TOS TODAY

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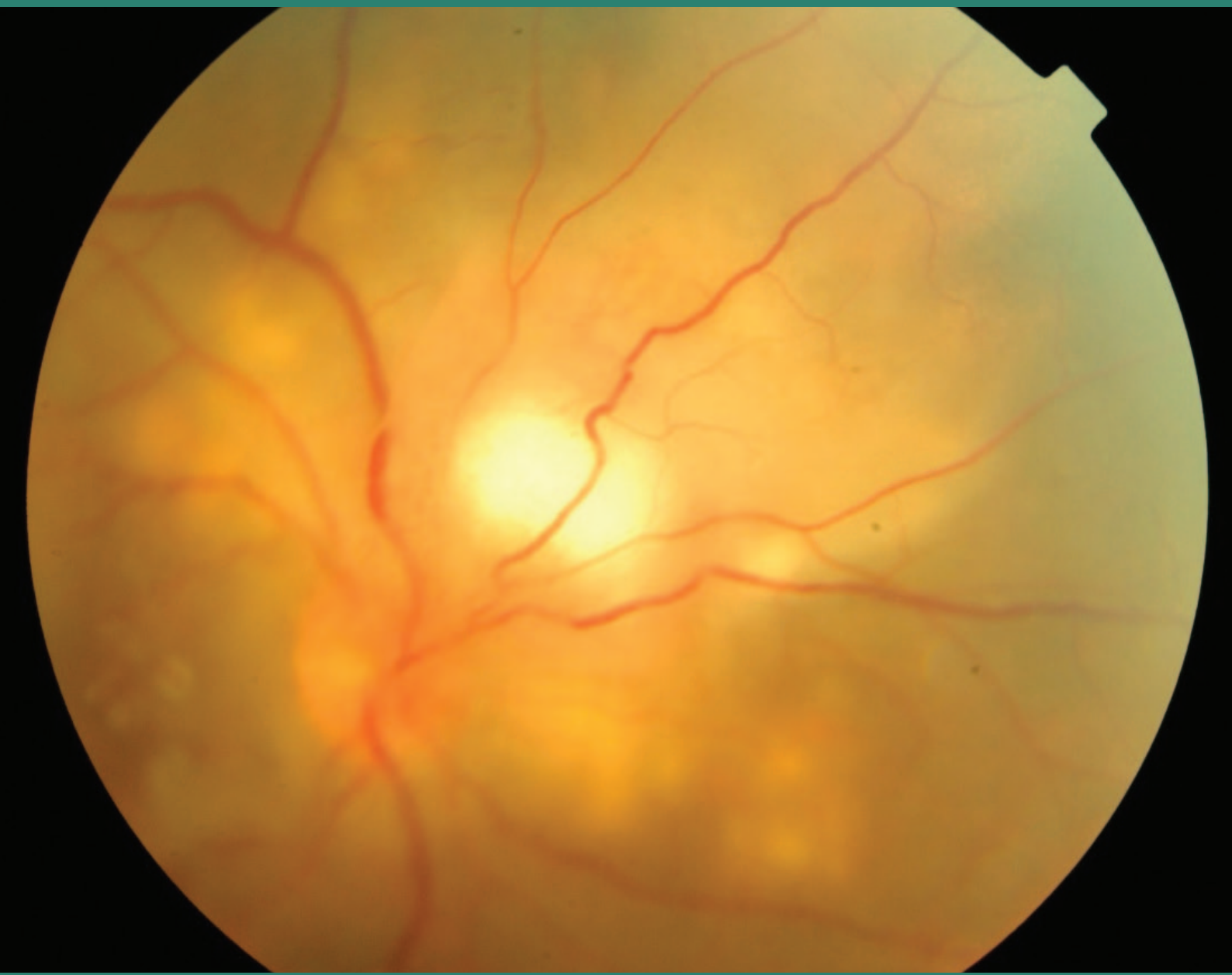

Choroidal Blastomycosis in a 33 year old man returning from endemic South America

www.tsos.co.in

WOS Meeting

Telangana Chapter organized at
Hyderabad under the aegis of HOA, April 30, 2017

From The Editor's Desk

Dear Colleagues & Friends,

Best wishes of the season!!

This issue of the Journal carries Guest articles from International leaders like Prof Mahipal S Sachdev, Prof Atul Kumar, Prof Padmamalini Mahendradas and Prof Anand Rajendran. There is also an amazingly well documented case of supposed conjunctival lithiasis from Kaktiya Medical College… the video demonstrating the extent of cleverness with which a child placed stones in her conjunctival sac and how these were retrieved is available on the TOS website www.tsos.co.in

We would like to congratulate the Local Organizing Committee for the stupendous success of the last Annual State Society Conference at Warangal in July 2017. Special gratitude is due to the Organizing Secretary Dr Bharath Kumar, the Chairman Scientific Committee Dr Pravin Krishna, and Seniors in the EC, Dr NS Reddy, Dr Manoj Mathur, Dr Nand Kishore, Dr Madhukar Reddy and Dr A Ravindra whose vigilance and guidance was invaluable.

With Intravitreal Avastin treatment having come under the scanner in our country in 2015-2016 due to multiple cluster infection events, reliance on the FDA approved drugs Ranibizumab (Lucentis) and Aflibercept (Eylea) as the standard of care agents has increased. This has significantly increased the financial burden of treatment for most middle and lower middle economic category families.

Novartis has responded by slashing prices of its Ranibizumab (as Accentrix) and Verteporfin (Visudyne for PDT) significantly for developing countries like ours.... and with patient support packages offering complimentary vials after purchase of a certain number of vials, the cost reduction been to the tune of 50-80% so that the same drug now costs in India a small fraction of what it costs in the developed countries…..however, keeping in mind that many patients need repetitive treatment with anti-VEGF agents every 4-12 weeks there is an urgent need for further cost reduction.

Indian biosimilars have risen to the occasion and agents like Razumab from Intas and Bevacizumab (Zybev) from Cadilla-Zydus have entered the market to bring down costs by a further 50%.

India is the first country in the world where Anti-VEGF biosimilars for Intravitreal Injections have been marketed. In other countries biosimilars can enter circulation only after the patent of the innovator product expires variably in 2020-2022.

The next Annual State Society Conference is in July 2018 at Sarojini Devi Eye Hospital, Hyderabad. Details will be notified soon.

We do hope you enjoy this journal.

Warm regards,

Sincerely,

Mallika Goyal, MD
Editor Publications
Telangana Ophthalmological Society

From The Desk of the President, TOS

TOS Presidents' Vision 2016-2019 - A 3 year Mission

President's Vision

I am overwhelmed & humbled by the privilege you have bestowed on me by electing me as your next president. To occupy the chair which has been held by so many luminaries like Drs N S Reddy, Pradeep Swarup, GC, Vishwanath & Ravi Kumar Reddy to name just a few & doing justice to it is no mean task. I assure you I will strive to do justice to the confidence you have placed in me.

My baby steps in the organisation were initiated & nurtured more than 12 years back by Drs NS Reddy, Pradeep Swarup, Vishwanath & Manoj. It is they who realised that I could be useful & take up some responsibility, guided me at every step. The result- I graduated from Joint-Sec to Treasurer-Gen secretary-VP –President elect & finally to President today.

Whenever I had doubts about my abilities to take up additional responsibilities- my back up system-my Family & friends-Madhukar, Mallika, Samba, Venkatesh, Prakash prodded me along , & reassured me I can do it. My presence here before all of you today is testimony to the kind of support & encouragement I received from them.

Dream Vision of the Presidential Trio



Dr Manoj Mathur/Dr Nand Kishore/Dr Madhukar Reddy

Dr Manoj Mathur when he took charge as the President planned meticulously and came up with an innovative agenda. We then realised that a year is too short a time to at least partly achieve what we set about doing. There 3 of us then put our minds to it and a 3 year vision has more likelihood of continuity of the tasks and success. Various target areas were identified leading to formation of action groups.

Our Progress report:

Membership drive subcommittee: Dr Sharat Babu to head, aim to increase membership especially from far-flung areas.

TOS HQ Office: Allotment secured for office space in Sarojini Devi Eye Hospital (SDEH). Plan to make it fully operational in a short time.

TOS web site: New look website in place will facilitate

Online member enrolment & payment

All year round Abstract submission

SB collect portal to help conference related transactions

Dedicated page to each member to upload & update his profile

Career Guidance team headed By Dr Gangadhar & assisted by chair ARC is functional. Members can avail the facility.

Hands on Training programme at willing private Institutes for a 2 week period is operational, list is available on website. Interested members can contact Dr Vidyawathi & Dr Rajalingam.

PR Committee aimed at protecting the interests of our members in case of unfavourable treatment outcomes. The team comprising of the Supdt SDEH, Dr K Viswanath, Dr Pradeep Swarup & Dr Ravi Kumar Reddy will be the single point of interaction with the media & Public on behalf of TOS & its members.

Anti Quackery wing team of Dr G Harikishan & Dr V Sree Kumar will liaise with the concerned authorities whenever such issues are brought to their notice.

OFWS, the FBS of erstwhile APOS is functioning under the TOS & needs to be actively encouraged.

TOS Achievement Awards:

- TOS Achievement Awards - aimed at suitably rewarding our members who have significantly contributed towards raising the scientific standards of the society or in its administrative efficiency through the way of awarding credits for various works. Members attaining a certain no of cumulative credits over a minimum 3 year period become eligible for these awards.
- This needs exhaustive planning, an unbiased approach and somebody willing to spare lot of time and effort.

I cannot think of anyone other than Dr Manoj Mathur for the job

Our Road Map For 2017-18:

Permanent Conference Facility: Realised during organising the 2nd TOC at Hyderabad. Large chunk of the revenue from the conference goes to 3rd party, instead can be utilised to develop infrastructure and facilities at our own institutes.

Currently SDEH and Kakatiya Medical College (KMC), Warangal which are under Govt can be explored as possible mid segment venues. TOS can support with periodic resources whenever conferences are held at these venues. NRI Alumini from these institutes can be approached for contributions. We will work out as mutually rewarding both to TOS & the institutes.

I request the Superintendents of SDEH & KMC along with Dr Rajalingam & Dr Sreekumar to assist us in actively exploring, identifying & liaising with the govt in achieving this.

District Chapters:

Strength of any state society is its district and rural units.

Propose to develop Vibrant and actively participating District units. There is an urgent need to bring our colleagues into limelight.

Propose short CME's, social gatherings, help in developing common ophthalmic diagnostic units on a co-operative basis.

I propose to tour at least 4 districts in the coming year & request Dr Manoj Mathur & Dr Madhukar Reddy to join me.

These are all tall tasks to achieve but if we all join hands WE CAN DO IT-TOGETHER LETS MAKE IT HAPPEN.

From all our members we look forward to:

Unrestricted Co-operation

Addictive Encouragement

Constructive Criticism

We ardently seek the blessings of all our senior colleagues

I finally conclude with four beautiful thoughts on LIFE

When you look back you get EXPERIENCE!

When you look forward you see HOPE!

When you look around you see REALITY! &

When you look within you find your REAL SELF.

THANK YOU ONE AND ALL!

Dr KB Nand Kishore

From The Desk of the Secretary, TOS

Dear Colleagues,

Telangana Ophthalmological Society wishes all its members a very Happy Diwali, merry Christmas and Prosperous 2018!

The Third Annual State Telangana Ophthalmological Conference was held at Warangal successfully. The arrangements by the LOC, Scientific Program designed by Dr Pravin Krishna were exemplary. The inspiring presentation by Dr GN Rao at the inaugural function was a reminder of our social responsibility. Scientific halls were full dispelling our doubts about the thin attendance.

The present membership now stands at around 900. Request all postgraduates to become Life Members soon after they join their course. Last minute applications for life membership may disappoint the applicants with regard to having their papers accepted at the conference.

The Journal, TOS Today, is eager to see you more frequently, but lack of enough contributions from members is becoming a stumbling block. TOS is keen to see an enhanced participation from members in this front also.

Over the years the CME calendar has become more hectic and crammed resulting in near overlapping of many scientific events in our area. We all agree that our State Annual Conference should be given the pride of place.

At the GBM of TOS a resolution was adopted that ideally no scientific event be planned at least 4 weeks prior and 2 weeks after the State Conference. This is in the interest of both the organisers and our members. We seek cooperation from all institutes, district chapters and members planning to organise CMEs to go by July 2018 guidelines.

The next Annual State Conference will be held at Hyderabad. The dates and other details will be finalised soon.

With regards,

Dr. Aitharaju Ravindra, MS, DO
Hony. General Secretary
Telangana Ophthalmological Society

The forthcoming Annual Telangana Ophthalmological Society Conference is in July 2018 at Sarojini Devi Eye Hospital, Hyderabad.

From The Desk of Organizing Secretary, TOC 2017

An Exhilarating Experience

Having thrilled with the amazing organization of the 2nd State Conference of the Telangana Ophthalmological Society at Hyderabad last year, we were enthused to arrange the following Conference at Warangal under the aegis of our unit, Warangal Ophthalmological Association. At the same time, we were afraid we were going to try daredevil stunts. However, we breathed a sigh of relief when the Orugallu Eye Con 2017 came to an end but was not brought to an end.

It could happen successfully under the watchful eye of the national and state leadership & under the able guidance of the experienced elders who came to our city to grace us with their pleasant presence for three days. It was possible also as it was the apple of each participant's eye.

The local organizers split into different Committees and strained every sinew to make the venue suitable for the deliberations & convenient for the participants.

My former students from all over Telangana and other states 'tied on wings and came' to share the burden when I informed them of the Conference. Actually, they did not feel it onerous as they found themselves laden with a lot of useful information at the end of the Conference. They said, "the Conference gave us fascinating insights into the subject of our study and the object of our profession".

Other developing ophthalmologists of the state and even some senior participants felt the event was able help in improving their skills and acquiring new ideas related to their job.

All this was he result of the interaction of the participants with some of the living legends of ophthalmology – the outcome of the listening to the wonderful Orations of some eminent experts and the scholarly presentations of others at the Conference.

The theme of the Conference Sight-Insight appeared to have materialised successfully.

The financial support was generously provided by some of our Members and the trade. And, the large number of registrations was also helpful in overcoming the budget crunch.

For me, the Organizing Secretary, it was an exhilarating experience to be shoulder to shoulder with others of different strata at various levels when the luminaries of sight shed light on us.

Dr GR Bharath Kumar
Organizing Secretary, TOSC 2017

Telangana Ophthalmological Society Executive Committee

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Guest Article

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Femto-Laser Assisted Cataract Surgery

Dr Mahipal S Sachdev, Dr Hemlata Gupta, Dr Gitansha Sachdev, Dr Charu Khurana, Centre for Sight, New Delhi

Introduction

Cataract surgery by femtolaser technology has been commercially available since 2011 and has changed the way cataract surgery is performed across the world. Modern cataract surgery is fast becoming a refractive surgical procedure. Despite the excellent results obtained with the current technology, perfection is demanded by patients and surgeons alike. In this article we will discuss how through automation of many of the most delicate and critical steps of the procedure, this technology has the potential to improve the clinical outcome of both standard and premium cataract surgery.

Femtosecond laser

The femtosecond laser is useful in ocular surgeries due to its ultrafast pulses in the range of 10^{-15} seconds and its decreased energy requirements for tissue destruction. The currently available femtosecond laser devices use a near infrared laser with a wavelength of about 1053nm. Unique, ultra-short duration laser pulses (10^{-15} seconds) allows its use as a "laser blade" in a process called photodisruption or photodissection allowing for reduced unintended destruction of surrounding tissues [1].

Femtosecond lasers: Mechanism of action

The Femtosecond laser causes tissue disruption with its near-infrared scanning pulse focused to 3 µm with an accuracy of 1 µm and is not absorbed by optically clear tissues. This makes femto-assisted surgery amenable to anterior chamber targeting at various depths, as the anterior chamber provides an optically clear tissue space [2]. The near-infrared wavelength is not absorbed by the cornea, and the waves are known to dissipate approximately 100 µm from the lens tissue target. Photo-disruption is essentially induced by vaporization of target tissues, which occurs through the following steps: the focused laser energy increases to a level where a plasma is generated; the plasma expands and causes a shock wave, cavitation, and bubble formation; and then the bubble expands and collapses, leading to separation of the tissue [3]. The spots are not completely overlapping so blunt dissection is used to make entry.

Currently available devices

These include Alcon LenSx (Alcon Laboratories, Ft Worth, TX, USA), OptiMedica Catalys (Optimedica Corp, CA, USA), LensAR(LensAR Inc, FL, USA) and Technolas (Technolas Perfect Vision GmbH, Germany). All laser systems share a common platform which includes an anterior segment imaging system, patient interface and femtosecond laser to image, calculate and deliver the laser pulses. The specific technology to achieve these steps differs between the individual lasers with notable differences in imaging and docking systems and laser treatment algorithms.

Steps of surgery

Different laser platforms have different techniques of docking and rest of the steps. In this article we describe steps of femto-assisted cataract surgery using OptiMedica Catalys (Optimedica Corp, CA, USA).

Standard pre-operative dilating drops and NSAIDs are administered as for routine cataract surgery. Surgery can be done under topical anaesthesia or peribulbar block. Peribulbar block if required for cataract surgery is given after femtolaser assisted steps are done.

The initial steps for the procedure involve selecting the capsulotomy size, incision location, extent of limbal relaxing incisions and lens chopping technique. All primary incisions are placed on the steep axis while a secondary incision was placed 50-60 degrees away depending on surgeon preference. Lens chopping technique is selected based on the grade of nuclear sclerosis and surgeon's preference. Arcuate incisions are performed for refractive errors between 1 – 2 diopters. Patients with higher cylinder are recommended toric IOLs.

Docking

After all parameters are selected, the sterile patient liquid interface system is docked onto the patient's eye. When adequate applanation occurs, suction is applied and the markings on the live optical coherence tomography image are verified including capsulotomy size and centration, primary, secondary and arcuate incision location and pattern of lens fragmentation (Figure 1). During all these steps patient has to remain still. The surgeon chooses a lens fragmentation pattern based upon the grade of cataract and surgeon's preference. Surgeon adjusts and approves safety zone from posterior capsule (mostly 500 to 800 microns) that is visualised on live OCT image. The laser is then fired with the duration of the laser being 30 seconds to one min depending on the degree of lens softening selected by the surgeon.

Fig 1: Live Optical coherence tomography image is verified including capsulotomy size and centration and lens fragmentation pattern

Anterior Capsulotomy

A perfectly sized and centered capsulotomy is formed with the femto-second laser. It is kept between 5.0 to 5.7 mm depending on the type of IOL being implanted and the pupil size. In case the pupil size is smaller, the capsultomy can be adjusted intra-operatively making sure it is slightly away from the pupil to avoid the laser from hitting the iris tissue. The anterior capsulotomy is created by scanning a cylindrical shell extending from a position in the lens volume through the capsule and into the anterior chamber. An integrated OCT-guided femtosecond laser system enables precise cutting of the anterior lens capsule creating continuous sharp-edged anterior capsulotomies of exact size, shape, and position.

Lens Fragmentation

Nucleotomy of the lens is achieved using femtosecond laser fragmentation, into patterns as desired by the surgeon. Published studies have shown that FSL systems reduce ultrasound energy necessary for phacoemulsification. This allows clear visualization of the segmentation and softening pattern of the nucleus below. The lens was fragmented into the desired pattern.

The lens patterns may be specified as Chop, grid Cylinder or combined chop and cylinder patterns.the patterns the laser creates can be customized in term of number quadrants, sextants or octants. This additional step has been shown to reduce the average time and energy required to break up and remove the lens by approximately 50% [4] .

Arcuate Incisions

After determination of the topographic amount of cylinder and the steepest corneal axis the surgical plan is choosen based on a nomogram.

Primary and Secondary Incisions

Primary and secondary incisions were created by the femto-second laser according to sepcified location parameters. The geometry of the Primary Incision Pattern is constructed from one, two or three angled planes arranged along a defined arc at the corneal periphery. The planes are connected to create a single opening. The Primary Incision Pattern may represent a completely penetrating cut or a partial thickness cut. Partial thickness cuts can be created and later opened manually by the surgeon. Secondary incision pattern is similar to primary incision except that it is only a single plane incision located along an arc on the corneal periphery.

Once the femto-second laser is complete the patient is shifted into the operating room. The secondary incision is opened, visco-elastic introduced into the anterior chamber and then primary incision is opened. The capsulotomy is removed in a circumferential fashion if it is not complete taking care to avoid tags and leave incomplete areas for the end. A gentle hydro-dissection is performed and phaco-emulsifiction done along the pre-placed quadrants of the selected nuclear chopping lines(Figure 2).

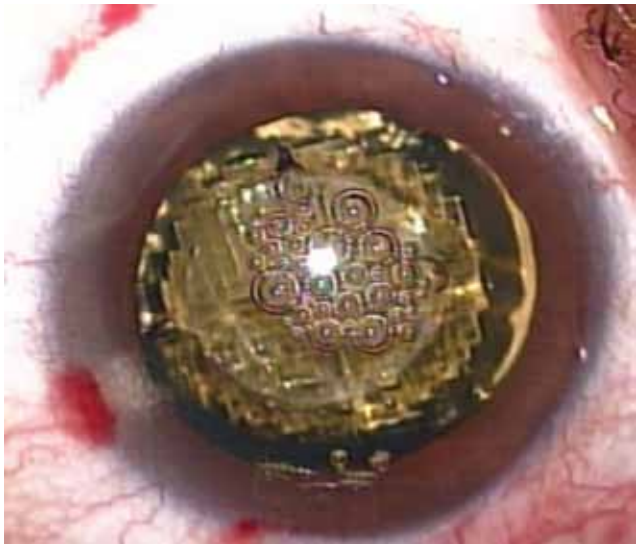

Figure 2: Grid pattern of lens fragmentaion

At this time, the standard divide-and-conquer or stop and chop technique can be used. Once the nuclear material has been removed, the surgeon may find that the removal of cortical material is slightly more challenging than with conventional phacoemulsification. At times, it may be difficult to visualize the edge of the cortex because the edge may correspond to the edge of the capsulorhexis cut by laser. Although this perfect safety zone ideally protects the capsule, it may be more difficult to extract the residual cortical material from the bag, the most challenging area being the subincisional cortex. The ease of cortical removal improves during the learning curve and appears to be an insignificant issue for experienced users. Bimanual techniques can be useful when faced with subincisional cortex or with cortex that is thicker than usual and is flush with the underlying capsule. Irrigation and aspiration is then completed and the IOL placed in the bag followed by visco-elastic removal. Standard post-operative antibiotic and steroid drops are administered for 4 weeks.

Challenges and complications

Narrow palpebral aperture, kyphosis, scoliosis, severe claustrophobia and inability to lie down calm and straight are challenges for successful docking. Small pupils initially presented as a challenge to the surgeon, as they limit the size of capsulorrhexis. Adding to this, applanation of the cone and firing of laser can further reduce the diameter of the pupil.

Femtosecond Laser assisted Cataract surgery is now being used in patients having small or incompletely dilating pupils with intraoperative use of Malyugin pupil expansion ring. Case reports showing favorable results with the use of this exciting technology in patients with phacomorphic glaucoma, subluxated lens, complicated catract, eyes with pseudoexfoliation syndrome and pediatric cataract has increased the inclusion criteria tremendously. Suction loss can be encountered with the procedure but is less common than Femto-lasik. Patient with nystagmus and psychiatric disorder may not be able to comply. Corneal opacification may interfere with laser photodisruption and alter the quality of corneal incisions and may lead to capsular tags an incomplete capsulotomy may be encountered anytime, but with the improvement of technology it is less encountered with. Surgeons should be well versed with all the steps of traditional phacoemulsification as he could have to convert the procedure into it if the need arises.

Femto assisted cataract surgery in difficult situations:

Brunescent cataract

Cataract surgery can be challenging when dealing with a very hard, brunescent cataract with a decreased protective epinuclear-cortex between the hard nucleus and the fragile capsule. Femtosecond laser-assisted cataract surgery can make the procedure safer and therefore may be the preferred technique. Gentle maneuvers protect the zonules and capsule, and reduced energy use means reduced likelihood of damaging the endothelium [5]. Using the Catalys platform , grid pattern is made creating 200 or 300 µm cubes. Hydrodissection should then be done particularly carefully, since there may be gas in the capsular bag.

Femtosecond cataract surgery can decrease the complexity of such surgery and provide a safe surgical procedure with an optimal postoperative result and a happy patient.

Traumatic cataract

Manual capsulorhexis may be challenging in traumatic cataract cases with anterior capsule laceration with or without lens subluxation or in hereditary systemic disease such as Marfan's syndrome. Nagy et al.described the use of FLACS in three cases who had penetrating eye injury and traumatic cataract with a radial tear in one of the three cases [6]. FLACS was performed successfully in these cases showing the benefice of this technique is such high-risk patients.

Schultz et al reported the use of FLACS for a decentered cataractous lens of a child with Marfan's syndrome, showing the advantage of the femtolaser for regular capsulotomy in these challenging cases of lens subluxation [7]. The effect of this surgery being lifelong in children, the accuracy provided by the laser technique is of significant importance.

Polar cataract

Posterior polar cataracts present a challenging situation to the cataract surgeon due to their association with posterior capsular defects and posterior capsular rupture (PCR). Reported rates of PCR in polar cataract is between 0 to 40 % as compared to 1.1 percent in routine phacoemulsification [8].

Live anterior segment OCT incorporated in femtolaser platform not only shows the status of the posterior capsule but also allows the surgeon to keep a safety margin from the posterior capsule (Fig 3). This ensures that there is no inadvertent damage to the posterior capsule due to collateral effects of the photodisruption created by the laser. The femtodelineation technique offers a lower PCR rate and enhanced safety during surgery in these eyes [9].

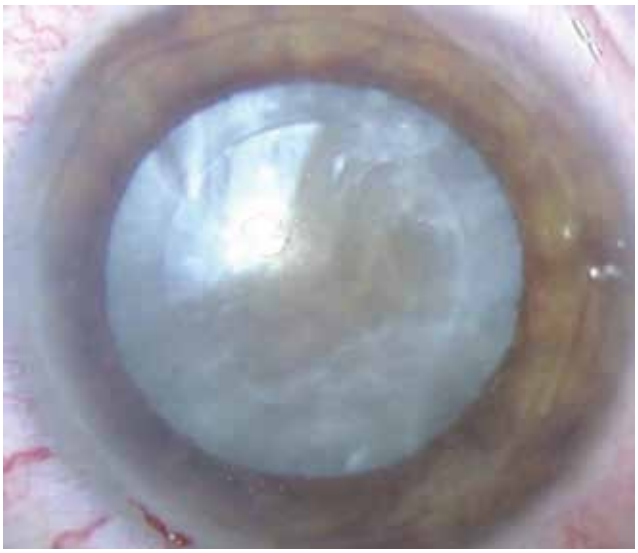

Figure 3: Free floating capsulorhexis in white intumescent cataract

Paediatric Cataract

The high elasticity of the lens capsule renders the capsulorhexis more difficult and may lead to decentred, too big or too small capsulorhexis, and eventually radial tears. Predictable refractive results being of most importance in children, and related to the well-centered and sized capsulorhexis, FLACS may play a major role in the management of paediatric cataract surgery in the future [8].

Phacomorphic Glaucoma

Another difficult surgical situation is the presence of shallow anterior chamber as seen in phacomorphic glaucoma (Fig 4). Complete capsulorhexis with femtolaser may make the procedure very safe [10].

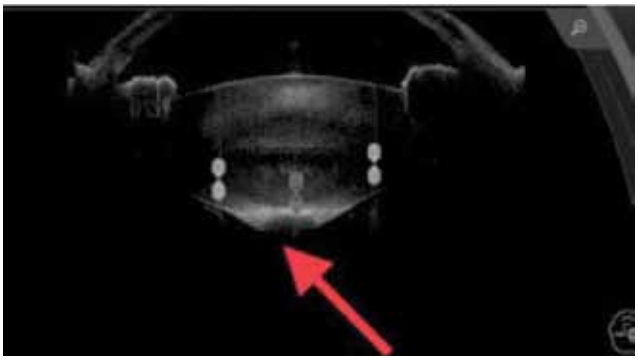

Figure 4: ASOCT showing pre-existing posterior capsular defect in Polar Cataract

Discussion

Femto-second lasers have brought a revolution in ophthalmology with their multiple uses in refractive surgery, penetrating keratoplasties and intra-corneal ring segments among many others. Introduction of the femto-second laser for cataract surgery is a fascinating technique as it targets the very basics of an ophthalmic practice. With its greater precison and accuracy, femto-second lasers are targeted at making cataract surgery similar to a refractive procedure and reduce the complication rate. Studies published since the introduction of FLACS have shown better reproducibility in terms of capsulorhexis diameter and centration, corneal wound construction and decreased ultrasound energy and time of lens fragmentation and liquefaction [11,12,13,14]

Because of the reduction in the use of ultrasound, FLACS is thus expected to reduce capsule complication and corneal endothelial cell injury . A few published studies have reported a reduction in ultrasonic phacoemulsification with femtosecond-laser lens fragmentation or liquefaction [13]. Nagy et al published the results of the first cases on ex-vivo porcine eyes and showed a 43% reduction in phacoemulsification energy and a 51% decrease in phacoemulsification time (effective phacoemulsification time = EPT) with the LenSx system compared with a 'divide-and-conquer' manual technique [14].

Femtosecond laser-assisted cataract surgery can make the procedure safer in brunescence cataract. Palanker et al reported that Catalys pre-treatment of the lens in a series of 29 eyes decreased the perceived hardness of a nuclear sclerotic cataract by two grades, from LOCS IV to LOCS II, and reduced ultrasound energy use (measured as cumulative dispersed energy) by almost 40% relative to a control group undergoing conventional cataract surgery [15].

Femtolaser capsulotomy may provide more stable anteroposterior, rotational and axial IOL position, especially for multifocal, toric and presbyopia correcting IOL. Current studies support the safety and efficacy of femtosecond laser assisted cataract surgery. Risk of endophthalmitis seems to be lesser in FLACS as compared to conventional surgery due to better self sealing corneal incisions. Further comparative studies would be needed. With improvement of technology ansd surgeons becoming more and more comfortable with the use of this technology, the indications are increasing for potential use of this technology. As of now femtosecond laser assisted cataract surgery seems to be a safe, efficient, and highly precise mode of treatment modality but further prospective multicentric studies with larger number of eyes can demonstrate the potential clinical benefits of this new exciting emerging technology.

Conclusions

Femto-second laser assisted cataract surgery offers improved result after a short learning curve. Improved UCVA & BCVA, short phacotime, tackling of co-existing astigmatism and reduced endothelial cell loss are advantages. Longer follow up in greater number of cases is needed to fine tune this technology for easier and widespread use.

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Guest Article

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Current status of Small Incision Lenticule Extraction

Mahipal S Sachdev, Hemlata Gupta, Gitansha Sachdev, Centre for Sight, New Delhi

Introduction

Femtosecond lenticule extraction (also known as ReLEx®, SMILE or FLEx) has emerged in recent years as an alternative paradigm to FS-LASIK. The early outcomes of FLEx surgery, which is considered a step toward transition to SMILE, were first published in 2008, whereas the early outcomes of SMILE were first reported in 2011. SMILE became commercially available in 2012[1]. ReLEx® or SMILE requires only a femtosecond laser to perform the entire refractive procedure, and it has various clinical, practical and economic advantages over the more traditional and well-known two-laser solution of LASIK.

SMILE has become a preferred alternative to LASIK for myopic correction within Europe and Asia. This procedure is CME approved in Europe and got US Food and Drug Administration approval in 2016. It is already available in more than 600 clinics in 61 countries worldwide. This is currently available only on a single femtosecond laser platform which is VisuMax (Carl Zeiss Meditec AG, Jena, Germany). This platform enables us to perform two different procedures: Femto second lenticule extraction (FLEx) and small-incision lenticule extraction (SMILE) [2].

Selection Criteria

Currently ReLEx SMILE is available as a treatment modality for myopic correction of upto – 10 Diopter spherical equivalent, with a maximum astigmatic error of up to – 6 D. The procedure is currently not available for hyperopic correction[3].

Patient selection criteria are similar to LASIK[4]. The patient should be 18 years or older with a stable refraction for at least a year. Preoperative corneal topography to rule out formefruste keratoconus, posterior keratoconus and pellucid marginal degeneration is mandatory. A minimal corneal thickness of 475 to 500 microns and a residual stromal bed thickness of 250 to 275 microns is required in accordance with precautionary measures to reduce the incidence of post LASIK ectasia. The procedure is contraindicated in patients with lenticular changes, glaucoma or other pre-existing ocular diseases. Retinal breaks or holes, if any, should be treated prior to the procedure.

Visumax Laser System

SMILE procedure is performed using the state of the art VisuMax Femtosecond laser (Figure 1). The VisuMax software calculates the thickness of lenticule required for refractive correction and the femtosecond laser creates a refractive lenticule with a high degree of precision. The femtosecond laser system in the Visumax Laser system remains fixed.

The femtosecond laser is delivered through a curved contact glass attached onto the laser system optical aperture. A green blinking light serves as the fixation target. The contact glass is available in three sizes S, M and L.

An infrared illumination mode is incorporated into the laser system, which allows the surgeon to ascertain better the centration following initiation of suction. During the illumination mode pupillary dilatation occurs. The surgeon can thus also verify that the pupil dilates around the intended center.

Figure 1: VisuMaxLaser System

Procedure

The procedure is carried out under topical anaesthesia. The patient'seyes is painted and draped using all aseptic precautions. A standard eye speculum is used to keep the eye open.

There are 3 discrete phases involved in ReLEx SMILE

A) Initial docking with precise centration

The joystick attached to the movable bed is used to align the patient's eye to the curved contact glass. A proper head position is achieved by tilting the patient's head medially to avoid nasal contact with the cone of the contact glass interface. The patient is asked to focus on the green blinking light and maintain fixation. Precise centration should be verified before corneal contact with the applanation interface.

B) Verification and maintenance of suction during femtosecond laser delivery

Following proper centration and adequate placement of the contact glass on the patient's eye, suction is initiated to hold the cornea against the contact glass interface. Femtosecond laser pulses with a typical pulse energy of 120 – 170nJ are delivered with a pulse repetition rate of 500 KHz. Typical spot distance between each pulse is 2-5 microns. The femtosecond laser spots are fired in a spiral track, with either decreasing (posterior lenticule surface) or increasing spirals (anterior lenticule surface).

Femtosecond laser creates photodisruption by inducing a state of plasma that is generated by vapourisation of target tissue. The force of cavitation bubble creates a cleavage plane within the corneal stroma as each gas bubble disrupts the corneal tissue at its respective position. Hence the femtosecond laser is capable of creating cleavage planes at a predetermined depth within the corneal stroma with a high degree of precision.

The various tissue disruption planes created by the laser are as follows (Figure 2):

1. Posterior lenticule surface (from periphery to centre) followed by transition zone at the edge of the refractive zone (for spherocylindrical correction). The optical zone selected determines the diameter of the posterior lenticule surface.
2. Vertical edge incision along the perimeter of the lenticule.
3. Anterior lenticule surface (from centre to periphery), which extends about 0.5- 1 mm beyond the posterior lenticule surface.
4. Peripheral corneal incision for lenticule access and extraction.

   The incision is generally created superiorly or superotemporally to preserve the nasal and temporal nerve arcades [5] and to provide surgical convenience. The corneal incision varies from 250-300 degrees in cordal length for FLEx and 30-50 degrees in cordal length for SMILE.

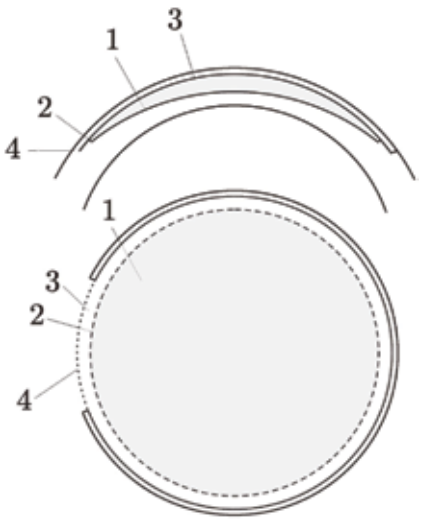

FIGURE 2: Cross Section of the Various Cleavage Planes Created During Femtosecond Laser Lenticule Extraction.

The entire laser procedure takes around 23 seconds irrespective of the refractive error to be corrected.

C) Performing manual lenticule extraction

Following completion of the femtosecond laser (treatment mode) the suction automatically turns off. The patient's eye is repositioned under the microscope (observation mode).

Femtosecond Lenticule Extraction (FLEx)

A small sharp-tipped instrument is used to open a small portion of the side cut incision, on the temporal side. A blunt spatula is then inserted in the cleavage plane created on the anterior side of the lenticule (i.e., the "flap cut"). It is then used to separate the tissue bridges in this cleavage plane, and then the flap lifted to a side, as in standard LASIK. A small sharp instrument is then used to enter into the cleavage plane on the posterior side of the lenticule to separate the edge of the lenticule. Once the edge of the lenticule has been separated, a corneal forceps is used to peel the lenticule from the underlying stroma. Once the lenticule has been peeled off and removed completely, a drop of Balanced Salt Solution (BSS) is placed over the remaining stroma. The flap is then refloated back in place. A PVA spear is used to wick excess fluid from the edges of the flap, so as to allow surface tension forces to keep the flap in place. After 30 seconds, the speculum is removed, and antibiotic drops instilled. Both eyes can be treated at the same time.

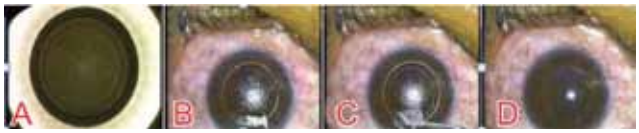

Figure 3 : Surgical Steps of the FLEx procedure

Small Incision Lenticule Extraction (SMILE)

The side cut incision is generally created superiorly or superior-temporally, in order to preserve the nasal and temporal nerve arcades, and to provide surgical convenience. A small sharp tipped instrument is used to open a small portion of the side cut incision. A small blunt spatula is inserted into the side cut incision, and the anterior surface of the lenticule separated from the overlying cornea. A small sharp instrument is then used to enter into the cleavage plane on the posterior side of the lenticule to separate the edge of the lenticule. A blunt spatula is then inserted through this edge below the lenticule and used to separate the posterior part of the lenticule from the underlying stroma (Figure 4). Once the lenticule is free from both surfaces, a small micro forceps is inserted to grasp the lenticule and extract it from the corneal stroma. After the lenticule is removed, the side cut incision site is patted down with a slightly wet PVA spear. After 30 seconds, the speculum is removed. Both eyes can be treated at the same time.

Post-operatively, with both procedures, patients may be prescribed mild steroids and antibiotics for a week and artificial tear supplements for a period of 4-8 weeks after the procedure.

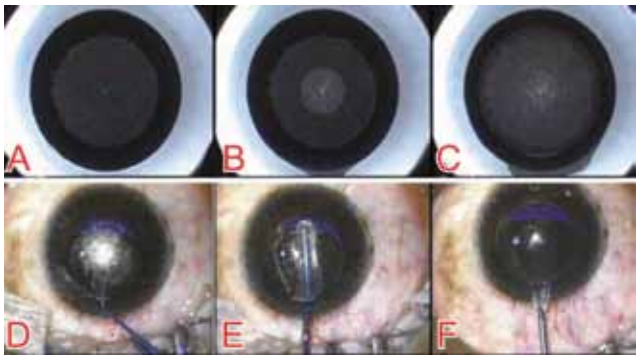

Figure 4 : surgical steps of SMILE procedure: Femtolaser assisted (A) posterior tissue disruption plane or lenticule cut (B) anterior tissue disruption plane or flap cut (C) Superior flap side cut incision. Manual (D) delineation of planes (E) dissection of planes (F) lenticule removal.

Complications

Intraoperative complications

1. Minor epithelial abrasions at incision site
2. Small tears at the incision
3. Difficulty in lenticule removal: During attempted lenticule delineation, incorrect tissue plane identification can result in primary separation of the posterior lenticule surface, resulting in its adherence to the stromal surface of the cap. In this situation, it is still possible to achieve lenticule separation, but it is more difficult.
4. Suction loss: Predisposing factors are longer duration of suction required in SMILE as compared to femtoassisted LASIK,Loss of contact between glass interface and cornea due to sudden eye or head movement, ocular factors including a small palpebral aperture, loose corneal epithelium, excessive reflex tearing, and poor fixation, fluid entry through suction ports or compressive forces against the contact glass resulting from intraocular gas-bubble transposition.

Postoperative complications

Symptoms of dry eye may be observed postoperatively, however the occurrence is less as compared to that with conventional LASIK[5].

Another complication arises due to unintended abandonment of residual intrastromallenticule fragments that may induce irregular astigmatism. Cases of epithelial ingrowth, microstriae and interface inflammation have been reported[6].

Advantages Over Femto-Lasik

1. Significantly shortened procedural time due to use of a single laser platform instead of the 2-platform procedure
2. Photo disruptive mechanism in SMILE, unlike ablative mechanism of excimer laser is independent of factors like corneal hydration, temperature, atmospheric humidity and depth of stromal ablation.
3. Increased refractive predictability over excimer laser particularly for higher refractive errors.
4. Significantly fewer total higher order aberrations particularly spherical aberration.
5. With femtosecond laser, the peripheral loss of fluence is not a factor at all, and no compensation needs to be carried out. So the amount of tissue required per diopter of treatment is smaller than that required with an excimer laser which compensates for the peripheral energy loss.
6. Reduced amount of energy is applied on to the cornea. Moreover the heat generated by an excimer laser is in a relatively shorter period resulting in adverse effects on corneal healing.
7. Reduced number of corneal nerves is severed due to smaller flap diameter and side cut incision, thereby reducing the incidence of postoperative dry eye.
8. No flap related complications
9. The small side cut incision heals relatively faster causing less patient discomfort.

Additional Uses of Smile Lenticule

Tailored stromal expansion with refractive lenticule for crosslinking the ultrathin cornea – Sachdev Technique[7]

Traditional corneal collagen crosslinking (CXL) requires a minimal stromal thickness of 400 microns for the procedure to be carried out safely and effectively in case of keratoconus. However patients with advanced forms of keratoconus often have thinner corneas, thus making the disease not amenable to traditional CXL.

We described a technique recently where using refractive lenticule, the thickness of the cornea is increased in the most physiological manner by adding stromal tissue whose biological and absorptive properties are the same as that of the cornea to be treated. Refractive lenticules of variable thickness can be obtained. Placement of the central lenticule over the apex of the cone enables us to augument the corneal thickness where required while sparing the remaining stroma to be crosslinked normally.

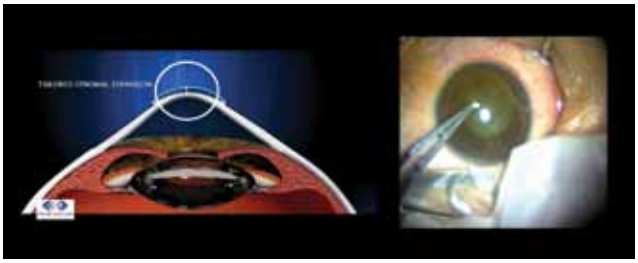

Figure 5: augmentation of stromal thickness by placement of lenticule over stromal bed following epithelial debridement

Femtosecond laser intrastromal lenticular implantation for hyperopia (FILI)[8]

Cryopreserved stromal lenticules obtained following SMILE for myopic correction are placed in a femtosecond created intrastromal pocket for the treatment of hyperopia.

It may potentially be a safe and effective alternative to excimer laser ablation for hyperopia because of the low risks of

regression, haze, flap-related complications, postoperative dry eye and higher-order aberrations.

Latest Advances: Circle Software

A recent adaptation of small-incision lenticule extraction software (Circle, Carl Zeiss Meditec AG) enables revision of the previously created cap by remodeling it into a larger diameter flap (with hinge) followed by excimer laser ablation[9].

In the CIRCLE procedure, the femtosecond laser is used to create a) an incision plane encircling the original "cap" cut as a lamellar ring and b) a side cut with hinge around the new incision plane and c) a "junction cut" which allows the original "cap" and the new incision plane to be part of one larger surface.

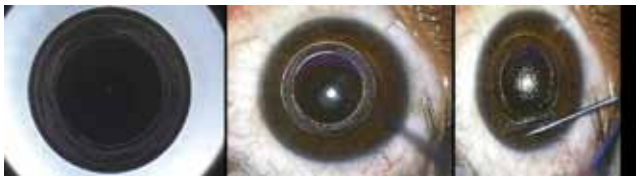

Figure 6: A lamellar ring is created at the same depth as the existing SMILE incision allowing the conversion of the SMILE cap into a hinged flap. The flap is then raised followed by excimer laser ablation.

Discussion

An analysis of the results of SMILE procedure indicates safety, efficacy and predictability of the femtosecond lenticule extraction procedure, with results for low to moderate myopia being more predictable than those for high myopia. Several groups have now published results of the procedure, including comparison of the results with results obtained for FS-LASIK[10, 11]. In general, the results seem to be similar to those obtained after FS-LASIK[12,13].

Sekundo et al [14] published their initial results on 10 eyes, which indicated that the procedure was effective, with over 90% of the eyes within 1 diopter of the intended correction. No eye lost 2 or more lines of best corrected visual acuity at 6 months. The study also revealed a high level of patient satisfaction.

Shah et al. [15] published results on SMILE with a single incision, where no flap was lifted, and the lenticule was extracted from a single small incision. In their study of over 50 eyes, they showed that at 6 months, 79% of the eyes had a UDVA of 20/25 or better. The study also revealed a high safety profile with only 5% of the eye losing a line of best corrected visual acuity.

While initial studies revealed a significantly slower visual recovery after ReLEx®, relative to FS-LASIK, Shah et al.[16] demonstrated that the slower visual recovery was at least partly due to the laser scanning patterns. Subsequently, the laser manufacturer made Scan Pattern B into the standard method of treatment.

Hjordtal et al [17] studied the results of ReLEx® in a relatively large sample (670 eyes of 335 patients) of patients with relatively high myopia (Pre-operative Mean Spherical Equivalent of -7.19+/- 1.03 D). At 3 months, 80% and 94% of the eyes were within +/- 0.5D and +/- 1.0D of the intended correction respectively. 84% of the eyes had a UDVA of 20.25 or better. In this study, as many as 2.4% of the eyes lost 2 or more lines of CDVA.

Vestergaard et al [10] published results of femtosecond lenticule extraction on a group (127 eyes) of moderate to high myopic eyes (Preoperative Mean Spherical Equivalent of -7.18 +/- 1.57 D). In this study, refractive outcomes were similar to those above, with 73% of the eyes having a UDVA of 20/25 or better at 3 months.

Kimaya et al [18]attempted to treat a group which comprised mainly of low to moderate myopia (Pre-operative Mean Spherical Equivalent of -4.26 +/- 1.39 D) with femtosecond lenticule extraction. In this study, at 6 months, all eyes were within +/- 0.5 D.

Kunert et al [19] attempted to perform Vector Analysis for a group of eyes with myopic astigmatism to analyse the results on astigmatism. They concluded that in terms of safety, predictability and efficacy, femtosecond lenticule extraction was similar to excimer lasers for the correction of astigmatism. There was a slight regression of the astigmatism correction (10%) with time. At 6 months, the mean error ratio was 0.68 +/- 0.75 (SD) and the mean correction ratio was 1.11 +/- 0.69.

Kimaya et al[20] compared a group of eyes treated with femtosecond lenticule extraction with eyes treated with wavefront guided LASIK in terms of asphericity and wavefront aberrations. They found that in myopic eyes, FLEx induces significantly fewer ocular fourth-order aberrations than WFG-LASIK, possibly because it causes less oblation in the corneal shape, but there was no statistically significant difference in visual acuity or in the induction of third-order aberrations and total higher order aberrations. It was suggested by Kimaya et al that FLEx is essentially equivalent to wavefront guided LASIK in terms of visual acuity and total higher order aberration induction, although the characteristics of higher order aberration induction are different.

The ReLEx® procedure has been shown by several studies to induce fewer spherical aberrations, though most of the same studies also show that the procedure induces higher third-order aberrations when compared to FS-LASIK[2]. As a sum total, in terms of higher order aberrations, the results of ReLEx® seem to be in line with those obtained after FS-LASIK.

In the near term, a review by the FDA of clinical data on SMILE in myopic astigmatism is anticipated, and ongoing research is exploring SMILE techniques for correction of hyperopia.

In conclusion, Femtosecond lenticule extraction surgery offers significant clinical, practical and economic benefits in comparison to conventional or femtosecond LASIK using the excimer laser.

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Guest Article

Myopic Traction Maculopathy: Current Concepts and Our Experience

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Introduction

Myopia is a leading cause of visual impairment in south east Asia region including India and is an important cause of blindness.[1]The North Indian Myopia Study (2015) estimated the prevalence of myopia in urban school children in New Delhi to be 13.1%.[2] In many south east Asian countries, degenerative myopia in the form of myopic traction maculopathy (MTM) is the most common cause of blindness.[3]

MTM is a term introduced by Panozzo and Mercanti to describe a condition with abnormal vitreomacular adhesions (vitreomacular traction [VMT]) and/or epiretinal membrane (ERM) in combination with degenerative fundus changes which may lead to macular damage in the form of foveoschisis, lamellar macular holes or macular detachments. Traction is a common phenomenon in all these manifestations. This condition is also referred to as myopic foveoschisis. MTM is seen in about 9% of high myopes with posterior staphyloma.[4] With the use of optical coherence tomography (OCT), it has been estimated that the prevalence of MTM in highly myopic eyes with posterior staphyloma ranged from 9% to as high as 34%.

There was only sparse literature on this entity a decade ago and currently this entity seems to be increasing in prevalence. This is due to a) increased detection of this entity owing to significant improvements in OCT technology and b) due to increasing prevalence of myopia per se. Over the last few decades Singapore, Japan, Hong Kong, China and Taiwan have seen many fold increase in prevalence of myopia in children completing school. The prevalence of pathological myopia and the blindness it causes is also proportionately increasing. Unfortunate fact is also that this causes vision loss that is permanent, often bilateral and affects the productive middle ages. Therefore the economic impact of this epidemic increase in cases of pathologic myopia and MTM to an individual and a nation is significant.

Pathophysiology

Myopic foveoschisis almost always occurs within the posterior staphyloma due to a gradual stretching or splitting of the retina between the non-compliant inner retina and the relatively flexible outer retina. Various factors leading to limitation of inner retinal flexibility include adherence of vitreous cortex to the retina after posterior vitreous detachment (vitreoschisis), presence of epiretinal membranes and inherent rigidity of the internal limiting membrane. These factors result in a centripetal traction, the effect of which is greatly enhanced in presence of centrifugal force exerted by progressive stretching and staphyloma in highly myopic eyes.

Traction from the retinal vessels is also thought to predispose to myopic foveoschisis. These can be observed on OCT images as retinal vascular microfolds on the retinal surface.

Highly myopic eyes develop vitreous liquefaction at a younger age and may have larger precortical vitreous pockets than normal eyes resulting in a high incidence of residual vitreous cortex on the retina after complete posterior vitreous detachment (PVD) in these eyes. This could result from splitting of the lamellar vitreous cortex (vitreoschisis) with the outermost layer of the cortex remaining on the retina. Contraction of this premacular vitreous generates anterior traction on the fovea and leading to MTM.

Clinical features

Myopic foveoschisis is often asymptomatic initially and progresses slowly and variably, leading to loss of central vision from foveal detachment or macular hole formation. Patients can present with reduced vision or central metamorphopsia or may remain asymptomatic for a long time. In a patient with pathologic myopia and posterior staphyloma with unexplained visual loss or visual symptom, an MTM should always be suspected. A retrospective study of myopic foveoschisis showed worsening of visual acuity in 69% and stable vision in 31% over a mean follow-up of 31.2 months.

For the diagnosis of MTM, OCT scanning is much more reliable than clinical evaluation based on symptoms and biomicroscopy. This is due to the thin retina in myopes with poor pigment epithelial contrast and pre-existing poor visual acuity in high mypoes due to various reasons such as focal chorioretinal atrophy, myopic choroidalneovascular membranes or amblyopia. But having a high clinical suspicion helps in anticipating a finding like MTM. On OCT, early stages of MTM presents as a splitting (retinoschisis) of the neural retina into a thicker inner layer and a thinner outer layer at the level of the external limiting membrane. Presence of "bridging columns" is characteristics of retinoschisis. Retinoschisis may also occur in outer retinal layers or both inner and outer layers simultaneously (compound foveoschisis). The lateral location of this schisis may be anywhere within the posterior staphyloma or all over the posterior staphyloma. Logically, subfovealschisis have poorer visual acuity and

prognosis. The outer layer may also get detached from the RPE in more advanced MTM giving the appearance on OCT of a dome-shaped neurosensory detachment external to a flatter shallow detached inner leaf of the neural retina. The other common OCT characteristics seen in patients with MTM are macular holes, epiretinal membranes, retinal microfolds due to inelastic and sclerotic arterioles, ILM detachment, ellipsoid zone discontinuity, paravascularmicroholes and patchy or diffuse chorioretinal atrophy.

Treatment

Myopic foveoschisis may remain anatomically and functionally stable for quite long duration, with case reports of spontaneous resolution over time. Therefore, initial observation can be offered to patient with good presenting visual acuity, minimal symptoms and early stages of MTM. But treatment is indicated in patients who are already in advanced stages of MTM such as macular hole or posterior pole detachment and in those who have documented progression on serial follow up. Treatment includes pars planavitrectomy(Figure 1) with internal limiting membrane peeling with gas tamponade with or without a macular buckle. Various combinations of these component parts of the surgery have been tested in MTM patients with results suggesting that vitrectomy with ILM peeling alone or with a macular buckle performs better than a macular buckle alone or a vitrectomy alone (without ILM peeling).

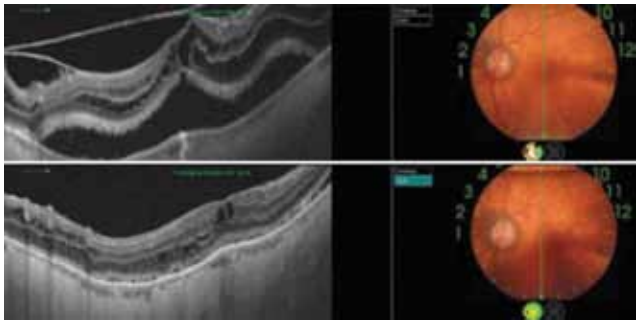

Figure 1. (a) Swept-Source OCT image at baseline of a patient with myopic traction maculopathy, (b) SS-OCT image at 6 months post-operative showing favourable anatomic success with complete resolution of myopic foveoschisis.

Our Experience

We recently reported our experience of treating eyes with MTM using vitrectomy and ILM peeling with gas tamponade.[5]We prospectively analysed the surgical outcomes of nine eyes with myopic traction maculopathy diagnosed on SD-OCT who underwent PPV with center-sparing ILM peeling for resolution of foveoschisis and improvement in best-corrected visual acuity (BCVA). Centre-sparing ILM peeling was accomplished with the help of microscope integrated OCT (miOCT)(Figure 2).

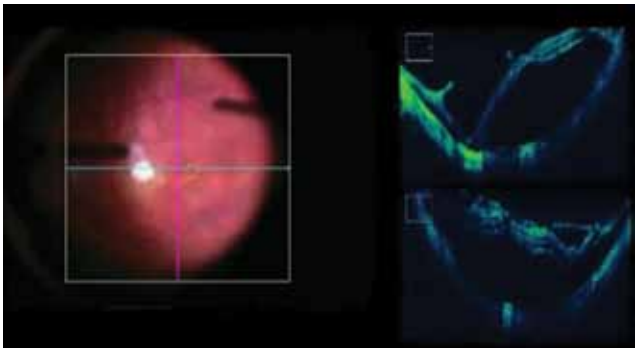

Figure 2. Intraoperative microscope-intergrated OCT (MIOCT) grab of a patient with MTM undergoing pars planavitrectomy showing ILM-schisis (white arrow) and neurosensory detachment (yellow asterix).

All patients were followed up for more than 9 months.We found that foveoschisis resolved in all 9 eyes on SD-OCT(Figure 3).

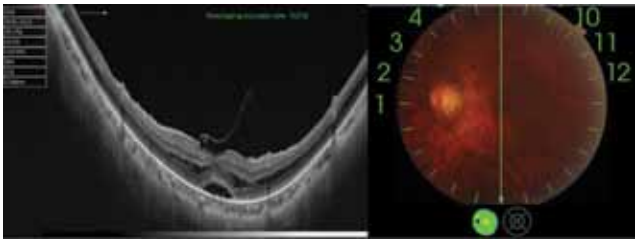

Figure 3. Post-operative SS-OCT of a patient with MTM showing spared foveal ILM tissue(blue arrow) with normal foveal contour alongwith small localised neurosensory detachment(yellow asterix).

At 36 weeks, therewas a significant improvement in mean BCVA fromthe preoperative BCVA (P = 0.0089) along with areduction in the CMT from 569.77 ± 263.19 to166.0 ± 43.91 um (P = 0.0039). None of the eyes showed worsening of BCVA or development of fullthicknessmacular hole in the intraoperative or follow-upperiod.

Vitrectomy with ILM peeling in pathologic myopes is not without its set of problems. The following are the challenges we faced in surgery for MTM:

Longer axial length: The patients with myopic traction maculopathy have pathological myopia and hence long axial length. Long axial length led to difficult instrumentation, especially in procedures like ILM peeling which require a lot of expertise.

Thinned out retina & severe CR degeneration: This lead to poor contrast even after staining with Brilliant blue G, leading to difficulty in viewing the ILM and posterior pole. Also, complete ILM peeling from over the staphyloma may lead to destabilization of Muller cells resulting in deroofing of cystic spaces and subsequent development of macular hole. MIOCT is very useful to prevent this complication(Figure 4).

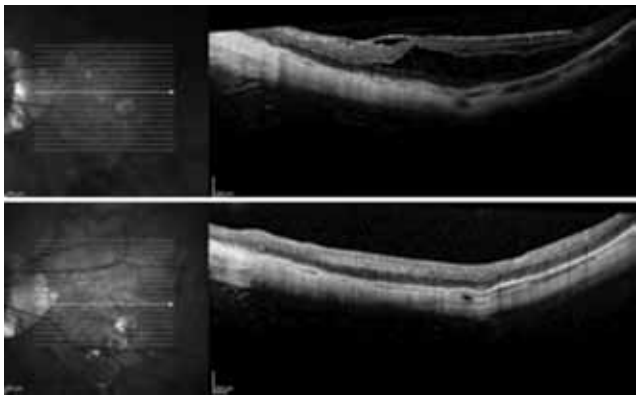

Figure 4. (a) Pre-operative Spectral-Domain OCT (SDOCT) of a patient with MTM showing foveoschisis and parafovealschisis corresponding to vision loss. (b) Post-operative 6month SD-OCT of same patient showing attached retina.

3. Previous refractive surgery: Presence of previous refractive surgery in eyes with pathologic myopia is not uncommon and leads to difficulty in visualization of fundus.[6] Intraoperativedifficulties during PPV in patients with phakic-IOL (pIOL) included problems in visualization due to aberrations produced at the edges of the optical zone of the pIOL. The aberrations were present with both the contact flat lens (Chalam Direct SSV® lens Volk Optical, Inc.) and wide angle viewing system (MiniQuad XL® contact wide angle viewing lens, Volk Optical, Inc.) as well as non-contact systems (Resight, Ziess). The aberrations were more marked while using the Chalam direct SSV ® lens probably due to a smaller field of view, a higher magnification and an increased depth of focus. The presence of ghost images at the edges of the optical zone caused difficulties in intraoperative visualization especially during critical steps such as internal limiting membrane peeling and peripheral vitrectomy. The presence of such aberrations while manipulating in the macular region can also increase the risk of complications.

Prognosis

Myopic foveoschisis without macular hole undergoing vitrectomy with ILM peeling has good anatomic and functional outcomes although complete resolution of foveoschisis may take a few months. Prognosis in patients with macular hole or posterior pole RD is more guarded.

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Guest Article

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Optical Coherence Tomography Angiography in Normal and Uveitic eyes

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Introduction

Unique invention in the field of OCT technology is Optical Coherence Tomography Angiography (OCTA) which is a dye and contact free non-invasive technology that utilises motion contrast imaging attributed to movement of erythrocytes to produce high resolution three-dimensional imaging of different segments of retina and choroid at microcirculation level and localization of abnormalities[1]. Blood flow being the only dynamic structure in retina and choroid, OCTA effectively identifies important vascular changes in different retinal and choroidal diseases.The basis of OCTA is to repeatedly scan a particular region and then examine the resultant images for changes in blood flow through vessels.It is based on the principle where area with high flow rate like in blood vessels has larger difference in between the scans unlike no or limited flow area which are near normal to previous scanned area.

Two different wavelengths of light are being utilised in OCTA. Longer wavelength's have a better tissue penetration offering better immunity over lenticular opacities giving improved visualisation and localisation of lesions in deeper tissues. Table 1 illustrates difference between Swept source (SSOCTA) and Spectral Domain OCTA(SDOCTA)[2].

Table 1

	SSOCTA	SDOCTA
OCTA	Swept source	Spectral Domain
Wavelength	1050 nm	840 nm
Light Source	Tunable swept laser	Broad band near infrared superluminiscent diode
Detector	Single photodiode	Spectrometer
A scans per second	400000	70000
Total A scan	4 x 1600 x 400	2 x 304 x 304
B scan repetition	4	2
Axial resolution	10 µm	5 µm
Acquisition time	6.7 seconds	3 seconds

OCTA device has an infrared laser that is projected on the posterior retina and the reflected light from motion contrast is detected as analogue which is converted later into digital signal by computer. Multiple A scans of a particular area of interest is compiled into a B scan which is analysed for both cross sectional structural and blood flow information. The motion contrast is mainly detected by 2 methods – Phase variance and speckle or amplitude decorrelation.

Phase variance is based on wave property of reflected light. Blood cells which move induce a variation in the phase of light reflected from one instant to the next, and this variance can be measured.[3] Each reflecting point of light forms a secondary spherical wavefront which superimpose to form small regions where the light waves constructively or destructively interfere. With coherent light as used in OCT, these discrete areas of constructive interference are the small speckles which change more rapidly over time.[4] The amplitude of light wave of different B scans are recorded at the two-time points over same area are compared.This change of reflection strength is referred as amplitude decorrelation.

It is crucial to note various factors which lead to low signal noise ratio (SNR) like saccadic eye movements, various cycles of cardiac rhythm, fixation difficulties. To improve SNR, some OCTA utilise certain image processing procedures like split- spectrum amplitude decorrelation angiography algorithm(SSADA). The SSADA algorithm considers the differences in the amplitude of the reflected light between the consecutive B-scans at each spatial location within the collected data. The algorithm splits the OCT image into multiple bands thereby increasing the usable image frames with low axial resolution so that they are less susceptible to eye motion. The amplitude of reflected light from the static tissue does not fluctuate between B-scans and has a low decorrelation value unlike blood flow which is represented by a high decorrelation value.[5]This averaged technique improves SNR by averaging the decorrelation values across various number of B scans and number of band width segmentations and also has high level of connectivity in the image thereby showing a smooth and continuous image of vasculature.

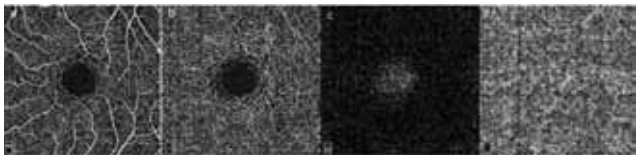
Most common commercially available OCTA machines areZeiss Angioplex™ angiographic imaging on CIRRUS™ HD OCT platform, Angiovue Imaging System (Optovue, Inc., Inc, Freemont,CA), Topcon with OCTA Ratio Analysis, Heidelberg engineering with Tru Track™

In our article , we elaborate use of Angiovue Imaging System (Optovue, Inc., Inc, Freemont, CA) which utilises SSADA in uveitis. Once the data is processed using the above algorithm, the images of retina and choroidal vasculature are presented as four zones[6] – superficial plexus, deep plexus, outer retina, choriocapillaries (Table 2, Figure 1)

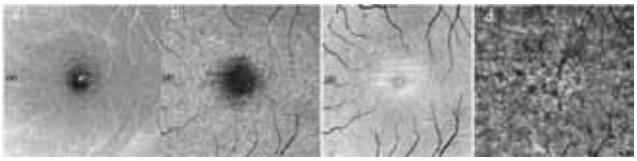
Table 2

	Superficial Layer	Deep Layer	Outer Retina	Chorio capillaries
Vasculature involved	Superficial retinal plexus	Deep retinal plexus	Normally doesn't show any vascular plexus	Chorio capillaries
Pattern of vessels	Centripetal pattern which are continuous with perifoveal arcade with regular meshes	Close knit pattern of vessels distributed around the avascular zone	Normally doesn't show any vascular plexus	Coarse texture
Upper offset	3 µm from ILM	15 µm from IPL	70 µm from IPL	30 µm from RPE
Lower offset	15 µm from IPL	70 µm from IPL	30 µm from RPE	60 µm from RPE
Thickness	10 µm	30 µm	30 µm	10 µm
Layers	Retinal nerve fiber layer, ganglion cell layer and superficial inner plexiform layer	Deep inner plexiform layer, inner nuclear layer, outer plexiform layer, and superficial outer nuclear layer.	Deep outer nuclear layer to the external limiting membrane.	Chorio capillaries

Figure 1



- **OCT Angiogram Fields of View and Segmentation Layers on Angiovue.** The normal left eye of a 40 year old indian woman using the Angiovue optical coherence tomography angiography (OCTA) software of the RTVue XR Avanti (Optovue, Inc., Fremont, CA). Fig (1) Full-thickness (internal limiting membrane to Bruch's membrane) 3 x 3 mm OCT angiogram. **1(a)** 3 x 3 mm OCT angiogram of the "Superficial" inner retina. **1(b)** 3 x 3 mm OCT angiogram of the "Deep" inner retina. **1(c)** 3 x 3 mm OCT angiogram of the outer retina shows absence of vasculature. The white represents noise. **(1(d)** 3 x 3 mm OCT angiogram of the choriocapillaris is generally homogenous. There is black shadowing from retinal vessels.



OCT EnfaceAngiogram Fields of View and Segmentation Layers on Angiovue. The normal left eye of a 40 year old indian woman using the Angiovue optical coherence tomography angiography (OCTA) software of the RTVue XR Avanti (Optovue, Inc., Fremont, CA). Fig (1) Full-thickness (internal limiting membrane to Bruch's membrane) 3 x 3 mm OCT angiogram enface . **(a)** 3 x 3 mm enface OCT angiogram of the "Superficial" inner retina. **1(b)** 3 x 3 mm enface angiogram of the "Deep" inner retina. **1(c)** 3 x 3 mm enfaceOCT angiogram of the outer retina shows absence of vasculature. The white represents noise. **(1(d)** 3 x 3 mm enface OCT angiogram of the choriocapillaris is generally homogenous..

OCTA over FFA has varied advantages. It doesn't utilise any dye like in FFA so it safe, non-invasive and can be done repeatedly on follow up examination if required. It can be acquired within seconds. Leakage of dye cannot be evaluated thereby it precisely delineates the boundaries and areas of capillary dropout and neovascularization net. Three-dimensional imaging allows evaluation of foveal capillaries in multiple planes at various layers of retina and choroid. Lack of blood vessel wall staining allows better visualization of blood column abnormalities, collateral, arteriovenous shunts and dilated blood vessels. Limitations include limited field of view in OCTA. Nevertheless, FFA/ICGA still considered the first line diagnostic modalities in case of differentiating various uveitic entities as OCTA presents with many artifacts.

Significant changes in retinochoroidal vasculature are noted in pathophysiology of uveitis. The novel OCTA technology aids in determining the pathology and natural history of uveitic entity. Our article presents brief overview of usefulness of OCTA in uveitis.

Retinal Vasculitis

Retinal vasculitis is an inflammatory disease mainly involves the retinal vessels. It is mostly idiopathic or seen in association with systemic diseases. Clinically, active disease appears as sheathing or cuffing around affected vessel segments. Fundus Fluorescein angiography is very sensitive in diagnosing active vasculitis which characteristically shows leakage of dye either focal as seen in sarcoidosis or more diffuse in Behçet's disease and Eales disease. It also delineates non perfusion areas and neovascularisation of disc and elsewhere. Leakage of dye from the optic nerve head arises from dilated capillaries, which may be due to either to primary infiltration as in sarcoidosis or secondary vascular changes induced by intraocular inflammation.

OCTA in retinal vasculitis gives detailed microvascular changes in vasculitis on progression like capillary dropout or loss in the superficial retinal capillary plexus, enlargement and/or irregularity of the foveal avascular zone, capillary remodelling, and normal flow in the presence of exudates[7]. The deep capillary plexus are thought to be more prone to ischemia being watershed zone, compared to the superficial plexus but projection artefacts have to addressed.[8]

Vogt Koyanagi Harada Syndrome

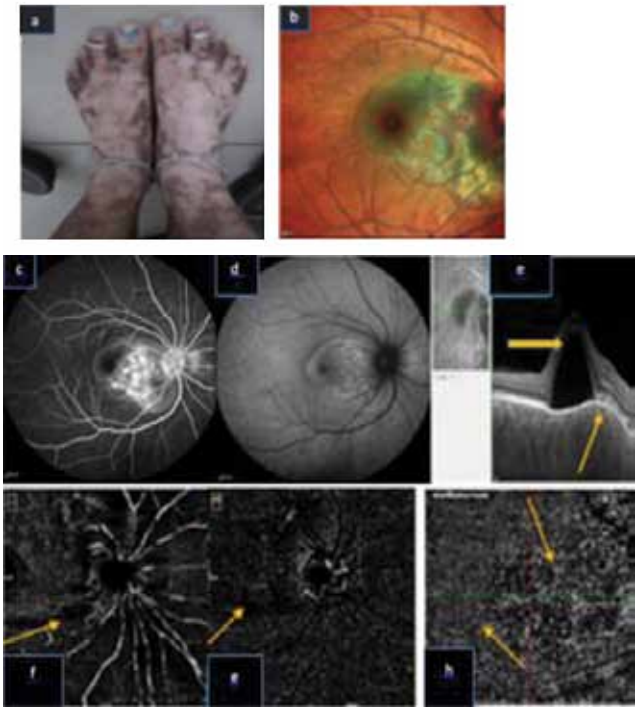
Vogt Koyanagi Harada Syndrome (VKH) is a multisystem disease which presents as bilateral granulomatous inflammation often

with along with or without additional neurologic, integumentary, and auditory symptoms. It is presumed secondary to autoimmune sensitization of T lymphocytes against melanocytes in genetically susceptible individuals notably HLA-DRB1*0405, which recognizes melanocyte proteins. Clinical presentation may vary from bilateral granulomatous uveitis, mild vitritis, multifocal serous retinal detachments with yellowish white retinal exudates, granulomata (Dalen Fuchs Nodules) and optic disc edema and hyperaemia. Classic histopathologic characteristics in acute VKH include granulomatous inflammation with primarily involvement of choroidal stroma sparing choriocapillaries and leading to bilateral exudative retinal detachment. As disease advances in chronic stages, non-granulomatous uveitis is noted which later presents as granulomatous uveitis in chronic recurrent disease and finally might involve choriocapillaries and outer retina. In chronic VKH the peripheral fundus scars noted are focal chorioretinal atrophy with loss of retinal pigment epithelium.

FFA typically shows variable size and shapes of pinpoint hypofluorescent areas in early phase which later become hyperfluorescent suggestive of pooling of dye in the areas of exudative retinal detachment. ICGA shows multiple hypocyanescent dark spots with disc hyperfluorescence in early and late phases suggestive of inflammatory foci suggestive of choriocapillaries ischemia.Corresponding to these areas of dark spots noted on ICGA, En face OCTA usually demonstrates multiple areas of dark foci of flow void suggestive of choriocapillaries hypoperfusion which are of variable size and shape. The corresponding OCTA structural en face image and on cross sectional OCT, there is no evidence of signal loss suggesting these areas to be of true flow void areas.[9]

One case report demonstrated a pattern of concentric cycles in deep capillary plexus, corresponding to disruption of the outer plexiform layer due to intrusion of the outer nuclear layer on OCT. Choroidal Granulomas usually presenting in deeper choroidal layers of choroid which are at times noticed on Enhanced Depth Optical Coherence Tomography are not evaluated by OCTA.

OCTA helps in differentiating VKH from CSCR (Central Serous Chorioretinopathy)[11]. VKH is primarily a diffuse stromal choroiditis with secondary involvement of the choriocapillaris, whereas as CSC occurs a result of choroidal vascular hyperpermeability. Both entities are seen as dark areas in choriocapillaries on OCTA (Figure 2) but these areas are found even in the areas not corresponding to the areas of subretinl fluid on enface images in VKH suggesting true flow void areas implying chorocapillaries ischemia which corresponds to choroidal inflammatory foci on ICGA unlike in CSCR where structural enface images showed signal loss due to the shadowing effect of the overlying retinal structures, sub retinal fluid, and RPE, thereby confirming that such dark hyporeflective areas did not represent true flow void. OCTA also helps as an adjuvant in determining course of disease, treatment response, resolution of disease and recurrences.



• Fig (a)Showing vitiligo patches. Multi Color Fundus Photo RE (b) showing subretinal fluid (SRF) temporal the to the disc. FFA (c) showing pinpoint leakage Corresponding FAF (d) showing hyperautofluorecence at the area of SRF. EDI OCT (e) showing large neurosensory detachment with choroidal granuloma (yellow arrow). On OCTA deep capillary plexus (f) showing cystoid spaces and outer retina (h) and choriocapillaries (i) showing flow void areas (yellow arrows)

Figure 2: A case of 42 years female diagnosed with complete VKH disease

Birdshot Chorioretinopathy

Birdshot chorioretinopathy (BCR) is chronic progressive posterior uveitis which is bilateral in nature. HLA – A29 is considered to have a central role in the pathogenesis of BCR. Autoimmunity triggering focal non-granulomatous T cell infiltration throughout the choroid particularly within choroidal stroma is considered as main door to the immune response to initiate which further leads to many inflammatory and immune sequelae like exposure to multiple highly immunogenic antigens like retinal S-antigen and Intraretinal-Binding Protein (IRBP) which further contribute to extensive retinal autoimmunity finally leading extensive tissue damage especially choroid and also retina.

Minimal symptoms in early stages of disease leads to significant delay in diagnosis which ultimately leads to irreversible tissue damage and visual loss. Research diagnostic criteria for BCR as defined at the 2006 UCLA international workshop is defined as follows Essential criteria being bilateral involvement with three or more characteristic birdshot lesions inferior or nasal to the disc in one eye, mild anterior chamber inflammation (less than 1+ cells on SUN score), low-grade vitreous reaction (no more than 2+ on SUN score). Birdshot lesions were defined as cream-coloured, elongated, choroidal lesions with irregular borders, the long axis of which is radial to the optic disc. Additional supportive features are presence of HLA-A29 positivity, retinal vasculitis, and Cystoid Macular Edema.

The BCR choroidal lesions appear hypofluroscent on ICG which correspond to choroidal lymphocytic aggregates

on histopathology. On OCT, disruption of retinal pigment epithelium and break in ellipsoid zone with retinal thinning is noted corresponding to these lesions. Optical coherence tomography angiography demonstrated dark spaces in the choriocapillaries layer suggestive of absence of choriocapillaries which corresponds either to a complete atrophy of choroidal vessels or greatly diminished blood flow on OCTA[12]. Sometimes, larger choroidal vessels which are usually seen deeper in the choroid are seen traversing these dark areas of decreased choriocapillaries flow. It is a speculation whether these larger vessels represent a compensatory response by the choroid or if they are simply Sattler's layer vessels which are pushed into an atrophic choriocapillaries area.

Various studies demonstrated various retinal changes in deep retinal plexus on OCTA like abnormally tortuous vessels, capillary loops and focal dilatations, increased intercapillary predominantly in perifoveal and temporal regions. In BCR, pathogenesis involved in retinal neovascularisation was prominently inflammation in the absence of capillary non perfusion[1].

But as OCTA demonstrated increase in intercapillary space which is increasing speculations of relative ischemia that could be proangiogenic, in spite of absence of the obvious areas of capillary nonperfusion. BCR has been reported in the western literate and it is not seen in our country

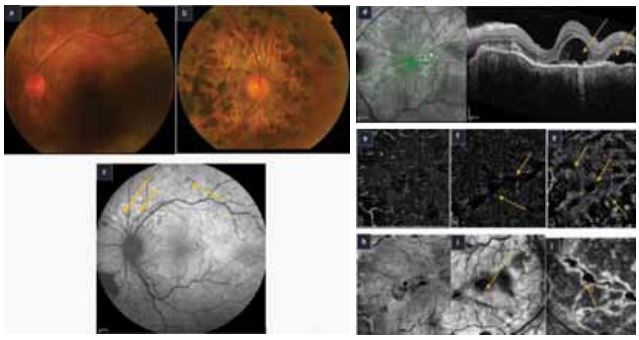
Serpiginous Choroiditis

Serpiginous Choroiditis is a chronic, bilateral, asymmetric, recurrent, progressive disease of unknown aetiology predominantly affecting the retinal pigment epithelium, choriocapillaries and choroid. It classically starts at the juxtapapillary or peripapillary area and progresses centrifugally from the disc to involve the macula. Because of its peculiar extension in a serpiginous fashion, hence the name. The disease has a destructive course. On resolution of disease, degeneration of retinal pigment epithelium and choroid sets in with further fibrous scarring and pigment hyperplasia. Sub retinal neovascular membrane (SRNVM) formation sometimes seen in chronic course.

Histopathology shows predominant atrophy of choriocapillaries, further RPE and photoreceptor layer. Lymphocyte infiltration at the margins of the lesion noted. Large choroidal vessels are generally spared. Although aetiology is unknown, association of various infective agents has been implicated in aetiopathogenesis of Serpiginous Choroiditis . It presents as various patterns of placoid progression like classical peripapillary, macular, ampiginous and serpiginous like choroiditis.

OCTA demonstrated notable changes in the choriocapillaries layer like presence of dark fluid void areas suggestive of decreased vascularity whereas the retinal vasculature showed no significant findings other than a hyporeflective round space, which was considered to be due to intraretinal fluid. (Figure 3) On correlating these corresponding areas of flow void on en face OCTA and OCT decreased signal transmission noticed is either secondary to presence of outer retinal deposits or RPE hyperplasia or nonperfusion of the layer in active stage[13]. Continuous progression of the lesion growth at the active edge showed increase in the areas of choriocapillaries flow void areas which is similar to clinically observed serpiginous pattern later forming large placoid/ geographic areas of choroiditis. Healing stage showed prominent underlying medium to large choroidal vessels among the flow void areas which represent it as a true loss of choriocapillaries and non perfusion and also localized tuft of vessels within which do not extend into the outer retina. These vessel tufts on multimodal imaging were isofluorescence on ICGA and stippled fluorescence on FFA with no dye leakage and on EDI OCT of corresponding area in active disease showed hyporreflective areas in thickened choriocapillaries which in healing stage showed atrophy of choriocapillaries layer [13]. The pathogenesis of such vascular changes may be linked to the development of ischemia in the inner choroid and surrounding milieu.

Figure 3: A case example of 50 year old female diagnosed with healed Serpiginous like choroiditis(SLC) in RE and active SLC in LE



* Color Fundus Photo (b) of RE showing healed chorioretinal scars. LE (a) showing active yellowish subretinal leisons in the posterior pole. Fundus autofluorescence FAF (c) of LE showing hypoautofluorescent lesions surrounded by hyperautofluorescence (yellow arrows). EDI OCT (d) showing thickened RPE and Choroid (yellow arrows) with multiple pockets of subretinal fluid . OCTA of deep Capillary plexus (e) showing hypointense cystoid spaces (yellow arrows) with flow void areas in outer retina and choriocapillaries (f) and (g). Corresponding Enface OCT showing void areas in all the three layers viz deep capillary plexus (h), outer retina (i) and choriocapillaries (i) (yellow arrows)

Idiopathic Multifocal Choroiditis

Idiopathic multifocal choroiditis (MFC) being a disease of unknown aetiology is an ocular inflammatory disease mainly affecting young aged female patients. Clinically, typically manifests as punched-out chorioretinal lesions in the posterior pole and peripheral retina with minimal vitreous reaction. The natural course being with recurrent episodes of inflammation leading to structural damage which on later resolution develops into chorioretinal atrophy, RPE pigmentary changes and scarring or resolves completely without any clinical sequelae. Major cause of potential vision loss in MFC includes choroidal neovascularisation.

A study[14] classified macular lesions secondary to multifocal choroiditis as active and inactive inflammatory lesion with or without choroidal neovascularisation based on multimodal imaging which includes Fundus photographs, FFA, FAF and OCT.

Inactive inflammatory lesions were defined as chronic stable yellowish or atrophic lesions clinically, hypoautofluorescent on FAF, RPE elevation with homogenous sub-RPE hyperreflective

material without RPE rupture and subretinal material on SDOCT and early hypofluorescence with late staining without late leakage on FFA. Atrophic lesions show RPE and outer retina atrophy on SDOCT with corresponding window defect without late leakage on FA.

Active inflammatory lesion presents as yellowish lesions while inflammatory CNVM is most commonly type 2. Both of them have similar features on conventional multimodal imaging like a ring of hyperautofluorescence surrounding hyperautofluroscent centre on FAF, RPE elevation with break in RPE with disruption of the ellipsoid and interdigitation zones and subretinal hyperreflective material deposition on SDOCT and early isofluorescence and late leakage on FFA.

OCTA helps in differentiating active inflammatory lesions from choroidal neovascular membranes noted with active and inactive lesions which is a diagnostic challenge. OCTA at the level of RPE (in outer retina) failed to demonstrate any flow in case of active lesions unlike CNVM which demonstrates highly organized dense flow in neovascular network in outer retinal and choriocapillaries segmentation with RPE dehiscence. They were also demonstrated along with inactive fibrotic scars which are known as quiescent CNV on OCTA which need close observation and no treatment as natural history is unknown[14]. Atrophic inactive lesions on OCTA demonstrated visualization of large choroidal vessels in choriocapillaries segmentation.

Bechet Disease

Bechet disease (BD) is a chronic multisystem disorder of unknown aetiology characterized by chronic non- granulomatous occlusive vasculitis with relapses and recurrences eventually leading to blindness. Classical ocular findings include mobile hypopyon, non-granulomatous pan uveitis, diffuse vitritis, occlusive vasculitis with retinal infiltrates, and optic disc edema. Inspite of being invasive procedure, FFA remains first line diagnostic tool in evaluating complications of Bechet's disease like peripheral retinal ischemia, neovascularization, cystoid macular edema, macular ischemia. It includes recurrent oral and genital ulcerations and also affects multiple organs including the heart, joints, and central nervous system.

OCTA demonstrates severe involvement of deep capillary plexus(DCP) as compared to superficial capillary plexus(SCP). A well delineated hypointense greyish area of retinal capillary nonperfusion/ hypoperfusion which are areas devoid of flow with capillary disorganization and telangiectasias formation noted secondary to ischemic process in both layers. DCP is more vulnerable to early ischemic process as it is the early intraretinal watershed zone between inner and outer circulation.[15] Deep capillary plexus demonstrates perifoveal capillary network disorganization like rarefied, dilated or shunting vessels. Inspite of no leakage on FFA and edema noted on SDOCT, early changes in the DCP suggestive of macular ischemia are noted suggestive of probable pathogenesis of Bechet's disease leading to progressive profound vision loss.

Certain cases also demonstrated cotton wool spots suggestive of retinal nerve fibre layer defects which are demonstrated as localised hypointense greyish area in both DCP and SCP when screened at the level of retinal nerve fibre layer defects. These usually demonstrate resolved retinal infiltrates or previous inflammatory attack of optic nerve.[15]

The flow void areas OCTA need to be differentiated from the grayish nonperfused areas which appear secondary to peripheral displacement of retinal capillaries owing to macular edema. Increase in FAZ is also noted in various studies in DCP and SCP layers in Bechet's disease mainly due to macular ischemia.

Choroidal Neovascular Membrane Secondary to Inflammation

Inflammatory choroidal neovascularization is a potential complication of uveitis often resulting in severe vision loss. In addition to VEGF, inflammatory mediators are widely thought to be involved in the development of choroidal neovascularization. FA has been the gold standard imaging modality in the diagnosis of choroidal neovascularisation. Fluorescein angiography demonstrates a hyperfluorescent area that leaks during the late phases of the angiogram. Chorioretinal inflammatory lesions can be confused with inflammatory choroidal neovascularization, since the leakage can be a hallmark of both lesion types.[16]But choroiditis lesions are generally hypofluorescent in early phase of FA and later become hyperfluorescent unlike CNVM which shows hyperfluorescence in early phase. Traditional OCT scans may reveal subretinal hyper-reflective tissue (non-specifically referred to as SHRM or subretinal hyperreflective material) that can be consistent with type 2 neovascularization, but this is not a universal finding of CNV activity and the lesion (SHRM) can represent other abnormalities such as subretinal fibrin or fibrosis. By contrast, 3X3 mm OCTA can identify a neovascular network in the subretinal (type 2) or sub RPE (type 1) compartment, confirming the presence of CNV. The area and the density of the inflammatory CNV can be quantified. It is possible that OCTA will provide a more precise and practical means of evaluating for inflammatory choroidal neovascularization versus dye-based modalities.[17]

Levison et al[18] in a prospective, descriptive case series of 12 patients including 7 patients with Punctate Inner Choridopathy (PIC), complicated by inflammatory choroidal neovascularization compared OCTA and conventional dye test. Inflammatory choroidal neovascularization was identified in 11 of the 12 patients (15eyes). OCTA detected inflammatory choroidal neovascularization even when the conventional dye test failed to clearly identify the neovascular lesion. The authors also described an associated ring of hyporeflectivity around the CNV as identified by OCTA suggestive of neovascular activity.

Cheng et al[19] investigated 52 eyes with Multifocal Choroiditis (MFC) .In their study, the authors detected 23 inflammatory choroidal neovascular membranes that on FA exhibited the classic appearance of early well defined hyperfluorescence with late leakage. OCTA demonstrated well-circumscribed vascular networks of different shapes in 20 of 23 cases. They also studied

inflammatory lesions on OCTA and found that among 34 lesions, 32 showed no blood flow signal in outer retina. They identified 3 cases of "quiescent CNV'' without active leakage on FA but OCTA revealed a blood flow signal and detailed microvascular identification of the CNV was noted. The authors hypothesized that these cases of "quiescent CNV" consisted of mature vessels associated with fibrotic tissue.

Zahid et al[20] retrospectively analysed the OCTA findings in 18 eyes with MFC. Eleven eyes in their series illustrated purely sub-retinal lesions that demonstrated neovascular flow on OCTA. Five eyes had mixed sub-RPE and subretinal lesions and OCTA flow was detected in all 5 cases, either active (1/5 eyes) or inactive (4/5 eyes). Their use of OCTA to detect the neovascular membrane resulted in a much higher frequency (83%). and in some cases it also detected membranes missed on conventional dye-based imaging (FA).They concluded that [12] OCTA demonstrated a higher sensitivity in detecting neovascular membranes in cases of MFC

Carnevali et al[21] studied twenty-two eyes of 20 patients with quiescent CNV. They described CNV shape on OCT-A as circular in 8 eyes and irregular in 10 eyes. CNV core was visible in 2 eyes. CNV margin was considered as well-defined in 15 eyes, and poorly-defined in 3 eyes. CNV margin showed small loops in 9 eyes and large loops in the other 6 eyes. CNV location was foveal-sparing in 12 eyes. In 4 out of 22 eyes it was not possible to classify the CNV "shape", "core", "margin", and "location" ether because the vascular network was not clearly shown (3 cases) or because it was not visible at all (1 case). In their study they found that the sensitivity and specificity of quiescent CNV detection by OCT-A turned out to be 81.8% and 100%, respectively.

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Guest Article

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Pachychoroid : The Disease Spectrum

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The term pachychoroid (pachy-[prefix]: thick) had been proposed as a term indicating an abnormal and permanent increase in choroidal thickness. It was employed first by Freund and workers to describe a new disease entity called pachychoroid pigment epitheliopathy and pachychoroid neovasculopathy.[1,2]

Spaide was the first to visualize the choroid utilizing spectral–domain ocular coherence tomography (SD-OCT) with the enhanced depth imaging (EDI) mode and Imamura et al related the choroidal findings in cases of central serous chorioretinopathy.[3]

Choroidal thickness is affected by age, refraction value, axial length, circadian rhythm and many other factors. While choroid thickness varies in many different studies, subfoveal choroid thickness is reported to range from 220 µm to 350 µm.[4,5] A thick choroid is defined as a choroid with a thickness of 390 µm and higher.

Abnormal choroidal blood flow leads to photoreceptor dysfunction and eventually, death. It has been shown to have vital roles in the pathophysiology of many diseases such as central serous chorioretinopathy (CSC), age-related macular degeneration (AMD), pathologic myopia, Vogt–Koyanagi–Harada (VKH) disease. ICG angiography is used to visualize the choroid. Optical coherence tomography, remains however, the most popular modality for the study of the choroid. Advanced features, such as Enhanced depth Imaging (EDI), OCT Angiography have helped evaluate the choroid in greater detail. Swept source OCT, a novel technology, allows for high resolution imaging from the vitreous to the choroid. Wide-field Imaging technology allows for extensive lateral extent of imaging permitting assessment of a large area of the choroid at once.

Diffuse choroidal thickening (thicker than 395 µm) and localized choroidal thickening was detected in relatives of patients with central serous chorioretinopathy. Evidence of isolated pachychoroid in all generations, suggested that pachychoroid could be a dominant inherited condition.[6]

The pachychoroid phenotype features are : 4,5

1) Reduced fundus tessellation on clinical examination or white light photography
2) Relatively increased choroidal thickness, which may be focal or diffuse;
3) pathological dilation of outer choroidal (Haller) vessels, referred to as “pachyvessels”; and
4) loss of choriocapillaris and Sattler layers overlying pachyvessels
5) choroidal hyper-permeability

Pachyvessel morphology - Normally in ICG angiography, choroidal veins disappear close to the macula, whereas in this disease group they may come in close proximity to the macula, often passing by it.

Pachychoroid diseases are a spectrum of 4 different disease groups1-5. These groups are essentially the stages of the disease itself, as the increased severity in the previous group leads the patient to the next group of disease.

The spectrum comprises the following 4 disease groups1-5:

1. Pachychoroid pigment epitheliopathy (PPE).
2. Central serous chorioretinopathy (CSC).
3. Pachychoroid neovasculopathy (PNV).
4. Polypoidal choroidal vasculopathy (PCV)

Pachychoroid Pigment Epitheliopathy (PPE).

Pachychoroid pigment epitheliopathy (PPE) refers to such changes that are believed to represent a forme fruste or precursor of CSCR.[1]

The characteristic findings of PPE are

- the presence of the pachychoroid phenotype,
- reduced fundus tessellation,
- a variety of RPE abnormalities -small RPE detachments (pigment epithelial
- detachment),
- absence of subretinal fluid,
- absence of subretinal fluid history, and
- absence of drusen.

The PPE may be isolated or may occur in the fellow eyes of patients with pachychoroid spectrum of diseases. It differs from CSCR in that subretinal fluid is not observed in this disease. 1 There are only changes in the retinal pigment epithelium to be found. 1 It is, therefore, frequently confused with other diseases such as age-related macular degeneration, pattern dystrophy, and pigment epithelitis.

The degenerative process may begin with RPE alterations before subretinal fluid accumulation. The PPE lesions, commonly seen above pachy-vessels, may be an indicator of photoreceptor apoptosis. 1

The pachyvessels, run in close proximity to the RPE-Bruch membrane complex, suggesting that they are mechanistically involved in inciting pigment epitheliopathy.1 Numerous, scattered small elevations of the RPE representing focal hyperplasia and sub-RPE drusen-like deposits are seen on OCT. Occasionally, small serous PEDs may be found. Swept source and EDI-OCT have also additionally shown - increased choroidal thickening, pachy-veins in Haller's layer, thinning in Sattler's layer and choriocapillaris and Outer Nuclear Layer thinning. Choroidal hyperpermability, demonstrated by mid-phase hyperfluorescence on Indocyanine green angiography, underlies these areas of RPE disturbances. On Fundus autofluorescence, areas of granular hypoautofluorescence and mixed stippled hyper- and hypoautofluorescence are seen while the signs of antecedent subretinal fluid, such as gravitational tracts, zonal areas of hyperautofluorescence or focal areas of speckled hyperautofluorescence are never seen in PPE. 1

Central Serous Chorioretinopathy (CSC)

Central serous chorioretinopathy, one of the most common of retinal disorders, is now being ascribed to the pachychoroid spectrum. It manifests as an idiopathic noninflammatory serous macular detachment, with or without serous pigment epithelial detachment (PED). It is the second stage of this pachychoroid disease spectrum. As is now being increasingly recognised, effusion under the retina occurs due to choroidal hyperpermeability. The predisposing factors are – having a Type A personality, hypochondria and hysteria, steroid use/ Cushing syndrome, pregnancy, systemic hypertension, Japanese/Southeast Asian racial characteristics, SLE, and psychopharmacological medication.Chronic or recurrent CSC can persist beyond the sixth and seventh decades with atrophic and/or neovascular sequelae. Progressive pathologic changes in the RPE result in ill-defined leakage.

OCT angiography may demonstrate pachy-veins in the Haller's layer. The Fundus fluorescein angiogram features of the ink-blot leak or the smoke-stack leak are well documented. Indocyanine green angiography typically shows dilated choroidal vessels that are diagonally oriented and that communicate with the vortex ampullae. In areas where FA shows leakage at the level of the RPE, ICGA shows choroidal hyperpermeability. (Fig 1a,1b) Fundus autofluorescence shows the chronic presence of fluid as focal areas of speckled hyperautofluorescence , and occasionally linear ,descending tear-drop tracts with a mottled fluorescence.

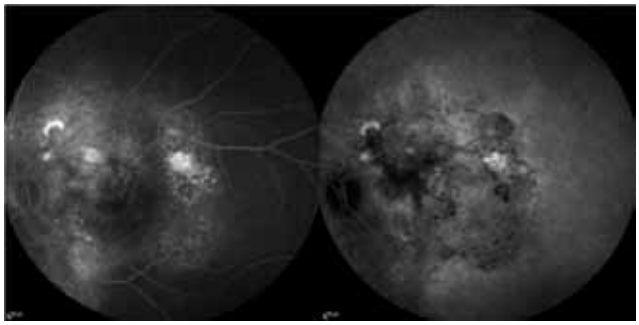

Figure 1a: FFA and ICGA images of a case of chronic CSCR showing the intense hyperfluorescence from leaking areas on the FFA with the corresponding choroidal hyperpermeable areas showing up as areas of hypercyannescence on the ICGA. A tear-drop tract is also evident.

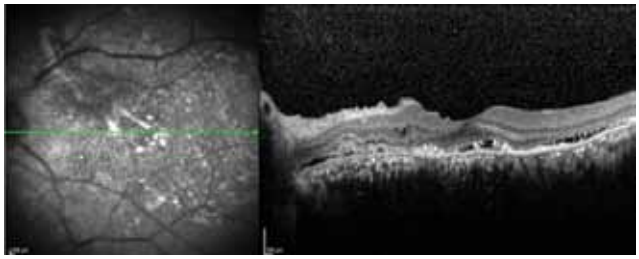

Figure 1b:The OCT shows the Chronic CSCR with diffuse shallow subretinal fluid with extensive subretinal precipitates and fibrin alongside a thickened choroid.

Pachychoroid Neovasculopathy (PNV)

Described by Freund and colleagues in 2015, it is the development of a type 1 CNV as a late complication of PPE and chronic CSC.2 In susceptible individuals, long-term effects on the RPE, Bruch's membrane and choriocapillaris induced by a hyperpermeable, chronically exuding pachychoroid may lead to the subsequent development of neovascularization of the pachy-veins at Haller's layer. Ayounger age at onset of neovascularization, a relative absence of drusen, and a thick choroid with pachyvessels distinguishes PNV from age-related macular degeneration and other degenerative conditions that predispose to type 1 neovascularization.2,4 As the neovascular tissue of PNV evolves, some eyes may develop polypoidal choroidal vasculopathy. OCT shows up its most important characteristic - the presence of broad shallow elevation of the RPE representing neovascular proliferation within Bruch's membrane. 2,4 This form of type 1 neovascularization is typically found overlying an area of localized choroidal thickening with dilated choroidal vessels (double layer sign on OCT). 2,4 OCT angiography highlights the complex, tangled vein network under these irregular shallow PEDs. FA shows an ill-defined hyperfluorescence and/or late leakage of indeterminate origin with a corresponding PED on cross-sectional structural OCT. Indocyanine green angiography often reveals both mid-phase patchy areas of choroidal hyperpermability and a discrete plaque of late hyperfluorescence corresponding to type 1 neovascular tissue. 2 (Fig 2a,2b)

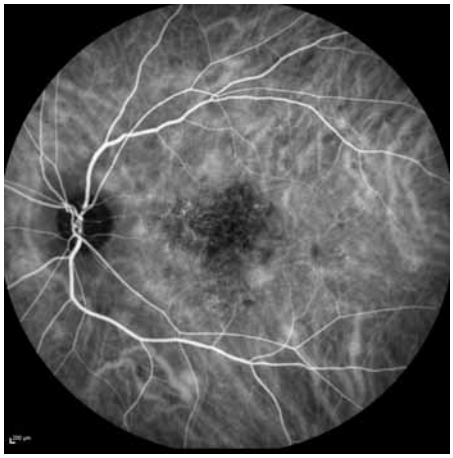

Figure 2a: Pachychoroid neovasculopathy (PCN) with areas of distinctive choroidal hyperpermeability seen as hypercyanescent zones on the ICGA

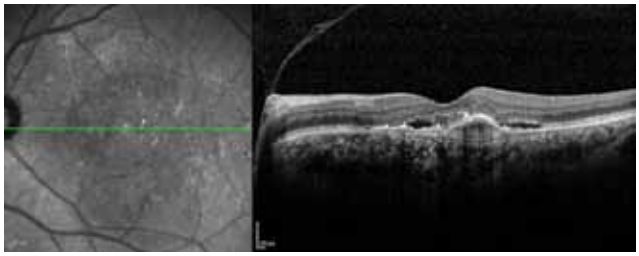

Figure 2b: OCT image of the PCN case with elevated RPE resembling a type I CNVM. A thickened choroid is also noted.

Polypoidal Choroidal Vasculopathy (PCV)

Yannuzzi and colleagues assert that 7.8% of the exudative AMD cases are PCV while studies from Asia place that incidence in the range 25-50%.7-9 Choroidal capillary proliferation under the retinal pigment epithelium with terminal aneurysmal development in the form of polyps is the hallmark.10 These choroidal vascular polyps, the pathognomonic lesions, are clearly diagnosed by ICG angiography. The polyps are easily identified on ICGA as early focal areas of intense hyperfluorescence that may show either late leakage or "wash-out" appearance depending on their degree of activity. 10(Fig 3a,3b) These polypoidal structures cause leakage or hemorrhage beneath the RPE and neurosensory retina.

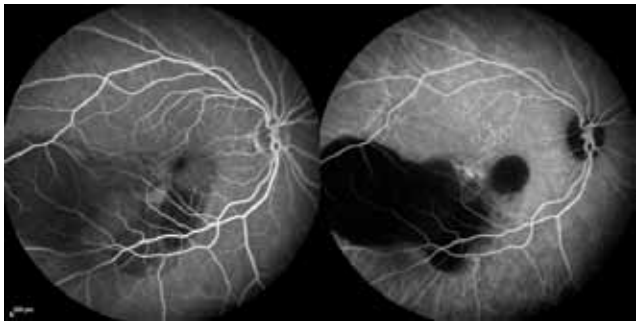

Figure 3a: A case of Polypoidal choroidal vasculopathy (PCV) with massive subretinal haemorrhage seen as blocked fluorescence on FFA and ICGA. The ICGA additionally shows the branching vascular network (BVN) terminating in the classic polyps seen as the hypercyanescent bulbs.

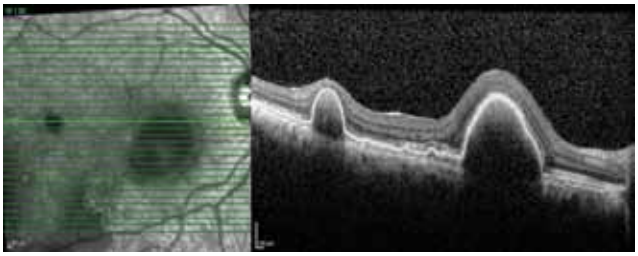

Figure 3b: The tall peaked, thumb shaped PEDs typical of PCV are noted on OCT with the thickened choroid.

The spectrum of clinical lesions range from serosanguinous PEDs and neurosensory detachments to bullous retinal detachments and vitreous hemorrhages. 10 The OCT features of PCV cause it to be likened to a form of type 1 neovascularization and it remains a close differential to neovascular AMD.11 In patients with type 1 CNV secondary to CSC or pachychoroid pigment epitheliopathy (pachychoroid neovasculopathy), this neovascularization becomes polypoidal, and lipid and hemorrhagic leakage occurs through these polyps. Acloser examination of the choroids of many patients diagnosed with PCV reveals features consistent with those of the pachychoroid phenotype: minimal or absent drusen, thicker choroids than are typical for AMD, pachyvessels with inner choroid attenuation, and shallow irregular PEDs.1,2,4

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Case Report

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Acanthamoeba Keratitis Masquerading as Dendritic Keratitis

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A 23 year old male patient presented with complaints of redness, pain, photo phobia and foreign sensation in the right eye since 10 weeks. He was diagnosed as Right Eye Herpes keratitis elsewhere and was already on Acyclovir eye ointment and FML eye drops for 9 weeks. The patient did not give us any history of trauma,steroid usage and contact lens usage.

On examination, his best corrected visual acuity was 20/40 in the RE and 20/20 in the LE.

Slit lamp examination of the right eye revealed congested conjunctiva. Cornea showed dendritic lesion on the epithelium which stained positive with fluorescein (Figure 1).

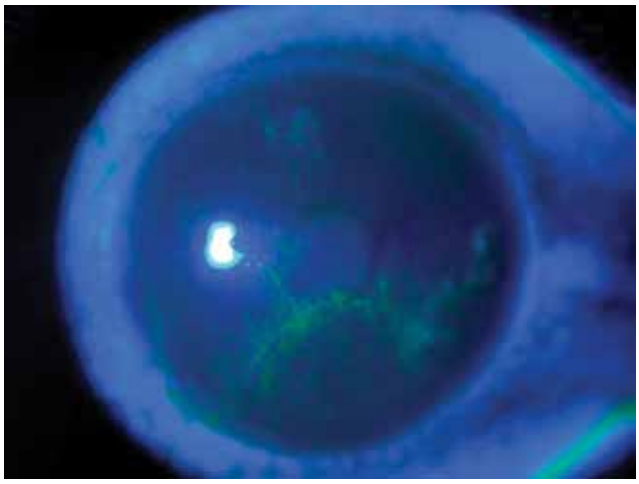


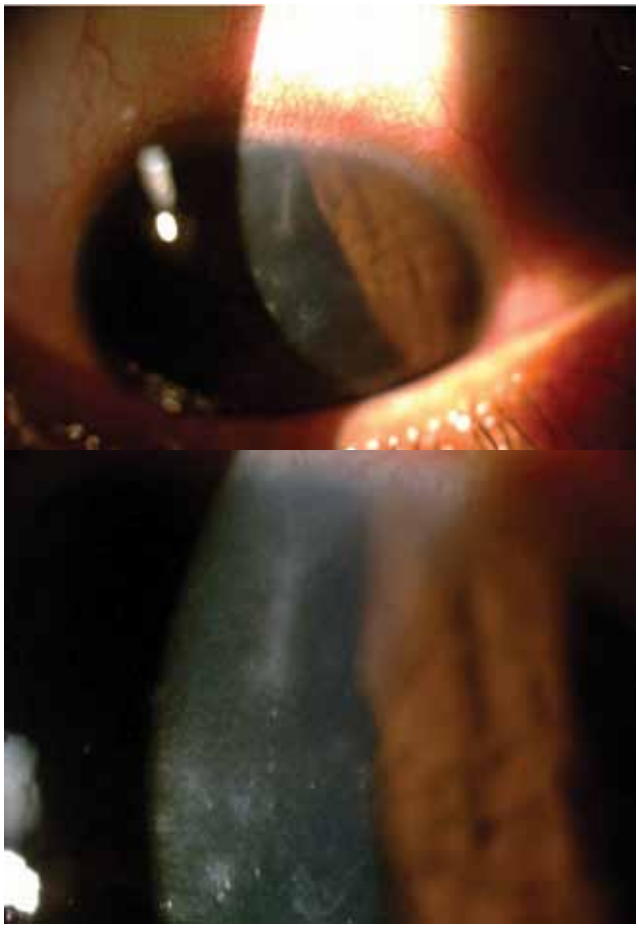
Figure 1: Initial presentation with a dendritic epithelial lesion.

The epithelium around the dendrite also showed a lot of diffuse punctate staining. Anterior chamber was quiet.Pupil was normal size and reacting to light. Lens was clear, Fundus was normal. IOP was 12mm hg. Left eye was within normal limits. With a presumed clinical diagnosis of chronic herpetic epithelial keratitis, the patient was started on oral acyclovir 400 mg 5 times a day in addition to topical antiviral drugs. The patient came back for review after 10 days.There was no improvement. In fact, the dendritic lesion became elevated, larger and involving a different area of the corneal epithelium (Figure 2).

Figure 2: Subsequent presentation as more extensive elevated dendrite.

The unhealthy epithelium was debrided and a Bandage Contact Lens(BCL) was placed. Oral antiviral treatment was continued along with topical antibiotics and lubricants. The patient was reviewed after 1 week.

The patient was symptomatically better but presented with radial keratoneuritis(Figure 3) and spotty stromal infiltrate which made us suspect acanthamoeba keratitis.



Figures 3a & 3b: Final presentation with radial keratoneuritis with nummular stromal infiltrates.

Corneal scrapings were taken for microbiologywhich showed acanthamebadouble walled cysts on smear examination. The patient was placed on PHMB 0.02% eye drops and chlorhexidine 0.2% eye drops every hour.After 1 week the patient was symptomatically much better,the size of the corneal infiltrates decreased in size and keratoneuritis subsided.After 6 weeks of medical treatment the infiltrate completely subsided and the patient had a mild residualnebular corneal scar (Figure 4).

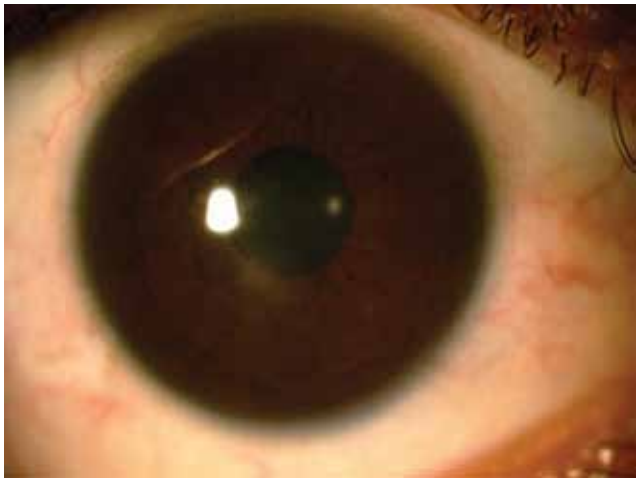


Figure 4: Resolved acanthamoeba keratitis with nebular scar.

We subsequently asked the patient regarding history of trauma, contact lens wear,swimming in pools: he gave history of taking holy bath in a pond a week prior to onset of symptoms, suggesting the possible source of infection.

Discussion

Acanthameba are free living amoebae causing human infections.Two of the eight known species of Acanthamoeba, A. castellanii and A.polyphaga, are responsible for most of the infections. Acanthamoeba keratitis is a sight-threatening disease that carries a favorable prognosis when diagnosed and treated early in the disease course. The three major risk factors for Acanthamoeba keratitis are contact lens use, exposure to contaminated water, and corneal trauma[1].

The initial symptoms of Acanthamoeba keratitis include photophobia, tearing, and pain[2].The pain is typically very severe, seeming to be disproportionate to the signs[3] The early signs include pseudo-dendritic epithelial lesion,[4,5] radial keratoneuritis,[6]limbitis, along with the finding of decreased corneal sensation.[7] Therefore, herpes simplex keratitis is the most common misdiagnosis because of the similarity of its early signs to Acanthamoeba keratitis.[8,9]Misdiagnosis may delay the appropriate treatment. The ring corneal infiltrate, which is the hallmark of the disease, may then appear, but it is a relatively late sign.[10]

Clinical suspicion is the first and most vital step in managing Acanthamoeba.Acanthamoeba trophozoites or cysts can be demonstrated with corneal scrapings via wet mount, stains, histopathologic examination, or culture. Corneal scrapings are stained with calcofluor white and specimens are examined with fluorescent microscopy . Amoebae may be cultured on non-nutrient agar (NNA) overlaid with organisms such as Escherichia coli.Confocal microscopy has recently become a powerful tool for rapid diagnosis of the infection in vivo. The advantage of in-vivo microscopy is that biopsy is not needed and the diagnosis is instantaneous on observation of the round hyper-reflective lesions of amoeba cysts.

Successful treatment of keratitis consists of early diagnosis and aggressive medical therapy. Topical therapies are a Biguanide (polyhexamethylenebiguanide PHMB 0.02% or chlorhexidine 0.02%) in combination with a diamidine (propamidineisoethanoate 0.1% or hexamidine 0.1%). These topical antimicrobials are administered every hour immediately after corneal debridement or for the first several days of therapy. These agents are then continued hourly during waking hours for 3 days depending on clinical response. Two weeks may be required before a response is observed, and the total duration of therapy is a minimum of 3-4 weeks. Some advocate treating for 6-12 months. When therapy is discontinued, close observation is warranted to rule out recurrent disease.

A medical cure can be achieved in Acanthamoeba keratitis using specific anti-amoebal agents, but the cornea often remains scarred and requires optical penetrating keratoplasty for visual restoration.Much evidence pointed out that successful recovery of excellent vision depended mainly on early diagnosis and appropriate treatment.[11]

Our patient was unique as the dendrite like presentation was quite "viral" in its appearance and persisted for several weeks after the initial presentation. Also, interestingly the clinical presentation was changing in serial visits, including the configuration and location of the epithelial dendrite. Finally, the appearance of the pathognomic sign of Radial Keratoneuritis helped to clinch the diagnosis that was confirmed microbiologically. Prompt initiation of anti-acanthamoeba treatment helped bring relief to the patient and led to resolution of the infection.

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Case Report

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A Rare Case of Conjunctival Stones: A Mystery or Mislead?

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Background

Many medical mysteries can be fascinating. Seeing a woman cry stones or crystals is baffling to many people, while doctors are left speechless.

Case Report

An 8-year-old female child complained of irritation in the left eye while crying stones& threads for the last 2 months. She was asymptomatic till 2 months prior to presentation. There were tears too while crying. Most of events happened in afternoon and evening, never when she was asleep. It was supposedly very painful. There were no similar complaints in the family.

Slit-lamp examination revealed no abnormality. As per the history given, the conjunctival cul-de-sac became filled with innumerable such stones of different sizes ranging from 2 mm to 5 cm and different colours associated with threads (Figure 1). The stones were hard in consistency. The chemical analysis of stones showed these to be composed of phosphate. Hematological and biochemical investigations revealed no abnormality.

Figure 1: Stones & thread that supposedly came from child's eye brought by her family

She subsequently visited the OPD with stones in situ (Figure 2). There was no explanation as to why she produces the stones. There is no known disease or condition of which the stones are as a symptom.

Figure 2: Child presenting with conjunctival stones at different visits

Conjunctival concretions (conjunctival lithiasis)

These are small vascular, granular, yellowish-white deposits produced due to conjunctival degeneration and are found in the sub-epithelium of palpebral conjunctiva and fornix (junction between palpebral and bulbar conjunctiva) in the elderly or in patients with chronic inflammatory conditions[1-3].

We then recorded a video of a stone being extricated from her temporal conjunctiva in the OPD (Figure 3). Video is posted in the TOS website www.tsos.co.in.

Figure 3: A still from a video demonstrating the removal of a conjunctival stone from the eye

Discussion, Conjunctival concretions (Conjunctival lithiasis)

Conjunctival concretionsare described in literature as small vascular, granular, yellowish-white deposits produced due to conjunctival degeneration; these are found in the sub-epithelium of palpebral conjunctiva and fornix in the elderly or in patients with chronic inflammatory conditions1-3.

Deposits have been found in patients as young as 20 years old, with the usual age range 30-80 years. They appear as multiple tiny inclusion cysts containing yellowish-white deposits of inspissated mucous and degenerated epithelial debris including keratin. They are frequently discrete, but confluent concretions are not uncommon.

In one study, no significant difference was found between the localisation on the upper and lower eyelids, right or left eye, and most of the concretions were superficial and hard, and mainly single. Associated dysfunction of meibomian glands is also noticed in some patients. They are usually idiopathic, but they have also been associated with:

1. Chronic atopic keratoconjunctivitis
2. Following post-trachomatous degeneration
3. Sulphadiazine eye drop administration

Conclusion

It's not implausible that the patient in this case could plant stones in her eyes and reveal them at an appropriate time. It's worrisome that people are attributing the claim as true without a medical exam, and, worse, ascribe it to some superstitious cause.

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Role of Ocular Imaging Biomarkers in Diabetic Retinopathy (DR)

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A biomarker can be defined as a characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacologic responses to a therapeutic intervention. Biomarkers could be useful (i) to detect early disease, (ii) to identify diabetic patients most prone to progressive worsening, in whom intensified therapy could be prioritized, (iii) to monitor the effectiveness of new drugs for DR before advanced DR stages have developed and (iv) to prognostigate the visual recovery following treatment of DR.

In the present article, we enumerate various ocular imaging based biomarkers of relevance in DR.

Ocular Imaging based biomarkers in DR

1. Fundus image based Classification of DR
2. Retinal Blood flow changes
3. Oxygen saturation in retinal vasculature
4. Retinal vessel calibre
5. Retinal vessel geometry
6. Neural retina assessments: electroretinograms
7. Ocular coherence tomography (OCT)

Ocular-Imaging based biomarkers in DR

1. Fundus Imaging based classification of DR

The absence or presence, type, and severity of retinal vessel lesions diagnosed by ophthalmoscopy or by mydriatic or non-mydriatic retinal photography are biomarkers of diabetic retinopathy status. These markers are used in routine clinical practice and in research [1]. Diabetic retinopathy may be asymptomatic for years, even at an advanced stage, so screening is essential to identify, monitor, and guide the treatment of retinopathy. New wide-angle imaging systems using scanning laser ophthalmoscopes can visualize up to 200 degrees (82%) of the retina; this improves coverage of the mid and peripheral retina and offers improved prognostic value in diabetic retinopathy over conventional 7-field ETDRS photography [2]. Figure 1 shows the clinical photos based on International Clinical Diabetic Retinopathy (DR) Disease Severity Scale. Figure 2 shows the clinical photos based on the international clinical classification of Diabetic Macular edema (DME).(3)

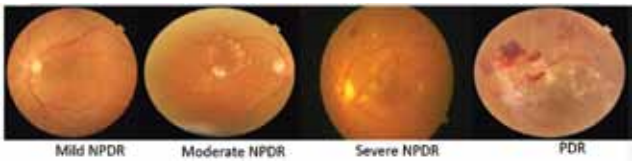

Figure 1: International Clinical Diabetic Retinopathy (DR) Disease Severity Scale

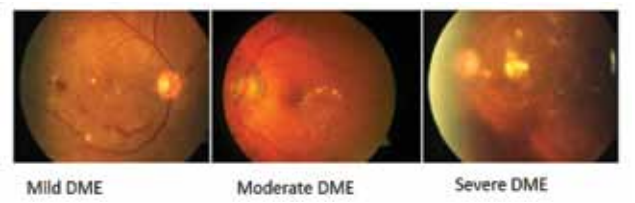

Figure 2: International Clinical Classification of Diabetic Macular Edema (DME)

2. Retinal Blood flow changes

Kohner suggested that blood flow abnormalities were pathological, and an early biomarker for progression of diabetic retinopathy [4]. Using more modern techniques, such as Doppler flow velocity waveform analysis, even earlier changes in blood flow have been identified, prior to the onset of clinically overt retinopathy, and even in prediabetic (impaired glucose tolerance) subjects . Later in the retinal disease process, retinal arterioles dilate, which causes increased blood flow [5] and accelerated progression to diabetic macular edema (DME) and PDR [6].

3. Oxygen saturation in retinal vasculature

Oxygenation of blood in retinal vessels can also be quantified non-invasively based on the differential light absorbance of oxyhemoglobin and deoxyhaemoglobin . Oxygen saturation has been found to be higher in retinal venules of diabetic than non-diabetic subjects [7] and to decline further with increasing severity of diabetic retinopathy [7]. This may relate to loss of metabolically active retina, anatomical damage to retinal vessels, and arterio-venous shunt formation in the retina, all of which reducing oxygen consumption

4. Retinal vessel calibre

Retinal arterial and venous caliber and their ratio, even in the presence of an apparently normal retina, are potential

early biomarkers of subsequent risk for diabetic retinopathy, and also for diabetic nephropathy [8]. Relationships between retinal vessel characteristics such as retinal venule caliber were reported as a biomarker of subsequent vision loss 30 years ago [9]. Both cross-sectional [10] and prospective studies [11] showed strong evidence of venular widening to be associated with diabetic retinopathy.

5. Retinal vessel geometry.

Greater retinal arteriole tortuosity was independently associated with retinopathy and early stage of nephropathy , cross-sectional study of young type 1 diabetes patients [12]. In longitudinal studies in a pediatric cohort of type 1 diabetes patients, greater simple tortuosity and lower arteriolar length to diameter ratio were independently associated with incident retinopathy [13].

6. Neural retina assessments: electroretinograms and visual evoked responses

There is general agreement that both neural and vascular changes are linked with diabetic retinopathy, but there has been controversy as to which occurs first. Availability and sensitivity of the tools used to assess retinal vascular and neural structure and function are important to answer this question. The links between retinal-neural and retinal-vascular function and structure, and the changes in diabetes, are not yet fully understood. Retinal glial cells are involved in the interaction between vascular, neural, and retinal compartments. Müller cells that surround retinal blood vessels are assumed to modulate vascular permeabilty, blood flow, and vascular cell survival [14-15].

ERG involves electrical stimulation, and has several subtypes, including flash and multifocal ERGs. Using these tools, differences have been identified in (neural) retinal function and at different disease stages between subjects with and those without diabetes, with the abnormality increasing with severity of the (more readily classified) vascular stages of retinopathy. Abnormal ERG can be present in diabetic subjects even in the setting of apparently normal retina [15].

In cross-sectional studies, ERG changes have been noted in type 1 diabetes patients with a disease duration of only one year [16]. In longitudinal, clinical, observational studies, multifocal ERG-readings predicted the location of new retinopathy that developed 1-3 years later [17]. Therefore, neurodegeneration could be a useful biomarker to predict the future development of microvascular damage in the diabetic retina. A portable ERG device has recently been released for non-mydriatic use in the primary care setting for the detection of late-stage diabetic retinopathy .

7. OCT imaging based biomarkers

Ocular coherence tomography (OCT). The OCT is another clinical biomarker that is now widely used in clinical practice. OCT provides images of the multiple retinal layers, and measures the thickness of the various layers. In diabetes-related retinal-neural degeneration, retinal ganglion cells and nerve fiber layers are thinned. The OCT is also very useful in identifying and quantifying retinal edema, including macular edema, which can be difficult to detect by retinal photography or fundoscopy. Abnormalities in OCT can be detected even in diabetic patients with a normal fundoscopic examination [15].

In a recent systematic review including 14 studies of OCT in type 1 and type 2 diabetes patients, retinal neurodegenerative changes were noted, even in the absence of diabetic retinopathy. Several layers in the retina and the mean retinal nerve fiber layer around the optic nerve head were significantly thinner in people with type 2 diabetes than in non-diabetic subjects. In type 1 diabetes patients with no retinopathy, the OCT was normal, but abnormal in those with retinopathy [18].

In a recent study in the Veterans Affairs Diabetes Trial including adults with type 2 diabetes and current or prior diabetic macular edema, early (4-month) changes in the disorganization of the retinal inner layer (DRIL) were detected by OCT (Figure 3), and were independent predictors of subsequent changes in visual acuity in a 1-year follow-up [19].

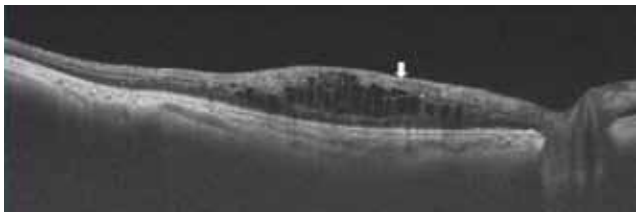

Figure 3 : Disorganization of the retinal inner layer (DRIL)

DRIL is a novel, noninvasive parameter that appears to be highly correlated with VA in eyes with either current or resolved DME. Early changes in DRIL extent are also predictive of longer-term VA outcomes in eyes with baseline DME.. These findings are important because a reliable biomarker of VA in patients with DME has yet to be firmly established. If foveal DRIL is validated in future studies as a marker of VA outcomes, its assessment would allow more effective estimation of VA potential, thus directly affecting patient counselling, disease management, and subject selection for DME clinical trials. Below picture of OCT describes the prognosis of Diabetic macular edema based on the various OCT presentation.

External limiting membrane disruption:

The external limiting membrane (ELM) is a structure that separates the inner segments from the outer nuclear layer, where the Müller cells are joined to the photoreceptor cells. The ELM serves as a skeleton to keep the photoreceptors

aligned. The ELM has been hypothesized to maintain a protein balance between the photoreceptor layer (ISL) and the outer nuclear layer (ONL). Recently, several studies have shown that ELM interruptions visible on spectral-domain optical coherence tomography (SD-OCT) are associated with lower visual acuity outcome in patients with clinically significant DME (CSME).(20) Possibly this is because the integrity of the ELM has a critical role in restoration of the photoreceptor microstructures and alignment.

ELM status is likely be as closely related to VA as is the IS/OS status in DMO and it may be useful in the evaluation of prognosis for the treatment of DMO(21). (Figure 4)

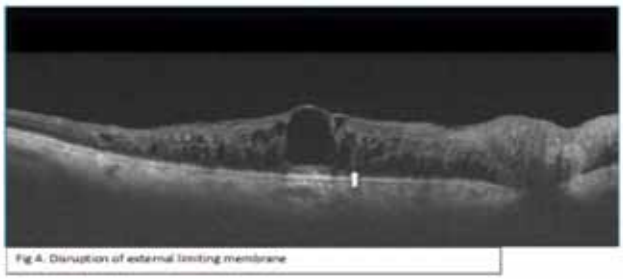

Figure 4: Disruption of External Limiting Membrane

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Challenges in Retinopathy of Prematurity Screening Programme in India

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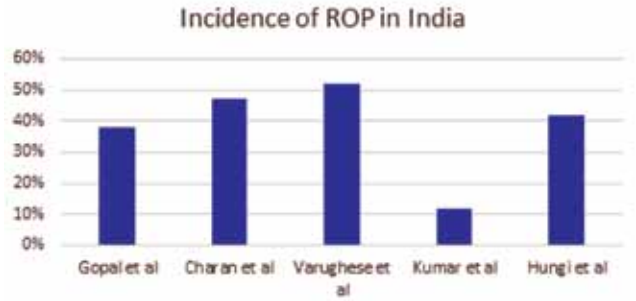
Abstract

Retinopathy of prematurity (ROP) remains one of the leading causes of childhood blindness worldwide. The level of neonatal care is improving due to which more pre-term babies are surviving. Unless adequate facilities for screening and treatment of ROP are provided, the visual impairment due to ROP is likely to increase continuously. A multi-disciplinary team approach is essential for an effective ROP screening programme. It involves good coordination between several team members including neonatologists, ophthalmologists, nurses, health-care workers and parents. For a successful program, every person in the team has a crucial role to play. There are many challenging situations that one has to face in these programmes. These situations and possible solutions to overcome them need to be analysed, which helps in expanding the ROP screening programmes both in quality and quantity all over the country.

Key words: challenges, retinopathy of prematurity, screening

Retinopathy of Prematurity (ROP) remains one of the leading causes of childhood blindness worldwide.[1] The world is currently witnessing the third epidemic of ROP[1,2] and it is emerging as a major public health concern in low and middle-income countries including India.[2] Approximately 15 million babies are born preterm (<37 weeks by definition) worldwide each year and India has the highest number of preterm births.[3] The current third epidemic is due to increasing number and survival of preterm births in the developing world as a result of better Obstetric and Neonatal services.[1]

Over 60% of preterm births occur in Africa and South Asia. India is among the 10 countries with the highest numbers of preterm births in the world.[4] In 2010, there was an estimate of 3,519,100 preterm births in India. India accounted for nearly 10% of the worldwide estimate of blindness and visual impairment due to ROP.[5] The incidence of ROP in India varies between 11.9 to 51.8% in studies done between 1992-2008 at various centres.[6-10]



The level of neonatal care in India is improving due to the establishment of number of Neonatal Intensive Care Units (NICU) and Special Newborn Care Units (SNCU) in both the public and private healthcare sectors. Unless adequate facilities for screening and treatment of ROP are provided, the visual impairment or blindness due to ROP is likely to increase continuously. ROP related blindness can be prevented only by rigorous screening programmes to cover all the 'at risk' babies. Multidisciplinary Team approach is essential for an effective ROP screening programme. It involves good coordination between several team members including Obstetricians, Neonatologists/ Paediatricians, ROP nurses, Ophthalmologists, Health care workers and Parents. For a successful programme every person in the team has a crucial role to play. Therefore, there are many challenging situations that one will face. The various challenges are discussed below with some possible solutions to overcome them.

1. Existing scenario in NICU/SNCUs – ROP screening

Challenges: Under the Indian Newborn Action Plan (INAP) - 2014 a nationwide network of facility-based newborn care has been established at various levels. 548 Special Newborn Care Units (SNCUs) for sick and small newborns have been established at district/sub-district hospitals or medical colleges. This number of SNCUs is increasing. Many neonatal care units have come up even in the private sector leading to better survival of these small babies, thus increasing the need for ROP screening. It is very alarming to find that there are many NICU/SNCUs which do not have ROP screening facility. The guidelines from the National Neonatology Forum (NNF) for the accreditation of level 2 neonatal unit mention "mandatory" and "essential" criteria.[11] The availability of ophthalmologist for ROP screening is included under essential criteria (but not mandatory) and therefore many level 2 units get accreditation without having these facilities.

Azad et al[12]reported that out of the 115 babies who presented with stage 5 ROP the vast majority (109 babies) had never been screened for ROP. Similar findings have been reported in another study of 66 cases of stage 5 ROP in India.[13] This shows that there is lack of screening in many places. In an Indian study by Sathiamohanraj et al[14], it was noted that out of 83 paediatricians only 54 were aware of ROP, 33 knew that ROP is preventable, and 38 did not know when ROP screening should start. Another Indian study done by Patwardhan and colleagues also found

that only 58% of the interviewed paediatricians always referred premature infants for ROP screening although all of them were aware of it.[15] Non-availability of any trained ophthalmologist in nearby areas (41% of those not screening) or absence of ophthalmologists affiliated to hospitals with inpatient care (23%) were the major deterrents in effective screening of predisposed neonates was found in this study. A similar study by Kemper et al, also reported non-availability of a trained ophthalmologists as major hindrance in ROP screening.[16]

Possible solutions: For escalating the screening programmes to every neonatal unit in India the NNF accreditation guidelines for level 2 units must include the availability of Ophthalmologist for ROP in the mandatory item list.[17]Studies have shown there is some lack of awareness and motivation among paediatricians regarding ROP screening. This gap can be filled by conducting CMEs and workshops on ROP. Training and awareness programmes may be conducted for both paediatricians and ophthalmologists together and improve the rapport among them. Training of ophthalmologists in ROP screening also must be made mandatory in postgraduate and fellowship programmes. In case of non-availability of ophthalmologist or other facilities the unit may take the help of other institutes/organisations who can provide these services. Public-private partnerships may also be encouraged.

2. Identification of babies who need ROP screening

Challenges: Sometimes an 'at risk' premature baby may not be screened or the screening may be delayed. It is very important to screen all the babies at risk and not to miss even a single baby who meet the criteria mentioned in the National Guidelines. In a pilot survey of the prevailing clinical practices regarding screening for ROP among paediatricians in India it was found that only 14.5% followed standard guidelines.[15]The American Academy of Pediatrics recommends ROP screening for babies ≤1500 g birth weight or ≤30 weeks gestational age and those infants >1500 g or >30 weeks with an unstable clinical course or at high risk for ROP.[18] As per the Canadian guidelines, ROP screening is recommended for GA of ≤30 weeks or birthweight ≤1250 g [19] and the United Kingdom recommends screening for <32 weeks or <1501 g.[20] Due to the variation in the standard of neonatal care in low and middle- income countries, larger and more mature babies are at risk of developing sight-threatening ROP.[1] So, the guidelines mentioned above, followed in the high-income countries with relatively uniform high level of neonatal care cannot be followed in India. It is imperative to be aware of the National Guidelines to be followed. As these babies are not born with ROP and the disease sets in only after few days, the timing of the 1st screening is also important as early screening will be of no use and delay in screening and treatment may lead to blindness, especially in Aggressive Posterior ROP.

Possible solutions: The Neonatologist/Paediatrician, Ophthalmologist and ROP Nurse of the unit should be well versed about when to screen and whom to screen. The NNF recommends that all infants weighing ≤1750g at birth and/or born at < 34weeks gestation should be screened for ROP. Infants with birth weight of 1750-2000g or gestation age of 34-36 weeks should also be screened if they have risk factors like ventilation, prolonged oxygen therapy, hemodynamic instability or adverse respiratory or cardiac disease.[21]

Once a baby meeting the above mentioned criteria is admitted the details and the date of 1st screening should be documented in 'ROP Diary'. Maintaining an 'ROP Diary' plays a vital role in ensuring screening of every 'at risk' baby in the unit. Other methods like low cost-red alert cards attached to cots/cradles of the babies requiring screening as shown in REDROP study by Vinekar and colleagues can be implemented to increase the enrolment for screening.[22] Coloured wrist bands or stickers on the files may also be used.

To avoid inconsistencies, ambiguity, and confusion about the timing of first screening Jalali S et al [23]recommended to complete one ROP screening session definitely before 'Day 30' of life and by 'Day 20' of life in smaller babies (possibly less than 30 weeks and/or birth weight less than 1200gms). This day 30 and day 20 strategy helps in the compliance of timing of screening as date of birth is well known to all and easy to be followed by all the care providers. Once a baby requiring screening is identified it is the responsibility of the Neonatologist/Paediatrician and ROP Nurse to inform and explain the parents about the need and procedure of screening. The date of the 1st screening should also be informed.

3. Examination

Challenges: Apart from Neonatologists and Ophthalmologists, ROP Nurses play a very crucial role in the screening programmes. The ROP Nurses help in preparing and monitoring the vitals during examination. The first and foremost step in preparing the baby for screening is the dilution of the dilating drops which is followed by administration. Knowledge about the dilution and proper administration is very important as errors in dilution and administration of dilating drops may even be fatal. During the examination, pain and infection control, monitoring of the vitals are other important issues to be taken care of.

Possible solutions: ROP nurse should be well versed with the concentration to be used and should be familiar with the steps of dilution. To prevent any errors in dilution one may paste charts with the steps of dilution in the unit. These charts will also help the other nurses in diluting the drops when the regular ROP nurse is unavailable. We can use measures like hand washing, using sterile instruments, sucrose solution, proper swaddling, pacifiers, etc for comfort of the baby and control of pain and infection.(figure 1) (figure 2).

Figure 1: Sterile speculum and Vectis for examination

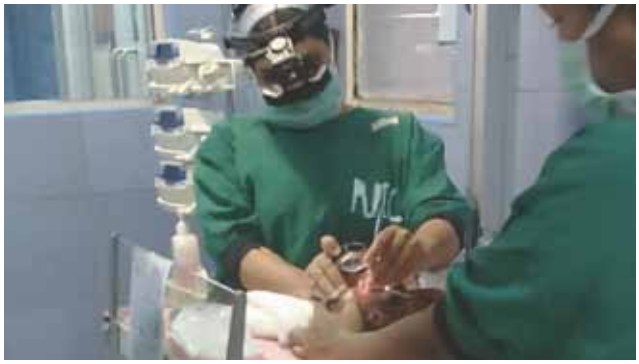


Figure 2: Examining baby in NICU with all aseptic precautions

It is always advisable to perform the screening under the supervision of Neonatologist/paediatrician, to prevent any untoward events. If the babies are screened in the Ophthalmology clinic it should be equipped with basic supports like pulse oximeter, suction apparatus and oxygen. One must also ensure that the babies are kept in a warm environment with facilities for feeding. Above all, every person handling these babies should be gentle and have patience.

4. Documentation and Counselling

Challenge: The clinical work is often done with perfection but we fail to document it properly. We are also not particular about informing and counselling parents about ROP and its management. Lack of this documentation and informed consent leads to unmet expectations from parents and causes medico-legal tangles.

Possible solutions: However, nowadays documentation and communication has improved in many hospitals due to implementing standards and third-party accreditations like NABH, JCI etc. Parents should be educated about the disease, its risk factors, chances of progression, treatment options available etc. The counselling will be more effective when done in simple and local language and also by showing retinal images with ROP. The date of next follow-up and the need for regular follow-up also should be informed. Informative and attractive pamphlets may also be given. A good counselling helps in improving the compliance and the follow-up rate.

5. Follow-up

Challenges: The most common challenge faced in the ROP screening program is lack of compliance and follow-up. As ROP is a disease which requires multiple visits to discharge them from the screening program, lack of follow-up can lead to progression of disease and blindness, which is very much preventable. The reasons for reduced compliance are; lack of knowledge about the disease and importance of frequent follow-ups, lack of proper communication to the parents and long distances between their home and the units especially for those who are from rural areas.

Possible solutions: The attrition of follow-ups can be reduced by proper counselling of the parents about the disease, its chances of progression and need for regular follow-up visits. The date of scheduled screening should be clearly mentioned in the ROP cards. A proper counselling and reminder by the neonatologist will have a better impact as the parents are most confident on them because they are the ones due to whose efforts, their babies survive. ROP nurses also play a major role as they have intimate relation with the babies and parents. In rural areas, Accredited Social Health Activist (ASHA) workers play a pivotal role by improving the compliance for screening and also in regular follow-up visits. They also play important role in prevention of ROP by educating the mothers about the importance of breast feeding, kangaroo mother care and practises to prevent infection. Increasing awareness about ROP through print and electronic media and other advocacy activities help in improving the compliance and preventing blindness due to ROP. As every individual in the country has some knowledge about vaccinations in a new-born, measures must be taken to create similar awareness about ROP such that parents themselves ask for screening in their babies if born pre-term.

6. Availability of infrastructure and trained personnel

Challenges: ROP screening requires a committed Ophthalmologist with special training to detect ROP and also to treat. The most important bottleneck in the ROP screening programme is the availability of Ophthalmologist who is skilled to detect and treat ROP. Most of the Neonatal units in India do not have ROP screening due to non-availability of skilled Ophthalmologist. As ROP was not included in the curriculum we do not have trained Ophthalmologists skilled in ROP screening.

Possible solutions: We can overcome this gap in near future by giving good exposure and hands-on training to all the postgraduate students. ROP screening should be included in the postgraduate curriculum, regular postings should be given. If the unit does not have ROP screening programme, it must be made compulsory to depute them for few weeks to any unit where the facility is available. Even in undergraduate training ROP should be included in Public Health and each and every graduated doctor should be aware of this preventable cause of childhood blindness. ROP programmes must be made mandatory in institutes offering Retina Fellowship programmes. More workshops and academic meetings need to be held for escalating the ROP programmes(Figure 3). Regular training activities must be conducted for the Neonatologists and ROP nurses about screening programmes and in better neonatal care practices which help to prevent ROP.

Figure 3: CME (MISSION ROP) conducted for paediatricians and ophthalmologists on World Prematurity Day.

All the infrastructure required for screening programme like dilating drops, speculum, depressors and indirect Ophthalmoscopes must be made available in all the Neonatal units. Portable Laser machines also need to be available at least at the District headquarters hospitals or Medical college NICU/ SNCU which can cover the smaller units around it.

Conclusion:

As new-born care facilities are expanding at a fast pace,we need to overcome all the challenges and be prepared to screen the greater number of preterm babies surviving. As ROP is a dynamic time-bound disease with a narrow period of time for screening, a timely screening and treatment can prevent blindness in most of these babies. A day should come in near future when all the 'at risk' babies are screened in every corner of India.

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Central Choroiditis in the Young, Clinical Pearls from Case Series

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This is a case series of 3 young male patients with central choroiditis; the aim is to highlight the differences in management in a situation where achieving quiescence urgently is crucial to prevent foveal scarring and irreversible visual loss.

Case 1

29 yo male presented on January 18, 2016 with right eye vision drop for 3 days. He had no systemic complaints or disease.

Right eye had active paracentral choroiditis (Figure 1); left eye was normal. In view of the urgency to save foveal centre from involvement he was placed on high dose oral steroids (1 mg/kg body weight).

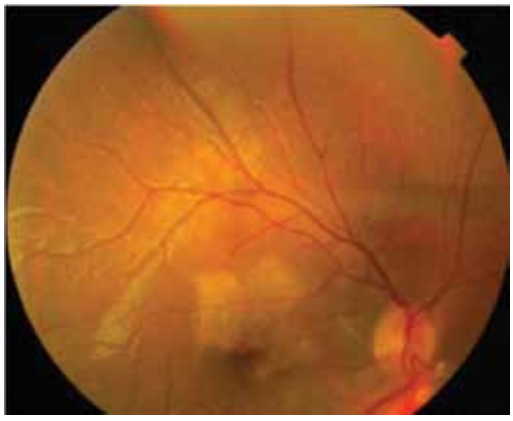


Figure 1: Paracentral choroiditis 3 days after starting oral steroids

There was inadequate response while on oral steroids even at 8 weeks (Figure 2). By the end of 5 months of treatment there was enough resolution to consider discontinuing steroids (Figure 3).

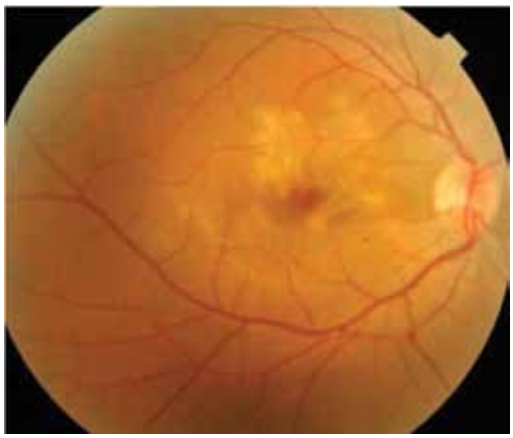


Figure 2: Persisting activity at edges of lesion even while on oral steroids for 2 months

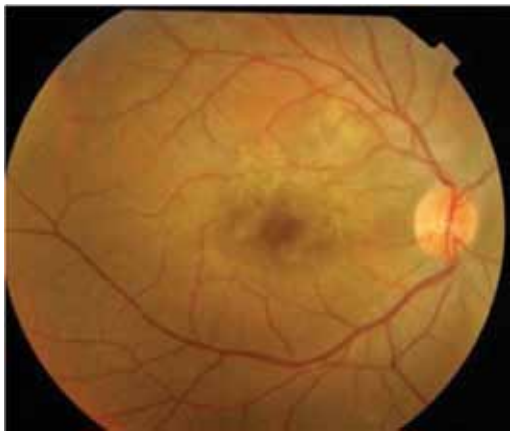


Figure 3: Resolution of activity while on oral steroids for 5 months

However, within 2 weeks off steroids, he developed new active choroiditis lesions (Figure 4).

Figure 4: New active lesions 2 weeks after discontinuing oral steroids

At this time he was investigated for possible underlying etiology including sarcoidosis, TB etc: Haemogram, Serum Calcium, Serum Angiotensin Converting Enzyme, Serum HIV, Chest X-Ray, QuantiFERON-TB Gold, Mantoux Test.

Mantoux skin test returned strong positive with 13 mm induration; all other results were normal/negative.

He was placed on antitubercular therapy along with oral steroids (tablet prednisone tapered off over 4 weeks). 10 days later central lesions were resolving more optimally than they had with oral steroids only (Figure 5), though there was a new active lesion seen inferior to disc at this time (Figure 6). This resolved well after 5 weeks of continuing therapy (Figure 7).

Figure 5: Resolving activity at 10th day after starting anti-tubercular therapy

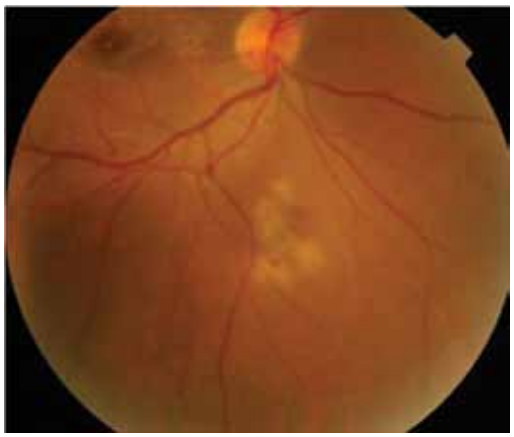


Figure 6: New active lesion on 10th day after starting anti-tubercular therapy

Figure 7: New lesion resolved by 5th week after starting anti-tubercular therapy

At the end of 6 months of AntiTB therapy all choroiditis was completely resolved, and there has been no reactivation of lesions since (Figure 8).

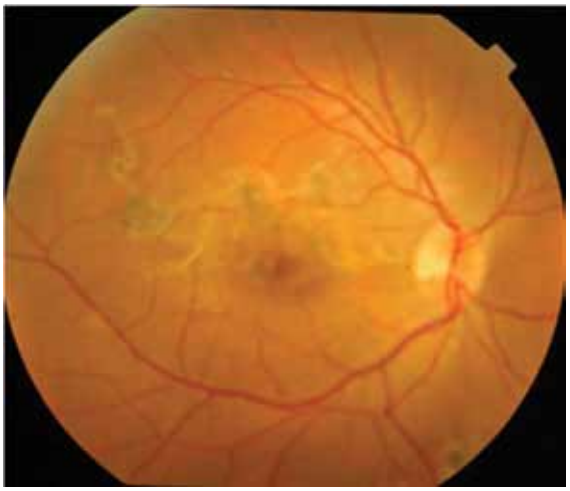


Figure 8: All lesions healed completely by 6 months after starting anti-tubercular therapy with no reactivation

This case highlights the importance of testing for latent TB in every case of choroiditis even in the first event, and strongly consider additional antiTB therapy in event of test results returning positive.[1-4]

Case 2, Feb 11, 2015

40 yo male patient presented with right eye floaters for 10 days in February 2015.

VA was 6/6 each eye. Right eye had active paracentral choroiditis; left eye was normal. He was placed on oral steroids (1 mg/kg body weight).

Investigations returned normal/ negative: Haemogram, Serum Calcium, Serum Angiotensin Con-verting Enzyme, Serum HIV, Chest X-Ray, QuantiFERON-TB Gold, Mantoux Test.

Even while on high dose steroids there was reactivation of some lesions & extension of scarring into the foveal centre (Figure 9). Steroids were discontinued after 3 months use.

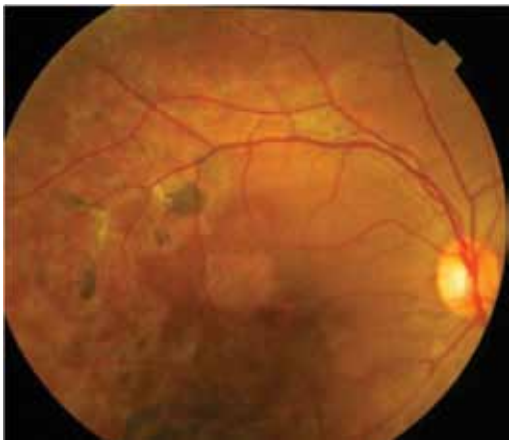


Figure 9: Right eye healed choroiditis after 3 months oral steroids

About 18 months later, in Nov 2016, he presented with active choroiditis in left eye (Figure 10). He was placed on high dose oral steroids (80 mg/d). Even while on oral steroids at 1 week, Nov 12, 2016, the central lesion was enlarging (Figure 11), and there was a new peripheral lesion (Figure 12).

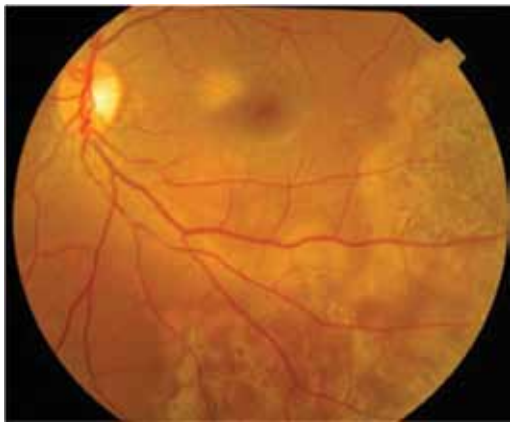


Figure 10: Left eye new active choroiditis 18 months after Right Eye was affected

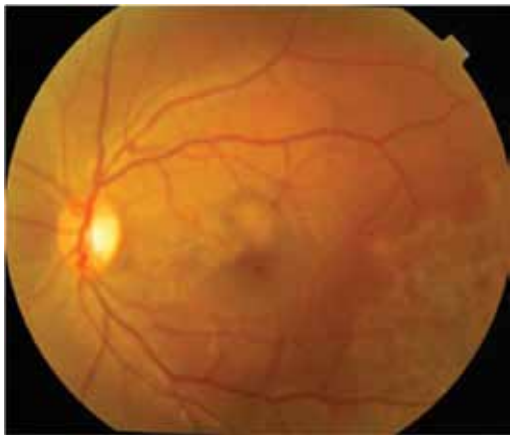


Figure 11: Left eye enlarging central lesion 1 week after starting oral steroids

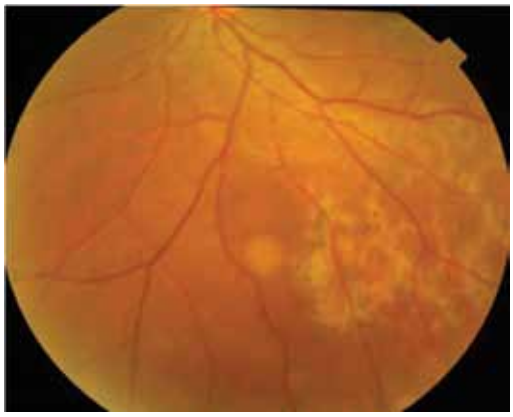


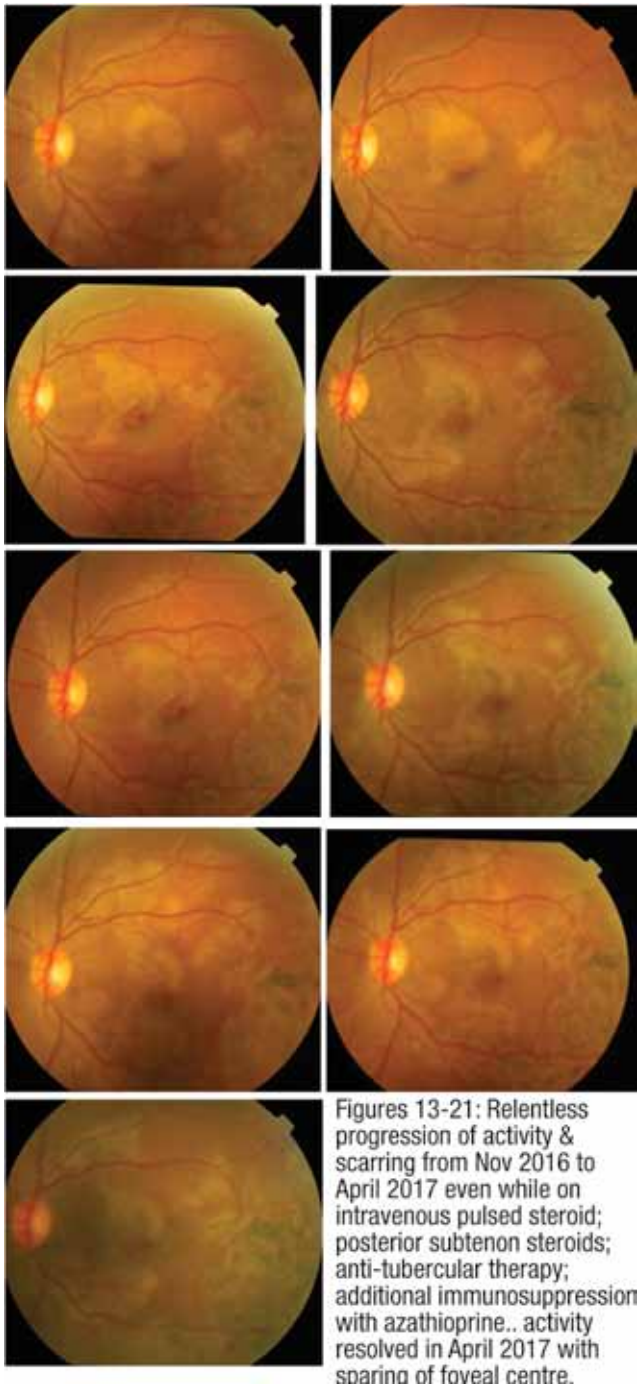
Figure 12: New active peripheral lesion 1 week after starting oral steroids

On Nov 12, 2016, in desperation, AntiTB therapy was initiated even though all relevant test results were negative.

Lesion was still enlarging, so oral steroids were replaced with intravenous high dose pulsed steroids for 11 days (in addition to antiTB therapy). Lesions continued to enlarge gradually and new lesions were seen so even while on intravenous steroids he was treated with an injection of pos-terior subtenon 20 mg triamcinolone acetonide. Alternative immunosuppression was also added (tablet Azathioprine 50 mg/d).

Subsequently for continuing activity 2 further injections of subtenon triamcinolone acetonide and further 4 injections of 1 gram intravenous methylprednisolone.

There was relentless progression of choroiditis (Figures 13-21) even while patient was on maximal systemic steroids, additional periocular steroids, alternative immunosuppression and antiTB thera-py. Lesion activity did finally resolve with foveal sparing and good visual outcome.



Figures 13-21: Relentless progression of activity & scarring from Nov 2016 to April 2017 even while on intravenous pulsed steroid; posterior subtenon steroids; anti-tubercular therapy; additional immunosuppression with azathioprine.. activity resolved in April 2017 with sparing of foveal centre.

Relentless placoid chorioretinitis (RPC) or Ampiginous choroidopathy was first described in 6 cases by Jampol & Yannuzzi et al in 2000 (Figure 22). [5]

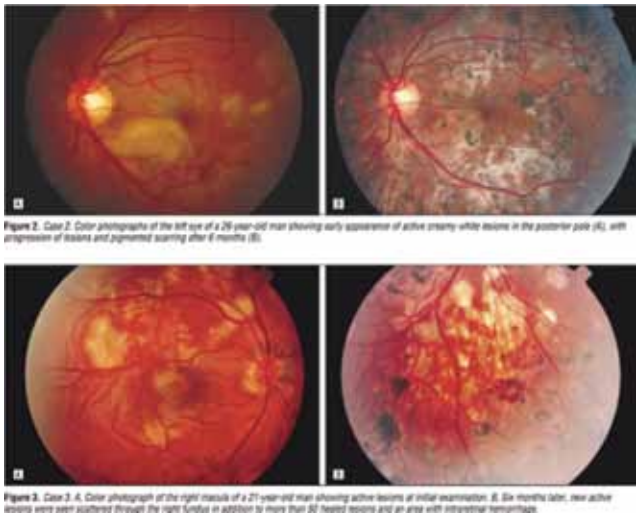


Figure 22: Cases of Relentless placoid chorioretinitis (RPC) or Ampiginous choroidopathy described Jampol & Yannuzzi et al in 2000

Salient features described include:

- Presence of 50 to hundreds of posterior & peripheral retinal lesions
- Older, healing lesions accompanied by new active placoid lesions
- Prolonged activity 5 - 24 months (mean 9 months)
- Relapses common even several years after quiescence
- Central vision is affected in all cases
- Systemic steroids improves visual outcome, reduces duration of active disease
- Can progress & recur despite steroid therapy
- Alternative immunosuppression helpful

Probably a hypersensitivity reaction to Mycobact tuberculosis; TB testing (with tuberculin skin testing or QuantiFERON-TB Gold) mandatory, especially when in endemic regions

Case 3

37 year old male patient presented in February 2014 with left eye central choroiditis. All test re-sults, including Mantoux Test, were negative. He was treated with high dose oral steroids with resolution of activity. He was then placed on Azathioprine for maintenance.

He developed aseptic necrosis of femur subsequently, likely from the prior steroid use. Azathio-prine caused altered liver function tests (LFT), hence alternative immunosuppression cover was re-moved.

He presented again in February 2017 with both eyes symptoms; at this time, right eye had new lesions of active paracentral choroiditis (Figure 23) and left eye had reactivation of activity along with paracentral choroidal neovascular membrane (CNVM) with bleed and fluid inferotemporal to macula (Figures 24-26).

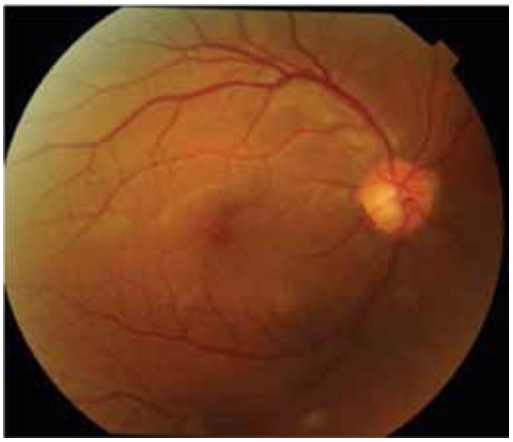


Figure 23: Right eye resolving central choroiditis lesions

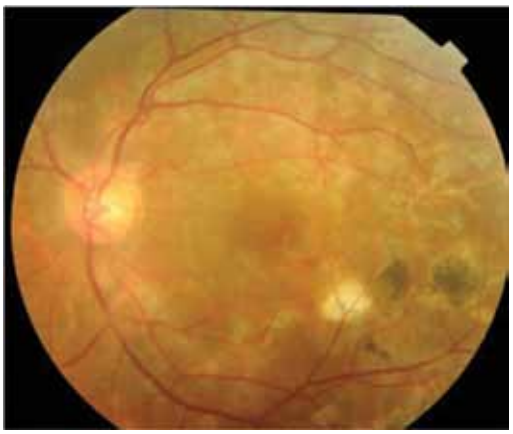


Figure 24: Left eye choroiditis scars with CNVM inferotemporal to macular centre

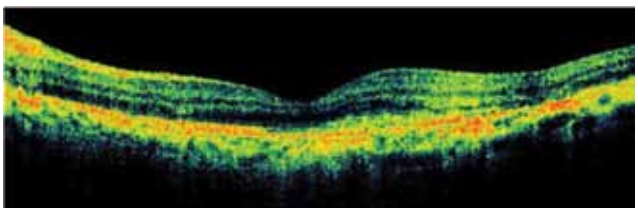


Figure 25: Left eye sub macular exudate & heme temporal to centre

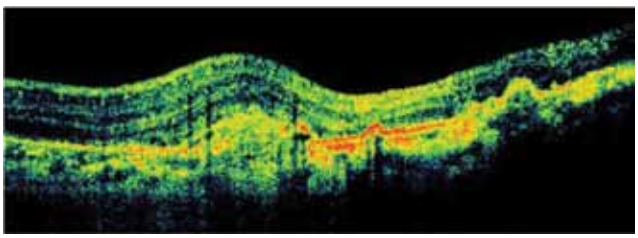


Figure 26: Left eye CNVM with intraretinal and sub retinal fluid

This time systemic steroids could not be given due to history of femur necrosis, and azathioprine was also contraindicated due to altered LFT.

He was treated with biologics, Adalimumab (Exemptia) 40 mg subconjunctival once in 2 weeks for 3 doses under rheumatologist guidance which led to resolution of activity.

Maintenance alternative immunosuppression is achieved with mycophenolate mofetil 2000 mg daily which he is able to tolerate well.

The left eye CNVM has been treated with 4 injections of intravitreal ranibizumab but fluid persist-ed and combination therapy (photodynamic therapy + intravitreal ranibizumab injection) was done with resolution (Figures 27-28).

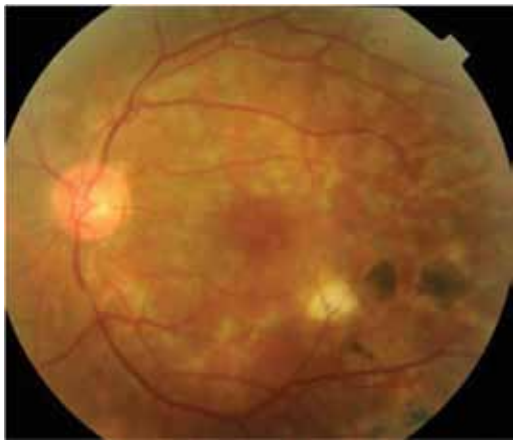


Figure 27: Left eye scar from CNVM; active CNVM regressed with resolution of heme and fluid following combination therapy

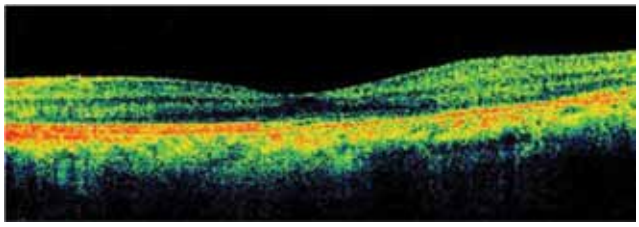


Figure 28: Left eye all sub macular exudate & heme resolved following combination therapy

Clinical pearls: Biologics maybe used to control central or paracentral choroiditis when steroids and other agents are contraindicated due to complications or adverse effects. [6-7]

CNVM is another complication of healed choroiditis that requires vigilance for early detection and management.

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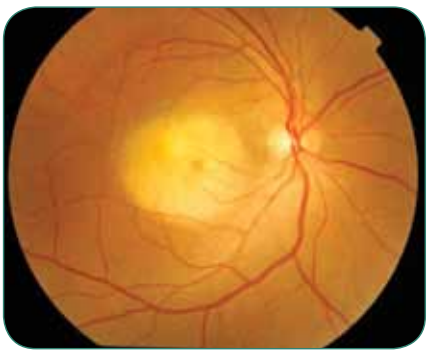
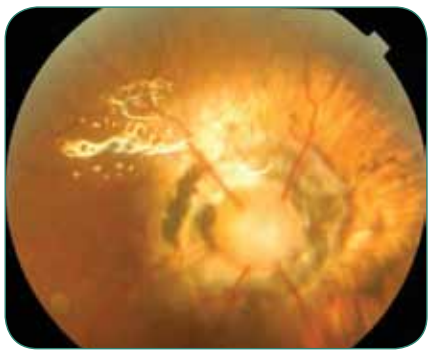
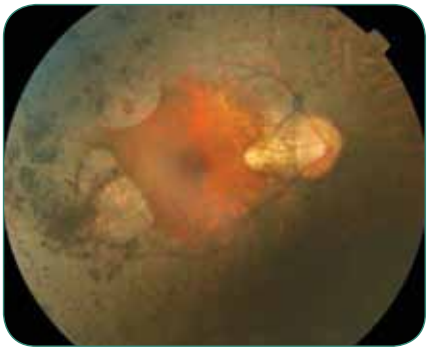


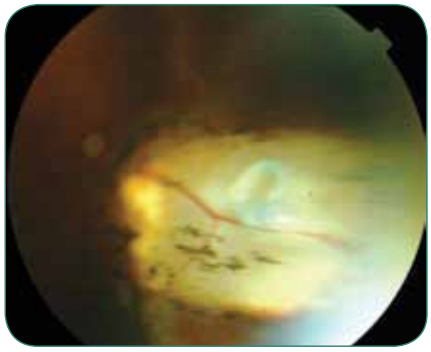
Exudative CSCR



Silicon Oil in-situ following retinal reattachment surgery in morning glory syndrome



Gyrate atrophy sparing the macular centre



Scleral suture (green ethibond) seen through the retina and choroid

3rd Annual Conference of

Telangana Ophthalmological Society

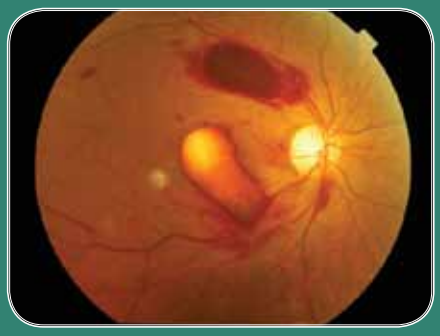
Warangal, July 21-23, 2017

Award Winners at TOC 2017

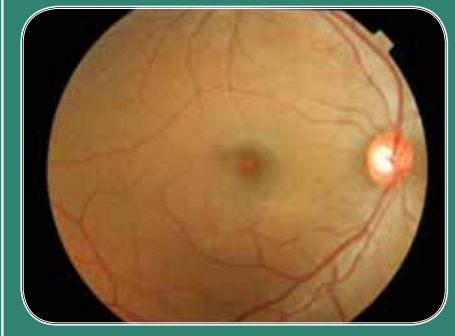
Sl No.	Category	Winner	Title of Presentation
1	Dr Swarup Video Session	Dr Anil K Mandal	Management of Congenital Glaucoma Associated with Stickler Syndrome
2	Dr P Ramchander Competitive Free Paper Session	Dr Tarjani Dave	Orbital Implant Migration - Are we Thinking Correctly?
3	Dr N Subramanya Reddy Competitive Free Paper Session for Post Graduates and Residents	Dr Arushi Gupta	OCT Angiography in Best Disease
4	Dr Manoj Mathur Competitive Poster Session	Dr Bhava Tharini M	Wavefront Optimized Ablation Vs Topography Guided Surface Ablation in Myopic Eyes - Comparative Study of Visual Outcomes
5	Dr VV Ramana Rao Grand Rounds	Dr Divya Pothala	Secondaries in Orbit from Thyroid Carcinoma
6	Dr Gangadhar Reddy Quiz	Dr Pavani P, Dr Pragathi G Dr Chiranjeevi Chowdary	Runners Up Team Dr Laxmipriya P, Dr Charul Singh Dr Sharat

Choroidal Lymphoma

Radiation Retinopathy and Optic Neuropathy

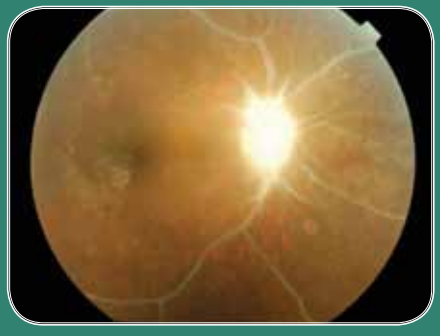


Haemorrhages in AML



Welding arc Maculopathy

Choroidal Rupture following Concussion



Behcet's Disease

Dr Alpa Atul Poorabia
Secretary- Telangana State Chapter
Mobile: +91 9885315270

WOS (Women Ophthalmologists Society) was formed in February 2015 at AIOC2015, Delhi, with the mission to boost professional environment and encourage more women to come forward and partici-pate in all aspects of professional carrier.

WOS initiated an assessment survey among the women ophthalmologists about which kind of activity or initiative WOS should take to help them for betterment of their personal and/or professional growth. Based on their valuable inputs and various individual suggestions, WOS is now working on many pathbreaking programs, some of them are already started and some of them are in the pipeline.

WOS started its first visionary Mentor-Mentee program in February 2017. In this program, list of the people who want to learn something and list of those people who wish to mentor them was finalized. Depending on the input, mentees were assigned to designated mentor.

During the second annual WOS conference in Jaipur from 25-26th November, 2017, all Mentees will be having face to face meeting with their Mentors to discuss their achievements and what needs to be done for further betterment. This is one of the highly successful programs of WOS.

And now WOS has announced another path breaking program, "Women in Research", WINR program.

WOS: Research Made Simple: Research For All = WINR Program.

This is the 1st of its kind in the world. This plan will make research (whether clinical or molecular) an arena of each member and will change the way research is looked at in India. And so many women, whether in Institution or solo practitioner, will be a part of this change. The good news is that even any member woman who has till now not been into any kind of research and studies can now be a part of this. And I am sure, a new chapter in history will be created. WINR has 2 arms: The Clinical Research Arm and the Molecular Research Arm.

Interested WOS members are invited to send 2 new research ideas on which they propose to do a study, which will be based on whatever kind of work they are doing. The idea will be reviewed by a support group who are experts in their field. If the support group finds the idea feasible, the WOS member will be given an opportunity to give a 4-minute presentation in front of the support group of the selected idea at the WOS Annual Conference in Jaipur on 24th/25th November 2017 in the WINR session. Here the study protocol would be discussed and thereafter the study will be started by the WOS member, the support group members will be constantly a part of this study as a guiding force for a year. There would be a midyear meeting to review the progress of the study. At the end of 1 year the WOS member can either present the analyzed data or preferably publish the data as a study in a journal. The support group will also guide on how to publish. I am sure this will be a wonderful opportunity for many women who aspire to work on research front.

WOS-Telangana State Chapter in collaboration with HOA organized a very successful 2nd meeting on 30th April 2017 which included elaborative topics on Challenging Situations in Cataract, Glaucoma and Refractive surgeries along with discussion on NABH and topics beyond routine Ophthalmology. One of the moto of WOS is to encourage more and more women to come forward and present on Dias. In this meeting, local WOS chapter provided opportunity to two new speakers, Dr Madhavilatha and Dr Renu Dubey, and both of them delivered their presentation with confidence and got accolades from audience. In this meeting, WOS-Telangana state chapter invited faculty like Dr Chitra Rammurthy and Dr Mohita Sharma (Secretary WOS). Dr Vidyavathi and Dr Subhadra Jalali were felicitated for their remarkable contribution in Ophthalmology by "Prathiprajana Award" by Maxivision Eye Hospital. Ms Manju Latha Kalanidhi, who is very well known senior health journalist, attended the meeting and de-livered an interesting presentation "Healthcare consumerism – The future is here"

WOS is having 2nd Annual conference on 25th & 26th November, 2017 at Jaipur, which will be attended by so many International and National faculty including Dr Susan Mc Donald (President, Women in Ophthalmology-WIO). It has a very exciting and very different kind of program, which will help us to face many challenges in our day to day clinical practice.

I hope that WOS will bring the much needed change in the way women practice and will be able to provide ample opportunity for their professional growth.

Suggestions are most welcome.

Regards,

Dr Alpa Atul Poorabia
Secretary- Telangana State Chapter
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Concept of Family Benefit Scheme of Ophthalmologists Family Welfare Society

A need of every ophthalmologist

Dr SV Katta; Late Dr. P. Ranga Reddy
Email: OFWS2016@gmail.com

Introduction

Concept of social bondage develops in four ways

1. Blood relationship
2. Professional referral relationship
3. Service to the known
4. Service to unknown through passing on knowledge, timely help etc. First 3 are limited. 4th is unlimited. Let us develop fraternity contribution concept in the society & enjoy the thrill of passing on to next generation the sanctity of this concept

History

UK surrounded with ocean & depending on exports & imports was prone for oceanic calamities. In 1798, at Lloyd's coffee club, marine exporters formed society & concept of insurance, which is being followed by LIC of India & all insurances of the world. Dr Ranga Reddy & Dr NS Reddy, following this ideology, floated the idea of FBS. Dr C Srirama Murthy later successfully implemented it at both State (for APOS) and National (for AIOS) levels

Purpose

1. Financial growth as per statistics
2. Spiritual growth: feeling of camaraderie among all
3. Contentment
4. Security: Registered body having constitution.

Aim

Passing on to next generation benefits of contribution

Age related admission fees maintained as deposits; the interest gained is maintained for office expenses. Fraternity contribution is delivered to deceased family in the right time

Recently after seeing our concept, twin cities taxpayers association, ENT association, Paediatric association, Gynecology association & Psychiatry association are planning to follow this model

Search:

1. In you tube.com, search family benefit insurance presentation on 2nd October 2009
2. In www.tos.org, search ofws

Application for membership of Family Benefit Scheme of Ophthalmic Fraternity Welfare Society

Name:

S/O / W/O:

Age: Sex: Date of Birth:

Address for Correspondence:

Your Photo

E-Mail: Mobile: APOS LM.No:

Admission Fee: Rs.1000/- same for all along with

Up to 35 years	Rs. 2500/-	35 to 40 years	Rs. 5000/-
40 to 45 years	Rs.10000/-	45 to 55 years	Rs. 15000/-

I enclose D.D. No. Dated for Rs.

Drawn on bank towards membership of FBS-OFWS

Declaration:

I solemnly declare that I do not have a history of any acute illness in the last 12 months (acute disease/ cardiac or malignant disease). I further agree to abide by the rules & byelaws of OFWS FBS. I accept any decision of EC/MC of OFWS as final.

Nomination: I herewith nominate the following as my beneficiaries

Sl. No.	Nominee's name	Age	Sex	Relationship	Signature/photo
1					
2					

In case of Minor

1					Guardian's signature
2					

Name & address of the Guardian who represents minor:

Witnesses: 1. 2.
(Signature/ Name & Mobile)

Date:

Place: Signature:

OFWS Secretariat office: Dr C SHARATH BABU
Alankar Circle, Warangal - 506 011
Mobile: Nutan Sleh – 95504 66800
EMAIL:ofws2016@gmail.com

Enclosures: 1) Age proof 2) D.D

APPLICATION FOR LIFE MEMBERSHIP OF TELANGANA OPHTHALMOLOGCAL SOCIETY, HYDERABAD.

PHOTO OF MEMBER

Applied For Life Member ☐ Member in waiting ☐

Existing APOS members need not apply.

Name (In Block Letters) .....

Father's / Husband's Name : .....

Age : ..... Sex : ..... Date of Birth .....

Native District .....

Address (Present) .....

.....

.....

Address (Permanent) .....

.....

.....

Mobile : ..... E-mail: .....

Designation : .....

Academic Qualification : .....

MBBS Year : PG DO. MS DNB Year (For Life Member)

Joined PG in Ophthalmology Year : (for member in - waiting)

Note : Existing APOS members need not apply.

Date : ..... Signature of the candidate

Membership Fee : Rs. 2,000 for Practitioners
Rs. 1,500 for PGS

DD/At Par Cheque No. :

Remarks of Secretary :

DD/Cheque in favour of **"TELANGANA OPHTHALMOLOGICAL SOCIETY"** Payable at Hyderabad.

Kindly send the completed forms to : **Dr. A. RAVINDRA,** Teja Eye Hospital, H.No. 6-2-58, Kakaji Colony, Hanamkonda- 506001. Cell: No. 98664 26367

TELANGANA OPHTHALMOLOGICAL SOCIETY

HYDERABAD.

Mail the duly filled in Registration Form along with your Cheque/DD to:

Dr A Ravindra
Teja Eye Hospital,
H.No. 6-2-58,
Kakaji Colony, Hanamkonda- 506001
Cell: No. 98664 26367

NOTES

NOTES

3rd Annual Conference of

Telangana Ophthalmological Society

Warangal, July 21-23, 2017

WCPOS IV

4th World Congress of Paediatric Ophthalmology and Strabismus

See You in Hyderabad, India

1-3 December 2017

Registration and Hotel Booking
Available Online