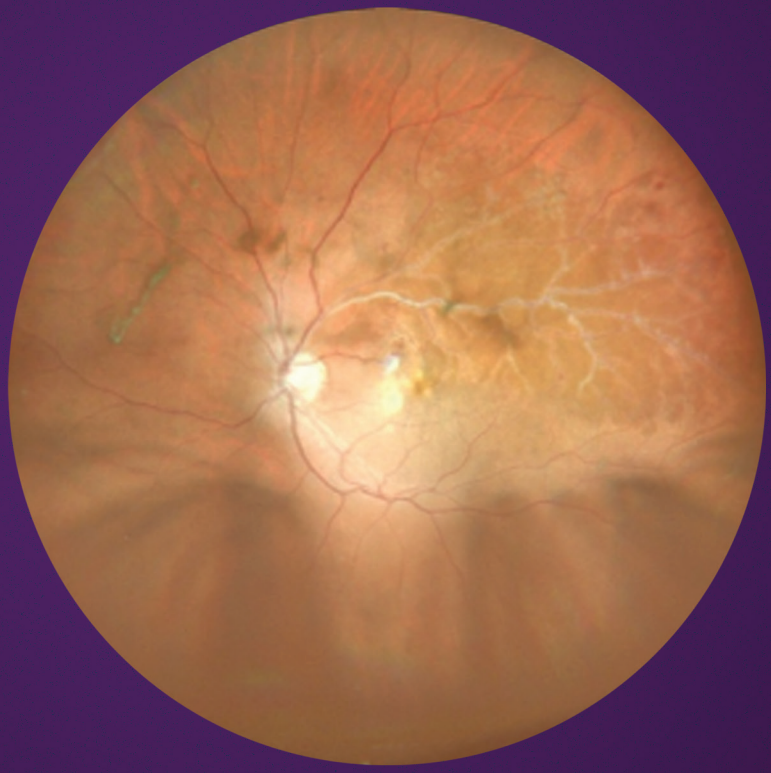
MPSOS TIMES

Vol 03 (Retina) Jul - Sep 2023



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*Data on File; wAMD: Wet Age-Related Macular Degeneration; DME: Diabetic Macular Edema;
RVO: Retinal Vein Occlusion; mCNV: Myopic Choroidal Neovascularization,
PDR: Proliferative Diabetic Retinopathy, ROP: Retinopathy of Prematurity

XLVI Annual Conference of M.P. State Ophthalmic Society

"Nayan Kumbh"

27th-29th Oct 2023, Hotel Anjushree, Ujjain, M.P

Hosted by: Ujjain Divisional Ophthalmic Society

Theme: "Collaborate & Connect".

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2022-2024

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MESSAGE

DR S K PARWANI

To,

The Scientific Committee chairman
MPSOS

Madam ,

It is great pleasure to know that the M P State Ophthalmic Society is coming out with MPSOS TIMES Retina Volume in the coming State conference at Ujjain.

The subject of retina has always been a challenging field. With the advancements in both diagnostic and operative technology, a new horizon has emerged in the management of retinal problems resulting in better visual outcome.

I am sure that this publication by your team will be useful for both students and practising ophthalmologists.

I wish you and your team a great success in this and all future endeavours.

With warmest Regards,

S K Parwani

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MESSAGE

DR ATUL MISHRA

Dear friends,

Congratulations to MPSOS on the launch of your inaugural journal focusing on retinal disorders. This significant achievement reflects your dedication to advancing the field of ophthalmology and improving patient care.

I applaud the entire team for their hard work and commitment in bringing this valuable resource to the medical community.

In today's rapidly evolving medical landscape, staying current with the latest research and treatment guidelines is essential.
The importance of updated text material in the treatment of retinal disorders cannot be overstated. In the constantly evolving field of ophthalmology, staying abreast of the latest research findings, treatment protocols, and technological advancements is crucial for providing the best possible care to patients with retinal conditions. This journal will serve as a valuable resource for ophthalmologists, providing them with access to evidence-based guidelines and innovative approaches that can lead to improved diagnostic accuracy and more effective treatment strategies. Additionally, it facilitates the dissemination of knowledge and fosters collaboration among experts, ultimately advancing the understanding of retinal disorders and leading to better patient outcomes. Embracing the power of updated text material empowers healthcare professionals to deliver cutting-edge care and ensures that patients receive the most up-to-date and tailored treatments available.

One striking example that underscores this significance is the evolution of diagnostic modalities such as OCTA. The emergence of innovative approaches, such as the integration of advanced imaging techniques like optical coherence tomography angiography (OCTA), has revolutionized our understanding various retinal conditions. These new diagnostic modalities have paved the way for more targeted and effective interventions, ensuring better outcomes and quality of life for patients.

As readers engage with your journal, they will undoubtedly benefit from the wealth of insights and information it provides. I am confident that your dedication to producing updated and comprehensive text material will have a lasting impact on the field of retinal disorders and beyond.

Once again, congratulations on this remarkable achievement, and I look forward to the continued success of the MPSOS Society and its contributions to the medical community.

Warm regards,

Dr. Atul Mishra

Director, R.K Mishra Eye Hospital, Jabalpur
Email: dratulmishra@live.com

MESSAGE

DR PRADEEP VYAS

It gives me immense pleasure to write these words for the breakthrough work by scientific committee of MPSOS by bringing out the first volume of scientific magazine of MPSOS.

50 years since it's inception, MPSOS has paved way for generations of ophthalmologists in career and academic progression.

At this juncture, the responsibility taken up by the editorial and scientific team of MPSOS to boost the scientific aptitude of our association through this magazine, will remain a milestone in the history of MPSOS.

I wholeheartedly congratulate the entire scientific team of MPSOS in giant leap forward.

Dr. Pradeep Vyas

Ex-president, MPSOS
Organizing Chairperson, Nayan Kumbh 3.0
MPSOS Conference 2023, Ujjain

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MESSAGE

Dr. ARVIND KUMAR DUBEY

Dear Dr.Vinita,

Kindly accept my sincere apologies for not being able to make to Ujjain Conference, but having seen your working, involvement and intensity I most sincerely congratulate you for the work you are doing to upscale the standard of scientific interaction in MPSOS.

You have been passionately involved through out the year conducting several online and off line activities,which sets a great precedence.

I carefully read the scientific programme for Ujjain, I can say most comprehensive, meticulous and inclusive.

Kindly accept my compliments for your hard work and also my appreciation for your team.

Dr. Arvind Kumar Dubey
Senior Retina Expert, Gwalior
Email: adubey56@gmail.com

MESSAGE

DR PUSHPA VARMA

Dear Dr Vinita Ramanani,
Scientific Committee Chairperson MPSOS,

It is with great pleasure let me have the apportunity to congratulate you for your absolute dedication and meticulous work for the MPSOS.

Since begining I have seen great sincierity in your every act as a scientific Chairperson for the up coming conference to be held at Ujjain.

I am sure the conference will be a great great success under your able guidance . Wishing you all the success from the depth of my heart.

With highest regards,

Dr Pushpa Varma
Senior Eye Specialist Indore and Ex Dean MGM Medical College
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CONTENTS

EDITORIAL

HOPE FOR RESTORING VISION IN RETINAL DYSTROPHY, CHALLENGES

Dr.Swarna Biseria Gupta

Ex Dean, D.M.E & Director RIO, M.P
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It gives me immense pleasure to write this editorial for MPSOS times, which has special focus on retina in its third edition. Retinal diseases are one of the most frequently seen disorders in our OPD, need time and patience for management. These encompasses several medical, uveitic, developmental and surgical pathologies. This issue of MPSOS Times is an important knowledge dissemination platform for the general ophthalmologists, Research fellows, post graduate students and faculty members.

“I FOUND THAT LUCK IS QUITE PREDICTABLE. IF YOU WANT MORE LUCK, TAKE MORE CHANCES, BE MORE ACTIVE, SHOW UP MORE OFTEN”- Brian Tracy.

The ultimate measure of a man is not where he stands in moments of comfort and convenience, but where he stands at times of challenge and controversy, said Martin Luther King ,jr.

Such a challenge for us is retina, as Retinal morbidity is an emerging cause of visual impairment.

Historically, retinal disease has had a low priority in prevention of blindness programmes in developing countries in comparison to cataract, glaucoma, cornea and community ophthalmology. Amongst retinal disease , diabetic retinopathy and age related macular degenerations get importance and are treated effectively.

The implications of its precise diagnosis and the controversies surrounding its optimal management have direct implication on quality of life. The scientists have found a new way to activate dormant cells in the retina that could restore vision. New research has found a solution to the problem of degenerative retinal diseases, which has made the lives of millions of people worldwide miserable.

According to a statement by the institute, researchers led by universite de Montreal’s Michel Cayouette have found a way to reactivate dormant cells in the retina and transform them to ultimately replace cells lost in retinal degeneration.

The research team, discovered that cells that lie dormant in the retina(glial cells) can be induced to transform into cells sharing some properties with cone photoreceptors, which are responsible for color perception, reading and driving.

Inherited retinal degenerations are caused by the loss of light- sensitive cells in the retina.When these cells degenerate due to disease, they are not replaced, and the patient suffers vision loss that can progress to total blindness. Rods and Cone photoreceptors are the main cellular units responsible for visual photo-transduction. It is a process by which light signals are converted into action potential within the retina and facilitate the brain’s

perception of an image. During this process, light sensitive pigments are generated and recycled. Retinal dystrophy occurs due to abnormalities in photoreceptors as well asdefects in photo-transduction.

Retinal dystrophies present with visual loss ranging from night blindness, color blindness, constricted fields, central scotoma to complete blindness. Affecting about one in 4,000 individuals, may or may not be associated with syndromic manifestations. Mutations in more than 120 genes coding for protein present or involved in retinal cells, rods and cones, photo-transduction, visual cycle or gene regulation may be causative. Inheritance is seen in all forms, autosomal dominant, recessive, x- linked and mitochondrial, genetic overlap may be there.

Next – generation sequencing and chromosome microarrays provide better diagnostic abilities and genetic counseling and therapy.

Retinal dystrophy can be classified as rod- dominated, cone –dominated and generalized.

ROD- DOMINATED RETINAL DYSTROPHIES

A genetic disorder affects rods, in the form of progressive (retinitis pigmentosa) or stationary (congenital stationary night blindness). Patients have night blindness, constricted field of vision may progress to complete blindness, if cones are involved in late stages. Congenital stationary night blindness shows autosomal dominant, recessive incomplete and complete form of x- linked inheritance.Two variants exist with normal fundus(Nougaret, Schubert Bornschein and Riggs - type and with abnormal fundus, fundus albipunctatus, and oguchi disease).

CONE- DOMINATED RETINAL DYSTROPHIES

Progressive cone or cone- rod dystrophy occur sporadically or withautosomaldominant, recessive or x- linked inheritance patterns.Patients have macular atrophy with complete or incomplete achromatopsia.

GENERALISED DYSTROPHY

Simultaneous involvement of rod and cone receptors, associated with progressive and severe involvement of visual functions. Leber's congenital amaurosis, choroidermia are sex linked and show progressive diffuse degeneration of the choroid, RPE, and retinal photoreceptors. Patients present in the first decade with night blindness and have severe visual loss after about 50 yrs of age.

MACULAR DYSTROPHY

A group of Mendelian inheritance including Best, Stargardt, Sorsby, Pattern dystrophy and others, show autosomal dominant, autosomal recessive, x- linked recessive and mitochondrial inheritance. Approximately 20% patients have positive family history.

NOTHING CAN BE DONE -

Isno longertrue for patients with Retinal dystrophies . Electrophysiological testing and auto-fluorescence imaging help to diagnose and predict the patient's course of disease. Better phenotyping, genotyping is essential for management approach. With increased life expectancy, the problem posed by these conditions is magnified. New gene – based treatment has promising role.

Newer treatment strategies directed towards stem cell and gene therapy are in trials. Luxturna provides RPE65 gene replacement therapy delivered by viral vectors to restore the visual cycle in early LCA has shown beneficial effect. In extensive, advanced involvement gene therapy is not effective and pluripotent stemcell transplant or retinal implants may be tried. Sub retinal transplantation or intravitreal injections of human embryonic stem

cell- derived retinal pigment epithelial cell,autologous bone marrow- derived mononuclear cells, undifferentiated umbilical cells are under trial.

Retinal prosthetics eg -light- sensing microchips are implanted into the retina and transmit impulses via the remaining neural network to the brain. A visual prosthesis is an artificial organthat transmits visual information by artificially stimulating a part of nervous system .Latest developments in retinal implants are developed to restore the vision in patients with dystrophy.

Such as" Argus11", provides rudimentary vision to blind patients, this device has 60 pixels,it can enable patients with advanced disease, to read large letters,determine the location of moving objects or people.

A device called the "Prima "was recently implanted under the retina of a patient with advanced dystrophy.It is activated by infrared light projected be special glasses. It has 378 electrode, provides more pixels than Argus.

Nanoretina, reported improved visual experiences, deliver infrared light to chip.

Other approaches to restore vision, are Brain implants, an electric chip directly stimulate the part of brain

Lab- Grown Retinas may restore vision to peoples with damaged retina, scientists developed a way to grow organised clusters of cells, called organoids, resembles the retina. They coaxed human skin cells reprogrammed to act as stem cells to develop into layers of several types of retinal cells that sense light andultimately transmit what we see to the brain.

ENHANCING VISUAL HEALTH CARE

An interdisciplinary team approach involving ophthalmologists, geneticists, genetic counselor and physician, providing patientcare would lead to the bestoutcomes.Genetic analysis, genetic counseling and the Gene therapy is the main essence of dystrophies. An up to date refraction, low vision aids, visual rehabilitation and support services are essential steps in the management of any advanced retinal dystrophy.

In conclusion, deprivation of eye sight has been perceived as the most severe form of punishment, second only to loss of life, Hence there is need for sensitization to problem, together with establishment of guidelines for preventing onset of depression, by including cognitive behavioral therapy and rehabilitation.

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Expert Opinion on Diabetic Macular Edema

Expert Panelists

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Q1: What are the investigations required for diagnosis and management of DME?

DR GAJENDRA CHAWLA

Managing DME is often challenging & require multi-disciplinary approach. An endocrinologist referral should be done for better metabolic control. Strict glycemic control is crucial in treatment of DME.

Apart from complete ocular examination, OCT is imperative& should be considered first &foremost investigation in managing DME including best corrected visual acuity. OCT helps us in better understanding & classifying the DME. Based on presence of various biomarkers like neurosensory detachment, hard exudates DRIL, vitreo macular traction, retina specialist can decide his treatment preference in given patient.

OCT-A is newer modality available to asses macular perfusion and ischemic maculopathy. Fluorescein angiography was considered gold standard investigation to asses DME in past, but with the availability of non-invasive OCT & OCT -A, its uses are gradually reducing however, in absence of OCT- A, it's quite helpful in assing macular perfusion. Ultra wide field angiography is another newer modality to evaluate peripheral retinal perfusion but it is quite expensive and is available only in limited centres.

DR DHANANJAY SHUKLA

In the era of macular photocoagulation, the operative description for DME was clinically significant macular edema (CSME); naturally, the diagnosis was clinical. If CSME does not involve the central fovea and the best-corrected visual acuity (BCVA) is good, still no diagnostic investigations are needed for prior to focal laser treatment of CSME. If BCVA is poor (<6/12) and CSME spares the foveal center, fluorescein angiography (FA) or OCT angiography (OCTA) is required to look for macular ischemia. In the current era of anti-VEGF treatment, the operative description is center-involving DME, which needs OCT for confirmation. If BCVA is disproportionately poor, OCTA/FA

may again be needed (see above).

For management of DME, the operative phrase is: TREAT THE PATIENT, not the eye. Systemic assessment is often overlooked, but is critical for the safety, choice, and redundancy of ocular treatment. For example, if the patient has excessive macular lipid exudation and deranged systemic lipids (note that these two entities may not necessarily coexist), intravitreal corticosteroids are preferred as first line treatment over anti-VEGF drugs. If the signs of hypertensive retinopathy (HTR: flame heme, cotton-wool spots) dominate over the clinically visible severity of diabetic retinopathy (DR), merely controlling blood pressure may alleviate the maculopathy.

DR ARUN BHARGAV

- OCULAR DIAGNOSTIC MODALITIES FOR DETECTING DME INCLUDES –
- OCT AND OCT- A ARE IMPORTANT NON INVASIVE TOOLS IN UNDERSTANDING THE PATHOGENESIS AND MANAGEMENT OF DME.
- APART FROM THICKNESS OCT BIOLOGICAL MARKERES INCLUDES - CHANGES WITHIN THE RETINAL LAYERS (OUTER AND INNER RETINA) AND CHANGES AT THE VITREOMACULAR INTERFACE, DRIL, LOSS OF INTEGRITY FOR OUTER RETINAL LAYERS, THICKNESS OF RETINAL LAYER, INTRARETINAL CYSTIC SPACES, PEARL NECK SIGN, SUBFOVEAL NSD.
- OCT ANGIOGRAPHY (OCT-A) ALLOWS VISUALIZATION OF THE RETINOCHOROIDAL CIRCULATION AND ALLOWS THE SEPARATION OF CAPILLARIES AT THREE DIFFERENT LEVELS. OCT-A CAN IDENTIFY FAZ, MICROANEURYSMS, AREAS OF CAPILLARY NONPERFUSION AND NEOVASCULARIZATION MUCH BEFORE THEY ARE APPRECIATED CLINICALLY. THEY ALSO GIVE AN IDEA OF CHOROIDAL THICKNESS.
- FUNDUS FUORESCEIN ANGIOGRAPHY (FFA)IS STILL AN IMPORTANT INVESTIGATIVE MODALITY TO CLASSIFY DME INTO FOCAL, DIFFUSE OR ISCHEMIC MACULOPATHIES . IMPORTANT FOR PLANNING THE LASERS.
- FUNDUS AUTOFUORESCENCE BEING A NON-INVASIVE INVESTIGATION CAN ALSO BE AN AID IN DIAGNOSING DME AS EXCESS ACCUMULATION OF LIPOFUSCIN PIGMENT AND DEPLETION OF LUTEAL PIGMENT AT RETINAL PIGMENT EPITHELIUM (RPE) LEADS TO AN INCREASED FAF SIGNAL.

SYSTEMIC INVESTIGATIONS FOR DME INCLUDES

HAEMATOLOGICAL TEST INCLUDES ESPECIALLY BLOOD SUGAR LEVELS, HBA1C, LIPID PROFILE, RENAL FUNCTION TEST AND ASSESSMENT OF SLEEP APNEA. ALL THESE FACTORS IF DETRANGED ARE KNOWN TO RISK FACTORS FOR INCREASED DME.

DR GANESH PILLAI

The main investigation that has changed the diagnosis and management of DME is OCT Macula. OCT is like the smartphone of Ophthalmology.

It has brought a new dimension to the management of DME.

We also have adjunct investigations for DME and Diabetic Retinopathy like Fundus Flurosceine Angiography which are used to identify macular ischemia and concurrent pathologies of Diabetic Retinopathy.

Q2: What is the treatment protocol for DME?

GAJENDRA CHAWAL

The treatment protocol for DME involve a combination of approaches tailored to individual patients need. We insist strict Glycemic control in all patients and also other metabolic disorder. As far as ocular treatment protocol is concerned, Intravitreal pharmacotherapy remains the choice of treatment for centre involved DME. Our first preference is AntiVEGF in all DMEs including pseudophakic except recent history of myocardial infant/stroke patients. Once macular thickness is reduced or it become non centre involving DME, we supplement with focal laser. For Tractional diabetic macular odema vitreous surgery is the only choice.

DR DHANANJAY SHUKLA

This is a long-answer question. First, we assess the retinopathy, and associated findings like HTR, cataract and intraocular pressure. Then we assess the systemic parameters and correlate them with fundus findings. Once a decision is made to perform macular focal photocoagulation, the follow-up is performed at 3-4 months. When central DME warrantsintravitreal injection, the choice of intravitreal anti-VEGF agents/corticosteroids depends on the severity of edema, lens status, intraocular pressure, and systemic status (see above). After the injection, the eye is reviewed for complications in the immediate postoperative period (infection, glaucoma, allergy, vitreous heme); the effect of the injection is assessed at about one month for anti-VEGF drugs and re-injection considered. These intervals are shortened considerably in a post-vitrectomy eye. Intravitreal steroid is typically not repeated before 3-4 months.

DR ARUN BHARGAV

REAL WORLD EXPERIENCES EVIDENCED ON MUCH RESEARCHED CLINICAL TRIALS HAVE OUTLINED TREATMENT PROTOCOLS ON THE BASIS OF VISION, FOVEAL INVOLVEMENT, PRESENCE OF PROLIFERATIVE RETINOPATHY, PRESENCE OF CATARACT AND IN VIEW OF SERIOUS SYSTEMIC DISEASES LIKE DIABETIC NEPHROPATHY, AND OTHERS.

PATIENTS WITH POOR SYSTEMIC CONTROL SHOULD FIRST INITIATE CONTROL OF RISK FACTORS PRIOR TO STARTING THE TREATMENT FOR RETINOPATHY

CATEGORY A: PATIENT WITH VISION BETTER THAN 20/40 AND NCI DME CAN BE OBSERVED.

CATEGORY B: PATIENTS WITH VISION LESS THAN 20/40 AND CI DME NEEDS ANTIVEGF THERAPY OR STEROID BASED IMPLANTS. CHOICE OF ANTI VEGF USED CAN BE BASED ON THE PATIENT NEPHROPATHY STATUS, CARDIAC AND NEUROLOGICAL FUNCTIONS. IN AN EVENT WHERE ANTI VEGF ARE CONTRAINDICATED THEN STEROID BASED IMPLANTS CAN OFFER A GOOD CHOICE. LASER CAN BE ONLY PLANNED AS A RESCUE TREATMENT IN THE WAKE OF RECURRENT OR RECALCITRANT DME.

CATEGORY C: PATIENTS WITH CI DME AND PDR NEEDS FIRST ANTI VEGF FOLLOWED BY PAN RETINAL PHOTOCOAGULATION.

CATEGORY D: IN WAKE OF CATARACT WITH DME BEST TO EITHER FIRST INJECT ANTI VEGF OR STEROID IMPLANT THEN PLAN CATARACT SURGERY OR CAN ALSO BE DONE AS A COMBINE PROCEDURE IN ONE SITTING.

CATEGORY E: PATIENTS WITH SERIOUS NEPHROPATHY ON DIALYSIS OR POST

RENAL TRANSPLANT NEEDS A NEPHROLOGIST CLEARANCE AND STEROID BASED IMPLANTS FORM THE FIRST CHOICE

ANTI VEGF ARE CONTRAINDICATED IN PATIENTS WITH RECENT HISTORY OF CVA OR CARDIAC INSULT.

CATEGORY F: PATIENTS WITH RECALCITRANT DME WHERE ALL TREATMENTS HAVE FAILED CAN BE PLANNED FOR SURGICAL MANAGEMENT.

CATEGORY G: PATIENTS POST VR SURGERY HAVING DME SHOULD BE TREATED WITH ANTI VEGF OR STEROIDS BASED IMPLANTS

DR GANESH PILLAI

After acquiring the relevant Investigations, we classify the type and extent of DME.

Important things we look at are-- type of DME, foveal involvement, affect on vision, chronicity, and presence of OCT Biomarkers.

Treatment Protocols mainly are of two types - PRN or Treat and Extend.

DME is Primarily treated with intravitreal ANTI-VEGF therapy.

Intravitreal (Dexamethasone implant) or periocular corticosteroids are also used for pseudophakic patients, chronic cases or those who are unresponsive to Anti VEGF Therapy.

Q3: What is the current role of LASERS in management of DME?

GAJENDRA CHAWLA

In the era of Intravitreal pharmacotherapy, laser still play a significant role in management of DME especially non central involved macular odema, focal laser remains first choice of treatment. We use lasers as adjunctive treatment in certain cases or in combination with antiVEGF injections for optimal results in centre involved DME patients, Laser PRP along with anti VEGF has shown beneficial effect and resulted in lesser number of injections in presence of significant peripheral non perfusion areas.

DR DHANAJAY SHUKLA

A: As mentioned earlier, lasers are still relevant for fovea-sparing CSME, or after the centre is dry post-injection. Though green wavelength is most used, yellow is arguably much safer for macular treatment to prevent collateral damage. The other safe laser modality is micropulse, though I have no experience with it.

DR ARUN BHARGAV

CURRENT INDICATIONS FOR LASER IN DME INCLUDES PATIENTS WITH NON-CENTER INVOLVED CSME, FOCAL AND GRID LASER THERAPY OFFERS RELATIVELY STABLE VISUAL ACUITY.

SELECTIVE LASER THERAPY (SRT) CAN BE USED FOR CI DME ESPECIALLY FOR RECALCITRANT AND RECURRENT DME.

LASER CAN ALSO BE PLANNED AS ADJUNCT TO INTRAVITREAL INJECTIONS.

DR GANESH PILLAI

LASERS have a very limited role in the management of DME today.

Green/Yellow Laser may be used for Occlusion of Leaking Microaneursyms

Treatment of circinate retinopathy. Or Treatment of background retinopathy

Subthreshold Macular Grid Lasers are upcoming, but have variable success, when compared to intravitreal injections.

Q4: What are the newer innovations in surgical management?

DR GAJENDRA CHAWLA

In recent years there has been several noteworthy newer innovations in surgical management procedures. Development of smaller gauge (23, 25 & 27 gauge) minimal invasive vitreous surgery (MIVS) has changed the surgical approach of VR surgeon resulting in faster recovery& improved outcome. Advancement in visualization system and microscope, chandler light, microsurgical instruments has helped VR surgeons to achieve surgical goal more efficiently, & they are able to deliver optimum results in most difficult cases also.

DR DHANANJAY SHUKLA

The innovations in surgical management are in the instrumentation (e.g., safer ILM peeling by Finesse Loop, 25-27G small gauge vitrectomy), equipment (e.g., heads-up 3-D vitrectomy with better teaching and recording facility), monitoring (e.g., intraoperative OCT to verify extent of membrane removal), and adjuvants (e.g., intravitreal steroids to speed up DME resolution).

DR ARUN BHARGAV

DME SHOULD BE CLASSIFIED FURTHER ON THE BASIS OF VITREO MACULAR TRACTION OR ADHESION. PPV PLAYS A MAJOR ROLE IN THE MANAGEMENT OF DME WITH VMTS, IN ORDER TO REDUCE MACULAR THICKNESS AND PROMOTE VISUAL ACUITY GAIN BY REMOVING RETINAL TRACTION EXERTED BY THE POSTERIOR HYALOID OR AN EPIRETINAL MEMBRANE. RECALCITRANT DME PATIENTS CAN ALSO BE TREATED WITH VITRECTOMY WITH ILM PEELING. THE NEWER GAUGE 27 G OFFERS EVEN FURTHER SAFER SURGICAL TECHNIQUES.

DR GANESH PILLAI

Surgical Management of DME mainly deals with the correction of co-existing pathologies that may contributing to the DME and vision loss, like ERM(Epiretinal Membrane), VMT (Vitreo Macular Traction) or Macular Holes

Newer advancements like Port Delivery System of Anti VEGF are upcoming, but have limited role in today's routine practice.

Q5: Prognostic factors for good visual recovery in case of DME?

GAJENDRA CHAWLA

Shorter duration, better baseline visual acuity, absence of proliferative diabetic retinopathy and smaller central macular thickness on presentation are few better prognostic factor for good visual outcome in DME. Strict glycemic control, hypertension and other metabolic control are essential in preventing further deterioration of visual acuity. Early diagnosis and prompt appropriate aggressive treatment with regular followup remain the key for maintaining good vision.

DR A SHUKLA

Systemic parameters include glycemic status & control of associated diseases; ocular parameters include severity of DME & integrity of outer retinal bands and RPE (OCT), macular perfusion status (OCTA or FFA), and central lipid exudation.

DR ARUN BHARGAV

- POOR OCULAR PROGNOSTIC FACTORS INCLUDES POOR VISUAL ACUITY, POST COMPLICATED CATARACT SURGERY, PRESENCE OF ISCHAEMIC MACULOPATHY, PRESENCE OF EXUDATION AT THE FOVEA, PRESENCE OF VITREOMACULAR ADHESION AND TRACTION.
- POOR SYSTEMIC PROGNOSTIC FACTORS INCLUDES POOR GLYCEMIC CONTROL, UNCONTROLLED DIABETIC NEPHROPATHY, ANEMIA, SLEEP APNEA AND MULTIPLE ENDOCRINE DISORDERS.

DR GANESH PILLAI

Poor Prognosis of DME is associated with the following

1. Chronicity of Macular Edema (multiple hard exudates/exudate at fovea)
2. OCT Biomarkers
3. Poor Response to Anti VEGF or intravitreal Steroid Therapy
4. Longstanding Diabetes/Poor Control of Diabetes
5. Poor Systemic Status-- Dyslipidemia, Hypertension, Anemia, Chronic Kidney Disease, etc.

Q6: How do you manage chronic recurrent DME with or without hard exudate plaque?

GAJENDRA CHAWLA

Managing chronic recurrent DME with or without hard exudates plaque remains challenge for any retina specialist. First & foremost, optimizing glycemic control, hypertension, lipid profile is crucial and here role of Internist/Physician is very important. Intravitreal steroids is primary choice or early switching to steroid may be beneficial in treating chronic recurrent DME. Additionally focal/grid laser treatment by conventional or sub threshold Micropulse laser can be considered in such cases.

DR DHANAJAY SHUKLA

ANS: My anti-VEGF of choice is aflibercept in severe, chronic, or recurrent DME. In the presence of hard exudates, dexamethasone depot (Ozurdex) works best.

DR GANESH PILLAI

Patients with Chronic Recurrent DME require more frequent injections and a close Follow up.

Chronic Recurrent DME respond better to intravitreal steroid therapy than to intravitreal Anti - VEGF. ANTI-VEGF therapy with periocular steroids Or Intravitreal Dexamethasone Implant are good options.

Newer ANTI-VEGF molecules like aflibercept and brolucizumab (pagenax) may be a more suitable choice for such patients.

CASE REPORT

A Rare Case of Serous Retinal Detachment with Subretinal Fibrin like Material Associated with Preeclampsia, Abruptio Placentae and Intrauterine Fetal Death

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A 31 year old female reported to the hospital with chief complaints of diminution of vision in the left eye since one day postpartum. She was primigravida with 29 weeks and six days of gestation and admitted under gynecologist with blood pressure of 160/110 mm of Hg, distension of abdomen, vomiting, absent fetal heart sound and heavy bleeding per vagina four days back. On evaluation, she was diagnosed with abruptio placentae and IUFD. She was a known case of hypothyroidism and gestational diabetes mellitus without any ophthalmic ailment. Laboratory tests on admission revealed hemoglobin - 7.7 gm/dl, erythrocyte count – 2.75 mil/cu.mm, total leucocyte count – 16300 cell/cu.mm, platelets count – 196 10^3/µl, prothrombin time – 14 sec, serum urea – 18.92 mg/dl, serum creatinine – 0.94 mg/dl and random blood sugar – 102 mg/dl. She was taken for emergency caesarean section and stillborn infant was delivered. Five units of blood transfusion and four units of fresh frozen plasma were given. Post-surgery patient recovered well with supportive treatment.

On ocular evaluation, best corrected visual acuity (BCVA) was 20/20 in the right eye while 20/40 in the left eye. The intraocular pressure and the anterior segment findings were within normal range. On posterior segment evaluation, right eye was normal while left eye revealed SRD with SRFM over posterior pole. There was no evidence of vitritis, disc edema, hemorrhage or vascular occlusion. On multi-modal imaging, Optical coherence tomography (OCT) revealed sub retinal fluid with fibrin like material and hyper-reflective dots in intraretinal andAbstract –

A 31 year old female reported with unilateral serous retinal detachment (SRD) with subretinal fibrin like material (SRFM) following preeclampsia, placental abruption and intrauterine fetal death. The best corrected visual acuity was 20/40 in the affected eye. Fundus angiography and optical coherence tomography (OCT) revealed choroidal ischemia with SRD and SRFM which resolved gradually over 4 weeks post treatment. The presence of SRFM with SRD could be due to intense ischemia and inflammation following transient disseminated intravascular coagulation, hypovolemia and hypertension. Multimodal imaging modalities can

help in early diagnosis and prompt management.

Keywords – subretinal detachment, abruptio placentae, intrauterine fetal death, preeclampsia, subretinal fibrin like material

Introduction –

Abruptio placenta refers to premature separation of normally implanted placenta from the uterus affecting about 1-2% of pregnant woman.[1] Preeclampsia is one of the most common causes for placental abruption. Serous retinal detachment (SRD) following placental abruption and intrauterine fetal death (IUFD) is a rare entity.[2-4] The presence of subretinal fibrin like material (SRFM) with SRD has been reported in association with pregnancy induced hypertension (PIH) in a singular case report. This is a rare case report describing unilateral SRD with SRFM following preeclampsia, abruptio placentae and IUFD.

Case Report –

subretinal area in the left eye (figure 1). Fundus fluorescein angiography (FFA) displayed multiple lobules of early hypofluorescence with late hyperfluorescence and leakage around macular area suggesting choroidal hypoperfusion in the left eye (figure 2). At ten days after the onset, left eye BCVA improved to 20/20 with complete resolution of SRD and SRFM without any treatment. However, OCT scan revealed irregularity of the ellipsoid zone (EZ) and the retinal pigment epithelium (RPE) layer with restoration of external limiting membrane (figure 3A). At four weeks, there is near complete restoration of the EZ while mild irregularity is seen in the RPE (figure 3B).

Discussion -

In the present case, SRD and SRFM were associated with preeclampsia, abruptio placenta and IUFD which appeared within one day of postpartum. FFA results have revealed delayed choroidal perfusion with late leakage suggesting that dysfunction of the choroidal circulation was primarily involved.

Vision loss in association with abruption of placenta, IUFD and preeclampsia can be attributed to posterior reversible encephalopathy syndrome, retinal vasculopathy and optic neuropathy. SRD is associated in 1-2 % case of severe preeclampsia.[2] Preeclampsia occurs after 20 weeks of gestation secondary to abnormal placentation and can lead to thrombocytopenia and placental abruption.[5] Complete abruption of placenta and IUFD can lead to series of events which can cause release of thromboplastin into maternal circulation and hence activating extrinsic coagulation pathways leading to disseminated intravascular coagulation (DIC).[6] The microangiopathic hemolytic anemia and vascular endothelial damage via platelet adhesion and activation would facilitate diffuse, generalized fibrin formation.[7] Also, severe maternal bleeding leads to loss of blood and circulatory volume causing hypovolemia. Fujii et al reported two cases of SRD with placental abruption and preeclampsia.[8] Komoto S et al reported one case with bilateral SRD associated with SRFM in a case of PIH secondary to blood loss, PIH induced choroidal vasoconstriction and increased blood viscosity.[9] However, in present case, associated abruption placentae and IUFD can cause transient DIC in addition to other factors

resulting in choroidal ischemia. Though bilateral involvement is commonly seen, unilateral involvement may occur as reported by Fujii A et al in one of their cases.[8]

The vulnerability of macular choriocapillaris to embolism can be attributed to the fact that terminal arterioles supplying the area are usually short and vertical, as compared to peripheral choroid where series of dichotomous branching occurs before terminating into vascular bed.[10] This choroidal hypoperfusion leads to damage to overlying RPE layer resulting in decrease pumping of fluid from the sub retinal space into the choroid causing SRD. SRFM is probably due to migration of larger molecule, such as fibrin and fibrinogen, due to intense choroidal hypoperfusion, extensive RPE damage and increased choroidal hyperpermeability.[9] As the triggering stimulus for preeclampsia and transient DIC is resolved (removal of placenta and delivery of stillborn baby in this case), choroidal perfusion and RPE function gradually improves resulting in resolution of SRD and SRFM with visual recovery. In our case, at four weeks after the onset, SRD and SRFM resolved completely with restoration of the EZ, however mild RPE irregularity persisted. The management of SRD in such case is conservative with good prognosis.

Conclusion –

SRD with SRFM is a rare association with preeclampsia, abruptio placenta and IUFD secondary to acute intense choroidal ischemia. It occurs due to transient DIC, blood loss and vasospasm. Early diagnosis, prompt management with serial observations can result in favorable outcome. To the best of our knowledge, there have been no reports about SRD and SRFM associated with preeclampsia, abruptio placentae and IUFD.

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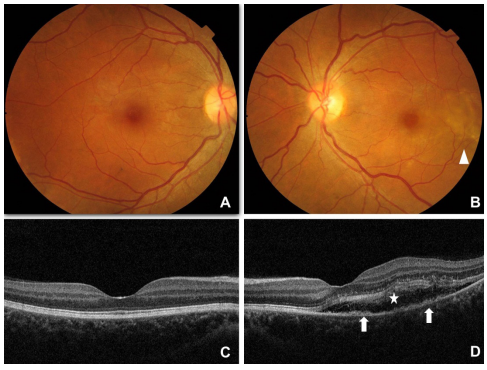

Figure 1

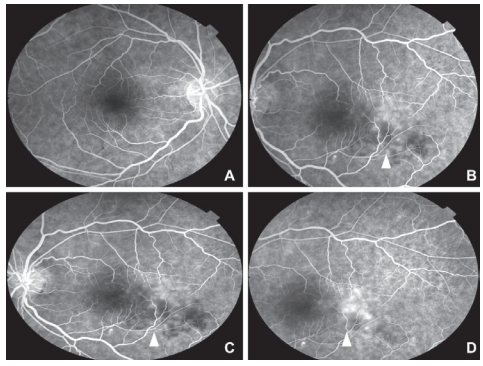

Figure 2

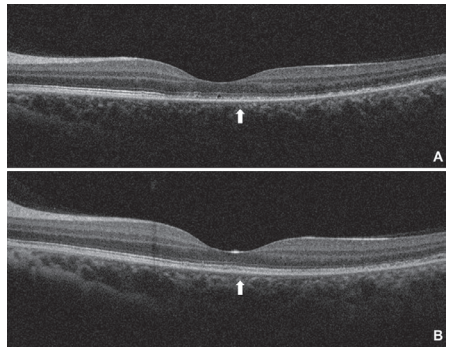

Figure 3

LEGEND TO FIGURES –

Figure 1 - Fundus photographs showing normal findings in the right eye (A) while serous retinal detachment (SRD) and subretinal fibrin-like material (SRFM) (white arrowhead) in the left eye (B). Optical coherence tomography scans exhibiting normal foveal contour in the right eye (C) while SRD with SRFM (white star), irregular retinal pigment epithelium (white arrow) and hyper-reflective dots in the left eye (D).

Figure 2 – Fundus fluorescein angiography exhibiting normal filling pattern in the mid phase of right eye (A) while delayed choroidal perfusion with late leakage in the early (B), mid (C) and late phase (D) of left eye (white arrowhead).

Figure 3 - At day 10 of onset, optical coherence tomography scan showing resolution of serous retinal detachment and restoration of external limiting membrane with irregular ellipsoid zone (EZ) and retinal pigment epithelium layer (RPE) (white arrow). At 4 weeks, there is restoration of EZ while mild irregularity in RPE persists (white arrow).

ONE MINUTE TIP

HOW TO READ MACULAR OCT

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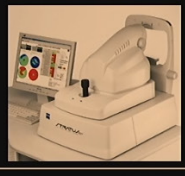
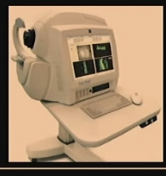
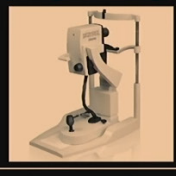
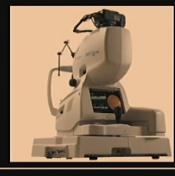
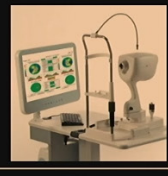
Optical coherence tomography (OCT) is an imaging modality first developed in the early 1990s. It uses light beams and their pattern of back-scattering to build high resolution cross-sectional images of the retina and optic nerve. This allows for rapid imaging of living tissue with micron-level resolution to depths of several millimeters without any ionizing radiation

Over the years with the evolution of technology it has been able to provide ophthalmologist with faster scan imaging, higher resolution and enhanced depth analysis which has been put in the table A

Commerical OCT Devices

	Carl Zeiss Meditec	Carl Zeiss Meditec	Heidelberg Engineering	Topcon	Optovue
					
Device	**Stratus OCT**	**Cirrus HDOCT**	**Spectralis OCT**	**3D-OCT**	**iVue**
Type	Time-domain	Spectral-domain			
Scan Speed	400	27,000	40,000	20,000	26,000
Axial Resolution	10 μm	5 μm	7 μm	6 μm	5 μm
Device		**Cirrus HDOCT**	**Spectralis OCT2**	**3D-OCT-1**	**Avanti**
Type		Spectral-domain			
Scan Speed		68,000	85,000	50,000	70,000
Axial Resolution		5 μm	7 μm	6 μm	5 μm

While scanning the macula on OCT it helps ophthalmologist in

1Macular OCT imaging in Glaucoma

2 Macular OCT imaging in Retina

I will be discussing the latter part (retina)

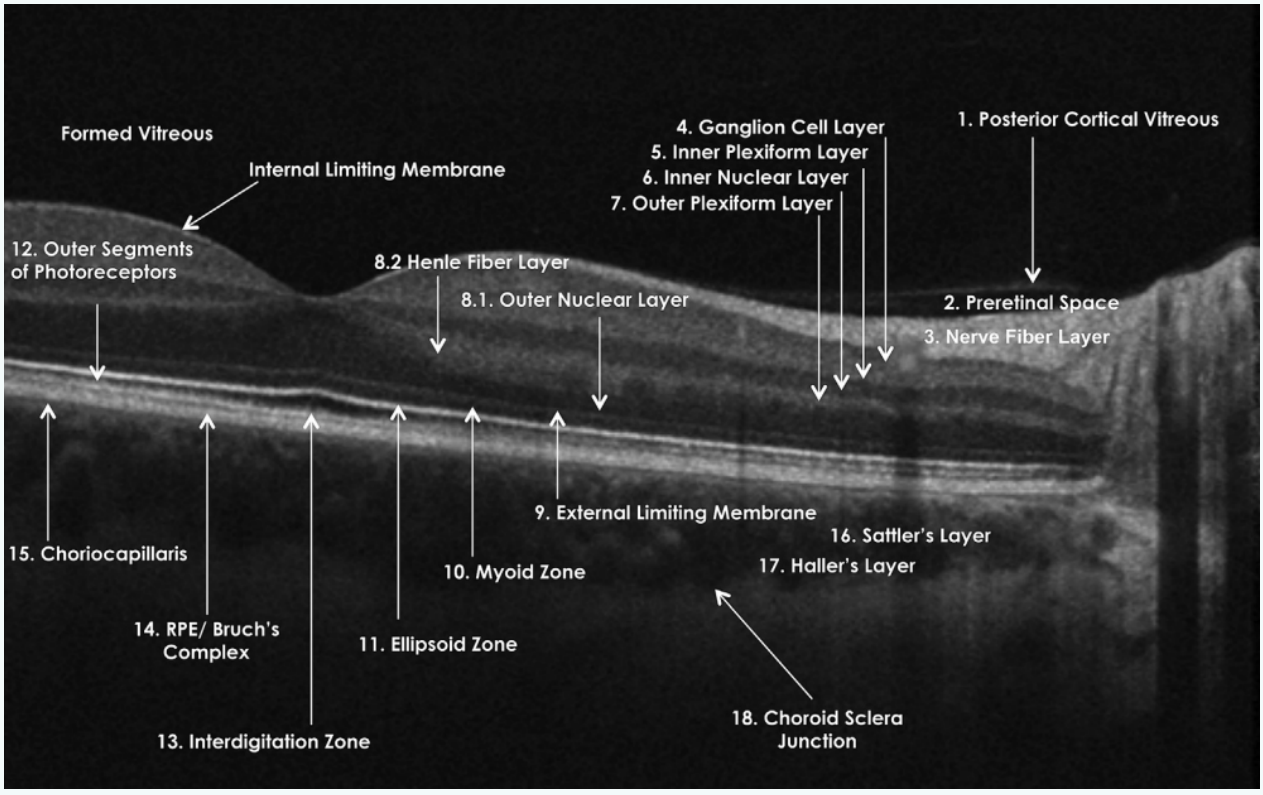
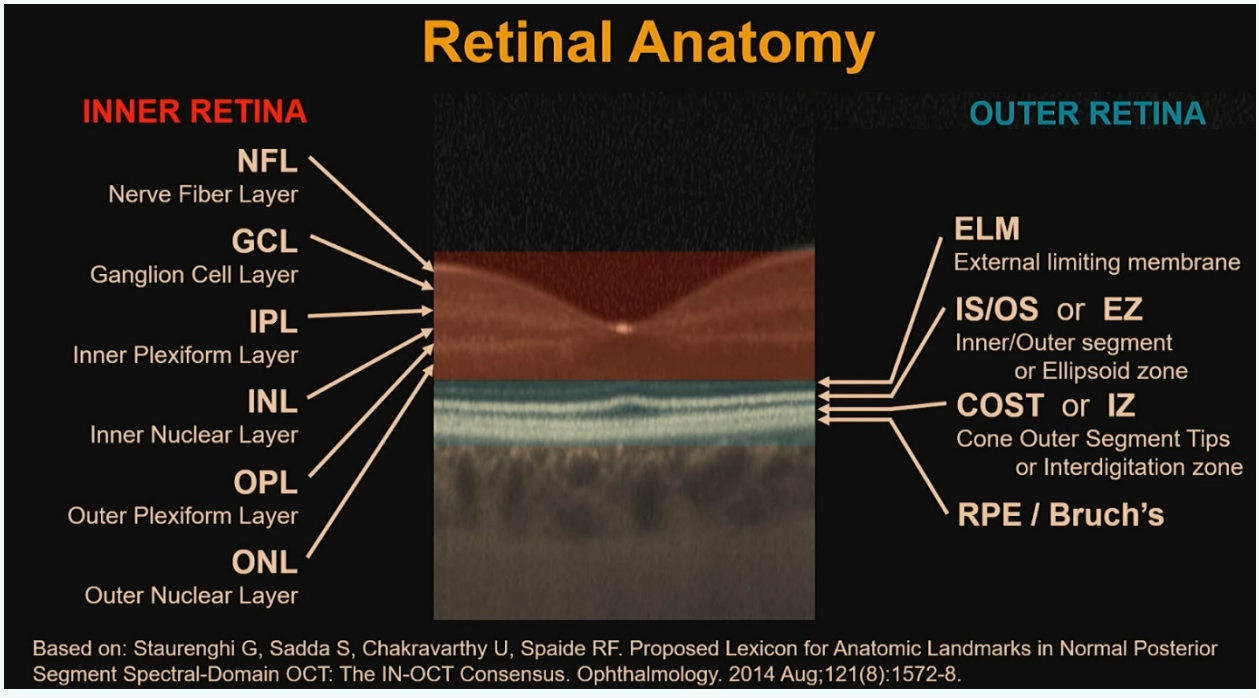


Figure 1 ,2 shows an SD-OCT image from a healthy patient. The important layers from the inner to outer retina are: vitreous (VIT), nerve fiber layer (NFL), ganglion cell layer (GCL), inner plexiform layer (IPL), inner nuclear layer (INL), outer plexiform layer (OPL), outer nuclear layer (ONL), external limiting membrane (ELM), inner segment ellipsoid zone (EZ), retinal pigment epithelium (RPE) and choroid (CHR).



By practicing these Eight steps, you will be better able to accurately (and comfortably) use Macular OCT to your clinical advantage

1. Know what's normal and familiarize yourself with anatomy

Familiarize yourself with how a normal OCT look. There should be nine layers of the retina itself, alternating in light and dark bands. We also want to consider the vitreoretinal interface above and the choroid below. The foveal depression should be symmetric and centrally located in the image.

Layers

Inner retina

- Nerve fiber layer
- Ganglion cell layer
- Inner plexiform layer
- Inner nuclear layer

Outer retina

- Outer plexiform layer
- Outer nuclear layer
- External limiting membrane
- Photoreceptor IS/OS (inner segment/outer segment)
- Retinal pigment epithelium

Become familiar with the layers of the retina and what "normal" looks like. For an in-depth classification of retinal layers pertaining to OCT, be sure to check out Proposed Lexicon for Anatomic Landmarks in Normal Posterior Segment Spectral-Domain Optical Coherence Tomography. (Fig 1,2)

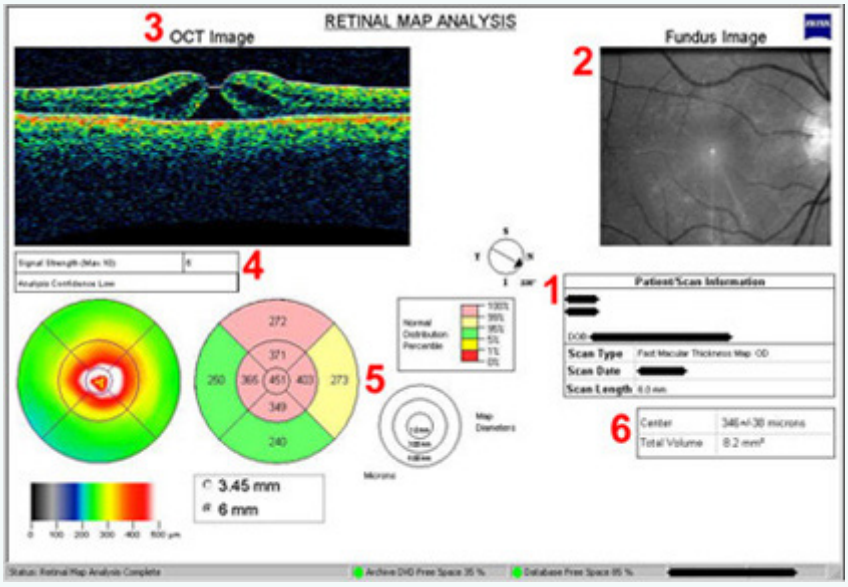
Here are a few key areas to keep in mind:

- Inner segment/outer segment (IS/OS) junction — i.e., inner segment ellipsoid zone. Loss of this thin reflective line represents a defect in the photoreceptors and typically has a profound negative impact on the patient's vision.
- Retinal pigment epithelium layer. You can see irregularity in primary RPE abnormalities such as pattern dystrophy. For example, it's common to see hyperreflective focal elevation of the RPE in the case of abnormal accumulation of material such as drusen or blood. In addition, hyporeflectivity under the RPE can represent a pigment epithelial detachment. Hyporeflectivity between the RPE and retina is a common indication of subretinal fluid.
- Outer retina. Fluid often accumulates in this location in macular edema related to inflammation, diabetes and vascular abnormalities such as vein occlusion or choroidal neovascularization. Nuclear layers are generally hyporeflective, and plexiform layers are generally hyperreflective in both the outer and inner retina.
- Inner retina. The inner retinal anatomy's integrity is a good reflection of the eye's vascular status. In chronic ischemic situations, you'll notice atrophy.

2. Check the name and date of image: This may seem obvious, but just as we learned in medical school when reading any imaging test like an X-ray, you must confirm that you have the right patient! The date is equally as important .

3. Center the image: Make sure the OCT image is centered on what you are interested in imaging.

4. Review the OCT image: This is the main reason why you order the OCT. Ensure that the image is in the center and the lines are placed appropriately. These lines are what generate the thickness measurements by the automated software.



5. Confirm the signal strength: If there is something in the visual axis (i.e. cataract, corneal opacity), it may prevent the OCT from obtaining a good quality imageyou want a signal strength of at least 6

6. Review the central subfoveal mean thickness (CSMT): This grid map gives you nine areas with measurements of thickness. The center grid number refers to the central subfoveal mean thickness, which is the circular area (1 mm diameter) centered around the center point. Compared with other measurements (versus center point thickness and total volume), it has a higher reproducibility and stronger correlation. Recent studies have shown that it is the preferred metric for following retinal thickness measurements (i.e diabetic macular edema).
7. Double-check the center point thickness (CPT) and its deviation: This box gives both center point thickness and total volume. The CPT is based on the intersection of six radial line scans (compared with 128 macular thickness measurements made for the CSMT. A deviation greater than 10 percent of the CPT implies an unreliable measurement.

8. Correlate clinically and make the diagnosis.
We often joke about the "clinical correlation required" comment on radiology reads; however, considering the clinical information at hand can often give you important tips to help with the diagnosis. Here are some guidelines to help you with your diagnosis:

Epiretinal membrane

- Locate pathology: Above the internal limiting membrane (ILM), so in the vitreoretinal interface
- Describe pathology: Hyperreflective membrane causing traction on the inner surface of the retina
- Correlate clinically: Older patient, often with a posterior vitreous detachment (PVD), describing central distortion

Diabetic macular edema

- Locate pathology: In the inner nuclear layer (INL) and outer plexiform layer (OPL)
- Describe pathology: Hyporeflective cysts
- Correlate clinically: Diabetic patient, central vision blurry, often worse with worsened diabetic retinopathy

Central serous retinopathy

- Locate pathology: Outer retina, under RPE

- Describe pathology: Hyporeflective serous fluid
- Correlate clinically: Typically younger male, high-strung patient under stress or taking steroid medications or hormones

Drusen/Age-related macular degeneration (AMD)

- Locate pathology: Outer retina, Bruch's/RPE
- Describe pathology: Hyperreflective bumps
- Correlate clinically: Usually older, fair-skinned patients, minimal or no symptoms

Common Disease Presentations

Let's review some clinical images to demonstrate a few important SD-OCT findings.

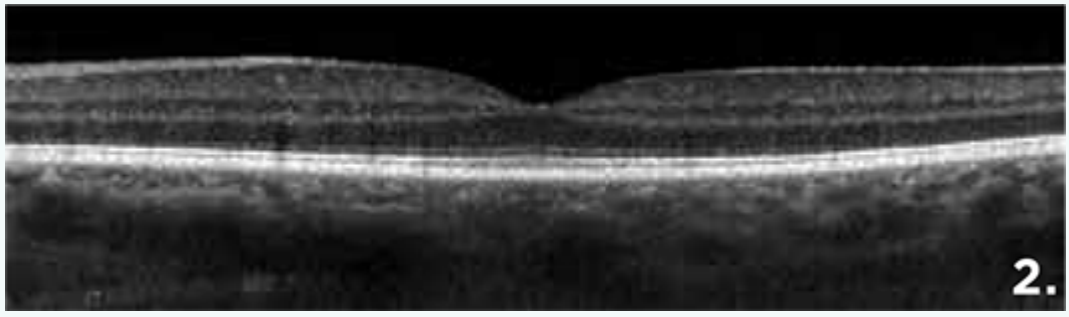


Figure 2 is an image from a healthy patient. Note the foveal contour and CMT (250 µm).

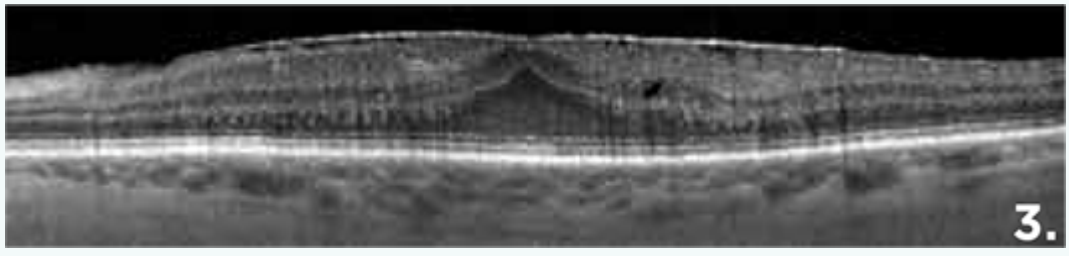


Figure 3 demonstrates an epiretinal membrane on the surface of the retina with a thickened CMT and loss of foveal contour.

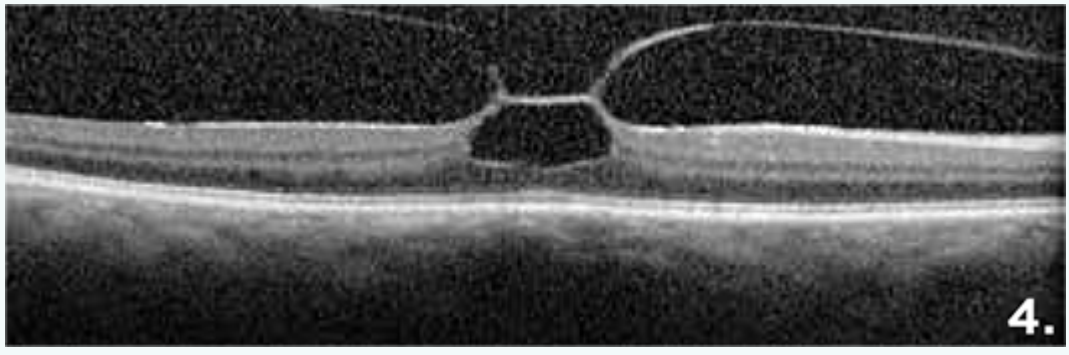


Figure 4 depicts vitreomacular traction with an inverted foveal contour and underlying cavitation.

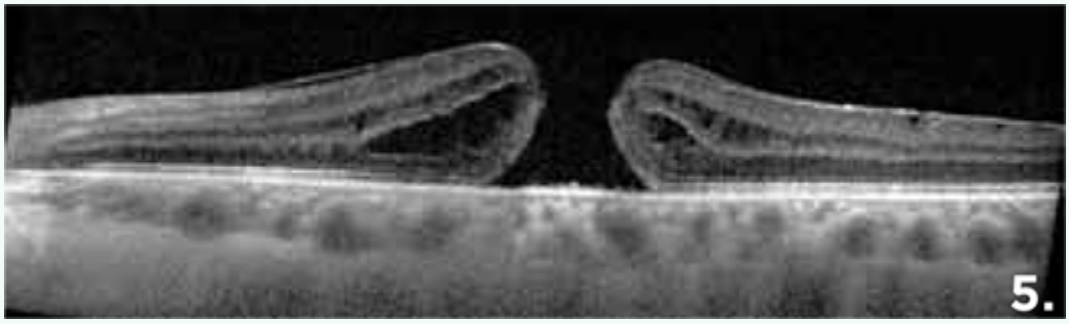


Figure 5 demonstrates a full-thickness macular hole.

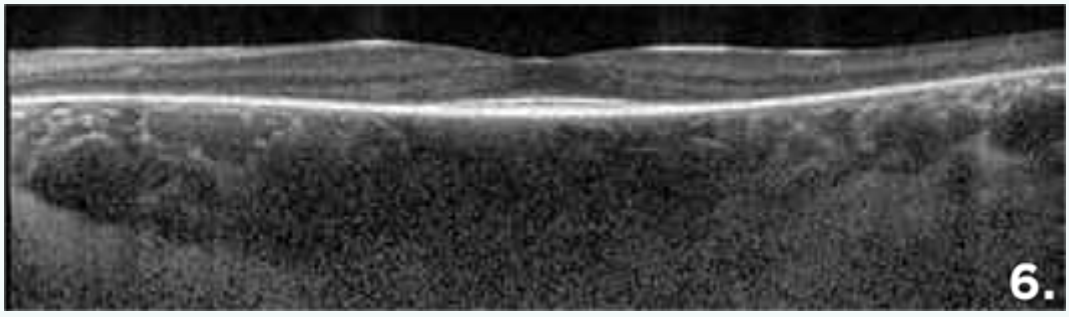


Figure 6 shows severe thinning of the outer retina with sparing of the central EZ. This patient has retinitis pigmentosa.

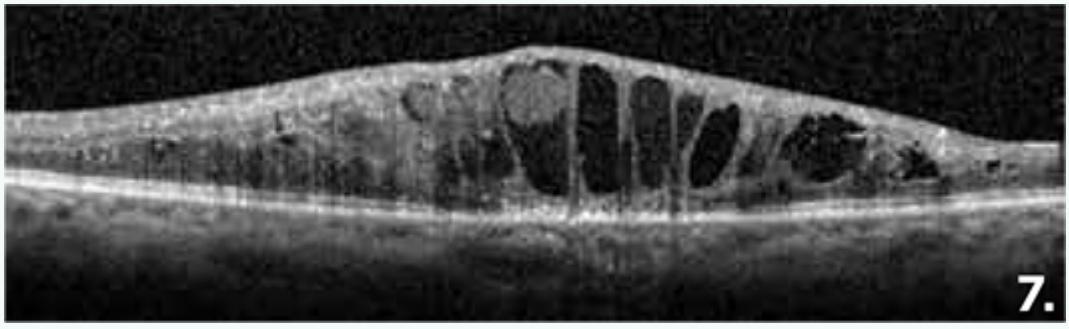


Figure 7 shows diffuse diabetic macular edema with EZ loss at the fovea.

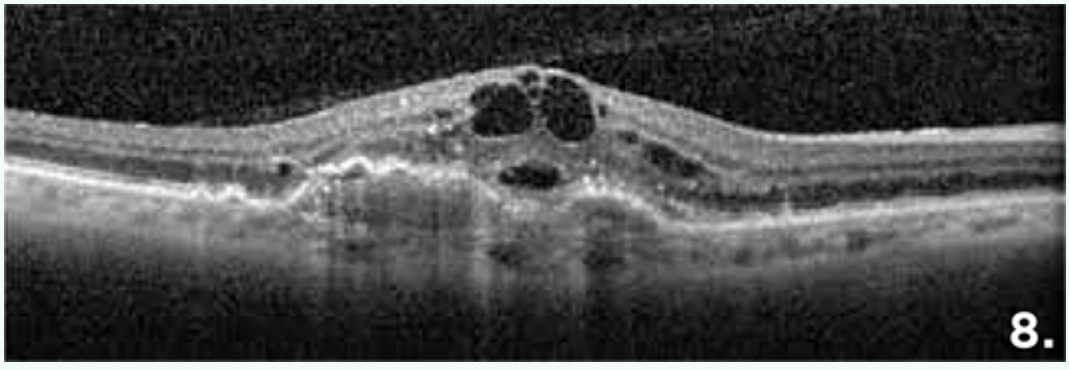


Figure 8 shows a choroidal neovascular membrane in a patient with exudative age-related macular degeneration. Note the fibrovascular pigment epithelial detachment, retinal edema and subretinal fluid.

ONE MINUTE TIP

COLOR CODED FUNDAL DRAWING

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INTRODUCTION

The history of standardizing color-coding in Ophthalmology dates back to 1969 when Schepens described the graphic documentation of retinal disorders.[1] This is extremely vital in the field of Ophthalmology, where the eye being a small and compact structure has innumerable microscopic findings for which documentation with standardized color-coding becomes vital for quality patient care.[2] It serves as standardized means of communication between ophthalmologists, educating trainees and postgraduates, and helping in the easy follow-up of disease course over a period.

PREREQUISITES OF DRAWING

Examination table, indirect ophthalmoscope, 20 D lens, scleral depressor, colored pencils (red, blue, green, yellow, brown, and black), fundus drawing chart, and eraser. Fundus drawings are made on a standard fundus chart, Amsler–Dubois chart, which contains three concentric circles – the innermost circle represents the equator, the middle circle represents the ora serrata, and the outer one is the junction between the pars plana and plicata [Figure 1]. The radial numbered in Roman numerical is used to designate the location and extent of the lesions in clock hours. The macula is drawn centrally, and the optic nerve head is located

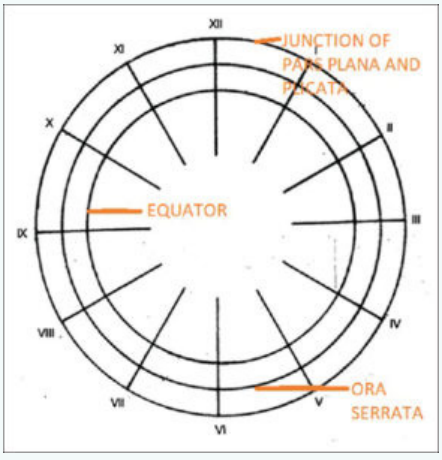


Figure 1: Amsler–Dubois chart

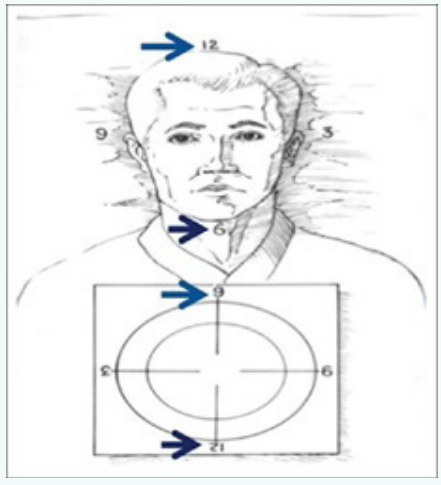


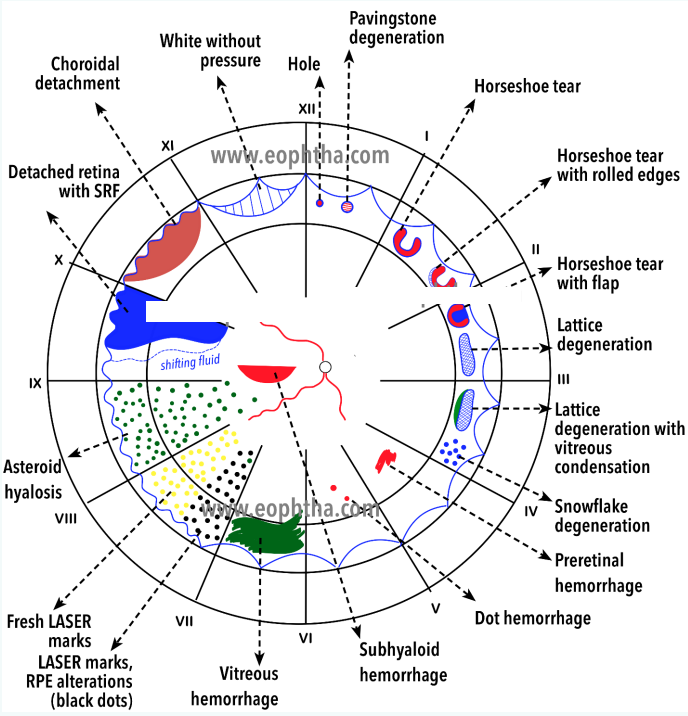
Figure 2: Amsler chart compared to patient position

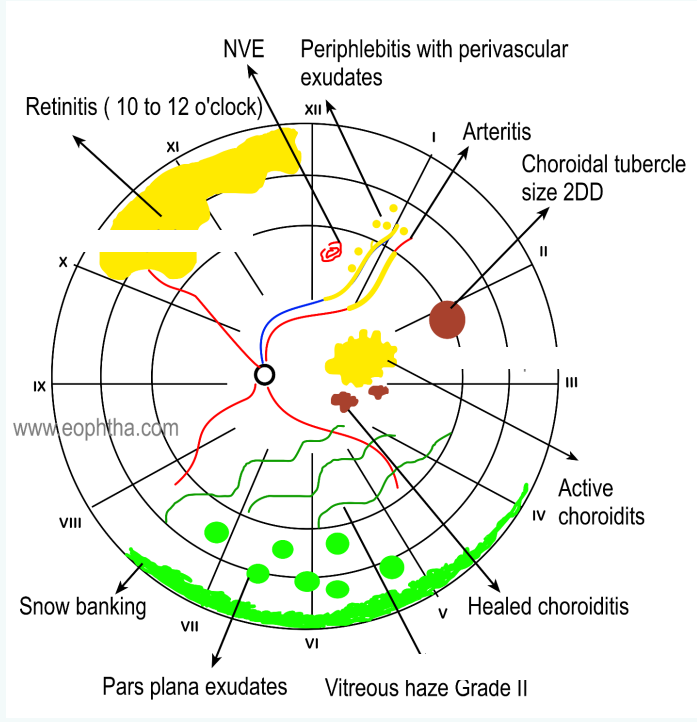
COLOR CODING

Detailed color-coding guidelines for posterior segment schematic representation

Color	Representation	Posterior Segment Structure or Pathology
Red Color	Solid	Retinal arterioles, elevated neovascularization, vascular abnormalities/anomalies, vascular tumors, vortex veins, attached retina, hemorrhages (pre-retinal and intraretinal, sub hyaloid), the open interior of conventional retinal breaks (tears, holes), the open interior of outer layer holes in retinoschisis, and macular edema
	Dot	Macula
	Cross-lines	Open portion of a giant retinal tear or large dialyses, inner portion of CRA, inner portions of thinned retinal areas, open portions of retinal holes, and the inner layer of retinoschisis
Blue	Solid	Detached retina, retinal veins, outlines of retinal breaks and Ora Serrate, meridional, radial, fixed and circumferential folds, VR traction tufts, retinal granular tags and tufts, an outline of flat neovascularization, outline of lattice degeneration, outline of thin areas of the retina, and intraretinal cysts
	Cross-lines	Inner layer of retinoschisis, white with or without pressure, detached pars plana epithelium anterior to the separation of ora serrate, rolled edges of retinal tears (curved lines)
	Stippled/circular shapes	Cystoid degeneration
	Interrupted blue line	Outline of change in area or folds of the detached retina because of shifting fluid
Green	Solid	Opacities in media, vitreous hemorrhage, vitreous membranes, hyaloid ring, intraocular foreign body, retinal operculum, cotton wool spots, and outline of elevated neovascularization
	Stippled lines or dots	Asteroid hyalosis, frosting or snowflakes on cystoid degenerations, retinoschisis, or lattice degeneration
Brown	Solid	Uveal tissue, pars plana cysts, ciliary processes, striae ciliaris, pigment beneath the detached retina, an outline of CRA beneath the detached retina, pigment epithelial detachment, outline of posterior staphyloma, malignant choroidal melanomas, edge of buckle beneath the detached retina, and choroidal detachment
Yellow	Solid	Intraretinal edema, intraretinal subretinal hard exudate, deposits in the retinal pigment epithelium, detached maculae, retinal separations, post cryotherapy or laser retinal edema, drusens, long and short ciliary nerves, and venous sheathing
	Stippled lines	Drusens

Black	Solid	Edge of buckle beneath the attached retina, outline of CRA, hyperpigmentation because of the previous treatment with Cryotherapy or Diathermy, naevi, sclerosed vessels, outline of long and short post ciliary vein and nerve, pigment in the choroid, or pigmented epithelial hyperpigmentation in areas of the attached retina, pigment in detached retina, pigmented demarcation lines at the attached margin of a detached retina or within the detached retina





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SURGICAL SECRETS

Scleral Fixated IOL – An Overview of Various Techniques

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1. In cases of trauma, complicated cataract, metabolic or inherited conditions such as Marfan's syndrome, and pseudoexfoliation, intraocular lens placement becomes a surgical challenge due to insufficient capsular support. Options for IOL placement in such cases include:

oAnterior chamber intraocular lenses (ACIOLs)
oIris-fixated posterior chamber intraocular lenses (PCIOLs)
oScleral-fixated intraocular lenses (SFIOLs)

2. SFIOL involves sutured/sutureless fixation of the IOL to the sclera, closely approximating the normal anatomic position of a within-thebag IOL.
3. It Does not affect pupillary dilation like iris claw lenses and has lower incidence of complications such as corneal endothelial decompensation, pupillary block, angle closure, and ocular inflammation compared to other options.
4. SFIOL is contraindicated in cases of:
    - o Uveitis
    - o Co-existing retinal detachment
    - o Extensive lattice degeneration with a family history of RD or major retinal disease
    - o Uncontrolled glaucoma and extensive limbal scarring

5. Pediatric Consideration

Various case series of children implanted with mainly sutured SFIOLs have shown promising results.[1,2,3] It is advisable to wait until at least 6 years of age for the eyeball to reach adult measurements. However, children are more prone to complications, so the need for long-term follow-up and second surgery if required should be well explained to the parents.

6. Timing of the Surgery

SFIOL can be done as a primary procedure in a planned manner or later once the media have cleared and the wound has healed. Studies have not shown any difference in the visual outcome or complication rates in either primary or secondary implantation.[4,5] The decision should be based on the surgeon's comfort level and the patient's needs.

7. Anterior Vitrectomy vs Pars Plana Vitrectomy Approach

The visual outcomes are similar with both approaches, but the chances of late complications such as retinal detachment are higher with the anterior vitrectomy approach.[6] A pars plana approach allows the surgeon to better deal with complications such as lens matter dislocation.

8. Sutured or Sutureless Fixation

The sutured method is more durable and provides stable long-term results. The sutureless fixation is easier and quicker, avoiding suture-related complications. However, it is a recent technique and lacks long-term data.

9. Choice of IOL

For sutured IOLs, a one-piece, rigid IOL with eyelets on the haptic for passing the suture is chosen. It has a larger optic size of 6 mm and a larger overall diameter of 13.5 mm, ensuring good centration. For sutureless technique, a 3-piece foldable lens is used.

10. Suture Considerations

A non-absorbable double-armed, 10-0 polypropylene (prolene) suture with both straight needles is preferred. However, suture degradation and late dislocation of IOL are reported in approximately 27.9% .[7] Nowadays, 9-0 prolene is the preferred choice. Polyetrafluoroethylene (Gore-tex) suture is also being used for fixation, but long-term data is still awaited.

11. Surgical Principles
    - IOL should be placed correctly in the sulcus.
    - The haptics should be placed and fixed exactly 180 degrees apart.
    - The corneal center and points of haptic fixation should be in a straight line.

These principles are essential to achieve a well-centered SFIOL.

12. Surgical Technique of Sutured SFIOL
    - AB INTERNO: Originally described technique where the sutures are passed inside-out at the ciliary sulcus.[8] However, this is a blind procedure and not practiced.
    - AB EXTERNO: The preferred method in which needles are inserted from outside to inside after marking the ciliary sulcus. This gives a more stable IOL position. [9]

13. AB EXTERNO Techniques of Sutured SFIOL
    - Two-point fixation:
        - 3 and 9 o'clock positions are marked on the cornea.
        - Partial-thickness scleral flaps 3x3 mm are made at these points.
        - The straight needle of either 10-0 or 9-0 polypropylene suture is inserted at 1mm posterior to the surgical limbus, a 26G hollow needle is inserted from the opposite side, and the polypropylene needle is docked into it and taken out from the opposite side.
        - The suture is externalized through a superior corneal tunnel, cut in the middle, and each end is tied to one haptic of the IOL.
        - The IOL is placed posterior to the iris with the haptics resting in the ciliary sulcus while the sutures are pulled out. Each suture is tied onto itself to secure the haptics to the sclera.

    - Four-point fixation:
        - After making scleral flaps, the double-armed 10-0 polypropylene suture is cut in the middle, and one needle is passed under the scleral flap 1mm behind the limbus and 1mm on one side of the radial mark. This is docked into a 26G needle inserted through the superior corneoscleral tunnel.

- o After taking out the needle, the suture is passed through one of the eyelets on the haptic.
- o Now the 26G needle is passed into the eye under the flap of the same side, 1 mm from the limbus and 1mm from the radial mark on the other side. The 10-0 needle is inserted into the eye through the superior section and docked into the 26G needle and taken out under the flap.
- o The same steps are repeated on the opposite side.
- o The IOL is inserted in the eye, positioned in the sulcus, and the sutures are tied, and knots are buried. The scleral flaps can be sutured or glued.

14. Surgical Techniques of Suture-less SFIOLS

Gabor and Pavlidis described the exteriorization of the haptics of a three-piece IOL for a sutureless SFIOL fixation.[10] This technique is further modified by Agarwal et al. [11] , popularly known as the glued IOL.

- The glued IOL technique:
  1. Partial-thickness scleral flaps are created 180 degrees apart.
  2. 25 G sclerotomies are created under the flaps 1 mm posterior to the limbus.
  3. A 26G needle is used to create intrascleral tunnels of 2-3 mm on either side, parallel to the limbus but facing in opposite directions.
  4. A 3-piece foldable IOL is inserted through the corneoscleral section.
  5. Intraocular end-gripping forceps is introduced through the sclerotomy on one side, and the leading haptic is exteriorized, followed by the trailing haptic.
  6. Haptics on each side is then inserted into the scleral tunnels and are tucked intrasclerally.
  7. Fibrin glue is applied to seal the scleral flaps and the conjunctiva.

Recently, Yamane et al. described a modification of the glued IOL technique which has become very popular. [12]

1. After the haptics are exteriorized, their tips are cauterized. The heat shortens the length of the haptic outside the eye and creates a bulbous flange whose diameter is larger than the thickness of the haptic.
2. This flange lodges within the substance of the sclera and is completely covered by the conjunctiva, thus fixating the IOL to the sclera without any sutures, flaps, or glue.

15. Complications

Complications may include hypotony, increased intraocular pressure, intraocular bleeding, macular edema, suture erosion, haptic exposure, IOL tilt, IOL dislocation, glaucoma, retinal detachment, and endophthalmitis.

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SURGICAL SECRETS

OCT Angiography (OCTA)

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Introduction

Optical coherence tomography angiography (OCTA) can image the retinal vasculature in vivo, without the need for contrast dye. This technology has been commercially available since 2014, however, much of its use has been limited to the research setting. Over time, more clinical practices have adopted OCTA imaging. This technology allowed for the visualization of retinal microvasculature in vivo. Prior to OCTA, fluorescein angiography (FA) and indocyanine green angiography (ICGA) were the mainstay modalities for retinovascular visualization. These imaging modalities generated a 2-dimensional en face view of the retinal vasculature and did not allow for the individual visualization of retinal capillary plexuses. OCTA offered in vivo visualization of the retinal microvasculature in a depth-resolved fashion, without the need for time-consuming dye administration. Prior to its commercial release, OCTA was available as a research tool. Investigational groups explored the utility of OCTA in a wide range of ocular pathologies including age related macular degeneration (AMD), diabetic retinopathy (DR), and uveitis. In this article, I have tried to give a glimpse of clinical utility of OCTA that I have come across in practice. I had started working on OCT-A in 2020 and the images in this article are from the equipment that is Topcon Triton Plus OCT installed at Sankara Eye Centre, Indore.

Functioning principle

OCTA was developed as an extension of OCT imaging. OCTA technology utilizes motion contrast to detect blood flow. When two successive images of a scene are taken, stationary objects will not change, while moving objects will become apparent. OCTA captures successive A-scans at the same retinal location, and each scan capture is separated by a brief lapse in time. As light is reflected back, a difference in signal will be detected between the two scans. This difference is due to motion between the scans and is termed decorrelation signal. Because the retina is a static tissue, the decorrelation signal is attributed to the movement of blood through the retinal vasculature. Thus, a decorrelation map is generated that mirrors the flow of blood in the back of the eye, rendering a representation of its vascular networks.

Advantages

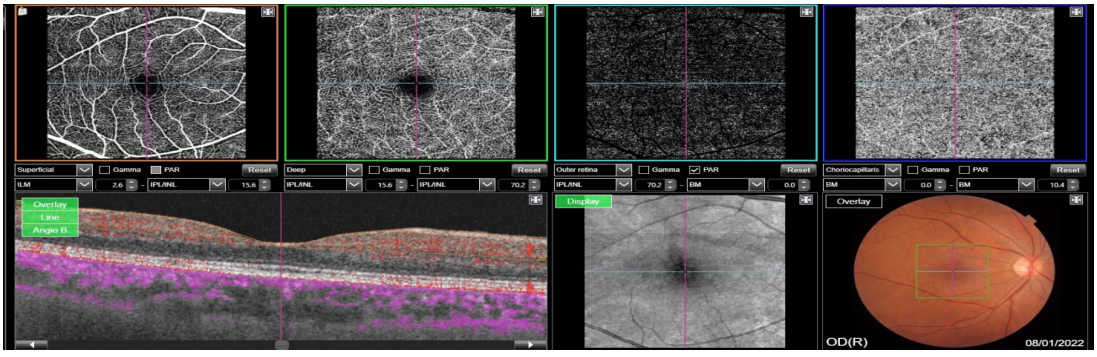
OCTA generates high-contrast images that are not obscured by dye leakage from vessels, leading to more pronounced definition of retinal vasculature. This dye–free imaging modality does not expose patients to the risks associated with contrast dye, which range from mild allergic reactions to anaphylaxis. Patients that have relative contraindications to dye imaging, including those with renal failure or poor intravenous access, can undergo OCTA imaging without limitations. OCTA imaging is fast and thus useful for patients that require recurrent

vascular imaging, such as those being treated with vascular endothelial growth factor antagonists (anti-VEGF) for wet AMD or diabetic macular edema (DME). Though FA remains the gold standard for imaging of the retinal vasculature, OCTA was found to be of great utility for detecting complications of retinal vein occlusion, DR and AMD

Limitations

Beyond its many advantages OCTA has a number of limitations. As aforementioned, flow detection on OCTA requires scanning a single location multiple times. This increases imaging times, especially for larger scan areas. Furthermore, device hardware and software are highly variable between OCTA manufacturers.Clinicians must be aware that such differences exist, as they can affect imaging results. When possible, patients should be imaged on the same device to assure accurate comparison between visits. Though dye-free OCTA imaging is safer for patients, it has some disadvantages. OCTA cannot visualize dye leakage, a common landmark of inflammatory vascular pathology and a sign of blood-retinal barrier breakdown. OCTA cannot provide information on transit time or vascular filling either.

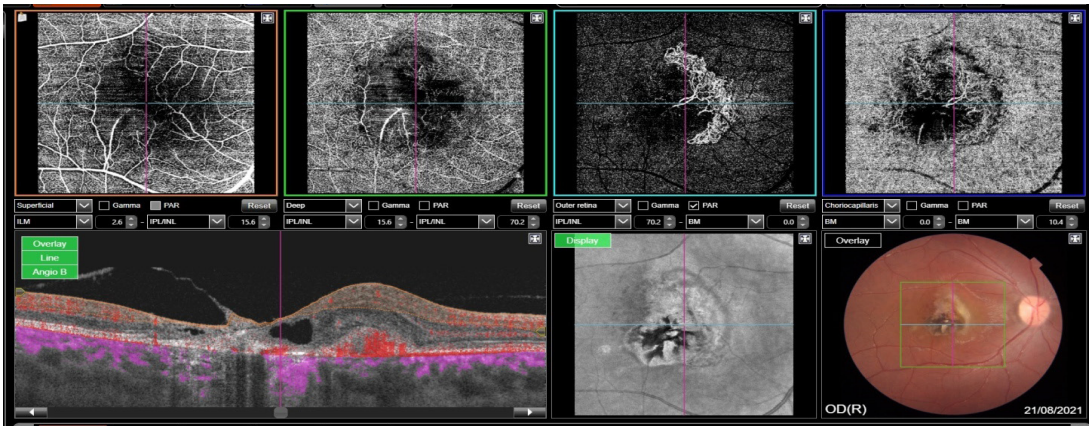
Reading OCTA imaging output



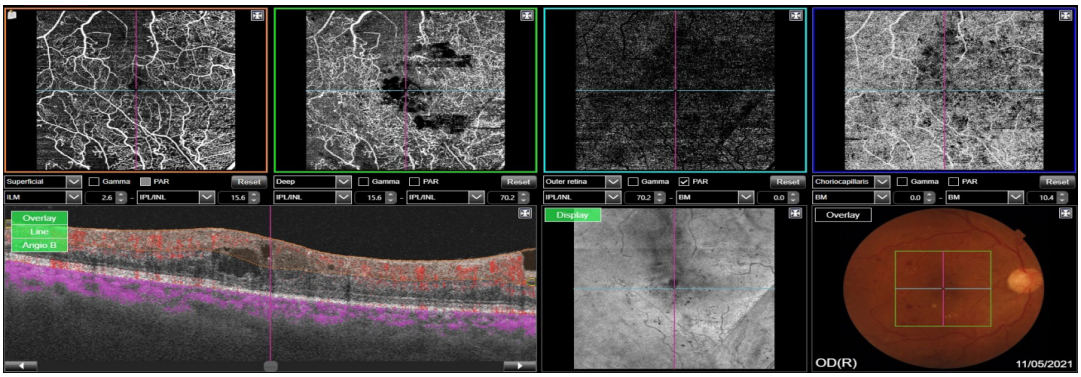
The report is generated in different slabs along with the fundus imaging and B scan. Superficial and deep retinal plexus slabs are helpful in assessment of primarily retinal pathologies like DR and RVOs. Outer retinal and choriocapillaries slabs are helpful in assessment of AMD and other subretinal and choroidal pathologies. B scan with flow signal (marked red) gives an indication of flow direction which is helpful in identifying active vasculature.

Case examples

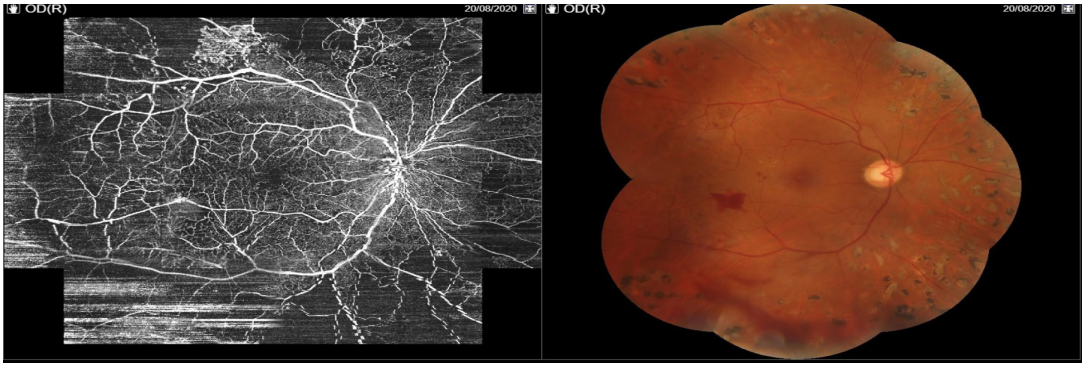
Active CNVM (seen on Outer retinal slab) nasal to subfoveal scarring in a 19 year old female



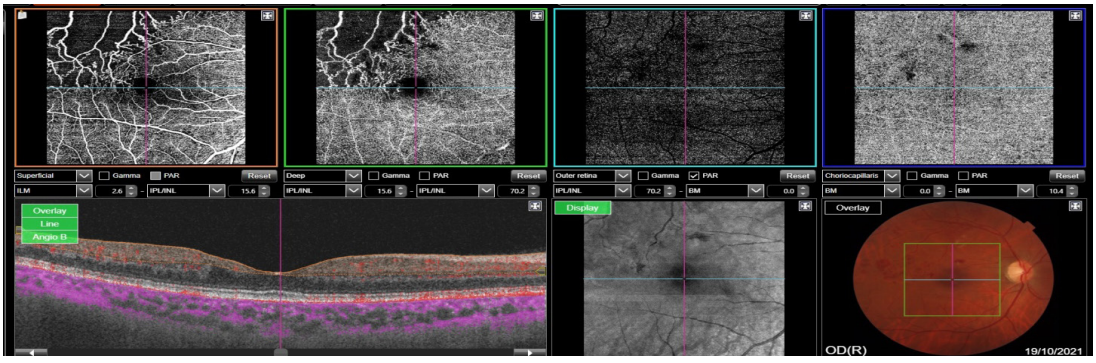
Macular ischemia associated with DME visible in superficial and deep retinal plexuses



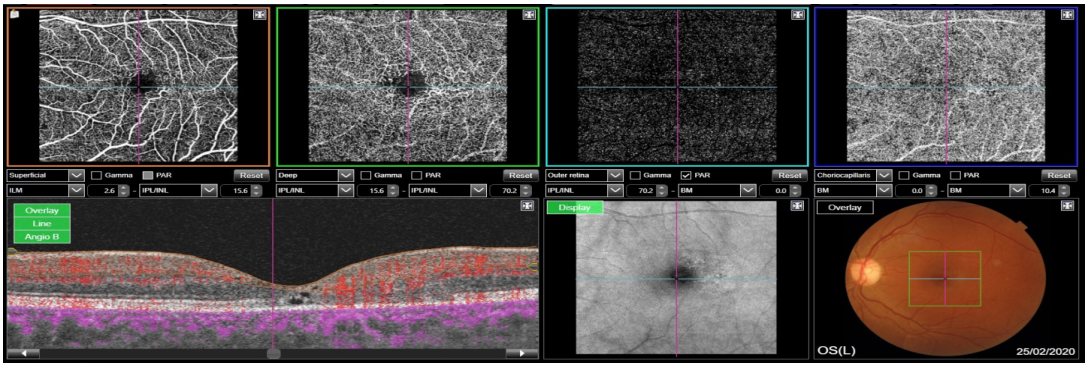
OCTA Montage picture of PDR with peripheral CNP and multiple NVE



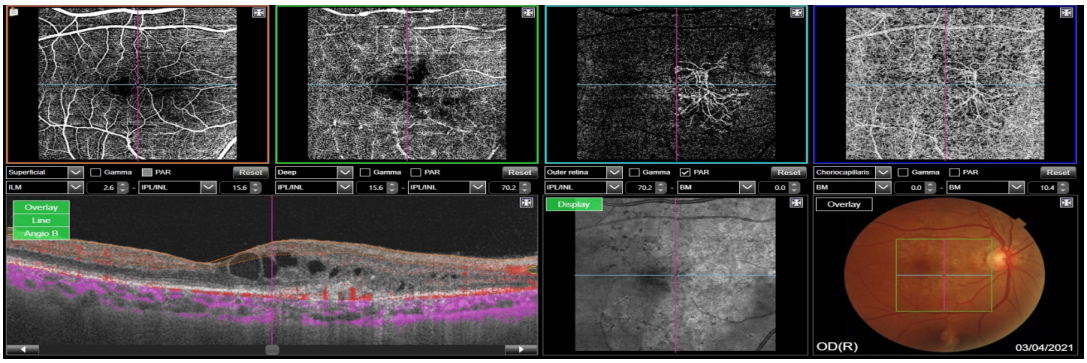
BRVO with ischemia sparing fovea



MacTel (parafoveal telangiectasia) with right angled vessels in deep retinal plexus



Exudative AMD with CNVM seen in outer retinal slab and flow signal on B scan



Conclusion

OCTA is a useful non-invasive clinical tool for fast assessment of retinal and subretinal vascular pathologies. Artefacts and inability to image leakages limit its use as a replacement of conventional angiography.

SURGICAL SECRETS

Micropulse Laser: What is it and its Uses?

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Micropulse is a laser modality that divides a continuous stream of laser into several short bursts separated by pauses (off time).

The whole pulse duration is called a pulse envelope, which is divided into 100 micropulses, and each micropulse has an on time and off time with a ratio depending on the duty cycle (the ratio between the on time and the whole micropulse) (Fig1). For example, for a 200 ms envelope which is divided into 100 micropulses, the micropulse duration on time will be 0.1–0.3 ms for a 5% and 15% duty cycle, respectively.[1]

Benefits

- Micropulse laser causes stimulation of a biological response that restores the proper pump function of RPE cells, resulting in enhanced and rapid absorption of subretinal fluid.[1]
- Subthreshold response produces an RPE- confined photothermal effect with sparing of the neurosensory retina.
- Temperature rise will be below the threshold for coagulation, in contrast to the conventional continuous-wave laser which leads to coagulation of the inner retina. Thus, directly heated tissue remains viable and can produce a stress response that induces production of beneficial anti-angiogenic intracellular biological factors.
- Confluent burns can be made in area with SRF.
- Yellow laser (577nm) is poorly absorbed by xanthophyll pigment, so it can be used for treatment over the fovea without risk of post-laser scotoma.
- Being absorbed by both melanin and oxyhemoglobin, 577 nm laser causes less scatter during treatment and allows use of less power and shorter pulse durations as compared to 810nm laser.[2]

1. Titration: Because of the variation of fundus pigmentation across different individuals, among patients of different ethnicities, it is advisable to titrate the laser parameters before performing treatment on or near the foveal region.
2. Test burns: begin with low power and subsequently step-up power. Burns are applied to healthy retina in the extra- foveal region (e.g., outside fovea but within vascular arcades) or on nasal side of the optic disc until threshold burn is achieved (i.e., a barely visible whitening of retina over the laser spot).
3. The threshold laser burn settings then are converted to treatment settings according to a preset algorithm.

It should be noted that currently there is no international consensus in titration nor in the subsequent conversion to treatment laser settings. Hence the confusion. However numerous groups are trying to come to a working consensus. Author has developed and followed a standard set of parameters that are seemingly working and reproducible.

Moreover, since multiple MPL systems are available that deliver laser pulses of different wavelengths (e.g., 577 nm in Supra Scan and 810 nm in Iris Medical Oculight Six Laser), standardization of titration becomes difficult.

SMPLT in retinal diseases other than CSCR

1. Diabetic macular edema (DME)
2. Severe NPDR or Proliferative Diabetic Retinopathy
3. Macular edema Secondary to RVO

1. DME

– Anti-VEGF injections unequivocally are the gold standard therapy for treating DME. Second line therapy are intravitreal steroids in the form of either a self-eluting dexamethasone implant, or intravitreal triamcinolone acetate (IVTA)[3]
– The visual acuity gain after anti-VEGF therapy in DME is generally superior to SMPLT.[4] However, treatment with intravitreal injections is expensive and requires multiple injections. SMPLT is much cheaper, and it would be economically rational to include it in the process of treating DME.

Moisseiev et al. concluded that combination therapy—anti-VEGF (in this case, ranibizumab) plus SMPLT—significantly reduced the burden of intravitreal injections, 2.6 for combined group versus 9.3 for mono therapy groupat the end of the follow-up.[5]

– EURETINA accepts the use of SMPLT for DME as an option in cases with early diffuse retinal edema and good visual acuity.[3] SMPLT is rarely effective in treating edema larger than 400 µm.
– Central DME with CMT is the only clinical scenario for SMPLT.[6] Patients who can be considered for SMPLT in this category are:

• Patients reluctant to receive intravitreal treatment.
• Patients in whom antiVEGF therapy is contraindicated due to systemic reasons.
• Patients who can not bear financial burden of intravitreal therapy.

To fully understand role of SMPLT in DME, clinical trials comparing anti-VEGF therapy and SMPLT are required to gain a better understanding of treatment response and thus clear-cut consensus protocols can be formulated.

SMPLT in Severe NPDR or Proliferative Diabetic Retinopathy

– Classic panretinal photocoagulation (PRP) has a proven track record of long-term stability of PDR regression, with most eyes remaining stable for as long as 15 years after laser with no additional treatment. Its efficacy has been corroborated by DRS and ETDRS studies.[7],[8]
– Disadvantage of PRP is that it results in peripheral visual field defects and general visual field narrowing, which could be troublesome, especially for younger patients.

– AntiVEGF therapy has been tried as an alternative to PRP in the landmark Protocol S with functional success; however, it requires regular intravitreal injections and numerous follow-up visits, which is

difficult to execute in a real-worldscenario. Field loss has been shown to occur with antiVEGF therapy as well. [9],[10]

- Subthreshold micropulse pan retinal photocoagulation has also been tested as an alternative to classic PRP in a couple of trials. Jhingan et al[11] studied a group of 10 patients (20 eyes) with severe NPDR or low-risk PDR, in which each eye was randomized to receive either classic PRP or SMPLT PRP. At nine months, only one eye in SMPLT group progressed to Vitreal hemorrhage and then required classic PRP. Eyes in classic PRP group had reduced Low Contrast Visual Acuity (LCVA), visual field index, and scotopic b/a ratio in comparison to the SMPLT group. However, the changes were not clinically significant.

Conclusion: SMPLT for PDR must be considered as experimental and further randomised clinical trials with long term follow-up are required to ascertain its long-term stability and safety as an alternative to classic continuous wave (CW) PRP.

SMPLT in Macular edema Secondary to RVO

- Theoretically, SMPLT should reduce inflammation and improve the fluid elimination due to RPE stimulation (production of cytokines and boosting RPE pump). In RVO, inflammatory processes and vascular hyperpermeability are probably more intense in comparison to other retinal vascular diseases.
- That could explain why results of SMPLT of macular edema secondary to RVO are not always satisfactory. In these cases, SMPLT is probably not potent enough to overweigh the benefits of intravitreal steroid or antiVEGF treatment.
- Current research is divided on the role of SMPLT in ME. due to RVO.

a) Research by Parodi et al[12],[13] show significantly better results with the use of intravitreal triamcinolone or bevacizumab than SMPLT.

b) A study by Terashima et al[14] concludes that combination therapy of IVR and SMPLT is effective to treat branch retinal vein occlusion cystoid macular edema, by decreasing the frequency of IVR injections while maintaining good visual acuity.

c) Özurt et al[15] showed that IVR or SMPLT treatment for ME due to BRVO were not found superior to each other for reducing ME and increasing VA for 1-year period.

Conclusion: Further research is needed to understand role of SMPLT in the treatment of ME in RVO. However, patients with limited macular edema or patients that were disqualified from intravitreal therapies due to systemic conditions or financial reasons could still benefit from SMPLT.

Micro pulse laser for Chronic CSR

Central serous chorioretinopathy (CSCR) is a macular disorder which characteristically has a serous detachment of the neurosensory retina due to leakage at level of retinal pigment epithelium (RPE), secondary to hyperpermeability of choriocapillaris. It typically occurs in young, middle-aged males. The male to female ratio is 3: 1; females with CSR tend to be older. It is also associated with exogenous steroid use in any form, Helicobacter pylori infection, renal dialysis, systemic hypertension, psychological stress, pregnancy, and sleep apnoea syndrome.

Patients complain of symptoms such as blurred vision, decreased contrast sensitivity, metamorphopsia, micropsia, decreased colour vision.

Clinical Course

It can be divided into acute and chronic forms.

- Acute CSCR: self-limiting course with spontaneous resolution within 3-4 months for most patients.
-Chronic CSCR: The definition of the duration of chronic CSCR is inconsistent; some studies have used 3 months and some studies have used 6 months. However, in most studies, it is defined when disease course lasts for more than 6 months with no documented resolution, and is characterised by longstanding SRF, chronic RPE changes, RPE tracts, photoreceptor shedding, sub retinal fibrin.

Micro pulse laser studies for CSR

Name of group	Laser	No. of sessions	Sample size (eyes)	Follow up (In months)	Resolution parameter	Complete resolution (%)	Partial resolution (%)
Lanzetta et al[16]	810 nm Diode	1-5	24	14	Anatomical SRF	71%	4%
Ricci et al[17]	810 nm Diode ICG Enhanced	1	7	12	Anatomical SRF	71%	29%
Chen et al[18]	810 nm Diode	1-3	26	6	Anatomical SRF	55%	20%
Yadav et al[19]	Yellow micropulse(577nm)	1	15	2	Anatomical SRF	40%	60%

Stepwise process of doing SMPL therapy

Treatment options

1) Observation: Initial treatment of choice in acute CSR. Exceptions to observation is reserved for few special situations such as, patients with marked initial drop of vision, those with massive subretinal fluid, monocular patients, and those seeking rapid visual improvement due to occupational causes. In

such situations, treatment with Subthreshold micropulse yellow laser (SMPYL) can be considered.

2) PDT: Considered gold standard treatment but due to low availability of the dye, Verteprofin, and need for specialised laser, the role of PDT has diminished greatly.`
3) Laser
   a) PDT: Considered gold standard treatment but due to low availability of the dye, Verteprofin, and need for specialised laser, the role of PDT has diminished greatly
   b) Continuous wave 532nm thermal laser:

-Used to be treatment of choice for extra foveal leaks. However, it can cause central or paracentral scotomas, contrast sensitivity loss, accidental foveal damage, retinal distortion, or choroidal neovascularization.
- Even though focal laser potentially seals leak seen on fluorescein angiography (FFA) and leads to resolution of subretinal fluid, it does not alter the choroidal hyperpermeability, thus the risk of recurrence remains.

   c) Subthreshold micro pulse yellow laser: Has demonstrated to be effective in reducing SRF, improving BCVA, along with option of re treatment. It is covered in detail later in the article.

4) Anti VEGF: Use case only in situations where there is a secondary CNVM. In cases without CNV, there is no evidence of elevated VEGF levels in plasma or aqueous, suggesting that anti-VEGF agents would be ineffective for the treatment of acute or chronic CSCR.
5) Oral medications: Reserved for patients who decline first line invasive treatments like laser.

a) Mineralocorticoid Receptor Antagoists
-Spiranolactone 50mg daily
-Eplerenone 25- 50mg daily

Both reduce SRF & choroidal thickness, but VA change is not statistically significant. Spironolactone may be superior but has greater side effects: hyperkalemia, gynecomastia, reduced libido, menstrual changes.[20]

b) Rifampicin: Since it is an inducer of cytochrome P4503A4, it was postulated that induction of cytochrome P450 3A4 may increase metabolism of endogenous steroids leading to improvement of CSCR manifestations. However, due to a risky side effect profile and lack of constructive evidence to support its usage, it is reserved as a last-ditch effort option at discretion of treating Ophthalmologist. Patient should be explained clearly the risk/benefit ratio of undergoing such treatment.

c) Methotrexate (MTX): Studies by Kurup et al[21] and Abrishami et al[22] indicate statistically significant improvement in BCVA, CMT, SRF, and total macular volume. No MTX-associated toxicity was detected in both studies. MTX may have a role in the treatment of chronic CSCR as evidenced by these results. However, these were uncontrolled studies and therefore randomised, controlled clinical trials are warranted to further investigate the effects of methotrexate in these patients.

There are three main types of subthreshold therapy currently available.

1. Subthreshold micropulse laser (MPL) therapy
2. Endpoint management (EPM) laser therapy
3. Selective retinal pigment epithelium therapy (also known as Selective retina therapy, SRT)

We shall limit this article to micro pulse therapy since the authors have no first-hand experience of the other two mentioned modalities of treatment.

Example:

Pulse ON: 0.1 ms (100 µs)
Pulse OFF: 1.9 ms (1900 µs)
Period (T): 2.0 ms (2000 µs)
Duty Cycle (%) : 5% (01/2.0 x 100)

SMPL, Sub Threshold Micropulse Laser, can be delivered in the green (532nm), yellow (577nm) or Red (810nm) range. The SMPYL (Sub Threshold Micropulse Yellow Laser) is believed to be the best wavelength for the Micropulse effect. At Vision Plus Eye Centre, Noida we have been using the SMPYL for past 7 years and here are a few examples of cases treated by us.

CASE1: CHRONIC CSCR ,5 Years, 51 years male doctor, had earlier received eplerenone without response.

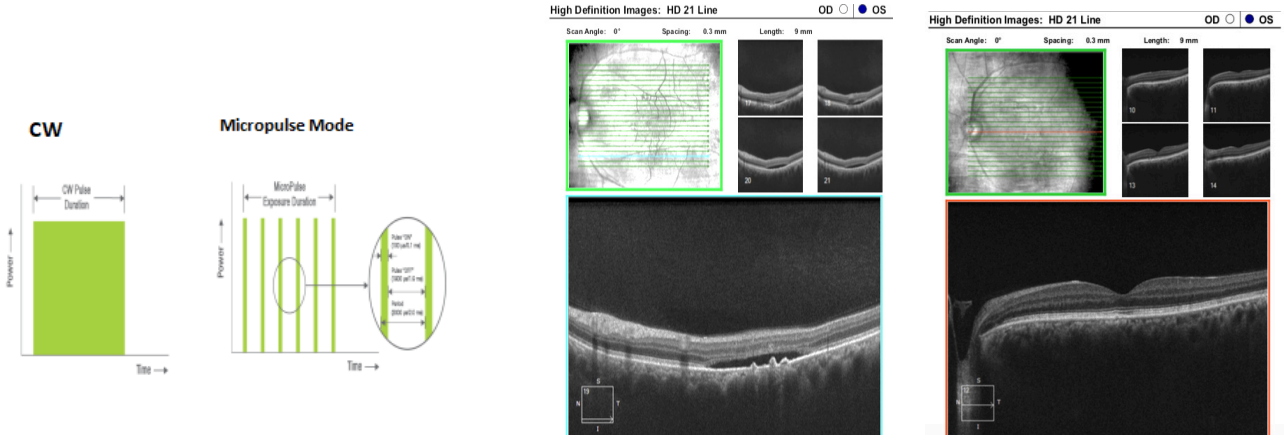

Fig 1

Fig 2a (OCT showing SRF and PEDs) 51 Years male, Chronic CSCR for 5 years, No CNVM on OCTA, VA 6/9p; Treated with MPLT 5% DC (6th Nov 2018)

Fig 2b (OCT Post Treatment) Feb 2019, SRF completely resolved VA 6/6 no metamorphopsia. Patient has received three MPLT from 2018-2022 and is doing well.

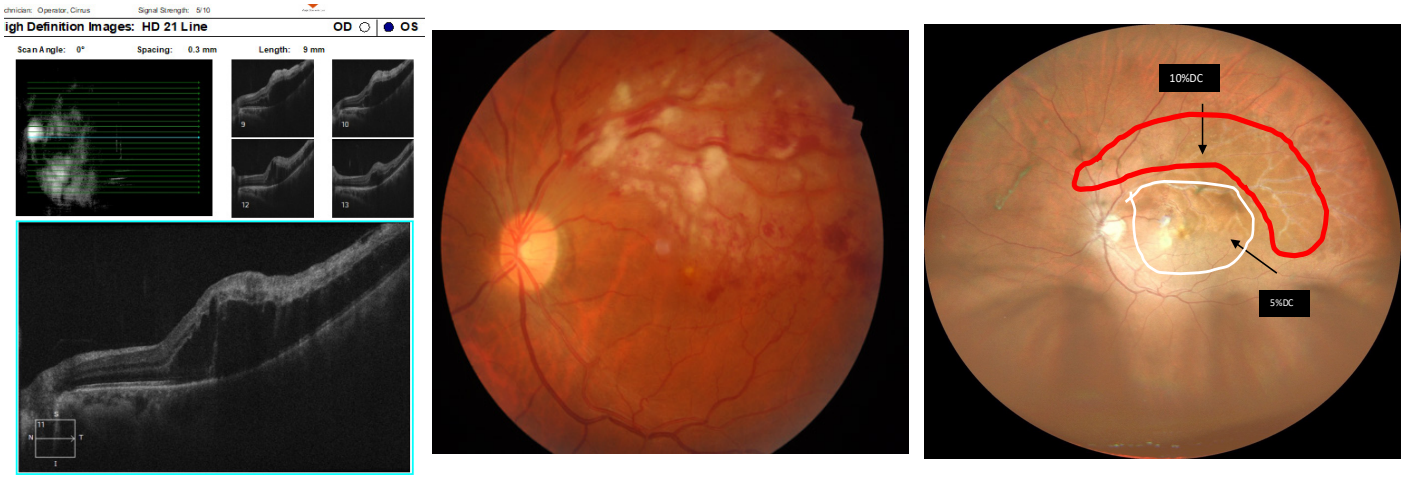

Fig 3 a & b OCT and Fundus photo : 39 years male , 7th May 2020, LE VA 6/36, RE 6/6; LE Upper temporal BRVO post Covid Vaccination.Received intravitreal Injection Accentrix 13th May 2020 and second Inj July 2020

Fig 3c: Fundus photo showing the pattern of SMPYL received: combination of 5%DC to the macular area and 10%DC to the extramacular area of CNP (Capillary Non-Perfusion) in September 2020

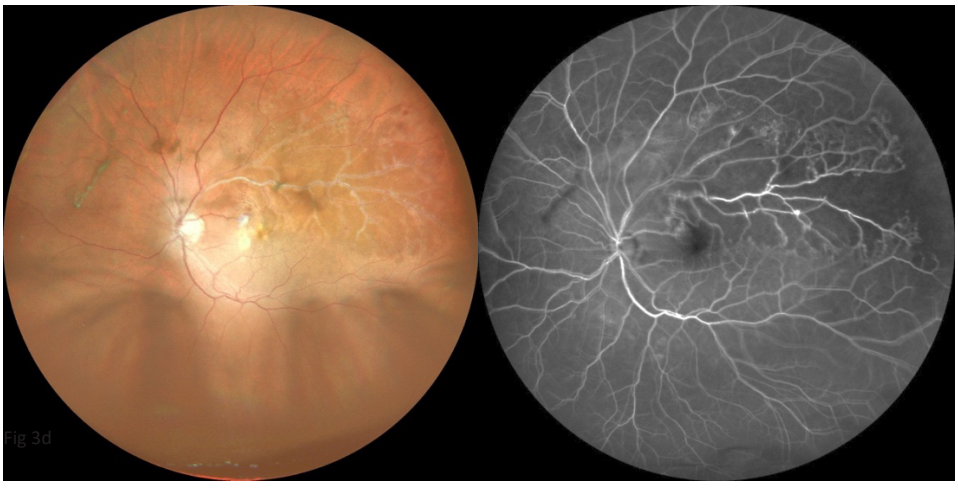

Fig 3d: Ultrawide Field Fundus photo and UWF FFA (July 2022) showing UTBRVO with a Large area of CNP, A small NVE and no macular edema 2 years after SMPYL Patient underwent repeat SMPYL with a Focal CW laser to the NVE. He is doing well.

Case 1: In cases of CSCR we have had near 100% success with SMPYL.
Case 2: demonstrates the ability of SMPYL to help in reducing the number of intravitreal injections of anti VEGF and avoiding the damaging CW laser therapy. This will possibly transform into a long-term preservation of field (along-term study in this regard is underway).

We now have a series of cases of Chronic CSCR, BRVO with Macular edema, Persistent DME with and without ERM, Persistent macular edema with ERM when patient is not willing for surgery, Post Radiation macular edema, Retinitis Pigmentosa associated CME and CRVO with Macular edema; and early PDR where patient has consented for SMPYL with a combination of CW Yellow laser treatment. We are in the process of collating our data and will be publishing soon. Our experience with Chronic CSCR is particularly very satisfying with near 100% success in the reduction of SRF. In the absence of availability of PDT in India SMPYL is an excellent substitute. I feel larger studies from multiple centers will help this treatment becoming widely acceptable.

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SURGICAL SECRETS

Scleral Buckling

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In the present era of cutting-edge technology does scleral buckling surgery still holds any significance? Development of modern vitrectomy machines with sophisticated sensors, software and 23, 25 and 27-G state of the art cutters have taken the management of retinal detachment to the next level. The whole story commenced with **Jules Gonin**[1] presenting his original concept in 1931 that retinal detachment is caused by retinal break and reattachment could be achieved by accurate localization of break, inducing inflammation around it with diathermy and by simultaneous drainage of Subretinal fluid. Even though **Deutschmann**[2] and **Biett**[3] in 1933 reported freezing the retinal break with solid carbondioxide to obtain adhesion, the instrumentation for diathermy application were more satisfactory and results more reliable during that period.

Around 20 years later in 1953, **Custodis**[4] revolutionized the scenario of retinal reattachment surgery by introducing an extraocular detachment technique ***Custodis Operation***, which eliminated the three major hazards namely: scleral perforation for SRF drainage, intravitreal injection to restore vitreous volume and scleral dissection to preserve the scleral wall from scleral necrosis.

It was **Lincoff** who reestablished the Custodis operation which was being abandoned because of high rates of extraocular infection and endophthalmitis as reported by **Schepens et al**[5]. Lincoff also experimented with carbondioxide applicators borrowed from dermatologists and along with McLean modified the Cooper- Linde Neurosurgical unit to create a device suitable for eye. By replacing the diathermy with cryosurgery and substituting irritative polyviol with silicone sponge **Lincoff and McLean**[6] re-popularized the local buckling. Subsequently ***Schepens technique***[7] introduced solid silicone structures buried in lamellar scleral dissection along with cryopexy.

Scleral buckling still is a gold standard in uncomplicated Rhegmatogenous RD and PVR upto C1. Specially in cases who cannot take prone position like medical conditions, morbidities and mentally challenged patients.

The current approach to Scleral buckle surgical technique is highly individualized but the basic principle remains universally accepted, namely:

1. Identification and accurate localization of breaks
2. Creation of chorio-retinal adhesion
3. Appositional closure of retinal breaks by indentation of RPE to relieve dynamic traction

With the advancements and improving results and safety of vitrectomy the indications for scleral buckling has been shrinking. Currently it is done for following indications:

- Fresh retinal detachments
- Retinal detachments with PVR not more than C1
- Phakic retinal detachments
- High myopia

- Absence of PVD / young patients
- RRD with breaks anterior to equator
- RRD secondary to retinal dialysis

Scleral buckling is not preferred in retinal detachments with

- Scleromalacia or severe scleral thinning
- Extensive PVR
- Giant Retinal Tears
- Hazy media (vitreous haemorrhage or significant cataract)
- Pseudophakia with PCO
- Retinal breaks posterior to equator
- Previous Glaucoma surgery is a relative contraindication

Standard steps of scleral buckling surgery includes ***anaesthesia*** which is peribulbar or general anasthesia as per the surgeon preference and the established standard of care. It may require supplementation during the procedure which is usually subtenon injection.

Lid and skin preparation is done with 10% povidone iodine solution followed by isolation of lashes with adhesive drapes. Ocular surface is irrigated with povidone iodine 5% solution, which is left in conjunctival cul de sac for 1 minute.

Peritomy and isolation of muscles

360 peritomy with relaxing cuts at 3 and 9 o clock position is done with blunt tipped Wescott scissors. Blunt tipped Stevens' tenotomy scissors are used to expose sclera by going under and spreading between the recti and relaxing the tenons wherever required.

Muscle isolation is done using muscle hook and fenestrated muscle hook or reversed needle. Muscle attachments and overlying tenons may be gently stripped using a cotton tip applicator. Care must be taken not to damage recti and their ligaments and vortex veins.

Indirect ophthalmoscopy & localization of breaks

Thorough Indirect ophthalmoscopic examination is done to localize all the breaks and latices. Identification of primary break. During localization eye is stabilized by the assistant who holds the traction sutures 180 degrees apart. The breaks are marked with forceps tip or Gass and O'Conner localizer then finalized by sterile marker pen or flat tip diathermy.

- Small retinal breaks – single spot
- Large HST – three spots; most posterior & two anterior horns
- Multiple breaks (circumferential buckle) – most posterior break in each quadrant is marked
- Dialyses – two ends & posterior extent of mid-point
- Bullous RD – marking is done
    - From anterior margin and indentation is moved posteriorly
    - Alternatively - SRF drainage before localising breaks

Treatment of retinal breaks

The aim is to create adhesion between RPE and retina which can be accomplished by using diathermy, cryotherapy or laser. Each technique is effective and selection is influenced by experience, preference of surgeon and features of individual case.

Diathermy is used infrequently because of the adverse effects like shrinkage, necrosis of sclera, penetration of globe resulting in retinal hole and intraocular hemorrhage. Cryotherapy is now the preferred technique since

trans-scleral application is possible without significant damage to sclera/long posterior ciliary artery and nerve and vortex veins. Cryotherapy is applied as contiguous lesions completely surrounding the break under direct visualization by IDO. The end point of Cryotherapy is distinct whitening of neuro sensory retina.

- Avoid treatment of bare RPE within large tear which causes dispersion of RPE cells and breakdown of blood-ocular barrier resulting in PVR, vitreous haze, CME, choroidal detachment, exudative retinal detachment and macular pucker.
- D-ACE technique- localisation and cryotherapy are delayed until after drainage of SRF and intraocular injection of air.

Laser photocoagulation requires clear media and causes less inflammation and dispersion of RPE. Confluent medium intensity burns surrounding the break preferably extended to the ora is the desired end point. Trans-scleral diode laser (DioPexy probe) can be used to achieve chorioretinal adhesion under IDO visualization. Treatment is started in attached retina to establish power and duration settings before treating detached retina.

Scleral buckling

The aim of buckling is *to relieve vitreous traction and close retinal breaks*. A buckle may be exoplant (sutured on the surface of sclera) or implant (placed beneath flaps after lamellar scleral dissection). Buckling materials may be as follows:

- Gelatin (hydrolized collagen)
- Solid silicone
- Silicon sponge

Solid silicon rubber is the preferred material. Handling difficulties are encountered after hydration of gelatin material. Silicon sponge can result in uneven indentation when used for encirclage and carry a higher risk of infection and erosion through the conjunctiva. A segmental buckle maybe circumferential or radial. A radial buckle is used for large horse shoe tear to counter tendency of fish-mouthing which is common with circumferential buckle.

Suture placement

5-0 non absorbable suture on spatulated micropoint needle should be used with half scleral thickness depth in each bite for secure fixation. Needle tip should be visualized at all times while passing through the sclera. Posterior bite should be at least 3 mm posterior to the break. In circumferential buckles, mattress sutures are passed parallel to the limbus. The anterior sutures are placed along the line of ora and the rule of 1.5 times the width and 3 mm posterior to the retinal break is followed as with radial buckles. The posterior edge of the break is to be placed at the center of the buckle. The suturing technique may be modified to avoid injury to intra-scleral portion of the vortex vein. Care must be taken in very soft eyes to avoid perforation.

Encircling elements

Solid silicone or sponge can be used for encircling which are secured with horizontal mattress sutures/ lamellar scleral pockets. Width and location of the encircling element depends on the pathology and the placement is such that the posterior edge of the pathology (Lattice degeneration/ vitreo retinal adhesion or vitreous base) should be placed on the crest of the buckle. Encircling buckles are indicated in following situations:

- Multiple breaks in different quadrants
- Aphakia
- Pseudo phakia
- High myopia
- Extensive lattice degeneration
- PVR greater than Grade B
- RD caused by trauma
- RD without a recognizable retinal break
- Giant retinal tears

Disadvantages being need for 360º peritomy, myopic shift and need for SRF drainage.

- Encircling should be avoided in patients with Sickle Cell Disease
- Excessive tightening may predispose to fish mouth phenomena and anterior segment ischemia

No.240 (2.5 mm) solid silicon band is preferably used encircling material which is secured with a Silicone sleeve or tantalum clips or clove-hitch knot of 5-0 polyester suture. No.41 band is commonly used by many surgeons in RD in aphakic, pseudo phakic eyes and in eyes without recognizable break.

Drainage of subretinal fluid

Drainage of SRF brings the retina closer to the RPE and increases the indentation effect of buckle. There exists a lot of variation in indication and technique amongst the surgeons. It is also fraught with most serious complications of Buckling surgery. The common accepted indications for drainage:

1. Highly elevated (bullous) detachments: D-ACE (Drain-Air-Cryo- Encirclage) sequence allows accurate localization and more effective cryotherapy and also better buckle.
2. Chronic inferior detachments: Viscous SRF absorbs very slowly and breaks may settle less readily on the buckle.
3. PVR: Drainage of SRF increases the buckle effect and may help in closure of retinal breaks which may otherwise remain open because of stiff retina and epiretinal membrane.
4. Aphakic detachments in highly myopic eyes: Drainage allows closure of break by retinal opposition to RPE which otherwise may not close due to persistent passage of fluid vitreous through the break.
5. Glaucomatous eyes and eyes with recent intraocular surgery: These eyes may not tolerate high intraocular pressure associated with non-drainage procedure.
6. Giant retinal tear

Selection of drainage site is done preferably in

1. areas with sufficient SRF
2. Horizontal meridian
3. Nasal quadrant whenever possible
4. Beneath the scleral buckle
5. Areas treated with Cryo should be avoided
6. Areas near vortex vein is avoided

Choice of technique of drainage depends on the preference of the surgeon. The proposed site should be easily accessible and can be viewed without significant manipulation of globe. The different techniques used for drainage

are as follows:

1. Choroidal knuckle & needle drainage
2. Suture needle [8]
3. Choroidal knuckle and drainage by argon laser (Endo laser) probe [9,10]
4. Needle drainage under IDO visualisation (**Charles**)[11]
5. Modified needle drainage by bent 27 G hypodermic needle **Pearce et al** [12]
6. Finalize encircling buckle with permanent sutures and elevated IOP before needle drainage [13]

Hypotony of globe should be prevented by external pressure and intravitreal injection of gas/BSS to prevent choroidal detachment and subretinal haemorrhage. In case of intraocular haemorrhage, digital indentation of the globe in inferotemporal quadrant under ophthalmoscopic observation to achieve blanching of choroidal vasculature and occlusion of central retinal artery for 5 minutes. Pressure is reapplied for 2 mins if bleeding persists upon release of pressure.

Closure of conjunctiva

Tenon's capsule and conjunctiva is irrigated with BSS containing antibiotic or Povidone-Iodine. They should be closed in two separate layers with Vicryl 8-O sutures followed by sub conjunctival injection of antibiotic and steroids.

Special situations

1. ***No break identified***: Cryotherapy applied to cover suspected sites based on Lincoff's Rule and No. 41 encircling band is placed after SRF drainage.
2. ***Prior glaucoma filtering surgery***: Avoid inadvertent injury to bleb and these are best managed with segmental buckle. Excessive elevation of intraocular pressure avoided.
3. ***Recent cataract surgery***: Wound is reinforced by sutures if necessary before proceeding.
4. ***Anterior chamber/Iris supported IOL***: Avoid paracentesis. Injection of Sodium Hyaluronate may be considered if anterior chamber becomes shallow.
5. ***Giant Retinal tear***: These are preferably managed by Vitrectomy procedures or a low, broad buckle should be used to provide adequate support posteriorly along with adequate cryotherapy to seal the edges to prevent anterior leakage of fluid beneath the anterior flap.

Complications

Intraoperative complications related to block like ***perforation*** can be avoided by keeping in mind that many of these eyes might be having staphyloma***. Bradycardia and oculocardiac reflex*** can occur while ligating and handling recti. Gentle handling and careful evaluation of the insertion can avoid ***Muscle disinsertion.***

Scleral Perforation can happen during suture placement and can be identified as leakage of viscous fluid with choroidal pigments. It can be avoided by avoiding the areas of scleral thinning. IDO should be done to rule out accidental retinal breaks and subretinal haemorrhage. In case of ***retinal break***, cryotherapy and buckle to support the break should be done. ***Bleeding*** can be stopped by application of external pressure to the site of perforation and positioning the eye to prevent subfoveal migration of blood. In case of massive sub retinal bleeding, immediate vitrectomy with internal drainage should be considered.

Corneal Clouding: Mild oedema is managed by rolling a dry cotton-tipped applicator over the cornea. Severe epithelial oedema requires epithelial debridement taking care to spare 2 mm rim of peripheral epithelium.

Miosis usually occurs as a result of hypotony, excessive cryotherapy or during paracentesis. Preoperative instillation of 0.3% flurbiprofen may reduce chances of intraoperative miosis. Additional mydriatic drops or intracameral 0.1 ml epinephrin 1:10000 may be used.

Complication associated with SRF drainage:

1. ***Dry tap***: Prevented by appropriately choosing the site of drainage and inspecting the retina immediately before drainage.
2. ***Retinal break***: Occurs because of injury with needle and is treated with cryotherapy and supported by buckle
3. ***Retinal incarceration***: Is characterized by *dimpled appearance of retina with radiating folds* from the site of incarceration. All pressure over the globe is removed and site of incarceration should be supported by buckle.
4. ***Intraocular haemorrhage***: Treated by elevation of intraocular pressure (by digital compression and traction on sling sutures) and appropriate positioning to avoid subfoveal accumulation of blood.
5. ***Choroidal hemorrhage***: Characterized by appearance of dark blood at the drainage site and is managed by immediate closure of drainage site and elevation of intraocular pressure above systolic pressure for 5 minutes. The site of drainage should be positioned inferiorly to prevent migration of blood beneath the fovea.
6. ***Fish mouth phenomenon***: Is treated by radially oriented buckle/ decreasing buckle height/ injecting gas or air.

Post operative complications:

Glaucoma: Angle closure glaucoma may occur because of choroidal detachment. It is manage with steroid, anti glaucoma medication, cycloplegic drugs. If the A/C doesn't reform within 7 days, choroidal darinage and A/C reformation should be done.

Anterior segment ischaemia: High buckle, disinsertion of more than one rectus, sickle cell disease or trait predispose to anterior segment ischemia. Characterized by stomal edema, anterior uveitis, elevated IOP, cataract and shallow anterior chamber. Mild cases may respond to topical steroids and cycloplegics. The severe cases though require release of the encircling band.

Ciliochoroidal detachment: Predisposing factors are excessive cryotherapy or laser, hypotony, damage to vortex veins, advanced age and hypertension. Serous choroidal detachment is more common and most resolve spontaneously. Drainage is indicated in kissing choroidals. Haemorrhagic detachments are less common and prognosis poor.

Persistent retinal detachment: SRF may persist for quite some time specially in non-drainage cases. If the configuration is concave, no obvious open breaks and macula is attached, such cases can be kept under observation. Persistent detachment with open break in superior quadrant may be managed by air/ gas and others may require buckle revision or vitrectomy.

Proliferative vitreoretinopathy: PVR is one of the important reasons for redetachment. It requires vitrectomy.

Infection of buckle elements and extrusion: Infection is seen more commonly and sooner in silicone sponge than solid sponge[14]. Coagulase negative staphylococci is the usual offending agent. These cases present with pain, conjunctival congestion, purulent discharge and tenderness. Infection and exposure usually mandates removal. If it occurs in the first post operative month, removal is delayed to allow retina to attach and managed with antibiotics and analgesics.

Other common complications encountered are ERM, intrusion of buckle, diplopia, refractive error, ptosis, CME, Optic atrophy, sub-foveal pigment migration and retinal vein occlusion, scleral abscess and endophthalmitis.

Scleral buckling surgery has been reported to achieve reattachment for a minimum period of 6 months in 75-95% of cases with a single operation[15]. Visual acuity of 20/30 or better at 6 months have been reported in 90% of macula-on detachment with good preoperative visual acuity whereas 10% suffered decreased visual acuity despite anatomically successful operation. A duration of macular detachment of more than 10 days was associated with poorer visual outcome[16]. The success rate is reduced in presence of GRT, previously failed scleral buckle surgery, trauma, PVR, aphakia and in cases with no apparent retinal breaks[15]. In order to achieve successful anatomical and visual outcome, careful preoperative evaluation, adequate planning and timely intervention are of paramount

importance. The surgeon should plan the surgery with minimal intervention and flexibility to allow modification of procedure based on intraoperative developments.

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PEARLS OF WISDOM

Screening for retinopathy of prematurity

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Introduction

Retinopathy of prematurity (ROP) is a leading cause of childhood vision loss worldwide [1]. Approximately 32,300 infants worldwide are diagnosed with irreversible vision impairment due to ROP annually, of which approximately 20,000 become blind or severely visually impaired. The incidence of ROP in India is reported to vary between 38 – 51.9 % in low birth weight infants.[1-3] Out of the approximate 26 million annual live births in India, approximately 8.7% of newborns in India are < 2000 grams in weight. ROP evolves over 4-5 weeks after birth. This relatively slow evolution gives a small window of opportunity to effectively conduct retinal examinations and timely interventions.

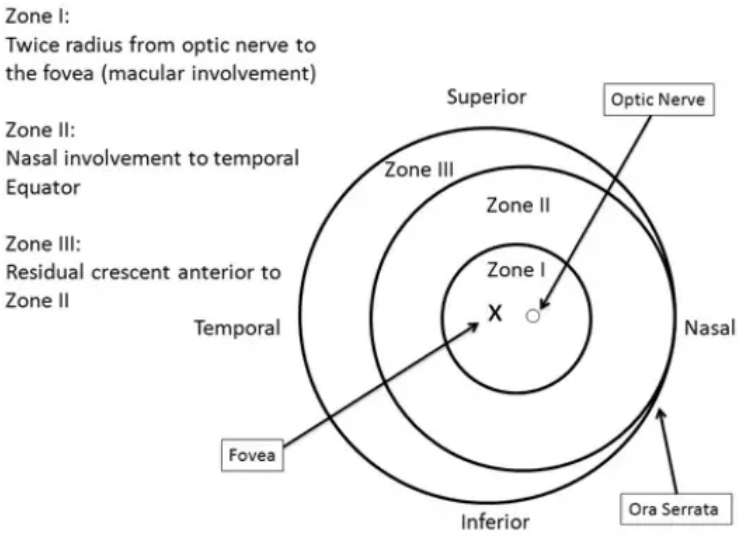
Classification

Now, a third revision, the International Classification of Retinopathy of Prematurity, Third Edition (ICROP3), is being followed.Each eye should be classified using the following examination parameters, defined in this article: zone, plus disease, stage, and extent. If aggressive ROP (A-ROP) is present, it should be noted.

Location of Vascularization: Zone

Retinal vascularization commences around the thirteenth week of gestation, proceeding centrifugally from the peripapillary region to the peripheral retina, which is fully vascularized by approximately term.

Three concentric retinal zones are centered on the disc and extend to the ora serrata The location of the most posterior retinal vascularization or ROP lesion denotes the zone for the eye. The most posterior region, zone I, is defined by a circle with radius twice the estimated distance from the optic disc center to the foveal center. Zone II is a ring-shaped region extending nasally from the outer limit of zone I to the nasal ora serrata and with a similar distance temporally, superiorly, and inferiorly. The committee defined a region of 2 disc diameters peripheral to the zone I border as posterior zone II to indicate potentially more worrisome disease than ROP in the more peripheral zone II .



The committee introduced the term notch to describe an incursion by the ROP lesion of 1 to 2 clock hours along the horizontal meridian into a more posterior zone than the remainder of the retinopathy. When present, this should be recorded by the most posterior zone of retinal vascularization with the qualifier "secondary to notch" [Image 2].For example, ROP in zone II in most places, but with a temporal notch extending into zone I, should be noted as "zone I secondary to notch," thereby distinguishing it from an eye in which most disease is present in zone I.

Zone III is the residual crescent of peripheral retina that extends beyond zone II.

Plus and Preplus Disease

The terms preplus and plus represent a continuous spectrum of retinal vascular changes.

Stage of Acute Disease (Stages 1–3)

In the developing premature infant, the retina is vascularized incompletely .When no ROP lesion is present, the Committee suggests using the term incomplete vascularization, accompanied by the zone of vascularization (e.g., "incomplete vascularization into zone II"), rather than using terms such as no ROP or immature retina. When acute ROP vascular features develop at the junction of vascularized and avascular retina, the term stage is used to describe the appearance. If more than 1 ROP stage is present in the same eye, the eye is classified by the most severe stage.

Stage 1: Demarcation Line

The demarcation line is a thin structure at the vascular–avascular juncture ,which is relatively flat and white, lies within the plane of the retina.

Stage 2: Ridge

The hallmark of stage 2 ROP is a ridge with width and height that evolve from the demarcation line . The ridge may vary in height and its color may appear to range from white to pink.

Stage 3: Extraretinal Neovascular Proliferation

In stage 3 ROP, extraretinal neovascular proliferation extends from the ridge into the vitreous and is continuous with the posterior aspect of the ridge, causing a ragged appearance as proliferation becomes more extensive. [Image 4]

Aggressive Retinopathy of Prematurity

The Committee recommends use of the new term aggressive retinopathy of prematurity (A-ROP) to replace aggressive-posterior ROP .[Image 1]

The hallmark of A-ROP is rapid development of pathologic neovascularization and severe plus disease without progression being observed through the typical stages of ROP.

Retinal Detachment (Stages 4 and 5)

Stage 4: Partial Retinal Detachment

Stage 4 describes partial retinal detachment, which either spares [stage 4A] [Image 5]or involves [stage 4B] the fovea.

Stage 5: Total Retinal Detachment

Extent

Extent of disease is classified using 30° sectors with boundaries along clock-hour positions .

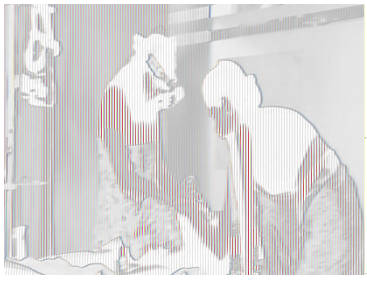
WHEN ROP SCREENING IS TO BE DONE

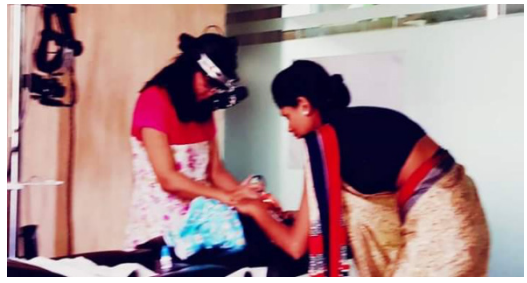
At 21 to 30 day of life or before discharge whichever is earlier.

- Birth weight <2000 g
- Gestational age <34 weeks
- Infants born at <28 weeks and weighing <1200 g are at high risk of developing severe ROP
- In presence of ARDS, sepsis, multiple BT, multiple births (twins/triplets), apneic episodes, intraventricular hemorrhage
    - o Screening should be considered even for babies >37 weeks gestation or >2000 g birth weight.

HOW TO SCREEN for ROP

By indirect ophthalmoscopy with scleral depression.





Scan QR code to know how to dilate the pupil for ROP screening.

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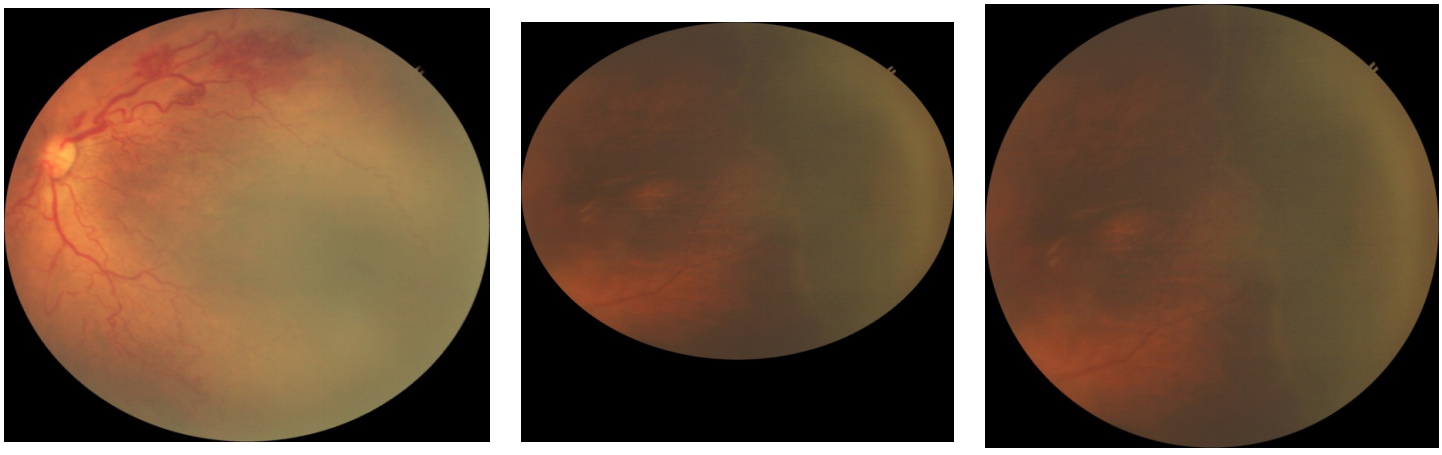

Image 1 (Left): Aggressive Retinopathy of Premuturity (AROP) in a 1200 g baby .Image Courtsey: Macretina Hospital

Image 2 (Middle): Notch as defined in ICROP-3 .Image Courtesy: Macretina Hospital

Image 3 (Right) Stage 2 ROP Image Courtesy: Macretina Hospital

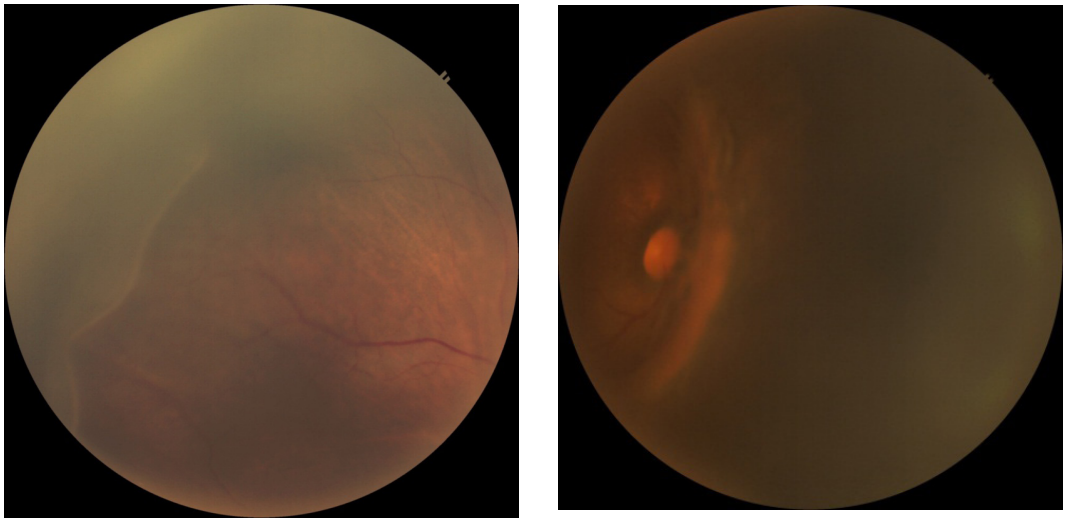

Image 4 (Left) Stage 3 ROP.Image Courtesy: Macretina Hospital

Image 5 (Right): Stage 4a ROP.Image Courtesy: Macretina Hospital

PEARLS OF WISDOM

"CURRENT ROLE OF ANTI VEGF& LASER IN AROP"

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In 1953, Reese et al (1) first described retrolental fibroplasia. The international classification of retinopathy of prematurity (ICROP) was developed in 1984 by 23 ophthalmologists from 11 countries (2) and importance of treating it early was emphasized. In 2005, revised ICROP classification was put forward highlighting aggressive posterior ROP, recognition of "pre-plus" form of disease and anatomical definition of zone 1. (3) Recently in 2021, the third revision of the International Classification of retinopathy of prematurity (ICROP3) (4) was done taking into consideration the latest advances and understanding of the disease.

In normal retinal development, vasculogenesis begins around 17 week postmenstrual age. Normal development can continue until about 39-40 week. The retinal vascular development comprises of initial vasculogenesis, responsible for formation of four major arcade vessels, and the second phase, angiogenesis, which completes the rest of the vasculature.

Interference in vasculogenesis results in aggressive ROP, while the classical staged ROP is correlated with disruption of angiogenesis. (5) The term "aggressive posterior ROP" (earlier referred as rush disease) has been replaced by "aggressive ROP" as this condition has been increasingly seen in larger premature infants with vasculature beyond zone 1. (5)It is characterized by rapidly progressing vascularization without typical stages seen usually in ROP and can cause retinal detachment within few days.Among all infants demonstrating aggressive ROP, lower birth weight, lower gestation age (<29.5 weeks) and preretinal hemorrhages are at the greatest risk for unfavorable outcome. (6) Although staged ROP and aggressive ROP are different, ridge tissue (simulating staged ROP) and flat neovascularization (simulating aggressive ROP) may coexist in the same eye. Hybrid ROP has a preponderance of posterior zone 2 disease and comparatively higher birth weight and gestational age as compared to aggressive ROP. (7) The differences in staged ROP and aggressive ROP can be made by the pattern of vessels which are dichotomously branched in staged ROP as compared to loops and shunting seen in aggressive ROP. Also, in staged ROP new vessels start at junction of vascular and avascular retina and they grow vertically in vitreous while in aggressive ROP new vessels can start at any place (especially nasally) with no definitive junction enclosing multiple pockets of avascular retina and are flat and ill defined.

The standard treatment in type 1 ETROP is by near confluent laser therapy. The main challenge is treatment of aggressive ROP in which despite laser treatment the outcomes are poor, with unfavorable structural outcomes ranging from 14.3% to 28.6%. (8) In some cases,fibrovascular traction usually begins 1 to 3 weeks after laser treatment, with a rapid progression to retinal detachment, despite adequate laser treatment.Laser treatment is also associated with risk of high myopia and limited field of vision. (9)

The treatment of aggressive ROP has been under debate since long. Some prefer doing early laser photocoagulation while others rely on anti VEGF therapy. Each of the procedures has their pros and cons. The advantages of laser therapy is its permanent effect on disease and finite follow up while disadvantages comprise of long learning

curve for perfect laser therapy, use of general anesthesia or sedation with associated risk of life and developmental issues, restriction of peripheral fields, inflammation, cataract formation, anterior segment ischemia and even after perfect laser aggressive ROP may progress to retinal detachment. The advantages of anti VEGF therapy is that it can be given easily at bedside even in sick intubated babies who cannot tolerate a prolonged laser therapy, in eyes with non-dilating pupils, doesn't require long learning curve to master the technique as compared to perfect laser therapy, less laser spots and treatment time if lasers are done later, less visual field loss and less myopia. The use of intravitreal anti-VEGF injection results in prompt regression of ROP, with the potential for further retinal vascular development. However, recurrence and reactivation of disease requiring longer follow up is the main concern with anti VEGF monotherapy which can occur even after months to years of injection. Thus, in patients with aggressive ROP especially in zone 1, combination of anti VEGF injection and laser have favorable structural outcome. (10)

There is still a debate on early vs deferred laser after anti VEGF injection in aggressive ROP.Though, eyes undergoing deferred laser require a fewer number of laser spots and have less myopia at 6 months after laser. Also, posterior zone I aggressive ROP can be managed with combined treatment with anti VEGF injection and zone I sparing laser ablation (after around 4 weeks of injection) in order to preserve large part of central retina. (11) However, eyes planned for the deferred laser after anti VEGF injection requires regular vigilant follow up for early identification and treatment of recurrence.

Several factors influence the 'ideal' timing of laser intervention after anti VEGF injection including response to the drug, recurrences, vascular growth into the retina beyond zone 1, weight of the baby, post menstrual age, systemic conditions and follow-up compliance (especially in rural areas). In some cases, vascularization can progress into more peripheral zones before they either stop or show signs of recurrence or worsen. Ideally, if compliant follow-up is ensured and enforced, a schedule of weekly imaging until a postmenstrual age of 44 weeks, fortnightly until 52 weeks and monthly thereafter may help to detect the growth of vascularization as well as any recurrence that requires early intervention.Laser is performed if there is active flat neovascularization (confirmed on angiography), arrest of vascularization or unwillingness to follow-up further. With this approach larger part of retinal area can be spared from laser ablation. (12)

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EVIDENCE BASED APPROACH

Post-operative endophthalmitis: Prevention and management strategies

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Introduction

Post-operative endophthalmitis is a rare but severe complication of cataract surgery. Post-cataract surgery endophthalmitis (PCE) incidence ranges from 0.02% to 0.71%.[1,2] Since most cases with PCE land up with suboptimal outcomes, prevention remains the best strategy in the fight against endophthalmitis. Good operation theatre practices, sterilisation of operation theatre and instruments, preoperative povidone-iodine eye drops and perioperative intracameral antibiotics are the strategies for preventing endophthalmitis.

The current management of PCE is guided by the Endophthalmitis vitrectomy study (EVS)[3] recommendations published over two decades ago. However, with the evolution of micro-incision vitrectomy systems (MIVS), PPV as a primary intervention has become safer and more accessible.[4] It has led to a debate on whether our approach to managing PCE needs to change. The emergence of antibiotic resistance and changes in the microorganism profile have added fuel to the debate.[5]

Prevention of postoperative endophthalmitis

Preoperative Povidone-Iodine

Applying 5% povidone-iodine in the conjunctival sac for a few minutes can significantly reduce the microbial load to levels comparable to those achieved with preoperative antibiotic drops.[6] A trial by Speaker and Menikoff found povidone-iodine to be more effective than silver protein solution in lowering the incidence of culture-positive endophthalmitis ($P < 0.03$). Furthermore, povidone iodine use was not associated with any significant adverse reactions.[7] It is therefore recommended to use 5% povidone-iodine in the eye before surgery to decrease the incidence of endophthalmitis.

Perioperative Antibiotics

Studies have demonstrated a significant decrease in the incidence of endophthalmitis with intracameral antibiotics such as moxifloxacin. In a study carried out at our centre, intracameral moxifloxacin reduced the occurrence of endophthalmitis from 0.18% to 0.08%. This reduction was more noticeable in patients from lower socioeconomic backgrounds. Given its ease of administration and established safety record in India, moxifloxacin was deemed suitable for intracameral use in our setting.[8]

However, the role of adjuvant topical antibiotics in the postoperative period for endophthalmitis prophylaxis remains unclear. Recent studies have stated that there is no benefit of using topical antibiotics in reducing

endophthalmitis rates[9,10]

Management of Postoperative endophthalmitis

Endophthalmitis vitrectomy study (EVS, 1995)[3]

The management of PCE currently follows the guidelines set by the EVS, a Randomised Clinical Trial that took place in the United States and was published in 1995. A vitreous tap with intravitreal antibiotics was recommended if the presenting vision was hand motions or better and if the vision was light perception or worse, a vitrectomy was suggested. The EVS indicated that diabetic patients might benefit from vitrectomy regardless of their presenting visual acuity, although statistical significance was lacking, making either tap and inject or PPV appropriate. The study also concluded that systemic antibiotics did not provide any benefits if intravitreal antibiotics were given.

Intravitreal antibiotics

The EVS study still guides the current antibiotics preference. The EVS study gave intravitreal vancomycin (1mg in 0.1 ml) and ceftazidime (2.25mg in 0.1 ml). (Figure 1)

In the EVS, acute PCE was most commonly caused by gram-positive cocci, with gram-negative organisms and fungi being less frequently involved. However, two studies conducted in India found that a substantial proportion of POE cases (24.3% and 41.7%) were caused by gram-negative organisms[11,12], while fungi accounted for approximately 8% of cases.[13] Additionally, the administration of Moxifloxacin as an intra-cameral antibiotic prophylaxis has resulted in a rise in postoperative endophthalmitis (POE) caused by gram-negative organisms.[13]

The emergence of antibiotic resistance is another concern currently. In the EVS study, all gram-positive organisms were 100% susceptible to vancomycin. However, a review of endophthalmitis studies on PubMed between 1999 and 2015 found 27 cases caused by gram-positive organisms with reduced susceptibility or resistance to vancomycin.[14] A retrospective study conducted in India found that 11.1% of culture-proven endophthalmitis cases were caused by gram-positive organisms resistant to vancomycin.[15]

In EVS, 89.5% of gram-negative organisms were susceptible to amikacin and ceftazidime. In contrast, a survey from India revealed that approximately 40% of gram-negative organisms were resistant to ceftazidime and responded to imipenem.

These findings raise the question of whether it's time to reconsider the empirical antibiotic combination recommended by EVS for the management of POE.

Pars plana vitrectomy and the evolution of MIVS

The EVS recommended core vitrectomy without induction PVD in eyes presenting with visual acuity of light perception or worse. The reluctance for early vitrectomy in eyes with better vision was primarily due to the fear of iatrogenic breaks and retinal detachment. The advancement in vitrectomy technology, now offers increased safety in the proximity of the retina due to better fluidic control, adjustable cutting rates and variable port openings/duty cycle.[4] Additionally, wide-field viewing systems have made performing PPV safer and more accessible. As a result, the threshold for PPV in the management of POE has decreased. In the past decade, several studies have shown that immediate pars plana vitrectomy (PPV) results in better outcomes and reduces the need for additional interventions. Also, the guidelines from the European Vitreo-Retinal Society (EVRS) recommended immediate PPV for managing postoperative endophthalmitis (POE), even in cases where visual acuity is better than the perception of light (PL). Similarly, The EVRS Endophthalmitis Study Report 1, which examined the preferred practice pattern for managing POE and post-intravitreal endophthalmitis, noted that PPV was often performed regardless of the initial vision.[16] However, a meta-analysis of 15 case series revealed that vitreous tap and injection (T&I) was equally effective as PPV in treating post-cataract, post-intravitreal injection, and post-PPV endophthalmitis, though the specific

inclusion criteria varied across the studies.

At our centre, we performed a small randomised controlled trial to study the effectiveness of immediate pars plana vitrectomy (PPV) versus tap and inject (TAI) in patients with endophthalmitis after cataract surgery.[17] Our study included patients who presented with hand movement or better vision. Immediate PPV resulted in earlier recovery, lesser interventions, and a more significant change in visual acuity than T&I in eyes with PCE presenting with visual acuity of ≥HM. Although the final visual acuity was similar between the two primary groups, delaying PPV may adversely affect the outcomes. The most significant improvement was observed in patients with hand movement vision.

The current practice pattern favour early PPV; however, one needs to be aware that if the facility for PPV is not available, intravitreal antibiotics before referral to a vitreoretinal centre significantly improve the outcomes and should be promptly given to all suspected patients of PCE.[18]

Conclusion

Intravitreal antibiotics have been the cornerstone treatment for over 25 years in managing POE. However, with changes in microbial profile and antibiotic resistance, it may be necessary to change the empirical intravitreal antibiotics. A significant preference shift from EVS guidelines is primary PPV, regardless of the presenting visual acuity.

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Figure legends

Figure 1A: Anterior segment photograph showing hypopyon at presentation post cataract surgery

Figure 1D: Fundus photograph showing severe vitritis at presentation

Figure 1B: Anterior segment photograph on postoperative day 1 of intravitreal vancomycin and ceftazimdime showing resolution of hypopyon

Figure 1E: Fundus photograph showing improvement in media clarity and reduction in vitritis on postoperative day 1

Figure 1C&F: Anterior segment and fundus photograph at 1 week follow up showing significant improvement

Figure 2A: Anterior segment photograph at presentation showing anterior chamber reaction

Figure 2D: Fundus photograph showing severe vitritis at presentation

Figure 2B&2E: Anterior segment and fundus photograph on postoperative day 1 of PPV+intravitreal antibiotics showing significant improvement and reduction in vitritis

Figure 2C&F: Anterior segment and fundus photograph at 1 week follow-up showing further improvement

Figure 1

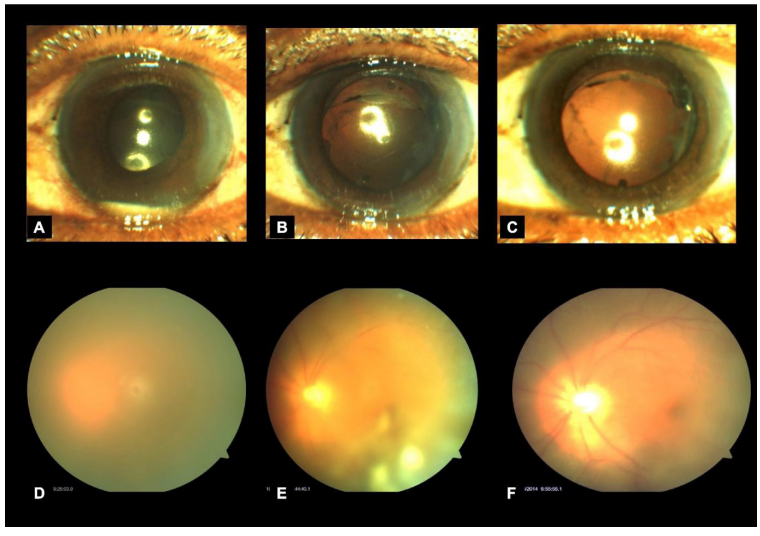

IMAGE ESSAY

Presumed ocular Tuberculosis presenting as Optic nerve head granuloma

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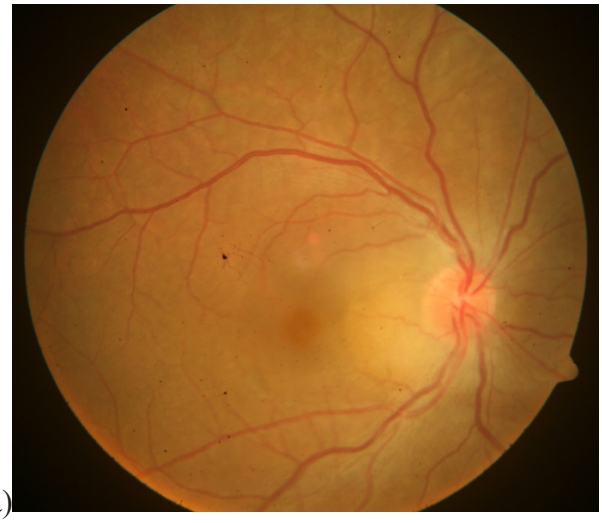
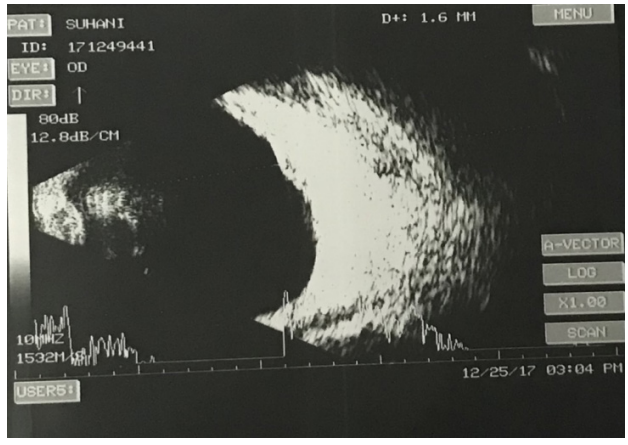
A 30 year old female patient presented with sudden painless diminution of vision in Right eye since 4 days. Best corrected visual acuity of (BCVA) was 6/18 in right eye and 6/6 in left eye. Anterior segment and intraocular pressure were normal in both the eyes. There was no RAPD. Dilated fundus examination showed a granuloma>1 DD in size involvingthe lower margin of right optic nerve head and adjacent choroid with peripapillary subretinal fluid (Fig.1a). Left eye fundus examination was unremarkable.

Right eye Bscan showed a homogenous elevated lesion at the posterior pole with increased choroidal thickness (Fig.1b).

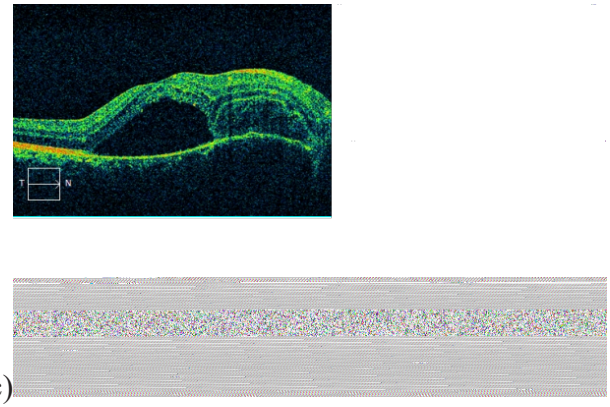
Optical coherence tomography (OCT) across the disc showed a lobulated hyper-reflective lesion (shown by arrow)with back-shadowing and peripapillary subretinal fluid(Fig.1c).OCT also showed attachment of the retinal pigment epithelial-choriocapillaris layer and the neurosensory retina over the granuloma (“contact” sign) shown by arrow(Fig.1d).

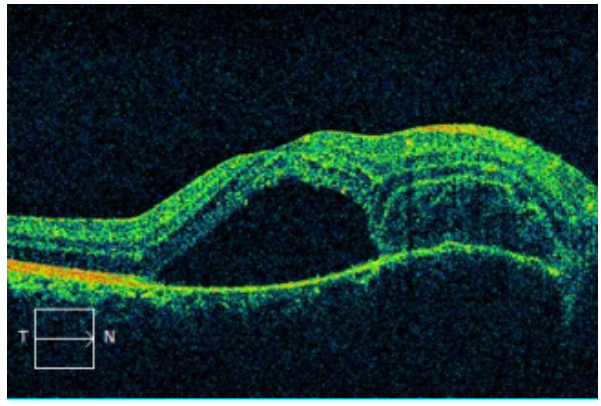


(1a)

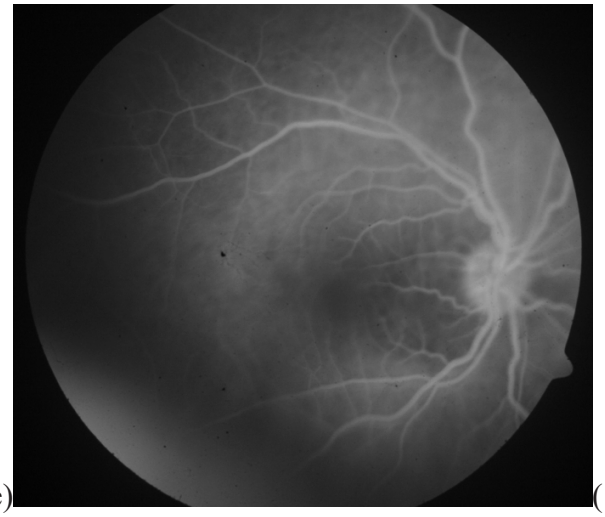


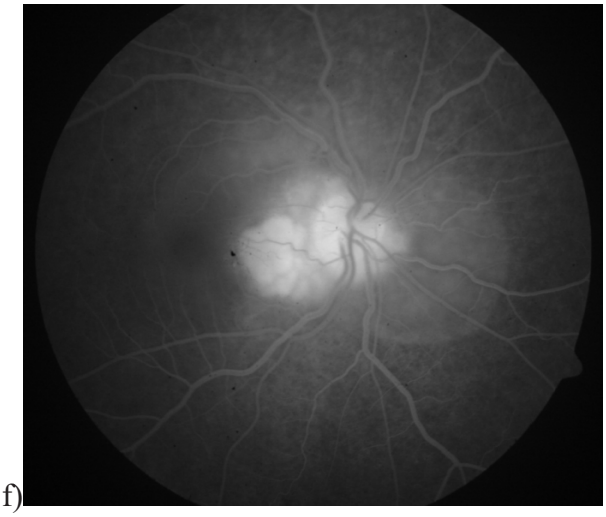
(1b)

(1c) 

(1d) 

Fundus Fluorescein angiography (FFA) showed a hot disc, early hypofluorescence(Fig.1e) in the involved area and late hyperfluorescence clearly delineating the granuloma involving both disc and adjacent choroid with peripapillary pooling of dye(Fig.1f).

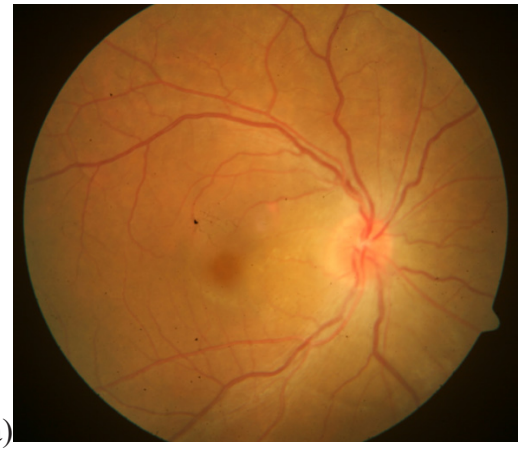
(1e) 

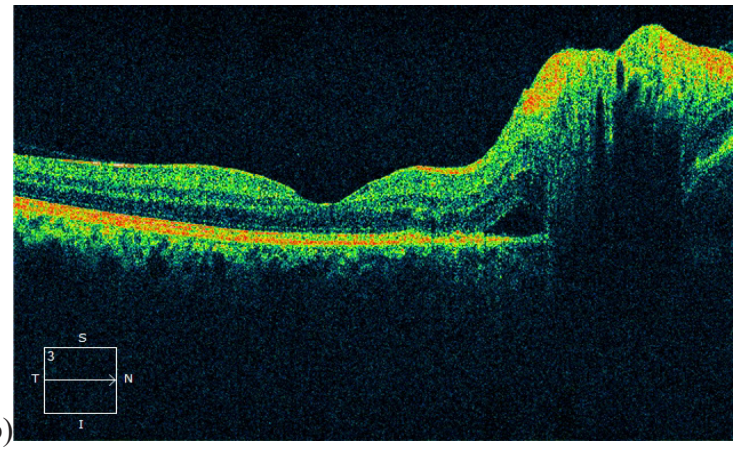
(1f) 

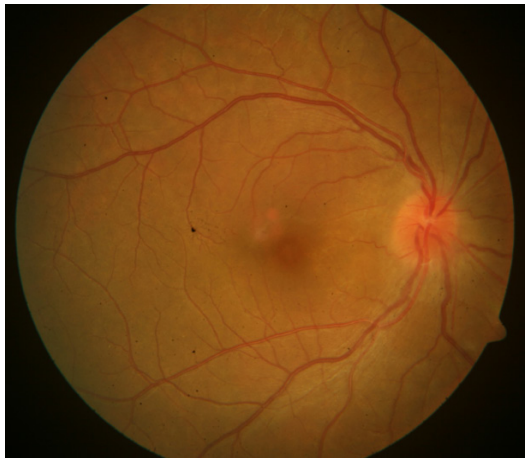
Complete blood counts, liver and Kidney function test, HIV/VDRL test,Toxoplasma titres,Serum Angiotensin converting enzyme levels (ACE)(12.5 U/L) were WNL.

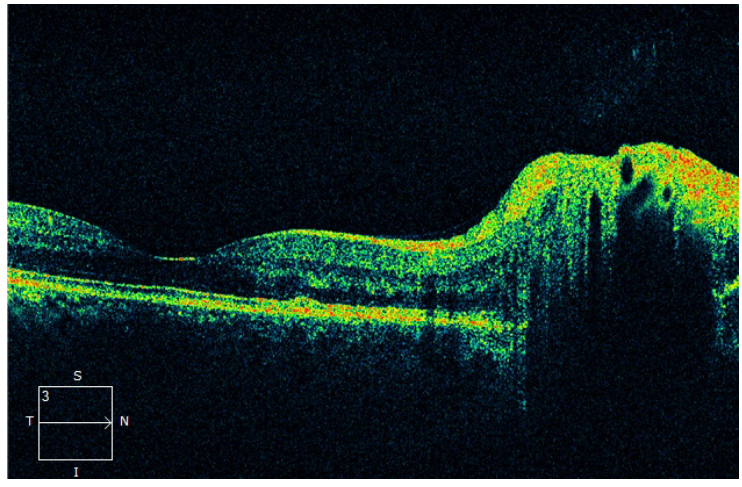
Elevated ESR(50mm/hr), strongly positive Mantoux test (20x20 mm induration) and a highly positive IGRA-QuantiferonTB gold test (338.53pg/ml) were seen . HRCT chest did not show any abnormalities. Sputum for AFB was negative.

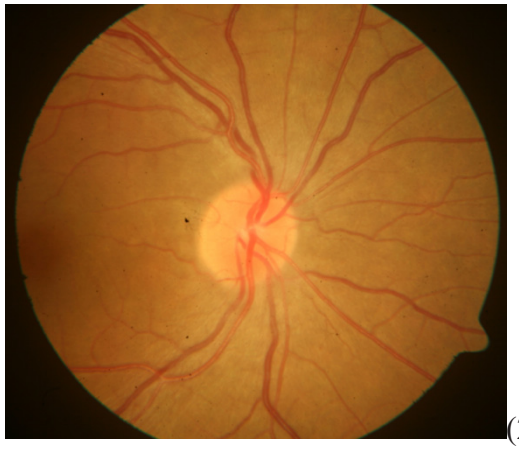
A diagnosis of Presumed Tuberculous optic disc granuloma was made. ATT was started along with Oral prednisolone in a dose of 1mg/kg/Day. Regression of granuloma and resolution of fluid was noted from 2 weeks (Fig. 2a,2b) with significant resolution at 2months(Fig.2c,2d). Complete resolution was observed at 4 months(Fig 2e,2f).

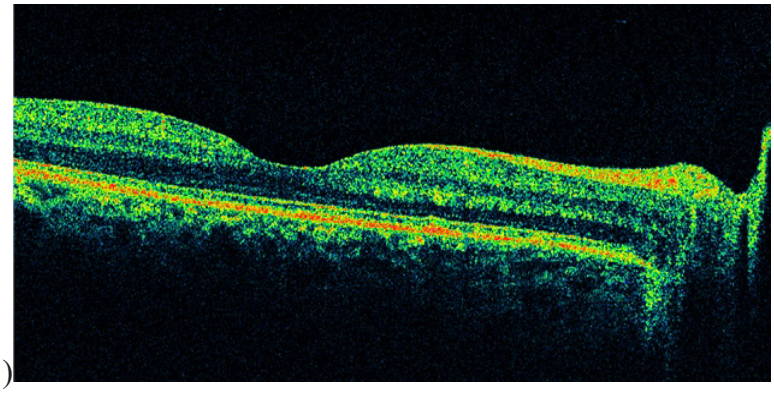
(2a) 

(2b) 

(2c) 

(2d) 

(2e) 

(2f) 

BCVA continued to improve from 6/12 at 2 weeks to 6/6 at 2 months, maintained in subsequent follow-up visits. ATT was continued for 6 months. Oral steroids were tapered over 2 months. No recurrence was observed at 1 year follow-up.

Tuberculous optic disc granulomas are rarely reported.They are typically unilateral and majority of patients present with painless loss of vision(1,2,4). According to a study, TB papillitis was reported in 51.6%,

neuroretinitis in 14.5%, retrobulbar neuritis in 8.1% and optic nerve tubercle in 11.3% (3). Visual outcomes are generally good (3). The “contact” sign on OCT is attributable to inflammatory adhesions overlying the granuloma that cause the neurosensory retina to stick to the retinal pigment epithelium at that point and not pathognomic of TB granuloma(5).Treatment of ocular TB is the same as for pulmonary TBand concomitant steroid therapy is often required.

In our case ATT and oral steroids were promptly started with remarkable improvement in visual acuity and ocular findings.

Declaration of patient consent

The author certifies that appropriate patient consent forms have been obtained for publishing their images and clinical information in journals. The patient understands that their names and initials will not be published and due efforts will be made to conceal their identity.

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5. Salman A, et al. Optical coherence tomography in choroidal tuberculosis. Am J Ophthalmol. 2006 Jul;142(1):170-2.

BEYOND EYE

ELECTROPHYSIOLOGY – SCIENTIFIC BASIS OF VISION

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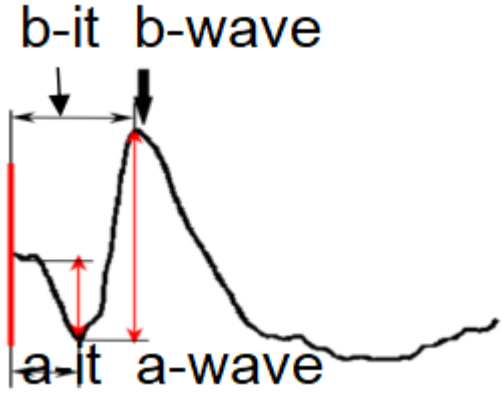
- Electroretinogram (ERG) usually represents the summed activity of the retina in response to a light flash or local electrophysiological responses from different regions of the retina

- Visual Evoked Potentials (VEP) are massed or local electrical signals generated by occipital cortical areas in response to visual stimulation.

- Electroculogram (EOG) is the measure of the functional state of the outmost retinal layers.

Full-Field Electroretinogram (ERG)

- Represents the summed activity of the retina in response to a light flash.
- Isolate responses of different retinal cells dominated by extramacular rods and cones, it is most useful in patients suspected of having widespread retinal disease.
- Essential in diagnosis of numerous disorders including cone dystrophy, retinoschisis, congenital stationary night blindness, Leber congenital amaurosis, rod monochromatism, and paraneoplastic retinopathies.



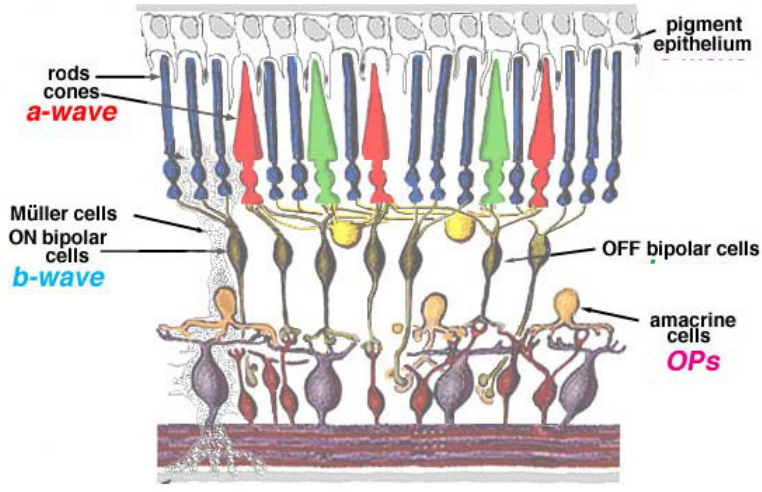
Basic ERG waveform & components

A typical response to white light contains an early, corneal-negative component (**a-wave**) and a slower, corneal-positive component (**b-wave).**

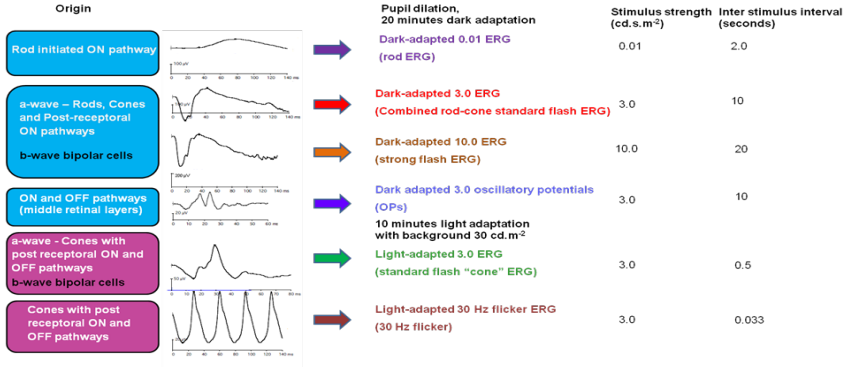
a-wave is know to be generated within the photoreceptor layer and directly reflects photoreceptor activity.

b-wave is generated at the level of the inner nuclear layer, probably by Muller cells, and indirectly reflects bipolar cell activity.

Oscillatory Potentials (OPs) are thought to reflect activity initiated by **amacrine cells** in the inner retina (Wachtmeister& Dowling 1978).



Standard Full-field ERG Protocol ISCEV Standard

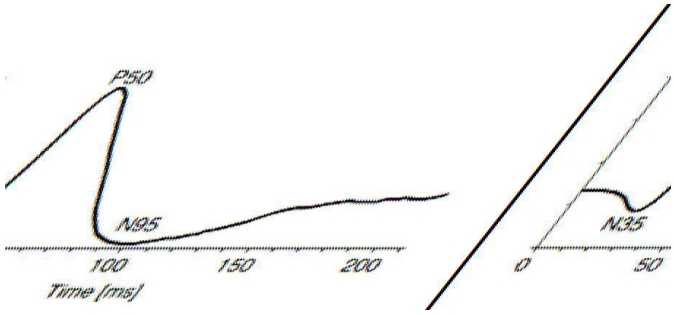


Pattern ERG (PERG)-

- Retinal response evoked by viewing a pattern stimulation (usually black and white checkerboard or bar gratings)
- Provide information of the retinal ganglion cell function and function of macular.
- Clinically, the PERG can be used in a patient with an abnormal visual evoked potential to establish whether a retinal (macular) disorder is present, and thus differentiate between macular and optic nerve dysfunction as a cause for the VEP abnormality.
- It can also directly demonstrate retinal ganglion cell dysfunction.

PERG Waveform

N35: small negative peak at 35 ms
P50: large positive peak at 45-60ms, Produced by retinal ganglion cells and other retinal cellular elements
N95: large negative peak at 90-100ms, Predominantly by retinal ganglion cells.

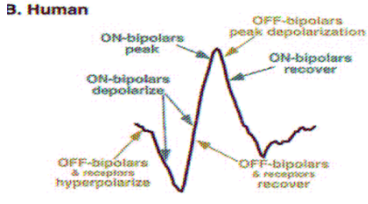


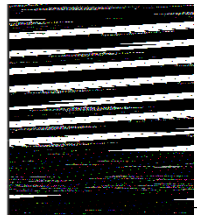
Multifocal ERG (mfERG)

- A method of recording local electrophysiological responses from different regions of the retina
- Provides a topographic measure of light-adapted (photopic) retinal electrophysiological activity

- Retina is stimulated with an array of hexagonal elements (typically 61 or 103, occasionally 241), each of which has a 50% chance of being illuminated every time the frame changes.
- Each hexagon goes through a pseudo-random sequence (the m-sequence) of black and white presentations and has the probability of 0.5 of reversing on any frame change
- By correlating the continuous ERG signal with the sequence of on-and off-phases of each element, the local ERG signal is calculated.
- mfERGs are a mathematical extraction of the signal and are not direct electrical potentials from local regions of retina.
- The waveform of the local mfERG response can be influenced both by

  – preceding (adaptation effects)
  – subsequent stimuli (induced effects)
  – the response to light scattered on other retinal area.





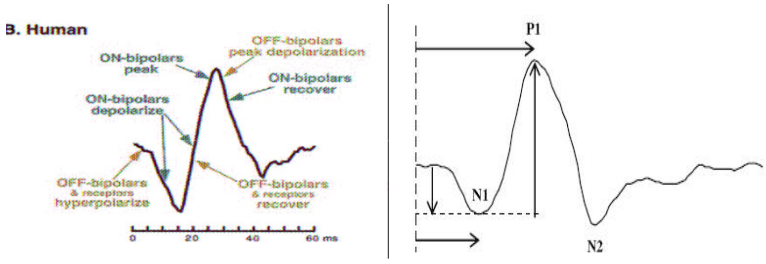
– Quality of fixation

mfERG Waveform

First-order response or first-order kernel
A biphasic wave with an initial negative deflection (**N1**) followed by a positive peak (**P1**). There is usually a second negative deflection after the positive peak (**N2**).

N1- cells contribute to a-wave of full-field cone ERG
P1- cells contribute to cone b-wave and oscillatory potentials

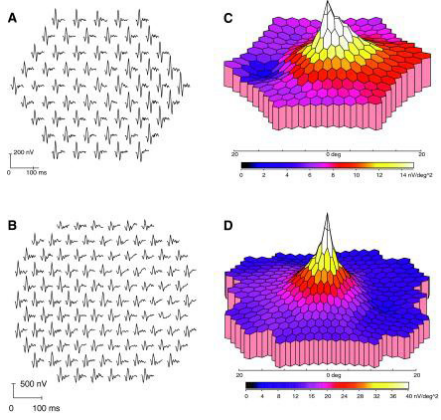
mfERG responses are not “little ERG” responses. Therefore, it is inappropriate to use “a-wave” and “b-wave” to describe features of mfERG waveform.



mfERG Traces:

3D Response Density Plots: Shows the overall signal strength per unit area of retina

Advantages: for visualizing areas of abnormality and for comparing the mfERG results to visual fields from perimetry.
Disadvantage: Information about the waveform is lost
A central peak in the 3-D plot can be seen in some records without any retinal signals



Highly dependent on how the local amplitude is measured.

mfERG: Group Averages

- Most commonly used display is response density in which the responses from each elements in each ring are summed and then divided by the area of the elements responses by rings (from center to peripheral)

- Helpful for comparing quadrants, hemiretinal areas, normal and abnormal regions of two eyes, or successive rings from center to periphery.

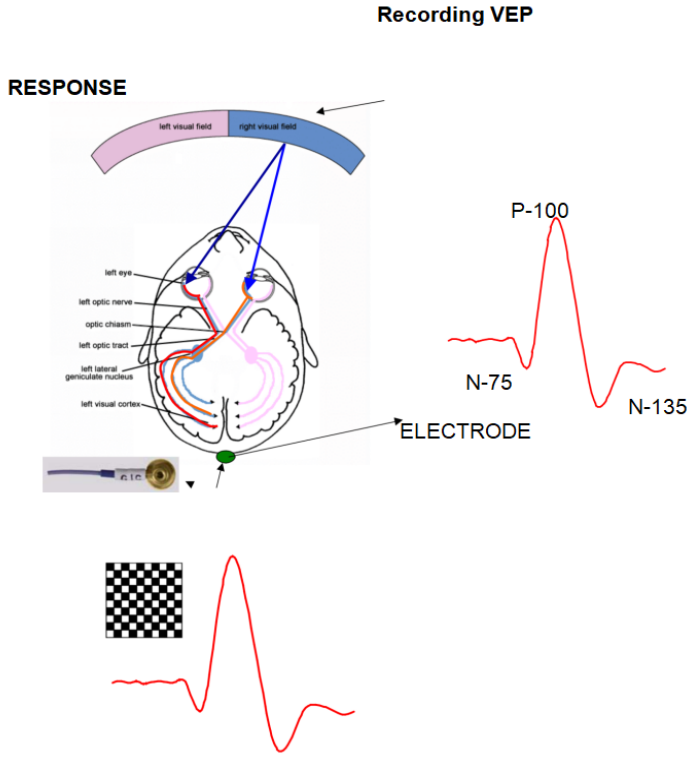
VEP

As visual cortex is activated primarily by the central visual field, VEPs depend on functional integrity of central vision at any level of the visual pathway including the eye, retina, the optic nerve, optic radiations and occipital cortex.

Clinical VEPs are used to evaluate the integrity of the visual pathway from the retina to the occipital cortex, Identify the site and nature of neurological lesions.
Evaluate different aspects of visual function such as **acuity**, contrast sensitivity, and **binocular vision.**

Transient vs Steady-state

<u>Transient</u>: relevant brain mechanism must return to its resting state before the next stimulus
<u>Steady-state:</u> repetitive potential whose discrete frequency components remain constant in amplitude & phase over a long time period.



Transient: Pattern-reversal VEP

VEP Is elicited by a checkerboard-like stimulus of alternating black and white square checks that reverse in a regular phase frequency

Waveform consists of a N75, P100 and N135 peaks.

P100 is usually a prominent peak that used for analysis.

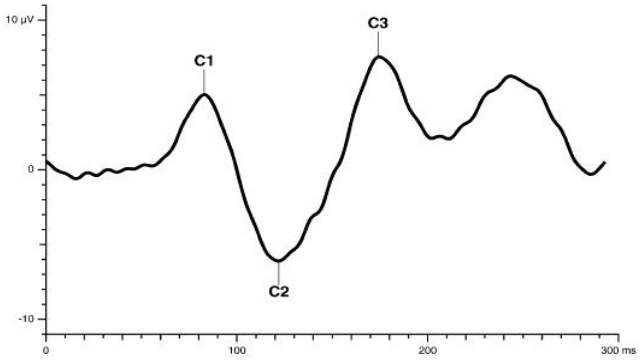
P100 shows relatively little variation between subjects, minimal within subject interocular difference, and minimal variation with repeated measurement over time. P100 peak time is affected by non-pathophyiologic parameters such as pattern size, pattern contrast, mean luminance, signal filtering, patient age, refractive error, poor fixation and miosis.

Transient: Onset/offset VEP

•VEPs are elicited by checkerboard pattern abruptly exchanged with a diffuse gray background.

•This stimulus is best suited for the detection/confirmation of malingering and for use in patients with

nystagmus.
•Less sensitive to confounding factors such as poor fixation, eye movements or deliberate defocus.
Response consists of •C1 (positive ~75 ms) •C2 (negative ~ 125 ms) •C3 (positivee ~ 150 ms)
Amplitudes are measured from the preceding negative peak.



WHO ARE CLIENTS/PATIENTS FOR ELECTRO DIAGNOSTIC TEST

1. Patients with media opacities, whose eyes have: symptoms suggestive of known neurological or ophthalmological disease
2. Assessment of retinal and optic nerve function following trauma
3. Unexplained visual loss,medico-legal problems.
4. Prior to corneal grafting or cataract surgery.
5. Vitreous hemorrhage (in DR or following trauma, RD).
6. Patients with uveitis or inflammatory eye disease
7. Patients with suspected disease or carrier status of inherited visual disorders
8. Patients requiring quantitative assessment of disease progression
9. workman's compensation,defects associated with psychiatric disturbance, mental or physical handicap,pediatric neuro-ophthalmic practice,

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MPSOS SCIENTIFIC ACTIVITIES

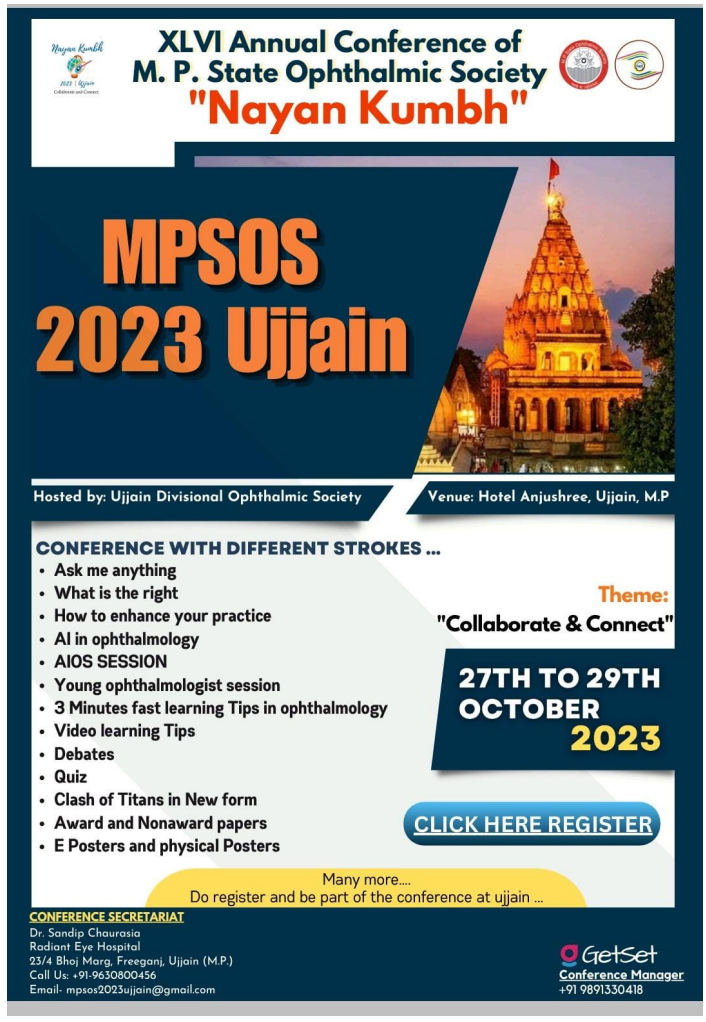
XLVI Annual Conference of M.P. State Ophthalmic Society

"Nayan Kumbh"

27th-29th Oct 2023, Hotel Anjushree, Ujjain, M.P

Hosted by: Ujjain Divisional Ophthalmic Society

Theme: "Collaborate & Connect".




MADHYA PRADESH
OPHTHALMOLOGICAL SOCIETY
webinar on
Publish and Flourish Part 2
Date : 23th July 2023, Sunday | Time : 11:00 Am - 1:00 Pm
Moderators
Dr. Vinita Ramnani
Dr Mita Joshi
Coordinators
Dr. Pankaj Choudhary
Dr. Shubhra Mehta
Dr. Rakesh Shakya
Panelists
Dr. Pushpa Varma
Dr. Parvez Siddiqui
Dr. Gajendra Chawla
Dr. Sujata Lakhtakia
Dr. Vasudha Damle
Dr. Shweta Walia
Speakers
12 min each
Dr. Divya Trivedi
Composing Abstracts & Compiling Data
Dr Chaitra Jaydev
Framing a research question Different Study Designs
Dr. Sabyasachi Sengupta
Statistics - A Quick Primer
Dr. Kavita Kumar
Research Ethics
Dr. Bharat Gurnani
Which journal to choose
Dr. Vanathi M
How & Why should you publish in IJO
Dr. Vinita Ramnani
Chairperson Scientific Committee, MPSOS 2022-24
ramnanivinita@yahoo.co.in | +91 9893091671


FRAMING QUESTIONS AND STUDY DESIGN
Dr. Chaitra Jayadev
DOMS, FVR, FICO (USA), PhD (Netherlands)
Head, Vitreoretina Department
Narayana Nethralaya, Bangalore


Shubhra Mehta
Dr.Bhavana Cho...
Dr Anushree
Rupsa(Clirent)

MPSOS List of Conferences Held

S no	YEAR	PLACE	NAME OF CONF	PRESIDENT	SECRETARY	CHAIRPERSON
1	1973	Jabalpur		Dr R K Mishra		
2	1974	Gwalior		Dr M L Agrawal		
3	1975	Indore		Dr MC Nahata		
4	1976	Rewa		DR SP Shrivastav		
5	1977	Raipur		Dr I M Shukla		
6	1978	Bilaspur		Dr IM Shukla		
7	1979	Bhopal		Dr Santokh Singh		
8	1980	Gwalior		DR SP Shrivastav		
9	1981	Jabalpur		Dr R K Mishra		
10	1982	Indore		Dr M C Nahata		
	1983	.....		No conference		
	1984	.....		No conference		
11	1985	Bhopal		Dr S Hafeez Ahmed		
12	1986	Durg		Dr I M Shukla		
13	1987	Indore		Dr V Kalevar		
14	1988	Gwalior		Dr Balendu Shukla		
15	1989	Jabalpur		Dr S C Batalia		
	1990	.....		No conference		
16	1991	Raipur		Dr P K Mukherjee		
17	1992	Indore		Dr P C Mittal		
18	1993	Chitrakoot		Dr S C Jain		
19	1994	Jagdalpur		Dr P Kothari		
20	1995	Rajnandgaon		Dr R A Siddiqui		
21	1996	Bhopal		Dr V A Joshi		
22	1997	Bilaspur		Dr J L Arya		
23	1998	Gwalior		Dr P Dutta		
24	1999	Jabalpur		Dr P S Soan		
25	2000	Raipur		Dr Anand Saxena		
26	2001	Chitrakoot		Dr B K Jain		
27	2002	Bhopal	FORESIGHT	Dr S K Govil	Dr Kavita Kumar	Dr AK Dubey
28	2003	Gwalior		Dr H C Setiya	Dr Salil Kumar	Dr AK Dubey
29	2004	Indore	ORBIT	Dr SR Khasgiwala	Dr Salil Kumar	Dr Pushpa Varma
	2005	Leap year		No conference		
30	2006	Jabalpur		Dr G D Hoonaka	Dr OP Agrawal	Dr Pushpa Varma
31	2007	Chitrakoot	CHITRANAYAN	Dr MK Rathore	Dr OP Agrawal	Dr Pushpa Varma
32	2008	Ujjain	NAYAN - KUMBH	Dr Pradeep Vyas	Dr OP Agrawal	Dr US Tiwari
33	2009	Bhopal		Dr Salil Kumar	Dr OP Agrawal	Dr US Tiwari
34	2010	Gwalior	MANTHAN	Dr Subodh Garg	Dr Purendra Bhasin	Dr US Tiwari
35	2011	Indore	SU-DRISHTI	Dr Hemant Doshi	Dr Purendra Bhasin	Dr Kavita Kumar
36	2012	Jabalpur	EYE- INFO	Dr P S Chhabra	Dr Purendra Bhasin	Dr Kavita Kumar
37	2013	Chitrakoot	CHITRAKOOT	Dr P C Dwivedi	Dr Mahesh somani	Dr Kavita Kumar
38	2014	Sagar	SULOCHAN	Dr P Bhargava	Dr Mahesh somani	Dr Shreya Thatte
39	2015	Ujjain	NAYANKUMBH	Dr Pradeep Vyas	Dr Mahesh somani	Dr Shreya Thatte
40	2016	Bhopal	SUNAYAN	Dr Harnath Singh Patel	Dr Vijay Nichlani	Dr Shreya Thatte
41	2017	Gwalior	NAYANDEEP	Dr U S Tiwari	Dr Vijay Nichlani	Dr Purendra Bhasin
42	2018	Indore	PRATIBIMB	Dr SK Parwani	Dr Sandeep Chourasi	Dr Purendra Bhasin
43	2019	Jabalpur	DRASHTICON	Dr Hitesh Agrawal	Dr Sandeep Chourasi	Dr Purendra Bhasin
	2020	.....	No conference due to Covid			
44	2021	Rewa - Virtual	NETROTASAV	Dr Sahshi Jain	Dr Gajendra Chawla	Dr Vijay Nichlani
45	2022	Sagar	ELAKSHI	Dr Ashok Singhai	Dr Gajendra Chawla	Dr Vijay Nichlani
46	2023	Ujjain	NAYANKUMBH	Dr Arvind Bhargav	Dr Rajeev Gupta	Dr Vinita Ramnani
47	2024	Bhopal				
49	2025	Gwalior				
50 th	2026	Indore	Golden jublee			

All efforts have been made to collect information- any act of omission or errors are unintentional.

List of Winners of MPSOS Conferences

MPSOS CONFERENCE- YEAR WISE LIST OF SCIENTIFIC AWARDS AND AWARDEES NAMES (2014 ONWARDS)		
Sagar 2014-(Chairperson -Dr Shreeya Thatte)	Ujjain 2015- (Chairperson- Dr Shreeya Thatte)	Bhopal- 2016 (chairperson- Dr Shreeya Thatte)
Dr D N S Choudhary Award (Best of Best Free Paper)	Prof. DNS Chaudhary Award | Best of the Best Free Paper	Prof. DNS Chaudhary Award | Best of the Best Free Pape
Dr	Dr Aditya Sharma	Dr Shuruti Kochar Maru
Prof I B Goel Memorial Award (Pediatric)	Dr. G. S. Wagle Oraton Award	Prof. B. Shukla Award Gross root Paper
Dr Salil Kumar	Dr Purendra Bhasin	Dr Prerna Upadhayay
Smt Narsingh Bahadur Memorial Award (Glaucoma)	Dr. P. L. Dubey Memorial Award (Retina)	Prof. R.P. Dhanda Memorial Award (Cornea FP)
Dr Tanuja Kate	Dr Aditi Dubey	Dr Prerna Upadhayay
Sadguru Seva Sangh Best Video Award	Dr. H. C. Setiya Award Session (Video Session)	Shri Sadguru Seva Sangh Video Award (Video Session)
Dr Vinita Ramnani	Dr Prashant Bhartiya	.....
Smt Prabha Devi Mishra Award (Lady Ophthalmologist)	Smt. Mani Heeresh Chandra Memorial Award (Orbit & Oculopla	Low Vision Aid Paper award
Dr Anamika Diwedi	Dr GVN Rama Kumar	
		Teacher's Award
		Dr Praveen Khare
		Dr. J. K. Raizada Award (Cataract)
		Dr Bhawana Sharma
Dr Ramesh Krishna Agarwal Award	Dr. Ramesh Krishna Agarwal Award	Dr. Ramesh Krishna Agarwal Award
Dr Amit Yadav	Dr Satendra Singh	Dr Amit Solanki
Dr Kumud V A Joshi Award	Dr. Kumud V A Joshi Award (PG)	Dr. Kumud V A Joshi Award (PG)
Dr Nilesh Jaiswal	Dr Harsha Saxena	Dr Rimpi Rana
PG's Quiz Winning Quiz Team Runner up team	PG's Quiz Winning Quiz Team Runner up team	PG's Quiz Winning Quiz Team Runner up team
	Bhopal Jabalpur	Chitrakoot GMC Bhopal
	Dr Rishi Gupta Dr Swapnesh Sukhdeve	
	Dr Yamini Dr Sarika Chouhan	
	Dr Sadananand Shetty Dr Harsha Saxena	
GWALIOR 2017 (Chairperson- Dr Purendra Bhasin)	INDORE 2018(Chairperson- Dr Purendra Bhasin)	Jabalpur 2019 (Chairperson -Dr Purendra Bhasin)
Dr D N S Choudhary Award (Best of Best Free Paper)	Prof. DNS Chaudhary Award | Best of the Best Free Paper	Prof. DNS Chaudhary Award | Best of the Best Free Pape
Dr Bhavna Sharma	Dr Pulak Paul	Dr Sangeeta Kumari
Prof I B Goel Memorial Award (Pediatric)	Dr. G. S. Wagle Oraton Award	Prof. B. Shukla Award Gross root Paper
Dr Bhushan Ghodke	DR Vasudha Damle	Dr Arpita Sthapak
Smt Narsingh Bahadur Memorial Award (Glaucoma)	Dr. P. L. Dubey Memorial Award (Retina)	Prof. R.P. Dhanda Memorial Award (Cornea FP)
Dr Shreya Thatte	Dr Arun Bhargava	Dr Harsha Saxena
Sadguru Seva Sangh Best Video Award	Dr. H. C. Setiya Award Session (Video Session)	Shri Sadguru Seva Sangh Video Award (Video Session)
Dr Rakesh Shakya	Dr Amit Solanki	Dr Pererna Phadnis
Smt Prabha Devi Mishra Award (Lady Ophthalmologist)	Smt. Mani Heeresh Chandra Memorial Award (Orbit & Oculopla	Low Vision Aid Paper award
Dr Bhavna Sharma	Dr Saroj Gupta	Dr Prerna Upadhyay
		Teacher's Award
		Dr Aditi Dubey
		Dr. J. K. Raizada Award (Cataract)
		Dr Vinita Ramnani
Dr Ramesh Krishna Agarwal Award	Dr. Ramesh Krishna Agarwal Award	Dr. Ramesh Krishna Agarwal Award
Dr Prabha Gupta	Dr Ankita Aishwarya	Dr Sannyam Malhottra
Dr Kumud V A Joshi Award	Dr. Kumud V A Joshi Award (PG)	Dr. Kumud V A Joshi Award (PG)
Dr Satender Kumar Singh	Dr Namrata Gupta	Dr Sapna Sabnani
PG's Quiz Winning Quiz Team Runner up team	PG's Quiz Winning Quiz Team Runner up team	PG's Quiz Winning Quiz Team Runner up team
SAIMS Indore Index medical college	MGMMC Indore RD Gardi medical	SSMC REWA AIIMS Bhopal
Dr Satender Kumar Singh Dr kyanat Patel	college	DrAwantika Chawde Dr Sunita Sabharwal
Dr Yati Gothwal Dr Deepti Majumdar	Dr Varun Upadhayay Dr Aviral Hooda	Dr Anjali Virani Dr Khushboo
Dr Shilpi Aggarwal Dr Ena Choudhary	Dr Awani Dubey Dr Richa Sharma	chouhan
Cataract Free Paper	Cataract Free Paper	
Dr Saumya Haldar	Dr Manbir Singh	
Cornea & Refractive Free Paper	Cornea & Refractive FP	
Dr Shruti Kochar Maru	Dr Shreya Thatte	
	Free Paper - (Glaucoma)	Free Paper - (Glaucoma)
	Dr pulak paul	Dr Amit Gupta
Retina Free Paper		Free Paper - Retina
Dr Manbir Singh		Dr Chahveer Singh Bindra
Ocular Trauma Free Paper		
Dr Salil Kumar		
Orbit Oculoplasty		Orbit & Oculoplasty Free Paper
Dr Sangeeta Kumari		Dr Sangeeta Kumari
Community Ophthalmology		Community Ophthalmology Free Paper
Dr Deepti Mujumdar		Dr Parvez Siddiqui Dr Payal Gupta
	Free Paper (Others)	Free Paper (Others)
	Dr Abha Shukla	Dr Amrita Tiwari

Cont..

REWA-Virtual 2021(Chairperson - Dr V K Nichlani)	SAGAR 2022 (Chairperson-Dr V K Nichlani)	UJJAIN 2023 (Chairperson- Dr Vinita Ramnani)
Dr D N S Choudhary Award (Best of Best Free Paper)	Prof. DNS Chaudhary Award | Best of the Best Free Paper	Prof. DNS Chaudhary Award | Best of the Best Free Pape
Dr Anu Litoriya	Dr GVN Rama Kumar	
Prof I B Goel Memorial Award (Pediatric)	Dr. G. S. Wagle Oraton Award	Prof. B. Shukla Award (Gross root Paper)
Dr Vinita Ramnani	Dr Vinita Ramnani	
Smt Narsingh Bahadur Memorial Award (Glaucoma)	Dr. P. L. Dubey Memorial Award (Retina)	Prof. R.P. Dhanda Memorial Award (Cornea)
Dr Anu Litoriya	Dr GVN Rama Kumar	
Dr. H. C. Setiya Award Session (Video Session)	Sadguru Seva Sangh Best Video Award	Dr. H. C. Setiya Award (Video)
Dr paresh Nichlani	Dr paresh Nichlani	
Smt Prabha Devi Mishra Award (Lady Ophthalmologist)	Smt. Mani Heeresh Chandra Memorial Award (Orbit & Oculopla	Low Vision Aids award
Dr Sujata Lakhtakia	Dr Shweta Walia	
Dr Ramesh Krishna Agarwal Award	Dr. Ramesh Krishna Agarwal Award	Dr. Ramesh Krishna Agarwal Award (poster)
Dr Rahul Choubey	Dr Rakesh Shakaya	
Dr Kumud V A Joshi Award	Dr. Kumud V A Joshi Award (PG)	Dr. Kumud V A Joshi Award (PG)
Dr Shivani Bansal	Dr Dhirendra Kumar Pandey	
PG's Quiz	PG's Quiz	PG's Quiz
PG's Quiz Winning Quiz Team Runner up team	WINNERS -SNC, Chitrakoot RUNNER UPS -SAIMS,	
SNCB Jabalpur Aurobindo medical college	indore	
Dr Ashul Chawla Dr Yashas Goyal	Dr Darshan Shah Dr Dhruv Agarwal	
Dr Ritika Tripathi Dr Garima Tiwari	Dr Aishwari Rewankar Dr Haritima Sharma	
Dr Richa Tripathi Dr Aviral Vasudev	Dr Rohan Porwal Dr Komal Jaiswal	
Cataract Free Paper	Cataract Free Paper	
Dr Dherendra Singh		
Cornea & Refractive Free Paper	Cornea & Refractive FP	
Dr Prabha Gupta		
free paper - glaucoma	Free Paper - (Glaucoma)	Free Paper - (Glaucoma)
Dr Parvej Siddiqui	Dr Yuri Kashiv	
Retina Free Paper	Retina Free Paper	Free Paper - Retina
Dr Anamika Diwedi	Dr Manisha singh	
Ocular Trauma Free Paper		
Orbit Oculoplasty	Orbit Oculoplasty	Orbit & Oculoplasty Free Paper
Dr Parvej Siddiqui	Dr Himanshu Gaikwad	
Community Ophthalmology	Community Ophthalmology	Community Ophthalmology Free Paper
	Dr Priyanka Mandraha	
Free Paper (Others)	Free Paper (Others)	Free Paper (Others)
Dr MA Khurrum	Dr Shlok O Singh	
Dr Gokuldas memorial award (2021) cataract complications	Dr Gurdeep Singh Memorial Award (2022)	Dr. J. K. Raizada Award (Cataract)
Dr OP Agrawal	no entery received	
CLASH OF TITANS (2021)	CLASH OF TITANS (2022)	CLASH OF TITANS
RDOS- Dr Gutam Singh Pawar, Dr Rakesh Shakya,	IDOS - Dr OP Agrawal, Dr Satish Premchandani,	
Dr Asheesh Bajaj, Dr Ayush Bajaj, Dr prashant Borde	Dr Shweta Walia, Dr Bhagyesh Pore	
	TEACHERS AWARD - every year	Teacher's Award (below 40 years)
	Dr Sonam Verma	
	Non teachers award - every year	Non teachers Award (below 40 years)
	Dr Harshdeep Singh Gabba	DR HARIPAD DATTA AWARD
All efforts have been made to collect information- any act of omission or errors are unintentional.		

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InDME

EURETINA Guidelines Recommendation

Aflibercept is the drug of choice in DME eyes with baseline BCVA below 69 letters, as it shows superiority to bevacizumab over 2 years and over ranibizumab in the first year of treatment[1]

ABBREVIATED PRESCRIBING INFORMATION

EYLEA™ (AFLIBERCEPT) INTRAVITREAL INJECTION IN VIAL. Approved name(s) of the active ingredient(s): One ml solution for intravitreal injection contains 40 mg aflibercept. Each vial provides a usable amount to deliver a single dose of 50 µl containing 2 mg aflibercept. Indication: EYLEA™ is indicated for the treatment of (i) neovascular (wet) age-related macular degeneration (wet AMD), (ii) diabetic macular edema (DME). Dosage Regimen: For initial EYLEA treatment is initiated with one injection per month (every four weeks) for three consecutive doses, followed by one injection every two months (8 weeks). Based on the physician's judgement of visual and/or anatomic outcomes the treatment interval may be maintained at two months or further extended using a treat-and-extend dosing regimen, where treatment intervals are gradually increased to maintain stable visual and/or anatomic outcomes. If visual and/or anatomic outcomes deteriorate, the treatment interval should be shortened accordingly. There is no requirement for monitoring between injections. Based on the physician's judgement that monitoring visits may be more frequent than the injection visits. Treatment intervals greater than 4 months (16 weeks) between injections have not been studied. Dosage Regimen for DME: the recommended dose for EYLEA is 2 mg aflibercept (equivalent to 50 microliters solution) administered by intravitreal injection monthly (every 4 weeks) for the first 5 consecutive doses, followed by one injection every 2 months (8 weeks). Method of administration: Intravitreal injections must be carried out according to medical standards and applicable guidelines by a qualified physician experienced in administering intravitreal injections. Following intravitreal injection patients should be instructed to report any symptoms suggestive of endophthalmitis (e.g. eye pain, redness of the eye, photophobia, blurring of vision) without delay. Each vial should only be used for the treatment of a single eye. Contraindications: Known hypersensitivity to aflibercept or to any of the excipients; active or suspected ocular or periocular infection; active severe intraocular inflammation; known or suspected septic endophthalmitis; and retinal detachment. Warnings and precautions for use: Endophthalmitis and retinal detachments may occur following intravitreal injections. Patients should be instructed to report any symptoms suggestive of endophthalmitis or retinal detachment without delay and should be managed appropriately. Increases in intraocular pressure have been seen within 60 minutes of an intravitreal injection. There is a potential risk of immunogenicity and arterial thromboembolic events following intravitreal use of VEGF inhibitors. EYLEA should not be used in pregnancy unless the potential benefit outweighs the potential risk to the foetus. Women of childbearing potential have to use effective contraception during treatment and for at least 3 months after the last intravitreal injection of aflibercept. Undesirable effects: Very common: Conjunctival haemorrhage, eye pain, Common: Retinal pigment epithelial tear, detachment of the retinal pigment epithelium, cataract, cataract cortical, cataract nuclear, cataract sub capsular, corneal erosion, corneal abrasion, intraocular pressure increased, vision blurred, vitreous floaters, vitreous detachment, injection site pain, foreign body sensation in eyes, lacrimation increased, eyelid oedema, injection site haemorrhage, psoriatic lesions, conjunctival cysts, ocular hyperaemia. For full listing of undesirable effects, please refer to the full prescribing information. Please contact Bayer Zydus Pharma (Haryana) Limited, Bayer House, Central Avenue, Hiranandani Estate, Thane, Maharashtra, India Pin- 400607 Email: medicalinfo.india@bayerzydusPharma.com, Invoice: Based on CDSCO Version 16 dated 30 Oct 2020, SPC version 01 dated 16 Nov 2020 & US PI dated May 2008. PI: Version No: EY_2008_01 dated 16 Apr 2020. Date of revision of ABP: April 2022. For the use of Healthcare professionals only.

Reference: 1. Schmidt-Erfurth, U., Garcia-Arumí, J., Bandello, F., Berg, K., Chakravarthy, U., Gerendas, B. U., & Lanzetta, P. (2017). Guidelines for the management of diabetic macular edema by the European Society of Retina Specialists (EURETINA). Ophthalmologica, 237(3), 185–222.

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