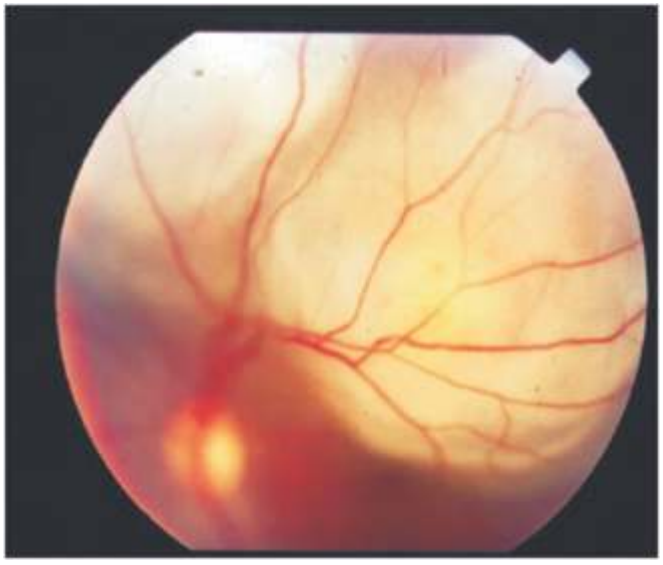

Figure 4 : Tubercular Sub retinal Abscess

neovascularisation and capillary non perfusion areas may be seen.

OCT : helps differentiate melanoma from CSR, wet ARMD, choroidal tumors. Contact sign (localized adhesion between chorio capillary RPE layer and overlying neurosensory retina is seen in tubercular granuloma.

Inflammatory cells show increased reflectivity in deeper layers (unique of TB)

B scan helps to rule out tumor from Tubercular sub retinal abscess

Investigations :

Hemogram : ESR , MT (can be false positive especially in our country)

CXR / CT scan

Quantiferon Gold test based on gamma interferon assay is useful / BActec MGIT 960 system

Histopathology and microbiological confirmation : PCR test from ocular specimen is diagnostic.

Treatment :

AKT should be initiated under physician supervision. Systemic steroids should be initiated for 4-6 weeks to protect tissues from delayed type of hypersensitivity. Steroids alone should never be used. In cases of MDR TB cases newer fluoroquinolones(levofloxacin/ Moxifloxacin) or aminoglycosides (Kanamycin/ Amikacin) may be initiated.

Ocular Syphilis :

Can mimic any of the other uveitic entities. It is the most common intraocular bacterial infection. Cases are remerging with the advent of AIDS .

1-2 % of HIV positive patients have ocular syphilis.

Clinically resemble other entities may present as chorioretinits , neuroretinitis,salt pepper retinopathy, optic neuritis, papilloedema, perineuritis. Sometimes resemble APMPPE. Unusual presentation is an acute necrotizing retinopathy. Ocular syphilis may be the first presentation of systemic disease and associated neurological abnormalities.

Lab Investigations :

They are a key to diagnosis. VDRL, Rapid plasma reagin test and TPHA are used. FTA-Abs is specific and can be used for initial diagnosis, is more reliable and has low false positivity. In HIV patients test may be negative. PCR more sensitive.

Treatment :

Ocular syphilis should be treated same as neurosyphilis. Long acting penicillin is drug of choice.

Viral Retinitis :

In any case of retinitis, viral infection is the first thing to be suspected. The common virus implicated are Herpes Zoster and Simplex, Cytomegalo virus,Chikangunya, Rubella.

Herpes is one of the most common infections to cause uveitis. It can be seen in both immunocompetent as well as immuno-compromised patients. Post uveitis may be seen as part of other systemic disease. Mostly the uveitis is acute and fulminant and may result in complications like RD and proliferative vitreoretinopathy. Clinical presentation depends on the immune status of the patient. It may present in 2 common forms :

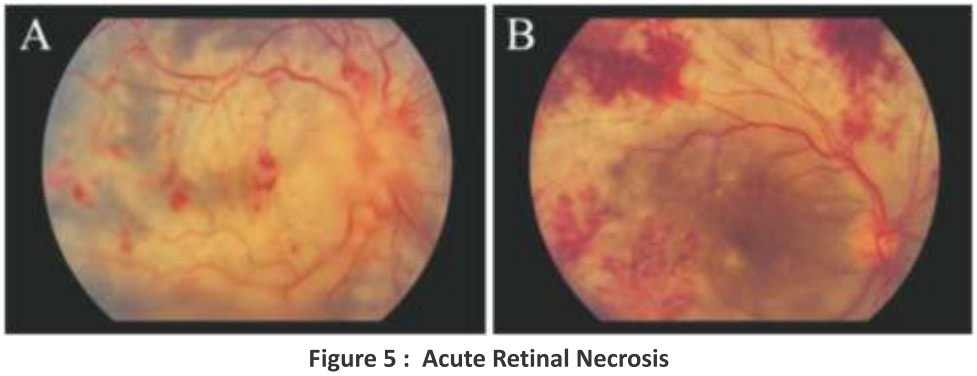

Figure 5 : Acute Retinal Necrosis

Acute Retinal Necrosis :

Is classical presentation of herpetic viruses. It includes the triad of

Vitritis

Arteritis

Periphelbitis

With associated peripheral retinal necrosis and is diagnostic of ARN. Posterior pole is spared in the early stages.

Progressive outer retinal necrosis :

It is frequently bilateral and seen more in immuno-compromised states. The early stages show a typical cracked mud appearance (Photo 6) necrotizing retinitis with outer retinal whitening with minmal vitritis at the posterior pole and sparing the retinal vessels in early stage. Rapid progression to RD and optic atrophy may be seen.

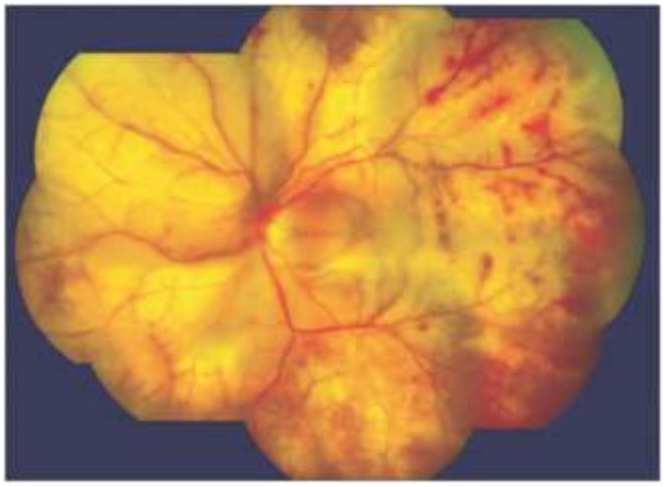

Figure 6 : Cracked Mud appearance (PORN)

ARN and PORN usually are manifestations of same virus with different immune status.

Viral posterior uveitis rarely may be seen as patchy , single or multiple retinitis patches which includes acute choriditis / chronic choroiditis in children or as necrotizing retinal vascultits in adults

CMV Retinitis :

Cytomegalo virus (CMV) is seen in mostly immuno-compromised or HIV or Post organ transplant patients. They show typical granular/ pizza pie appearance and/ or typical brush fire pattern (Photo 7) of spread along blood vessels.

Chikungunya retinitis :

Mimics herpetic or CMV retinitis

Diagnosis is based on history of chikungunya and by detection of specific antibodies to virus from serum or intraocular fluid. It is usually a self limiting disease. Acyclovir and Gancyclovir

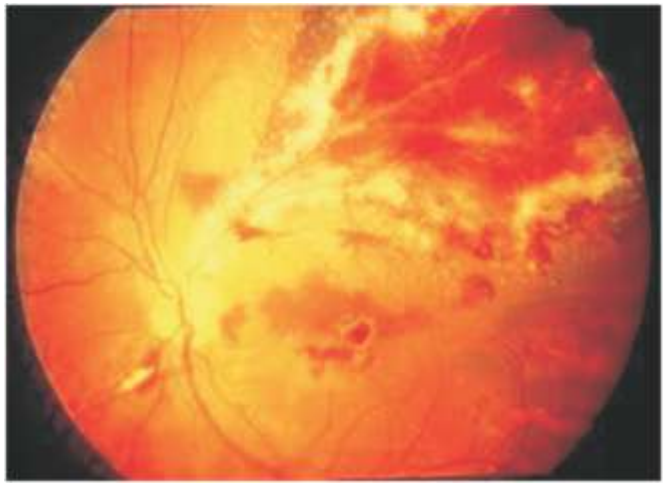

Figure 7 : CMV Retinitis

found to be useful.

Rubella Virus :

Can cause ocular lesions and have typical salt pepper appearance mimicking retinitis pigmentosa.

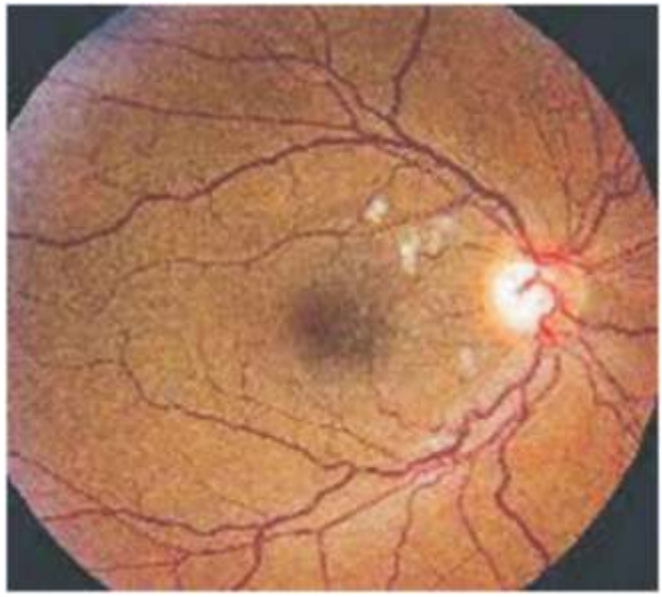

Figure 8 : Rubella Viral infection

Treatment :

Long term systemic antiviral drugs such as acyclovir or valaciclovir are treatment of choice in case of herpetic retinitis. Acyclovir is very effective against HSV and HZV. Treatment with acyclovir reduces infection with fellow eye from 70 to 13%. In resistant cases systemic treatment along with intraocular Foscarnet and Gancyclovir may be given.

For CMV retinitis systemic Gancyclovir is treatment of choice alongwith Intravitreal foscarnet or gancyclovir injection when macula is threatened.

Commonly used systemic antiviral agents include the

following which can be tapered slowly over months following resolution of the acute herpetic phase of ARN:

- Acyclovir (13 mg/kg/dose divided every 8 h IV for 7 days, followed by 800 mg five times daily orally for 34 months)
- Famciclovir (500 mg orally q8h)
- Valacyclovir (10002000 mg orally q8h for induction)
- Ganciclovir (500 mg IV q12h)
- Valganciclovir (900 mg twice daily orally for 3 weeks induction, then 450 mg twice daily po for maintenance)

Antiviral agents administered intravitreally can provide beneficial adjunctive therapy, especially if retinitis is threatening the macula or optic disc:

- Ganciclovir (200-2000 ug per 0.1 mL)
- Foscarnet (1.2-2.4 mg per 0.1 mL)

Steroids may have a beneficial therapeutic effects if initiated 24-48 hours after the start of antiviral therapy or once regression of retinal necrosis been demonstrated:

- Prednisolone (0.5-2.0 mg/kg/day orally for up to 6-8 weeks)

Aspirin may minimize vascular thrombosis and propagation of further retinal ischemia and necrosis. Topical steroid drops and cycloplegics have also been shown to be beneficial, depending upon the degree of anterior segment inflammation. Topical antiviral therapy has been shown not to be efficacious in treatment of ARN.

Prophylactic laser barrage adjacent to retinitis lesion are known to prevent retinal detachment although no randomized trial has confirmed it.

Fungal Uveitis :

Endogenous fungal infections are common in immuno-supressed patients and organ transplants. Infections occur from yeats and molds.

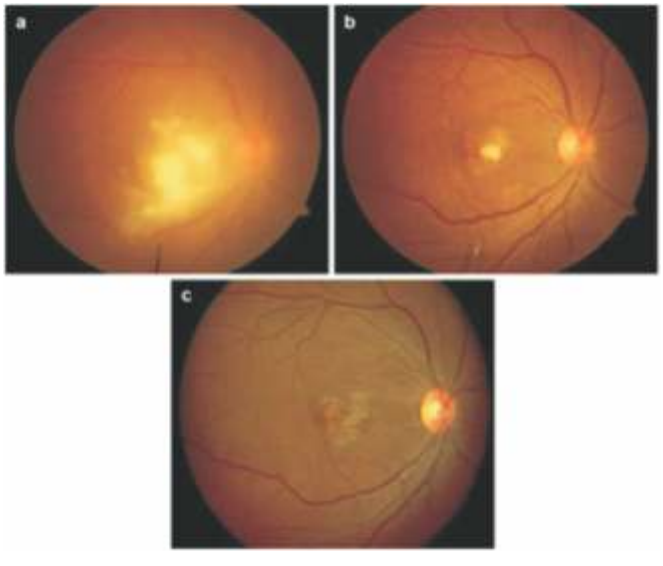

Figure 9 : Candidiasis

Candidiasis : most of the endogenous endophthalmitis are due to candidal infection. Choroidal infiltrates enlarge into small, subretinal white nodule aand penetrates through retina in a mushroom type pattern. Inflammatory reaction is mild to moderate unless vitreous seeding has occured. Vision is good in early stages. Vitreous sample from vitrectomy is better than vitreous tap.

Choroidal and sub retinal lesions can be treated with oral Fluconazole or oral voriconazole, retinal or vitreous infections require intravitreal amphotericin (5micr/0.1 ml) or voriconazole (50-100micr/0.1 ml).

Aspergillus Infections :

Appear usually as diffuse yellow chorioretinal plaque(Photo 10.) which involves macula and vitreous. It does not bud and produce large sub retinal nodules. Aspergillus may spread intravascularly and show poor visual outcomes. Infections are poorly tolerated than candidiasis. Rapid growth is characteristic. Nodules are denser and appear to be surrounded by pseudocapsule. Common in broncho-pulmonary aspergillosis and intravenous drug abuse.

Treatment is same as for candidial endophthalmitis

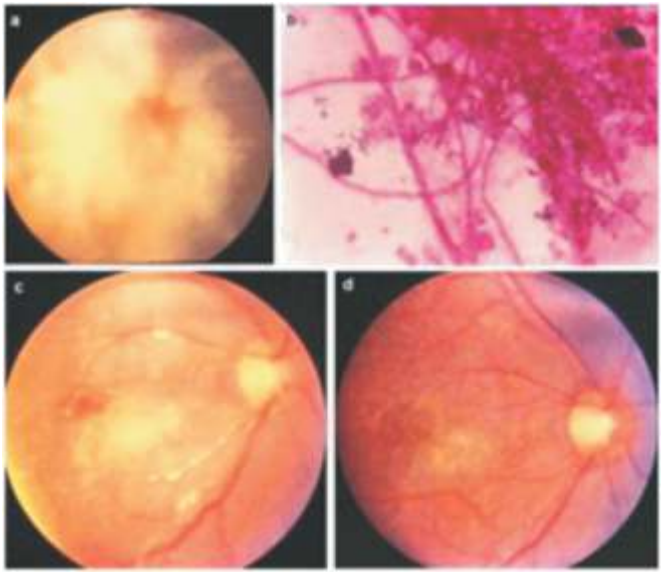

Figure 10 : Aspergillus infection

Non infectious Posterior Uveitis :

Mostly termed as White Dot Syndrome (WDS) based on clinical appearance and behavior.

FFA and ICG are usually diagnostic. Lab investigations are not routinely required for diagnosis but essential for therapy related side effects.

Many patients have prodromal, viral like illness followed by onset of ocular condition. Mostly they are self limiting. Pt complain of blurring of vision, photopsia , floaters scotoma or metamorphopsia.

Various WDS are :

APMPPE

MEWDS

Serpiginous Choroiditis

MFC

PIC

Presumed Ocular Histoplasmosis

Characteristics of lesions	Diagnosis
Subtle lesions	MEWDS
Prominent lesions	MFC
Placoid lesions	APMPPE if discrete & if they are coalesced, serpiginous choroidits consider ampigenous or serpigenous choroiditis
Discrete Lesions	MEWDS, DUSN, MFC, Birdshot Retinopathy

APMPPE :

It is an inflammatory retino choroidal disease. Patient present with sudden dimness of vision. Clinical examination shows multiple yellow white flat inflammatory lesions in RPE and at the choriocapillary level. New lesion are seen mostly in periphery. It has a self limiting course. Visual recovery usually good recurrence although rare may be seen within 6 months.

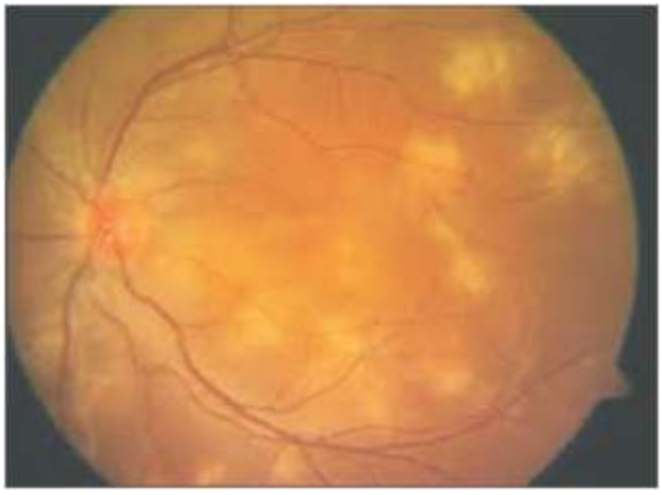

Figure 11 : APMPPE

MEWDS :

It is a rare disorder with unknown etiology. It may present with acute painless, unilateral dimness of vision. Viral prodrome may be seen in 50 % of cases. Lesions appear deep in outer retina or at the RPE level (Photo 6). Condition may present with optic neuritis vasculitis or vitritis. Dots and spots may be seen. Small dots in inner retina or at the level of RPE, larger spots are more external sub RPE level. Shows distinct morphology macular granularity , transient in nature , self limiting lack of sequel and rapid recovery is seen. Visual recovery is good within 3-9 weeks. No scarring seen usually. Occasionally enlarged blind spot may be seen. Rarely CNVM may occur.

Figure 12 : MEWDS

Serpigineous Choroidits (GHPC) :

Also known as Geographic helicoid peripapillary choroidopathy(Photo 13). It is a bilateral, asymmetric chronic recurrent progressive disorder. It presents as grey white sub retinal infiltrates. Inflammation is at the level of RPE chorio capillary complex. The clinical course is progressive with multiple recurrences leading to significant visual loss when macula gets involved. Patients notice photopsia, scotomata. Anterior segment is usually quiet. Active lesions are seen at border of active lesions. Appear in interlocking polygonal pattern that spreads out in periphery from optic nerve. Macular involvement is common. In some cases aqueous or vitreous flare may be seen.

Clinically classified as

Peripapillary

Macular

Amphigenous types

Amphigenous variety mimics placoid lesions of APMPPE and coalesced lesions of GHPC. Persistent placoid maculopathy is a

resistant form and resembles macular GHPC. Majority of eyes develop CNVM resulting in central vision loss. Vitreous and aqueous flare may be seen.

Tubercular etiology should be ruled out.

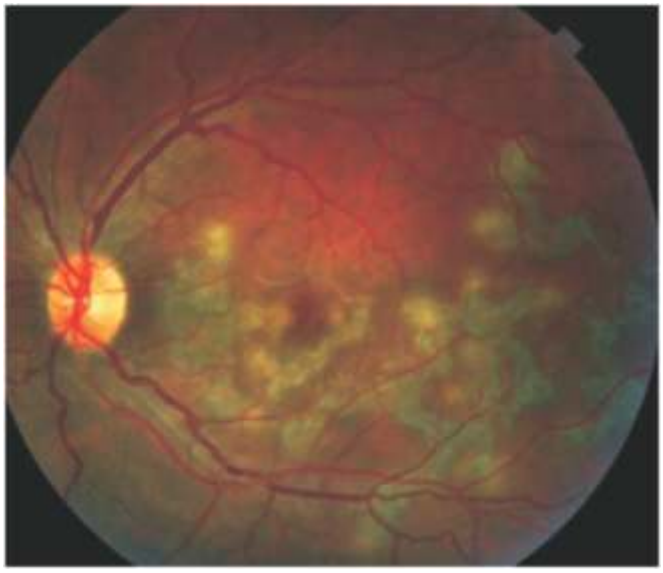

Figure 13 : Serpeginous Choroiditis

Multifocal Choroiditis (MFC) :

Seen bilaterally in 66-97% of cases. Optic disc edema edema, peripapillary scarring and prominent linear CR streaks are also seen. Patients may present with CME and CNVM.

Puntate Inner Choroidopathy (PIC) :

Occurs in myopic women. Idiopathic inflammatory multifocal chorioretinopathy presents with acute bilateral loss of vision photopsia and scotoma. Anterior segment is quiet and vitreous clear. Treatment advised only for if there is presence of CNVM or sub retinal fibrosis. Prognosis is excellent except in presence of CNVM.

Subretinal Fibrosis(SFU) :

Progressive subretinal fibrosis with multifocal lesions of RPE and choroid can be seen in associatin with PIC and recurrent MFC. Steroids benefit in early stages , progressive fibrotic sub retinal lesions leads to severe and permanent visua loss. Fibrosis occurs at the site of inflammation and turbid SRF lies over these lesions. It is also seen in serpigenous choroiditis SLE asso CSR and onchocerciasis. Infliximab has given good results.

Presumed Ocular Histoplasmosis Syndrome (POHS) :

Usually seen in endemic areas of histoplasma capsulatum. Presents asymptomatically or with central scotoma. Histoplasma has never been isolated from choroid and hence the name. CNVM managed by argon laser for extrafoveal and krypton laser for juxtafoveal lesions.

An association of HLA-DR15/HLA DQ6 and development of CNVM in POHS has been seen.

Birdshot Chorioretinopathy :

More than 90% of these are HLA -29 positive. Associated non granulomatous seen in 25% cases. Birdshot term is used because pattern of lesions resembles shotgun scatter of birdshot.

Acute retinal pigment epithelitis (ARPE-Krills's disease)

Usually presents with unilateral blurred visionand metamorphopsia in young adults without significant prodromal flu like illness. Round macular lesions are hall mark of the diease and manifested by transient and subtle RPE alterations. Discrete clusters of small, hyperpigmented spots, dark grey spots at the RPE level surrounded by yellow white haloor area of macular depigmentation affects vision, grey dots and halo fade with resolutionand become undetectable clinically. Vitritis is rare, VEP and ERG are normal, EOG may be abnormal. FFA to t rule out CSR and no treatment required.

Treatment of non infectious PU :

First of all the diagnosis of non infectious uveitis should be confirmed.

Systemic steroids are the main stay of treatment. In vision threatening cases iv methylprednisolone pulse therapy 1 gm per day for 3 days followed by oral steroids should be given under physician care. In recurrent cases/ intolerant to steroids Immuno supressives may be added with/ without systemic steroids. The commonly used ones include antimetabolites like azathioprine, methotrexate and T cell suppressors like cyclosporine and tacrolimus, cyclophosphamide and chlorambucil may be given all under physician guidance.

Local drug delivery like Intravitreal triamcinolone and dexamethasone may be given for refractory CME. Inflammatory CNVM treated with intravitreal avastin with steroid.

Masquerade Syndromes :

These are non uveitic conditions that mimic and present like uveitis. Have to be differentiated neoplastic diseases from uveitis.

Lymphoma and secondary malignancies resemble viral retinitis. Lymphoma should be rules out before initiating therapy when atypical presentation of viral retinitis intermediate uveitis are seen. The common malignancies in children are leukemias and retinoblastoma, in adults melanoma and metastasis.

Choroidal melanoma mimics choroidal abscess.

B scan is an important tool to differentiate:-

Table 1. Comparative Characteristics of clinical presentations of White dot syndrome

	APMPPE	BIRSHOT	PIC	MEWDS	MFC	GHPC	POHS
Age	Young (20-40)	Middle aged (40-60)	Middle aged (Myopes)	Young myopes (20-40)	Myopic (20-40)	Variable (30-60)	Middle aged
Sex	M=F	F>M	F>M	F>M	F>M	M>F	M=F
Laterality	Bilateral asymmetric	Bilateral	Bilateral	Unilateral	Bilateral asymmetric	Bilateral asymmetric	Bilateral
Viral illness	+	-	+	+	+/-	-	+/-
Onset	Abrupt	Insidious	Abrupt	Abrupt	Insidious	Variable	Abrupt
Duration	Weeks months	Chronic	Weeks months	Weeks months	Chronic	Chronic	Chronic
Recurrence	Rare	Recurrent	Recurrent	Rare	Recurrent	Recurrent	Rare
Vitritis	Mild	Moderate with disc edema, CME	Absent	Mild	Moderate and anterior uveitis	Mild	Absent/Mild
ERG/EOG HLA	Abnormal EOG B7, DR2	Abnormal ERG A29	Abnormal -	Abnormal ERG -	Abnormal ERG -	Normal B7	Abnormal HLA-DR2 HLA-B7
Fundus Active	Multifocal, flat grey-white placoid lesions primarily posterior pole at the level of RPE	Multiple depigmented yellow white patches scattered throughout fundus in post equatorial region these radiate from optic nerve and follow large choriodal vessels	Multiple discrete yellow round lesions (50-300 microns) at the level of RPE and inner choroid concentrated at the posterior	Multiple small (100-200micr) slightly indistinct yellow white spots distributed over posterior fundus especially at perifoveal and peripapillary regions at the level of RPE	Multiple yellow-grey lesions at the level of choroid and RPE mid periphery (50-100micr)	Macular, peripapillary or amphigenous Irregular grey white or cream yellow sub retinal infiltrates at the level of choriocapillaries and RPE-snake like pattern	Peripapillary atrophy, atrophic chorioretinal lesions, cnv, punched out yellow lesions, linear streaks-mid periphery
Fundus healed	RPE clumping and hyper pigmentation	Lesions have hyperpigmented edge but frequently hypopigmented at centre		Heals rarely by scarring	Punched out atrophic scars that develop pigmentation over time	Heals from centre towards periphery	Scars
Pathogensis	DTH	Auto immune		? hormonal		Idopathic/ infective	

Melanoma have high surface reflectivity and low to moderate internal reflectivity or acoustic while Metastases have high surface reflectivity and moderate to high internal reflectivity, acoustic hallowing and choroidal excavation not seen. Another important tool is vitreous/ retinal biopsy.

AIDS related posterior uveitis :

Patient with AIDS are susceptible to infections like toxoplasma, syphilis and tuberculosis. Can have atypical ocular manifestations.

Table 2. Comparative characteristics of FFA and ICG features of White Dot Syndrome

	APMPPE	BIRSHOT	PIC	MEWDS	MFC	GHPC	POHS
FFA active	Early hypo fluroresence and staining in late phase	Mild hyper fluorescence and staining in late phase	Early hypofluoresc ence and late hyperfluoresc ence	Early patch hyperfluoresc- with late deep staining or RPE and peripapillary area. Leakage from optic disc an retinal capillaries. Early fluorescence wreath like pattern. Choroidal background fluorescence between lesions is normal	Early hypofluoresc ence and late hyper fluorsecence	Early hypofluoresc ence and late hyper fluorescence. Choroidal vessels easily seen	Early hypofluoresc e and late hyper fluorescence confirms CNVM
FFA inactive	Window defects	Window defects	Window defects	Window defects	Window defects Early hyperfluoresc ence and late	Window defects	Window defects ICG
ICG active	Marked choroidal hyperfluorescen in both early and late phase Large choroidal vessels seen		CNVM reveals hyperfluorescen	Multiple hypofluorescent spots in posterior pole and hyper fluorescence around optic nerve head especially in patients with enlarged blind spots	Hypofluorece CNVM reveals hyper Fluorescence		
ICG active	Choroidal hypoflurorecence			Hypofluoresc- ent spots persists until patient recovers	Hypofluroresc ent		
ERG/VEP		Unilateral negative ERG		Markedly reduced a wave and receptor potential anmplitudes suggesting primary RPE involvement			
Visual fields				Enlargement of blind spot. No correlation of field defects with lesions.			

To summarize :

Posterior uveitis have characteristic clinical features and diagnosis is mostly clinical. Infective should be differentiated from non infective as the management of both of them are diametrically opposite. Infective cases need to be managed with specific anti infective therapy and steroids. Empirical use of systemic steroids and immunosuppressives shoud be avoided in all cases. Ancillary tests like FFA, ICG, OCT and USG help to diagnose the etiology. Intraocular fluid testing with PCR and antibodies is also useful. All cases should be followed up even after resolution of the disease to look for any sequlae like CNVM, hemorrhage or retinal breaks. Macular or optic nerve damage can cause irreversible damage. Hence proper diagnosis is a must.

Hermann Ludwig Ferdinand von Helmholtz *(1821-1894) introduced the ophthalmoscope on December 6, 1850 and revolutionized ophthalmology. The ophthalmoscope, the most important invention for ophthalmologist clinicians, is an instrument that allows the ophthalmologist to look inside a person's eye and see the details of the living retina. Ophthalmoscopes allow physicians to diagnose eye diseases and prevent blindness.*

Helmholtz *created the ophthalmoscope during the preparation of an experiment to demonstrate the law of conservation of energy. For the experiment, Helmholtz constructed a crude instrument made of cardboard, glue and microscope glass plates. Helmholtz's instrument showed that light entering the pupil is reflected back to its source. Furthermore, the light follows the same path out of the eye as it took entering the pupil. Using his makeshift ophthalmoscope, Helmholtz could place his eye in the path of the rays of light entering and leaving the patient's eye, thereby allowing the patient's retina to be seen.*

Retinal Detachment and its Management

Khyati Singhai[1], Arun Bhargava[2]

Retinal detachment (RD) is a separation of the neurosensory retina from the retinal pigment epithelium with the accumulation of fluid in the potential space between them.

Types of RD :

A) Rhegmatogenous - associated with break(s)

B) Tractional associated with traction, without breaks

C) Exudative - due to fluid exudation which may resolve spontaneously For understanding the basic principles underlying the surgery, further discussion will be limited to rhegmatogenous retinal detachments.

PATHOGENESIS :

There are no anatomic junctions between the NSR and RPE, but weak mechanical forces are responsible for their adhesion. These include the active and passive forces of choroidal oncotic pressure and the RPE pump, creating a pressure gradient between the two. The interpho- toreceptor matrix, consisting of a variety of molecules including the glycosaminoglycans chondroitin sulphate and hyaluronic acid, and the RPE microvilli, enveloping the photoreceptor outer segments also contribute to these adhesive mechanisms. The metabolic state and oxygenation of the RPE affects this overall adhesion. Any RD is by definition an accumulation of subretinal fluid between NSR and RPE. The two prerequisites for the development of Rhegmatogenous RD are liquefaction of the vitreous; an RRD will not occur without first some degree of liquefaction of the formed vitreous gel that precedes posterior vitreous detachment (PVD), and supplies the low viscosity fluid that is able to flow through retinal breaks, and a retinal break through which fluid gains access into the subretinal space.

RISK FACTORS :

1. Vitreous liquefaction

2. Partial / complete posterior vitreous detachment

3. Retinal tears at sites of vitreoretinal adhesions

Predisposing Conditions :

1. Myopia
2. Aphakia
3. Trauma
4. Lattice degeneration
5. Complicated cataract surgery
6. Proliferative retinopathies - Diabetes, BRVO, sickle cell, ROP
7. Infections
8. The most common hereditary conditions associated with RD are axial myo- pia and lattice degeneration.

- Familial vitreoretinal disorders
- Wagner Jansen Stickler vitreoretinal dystrophy
- Axial myopia
- Lattice degeneration
- Goldmann Favre vitreoretinal degeneration
- Xl RD inked juvenile retinoschisis
- Familial exudative vitreoretinopathy
- Snowflake vitreoretinal degeneration

RISK TO FELLOW EYE :

The fellow eye in patients with RRD is at a higher risk. The Scottish Retinal Detachment Study found a prevalence of bilateral RRD of 7%. Interestingly, in the same cohort, retinal tears were found in 8% of fellow eyes in patients with primary RRD, which underscores the need for a thorough dilated fundal examination of the fellow eye.

PREVENTION : Prophylactic treatment to fellow eye following RRD. Although RRD may develop in a fellow eye from preexisting retinal lesions, most subsequent RRDs (at least 50% and possibly as high as 80%90%) in the fellow eye will occur from ophthalmoscopically normal areas of retina, therefore, prophylactic treatment with either laser or cryotherapy to funduscopically abnormal areas does not completely reduce the incidence of fellow-eye RRD.

CLINICAL FEATURES :

Patients with RRD may present with floaters, photopsia and “curtain” defect that obscures part of the visual field. Visual acuity (VA) ranges from excellent to poor, depending on whether the macula is still attached. In patients with macula-off RRD, vision usually is decreased. If the area of detachment is large, an afferent pupillary defect may be present.

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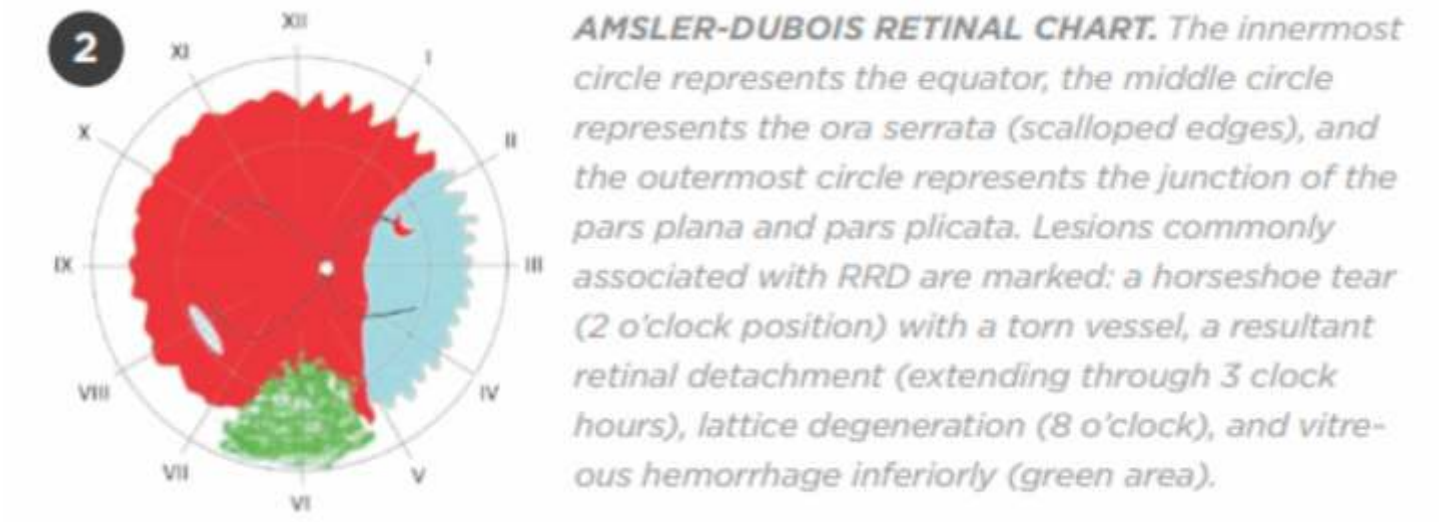
Intraocular pressure (IOP) can be low or high. Low IOP results from increased outflow of intraocular fluid through the subretinal space and peripapillary connective tissue, particularly if the optic disc border is involved. High IOP may occur with chronic RRD, in which photoreceptor outer segments transgress into the anterior chamber and trabecular meshwork, impeding aqueous outflow. This is also known as Schwartz-Matsuo syndrome. Other features of chronic RRD may include a pigmented demarcation line at the detachment border, intraretinal macrocysts, atrophic thinned retina, subretinal white precipitates, and signs of proliferative vitreoretinopathy(PVR) such as fixed retinal folds. Assessment of RRD requires a thorough 360-degree fundus examination. When visualization of the fundus is poor, as in patients with dense cataract or vitreous hemorrhage, an ultrasound B-scan may be useful.

EXAMINATION :

Binocular indirect ophthalmoscopy (BIO) of the fundus. BIO with a lens of 20 or 28 D allows visualization of the peripheral retina. For some eyes, scleral depression during indirect ophthalmoscopy or contact fundus lens examination using the slit lamp (e.g., Goldmann 3-mirror lens) may help view smaller peripheral retinal breaks.

This should include the following:

1. Identify the extent of detachment. The detached area will appear opaque and corrugated, with undulating retinal folds during eye movement. The borders of the detached tissue usually are convex, and the subretinal fluid is clear and nonshifting. Other features that may accompany RRD include a positive Shafer's sign (pigment in the anterior vitreous), vitreous hemorrhage, and lower IOP than in the fellow eye.
2. Find all retinal breaks, which will help guide the surgical approach. It is important to note the size, number, and location of each break. Lincoff rules are useful for identifying the precise location of the retinal break in cases of primary RRD. If there are multiple breaks, the highest retinal hole is considered the primary hole. (See "Lincoff Rules.") The location of retinal detachment plays a major role in management and prognosis.
3. Determine whether the RRD is macula-on or macula-off (Fig. 1). Although visual prognosis is much better for macula-on RRD that spares the fovea, urgent intervention is still needed.
4. Check for associated features. Retinal lesions that predispose to retinal breaks, such as lattice degeneration, should be identified. Also look for features that might affect management and prognosis, such as coexisting vitreous hemorrhage and PVR.
5. Document the findings on an Amsler-Dubois chart or in the electronic medical record, using color codes and symbols to represent retinal lesions.



AMSLER-DUBOIS RETINAL CHART. *The innermost circle represents the equator, the middle circle represents the ora serrata (scalloped edges), and the outermost circle represents the junction of the pars plana and pars plicata. Lesions commonly associated with RRD are marked: a horseshoe tear (2 o'clock position) with a torn vessel, a resultant retinal detachment (extending through 3 clock hours), lattice degeneration (8 o'clock), and vitreous hemorrhage inferiorly (green area).*

DIAGNOSIS :

Finding retinal break (RB) causing Rhegmatogenous retinal detachment (RRD) is a critical step before vitreoretinal surgery (VRS). The detection of the RB is the first stage of the management of RRD. Possible localizations of RBs in the eyes with RRD are supero temporal (60%), superonasal (15%), inferotemporal (15%) and inferonasal (10%) quadrants. Modified Lincoff's rules are rules to detection of primary RBs based on the configuration of sub retinal fluid (SRF) and the localisation of RRD in retrospective analysis of 1,000 cases of RD. Modified Lincoff's rules include followings :

I. Inferior RD with equal SRF levels on both sides of the OD: A primary RB will be at inferior, at 6 o'clock position (Figure 1A).

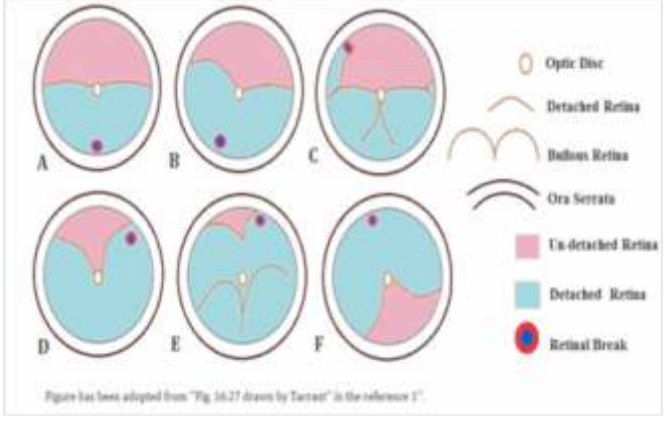
II. A shallow inferior RD in which the SRF is slightly higher on the temporal side: A primary RB will be located inferiorly on that side (Figure 1B). On the other words, in "Lateral" RRD that means inferior swallow RRD with SRF higher on one side of the optic disc (OD) (superotemporal or superonasal RRD), primary RB is within 1.5 clock hours of the higher border of RRD.

III. A bullous inferior RRD: A primary RB will usually exist above the horizontal meridian (Figure 1C).

IV. A diffuse RRD with a superior attached wedge retina from OD to oraserrata: Because of the primary RB located in the upper nasal quadrant, the SRF will revolve around the OD and then rise on the temporal side until it is level with the primary RB (Figure 1D).

V. A subtotal RRD with a superior wedge of the attached retina:A primary RB will locate in the periphery nearest its highest border (Figure 1E).

VI. A diffuse RRD with an inferior attached wedge retina from OD to oraserrata: When the SRF crosses the vertical midline above; the primary break is near to 12o'clock, the lower edge of the RRD corresponding to the side of the break (Figure 1F).



INVESTIGATIONS :

ULTRASONOGRAPHY - If the fundus view is obscured, dynamic B-scan ultrasonography is helpful to confirm RRD and determine the status of macular involvement, presence of posterior vitreous detachment, location of retinal break (occasionally), and chronicity of RRD (mobile or fixed), AXL, choroidal detachment, posterior staphyloma, to violate ICM (Inter Calary Membrane) break of coloboma. Typical ultrasound findings for RRD include high reflectivity, a high spike on the A-scan, a membrane within the vitreous cavity, and mobility during eye movements. Posterior vitreous detachment is characterized by a posterior hyaloid face, low reflectivity, a low spike on the A-scan, and a high degree of mobility during eye movements.

OCT - PVD, Macular edema, Macular Hole, Macular cyst. Optical coherence tomography (OCT) is not routinely required to assess macular status, as this can be established by BCVA and clinical examination, with preoperative BCVA determining potential postoperative BCVA. However, OCT and ultrasound imaging may be useful in assessing the presence of PVD, as this can influence the surgical approach.

Surgical management of RRD :

The main target of RRD management is to achieve retinal reattachment. Although the benefit for treatment of asymptomatic (chronic) RRD remains unclear, symptomatic RRD is a clear indication for surgery. On presentation, RRD is usually divided into 'macula-on' where the foveal centre is not involved, and 'macula-off' where the fovea is detached. People with macula-on RRD typically have good initial best-corrected visual acuity (BCVA) and a better visual prognosis with successful surgery. Macula-off RRDs have lower initial BCVA and worse visual prognosis even with successful reattachment of the retina. However, in macula-off RRDs postoperative BCVA is better in patients with 13 days of visual loss compared with 46 days, and hence these patients also need to be treated as a matter of urgency. Indeed it is likely that prognosis reduces linearly for every day that the macula remains detached.

SURGICAL APPROACHES :

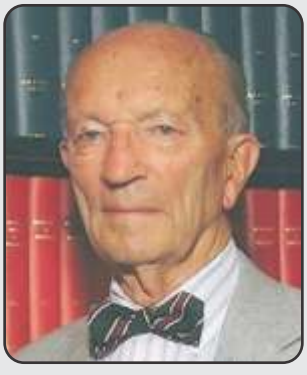
There are three main current options for the management of RRD, namely pneumoretinopexy (PnR), SB, PPV , SB+ PPV. The choice of surgery will depend on various factors, including number, location and size of retinal breaks present and the presence of any PVR; the ability of the patient to posture in order to allow optimum positioning of intraocular tamponade agents; lens status and surgeon's experience and preference. PVR remains the most predictive variable for failure of primary surgery with success rates dropping from 90% to 68% if PVR is present preoperatively. It is characterised by cellular proliferation affecting both surfaces of the detached retina and the vitreous base, resulting in the formation of contractile periretinal membranes. PVR can also occur following surgery and is one of the chief causes of failure, along with new break formation as well as missed retinal breaks. Although several studies have identified a number of clinical risk factors for PVR developing and causing primary failure, including vitreous haemorrhage, PVR at presentation, aphakia, uveitis, RRD associated with trauma, duration of detachment and presence of choroidal detachment preoperatively.

CONCLUSION :

Although RRD is now routinely treated, the success rate remains stubbornly less than 100%, and typically around 85% with most large modern series. Potentially, optimising retinal

PROCEDURE	SUCCESS RATE	VISUAL OUTCOMES	LIMITATIONS
PnR (PNEUMO RETINOPEXY)	73 81%	more than SB & PPV	Raised IOP, Endophthalmtis
SB(SCLERAL BUCKLE)	84%	more than PPV	Scleral perforation, retinal incarceration & CD
PPV(PARS PLANA VITRECTOMY)	89%	more than SB or PnR	Iatrogenic retinal tears, lens touch & cataract formation
SB+PPV (COMBINED)	93.5%	More than all 3 , visual acuity >0.4 LogMAR	Higher invasity of surgical procedure, iatrogenic breaks, post surgical macular edema.

break detection and effective, rapid onset retinopexy, obviating the need for tamponade could help improve primary success rates. PVR remains a common cause of failure and new strategies to prevent and treat PVR are required. The optimum method to repair detached retinas to allow maximal visual recovery, especially in macula-involving cases, is gradually becoming more defined, but surgeon experience and preference will still remain major factors affecting technique choice.



Charles Louis Schepens *(March 13, 1912 March 28, 2006) was an influential Belgian (later American) ophthalmologist, regarded by many in the profession as "the father of modern retinal surgery".*

Schepens *invented the binocular indirect ophthalmoscope (BIO), which is routinely used to look at the retina. His original BIO is now in the collection of the Smithsonian Institution. It has been reported that Schepens assembled the prototype for his BIO from metal scraps collected from the streets of London during the German blitz. He was also a pioneer of surgical techniques such as scleral buckling for the repair of retinal detachments. The use of these techniques has raised the success of retinal reattachment surgery from 40% to 90%. During his career, Schepens wrote four books and over 340 research papers*

Microincision Vitrectomy Surgery - A Review

Deepanshu Agrawal

Introduction :

Dr Robert Machemar is known as the father of vitreoretinal surgery. He introduced Pars plana vitrectomy (PPV) in 1971 where he had used a single port, 17-gauge (1.5mm) system with a drill tip connected to a micromotor.[1] This system allowed aspiration, grasping and cutting of the vitreous. Although rudimentary by today's standards, this tremendous innovation allowed the treatment of previously blinding diseases, such as non-clearing vitreous hemorrhages and retinal detachments with proliferative vitreoretinopathy. Following this innovation, Conor O'Malley and Ralph Heinz, for the first time, introduced a three-port pars plana vitrectomy system where in they had used 20-gauge (0.9mm) instrumentation and it became the standard of care.[2] This new approach required closure of sclerotomy sites with sutures after removal of the ports and proved to be successful.

In 2002, Gildo Fujii et al. developed a 25-gauge (0.55mm) transconjunctival vitrectomy system.[3] A major disadvantage of the first 25-gauge instruments was the flexible instruments, which made surgery procedures more challenging. In 2005, Claus Eckardt introduced 23-gauge (0.72mm) sutureless vitrectomy instruments that, compared to the ones from Fujii et al., offer more stability due to their larger physical size.[4] In the last decades numerous technical improvements of 25-gauge instrumentation were implemented, which enhanced instrument rigidity, increased vitreous cutting rate and brightness of light sources. The trend towards yet smaller gauge instruments continued with the development of a 27G sutureless vitrectomy system by Oshima in 2010.[5] The overall advantages of smaller vitrectomy systems are less risk for intra and post-operative complications, increased post-surgical patient comfort and faster recovery time due to less traumatic intraocular access through smaller sclerotomies.

Small gauge vitrectomy, with its smaller instrumentation intended to be transconjunctival, self-sealing, and sutureless, has theoretical advantages including decreased ocular trauma and inflammation, decreased corneal astigmatism, reduced operating times, faster post-operative recovery, increased patient comfort, reduced conjunctival scarring, and conjunctival preservation, especially in patients with prior or pending glaucoma surgery.[6-8] In addition, smaller gauge vitrectomy instruments are better suited to the narrower spaces of pediatric eyes. However, miniaturization of instruments limits instrument diameter and lumen, with counterproductive effects on instrument flexibility, efficiency, and performance. Advances in wound construction, instrumentation, fluidics, cutter technology, illumination, and wide-angle viewing systems (WAVS) have overcome the handicaps of smaller gauge instrument size and are discussed in detail as follows.

Trocar / Cannula System :

Standard 20G vitrectomy surgery requires conjunctival incisions and sclerotomies of 0.9mm diameter. Smaller gauge vitrectomy using transconjunctival trocar / cannula systems, have reduced the scleral incision diameter to 0.7mm for 23G, 0.5 mm for 25G, and 0.4mm for 27G [Figure 1]. The trocar / cannula system theoretically creates less traction on the vitreous base during instrument entry and exit. The once only placement of the cannulas maintains the alignment between the conjunctiva and sclera and is less traumatic to wound borders than the repeated insertion and withdrawal of instruments through a 20G sclerotomy. It also increases the chances of self-sealing sclerotomy closure and minimize the risk of suture-related inflammatory reaction, or subsequent atrophy and thinning over the sclerotomy site. Cannulas also allow for interchangeability of instrument and infusion sites, allowing for improved access.[9] Placing the sclerotomies closer to the horizontal meridian reduces the need to rotate instruments significantly for peripheral and superior access and avoids displacement of the infusion as the eye is rotated inferiorly.[10]

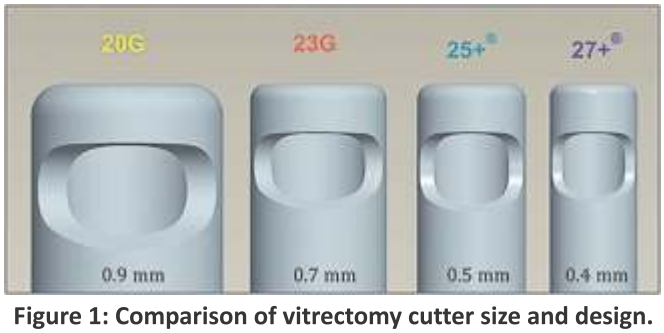


Figure 1: Comparison of vitrectomy cutter size and design. The 27G cutter is less than 50% the size of the 20G cutter.

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Wound Construction :

Wound construction in smaller gauge vitrectomy systems is a critical step and affects whether the sclerotomy seals well at the end of surgery. Early incisions were straight (perpendicular to the sclera) in 25G systems, as better self sealing was expected with the smaller diameter incisions.[3] This was changed to an angled (oblique) scleral incision after studies showed better wound closure and reduced risk of hypotony compared with straight incisions [Figure 2].[11,12] One- and two-step techniques of angled wound construction have been described. The original two-step technique for 23G vitrectomy, as described by Eckardt, involves displacement of the conjunctiva and stabilization of the eye with a pressure plate, followed by use of a sharp angled MVR blade to create the initial slit opening in the sclera followed by insertion of the blunt trocar, onto which the cannula is mounted.[4] This technique allows more consistent wound creation, but it may sometimes cause difficulty in finding the initial point of trocar insertion. The modern one-step technique involves entry by a sharp trocar with a mounted cannula. Cannulas are quick to insert and easily removed from the trocars without need for a second instrument, but it may be necessary to apply a slightly higher pressure to insert the microcannula at an oblique angle, which can cause problems in eyes with recent corneal or scleral wounds.[10] In general, longer (more oblique) the intrascleral path, the better the wound apposition. In Zorro's incision, the blade is inserted obliquely at an angle of 10 to 15 degrees and enters the vitreous without straightening.[13] Pollack improved on this by suggesting a biplanar incision, where the trocar is inserted at a 5-degree angle to the sclera until 50% scleral depth, and then raised to a 30-degree angle to the sclera. Trocar entry at 30 degrees produces a longer tunnel length of 1.414 mm, compared with a tunnel length of 1.154mm produced by trocar entry at 45 degrees. The 30% increase in tunnel length results in more watertight closure.[13] Alternatively, the trajectory may also be made very tangential to the sclera at about 5 degrees and then tilted up to a more perpendicular angle after the cut is made through the sclera in order to avoid impaling the retina. Conjunctival and scleral vessels should be avoided where possible, to reduce post-operative subconjunctival hemorrhage.

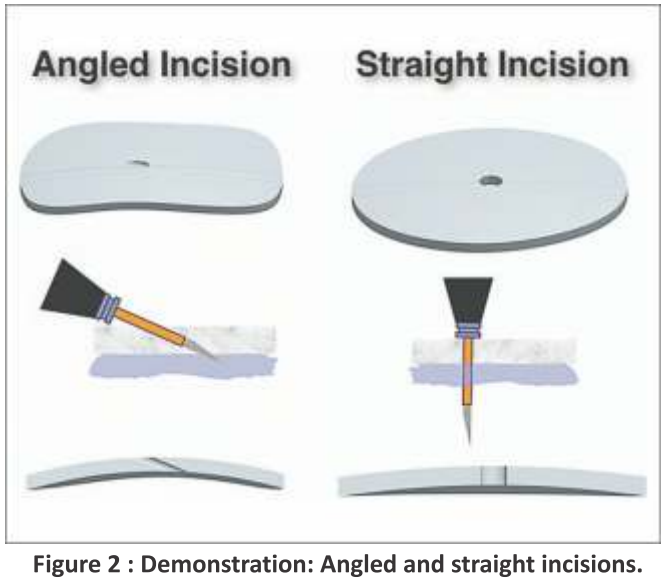

Figure 2 : Demonstration: Angled and straight incisions.

Valved Cannula System :

Newer valved cannula designs remove the need for plugs and consist of a cap-like silicone membrane mounted onto the cannulas (DORC, Dutch Ophthalmic Research Corporation, Zuidland, the Netherlands), or built into the cannula head (Alcon, Fort Worth, Texas, US). They help maintain a closed system, provide more stable intraocular pressure (IOP) control during instrument exchange, and reduce the amount of infusion. High infusion flow can cause turbulence when working with perfluorocarbon liquids, direct mechanical trauma to the retina, ballooning of the retina if the infusion is directed toward a retina break, or increased dehydration if fluid-air exchange has already been performed. Valved cannulas address the problem of high flow from the infusion through open cannulas during instrument exchange due to IOP compensation features, which can lead to a “fountain effect” at the open cannulas and dislodge plugs, or cause vitreous or retinal incarceration at the sclerotomies.[14] However, valved cannulas can lead to increased friction between the instrument and the valve, and difficult entry for soft or flexible tip instruments, such as the soft tip backflush cannula, or the diamond dusted membrane scraper (DDMS). Entry of such instruments requires straight entry at the centre of the valve aligned with the cannula direction, or a second instrument to act as a glider displacing the valve leaflet.[15]

Cannula Removal and Wound Closure :

The self-sealing ability of a sclerotomy wound is affected by wound architecture, scleral tunnel length, scleral elasticity, wound apposition by residual vitreous, surface tension of a gas bubble, and intraocular pressure. To facilitate approximation of the wound edges, the cannulas should be withdrawn in a tangential trajectory. Infusion pressure can be decreased prior to cannula removal to minimize vitreous prolapse.[10] Infusion pressure may be activated to raise internal pressure while concurrent external pressure on the wound facilitates the angled incision tunnel to collapse and close.[16] Some vitreous remnant may also plug the ports to an extent during cannula removal. However, there is no increased rate of retinal detachment attributable to this.[17] If a wound leak is still detected at the end of surgery, absorbable sutures can be placed, especially in the setting of leaking silicone oil. Leakage from sclerotomies is more likely in highly myopic eyes with low scleral rigidity, in eyes with scarred conjunctiva or sclera from

previous surgery, in Marfan's syndrome,[18] and in young children.[10]

Instrument Rigidity, Functionality, and Array :

Rigidity of instruments is dependent on material, thickness, diameter (gauge), and length.[19] As the trend towards smaller gauge continued, problems with instrument array and tool flexure arose. Initially, 25G vitrectomy was primarily utilized in macular surgeries. As the range of instruments for small gauge systems increased, surgeons applied 25G and 27G systems to cases requiring more extensive peripheral vitrectomy, and flexibility of the smaller cutter was a problem, especially when using the instruments to affect eye rotation for peripheral access and visualization. Hubschman et al. demonstrated that 23G and 25G cutters were less stiff than 20G cutters. Even within the same gauge group, cutter stiffness varied due to differences in internal diameter among 25G and 23G vitrector probes.[20] Optimal positioning of the sclerotomies close to the horizontal meridian, avoiding the supraorbital rim and bridge of the nose, wide-angle viewing systems, and scleral depression, all minimize the need for eye rotation and problems related to tool flexure. Newer generation 25G and 27G cutters, endoilluminators, and laser probes are now stiffer, and newer forceps are shorter to increase the stiffness. Due to the improvements in instrument stiffness, its application to a wider range of surgical indications, including simple and complex retinal detachments, macular surgeries, tractional retinal detachments, and stages 4 and 5 retinopathy of prematurity.[21-27] Phacofragmatome for removal of dense dropped nuclear fragments have aditionally been limited to 20G, but a 23G fragmatome has recently been introduced (DORC, Zuidland, the Netherlands).

Fluidics of Vitrectomy :

Infusion Flow Rates : Reduction in internal diameter of the infusion cannula in smaller gauge systems increases frictional forces and loss of pressure head and decreases volume flow at the infusion tip entry into the eye, as per Poiseuille's law, which states that flow of an incompressible viscous fluid is proportional to the fourth power of radius of the transmitting tubing and inversely proportional to its length.[14] The volume flow rate decreases by a factor of sixteen when the inner tubing radius is reduced by half. In addition, the volume flow rate is directly proportional to the pressure differential and inversely proportional to the fluid viscosity.

Higher infusion pressures in the range of 40-50mmHg may be a way to compensate for this and allow higher flow rates in smaller gauge systems, but may affect eyes with compromised ocular perfusion.[10] Infusion fluid can be infused into the eye either by a gravity-fed system or a pressurized system. In gravity-fed systems, infusion pressure, measured in centimetres of water, is equivalent to the bottle height above the eye. In vented gas-forced infusion systems, the infusion bottle itself is pressurized and allows for rapid infusion pressure control via console-controlled venting.[14] In the Constellation system (Alcon, Fort Worth, Texas, US), the infusion is pressurized within the console cassette, which should ideally be placed at eye level. The integrated pressurized infusion has internal, noninvasive sensors that constantly measure flow into the eye through the infusion line and cannula and integrate it through the microprocessor of the computer. The resistance is measured during machine priming. Vitrectomy creates a pressure gradient that the machine senses and compensates for by increasing infusion.[19]

Cutter Flow Rates : Vitreous cutters developed based on the VISC had different drive systems. The electric cutter maintained a constant duty cycle (percentage of time the cutter port is open relative to each cutting cycle) with increased cut rate, but it was heavy and the electric motor in the handpiece led to easy muscle fatigue. The pneumatic cutter was first reported by O'Malley and Heintz in 1975.[2] Until quite recently, pneumatic cutters employed a single pneumatic pulse from a pneumatic energy source located in the machine to close the cutter guillotine blade and relied on a spring to open it to complete a duty cycle. Pneumatic cutters were smaller and lighter, but as the mechanical properties of the spring remain constant, as the cut rate increased, the inability of the spring to keep up with the pneumatically driven closure increased the time the port is closed, thereby decreasing the duty cycle.[28] Engineering advances led to

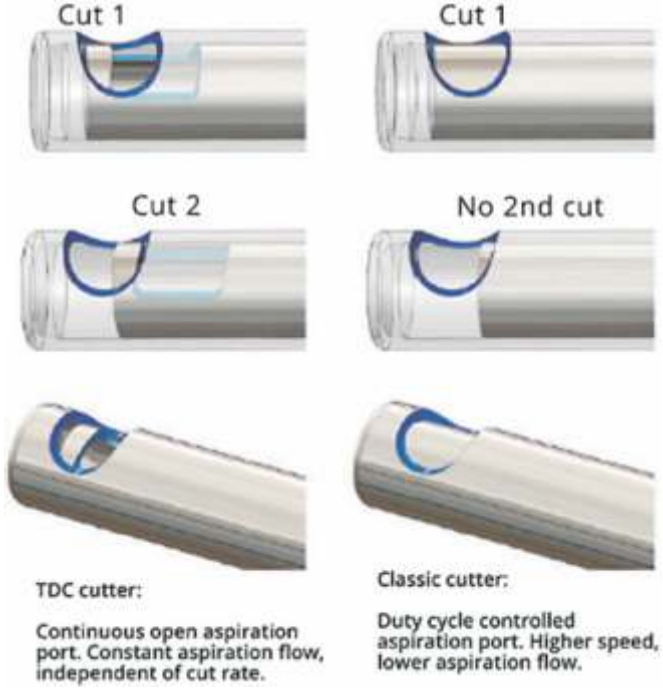

Figure 3: Key feature of the DORC twin-duty cycle 27G vitrector and comparison with classic cutter.

newer dual pneumatic drive cutters, which replaced the passive spring return phase with a second pneumatic piston that actively pushes the guillotine blade into the open position. This allowed a higher duty cycle at ultrahigh-speed cut rates up to 7500 cuts per minute and allowed surgeons to vary the duty cycle between 50% (50/50), less than 50% (shave mode), or more than 50% (core mode).[9] The latest twin duty cycle (TDC) cutter design on the Enhancing Visual Acuity (EVA) vitrectomy system (DORC, Zuidland, the Netherlands) has a second port in the internal guillotine blade of the pneumatic cutter. The concept of a double-port cutter was originally patented by Hayafuji more than 20 years ago. With two cutting edges, it cuts both forward and backward, nearly eliminating any port closed time, resulting in a 92% duty cycle independent of cutting speed and allowing cut rates to be doubled to reach 16,000 cuts per minute [Figure 3]. With the smaller 27G cutters, increased cutting rate and duty cycle improve cutting efficiency, without unduly increasing fractional forces.[29]

Illumination :

The first light source for vitrectomy originated from an external slit illuminator. In 1976, Peyman introduced endoillumination for 20G vitrectomy using a fibre optic inserted into the vitreous cavity.[30] Coaxial and slit lamp transcorneal illumination from the operating microscope produce scattered light (glare), while endoillumination minimizes light reflections and light scattering from the viewing system lens, cornea, lens, and vitreous.[31] Modes of endoillumination include light pipes, chandelier lights, and illuminated instruments. Handheld light pipes allow techniques of focal bright illumination, specular illumination where light shone at a critical angle causes an almost transparent surface to glow, highlighting surface irregularities, as well as retroillumination by reflecting the endoilluminator off the surface of the retina, retinal pigment epithelium, choroid, sclera, or off the cutter.[31] Compared to 20G light pipes, 23G and 25G endoilluminators had reduced light transmission due to reduced surface area of the fibre optic by 40% and 50%, respectively, and therefore required higher power sources. Initially, high arc lamps (xenon and mercury vapour) provided the high power output required for small gauge endoilluminators.[31] Newer light emitting diode (LED) light sources provide up to 40 lumens without degradation of light output, can last more than 10,000 hours, & are therefore particularly suited for smaller gauge endoillumination. Chandelier light and illuminated instruments were developed to allow bimanual surgery. Chandelier illumination is fixed in the sclera and provides wide-angle diffuse lighting. Illuminated picks provide focal bright light at the surgical dissection site, allowing clearer delineation of the surgical dissection planes. Illuminated lasers, scissors, forceps, and infusions are also available. Phototoxicity from high intensity light sources can be reduced by starting with low intensities, lowering intensities when switching from 25G to 20G endoilluminators, shortening exposure times to the macula, and maximizing working distances between the tip of the endoilluminator and the retina.[31] Newer LED light sources, used in LEDStar (DORC, Zuidland, the Netherlands) and integrated in EVA (DORC, Zuidland, the Netherlands), have built-in adjustable yellow filters to minimize phototoxicity.

Wide-Angle Viewing Systems (WAVS) :

In conjunction with developments in MIVS, enhancements in wide-angle viewing systems (WAVS) have improved panoramic viewing of the surgical field and enhanced safety and efficiency. They reduce the need for eye rotation, head repositioning, or scleral indentation and are particularly advantageous when using the smaller gauge cutters. Most WAVS consist of two components: an indirect ophthalmoscope lens placed on or above the cornea and a prismatic stereo reinverter that reinverts the image. WAVS are broadly classified into contact and noncontact viewing systems. Contact WAVS have a fixed field of view depending on the lens dioptric power, whereas the field of view in noncontact systems varies depending on the distance between the ophthalmoscope lens and the cornea.[32] Two contact-based wide-angle lens systems, ClariVit and HRX (Volk Optical Inc), are available. They provide approximately 10 degrees wider field of view than noncontact systems and provide superior image quality as they eliminate corneal aberrations and light reflections by directly placing the lens on the cornea. However, an experienced assistant is needed to hold the lens, and therefore more surgeons prefer to use the noncontact systems. Noncontact systems widely used include BIOM (Binocular Indirect Ophthalmo Microscope; Oculus, Wetzlar, Germany), OFFISS (Optical Fibre Free Intravitreal Surgery System, Topcon Medical Systems, Oakland, NJ), Resight 700 (Carl Zeiss Meditec AG, Jena, Germany), and Peyman-Wessels-Landers vitrectomy lens (Ocular Instruments, Bellevue, WA).[33]

Complications Associated with MIVS :

Intra-operative: Rise in intraocular pressure to more than 60mmHg has been measured during insertion of the trocar cannula complex,[33] but newer sharper trocar blade designs have improved ease of entry. Increased intraocular pressure and globe deformation can open recent corneal or scleral wounds, and placing sutures prior to port insertion reduces the risk of wound gaping and hypotony.[34] Displacement of the infusion cannula, during scleral indentation and eye rotation, can lead to serous or haemorrhagic choroidal detachment.[35] It can also occur in eyes with choroidal edema, such as in re-detachment surgeries.[36] The dislocated infusion can be

quickly moved to one of the other two ports to repressurize the eye. Avoiding excessively long scleral tunnels and placing the infusion closer to the horizontal meridian prevent easy dislodgment by the inferior lid or speculum. Cannula dislodgement can occur when instruments are withdrawn from the eye as a result of increased friction between the instrument and cannula wall, such as when removing forceps or scissors without fully closing the jaws.[10] Sclerotomies situated over areas of scleral thinning, such as in eyes with repeat surgeries, may have reduced friction between the cannula and the sclera and predispose to cannula dislodgement.[13] The dislodged cannula can be mounted onto a trocar and reinserted through the same scleral tunnel, or if it cannot be found, a new sclerotomy can be made. Rarely, breakage and intraocular dislocation of a segment of a cannula tip have also been reported.[36] Use of hybrid 20G / 23G or 20G / 25G systems, such as during phacofragmentation, can create infusion/outflow mismatch if care is not taken to raise infusion pressures to match egres.[37] Entry site breaks are not common in small gauge vitrectomy.[38]

Post-operative : Wound sealing of the sclerotomies was the main problem in the development of sutureless small gauge vitrectomy systems. The hypotony is usually transient, but can sometimes be severe, leading to choroidal detachment or haemorrhage, hypotonous maculopathy, or gas escape, and inadequate tamponade.[10,39] Furthermore, initial reports suggested higher rates of endophthalmitis. This may be due to contamination from conjunctival flora, ingress associated with postoperative hypotony, and vitreous wick effect at unsutured sclerotomies.[40] India ink passage has been demonstrated in eyes with unsutured 25G, 23G (straight or beveled), and 20G sclerotomies, compared to no entry of India ink in eyes with sutured sclerotomies.(12,41) Kunimoto et al. reported an endophthalmitis incidence of 0.23% for 25G PPV compared to 0.018% for 20G.[42] and Scott et al. identified an endophthalmitis incidence of 0.84% for 25G PPV compared to 0.03% for 20G in their cohort studies.[40] This may have been related to variations in sclerotomy construction, as a straight incision was found to have increased risk of endophthalmitis compared with a beveled approach. A systematic review by Govetto et al. did not find an increased risk of endophthalmitis for microincisional vitectomy systems compared to standard 20G vitrectomy.[43]

Summary and Future Directions for MIVS :

Significant strides in microincisional vitrectomy system fluidics, instrumentation, illumination, and viewing systems have been made in recent years, and MIVS has all but replaced 20G systems for a wide variety of vitreoretinal surgical indications. Retinal specialists have shifted away from 20G systems to smaller sutureless systems that have reduced operative times, surgical trauma, inflammation, astigmatism, and improved patient comfort, postoperative recovery times, and patient satisfaction. This drives the quest toward even smaller gauge systems, although this is tempered by the engineering challenges, surgical learning curves, availability, and, importantly, the higher costs. Careful case selection and optimization of surgical techniques for small gauge systems are important for surgical success.

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Dehemoglobinized heme at fovea in case of Valsalva Retionopathy : To observe or to operate ?

Neha Sharma[1], Dhaivat Shah[2]

INTRODUCTION :

Valsalva retinopathyis a condition that presents as sudden, painless loss of central vision primarily due to rupture of superficial retinal capillaries secondary to raised venous pressure following elevated intrathoracic or intra-abdominal pressure.[1] Most of the cases are self-resolving, but surgery is indicated in cases of long standing non resolving haemorrhage, especially at the macula.[2] The usual recovery period ranges anywhere between 4-8 weeks, during which the haemoglobin in the blood undergoes the process of de-haemoglobinization and reabsorption.[2] If this process is delayed, especially when there is macular involvement, an intervention is necessary. Interventions include puncturing the posterior hyaloid face and internal limiting membrane with a neodymium-doped yttrium aluminium garnet (Nd-YAG) laser, pneumatic displacement of the haemorrhage by an intravitreal injection of gas with or without recombinant tissue plasminogen activator, or pars plana vitrectomy with ILM peeling.[3,4,5]

Here we state a case where the patient presented to us with Valsalva retinopathy at macula after one month of the episode, and the dehemoglobinized blood self-resolved over a period of 3 months with functional improvement in visual acuity.

CASE REPORT :

A 24 year old male, presented with a complaint of diminution of vision and appearance of a black spot in front of right eye since 1 month. He gave a history of having multiple vigorous episodes of vomiting before a month while being treated for jaundice. Best corrected visual acuity (BCVA) and Intraocular pressure in right and left eye were 20/200 and 20/20, and 14 and 12 mmHg respectively. On right eye fundus examination, a yellowish area with well-defined margins was noted over macula.On OCT, a well-defined elevated hyperreflective area with significant back shadowing was seen at the fovea. Based on these findings, a diagnosis of Valsalva retinopathy was laid Fig 1(a.b)

The patient also stated that since the time of occurrence of the event, there has been some improvement in the visual acuity over a period of 1 month. With this history of subtle improvement and noting the clinical de-hemoglobinization of blood, the patient was advised observation for a period of 2 weeks. The patient was clearly counselled that a surgical intervention would be warranted if the blood does not resolve by then.

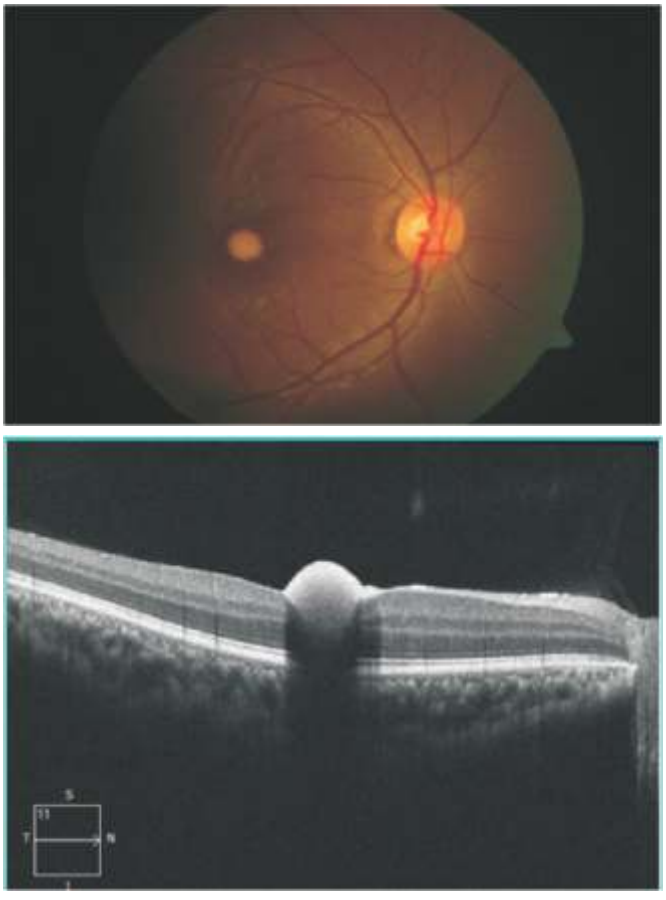

Fig 1 (a,b) Fundus photo of RE showing yellow,round deposit at the macula and OCT image corresponding to clinical finding,showing homogenous hyperreflective deposit at sub ILM level with backshadowing is Dehemoglobinized heme.

However, 2 weeks later, the patient followed up with significant improvement in visual acuity (20/60). Clinically, there was marked reduction in the elevation of yellow spot at the macula Fig 2(a,b)

DISCUSSION :

Patient presenting with Valsalva retinopathy usually have history of retching, vomiting, heavy weight lifting etc. followed by sudden diminution of vision. These bleeds are located commonly at sub ILM level or subhyaloid levels. The location of bleed determines spectrum and severity of symptoms. Large bleeds can be boat shaped along with large dome

Choitram Netralaya, Indore

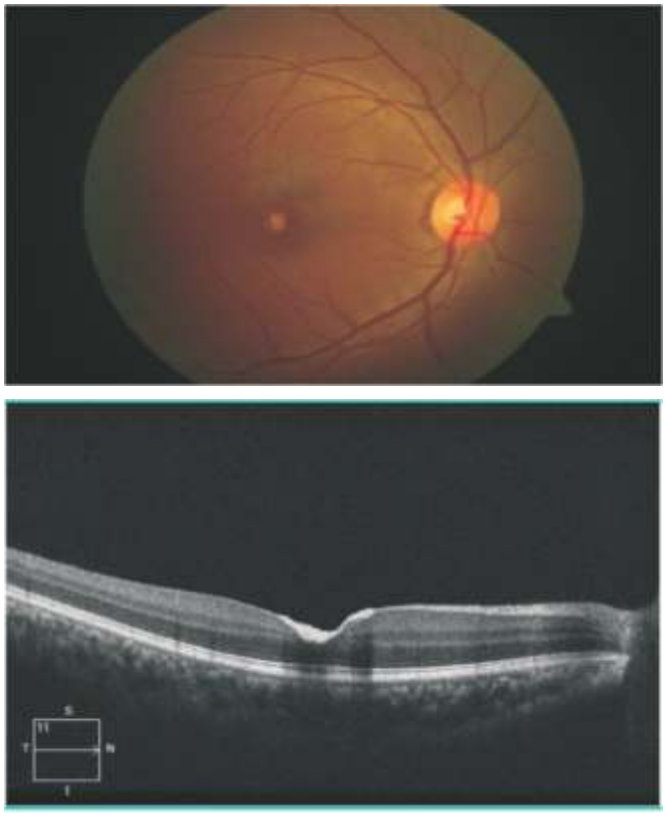

Fig 2 (a,b) Fundus photo and OCT of the same patient on follow up showing reduction in size dehemoglobinized heme deposit and reduced back shadow on OCT respectively.

shaped elevated haemorrhage, and macula being the gravitational centre, becomes a common site for occurrence. [6,7]

Treatment depends on the location and time of presentation of the condition. Fresh onset, small and extra-foveal haemorrhages can be observed for a period of 2-6 weeks for self-resolution. But a large macula involving bleed, especially more than 4 weeks old, usually needs intervention. [8]

As in our patient, the time was onset of injury was already a month old. Also, the colour of the heme had changed from red to yellow, meaning dehemoglobinization had taken place. Such a blood seldom tends to resolve on it's own. Hence, we gave option of observation vs surgical intervention for the same (Vitrectomy + ILM peeling), explaining pros and cons of both. The patient was willing to wait for spontaneous resolution.

To our surprise, on one month follow up, he had drastically improved in visual acuity (20/60) as well as extent and density of heme.

Conclusion :

In such cases of Valsalva maculopathy, a chance of observation should be given instead of directly jumping onto surgical intervention.

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Dr. Meyer *- Schwickerath photographed coagulating the retina using a xenon coagulator.*

Feed me Some : Imaging Feeder Vessels in a case of Retinal Angiomatous Proliferation

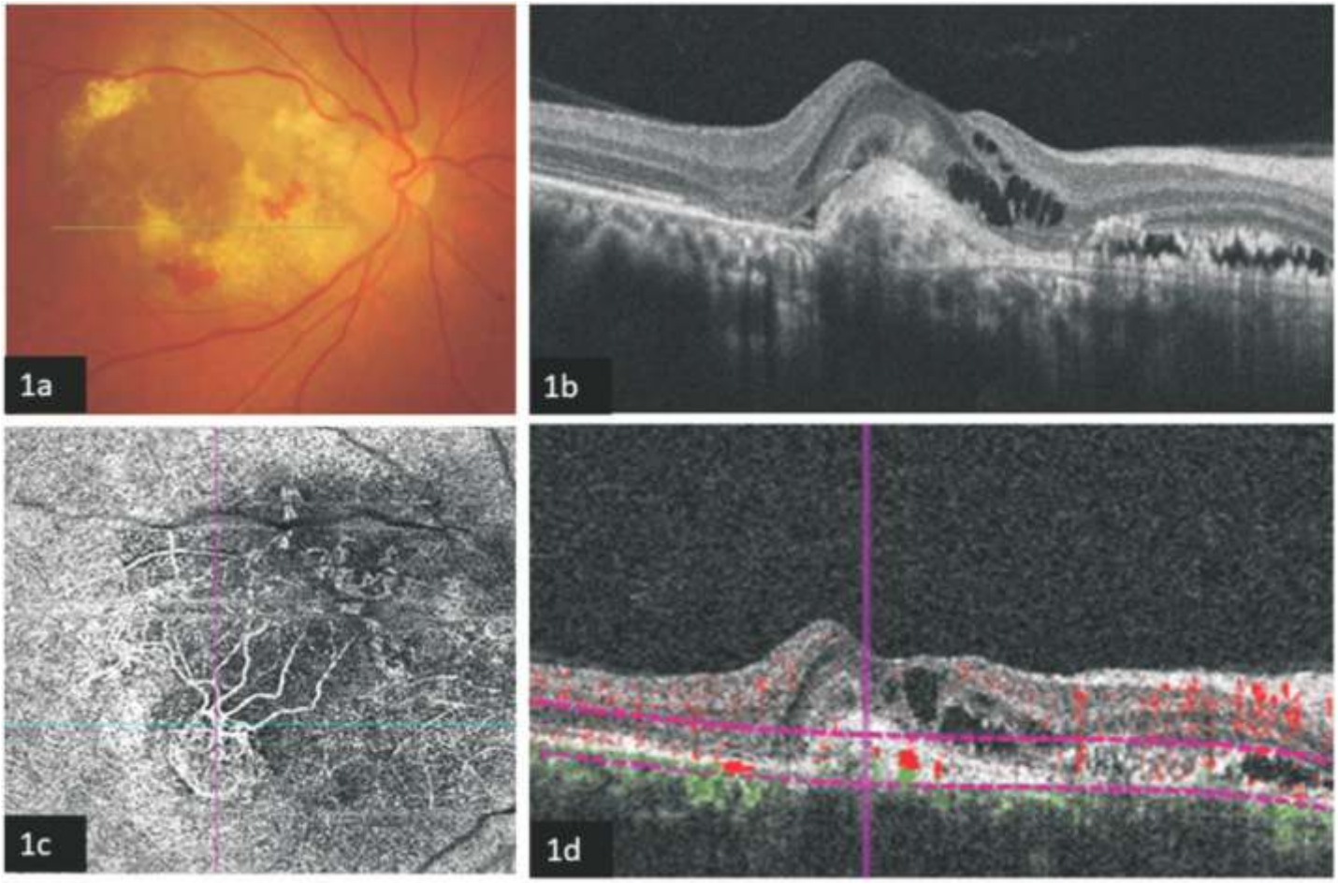
Achal Singhal[1], Mradula Gangwar[2], Dhaivat Shah[3]

Introduction :

Retinal Angiomatous Proliferation (RAP) was described by Yannuzzi et al. as neovascularization that begins in the intraretinal architecture and progresses posteriorly into sub-retinal space, contradictory to the Type 1 and 2 choroidal neovascular membranes. It was believed to eventually reach the choroidal circulation and form a retinal-choroidal anastomosis. It is commonly noted after the 5th decade and the occurrence is higher in hypertensives and smokers. [1]

Case Report :

A 58-year-old male patient, a chronic smoker, came to our OPD with complaints of a diminution of vision in the right eye (BCVA: 2/60). On examination, the following findings were observed in the patient.



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On fundus examination (Image 1a) - Large areas of exudation with multiple superficial and deep hemorrhages at the macula were noted.

On SD-OCT imaging (Image 1b) - Multiple intraretinal spaces were seen along with shallow subretinal fluid and hyperreflective dots (indicative of phagocytosed photoreceptors). On the foveal area - a hyperreflective

membrane was noted which seemed to dip down and establish a retino-choroidal anastomosis.

On OCT-A imaging (Image 1c) - In the ORCC complex, the neovascularization frown, correlating with the membrane complex on the fundus and structural OCT, was visible which was noted to be supplied by small feeder vessels coming from the superior aspect of the fovea.

On OCT-A blood flow analysis imaging (Image 1d) - The blood flow analysis showed increased blood flow signals at the level of the membrane (indicative of increased color signal).

Discussion :

Based on the findings of the above investigations and clinical examination, the patient was diagnosed with a Case of CNVM Type 3, also described as RAP, and was managed by Anti VEGF injections. This condition usually requires more injections as compared to Type 1 and 2 CNVMs, and the visual prognosis is guarded.[2] Hence, it's very important to counsel the patient before initiating the treatment that the treatment would be long-term and the aim would be preservation of existing vision.

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Lawrence A. Yannuzzi, *pioneered the use of indocyanine green (ICG) angiography for managing chorioretinal diseases, and through his clinical research, discovered new macular diseases and manifestations of known disorders such as central serous chorioretinopathy, idiopathic polypoidal choroidal vasculopathy, and retinal angiomatous proliferation (RAP). He also developed The Yannuzzi Lens, with a flange to compress sclera and raise intraocular pressure without distorting fundus image. It was used to stop choroidal perfusion and bleeding while using Krypton Red Laser for juxtafoveal photocoagulation of choroidal neovascularization. The Krypton laser was also useful for avoiding absorption of laser light by macular pigments, particularly in Pathologic Myopia and Pseudo Xanthoma Elasticum.*

Irvine - Gass Syndrome

Ashima Monga[1], Deepanshu Agrawal[2], Dhaivat Shah[3]

ABSTRACT :

Irvine-Gass Syndrome, commonly known as Pseudophakic cystoid macular edema (PCME), is one of the commonest complications post-cataract surgeries, leading to a decrease in vision. Although the pathogenesis of PCME is not completely understood, the contribution of surgical inflammation is established evidence. Consequently, anti-inflammatory medicines, including steroids and non-steroidal anti-inflammatory drugs, have been postulated as having a role in both the prophylaxis and treatment of PCME.

CASE :

A 70 year-old healthy male, presented with sudden, painless diminution of vision in the left eye (OS) since 15 days. Patient had undergone an uneventful manual small incision cataract surgery (MSICS) with posterior chamber intraocular lens implantation (PMMA) 2 months back. He was discharged on standard topical treatment of moxifloxacin 0.5% and prednisolone 0.1% (tapered weekly) combination, with a visual acuity of 20/32. Patient had presented 2 months following surgery, with a best corrected visual acuity (BCVA) in OS of 20/200. Slit lamp examination revealed clinically unremarkable OS anterior segment with well placed posterior chamber intraocular lens. Dilated fundus examination (OS) revealed an attached retina with cystic spaces at macula which were confirmed on optical coherence tomography (OCT) (central foveal thickness of 633 microns) [Figure 1A]. Patient was diagnosed as a case of Pseudophakic cystoid macular edema (PCME) and was started on topical non-steroidal anti-inflammatory drug(NSAIDS), Nepafenac 0.1%,three times a day, and was asked to review after 6 weeks.

On the 6th week follow-up visit, BCVA in OS had improved to

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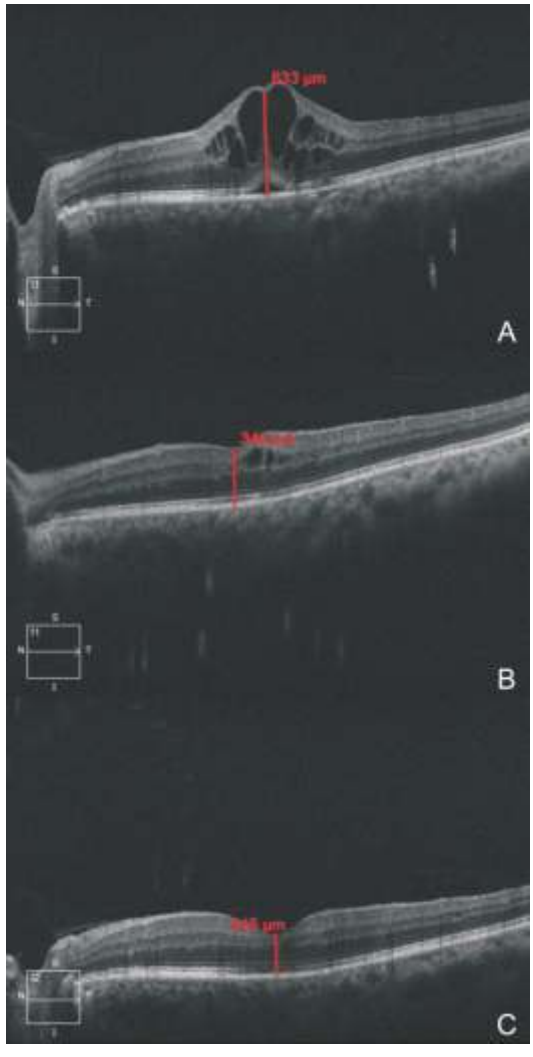

Figure 1: Optical Coherence Tomography - Macula (HD 21 Line scan, through fovea) scans at presentation and on follow-up.

1A: OCT - Macula scan depicting tall, multiple intra-retinal hypo-reflective cystic spaces, distributed equally over fovea, along with presence of subretinal fluid. (Central foveal thickness: 633 microns)

1B: OCT - Macula scan at 6 weeks follow-up, shows resolved subretinal fluid. Intra-retinal cystic spaces have reduced in height as well as number. (Central foveal thickness: 346 microns)

1C: OCT - Macula scan at 10 weeks follow-up, shows completely resolved intra-retinal cystic spaces. (Central foveal thickness: 245 microns)

20/80. OCT evaluation revealed reduced height of cystic spaces with minimal intra-retinal hypo-reflective spaces (central foveal thickness: 346micron) [Figure 1B]. He was continued on the topical treatment and was asked to review after 4 weeks. One month later, OS BCVA improved to 20/32 and there were no signs of CME clinically, as well as on OCT (Central foveal thickness: 245micron) [Figure 1C]. Patient was asked to continue topical medication for 1 month more and review thereafter.

DISCUSSION :

Irvine-Gass Syndrome also known as PCME is a common cause of diminution of vision following cataract surgery. It was first described by Irvine in 1953 in his Proctor lecture,[1] and then characterized angiographically by Gass and Norton in 1966.[2] Its incidence ranges from 1-30% after extra capsular cataract extraction. However, the incidence of clinically relevant CME is 1-2% in the absence of any other risk factors.[3]

The Pathophysiology of PCME can be attributed to accumulation of fluid in the inner nuclear and outer plexiform layer due to increase in perifoveolar capillary permeability. It is associated with intraocular inflammation and mediated by release of prostaglandins and leukotriene. The incidence of CME peaks at 6-10 weeks after surgery. About 95% resolve spontaneously in uncomplicated cases.[4] Various risk factors exist for PCME including pre-existing retinal conditions like diabetic retinopathy, uveitis, vein occlusions, retinitis pigmentosa etc. to intra and post-operative factors like posterior capsular rupture, vitreous loss, iris manipulation, poorly controlled post-operative inflammation.[4]

Diagnosis is generally clinical with anterior segment showing nil to minimal congestion or reaction. Fundus evaluation reveals an isolated CME with no associated vascular changes, drusen or hemorrhages. OCT shows cystoid spaces of low reflectivity and associated retinal thickening. The early phase of fundus fluorescein angiography (FFA) shows leakage around the fovea and disc leak. This differentiates PCME from diabetic CME. In practice, PCME is defined by patient's history of visual impairment post-cataract surgery along with clinical assessment of macular edema which can be further confirmed up on OCT, meaning not every case requires a FFA for confirmation of diagnosis.[5]

Post-operative use of anti-inflammatory topical drugs has a prophylactic action against early CME. Medical therapy also starts with a course of NSAIDS and corticosteroids. Topical therapy may take 3-6 months to resolve chronic CME and risk of relapse after cessation of drugs is also present. Corticosteroids are frequently used in treatment of PCME as they block the inflammatory cascade and also encourage fluid absorption by retinal pigment epithelium. Intravitreal/ posterior sub-tenon injection of steroid has shown benefits in PCME who are not responding to medical therapy. Off label use of anti-VEGF or systemic carbonic anhydrase inhibitors in cases of chronic non-responsive CMEs is controversial.[4,5]

The Irvine-Gass Syndrome remains a therapeutic challenge to the cataract surgeons.[5] With the advancement in surgical techniques and better investigative tools over the past decade, the detection and management of PCMEs has improved.[6] However, prophylaxis and treatment measures have largely remained the same. We believe that all patients undergoing cataract surgery do not require to be prescribed NSAIDS prophylactically as a blanket therapy. Only patients with above discussed pre, intra or post-op risk factors are suited to be initiated on NSAIDS post-op. Pre-op counseling holds utmost importance specifically in these cases, looking at the higher expectation of the patients in today's era.

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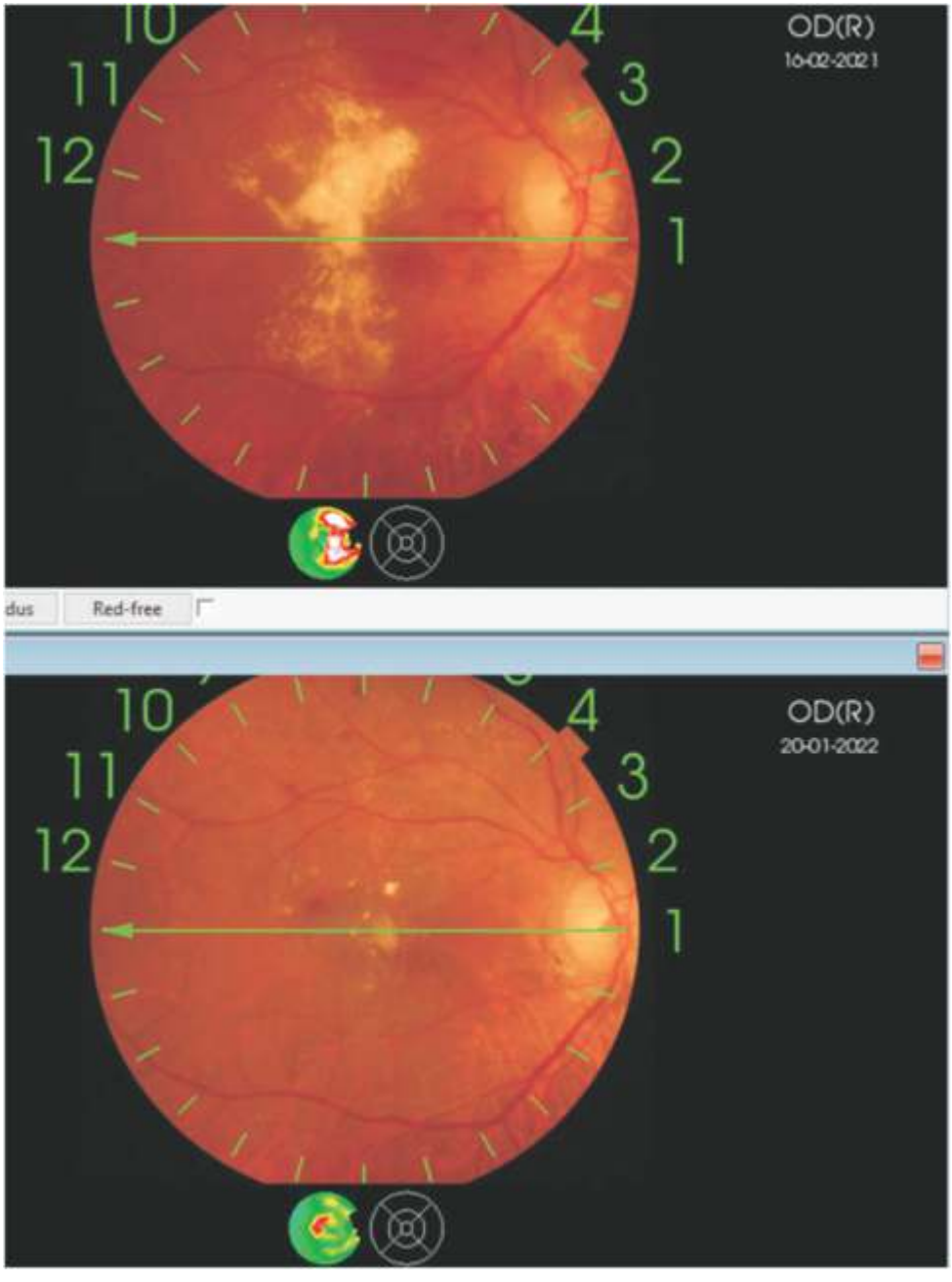
Retina Case Report

G.V.N. Rama Kumar

Hard exudate plaques do disappear!



55 year old female was treated for Diabetic macular edema with hard exudates plaque in her right eye. She was given intravitreal injections of Aflibercept in her right eye 4 times in a period of 11 months. The hard exudate plaque almost disappeared at last follow up

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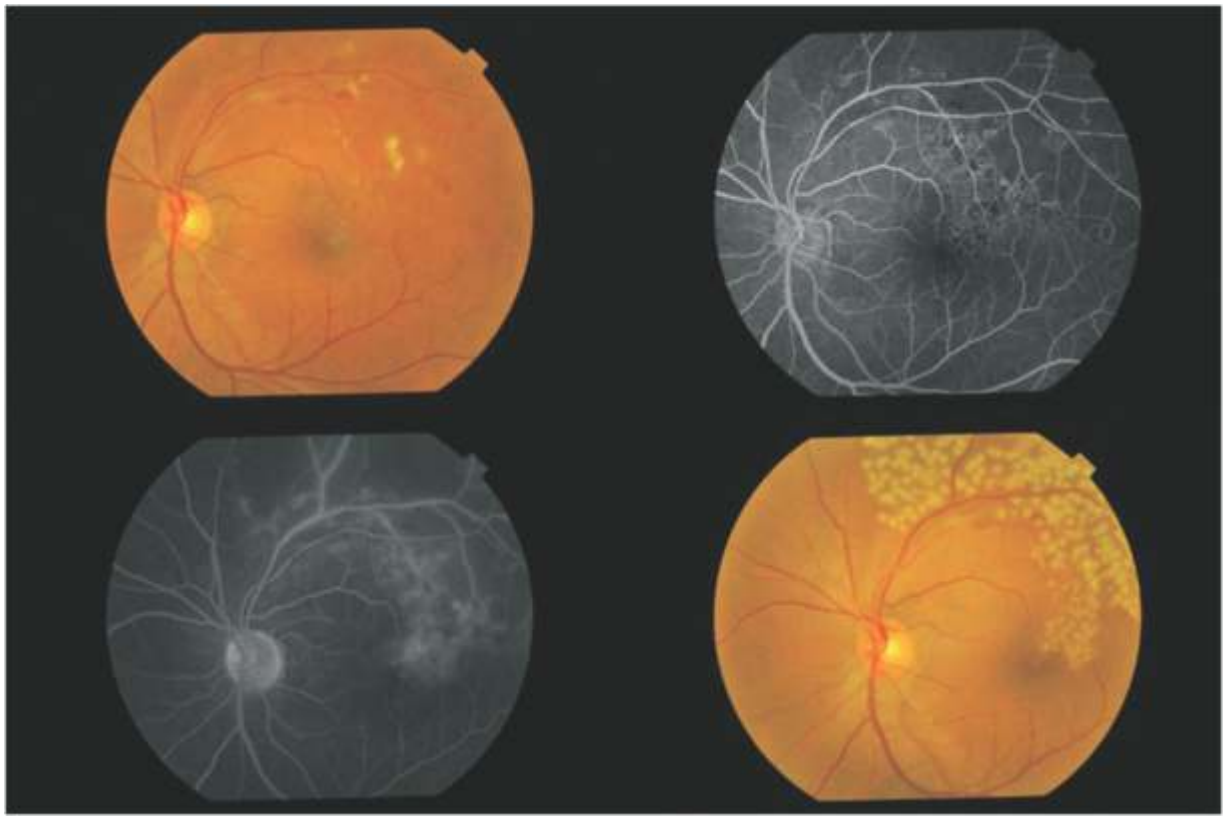
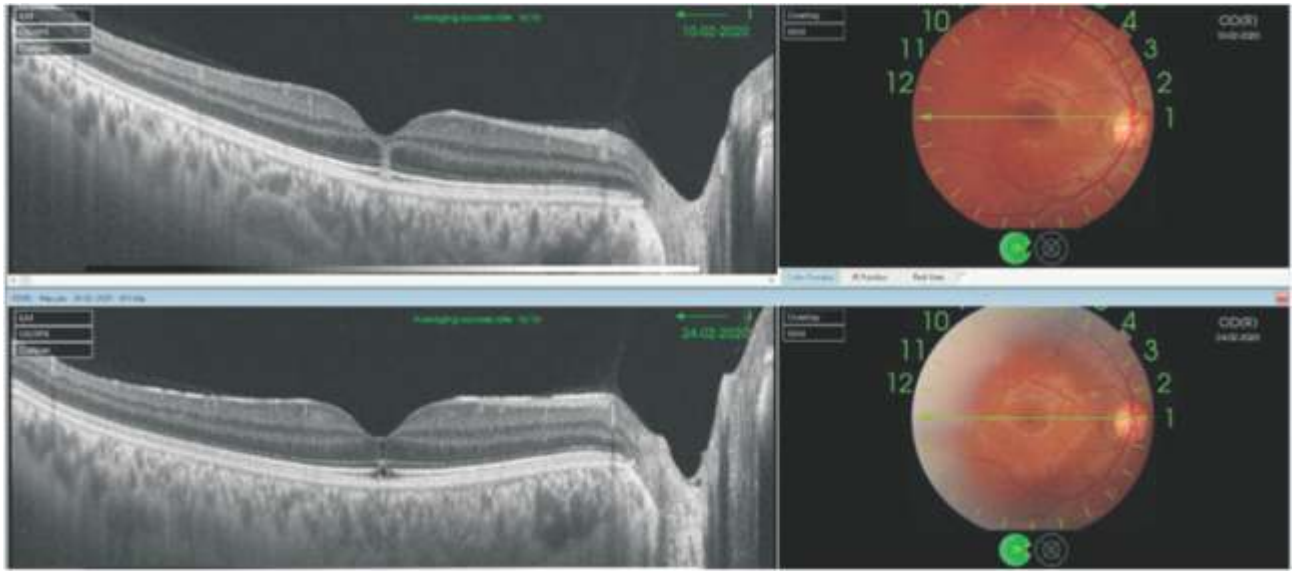
Recommended pattern of sectoral laser photocoagulation for BRVO after disappearance of retinal haemorrhages



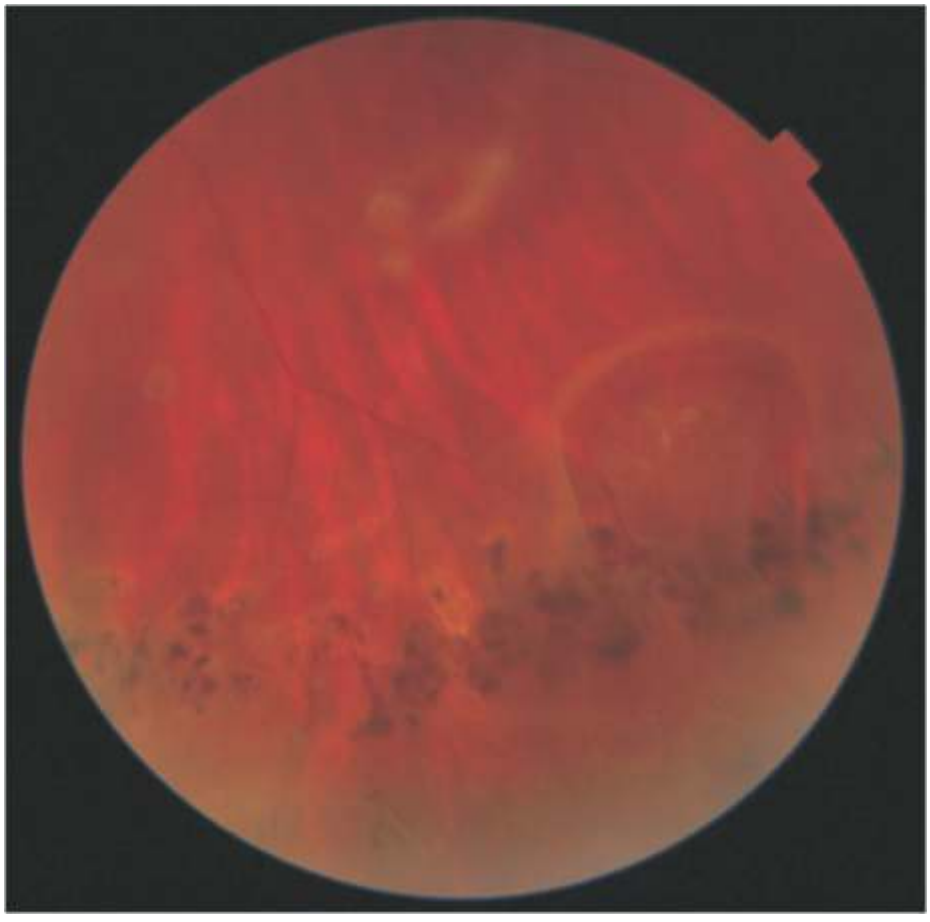
This image is self explanatory. Sectoral laser for BRVO with NVE should be done to the area of avasular retina. Laser spots to avascular area at macula should be grid pattern low intensity burns with good spacing. Beyond macula, the avascular retina should be covered with confluent laser spots with minimal spacing.

Acute photic retinitis results in outer lamellar macular hole in 10-15 days



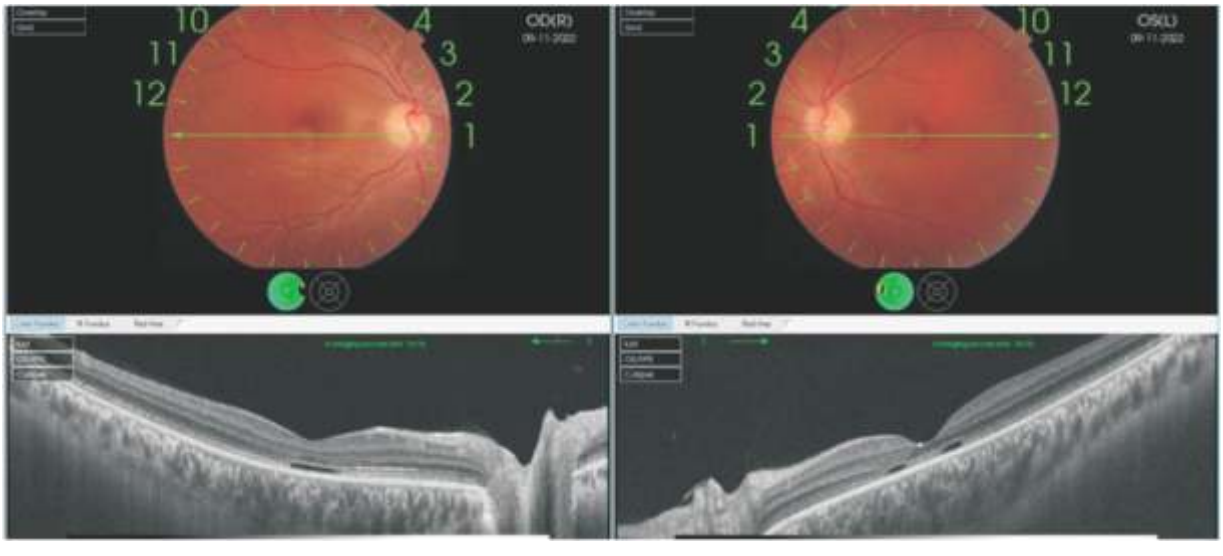
30 year old male had dense amblyopia in his left eye with BCVA of 3/60. Right eye was the better eye with good vision. He wanted to further strengthen his right eye and he did sun gazing with his right eye during a prayer as suggested by some spiritual guru. He developed sudden DOV and presented to us on the same day. As it was fresh case of photic retinitis, high dose oral steroids and other supportive treatment including antioxidants were prescribed. Eventually, the eye developed outer lamellar macular hole at fovea in 2weeks and his vision got stabilised at 6/9

HST posterior to laser barrage done for pigmented lattice!



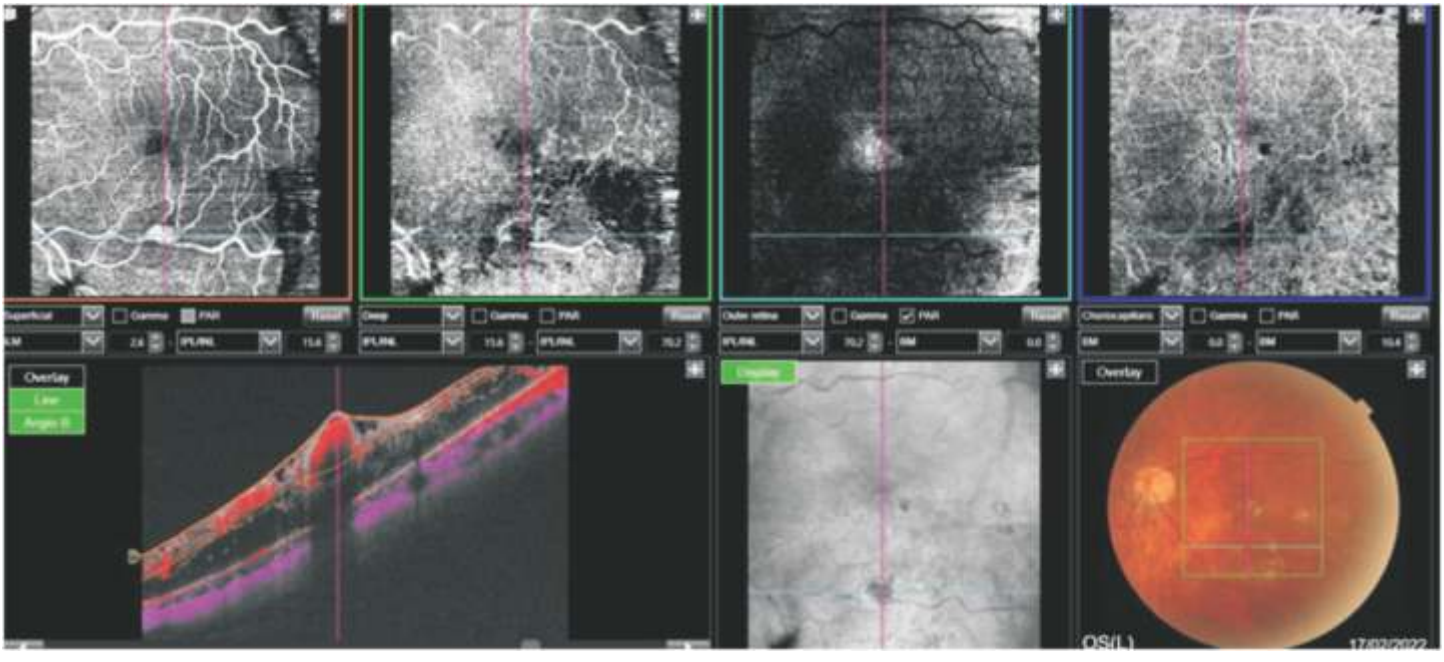
38 year old male with pathological myopia in both eyes underwent laser barrage(elsewhere) to inferior pigmented lattice 5 years back. He presented to us with sudden onset of floaters in left eye and on examination, he was found to have PVD with horse shoe tear (HST) posterior to the previous laser barrage. This case cautions about the fact that laser barrage to lattice, though reduces the risk of retinal breaks around lattices, itself can result in breaks in the thin retina associated with pathological myopia.

Cone dystrophy can be diagnosed without ERG

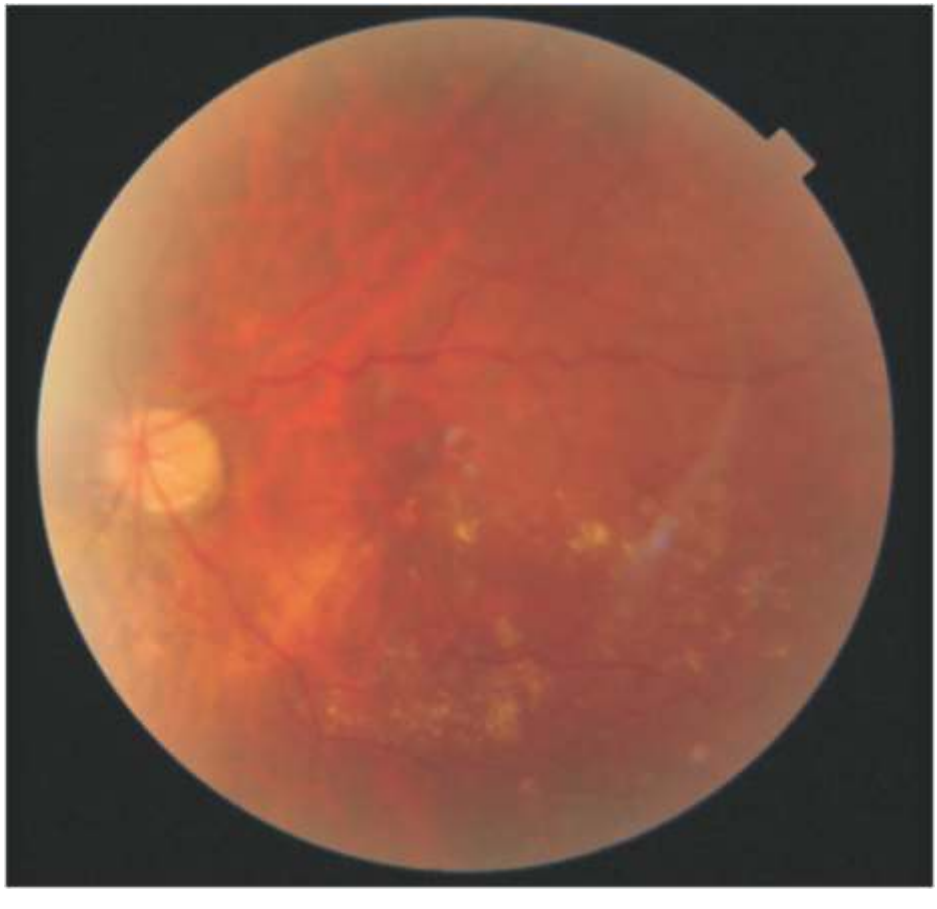


Onset of symptoms at around 3-5 years of age, associated photophobia, colour vision deficiency, nystagmus, symmetrical vision loss, symmetrical fundus picture and loss of cones at fovea (as seen on OCT here) are almost always diagnostic of Cone dystrophy. Target maculopathy, if seen, either on fundus picture or on autoflorescence, adds to the diagnosis.

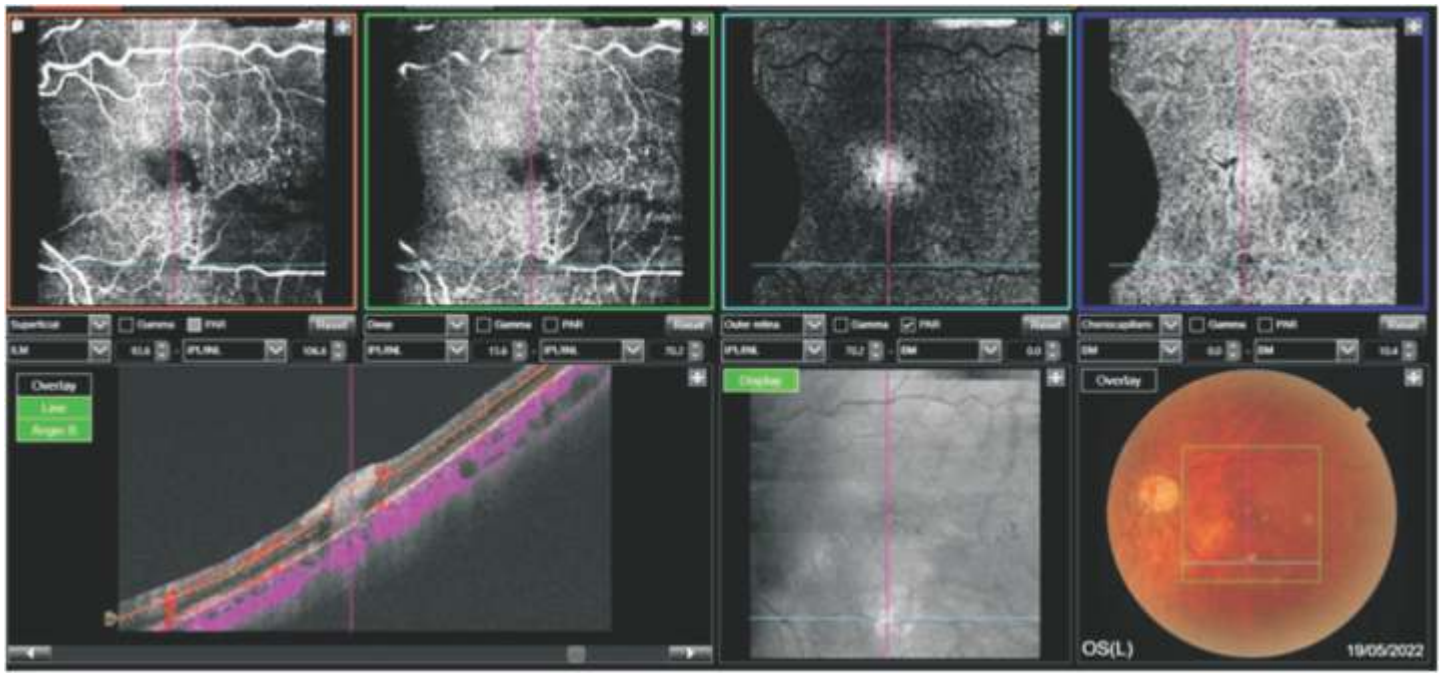
Diagnosis, treatment and follow up of Retinal arterial macroaneurysm with the help of OCT angiography



52 year old female presented with gradual loss of vision in her left eye. There was no history of DM but she was a known hypertensive on medication. BCVA was 6/18 in left eye and there were multiple hard exudates at macula with foveal thickening. OCT angiography revealed retinal arterial macroaneurysm (RAM) along inferior macular arteriole. Foveal thickening resolved and RAM got sclerosed with vision improvement to 6/6 at 3 months follow up

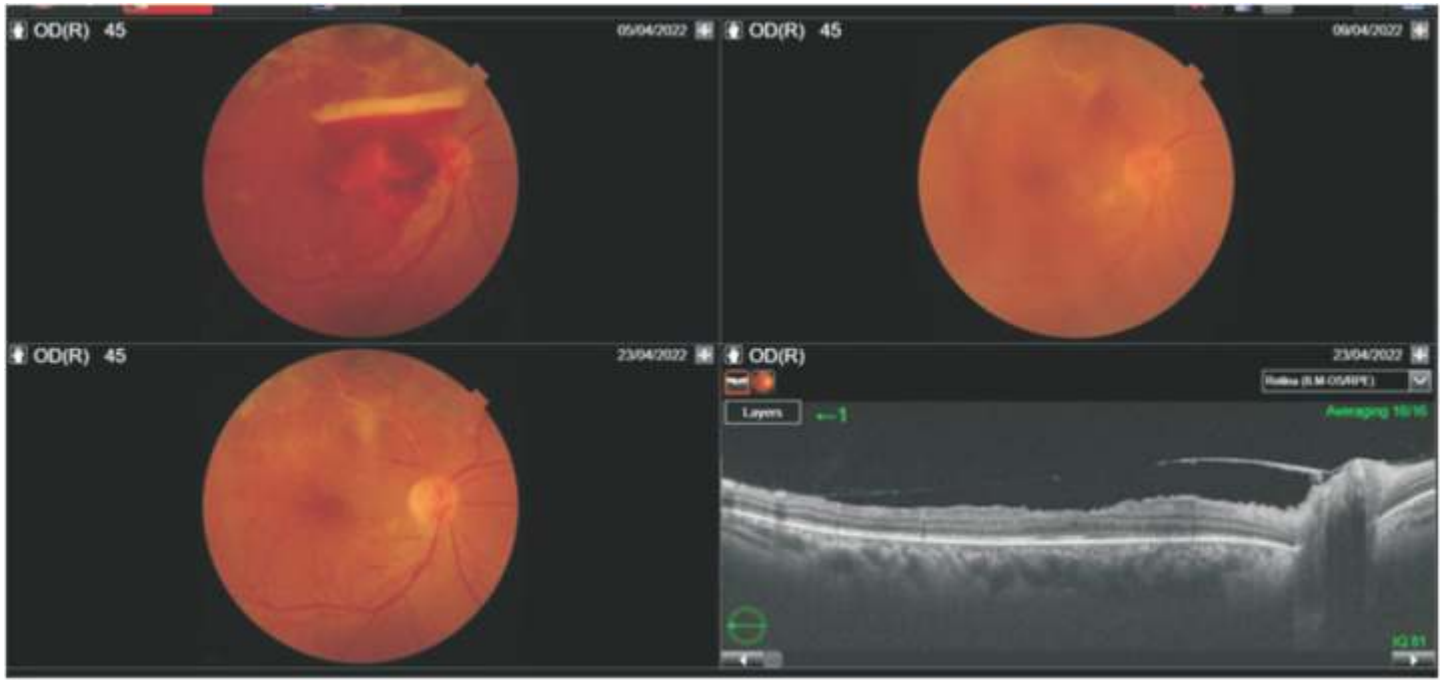


She was given intravitreal anti VEGF injection followed by focal laser to RAM 1 month later.



Foveal thickening resolved and RAM got sclerosed with vision improvement to 6/6 at 3 months follow up

Diagnosis, treatment and follow up of Retinal arterial macroaneurysm with the help of OCT angiography



48 year old male had BRVO 2 years back and sectoral laser PRP was done elsewhere. He developed sudden diminution of vision with a central scotoma. He consulted elsewhere and presented to us 2 weeks after onset of symptoms. On examination, he was found to have premacular subhyaloid hemorrhage with a fluid level and dehemoglobinised blood at the top.

Subhyaloid hemorrhage associated with valsalva retinopathy without any vasculopathy is usually treated with YAG laser hyaloidotomy. Similar bleeds associated with fibrovascular proliferation of diabetic retinopathy and RVO are supposed to need VR surgery because of risk of worsening of traction on retina with YAG laser. In this patient, hyaloidotomy was done with green laser to minimise the risk of traction and to avoid VR surgery. Though there was no immediate drainage of bleed, it cleared subsequently in 2 weeks with vision improvement to 6/9

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