian gland disease(MGD) or ulcer in patients with atopy.

PA: What is the role of corneal biopsy and when should it be done?

NVP: In deep seated corneal infections, which clinically looks infective, but unresponsive to therapy, a corneal biopsy is useful.

NS: Corneal biopsy can be a useful tool in cases where repeated smear examinations and cultures of corneal scrapings fail to demonstrate the presence of any microorganisms. Detailed microbiological examination of certain cases of deep mycotic keratitis and intrastromal abscesses can be greatly aided by obtaining microbe-infested tissue through a corneal biopsy.

UM: Corneal biopsy is indicated when an infective etiology is clinical suspected and routine cultures are sterile. It may be prudent to perform biopsy in deep keratitis with no epithelial defect.

PP: Corneal biopsy is performed:

1. When the infiltrate is deeper than what corneal scraping can collect or
2. When the clinical behavior does not match the microbiological diagnosis or
3. to confirm a suspicion of viral involvement in a mixed infection or
4. to rule out active infection, for example in acanthamoeba keratitis.

RA: In a definitely suspected fungal aetiology with deeper exudates or corneal abscess with intact epithelium.

PA: When should one think of therapeutic keratoplasty before infective process involves limbus/ posterior chamber?

NVP: If it is well established that the ulcer is not showing signs of healing, it would be better to go for early therapeutic keratoplasty, much before it involves the limbus.

NS: The most common indication of therapeutic keratoplasty in cases of corneal ulcer is corneal perforation. Sometimes, it can be undertaken to decrease the infective load of the cornea that is not getting controlled with maximum medical therapy.

UM: Therapeutic keratoplasty should be planned early rather than late. Results are better when the limbus is spared. If the ulcer is not responding to antimicrobial therapy and is relentlessly spreading to periphery, we should intervene early.

PP: Therapeutic keratoplasty is indicated

1. When the infection is clearly not responding to conservative medical treatment.
2. threatened perforation
3. likelihood of spread to limbus / sclera
4. endophthalmitis, where it may need to be combined with vitrectomy
5. threatened panophthalmitis.

RA: Therapeutic keratoplasty before limbal involvement,but if ulcer shows signs of localization or vascularization I wait. There is high incidence of reinfection with exudates in AC.

PA: Can therapeutic keratoplasty be done with glycerine preserved cornea if there is a shortage of good quality tissue?

NVP: Any eye bank corneal tissue is useful in such cases. The main aim is to remove the infected cornea and thereby quieten the eye for a future optical keratoplasty, if necessary.

NS: Lack of developed eye banking networks and a critical shortage of tissue suitable for transplantation are the major limiting factors for a sight restoring keratoplasty in the developing world. An eye saving approach can be adopted by the use of glycerin preserved corneal tissue in emergency conditions in the absence of availability of good quality donor tissue. The same can later by exchanged with a good quality donor tissue to restore the vision in these cases. Glycerine preserved eyes have also been used for lamellar keratoplasty in a few studies and the grafts have been noted to clear up postoperatively providing good visual rehabilitation to the patients. Also there have been recent reports of decreased incidence of graft rejection episodes with the use of glycerin preserved corneas.

UM: I am personally not in favour of this approach. In fact, this eye is severely inflamed and we should try to give it a good chance to survive by using a reasonably good quality of tissue. The success of keratoplasty is best in the first attempt.

PP: I have no experience with glycerin-preserved cornea and cannot comment.

RA: deally one should prepare good grade cornea for a compromised eye .Usually the practice is opposite, have done very few therapeutics with glycerine preserved cornea and experience has not been encouraging. Re-infections are common, in the current scenario where availability of corneas has improved I avoid this practice. In case of imminent corneal perforation or perforation one may tide over with glue and BCL and arrange for good quality cornea.

PA: What should be the size of the donor graft?

NVP: The usual recipient / donor disparity of 0.5 mm is mandatory. However in larger grafts beyond 9.0mm, a 1mm disparity may be helpful to achieve a better postoperative result and reduce the incidence of peripheral anterior synechieae and glaucoma. In certain conditions, a sclerocorneal graft may be indicated, depending upon the involvement of the original ulcer.

NS: The donor button in cases of therapeutic keratoplasty is always terphined after the preparation of recipient bed, as necrosis around the host cut may require additional trimming and may alter the size of the graft. Donor button is punched from the endothelial side, and is usually 0.5-1mm larger than the host cut. In cases of perforated corneal ulcers, the memory of the prolapsed iris may lead to formation of peripheral anterior synechiae and recurrent prolapse of the iris through the graft host junction. Oversizing the graft by 0.5-1 mm helps in preventing this complication. We generally take 1mm oversized grafts in such cases as this helps in better maintaining the anterior chamber postoperatively. (Sharma N et al. Curr Opin Ophthalmol. 2010;21 (4):293-300).

UM: The recipient bed should be trephined around 1 mm beyond the area of infiltration. Donor tissue should be 0.5 to 1 mm larger than the recipient bed.

PP: The donor graft is usually 0.5mm oversized with respect to the recipient bed.

This allows better apposition and easier suturing of the graft-host wound. It also reduces the likelihood of peripheral synechiae and secondary glaucoma postoperatively.

RA: Prefer clear corneal host periphery and donor cornea may be 0.5-1.0 mm larger than the host bed for the ease in chamber formation esp. if there is present lot of exudation. Sometimes patch graft or cresentic grafts are also necessary.

PA: What is your take on cocktail regimen of treatment with antibacterial, antifungal and antiviral?

NVP: On retrospective analysis of corneal ulcer cases the incidence of mixed corneal infections is extremely low and hence cocktail therapy is not justified. However, in some cases, when the distinction between the aetiologies are not clear, antifungals can be used alongside antibacterials.

NS: The treatment of corneal ulcer should always be based on the microbiological reports. It is prudent to have a documentation of the infecting organism and a specific targeted approach is recommended in the management of corneal ulcers. There is no role of cocktail regimens.

UM: Not a good idea! In fact, topical medication may interfere with healing.

PP: Empirical cocktail regimens of antibacterial, antifungal and antiviral drugs should be strongly discouraged. If the clinician does not have access to microbiological facilities, he must exercise his clinical judgment. If he lacks both, the case must be referred to an appropriate centre.

RA: To be avoided and is a clear observation that doctor doesnot understand the disease. Hardly successful and increases drug toxicity and reduced drug penetration.

PA: Is there a role of amnion membrane grafting?

NVP: AMG is done in our centre for neurotrophic corneal ulcers and as tectonic support in large descemetocoele, if the patient is not willing/suitable for keratoplasty. We do not perform AMG for active infectious keratitis.

NS: Whereas some studies show that amniotic membrane has some antimicrobial properties, the others speculate that the mechanism of antimicrobial activity is the induction of a physical barrier to prevent bacterial invasion. AMG finds a role in cases of resolving keratitis cases. AMG has been found to have a good penetration of antimicrobial agents. Some studies have also demonstrated the efficacy of amniotic membrane in cases with keratitis due to Pseudomonas and viral keratitis. AMG is also an effective alternative for treating persistent neurotrophic ulcers (multilayered AMG), non traumatic corneal perforations and descemetoceles.

UM: Amniotic membrane may help in assisting healing of neurotrophic ulcers. It acts like a bandage contact lens and a substrate over which cells can grow, apart from releasing growth factors that support healing.

PP: Amniotic membrane transplantation has been found to be very useful in treating non-healing epithelial defects, in reducing inflammation and hence the cascade of events that result in stromal necrosis and perforation. It can also be used as a patch graft for an impending perforation to avoid a therapeutic keratoplasty.

RA: There are numerous reports of the promotion of healing process in severe bacterial keratitia, fungal keratitis and herpetic keratitis. In all these cases intensive antibacterial and antifungal treatments ere given prior to AMT.I have used AMT as a patch and graft in ulcers with stromal thinning with or without limbal deficiency and found encouraging results in 60% cases.

PA: Any non conventional therapy you feel has worked in your experience?

NVP: In cases of deeper mycotic keratits, intrastromal amphotericin B has shown to be of some use. Tarsorhapy shows good results in indicated cases such as neurotrophic keratitis.

NS: We have treated a few cases of fungal keratitis with intrastromal and intracameral amphotericin B and voriconazole. A favourable response has been seen in

these cases.(Prakash G, Sharma N et al. Am J Ophthalmol 2008;146;56-9).

UM: Tarsorraphy should be planned early in neurotrophic cases.

PP: Recently corneal collagen cross linking with UV-A has been tried in 2 patients with bullous keratopathy who also had infectious keratitis – one with Streptococcus viridans and another where smear was negative. Kozobohs V et al. Cornea 2010; 20; 235-238. Both showed a significant improvement in their clinical picture.

UV-A is known to have an anti-microbial effect, but we need to have a better understanding of its penetration in diseased corneas to ensure the safety of the procedure.

RA: Addition of 20%autologous serum, frequent lubrication, tarsorrhaphy.

PA: What would be the prognosis in most cases?

NVP: If the underlying etiology is corrected the ulcer heals well with scarring and minimal vascularisation and may not necessitate any further surgical intervention. However some of these cases need therapeuctic keratoplasty and the culture of the corneal button helps to guide postoperative medical management. Recurrence of infiltrate in the graft, scleral and posterior segment involvement is a poor prognostic sign.

NS: The prognosis in such cases is usually guarded. The same is also determined by the microorganism causing the keratitis e.g. cases of Nocardia keratitis generally carry a fair prognosis, cases of mycobacterial keratitis have a poor prognosis with only half the patients responding to the treatment instituted.

UM: Depends on the etiology. Therefore, it is of utmost importance to arrive at an etiological diagnosis clinically or with the aid of laboratory services.

RA: If properly diagnosed and managed prognosis is good in terms of ulcer healing and reepithelialization and stromal tissue recovery. Management of coexisting raised intraocular pressure also aids in ulcer healing.

PA: What would be the factors deciding the prognosis?

NVP: Severity of the ulcer, causative organism, response to available antimicrobial therapy and susceptibility pattern, compliance of the patient, and the timing of surgical intervention will be crucial factors.

NS: The prognosis in these cases depends on a number of factors including the type of microorganism isolated, the stage at which the treatment has been initiated, the time of referral to a higher center for detailed assessment and appropriate management has been started.

UM: Etiology, extent of involvement and patient's systemic status.

RA: Proper diagnosis, close follow up, infective vs non infective aetiology, early corneal graft with good quality tissue by senior corneal surgeon, specific management, avoid cocktails, attend to trichiasis foreign body, IOP, Prior history of use of steroids delays healing. Management of coexisting systemic problem with immunosuppressives. Look for tubercular focus. Consult the cornea doctor whenever picture is not clear. Don't hesitate.

PA: Any tips and tricks you would like to share for such difficult cases?

NVP: Sometimes, it is wise for the physician to look at the medicines used by the patient. In some cases, we have found that the drugs were not used in the prescribed manner (for example, Homatropine was used hourly, while the anti infectives were used twice a day). Culture of the drops especially, Natamycin is useful in some cases to identify causes for secondary bacterial contamination.

NS: A diligent search for the causative microorganism and a targeted treatment approach is the key to successful management of such cases. A thorough scraping should be done from the base and margins of the ulcer. Contact lenses, contact lens cases, contact lens solutions should be subjected to culture in cases with contact lens induced keratitis. In cases of infectious keratitis post LASIK, the flap may need to be lifted and scraping may be obtained from the residual stromal bed. Infections following keratoplasty especially DSAEK may need to be subjected to a corneal biopsy to isolate the pathogenic organism.

UM: Take a detailed history including systemic history, examine the fellow eye first, check corneal sensations, order special microbiologic stains and culture according to the clinical suspicion, examine the adenexae and perform corneal biopsy when indicated.

PA: When should one refer the case to a higher centre?

NVP: Whenever, doubts exist that the ulcer is not responding, and if there is no access to corneal tissue, it is better to refer the case earlier, so that early TPK can be planned and the outcomes of salvaging the globe is better.

NS: All cases of infectious keratitis not responding to conventional therapy within 48-78 hours should be referred to the higher centre for evaluation and management. In centers where the microbiology facilities are not available, all cases of corneal ulcer should be referred for a basic microbiological work up and specific antimicrobial therapy. Paediatric cases, cases with impending or actual perforation should be referred immediately to a higher center for further management.

UM: An ulcer not responding with your management for more than two weeks, should be referred to a higher centre.

RA: If ulcer is not responding to your line of management for 2-3 days an expert opinion is must and don't take therapeutic keratoplasty lightly.

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Lens Induced Glaucoma

Baswati Prasanth *MS DNB,* ***Suneeta Dubey*** *MS,* ***Monica Gandhi*** *MS*

Cataract accounts for 50-80% of the world's blind. In the developing world existing financial, cultural and psychosocial barriers refrain accessibility to existing surgical services thereby increasing the visual morbidity. There is an ever increasing backlog of cataract due to the population explosion, increased life expectancy and low productivity in terms of utilisation of the available surgical services. The uptake of eye care services by the rural community has also been suboptimal in countries like India1 where lens induced glaucomas are a common cause of ocular morbidity.

Under Vision 2020, the global initiative of the WHO and voluntary service organizations to reduce significantly avoidable blindness by the year 2020, goal is to increase the cataract surgeries performed, particularly in the developing nations. Currently, it is estimated that about 12 million cataract operations are performed each year the world over with a way below target of about 20 million cataract operations by the year 2010 and ultimately reach a target of 32 million people receiving cataract surgery annually by 2020.

While the prevalences of morbidity and visual impairment due to primary glaucomas have been fairly well established by population surveys in the west and, recently, in the developing world, the issue of blindness from secondary glaucomas has received little attention from most investigators. Information on secondary glaucomas in published eye surveys is limited and the cause of glaucoma seldom identified. Based on the WHO Blindness Data Bank, Thylefors and Negrel, in their world estimate of glaucoma blindness, found it was not possible to determine the number of blind from secondary glaucoma, although they estimated the world prevalence to be 2.7 million[2]. Quigley estimated the mean prevalence of secondary glaucomas to be 18% of the mean prevalence of primary open angle glaucoma in the world[3]. As per the published data from the Glaucoma Services at the Aravind Eye Hospital in the year 2000, the Common Causes of Secondary Glaucomas are as shown in Table[4].

Lens induced glaucoma thus forms a major cause of secondary glaucoma in our country and the visual impairment and ocular morbidity associated with such condition calls for a better insight into the diagnosis and management of these glaucomas.

Morphological Types

Lens-induced glaucoma in the elderly can be subdivided into two major categories.

Secondary Angle Closure Glaucoma: The first category relates to a blockage of the anterior flow of the aqueous humor due to the lens that results in an increase of intraocular pressure (IOP).

Phacomorphic Glaucoma: Condition(s) related to the size of the lens

- Intumescent cataract
- Traumatic cataract

Mechanism

- Pupillary block
- Direct angle closure
- Combination

Phacotopic Glaucoma: Condition related to the site of the lens

- Subluxated
- Dislocated

Mechanism

- Direct angle closure
- Pupillary block

Secondary Open Angle Glaucoma: The second category is characterized by the blockage of the trabecular meshwork from lens proteins (phacolytic glaucoma), lens material or debris, and rarely by phacoanaphylactic response to lens material.

Phacolytic Glaucoma (Lens Protein Glaucoma): Condition(s) related to soluble lens proteins.

Mechanism

- Heavy Molecular Weight protein [HMW]
- Macrophagic response to the lens Protein

Lens Particle Glaucoma: Condition related to lens particles

Table1: Common causes of Secondary Glaucoma[4]

Diagnosis	Number of Individuals with Secondary Glaucoma	Percentage of Total Glaucoma
Lens induced glaucomas	158	2.5
Neovascular glaucomas	58	0.95
Pseudophakic glaucomas	38	0.62
Uveitic glaucomas	25	0.4
Traumatic glaucomas	16	0.26
Steroid-induced glaucomas	12	0.2
Secondary glaucomas of Unspecified cause	60	1.0

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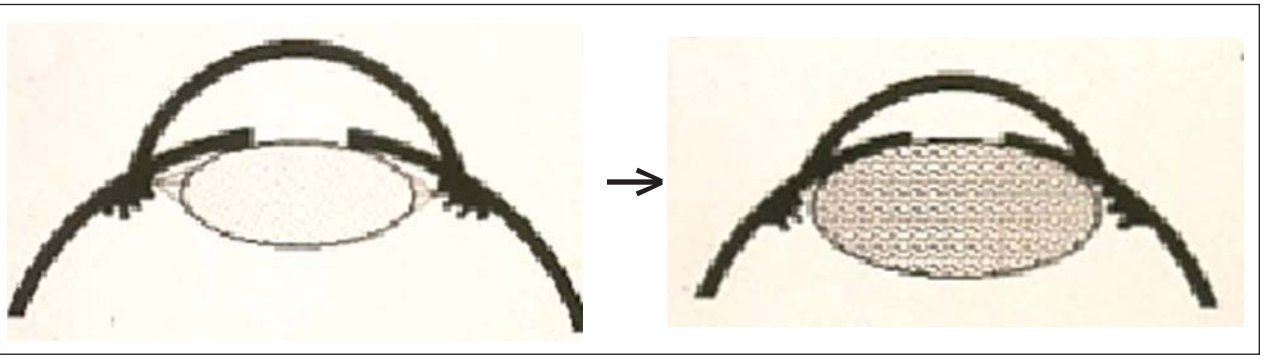

Figure 1: *Swollen lens → Pupillary block → Iris bombe → Angle closure*

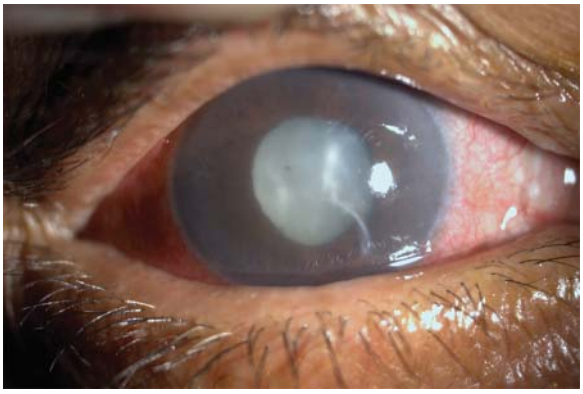

Figure 2a: *Slit lamp photo after control of intraocular pressure with medical therapy*

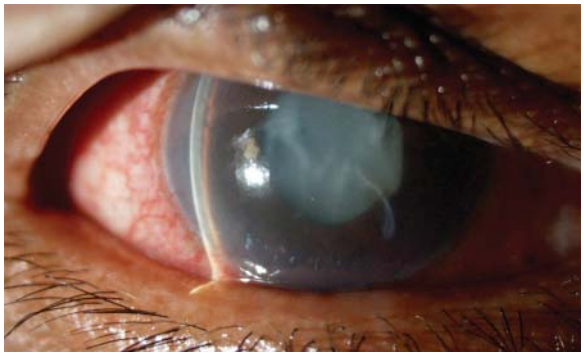

Figure 2b: *Reveals shallow anterior chamber in the same patient*

Mechanism

- Blockage of Trabecular Meshwork by lens particles e.g - retained lens matter following cataract surgery
- Post. YAG capsulotomy

Phacomorphic Glaucoma

Phacomorphic glaucoma often occurs from an immature cataract, but may also occur from spherophakia in Weill–Marchesani syndrome. It is encountered more in developing countries and seen commonly in smaller eyes (hyperopic) with a shallow anterior chamber. It is predisposed by rapidly developing intumescent cataract or traumatic cataract. It is seen more often as compared to other lens induced glaucomas. In a retrospective study conducted at Nepal Eye Hospital[5] out of 40 cases reviewed, 26 (65%) were phacomorphic glaucoma and 14 (35%) were having phacolytic glaucoma. In another study[6] that dealt with the frequency and types of lens-induced glaucoma the percentage of phacomorphic glaucomas was again reported higher (72%) than phacolytic (28%). The authors also reported a female preponderance (55%) which has been substantiated by other studies also[7,8] and could be a result of the anatomical predisposition.

Mechanism: Involves a pupillary block characterized by the obstruction of the aqueous outflow by the apposition of the iris root to the trabecular meshwork. A senile cataractous lens that has progressed enough to become intumescent or a traumatic intumescent lens with an increased anteroposterior diameter, leads to pupillary block. (Figure 1)

Clinical features: Phacomorphic glaucoma rarely presents asymptomatically as chronic angle-closure glaucoma, however it more often presents as acute angle closure glaucoma. This is characterized by ocular pain, headache, blurred vision, perception of halos around lights (due to the corneal edema), and also nausea, vomiting, bradycardia, and diaphoresis due to the vasovagal response.

Clinical examination reveals reduced visual acuity mostly secondary to corneal edema, circumcorneal congestion, a mid-dilated pupil, and, an intumescent cataractous lens. Anterior chamber appears shallow both centrally and peripherally with the presence of flare. The fellow eye shows the same anatomic predisposing factors and usually a cataract. (Figure 2a&b) reveals Slit lamp photographs of a 60 years male presenting with hazy cornea, intumescent cataract with shallow anterior chamber

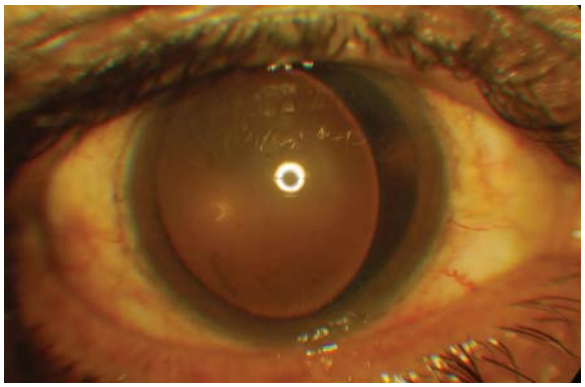

Figure 3: *Slit lamp photos showing subluxated lens*

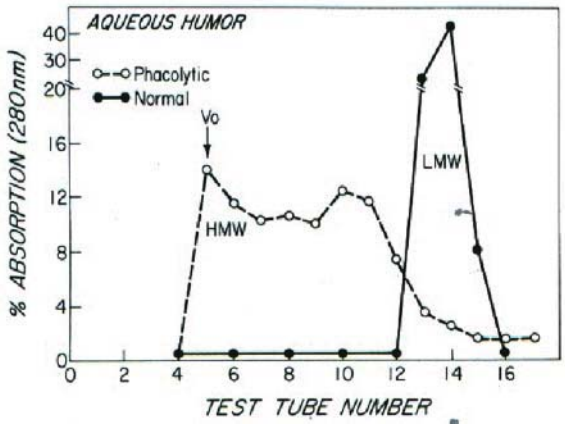

Figure 4: *Heavy molecular weight (HMW) protein in the aqeous humor of patients with phacolytic glaucoma*[13]

and intraocular pressure of 54mm Hg. Patient was diagnosed as Phacomorphic glaucoma and underwent extracapsular cataract extraction following medical control of intraocular pressure.

Management: Initially, IOP-lowering medications are used. Hyperosmotic agents (mannitol IV), carbonic anhydrase inhibitors and topical betablockers, are the mainstay of medical treatment. In resistant cases Yag iridotomy can be performed. If the fellow eye is also anatomically predisposed to angleclosure glaucoma, prophylactic laser iridotomy should be performed.

After IOP control to a safe level and the reduction of corneal haze cataract extraction, is done to erase the major causative factor of angle-closure glaucoma. Extracapsular cataract extraction with in the bag PCIOL implantation is the usual procedure of choice. Good visual recovery with control of IOP has been reported by most of the studies following this procedure[5-7,9]. A study[10] done to evaluate the intraoperative and postoperative complications of Phacoemulsification in phacomorphic glaucoma revealed it to be a relatively safer procedure with good visual recovery and minimal intraoperative and postoperative complications.

Role of Combined surgery: Usually cataract extraction alone is sufficient to reduce the intraocular pressure and control the inflammation. However, when the duration of presentation is prolonged (more than 72 hours) or intraocular pressure cannot be sufficiently controlled with maximal medical therapy for more than 07 days, a combined trabeculectomy with cataract surgery is contemplated.

Ectopia Lentis

This clinical entity may present as an isolated inherited form or may be associated with systemic disorders such as Marfan's syndrome, homocystinuria, Weill–Marchesani syndrome, hyperlysinemia, and sulfite oxidase deficiency that results in a defective zonular apparatus. Dislocation of the crystalline lens may also be secondary to trauma.

Clinical features: Dislocation may cause pupillary block and result in angle-closure glaucoma or a dislocated lens may directly encroach upon the angle. The patient experiences a red and painful eye, decreased visual acuity, and sometimes headache, nausea, and vomiting. Signs include phacodonesis, iridodonesis, shallowing of the anterior chamber either symmetrically or asymmetrically, and a difference in the depth of the anterior chamber between the two eyes. (Figure 3) highlights the anterior segment photo of a 58 years male who presented with history of trauma of 04 days duration. Examination revealed lens subluxation of > 180^0 with an intraocular pressure of 34 mmHg. There was no angle recession on gonioscopy, however the temporal, inferior and partly superior angle was encroached by the subluxated lens. IOP was reduced medically and patient was advised lens extraction with intraocular implantation.

Management: The therapeutic approach depends on the degree of dislocation and the symptoms. In cases of partial subluxation within the pupillary space that does not cause significant visual impairment or pupillary block glaucoma, a conservative nonintervention strategy can be followed. When the previous condition is accompanied by pupillary block, then a laser peripheral iridectomy is the appropriate solution. Total anterior dislocation requires removal of the lens.

Phacolytic Glaucoma

This acute open-angle glaucoma is the result of the leakage of lenticular material from senile hypermature or Morgagnian cataract through an intact lens capsule. Its less commonly seen as compared to phacomorphic glaucoma. At the beginning of the century Gifford described glaucoma associated with hypermature cataract and suggested that it could be prevented and cured by timely cataract extraction. Since then various authors Irvine and Irvine (1952), Flocks, Littman and Zimmerman (1954) and Chandler (1958) have described various lens induced glaucomas.

Mechanism: The original theory about the pathogenesis of this condition was that the macrophages were the major culprit of increase in IOP by blocking the trabecular meshwork[11,12]. Lens material may cause blockage of outflow of the aqueous at the drainage angle and this may occur after injury (including cataract surgery) or when lens material leaks through the lens capsule of immature/hypermature lens. Macrophages, attempting to remove this material, together with the abnormal lens material itself may cause blockage at the angle of the anterior chamber.

Later research by Epstein and colleagues[13] Yanoff and Scheie[14], and Dueker[15] emphasized the role of heavy molecular proteins (HMW) leaking from the lens in the obstruction of the aqueous outflow and de-emphasized the role of the macrophages. In a series of patients[13] aqueous humor was obtained by paracentesis at the time of cataract surgery from six patients with phacolytic glaucoma and from six control patients with immature cataracts. Quantities of heavy-molecular-weight (HMW) protein sufficient to obstruct aqueous outflow were identified in all six phacolytic aqueous humor specimens but in none of the controls. Three of the hypermature cataractous lenses from the cases of phacolytic glaucoma were found to have 14-fold greater quantities of HMW protein in their liquefying cortex than were present in the cortex of immature cataractous lenses. (Figure 4)

As a consequence, the examination of the anterior chamber fluid for the presence of HMW protein has become an important diagnostic aid in suspected and atypical cases of phacolytic glaucoma, but is not yet widely available.

Clinical Features: Patient usually presents with a painful red eye. The pain is acute in onset and maybe associated with nausea, vomiting. There is history of gradual decline of visual acuity reflecting the slow maturation of the cataract.

Clinical examination reveals Circumcorneal congestion, corneal edema, a heavy flare, and aqueous cells with a deep anterior chamber. (Figure 5a,b) Soft white patches on the lens capsule can be observed (Figure 5c), which are aggregates of macrophages trying to seal the site of leakage[11]. Whitish material may be seen floating in the anterior chamber which are calcium oxalate or cholesterol crystals. Intraocular pressure is very high and gonioscopy reveals open angles. The fellow eye usually has a mature cataract and a deep anterior chamber.

Management: Initial treatment of phacolytic glaucoma is focused upon acute lowering of IOP using a combination of topical and systemic IOP-lowering agents. Topical steroids by reducing the inflammation also may facilitate IOP lowering and decrease pain. Medical therapy however is only a temporizing measure until cataract surgery can be scheduled. The definitive treatment involves cataract extraction. Extracapsular cataract extraction is the procedure of choice owing to poor visibility. This generally has to be accompanied by thorough anterior chamber irrigation to remove all the lenticular material. Manual sutureless extracapsular cataract surgery (MSICS) has been reported by some surgeons to be beneficial with early visual recovery and good IOP control. In a series of thirty three cases Venkatesh and coworkers[16] reported this technique to be a better and safer technique in advanced cases but a challenging situation for the surgeons.

Role of Combined surgery: Combined cataract and trabeculectomy was found to result in significant IOP lowering as compared to cataract surgery alone in a group of patients with phacolytic glaucoma where symptoms existed for more than 07 days or intraocular pressure was not controlled preoperatively with maximal medical therapy[17].

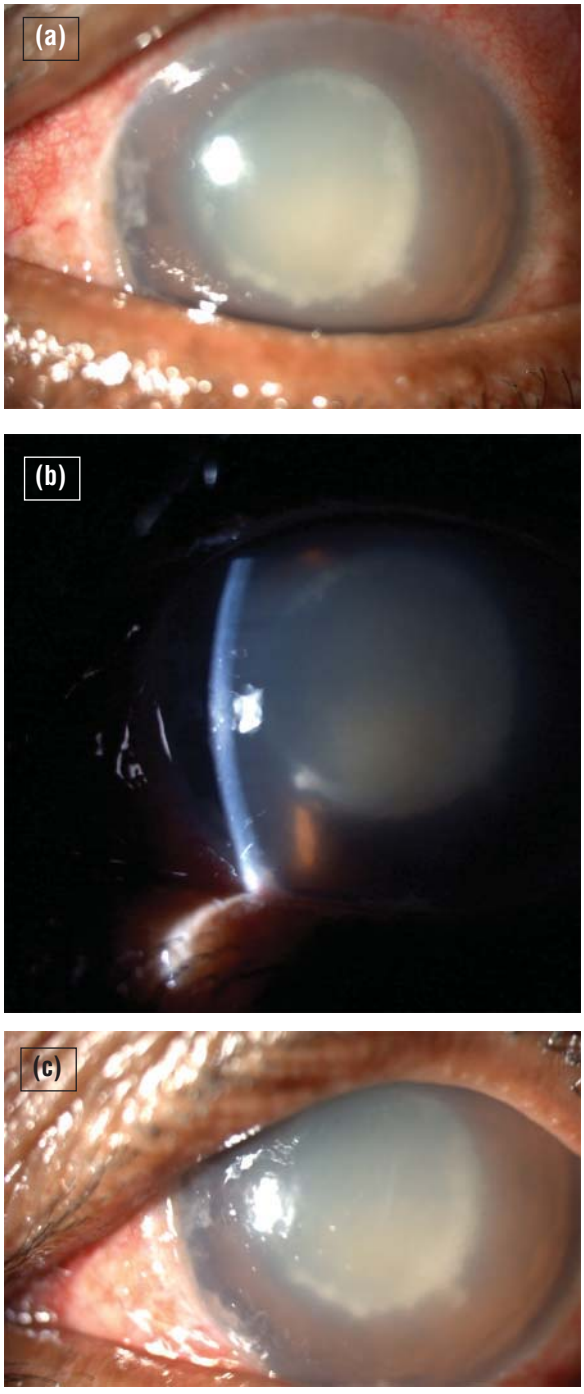

Figure 5a,b&c: *Slit lamp photograph of a 68 years male presenting with phacolytic glaucoma*

Lens-particle Glaucoma

Is a form of lens-induced glaucoma with open-angles which is associated with a grossly disrupted capsule and the presence of obvious fragments of lens material in the anterior chamber. It may occur after cataract surgery, trauma to the lens, or Nd:YAG posterior capsulotomy.

Mechanism: Obstruction of the aqueous outflow at the trabecular meshwork by the floating lens particles or engulfed macrophages. Enucleated human eyes were perfused via the anterior chamber at 25 mm Hg pressure with lens particles (whole lens homogenates) in one series of experiments and with soluble lens proteins from human cataractous lenses in another series. Adding 1% of a

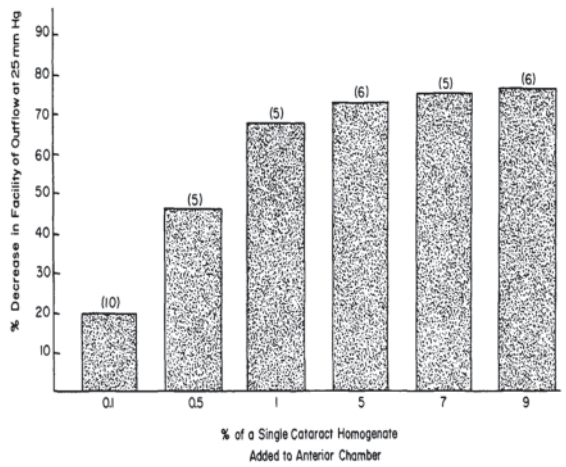

Figure 6: *Effect produced on facility of aqueous outflow by varying the proportion (%) of a cataractous whole-lens homogenate added to the anterior chamber of perfused eyes. The number of eyes in each group is shown in parentheses*

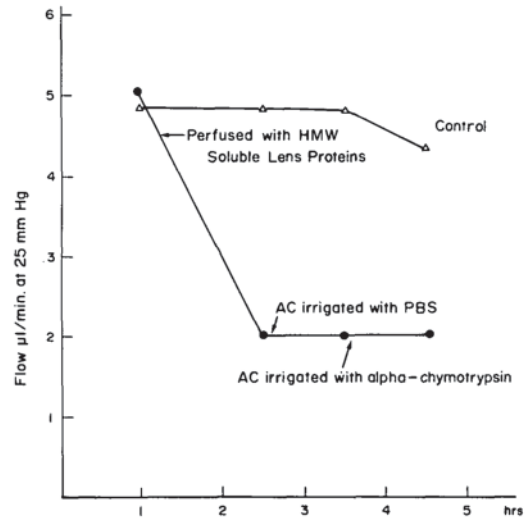

Figure 7: *Effect of specially isolated heavy molecularweight (HMW) soluble lens proteins from humancataracts (1 mg/ml; MW over 150 X 106) on fluidoutflow. Ten enucleated human eyes were perfusedfor 1 hr with HMW soluble lens proteins (filled circles). Six controls were sham-treated witha protein-free solution (triangles). Attempts were made to reverse the effect by washing out the anteriorchamber.*

homogenate of a single cataractous lens to the anterior chamber induced a 68% decrease in outflow. (Figure 6) Perfusion with HMW lens proteins also produced a severe obstruction of fluid outflow which was not relieved by irrigation of the anterior chamber with balanced salt solution or alpha-chymotrypsin[18]. (Figure 7)

Clinical features: Patients may be asymptomatic or depending on the severity of intraocular inflammation and elevated IOP may present with monocular eye pain, redness, and blurring of vision. On interrogation, patient often gives a recent or remote history of trauma or intraocular surgery, particularly cataract extraction[19,20]. The onset of lens-particle glaucoma has been reported to occur many years after cataract surgery[21,22]. In an unusual case report, lens particle glaucoma was seen 65 years after congenital cataract extraction which was successfully treated with parsplana vitrectomy[20]. On examination, variable degree of inflammation may be seen ranging from small free-floating fragments of cortex to a layered pseudohypopyon. Anterior chamber angle is open by gonioscopy, although inflammatory synechia may be observed in severe cases. Particles of cortex or nucleus may be seen dislocated into the vitreous cavity.

Treatment: Initially, a trial of medical antiglaucomatous therapy may be attempted. Owing to the inflammatory nature of the condition miotics are best avoided. Steroids are given in mild to moderate doses depending on the severity of the inflammation. A large amount of lens material in the anterior chamber or vitreous cavity resulting in uncontrollable glaucoma however warrants surgical removal.

Phacoanaphylactic Glaucoma

Phacoanaphylactic glaucoma is a rare entity due to an inflammatory reaction directed against own lenticular antigens with elevation of the IOP due to involvement of the trabecular meshwork by the inflammation or by obstruction from inflammatory cells. Verhoeff and Lemoine in 1922, first drew attention to this form calling them "endophthal¬mitis-phaco-anaphylactica". The presumption was that such cases were allergic in nature, the allergen being their own lens protein. On the basis of the finding that those persons who suffered the violent type of ocular symptoms when tested intradermally displayed a positive skin test to lenticular-protein and when given a desensitising course of intra muscular injection of lens protein rapidly showed an amelioration of their symptom, this was thought to be a hypersensitivity reaction.

Mechanism: Similar to the lens particle glaucoma, there is usually a preceding disruption of the lens capsule. However, in contrast to lens particle glaucoma, there is usually a latent period of 24 hours to 14 days between the trauma and the onset of inflammation. The patient is sensitized to his own lens antigens and these proteins are kept in an immunologically privileged site within the lens capsule. After an eye surgery or other trauma to the lens capsule, these lens antigens are exposed to the circulation, they may be recognized as 'foreign' by the individual's immune system and they incite an inflammatory response. The mechanism causing the reaction seems to be an Arthus-type immune complex reaction mediated by IgG and the complement system[23,24]. A matter not completely elucidated is why some patients develop this reaction and others do not.

Clinical Features: The clinical signs include lid edema, chemosis, conjuctival injection, corneal edema, heavy anterior chamber reaction, posterior synechiae, and mutton fat keratic precipitates. The predominant presentation in such a condition is usually that of a chronic granulomatous type of inflammation with associated glaucoma which may be a rare feature. Also, a point of interest is to note that the inflammation may be centred on the lens matter of the primarily involved eye or the fellow eye.

Management: Initial measure is to control the inflammation medically with the help of steroids and raised IOP if present requires antiglaucoma treatment. When medical measures are inadequate, the retained lens matter has to be removed surgically.

Conclusion

Lens induced glaucoma is a preventable and curable disease. Unlike developed countries, Lens induced glaucoma is not uncommon in our population, where people cannot afford to undergo cataract surgery in time. Definitive treatment of this condition most often involves removal of the cataractous lens alone or combined with filtration surgery, once the high intraocular pressure is satisfactorily controlled. The results in response to visual recovery and IOP control is quite encouraging and earlier the lens extraction, better is the outcome in terms of vision and IOP control. Combined surgery is most often reserved for only those cases with a late presentation(more than 03 days), where symptoms are not controlled with medical therapy for more than 07 days, IOP not controlled with maximal medical therapy, extensive peripheral anterior synecheiae noted (more than 180^0) and optic nerve head cupping noted in USG B scan. Looking at the burden of ocular morbidity imparted by this entity and the successful outcomes associated with an early intervention, there is a great need to impart health education to the public about the importance of timely surgery.

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Intermediate Uveitis and Pars Planitis

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The IUSG (International Uveitis Study Group) suggested the term Intermediate uveitis (IU) to denote an idiopathic inflammatory syndrome, mainly involving the anterior vitreous, peripheral retina and the ciliary body with minimal or no anterior segment or chorioretinal signs. Intermediate uveitis is however associated with various conditions like Sarcoidosis, Multiple sclerosis, Lyme disease, peripheral toxocariasis, Syphilis, Tuberculosis, primary Sjogren syndrome and infection with Human T-cell lymphotropic virus type–I (HTLV-I). More recently, according to Standardization of Uveitis Nomenclature (SUN) working group's international workshop for reporting clinical data, the term pars planitis is reserved for IU without any associated infection or systemic disease and presence of snowbanking. It is the most common form of IU, constituting approximately 85-90% of cases.[1]

Epidemiology & Pathogenesis

IU represents 11-15% of uveitis patients. Incidence is higher in the children. Pars planitis occurs mostly in 5-40 years of age. However cases associated with HTLV-1, develop IU in the 7th -8th decade. Pars planitis has a bimodal distribution with one peak in the second decade and another peak in the third or fourth decade. Overall, there is no sex predilection though pars planitis in younger age group of 5-15years has a male preponderance and females are more commonly affected in the older age group of 20-40 years.[1]

Pathogenesis: Pars planitis is likely to be an autoimmune response to vitreous, ciliary body or peripheral retinal tissues. Heritable or environmental factor may play a role.

- Multiple Sclerosis - autoimmune cause is suspected. 20% of patients with IU have a chance of developing multiple sclerosis or optic neuritis during 5 years period.
- Lyme disease – infection + autoimmune cause
- HTLV-1 infection– immune system disregulation[2]
- Several HLA types are reported: HLA DR-15 (suballele at HLA-DR2) has highest association with MS[3]

Clinical features

Onset of pars planitis is usually insidious & gradual. Smith et al described clinical course and progression of disease in three patterns. 4 Pattern 1 patients (10%) have a self-limiting course with no exacerbations. A prolonged course without any exacerbations (60%) constitutes pattern 2 patients. Patients with pattern 3 diseases (30%) have a chronic smoldering uveitis with remissions and one or more exacerbations. Pars planitis is bilateral in 70-80% of cases although asymmetry may be marked. The patient presents with complaints of floaters, blurred vision and rarely with initial complaint of mild pain, photophobia and redness.

On examination, vitritis is a characteristic feature of IU, and it is typically described as vitreous haze ranging from trace to 4+. Vitreous clumps of inflammatory cells - vitreous snowballs, are common. They are usually located inferiorly, but may be found throughout the vitreous cavity. Posterior keratic precipitates and vitreous strands may be present. Exudates along inferior pars plana (may extend along whole circumference) called as snowbanking are pathognomic of pars planitis. Snowbanking in quiescent stage is smooth & shiny and fluffy in appearance in acute stages. Peripheral vascularization may develop which may lead to vitreous hemorrhage. Peripheral retinal perivasculitis

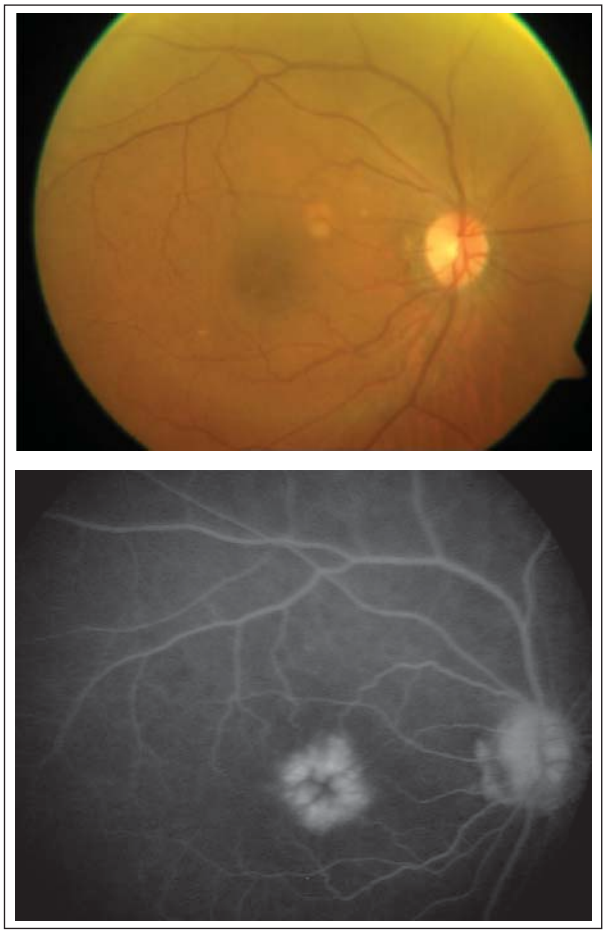

Figure 1a: *Fundus photograph and FA showing presence of cystoid macular edema in a patient with pars planitis. Mild vitritis is also present*

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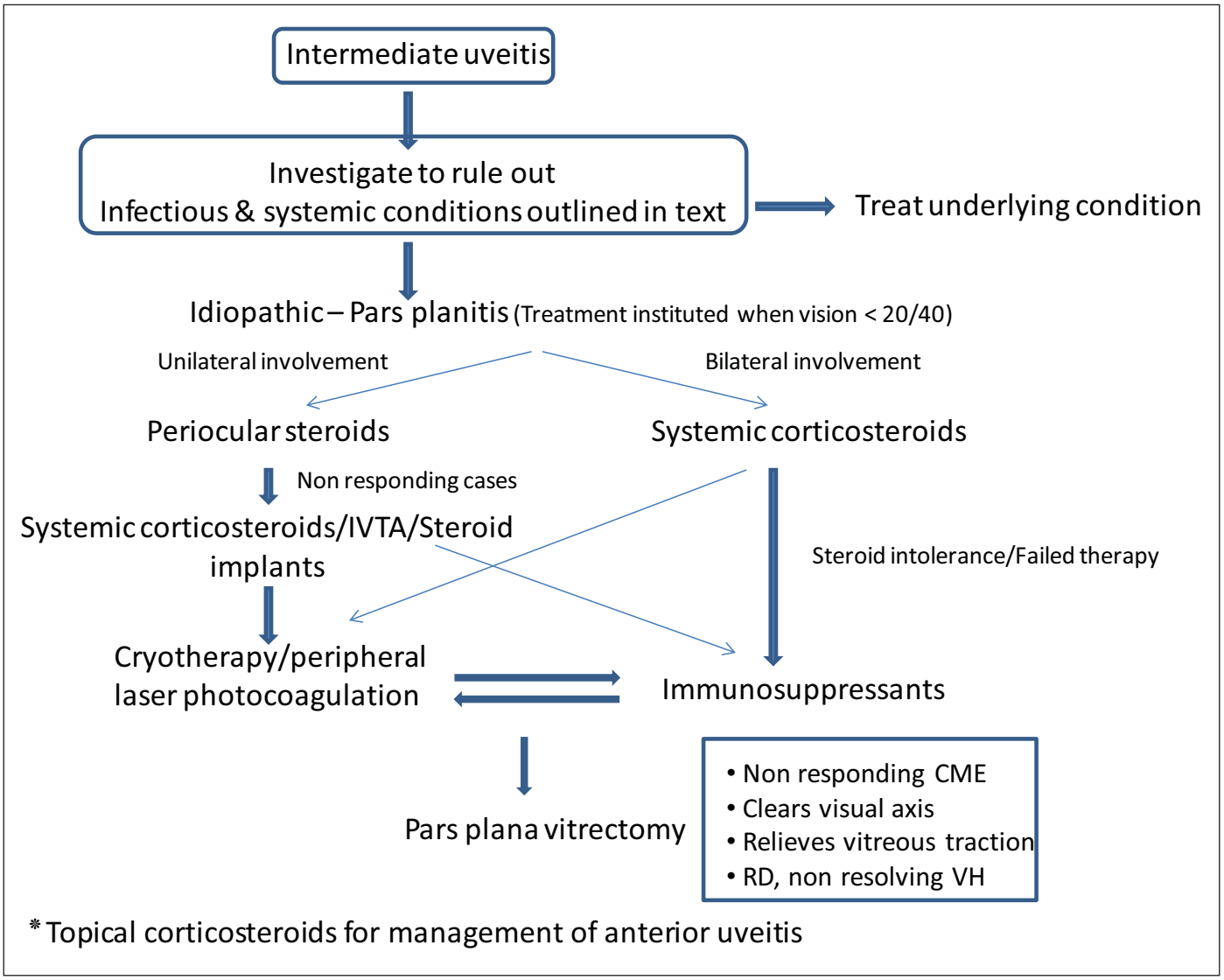

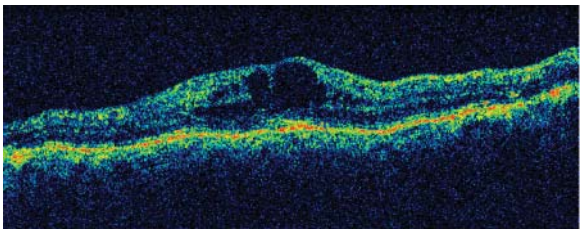

Figure 1b: *OCT reveals macular edema with presence of cystic spaces*

(especially periphlebitis) and perivascular sheathing are common in pars planitis.

Cotton wool spots & retinal hemorrhages may occur especially in cases associated with HTLV-1 infection. Posterior vasculitis is also more severe in such cases.

Anterior chamber examination may show mild inflammation with cell rarely exceeding 2+. Other findings may include small to moderate KP's, autoimmune endotheliopathy, corneal edema and trabecular precipitates. Post synechiae formation is very rare. Presence of 3-4 clock hours of post synechiae challenges the diagnosis of IU.

Complications

- Cataract (15-60%): Inflammation or steroid therapy are responsible[5]
- Cystoid macular edema (50%): CME is the most common cause of decreased visual acuity in pars planitis. Ocular inflammation & / or vitreous tractions may be responsible for it. CME of long duration may lead to macular degeneration & permanent loss of vision.[5]
- Glaucoma (10%)
- Neovascularization: Mostly peripheral but rarely NVD, NVE or NVI (severe disease) may develop.
- Vitreous hemorrhage (<5%)
- Papillitis and optic nerve edema – not common
- Band shaped keratopathy
- Retinal traction leading to dragged disk vessels or retinal tear or macular heterotropia
- Rhegmatogenous or tractional retinal detachment (5-15%)
- Cyclitic membrane formation – leading to hypotony and phthisis bulbi

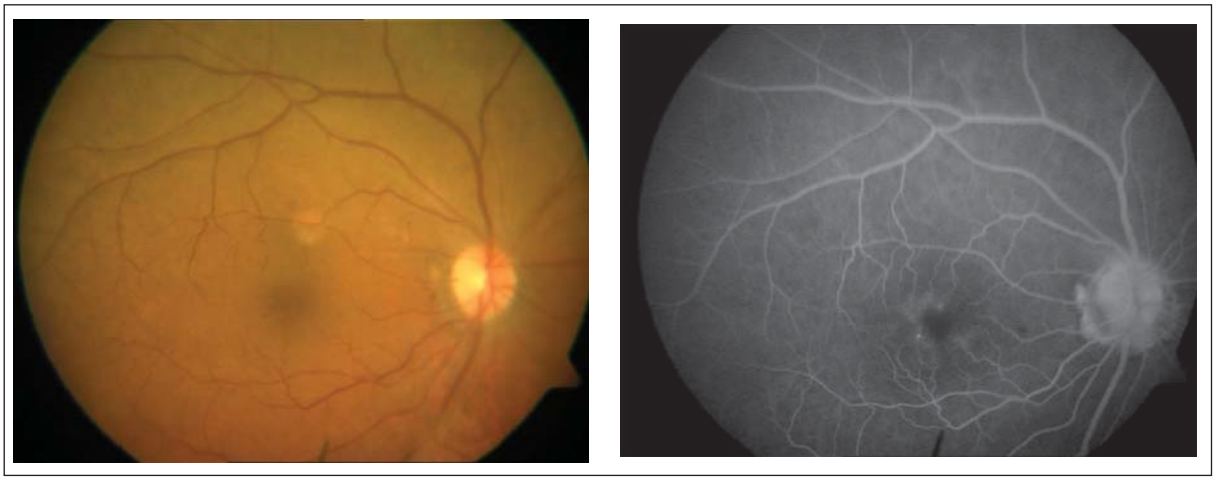

Figure 2a: Fundus photograph and FA showing resolved cystoid macular edema following Posterior sub-Tenon's injections of Triamcinolone

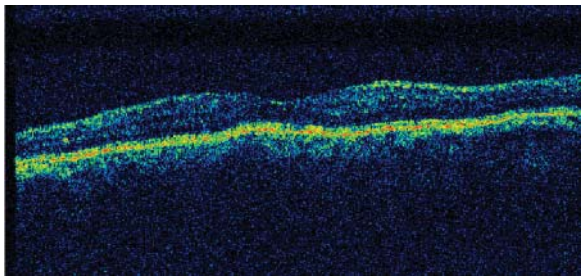

Figure 2b: *OCT also shows resolved macular*

Diagnosis & investigations

Diagnosis is clinical. Investigations must be carried out to rule out other causes of intermediate uveitis.

- Blood counts – WBC abnormality as in malignant masquerade syndrome, ESR, Montoux
- Syphilis serology (VDRL), Lyme disease serology
- Chest X-ray to rule out sarcoidosis, TB
- MRI for Multiple Sclerosis related changes
- ACE levels, serum lyzozyme, serum calcium levels, gallium scan if sarcoidosis is strongly suspected
- Serum HTLV-1 testing in patients from endemic areas

Fluorescein angiography (FA)

- Fluorescein angiography is useful in determining the presence and extent of CME.
- The angiogram provides information about the integrity of the retinal vasculature. Staining of the vessel walls and/or leakage indicates a perivasculitis.
- Retinal neovascularization and optic nerve edema can be recognized easily.

B-scan ultrasonography

- When media are obscured by vitreous hemorrhage, inflammatory debris, cyclitic membrane, or cataract, B-scan ultrasonography can be useful.
- B-scan ultrasonography helps to rule out retinal detachment, and cyclitic membranes.

Optical coherence tomography (OCT)

- OCT has replaced traditional fluorescein angiography as the imaging modality of choice in establishing a diagnosis of CME.
- OCT can help demonstrate the presence of cysts in the fovea and measure macular thickness. OCT is a highly sensitive, noninvasive method to help diagnose CME and provides the best method to monitor the therapeutic response of patients to treatment as macular thickness appears to correlate with visual acuity to some degree.
- OCT can also help demonstrate the presence of epiretinal membranes, a complication of chronic ocular inflammation.

Ultrasound microscopy (UBM)

- UBM may show features that are not clinically obvious, such as uveal thickening, the exact nature of inflammatory condensations in the vitreous, and vitreoretinal adhesions with traction.

Treatment

Being a chronic inflammatory disorder, the aim of the treatment in IU is to eliminate or diminish vision threatening complications and preserve vision. If CME is treated until resolution and kept from returning by adequate control of inflammation, the long

Immunosuppressive therapy for Pars planitis			
Medication	**Mechanism of action**	**Dosage**	**Complications**
	Antimetabolites		
Methotrexate	Folate analog; inhibits dihydrofolate reductase	7.5-25mg/week	GI upset, fatigue, hepatotoxicity, pneumonitis
Azathioprine	Alters purine metabolism	100-250mg/day	GI upset, hepatotoxicity
Mycophenolate Mofetil	Inhibits purine synthesis	1-3g/day	GI upset
	Alkylating agents		
Cyclophosphamide	Cross links DNA	1-2mg/day	Hemorrhagic cystitis, sterility, risk of malignancy
Chlorambucil	Cross links DNA	2-12mg/day	Sterility, risk of malignancy
	Inhibition of T-lymphocytic signals		
Cyclosporine	Inhibits NF-AT (nuclear factor of activated T lymphocytes) activation	2.5-5mg/Kg/day	Nephrotoxicity, hypertension, gingival hyperplasia, GI upset, paresthesis
Tacrolimus	Inhibits NF-AT activation	0.1-0.2mg/kg/day	Nephrotoxicity, hypertension, Diabetes mellitus

term visual prognosis can be good, with nearly 75% of patients maintain visual acuity of 20/50 or better.

Topical corticosteroids may be required for management of anterior uveitis if present.

Kaplan's 4 step approach for treatment of vision loss secondary to pars planitis.[6]

Treatment should be instituted when vision drops to 20/40 (6/12) that lasts 1-2 months. Mild cases without CME may require no treatment.

- Corticosteroids
- Cryotherapy
- Surgery – pars plana vitrectomy
- Immunosuppressants

Corticosteroids

Corticosteroids can be given by oral or periocular route. Periocular steroids minimize the systemic complications. Posterior sub-Tenon's (PST) injections of steroids are repeated every 4-6 weeks until CME resolves or vision returns to 20/20 (6/6).

Triamcinolone acetonide has small particle size and lacks carrier vehicle (which can cause orbital inflammation) and hence it is preferred for PST. The drug is also well tolerated in the vitreous cavity. Vehicle of methyl prednisolone acetate depot preparation causes severe retinal & retinal pigment epithelial scarring & atrophy when injected intravitreally.

Periocular injection complications: Ocular perforation, glaucoma, cataract

Systemic therapy is initiated if local measures are not effective and reserved for bilateral or more severe cases. Systemic prednisolone is given 1mg/kg in divided or single dose, and tapered over 6-12weeks depending on response.

Complications of systemic therapy: Iatrogenic cushing syndrome, DM, GI upset or ulceration, mood swings, psychiatric difficulties, weight gain, insomnia, HTN, congestion heart failure, osteoporosis, muscle weakness, acne, growth suppression in children.

Ocular cataract & glaucoma

In cases of severe inflammation, intravitreal triamcinolone (4mg in 0.1ml) is effective. The effect is usually transient, lasting around 3 months, but can be repeated although the side effects of cataract and raised intraocular pressure are increased in frequency with intraocular versus periocular corticosteroid injections. This has led to the development of new intraocular corticosteroid devices which are designed to deliver sustained-release drugs and obviate the need for systemic immunosuppressive treatment. The first such implant was Retisert, which is surgically implanted (in the operating theatre) and is designed to release fluocinolone over a period of about 30 months. More recently, Ozurdex, a 'bioerodible' dexamethasone implant which can be inserted in an office setting, has completed phase III clinical trials in patients with intermediate and posterior uveitis. This implant lasts approximately 6 months, and has been found to be effective with a much better side effect profile than Retisert or intravitreal triamcinolone injection, at least for one injection. [7]

Cryotherapy

When steroid therapy fails, cryotherapy can be applied peripherally at vitreous base to ablate the snowbank (peripheral laser photocoagulation is also effective).

Pars Plana Vitrectomy

Pars plana vitrectomy removes vitreous membranes, vitreous opacity in visual axis & eliminates vitreous traction. Other indications are retinal detachment, non resolving vitreous hemorrhage and CME not responding to medical therapy. Surgery has the theoretical benefit of removing vitreous antigens, inflamed cells & mediators. Vitreous separation of posterior hyaloid may also help in reducing CME. [8]

Immunosuppressants

Severe uncontrolled disease/corticosteroids intolerance and patients who do not respond to other treatment modalities are managed with immunosuppressive therapy.

Methotrexate, Cyclosporine, Azathioprine, Chlorambucil, Cyclophosphamide, Tacrolimus and mycophenolate mofetil are drugs used for refractory pars planitis. Combination therapy (prednisolone + immunosuppression/or 2-3 different immunosuppressants) can be used for severe disease.

Treatment failure should not be assumed at any step without at least 3-6 month of giving treatment.

Differential diagnosis of IU (Pars planitis)

Unilateral conditions

- Coat's disease - unilateral & intraretinal angiomatosis
- Intraocular tumours - Retinoblastoma, malignant melanoma, medulloepithelioma
- Fuchs heterochromic iridocyclitis
- Infection by P. acnes
- Retained intra-ocular foreign body or chronic RD

Unilateral or Bilateral conditions

- Sarcoidosis should be suspected in all cases with IU
- Lyme's disease
- Cat scratch disease
- Wegener's granulomatosis, Behcet's disease, Inflammatory bowel disease – significant vitritis is present but there is no snow banking
- Large cell lymphoma in patients older than 40 years may mimic pars planitis. Vitreous membranes simulating snow bank may occur.

Bilateral conditions

- Senile vitritis – idiopathic vitritis of older patients. There is absence of snowbanking and retinal vasculitis, fewer snowball & CME may be present. Senile vitritis is a diagnosis of exclusion.
- Amylosis can cause vitritis, however no snowballs or CME is present.
- Whipple disease – Absence of snowball opacities.

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Retinal Vein Occlusions: Current Treatment Modalities

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Retinal vein occlusion (RVO) is the second most common retinal vascular disease after diabetic retinopathy. Macular edema leads to vision loss in many patients with either central or branch retinal vein occlusions (CRVO or BRVO). BRVO is the more common of the two presentations, accounting for approximately 80% of RVO.

Therapies in BVOS have two aims: to reduce macular edema, and to reduce macular edema, and to prevent NV caused by retinal ischemia. In the past, grid pattern laser photocoagulation was the only treatment option available for either condition based on the findings of the BVOS. However, new medical and surgical therapies have emerged in recent years, and more prospective treatments are on the horizon.

Laser photocoagulation

The BVOS remains the largest randomized prospective trial that evaluated the efficacy of grid pattern argon laser photocoagulation for the treatment of macular edema. After a 3 year follow up period, 65% of treated eyes gained 2 or more lines of vision, as opposed to 37% of untreated eyes.

The Branch Vein Occlusion Study (BVOS) showed that laser photocoagulation was a viable treatment option for macular edema secondary to branch retinal vein occlusion (BRVO)[2]. This finding was reaffirmed by recent results from the Standard Care vs. Corticosteroid for Retinal Vein Occlusion (SCORE) study[3]. Results from the SCORE study showed that laser treatment was comparable to steroid injections for the treatment of macular edema secondary to BRVO. Furthermore, the overall safety profile was greater for the laser treated group. So, based on these data, laser treatment remains the standard of care for macular edema associated with BRVO.

Although clinical trials, such as the Central Vein Occlusion Study (CVOS), showed moderate effectiveness of laser photocoagulation in patients with macular edema secondary to CRVO, there was no statistically significant effect on visual acuity when compared to untreated patients[4].

Intravitreal Steroids

Triamcinolone acetonide (TA), a long acting steroid derivative, has anti inflammatory effects, reduces the permeability of endothelial cells, and stabilizes the blood retina barrier. Intravitreal TA has been associated with risks of acute and sterile endophthalmitis, retinal tears and detachment, vitreous hemorrhage, cataract, and increased intraocular pressure.

The SCORE Study

To evaluate the clinical benefits of triamcinolone for treating macular edema associated with vein occlusion, researchers organized a multi-center clinical trial known as the Standard Care vs. Corticosteroid for Retinal Vein Occlusion (SCORE) study. This study, conducted at 84 clinics across the country, was sponsored by the National Eye Institute, part of the federal government's National Institutes of Health.

Impact on Clinical Practice

Through the SCORE CRVO trial, scientists identified the first long-term, effective treatment to improve vision and reduce vision loss associated with macular edema due to CRVO. A large clinical trial had never before shown that patients with CRVO could experience a visual improvement with treatment. Therefore, clinicians may now offer people who have macular edema associated with CRVO a low-dose corticosteroid injection that could increase their chance of visual improvement.

The SCORE BRVO trial was the first large, long-term clinical trial to show that laser treatment and eye injections of corticosteroids have a similar impact on vision for patients who have macular edema due to BRVO. However, laser treatment may still remain the best proven treatment option because it is associated with fewer complications for patients. In future clinical trials, laser may also serve as the best benchmark with which other BRVO treatments should be compared.

Sustained Intravitreal Drug Release Devices

In June 2009, Ozurdex (dexamethasone, Allergan) became the first FDA-approved intravitreal steroid implant for the treatment

Figure 1: *Ozurdex implant with applicator*

Tirupati Eye Centre, Noida
*Sai Retina Foundation, Delhi

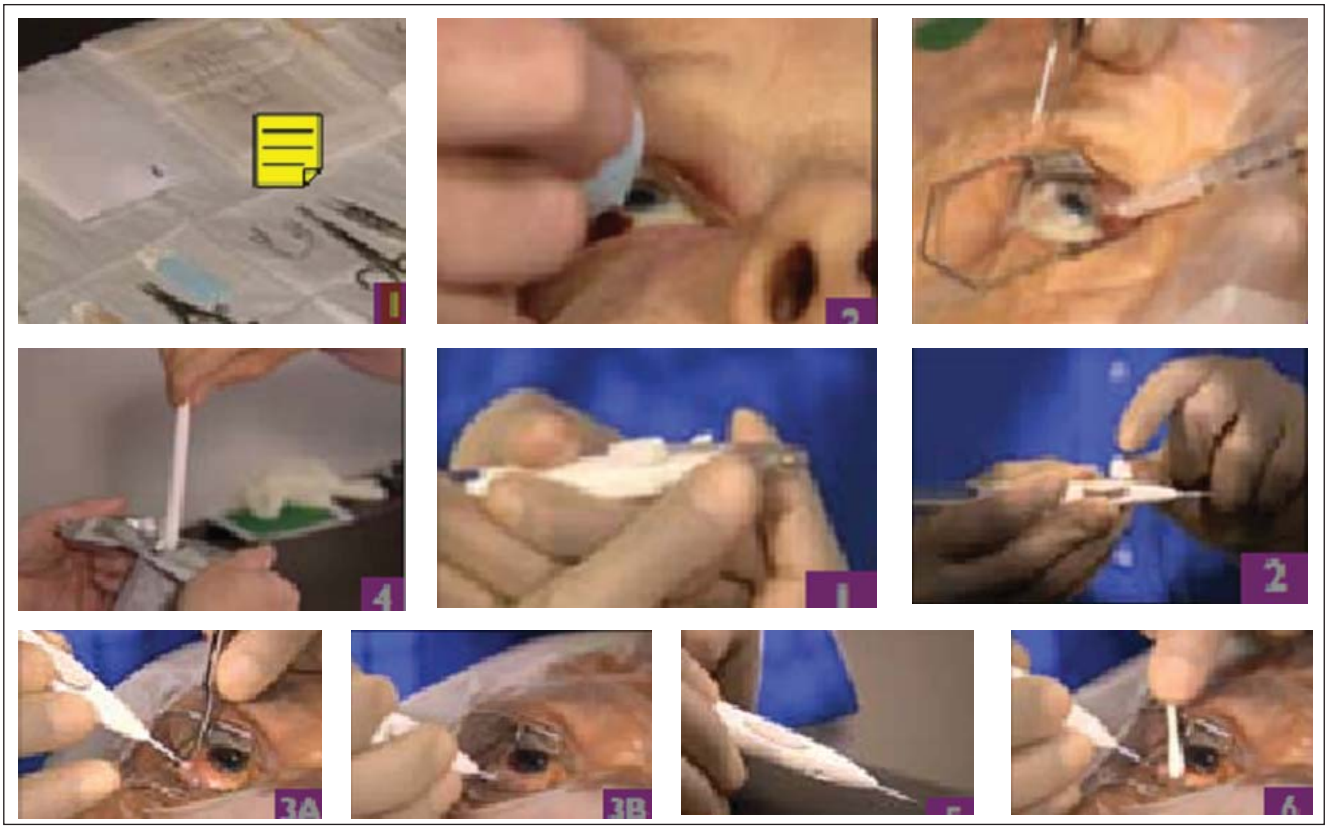

Figure 2: *Stepwise intravitreal injection of Ozurdex*

of macular edema secondary to retinal vein occlusion5. Ozurdex is a biodegradable polymer implant that is injected through a 22-gauge applicator (Figure 1). It contains 0.7 mg dexamethasone in the NOVADUR™ solid polymer drug delivery system which biodegrades into lactic acid and glycolic acid.

The delivery system contains dexamethasone, a potent, highly soluble corticosteroid with a short half-life. The effects of Ozurdex typically persist for one to three months, but longer periods of action have been shown clinically[6,7,8]. Also, because its therapeutic effects may last more than three months, fewer re-injections are required.

FDA approval of Ozurdex was based on two parallel clinical trials[5]. Approximately 1,300 subjects participated in these two double-masked, randomized studies[5]. Two thirds of the subjects had macular edema associated with BRVO and one third had macular edema associated with CRVO. Most subjects had macular edema for more than three months.

Subjects were randomly assigned to receive either a single Ozurdex implant or a placebo injection. The studies showed that subjects in the treatment group achieved a visual improvement of three lines or more compared to patients in the placebo group[5,7].

This improvement was observed in 20% to 30% of treated subjects within one to two months, compared to just 10% of the subjects in the placebo group. Optimal visual improvement was observed at day 60, and a significant difference between the two groups was noted by day 90[7].

Stepwise technique of intravitreal injection of Ozurdex (Figure 2):

Step 1: Both preparation and the intravitreal injection procedure should be carried out under controlled aseptic conditions, which include the use of sterile gloves, a sterile drape, and a sterile eyelid speculum (or equivalent).

Step 2: Administration of a broad-spectrum microbicide is recommended prior to the injection.

Step 3: Subconjunctival` anesthesia an also be administered before the intravitreal injection. This is an optional step as injection can be carried out under topical anesthesia also. Subconjunctival anesthesia was used in the phase 3 clinical trials of Ozurdex intravitreal implant.

Step 4: Remove the foil pouch from the carton and examine it for damage. Then, in a sterile f field, open the foil pouch and gently place the applicator on a sterile tray.

Step 5: Maintaining aseptic technique, carefully remove the cap from the applicator.

Step 6: Hold the applicator in one hand and pull the safety tab straight off the applicator. Do not twist or flex the tab.

Step 7: Holding the long axis of the applicator parallel to the limbus, engage the sclera at an oblique angle with the bevel of the needle up (away from the sclera) to create a shelved scleral path. Advance the tip of the needle within the sclera for about 1 mm (parallel to the limbus), then redirect toward the center of the eye

and advance until penetration of the sclera is completed and the vitreous cavity is entered. The needle should not be advanced past the point where the sleeve touches the conjunctiva.

Step 8. Slowly depress the actuator button until an audible click is noted.

Step 9. Before withdrawing the applicator from the eye, make sure that the actuator button is fully depressed and has locked flush with the applicator surface.

Step 10. Remove the needle in the same direction as used to enter the vitreous.

Step 11. Properly dispose of applicator.

Each applicator can only be used for the treatment of a single eye. If the contralateral eye requires treatment, a new applicator must be used, and the sterile field, syringe, gloves, drapes, and eyelid speculum should be changed before ozurdex is administered to the other eye.

Caution

The most common ocular adverse reactions reported by greater than 2% of the patients in the first 6 months included increased intraocular pressure (25%), conjunctival hemorrhage (20%), eye pain (7%), conjunctival hyperemia (7%), ocular hypertension (4%), cataract (4%), vitreous detachment (3%), and headache (3%). Patients should be monitored following the injection. Corticosteroids should be used cautiously in patients with a history of ocular herpes simplex. Corticosteroids should not be used in active ocular herpes simplex.

Recommended follow up

Patients should be monitored for elevation in intraocular pressure and for endophthalmitis. Monitoring must consist of the following:

- Reperfusion check of the optic nerve head immediately after injection.
- Tonometry within 30 minutes following the injection.
- Biomicroscopy between 2 and 7 days following the injection Patients should be counseled regarding the risk of potential complications, particularly endophthalmitis.
- Inform patients of the need to be vigilant for new symptoms in the days following intravitreal injection.
- Stress the importance of seeking immediate care from an ophthalmologist if the eye becomes red, sensitive to light, painful, or develops a change in vision.

Also advise patients that they may experience temporary visual blurring after receiving an intravitreal injection.

Standard of care continues to evolve. Current clinical trial results and the approval of new therapies, such as Ozurdex, provide new, viable treatment options for patients who present with macular edema secondary to retinal vein occlusion.

Intravitreal anti VEGFs

Recently, there has been interest in the use of vascular endothelial growth factor (VEGF) inhibition in the treatment of RVO because of the observation of increased VEGF in the vitreous and aqueous of patients with these conditions[4]. Two randomized controlled trials assessed the efficacy and safety of intravitreal ranibizumab (Lucentis, Genentech) in BRVO and CRVO. The results showed that with intensive, monthly treatment, patients achieve very good results; superior to anything we have seen previously with other treatment modalities[6].

However, initial subgroup analysis from BRAVO and CRUISE suggests that there was a definite benefit for patients who presented with a short duration of disease as well as those who presented with long duration, compared with patients with comparable duration of disease in the control arm. In other words, no matter the duration of the disease, treated patients did better than sham-treated patients.

Because patients with RVO are in general younger than our patients with AMD and more likely to be involved in the working world, it is often important that they recover their vision quickly so that they can drive to work and function in the workplace. The month or two of improved visual acuity that an early injection of a VEGF inhibitor can potentially deliver might help these patients keep their jobs.

As with any treatment, the risk-benefit ratio must be considered. In these studies the rates of adverse events like endophthalmitis, retinal tear, vitreous hemorrhage were very low, but the benefit rate and the chance of improving BCVA was high. So from the point of risk-benefit ratio there is not a great rationale for delaying treatment, unless the patient's other eye is healthy and the RVO is not affecting his or her lifestyle.

The BRAVO and CRUISE trials used monthly dosing schedules, In practice, most physicians will probably give their RVO patients several monthly injections as a loading dose and then either treat on an as-needed basis (PRN) or with a treat-and-extend strategy. In patients with BRVO who require continued treatment, the physician may decide to add grid laser photocoagulation. When laser is added to anti-VEGF therapy in BRVO patients, it is recommended to perform laser a week or so after the anti-VEGF injection.

The SCORE study found laser alone superior to steroids in patients with BRVO, but that study did not compare laser with anti-VEGF regimen. It is unlikely that laser would provide a benefit in BRVO that would be as robust in the short term as the response to anti-VEGF treatment in BRAVO. In the long term, for instance at 2 years, laser might yield a similar response to anti- VEGF therapy, but as noted earlier, working-age patients and indeed, patients of any age may benefit from earlier visual recovery.

Regarding the choice of anti-VEGF agent, the Comparison of AMD Treatment Trials (CATT) is testing the efficacy of ranibizumab versus bevacizumab in AMD. The results of these trials, expected by next year, may shed some light on the relative efficacy of these two agents in other diseases such as RVO.

Steroids versus anti VEGfs: which to choose?

It is still not clear as to which therapy has an edge. In terms of complication rate anti VEGFs seem to score better. In the aforementioned landmark studies, BRAVO and CRUISE, after six

months of ranibizumab therapy, between 55% and 61% of patients with branch retinal vein occlusion (BRVO) gained at least three lines of BCVA, and about 47% of patients with central retinal vein occlusion (CRVO) gained at least three lines of BCVA[2,3].

Ozurdex data found 41% of patients gained at least three lines of vision.[4] However, adverse effects occurred more frequently. Twenty-five percent of patients experienced elevated IOP, 20% developed a conjunctival hemorrhage and 4% required cataract surgery[5]. By contrast, among nearly 800 patients studied in the BRAVO and CRUISE trials, researchers found only one case of retinal detachment, one case of endophthalmitis and one case of vitreous hemorrhage[2,3].

From a pragmatic standpoint, steroids retain considerable appeal, mainly due to their much easier dosing schedule. Patients with RVO tend to be younger, with more family and work obligations than age-related macular degeneration patients. The monthly hassle of intravitreal anti-VEGF injections may prove onerous to some. Also, retina specialists with a large cohort of AMD patients receiving anti-VEGF injections on a monthly schedule might lack the capacity to introduce another regimen that requires frequent injections.

Meanwhile, studies have found that 20% of Ozurdex recipients required only a single injection for an entire year (4). The implant is designed to be delivered once every six months, but that is still a great advantage over the nine or 10 injections a year necessary in ranibizumab therapy.

Much depends on the patient's needs, since every eye is different; we need to customize therapy as we follow patients along.

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Tarsorrhaphy: A Corneal Surgeon's Viewpoint

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Tarsorrhaphy is a surgical procedure which consists of fusion of upper eyelid with inferior eyelid.[1] This is a simple technique that is performed separately or combined with other surgeries to treat a number of oculoplastic diseases such as lagophthalmos, facial paralysis, ectropion, entropion, proptosis, thyroid disease and other conditions.[2,3]

In services of Cornea and External Disease tarsorrhaphy is used as a treatment option for obtaining a protection of the cornea or to accelerate the healing of corneal ulcers. It is a useful tool for treatment of persistent epithelial defects secondary to exposure keratitis, neurotrophic keratopathy and dry eye syndrome. It can be combined with penetrating keratoplasty (PK) and other surgeries to promote surface healing.[4] Generally, tarsorrhaphy is underused as a prophylaxis and treatment option for recalcitrant surface healing problems.

Healing of wounded epithelium

In normal eyes, small to moderately sized abrasions are expected to heal within 12 to 24 hours. Larger defects may require a longer time to heal but usually heal within a week or two. Persistent (non healing) epithelial defects (PED) result when the epithelium fails to regrow over a defect within the expected time course.[5] Out of the several factors, main factors considered responsible for poor-healing of an epithelial defect are loss of endogenously released various growth factors, essential neurotrophic factors released locally within the epithelium from the trigeminal nerve, increased exposure, and dry eye.[6] Persistent epithelial defects and stromal ulceration have numerous causes (Table). Immune or neurologic disease (e.g. neurotrophic cornea, facial palsy), tear film anomalies, infections, metabolic disease, medications, contact lens wear, trauma, neoplasms, and chronic eye rubbing may produce persistent defects and stromal ulceration. They may be idiopathic as well.[7]

Persistent epithelial defects occur frequently and are difficult to treat, and result in significant ocular morbidity and visual loss.

Persistent epithelial defect post penetrating keratoplasty

Epithelial defects frequently occur in the donor cornea in the early and, less commonly, in the late postoperative period after penetrating keratoplasty (PK). Reepithelization in early postoperative period is critical for wound healing, improved visual acuity, graft transparency, graft survival, and protection against infection and melting. If the recipient surface has a healthy ocular surface, re-epithelisation is uneventful. In patients with ocular surface disease PED may lead to graft failure. In patients in whom problems with epithelisation are anticipated, a permanent or temporary tarsorrhaphy at the time of keratoplasty or early in the postoperative period is the most powerful and prophylactic treatment modality.[8]

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Causes of Persistent Epithelial Defects

- Medications
  - Topical
  - Systemic
- Contact lens wear
- Chronic eye rubbing
- Traumatic
  - Mechanical
  - Chemical
  - Radiation
- Metabolic
  - Intrinsic
  - Acquired
- Post infectious
- Immune
  - Rheumatoid diseases
  - Non rheumatoid collagen vascular diseases
  - Mooren's ulcer
  - Dermatologic
- Neurological
  - Neurotrophic
  - Neuroparalytic
- Tear film anomalies
- Lid- surfacing abnormalities
- Idiopathic

Strategies of treatment for PED include removal of any identifiable aetiologies and promotion of epithelial healing. For protection of corneal surface and promotion of healing various options are described in the literature, including: ointments and lubricants, autologous serum, contact lens, punctual occlusion, amniotic membrane transplantation, conjunctival covering and tarsorrhaphy.[9,10]

Role of tarsorrhaphy

In nonhealing epithelial defects, tarsorrhaphy is reported to have 80% to 100% success rate for complete healing.[11,12]

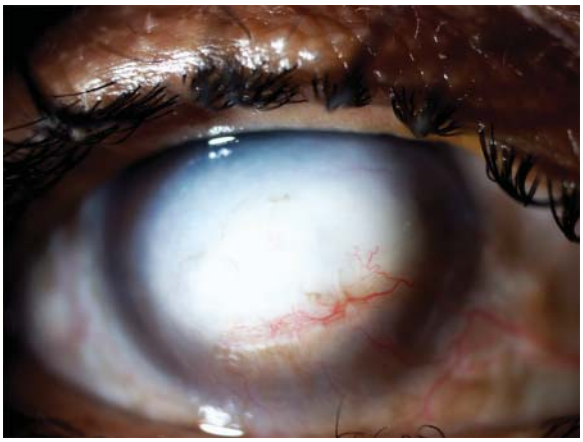

Figure 1a: *Preoperative Status*

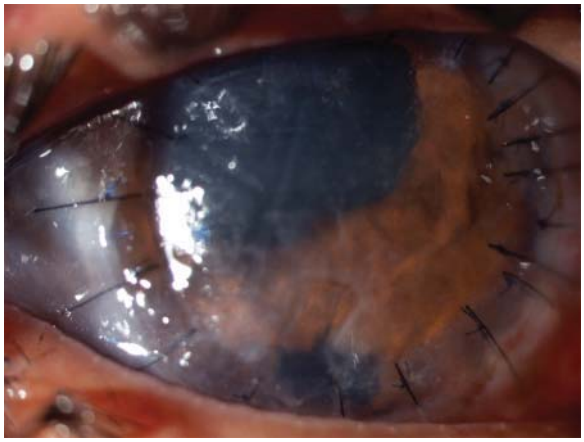

Figure 1b: *Non Healing Defect Post Keratoplasty*

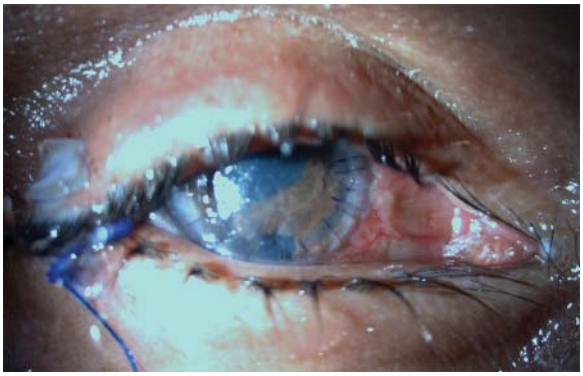

Figure 1c: *Post Tarsorrhaphy*

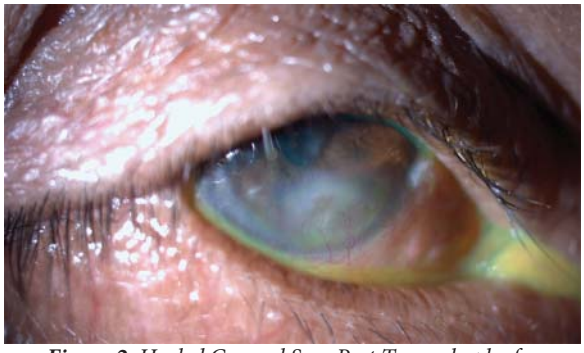

Figure 2: *Healed Corneal Scar Post Tarsorrhaphy for Persistent Epithelial Defect Due to Facial Nerve Palsy*

Tarsorrhaphy mainly helps by

a. Decreasing the palpebral fissure width and hence decreasing the evaporation rate of tears. Preservation of patient's own tears along with lubricants brings the exposed cornea into contact with the superior and inferior lacrimal strips even with small movements of the eye, continuously redistributing the tear film over the epithelial defect.

b. Immobilization of the lid over the epithelial defect decreases the traumatic effect of the moving lids on the healing epithelium.

There may be other mechanisms related to the neurotrophic effects on re epithelisation but they require further understanding.

Due to above described mechanisms, tarsorrhaphy is an effective modality in the treatment of persistent epithelial defects in cases of exposure, xerosis, neurotrophic keratopathy and post penetrating keratoplasty. It also has a role in the treatment of chronic corneal ulcerations and prevention of perforations. Small descmetoceles and very small peripheral perforations heal after tarsorrhaphy as it provides a smooth tarsal conjunctival surface which protects descmet's membrane and promotes re epithelisation.[13]

Types of tarsorrhaphy[14,15]

Tarsorrhaphy aims at closing eyelids, either temporarily or permanently. Four basic types of tarsorrhaphies are:

I. *Temporary (short-duration) tarsorrhaphy without sutures:* Lids closed with tape or adhesive glue. It lasts for a few days. Temporary glue tarsorrhaphies although have an advantage of being an easily performed outdoor procedure, run the risk of glue migrating onto posterior aspect of the lids and premature separation.

II. *Temporary suture tarsorrhaphy, with or without a bolster:* Suture materials like catgut, silk, nylon or prolene can be used. It may last up to 4 to 6 weeks.

III. *Permanent suture tarsorrhaphy:* It is performed by excising opposing lid margins so that they heal together and form a strong adhesion. Absorbable sutures that are buried or non absorbable sutures over bolsters or with exposed knots can be used. These tarsorrhaphies can be opened later on.

IV. *Permanent suture tarsorrhaphy with lamellar resection:* It is the most extensive type of tarsorrhaphy. It involves mobilization

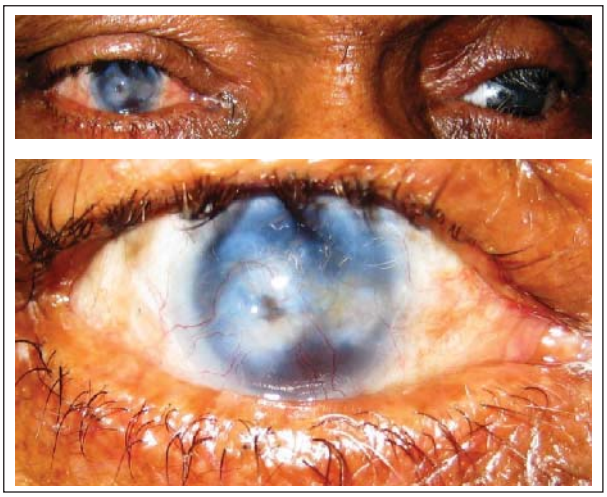

Figure 3a: *Preoperative Picture of a one Eyed Patient with Vascularized Corneal Scar in Right Eye and Prosthetic Eye in Left Eye*

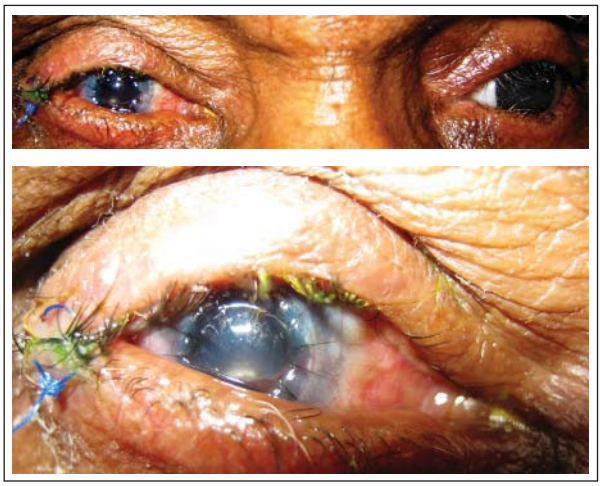

Figure 3b: *1 Day Postoperative Picture: Status Post Keratoplasty and Simultaneously Performed Lateral Tarsorrhaphy*

of skin or tarsal plate flaps. Eyelids are split into anterior and posterior lamellae. Either lamella can be resected in the upper or lower lid and filled with advancement of the opposite lamella. It is difficult to reverse these tarsorrhaphies at a later date.

A tarsorrhaphy should be performed in a manner that results in maximal coverage of the defect. Depending on the temporal or nasal location of defect, a lateral or medial tarsorrhaphy can be performed. Some cases may require the placement of both lateral and medial tarsorrhaphies.

A temporary or permanent can be performed depending on whether the clinical diagnosis is a short-term problem or a long-term irreversible condition. It can be used alone or in conjunction with other techniques both before and after perforation such as tissue adhesive, conjunctival flap, amniotic membrane transplantation, penetrating keratoplasty and patch grafting.

A lateral or central suture tarsorrhaphy with or without bolsters can be performed along with penetrating keratoplasty in high risk cases. The sutures remain effective for several weeks during the critical re-epithelisation process and then can be easily removed without sequelae. Keratoplasty should always be combined with permanent tarsorrhaphy in a one eyed patient with extensive corneal vascularisation and/ or ocular surface problems.

Commonly seen complications of tarsorrhaphy can be localized trichiasis, lid margin deformities, suture granulomas, focal cellulitis, premature separation of tarsorrhaphy and distichiasis.[16] Entropion can also occur after lysis of a permanent tarsorrhaphy.

Advantages of tarsorrhaphy over patching

In an Indian study, complete healing of epithelial defects and symptomatic relief after keratoplasty was found to be faster in the tarsorrhaphy group than in the patching group.8 Another study comparing bandage contact lenses, pressure patching, and temporary tarsorrhaphy for non healing epithelial defects after epikeratoplasty concluded that the eyes with the tarsorrhaphy healed significantly faster.[17]

Tarsorrhaphy definitely has advantages over patching as it allows more oxygen to get to the corneal epithelium than a totally closed or patched eye. Allowance for the administration of eye drops, retention of partial eye sight, and examination of the cornea by having the patient adduct the eye are other advantages of tarsorrhaphy over patching. Also the factors like reduced tear turnover, decreased elimination of metabolic waste products, and high local temperature with patching are eliminated with tarsorrhaphy.[9]

The use of therapeutic bandage contact lens is effective but increases the risk of infectious keratitis.

Conclusion

Tarsorrhaphy is a very effective and safe procedure in the management of nonhealing epithelial defects and other surface problems with good success rate and minor complications. It should be performed sooner rather than later when persistent epithelial defects do not respond to medical therapy.

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Topical and Intracameral Anaesthesia for Cataract Surgery

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Topical and intracameral drug administration is now the preferred method for anaesthesia in cataract surgery and an increasingly preferred method for anaesthesia in anterior and posterior segment surgeries. First introduced in Europe at the end ofthe19th century when cocaine was widely available to ophthalmologists[1], it almost fell out of use in favour of retrobulbar injections following the introduction of newer and less toxic anaesthetic agents such as procaine[2]. Modern topical anaesthesia in cataract surgery began in 1991, when Fichman performed a series of phacoemulsifications under topical anaesthesia using 0.5% tetracaine[3]. Intraocular irrigations of anaesthetic agents to improve analgesia were postulated in 1993[4], and the first results on a large series of patients were published in1997[5,6].

Bases of Topical/Intracameral Anaesthesia in Cataract Surgery

Physiological Bases

Ocular sensitivity is based on terminations of the 5th cranial nerve, especially distributed to the cornea and to the ciliary body in the anterior part of the eye. These fibres are generally non-myelinated, types A-delta and C. They are able to carry sensation of pain, temperature and touch, and are blocked by lower concentrations of drugs in comparison with motor fibres.

Pharmacological Bases

Sensory termination block is the most important feature of topical anaesthesia. It involves the inhibition of sodium channels at nerve endings or receptors by the anaesthetic agents, thus blocking the production (and not the transmission) of nervous impulse.

Surgical Bases

In cataract surgery, the absence of ocular akinesia makes small incision surgery with phacoemulsification mandatory. Usually two incisions are made, allowing the surgeon to stabilize and to direct the eye with two instruments. Not only is akinesia no longer needed, but the retained ocular motility can be of help in some passages of surgery if the patient follows surgeon's instructions. The reduced length of the incisions makes them less painful, as lower numbers of nerves are cut compared with wider incisions. During surgery, instruments are moved as levers through the incisions thus pre-venting fluid leakage and excessive intraocular pressure variations, a possible cause of ciliary pain. Careful hydrodissection prevents excessive ciliary body stimulation by zonular fibres during nucleus rotation. During surgery, a decrease of ocular sensitivity is provided by the frequent use of cold irrigation solution. In addition, there are no painful maneuvers typical of extracapsular surgery like muscle sutures, conjunctival incisions, iris manipulations and tissue sutures. The advantages of topical anaesthesia over periocular injections include not only a higher safety level, but also better consistency of analgesia during surgery and lower intraocular pressure. Moreover, the limited amount of drug employed inhibits the general side effects commonly observed with local anaesthesia. The return of sensitivity soon after surgery makes it possible to immediately detect any unexpected ocular pain suggestive of complications.

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Drugs Employed

The chemical compounds employed for ophthalmic topical anaesthesia are tertiary amines composed of an aromatic hydrophobic ring, usually benzene, and an amidic hydrophilic group, with an ester (proparacaine, tetracaine, benoxinate) or an amidic (lidocaine, etidocaine, mepivacaine, ropivacaine) intermediate chain. The ester compounds are rapidly hydrolysed by plasmatic esterases, and to a lesser extent by tissue esterases. The amide compounds are degraded more slowly, and mainly outside the eye in the liver, and therefore are endowed with longer duration of action.

Ester-Bound Compounds

Tetracaine 1% and 2%

Tetracaine was the first anaesthetic employed topically for cataract surgery[7], but at present it is less used because of its short duration of action and because of the esterase deficiency that can lead to toxic reactions in some patients. After its action begins within 1min and lasts for10–15min. Tetracaine is considered more toxic than other agents for the corneal epithelium.

Proparacaine 0.5%

It is considered safer than other ester-bound compounds. It is less irritating and less painful on instillation than benoxinate, and does not show bacteriostatic properties. The onset of action is a matter of seconds, but the duration is usually shorter than 10min.

Benoxinate 0.4%

Its instillation is painful, and its corneal epithelium toxicity is also high. It has bacteriostatic properties. After touching the cornea, the anaesthetic action takes place in seconds, and lasts up to 10min.Being rapidly degraded by ocular esterases, its activity on intraocular structures is less strong than other compounds.

Amide-Bound Compounds

Lidocaine 1%–4%

Currently, lidocaine may be the most employed topical anaesthetic in cataract surgery, and it is the most employed for intracameral

irrigations[8]. Instillation is rather painful because the pH of the solution is usually below 6. At the corneal surface, the onset of anaesthesia is slower than with ester compounds. After eyedrop application, lidocaine crosses rapidly the corneal epithelium and stroma, exerting its sodium channel blockade on the cornea by first. As a result, temporary epithelial and stromal swelling can sometimes be observed even with unpreserved preparations. Lidocaine is not degraded within the eye, and therefore it can exert its anaesthetic effect on anterior chamber structures for a long period, up to 20min.

Bupivacaine 0.50%, 0.75% and 2%

Bupivacaine has been extensively used as local anaesthetic because of its potency and long duration of action, and despite the relatively slow onset of activity. The intraocular penetration is good because bupivacaine is extremely liposoluble. The duration of the effect is about 10min longer than with other amide agents.

Ropivacaine 1%

Ropivacaine is a long-lasting anaesthetic agent that provides up to 12h of postoperative analgesia. The onset of action is rather slow as it happens with bupivacaine, but ropivacaine has lower cardiac and central nervous system toxicity.

Mepivacaine 2%

Mepivacaine is an amide anaesthetic with rapid onset of activity. Because of its poor corneal penetration, its use in cataract surgery has been mainly limited to intraocular injections in patients showing pain after topical anaesthesia with 2% bupivacaine[9].

Influence of Formulation

pH

Topical anaeshtetic drops frequently have pH between 5 and 7, that contributes to the burning sensation on the first eyedrop application. The pH of these solutions can be raised by further diluting the drugs in BSS or BSS plus, approaching the physiologic normal of 7.2–7.4. However, some anaesthetic agents could be unstable in solutions at pH above 7 at certain concentrations; this is especially true for bupivacaine and mepivacaine, while lidocaine can be buffered to 7.4 without precipitating even at the 4% concentration.

Preservatives

Corneal epithelial swelling has been frequently observed with preserved formulations of lidocaine, and the intraocular safety of preservatives remains even more controversial. Although some preservatives like benzalkonium chloride increase corneal penetration, the use of unpreserved formulations of anaesthetic agents both for topical application and for intraocular irrigation5 is recommended.

Temperature

Temperature influences the stability of solutions. A warm solution is probably more stable, better tolerated by patients and therefore more active. Therefore ampoules should not be refrigerated immediately before use.

Routes of Administration

Eyedrop Instillations

Usually the anaesthetic agent is applied to both eyes, to prevent blinking and Bell's phenomenon elicited by the non-operated eye. This practice allows the patient to keep both eyes open without effort during surgery. However, because of corneal epithelial toxicity of the drugs and lack of hydration, some vision impairment in the nonoperated eye for the first postoperative week has to be anticipated in the patient. Unpreserved eyedrops of the selected drug are instilled in the 10–60min preceding surgery.

Anaesthetic agents of the ester group cannot have systemic side effects at commonly employed doses, as they are rapidly degraded by tissue and plasma esterases. Anaesthetics of the amide type are metabolised in the liver, and some concern arose about possible general effects. With topical anaesthesia the potential risks of needle injections are avoided, but still local side effects can occur. Apart from burning sensation, the instillation of an anaesthetic agent into the conjunctival sac impairs the tear film because of dilution and because of the pH of applied solutions. These epithelial side effects can impair visibility during surgery, and are an argument favouring the reduction of eye drops instillation and the adjunct of intracameral anaesthetic irrigation.

Gel Application

Gel formulations of anaesthetic agents have been employed to prolong the contact between the drug and ocular surfaces. A single application of lidocaine 2% gel into the conjunctival sac has been found as effective as repeated eye-drop instillation in providing anaesthesia for cataract surgery[10,11,12,13,14], with more elevated intraocular drug level[11]. Improvements over eyedrop instillations include less burning on application and less corneal dehydration.

Drug-Soaked Sponges

The advantages of sponges could be a lesser amount of drug in contact with corneal epithelium, although the effects on ocular surface have never been compared to that of eyedrop instillation.

Intracameral Irrigations

The most employed drug is lidocaine at 1% concentration, probably because of simplicity in preparation. Lidocaine 1% is mainly prepared at surgery from 4% solutions by diluting in BSS or BSS plus, with obtained pH of 6.39 and 7.11, respectively[15].Other drugs tested for intracameral irrigation are bupivacaine[16,17] and mepivacaine[9]. After being delivered into the anterior chamber, a part of the anaesthetic drug is rapidly absorbed by iris, ciliary body and cornea, while the drug still present in solution is removed by subsequent anterior chamber irrigations, thus limiting tissue exposure[18]. With the commonly used irrigation of 1% unpreserved lidocaine, anterior chamber levels of the drug are 100 times more elevated than after eye drop application. The intraocular irrigation can be repeated in pro-longed or complicated surgeries, because lidocaine is rapidly removed from ocular tissues by irrigating BSS[18]. Every surgeon should check the pH and the osmolarity of injected solutions. The safety of intracameral irrigations with lidocaine and other anaesthetic agents has been extensively studied starting from the amaurosis encountered in some patients after

posterior capsule rupture[19]. Experimental studies on rabbits showed the lack of toxicity of common preparations both for corneal endothelium[20,21,22,23] and for the retina[17]. Reversible cellular swelling could be observed when the concentration was at least 1%[22], with permanent damage only at 2%.

Viscoelastic-Borne Anaesthesia

Solutions combining ophthalmic viscosurgical devices with lidocaine have recently been developed. The purpose was to avoid the additional step of irrigating the anterior chamber with lidocaine solution, and to prolong anaesthesia time to cover little delays in the completion of surgery. One system is based on methylcellulose[24], while a second system is based on sodium hyaluronate[25].

Patient Selection and Counseling

Topical/intracameral anaesthesia was not appreciated immediately as an universal procedure, but as a procedure requiring patient selection. Grabow[26] was one of the first addressing difficulties in applying topical anaesthesia to some patients, like foreigners and those affected by deafness, dementia and uncontrolled eye movements. In addition, patients unable to co-operate during tonometries or A-scan measurements were not considered good candidates for topical anaesthesia[27]. At present topical and intracameral anaesthesia are considered the standard technique for cataract surgery. Some of the old contraindications remain, but the most part have been overcome by the confidence both of surgeons and of patients. As patients now expect to be operated under topical anaesthesia, little instructions have to be given before surgery. On the contrary, too much dialogue could increase patient anxiety. We tell patients simply that anaesthesia will be present, but with no needle injection; that anaesthesia can be increased at any time during surgery should they perceive pain; that the lack of burning sensation on eye drop application is the proof of achieved anaesthesia; and that eye movement will not affect surgery.

Surgery Adaptation

A few adaptations have to be made to adjust cataract surgery for topical anaesthesia. Usually surgeons appreciate the lower posterior vitreous pressure as compared with peribulbar injections, due to the lower pressure in the orbit. However, the corneal surface rapidly dries during surgery, and must be frequently irrigated. The microscope light frequently causes patient discomfort, especially with subcapsular posterior cataracts and in young patients, and some-times it must be reduced during the first phases of surgery. Toothed forceps are more likely to cause discomfort than notched forceps. Corneal tunnels without conjunctival incisions or diathermy are likely to be better tolerated than scleral incisions. Additional eye drop instillation after any conjunctival opening and before further manipulation must be considered. The eye can be better stabilized by a second instrument within the side port incision than grasping the sclera. Cataract extraction should be made with phacoemulsification, because the manoeuvres required for manual fragmentation could be more traumatising for the eye. IOL implantation should not stretch the incision, as at that point analgesia is lower than at the beginning of the procedure. The lids must remain free from trauma because they are not anaesthetised, a condition evident on draping removal.

Management of Complications

The short duration of topical and intracameral anaesthesia points out the necessity to repeat instillations and/ or irrigations in prolonged surgeries. Even repeated iris touch are painless if sufficient amount of drug is present in the anterior chamber. Topical anaesthesia has not been associated with a higher complication rate in published studies, but complications can nevertheless occur, as with peribulbar anaesthesia, and have to be managed safely and efficiently. Even suprachoroidal haemorrhages have been reported[28]. Posterior capsule rupture and anterior vitrectomy typically cause little or no additional pain. Scleral fixation of the intraocular lens can be achieved with intraocular irrigation of unpreserved lidocaine[29] but profound amaurosis has to be expected. Probably, the only manoeuvres requiring either subconjunctival or peribulbar injection of anaesthetic agents to block the eye is a pars plana incision or incisional enlargement to convert to extracapsular extraction.

At present there is a push from patients towards topical anaesthesia, which is now regarded as the standard technique in many countries. This change in mentality will probably further increase success rates and will extend topical anaesthesia to other procedures on the anterior and posterior segment.

Schedule for topical/intracameral anaesthesia for phacoemulsification

1. Base drug Lidocaine 4% 0.5ml without preservatives
2. Topical instillations one drop in both eyes every 5min at times –15,–10,–5min
3. Intracameral irrigation1% Lidocaine obtained adding 1.5ml of BSS to 0.5ml of the 4% solution (pH6.4) approximately 1–1.5ml used to obtain hydrodissection
4. Topical repeated in case of conjunctival manipulation
5. Intracameral repeated in case of prolonged surgery/ complications

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Management of Preexisting Astigmatism

Harbansh Lal MS, Swapna Parekh, Garima Lakhotia, Ikeda

Phacoemulsification, in recent years has changed the fundamental aim of cataract surgery, that was, the replacement of an opacified crystalline lens with an artificial lens. Now the aim of modern cataract surgery is to have uncorrected visual acuity (UCVA) as good as best corrected visual acuity (BCVA). To achieve this one needs to address the issue of PREEXISTING ASITGMATISM (PEA) at the time of planning cataract surgery.

Incidence of astigmatism

- About 36 to 45% of patients have astigmatism of > 1D, out of which 78% have < 1.5D, 20% have 1.5 - 3.0D and 2% have >3.0 D.
- The 3-year incidence rate of astigmatism was 33.6% (cylinder power of 0.5 D or worse) or 11.5% (cylinder power of 1.0 D or worse). When measured in children age 7 – 9 years. Myopic children had a higher incidence rate of astigmatism than nonmyopes.
- The incidence of astigmatism in adult Navajo population was found to be 4% of with the rule astigmatism of atleast 2 diopters or more in atleast one eye.
- Astigmatism was found to exist in about 63% of the eyes when studied in 1112 patients from a military optometric clinic. It was found that with-the-rule (WTR) and against-the-rule (ATR) astigmatism were the predominant types of astigmatism, and that approximately 70% of astigmatism found required 1.00D of correcting cylinder power or less.

The chief methods of correcting preexisting astigmatism during cataract surgery are:

- Limbal relaxing incisions (LRIs)
- Opposite clear corneal incisions(OCCIs)
- Toric intraocular lens (Toric IOLs)

Limbal relaxing incision

Astigmatic Keratotomy was a routine procedure for correction of astigmatism for patients undergoing radial keratotomy for myopia. These incisions were made parallel to the limbus, in between the radial incisions of the radial keratotomy and had a depth of about 90% of the corneal thickness. Various surgeons, Muller-Jensen et al (2000), Budak et al (2000), Gills et al (2002), Wang et al (2003), Shen et al (2004), Kaufmann et al (2005) have established the effectiveness of limbal relaxing incisions in correction of preexisting astigmatism.

Correction depends upon the length of the incision (4 to 8 mm), the number of incisions(single, doubled, paired doubled), the type of the incision (uniplanar, biplanar, triplanar and hinged), the distance from the centre of the cornea (the nearer the relaxing incision to the centre of the cornea, the greater the relaxing effect), the depth of the incision (about 600 microns depth gives good results) and the age of the patient (more the age of the patient, the greater will be the effect).

It is advisable to use a diamond knife with a guard for more predictable results. Every surgeon needs to establish his own nomogram, however the following nomograms may be helpful and can serve as a useful guide.

Gills nomogram

Amount of astigmatism	Number of incisions	Length of incisions
1D	1	6
2D	2	6
3D	2	7
4D	2	8
>4 D	2	10
		2 mm for every D over 4D at mm OZ

The NAPA nomogram

Nichamin Age and Pach Adjusted Intralimbal Arcuate Astigmatic Nomogram.

With the Rule Astigmatism (10 degrees arc= 1 mm)

Pre Op Cyl (Diopters)	Paired Incisions in degrees of arc 50- 60 years of age
0.75	30
1.00	35
1.25	40
1.50	45
1.75	50
2.00	55
2.25	60
2.50	65
2.75	70
3.00	80

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Against the Rule Astigmatism (10 degrees arc= 1 mm)	
Pre Op Cyl (Diopters)	**Paired Incisions in degrees of arc 50- 60 years of age**
0.75	35
1.00	40
1.25	45
1.50	50
1.75	55
2.00	60
2.25	65
2.50	70
2.75	75
3.00	80

Donnenfeld nomogram (1 clock hour= 30 degrees)		
Preoperative Astigmatism	**Number of Incisions***	**Length of Incisions, Clock Hourst**
0.50D	1	1.5
0.75 D	2	1
1.50 D	2	2
3.00 D#	2	3

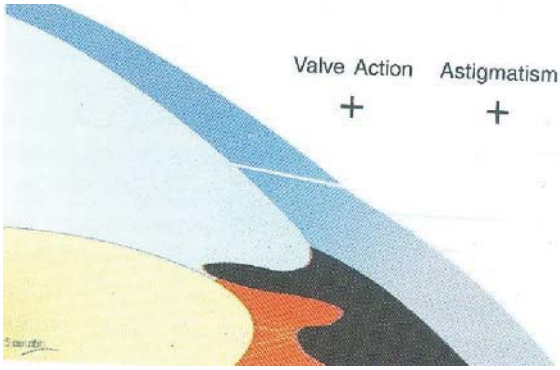
** All incisions are placed 0.5 mm from the limbus in the correct asix.*

 # Patients who have against-the-rule astigmatism or who are less than 45 years old may benefit from slightly longer incisions. Shorter incisions may be indicated for patients older than 65 years.

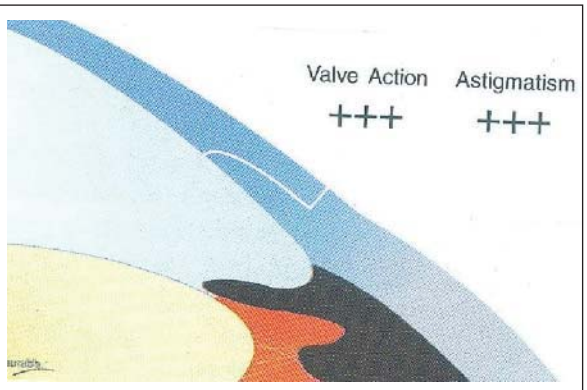
Limbal relaxing incisions can treat upto 4.0 D of astigmatism; however outcomes are less predictable with higher levels of astigmatism due to variable tissue healing.

Limitations

- *Regression:* The higher the astigmatism, the greater is the regression. Most regression occurs in eyes with more than 3.5 D of astigmatism and maximum regression can be seen between 1 to 3 months.
- Diamond knife is preferable.
- *Mechanical instability:* LRIs may lead to weakening of the globe which is prone to rupture or trauma.
- *Ocular surface discomfort:* Post operative tear film instability is seen.



Unipolar



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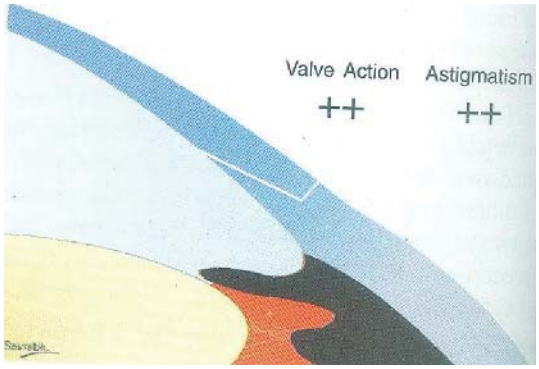
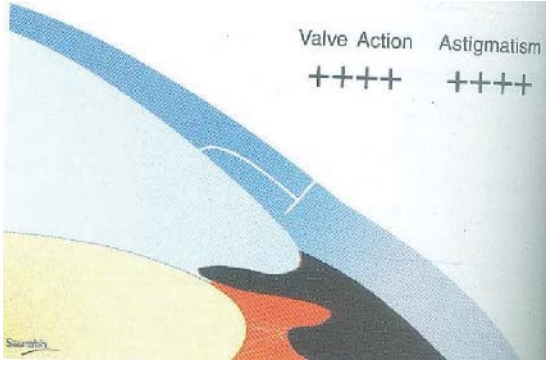


Tripolar



Hinged

Against the Rule Astigmatism (10 degrees arc= 1 mm)	
Pre Op Cyl (Diopters)	**Paired Incisions in degrees of arc 50- 60 years of age**
0.75	35
1.00	40
1.25	45
1.50	50
1.75	55
2.00	60
2.25	65
2.50	70
2.75	75
3.00	80

Donnenfeld nomogram (1 clock hour= 30 degrees)		
Preoperative Astigmatism	**Number of Incisions***	**Length of Incisions, Clock Hourst**
0.50D	1	1.5
0.75 D	2	1
1.50 D	2	2
3.00 D#	2	3

** All incisions are placed 0.5 mm from the limbus in the correct asix.*

 # Patients who have against-the-rule astigmatism or who are less than 45 years old may benefit from slightly longer incisions. Shorter incisions may be indicated for patients older than 65 years.

On Axis Cataract Incision and Opposite Clear Corneal Incisions

Phaco-incision is considered to be astigmatically neutral. Typically, a 3.2 mm incision induces 0.25 to 0.50 D of astigmatism. If we were to place this incision on the steep axis, it can be used to lower the PEA.

In phacoemulsification, adding an identical, penetrating CCI opposite the initial CCI can be used to reduce the preexisting astigmatism. The paired opposite CCIs are placed on the steepest meridian axis to flatten it. One CCI is used to perform cataract surgery and the opposite CCI is made to enhance the flattening effect on the corneas to modulate PEA.

It is possible to correct about 1.0 to 4.0 D of astigmatism by this method so that majority of the patients are able to achieve UCVA as good as BCVA. In the patients desirous of going in for accommodative or multifocal IOLs, using on axis incision, the PEA can be brought down to within 1.0 D which is mandatory for the use of these IOLS.

The biggest advantage of this technique is the stability of the cornea which is achieved in 2 weeks time. Also, postoperatively, there are minimum fluctuations in vision and minimal regression.

Additional point in favour of this method is that no extra instruments or training is required. The surgeon has to learn to mark the axis correctly and he may have to shift the position of the operating chair depending upon the site of incision.

The amount of correction depends upon:

- Types of incisions
- Site and location of the incision
- Size of the incision
- Amount of astigmatism

Types of incisions

The incisions may be:

- Unipolar
- Bipolar
- Tripolar
- Hinged

If all the parameters remain same, then as one progress from unipolar to hinged incision, the amount of astigmatism corrected will be nearly doubled.

Site and location of the incision

A superiorly placed incision gives more correction than a superotemporal or a superonasal incision which in turn provides more correction than a temporally placed incision. (Superior >

Preop Astigmatism (D)	Age (Y)	Number	Length (Degrees)
WTR*			
0.75–1.00	<65	2	45
	≥65	1	45
1.01–1.50	<65	2	60
	≥65	2	45 (or 1 × 60)
>1.50	<65	2	80
	≥65	2	60
ATR/oblique*			
1.00–1.25†	—	1	35
1.26–2.00	—	1	45
>2.00	—	2	45

WTR = with the rule; ATR = against the rule
†Combined with temporal corneal incision
‡Especially if cataract incision is not directly centered on steep meridian

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superotemporal/superonasal >Temporal). This may be because of more slippage of the wound and more importantly the superior incision is more central in location as the vertical diameter is less than the horizontal diameter of the cornea.

Size of the incision

The lesser the width of the incision, the more will be the correction.

Amount of astigmatism

The more is the preexisting astigmatism, the greater is the correction achieved.

Lever and Dahan (2000) corrected astigmatism of 2.06D using incisions ranging from 2.8 to 3.5 mm. Zemaitiene R et al (2003) used incision sizes ranging fro 3 to 4 mm to achieve an astigmatic correction of 0.67 D. Tadros A, Habib M et al (Feb 2004) corrected astigmatism of 1.3 D using incision size of 2.85 to 3.5 mm. Abid Mahamood Qamar et al (2005) used incision size of 3.2 mm to correct astigmatism upto 1.25 D. Khokar et al (2006) corrected astigmatism of 1.66 D using 3.2 mm paired OCCI.

Nomograms

The following nomograms can be used as a guideline:

The following nomogram has been devised by Dr. Harbansh Lal based on his experience

Type of Incision	Amount of Astigmatism Corrected
Single steep axis	< 0.50 D
OCCI 3.2 mm incision	0.5 to 1 D
OCCI 3.5 mm incision	1 to 1.5 D
OCCI 4.0 mm incision.	1.5 to 2 D
OCCI 4.5 mm incision	2 to 2.5 D
OCCI 5.0 mm incision	2.5 to 3 D

The amount of astigmatism corrected depending upon the type of incision:

Hinged > Triplanar > Biplanar > Uniplanar

The amount of astigmatism corrected depending upon the site of incision:

Superior > Oblique > Temporal

Do I Need to Change IOL Power?

No, I do not need to change IOL power due to the coupling effect.

Coupling Effect

Cravy has described gauss's law of elastic domes – "for every change in curvature in one meridian there is an equal and opposite change 90 degrees away". This phenomenon of corneal behaviour is known as the coupling effect.

Corneal coupling is ratio of magnitude of corneal flattening or steepening in axis of surgery divided by magnitude of flattening or steepening 90 degrees away.

Coupling ratio

Amount of flattening in the incision meridian

Induced steepening in the opposite meridian

Coupling ratio =1, indicates that the spherical equivalent is unchanged.

Limitations of OCCI

The major limitation of OCCI is the limited amount of correction induced.

Toric Intraocular Lens

The advantages of toric IOLs are that they do not require the additional surgical skills needed to create clear corneal incisions and they can be implanted using standard cataract surgical techniques.

One major drawback of toric IOLs is that rotation of these IOLs following implantation can decrease the efficacy. For this reason, proper alignment of a toric IOL during surgery is critical. It is also important that the toric IOL shows rotational stability postoperatively to maintain the full intended effect.

Surgical Pearls

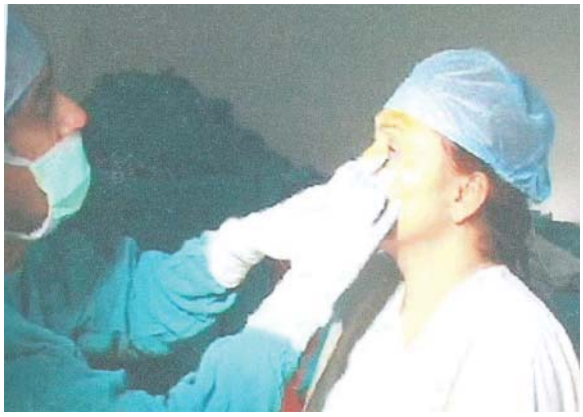
As in all patients, marking of the axis should be done in the sitting position first and then in the supine position.

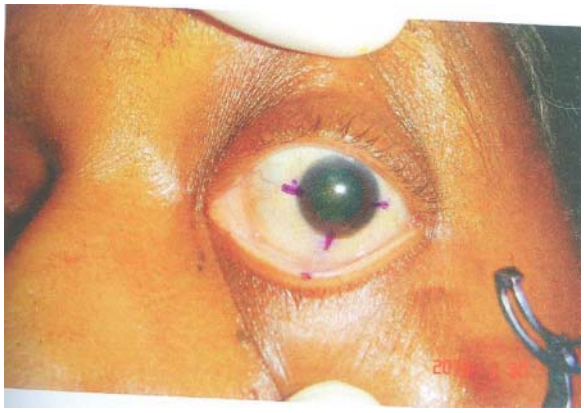
Incision should preferably be placed on the steep axis. As a result, the corneal astigmatism will be partially corrected and the cylindrical power required in the IOL will be less. One degree of off-axis rotation can result in a loss of up to 3.3% of the IOL's cylinder power. This means that approximately one-third of the astigmatic correction is lost if the toric IOL is rotated 10 degrees off-axis. Similarly, two thirds of the effect is lost with a rotation of 20 degrees off-axis. From this, one can infer that if the cylindrical power used in the IOL is less, the loss of effect will be less, if by chance the IOL gets misaligned. Steep axis incision should be performed until and unless one needs to give an incision, which is technically difficult. For example, an inferotemporal / Superonasal incision will be difficult for a right handed surgeon.

The capsulorrhexis should be smaller in size than the size of the IOL optic, should be well centered and should cover the optic equally on all side so that IOL misalignment is reduced. If the capsulorrhexis is eccentric or irregular, it can cause decentration and misalignment of the IOL. It is always better to err on the side of a smaller CCC. An ideal size would be about 4.5-5.5 mm.

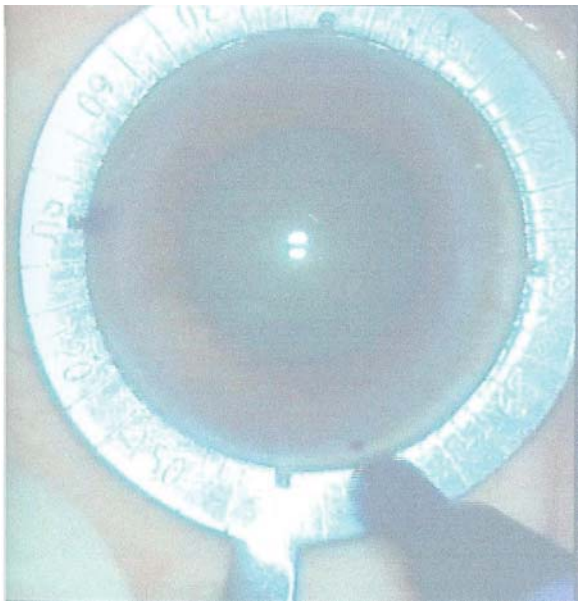
Cohesive viscoelastics should be used for IOL implantation as they can be removed completely from the capsular bag at the end of surgery, thus preventing rotation of the IOL. Dispersive viscoelastics should be avoided as they cannot be removed completely from the capsular bag, which may result in rotation and misalignment of the IOL.

Toric IOL should be placed 20-30 degrees away from the desired position and then rotated clockwise. After removing all the viscoelastic, final positioning of the IOL should be done. Irrigation





Marking of the Axis of Astigmatism in the Sitting Position



Marking of the Axis of Astigmatism in the Supine Position

port of the bimanual or coaxial system is used as an anterior chamber maintainer, through the sideport while doing the final positioning of the IOL. Before withdrawing the irrigation port, air should be placed in the anterior chamber so that the IOL is pushed back into position.

Any doubts in the positioning of the IOL, it is advisable to correct the misalignment, then and there before closing the eye.

Post operatively, one should attempt to determine the correction of corneal astigmatism by measuring the axis of keratometry readings and the refraction.

If the astigmatism left is > 1 D and if this is due to misalignment of the lens realignment should be done within 1-2 weeks. Alternatively, if the patient is very demanding, corneal procedures for correcting residual astigmatism may be used.

The results of corneal procedures like LRIs and OCCIs are variable as they depend upon a large number of factors like site, size, number, length, depth of the incision, age of the patient, amount of astigmatism and many other factors. The correction obtained by a Toric IOL is much more predictable, if aligned correctly.

Techniques of Correction

Treat Only Corneal Astigmatism

Astigmatism is of three types: Corneal, lenticular and retinal. The amount of astigmatism contributed by the retinal slope cannot be assessed. Lenticular astigmatism is treated by removal of the crystalline lens and implantation of an IOL. However, the contribution of the IOL to astigmatism cannot be predicted. Corneal astigmatism is the only astigmatism which can be assessed and corrected. Also, DO NOT TREAT THE REFRACTIVE ERROR. For example, if a patient has – 2.5 D of astigmatism, but keratometry shows only 0.5 D of astigmatism, then treat only 0.5 D.

For correct assessment and treatment of corneal astigmatism, following are important:

Keratometry

Keratometry can be done by the following methods

- Manual keratometry.
- Automated keratometry
- IOL Master.
- Topography

Manual Method is the Gold Standard. Certain points should be kept in mind while proceeding with manual keratometry:

- The patient should be asked to look into the barrel of the machine. Measurements should be taken of the central part of the cornea.

- The instrument should be clean.
- There should be no play in the instrument.
- Standardisation should be done regularly either with an artificial eye or with a calibration sphere.
- The instrument should be properly centered.
- Patient's eyelashes should not come into the view.
- If the patient's tear film dries up, topical lubricants may be used.
- Inter operator differences should not be there; if differences are present, it means that either operators are not trained or instrument is not correct.
- If the operator has any refractive error, it should be corrected.
- Tonometry should always be done after keratometry.

Any of the above mentioned methods may be used, however the most important point is to maintain consistency of readings. Also, same method should be used post operatively for analytical purposes.

Marking of the Axis of Astigmatism is the most important step and should be done accurately. There may be torsion in the supine position of 5-6 degrees in majority of the patients. So, initially the axis should be marked with the patient in the sitting position and then in the supine position.

Conclusion

OCCIs and LRIs, both correct astigmatism and can be combined together for better effect. Because of better post operative stability of refraction, OCCI seems to be a better option.

TORIC IOLs are more predictable than LRIs and OCCIs for correction of PEA but the cost, the exact operative positioning and the post operative rotational stability remains an issue.

OCCI can also be clubbed with toric IOLs to increase the range of astigmatism corrected. It can also be safely used with multifocal and accommodative IOLs. The patients who are left with < 1.0 D of astigmatism remain comfortable, particularly if the astigmatism happens to be against the rule (ATR), as the patient will experience increased depth of focus due to pseudoaccomodation. Probably, better nomograms for OCCIs is the need of the day.

Thus, with a little extra effort of marking the axis correctly and changing the operating chair according to the site of incision, the pre existing astigmatism may be tackled. This proves to be a rewarding experience for the surgeon and a highly satisfactory experience for the patients.

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Scheme for Participation of Voluntary Organisations

Preamble

Blindness is a major public health problem in India with an estimated 12 million blind persons in the country. To tackle this problem, National Program for Control of Blindness was launched in 1976 with the goal to reduce the prevalence of blindness from 1.4% (1974) to 0.3% by the year 2020 by developing eye care infrastructure human resources, improving accessibility quality of eye care services. As per the survey of 2007, level of prevalence of blindness has come down to 1.0%

Cataract is the dominant cause of blindness as it accounts for nearly two third of blind population. The purpose of cataract surgery is to restore vision of the affected person through provision of package of services that can enable the person to gain sight and return to his normal working as before. Refractive errors, childhood blindness, glaucoma, diabetic retinopathy, low vision, ocular injury, age-related macular degeneration, Retinopathy of Prematurity (ROP) and corneal blindness are other important causes of blindness.

The Eleventh (11th) five year (2007-12) plan aims at making National Program for control of Blindness address issues leading to blindness in a comprehensive manner i.e. management of Diabetic Retinopathy (DR), Glaucoma, Squint, Kerato Plasty, Retinopathy of Prematurity, (ROP), low vision etc. in addition to cataract, refractive errors and other ongoing schemes of Tenth five year plan of programme. The National Programme for Control of Blindness (NPCB) has been able to deliver effective & efficient eye care services through successful Public Private Partnership (PPP). The focus of NPCB is specifically targeted towards providing services in rural/tribal and other difficult areas. In addition to ongoing schemes, financial assistance for schemes has been revised and/or new scheme introduced with the approval of Eleventh five year plan.

Schemes for Voluntary Organizations

The purpose of the schemes are to develop eye care infrastructure and to provide appropriate eye care services to reduce the prevalence of blindness. Following schemes are presently available for the voluntary sector:

A Non-recurring Grant-in-aid

I. Non-recurring Grant-in-aid to District Health Societies (NPCB) for release to NGOs for strengthening/expansion of Eye Care Units in rural and tribal areas (upto maximum Rs. 30.00 lakhs);

II. Non-recurring Grant-in-aid for Eye Banks in Government/ Voluntary Sector (upto maximum Rs. 15.00 lakhs);

III. Non-recurring Grant-in-aid for Eye Donation Centres in Government/Voluntary Sector (upto maximum Rs. 1.00 lakhs).

IV. Non- recurring Grant-in-aid for Development of Mobile Ophthalmic Units with Tele-Ophthalmic Network and few fixed Tele-Models (upto maximum Rs. 60.00 lakhs).

V. Non recurring Grant-in-aid for PHC/Vision Centres in Government and Voluntary Sector (upto maximum Rs. 50 Thousand).

B Recurring Grant-in-aid

VI. Recurring Grant-in-aid for free cataract operations and other eye diseases by voluntary organizations/PRI etc. in camps/fixed facilities

VII. Recurring Grant-in-aid for Eye Banks in Government/ Voluntary Sector.

VIII. Recurring Grant-in-aid for Eye Donation Centres in Government/Voluntary Sector

General Eligibility Conditions

The purpose of the schemes are to develop eye care infrastructure and to provide appropriate eye care services to reduce the prevalence of blindness. Following schemes are presently available for the voluntary sector:

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II. Non-recurring Grant-in-aid for Eye Banks in Government/ Voluntary Sector (upto maximum Rs. 15.00 lakhs);

III. Non-recurring Grant-in-aid for Eye Donation Centres in Government/Voluntary Sector (upto maximum Rs. 1.00 lakhs).

IV. Non- recurring Grant-in-aid for Development of Mobile Ophthalmic Units with Tele-Ophthalmic Network and few fixed Tele-Models (upto maximum Rs. 60.00 lakhs).

V. Non recurring Grant-in-aid for PHC/Vision Centres in Government and Voluntary Sector (upto maximum Rs. 50 Thousand).

B Recurring Grant-in-aid

VI. Recurring Grant-in-aid for free cataract operations and other eye diseases by voluntary organizations/PRI etc. in camps/fixed facilities

VII. Recurring Grant-in-aid for Eye Banks in Government/ Voluntary Sector.

VIII. Recurring Grant-in-aid for Eye Donation Centres in Government/Voluntary Sector

General Eligibility Conditions

3.1 Voluntary Organization/NGO:

For the purpose of all the above schemes, a voluntary organization will mean;

a) A Society registered under the Indian Societies Registration Act, 1860 (Act XXI of 1860 or any such act resolved by the State) or a charitable public trust registered under any law for the time being in force;

b) Track record of having experience in providing health services preferably eye care services over a minimum period of 3 year;

c) Properly constituted managing body with its powers duties and responsibilities clearly defined and laid down in a written constitution.

d) Services open to all without distinction of caste, creed, religion or language

e) Having available well trained staff, infrastructure and the required managerial expertise to organize and carry out various activities under the scheme; and

f) Agreeing to abide by the guidelines and the norms of the program. Definitions applicable for grant under the National Programme for Control of Blindness schemes:

3.2 Private Practitioner

a) MD/MS Eye surgeon with two year of work experience in ophthalmology and not working with government on regular/ full time basis and shall be:

b) Providing services to population residing in rural/urban/ tribal/hard core/unserved and/or under-served area as mutually agreed by District/State health society.

c) Agrees to abide by the programme guidelines/norms as announced from time to time.

d) Ensure proper maintenance of records for scrutiny and send regular report to district health society.

3.3 Eye Bank (EB)

An Eye Bank will mean an organization that is:

i) Registered under "The Transplantation of Human Organs, Act 1994";

ii) Provide a round the clock public response system for eye donation;

iii) Coordinate with donor families and hospitals to motivate eye donation;

iv) Harvest corneal tissue not less than 50 eyes in a year.

v) Collect/Process and evaluate the collected tissue and blood for serology;

vi) Distribute tissue in an equitable manner to organizations having capacity for corneal transplantation;

vii) Ensure safe transportation of tissue.

viii) Conduct health personnel and public awareness programs on eye donation

Details of Schemes

Non-recurring Grant-in-aid to District Health Societies (NPCB) for release to NGOs for strengthening/expansion of Eye Care Units in rural and tribal areas (upto maximum Rs. 30.00 lakh);

The scheme seeks to enhance capacity to provide free and subsidized Eye Care Services for underserved affected population in rural including tribal areas. The purpose of the Scheme is to encourage voluntary organizations to expand or upgrade eye care services for providing quality Eye Care services to the affected persons in rural including tribal populations of the country. The scheme offers opportunity to develop capacity for sustainable eye care delivery in the NGO/) sector in areas having inadequate eye care facility.

Three guiding principles influencing the design of this scheme are:

- Long term sustainability;
- Provision of quality eye care services and
- Equivalent resource participation by NGO.

The grantee institute would submit the details of organization and area of service as per Annexure-I.

Financial assistance

Under the scheme, financial assistance will be provided up to a maximum of Rs. 30 Lakh (with equal contribution from NGO in the form of building, equipment and vehicle(s) or cash from management / donations) for any of the following purposes:

a) Construction, renovation & furnishing*

b) Ophthalmic equipment, instruments and other machines (List attached)

*(*Not more than 33% of GIA can be utilized on capital works/ construction activities)*

Recent investments made by the NGO on above-mentioned items during preceding three years (03) can be taken as contribution from NGO as matching grant. For longterm sustainability and resource participation, following recurring costs shall be borne by the NGO:

a) Salaries of staff

b) Cost of consumables

c) Costs on maintenance of equipment and vehicles, POL, etc.

d) Administrative overheads

Eligibility Criteria

The organization should have:

a) Should satisfy general eligibility conditions mentioned at page no. 3 of the document

b) Organizations having experience in providing eye care services will be given preference.

c) Operated on at-least 500/1000 cataract cases or combination of cataract and other ophthalmic diseases (as approved in the scheme) in the preceding 1 year / 2 Year of application. In case of difficult terrain (e.g. North eastern states), relaxed criteria of 300/600 operations in the preceding 2 Year shall be applicable.

d) Facility should be well connected and should have electricity & water supply

Population to be served

Population pockets (to be identified by the District Health Society/ DPM) of 3 to 5 Lakh (only 50,000 in case of sparsely populated rural tribal / hilly / desert / difficult terrains) people will be covered by the applicant NGO. However, patients from other adjoining areas can also be operated either as walk in or community screening.

Infrastructure Requirement:

a) Manpower requirement:

Category of personnel	Minimum No.	Minimum No. in difficult terrain
Ophthalmic Surgeons	2	1
Para Medical Ophthalmic Assistant (Ophthalmic Assistant / Technician / Optometrists / Ophthalmic Nurse.)	4	2
Support Staff (Counselor / Social worker/Accountant / Administrator)	2	1

b) At-least 15 bed IPD facility

c) In addition, the applicant NGO should have adequate infrastructure and equipment for OPD services, Operation and Management of admitted patients.

Expected Output: NGOs receiving non-recurring grants Shall

i) Commit to take the responsibility of active screening of population of villages allocated by the District Health Society and in addition, cater to the patients from adjoining area.

ii) Prepare and maintain village wise Blind Registers in prescribed format (Annexure II)

iii) Complete the construction & procurement of equipments & vehicle, if any within one year after following due procedures.

iv) Provide & maintain Cataract Surgical Cards for the patients operated and other OPD / Indoor wards records (Annexure – III)

v) The NGO should be committed to perform free of cost operations of 1) Cataract and/or other ophthalmic diseases like Diabetic retinopathy, Glaucoma, Keratoplasty, Childhood Blindness- Squint correction, ROP, Retinoblastoma upto a value of 50 % of the sanctioned amount. For the purpose of this scheme, the deemed value of one cataract operation is Rs. 750/-only and for other diseases it is Rs. 1000/- per case.

vi) Maintain proper record & submit monthly report on cases screened, treated and operated in the prescribed Performa (Annexure – VII a, and VII b) in addition to reports as may be sought from the institution from time to time.

vii) Prepare and maintain Diabetic Retinopathy Register (Annexure – XII), Glaucoma Register (Annexure – XIII), Squint Register (Annexure – XIV), Keratoplasty Register (Annexure XV).

Procedure for Approval of Grants

Two copies of application in prescribed formats (Annexure I) would be submitted by applicant NGO along with necessary documents in support of qualifying criteria to the State Programme Officer (SPO), NPCB. The SPO would examine the proposal in terms of eligibility criteria, and depute a team of expert(s) (2-3) from the State/district to visit the NGO for assessing present facilities and requirements. This entire work should be completed within maximum of three months from the date of receipt of applications complete in all respects. The SPO may thereafter, forward his recommendation to the competent authority for final disposal.

Competent authority

Secretary (Health)/Mission Director NRHM of the State would be the competent authority to approve/reject applications in writing giving reasons for rejection, in case of disapproval.

Release of Grant

The NPCB shall release funds for this scheme to State Health Society on the basis of proposal in the State PIP. The State Health Society shall release Grant-in-aid to approved grantees in two installments on Execution of Bond on a Hundred Rupee Non-Judicial Stamp paper by the grantee institution /NGO in the prescribed Pro forma (Annexure–IV).

Penalties

The Government of India/State Government reserves the right to inspect the premises /accounts of the NGO. Any violation of conditions will lead to suspension of any Government grant to the organization in future.

Disposal of Assets

NGO shall maintain a register of Assets acquired wholly or substantially out of Government grants as per the prescribed Performa at (Annexure–VI). Assets acquired wholly or substantially out of the Government grants will not be disposed of, encumbered or utilized for any purpose other than those for which the grants are sanctioned. If such assets are disposed of after due sanction, the money thus received will be credited to State Health Society. Goods declared as obsolete and unserviceable or condemned as per the prescribed procedure may be disposed by NGO after prior approval of State Health Society.

Monitoring and Evaluation

The State Programme Officer/district Health Society shall inspect the work done as and when required and shall also obtain monthly report from the NGO of the work done. The grantee NGO shall be duty bound to submit such reports on a timely basis.

Audited Statement of Accounts & Utilization Certificate

NGO shall get its accounts audited by a Chartered Accountant and submit these accounts within three months of the closure of every

financial year till the completion of conditions in the prescribed Bond to the State Health Society under intimation to the District Health Society. NGO will also have to furnish a certificate of actual utilization of the non-recurring Grant-in-aid for the purpose for which it was received within a period of 3 months of the closure of the financial year. Utilization Certificate shall be submitted in the prescribed Performa at Annexure – VIII. The account of NGO shall be open to inspection by the sanctioning authority whenever the institution is called upon to do so.

Nomination by Government

The State Government / State Health Society may nominate one officer as its representative to the governing body of the NGO receiving Grant-in-aid.

List of equipments that can be procured from Non-Recurring GIA to NGOs for Strengthening/Expanding Eye Care Facility

S.No	Component
A	***Ophthalmic Equipments***
1	Operating Microscope with Assistantscope & Camera attachments
2	A-Scan Biometer
3	Keratometer
4	Slit Lamp
5	Yag Laser
6	Applanation Tonometer
7	Auto Refractometer
8	Vitrectomy Unit
9	Flash Autoclave
10	Automated Perimeter with field analyzer
11	Phacoemulsifier
12	Double Frequency Yag Laser/Argon Green Laser with delivery systems
13	Fundus Fluorescein Angiography Camera
14	B- Scan
15	Surgical instruments for various eye specialties
16	IOLs
17	Sutures 4-0, 8-0, 10-0
B	***Surgical instruments for various eye specialties***
C	***Furniture & Fixtures of Operation Theatres & Ward***
D	***Mobile Ophthalmic Unit with diagnostic equipments and minor surgical instruments***
Maximum Assistance = Rs. 30 Lakh	

Reprint from Scheme for Participation of Voluntary Organisation, National Programme for Control of Blindness July, 2009 Edition.

Forthcoming Events: National

October 2010

28-31 NEW DELHI

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

November, 2010

12-14 NEW DELHI

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

27-28 NEW DELHI

Mid-term Conference
Delhi Ophthalmological Society
Venue: India Habitat Centre, Lodhi Road, New Delhi
Contact Person & Address
Dr. Amit Khosla, *Secretary DOS*
Room No. 2225, 2nd Floor, New Building,
Sir Ganga Ram Hospital,
Rajinder Nagar, New Delhi - 110 060
Ph.: 011-65705229, ***E-mail:*** dosrecords@gmail.com
Website: www.dosonline.org

December 2010

2-4 MYSORE

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

4-5 VARANASI

45th Annual Conference of U.P. State Ophthalmological Society
For more information :
Dr. M.K. Singh
Organising Secretary
Deptt. of Ophthalmology Institute of Medical Sciences
Banaras Hindu University Varanasi
Mob.-9415812264,0542-6703601
E-mail: kashieyecon2010@gmail.com

18-19 JAIPUR

Annual Conference of Strabismological Society of India
Contact Person:
Dr. Virendra Agarwal
104, Shyam Anukampa, Opp. HDFC,
Ahinsa Circle, C-Scheme, JAIPUR
(M) 09829017147, 09414043006
Website: www.strabismusindia.com

January 2011

16-17 CHENNAI

Chrysalis 2011
International Conference on Oculofacial Reconstructive and Aesthetics Surgery
Organizing Secretary
Dr. Shubhra Goel
E-mail: chrysalis2001@snmail.org
Ph.: 044-28254177, ***Mobile:*** 91-9382832910,
Website: www.sankaranethralaya.org/chrysalis2011

February 2011

3-6 GUJARAT

69th AIOS Annual Conference
Gujarat University Convention Centre, Ahemadabad
Conference Secretary,
Dr. Tejas D. Shah
Amdavad Eye Laser Hospitals Pvt. Ltd.
Vision Complex, Polytechnic Cross Roads,
Ahmedabad, 380015. India
Fixed: +91 79 26303208, ***Fax:*** +91 79 26303308
Website: www.aioc2011.com,
E-mail: info@aioc2011.com
Conference Help Line: 96248 96248

April 2010

15-17 NEW DELHI

Annual Conference
Delhi Ophthalmological Society
Venue: Hotel Ashok, Chanakya Puri, New Delhi
Contact Person & Address
Dr. Amit Khosla, ***Secretary DOS***
Room No. 2225, 2nd Floor, New Building,
Sir Ganga Ram Hospital,
Rajinder Nagar, New Delhi - 110 060
Ph.: 011-65705229, ***E-mail:*** dosrecords@gmail.com,
Website: www.dosonline.org

Forthcoming Events: International

October, 2010

6-9 GREECE

EVER 2010 Congress
Venue: Creta Maris, Creta, Greece
Further Information:
EVER Office
Kapucijnenvoer 33, 3000 Leuven, Belgium
Fax +32 16 234 097
Email: ever@ever.be

16-19 CHICAGO, USA

American Academy of Ophthalmology
Venue: Chicago, IL, USA
Contact Information:
American Academy of Ophthalmology
655 Beach Street
San Francisco, CA 94109-1336
Tel.: 415.447.0320, ***Fax:*** 415.561.8576
EMAIL:meetings@aao.org

March, 2011

20-24 SYDNEY, AUSTRALIA

APAO
Venue: Sydney, Australia
Further Information:
Congress Secretariat
GPO Box 3270
Sydney NSW 2001
Ph: +61 (0) 2 9254 5000
Fax: +61 (0) 2 9251 3552
Email: info@apaosydney2011.com

Monthly Clinical Meetings Calendar 2010-2011

Max Eye Hospital 25th July, 2010 (Sunday)	**Midterm Conference of DOS** 27th & 28th November, 2009 (Saturday - Sunday)
Sir Ganga Ram Hospital 29th August, 2010 (Sunday)	**Bharti Eye Foundation** 26th December, 2010 (Sunday)
Army Hospital (R&R) 26th September, 2010 (Sunday)	**Guru Nanak Eye Centre** 30th January, 2011 (Sunday)
Centre for Sight 31st October, 2010 (Sunday)	**Safdarjung Hospital** 27th February, 2011 (Sunday)
Shroff Charity Eye Hospital 21st November, 2010 (Sunday)	**Mohan Eye Institute** 27th March, 2011 (Sunday)

Annual Conference of DOS 15th to 17th April, 2011 (Sunday)

Delhi Ophthalmological Society

***SUBMISSION** online www:dosonline.org*

(LIFE MEMBERSHIP FORM)

Name (In Block Letters)_____

S/D/W/o _____ Date of Birth_____

Qualifications_____ Registration No._____

Sub Speciality (if any) _____

ADDRESS

Clinic/Hospital/Practice _____

_____ Phone _____

Residence_____

_____ Phone _____

Correspondence _____

_____ Phone _____

Email _____ Mobile No. _____

Proposed by

Dr. _____ Membership No. _____ Signature _____

Seconded by

Dr. _____ Membership No. _____ Signature _____

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

I agree to become a life member of the Delhi Ophthalmological Society and shall abide by the Rules and Regulations of the Society.

(Please Note : Life membership fee Rs. 3100/- payable by DD for outstation members. Local Cheques acceptable, payable to Delhi Ophthalmological Society)

Please find enclosed Rs._____in words _____ by Cash

Cheque/DD No._____ Dated_____ Drawn on_____

Signature of Applicant with Date

Three specimen signatures for I.D. Card.

FOR OFFICIAL USE ONLY

Dr._____has been admitted as Life Member of the Delhi Ophthalmological Society by the General Body in their meeting held on_____

His/her membership No. is _____. Fee received by Cash/Cheque/DD No._____ dated_____ drawn on _____.

(Secretary DOS)

INSTRUCTIONS

1. The Society reserve all rights to accepts or reject the application.
2. No reasons shall be given for any application rejected by the Society.
3. No application for membership will be accepted unless it is complete in all respects and accompanied by a Demand Draft of Rs. 3100/- in favour of **"Delhi Ophthalmological Society"** payable at New Delhi.
4. Every new member is entitled to receive Society's Bulletin (DOS Times) and Annual proceedings of the Society free.
5. Every new member will initially be admitted provisionally and shall be deemed to have become a full member only after formal ratification by the General Body and issue of Ratification order by the Society. Only then he or she will be eligible to vote, or apply for any Fellowship/Award, propose or contest for any election of the Society.
6. Application for the membership along with the Bank Draft for the membership fee should be addressed to Dr. Amit Khosla, Secretary, Delhi Ophthalmological Society, Room No. 2225, 2nd Floor, New Building, Sir Ganga Ram Hospital, Rajinder Nagar, New Delhi - 110 060
7. Licence Size Coloured Photograph is to be pasted on the form in the space provided and two Stamp/ Licence Size Coloured photographs are required to be sent along with this form for issue of Laminated Photo Identity Card (to be issued only after the Membership ratification).
8. Applications for 'Delhi Life Member' should either reside or practice in Delhi. The proof of residence may be in the form Passport/ Licence/Voters Identity Card/Ration Card/Electyricity Bill/MTNL (Landline) Telephone Bill.

Online Journal Available

Dear DOS Members,

The DOS members can get the full text articles of the current issues as well as many back issues of these subscribed journals. You need to send the request for the article needed via email: **dos_library@rediffmail.com.** We will email you full text.

Dr. Harbansh Lal
Library Officer, DOS
E-mail ID is: dos_library@rediffmail.com

1. Acta Ophthalmologica –(2008-2010)
2. Acta Ophthalmologica Scandinavica –(2001-2007)
3. Acta Ophthalmologica Scandinavica Supplement –(2002-2005)
4. Archives of Ophthalmology –(1995-2010)
5. British Journal of Ophthalmology –(2008-2010)
6. Clinical & Experimental Ophthalmology –(2001-2010)
7. Contemporary Ophthalmology –(2005-2010)
8. Cornea –(2000-2010)
9. Current Opinion in Ophthalmology –(1999-2010)
10. Evidence-Based Ophthalmology –(2008-2010)
11. International Ophthalmology Clinics –(2000-2010)
12. Journal of Glaucoma –(2001-2010)
13. Journal of Neuro-Ophthalmology –(2001-2010)
14. Journal of Pediatric Ophthalmology & Strabismus –(2008-2009)
15. Journal of Refractive Surgery –(2008-2009)
16. Ophthalmic Plastic & Reconstructive Surgery –(2000-2010)
17. Ophthalmic Surgery, Lasers & Imaging –(2008-2009)
18. Ophthalmology Management –(2008-2010)
19. Retina –(2000-2010)
20. Techniques in Ophthalmology –(2003-2010)

Delhi Ophthalmological Society
Monthly Clinical Meeting, October 2010

Venue: Centre for Sight, B-5/24, Safdarjung Enclave, New Delhi - 110029
Date & Time: 31st October, 2010 (Sunday), 10:00 A.M.

Scientific Meeting 10:00 a.m. Onwards

Clinical Cases:

1	Intacs for pos lasik ectasia	:	Dr. Ramendra Bakshi
2.	Flipped Iris Claw Lens	:	Dr. Charu Khurana

Clinical Talk:

Posterior Chamber IOL fixation with Glue : Results and complications	:	Dr. Mahipal S. Sachdev

Mini Symposium: ***Challenges in Ophthalmology***
Chairpersons: *Dr. V.K. Dada,* ***Co-Chairman :*** *Dr. H.K. Tewari,* ***Convener :*** *Dr. Harsh Kumar*

1.	Challenges in Glaucoma	:	Dr. Harsh Kumar
2.	Sutureless Vitrectomy	:	Dr. Avnindra Gupta
3.	Recent advances in Ptosis Surgery	:	Dr. Vikas Menon
4.	Recent Advances in Amblyopia	:	Dr. Renu Grover

20 Early Bird Prizes & Lunch Sponsored by M/s. Syntho Pharmaceuticals

DOS Skill Transfer Workshop on Basic Oculoplasty

Dear DOS Member/Student,

The **Skill Transfer Workshop on Basic Oculoplasty** will be held on Sunday, 19th December, 2010 at Auditorium, Gurunanak Eye Centre, MAMC, Maharaja Ranjit Singh Marg, New Delhi from 9:00 a.m. to 4:00 p.m.

Please register by Phone or E-Mail to your respective co-ordinators with the following :
(1) Name (2) Affiliation (3) Postal Address (4) E-Mail Address (5) Mobile No

Our E-Mail ID is : dosrecords@gmail.com
Our Phone No. is : 011-65705229

If you have any query, please do not hesitate to contact us.

With regards,

Dr. P.V. Chadha
President

Dr. Ruchi Goel
Workshop Co-Ordinator

Dr. Amit Khosla
Secretary

ATTENTION !
MD / MS / DNB / DO / OPHTHALMOLOGY STUDENTS

DOS TEACHING PROGRAMME

DOST
OPHTHALMOLOGY
A Two Days Exhaustive Ophthalmology Training

VENUE
8th & 9th January, 2011
Saturday & Sunday,
Army Hospital (R&R),
Auditorium,
Delhi Cantt., Delhi

The Delhi Ophthalmological Society organizes it's third Teaching Programme "DOST-3" aimed at teaching the Post Graduate (MD/MS/DNB/ DO Ophthalmology) Students all over India. A two day exhaustive course for Post-Graduate Students.
All the Members & Students are welcome to attend !

Registration Fee

Category	Upto 15th December, 2010
DOS Member / Student	₹ 300
Non Members / Student	₹ 500

Highlights

- OSCE / Case Presentation
- Basic Sciences
- Lens
- Oculoplasty
- Refraction
- Glaucoma
- Retina
- Cornea
- Squint
- Cataract