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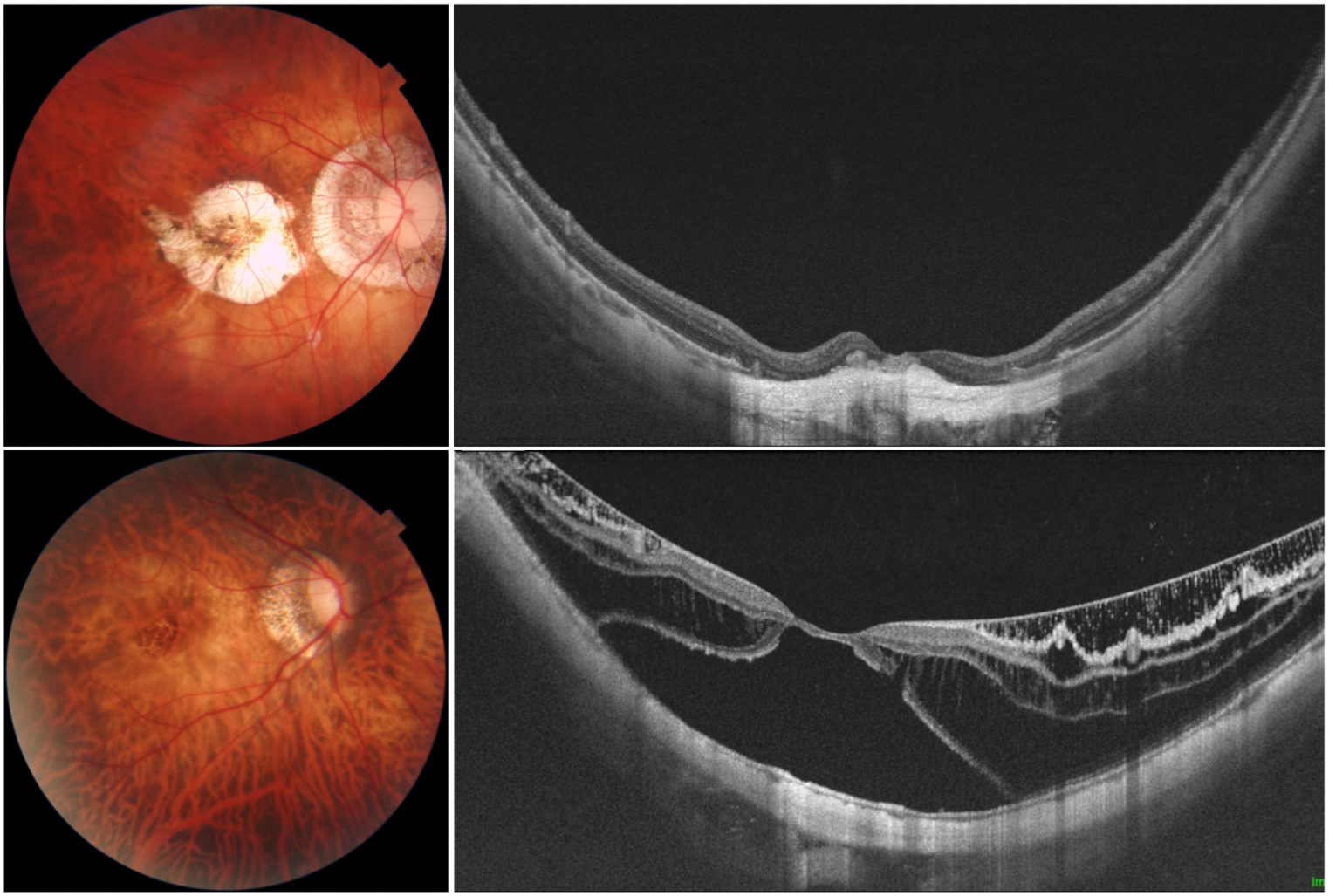
JUNE 2022

The Official Newsletter of the

VITREORETINAL SOCIETY-INDIA



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Cover Image : Colour Photo and OCT Image of
- Atrophic Maculopathy
- Myopic Traction Maculopathy

Courtesy : **Dr. Pradeep Sagar B K**
Sankara Eye Hospital, Shivamogga

Creative Credit : **Dr. Shwetha Suryakanth**
Sankara Eye Hospital, Bengaluru

FROM THE PRESIDENT'S DESK

Dear Friends

The year 2022 seems to be going well as the Covid situation seems to be steadily improving.

The preparations for the annual meeting in Nagpur in December are underway. The LOC is working very hard to make this meeting very memorable. The registration and the abstract submissions are open now! I urge all of you to participate actively by sending as many submissions as possible.

Dr. Mahesh Shanmugam has conducted a unique Webinar on the well being of the vitreoretinal surgeon, which drew excellent response from all! He has planned more Webinars in the times to come. Dr Mahesh is also bringing monthly editions of 'Retina Roundup'- a collection of important journal articles. I urge all of you to take advantage of this. It is also available on YouTube in the audio format.

The safety committee of VRSI is keeping tabs on the adverse reactions reported by the members, which have been fortunately, very few. Please report any adverse reaction to any drug (especially intra vitreal injections) promptly. The details are available on the website. VRSI website has also gone through many changes to make it more useful to the members.

Wishing the much awaited All India Ophthalmic conference at Mumbai a grand success ! VRSI has a stall there wherein registration for the Nagpur conference can be done.

Take care, Stay Safe and let us meet at Nagpur !

Regards
Dr. N S Muralidhar
Honorable President - VRSI

FROM THE HONORARY SECRETARY'S DESK

Dear Seniors and Friends,

Our journey as the new governing council has begun under the able leadership of Dr. N.S Muralidhar. We have various sub committees performing their respective roles and focusing on various aspects of working of the society. We are in the process of modifying the website with addition of new features so that it becomes more user friendly both for our members and patients. We are having video and image competition every month and reporting of adverse drug reactions. Many more activities are being planned in the coming months.

We had an overwhelming response to the webinar titled "Well-being for the Vitreoretinal Surgeon" and pre-recorded retina round ups released every month by our scientific convener Dr. Mahesh P Shanmugam.

We have not had a physical annual conference for last two years and therefore this year we all are looking forward to meeting everyone in person at Nagpur from 2-4th December, 2022. We have an excellent scientific program in place with a galaxy of national and international speakers.

I request all the members to register and participate in the forthcoming annual conference and make it a grand success.

Regards
Dr. Manisha Agarwal
Hon. General Secretary
VRSI

FROM THE CONVENER, SCIENTIFIC COMMITTEE'S DESK

Dear Friends,

It has been an exciting start to the new governing council of the VRSI so far. The new editorial team comprising of dynamic youngsters is bringing in this second issue of the newsletter, which will now focus on a specific theme in each issue. This issue is on myopic maculopathy is spear headed by Dr. Pradeep Sagar.

We have so far been dependent on western collaborative studies for answers to key questions in vitreoretinal diseases and surgery. After all, we are the second largest population and also the home to the second largest diabetic population in the world. It is time that we generate our own quality data, define treatment goals tailoring them to suit our specific demands in India. Towards this end, we have completed the first multicentric study under the VRSI and the manuscript is being drafted currently. I do hope that we can make a meaningful impact to our practices in India and elsewhere with the collaborative studies in the next few years.

All of us are looking forward to the annual meeting this year which will focus on inclusivity, more discussion time than presentation time and hopefully a more rounded program that should interest each and every one of you. Let us all share our research in the forthcoming meeting and make it more meaningful and rewarding.

Warm regards and thanks for all the support.

Dr. P. Mahesh Shanmugam
Convener, Scientific Committee, VRSI.

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 works (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.

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INTRODUCTION TO THE ISSUE....

The posterior segment complications are the leading cause of legal blindness in patients with myopia. With the increase in prevalence of myopia, we would encounter the posterior segment complications of myopia often. This issue of VRSI newsletter focuses on 'Myopic maculopathy'.

The current issue deals with management of common posterior segment complications like myopic choroidal neovascular membrane (CNV), dome shape macula and myopic traction maculopathy (MTM).

CNV in myopia behaves differently compared to AMD. Dr Pradeep Venkatesh briefs us regarding this important topic.

Dome shape maculopathy is a poorly understood entity and Dr Mukesh Jain has written a concise article regarding this entity.

Management of myopic traction maculopathy and myopic retinal detachment is challenging for every vitreo-retinal surgeon. Tips related to surgical management of MTM is discussed in a questionnaire by two experts in the field : Dr Barbara Parolini and Dr VR Saravanan.

Macular buckle and scleral imbrication are some of the less commonly performed surgeries for MTM. Dr Pradeep Susvar and Dr Chetan Rao shares their experience in macular buckling.

Dr Payal Shah has made an attempt to explore the newer terminologies and treatment options described in myopic eyes in the last decade in a section on 'Newer concepts in myopia'.

Finally, Dr Sriram Simakurthy summarizes all about the management of myopic maculopathy in a schematic.
We hope that this issue gives an insight into myopic maculopathy.

Thank you
Dr. Pradeep Sagar
Deputy Editor, VRSI

INTRODUCTION TO THE ISSUE....

CHOROIDAL NEOVASCULAR MEMBRANES IN MYOPIA - BRIEF OVERVIEW

Dr. Pradeep Venkatesh, MD
Dr. Rajendra Prasad Centre for Ophthalmic Sciences
All India Institute of Medical Sciences, New Delhi

Myopia is the 2nd most common cause of choroidal neovascularization after age related macular degeneration and can lead to irreversible visual loss.[1] Choroidal neovascularization in patients with pathological myopia occurs in about 5-10% of cases [worldwide pathological myopia prevalence being about 1 to 3 percent].[2] Risk factors include the severity of myopia, gender, age, concurrent pathology, trauma and ethnicity. The term neovascularization generally brings to mind that it is a result of tissue hypoxia and nonperfusion. However, unlike in patients with proliferative vascular retinopathies and proliferative diabetic retinopathy, no evident nonperfusion of tissue is seen on fluorescein angiography. Hence, choroidal neovascularization likely results from a combination of two factors in tandem- prolonged breach in the Bruch's membrane and imbalance in the local proangiogenic [VEGF] and antiangiogenic [PEDF] milieu. Breach in the Bruch's membrane could be clinically visible [lacquer crack] or subclinical but is an essential precursor for the development of choroidal neovascularization. These new vessels could be of two forms- true neovascularization or merely an ingrowth of the choriocapillaris.

The Bruch's membrane, despite being only about 3µ in thickness, is composed of 5 layers- middle elastic, outer collagenous, inner collagenous layer and basement membrane of the choriocapillaris endothelial cells on the outside and basement membrane of the retinal pigment epithelial cells, on the inside. Other than providing structural support and acting as a segregating barrier between the highly vascular choriocapillaris and avascular retina and retinal pigment epithelium, this membrane probably also plays the role of contact inhibition and of a nano-filter [precisely controlling the amount of molecules, including VEGF, which reaches the choriocapillaris from the basolateral aspect of the retinal pigment epithelium]. In myopia, it is likely that breaks develop in the Bruch's membrane at an earlier age and are likely to be larger and visible as lacquer crack, as it is structurally weak or due to abnormal forces of stretching or a combination of these. The duration for which the break persists without 'healing' may be important in the pathogenesis of myopic CNV, a possibility because not all patients with lacquer crack develop CNV.

Choroidal neovascular membranes in myopia have features very distinct from those seen in patients with neovascular age related macular degeneration [nAMD], as well as idiopathic CNV [idCNV] and inflammatory CNV [inCNV]. Differences exist in the age, gender, refractive status and its severity, morphological characteristics [shape, size and location of the membrane as well as pattern and caliber of the new vessels] and functional characteristics [velocity of blood flow and permeability or severity of leakage]. In addition, the natural history [disease course without any intervention] as well as response to treatment and prognosis of myopic CNV [mCNV] is different from that seen in nAMD. In mCNV, the new vessels are usually located between the Bruch's membrane and retinal pigment epithelium [RPE], are smaller is size, have a discrete pattern and leak less [on fluorescein angiography]. Subretinal hemorrhages when present are generally small and thin layered. Unlike nAMD, these membranes do not have concurrent pigment epithelial detachments [PED]. In its natural course, mCNV resolves by scarring [Forster-Fuchs' spot], but the scar too is small in size. Eventually the scar is replaced by atrophy. Hence, three stages of mCNV are recognized- active, scar and atrophic. Visual recovery depends on the presence of concurrent myopic macular pathology [e.g., severe atrophy of the overlying retina] and the number of recurrences [always much less than nAMD]. In some patients there could be other features of high myopia such as myopic traction maculopathy, dome shaped macula and posterior staphyloma and all of these have a bearing on the development of mCNV and its response to

	CNV in Myopia	CNV in AMD
Type	Type 2	Types 1/ 2/ 3/ PCV
Age	30-60 years	Above 50 years
Laterality	Unilateral/ delayed bilaterality	Early bilaterality
Refractive status	High/ Pathological myopia	Emmetropia/ simple myopia
Major symptom	Metamorphopsia	Diminution of vision
Drusen	Absent	Common
Chorioretinal atrophy [macula]	Common	Infrequent [only if concurrent GA]
Lacquer cracks	Common	Absent
Size at presentation	Small	Medium to large
Subretinal and intraretinal fluid	Minimal	Significant
Leakage on FFA	Minimal	Appreciable
Daily growth of membrane	Not measured but likely negligible	5-7 microns/day
Response to anti-VEGF	Satisfactory and sustained	Satisfactory and ill-sustained
Number of injections over 24 months	1-6	> 15
Need for 3 loadings doses	Not necessary	Preferred
Effect of PDT	May have adverse effects	May be marginally beneficial
Main RCTs	RADIANCE/ REPAIR/ BRILLIANCE	Innumerable
Need for sustained drug delivery	Not warranted	Important necessity
Predominant cause of visual loss [long term]	Chorioretinal atrophy [creeping]	Subretinal scarring and fibrosis

Table 1- Summary of differences between choroidal neovascular membrane in patients with myopia / pathological myopia and those with exudative age related macular degeneration

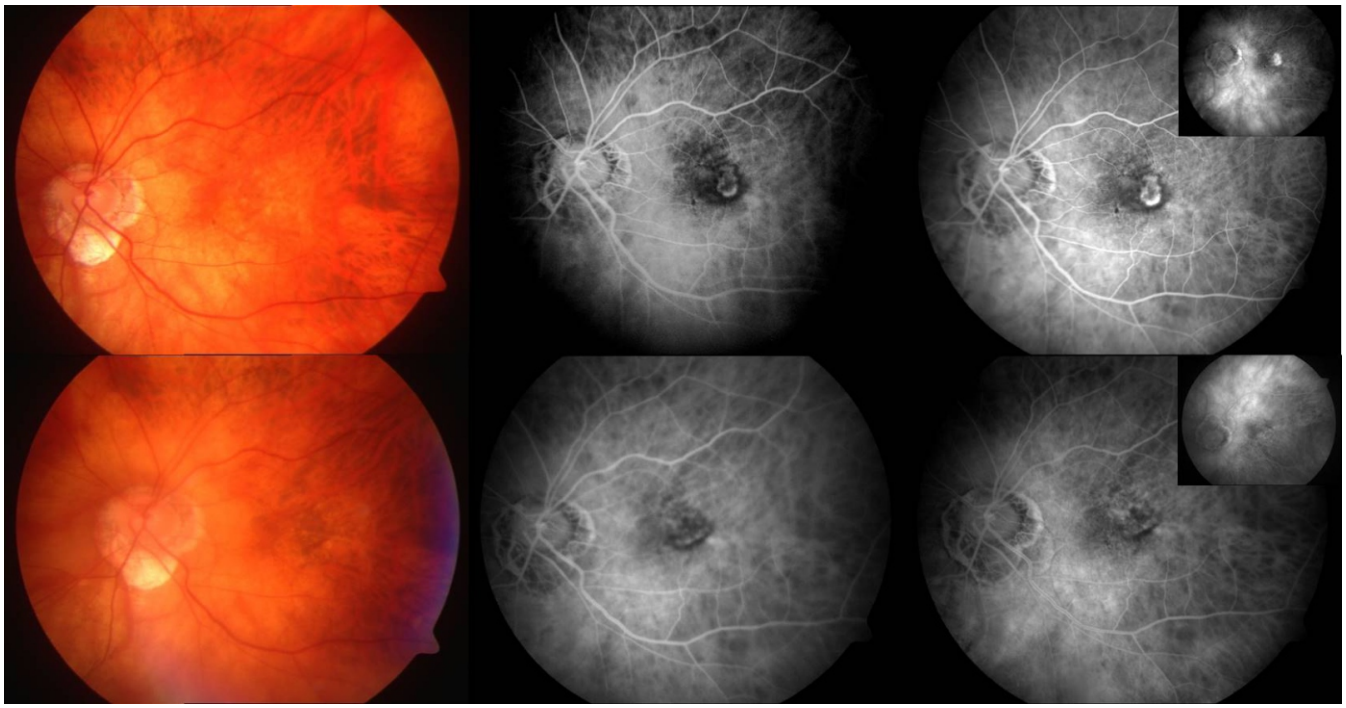

Figure 1 : "Pre and Post laser [PDT] fluorescein angiographic sequence of a patient with high myopia and active choroidal neovascular membrane. Note resolution of the membrane but concurrent increase in chorioretinal atrophy".

treatment. Association with punctate inner choroidopathy [PIC] may also be evident. Importantly, it is important not to neglect screening of the retinal periphery [in both eyes] for the presence of lesions that could predispose the eye to retinal detachment [lattice/ holes/ tears]. It may be best to treat these peripheral lesions before the initiation of intravitreal pharmacotherapy to manage mCNV.

Patients with mCNV present with symptoms of sudden blurring of vision and/or distortion of vision. On fundoscopic examination, loss of normal choroidal reflex and severity of choriocapillaris atrophy may make the detection of a greyish membrane more difficult. In addition, the symptoms could be only because of lacquer crack with/ without subretinal hemorrhage but no CNV. Important investigative modalities that are useful in confirmation of diagnosis, prognostication and follow up include serial fundus photography [30 degree], red free imaging [sensitive in identification of small subretinal hemorrhage], autofluorescence imaging, fluorescein angiography, indocyanine green angiography, optical coherence tomography [OCT] and OCT-Angiography [OCT-A] [Figure 1 and 2]. mCNV membranes are frequently between 1/5th to 1/4th disc area and smaller the membrane, more challenging it is to confirm its presence and to evaluate its response to treatment. Most mCNV membranes are either subfoveal or juxtafoveal. A few studies [BRILLIANCE] however have included extrafoveal mCNV [small number of eyes] in their analysis.

In the pre-pharmacotherapy era, attempts were made to treat mCNV using conventional thermal laser, transpupillary thermotherapy and photodynamic therapy. Over a little over than a decade, it is now firmly established that these modalities actually caused more harm in the long term, by inciting an insidious and prolonged chorioretinal damage. As expected, the current standard of care is intravitreal pharmacotherapy with any of the anti-VEGF drugs. The response to therapy is quite gratifying compared to that with nAMD and in addition, no loading dose [3 monthly injections] is necessary and control is achieved with fewer injections. Differences between mCNV and nAMD membranes is summarized in table 1. Unlike the innumerable studies on the use of anti-VEGF therapy for nAMD, there are only limited randomized studies on mCNV. A summary of all the studies on mCNV with their salient features and observations is shown in table 2.[3-18]

The evidence gathered so far by studies indicate that delayed diagnosis, age above 50 years, CNV size more than 400 microns, larger breaks in Bruch's membrane, Lacquer crack extending to the foveal centre, depth of posterior staphyloma, presence of concurrent MTM, prior treatment with photodynamic therapy and associated chorioretinal atrophy [CRA] are poor prognostic indicators for CNV in myopia. PDT has an adverse impact on outcomes compared to treatment naïve patients [managed with anti-VEGF intravitreal pharmacotherapy] because it is likely to have resulted in subclinical damage to the retinal pigment epithelium and choriocapillaris, owing to its ability to induce transient choroidal ischemia and thrombosis of choroidal vessels [at the time of initial treatment]. These effects of PDT are also likely to be aggravated in myopic eyes compared to those with exudative AMD as they already have compromised RPE-Bruch's-choriocapillaris.

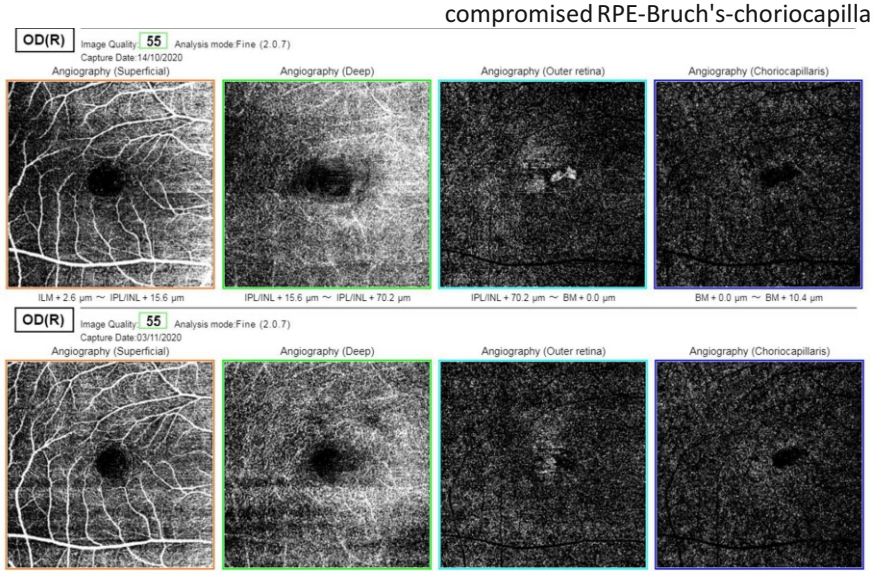

Figure 2 - "Pre and Post injection [Ranibizumab] OCTA images of the right eye of a patient with high myopia and lacy network of the choroidal neovascular membrane. Note resolution of the membrane [outer retinal slab] within a few weeks of treatment".

	Sample size [eyes]	Baseline / final VA or change in letter score	Base line / final central macular thickness [microns]	Treatment	Observation(s)
Wang et al [2021]	64	0.83/ 0.50	263/ 209	Conbercept	Mean CMT was significantly higher [~360µ] in those with recurrence. Risk of recurrence increased 2X for each 100µ higher [262µ] CMT.
Chen et al [BRILLIANCE Study, 2019]	457 [3 groups]	~ 10 letters	Mean reduction of 81µ	Ranibizumab	Confirmed similar efficacy of Ranibizumab in south-Asian eyes. Retinal detachment and endophthalmitis developed in one patient each.
Iacono et al [2017]	48	0.50 / 0.40	NA	Bevacizumab / Ranibizumab	Positive predictors were early diagnosis, higher baseline BCVA, smaller CNV size, hyperfundus FAF CNV pattern
Ikuno et al [MYRROR Study, 2015]	122 [2 groups]	~ 14 letters		Aflibercept	Median number of injections was 2. Inclusion of sham injection as control group was questionable
RADIANCE [2014]- Phase 3	277 [3 groups]	~ 11 letters	NA	Ranibizumab / PDT	67% showed resolution of leakage at 12 months. Median injection was 2-4.
REPAIR [2013]- Phase 2	65	~ 14 letters	Mean reduction of 135µ	Ranibizumab	Median number of injections [12 month follow up] was 3. 1 patient developed culture negative endophthalmitis
Franqueira et al [2012]	40	9 letters	Mean reduction of 80µ	Ranibizumab	BCVA improves every year [5 letters at 12 months to 9 at 36 months]
Yoon et al [2012]	40	0.48 / 0.28	NA	Bevacizumab / Ranibizumab	Foveal lacquer crack and peripapillary atrophy were negative predictors. 1 injection was adequate in 30% [12 months]
Iacono et al [2011]	48	~ 2 line improvement	216/ 205	Bevacizumab / Ranibizumab	3 line improvement in 30% [IVR} to 45% [IVB]. More injections in IVB over 18 months.
Kuo et al [2011]	56	1.09 / 0.77	271/ 228	Bevacizumab	Degree of myopia and presenting VA were important prognostic predictors independent of patient age.
Wakabayashi et al [2011]	31	0.34 / 0.18	NA	Bevacizumab	Single injection followed by PRN was as effective as 3, monthly loading group
Yoon et al [2010]	144 [3 groups]	0.56 / 0.27	NA	Ranibizumab / Bevacizumab	Anti-VEGF alone is superior to PDT as well as combination therapy
Silva et al [2010]	34	0.66 / 0.50	308 / 263	Ranibizumab	Prior PDT eyes also showed improvement
Lallolum et al [2010]	32	20/100 to 20/50	336 / 233	Ranibizumab	47% had more than 3 line improvement
Ikuno et al [2009]	63	0.57 / 0.33	NA	Bevacizumab	BCVA improved or remained stable in 95% at 12 months. Absence of CRA at baseline was a useful predictor.
Hayashi et al [2009]	156	0.68 / 0.45	372 / 301	Bevacizumab	IVB improves VA, arrests CNV and is better than PDT [in which CNV continued to grow] at 12 months FU

Table 2- Summary of studies on choroidal neovascular membranes in myopia [only RCTs and those with statistically significant sample size (> 30) and 12 or more months of follow up have been included]

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DOME SHAPED MACULOPATHY - A BRIEF REVIEW

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Introduction

The prevalence of myopia and high myopia is increasing world-wide. Published data show that nearly 22.9% and 2.7% of the world population have myopia and high myopia, respectively.[1] Progressive globe elongation causes significant visual morbidity resulting from myopic maculopathy- atrophic, tractional and neovascularization.[2]

Dome Shaped Maculopathy [DSM] was first described by Gaucher and associates in 2008.[3] In this article, we present a quick overview of the available literature on the pathophysiology, classification, clinical features, multi-modal imaging and complications associated with DSM.

Historical Perspective

The initial definition by Gaucher and associates[3] of DSM as a convex forward bowing of the macula was qualitative in nature, thus subjected to observational bias. Ohsugi et al. and Ellabban et al. defined DSM if inward bulge of the macular retinal pigment epithelium (RPE) was >50 µm in the vertical or/and horizontal section of the OCT image [Figure 1].[4-5]

Hypotheses on Formation of DSM

Imanura et al. found that the sub-foveal scleral thickness in eyes with DSM was significantly greater than eyes with no DSM (570 +/- 221 microns versus 281 +/- 85 micron).[6] This increased scleral thickness was localized to the sub-foveal region with significant scleral thinning noted in all four para-foveal quadrants similar to high myopic eyes without DSM. Experiments from various animal models support that DSM results from the process of adaptive emmetropization.[7]

Fang et al. postulated that focal relaxation of the posterior sclera by the localized absence of Bruch's membrane as noted in DSM eyes would allow inward bowing of the macula even when the eyeball is expanding out.[8]

Natural History

Asymmetric progressive elongation of the globe and scleral thinning results in increase in the bulge height. Ellabban AA

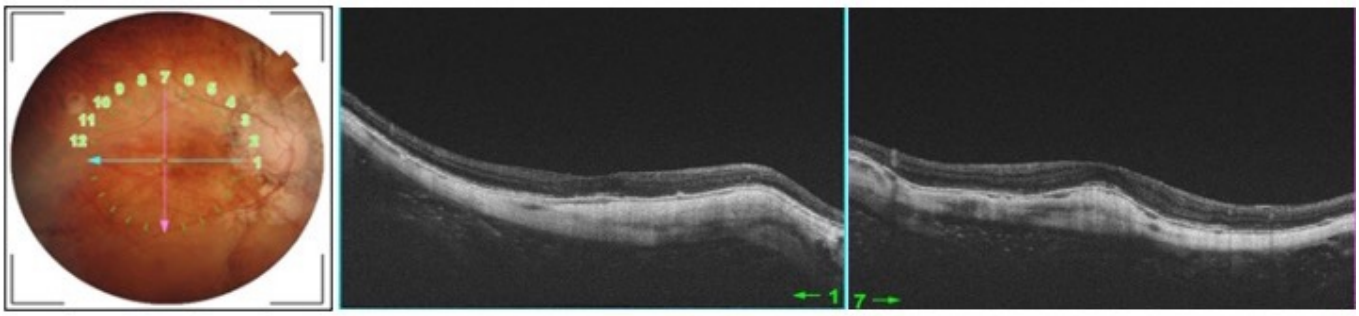

Figure 1. Optical Coherence Tomography scan of horizontal and vertical sections through the fovea in a high myope using swept source technology. The left images shows the posterior pole fundus photographs with tessellated back-ground. The middle image representing the horizontal section shows a relatively flat macular contour. The right image representing the vertical section shows a convex forward bowing of the foveal contour with normal retinal anatomy overlying it.

et al. noted that, over a mean follow up of 2 years, the bulge height increases from 136.5 to 157.6 microns with a generalized choroidal thinning from 28.3 to 22.9 microns.[9]

Symptomatology

Presentation varies from being asymptomatic to metamorphopsia and gradual mild to moderate visual loss over many years.

Fundus Features

Morphology Of DSM

According to the orientation of the protrusion, 3 anatomical types of DSM have been described.

1. Horizontally-oriented Dome: Most common type. Vertical OCT shows the anterior protrusion of the macula in the background of outward bowing in the peri-macular region.
2. Round Shaped Dome: Second most common. Both the vertical and horizontal OCT shows an anterior protrusion of the macula in the background of outward bowing in the peri-macular region.
3. Vertically oriented dome: Least common type. The horizontal OCT shows the anterior protrusion of macula in the background of outward bowing in the peri-macular region.

OCT scan of 9 mm and above with both vertical and horizontal sections at the fovea is required for accurate diagnosis of DSM.

Macular Changes

The presence of DSM is associated with an increased risk of complications.

The OR for sub-foveal retinal detachment, foveal retinoschisis, extra-foveal retinoschisis, macular hole and choroidal neovascularization (CNV) calculated was 35.5, 0.06, 1.6, 0.8, and 0.8, respectively.[9]

Serous Retinal Detachment Without Associated CNV

The incidence of subretinal fluid (SRF) in DSM is highly variable. Multiple hypotheses have been postulated to explain the accumulation of SRF in DSM .

The localized scleral bulge results in significant stress to the retinal pigment epithelium with resultant atrophy.[4] Moreover, compression of choroidal tissue at the border impairs choroidal flow increasing the choroidal thickness and promoting SRF accumulation.[10]

Patients complain of gradual onset decrease in vision/metamorphopsia, although few may be asymptomatic. Over a follow-up of 2 years with no intervention, eyes showed considerable fluctuations in the amount of SRF.[11] However, the vision remains relatively stable.[11] There is little evidence to support effectiveness of any reported treatment modalities at present.

CNV

The development of CNV is independent of the presence of DSM and present with sudden onset decrease in vision, metamorphopsia, and/or scotoma. Intravitreal anti-VEGF drugs are the standard of care for myopic CNV even in eyes with DSM. DSM does not alter anti vascular endothelial growth factor (anti-VEGF) response in myopic CNV.[12-13]

Differential Diagnosis

1. **Choroidal Hemangioma:** A localized bulge with SRF can be confused with choroidal hemangioma.[14] OCT-EDI or SS-OCT would be sufficient in most cases.[47] DSM on fundus fluorescein angiography (FFA) shows occasional staining from sub-retinal fluid while on ICG it is iso-fluorescent. Choroidal hemangioma on FFA shows early diffuse hyper-fluorescence with leakage, while on ICG shows bright hyper-fluorescence with characteristic "washout" phenomenon.[14]
2. **Inferior Staphyloma :** In myopic eyes with inferior staphyloma, the superior edge of the staphyloma at fovea causes a sudden sharp slope. This can be seen like a similar macular "bend" on OCT.[15]

Conclusion

DSM is an anterior forward bowing of the macula in a small proportion of eyes with high myopia. Localized scleral thickening results from regional variation in the bio-mechanical properties of sclera and asymmetric scleral growth during emmetropization. The bulge increases minimally over time resulting from progressive axial elongation. SRF and extra-foveal retinoschisis are commonly associated complications. CNV seems unrelated to DSM and responds well to Anti-VEGF injections.

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CHALLENGES IN SURGICAL MANAGEMENT OF MYOPIC RETINAL DETACHMENT AND MYOPIC TRACTION MACULOPATHY

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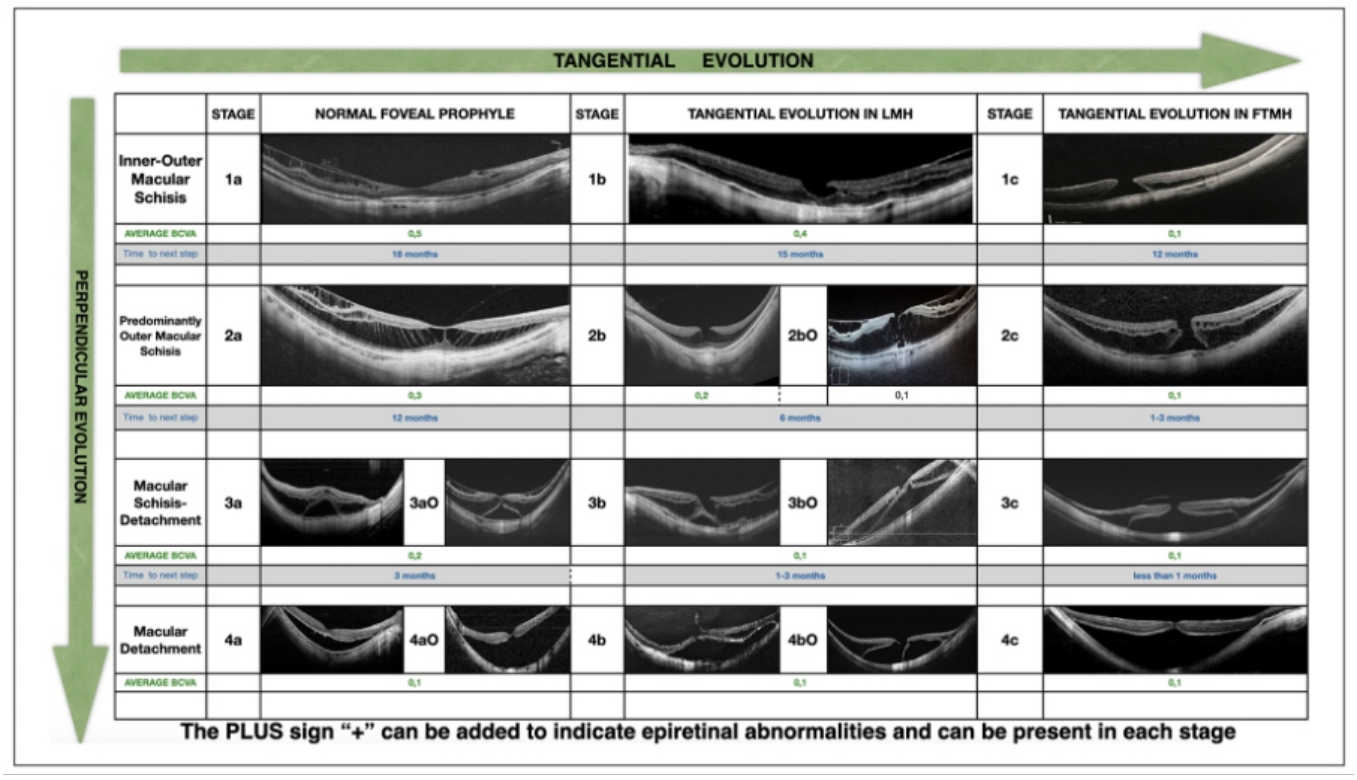
1. Case selection for intervention in MTM?

BP: We simply cannot speak about MTM as a whole and unique disease. It is a spectrum of clinical pictures and evolve in different stages. The management must be different per each stage. I summarized the indication for intervention in one table where I advised different management per each stage of MTM. Please see the table attached as figure.

VRS: Young patients who complain of drop in vision or significant distortion are good candidates for surgery. Eyes with significant atrophic maculopathy are poor candidates for surgery



2. Indications for using encirclage in MTM and RD?

BP: Encircling buckle should be used for

A. RD with peripheral breaks that can be supported by the episcleral element

B. In addition to PPV in presence of PVR with anterior contraction of the vitreous base even in absence of peripheral breaks

Maculoschisis and macular detachment should be treated with a macular buckle

VRS: Encirclage is seldom needed except in cases of intraoperative iatrogenic peripheral retinal tears

3. Measures you take to reach the posterior pole in longer eyes during various steps of surgery

BP: Macular buckle can be inserted with big bayonet forceps like the ones used for general surgery. I have designed a simple 'intromarker' that can be used and assembled to the AJL macular buckle to ease the technique of insertion

The peeling of ERM and ILM is easier with long peeling forceps and can be further eased if the eye is treated first with macular buckle to shorten the eye

VRS: Some tips to reach the posterior pole in long eyes include posterior placement of sclerotomy, removing cannulas and using forceps through bare sclera, approaching macula from temporal sclerotomy and finally using specially manufactured long instruments

4. Tips for complete removal of posterior hyaloid from the surface of retina

BP: Triamcinolone staining is extremely useful to highlight the hyaloid and should be injected multiple times. If the retina is attached, triamcinolone can be used alone. If the retina is detached, we should stain with triamcinolone paying attention to avoid subretinal injection (toxic) and then attach the retina with PFCL. The granules of triamcinolone will be more visible under PFCL and PFCL itself will serve as a hand to hold the retina during peeling which can be done also with a Tano scraper gently passed onto the retina to remove the hyaloid

VRS: Staining vitreous with triamcinolone crystals aids in complete removal of schitic posterior vitreous. Doing ILM peeling also ensures that no overlying vitreous is left over

5. Would you consider ILM peeling in all cases ? If not, what are your Indications for ILM peeling ?

BP: ILM should be peeled only to close full thickness macular holes (FTMH) by using ILM flaps. It should never be touched in cases of maculoschisis and macular detachment without holes in order to limit the risk of inducing a iatrogenic FTMH, and especially in presence of an outer lamellar macular hole (OLMH)

VRS: I do ILM peeling in eyes with FTMH. ILM peeling is not mandatory in eyes where complete removal of posterior schitic vitreous has been ensured

6. Tips for ILM peeling/ Complete peel versus foveal sparing

BP: I strongly suggest not to peel the ILM in any case of MTM unless there is a FTMH

To release the traction in cases of schisis and detachment of the macula, we should push the sclera through the use of a macular buckle because the real cause of schisis and detachment in the macula is the elongation of the sclera

VRS: Good staining helps in easy peeling as myopic eyes have decreased contrast due to chorioretinal degeneration. Using BBG under air, making it heavy by mixing it with dextrose normal saline or staining under liquid per fluoro carbon helps to ensure good staining of the ILM.

Fovea sparing ILM peel is preferred in eyes with thin fovea due to outer lamellar hole formation to prevent postoperative secondary macular hole formation.

7. Will you consider complete peripheral vitreous shaving in MTM ?

BP: There is no need to shave the vitreous in MTM unless in presence of concomitant peripheral retinal breaks. In MTM we should limit the surgery to the treatment of posterior pole, in order to avoid the risk of iatrogenic peripheral lesions. Especially in cases of atrophic areas, it is extremely difficult to treat any lesion. Therefore a minimalist surgery is preferable if there is no peripheral pathology. In presence of rhegmatogenous lesion I suggest only peripheral laser.

VRS: I usually do not perform extensive or meticulous vitreous base shaving especially in the absence of peripheral retinal tears

8. Will you consider a tamponade agent in MTM other than macular hole. If so, what is the tamponade of choice?

BP: If there is no macular hole, there is no need for tamponade. We should pay a special attention in avoiding the use of silicone oil in high myopia for the high rate of toxic effect and secondary glaucoma which is always reluctant to treatment.

VRS: I usually use air as a short-term tamponade agent in the absence of macular hole or peripheral retinal tears

9. How to avoid the complications?

BP:

A. It is essential to treat anteroposterior tractions with a macular buckle and the tangential tractions (holes) with PPV and peeling
B. Avoid peeling ILM if there is no hole
C. Place a flap over any hole (macular or peripheral) if possible
D. Buckle peripheral holes if they coincide with an atrophic area
E. Follow the MTM Staging System Guidelines

VRS:

A. Instrument touch due to sloping surface of staphyloma can be avoided if time is taken to orient yourself to the contour of the posterior pole. Better visualization (depth perception) can also be achieved using contact lenses.
B. Preoperative assessment of axial length will also help to choose the length of forceps needed.
C. Careful posterior vitreous detachment induction and limited vitreous base shaving in MTM will help

UNCONVENTIONAL SURGERIES FOR MYOPIC TRACTION MACULOPATHY (MTM) – MACULAR BUCKLE AND SCLERAL IMBRICATION

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Extrapolating newer classification of MTM for surgical decision making

Parolini and associates recently published a new staging system to describe Myopic Traction Maculopathy termed as MTM Staging System (MSS) based on the OCT changes.[1] MTM can now be considered to evolve into two surgical indications, namely; macular schisis (MS), macular holes (MHRD), which may or may not be a part of macular retinal detachments (MRD) but invariably associated with posterior staphyloma (PS). Pathomechanism of the vector forces mainly narrow down to the 2 types of centrifugal force acting against the normal centripetal force acting at the retinal surface. The perpendicular type of centrifugal forces (pull) come primarily from the vitreous traction and the sclera expansion, whereas tangential type acts at the retinal inner architecture. This 'game of push and pull' of retina (fig.1), vitreous and sclera became the basis and rationale to choose appropriate surgical options to counteract these forces to manage MTMs.[2]

Of the three main approaches to manage MTMs namely vitrectomy, macular buckling (MB) and Scleral imbrications (SI), we discuss here the nuances of the latter two in detail as a relatively unconventional mode of managing MTMs.

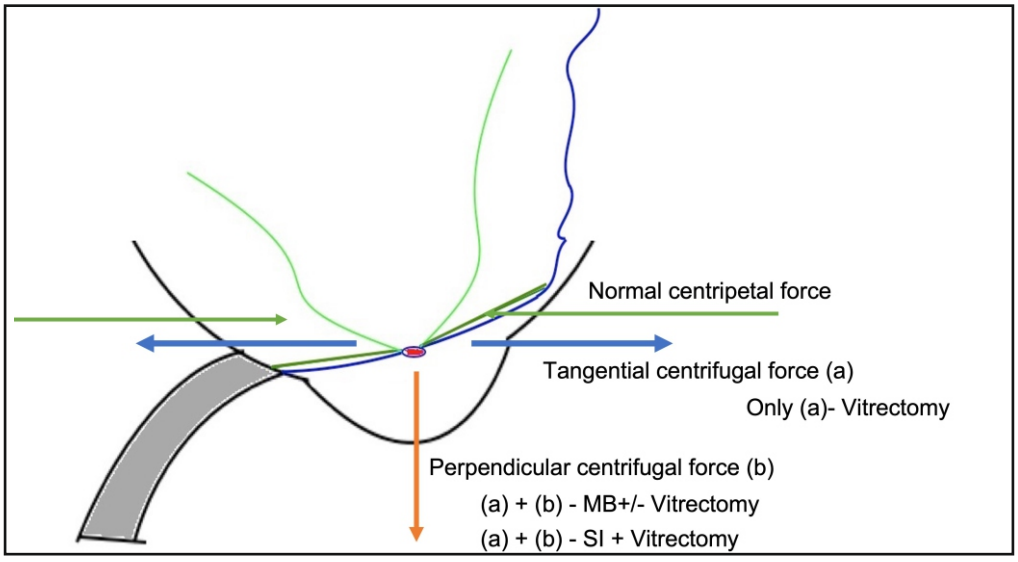

Fig 1 : Schematic representation of the forces acting at the posterior pole guiding the surgical options

Macular Buckling

We have explained the case selection challenges based on our case series in our recent article elaboratively.[3] MB is performed only if there is posterior staphyloma. Objective is to negate the centrifugal force which is anteroposterior in nature, by supporting PS from outside. Targeted indications for MB are mainly MRD and MHRD associated with PS.[3,4,5] Macular buckle is best suited for PS type 2 or 9, with no associated scleral bridging around the disc. A detailed examination of the staphyloma depth is done and in a deep staphyloma, MB is a more favorable option than vitrectomy which can be done for shallow contour. An axial length more than 29 mm and severe staphyloma (>4 mm) contour, both measured on ultrasound B Scan favor towards MB. Swept source OCT of 12 -15 mm wide scans give best details of all 3 layers (vitreous, retina and sclera) in relation to PS. If OCT shows MRD then MB alone can achieve traction relief, however if there is MHRD then MB combined with vitrectomy is necessary for best results.[2,3,4] Matsui group[6] in their large series, showed correlation of ophthalmoscopic fundus PS features with MRI images of staphyloma contour in myopic eyes. Study depicted the irregularities and overall shape of staphyloma using 3D MRI images of the posterior ocular wall, and this correlation aid in the planning and prognosticating the anatomical success of MB. Previous scleral buckle surgery and advanced glaucoma disc changes are few amongst the important contraindications for planning MB.[3]

Types of macular buckle material

Ando's Plombe, Morin Devin Wedge implants, AJL buckles are some of the popular materials studied and published in the literature.[4,5] Recent studies with donor sclera crosslinked with a specific collagen-based solution like genipin explored by Chinese group[7,8] is what is coined as posterior scleral contraction or reinforcement (PST/PSR). They postulated that the genipin crosslinked allogenic sclera act as buckle material, adhering well to the host sclera, promoting the entry of new vessels, thickening the new sclera and hinders the eye axial elongation. Suprachoroidal injection of long-acting hyaluronic acid[9] is yet another innovative technique to indent the macula without external choroidal compression. However, the procedure is limited by the temporariness of the effect of the buckle and technical difficulty in terms of sufficient volume and exact placement of the injection.

Surgical nuances of MB

Surgical steps[3] of any MB are simple but can be exhaustive, reason being that precise placement of the buckle is relatively a blind procedure. The buckle is passively manoeuvred posteriorly, without visualising any of the choroidal vessels and nerve traversing the posterior sclera. The surgeon need to be aware of the inherent risk of developing sub-macular bleed during this step. Indentation effect can be appreciated indirectly either ophthalmoscopically or by means of endo or trans illumination for the location and the height. Surgeons need to be aware of other potential complications during surgery or in the immediate post op period such as subretinal blood extending to the fovea, perforation, haemorrhagic choroidal detachment, high intra-ocular pressure which can potentially lead to permanent vision loss.

A 16-year review of literature by Mateo group[4] concluded that MB lead to significant and sustained anatomical restoration along with visual improvement or stability on long term follow up. Our case series recently published[3] using Morin Devin implants also showed encouraging outcome in terms of anatomical restoration and functional stability with improvement.

Scleral Imbrication

The scleral shortening technique introduced way back in 1893 by Alaimo and 1903 by Muller, postulates that the shortening of sclera reduces the axial length.[10] Scleral shortening was achieved initially by performing full thickness, followed by lamellar scleral resection with much complications, later replaced with scleral infolding (invagination) techniques and latter two techniques were also comparable.[10] The technique of SI has been used in extremely high myopic eyes having MRD or MHRD associated with staphyloma with the purpose of flattening the posterior staphyloma by shortening the axial length, thereby closing the hole and retinal reattachment.[11,12,13]

Surgical nuances of Scleral Imbrication

Scleral imbrication has been conventionally combined with vitrectomy, ILM peeling and a tamponade, where vitreous cortex traction relief by vitrectomy complement with the scleral shortening.[12] Around 2 or 3 mattress scleral sutures (for each of the temporal quadrant) are taken 6-9 mm wide (starting from the arc of recti muscle insertion) with 4 or 5-0 polyester suture, circumferential to limbus either before or after the vitrectomy procedure.[10,11,12,13]

MH closure and retinal reattachment (anatomical) and functional success were noted significant and also sustained

on long term in the literature.[12,13] Ando's study[13] highlighted two information which may be relevant for an appropriate case selection. They objectively assessed changes at the level of staphyloma utilising 3D MRI images (pre and post imbrication) by comparing relevant measurement like posterior axial length, depth, width and the angles of staphyloma. Another observation in their study was that eyes having MHRD limited to PS benefit more with imbrication than in cases having peripheral temporal detachment beyond staphyloma.

Initial reduction was significant in all the studies despite having a small amount of rebound of the axial length.[11,12] Despite the rebound axial length change, retinal attachment and macular hole closure was noted to be permanent.[11,12,13] Similarly, the astigmatism (both with and against the rule) was higher at initial visit and reduced over the period of time. As a part of the post imbrication refractive correction, toric intraocular lenses were implanted to overcome the astigmatic refraction.[11,12]

Scleral dehiscence, thinning and ectasia specially seen in supero temporal quadrant were noted as one important limiting factor to perform the procedure.[12] In such situations, surgeon had to plan an alternative surgical options of vitrectomy or other MB techniques.

Scleral imbrication combined with PPV and gas tamponade produced shortening of axial length and flattening of the posterior eye wall including the posterior staphyloma, thus facilitating the retinal reattachment and the closure of MH with the long-term stability in cases with refractory MHRD.

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NEWER CONCEPTS IN MYOPIA

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With the increasing incidence of myopia, a lot of newer treatment modalities have evolved for myopia ranging from topical medications like atropine, special spectacle lenses like defocus incorporated multiple segments (DIMS), highly aspherical lenslets (HALs), slightly aspherical lenslets (SALs) and progressive additional lenses (PALs), contact lenses like multifocal soft contact lenses (MF SCLs) and orthokeratology/Ortho-K lenses to refractive surgeries. In this article, we would be focusing on the newer concepts related to the posterior segment pathologies in myopic eyes

1. Newer terminologies

1a. Ridge shaped macula

Ridge shaped maculopathy (RSM) and dome shaped maculopathy (DSM) are convex inward protrusions of macula in myopic eyes described in the last decade. RSM is usually seen in children and adolescents and the macula is bulged usually in a single meridian forming a ridge.[1] These eyes usually do not have staphyloma, have a wider base and lesser bulge height compared to DSM and are associated with diffuse chorioretinal atrophy. DSM is usually seen in older individuals and associated with subretinal fluid and extrafoveal macular schisis.[2](Figure 1)

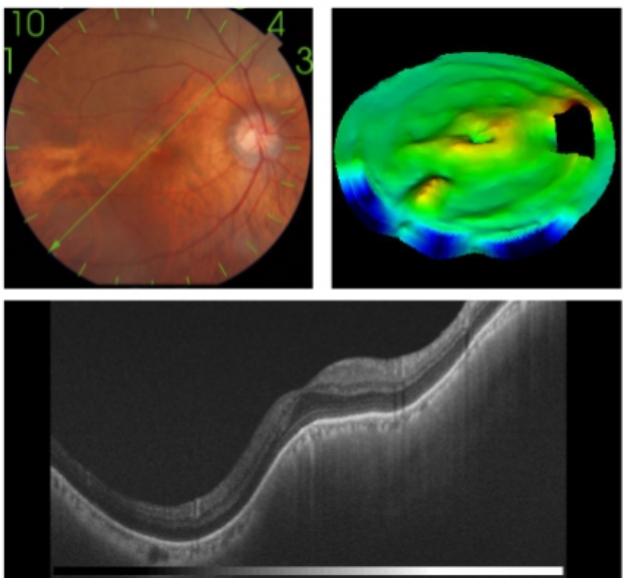

Figure 1: Ridge shaped maculopathy noted in single meridian.

1b. Macular pit

Macular pits are focal areas of absence of retina and choroid with associated scleral ectasia. They are usually seen within areas of chorioretinal atrophy in the macula and posterior pole.[3] It is believed to develop due to extreme scleral thinning. It is reported to develop near the areas of entry of short posterior ciliary artery into the sclera suggesting poor scleral strength in these areas. These areas are sensitive to stress and are at risk of perforation during retrobulbar block or during vitrectomy.[4](Figure 2)

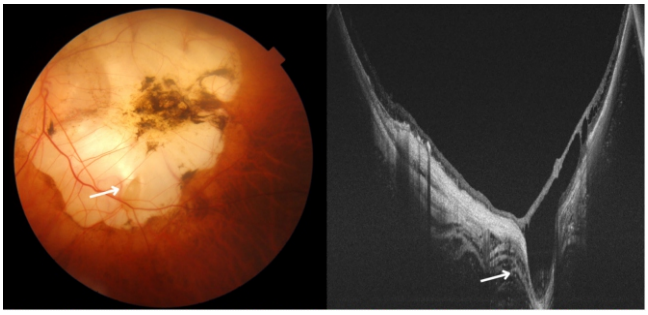

Figure 2: Color photo and OCT showing macular pit within CRA

1c. Intrachoroidal cavitation (ICC)

ICC is described as hyporeflective space underneath an intact retinal pigment epithelium (RPE) seen on optical coherence tomography (OCT). It can be peripapillary or at the macula. ICC is also noted around the areas of chorioretinal atrophy. In ICC, there is complete absence of choroidal vessels and the sclera is bowed posteriorly; while in focal choroidal excavation, only the choroid is affected with underlying sclera being normal. One theory is that ICC is formed due to the transudation of fluid from the intrascleral vessels due to increased transluminal pressure and overlying thin connective tissue.[5](Figure 3)

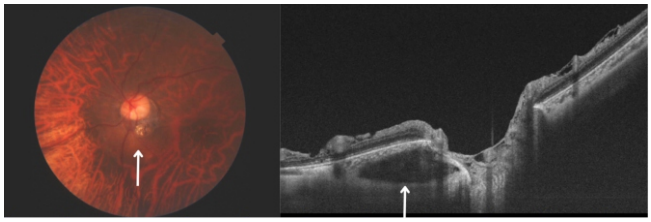

Figure 3: Color photo and OCT showing peripapillary intrachoroidal cavitation

Macular sink hole:
This term has been proposed for full thickness macular holes with myopic intrachoroidal cavitation with incarceration of retinal tissue. In one case report, the authors have shown area of RPE loss through which the retina is incarcerated directly from overlying macular hole to underlying intrachoroidal cavitation.[6]

Peripapillary sink hole:
Sinking of peripapillary retinal tissue into a sclerochoroidal cavity associated with retinal hole formation and posterior vitreous prolapse has been termed as myopic peripapillary sink hole.[7]

2. Classification and staging:

Newer classification and staging systems for myopic degenerative maculopathy (META-PM group) and myopic traction maculopathy (MTM) have been proposed.[8,9]

1.META-PM classification:

This group has classified degenerative lesions in myopic macula, based on fundus photographs into following categories:
Category 1: Tessellated fundus
Category 2: Diffuse atrophy
Category 3: Patchy atrophy (usually not centred at fovea)
Category 4: Macular atrophy (centred on the fovea, usually round, regular and appears around regressed fibrovascular membrane and enlarges with time)

Plus lesions: Lacquer cracks, myopic choroidal neovascular membrane (CNV) and Fuch's spot

Posterior staphyloma – macula involved or not (modified Curtin's classification)

2. MTM staging system:

A new staging system on myopic traction maculopathy (MTM) has been proposed by Barbara et al.[9] According to this MTM staging system, stages a to c evolve due to tangential traction, while stages 1 to 4 evolve due to perpendicular traction. Stage 1 is defined as inner/ inner-outer macular schisis, stage 2 is predominantly outer macular schisis, stage 3 is macular schisis and subfoveal detachment and stage 4 is macular detachment extending to the entire posterior pole with disappearance of schisis. Stage a (1a-4a) has no foveal defect, stage b (1b-4b) comprises of inner lamellar macular hole while stage c (1c-4c) comprises of full thickness macular hole. In addition, presence of outer lamellar macular hole is denoted by "O"(can occur in stage 2,3 or 4) and presence of epiretinal membrane is denoted by "+" (can occur in any stage). (Figure depicting the staging system is included in the article 'Challenges in surgery in myopic retinal detachment and myopic traction maculopathy')

3. Adjuvants in failed myopic macular holes:

Myopic macular hole associated retinal detachments (MHRD) are challenging to manage for every vitreoretinal surgeon. Various techniques have been tried to close myopic macular holes (MMH). Apart from the various internal limiting membrane (ILM) peeling techniques like circular or semicircular peel with temporal or superior hinged flaps, the Texas Taco technique,[10] pedunculated flaps, free flaps and anterior lens capsule have been used. Human lyophilized amniotic membrane graft (AMG) is being increasingly used to close persistent or recurrent myopic macular holes or macular holes with detachment.[11] Garcin et al. have suggested to prefer overlay versus inlay technique to place AMG, owing to its scaffold like action to promote centripetal migration of cells, prevention of foveal gliosis and avoiding RPE and neuroretinal injuries.[12] (Figure 4)

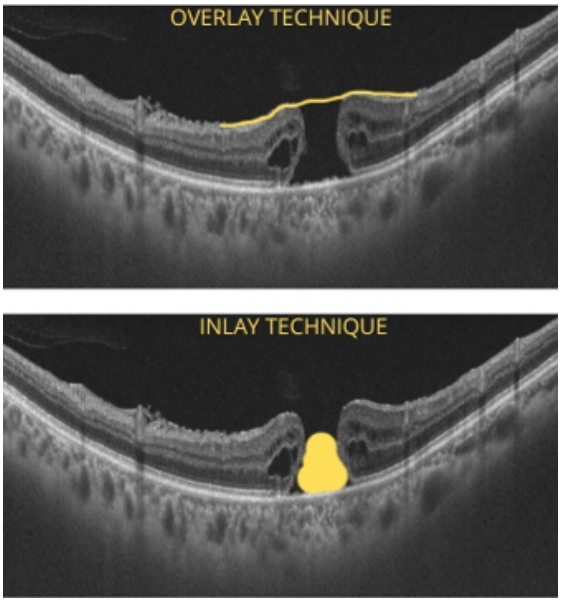

Figure 4: AMG – Representation of overlay and inlay techniques in macular hole repair.

Autologous blood has also been tried to close macular hole in MHRD. A study by Lai et al has shown 96% retinal reattachment rate with single surgery (vitrectomy with inverted ILM flap + autologous blood clot + gas) and 100% with second surgery.[13] In this technique, the authors peeled the ILM and filled the macular hole with the ILM. At this point, patient's blood drawn from the antecubital vein was injected gently over the macula to form a macular plug. The authors claim that the advantage with this technique

includes combination of two techniques leading to a higher chance of hole closure, faster wound healing and lesser postoperative prone positioning.

4. Posterior scleral reinforcement:

Posterior scleral reinforcement (PSR) has been tried using donor sclera and genipin-cross-linked sclera to strengthen the weak posterior sclera, reduce retinal stretching, contain the posterior staphyloma, and limit its progression. Peng et al. have shown 3 year results of posterior scleral reinforcement (PSR) using donor sclera as a U shaped scleral belt passing posteriorly from the insertion of superior to inferior recti.[14] Posterior sclera can be exposed by placing traction sutures for the inferior and lateral rectus muscles and then lifting the inferior oblique with a muscle hook. The scleral strip is then sequentially passed underneath the inferior oblique, lateral rectus and inferior rectus muscles and secured. This technique has shown to prevent progressive axial length elongation and stabilize the refractive error. Qi et al found better closure of macular holes and collapse of foveoschisis with PSR along with vitrectomy compared to vitrectomy alone in extreme myopes with axial length ≥ 30mm.[15]

Pan et al. have performed PSR with genipin cross-linked sclera and found 78.1% retinal reattachment rates.[16]
Genipin is a natural plant based crosslinking agent derived from Gardenia fruit with good biocompatibility. It has natural coloring properties due to the presence of iridoid blue based pigments. Animal studies have shown that genipin induced crosslinking helps to strengthen the biomechanical properties of sclera.[17]

5. Sutureless macular buckle

Macular buckle is another challenging surgery to support the posteriorly stretched sclera. A novel fiberoptic guided sutureless macular buckle designed using 7mm solid silicon tire and a sponge guided by a discarded chandelier illumination fiber cable has been described by Bedda et al which is fixed in place using sterile topical adhesive called Histoacryl Blue.[18]

6. Deep learning in myopia:

Finally, deep learning and artificial intelligence are also being applied to identify and segment lesions in pathological myopia based on fundus photos and also identify vision-threatening lesions like foveoschisis, macular hole, RD and CNV based on OCT images.[19,20]

To conclude, a lot of newer terminologies and therapies have evolved to treat myopia and its consequences, although we still have a long way to go.

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MANAGEMENT OF MYOPIC MACULOPATHY IN A SCHEMATIC

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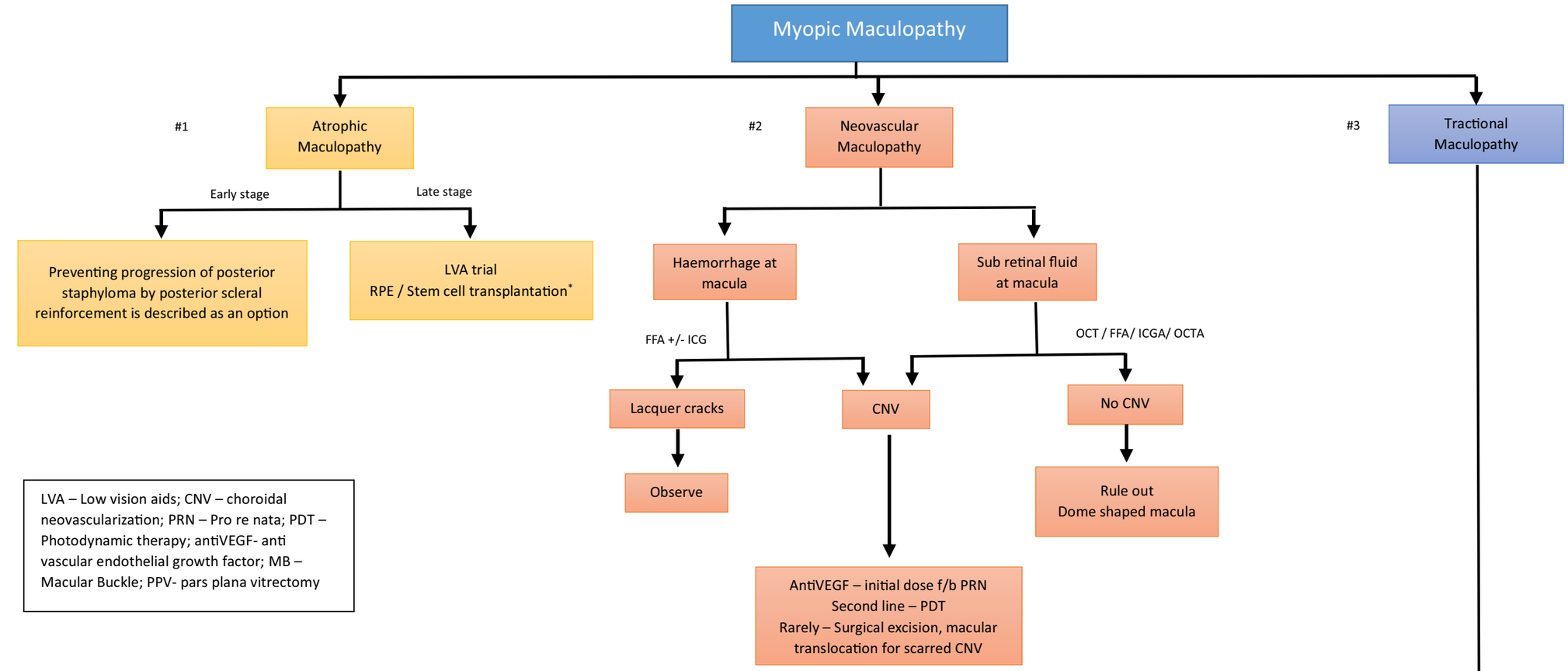

LVA – Low vision aids; CNV – choroidal neovascularization; PRN – Pro re nata; PDT – Photodynamic therapy; antiVEGF- anti vascular endothelial growth factor; MB – Macular Buckle; PPV- pars plana vitrectomy

MB or PPV is surgeon's choice. Only few surgeons consider MB during primary surgery. Few surgeons consider MB in cases with failed or recurrent cases. Few surgeons manage with PPV alone

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