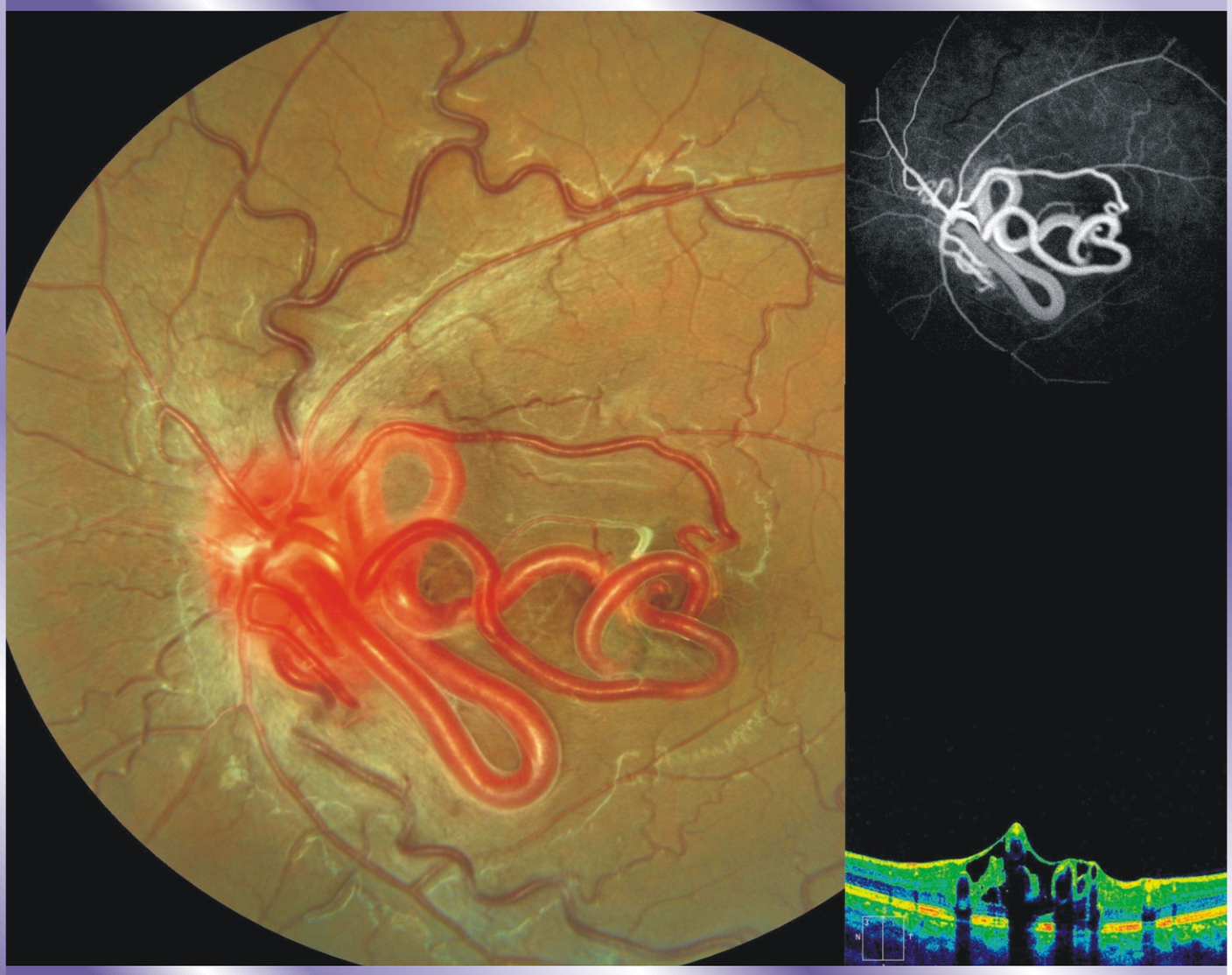
SEPTEMBER 2020

The Official Newsletter of the

VITREO RETINAL SOCIETY-INDIA



Official Website : www.vrsi.in

NOVARTIS

STRENGTH IN EVIDENCE

The efficacy and safety of Accentrix® were rigorously studied in 500+ clinical trials[1]

Accentrix®
ranibizumab

A vision to believe in

References:
1. CHRISTIAN PRÜNTE, NICOLE ETER Retina Today - March 2018

Contents

Editor
Dr. Anand Rajendran

VRSI Executive 2019-20

President
Dr. Shobhit Chawla

Secretary
Dr. Raja Narayanan

Convenor, Scientific Committee
Dr. Anand Rajendran

Vice-President
Dr. NS Muralidhar

Ex-President
Dr. A Giridhar

Treasurer
Dr. Hemant Murthy

Joint Secretary
Dr. Manisha Agarwal

Joint Treasurer
Dr. Prashant Bawankule

Executive Committee Members
Dr. Pukhraj Rishi
Dr. Chaitra Jayadev
Dr. Manoj Khatri

From the President's Desk

Dr. Shobhit Chawla
Medical Director and Chief - Vitreo Retinal Services
Prakash Netra Kendr
Lucknow
shobhitchawla1412@gmail.com

Dear friends and colleagues

As the year 2020 moves ahead with a changed world order , and the world waits expectantly for the virus to let go its hold or the vaccine to surface, let us wish and pray for the same . Friends, the next issue of VRSI newsletter has participation of Dr Lingam Gopal in the field of Management of Choroidal Coloboma related Retinal detachment. Dr Gopal's work in this field has been well recognised, received internationally, it has been the basis of our management of this challenging situation through the years . The spotlight article is anchored by our scientific convenor Anand Rajendran and Rupak Biswas on ocular trauma management. Hoping for the best for everyone and the world as the festive season lurks round the bend in the next quarter.

> "Learn from yesterday, live for today, hope for tomorrow. The important thing is not to stop questioning."
>
> – Albert Einstein

Regards and best wishes
Dr. Shobhit Chawla
President
Vitreo-Retinal Society of India

From the Honorary Secretary's Desk

Dr. Raja Narayanan
Director-Head, Clinical Research Consultant
Smt. Kanuri Santhamma Centre for Vitreo Retinal Diseases
Kallam Anji Reddy Campus, Hyderabad
narayanan@lvpei.org

Dear Friends

Warm greetings from VRSI. An excellent issue of VRSI Newsletter of September 2020 has been compiled by Dr. Anand Rajendran. I am sure that you will find the articles extremely valuable for your daily practice, and to provide the best care to your patients. I take this opportunity to request you all to submit your interesting cases, articles and innovations to the VRSI newsletter, which will help improve the scientific knowledge base of our members.

We are working to bring online content to our members on various topics, and a brief conference is being planned in December 2020. Stay safe, and we shall meet soon online.

Regards
Dr. Raja Narayanan
Hon. Secretary
Vitreo-Retinal Society of India

From the Convenor, Scientific Committee's Desk

Dr. Anand Rajendran
Professor & Head
Vitreo-Retinal Service,
Aravind Eye Hospital, Chennai
anandrjn@gmail.com | convener.scientificcom.vrsi@gmail.com

Dear Friends and Colleagues

It has been a pleasure bringing out the September edition of the VRSI Newsletter. In this issue, we have Lingam Gopal, a retina surgeon of global repute, giving us an elaborate account of the work that he is an acknowledged to be an expert on - "Management of Retinal detachment with Coloboma of Choroid" in the 'StalwartSpeak' section. The Spotlight article of the issue, anchored by Dr. Rupak Biswas and yours truly, is focused on "Management of Difficult Situations in Posterior Segment Trauma" with an eminent panel of national experts holding forth on a slew of challenging situations. The Retina Tech Section has Dr. Rupak Ray and colleagues highlight the burgeoning role of Multicolour Imaging in Macular pathologies. In the Innovator's Isle section, Dr. Ashish Ahuja, a young retinal specialist with a passion for innovation, describes his experience with and the multiple possibilities with 3D printing. Finally, Dr. Sangeet Mittal provides us an interesting case report on Purtscher's retinopathy.

We look forward to contributions from all members to future issues. As an unfortunate fallout of the COVID 19 crisis, the VRSI 2020 Annual Meet has had to be cancelled. An enthralling Virtual VRSI Conference is being planned in December, an event that promises to be a scientific fest. It has truly been a pleasure to bringing our members a series of high end webinars these past few months and we are thankful for the appreciation and positive feedback. We hope to see the same enthusiastic response and welcome your whole-hearted support to VRSI activities.

Dr. Anand Rajendran
Convenor
Scientific Committee
Vitreo-Retinal Society India

Guidelines - Manuscript Submission for VRSI Newsletter

Original articles:

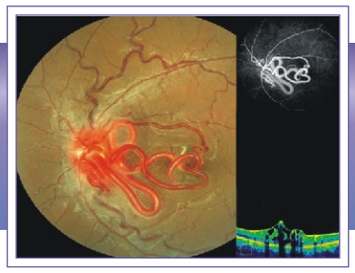
These include randomized controlled trials, intervention studies, studies of screening and diagnostic test, outcome studies, cost effectiveness analyses, case-control series, and surveys with high response rate. The text of original articles amounting to up to 3000 words (excluding Abstract, references and Tables) should be divided into sections with the headings Abstract, Key-words, Introduction, Material and Methods, Results, Discussion, References, Tables and Figure legends.

Case reports / Challenging case /Innovations / Instruments /Techniques :

New, interesting, challenging, rare cases, innovations, instruments and techniques can be reported. They should be unique and providing learning point for the readers. Manuscripts with clinical significance or implications will be given priority. These communications could be of up to 1000 words (excluding Abstract and references) and should have the following headings: Abstract (unstructured), Key-words, Introduction, Case, Discussion, Reference, Tables and Legends in that order.

The manuscript could be of up to 1000 words (excluding references and abstract) and could be supported with up to 10 references. Case Reports could be authored by up to four authors.

Mail to anandrjn@gmail.com, convener.scientificcom.vrsi@gmail.com



The Cover page Image - **"Winner of the VRSI July Retina Imaging Contest - Wyburn-Mason Syndrome: A rare phacomatosis"**
was contributed by
Dr. Deepak Bhojwani | VR & Uvea Consultant, Raghudeep Eye Hospital, Ahmedabad.

STALWART SPEAK

Management of Retinal Detachment with Coloboma of Choroid

Dr. Lingam Gopal
Assoc. Prof & Senior Consultant
Department of Ophthalmology
National University Hospital, Singapore
E mail id: lingamgopal@hotmail.com

Introduction:

Ocular coloboma is caused by defective closure of the embryonal fissure that normally takes place between 4-6 weeks of gestation.[1] This leads to absence of choroid and retinal pigment epithelium in the affected area. The neurosensory retina is replaced by a non-descript partly fibrotic tissue called inter calary membrane.

Although the iris, ciliary body, zonules, as well as optic disc can be involved in addition to the choroid and retina, the effect on vision is usually influenced mainly by involvement of optic disc and macula. The global effect of the developmental defect is the often coexisting microphthalmos. Occurrence of nystagmus, if present is usually a sequela to poor fixation.

Pathoanatomy of relevance for occurrence/ management of retinal detachment (RD):

1. The optic disc may be located within the fundus coloboma or outside. This has relevance in the ability to place laser comfortably, without risk, and effectively around the coloboma during surgery.[2]

2. The margin of the coloboma harbors the communication(s) between the sub inter calary membrane space and sub retinal space.[3] These communications are the root cause of coloboma related retinal detachments. The neurosensory retina splits at the margin of coloboma with the inner layers continuing as inter calary membrane (ICM) while the outer layers turn back and merge with the retinal pigment epithelium (RPE). This junction (zone of least resistance) is weak[4] and can get disrupted easily by natural forces (traction by taut ICM) or iatrogenic forces (during surgery in attempts to induce posterior vitreous detachment (PVD)).

3. Breaks in the inter calary membrane: These are not always obvious against the backdrop of lack of pigmentation in the coloboma.[5] However, from the surgical standpoint, they are not of much importance, since closure of these ICM breaks is not attempted nor is practical. One aims to surround the coloboma with a zone of adhesion between retina and RPE and affectively segregate the coloboma from rest of retina.

4. Fovea Vs the coloboma: If the centre of macula is well within the coloboma, it is not likely to be functional and hence can be ignored in the larger scheme of things. If the centre of macula is away from coloboma margin by more than 1000 microns, there would be enough space in between for careful laser photocoagulation and hence should not interfere with adequacy of laser around coloboma. If, however the fovea is just beyond the coloboma, any treatment of margin of coloboma is likely to destroy a functional fovea.

5. Microphthalmos Vs Microcornea: Corneal diameter may not always reflect on the size of the eyeball since there could be microcornea without corresponding microphthalmos.[6] Ideally one should perform ultrasound evaluation to measure the extra colobomatous antero posterior diameter of the eye to gauge the degree of microphthalmos. This may to some extent dictate where to place the sclerotomies in relation to the limbus.[7]

6. Morning glory syndrome Vs fundus coloboma with disc involvement: Morning glory syndrome also involves coloboma of the optic nerve head but seems to behave entirely differently from the routine fundus coloboma with disc involvement. In contrast to morning glory syndrome, one does not see any drag of the peri papillary retina into the colobomatous area; the region is not pulsatile (as is occasionally seen in morning glory), and one does not hesitate to use silicone oil as vitreous tamponading agent (something one would avoid in morning glory for fear of intra cranial spread of oil).

Unique issues with presentation and evaluation:

a) Difficulty in detecting subtle features (such as ability to be certain whether ICM near margin of coloboma is detached or not) due to often coexisting nystagmus, microphthalmos and young age of the patients. b) Children do not complain, and parents may have difficulty in identifying further drop in vision (due to RD) on top of a basically poor vision (due to coloboma). Hence often they are brought quite late.

Types of retinal detachment in eyes with fundus coloboma:

a) RD due to peripheral retinal breaks where coloboma is incidental: The RD stops at margin of coloboma; there is no ICM detachment; and peripheral breaks are obvious.

b) Retinal detachment due to coloboma (with or without contribution from peripheral breaks): Detachment of retina extends variably into coloboma as ICM detachment. i.e the sub retinal space communicates with sub ICM space through one or more breaks in the zone of least resistance; ICM breaksare often present.

c) ICM detachment without retinal detachment: Asymptomatic; Risk of spreading into clinical RD exists.

d) ICM detachment with traction retinal detachment just at border of coloboma: Can be picked up on OCT; May not be detectable on clinical examination; Could be responsible for pigmented patches of degeneration that are often seen beyond margin of coloboma

Other features:

Proliferative vitreo retinopathy (PVR) can be superadded; RDs tend to be chronic since vision loss is often accidentally detected; Cataract can coexist; In eyes with iris coloboma, the superior pupillary border can sometimes be resistant to proper dilation.

Management:

Scleral buckling:

RD unrelated to the coloboma can be managed by scleral buckling procedure. However, one must be certain that the RD is not extending into the coloboma before attempting simple buckling.

Vitreo retinal surgery:

The principles revolve around management of lens (when needed), induction of PVD, proper vitrectomy, fluid air exchange to settle the retina with endo drainage through ICM breaks or a drainage retinotomy, endolaser around the coloboma margin, and internal tamponade.

Variables:

1. Lens management:

In extremely microphthalmic eyes, one may need to sacrifice a clear lens in order to properly manage the posterior segment. In an otherwise normal/ near normal sized eye, coexisting cataract could be an indication for lens removal. The options for management include a full lensectomy, thus making the eye aphakic; or lens aspiration and retention of posterior capsule with plans to introduce secondary IOL at time of oil removal. One has to keep in mind that in young children the capsule tends to opacify very soon and gets adherent to iris quickly.

2. Role of encirclage:

In large colobomas and with no PVR, encirclage does not add much value. Encirclage may be useful in normal/ near normal sized eyes where lens is retained and in eyes with significant PVR.

3. Induction of PVD:

This could be the most troublesome step in the surgery. The violence of the maneuver can induce further breaks in the zone of least resistance. It is recommended to use forceps to induce initial separation of vitreous fibrils before the use of suction.

4. Laser application:

- Disc and fovea outside the coloboma: No specific issues
- Disc within the coloboma: Care needed to avoid accidental full thickness retinal burns while treating around the

functional border of the disc. Light burns and use of diode laser is recommended.

- Fovea at border of coloboma: In order to preserve the fovea, one may have to avoid treating on either side of the fovea. However there remains a risk of recurrence of RD when silicone oil is removed if the communication between sub ICM space and sub retinal space exists in this area.

- Treatment of rest of ora serrata: It is recommended to treat the rest of ora serrata to reduce risk of recurrent RD after oil removal. In microphthalmic eyes the sclerotomies may be closer to the ora serrata compared to a normal sized eye. This increases risk of small dialysis occurring in these meridians. They can remain undetected and result in recurrent RD after oil removal.

5. Choice of tamponade:

- Silicone oil is preferred considering the usual age of the patients, large area of coloboma margin that needs to be tamponaded and quicker visual rehabilitation especially in one eyed patients.[8]

- Gas can be used in selected cases where the site of break in zone of least resistance is evident (focal spillover of RD into coloboma); PVD is already present or the induction was atraumatic; there is no PVR, and the patient can posture well.

Postoperative issues:

- IOP and glaucoma: Intra ocular pressure monitoring is vital. One cannot rely on the disc and visual fields to guide the management of glaucoma if present. In young children one can use I-care to measure IOP in the clinic.

- Silicone oil removal should be done as soon as possible. Tendency for oil emulsification is high among the children if one procrastinates. Many parents (and surgeons) may be postponing removal of oil in order not to face the risk of recurrence of RD.

References:

1. Pagon RA. Ocular coloboma. SurvOphthalmol 1981;25:223-236.

2. Gopal L, Badrinath SS, Kumar KS, Doshi G, Biswas N. Optic disc in fundus coloboma. Ophthalmology 1996; 103:2120-2127

3. Gopal L. Khan B, Jain S, Prakash VS. A clinical and optical coherence tomography study of the margins of the choroidal colobomas. Ophthalmology 2007;114:571-580

4. Schubert HD. Schisis-like rhegmatogenous retinal detachment associated with choroidal colobomas. Graefes Arch ClinExpOphthalmol 1995;233:74 9.

5. Gopal L, Badrinath SS, Sharma T, Parikh T, Biswas J. Pattern of retinal breaks and retinal detachments in eyes with choroidal coloboma. Ophthalmology 1995;102:1212-1217

6. Toker E, Elcioglu N, Ozcan E, et al. Colobomatous-macrophthalmia withmicrocornea syndrome: report of a new pedigree. Am J Med Genet A 2003;121:2530.

7. Liu T, Zhang M, Xu B, Wang W, Lee J, Gao Y, Zhang X. Evaluation of the safety of sclerotomy incision in patients with choroidal colobomas with/without associated microcornea. Retina 2014;34:2300-2305

8. Gopal L. Kini MM, Badrinath SS, Sharma T. Management of retinal detachment with choroidal coloboma. Ophthalmology 1991; 98:1622-1627

SPOTLIGHT

Management of Difficult Situations in Posterior Segment Trauma

Authors

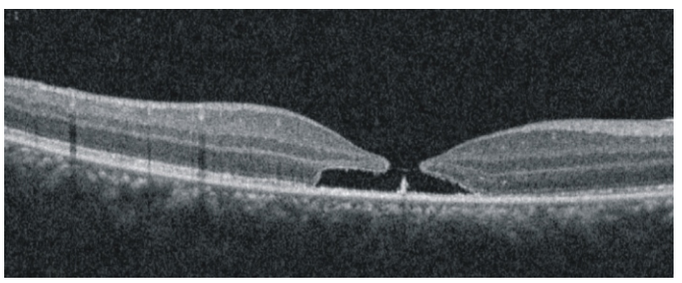
Dr. Rupak Kanti Biswas (RB)[1]
Dr. Anand Rajendran (AR)[2]
Dr. NS Muralidhar (NSM)[3]
Dr. Dhananjay Shukla (DS)[4]
Dr. S Natarajan (SN)[5]
Dr. Dhanashree Ratra (DR)[6]
Dr. Naresh Yadav (NY)[7]
Dr. VR Saravanan (VRS)[8]

Affiliation

1. Senior Consultant and Executive Director, BB Eye Foudation VIP, Kolkatta
2. Head, Retina-Vitreous Service, Aravind Eye Hospital, Chennai
3. President, Retina Institute of Karnataka, Bangalore
4. Senior Consultant, Retina–Vitreous Service, Ratan Jyoti Nethralaya, Gwalior
5. Chairman & Managing Director, Aditya Jyot Eye Hospital, Mumbai
6. Senior Consultant, Shri Bhagwan Mahavir Retina Service, Sankara Nethralaya, Chennai
7. Head, Retina Service, Narayana Nethralaya, Bengaluru
8. Senior Consultant, Retina-Vitreous Service, Aravind Eye Hospital, Coimbatore

SCENARIO 1:

RB / AR: A 26 year old male presents with history of trauma with cricket ballto RE 3 weeks back. VA was 6/24, N18. Fundus exam revealed macular hole and inferior speckled vitreous haemorrhage.



Questions:

How do you plan further management? How long would you observe before deciding on surgery?

What are the chances of spontaneous closure of the hole?

If Vitrectomy and ILM peeling is opted for, what technique of ILMP would you prefer? If partial RPE/macular atrophy or choroidal rupture is co-existent, would you alter your management or technique?

Answers:

NSM: 26 year old with traumatic macular hole with 6/24 vision can be observed for spontaneous closure. The history is of 1 month, hence I would wait for another month. If there is no spontaneous closure, then I would advise Vitrectomy.

However, the OCT of this patient shows unusual type of macular hole. There may be loss of tissue due to atrophy secondary to trauma. Hence the visual prognosis is more guarded.

In the largest series on traumatic macular holes published by Miller et al (1), eleven of the 28 holes closed spontaneously (39.3%) (Median 5.6 weeks range 1.767.3 weeks). All but 2 of these spontaneous closures occurred by 11 weeks, with only 1 occurring greater than 4.5 months after presentation. Hence I feel that a wait of 2-3 months is warranted before we intervene.

If surgery were needed, I would do a conventional surgery with ILM peel. Inverse flap is only planned in chronic holes and large holes. I would not alter my technique in the presence of co-existent RPE/macular atrophy or choroidal rupture.

DS: First, one should look for traumatic optic neuropathy (TON) by pupillary response (RAPD, indicates poor visual prognosis), then examine periphery for impact necrosis breaks which develop early.

This macular hole is small (separation at the edges), and has coexistent retinal atrophy rather than edema at the edges. I would observe for a minimum of a month (& can observe for a maximum of 6 months, if monthly follow-ups reveal a reduction in hole size). If hole stays the same/enlarges, I may operate early (2 months).

Spontaneous closure is likely if the macular hole is small, the patient is young, there is no PVD, and the hole occurs a bit later after trauma. Three of the above parameters are applicable in this case (no information on the last), and so waiting is the preferred option.

I do regular PPV+ILM peeling, no flapping/stuffing. RPE atrophy/choroidal rupture if present, change prognosis, but not the surgical technique.

SN: I will observe for three months. I will do every month serial oct. I will advise him to report on emergency if vision deteriorates.

There are 50 % chance of spontaneous closure of the hole.

I will do inverse flap ILM peeling technique.

If RPE/macular atrophy is there, will opt for amniotic membrane graft.

DR: Spontaneous closure of an idiopathic macular hole is rare. But in case of a traumatic macular hole the reported rate of spontaneous closure varies from 11% to as high as 40%. However, no clear cut predictive factors have been identified as yet. But holes with smaller overall diameter, tapered edges or bridging tissue on OCT and absence of intraretinal cysts have a higher chance of closing spontaneously. Therefore it has been advised to watch a traumatic macular hole for at least 6 months before deciding on surgical intervention.

Co-existing RPE atrophy, scarring or choroidal rupture through the fovea or adjacent to fovea is likely to compromise visual recovery. It can exert traction on the edges of macular hole and prevent closure even if surgical intervention is attempted. I would prefer to watch these holes without offering surgical correction. In cases where surgery is beneficial, my choice of procedure would be inverted ILM flap as it has a higher success rate especially where the basal diameter of the hole is larger.

NY: Traumatic macular holes in young people, can spontaneously close in 28% -40% of the cases, most holes close around 8 weeks. There are conflicting reports about OCT features, affecting traumatic hole closure. The absence of intra retinal cysts as seen in this patient, was found to increase the chance of spontaneous hole closure in one of the studies. The PVD status cannot be determined by the shown OCT. If the PVD is not present, one can safely wait before advising surgical intervention. In this case, I would like to wait for 6-8 weeks, before advising surgery. I would also like, to rule out traumatic optic neuropathy and other trauma related issues like traumatic retinal dialysis, RPE changes in macular area, photoreceptor damage, choroidal rupture etc. These associated changes can be picked up by performing, indirect ophthalmoscopy with depression, Fundus Fluorescein angiography and Fundus autofluorescence. These can have prognostic significance and the patient may need to be counseled accordingly.

The macular hole indices are not provided for this case. If the hole size is less than 450 microns, I will do Vitrectomy with ILM peeling and gas injection. If the hole size is larger than 450 microns, will perform inverted ILM peel.

VRS: Observe 3 weekly and follow-up with serial OCT to watch for spontaneous closure. Wait for 2 months and if the hole diameter is not shrinking or if it enlarges plan for vitrectomy with ILM peeling and gas tamponade.

Studies have put it at 40 - 70 percent.

Prefer to do inverse flap or temporal flap. Would consider retinal graft if the hole is more than 1500 microns or significant atrophy is present.

Take Home Pearls

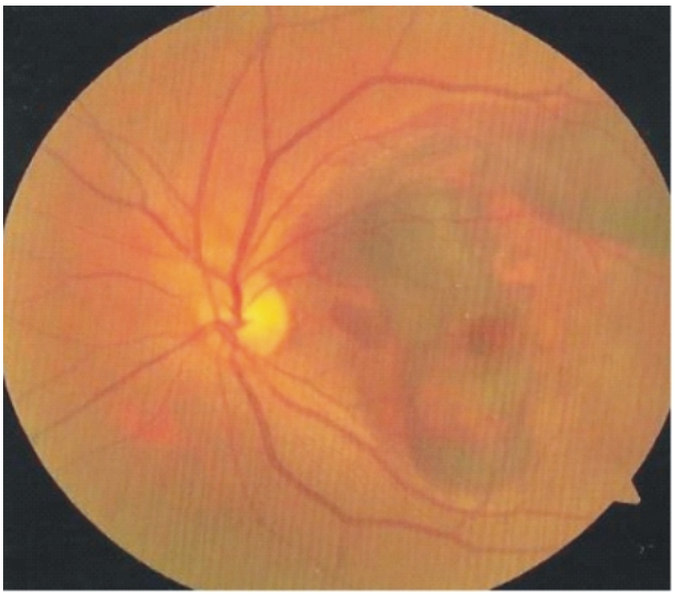
It is a good idea to watch a traumatic macular hole for 2-3 months before deciding for surgical intervention, as majority of spontaneous hole closure occurs during this period. Smaller hole size, tapered edges and absence of intra-retinal cysts are positive influencing factors for final hole closure. Visual outcome depends on associated changes like RPE atrophy, choroidal rupture under the hole, etc. Associated implications of trauma also to be kept in mind are traumatic optic neuropathy, retinal dialysis, zonular dialysis, traumatic cataract, etc and to be treated accordingly.

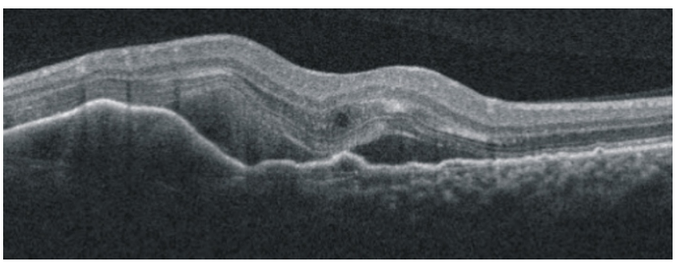
SCENARIO 2:

RB / AR: An 18 year old boy presents with history of blunt trauma, to the LE, with a football 1 week back. VA was 5/60. Fundus examination revealed sub-macular haemorrhage.

Questions:

Do you see a role for pneumatic displacement of traumatic sub-macular haemorrhage at this point and what are your criteria for pneumatic displacement and vitrectomy respectively?





What is the role of tPA in such cases?

What complications, if any, have you faced?

Answers:

NSM: In the case presented with traumatic submacular hemorrhage, I will consider pneumatic displacement, since there is significant hemorrhage.

I don't use tPA with pneumatic displacement, as I have obtained good results without its use.

Vitrectomy is reserved for thick arcade to arcade hemorrhage, or massive subretinal hemorrhage. I do use tPA during vitrectomy.

We have not faced any complications due to pneumatic displacement. Cataract (commonly) and Retinal detachment(rarely) have occurred after vitrectomy for submacular hemorrhage.

DS: Again, I begin with assessment for refraction (myopia), RAPD (TON), choroidal rupture, central fovea (not seen in this cut of OCT), peripheral retinal status, thickness of subretinal heme (SRH; thin here), and presence of sub-RPE heme (present here), and co-existing choroidal ruptures. Thin SRH commonly resolves sans intervention, at least in a young near-emmetropic person with healthy RPE (likely in this case). Sub-RPE heme is refractory to treatment.

If there is no TON, macular hole, commotio retinae or peripheral pathology AND the patient is older & myopic, SRH is thick AND EXPLAINS THE POOR VISION, I'd prefer pneumoretinopexy with 100% 0.3-0.4cc C3F8 (over vitrectomy) as the initial procedure: I have enjoyed good success with this simple procedure sans R-tPA, which is expensive, and may not contribute to the success, specifically in traumatic SRH.

The main issues with gas injection are avoiding fish-egging of the gas bubble, flat AC, and a gas leak into AC (with phacodonesis). To avoid these issues, start the patient on IV Mannitol 20% and tap AC repeatedly to make the eye mushy soft before injection. Don't tap (as far as possible) AFTER gas injection, if central retinal artery is perfused.

SN: Yes,there is a role for pneumatic displacement of traumatic sub macular haemorrhage.

My criteria: I will avoid vitrectomy for this case. Only if severe sub macular haemorrhage happens.

If there is clotted blood, I will use tpa.

One patient developed retinal detachment following vitrectomy.

DR: Submacular haemorrhage causes sudden, profound loss of vision. The toxic effects from the hemosiderin as well as the tractional and shearing forces from the contracting fibrin in the subretinal space rapidly lead to permanent damage to the photoreceptors. By 15 days there can be atrophy of the outer retina. Thus, the duration and the size of the haemorrhage are the main deciding factors. Pneumatic displacement with or without tissue plasminogen activator (tPA) is a relatively less invasive procedure and works well with moderate sized haemorrhage of less than 2 weeks duration. The tPA causes liquefaction of blood and the gas bubble helps in displacing it away from the fovea. Vitrectomy with or without tPA can be reserved for massive subretinal haemorrhage, with break-through vitreous haemorrhage which is more often seen in older population secondary to neovascular AMD or PCV. The tPA can be injected in the intravitreal or subretinal space. Failure to displace the blood can occur if it is already organized in the subretinal space. Other complications such as rebleeding, retinal breaks, detachment and PVR are rarely seen in case of massive haemorrhages. However, here the haemorrhage is beneath the retinal pigment epithelium which is evident from the darker colour of the blood on fundus photo and the irregular elevation of the RPE on the OCT. These sub-RPE haemorrhages are more difficult to displace and carry a less favourable prognosis. Inability of the tPA to reach the sub-RPE space might be the reason for nonclearance of the haemorrhage. To address this issue a two-step approach has been recommended by some, wherein the tPA is injected in the subretinal space during the initial vitrectomy followed by injection of 4-5 cc of heavy perfluorocarbon liquid. Around 7-15 days later a second surgery is performed for the removal of the PFCL and drainage of the liquefied blood. This allows for a better clearance of the sub-RPE haemorrhage. The visual outcomes however may remain unsatisfactory.

NY: The Fundus photograph shows sub macular hemorrhage secondary to blunt trauma. The OCT shows, sub retinal hemorrhage along with sub RPE hemorrhage involving the foveal area. The persistence of sub retinal blood can damage the photoreceptors within 24 hours, thus early pneumatic displacement of this subretinal blood from the macular area is desirable. The treatment approach usually depends up on the size and amount of hemorrhage along with the interval time between onset and treatment. As this patient has reported within 1 week of the trauma, the sub retinal hemorrhage is likely to get displaced by just gas injection (pure 0.3ml SF6). The gas, will pneumatically displace the blood away from the macula. If the patient had come after 2 weeks of injury, the patient would have required Gas injection and intra vitreal TPA injection (Can degrade the fibrin clot). Patients with massive sub macular hemorrhage (extending beyond the arcades) usually need vitrectomy with sub retinal/ intra vitreal TPA injection and gas injection. The subretinal TPA injection is given with the help of a fine 41 Gauge needle, the maneuver is technically challenging. Automated injector is desirable as it avoids the tremors associated with manual injection. The risks associated with sub retinal TPA are break through vitreous hemorrhage, intra op macular hole formation, RPE rip, retinal and RPE toxicity etc. Intra operative hole formation is more likely to happen in patients who have sub macular hemorrhage secondary to Retinal artery macroaneurysm (RAM). Correct formulation of the TPA dose is mandatory, I usually don't give more than 50 micrograms /0.1ml. Intravitreal TPA, can be used instead of sub retinal injection along with vitrectomy. The discontinuity in the ILM may provide access to the sub retinal space from the vitreous cavity. The most common side effect I have, encountered is break through vitreous hemorrhage after pneumatic displacement and intravitreal TPA injection.

VRS: Yes. Eventhough part of the haemorrhage seems to be sub RPE, considerable amount of blood is sub retinal and displacement can lead to partial if not total recovery of vision. Vitrectomy can be reserved for cases with significant vitreous haemorrhage or post displacement breakthrough vitreous haemorrhage.

I would reserve tPA for ARMD or PCV cases

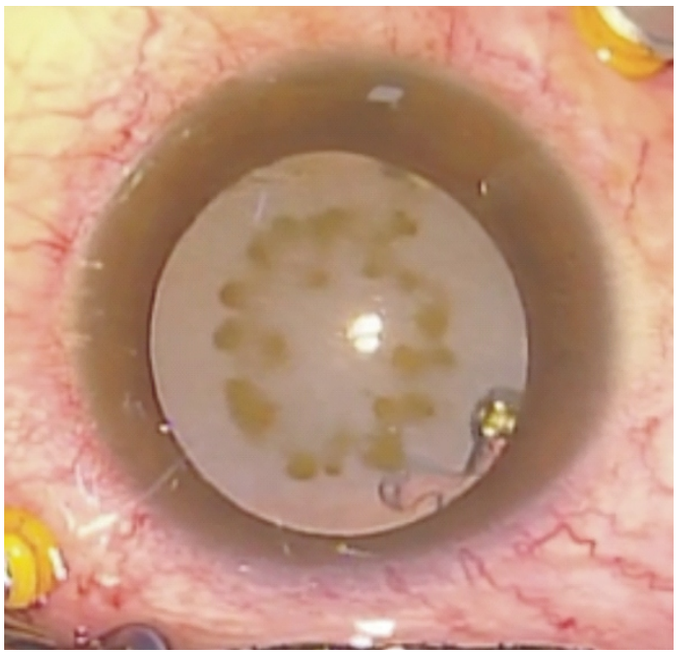
Failure to displace the blood. Breakthrough vitreous hemorrhage. Retinal detachment. Macular hole rarely.

Take Home Pearls

In case of thin SRH we can wait and watch for some time as they commonly resolve without intervention, at least in a young near-emmetropic person with healthy RPE and less than 2 weeks duration. The duration and the size of the haemorrhage are the main deciding factors for treatment. Early displacement of the haemorrhage is necessary to prevent photoreceptor damage. In fresh cases where the blood is not clotted only gas injection can suffice, whereas in case of clotted blood tPA may be necessary. Vitrectomy with or without tPA can be reserved for massive subretinal haemorrhage, with break-through vitreous haemorrhage which is more often seen in older population secondary to neovascular AMD or PCV. Complications such as rebleeding, retinal breaks, detachment and PVR are rarely seen in case of massive haemorrhages. The risks associated with sub retinal TPA are break through vitreous hemorrhage, intra op macular hole formation, RPE rip, retinal and RPE toxicity, etc.

SCENARIO 3:

RB / AR: A 53 year old man presents with history of penetrating trauma to the LE 1 year back. VA was FCCF. Siderotic cataract with entry wound found in inferior iris.Fundus examination revealedretinal detachment with sclerosed vessels.



Questions:

What is the prognosis in cases of retinal detachment with siderosis?

If the IOFB is incarcerated in the sclera with retinal scarring and fibrosis, how would you manage?

Answers:

NSM: The combination of siderosis and retinal detachment carries poor prognosis.

I would make all attempts to remove the foreign body in every case. If there is a risk of scleral incarceration site opening up, then we may be forced to leave the foreign body in situ. There is a risk of the foreign body disintegrating while catching it if it is old.

DS: Prognosis (you mean visual, I presume) is surprisingly good even in presence of a nearly flat ERG. I would check the size of longstanding IOFB with CT/X-ray preoperatively: if it has already disintegrated, only retinal detachment needs to be addressed.

If a small IOFB is impacted deep into posterior sclera with thick overlying scar, it has to be chronic. In this case, if there is no clinical or electrophysiological evidence of siderosis, I'd leave the IOFB alone. If IOFB is large (with great velocity, to penetrate and get deeply impacted posteriorly), it'd typically have already damaged the eye enough not to warrant removal anyway. If siderosis is present and progressive, scar tissue can be excised for IOFB removal. Sometimes, retina detaches when the scar is excised, so one should be prepared to deal with it.

SN: The prognosis is poor.

I will do vitrectomy, perform endo laser around the site of impact of IOFB under PFCL, then dissect the fibrosis , use the MVR blade to dissect the IOFB, use IOFB forceps to grasp and remove the foreign body. I will use 1000 cs silicone oil as tamponade.

DR: Siderosis per se is a poor prognostic indicator. When associated with retinal detachment it can mean further worsening of visual prognosis. However, isolated case reports and a case series have reported stabilization and rarely reversal of siderosis following surgical repair and removal of the foreign body. The changes in the electrophysiology due to siderosis were seen to remain stable for many years following surgical repair of retinal detachment. One case series has reported chance of visual improvement following removal of foreign body in nearly 60% eyes with 20% remaining stable and 20% worsening. It is worth considering surgery for such eyes, but at the same time one must be wary of complications such as cystoid macular edema, peripheral non-perfusion, retinal pigment epithelial atrophy, and secondary glaucoma which can lead to loss of vision. If pre-operatively the IOFB is seen to be incarcerated in the sclera with retinal scarring and fibrosis, one has to weigh the pros and cons of intervention. If the IOFB is well covered chances of the iron dispersion might be less and the IOFB can be left alone as it is not likely to cause much further damage. The site of incarceration plays a major role in this decision. If near the macula or the optic nerve head, it is better to be observed. Often

times, removal may entail more damage in terms of retinal break, detachment, optic nerve injury, massive bleeding or a large defect in the eyewall. However, if intraoperatively such a situation is noted, removal can be attempted by cutting the capsule over the IOFB and gently teasing it out. It is best to leave the rest of the capsule which is adherent to the retina undisturbed to avoid trauma to the retina.

NY: The clinical findings in siderosis bulbi, include iris heterochromia, cataract formation, retinal pigmentary changes, secondary glaucoma etc. The removal of IOFB can stop the progression of siderosis in most of the cases, thus early surgical removal is mandated. The post op visual acuity in siderosis bulbi cases after IOFB removal, depends upon the initial visual acuity, size of IOFB, location of the IOFB in the eye and presence of pre-op RD. The post op visual acuity, is going to be guarded in this particular patient because of the retinal detachment. Before taking up this patient for surgery, I would want this patient to undergo Electro retino graphy (ERG). Iron retino toxicity leads to more damage to the inner retina. As siderosis worsens the b wave decreases, causing the b -wave/ a- wave ratio to fall. The vision could be good in siderosis with ERG amplitudes of up to 50% and complete reversal is possible in amplitudes up to 40%. This patient has complicated cataract with RD and retained IOFB in the retinal periphery. This patient will need combined surgery, the cataract removal will help in reaching the retinal periphery without worrying about lens touch. The IOFB does not seem to be covered by any fibrous capsule, as such can be easily lifted by intra ocular magnet, after doing a thorough vitrectomy. In long standing cases the retained metallic IOFB can lose its magnetic property, and one may have to use intra ocular forceps. Once the IOFB is in the mid vitreous cavity, it can be removed either via limbal or sclerotomy route (hand shake approach) depending upon the size of the IOFB. The intra ocular lens can now be implanted and the retinal detachment can be settled by doing fluid air exchange and by using the appropriate tamponade. If the IOFB is incarcerated in the sclera with fibrosis, we may have to tease out the IOFB from the capsule, with the help of an MVR blade. If the surrounding retina is scarred and incarcerated, a relaxing retinotomy may be needed.

VRS: Very poor visual recovery in spite of successful retinal reattachment.

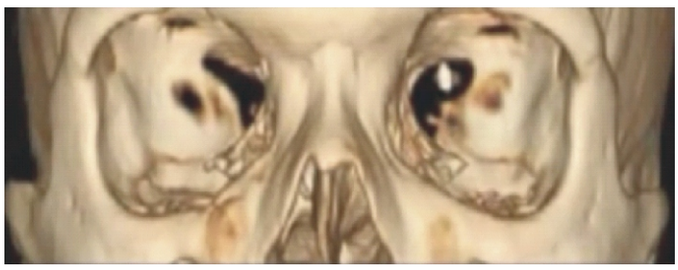
This risk of siderosis is very less if the FB is not exposed to vitreous fluid. If the chronic FB is under the choroid, I will leave it in situ with serial ERG every 4 - 6 months. If signal drop is noted go in and remove IOFB. Educate patient on not doing MRI if the IOFB is magnetic.

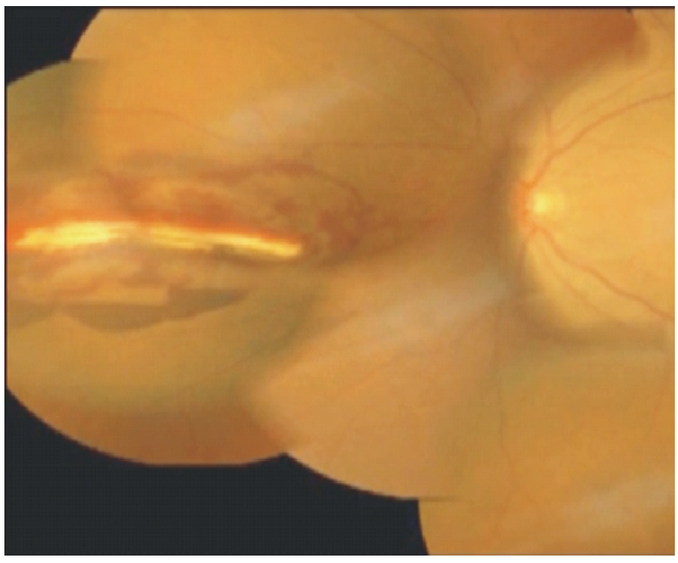
Take Home Pearls

Siderosis itself is a poor prognostic factor, on top of it retinal detachment causes further worsening. In some rare cases though, visual prognosis may be surprisingly good even in presence of a nearly flat ERG. Isolated case reports and a case series have reported stabilization and rarely reversal of siderosis following surgical repair and removal of the foreign body. So an early intervention is always better. The post op visual acuity in siderosis bulbi cases after IOFB removal, depends upon the initial visual acuity, size of IOFB, location of the IOFB in the eye and presence of pre-op RD. In long standing cases the retained metallic IOFB can lose its magnetic property, and one may have to use intra ocular forceps. If the IOFB is incarcerated in the sclera with fibrosis, the scar tissue surrounding it may have to be excised with the help of an MVR blade and the IOFB teased out from the capsule to remove it. Special instructions to the patient regarding avoiding MRI is very important.

SCENARIO 4:

RB / AR: A 45 yr old man presented with history of bomb blast injury to the Left eye 5 days back.VA was FCCF. Subconjunctival haemorrhage, hyphaema, hypotony noted. Fundus evaluation





showed choroidal rupture extending 6 mm nasal to optic disc with shallow RD in nasal periphery. 3D reconstructed CT Scan found a shrapnel that perforated the eye and was located very close to the optic nerve in the posterior part of the muscle cone.

Questions:

What should be the approach in this case?

How do you close the posterior scleral wound?What are the options for managing this posterior scleral wound?

Answers:

NSM: I would take up this case for exploration and scleral tear repair, Vitrectomy and endolaser with silicone oil injection.

Scleral wound is closed with 7-0 or 8-0 nonabsorbable sutures. If needed, the medial rectus can be isolated and detached to gain access to the posterior part of the scleral tear. After the scleral tear closure, vitrectomy is done along with PVD induction. Endolaser to retinochoroidal rupture site and silicone oil injection is done. The vitrectomy can be postponed by 10-15 days, if it is felt that water tight closure of the scleral tear is difficult to allow vitrectomy during the primary repair.

The orbital foreign body is best left alone, as efforts at removal may damage the optic nerve.

DS: Extraocular intra-orbital metallic foreign bodies (FB) are mostly inert, and may be left undisturbed. In this case, since FB may not be inert (blast fragment), I'd consult my oculoplasty colleagues, who might remove the shrapnel threatening the optic nerve (after detailed CT localization).

Since a clear fundus photograph could be obtained, I am assuming the intraocular inflammation is controlled and there is no GROSS hypotony in this case. A key concern is Hand Motions vision, difficult to explain by the fundus picture. Generally posterior scleral wounds self-seal within a week, and don't warrant repair. If there is no visual potential, I would only attempt laser barrage around the perforation. If there is hope for visual recovery, I would perform vitrectomy with release of any incarcerated vitreous (retina appears intact except for break causing the detachment), since this exit wound may invite severe fibrotic scar formation. If preoperative OCT shows any vitreoretinal traction, I might remove ILM at macula proactively. If silicone oil or gas leaks posteriorly at the end of the surgery, fibrin glue might be considered, though I have no personal experience with it.

SN: We should do vitrectomy. Since there is hypotony, I will inject air into vitreous cavity to firm up globe. Then tag muscle gently, place radial buckle to close the scleral perforation. Following that I will do vitrectomy, PFCL, endo laser around the perforation, silicone oil as tamponade.

DR: This is a very challenging situation and ideally requires a multidisciplinary approach. I would prefer to suture the posterior scleral wound by external approach using 6-0 or 7-0 vicryl suture. A hand-over-hand technique of suturing can be adopted where the ends of the preceding suture are purposely cut long and are held to provide traction and guidance to reach the posterior extent of the scleral tear. It would also be better to do cryo to this wound and place a radial buckle to give more support. More often than not this external approach is likely to be more successful in closing the wound and addressing the retinal detachment than an internal approach. The removal of the shrapnel from inside the muscle cone would need careful evaluation of possible injury to the optic nerve. It would be better to seek the help of an Oculoplastic surgeon for performing an orbitotomy and safe removal of the intraconal foreign body.

NY: The presence of sub conjunctival hemorrhage, hyphema, hypotony indicates that there is an occult scleral rupture in this patient. Occult rupture needs emergent repair, as delay can lead to expulsive choroidal hemorrhage, endophthalmitis etc. The fundus photograph shows, a large linear radially oriented scleral tear, the posterior extent is around, 5 DD from the optic disc. The scleral wound repair, in this patient needs to be planned under GA. Gentle peritomy and conjunctival separation is done around the medial rectus. Basically, step wise, opening of the conjunctiva to expose the scleral wound is needed. The wound edges are to be thoroughly cleaned, as this is blast case. The sclera, is a relatively thin tissue (0.3mm- 1.35mm), smallest adequate needle and suture must be used and I usually prefer 7 - 0 Vicryl suture. The approach of scleral wound repair, is called as close- as- you- go approach. Interrupted sutures are placed to close the wound, one end of the knot can be left long and this can be pulled to rotate the globe (Acts as traction suture). Try to reposition the uveal and retinal tissue, the prolapsed vitreous needs to be excised with a vitrector. In this case the medial rectus may have to be temporarily disinserted, so as to allow suturing. Closure of the laceration should continue, till it is safely possible without applying undue force on the globe. Very posterior lacerations can close on their own from the physiologic tamponade by the orbital tissue and scarring. As the medial rectus is already disinserted in this case, a 5 0 Dacron placed through the muscle tendon helps to rotate the globe and to access the area near the optic nerve (Malleable retractors and cotton applicators, used to reflect the posterior tenon capsule and intra orbital fat). The shrapnel, can thus be safely removed. The radial scleral wound here can be supported by a radial sponge. There is only a small area of localized RD nasally, which can be managed by gas injection after LIO laser/Cryopexy around the scleral wound. There is a possibility that this patient could develop recurrent RD later on, because of PVR. This may warrant vitreous surgery with adequate tamponade. Small posterior perforations, can open up during vitrectomy and these can be, plugged by scleral patch graft or amniotic membrane graft.

VRS: Usually vitreous incarceration into the wound causes scarring which will act like a barrage and no further medical or surgical intervention is required. If a rhegmatogenous RD is progressive the patient can be taken up for surgery after allowing a few days for the wound to scar (5 - 7 days). Intervening earlier will cause BSS leakage into the orbit apart from high risk of intraoperative hemorrhage. Removing the intraconal FB can be risky with intraoperative optic nerve damage and vision loss and requires the help of a neurosurgeon or ENT surgeon and may be safe to leave the FB insitu if there is no vision drop.

The wound heals on its own with fibrosis of sclera and episcleral tissue in a few days. If a rhegmatogenous RD is progressive the patient can be taken up for surgery after allowing a few days for the wound to scar (5 - 7 days).

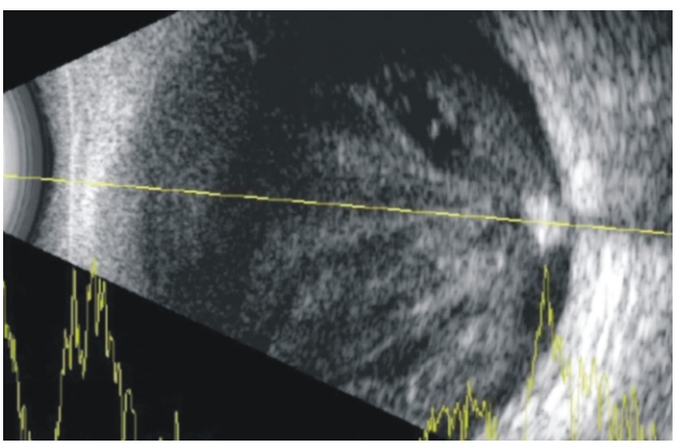
Take Home Pearls

Some posterior scleral wounds may self-seal within a week not requiring repair. If large and in presence of RD the scleral wound is closed with 7-0 or 8-0 nonabsorbable sutures. The medial rectus can be disinserted to gain access to the posterior part of the scleral tear to suture it. A hand-over-hand technique of suturing can be adopted where the ends of the preceding suture are purposely cut long and are held to provide traction and guidance to reach the posterior extent of the tear. A radial buckle can be placed at the scleral perforation. Vitrectomy is the only option here to repair the nasal RD. Laser around the tear with silicone oil or gas tamponade is needed. If it leaks posteriorly at the end of the surgery, fibrin glue might be considered to reinforce the tear.

The orbital foreign body is best left alone, as efforts at removal may damage the optic nerve.

SCENARIO 5:

RB / AR: A 44 year old man presents with history of trauma to the RE with an iron particle 2 weeks back. VA was PL +, PR accurate. Conjunctival congestion, corneal haze, hypopyon measuring 0.5 mm found. No fundusview. USG B-Scan revealed vitreous exudation along with an IOFB.



Questions:

What are the causative organisms you usually encounter in traumatic endophthalmitis?

Do you do PVD induction if there is a retinal tear with an IOFB incarcerated in the retina with endophthalmitis as seen here?

Do you use silicone oil even when the retina is attached?

What are your intravitreal antibiotics of choice is this setting and do you reduce the dose if injecting after silicone oil?

Answers:

NSM: Traumatic endophthalmitis is commonly caused by Gram +ve organisms. Bacillus species are the most common pathogens reported in the literature.

In the case presented, I would attempt PVD induction, and avoid only if the retina appears necrotic.

I use silicone oil in cases of severe endophthalmitis and co existent RD.

Vancomycin and Ceftazidime are my choice of antibiotics initially. Only if the culture results warrant, I would change the antibiotics. The dosage of antibiotics in silicone filled eyes is half of that used normally.

DS: Causative organisms for traumatic endophthalmitis are generally similar to postoperative endophthalmitis (at least in Indian scenario, where postoperative infections are varied and more severe than what EVS study found), though gram negative, fungal and polymicrobial infections are more common.

PVD induction is useful; but by no means mandatory. I focus on removal of infection load, not the IOFB, which I may leave behind for a 2nd intervention, if difficult to remove with CORE vitrectomy.

I don't use silicone oil, but I commonly perform fluid-air exchange, at least partially, and leave sclerotomies un-sutured in all cases.

My intravitreal antibiotic preference is Vancomycin +Ceftazidime +Dexamethasone, same as for postoperative endophthalmitis. I replace Ceftazidime with Amikacin if cornea is infiltrated; and add Voriconazole in agricultural/organic trauma.

SN: It could be multiple organisms.

Yes I induce PVD.
Yes.

I use vancomycin with clindamycin. I use the same dose with oil in severe infection.

DR: In the setting of endophthalmitis, the retina is edematous and fragile. Even the slightest traction or suction force can lead to a retinal break or worsen an existing break. And the chances of PVR are very high in endophthalmitis. Thus, it is advisable to do minimal manipulation of the retina. I prefer to clear the vitreous around the IOFB enough to remove it without exerting traction on the residual vitreous. But I certainly never attempt to induce a PVD. Even if there are thick exudates stuck to the retina along with the posterior hyaloid, in my experience aggressive attempts at removal only lead to further complications. I generally leave the residual vitreous along with the PHF attached to retina. In the post-operative period, very often the vitreous exudates clear and a spontaneous PVD is seen to occur. Or else a second surgery for the removal of the residual vitreous can be planned once the endophthalmitis has been stabilized and sterilized. If the retina is attached, I avoid silicone oil and prefer gas in order to tamponade the retinal break. But in the presence of retinal detachment silicone oil is essential. Intraoperatively, I inject full dose of the antibiotics in the fluid filled vitreous cavity before injecting the silicone oil. It is advisable to reduce the dose to half if injecting in the silicone oil filled cavity. The choice of antibiotics depends on the causative organisms. Generally, Bacillus is seen to be commonly present in the presence of an IOFB or soil contamination. Other gram positive organisms such as coagulase negative staphylococci, streptococci are also common. 10-15% infections are due to gram negative organisms such as pseudomonas and 10 % can be due to fungi. It is always better to combine 2 broad spectrum antibiotics such as vancomycin and ceftazidime. Third generation cephalosporins or fluoroquinolones are preferred for Bacillus infection. Antifungals such as voriconazole or amphotericin B can be added if fungal etiology is suspected.

NY: The patient has a retained metallic IOFB with traumatic endophthalmitis. The common causative organisms cultured in traumatic endophthalmitis are: Gram positive organisms like, Staph Epidermidis, Staph aureus, Streptococcus species, Bacillus (spore forming rod), Clostridium etc. The Gram-negative organisms are, Pseudomonas species, Proteus species etc. The Fungal species involved are Candida albicans and Aspergillus. The case discussed here, needs vitrectomy with intra ocular antibiotics injection and IOFB removal at the earliest. If the intra operative visualization is good, one should aim for as complete vitrectomy as possible. If the view is not good, one needs to be a little conservative in performing vitrectomy. If there is retinal break near the IOFB, clearing the vitreous is mandatory. Using intra ocular forceps with attached vitreous, can pull on the retina and enlarge the retinal tear. One needs to remember that the associated inflammation, helps in induction of the PVD. The PVD induction helps in performing a safer and complete vitrectomy, it also makes the grasping of the IOFB easier. The choice of initial antibiotic is always empirical, one needs to cover both Gm positive and Gm negative organisms. My first choice still is, intra vitreal Vancomycin (1mg/0.1ml) and Ceftazidime (2.25mg/0.1ml). The history regarding the injury is not provided, if there is soil contamination or rural setting, the causative organism could be Bacillus. Bacillus species respond well to intra vitreal Piperacillin / Tazobactam (250 microgame/0.1ml). All patients need to be started on systemic antibiotics also, my choice will be intra venous Ceftazidime or intra venous Clindamycin if suspecting Bacillus species. Silicone oil has been reported to decrease bacterial and fungal survival in vitro. The viscous silicone oil may act as a physical barrier to deprive the bacteria of nutrition, and the components of the oil may be toxic to the microbes. The other concern is the relative high number of Gm negative cases and high incidence of antibiotic resistance in our part of the world. The additional intervention of injecting SOI may help, mitigate the bacterial proliferation. Taking all these factors into account I would like to inject Silicone oil in this particular case. In vitro experiments have demonstrated that only small proportion of antibiotics mix with silicone oil. The intra vitreal concentrations of antibiotics are several orders of magnitude higher than the 90% minimum inhibitory concentration(MIC 90). This allows us to give half dose of the intra vitreal drug, without worrying about the efficacy in a silicone oil filled eye, where the aqueous volume is very less.

VRS: Bacillus species, Streptococcus pneumonia, staph aureus etc

If the view is clear or relatively clear and no significant retinal necrosis is noted would prefer to induce a PVD especially in the presence of a retinal tear.

No tamponade preferred if no retinal breaks or detachment is noted.

Full dose routine Vanco and Cefta till culture comes up with specific sensitivity results.

Take Home Pearls

Traumatic endophthalmitis can be caused by varied organisms. Bacillus species are the most common pathogens reported in the literature. If the intra operative visualization is good, one should aim for as complete vitrectomy as possible. If the view is not good, one needs to be a little conservative in performing vitrectomy. If there is retinal break near the IOFB, PVD is mandatory.Necrotic retina may be a hazard for PVD induction. Silicone oil has been reported to decrease bacterial and fungal survival in vitro. The viscous silicone oil may act as a physical barrier to deprive the bacteria of nutrition, and the components of the oil may be toxic to the microbes. Vancomycin and Ceftazidime are the choice of antibiotics initially. Antibiotics can be changed if the culture results warrant. Amphotericin B/ Voriconazole may be injected for suspected fungal infection. Half dose of intravitreal injections in silicone oil filled eyes is the general consensus.

SCENARIO 6:

RB / AR: A 12 year old boy presents with history of trauma to LE with cricket ball 2 weeks back. VA was 6/9, N6. Fundus examination revealed retinal detachment in the nasal half along with a retinal dialysis at supero-nasal quadrant from 10 to 12 O` clock position.

Questions:

Scleral buckling / Vitrectomy Which approach do you prefer for managing this case?

Your tips and tricks for a successful outcome?

Answers:

NSM: In this 12 year old boy with nasal detachment due to dialysis, my first choice would be to do a scleral buckling surgery. Since there seems to be no PVR changes, Vitrectomy may not be indicated.

Buckling is ideal in this scenario, as vitrectomy carries more challenges. Even in the presence of subretinal bands, we can get away with SB, as long as the bands are not causing retinal elevation.

While performing SB, it is important to make sure that the anterior part of the dialysis is supported fully.

DS: Scleral buckling: #287 (biconvex profile) segment with a #240 encircling band, SRF drainage SOS (if fluid present on buckle).

Use mannitol IV preoperatively to avoid SRF drainage if possible, and give oral and topical acetazolamide to aid the RPE pump, esp. with non-drainage. Inject air for internal tamponade if SRF is drained. Avoid heavy intraoperative cryopexy/postoperative laser, which may cause macular pucker.

SN: I prefer scleral buckling.

My trick

I do 360 degree peritomy, do gentle tagging of muscles,place sutures for buckle, make scleral tunnel for 240 band 360 degree, place Watzke sleeve,localise the break, gentle cryo, do SRF drainage, inject non expansible gas of 0.3 to 0.5 cc gas to manage hypotony. Tighten buckle gently to have 360 degree buckle effect. Tighten buckle suture to close the dialysis.

Usually the patient maintains 6/6 with minimal myopia.

I have extensive experience with this trick.

DR: Scleral buckle with or without subretinal fluid drainage is my treatment of choice in this scenario. Here the vitreous is likely to be a formed, viscous gel adherent to the dialysis. It is easy to appose the edges of the dialysis with a scleral buckle placed at the ora. It almost never requires bulky, large buckles but care should be taken to see that postoperatively the slightly receeded posterior edge of the dialysis falls on the buckle. Very often, the wide gaping dialysis which creates an illusion of being very posterior, flattens well after the drainage of subretinal fluid, with the edge settling far anteriorly than expected. PVR is almost never seen in these typical cases of dialysis. Thus these cases are more amenable to scleral buckling than vitrectomy.

NY: We all agree that Pars plana vitrectomy has gained favor over Scleral buckling (SB) in the management of rhegmatogenous retinal detachment (RD). However, SB still remains relevant for certain types of RD and Retinal dialysis associated RD is one such indication. In retinal dialysis, the retina gets disinserted from the vitreous base. It is important to differentiate retinal dialysis from giant retinal tear, as the surgical management of the two usually differ. Multiple studies have shown the safety and efficacy of scleral buckling in the management of retinal dialysis causing RD. I would perform scleral buckling in this patient. SB in patients with retinal dialysis is simpler as the break is located near the ora serrata (localizing and cryopexy is easier), The detachment is shallower in the periphery(may not need drainage), there is no PVD and the vitreous is more formed, the PVR is rarely seen, is a slowly progressive RD etc. In this case the retinal dialysis just extends for 2 clock hours , I would just place a segmental 279 buckle(Shallow effect) supporting the dialysis. This way I don't have to perform 360 degree limbal peritomy and tag all the muscles, the post-operative refractive change will be minimal this way. The vitrectomy in young people can be quite challenging, inducing PVD is not easy, there is risk of cataract, tamponade will cause refractive issues, trouble of maintaining position, risk of post-operative PVR (if Sx fails) etc. SB is an elegant and very useful surgery and all retina surgeons should be comfortable doing it. Even in this era of PPV, SB still has some indications and its not ethical to deny any patient when the eye condition demands it.

VRS: Scleral buckling preferred unless the dialysis is more than 6 clock hours with rolled edges.

Avoid very posterior placement of scleral buckle because it leads to leakage anteriorly with eventual reRD. Can use intraoperative gas if edges are very raised. Can do LIO laser in the postoperative period after successful retinal reattachment. Adequate control of postoperative inflammation with oral and topical steroids.

Take Home Pearls

It is important to differentiate retinal dialysis from giant retinal tear, as the surgical management of the two may differ drastically. Multiple studies have shown the safety and efficacy of scleral buckling in the management of retinal dialysis causing RD. Scleral buckling is the first choice in the described scenario. Care should be taken to see that postoperatively the slightly receded posterior edge of the dialysis falls on the buckle. It is also important to make sure that the anterior part of the dialysis is fully supported. Gas can be used if the edges of the break are raised. LIO in the post op period can reinforce the buckle.

Acknowledgements: The authors acknowledge the efforts of the following in the compilation of the manuscript.
Dr. Krishnendu Nandi,
Senior VR Consultant, BB Eye Foundation VIP, Kolkata.
Dr. Maharshi Maitra,
VR Fellow, BB Eye Foundation VIP, Kolkata.

RETINA TECH

Clinical applications of multicolor imaging

Authors
1. Rupak Roy (MS)[1]
2. Kumar Saurabh (MS)[2]
3. Sugandha Goel (DNB)[3]

Affiliation
1. B B Eye Foundation, Kolkata
2. Aditya Birla SankaraNethralaya, Kolkata-700 099
3. MM Institute of Medical Sciences & amp; Research, Ambala

Multicolor imaging (MCI) is a non-invasive retinal imaging modality available in Spectralis platform (Heidelberg Engineering, Heidelberg, Germany). It simultaneously acquires three reflectance images of the retina using three individual lasers of different wavelength: blue (488nm), green (515nm) and infrared (820nm). These penetrate the tissue to different depths, simultaneously capturing and depicting information originating from different retinal structures. The infrared reflectance (IR) image visualizes structures at the level of the outer retina and choroid. The green reflectance (GR) image allows imaging of retinal blood vessels, haemorrhages and exudates. The blue reflectance (BR) particularly provides details of the inner retina and the vitreoretinal interface such as epiretinal membranes, retinal nerve fibre layer (RNFL) thinning and macular pigment changes. The information from these three images are integrated to form a composite multicolor image.(Figure 1) [1]

Color fundus photography (CFP) is currently the most commonly used fundus photographic modality. CFP utilises broad spectrum of illumination and it is the imaging modality which best correlates with clinical ophthalmoscopy.[1] However it has various

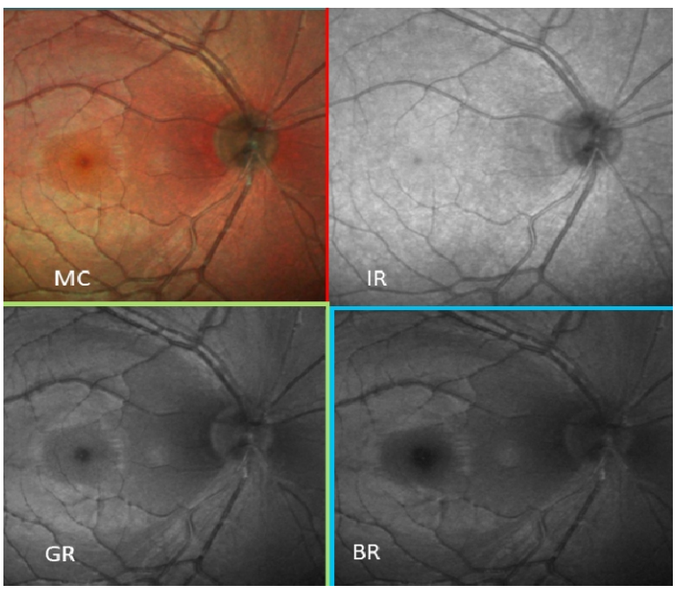


Fig.1: Composite image showing normal Multicolour composite image (MC), Infrared reflectance image (IR), Green Reflectance image (GR) and Blue reflectance image (BR)

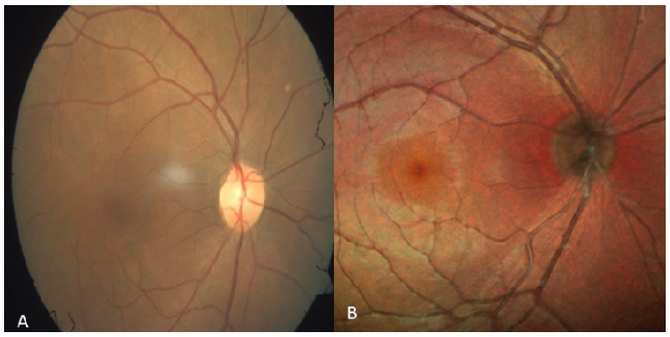


Fig.2: Image showing a standard normal white light colour fundus photograph (A) and a normal composite multicolour image (B)

limitations. It uses bright white light and needs dilated pupil, thus making it uncomfortable for patients. It is difficult to obtain good images in presence of media opacities. The images obtained have low contrast and lesion margins are not well delineated. MCI has the inherent advantages of much superior confocal scanning technology. Confocal technology results in sharp image with higher resolution and contrast. Inbuilt live eye-tracking technology and "noise" reduction techniques enhances image stability and clarity.[2] Other advantages include small pupil scanning and avoidance of discomforting white light, as used to obtain CFP. Other unique advantage of this modality is completion of all scan acquisitions in a single machine and utilisation of the rescan follow up mode of the Spectralis platform for patients having multiple image acquisition at multiple time points.[1]Multicolor images can be captured with both 30° and 55° field of view along with three reflectance images (BR, GR and IR). Figure 2 shows the appearance of colour fundus (Fig2 A) and a composite multicolour image (Fig 2B). Herein, we describe the utility of MCI in various retinal, choroid and optic nerve head disorders.

Retinal disorders:

Diabetic retinopathy

Early detection of diabetic retinopathy through screening programs and subsequent referral for therapy are vital to preserve vision in individuals with diabetes. Screening for retinopathy is undertaken using conventional CFP and relies on the identification of hemorrhages, hard exudates, and cotton-wool spots. In a study by Roy et al, they found out that hard exudates, cotton wool spotsand retinal hemorrhages were better seen on MCI and in GR images as compared to CFP, BR, and infrared imaging, respectively (Figs 3 and 4). The sensitivity and specificity of MCI to detect of these lesions were more than 90% with CFP as the comparator.[3] On multicolor composite image, microaneurysms are seen as dark red dots, hard

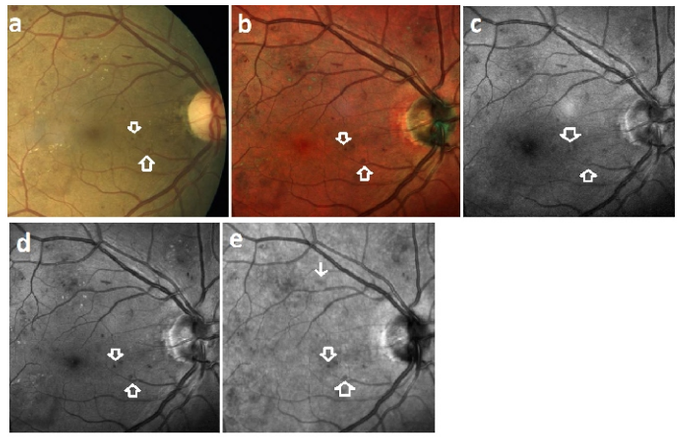


Fig.3: MCI (b) shows retinal hemorrhages which appear darker compared to CFP (a). Retinal hemorrhages are better defined in GR (d) image as compared to BR (c) and IR (e) images.

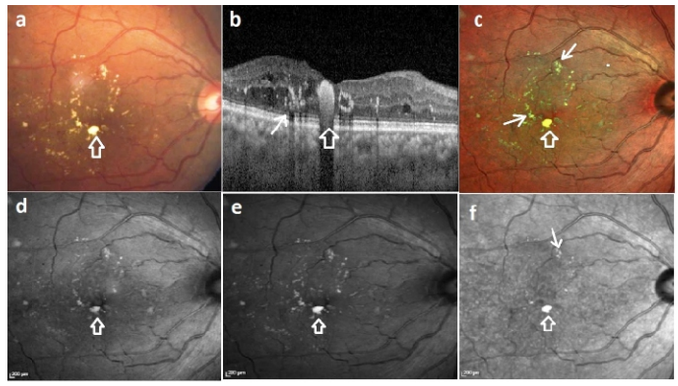


Fig.4: Color fundus photograph (CFP) (a) shows yellowish hard exudates (HEX) at macula. SDOCT (b) shows HEX plaque as a hyperreflective zone with a posterior shadow. Multicolor imaging (MCI) (c) shows multiple greenish yellow HEX. HEX are better visualized in green reflectance (GR) (e) image as compared to blue reflectance (BR) (d) and infrared reflectance (IR) (f) images.

exudates as greenish yellow and cotton wool spots as greenish white. MCI is a useful technique to detect different subtypes of retinal microaneurysms in diabetic retinopathy.[4] In proliferative diabetic retinopathy, MCI can easily detect subtle neovascularization which may be missed on CFP. Fibrovascular nature of neovascularization is seen better on MCI than CFP (Fig 5). Green reflectance highlights both vascular and fibrous components of neovascularization.[5] In a study by Saurabh et al, MCI was superior to CFP in detecting cysts of diabetic macular edema at fovea.[6] It can also be used to monitor progress of diabetic retinopathy.[7]

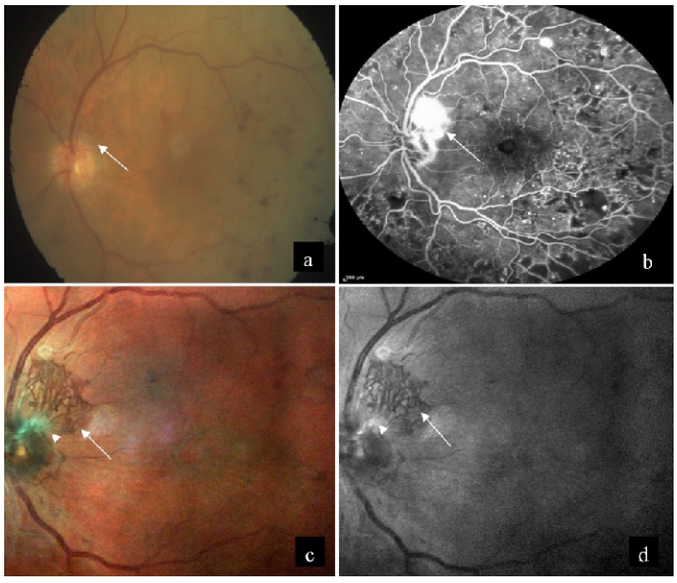


Fig.5: Neovascularization on the superior disc margin was visible in the color fundus photography of the left eye (a). Fundus fluorescein angiography showed leakage from NVD with multiple leaking microaneurysms (b). However when MCI is used, the fibrovascular nature of the neovascularization was seen in much greater detail this "tuft" is much more complex and extensive when seen with MCI than seen with color fundus photography (c). Green reflectance also provides clear evidence of both the vascular and fibrous components of this NVD (d).

Retinal vascular occlusion

Retinal vascular occlusions can visual loss when associated with macular edema and neovascularization. MCI can detect subtle macular edema and retinal thickening. Macular edema is seen as a greenish tinge[8] (Fig 6). Area of ischemia and hemorrhages are well visualized. MCI allows documentation of disease progression and the response to treatment. In a study by Schouten et al, they have described the role of MCI, particularly the BR component in diagnosis of concurrent macular telangiectasia type 2 and branch retinal vein occlusion.[9] It helps in guiding treatment decisions and prognosticating treatment outcomes.

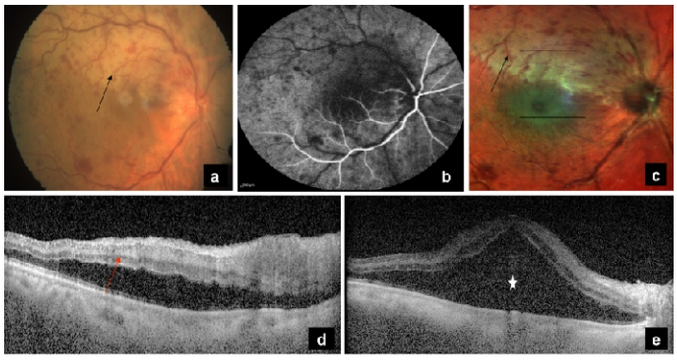


Fig.6: CFP (a) showing multiple haemorrhages, disc edema and pale ischemic area superiorly (black arrow). Fundus fluorescein angiography (b) showing delayed arterial dye filling and prolonged arteriovenous transit time. MCI (c) highlighting the area of ischemia (black arrow) and showing greenish tinge in the area of macular edema (white arrow). OCT showing hyperreflectivity of inner retinal layers (d) (red arrow) and increase in retinal thickness (e) in the corresponding areas.

Age related macular degeneration

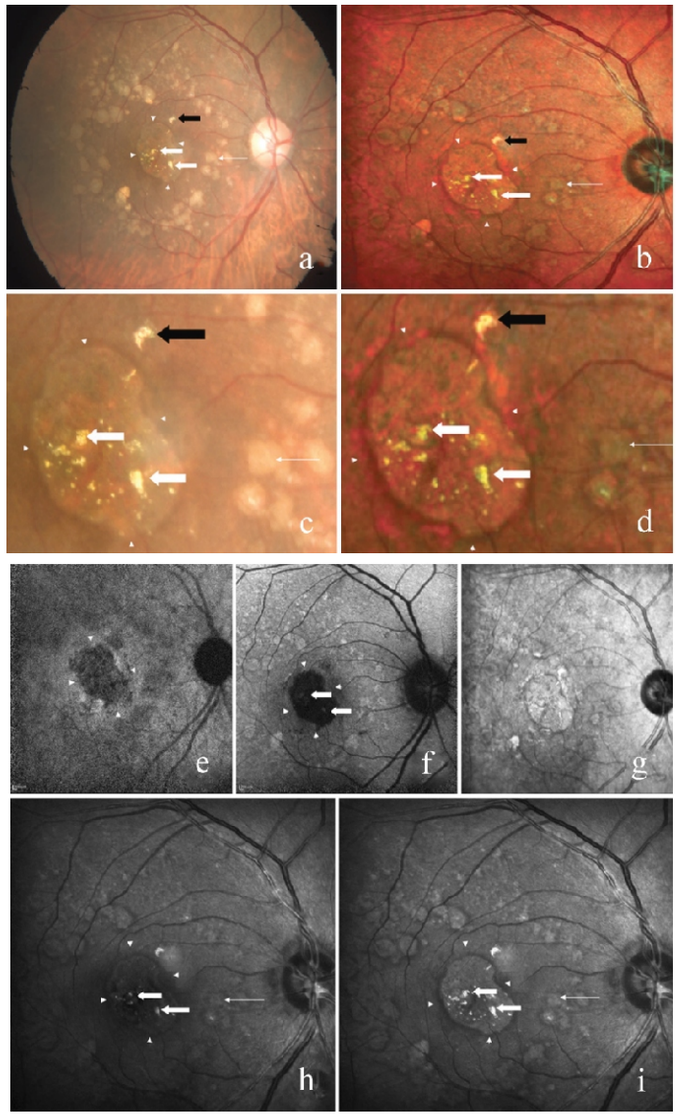


Fig.7: CFP (a) of the right eye showing refractile drusen as yellow, glistening deposits in the macular and perimacular area (thick white arrow), and soft drusen circumferentially (thin white arrow) with an area of central retinal pigment epithelium atrophy (arrowhead). Hyper-reflectant structure seen superonasal to the fovea is artifactual (black arrow). Multicolor

composite image (b) highlights the refractile drusen as small, yellow, glistening deposits (thick white arrow), whereas soft drusen appears as green with an orange border (thin white arrow). Magnified conventional fundus photography (c) and multicolor composite image (d) view of refractile drusen (thick white arrow), soft drusen (thin white arrow), and artifact (black arrow).Near-infrared autofluorescence (e) and blue autofluorescence (f) highlight the area of retinal pigment epithelium atrophy. IR(g) fails to highlight the drusen. BR (h) shows refractile drusen as hyper-reflectant lesions (thick white arrow). Soft drusen appears mildly hyper-reflectant with surrounding dark borders (thin white arrow). Arrowhead shows the area of retinal pigment epithelium atrophy. GR (i) distinctly highlights the refractile drusen as hyper-reflectant material (thick white arrow), whereas soft drusen as mildly hyper-reflectant with dark borders (thin white arrow) surrounding it with distinct retinal pigment epithelium atrophic area (arrowheads).

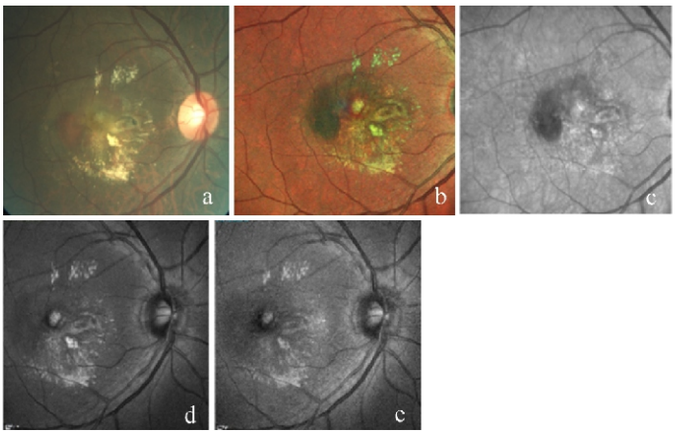


Fig.8: CFP (a) of right eye showing greyish white membrane with yellow hard exudates at macula suggestive of CNV. Multicolor composite image (b) highlights area of CNV as greenish tinge with greenish yellow hard exudates with corresponding IR (c), GR (d) and BR (e) images.

Age-related macular degeneration (AMD) is a common macular disorder affecting elderly people. It is characterized by the presence of drusen in the macula that can be complicated by choroidal neovascularization (CNV) or geographic atrophy (GA). MCI is an excellent tool for the measurement of area and width of GA and for the detection of foveal sparing.[10] GA is best visualized on IR images. Refractile drusen are seen in association with AMD and are thought to be an important prognostic biomarker for developing central GA.[11] In multicolor composite image, soft drusen appears green with orange borders, while reticular drusen stands out as glistening, light yellow structures (Figs 7a-7d). Refractile drusen are distinctly hyper‑reflectant in GR and BR, whereas soft drusen appear mildly hyper‑reflectant (Figs 7e-7i). Reticular pseudodrusen may represent a high-risk phenotypic feature for progression, can also be imaged more distinctly on multicolor image.[12] Reticular pseudodrusen can be distinguished from drusen on the basis of its appearance and identification on the GR images. Soft sub-RPE drusen are highlighted well on IR images.[13]MCI allows earlier detection of CNV and helps in monitoring during anti-VEGF therapy. CNV appears greenish on MCI (Fig 8).

Polypoidal choroidal vasculopathy

Accurate diagnosis of Polypoidal choroidal vasculopathy(PCV) is important because the clinical course of PCV differs from that of typical neovascular AMD and treatment options are different. Orange subretinal nodules or massive submacular haemorrhage is highly suggestive of PCV. Multicolor imaging is able to detect polypoidal lesions in most patients with PCV.[14] Both the polyps and branching vascular network (BVN) are best visualized using the multicolor composite or infrared reflectance image as the lesions are located beneath the retinal pigment epithelium (RPE).[15]Polyps appear as dark green oval lesions on multicolour composite image and BVN appear as mottled gray regions on IR.Associated pigment epithelial detachment (PED) and subretinal hemorrhage are also well visualized.

Macular hole

Macular hole is well visualized on MCI. Surrounding cuff of subretinal fluid is seen as greenish tinge (Fig 9). Associated RPE

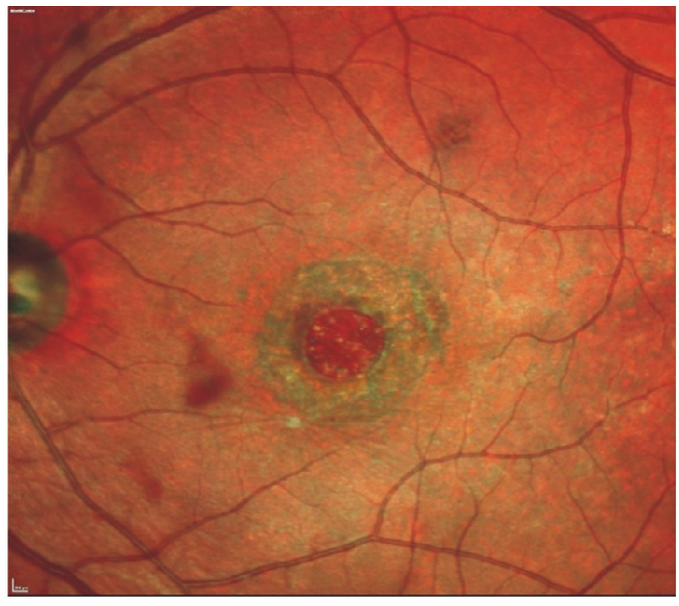


Fig.9: Multicolour composite image of left eye showing full thickness macular hole with surrounding cuff of subretinal fluid that is seen as greenish tinge.

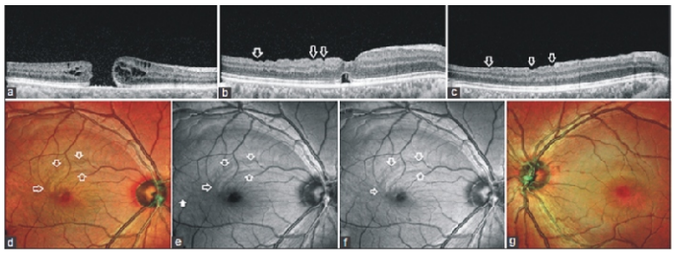


Fig.10: Preoperative line scan SDOCT image (a) through fovea shows full thickness macular hole. Postoperative SDOCT line scan (b) across fovea shows dissociated optic nerve fiber layer (DONFL) as irregular depressions in the retinal contour (white blank arrows) due to focal thinning of ganglion cell layer and hyporeflectivity of retinal nerve fiber layer. SDOCT line scan (c) superior to fovea also shows depressions in the retinal contour (white blank arrows) suggestive of DONFL. Multicolor image of right eye (d) shows closed macular hole with darker arcuate zone superior and temporal to fovea (white blank arrows) suggestive of DONFL defects. BR (e) shows arcuate zone of DONFL (white blank arrows). Discrete dissociated optic nerve fiber layer lesion is noted temporal to fovea (white solid arrow) which is not visible on multicolor image. GR (f) shows arcuate zone of DONFL (white blank arrows). Discrete DONFL lesion noted temporal to fovea in (e) is not visible in this GR image. Multicolor image of normal left eye (g) shows normal greenish hue which is uniform across around fovea.

atrophy can be seen. Delineation of margin and detection of subretinal fluid (SRF) is better than CFP. MCI is capable of detecting inner retinal alterations in patients with a history of internal limiting membrane (ILM) peeling, and may be clinically useful for monitoring anatomical changes associated with ILM peeling.[16] Dissociated optic nerve fiber layer (DONFL) is an after effect of ILM peeling for macular hole repair that is usually diagnosed on Spectral domain optical coherence tomography (SD-OCT).[17] DONFL is seen as darker arcuate zones on multicolour composite image. BR image delineates the DONFL better than GR (Fig 10)

Macular telangiectasia type 2

Macular telangiectasia type 2 (MacTel) is a bilateral retinal disease that seems to be limited to the juxtafoveal region of macula and usually diagnosed in the fifth or sixth decade of life. Key diagnostic features of MacTel include graying of parafoveal area, superficial retinal crystals, right angled venules and pigment hyperplasia. BR imaging has emerged as a promising rapid, non-invasive diagnostic tool for MacTel that shows a characteristic oval parafoveal area of increased reflectance.[18,19] Superficial retinal crystals and right angles venules are also better visualized on MCI (Fig11).

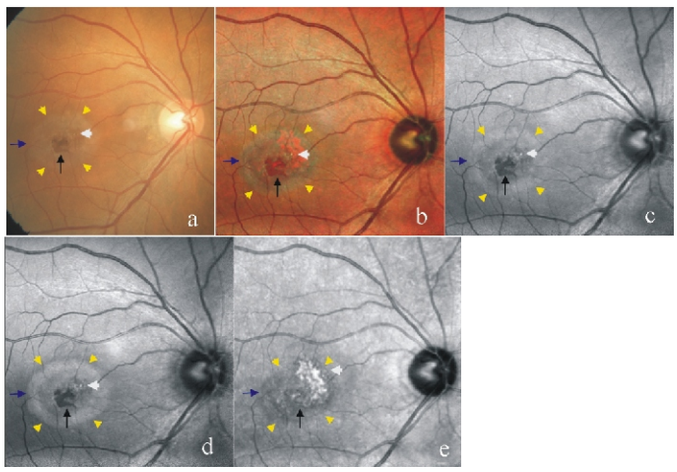


Fig.11: CFP) of right eye (a) shows yellow crystals (white arrow), dark pigments (black arrow), right angled venule (blue arrow) and graying (yellow arrow heads) at macular area.MCI highlights yellow crystals (white arrow), dark pigments (black arrow) as orange color, right angled venule (blue arrow) and larger area of graying (yellow arrow heads). Area of graying (yellow arrow heads) is better visualized in BR (d) as compared to GR (c) and IR (e). Right angled venule (blue arrow), pigments (black arrow) and crystals (white arrow) are better seen in GR (c) and BR (d) as compared to IR (e).

Central serous chorioretinopathy

Central serous chorioretinopathy (CSC) is characterized by serous retinal detachment and RPE detachment involving macula. He et al. have reported that MCI was superior in delineating area of SRF compared to CFP.[20] Venkatesh et al. have compared MCI with CFP and fundus fluorescein angiography (FFA) and reported that MCI was similar to FFA in identifying focal leaks.[21] They also reported that MCI was able to detect PED and RPEatrophy. Saurabh et al compared the ability of MCI to detect the lesions of CSC against conventional CFP.[22] SRF was seen as greenish hue over the retina on MCI. Keeping SDOCT as gold standard, MCI was able to pick SRF in more number of eyes than CFP. SRF was best seen on GR. (Fig 12) MCI detects PED as an elevation of retinal contour and renders it greenish hue which is darker than SRF because PED is present at deeper level than SRF. Smaller well-circumscribed size and surrounding pink ring differentiate PED from SRF on MCI. (Fig 13) PED was best detected on IR image due to its deeper location. RPE atrophy was seen better on IR image than BR or GR images. Focal leaks appear as orange spots on MCI. Pachyvessels are seen as bright orange vascular network.

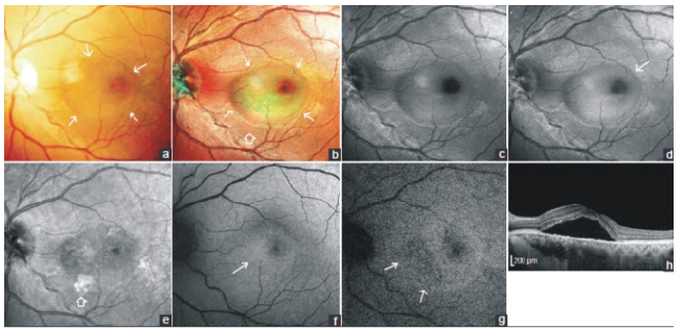


Fig 12: CFP of left eye (a) shows subretinal fluid (SRF) marked with white arrows as a bleb at the center of macula.SRF is seen more distinctly in MCI (b). Margins of SRF (white arrow) are better delineated than CFP. Not the orange-pink coloration of retinal pigment epithelium (RPE) atrophy near the inferior margin of SRF (blank arrow) BR (C) shows the SRF as bleb. GR (d) shows SRF better than BR. The temporal margin (white arrow) is better demarcated than BR image. SRF is seen less distinctly than BR and GR images and is detected as hyperreflectivity at the site of bleb of SRF (e). Note the hyperreflective RPE atrophy (blank arrow) coinciding with same seen on MCI in (b). On blue autofluorescence (BAF) (f) the foveal zone of normal hypoautofluorescence is reduced and hyperautofluorescence is seen inferonasal to fovea (white arrow) which coincides with zone of greenish hue seen on MCI. The normal hyperautofluorescence at fovea (g) due to maximum melanin content in infrared autofluorescence (IRAF) image is replaced by hypoautofluorescence due to the presence of SRF. Note the inferonasal hypoautofluorescence (white arrow) denoting RPE atrophy which is not seen on BAF and coincides with orange-pink discoloration seen on MCI in (b). SDOCT scan (h) through fovea shows the SRF.

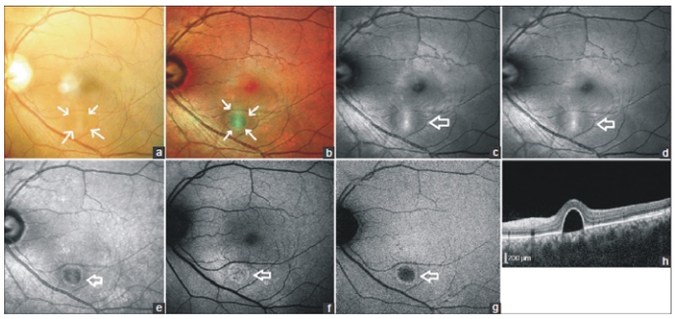


Fig.13: CFP of left eye (a) shows retinal pigment epithelial detachment (PED) as round elevation (white arrow). MCI (b) shows PED as a greenish lesion with pink ring surrounding it (white arrow). PED is seen more distinctly on MCI than CFP. BR (c) shows PED as circumscribed zone of central hyperreflectivity at peak flanked by zone of hyperreflectivity (blank arrow). GR (d) shows similar pattern as BR (blank arrow). PED appears as hyporeflective lesion (blank arrow) surrounded by rim of hyperreflectivity on IR image (e). BAF (f) shows stippled hyperautofluorescence (blank arrow) at the site of PED. PED appears as central hyperreflectivity (blank arrow) surrounded by hyperautofluorescent rim on IRAF image (g). SDOCT line scan (h) shows serous PED.

Epiretinal membrane

Muftuoglu et al. have studied epiretinal membrane (ERM) with MCI and have concluded that it was better visualized on MCI as

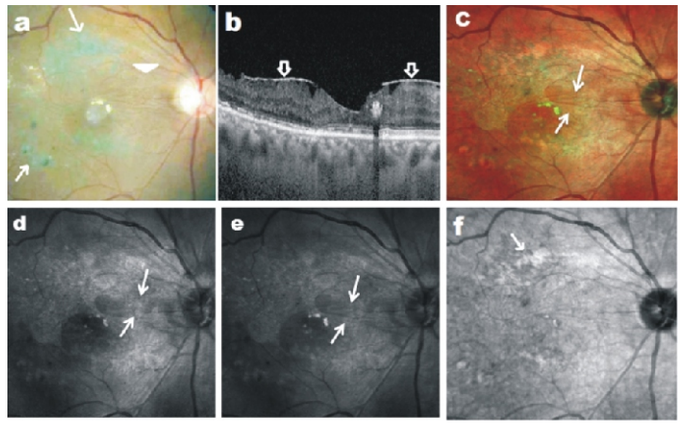


Fig.14: CFP (a) shows epiretinal membrane (ERM) as fine striataions (arrowhead) and laser marks (arrow) in a patient with diabetic retinopathy. SD-OCT (b) shows ERM. MCI (c) highlights ERM as bright greenish lines. Hyper-reflective folds of ERM are well visualized in BR (d) and GR (e) images as compared to IR image (f).

compared to CFP.[23] GR image is best suited to image hyper-reflective surface folds of ERM (Fig 14). Multicolor image highlights ERM as greenish retinal folds. The ERM area is more clearly detectable and widely demarcated inMCI than in CFP images.[24]

Retinal dystrophies

Utility of MCI has been described in various retinal dystrophies.

Best vitelliform dystrophy

Saurabh et al. have reported MCI characteristics of Best's Vitelliform Macular Dystrophy (BVMD). [25] Composite multicolour image revealed larger area of RPE atrophy in vitelli eruptive stage of the disease compared to CFP. Retinal elevation in the pseudohypopyon stage was better delineated on composite multicolour image and BR image. Subretinal lipofuscin was best seen in GR image (Fig 15).

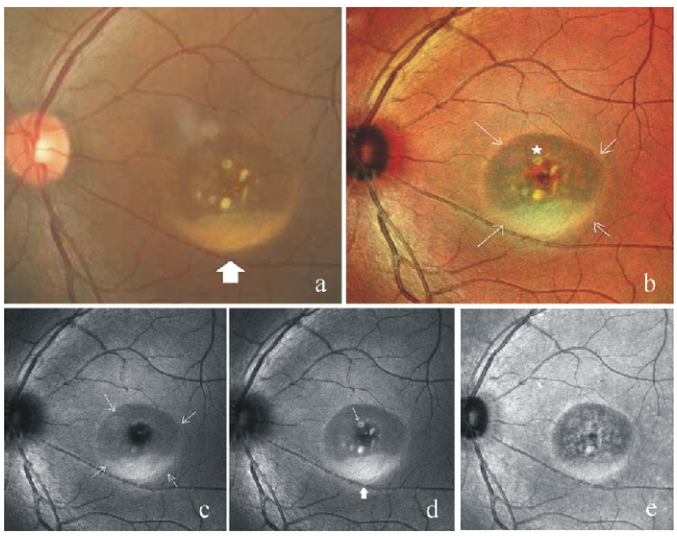


Fig.15: CFP of left eye (a) shows area of retinal elevation at the center of the macula with yellowish subretinal deposit at the dependent part (solid white arrow) suggestive of lipofuscin. Composite multicolor image (b) gives blue-green hue to the elevated retina and delineates the margin of elevation sharply (white arrows). Subretinal yellowish lipofuscin deposits (solid star) are brighter than composite CFP.BR image (c) shows well-circumscribed ring of retinal elevation (white arrow) with subretinal lipofuscin. Subretinal lipofuscin layered inferiorly (solid arrow) and scattered through the retinal elevation (white arrow) appeared brighter on GR image (d). IR image (e) shows RPE mottling within the area of retinal elevation.

Multifocal Best disease is a rare disease characterized by multiple sharply demarcated yellowish lesions in the posterior pole and beyond arcades. This is considered an atypical variant of BVMD. Shah et al have reported MCI characteristics in these patients.[26]

Multicolor image showed greenish hue over the macula suggestive of retinal thickening and multiple orange spots corresponding to yellow lesions. IR image showed multiple white hyper-reflective spots suggestive of alterations at RPE level, while GR and BR images failed to highlight the lesions clearly.

Stargadt disease

Stargardt macular dystrophy is an autosomal recessive inherited retinal disease. Yellow or white fish-shaped flecks and photoreceptors, RPE and choriocapillaris atrophy are the pathognomonic clinical features of this disease.[27] In both CFP and multicolor image, the white flecks of the fundus can be seen. However, the demarcation of the central area of atrophy is more clearly seen on MCI (Fig 16).[1]

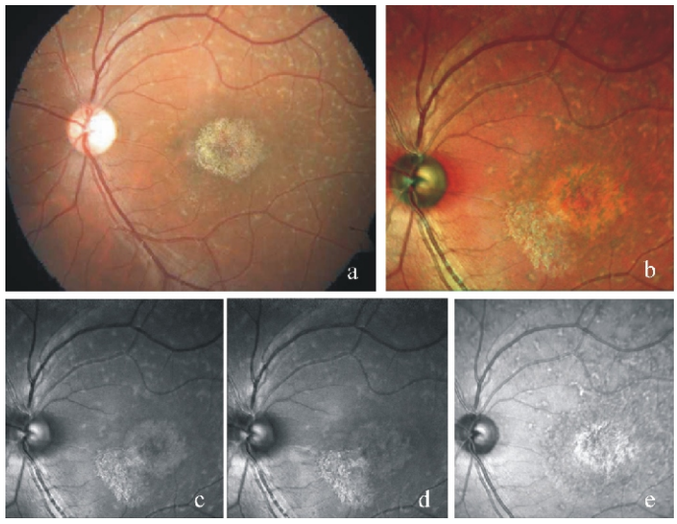


Fig.16: CFP of left eye (a) in Stargadt disease showing yellowish white retinal flecks and RPE atrophy at macula. Multicolor image (b) highlights the area of RPE atrophy and retinal flecks. Lesions are better visualized on IR (e) as compared to GR (c) and BR (d).

Pattern dystrophies

Pattern dystrophies includebutterfly-shaped dystrophy, reticular dystrophy, multifocal pattern dystrophy simulating fundus flavimaculatus, adult vitelliform dystrophy, and fundus pulverulentus.Roy et al. have reported MCI in a case of fundus pulverulentus.[28] It is a rare type of patterndystrophy that is characterized by coarse pigment mottling in the macula.

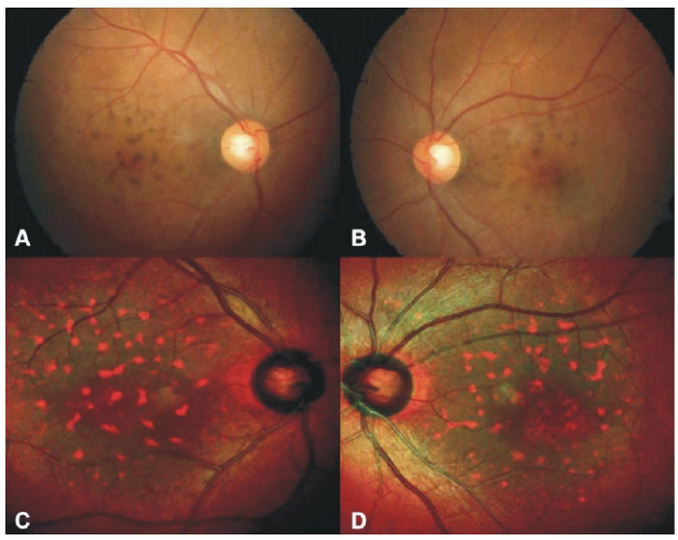


Fig.17: CFP (A and B) of the right eye and left eye showing coarse pigments on the posterior pole. Multicolor image (C and D) showing greenish hue and orange hyper-reflective spots over the macula.

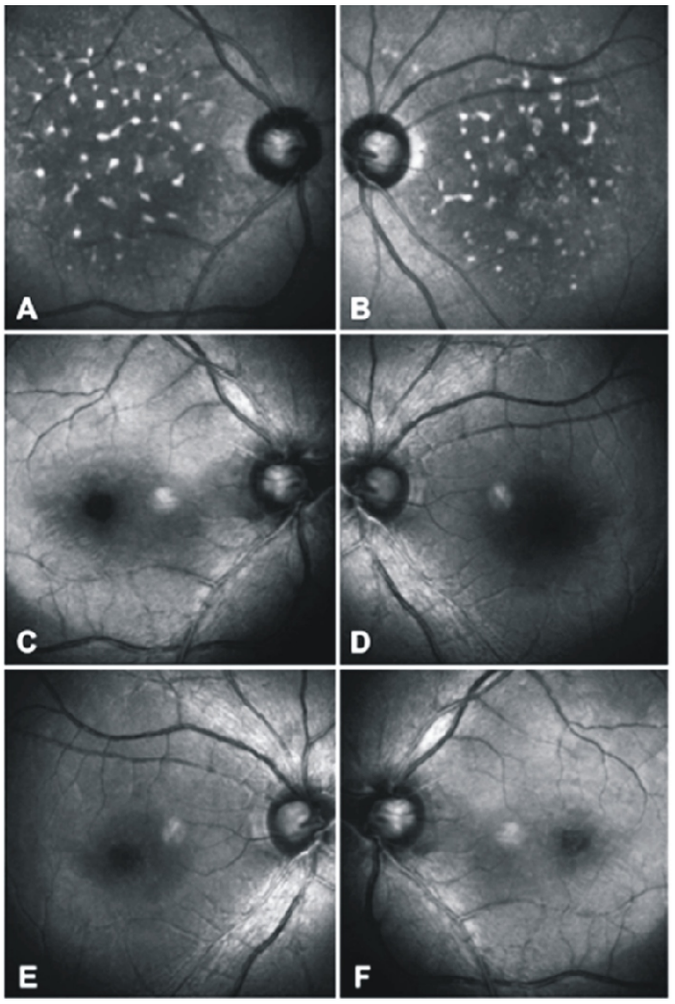


Fig.18: IR image (A and B) of the right eye and the left eye showing multiple white hyperreflectant spots over the posterior pole corresponding to the pigments suggestive of alterations at RPE level, whereas BR (C and D) and GR (E and F) images show no abnormality.

Composite multicolor image showed greenish hue over the macula suggestive of retinal thickening and orange hyperreflective spots suggestive of alterations at the level of RPE (Fig 17). IR imaging localized the lesions to the RPE level (Fig 18).

Dominant cystoid macular dystrophy

Dominant cystoid macular dystrophy is a rare inherited retinal disorder that primarily affects the macula. It is a unique retinal dystrophy because the appearance of cystic spaces in the macula heralds its onset with the rest of the retina being essentially normal. MCI helps in picking up the cystoid abnormality much efficiently as compared to the conventional CFP (Fig 19).[29]

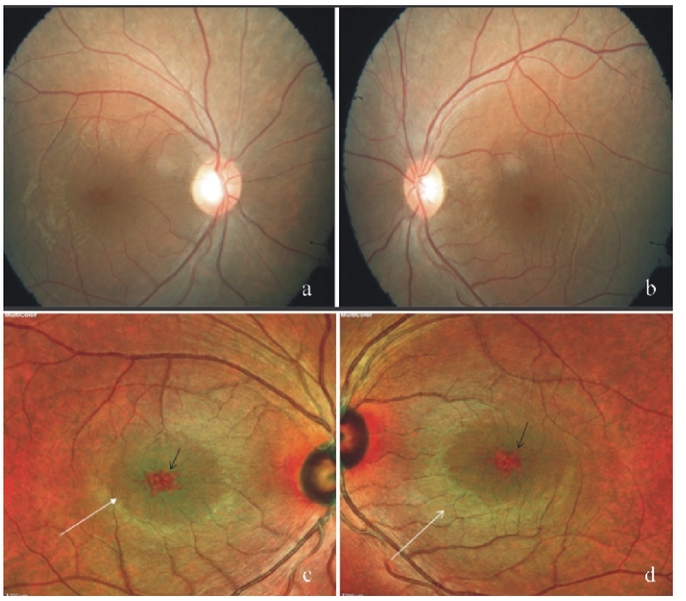


Fig.19: CFP of right (a) and left (b) eye showing cystic spaces at macula. Multicolor image of right (c) and left (d) eye showing greenish hue at posterior pole suggestive of retina thickening (white arrows) with central orange hyper reflectance at fovea (black arrows) suggestive of a cyst.

Retinitis pigmentosa

Retinitis pigmentosa (RP) is an inherited retinal disease caused by the degeneration of photoreceptors and RPE cells. MCI is capable of detecting macular changes and complications in patients with RP.In a study by Liu et al,MCI clearly defined the borders of the macular-sparing area corresponding to the relatively intact outer retinal structures on OCT images, particularly the status of the ellipsoid zone and external limiting membrane.[30]

Choroideremia

Choroideremia is a rare X-linked disorder thatcauses progressive degeneration of retina, RPE and choroid. Goel et al have reported MCI signatures in choroideremia.[31] MCI highlighted the residual RPE tissue at macula and the surrounding chorioretinal atrophy much better than CFP (Fig 20). The residual RPE tissue was well visualized in IR as compared to GR and BR (Fig 21). MCI can be used to evaluate viable retina and document disease progression in patients with choroideremia.

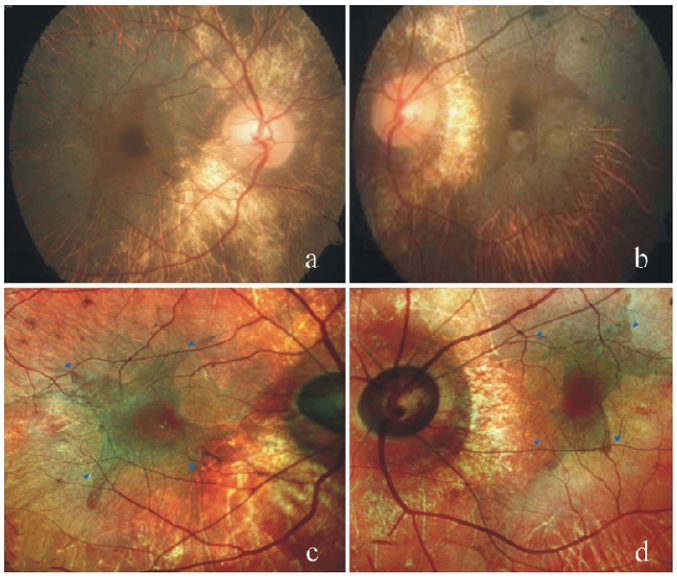


Fig.20: CFP (a and b) showing bilateral chorioretinal atrophy (CRA) and areas of retinal RPE disruption with sparing of the central macula. Multicolor image (c and d) highlighting the residual RPE tissue at macula (blue arrow heads) and surrounding CRA much better than CFP.

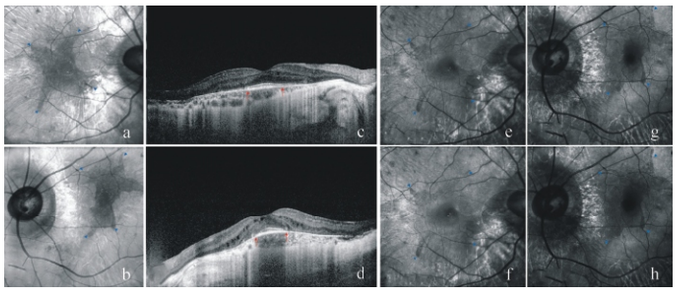


Fig.21: Area of residual RPE tissue (blue arrow heads) is well visualized in IR images (a and b). SDOCT showing retinal thinning and choriocapillary atrophy sparing central macula (red arrows) (c and d). GR images (e and g) and BR images (f and h) of both eyes showing area of residual RPE tissue (blue arrow heads) faintly as compared to IR.

Hydroxychloroquine toxicity

Hydroxychloroquine (HCQ) retinal toxicity is known to cause irreversible vision loss and it is a progressive condition which progresses even after stopping the drug. Therefore, early detectionof toxicity is of utmost importance to prevent visual morbidity. Saurabh et al have described the utility of MCI in obviating the retinal changes in HCQ retinal toxicity better than conventional CFP.[32] Composite multicolor image showed

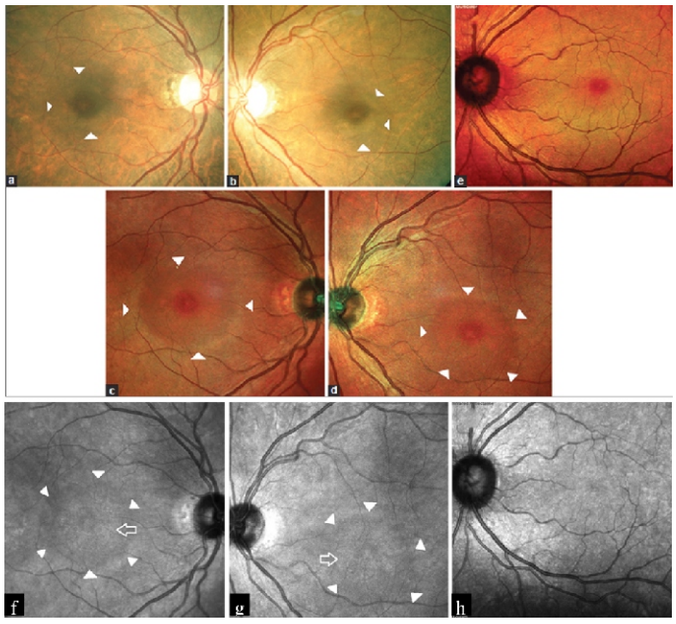


Fig.22: CFP of the right and left eye (a and b) show arcuate zone of hypopigmentation (white arrow heads) superior, temporal, and inferior to fovea. Multicolor images (c and d) of the right and left eye show ring of darker hue around fovea (white arrow heads) corresponding to the arcuate hypopigmentation seen on CFP and extending beyond. Multicolor image of the left eye (e) of a normal individual shows deep pink center of the fovea surrounded by a greenish hue corresponding which is missing in the eye with hydroxychloroquine retinal toxicity. IR images of the right and left eye (f and g) shows speckled hyperreflectance at the center of macula (blank white arrow). This area is surrounded by an arcuate zone of hyporeflectance (white arrow heads). IR image of the left eye (h) of a normal individual for comparison shows nondescript pattern of hypo and hyperreflectance at the macula; devoid of any arcuate zone as seen in hydroxychloroquine retinal toxicity eyes.

acircumscribed perifoveal arcuate area of darker hue sparing the fovea and IR image showed speckled hyperreflectance at the center of macula with an arcuate zone of hyporeflectance surrounding it (Fig 22). Larger area of retinal involvement could be appreciated in multicolor image as compared to CFP.

Torpedo maculopathy

Torpedo maculopathy is a rare, congenital anomaly of RPE characterized by the appearance of a "torpedo-shaped" lesion located temporal to the fovea. Venkatesh et al have reported multicolour imaging findings in Torpedo maculopathy. [33] MCI can be a useful tool in identifying the level of retinal and choroidal layer involvement in these patients.

Acute retinal pigment epitheliitis

Acute retinal pigment epitheliitis (ARPE) is a rare idiopathic inflammatory retinal disorder that causes dramatic vision loss, mainly in young adults. Clinical manifestations include central scotoma and pigment stippling in the macula surrounded by hypopigmented halos. Roy et al have reported MCI signature of ARPE. [34]ARPE lesion had a reddish color with inferior orange extension in multicolour composite image. The difference in color within same lesion could be attributed to differential topographical concentration and reflectance properties of the inflammatory debris of the ARPE lesion. The lesion could not be visualized in GR and BR imaging and was seen only in IR image, thus supporting the outer retinal origin of these lesions.

AMN

Acute macular neuroretinopathy (AMN) usually presentsas brown□red lesion near fovea and leads to variable disruption of photoreceptors seen as disruption of external limiting membrane and ellipsoid zone. Saurabh et al have reported MCI signatures in AMN.[35] Multicolor image showed a zone of pale-pink discoloration with loss of perifoveal greenish hue. Infrared

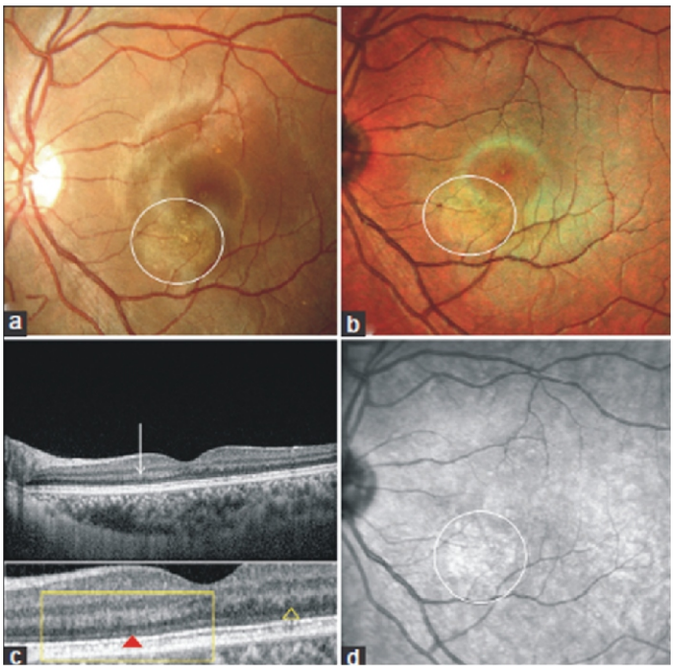


Fig.23: CFP of left eye (a) showing an area of yellowish brown depigmentation inferonasal to fovea (white circle); multicolor composite image (b) showing loss of perifoveal greenish hue over the area of acute macular neuroretinopathy lesion along with pinkish discoloration (white circle); line scan SDOCT image (c) through the lesion showing thickening of outer plexiform layer (OPL) on the nasal side of fovea compared with the temporal side (white arrow). The difference in the OPL thickness can be clearly made out in blown up image in the lower panel (yellow rectangle) comparing with the temporal OPL (yellow triangle). Note the subtle zone of discontinuity of external limiting membrane beneath the thickened OPL (red triangle) and thinning of outer nuclear layer; IR image (d) showing hyperreflectance corresponding to the AMN lesion (white circle).

---

channel aimed at picking the outer retinal lesions showed zone of hyperreflectance (Fig 23). The pseudocolor encoding for this channel is red which explains the pinkish discoloration of lesion amidst the greenish perifoveal hue on multicolor image.

Angioid streaks

Angioid streaks are irregular crack-like dehiscences in Bruch's membrane that are often associated with atrophic degeneration of the overlying RPE.[36] These can occur in isolation or in

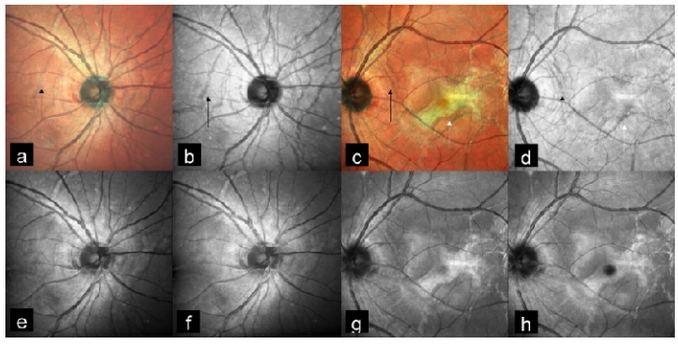


Fig.24: Multicolor image of both eyes highlights angioid streaks as dark orange color (a and c) (black arrows) and better visualized in IR (b and d) (black arrows) as compared to GR (e and g) and BR (f and h). Multicolor image of left eye showing CNV as greenish color (c) (white arrow) with corresponding IR (d), GR (g) and BR (h) images.

---

association with systemic diseases like Pseudoxanthoma elasticum, Paget disease, sickle cell trait disease and thalassemias. MCI highlights angioid streaks as dark orange color. These are better visualized in IRas compared to GR and BR. Associated complications like CNV are better seen on MCI (Fig 24).

MCI has some disadvantages when compared to conventionalCFP. MCI may require a slightly longerperiod of fixation to take the three separate images and artifacts must be taken into account. Clinician should be familiar with variations in the color produced in MCI. In conclusion, MCI is a useful modality to assess posterior segment lesions and may be used to replace CFP in some diseases.

References:

1. Tan AC, Fleckenstein M, Schmitz-Valckenberg S, et al. Clinical application of multicolor imaging technology. Ophthalmologica 2016;236:818.

2. Sergott RC. Retinal segmentation using multicolor laser imaging. J Neuroophthalmol. 2014;34 Suppl:S24-S28.

3. Roy R, Saurabh K, Thomas NR, et al. Validation of Multicolor Imaging of Diabetic Retinopathy Lesions Vis a Vis Conventional Color Fundus Photographs. Ophthalmic Surg Lasers Imaging Retina. 2019;50(1):8-15.

4. Arrigo A, Teussink M, Aragona E, et al. MultiColor imaging to detect different subtypes of retinal microaneurysms in diabetic retinopathy.2020. Eye.https://doi.org/10.1038/s41433-020-0811-6.

5. Goel S, Garg B, Shah AV, et al. Multicolor imaging in neovascularization of disc. Indian J Ophthalmol. 2019 Mar;67(3):408.

6. Saurabh K, Roy R, Goel S. Correlation of multicolor images and conventional color fundus photographs with foveal autofluorescence patterns in diabetic macular edema. Indian J Ophthalmol. 2020;68(1):141-144.

7. Ahmad MSZ, Carrim ZI. Multicolor Scanning Laser Imaging in Diabetic Retinopathy. Optom Vis Sci. 2017;94(11):1058-1061.

8. Goel S, Saurabh K, Roy R. Combined central retinal vein occlusion and branch retinal artery occlusion. Indian J Ophthalmol. 2018 Dec;66(12):1862-1863.

9. Schouten I, Goel S, Saurabh K, et al. Multicolour imaging signatures to diagnose concurrent macular telangiectasia type 2 and macular branch retinal vein occlusion. GMS Ophthalmol Cases. 2019;9:Doc36.

10. Ben Moussa N, Georges A, Capuano V, et al. MultiColor imaging in the evaluation of geographic atrophy due to age-related macular degeneration. Br J Ophthalmol. 2015;99(6):842-847.

11. Mishra S, Goel S, Roy SS, et al. Multimodal imaging characteristics of refractile drusen. Indian J Ophthalmol. 2019 Jan;67(1):128-129.

12. Badal J, Biarnés M, Monés J. Performance characteristics of multicolor versus blue light and infrared imaging in the identification of reticular pseudodrusen. Int Ophthalmol. 2018;38(1):199-206.

13. Venkatesh R, Pereira A, Thomas S, et al. Subretinal drusenoid deposits versus drusen on multicolor imaging. Indian J Ophthalmol 2020;68:1450-1.

14. Tan CS, Ting DS, Lim LW. Multicolor Fundus Imaging of Polypoidal Choroidal Vasculopathy. Ophthalmol Retina. 2019;3(5):400-409.

15. Tan ACS, Yanagi Y, Cheung GCM. COMPARISON OF MULTICOLOR IMAGING AND COLOR FUNDUS PHOTOGRAPHY IN THE DETECTION OF PATHOLOGICAL FINDINGS IN EYES WITH POLYPOIDAL CHOROIDAL VASCULOPATHY [published online ahead of print, 2019 Aug 27]. Retina. 2019;10.1097/IAE.0000000000002638. doi:10.1097/IAE.0000000000002638

16. Feng HL, Sharma S, Asrani S, et al. MULTICOLOR IMAGING OF INNER RETINAL ALTERATIONS AFTER INTERNAL LIMITING MEMBRANE PEELING. Retin Cases Brief Rep. 2017;11(3):198-202.

17. Saurabh K, Roy R, Mishra S, et al. Multicolor imaging features of dissociated optic nerve fiber layer after internal limiting membrane peeling. Indian J Ophthalmol 2018;66:1853-4.

18. Charbel Issa P, Berendschot TTJM, Staurenghi G, et al. Confocal blue reflectance imaging in type 2 idiopathic macular telangiectasia. Invest Ophthalmol Vis Sci 2008;49:11721177.

19. Charbel Issa P, Finger RP, Helb HM, et al. A new diagnostic approach in patients with type 2 macular telangiectasia: confocal reflectance imaging. Acta Ophthalmol 2008;86:464465.

20. He L, Chen C, Yi Z, et al. Clinical application of multicolor imaging in central serous chorioretinopathy. Retina 2018. doi: 10.1097/IAE.0000000000002441.

21. Venkatesh R, Agarwal SK, Bavaharan B, et al. Multicolour imaging in central serous chorioretinopathy. Clin Exp Optom 2019. doi: 10.1111/cxo. 12965.

22. Saurabh K, Roy R, Goel S, et al. Validation of multicolor imaging signatures of central serous chorioretinopathy lesions vis-a-vis conventional color fundus photographs. Indian J Ophthalmol 2020;68:861-6.

23. Kilic Muftuoglu I, Bartsch DU, Barteselli G, et al. Visualization of macular pucker by multicolour scanning laser imaging. Retina 2018;38:352–8.

24. Song JH, Moon KY, Jang S, et al. Comparison of MultiColor fundus imaging and colour fundus photography in the

evaluation of epiretinal membrane. Acta Ophthalmol. 2019;97(4):e533-e539.

25. Saurabh K, Roy R, Thomas NR. Multicolor imaging characteristics of Best's vitelliform macular dystrophy. Middle East Afr J Ophthalmol 2019;26:178-80.

26. Shah D, Saurabh K, Roy R. Multimodal imaging in Multifocal Best disease. Indian J Ophthalmol 2018;66:1313-5.

27. Cicinelli MV, Battista M, Starace V, et al. Monitoring and Management of the Patient with Stargardt Disease. Clin Optom (Auckl). 2019; 11: 151165.

28. Roy R, Saurabh K, Shah D. Multimodal Imaging in a Case of Fundus Pulverulentus. Retina. 2018;38(7):e55-e58.

29. Roy R, Saurabh K, Bhattacharyya S, et al. Multimodal imaging in dominant cystoid macular dystrophy. Indian J Ophthalmol. 2017;65:8656

30. Liu G, Du Q, Keyal K, et al. Morphologic characteristics and clinical significance of the macular-sparing area in patients with retinitis pigmentosa as revealed by multicolor imaging. Exp Ther Med. 2017 Dec; 14(6): 53875394.

31. Goel S, Saurabh K, Roy R. Multimodal imaging in a caseof choroideremia. Indian J Ophthalmol 2019;67:1470-1.

32. Saurabh K, Roy R, Thomas NR, et al. Multimodalimaging characteristics of hydroxychloroquine retinopathy. Indian J Ophthalmol2018;66:324-7.

33. Venkatesh R, Bavaharan B, Yadav NK. Multicolor imagingfindings in torpedo maculopathy. Indian J Ophthalmol 2019;67:295-7.

34. Roy R, Saurabh K, Thomas NR. Multicolor imaging in a case of acute retinal pigment epithelitis. Retin Cases Brief Rep. 2018;0:14.

35. Saurabh K, Roy R, Shah D, et al.Multimodal imaging characteristics of acute macular neuroretinopathy. IndianJ Ophthalmol 2019;67:403-4.

36. Georgalas I, Papaconstantinou D, Koutsandrea C, et al. Angioid streaks, clinical course, complications, and current therapeutic management. Ther Clin Risk Manag 2009;5:8189.

EYLEA™

(aflibercept solution for injection)

MORE TIME FOR WHAT MATTERS

In wAMD patients

WHAT YOU START TODAY MAKES A DIFFERENCE TOMORROW

RAPID MEANINGFUL IMPROVEMENTS FROM THE FIRST DOSE [2-6]

VA and ≥ 3-line gains from baseline at year 1 [2-6]

OUTCOMES MAINTAINED OVER TIME[7-9]

Maintained visual gains from baseline up to 4 years [7]

PROVEN RELIABILITY IN REAL LIFE SETTINGS[10-15]

Real life evidence comparable to clinical trials[10-15]

INNOVATIVE MODE OF ACTION[16-22]

1. Eylea (aflibercept solution for injection) Summery of product Characteristics India Bayer Zydus Pharma.2015 2. Data on file Bayer Zydus Pharma 3. Heier JS, Brown DM, Chong V, et al. Intravitreal aflibercept (VEGF Trap-Eye) in wet age-related macular degeneration. Ophthalmology. 2012;119(12):2537-2548. doi:10.1016/j.ophtha.2012.09.006. 4. Korobelnik J-F, Do DV, Schmidt-Erfurth U, et al. Intravitreal aflibercept for diabetic macular edema. Ophthalmology. 2014;121(11):2247-2254. doi:10.1016/j.ophtha.2014.05.006. 4. Schmidt-Erfurth U,Kaiser PK, Korobelnik J-F, et al. Intravitreal aflibercept injection for necrovascular age-related macular degeneration. Ophthalmology. 2014;121(1):193-201. doi:10.1016/j.ophtha.2013.08.011. 5. Brown DM, Schmidt-Erfurth U, Do DV, et al. Intravitreal aflibercept for diabetic macular edema: 100-week results from the VISTA and VIVID studies. Ophthalmology. 2015; 122(10):2044-2052. doi:10.1016/j.ophtha. 2015.06.017. 5. EYLEA (aflibercept solution for injection) Summary of Product Characteristics. Berlin, Germany: Bayer Pharma AG; 2015. 6. Data on file. Bayer HealthCare Pharmaceuticals Inc. 7. Clark WL. Longterm follow-up of intravitreal aflibercept injection (IAI) in patients with neovascular age-related macular degeneration. Poster presented at: American Academy of Ophthalmology Annual Meeting; November 15-19, 2013; New Orleans, LA. 8. Richard G, Monés J, Wolf S, et al. Scheduled versus pro renata dosing in the VIEW trials. Ophthalmology. 2015;122(12):2467-2503. doi:10.1016/j.ophtha.2015.08.014. 8 . Heier JS, Clark WL, Boyer DS et al Intravitreal aflibercept injection for macular edema due to central retinal vein occlusion two years result from theCOPERNICUS study Ophthalmology 201415B (1032-1036) 9. Ogura Y. Roider J. Korobelnik JF et al Intravitreal aflibercept or macular edema secondary to central retinal vein occlusion 18 months result of the phase 3 GALIED study AM J Ophtmatol 014159 (1032-1038) 10. Brown DM, Kaiser PK, Michels M, et al; ANCHOR Study Group. Ranibizumab versus verteporfin for neovascular agerelated macular degeneration. N Engl J Med 2006;355:1432–44. 12 Martin DF, Maguire MG, Ying GS, et al; CATT Research roup. Ranibizumab and bevacizumab for neovascular agerelated Group. Ranibizumab and bevacizumab for neovascular agerelated acular degeneration. N Engl J Med 2011;364: 697–908. 13. Gaudreault J, Fei D, Rusit J, et al. Preclinical pharmacokinetics of ranibizumab (rhuFabV2) after a single intravitreal administration. Invest Ophthalmol Vis Sci 2005;46:726–33 14. Mordenti J, Cuthbertson RA, Ferrara N, et al. Comparisons of the intraocular tissue distribution, pharmacokinetics, and safety of 125I-labeled full-length and Fab antibodies in rhesus monkeys following intravitreal administration. Toxicol Pathol 1999;27:536–44..15 Stewart MW. Predicted biologic activity of intravitreal bevacizumab. Retina 2007;27:1196–200. 16. Heier JS, Boyer D, Nguyen QD, et al. The 1-year results of CLEAR-IT2, a phase 2 study of vascular endothelial growth trap-eye dosed as-needed after 12-week fixed dosing. Ophthalmology 2011;118:1098e106. 15. Holash J, Davis S,Papadopoulos N,et al. VEGF-Trap: a VEGF blocker with potent antitumor effects. Proc Natl Acad Sci U S A 2002;99:11393–8. 17 Nguyen QD, Shah SM, Heiltz G, et al; CLEAR-AMD 1 Study Group. A phase 1 trial of intravenously administered vascular endothelial growth fact eovascularization due to age-related macular degeneration. Ophthalmology 2006;113:1522e32. ..19 Korobelnik JF, Do DV, Schmidt-Erfurth U, et al. Intravitreal aflibercept for diabetic macular edema. Ophthalmology 2014;121:2247–54.

Bayer Zydus Pharma

For further Information please contact:
Bayer Zydus Pharma, Bayer House, Central Avenue, Hiranandani Estate,
Thane, -400607 Maharashtra India. E-mail : medicalinfo.india@bayerzyduspharma.com

This advertisement is for informational purpose and by no means obligates or influences any medical practitioner to prescribe, recommend or purchase any products from Bayer or any of its affiliates. Please read full prescribing information before using the product.

ABBREVIATED PRESCRIBING INFORMATION

EYLEA™ SOLUTION FOR 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 neovascular (wet) age-related macular degeneration (wetAMD). Dosage Regimen wAMD The recommended dose for EYLEA™ is 2mg aflibercept , equivalent to 50µl. EYLEA™ treatment is initiated with one injection per month for three consecutive months, followed by one injection every 2 months. There is no requirement for monitoring between injections. Long term (after the first 12 months of treatment), it is recommended that patients continue to be treated with EYLEA™ every 2 months. 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 Know hypersensitivity to aflibercept or to any of the excipients, active or suspected ocular or periocular infection, active severe intraocular inflammation. Special warnings and special 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 hemorrhage, 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 edema, injection site hemorrhage, punctate keratitis, conjunctival hyperemia, ocular hyperemia. For full listing of undesirable effects, please refer to the full product insert. For further prescribing information, please contact Bayer Zydus Pharma Private Limited, Bayer House, Central Avenue, Hiranandani Estate, Thane, Maharashtra, India Pin-400607. Email: medicalinfo.india@bayerzyduspharma.com. Source: Based on CCDS / Version 10 / 19 Apr 2016; PI Rev 03 Nov 2016. Date of revision of API, 17 Nov 2016. For the use of healthcare professionals only.

LIN.MKT.06.2016.0840

For the use of healthcare professional only

INNOVATOR'S ISLE

Application of 3D Printing in Vitreo-retinal speciality

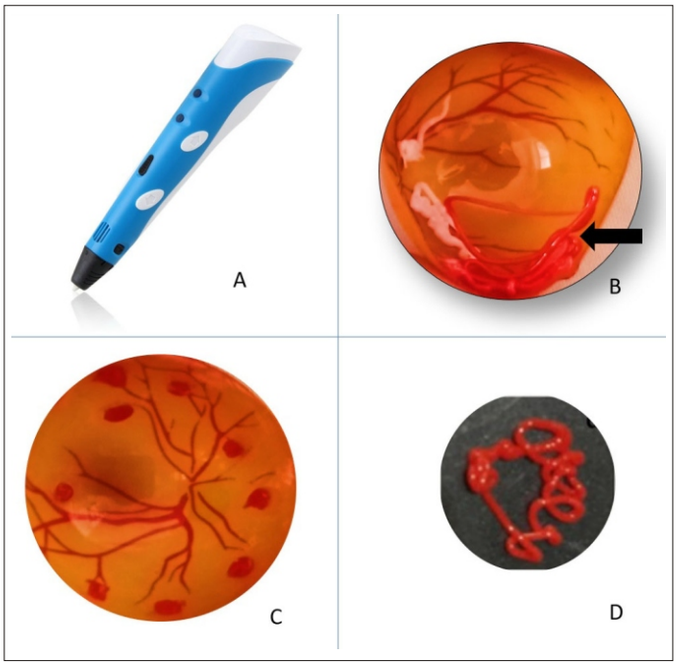
Dr. Ashish Ahuja
D.N.B., FICO (UK), F.A.I.C.O. (Retina) ,FAICO (Uvea).
Vitreo-Retina consultant
Sadhu Kamal Eye Hospital, Mumbai.

3D printing is a tremendously exciting new technology that is changing the face of modern manufacturing. Today, desktop 3D printers are cheaper and better than ever and are continuing to improve. 3D printing helps in designing customised instruments and devices, is environment friendly as it is an additive manufacturing process where we add layer by layer of the material to create the final prototype so wastage is minimal.
Application of 3D printing in the retina speciality is immense and keeps growing with time. In this article we will discuss some of the devices made with the help of 3D printing .

Dr. David Chow had designed 3D printed forceps handle for intra vitreal forceps which can be customised as per surgeons hand grip . [1] So this would enable development of forceps with various sizes to grip the instrument which would improve the dexterity. Also an individualized instrument catalogue could be generated for each surgeon in the future. Ten Hove used 3D printing to make a prototype transconjunctival vitrectomy trocar (21 and 22G) which they tested in pig eyes . [2]

3D doodling may be used which is a pen which extrudes a PLA or ABS filament at a high temperature which can be given any desired shape. Using the Aurolab retieye, different retinal diseases may be simulated for better training of doctors. We have made fundus models to simulate different retina pathologies like proliferative diabetic retinopathy, retinal holes, CRVO, DME with 3D printing which maybe used for patient education purpose and laser training of resident doctors and fellows.

A low cost (Rs 6000) ENT endoscopic light source maybe used



A : 3D doodling pen which extrudes ABS or PLA material to make the desired object.
B : Aurolabretieye modified with 3D printing to simulate proliferative diabetic retinopathy, lesion marked by black arrow signifies vitreous haemorrhage.
C : Aurolabretieye modified to simulate CRVO.
D: Sea – Fan neovascularisation.

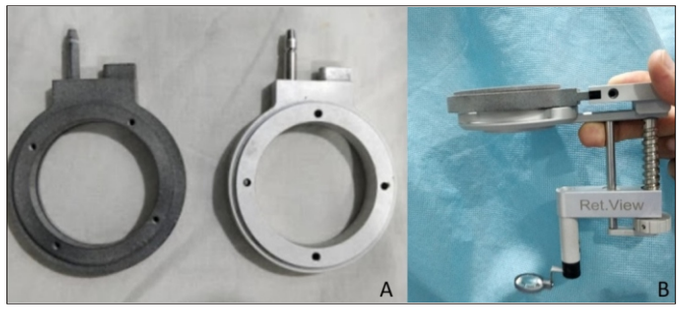
with a modified 3D printed connector to provide endoillumination during vitrectomy surgery. Light output is 10 watts, the device is battery operated and provides uniform illumination. This device may be used along with a chandelier light pipe and also for emergency purposes in case of power failure.

A : portable ENT endoscopic light source.
B : Ophthalmic 23G light pipe connected to ENT light source with a 3D printed connector.

3D printing maybe used to develop BIOM plates for Ret.View BIOM (by Appasamy). The material used is nylon and is a supportive plate to mount the BIOM onto the microscope .

3D printing can also be used for rapid prototyping of new surgical instrument designs ,we have designed a twin gauge trocar (23 -20 gauge , 23-25 , 23-25 gauge) , which could be used in cases with nucleus drop , IOFB removal .

ODocswas one of the first smartphone based fundus cameras developed via 3Dprinting and has been open sourced which is available for free download along with instructions, on Instructables[3]



A : 3D printed BIOM fixation plate
B : BIOM fixed on the 3D printed nylon plate which can be mounted on the microscope.

A company called Bionikodevelops 3D printed models of eyeballs for scleral buckling, internal limiting membrane peeling, suprachoroidal MIGS, and other ocular surgery training models.[4]

Furdova et al.used 3D printed models of the eye with intraocular tumours to plan stereotactic radiosurgery in 139 choroidal melanomas and 11 ciliary body melanomas. The authors concluded that 3D printed model of eye with tumour was helpful

3D printed Twin guage trocar with a cap (which has the 3.5 and 4 mm markings).

---

in planning the process to achieve the optimal scheme for irradiation which requires high accuracy of defining the targeted tumour mass and critical structures. [5]

Maloca et al. used 3D scans from an SSOCT device (DRI OCT Triton; Topcon, Tokyo, Japan) to 3D print choroidal vesselsand OCT Angiography scan from a Zeiss Cirrus HD-OCT Model 5000 with ANGIOPLEX (Review software 9.0.0.281; Carl Zeiss Meditec, Jena, Germany) to print retinal vessels at the macula in large sizes for study and demonstration purposes. [6,7]

A group from Japan had developed a 3D printed device to prevent fogging of the wide angle Resight lens used during vitrectomy surgery. The 3D printed device is mounted on the wide angle viewing system lens and is connected to the vacuum pump to remove the air between the lens and eye during the surgery to prevent fogging [8]

Shi et al. from Singapore developed a hybrid retina construct via three-dimensional (3D) bioprinting technology. 2 cell lines were used along with a 3D printed mesh to study the interaction of the RPE cells with the photoreceptor cells over 2 weeks period. This model would help develop effective strategies for the investigation of disease progression, drug metabolism and applications of tissue or organ transplantation. [9]

3D printing is quickly finding multiple uses in various aspects of ophthalmology and this is just the tip of the iceberg of many more potential uses of this technology.[10]

Conclusion

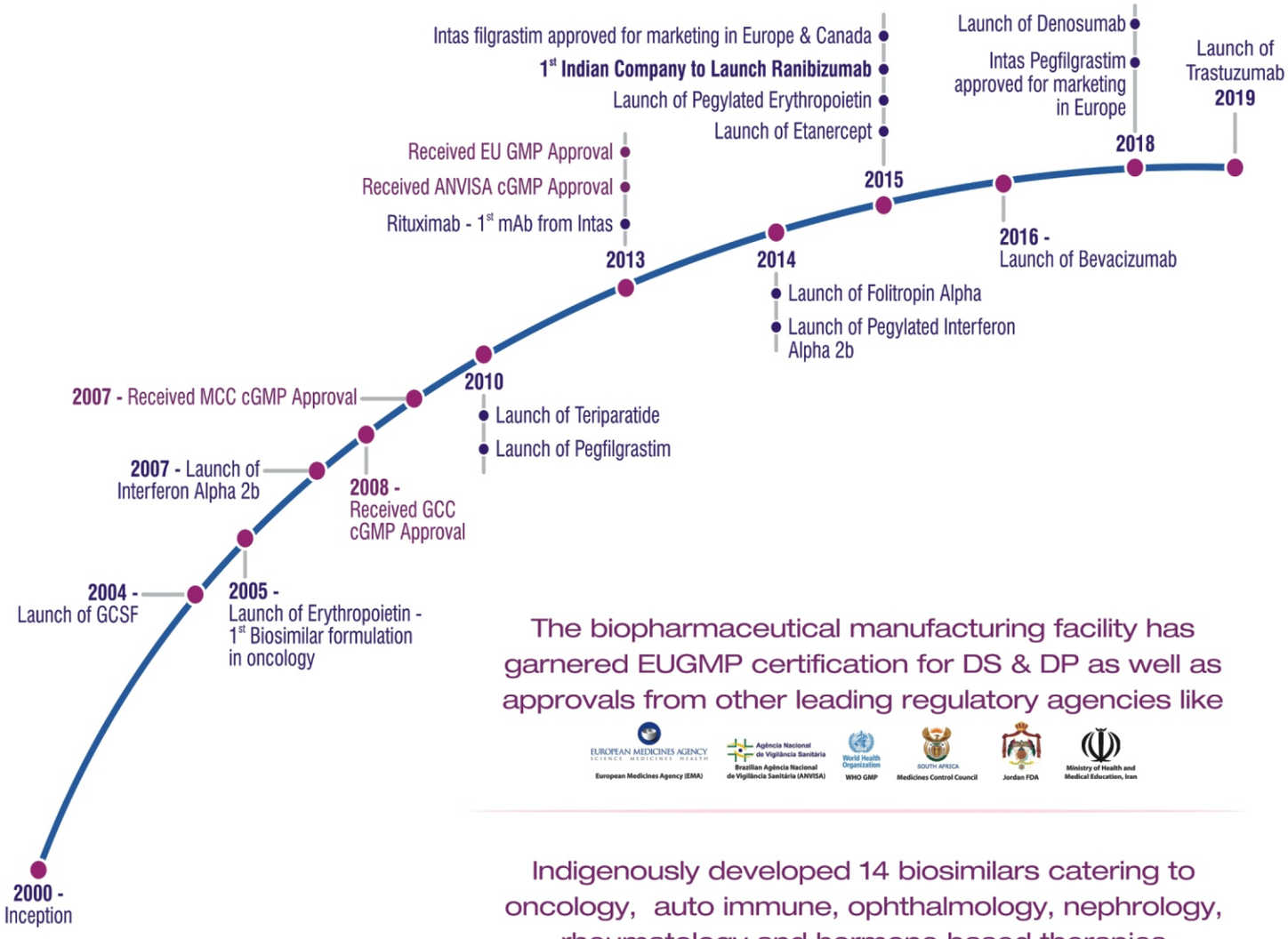
Future studies need to focus on sterilization of the 3D-printed instruments using chemical methods such as ethylene oxide gas, also biocompatibility studies would need to be carried out. Nano printing technology would be needed to print smaller gauge instruments. With the advancement in metal 3D printing the applications will keep growing with time.

References

1. 3D Printed Vitreoretinal Forceps. American Academy of Ophthalmology; 2016. Available from: https://www.aao.org.
2. Navajas EV, Ten Hove M. Three-Dimensional Printing of a Transconjunctival Vitrectomy Trocar-Cannula System. Ophthalmologica. 2017;237(2):119-122.
3. Ophthalmicdocs. Smartphone Ophthalmoscope oDocs Fundus. Available from: http://www.instructables.com/id/Retinal□Imaging□Device□OphthalmicDocs□Fundus.
4. Bioniko Website for 3D Printed Eye Surgery Simulators. Availablefrom:http://www.bioniko.com.
5. FurdováA, SramkaM, ThurzoA, FurdovaA. Earlyexperiences of planning stereotactic radiosurgery using 3D printed models of eyes with uvealmelanomas. ClinOphthalmol2017;11:267□71.
6. Maloca PM, Tufail A, Hasler PW, Rothenbuehler S, Egan C, Ramos de CarvalhoJE,Spaide RF. 3D printing of the choroidal vessels and tumours based on optical coherence tomography. ActaOphthalmol. 2019 Mar;97(2):e313-e316.
7. Maloca PM, Spaide RF, Rothenbuehler S, Scholl HPN, Heeren T, Ramos de CarvalhoJE, Okada M, Hasler PW, Egan C, Tufail A. Enhanced resolution and speckle-freethree-dimensional printing of macular optical coherence tomography angiography.ActaOphthalmol. 2019 Mar;97(2):e317-e319.
8. KusakaS, Tachibana K, TsujiokaD, HottaF, EguchiH, Shimomura Y. antifogging device to prevent moisture condensation during vitrectomy with noncontact wide-field viewing system. retina. 2017 jun;37(6):1215-1217.
9. Shi P, Edgar T Y S, Yeong W Y, et al., 2017, Hybrid three-dimensional (3D) bioprinting of retina equivalent for ocular research. International Journal of Bioprinting, vol.3(2): 138146 .
10. Akkara JD, Kuriakose A. The magic of three-dimensional printing in ophthalmology. Kerala J Ophthalmol 2018;30:209-15 .

INTAS

A Global Biopharma Player



The biopharmaceutical manufacturing facility has garnered EUGMP certification for DS & DP as well as approvals from other leading regulatory agencies like

European Medicines Agency (EMA) · Brazilian Agência Nacional de Vigilância Sanitária (ANVISA) · WHO GMP · Medicines Control Council · Jordan FDA · Ministry of Health and Medical Education, Iran

Indigenously developed 14 biosimilars catering to oncology, auto immune, ophthalmology, nephrology, rheumatology and hormone based therapies

DS: Drug Substance • **DP:** Drug Product • **EUGMP :** European Union's Good Manufacturing Practice

INTAS PHARMACEUTICALS LTD.

Corporate House, Near Sola Bridge, S.G. Highway, Thaltej,
Ahmedabad-380054, Gujarat, INDIA • Website: www.intaspharma.com

RAZUMAB®
Ranibizumab 0.5mg Injection

CASE REPORT

Intravitreal Bevacizumab for management of Purtschers Retinopathy

Authors:
1. Kanwaljeet H Madan M.S.*
2. Sangeet Mittal M.S.*
3. Jaswant Singh Thind M.S.*

Institution:
*Thind Eye Hospital Ltd.

Corresponding Author:
Dr Sangeet Mittal
Thind Eye Hospital Ltd, Jalandhar, Punjab

Abstract:

35 years male presented with sudden diminution of vision in both eyes following clavicular fractures in an accident. His Best Corrected Visual Acuity was 6/60 in OD and CF@1metre in OS. He was diagnosed as Purtscher Retinopathy in both eyes. He received 3 intravitreal injections of Bevacizumab at monthly intervals in both eyes. At 9 months follow up, vision improved to 6/6 in both eyes. This particular case showed improvement with Intravitreal Bevacizumab in Purtscher Retinopathy, a disease whose prognosis is otherwise unfavorable. There are no previous reports of use of Bevacizumab in the management of post-traumatic Purtscher Retinopathy.

Introduction:

Purtscher Retinopathy is a haemorrhagic and vaso-occlusive retinopathy associated with blunt thoracic trauma, head trauma and numerous non traumatic diseases like acute pancreatitis, fat embolization etc.[1] Purtscher Retinopathy leads to irreversible loss of vision and the disease may be bilateral in 60% of cases.[2] There is currently no established treatment for Purtscher retinopathy, although the literature consists of several case reports of treatment with corticosteroids, without certainty as to effect on the clinical course.[3] Despite treatment the visual recovery is poor.[4] We report for the first time a case of post traumatic Purtscher Retinopathy who was successfully treated with intravitreal injections of Bevacizumab.

Case:

35-year healthy male presented with sudden decrease of vision following a road side accident 2 days ago. He gave history of multiple fractures of clavicle (Fig 1) during the accident. On examination, his best corrected visual acuity was 6/60 in right eye and counting fingers close to face in left eye. Slit lamp examination was within normal limits. Fundus examination of both eyes revealed retinal ischaemia and infarction with cotton wool spots and intraretinal haemorrhages arranged in a petaloid fashion around the optic disk suggestive of Purtscher flecken (Fig 2A & 2B). FFA revealed areas of retinal ischaemia and non perfusion with abruptly ending arterioles (Fig 2C & 2D). OCT showed cystoid macular oedema with inner layer hyper-reflectivity due to Purtscher flecken (Fig 2E & 2F-blue arrows)

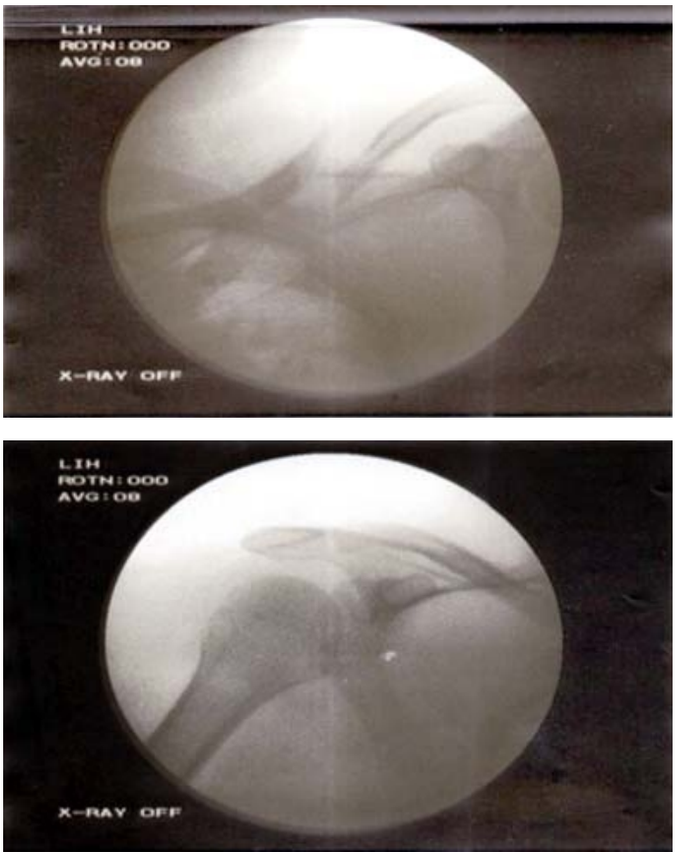


Fig 1: Clavicular Fracture

and outer layer hypo-reflectivity due to inner layer oedema secondary to capillary occlusion (Fig 2E & 2F-yellow arrows). A diagnosis of Purtscher Retinopathy was made and the patient was advised monthly injections of intravitreal Bevacizumab in both eyes. The off-label use of intravitreal Bevacizumab was explained to the patient and an informed consent was obtained from him. He underwent monthly injections of intravitreal Bevacizumab for 3 months in both eyes. On every follow up visit, fundus photographs and OCT were done (Fig 3). Fundus photographs at each visit revealed continuous decrease of cotton wool spots and intraretinal haemorrhages. OCT also showed continuous decrease in macular oedema and sub retinal fluid. The central retinal thickness decreased from 396µ to 243µ in right eye and from 451µ to 255µ in left eye. Best corrected visual acuity improved to 6/6 in right eye at 20 weeks and in left eye at 36 weeks.

Discussion:

Purtscher Retinopathy is described as a chorioretinopathy associated with indirect trauma, associated with a constellation of retinal findings including cotton-wool spots, retinal haemorrhages, optic disc oedema, and Purtscher flecken (areas of inner retinal whitening). The condition is most classically associated with chest compression trauma.[1] When the features of Purtscher retinopathy present without the history of frank trauma, it is called Purtscher like retinopathy. This can be due to acute pancreatitis, fat embolization, pre-eclampsia, haemolysis etc.[2] Our case gave history of trauma with multiple fractures of clavicle, hence a diagnosis of Purtscher Retinopathy was made. The diagnosis was confirmed by the classical features of Purtscher Retinopathy on FFA (hypo-perfusion with abruptly ending arterioles) and OCT (inner layer hyper-reflectivity due to Purtscher flecken and outer layer hypo-reflectivity due to inner layer oedema secondary to capillary occlusion).[3]

There are reports of successful treatment of Purtscher Retinopathy with high dose steroids.[5] The authors have attributed the use of steroids to their ability to stabilize the damage to neuronal membranes and microvascular channels, inhibition of granulocyte aggregation and complement activation. However, the treatment with steroids have not been established and remains controversial. In a systematic review of 110 eyes of Purtscher Retinopathy, the authors have found no benefit of the use of corticosteroids in the management.[3] Corticosteroids seem to have beneficial effect in Purtscher like

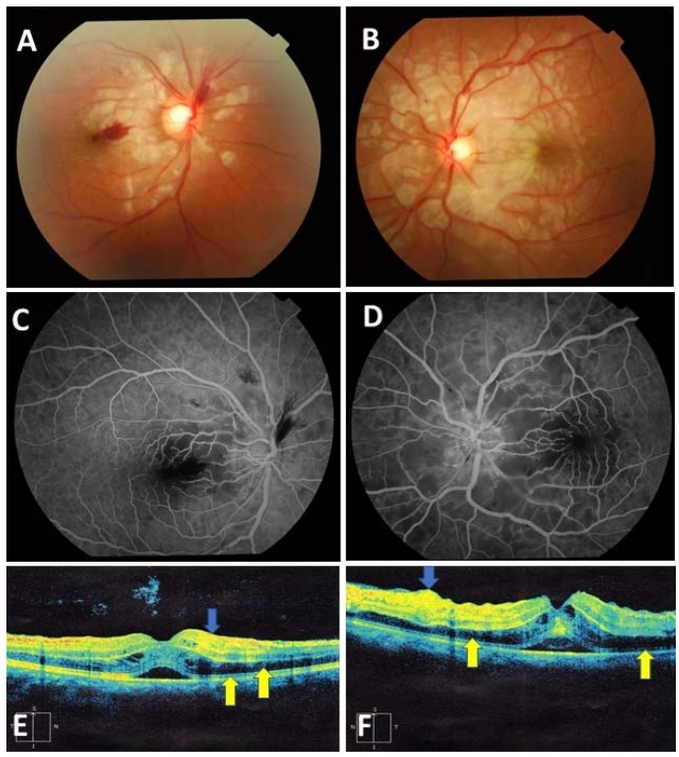
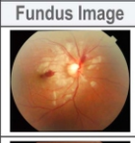
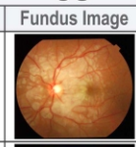
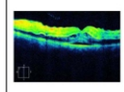
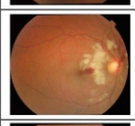
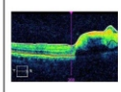
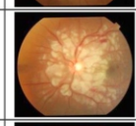
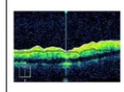
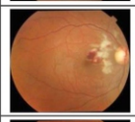
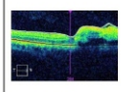
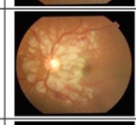
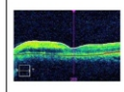
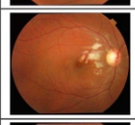
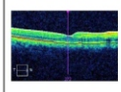
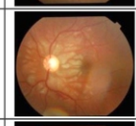
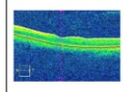
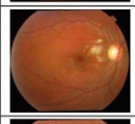
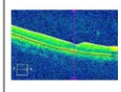
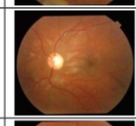
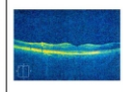
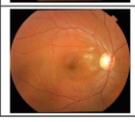
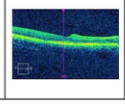
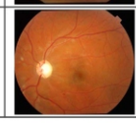
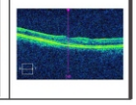


Fig 2: At Presentation (A & B) Fundus Photograph of OD & OS showing Purtscher Flecken (C & D) FFA of OD & OS showing retinal ischaemia with abruptly ending retinal arterioles (E & F) OCT scan of OD & OS showing inner layer hyper-reflectivity due to Purtscher flecken – blue arrows & outer layer Hypo-reflectivity due to inner layer edema

COURSE	OD			OS		
	Visual Acuity	Fundus Image	OCT Image	Visual Acuity	Fundus Image	OCT Image
At Presentation (IVB*-1)	20/200			CF@1Meter		
At 4 weeks (IVB*-2)	20/120			CF@2Meter		
At 8 weeks (IVB*-3)	20/60			20/200		
At 12 weeks	20/40			20/60		
At 20 weeks	20/20			20/30		
At 36 weeks	20/20			20/20		

*IVB=Intra Vitreal Bevacizumab

Fig 3: Follow up visits showing gradual improvement in both eyes

Retinopathy and not in post trauma Purtscher Retinopathy.[2]

The pathogenesis of Purtscher Retinopathy remains uncertain. It results from occlusion of small arterioles by intravascular micro-particles generated by underlying systemic conditions. Subsequent endothelial damage can cause incompetency of the micro vascular circulation, resulting in occlusion and ischemia.[6] Intravitreal injection of anti-VEGF drugs is mainstay for the treatment of many microvascular diseases of retina.[7] Bevacizumab may benefit the cases with macular oedema.[8] Nesmith et al have reported beneficial effects of single injection of intravitreal bevacizumab in a case of Purtscher like Retinopathy secondary to chronic pancreatitis and cirrhosis.[9] Beneficial effects of systemic eculizumab were also seen in a patient of Purtscher like retinopathy secondary to atypical haemolytic uremic syndrome.[10] Our patient was diagnosed to have post traumatic Purtscher retinopathy and received injections of intravitreal Bevacizumab at monthly intervals for 3 months in both eyes and a continuous improvement in disease was seen after every visit.

This is the first report of successful treatment of a patient with post traumatic Purtscher Retinopathy with intravitreal injections of anti-VEGF agents. Purtscher Retinopathy is an extremely rare condition with a poor visual outcome. We suggest this treatment be considered in similar cases.

References:

1. Sally A Buckley, Bruce J. Purtscher retinopathy. *Postgrad Med J* 1996; 72:409 -412

2. Agrawal A, McKibbin MA. Purtscher and Purtscher-like retinopathies: a review. *SurvOphthalmol* 2006; 51:12936

3. Miguel AIM, Henriques F, Azevedo LFR, Lourerio AJ, Maberley DAL. Systematic Review of Purtscher and Purtscher-Like Retinopathies. *Eye* 2013; 27:1-13

4. Yanoff M, Duker J S. *Ophthalmology,* 3rd ed. Edinburgh: Mosby; 750754

5. Atabay C, Kansu T, Nurlu G. Late visual recovery after intravenous methylpred nisolone treatment of Purtscher retinopathy. *Ann Ophthalmol* 1993; 25:330333.

6. Parc C. Purtscher-like retinopathy as an initial presentation of a thrombotic microangiopathy associated with antineoplastic therapy. *Am J Hematol* 2007; 82:486-8

7. Grisanti S, Ziemssen F. Bevacizumab: Off-label use in ophthalmology. *Indian J Ophthalmol* 2007; 55:417-20

8. *Holak HM, Holak S. Prognostic factors for visual outcome in Purtscher retinopathy. SurvOphthalmol2007; 52:117-8*

9. *Nesmith BL, Bitar MS, Schaal S. The anatomical and functional benefit of bevacizumab in the treatment of macular edema associated with Purtscher-like retinopathy. Eye2014; 28:1038-40.*

10. de Carvalho JER, Schlingemann RO, Oranje M, Bemelman FJ, van Schooneveld MJ. Reversal of threatening blindness after initiation of eculizumab in Purtscher-like retinopathy secondary to atypical hemolytic uremic syndrome. *Int Ophthalmol* 2018; 38:399-407

Notes

Notes

Game Changer

In 1968, Dick Fosbury revolutionized the high jump by developing a technique that elevated him to Olympic gold, raising the bar for athletes the world over.

It's time to rewrite the rules of vitreoretinal surgery.

- The ULTRAVIT® 7500cpm probe provides the benefit of **faster cutting** and **smaller vitreous bites** without fluidic compromise.[1]
- Trust in integrated and **stable IOP compensation**[2, 3]
- **Helps enhance patient outcomes** and achieve faster visual recovery with ALCON® MIVS platforms[4]
- **Increase efficiency during cataract removal** with OZil® Torsional Handpiece[5, 7]
- **Improve your OR set up time by 36%** with V-LOCITY® Efficiency Components[6]

Alcon®

a Novartis company

constellation® VISION SYSTEM

© 2015 Novartis 1/15 CON15001JADi

1. Abulon, et al. Porcine Vitreous Flow Behavior During High Speed Vitrectomy up to 7500 Cuts Per Minute. ARVO Poster, 2012. 2. Riemann C, et al. Prevention of intraoperative hypotony during vitreoretinal surgery: an instrument comparison. ASRS Poster Presentation, 2010.* 3. Buboltz, DC. New method for evaluating flow rates and intraocular pressures during simulated vitreoretinal surgeries. ARVO Congress Poster Presentations, 2010. Fort Lauderdale, FL.* 4. Nagpal M, et al. Comparison of clinical outcomes and wound dynamics of sclerotomy ports of 20, 25, and 23 gauge vitrectomy. Retina. 2009;29(2):225-231. 5. Davison JA, Cumulative tip travel and implied follow ability of longitudinal and torsional phacoemulsification J Cataract Refract Surg 2008; 34:986–990 6. Alcon data on file 954-0000-004. 7. Fernández de Castro, L. E. et al. (2010). Bead-flow pattern: Quantization of fluid movement during torsional and longitudinal phacoemulsification. J Cataract Refract Surg 36(6): 1018-1023.
*Based on bench lab testing.

The CONSTELLATION® Vision System is an ophthalmic microsurgical system that is indicated for both anterior segment (i.e., phacoemulsification and removal of cataracts) and posterior segment (i.e., vitreoretinal) ophthalmic surgery. The ULTRAVIT® Vitrectomy Probe is indicated for vitreous cutting and aspiration, membrane cutting and aspiration, dissection of tissue and lens removal. The valved entry system is indicated for scleral incision, cannulae for posterior instrument access and venting of valved cannulae. The infusion cannula is indicated for posterior segment infusion of liquid or gas. **Warnings and Precautions:** The infusion cannula is contraindicated for use of oil infusion. Use of disposables and handpieces other than those manufactured by Alcon may affect system performance and create potential hazards. Attach only ALCON® supplied consumables to console and cassette luer fittings. Do not connect consumables to the patient's intravenous connections. Mismatch of consumable components and use of settings not specifically adjusted for a particular combination of consumable components may create a patient hazard. Vitreous traction has been known to create retinal tears and retinal detachments. The closed loop system of the CONSTELLATION® Vision System that adjusts IOP cannot replace the standard of care in judging IOP intraoperatively. If the surgeon believes that the IOP is not responding to the system settings and is dangerously high or low, this may represent a system failure. To ensure proper IOP Compensation calibration, place infusion tubing and infusion cannula on a sterile draped tray at mid-cassette level during the priming cycle. Leaking sclerotomy may lead to post-operative hypotony. Refer to the CONSTELLATION® Vision System Operators Manual for a complete listing of indications, warnings, and precautions.

For the use of registered medical practitioners, hospitals or a laboratory

S/CON/P-910207/202002

DME*

Allergan

Ozurdex®

(dexamethasone intravitreal implant) 0.7 mg

*For the treatment of adult patients with visual impairment due to Diabetic Macular Edema (DME) who are considered unsuitable for, or insufficiently responsive to, non-corticosteroid therapy or are pseudophakic

Allergan India Pvt Ltd
Level 7, Prestige Obelisk No 3; Kasturba Road; Bengaluru; Karnataka; INDIA 560 01
Tel: +91-80-40707070; Email: IN-Allergan@allergan.com; Website: www.allergan.co.in

IN/2755/2018