BDOS SCIENTIFIC HIGHLIGHT

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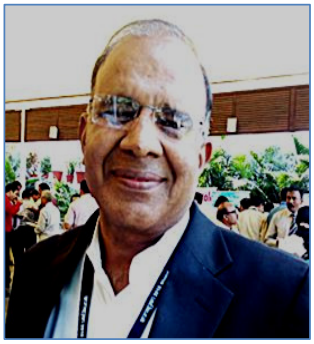

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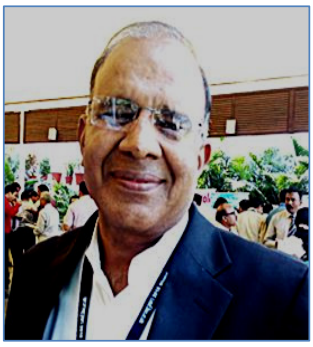

Dr Lalit Shrivastava
President, BDOS

Respected Members,

It is a matter of an immense pleasure to say something amongst our esteemed BDOS members. On behalf of BDOS, I am extremely thankful to everyone for their cooperation, suggestions and advice to improve the level of our activities.

In Corona pandemic, our team is carrying out so many digital webinars on different basic and recent topics under the guidance and great help of our Chief Patron Dr Salil Kumar Sir and with the herculean effort made every time by our Honorary Secretary Dr Vinita Ramnani as well as our energetic dynamic Secretary Dr Chahveer Singh Bindra.

It is my humble submission to all of you to raise our membership and deposit the activity fee. Secondly, participate in maximum numbers in our scientific activities.

Thank you
Best regards to everyone

Dr Lalit Shrivastava
President, BDOS

MESSAGE FROM BDOS HON. SECRETARY

Dr Vinita Ramnani
Hon. Secretary, BDOS

Respected Seniors and Dear Colleagues,

I am very proud and delighted to present 5th volume of BDOS Scientific Highlight. It has been a heart filled moment with feeling of gratitude and happiness on successful accomplishment of one year as a Honorary Secretary of Bhopal Divisional Ophthalmic Society.

Since beginning our efforts have been to turn every unturned stone towards betterment of our society. We have come a long way since January 2014 when I added a new feather to the cap of BDOS as a Clinical Secretary and presented the first ever volume of BDOS Scientific Highlight.

I acknowledge moral support & constant motivation from Chief Patron Dr Salil Kumar and President Dr Lalit Shrivastava along with all office bearers & executives of the BDOS team.

Editor Dr Chahveer Singh Bindra and his team have done justice to this issue of BDOS Scientific Highlight with the best of their abilities. We are grateful to all esteemed authors for articles and sharing their experiences to help us. I also appeal to all BDOS members to submit their articles and provide details of any achievements, publications or awards for the next issue.

The current issue of BDOS Scientific Highlights includes Guest editorial, Expert corner, Review articles, Recent updates and Case reports extremely well written along with articles beyond ophthalmology. I am sure that they will be helpful in upgrading our knowledge.

During this COVID pandemic, we have conducted a series of monthly webinars with your support and encouragement. I am really thankful for the active participation from you all. I take this opportunity to seek blessings and support to launch a new website of BDOS soon and to make BDOS directory of members for the holistic development of BDOS.

Thanks
Warm Regards

Dr Vinita Ramnani
Hon. Secretary BDOS

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EDITORIAL

Recent Consensus On AMD And PCV – A Paradigm Shift

Dr Chahveer Singh Bindra

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Age related macular degenereation (AMD) is the third leading cause of blindness globally. It is characterised by subretinal drusenoid deposits, basal linear and basal laminar deposits. The basal laminar deposit (BLamD) is located between the plasma membrane (pm) of the retinal pigment epithelium (RPE) and the basement membrane (bm). Between the basal lamina and the trilaminar Bruch's membrane is a collection of basal linear deposit. Both types of deposit occur internal to the inner collagenous layer of the trilaminar Bruch's membrane. Diffuse thickenings of these deposits may not be detectable by conventional ophthalmoscopy, but mounds of basal linear deposit appear as soft drusen. Subretinal drusenoid deposits are accumulations of material located above the RPE and appear as pseudodrusen. In early phases of AMD, the visual performance may show minimal changes. With time, vitelliform deposit may accumulate, pigment may migrate into the retina, drusen size may increase, and hypopigmentation and hyperpigmentation of the RPE may develop. Late phases of the disease may be characterized atrophy of the outer retina, thinning and loss of the RPE, and macular neovascularization (MNV).

MNV denotes neovascular disease in the macula from many causes. In AMD, the neovascularization may start in the outer retina, and therefore, the term choroidal neovascularization is not appropriate for the class. The risk for progression in the eye without AMD stigmata is nearly zero, and accordingly, one should not diagnose AMD in an eye without visible abnormalities. Gass1 classified MNV into Type 1 and Type 2 based on the location of the lesion in relation to RPE. This classification was based on the observation of different visual outcomes assumed to be because of differential locations of MNV. In the era of multimodal imaging, Freund et al introduced the term "Type 3" to indicate intraretinal MNV.[2,3]

Recently, the Consensus on Neovascular AMD Nomenclature Study Group (CONAN) recommended that these anatomic terms be used to describe MNV in future studies of nAMD and the clinical practice.[4]

With much more advanced techniques now available, such as OCT and OCTA, retina specialist and researchers believed it was time to conduct a comprehensive review all of the old definitions relating to wet AMD. The panel decide that familial old terms such as "occult CNV", "classic CNV" and "occult retinal choroidal anastomosis" should be replaced by more precise definitions such as "type 1 MNV", "type 2 MNV" and "type 3 MNV" respectively.

In type 1 MVN, the ingrowth of vessels arises from the choriocapillaris and extends up to and under the retinal pigment epithelium, leads to varying types of PEDs. With growth and expansion of the lesion, remodeling and enlargement of the feeding and draining vessels occurs within both the choroid and the lesion. Increased intralesional hydrostatic pressure coupled with breakdown of the outer blood-retinal barrier leads to exudation into the subretinal space (figure 1). Intraretinal fluid accumulation may occur in conjunction with breakdown of the external limiting membrane, or VEGF expression may induce intraretinal leakage independently. Mature forms of Type 1 MNV can exhibit partial resistance to anti-VEGF therapy, although, despite persistent SRF, the visual prognosis may remain stable with up to 5 years of continuous anti-VEGF treatment.[2]

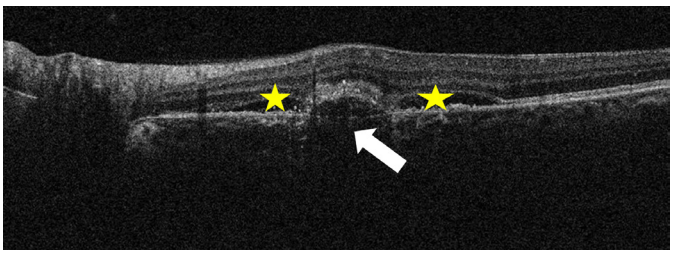

FIGURE 1: In type 1 MVN, the ingrowth of vessels arises from the choriocapillaris and extends up to and under the retinal pigment epithelium, leads to varying types of PEDs (White arrow). Increased intralesional hydrostatic pressure coupled with breakdown of the outer blood-retinal barrier leads to exudation into the subretinal space (star).

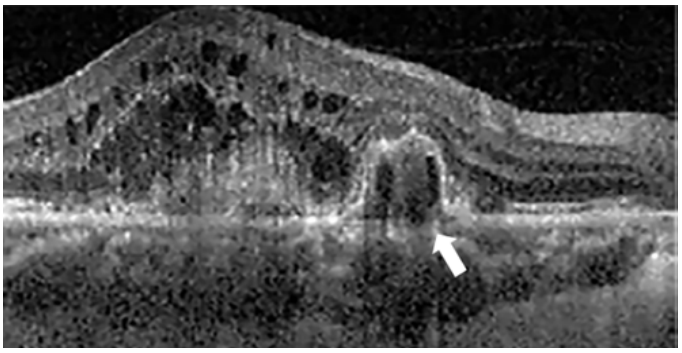

FIGURE 2: PCV expands slowly in the sub-RPE space. It may present with multiple PEDs, sharp PED peak, PED notch and rounded sub-RPE hyporeflective area (White arrow).

Polypoidal choroidal vasculopathy (PCV) is an important subtype of neovascularization defined by a branching vascular network and nodular vascular agglomerations also called polyps. PCV expands slowly in the sub-RPE space over time and may grow to considerable size before having any meaningful impact on vision (figure 2). The polyps may be pulsatile and are particularly prone to bleed. Because of the slow perfusion dynamics, most polyps remain undetected by OCT angiography. The polyp was the growth of cavernous thin-walled vessels immediately external to the RPE, above BM. Based on the presence of at least 3 of 4 OCT signs (multiple PEDs, sharp PED peak, PED notch and rounded sub-RPE hyporeflective area), De Salvo et al found the sensitivity and specificity to be greater than 90%.[5]

APOIS PCV Workgroup has proposed a set of practical, easily adopted 9 non-ICGA diagnostic features based on OCT and fundus photography for diagnosing PCV. Workgroup recommended revising the terms ‘polyp’ and ‘branching vascular network’ which are based on ICGA appearance to ‘polypoidal lesion’ and ‘branching neovascular network’, respectively.[6] These features comprise of sharp peaked PED, sub RPE ring like lesion, complex / multiloibular PED, double layer sign, thick choroid with dilated Haller’s layer vessel, fluid compartment, enface OCT-complex RPE elevation on OCT and extensive subretinal haemorrhage and orange nodule on color fundus photography.
Type 2 MNV refers to the proliferation of new vessels arising from the choroid into the subretinal space. Although these vessels traverse the sub-RPE space, the disease process in type 2 neovascularization is dominated by the subretinal portion (figure 3). Type 2 MNV occurs in conditions other than AMD that affect the RPE, such as angioid streaks, lacquer cracks, and chorioretinitis.

Type 3 MNV refers to a downgrowth of vessels from the retinal circulation toward the outer retina. The vascular proliferation is suspected to start from the deep capillary plexus in the retina with the vector of growth extending toward the outer retina. Increasing blood flow within the angiomatous proliferation is supplied by the retinal vessels, which seem to remodel overtime to handle the flow requirements. Scattered flecks of intraretinal hemorrhage (always outside the foveal avascular zone) and cystoid spaces are present, both of which may appear before the neovascularization. The neovascularization has the potential to leak and bleed. Old term used for type 3 MNV was retinal vascular anomalous complexes, retinal angiomatous proliferation (also known as RAP), and occult retinal choroidal anastomosis.

In mixed type 1 and type 2 MNV prominent neovascularization is present in the subretinal and sub-RPE compartments. Early-phase of fluorescein angiogram shows a well-defined area of neovascularization. Later-phase fluorescein angiogram shows pronounced leakage from the well-defined neovascularization and some punctate leakage from adjacent area. Patients with mixed type 1 and type 2 lesions can show apparent regression of the type 2 component after initiation of treatment.

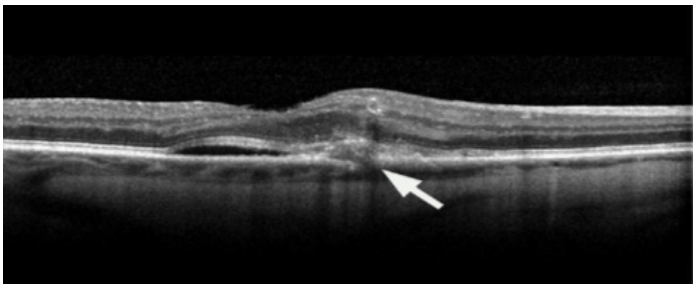

FIGURE 3: In type 2 MNV vessels traverse the sub-RPE space and enter the subretinal space with breach in the RPE (White arrow).

Age related macular degeneration may lead to atrophy or thinning of RPE or retina in late phases. It has been classified on basis of RPE and outer retinal layers involvement as follows:

A. Outer Retinal Atrophy (ORA) –
   a) In complete outer retinal atrophy (cORA), the ellipsoid zone and the interdigitation zone are not visible, the external limiting membrane may not be discernable, and the outer nuclear layer becomes thinner.
   b) In incomplete ORA, a discontinuous loss of the ellipsoid zone has occurred, and the interdigitation zone is typically not visible.

B. Retinal Pigment Epithelial and Outer Retinal Atrophy (RORA) -
   a) In complete RORA, an absence of the RPE is present, as manifested in OCT imaging by a loss of the RPE band with associated choroidal hypertransmission in OCT imaging, together with the findings of cORA in a zone of at least 250 mm
   b) Incomplete RORA is defined in OCT imaging by heterogeneous hypertransmission and associated fragmentary attenuation or loss in the reflectivity from the RPE band with overlying photoreceptor degeneration in zone less than 250 mm

To conclude advances in diagnostic imaging and learning's from therapeutic outcomes have substantially changed the understanding of neovascular AMD as a morphologic disease entity. The substitution of largely angiographic definitions by OCT based feature identification, the insight into the origins of neovascular development, including both the retina and choroid, and the differentiation of neovascular and atrophic pathways including their concominant occurrence will help us in understanding the disease, its prognosis and its universal acceptance.

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GUEST EDITORIAL

Ocular Surface – The Basics

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The term ocular surface describes the entire epithelial lining bordered by the superior and inferior eye lid margins, on to the back of the lid, into the fornices, and its reflection back over the globe including that which covers the cornea. Epithelial surface has two major areas of cornea and conjunctiva separated by limbus. The corneal epithelium is composed of non-keratinized, stratified squamous cells. The superficial layer of epithelium consists of flattened cells with microvilli. Coating this microvilli is a layer of glycocalyx, which interacts with the mucous layer of tear film and provides stability to tear film. The corneal epithelial cells have a life span of 7 to 10 days undergoing involution, apoptosis and desquamation. Beneath this, is a layer of wing cells and the deepest one is basal cell layer, anchored to basement membrane. At the corneo-scleral limbus, there is transition from the stratified, non-keratinized squamous epithelium to columnar epithelium with mucin secreting goblet cells of conjunctiva. The architecture of the limbus shows a palisade (of Vogt) arrangement. The limbal stem cells are situated in the basal layer of the palisade of Vogt.

The minute surface irregularities are masked by a smooth and regular overlying tear film. Tear film produces lubrication and hydration to the ocular surface. It is also a source of oxygen, immunoglobulin, lysozymes, lactoferrin. The tear film is composed of three layers. The most superficial layer is composed of oily secretion of meibomian glands. Because oil is less dense than water, the secretions float to the top of tear film, providing smoothness to the surface and a barrier against evaporation. The middle layer is formed by secretion of lacrimal and accessory lacrimal glands, which is aqueous in nature. The inner most mucin layer is produced by goblet cells. All the three layers form a gel on the ocular surface, responsible for debris removal and immune surveillance. Figure 1 shows the formation and factors responsible for the maintenance of preocular tear film

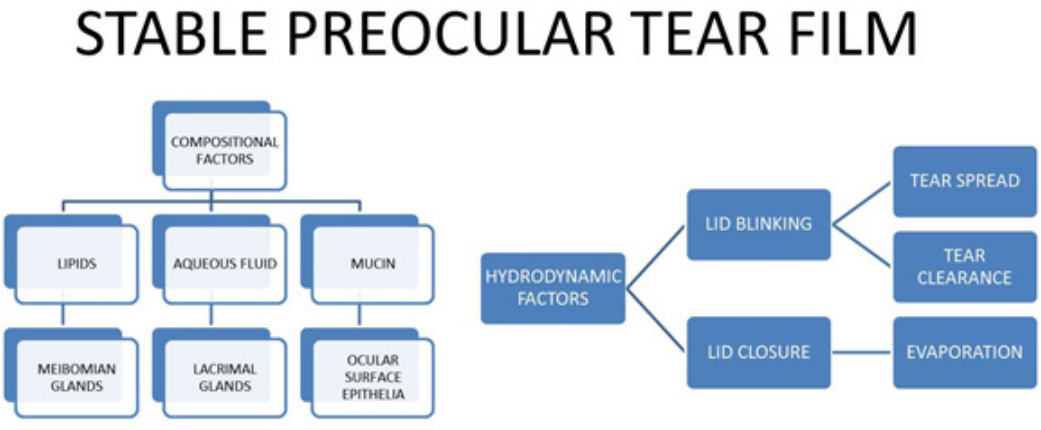

FIGURE 1: composition and hydrodynamic factors for preocular tear film

The ocular surface consists of two types of epithelial cells, conjunctival and corneal, both are anatomically continuous at limbus but the two cell phenotypes represent quite distinct sub- populations. The limbal palisades of Vogt and inter-palisade rete ridges are repositories of stem cells. When there is a demand for tissue regeneration, the stem cells divide. When a stem cell divides, one of the daughter cell remains as a parent and serves to replenish the stem cell pool, such daughter cells are called "transient amplifying cell." Stem cells are present in all self renewing tissues. These cells are long lived and have great potential for clonogenic cell division, responsible for tissue regeneration.

The primary function of the ocular surface is to provide clear vision and preventing microbial invasion. The mechanism, by which ocular surface health is ensured, is built into intimate relation between ocular surface epithelia and tear film, constituting ocular surface defense, which governs epithelia and tear film to work as one unit. All elements of ocular defense are integrated and work in concert; hence dysfunction of one element can affect other. Even if there is ample amount of tears with normal components, it cannot form a film without the hydrodynamic factor, which includes periodic and complete eye lid blinking to facilitate tear spread into a film to cover the entire ocular surface and tear clearance into nasolacrimal drainage system.

Therefore it is important to point out that ocular surface defense is integrated with epithelia via two neuronal reflex arcs. Both reflexes are sub served by the first branch of trigeminal nerve, controlling ocular sensitivity as the afferent sensory input and by parasympathetic branch, the motor branch of facial nerve as efferent output respectively. Such neuroanatomic integration explains the sensitive activity of ocular surface. The integration of these two reflex arcs is controlling compositional and hydrodynamic factors. Deficiency in ocular sensitivity can lead to a deficiency of compositional factors; this leads to inadequate blinking, resulting in increased exposure and finally leads to unstable tear film as shown in figure 2.

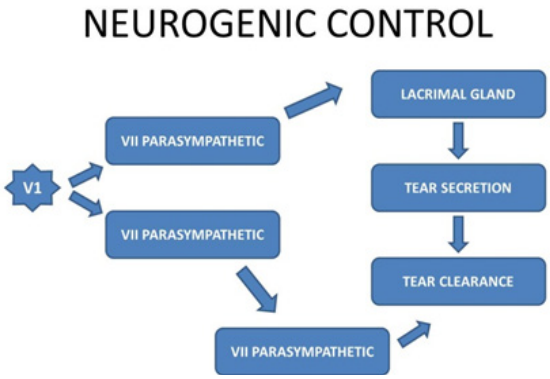

FIGURE 2: Showing the neurogenic control of preocular tear film

There are two types of ocular surface failure identified by impression cytology based on resultant epithelial phenotype, first one shows squamous metaplasia, second type shows stem cell deficiency. With the advent of limbal stem cell concept, Kenyon and Tseng developed the surgical procedure of limbal stem cell transplantation, for the patients with diffuse stem cell deficiency, by using auto graft. Afterwards cultured stem cells and use of "BIOENGINEERED CORNEA" advocated. Tseng and coworkers reported that amniotic membrane transplantation provides, support for growth of epithelial cells.

With any type of ocular surface failure, the aim should be to restore surface defense and to reconstruct with amniotic membrane or stem cell. The ocular surface is not a single structure, it is composite, has its specific anatomy and physiology; hence its disease involves a variety of etiological mechanisms.

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EXPERT VIEWS ON NEOVASCULAR GLAUCOMA

Expert Panelists

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Phaco & Glaucoma Consultant,
Chaitanya Netralaya

Dr. Madhulika (MM)
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Q1: **50 years old patient comes to OPD with chief complaints of pain, loss of vision, headache and redness. On evaluation BCVA is 6/60 in right eye and 6/6 in the left eye. Patient has history of diminution of vision in right eye since 3 months and was diagnosed as central retinal vein occlusion earlier. GAT is 38 in right eye and 16 in left eye. Anterior segment shows neovascularization of iris and angle with normal AC depth. Fundus evaluation shows 0.6 cupping with old central retinal vein occlusion in right eye while left eye was normal. What is the next set of investigations required?**

RKG: The investigations can be divided into ocular investigations and systemic investigations. Ocular investigations include- A) Gonioscopy - to look for new vessels in the angle and whether the angle is open or closed due to formation of peripheral anterior synechiae. This is important as line of management will be different in these two situations. B) Slit lamp biomicroscopy with 78D or 90D - for stereoscopic evaluation of optic disc. Documentation of disc findings with photographs or OD drawing for follow up. C) Perimetry – Visual field analysis, to correlate field changes with Optic disc findings and to document the damage. This will help in follow up to know the progression. D) OCT - Optic disc - RNFL and macula to document the glaucomatous damage and for progression. E) FFA- before performing panretinal photocoagulation. Systemic Investigations comprising of recording of blood pressure, HbA1c and fasting and post prandial blood glucose level and lipid profile.

VR: Neovascular glaucoma (NVG) post CRVO is a potentially blinding diseases, where beside good history and through clinical examination on slit lamp, Applanation tonometery, Gonioscopy and Fundus photo to document, investigations required are Fundus Fluorescein Angiography to see for extent of retinal ischemia, OCT macula to rule out macular odema along with Perimetry (HVF) & OCT (RFNL, ONH) to asses glaucoma damage. Good systemic examination with blood investigations like CBC, ESR, CRP, HOMOCYSTINE, LFT, RFT & lipid profile with Cardiac evaluation should be done.

SP: Blood investigations including complete blood count with hemoglobin, fasting and post prandial blood glucose level, HbA1C, serum homocysteine, blood pressure monitoring and lipid profile. OCT macula also needs to be done to rule out associated macular edema.

AB: Seeing the visual potential of 6/60, would definitely make all possible efforts to save this vision and also make sure to find and treat the cause of pathology, so that the other eye doesn't get similarly affected. History of any systemic illness, smoking and lifestyle is important and should be taken. Check blood pressure and blood glucose levels along with HBbA1C, Lipid Profile and complete blood count and ESR, Serum homocysteine, Gonioscopy despite neovascularisation (also look for whether angle is open or closed). If its angle closure stage medical treatment may not be effective. Stereoscopic biomicroscopy should also be done to look for the status of disc rim for any notches, excavations and cupping. Perimetry and fundus photo/ disc drawing for comparisons in follow up. Central corneal thickness should be done to ensure accurate IOP measurements. Fundus fluorescein angiography is done for any activity or laser requirement. OCT (macula) to look for any active cystoid macular edema

SD: My set of investigations include gonioscopy for NVA, Perimetry, CCT and OCT (RNFL)

MM: I will prefer ocular investigations comprising of fundus fluorescein angiography, OCT macula and RNFL (supportive), Visual field analysis, gonioscopy and applanation tonometry of both eyes. I would also prefer following set of blood investigations – FBS/PPBS, LFT, KFT, lipid profile and serum homocysteine at the initial onset.

Q2: What is the medical treatment of choice in this case and how frequent follow up is required?

RKG: Antiglaucoma drugs which decrease aqueous humour production like a combination of topical beta blockers and alpha 2 agonist can be started along with systemic carbonic anhydrase inhibitors. Once IOP decreases, oral CAI can be withdrawn and if required topical CAI can be added.Topical steroids and cycloplegics are given to control inflammation along with antiglaucoma drugs. Once ocular inflammation is controlled and if desired IOP is not achieved then PG analogues can be added. PG analogues are not so effective if a fibrovascular membrane obstructs the outflow path, and they may exacerbate ocular inflammation. Patient is followed alternate day till IOP is controlled with medical therapy then monthly for 3 months and then every 3 monthly. OD evaluation, gonioscopy and perimetry are done every 6 months.

VR: Delayed diagnosis or poor management can result in complete loss of vision; therefore effective management strategies to treat both the elevated intraocular pressure (IOP) and the underlying cause of disease (CRVO) are needed. Mainstay of medical treatment is to reduce aqueous production with topical beta blockers, alpha2 agonists and with topical and/or oral carbonic anhydrase inhibitor and Hyperosmotic agents as per IOP control. Topical prostaglandin analogues can be given after control of ocular inflammation. Miotics are contraindicated as they can increase inflammation and discomfort. Frequent administration of topical steroids and cycloplegics are recommended to reduce inflammation. It is necessary to closely follow these eyes and manage patients adequately to reduce the Blindness.

SP: Put on Tab Acetazolamide 250 mg BD for 3 to 5 days. Start locally Briminodine 0.3% with timolol 0.5% eye drop combination. In these types of cases close follow-up is needed. First follow-up is usually done within one week and further depending upon the response of AGM and retinal status.

AB: Medical treatments of choice are drugs that reduce aqueous production like topical beta blockers, carbonic anhydrase inhibitors and alpha2 agonists along with oral carbonic anhydrase inhibitors. Oral acetazolamide and intravenous mannitol should be used with caution in diabetics as concomitant renal disease may be present. Once IOP is controlled with topical and systemic treatment, systemic acetazolamide should be withdrawn and IOP checkup off acetazolamide should be taken after 3-4 weeks. Prostaglandin analogues I usually start after reducing inflammation with steroids.

AB: Remember PG analogues are not contraindicated in any inflammation but should be used with caution as they might increase inflammation. In my practice I have seen sometimes we get the desired IOP only after starting PG analogue even in uveitic glaucoma and also NVG. Topical Steroids and Cycloplegics should be aggressively started in all stages of NVG (even in angle closure glaucoma stage) to reduce inflammation. I have seen steroids and cycloplegics are often missed in prescriptions despite diagnosis and rest of the treatment being right. An eye with even a fair visual potential of finger counting vision or better, I usually follow up alternate day or daily till IOP is on decreasing trend and eye looks less inflamed. Thereafter, I review weekly for 3-4 weeks off systemic acetazolamide (i.e. only on drops) and thereafter 1 monthly 2monthly 4 monthly. Once IOP checkup 4 monthly and once a year Gonioscopy Stereoscopic Biomicroscopy and Perimetry (if vision not less than 6/60)

SD: I would like to start patient oral acetazolamide along with beta blockers, alpha agonists and carbonic anhydrase inhibitors. Prostaglandins are to be avoided. After starting AGM, I would prefer weekly follow up to begin with.

MM: Medical treatment would comprise of beta blockers, alpha agonists, carbonic anhydrase (both orally and topical) along with topical steroids and cycloplegics. Oral carbonic anhydrase should be started in TDS dosing for at least 7 days. Follow up to be done every 3rd day to begin with and then weekly depending on the response to treatment and IOP control.

Q3: In this case of Neovascular Glaucoma with CRVO, what is the role of anti VEGF and panretinal photocoagulation and when they are to be considered and in what sequence? What is the overall prognosis in such case?

RKG: Panretinal photocoagulation is considered in all cases of Neovascularization. It reduces the ischemic drive that produces VEGF. PRP helps in complete regression of iris, angle and retinal neovascularization. PRP take several weeks to exert its effect, so it is performed 3-4 weeks prior to surgery. VEGF is an important and predominant factor in the pathogenesis of neovascularization. Anti-VEGF injection leads to regression of both iris and angle neovascularization, and intraocular pressure control when the angle is open. The anti-VEGF agents also help by decreasing the pain associated with the inflammation in this disease. They are considered as an adjunct to definitive surgical procedures for NVG. Studies have shown that pre-treatment with anti- VEGF before glaucoma surgeries can significantly lower the frequency of hyphema. These drugs have transient effect so repeat injections may be required. Neovascular glaucoma is difficult to manage, failure rates are high. They require close monitoring and lifelong follow up. Early diagnosis and appropriate treatment can benefit some patients. Visual prognosis is always guarded.

VR: Medical management with intravitreal and or intracameral use (1.25mg/0.05ml) of Anti-VEGF along with retinal ablation wherever possible helps to control the IOP in the open angle stage of NVG. The introduction of Anti-VEGF agents in the management of NVG has proved to be a benchmark in both prevention and treatment of this disease, Anti-VEGF should be immediately injected after systemic antiglaucoma drugs, because this not only causes regression of the iris neovascularization but also control IOP, intraocular inflammation and pain and prevents bleeding in subsequent glaucoma surgery. In the Central Retinal Vein Occlusion Study (CVOS) PRP was indicated for FFA confirmed ischemic CRVO who developed 2 clock hours of NVI or NVA, and should be done as early as possible, may be 1-3 days of Anti VEGF injection and clearer media. In cases where PRP is not possible due to poor retinal view, intravitreal anti VEGF followed by surgical IOP control is planned and PRP can be done subsequently. Anti VEGF-induced regression of neovascularization is often temporary, PRP provides a more permanent reduction of the ischemic angiogenic stimulus to prevent recurrence of neovascularization and to minimize visual loss. Anterior retinal cryopexy (ARC) may be useful in cases of compromised posterior segment view and where affordability of anti VEGF is issue. Blindness caused due to NVG is totally preventable if diagnosis is made in early stages with aggressive management along with frequent follow ups.

SP: Pan retinal photocoagulation is done on urgent basis and aggressively on alternate day rather than 1 week apart. Anti VEGFs are also given to reduce VEGF drive and for associated cystoid macular edema. Intracameral anti VEGF can be given if media is hazy for pan retinal photocoagulation. Overall prognosis may be fair as it appears to be non-ischemic CRVO at present. Patient may recover some vision with prompt treatment.

AB: Unlike other glaucomas in managing NVG it's important to treat IOP as well as underlying cause of disease. So role of anti VEGF and PRP is important. Anti VEGF regresses neovascularisation of iris in 24-48 hours and also decrease inflammation and even IOP. This can be followed by PRP. But prophylactic PRP does not prevent the development of neovascularisation. Best time to consider PRP is early neovasularisation. Despite multiple modes of medical and surgical options advocated over years treatment of NVG is challenging and often not satisfactory in preventing visual loss in majority of patients due to the progressive underlying pathology.

SD: Anti VEGF injection are to be given followed by pan retinal photocoagulation. Overall prognosis is generally not good in such cases.

MM: There is definite role of anti-VEGF followed by Pan retinal photocoagulation. Prognosis depends on the control of blood sugar levels, response to anti-glaucoma treatment and regression of Neovascular iris with anti-VEGF.

Q4: What is the surgical treatment of choice if IOP is not controlled despite maximal medical therapy?

RKG: The surgical treatment of choice would be trabeculectomy with MMC. To increase the success rate of trabeculectomy, PRP and anti VEGF injection before surgery for regression of new vessels is essential. Topical steroids with cycloplegics are required to control inflammation. If trabeculectomy fails then glaucoma drainage implants are indicated.

VR: In advanced cases of NVG beside Anti VEGF and PRP, due to synechial angle closure surgical intervention for IOP lowering is required. The three surgical modalities often employed are trabeculectomy, tube shunts and cycloablation. The choice of surgical procedure is made depending upon underlying disorder as well as the clinical characteristics of each patient i.e., level of IOP, presence of active or regressed NVI, prior laser or anti-VEGF treatment, prior intraocular surgeries, degree of inflammation, stage of disease, degree of angle closure, severity of glaucomatous optic neuropathy and visual potential. My preferred choice is trabeculectomy with anti-fibrotic agents (mitomycin C) which reduces risk of failure, hemorrhage and inflammation. All patients should be followed up meticulously, though the treatment of NVG still remains a challenge, the advent of anti-fibrotic agents, tube shunts and anti-VEGFs has revolutionized its management and promises relatively better outcomes.

AB: In this patient with age of 50 years and other eye vision of 6/6, my surgical treatment of choice would be Trabeculectomy with anti-fibrotic agent (Mitomycin-C). But before this procedure, I will try to regress NVI with anti VEGF injections and PRP and decrease inflammation by aggressive topical steroids and cycloplegics. In case of Trabeculectomy failure will consider secondary glaucoma valve implants. In case of young or one eyed or both, I would consider Primary implant as chances of failure of conventional filtering surgery or trabeculectomy is high due to subconjunctival scarring.

SP: I will prefer trabeculectomy with mitomycin C along with intracameral Anti VEGF injection.

SD: Trabeculectomy with MMC is my surgery of choice. 5-fluorouracil should be used in follow up if vascularization is seen over bleb. If trabeculectomy fails, then glaucoma drainage device surgery should be considered.

MM: If no exposure to anti-glaucoma treatment earlier, augmented (MMC - 0.04 concentration) trabeculectomy under intravenous mannitol can definitely be given a shot (but only after careful analysis of VFA, ONH status and response to antiglaucoma treatment and to anti-VEGF). Though trabeculectomy has guarded success (especially with neovascular angle and synechial closure), we had done a study (though very limited cohort) which showed good success with trabeculectomy. Glaucoma drainage device is definitely the second choice.

Q5: In a case of absolute glaucoma, do you prefer cyclo-cryocoagulation or trans-scleral diode laser or evisceration?

RKG: These procedures are considered when visual potential is poor or it is a painful blind eye. Trans-scleral diode laser is preferred over cyclocryotherapy as it is associated with lot of pain, inflammation, hypotony and phthisis bulbi. If IOP is controlled with laser treatment then evisceration is not required.

VR: NVG is a potentially blinding disease, if not treated aggressively can leads to Absolute Glaucoma with painful blind eye, if severe pain not improving with strong cycloplegics and anti-inflammatory drugs as a last resort diode CPC is preferred, if facilities are available. If not cyclocryo which is cheaper and commonly available option should be preferred over evisceration. But remember Prevention and early diagnosis is the key to reduce visual loss caused by this devastating disease.

SP: My first choice is transscleral cyclophotocoagulation. If there is no response then cyclocryocoagulation can be considered. Enucleation surgery is preferred for very painful blind eye.

AB: Any cyclodestructive procedure should be reserved only for painful blind eye. I prefer transscleral diode laser as it cause less pain and better tolerated by patients and less associated with hypotony, inflammation and visual loss.

SD: In absolute cases, I prefer cyclocryotherapy.

MM: In absolute glaucoma, cyclo-cryotherapy has poor tolerability (due to IOP spike and conjunctival reaction). Trans-scleral diode laser is more promising with fewer side effects. The problem is the repeatability of these procedures required.

DISCLAIMER – All the answers are recommendations by the practicing consultant and are not in accordance with standard practice pattern. This opinion by expert panelists is compiled by Dr Chahveer Singh Bindra.

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REVIEW ARTICLE

Dry Eye Disease In Users Of Visual Display Devices

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Introduction

Various definitions have been proposed and International Dry Eye Workshop (DEWS) revised the definition of dry eye in 2007 which is widely accepted. According to the revised definition Dry Eye Disease (DED) "Dry eye is a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance and tears film instability, with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface."[1]

Normal tears are made up of 3 layers. Outer one is lipid layer which prevents evaporation and 90 percent of the tear volume is made up of water with a low concentration of salt which constitutes the middle layer. Innermost layer is mucin layer which binds tear film with epithelium. Constant working on screens of electronic devices leads to quick evaporation from the surface due to inefficient blinking [2] or change in composition like deficiency of mucin leading to unstable tear film. Position of monitor also plays a major role. Looking straight ahead or up instead of downward gaze reduces blink rate and increase evaporation of tears which leads to dry eye. Lighting, glare, display quality, refresh rates and radiation also contributes to dry eye.

Various parameters used to assess dry eye disease are Schirmer's test, break-up time (TBUT), tears film osmolarity, ocular surface staining and biochemical analysis. Apart from this, there are different Patient Reported Output Questionnaires which can be used subjectively (FDA PRO guideline & OSDI Questionnaire).[3,4] Protection of cornea is determined by TFBUT and blink rate. The Ocular Protection Index (OPI) demonstrates how the TFBUT and inter-blink interval (IBI) interact to protect the corneal surface. If the amount of time between blinks is longer than TFBUT, an insufficient tear film leaves the corneal epithelial cells unprotected, exacerbating the signs and symptoms of dry eye.

Epidemiology

Prevalence of dry eye disease varies from country to country and range from 5% to 35% in different age groups.[5-8] Approximately 25-30 million people are affected. The prevalence of DED in India is higher than the global prevalence and ranges from 18.5% to 54.3%.[9] There is role of various factors which enhance dry eye disease like prolonged working on computers[10], use of contact lenses, rheumatoid arthritis, Parkinson's disease, use of air conditioners, female patients and age of the subjects. In females it is higher in menopausal age due to imbalance between estrogen and androgen hormones leading to inflammation of lacrimal gland.[11] With advancing age, it's not only deficient secretion by lacrimal glands, probably they have used monitors for more number of years.

Clinical Assessment: Dry eye syndrome is characterized by eye irritation due to changes of the tear film. Symptoms include itching, foreign body sensations, mucous discharge and transitory vision blurring or less often photophobia and eye tiredness. Apart from record of visual acuity, fundus and slit lamp examination, examination of lacrimal system and meibomian glands, other important assessment tools like Schirmer's test, Tear film break up time (TFBUT) and Ocular surface disease index questionnaire (OSDI). [12-15]

All those factors which enhance dry eye disease like contact lens use; old age etc. should be excluded if we want to assess the role of monitors exclusively. Smart phone usage is another major contributor.
The total **OSDI** score is calculated on the basis of the following **formula**. There are 12 questions e.g. is patients sensitive to light?, then depending upon severity scores are given from 0-4 for one question and sum of scores of all questions is taken multiplied by 25 and then divided by number of questions answered.

Formula
OSDI = Sum of scores for all questions answered) × 25 / Number of questions answered
Thus, the OSDI is scored on a scale of 0 to 100, with higher scores representing greater disability.

Discussion

The TBUT depends on the integrity of the system at the interface of the tears and the epithelium. It is therefore related to the integrity of the microvilli of the surface epithelial cells, the cell surface related components, the intervillous mucus, and the mucus layer lying on the microvilli. These can be deranged in many external diseases and by topical medication. If TFBUT time is lesser than blink rate, cornea is unprotected. Mean blink rate is 22 per min in relaxed state and according to some of the studies, it's 10 per min when reading a book and 7 per min on the computers.[16] So probably it is the decreased blink rate which causes more evaporation of tears.

In Osaka study in Japanese office workers who were working on monitors, they found decreased concentration of a substance called Mucin5AC in tears.[17] Although it is commonly used, Vanley et. al criticized the use of the break-up time because of its poor reproducibility.[18]

Ian A. Mackie et al in one of the study in Moorefield's Eye Hospital assessed the association of tear lysozyme and tear flow volume. They found Schirmer's test is valuable in detecting the eye with severely depleted lysozyme secretion and unreliable for detecting the eye with moderately depleted lysozyme secretion or the questionably dry eye.[19] OSDI Questionnaire helps in early detection of DED, this subjective test assess the quality of life in a better manner.
According to The Odissey European Consensus Group, OSDI has poor reproducibility while DEWS report 2007 has accepted it. In a study by Titiyal et.al, based on OSDI questionnaire prevalence of dry eye was reported 32%.[20]
In another study done on prevalence of dry eye disease in 300 bank employees, majority of patients in the category of moderate to severe dry eyes were using monitors for more than 6 hours assessed by Schirmer's test and TFBUT, though not significant statistically. OSDI scores were found significant statistically in moderate to severe dry eye cases. Though exact correlation between objective and subjective tests was not found but results were not contradictory also.[21]
Now there are various emerging therapies, targeting underlying cause like intranasal tear neurostimulator, TrkA receptor agonist that acts on neurotransmitters, dexamethasone-loaded punctal plug, interleukin-1 receptor inhibitor administered as a topical drop, Thymosin-β4- a naturally occurring protein that promotes corneal surface healing, Rebamipide (a mucin stabilizer) seems to add newer dimensions in management of dry eye disease. There is a software-controlled, wearable eyelid technology that directs heat to the meibomian glands and helps in expressing secretions. Assessment of tears soluble factors by ELISA, quantification of interleukins are recent advances in therapy. [22,23]

Conclusion

With change of life style and inadvertent use of technology, most of the manual work is done on monitors. Higher stress level, use of air conditioners, improper position of monitors and constant staring is associated with poor blinking and so the associated symptoms of dry eye. This has affected overall work efficiency and so the output of organization. Patients' education, deliberate ergonomics of computer use, including screen height, blinking exercises, chair position, glare protection and artificial tear substitutes can minimize the symptoms of dry eye syndrome and prevent serious complications. OSDI score is a very informative tool for functional assessment.

Breaks and Blinking - Avoid eyestrain by preventing continuous use of eyes by taking short breaks. One of the golden rule is '20-20-20' rule, every 20 minutes, focus the eye on an object 20 feet (6 meters) away for 20 seconds or blink every time you hit the "ENTER" key or mouse click. Lowering the monitor by 10 to 20 degree from eye level can reduce the palpebral aperture and so reduces evaporation of tears.

Studies based on patient's subjective response through questionnaire like OSDI, evaluation of tear film stability helps in diagnosing the disease. *But studies based on biochemical analysis assessing individual components as deficiency of mucin or lipids, tears lysozymes are to be promoted on regular basis*. Target therapy seems to be promising. Assessment of tears soluble factors by ELISA, quantification of interleukins can add newer dimension to the management of disease.

Conflict of interest: Nil

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REVIEW ARTICLE

Phakic IOL's in High Myopia

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Introduction

Phakic Intraocular lens (IOL's) forms part of a technology that expands the range of refractive surgery to cover higher degrees of myopia, hyperopia, and astigmatism that were previously not possible to treat. It provides an effective solution to high refractive errors in candidates who are not suited for corneal refractive treatments. The options available for treatment of high myopia include excimer laser ablative procedures such as laser in situ keratomileusis (LASIK), photorefractive keratectomy (PRK), laser-assisted subepithelial keratectomy (LASEK) or Epi-LASEK, intraocular procedures such as intracorneal ring segments (ICRS or INTACS), clear lens extraction (CLE) or phakic intraocular lens/IOL (PIOL) implantation.

Excimer laser procedures are effective for the treatment of low and moderate myopia. Intracorneal ring segments may be used to treat low myopia but their results are highly unpredictable. Phakic IOL's provide for a better quality of vision not available with other techniques. The preservation of accommodation and minimal manipulation of the crystalline lens are other advantages over clear lens extraction.[1]

History of phakic IOLs

The concept of phakic IOL's was introduced in 1953 when Benedetto Strampelli implanted an anterior chamber IOL (ACIOL) for the correction of severe myopia in phakic eyes. These did not come into practice due to various complications like endothelial cell loss, iritis, pupillary block, and glaucoma. Joaquin Barraquer then introduced an IOL with elastic loops better adaptable to the anterior chamber for myopic phakic eyes. These IOL's also caused similar complications and thus did not become very popular.[2]

Over time phakic AC-IOLs progressively improved with the development of iris fixed IOL's by Fechner and Worst [3], angle supported AC-IOLs by Baikoff [4] and posterior chamber IOLs (PC-IOLs) with ciliary groove fixation by Fyodorov.[5]

Preoperative evaluation

A complete history of the refractive stability, comfort, and satisfaction with glasses or contact lenses, the age of first spectacle correction, any history of amblyopia or strabismus should be taken. The parameters that need to be considered preoperatively include:

a. Patient's Age and refraction– A well- motivated patient above 21 years of age who has achieved refractive stability and has less than 0.5 D of refractive change in 1 year is an ideal candidate for phakic IOL.[6] Both manifest and cycloplegic refraction should be performed. The best-corrected visual acuity in the undilated and cycloplegic state (after performing cycloplegia with homatropine or tropicamide) should be recorded.
b. Pupil size – A scotopic pupil size larger than the optical zone of the implant would lead to glare and halos which may be severely debilitating in the postoperative period. [7]

c. Corneal evaluation - endothelial status by specular microscopy, and corneal topography are important before planning a phakic IOL implantation. Phakic IOL implantation leads to endothelial cell loss in the postoperative period, so a healthy preoperative endothelium with a cell count of at least 2300 per millimeter square should be ensured. Corneal topography needs to be done to rule out keratoconus, corneal ectasias or any other corneal abnormality.[6] [8]
d. Anterior chamber depth - A substantially deep anterior chamber is mandatory for a phakic IOL. Most phakic IOL does require an anterior chamber depth of at least 3 mm.[9]
e. Anterior chamber angle and gonioscopy - The accepted range of iridocorneal angle aperture for phakic IOL implantation is more than or equal to 30 degrees, which corresponds to Shaffer grade 3 and 4 or Scheie grade 0 and 1. Gonioscopy needs to be performed preoperatively to identify narrow or abnormal angles, which may predispose to secondary glaucoma after phakic IOL implantation. [10]
f. Sulcus to sulcus (STS) measurements – This is essential for sizing of the phakic IOL and measurement of lens vault (distance between the posterior surface of the IOL and the anterior lens capsule) in case of a phakic posterior chamber IOL (PCIOL). An approximate calculation of the size of the IOL depending on the vault is made using the white to white (WTW) measurement by adding 0.5 mm to the measured value in myopes and subtracting 0.5 mm in hyperopes. WTW may be measured using calipers, scanning slit topography including Orbscan II (Bausch and Lomb, Technolas, USA), digital caliper, Zeiss IOL master 500 and 700 (Carl Zeiss Meditec, Germany), Lenstar (LS 900, Haag Streit, USA), Scheimpflug based devices such as Pentacam (Oculus, Wetzlar, Germany), Sirius (CSO, Florence, Italy), and Galilei (Ziemer, Port, Switzerland), ultrasound biomicroscopy (UBM), or digital ultrasounds such as the Artemis VHF (Arcsan Inc, Morrisson, Colorado).[11][12][13]. Direct measuring the STS diameter using ultrasound biomicroscopy or very high frequency (VHF) digital ultrasound may be a more reliable method for the size estimation of phakic IOL's. [14][15] Ideal vault size for phakic posterior chamber IOL's is 1 plus or minus 0.5 of the corneal thickness which is in the range of 250 to 750 microns. [16]
g. IOL Power Calculation – Biometry, keratometry, anterior chamber depth (ACD), lens thickness, preoperative refraction need to be accurately measured and the appropriate formula applied. Van der Hejde nomogram may be used for IOL power calculation.[17]
h. Peripheral retinal examination - Laser of retinal breaks, if present, are important to prevent complications of retinal detachments in the postoperative period, especially as pupil dilation remains limited with anterior chamber IOL's

Relative contraindications for phakic IOLs include: [6] cataract, chronic uveitis, low endothelial cell count, visually significant retinopathies, iris abnormalities, angle abnormalities and glaucoma.

Types of phakic IOL's

I. Anterior chamber IOL's:
These include iris fixed and angle supported IOL's.

a. Iris fixed –
Verisyse IOL, Abbott Laboratories, USA/ Artisan IOL, Ophtec BV, Netherlands– made of Polymethylmethacrylate (PMMA), FDA approved in September 2004 for myopia of -3.0 to -23.5 D and hyperopia of +1.0 to +12.0 D, with an overall length of 8.5 mm.[18] Artiflex and Veriflex are the flexible models, also available as toric IOL's in the range of −1.0 D to −13.5 D (spherical) and −1.0 D to −5.0 D(cylindrical).[19]
The Verisyse and Artisan IOL's are one-piece PMMA IOL's available in optic diameters of 5.0 and 6.0 mm. Veriflex and Artiflex IOL's have rigid PMMA haptics attached to a soft silicone 6.0-mm optic. The unfolded lens can be flexed and inserted through a 3.2 mm incision and returns to its original shape after unfolding inside the anterior chamber.

b. Angle supported -
Acrysof cachet IOL (Alcon, USA) - single-piece foldable hydrophobic acrylic IOL available for myopia of −6.0 D to −16.5 D. It was introduced in 2008 but later had to be withdrawn due to excessive endothelial cell loss. [20]
Phakic 6 (Ophthalmic Innovations International, Ontario, California) with and without heparin coating - single piece PMMA IOL with haptics angulated at 18 degrees - available for –2.0 D to–25.0 D of myopia, and +2 to +10 D of hyperopia[21]

ZSAL-4 (Morcher, Stuttgart, Germany)- one-piece PMMA IOL with haptics angulated at 19 degrees, available for myopia of -10.0 D to -23.0 D.[22]
NuVita MA 20 (Bausch & Lomb, Claremont, California) - PMMA IOL available for myopia of -6.0 D to -20.0 D[21] This was associated with pupil deformation and angle abnormalities and was eventually withdrawn due to excessive night vision problems.[23]
GBR/Vivarte (Ciba Vision, USA, and IOLTECH, France) - hydrophilic acrylic IOL with non-angulated PMMA haptics available for myopia of -7.0 to -25.0 D[24]
Kelman Duet implant (TEKIA, Irvine, California) - 2 piece silicone IOL with asymmetric tripod shaped PMMA haptics and a separate 5.5mm optic, available for myopia of –8.0 to –20.0 D. [21]
I-Care (Corneal Inc., France) - single-piece hydrophilic acrylic IOL available for myopia of -2.0 to -25.0 D, and hyperopia of +2.0 to +10.0 D.[25] It has four haptics which prevents rotation of the IOL and minimizes angle occlusion.
None of these IOL's are FDA approved.

II. Posterior chamber IOL's:

Phakic refractive lens (PRL) (Medennium Inc, Irvine, California/CIBA Vision, Duluth, Georgia/ Zeiss Meditec, Jena, Germany) –plate haptic silicone IOL available in sizes of 10.8 to 11.3 mm for myopia and 10.6 mm for hyperopia – usually not used for astigmatism correction as rotation is a possibility. It was available for myopia of -3.0 to -20.0 D and hyperopia of +3.0 to +15.0 D but was later withdrawn due to reports of zonular dehiscence and posterior subluxation.[23] [26][27]
Visian Implantable Collamer lens (ICL) (STAAR Surgical, Monrovia, California, USA) – a copolymer of porcine collagen/ hydroxyethyl methacrylate (HEMA). The Collamer material patented by Staar is made of 60% poly-HEMA, water (36%), and benzophenone (3.8%), with an optic diameter ranging from 4.9 mm to 5.8 mm and a total diameter of 11.5 to 13 mm for myopia and 11 to 12.5 mm for hyperopia. [10]
The Visian ICL V4c version has a central 360-micrometer hole, called KS-Aquaport that allows for natural aqueous humor flow, eliminating the need for an iridectomy. It received FDA approval in December 2005 for myopia of -3.0 to -20.0 D, hyperopia of +3.0 to +12.0 D and astigmatism of up to 6 D. Its toric version got approved in 2018 [6]
STAAR Surgical has also launched the EVO+ (Evolution in Visual Freedom) ICL and the EVO+ toric ICL in September 2011 for the correction of a wide range of refractive errors with premium outcomes. It consists of an optic size of 6.1 mm designed for larger pupils. This is not FDA approved.[28]
The Implantable Phakic Contact Lens (Care Group, India) is a hydrophilic acrylic posterior chamber phakic IOL available in both spherical and toric models in ranges -1.0 to -30.0 D for myopia, +1.0 to +15.0 D for hypermetropia and up to 10 D of astigmatism. It has an optic diameter range from 5.75 to 6.20 mm and an overall diameter range from 11.0 mm to 14.0 mm. The IPCL V2.0 model has a central 380-micrometer hole for aqueous flow and also to minimize light scattering. It has 2 more holes in the periphery of the optic for aqueous flow across the anterior chamber and 4 holes outside the optical zone. The IPCL V2.0 Presbyopic lens is available for correction of up to 4.0 D of presbyopia and has a refractive-diffractive trifocal design, catering to near, far, and intermediate vision. [29]

Indications for FDA approved phakic IOL's: [23]

Visian ICL - correction of myopia from -3.0 to -15.0 D and reduction of myopia from -15.0 to -20.0 D with less than 2.5 D of astigmatism at the spectacle plane in patients aged 21–45 years with an anterior chamber depth of more than 3.0 mm and refractive stability within 0.5 D for 1 year before implantation.
Artisan/ Verisyse IOL - Correction of myopia from -5.0 to -20.0 D with less than 2.5 D of astigmatism at the spectacle plane in patients aged more than 21 years with an anterior chamber depth of more than 3.2 mm and refractive stability within 0.5 D for 6 months before implantation.

Contraindications for FDA approved phakic IOL's: [24]

Visian ICL - Anterior chamber angle less than grade II determined by gonioscopy, pregnant or nursing females, and endothelial density in the range 1900–3875 cells/mm2 depending on age.
Artisan/ Verisyse IOL - Any angle abnormality, iris abnormalities such as peaked pupil or elevated iris margin, pregnant or nursing females, endothelial density in the range 2000–3550 cells/mm2 depending on age.

Surgical Technique:

Some IOL's such as the anterior chamber IOL's, PRL and Visian ICL 4 require preoperative laser iridotomy or intraoperative surgical iridectomy to avoid pupillary block in the postoperative period. [30]
Angle-fixated phakic lenses – The foldable IOL's can be inserted into the anterior chamber through a small incision of less than 3.5 mm with the help of forceps or injectors.
Kelman Duet anterior chamber IOL - It has a unique implantation technique in which the haptic is inserted into the anterior chamber through a 2 mm incision or less, and the optic is loaded into a cartridge and injected through the same incision with the help of an injector and then engaged into the haptics. It provides the option of an optic exchange, if required due to a change in refractive error.[21]
The non-foldable IOL's like the ZSAL-4 and NuVita require larger incisions.[21]
Iris-fixated IOL - A corneal or limbal incision is fashioned. After constricting the pupil pharmacologically, enclavation of the IOL is performed in which a portion of the mid-peripheral iris is tucked into the opposed claws of the IOL. The earlier non-foldable IOL's required a larger 5.5 mm incision while the foldable ones can now be inserted using smaller incisions. [31]
Posterior chamber IOL's - The Visian ICL and Care group IPCL can both be implanted through a 2.8 mm clear corneal incision and under topical anesthesia, wherein each footplate of the haptic is tucked into the ciliary sulcus. Loading of the ICL in its cartridge is done after filling it with viscoelastic, placing it in the loading area of the cartridge, and grasping with a fine long forceps (figure 1).[32] The PRL is injected into the posterior chamber through a 3.0 or 3.5 mm clear corneal incision and stabilized in the sulcus (figure 2). [33]

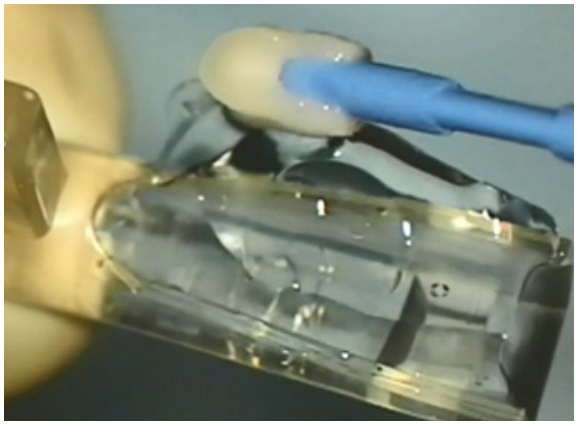

Figure 1. Loading of the ICL into cartridge.

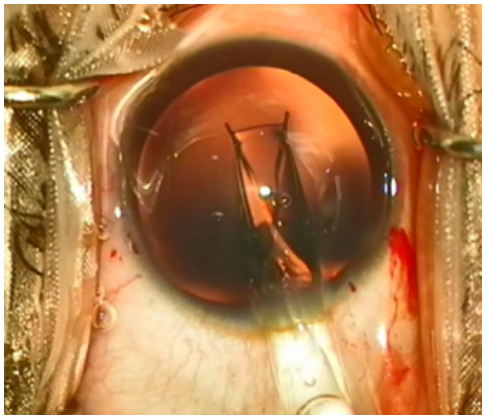

Figure 2. Insertion of ICL in sulcus through a temporal clear corneal incision.

Toric IOL's - For toric ICL implantation, preoperative marking needs to be made using manual methods like tonometer markers, electronic toric markers, Neuhann one-step toric bubble marker, Geuder-Gerten Pendulum marker or image-guided systems like Verion Image-Guided System (Alcon Laboratories, Texas, USA), Callisto Eye and Z align (Carl Zeiss Meditec AG, Dublin, USA), iTrace with Zaldivar Toric Caliper (Tracey Technologies, Houston, USA), and TrueVision 3-D Surgical System (TrueVision Systems, Santa Barbara, California, USA). [34][35] The ICL toric power calculator gives an implantation diagram to determine the axis of placement for appropriate toric effect. [36] In its toric version, the IPCL needs to be aligned between 0 and 180 degrees as the astigmatic component is incorporated accordingly.

Complications:

Endothelial cell loss – It is more common with ACIOL's. This may lead to corneal decompensation and if the endothelial count drops below 2000 cells per millimeter square, explantation of the IOL may be required. A 1% mean annual reduction in endothelial cell count has been seen in angle fixed IOL's when the distance between the corneal endothelium and the edge of the IOL is 1.43 mm. This increases to 1.7% when the distance reduces to 1.2 mm and is minimal when the distance is 1.66 mm.[37]
In a 12-year retrospective study on 144 eyes implanted with ICL, Moya et al reported 6.46% surgically induced endothelial cell loss during the first year, beyond which an average yearly decrease rate of 1.20% was noted. [38]

Pigment dispersion – may lead to lens deposits. Usually, no intervention is required.
Chronic inflammation and uveitis – It is more commonly seen with ACIOL's.
Pupil distortion – It is seen in ACIOL's. This may cause intractable glare and maybe cosmetically unacceptable.
IOL rotation – It is due to inappropriate sizing. It may lead to induced astigmatism in cases of toric IOL's.
Pupillary block and glaucoma – Pupillary block may occur due to inappropriate vaulting in the case of PCIOL's. It may resolve after pupil dilation and use of pressure-lowering agents but the definitive treatment remains the creation or enhancement of a previously created peripheral iridotomy. Retained viscoelastic material may also cause raised intraocular pressure (IOP). Angle fixed IOL's may block the angles and lead to a rise in IOP.
Glare and halos – when the scotopic pupil size is greater than the optic of the IOL. Miotic agents may be given for resolution.
Cataract formation – It occurs in case of low PIOL vault or undersized PIOL. This is mostly in the form of anterior subcapsular opacities that develop due to the pressure of the IOL on the crystalline lens. Silicone lenses such as PRL predisposes more to cataract formation than collamer of ICL. [39][40][41]
In an 8 year follow up of 41 eyes implanted with V4 ICL by Igarashi et al, asymptomatic ASC was reported in four eyes (9.8%). [42]
In a 5-year retrospective study, Brar et al reported that, in a total of 957 eyes, significant anterior subcapsular cataract (ASC) requiring explantation developed in four eyes (0.4%). [43]
Sanders et al, in their study on 106 eyes with -12.00 D or more of preoperative myopia reported clinically significant cataracts in seven eyes (6.6%), whereas no cataract occurred in 420 eyes with preoperative myopia less than -12.00 D. They thus concluded that a higher degree of baseline myopia more frequently predisposes to cataract formation. [44]
Retinal detachment - As most cases are high myopes, there are chances of rhegmatogenous retinal detachments (RRD) more than emmetropic eyes, in the range of 0.7% to 3.2%.[45] However, such eyes with high myopia are already predisposed to retinal detachment and the association of PIOL with RRD needs further evaluation.

Prognosis and outcomes:

Success with phakic IOL's largely depends upon accurate preoperative evaluation which determines the vaulting in case of a PCIOL and sizing in case of an ACIOL.[46]
With optimum calculations, these IOL's are known to effectively correct myopia of up to 20 dioptres(D) with good results. As more specialized instruments are developed, higher rates of successful implantation can be expected. Any residual refractive error may be corrected using bioptics, which is the combination of an intraocular procedure with a keratorefractive procedure. Pseudophakic ametropia may also be corrected with the help of phakic IOL's. [47]

Conclusion:

Phakic IOL's provide a good option for management of high refractive errors not amenable to other modalities of treatment, while maintaining the natural asphericity of the cornea and allowing the crystalline lens to retain its normal function. Further developments in phakic IOLs will help optimize lens designs to decrease long-term anterior segment complications; evolve better surgical techniques; and expand clinical indications.

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REVIEW ARTICLE

Mucous Membrane Graft (MMG)

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With the advancement in ophthalmic equipment's and surgical techniques ocular surface restoration is now possible by the utilization of different graft tissues either allograft or auto graft depending upon the ocular condition. Mucus membrane graft (MMG) has gained popularity recently for the restoration of ocular surface including correction of lid abnormalities, management of shallow or contracted socket, replacement of keratinized conjunctiva in Steven Johnson`s syndrome (SJS). Success rate of mucous membrane grafting is depending upon the host ocular condition, extent of abnormalities, surgical skill, graft harvesting site and techniques etc.
Graft survival and revival of ocular surface is still a challenging job for Oculoplastic surgeon`s to increase the success rate and minimize the complications for a successful graft outcome.

Mucous membrane graft (MMG)

a. Tarso-conjuctival graft: A posterior lamellar graft in eyelid reconstruction can be done by a free tarso-conjuctival graft obtained from the upper eyelid. Tarsus provides structural support for the upper eyelid and the adjacent conjunctiva. It is important to evert the upper eyelid preoperatively to ensure that the height of the tarsus is adequate. At least 3.5 mm of tarsus from the eyelid margin is required to support the upper eye lid for proper functioning.

b. Lip / Buccal mucosa: Lip and buccal mucosal graft harvesting is not an easy. Accessibility is a major problem the area is limited and difficult to approach. Lower lip is preferred site to obtain a graft. In case of buccal mucosa removal, special care must be taken while making incision to safeguard the parotid duct which opens directly opposite the upper molar teeth. (Figure 1). Mucous membrane grafting should always be performed under general anesthesia. MMG is better to remove manually, before removal it should be marked and inj. lignocaine 2% or normal saline must be injected over the area of interest for easy dissection of marked tissue. No suture is required usually and that area heals spontaneously within 2-3 weeks & hardly leaves any scar [2]

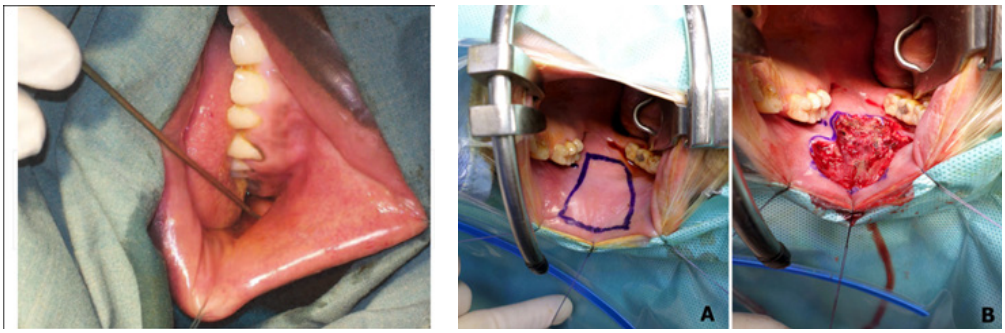

Figure 1. Location of parotid duct opening (a) incision marking (b) donor site

c. Hard palate graft: Hard palate mucosa is more suitable graft in case of lower lid retractor recession and lower lid cicatricial entropion because of its tough nature than lip or buccal mucosa. It has a rougher surface because of keratinized epithelium unlike lip mucosa and it hardly shrinks. Hard palate is an alternate option after lip or buccal mucosa for contracted socket reconstruction. [3]

d. Nasal septal cartilage graft: A nasal septal cartilage graft makes an ideal posterior lamellar replacement for lower eyelid reconstruction where the whole of the lower eyelid has been resected. It is usually used in conjunction with a Mustarde cheek rotation flap.

e. Auricular cartilage graft: For the tarsal replacement in upper or lower eyelid reconstruction, auricular cartilage graft is most preferred graft (patient's pinna) although easily accessible area and size of graft but in contrast to the hard palate graft, the auricular cartilage graft has lack of mucosa.

Indications:

- Ocular surface reconstruction in dry eye
- Steven Johnson's Syndrome (SJS) with symblepharon formation
- eyelid reconstruction as posterior lamella
- Conjunctival fornix reconstruction
- Shallow/Contracted socket reconstruction
- Major eyelid entropion
- Covering of conjunctival space following an enucleation etc.

Dry eye-associated ocular surface disease: Mucous membrane grafting in the clinical management of dry eye-associated ocular surface disease is now a day's done. [4]

- The reformation and maintenance of a conjunctival fornix requires the addition of epithelial tissue, or a basement membrane which can be populated by healthy host epithelial cells.
- A conjunctival or tarsal autograft is the ideal material.
- Oral mucosa does not contain goblet cells and therefore does not supplement the tear film: a full-thickness oral mucous membrane
- Nasal mucosal grafts contain goblet cells that may contribute mucous to the tear film.
- Split-thickness mucosal grafts contract more but are less bulky therefore should be used on the globe.
- Hard palate grafts are the thickest oral mucosal grafts and contract the least.

Mucous membrane graft (MMG) in Stevens Johnson`s disease with symblepharon: The acute stage of the SJS is characterized by bilateral catarrhal and membranous conjunctivitis.

- During the chronic stage of the disease, most patients have various alterations of the ocular surface, such as symblepharon, entropion, trichiasis, dry eye, limbal cell deficiency, conjunctival inflammation and corneal neovascularization.(Figure 2) [5,6]
- The major aim of treatment in late phases of SJS is ocular surface reconstruction to correct cicatricial sequalae & chronic inflammation.
- A full-thickness oral MMG is the simplest graft to use if a conjunctiva is not available. It is preferred over split-thickness mucosal grafts as it contracts less in comparison, though a split-thickness graft is preferable from the cosmesis point of view because of its lesser bulk and pinky appearance.[7]
- Mucosal tissue transplantation can be considered in situations where cost and inadequate infrastructure are concerned. The aim of treatment of the chronic phase of ocular surface disorders like SJS is restoration of the anatomical structures and physiologic properties of the ocular surface.

Reconstruction of fornix:

- Reconstruction of the Fornix If marked contraction of the conjunctiva of the socket is established, (Figure 3a and b) extra mucosa must be inserted. Reconstruction is performed under general anesthesia.
- The lower fornix is reformed and scar tissue is dissected by a transverse incision through the contracted conjunctiva of the lower fornix. After the incision is deepened with scissors to expose the periosteum of the inferior orbital rim, enough oral mucosal graft is inserted to create a new fornix without tension.

- Deepening of the reconstructed fornix should be supported by fixation to the periosteum of the orbital rim by sutures tied over strips on the skin. The conjunctiva of the upper fornix is incised transversely close to the upper border of the tarsal plate and reflected from the upper lid retractors. Then the dissection is continued beyond the uppermost extent of the fornix.
- The graft is inserted and supported with a conformer formed by one half of a silicone tube sutured in place with sutures which pass through the full lid thickness. Reconstruction of Filtering Bleb a lid speculum is inserted and 7/0 silk sutures placed through the clear cornea for retraction and exposure of the affected bleb. Xylocaine 1% is injected subconjunctival surrounding the atrophic bleb. A blade is used to perform a shallow keratectomy anterior to the bleb edge. The bleb is removed from the sclera. The edge of the residual flap is sutured edge to edge with the buccal mucous graft with a resorbable 9/0 suture.
- Postoperative Management Topical antibiotics and steroids are administered 4 times a day for approximately one month.
- The mucous graft tissue is initially white and avascular but after 1 week it is getting pink, vascularized and viable looking which further improves after 3 months.(Figure 3 and 4)
- Various alternative and in part newer techniques, oral mucosal grafting is still an up-to-date suitable procedure for the replacement of the conjunctiva. Advantages include easily accessibility of grafts in enough size even for repeated procedures, fast and cheap grafting in contrast to ex vivo methods and a high stability of the grafts. For some indications, nasal mucosal grafts might be superior to oral mucosal transplants due to the lack of goblet cells in the oral mucosa. The clinical experience of the benefit of ex vivo propagated mucosal epithelial cells for treatment of ocular surface disease and limbal stem cell insufficiency will probably markedly increase in the near future.

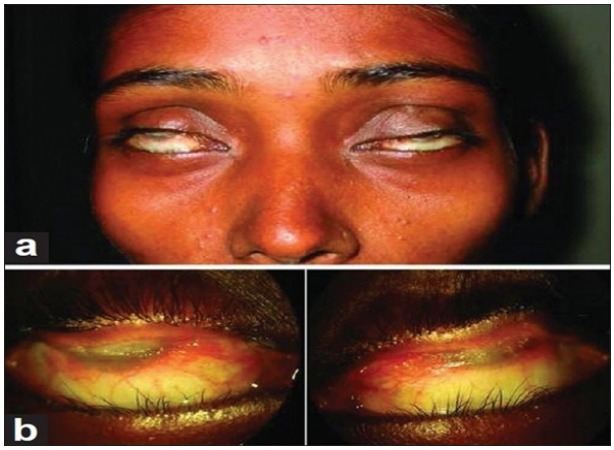

Figure 3 Early post-operative view (7th day) : – (a) gross appearance. (b) Close-up view of both eyes with the bandage contact lens and symblepharon ring seen in situ.

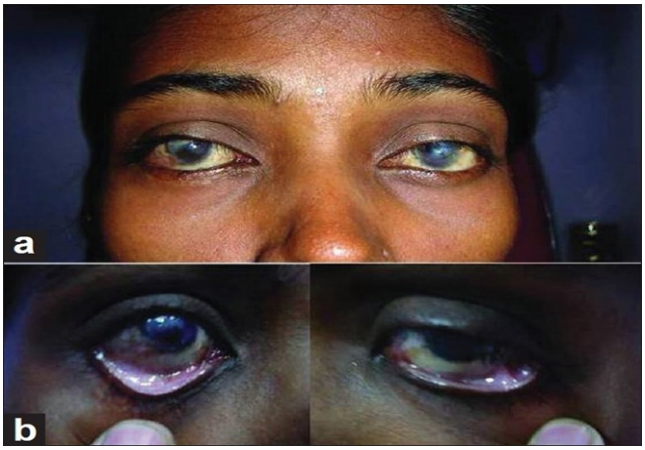

Figure 4. (a) Postoperative view (3 months): gross appearance. (b) Close-up view showing well reconstruction in the inferior fornix in both eyes

MMG for lid reconstruction:

A posterior lamellar mucous membrane graft is typically used for patients with a severe entropion with marked symblepharon, severe lagophthalmos and eyelid retraction.

- A graft is indicated if the patient is requiring a subsequent penetrating keratoplasty. Amniotic membrane may be used as an alternative graft if the patient agrees to the use of donor material.
- It is preferable to avoid the use of a hard palate graft for use in the upper eyelid as the corneal surface, which is often already compromised, can be damaged by its rougher surface. The procedure is usually performed under appropriate anesthesia as per the patient's age and co-operation.
- Size of the graft depends upon the size of defect. Usually larger grafts are required to fill a larger gap; these grafts can be harvested from lip or buccal mucosa.
- For anterior lamella of the lid, full thickness skin grafts can be taken either from contralateral lid or post auricular region, inner upper arm, supraclavicular region and nasolabial fold.
- For posterior lamella, tarsoconjunctival free graft is always a good match. In adverse condition, lip mucosa, buccal mucosa, nasal or hard palate are the best suitable tissues in worst condition few other available options like auricular cartilage may be tried. (figure 6)

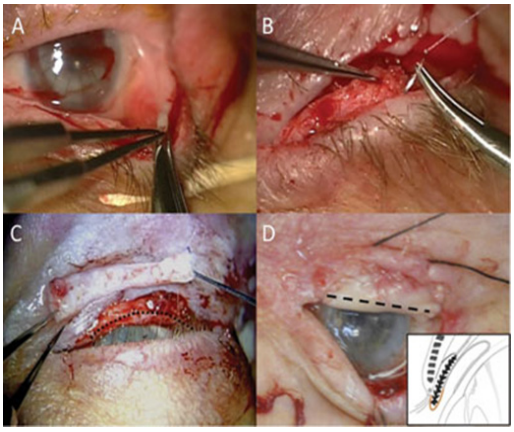

Figure 5. Lid reconstructions with mucous membrane graft

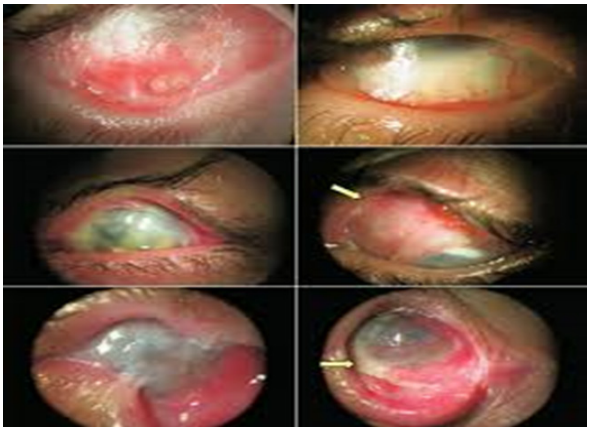

Figure 6. (a) Chemical injury with symblepharon (b) release of symblepharon with MMG

Choice of Graft Material: The ideal material for fornix reconstruction is a healthy conjunctival or tarsal autograft; however, especially in bilateral disease, there is limited availability. If conjunctiva or tarsus is not available, a full-thickness oral mucous membrane graft is a simple way to use. Split thickness mucosal grafts contract more than full-thickness grafts. They are therefore less suitable for fornix reconstruction. But they can serve as an alternative for replacement of the bulbar conjunctiva. Grafts from the hard palate are the thickest oral mucosal grafts. They contract the least but are more difficult to harvest than other mucosal grafts. So, they should be chosen if enough buccal or labial mucosa is not available or when it is desirable to avoid contracture. Oral mucosa does not contain goblet cells and therefore does not support the tear film.

Advantages and disadvantages of different graft materials for conjunctival replacement (modified from Henderson and Collin) [7]

Graft material	**Oral mucosa**	**Nasal mucosa**	**Amniotic membrane**
Stability	high	High	Low
Thickness	thicker	Thicker	Thinner
Color	pink	pink	colorless
Goblet cell replacement	No	Yes	No
Easy accessibility	Yes	No	Yes
Epithelial stem cells	yes	yes	no

MMG for fornix reconstruction:

Mucous membrane transplants have been used for over a century in the reconstruction of fornixes obliterated by symblepharon formation. Oral mucosal graft is commonly used during Oculoplastic surgeries to replace the ocular (corneal and conjunctival) surface as well as for reconstruction of the eyelid, the fornix and the socket. For reconstruction of the lid margin, oral mucosal graft can correct trichiasis and entropion. Recently, Iyer et al. [8] used mucous membrane grafting for correcting lid margin keratinization in Stevens-Johnson syndrome to reduce ocular surface inflammation and to improve patient comfort and visual acuity. However, their study excluded severe cases with extensive symblepharon, entropion, ocular surface cicatrization, and severe dry eye. In our experience, oral mucosal graft can be used not only for correcting distichiasis causing blink-related micro trauma, but also for incomplete closures that are frequently found in severe cicatricial ocular surface diseases. (Figure 6)

Surgical steps:
For reconstruction of posterior lamella after symblepharon release in post SJS/Chemical injury (fig. 6)

- 2-3 ml of bupivacaine with adrenaline mixed with lidocaine is injected along the upper lid skin crease.
- A 4/0 Silk traction suture or a cotton suture is placed horizontally through upper eyelid margin centrally and the eyelid is everted over a Desmarres retractor.
- All symblepharon are divided with Westcott scissor. The conjunctiva at the upper border of the tarsus is incised and dissected free from all subconjunctival scar tissue into the superior fornix and onto the bulbar surface of the globe.
- Next, a template is taken of the conjunctival defect or the size of the defect is measured.
- The lower/upper lip mucosa is infected with local anesthetic.
- The lower lip is everted with fingers or traumatic forceps. The lip mucosa is dried with a swab. The template is transferred to the lower lip mucosa or the required lip mucosa is marked with avoiding the vermillion border and frenulum. This is outlined with a sterile gentian violet. The marked incision line is gently incised with a no. 15 scalpel blade and the graft removed very carefully using blunt-tipped Westcott scissors and small-toothed forceps. The Westcott scissors should be kept under the surface of the graft with the edge of the graft drawn horizontally to ensure that the graft is not inadvertently perforated and the dissection is not taken too deep. Dissection in a deeper plane risks leaving areas of the lip with sensory loss. The graft donor site is compressed with topical adrenaline on a swab. Bipolar cautery is used to cauterize any bleeding vessels. The graft is carefully shaped with Westcott scissors and graft is then placed ensuring that the original graft surface faces upward; on the recipient bed and interrupted 7/0 Vicryl sutures are placed from the graft edge to the recipient conjunctival edge.
- The graft must be maintained in position with the use of a symblepharon ring when the graft is placed onto the globe or a conformer of an appropriate size and shape when the graft is placed centrally in an anophthalmic socket. If the graft is used to reconstruct a conjunctival fornix it should be held in place with a silicone retinal band and 4/0 Nylon fornix-deepening sutures. Topical antibiotic ointment is instilled into the eye. A compressive dressing is applied.[9]

Postoperative care of donor site
The patient is instructed to avoid any hot drinks or hot food for a period of 1-2 weeks. The patient is discharged on a broad spectrum oral antibiotic for a week and an oral antiseptic mouth wash for 2 weeks. Topical preservative free antibiotic drops are instilled into the eye four times a day for 2 weeks. A preservative free topical lubricant is used. The patient is instructed to sleep with the head elevated for 1-2 weeks. The symblepharon ring is maintained in place for a minimum period of 6 to 8 weeks. The patient must be reviewed twice weekly to ensure that the symblepharon ring does not cause any problems. [9]

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WHAT'S NEW

Imaging Innovation In Ophthalmology

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Introduction

Imaging in ophthalmology is an integral part of an ophthalmology department. It allows the clinician to record the findings from clinical ocular examination in an objective, reproducible, transmissible and durable manner. Many imaging modalities are of key importance in diagnosis of ocular pathologies. In recent times where doctor to patient ratio is decreasing with time it is not humanly possible for a clinician to give physical presence. Many diseases like diabetic retinopathy and ROP whose dreaded complications can be avoided with prior diagnosis and proper screening can be done using telemedicine for which again imaging comes into play. As a result, ophthalmic imaging is essential for diagnosis, treatment, and long-term monitoring of many ocular conditions. In addition, it plays a central role in ophthalmic disease screening, teaching, clinical trials and in virtual clinics and telemedicine.
With time imaging modalities have got very refined and have reached a great level of sophistication. This article provides brief details of the imaging devices available and their role in modern day practice. Also, it cites the recent innovations and low cost "Jugaad" available to us.

Recent advances

Portable imaging devices:
Portability is need of the hour as far as ophthalmology is concerned because of the following reasons-:

- Geriatric age groups of patients are majority.
- Bed side opinion for patients who cannot be shifted to OPD
- Tele medicine
- Screening for diabetic retinopathy and ROP

There has been a great enhancement in all the imaging devices for portability with following options:

1. **Portable anterior segment cameras**– There are devices to capture high definition anteriorsegment images and also with the help of integrated software one can diagnose things like tear film meniscus height, analyze meibomian gland function and complete dry eye evaluation. Also, it could be used to diagnose and keep a record of anterior segment disease in telemedicine.
2. **Portable Fundus cameras**- These devices are of great help to capture fundus pictures & have a very small learning curve. They come in handy for diabetic screening, disc evaluation for glaucoma screening.
3. **Portable OCT**- Nowadays, portable OCT devices are also available which is of great help for seeing patient bedside.
4. **Portable Contact fundus cameras**- Contact wide filed portable fundus camera are of utmost important for ROP screening in peripheral centers where physical presence of a clinician is not possible every time. We can also do angiography with these devices.

Wide field Fundus cameras

With these devices we can get up to 200º of fundus view in a non mydriatic eye of patient, with which we can monitor almost whole retina in a single image.

Innovations in Imaging

With the constant up gradation happening in instruments and with the increasing sophistication of newer devices the price factor has also gone up, which is a major factor of consideration for non-institutional practice and single ownership eye clinics. There have been many innovations aka "Jugaad" to develop devices with lesser capital and with comparable results. Smartphone being the epicenter of all these innovations as with smartphone we can get a very high end camera and a smooth processor in portable form.

Smartphone based imaging devices

Smartphone assisted Slit Lamp photography
It has a short learning curve but with small training one can master techniques of smart phone imaging. Free hand photography being the most primitive one in which one has to hold camera 5mm from the slit lamp eye piece with one hand to be used for support for smartphone and other hand to click images (Figure 1), do remember to turn on flash mode.

Figure 1: Free hand slit lamp photography

Figure 2: Slit lamp photography using adaptors

Figure 3: DIY adaptors using PVC pipes (Courtesy: John Davis Akkara)

Many of us find it difficult to master free hand technique and also we cannot take posterior chamber images as both our hands are occupied for this. To overcome this, we can use cheaply available adaptors which can be procured from online shops like amazon and eBay (figure 2), DIY adaptors like Dr. Biju Raju's DIYretcam,[1] (figure 3) can be easily made at home with readily available material following the instructions in the article published in IJO and has a very good ease of use.

While using adaptors as one hand is free we can place 90/78D lens and take videos and images of fundus as well (figure 4). To get more high resolutions a go pro or DSLR can be used with a C-mount as described by Ye Y, Jiang H, Zhang H et al [2]. It is an expensive option but gives a stable and promising results also as it uses a C-mount and a beam splitter eye piece is not utilized and we can get images while examining patients, now these systems are more refined and are available to us with cost lesser then commercially available systems.

Smartphone photography
Many techniques are been described to capture anterior segment and posterior segment high resolutions images of eye without taking assistance of a slit lamp, these are low cost ideas to convert your smartphone into an imaging device.

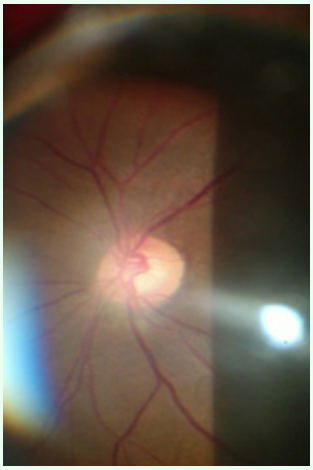

Figure 4: Slit lamp posterior segment photography

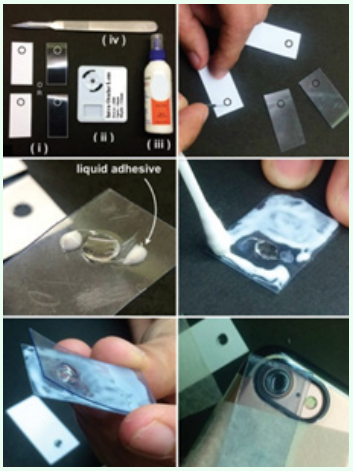

Figure 5: Using PMMA IOL for anterior segment imaging (Courtesy: Dr Prithvi Chandrakanth)

Anterior segment photography with intraocular lens as described by Dr. Prithvi Chandrakanth[3], using old expired PMMA PCIOL, hard plastic sheet and some stationery (figure 5) one can build a small attachment for smartphone and directly use it to capture images of anterior segment and adnexa. Steps can be followed as described in given article.

Smartphone-based Gonio-Imaging as described by Kumar et al[4], Patient lying in horizontal position goniolens to be placed on eye and with other hand keeping smartphone approximately 3inch away (depending on focal length of camera) high resolution images of angle can be obtained. It could be a very handy tool in screening programs and in satellite centre.

A fair quality of retinal images can also be obtained in a full dilated patient by taking a video and keeping flash light on and placing 20D lens 2 cm away from eyes and smartphone 30cm away from rim of condensing lens and later taking out screen shots of the desired frame from video. It has its own learning curve and requires a very cooperative patient (figure 6).

Keeping same principle and optics in mind and refining them Dr. Ashish Sharma[5] has innovated an adaptor MII Retcam which can be easily attached to a smartphone, preferably I Phone and using his own designed software good quality of retinal images can be taken. Many papers have been published showing the use of MII Retcam in retinal imaging, including ROP screening [6] and documentation.

Perimetry

Visual field analysis is an expensive but essential diagnostic tool in glaucoma clinic; it is cumbersome and difficult procedure for patient. Virtual Reality based visual field testing has been developed such as the VirtualEye[7] and PeriScreener[8] which chart visual fields in a pattern similar to the Humphrey field analyzer (figure 7).

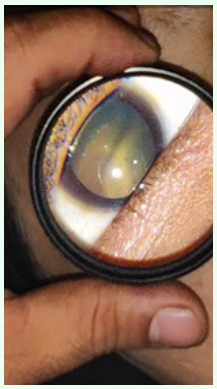

Figure 6: Free hand fundus imaging showing retinal detachment

Figure 7: Virtual Reality based visual field testing (Courtesy: Dr John Davis Akkara)

Conclusion

Innovation is need of the hour as they offer cheap alternative and sometimes with no extra cost. Smartphone have got benefit of a processing device which is faster than traditional computers, is very user friendly and is available in our pockets all the time. These images can be readily edited or marked showing the point of interest, can be shared through various platforms with our colleagues for a second opinion as well and not to forget its use in telemedicine and satellite centers.

Financial interest: Nil

Conflicts of interest: There are no conflicts of interest.

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RECENT UPDATE

Screening Of Retinopathy Of Prematurity In Tertiary Eye Care Centres

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INTRODUCTION

Retinopathy of prematurity (ROP) is a vasoproliferative disease that affects premature infants. The incidence of ROP in India varies from 38% to 51.9%among low birth weight babies.

The World Health Organization's "Vision 2020 programme" has identified ROP as an important cause of blindness in both high- and middle-income countries.

WHO estimates that there are 15 million preterm births a year (born at <37 weeks) and India has the largest number of preterm births in the world.[1]

NATIONAL PROGRAMMES

The exponential increase in services for preterm infants made it urgent to expand ROP programs in facilities where the majority of preterm infants receive care.

Three major programs cover the range of services for prevention of blindness from ROP India. They are Child Health, Ministry of health and family welfare; Rashtriya Bal Swasth karyakram and National programme for Control of Blindness and visual impairment. [NPCB and VI]

NPCB and VI are responsible for strengthening the public eye health system for screening and treatment of ROP. [2]

PATHOGENESIS

During intrauterine life, the foetal retina is in a hypoxic state. Regardless of gestational age at delivery, ROP begins to develop between 32 and 34 weeks after conception and has two phases. During the initial acute phase, the normal vasculogenesis of the retina is disturbed by the relative hyperoxia of the extra uterine environment and is further worsened if the baby requires oxygenation post-delivery. This results in obliteration of vessels and non-vascularisation of areas of the anterior retina. A second chronic phase follows due to the subsequent hypoxia, which in turn stimulates vascular endothelial

Growth factor (VEGF) in some cases leading to arteriovenous shunts and neovascularisation, occasionally leading to visual impairment.

RISK FACTORS

- Low birth weight
- Young gestational age
- High, unregulated oxygen at birth
- Poor postnatal growth
- Intraventricular haemorrhage, respiratory distress syndrome ,sepsis white race, blood transfusion and multiple births.[3]

SCREENING GUIDELINES [4]

- **WHOM TO SCREEN**

1] Birth weight less than 2000 gram
2] Gestational age less than 34 weeks
3] Gestational age between 34 to 36 weeks but with risk factors such as
A] Cardio-respiratory support
B] Prolonged oxygen therapy
C] Respiratory distress syndrome
D] Chronic lung disease
E] Blood transfusion
F] Neonatal sepsis
G] Poor postnatal weight gain

- **WHEN TO SCREEN**

1. Should receive first screening at 4 weeks of birth ,
2. Infants with period of gestation less than 28 weeks or less than 1200g birth weight should be first screened at 2-3 weeks after delivery.

- **DURATION AND FREQUENCY OF SCREENING**

In general, the screening examination will continue at least every two weeks until
1. Vascularisation of the retina reaches normal completion ,or
2. Until ROP regresses, or
3. Until ROP requiring treatment develops.

- **PREPARATION**

Personnel to carry out screening;
1. Preferably, the team should comprise an experienced ophthalmologist, medical office and a nurse especially of the SNCU.
2. Integrate ROP screening and management services with “National programme for control of blindness ‘’[NPCB’]

- **PLACE OF EXAMINATION**

Neonates are best examined in the neonatal unit itself under supervision of attending paediatrician/neonatologist. Place should be warm enough and clean. If babies are being examined at ophthalmologist’s office, there should be arrangement for basic resuscitation equipment.

- **EQUIPMENT CHECKLIST**

1. Indirect ophthalmoscope, preferably wireless one
2. 20, 28 or 30 D lens, as they allow easier viewing of the peripheral retina
3. Paediatric speculum
4. Infant scleral depressor
5. Dilator eye drops a] Tropicamide 0.5% b] phenylephrine 2.5% c] cyclopentolate 0.5%
6. Topical anaesthetic eye drops-proparacaine 0.5%
7. Topical antibiotic eye drops
8. Sterile cotton and gloves
9. ROP documentation sheet
10. Optional: Wide field digital camera.
11. Nestling of infant can significantly reduce the stress during procedure.

- **PROCEDURE FOR THE DIAGNOSTIC EYE EXAMINATION**

1. Baby should be well clothed and wrapped.
2. Pupil should be dilated 30 minutes before examination
3. Informed consent should be obtained from parents.
4. The baby is swaddled and arms restrained to minimise general movements.
5. Topical anaesthetic drop, proparacaine 0.5% instilled.
6. Lid speculum is inserted below lid margins
7. First anterior segment examination to be done with condensing lens focusing on cornea, iris and lens, dilated iris vessels.
8. Retinal examination to be done using 20D /28D lens.
9. Scleral depression to be done with wire vectis or paediatrics depressor.
10. Findings to be recorded according to international classification of ROP [ICROP] guidelines.
11. One drop of antibiotic to be instilled at the completion of examination
12. Baby should be observed for 15 minutes before handling to parents.

FOLLOW UP SCHEDULE

Zone of retinal findings	Stage	Follow up interval
Zone 1	Immature vasculature	1-2 Weeks
	Stage 1 or 2	1 Week or less
	Regressing ROP	1-2 weeks
Zone 2	Immature vasculature	2-3 weeks
	Stage 1	2 weeks
	Stage 2	1-2weeks
	Stage 3	1 week or less
	Regressing ROP	1-2 weeks
Zone 3	Stage 1or 2	2-3 weeks
	Regressing ROP	2-3 weeks

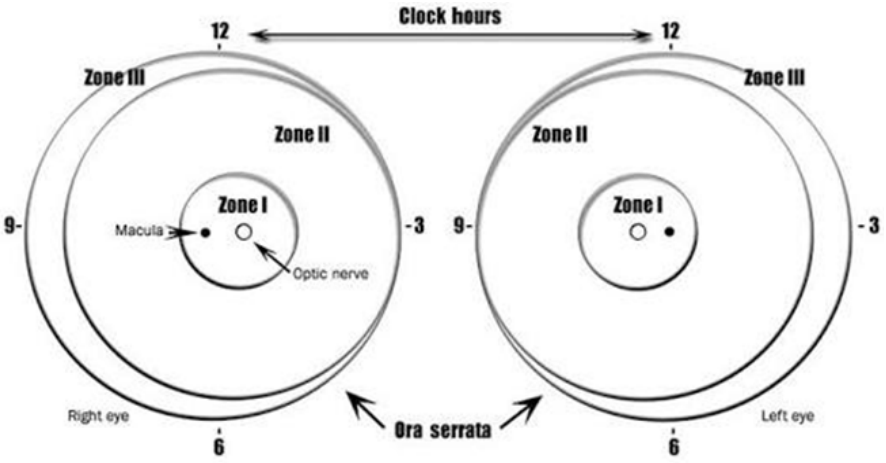
RECENT ADVANCES IN SCREENING METHODOLOGY

The retcam has revolutionised the technique for screening of ROP. It is digital imaging equipment for examining the paediatric fundus .It is a non-stressful way to screen premature babies and is easier to perform when compared to indirect ophthalmoscopy, performed by ophthalmologists. It is easily mobile and helps to access remote areas where sufficient facilities are unavailable. It offers better cardio respiratory stability during examination in children. It also helps to keep a photographic record of the screened patients. [5]

The KIDROP program is a well-structured tele-ROP tool in rural South India. Retinal imaging is performed by trained technicians who grade images and transfer them to experts with specially designed software to generate ROP report. The accredited technician determines on site whether the baby needs follow up/treatment/discharge based on the triage algorithm created by experts. [6]

LOCATION OF THE DISEASE

Three circular zones are defined with optic disc at the centre.



STAGES OF THE DISEASE

Stage 1-Demarcation line
It separates the normal vascularised retina from sharply contrasting avascular white/grey retina.
Stage 2- Demarcation ridge.
Stage 3- Extraretinal fibrovascular proliferation.
There is development of abnormal new blood vessels, and fibrous scar tissue. It is further divided into mild, moderate and severe.
Stage 4- Subtotal retinal detachment;
4A - Sparing macula.
4B - Involving macula.
Stage 5- Complete retinal detachment.
These are funnel shaped and mostly tractional in nature.

PLUS DISEASE

It is characterised by arteriolar tortuosity and venous engorgement of the posterior pole, iris vascular engorgement and vitreous haze.

PRE-PLUS DISEASE

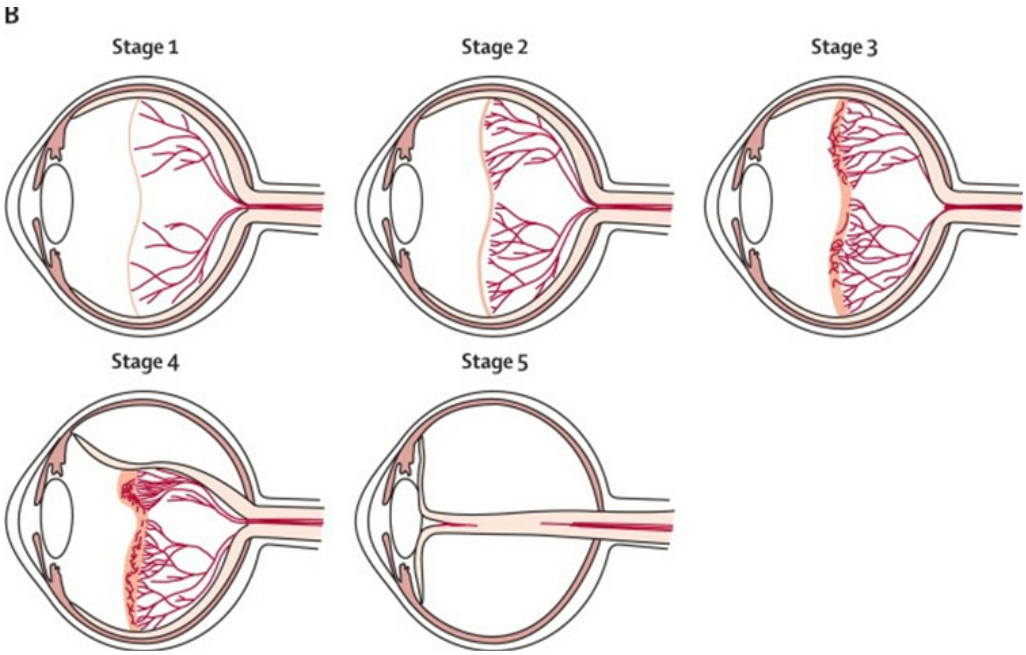
It indicates posterior pole tortuosity and dilatation that are not sufficiently abnormal to reach the criteria of plus disease, but is greater than that regarded as normal.

AGGRESSIVE POSTERIOR ROP

It is characterised by severe dilatation and tortuosity of the vessels which is out of proportion to the peripheral retinopathy. The disease is limited to the posterior pole in zone 1 or posterior zone 2 and usually does not progress through the classic stages 1-3 of ROP.

Shunting occurs between vessels intra-retinally and flat neovascularisation is noted. It extends circumferentially and if left untreated, very rapidly progresses to stage 5 within few days. [7]

The retinal vascular development in utero has two phases. The initial phase, vasculogenesis, is responsible for formation of four major arcade vessels, and the second phase, angiogenesis, completes the rest of the vasculature. Interference in vasculogenesis results in APROP/zone 1 ROP, while the classical staged ROP is correlated with disruption of angiogenesis. Flynn and Chan-Ling have described a hybrid form of ROP where normal vascularisation was present along upper temporal arcade and abnormal vessels simulating APROP were present along lower temporal arcade. They hypothesised that there may be an overlap between vasculogenesis and angiogenesis and both may be disrupted at the same time. This hypothesis may explain the simultaneous presence of abnormal fat new vessels [disrupted vasculogenesis] and ridge tissue [disrupted angiogenesis]. [8]



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CASE REPORTS

A Rare Case of Traumatic Phacocoele with Choroidal Detachment

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INTRODUCTION

Migration of lens into the subconjunctival space is a serious and rare complication of blunt trauma. It can be direct or indirect as described by Fejer et al[1]. Dislocation of lens into the subtenon's space with choroidal detachment is associated with complications such as occult scleral tear and retinal detachment. We report a case of traumatic subtenon's dislocation of crystalline lens with choroidal detachment after blunt trauma and its successful surgical management.

CASE REPORT

A 56 year old female, non -myope came with complaints of sudden onset pain and defective vision in the left eye following blunt trauma sustained due to injury by stick 7 days back. Patient did not give any history of prior ocular surgery or systemic condition such as collagen disorder predisposing to a weak sclera. Her best-corrected visual acuity was 6/9 in the right eye (OD) and perception of light in the left eye (OS). Intraocular pressure was 12 mmHg in OD and 10 mmHg in OS, respectively. On examination, her right eye had Grade 2 nuclear sclerosis with normal posterior segment. The anterior segment of left eye showed a well-defined oval yellowish subconjunctival mass of size 1.5 cm × 2 cm extending from 11'o to 9'o clock position (Figure1A) seen in the supero-nasal quadrant, suggestive of phacocoele with localised subconjunctival haemorrhage not extending posteriorly. There was corneal stromal edema and hyphema in anterior chamber. Pupil was updrawn with aphakia. Fundus view was hazy with red glow. Ultrasound of the left eye was suggestive of annular choroidal detachment however the retina was attached in all quadrants (Figure1B).

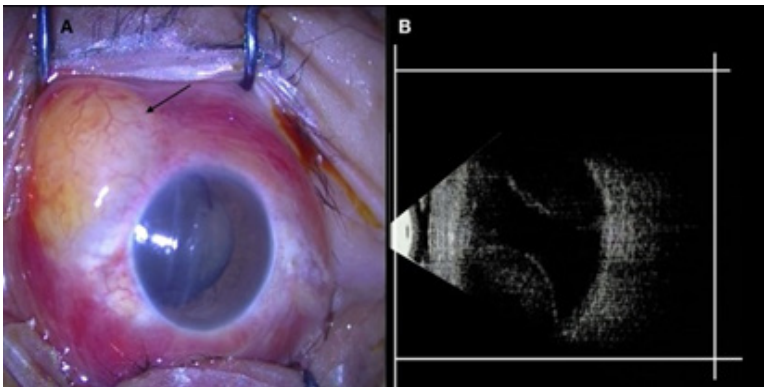

Figure 1: (A) Slit Lamp photograph showing superionasal migration of lens.
(B) Ultrasound showing choroidal detachment.

As part of operative procedure she underwent wound exploration, lens extraction, wound toileting and repair of scleral defect in the left eye under general anaesthesia. Peritomy was done anticlockwise from 12'o to 6'o clock position to expose sub tenons space. Spontaneously extruded cataractous lens was found lodged in the subtenon space of the Supero-nasal quadrant of the left eye. Circumferential scleral defect with ragged margins was noticed from 11'o to 6'o clock position, 2-3 mm away from limbus and parallel to it (Figure 2A & 2B).

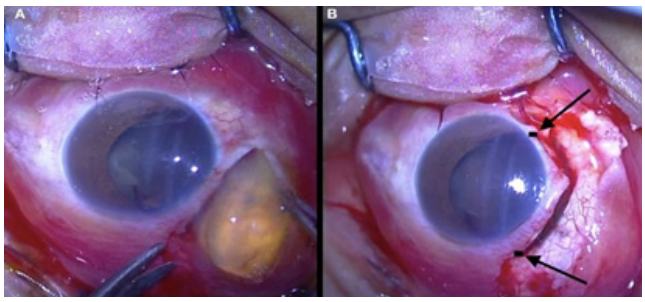

Figure 2: (A) Intraoperative photo showing phacocoele. (B) Intraoperative photo showing extent of scleral rupture

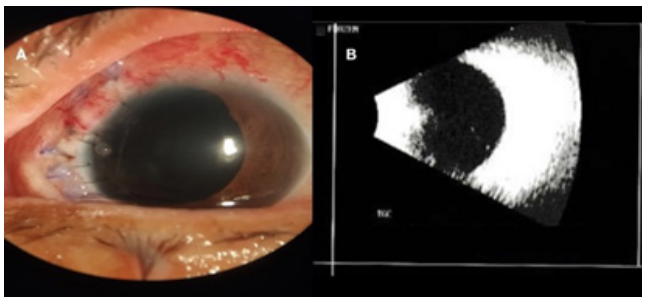

Figure 3: (A) Postoperative slit lamp photo. (B) Post-operative ultra sound showing resolved choroidal detachment.

Abscission of uveal tissue prolapsing through the defect was done followed by vitrectomy at the wound margin. Suturing of the scleral defect was done with 6 0 vicryl and 10-0 monofilament nylon. Limited anterior vitrectomy was done and the patient was left aphakic. Postoperatively, her vision improved to 6/24 with +11 dioptre lens. Choroidal detachment subsequently resolved on its own. She was explained about the need for placement of secondary scleral fixated IOL (Figure 3A& 3B).

DISCUSSION

Blunt trauma of sufficient force can result in indirect scleral rupture resulting in dislocation of lens into subconjunctival space. The rupture is in the equatorial direction for a length corresponding to the size of the lens, so that the entire lens is able to slip out through the gap pro¬duced. Proposed mechanism for this is sclera undergoes greatest stretching at the point opposite to the trauma and ruptures at the summit because of this stretch[1].Other mechanism proposed by Santos Bueso et al[2] concluded in blunt trauma, energy is transmitted from the site of impact to opposite orbital wall which leads to sclera rupture.

The incidence of phacocoele is very infrequent compared to other lens dislocations. McDonald and Purnell in 1951,[3] Bhupally et al. in 2015[4] reported that the incidence of phacocoele was only 13% and 1.13% in their respective study. More so phacococele associated with choroidal detachment is uncommon. Supero-nasal quadrant is the most common site for phacocoele which was also seen in our patient similar findings were also found by Fejér in 1928[1]. He also suggested phacococele is even rarer in paediatric age group due to scleral elasticity. Bhattacharjee et al. in 2007[5] reviewed eight cases of traumatic phacocoele which had favourable visual outcomes and outcome was inversely related to length of scleral rupture not in agreement with our patient which had almost 6 clock hour tear. Goel in 2018[6] reported a case of phacocoele following peribulbar anaesthesia. They hypothesised increase in IOP following inadvertent administration of local anaesthesia intraocularly to be the cause of scleral rupture and development of phacocoele. There have not been any case reports of phacocoele with choroidal detachment which was seen in our patient. The possible mechanism of choroidal detachment is trauma leading to scleral rupture causing sudden hypotony causing transudation of fluid from the ciliary body with loss of fluid from vitreous.[7]

CONCLUSION

Blunt trauma can result in indirect scleral rupture resulting in dislocation of lens into subconjunctival space. Phacocoele should therefore be suspected in a case of occult scleral rupture with localized hematoma and aphakia. Visual outcome depends on timely diagnosis, proper management, and associated ocular complications.

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Surgical Management of Suprachoroidal Hemorrhage Secondary to Penetrating Trauma

Dr Ashish Athale[1], Dr Prateek Gujar[2]

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2. Cornea Consultant, Sudarshan Netralaya, Bhopal

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INTRODUCTION

The suprachoroidal space is normally virtual because the choroid is in close apposition to the sclera. As fluid accumulates, this space becomes real, and the choroid is displaced from its normal position. Fluid accumulation, either serum like or blood, also can occur within the choroid, which is a spongy tissue. Suprachoroidal haemorrhage is defined as the accumulation of blood within the potential space between the choroid and sclera, with the source of the blood being the long or short posterior ciliary artery. This can occur spontaneously (rare), as a consequence of ocular trauma, during eye surgery, or after eye surgery.[1-3]

The extent of SCH ranges from localized, self-limiting haemorrhages[4,5] to expulsion of intraocular contents. It is important to recognize this complication early and to manage it expediently. Key considerations include early detection, optimized medical management with close follow-up, and appropriately timed minimally invasive surgery, in some cases.[4,6] Successful management increases the chance of visual recovery.[4] Limited, non-appositional SCHs may not require surgical intervention.[4] Rather, they may be observed, as spontaneous resolution may occur over a period of weeks to months. Indications for surgical drainage in¬clude retinal apposition “kissing choroidals”, uncontrolled IOP, flat anterior chamber, and rhegmatogenous retinal detachment.[1,4,7,8] There is no consensus on the best timing for drainage. In most cases, drainage is performed when serial B-scan ultrasonography shows signs of clot liquefaction (usually in 7-14 days).[5,7] We present a case of traumatic suprachoroidal haemorrhage, who after surgical intervention, showed significant visual improvement.

CASE REPORT

37 year old male patient, presented to our OPD on 25th July, 2019 with complaints of diminution of vision in right eye since 3 days. He had history of penetrating trauma with grinder paper 3 days back. He had consulted local general practitioner, who had advised antibiotic drops and sent him home. The patient sought opinion from Ophthalmologist after 24 hours. He was diagnosed as having corneo-scleral tear, for which he was operated 2 days back. He had no history of any systemic illness.

On examination, BCVA in right eye was perception of light, with faulty projection and in left eye was 6/9. Anterior segment showed sealed corneal-scleral tear, formed anterior chamber and membrane of clotted blood in pupillary area. Rests of the detail were not visible. Fundus in the right eye could not be visualized. Left eye was normal except for lattice degeneration inferiorly. B-scan showed haemorrhagic choroidal detachment in right eye (kissing choroidals), with few blood clots (fig 1).

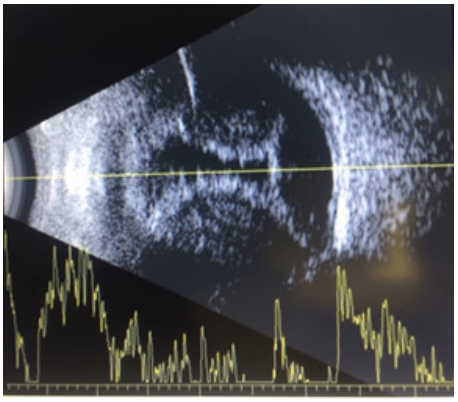

Figure 1 - B-scan showed haemorrhagic choroidal detachment in right eye with kissing choroidals along with few blood clots.

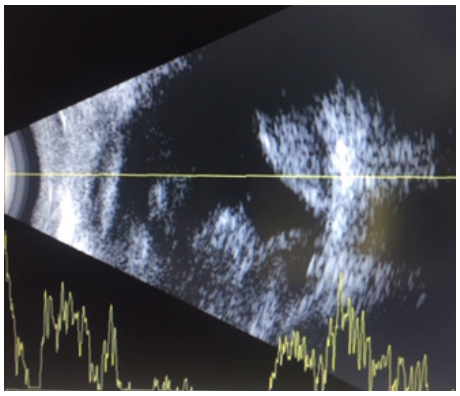

Figure 2 – After 3 weeks, B-scan showed reduced height of choroidal detachment in infero-temporal quadrant with liquefaction of blood clots in suprachoroidal space.

He was put on oral steroids 1mg/kg and serratiopeptidase and advised to continue local steroids and cycloplegics. On follow up after 5 days, patient was status quo. He was advised to come after 3 days. But he followed up after 2 weeks. Repeat B-scan showed reduced height of choroidal detachment in infero-temporal quadrant with liquefaction of blood clots in suprachoroidal space (figure 2). He was advised to undergo surgery under guarded visual prognosis. After one month of corneoscleral repair, patient underwent lensectomy, trans-scleral drainage of blood along with belt buckle, pars plana vitrectomy, endolaser and silicone oil tamponade in right eye. On table he was found to have total rhegmatogenous RD which was settled.

Post-operatively, in right eye, anterior segment was quiet. Retina was attached with silicone oil in vitreous cavity and good buckle effect in right eye. There was residual supra choroidal haemorrhage inferiorly, not involving macula. Intra-ocular pressure was normal digitally. BCVA improved to FC ¼ metres at 6 weeks and 1/60 at 10 months. After 10 months, patient was operated for silicone oil removal with SFIOL implantation in right eye. Post-operatively, he did well with attached retina and SFIOL in place (figure 3). BCVA in right eye improved to 6/36 at 6 weeks.

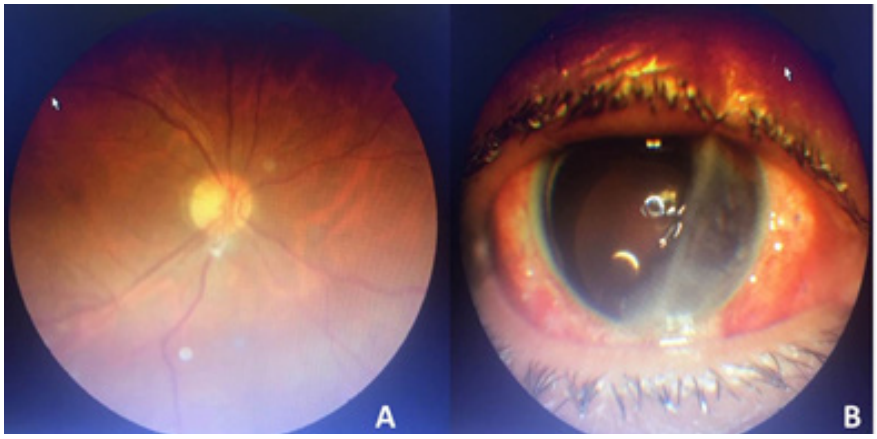

Figure 3 - Color fundus photograph shows well settled retina (A) with SFIOL in situ (B).

CONCLUSION

Haemorrhagic choroidal detachment is a devastating complication, whether after intra-ocular surgery or trauma. It can pose a management dilemma. Numerous approaches exist for the management of suprachoroidal haemorrhage. Although medical management is favoured when possible, a surgical approach that entails minimal manipulation and achieves maximal drainage is ideal for restoring ocular anatomy and facilitating visual recovery. With careful follow-up and management, it is possible to achieve fruitful vision, at least in some cases.

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BEYOND OPHTHALMOLOGY

"A Blessing in Disguise" - A True Story

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As I was wading away in my thoughts on a lazy COVID holiday, I happen to stumble upon a 'blessing-in-disguise' type of incident in the memory lane; one that took place during my Glaucoma fellowship at one of the most prestigious and toughest institute. Toughest in the fellowship was the early morning class, doors for which close at 7 AM sharp. If late, no fellow was allowed to enter the class; even the Director and the Chairman of the Institute had to abide by those rules.

What if you are a presenter in that class? The preparation for one presentation goes for 1-2 months and the aura for it starts building up way before the preparation starts.

I finished my scheduled presentation on Thursday and it went well and was in good mood. The following Friday evening all fellows had an invitation for a marriage party by one of our colleague. The party carried into the late evening and we thoroughly enjoyed the party after a long hectic week. On Saturday morning, probably the alarm didn't ring or we forgot to set it. Me and my roommate couldn't get up and go for the class. We thought occasional missing of class is OK.

But it was not OK!! As soon as I stepped in OPD, I heard the news that only few fellows attended the class today, so the faculty asked to give written explanation for the absence. Additionally, as a repercussion, the absent fellows were assigned all the department presentations for the whole month.

Guess what?? I was the only Glaucoma fellow who missed the class and there were 4 presentations of Glaucoma due that month. I had a sinking feeling as I had a steep and almost impossible mountain to climb ahead with no other option on the table as the rules and punishments there were very strict. The stories of some fellows who had to leave the institute without completing the degree reverberated across my puzzled mind. In the moment, I started imagining that my name will be associated with yet another imperfect failed story echoing through the perfect corridors of this great institute. I was drowning.

Lot of suggestions came from my fellow students and none was worth pursuing to get me out of this quagmire/ situation. It was deemed impossible to deliver 4 presentations in a month. My other 4 colleagues who were free now, as I was carrying their burden for the month were giving me free advices. I was drowning.

In search for finding the solution, I thought about the toughest time that I endured through my professional journey when I had to put in my best efforts. I recalled it was the times when had done studies for PG Examination, 20 subjects and all on tips and could make it successfully. Suddenly this thought changed everything. Why worry about 3-4 topics of such a small subject ophthalmology? Before this thought everything was impossible and after that thought nothing was impossible.

Thereafter, I didn't take any suggestion from anybody. I was after my faculty to finalise my cases and journals, and they too expressed lot of empathy and sympathy with me as they knew task ahead was tough for me.

With determination I embarked on this journey. My first case was after 3 days followed by Journal clubs, where statistics was new and tough for me. I was burning the midnight lamp everyday as I prepared for cases and presentations one after another. In my last case after I said the case is now open for discussion, the Director (also Glaucoma HOD) took the microphone and said

"Thanks Arpita letting us know even the finest details of this Journal topic, you cleared all my doubts I had, while I read this just before entering the class."

(He used to read every topic before class and was very good at stats. It was very difficult to impress him!)

I was surprised by his response as I could not believe the encouraging words I was hearing. His next sentence "On behalf of all I congratulate you the way you presented all your presentations and took the challenge. I am highly impressed". The class ended. No questions were asked after that. All started dispersing showing me thumbs up.

Thereafter, I could notice a kind of respect by my fellow colleagues, seniors, juniors and faculty members. Even the faculty who set me up to this task congratulated me.

The appreciation went on in fellow appraisal in presence of all glaucoma departments. Later we also published Letter to Editor from the Journals.

It just requires a determined thought to change impossible to possible!! If you keep at it, work hard and ready to dive full length, you can achieve the impossible!! Keep walking.

Thanked Almighty for his crucial presence.

BEYOND OPHTHALMOLOGY

From The Pen Of Ophthalmologist

Dr Khalid Khan

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"मन जब बना ही लिया"

हमने चलने का मन जब बना ही लिया,
धूप हमको लगी चांदनी की तरह.

हमने लडने का मन जब बना ही लिया,
जंग हमको लगी आशिकी की तरह.

हमने जीने का मन जब बना ही लिया,
मोत हमसे मिली जिंदगी की तरह.

हमने हंसने का मन जब बना ही लिया,
चोट पर हंस दिए गुदगुदी की तरह.

"सुविधा - दुविधा"

प्रेम में झंझट है, विवाह में सुविधा है
झंझट में रस है, यही तो दुविधा है

संघर्ष में झंझट है, संधि में सुविधा है
झंझट में रस है, यही तो दुविधा है

लहरो में झंझट है ,किनारे पर सुविधा है
झंझट में रस है, यही तो दुविधा है

गृहस्ती में झंझट है , संयास में सुविधा है
झंझट में रस है ,यही तो दुविधा है

जीवन में झंझट है , मृत्यु मे सुविधा है
झंझट में रस है, यही तो दुविधा है

"अभिनंदन"

खुशियो का तो सब करते हैं,
तू दुख का भी कर अभिनंदन,
मित्रो का तो सब करते हैं,
तू शत्रु का भी कर अभिनंदन.

बरखा का तो सब करते हैं,
तू पतझड़ का भी कर अभिनंदन,
गुलशन का तो सब करते हैं,
तू सहरा का भी कर अभिनंदन.

फूलो का तो सब करते हैं,
तू काँटो का भी कर अभिनंदन,
साहिल का तो सब करते हैं,
तू मझधारो का भी कर अभिनंदन.

भोर का तो सब करते हैं,
तू संध्या का भी कर अभिनंदन,
जीवन का तो सब करते हैं,
तू मृत्यु का भी कर अभिनंदन.

खालिद खान "गुस्ताख"

BDOS SCIENTIFIC ACTIVITIES

8th September 2019

NEW BDOS TEAM ELECTED

29th September 2019

FIRST EXECUTIVE BODY MEETING

2nd November 2019

SATURDAY CLINICAL MEETING AT GMC BHOPAL

24th November 2019

CME ON RETINA AND ROP AT HOTEL SAYAJI

17th - 24th November 2019

ROP CAMP BY BDOS MEMBERS

12th January 2020

SESSION 3 – ALL WE SHOULD KNOW ABOUT ENDOPHTHALMITIS (12.15 pm – 2.00 pm)

Session officials- Dr Salil kumar, Dr Prashant Bawankule, Dr A K Dubey, Dr Gajendra Chawla, Dr Mansi krishnani, Dr Alok Sen

PRESENTER NAME	TOPIC	TIME
Dr A K Dubey	Acute Post operative bacterial endophthalmitis - Nightmare for all	7 min
Dr Prashant Bawankule	Solving Dilemma - Post op Endophthalmitis VS TASS	7 min
Dr Shrinivas Joshi	Post Intravitreal injection Endophthalmitis Pearls to prevent & manage	7 min
Dr Alok Sen	Cluster endophthalmitis - how to investigate and manage	7 min
Dr Rama Kumar	Endogenous endophthalmitis - what all we should remember	7 min
Dr Vivita Ramnani	Bleb related endophthalmitis	7 min
Dr Rahul Shukla	Surgical wound infections in SICS and PHACO - How to prevent and treat	7 min
Dr Shardani Vyas	Infections following Corneal Surgeries	7 min
Dr Vijay Ramnani	Microbiological aspect of ocular infections	7 min
Dr Prashant Bawankule	Preventive measures for postoperative endophthalmitis - march to zero	7 min
Dr Shrinivas Joshi	Art and Science of managing postoperative endophthalmitis	7 min
	DISCUSSIONS	25 min

LUNCH – 2.00 pm- 2.30 pm

WET LAB- 2.30 pm – 4.00 pm

Indirect Ophthalmoscopy & Charting - Dr Ganesh pillay, Dr Kavita Gupta

B-Scan – Dr Vineet Gour, Dr Chahveer Singh Bindra

Intravitral injection technique - Dr Rahul Agarwal, Dr Anusha Ajwani

REGISTRATION IS COMPLIMENTARY BUT MANDATORY – Contact: Dr Chahveer Bindra (9826004896)

SYMPOSIUM SPONSORED BY- NOVARTIS HEALTHCARE PVT LTD

AIOS-ARC COMBAT EYE INFECTIONS SYMPOSIUM

12th January, 2020 (Time- 8.30 am - 4.00 pm)

Organised By

Madhya Pradesh State Ophthalmic Society

& Hosted By

Bhopal Divisional Ophthalmic Society

Venue: Hotel Sayaji, Bhopal (M.P)

DR PARTHA BISWAS – CHAIRMAN AIOS-ARC

DR AMIT PORWAL – AIOS-ARC MEMBER CENTRAL ZONE

DR SALIL KUMAR – CHAIRMAN ORGANISING COMMITTEE

DR LALIT SHRIVASTAVA – ORGANISING PRESIDENT

DR VINITA RAMNANI – ORGANISING SECRETARY

COORDINATORS FOR SYMPOSIUM

Dr Chahveer singh Bindra

Dr Hemlata Yadav

Dr Riddhima Deshpande

Dr Fazil Khurrum

Program Details - AIOS-ARC Comat Eye Infections

12th January 2020- Hotel Sayaji, Bhopal

Registration & BreakFast – 8.30 am- 9.00 am

Inauguration- 9.00 am - 9.30 am

Scientific Session- 9.30 Am – 2.00 pm

Lunch- 2.00 pm – 2.30 pm

Wet Lab- 2.30 pm – 4.00 pm

AIOS -ARC COMBAT EYE INFECTIONS SYMPOSIUM

12th January 2020

MPSOS OFFICE BEARERS

President – Dr Hitesh Agarwal

President Elect – Dr Sashi Jain

Hon. Secretary – Dr Gajendra Chawla

Vice President – Dr Lalit Shrivastava

Joint Secretary – Dr Meeta Joshi

Treasurer – Dr Saroj Gupta

Chairperson Scientific Committee – Dr V K Nichlani

Member AIOS Managing Committee – Dr Salil Kumar

Member AIOS Managing Committee – Dr A K Dubey

BDOS OFFICE BEARERS

President – Dr Lalit Shrivastava

Hon. Secretary – Dr Vinita Ramnani

Clinical Secretary – Dr Chahveer Singh Bindra

Vice President – Dr Pramod Chandra

Vice President – Dr Bhavana Sharma

Joint Secretary – Dr Vivek Som

Treasurer – Dr Rahul Agarwal

SESSION 1 –TIPS TO DIAGNOSE AND MANAGE EYE INFECTIONS (9.30 am – 11.15 am)

Session officials – Dr Parikshit Gogate, Dr P S Bindra, Dr Pramod Chandra, Dr R K Gupta, Dr Bhavana Sharma, Dr Amit Porwal

PRESENTER NAME	TOPIC	TIME
Dr Aditi Dubey	Pearls to diagnose and manage Conjunctivitis	7 min
Dr Preeti Bindra	Approach to Bacterial & Parasitic Keratitis	7 min
Dr Prateek Gujar	Advancements in treatment of Fungal Corneal Ulcer	7 min
Dr Pankaj Choudhary	Diagnosis and Management of Viral Keratitis	7 min
Dr Bhavana Sharma	PYTHIUM KERATITIS- What all should you know	7 min
Dr Salil Kumar	How to deal with Non Healing Corneal Ulcer	7 min
Dr Shreya Thatte	Management pearls for Perforated Corneal Ulcer	7 min
Dr Shweta Walia	All that matters in Orbital Cellulitis !!!	7 min
Dr Prashant Bhardiya	How to proceed for Cataract Surgery in Preexisting Corneal Conditions	7 min
Dr Purendra Bhasin	Pre and Post operative considerations in Cataract surgery in Uveitis	7 min
Dr Praveen Khare	DOS and DONT'S of Cataract Surgery in Oculoplastic Conditions	7 min
	DISCUSSION	25 min

SESSION 2 - KNOW THE PROTOCOLS (Time- 11.15 am – 12.15 pm)

Session officials- Dr Shrinivas Joshi, Dr Pradeep Vyas, Dr A C Agrawal, Dr M K Khurrum, Dr V K Nichlani, Dr Vivek Som

PRESENTER NAME	TOPIC
Dr Parikshit Gogate	Protocols for preoperative evaluation before Cataract surgeries
Dr Amit Porwal	Postoperative protocols after Cataract surgeries
Dr Shubhra Mehta	Guidelines to perform safe Cataract surgeries in camps
Dr Meeta Joshi	NASH guidelines for HIC
Dr Parikshit Gogate	Red eye post Cataract surgery - what next ?
Dr Kavita Kumar	OT - Design and Sterilization
Dr V K Nichlani	Sterilization of ophthalmic instruments
	DISCUSSION

AIOS -ARC COMBAT EYE INFECTIONS SYMPOSIUM

12th January 2020

AIOS -ARC COMBAT EYE INFECTIONS SYMPOSIUM

30th January 2020 - SECOND EXECUTIVE BODY MEETING

8th March 2020

GLAUCOMA AWARENESS RALLY

12th March 2020

CASE BASED DISCUSSION

8th - 14th March 2020

GLAUCOMA WEEK ACTIVITIES

8th March 2020

INTERNATIONAL WOMENS DAY

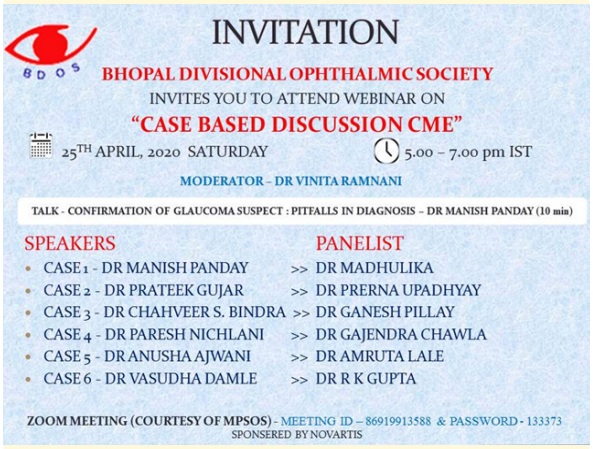
8th March 2020

INTERNATIONAL WOMENS DAY CELEBRATION

14th March 2020

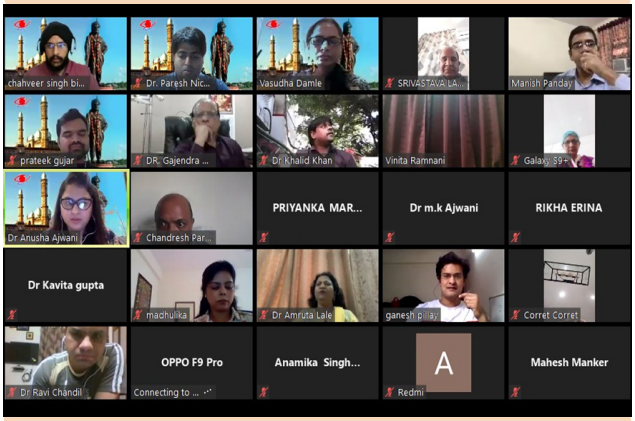
DIGITAL CASE BASED DISCUSSION CME

24th April 2020



DIGITAL CASE BASED DISCUSSION CME

24th April 2020



DIGITAL CASE BASED DISCUSSION CME

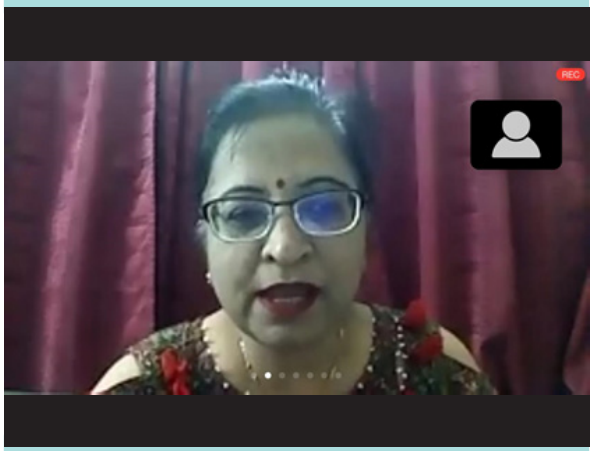
2nd May 2020

DIGITAL CASE BASED DISCUSSION

22nd May 2020

DIGITAL CASE BASED DISCUSSION CME

3rd August 2020



3RD VIRTUAL EXECUTIVE BODY MEET

26th July 2020

GLAUCOMA CASE BASED DISCUSSION

23rd August 2020

INVITATION

BHOPAL DIVISIONAL OPHTHALMIC SOCIETY

INVITES YOU TO ATTEND WEBINAR ON

"DEBATES ON CORNEA, REFRACTIVE AND CATARACT"

23rd AUGUST, 2020 SUNDAY 11.00 am – 1.00 pm IST

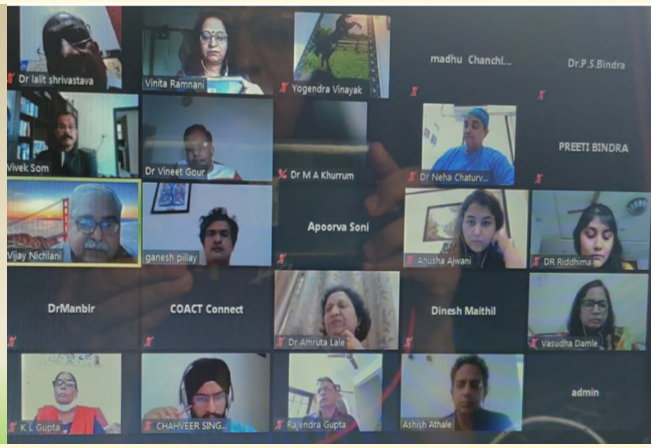
MODERATOR – DR VINITA RAMNANI

EXPERT PANELIST - DR SWARNA BISARIA GUPTA, DR PRAMOD CHANDRA, DR VIVEK SOM

1. Coexisting pterygium with cataract – Combined surgery (Dr Madhu Chanchlani) VS Two stage surgery (Dr Vasudha Damle) – Comment (Dr PS Bindra)
2. Preferred technique of cataract surgery in rock hard black cataract – SICS (Dr Apoorva Soni) VS Phaco (Dr Manbir Singh) – Comment (Dr MA Khurrum)
3. Preferred keratorefractive procedure – SMILE (Dr Ganesh Pillay) VS LASIK (Dr Anusha Ajwani) – Comment (Dr Amruta Lale)
4. Managing preexisting astigmatism in cataract surgery – Toric IOL (Dr VK Nichlani) VS LRI (Dr. Neha Chaturvedi) – Comment (Dr Prerna Upadhayay)
5. Phakic IOL – For all grades of refractive error (Dr Prateek Gujar) VS Only for extreme refractive error (Dr Preeti Bindra) – Comment (Dr RK Gupta)
6. Secondary IOL implantation – Scleral fixated IOL (Dr Riddhima Deshpande) VS Posterior iris claw lens (Dr Ashish Athale) – Comment (Dr Vineet Gaur)

4 min presentation followed by 2 min comment

SPONSERED BY AJANTA ANVAXX



DEBATES ON CATARACT & CORNEA

27th September 2020

वेबिनार

बंसल अस्पताल की नेत्र रोग विशेषज्ञ एवं बीडीओएस सचिव डॉ. विनीता रामनानी ने कहा

सकारात्मक बदलाव जो कोरोना संकट की वजह से देखने को मिला

कोरोना वायरस के चलते कई बड़े संकट हमारे सामने आए हैं लेकिन वर्तमान समस्यों के चलते भोपाल डिवीजनल ओफ्थेल्मीक सोसाइटी ने अपने सदस्यो के लिए होने वाली हर महीने की शैक्षणिक एवं अनुसंधान सम्मेलन को वेबिनार के जरिये चालू रखा है, ऐसा अध्यक्ष डॉ ललित श्रीवास्तव एवं सचिव डॉ विनीता रामनानी ने चर्चा में बताया

स्टार समाचार | भोपाल

डिवीजनल ओफ्थेल्मीक सोसाइटी (बीडीओएस) भोपाल के आई डॉक्टर्स की एक संगति है जिसमें सारे आई डॉक्टर्स मिलकर नित नई तकनीक एवं अनुसंधान की चर्चा करते हैं बंसल अस्पताल की नेत्र रोग विशेषज्ञ एवं बीडीओएस की सचिव डॉ विनीता रामनानी ने बताया कोरोना महामारी के चलते पूरे विश्व में वैज्ञानिक गतिविधियाँ डिजिटल (वेबिनार) होने से बहुत फायदा हुआ है। इन वेबिनार के जरिये न केवल भारत के वरन अंतर्राष्ट्रीय स्तर के वक्ताओं को भी घर बेठे एक आरामदेह माहोल में बिना कहीं जाए सुनने के साथ साथ ज्ञान एवं विचारों को एक दूसरे के साथ साझा करने का एक सुनहरा मोका है कोरोना महामारी की इतनी सारी नकारात्मक खबरों एवं परेशानियों के बीच अपनी लगन, मेहनत एवं निष्ठा के साथ एक सकारात्मक पहलू रखते हुए बीडीओएस® की टीम ने प्रतिकूल परिस्थितियों को अनुकूल परिस्थितियों में बदल अपने सदस्यों में उत्साह एवं नजदीकिया बढ़ा दी हैं।

बीडीओएस क्लिनिकल सेक्रेटरी डॉ चाहवीर सिंह बिंद्रा के अनुसार इसी कड़ी में 27 सितंबर रविवार बीडीओएस के वेबिनार में जी एम सी भोपाल के पास हुए अमेरिका के वरिष्ठ मशहूर रेटिना एक्स्पर्ट डॉ हरींदरजीत सिंह एवं लंदन की जानी मानी एम जी एम इंदौर से पढ़ी आई स्पेशलिस्ट डॉ अर्पिता जैन के व्याख्यान के साथ भोपाल शहर के जाने माने नेत्र रोग विशेषज्ञ भाग लेंगे।

सरकार द्वारा दिए निर्देशों का पालन करें : श्रीवास्तव

कोरोना महामारी के चलते बीडीओएस के अध्क्ष डॉ ललित श्रीवास्तव ने लोगों से आहवान किया है कि वो सरकार द्वारा दिये निर्देशों का पालन करें, सोशल डिस्टेन्सिंग का पालन करें, मास्क लगाएं, हाथ, मुँह एवं घर की स्वछ्ता बनाये रखें एवं बहुत आवश्यक होने पर ही घरो से बाहर जाए। बी.डी॰ओ॰एस॰ सचिव डॉ विनीता रामनानी का कहना है कि अपने स्वास्थ्य विशेषकर आँखों का खयाल रखें कोरोना महामारी के चलते आँखों की बीमारियाँ विशेषत: ग्लौकोमा (कांचबिन्द), डायबैटिक रेटिनोपेथी एवं आँखों की किसी भी प्रकार की तकलीफ जिसका पहिले से इलाज चल रहा हो तो नजरअंदाज ना करें

आंखों की तकलीफ के लिए तुरंत परीक्षण करवाएं

समय रहते आँखों की जाँच करवाएं, डॉक्टर द्वारा दशार्ये गए निदेशों का पालन करें एवं किसी भी तरह की आँखों की तकलीफ के लिए तुरंत नेत्र रोग विशेषज्ञ से परीक्षण करवाएँ। डॉ विनीता रामनानी ने बताया आजकल मास्क लगाने की वजह से वश्मे पर भाप (फॉग) जमा होने से काफी दिक्कतें आ रही है। वश्मे के काँच को धुंध से बचाने के लिए मास्क नाक पर अच्छी तरह से कसा हुआ होना चाहिए अगर विलपयुक्त मास्क हो तो उसे सही तरह से नाक पर दबाकर मास्क का इस्तेमाल करें अगर फिर भी भाप जमा हो रही हो तो मास्क के अन्दर ऊपर वाले हिस्से में टिशू पेपर की पट्टी इस्तेमाल करें मास्क नाक पर ढीला होने की वजह से मास्क के ऊपरी हिस्से से छूटने वाली गर्म सांस से आँखों के आँसू जल्दी विलुप्त होने के कारण आँखों में बहुत रुखापन आ रहा है – जिसे मास्क असोसिएटेड ड्राई आई कहते हैं रूखेपन से बचने के लिए यह सुनिश्चित करें कि मास्क नाक पर सही तरह से फिट हो एवं जरूरत पड़ने पर लुब्रिकेटिंग आई ड्राप्स का इस्तेमाल करें।

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