Vitreo-Retinal Society - India
Sept - Nov. 2012

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RETINA
an insight

The official newsletter of the
VITREO RETINAL SOCIETY - INDIA

Dr Gopal Lal Verma
President - VRSI

From the President's Desk

Vitreo retinal speciality had never before been so much exciting to ophthalmologist and public alike, as it is now. Primarily it has emerged so on three accounts; firstly introduction of newer antivegf drugs, secondly newer retinal imaging modalities and lastly newer instrument and equipment designs in smaller gauge vitrectomy. It is exhilarating to see more and more young ophthalmologists joining vitreo retinal brigade. In this issue Dr S Natarajan who holds the baton, is enlightening us on antiplatelet derived growth factor and its possible beneficial role in conjunction with ranibizumab. Dr Manish Nagpal with his realistic approach is discussing issue of rebleeds in 23 G TVS in vascular retinopathies. Dr Lingam Gopal is giving an insight from his vast experiences on understanding and treating choroidal coloboma.

Continuing with our trend , this issue features contribution of, another living legend in vitreo retinal speciality-Prof. Dr Hem Kumar Tewari, the most revered teacher who spent his entire life in business of retina and vitreous. Many of his disciples and trainees are highest skilled vitreo retinal surgeons in India and abroad.

The forthcoming annual meeting of vitreoretinal society-India at Guwahati by enduring efforts of Dr Satyan Deka and Dr Harsha Bhattacharjee and their team, is a testimony to leading role of our vitreoretinal colleagues doing par excellence as compared to any developed nation. This year VRSI has invited another pioneer Prof. Dr J K Ambati -Kentucky for Nataraja Pillai Oration and we are glad to nominate Prof. Dr TP Das for Prof. S S Hayreh award.

Finally, we all must remember that, we should work for a cause not for applause and live to express not to impress.

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1. Rizzo S, et al. Comparative Study of the Standard 25-gauge Vitrectomy System vs the New Ultra-high-speed Vitrectomy System. *Retina Today*; September Insert, 2010. 2. Data on File, Alcon Research 954-2020-003. 3. Nagpal M, Wartikar S, Nagpal K. Comparison of clinical outcomes and wound dynamics of sclerotomy ports of 20, 25, and 23 gauge vitrectomy. Retina. 2009;29(2):225-231. 4. Alcon data on file 954-0000-004.

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Editorial

Dr. S. Natarajan
Editor – In - Chief

Dear Friends,

Amidst the season of festivals, I bring you a brief introduction to recent advances in vitreoretinal surgery through this winter issue.

Along with the basics of mechanism of microplasmin in inducing PVD, we also bring forward a glance on Anti-PDGF in treatment of wet AMD. An insight on use of intraoperative OCT has made surgical demarcations more precise and thus needs a special reference. I also bring forward two new instruments, the holekamp diamond dusted soft tip drip dropper with fluted bulb and the nano tapered awh forceps, that further potentiates the ease of vitreoretinal surgery

Microplasmin (Ocriplasmin):[1]

Current concepts of the pathogenesis of macular disease are based on fibrocellular proliferation mediated traction at the vitreoretinal interface as a major cause.

Mechanical removal of the cortical vitreous however remains incomplete, leaving behind cortical vitreous fibrils on the inner limiting membrane (ILM). This seems to be a major cause of disease progression and treatment failure.

Plasmin is a serine protease that mediates the fibrinolytic process, modulates the extracellular matrix and does not degrade collagen type IV, a major component of basement membranes and the ILM.

Plasmin, however, has as yet not been available or approved for intravitreal application in humans. Therefore, plasminogen, the inactive precursor of plasmin has been proposed for intravitreal injection, necessarily followed by an additional administration of a plasminogen activator.

Microplasmin demonstrates efficacy in separating the posterior hyaloid from the ILM. Complete vitreoretinal separation is induced in a dose- and time-dependent fashion without morphologic damage to the retina thus is safe for the ocular ultrastructure. The major advantage of microplasmin compared with mechanical ILM peeling for complete vitreoretinal separation lies in the unchanged reactivity of retinal glial cells and neurons. Compared with autologous plasmin, microplasmin ensures the application of a pure substance at a defined dose.

Anti-PDGF in wet AMD:[2]

Treatment of neovascular macular degeneration with anti-VEGF monotherapy, is known to be was unsustainable in order to achieve the best possible results that requires closely spaced injections. Even in the best of studies, such as the ANCHOR and MARINA studies, two-thirds of patients didn't gain significant visual acuity.

The neovascular complex expands with a specific, specialized group of cells known as the tip cells. These are the only naked endothelial cells and act as scout or lead cells in expanding the size of the neovascular membrane. These produce platelet derived growth factor (PDGF), which matures and recruits pericytes that back cover the neovascular complex. The pericytes act as a protective armour against anti-VEGF monotherapy

Thus it would be desirable to combine anti-PDGF treatment with anti-VEGF treatment. The goal would be to have the anti-PDGF treatments chemically strip pericytes from the neovascular complex, rendering it susceptible to the anti-VEGF treatment.

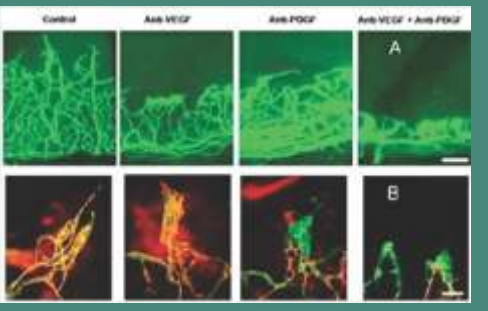
Ophthotech clinical phase 1 trial using a drug named E10030 (renamed Fovista) in combination with Lucentis (ranibizumab) to treat patients with advanced exudative macular degeneration has shown superior efficacy to Lucentis monotherapy. Phase 2b clinical trial results by revealed that Fovista, combined with anti-VEGF therapy increased positive visual outcomes compared with anti-VEGF therapy alone. The clinical trial showed that sequential injection of Fovista on the same day as Lucentis (ranibizumab) produced better visual acuity in patients at 6 months. The added benefit with combination therapy is reducing the burden of monthly anti-VEGF treatments in advanced cases. This combination therapy exhibits excellent safety profile[3,4]

Intraoperative OCT:[5]

The advent of optical coherence tomography (OCT) has greatly enhanced our understanding of vitreoretinal diseases. The use of OCT has recently been extended beyond the offices and clinics to perioperative and intraoperative settings. The new development in high-resolution and high-speed spectral domain OCT, along with the improvement in portability, has made the device more valuable than ever before.

(a) BIOPTIGEN[6]

The Bioptigen SDOCT instrument is a handheld, FDA-approved portable device that is able to produce high-resolution



Murine corneal neovascularization model. Combination treatment of anti-PDGF and anti-VEGF inhibits (A) and regresses (B) neovascularization more effectively than either agent alone.

(Figure 1.: Action of combination treatment eith anti-VEGF and anti-PDGF)

(Figure 2.: BIOPTIGEN)

(Figure 3.: SPECTRALIS)

(Figure 4.: OPTOVUE)

(Figure 5.: Fluted bulb drip dropper)

(Figure 6.: Diamond Dusted Soft TipILM polisher)

(Figure 8.: see-to-the tip design of Awh Forceps)

(Figure 7.: Awh forceps with larger grasping platforms)

retinal images. It offers flexibility with current SDOCT technology. With a convenient handheld scanner and mobile platform, the device adapts to a variety of clinical settings. Without the stability of a fixed platform, the handheld Bioptigen requires an experienced operator to obtain images of high quality.

(b) HEIDELBERG INTRAOPERATIVE SPECTRALIS PROTOTYPE[6]

The Spectralis is an FDA approved device for SD-OCT imaging. Designed similarly to operating microscope platforms, the intraoperative Spectralis is mounted with an adjustable arm that provides stability and controlled movements. It is suited for the sterile operating field, as it has the option of hands-free foot pedal control to position the device precisely.

When combined with eye-tracking technology, it acquires high quality, precise and reproducible images in the operative setting. But its size and bulkiness limit its effective motility.

(c) OPTOVUE[6]

The iVue is an FDA-approved device contained in a compact wheeled platform, with the ability to obtain images in clinical settings via a slit-lamp control. The device can also be detached and used as a handheld unit, suitable for use in the operative setting. It has a flat-screen monitor and a foot-pedal option for image acquisition. More recently, Optovue obtained clearance by the FDA for its iStand, a novel, multidirectional mounting stand that allows imaging of patients in the supine position, a modification that may enhance intraoperative imaging.

The drawbacks stem from the handheld nature of the device. The iVue proves fairly bulky and heavy when attempting to image a patient in the supine position. However, the new iStand will address most of these shortcomings. This dual utility allows for perioperative image comparison.

Holekamp Diamond Dusted Soft Tip Drip Dropper with Fluted Bulb:

This unique design combines the Drip Dropper with the popular ILM polisher thus eliminating an instrument exchange. The fluted bulb acts like a standard handpiece providing reflux and passive aspiration. The instrument is available in 20G, 23G, 25G, and 27G.

Benefits:

Ultra-low, non metallic bushing design, Low force, Short stroke, Low glare, Throughlumen for cleaning, infusion/aspiration of fluids, Onepiecetips react to every movement of handle. Handle is completely round when the instrument is fully closed

iQ™ NanoTapered™ Awh Forceps

Features much larger grasping platforms to minimize membrane shedding. Maintains see-to-the tip design for ILM and general membranes. This forceps introduces the Katalyst Nanotapered™ design which allows the tips to close first, yet maintain the large grasping surface. The instrument is available in 20G, 23G, 25G, and 27G.

My experience of using these new instruments was excellent. The following link of the surgical video could demonstrate the same. = http://www.youtube.com/watch?v=d0X14U5WZ08

References:

1. Arnd Gandorfer, Matthias Rohleder, Charanjit Sethi, Dominik Eckle, Ulrich Welge- Lu¨ssen, Anselm Kampik, Philip Luthert, and David C h a r t e r i s . P o s t e r l o r Vitreous Detachment Induced by Microplasmin. Investigative Ophthalmology & Visual Science, February 2004, Vol. 45, No. 2
2. Pravin U. Dugel, Anti-PDGF, anti-VEGF combination may be game changer in wet AMD treatment. Ocular surgery news, June 2012.
3. Combining anti-PDGF with anti-VEGF may improve visual acuity, reduce AMD treatment burden: A phase 2b clinical trial found better results with combination therapy than with anti-VEGF monotherapy. OSN Retina, July 2012
4. Jordi Mones, Inhibiting VEGF and PDGF to Treat AMD: Drugs that combine regression of CNV with resolution of permeability in wet AMD m a y e n h a n c e visual outcomes. Review of Ophthalmology, Sept 2011.
5. S. K. Steven Houston, Audina M. Berrocal, Timothy G. Murray. Intraoperative OCT: An Update on Current Technology. An evaluation of three devices surgeons can use during procedures. Retinal Physician, Jan.2011
6. Huang LL, Hirose T. Portable optical coherence tomography in management of vitreoretinal diseases: current developments, indications, and implications. Semin Ophthalmol. 2012 Sep;27(5-6):218-25

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Dr. Manish Nagpal MS, DO,FRCS
Editor

Incidence of rebleed following 23 gauge TSV for vitreous hemorrhage due to vascular etiologies.

Dr Manish Nagpal, Dr Sidharth Bhardwaj

Pars plana vitrectomy (PPV) is a well-established treatment for vitreous hemorrhage due to various causes. Traditionally, most vitrectomy surgical systems utilize the 20-gauge instruments; however the newly developed 23-gauge system allows for small incision, self-sealing, sutureless transconjunctival pars plana sclerotomies. This offers a number of potential advantages including decreased surgical trauma, less postoperative inflammation, and faster postoperative recovery time. Eliminating suturing may also shorten total operating time.

Early postoperative vitreous hemorrhage may occur due to residual blood clots in the peripheral vitreous, iatrogenic injury and incomplete removal of fibrovascular tissue. A common cause of recurrent hemorrhage is new vessel growth at the inner sclerostomy sites associated with fibrous traction which has been demonstrated on UBM or 20-MHZ highresolution anterior-segment ultrasonography.

The incidence of POVH in patients undergoing 20 gauge pars plana vitrectomy (PPV) for vitreous hemorrhage in proliferative diabetic retinopathy (PDR) is around 9 to75%[1-3] However, there are no studies till date to report exclusively the final outcomes of eyes that undergo sutureless vitrectomy using 23 gauge for vitreous hemorrhage exclusively due to vascular etiologies.

The purpose of our study was to study incidence of rebleed following 23 gauge TSV and interventions done for the same.

Materials and Methods

After institutional board review approval, charts of patients who underwent 23 gauge TSV by a single surgeon (MN) over a 5 year period for vitreous hemorrhage due to vascular etiologies were reviewed.

Data abstracted from these charts included patient age, gender, date of operation, indication for surgery, operative eye, visual acuity (VA) (preoperative visit, postoperative day 1, 30, 90 and 180), intraocular pressure (IOP) (preoperative visit, postoperative day 1, and monthly postoperative visits), phakic status (phakic, cataract, pseudophakic, aphakic), anterior and posterior segment findings, preoperative laser photocoagulation and anti-VEGF injections. Ultrasonography findings were noted in eyes which had a preretinal hemorrhage that obscured clear fundus visualization. Detailed history of co-incidental and past systemic and ocular pathologies and procedures was noted. Data regarding the onset and treatment of recurrent vitreous hemorrhage, and postoperative follow-up were also compiled.

Presence of co-incidental ocular pathologies (e.g. glaucoma, uveitis, retinal degenerations and dystrophies etc.) and retinal detachment, and blood dyscrasias associated with abnormal coagulation were considered as exclusion criteria. Subjects that underwent silicone oil injection or simultaneous vitrectomy with any other surgery were also not included in the study. Any patient who required sutures to be taken for one or more ports at the end of surgery was also excluded.

All the patients were operated under peribulbar anesthesia. The operative technique was 3-port 23 gauge PPV using self-sealing sclerotomies. Immediately prior to making the incisions, the eye was washed with a jet of saline and a few drops of povidone iodine drops were instilled to address conjunctival flora. The Trocar Fixation plate (pressure plate forceps) from Asico (Westmont, IL) was used so as to stabilize the globe while making the biplanar incision (Figure 2). The pressure plate forceps have an incorporated caliper to measure distance from the limbus apart and have serrations on the undersurfaces, allowing a good hold on the conjunctiva for misalignment over the proposed scleral entry. Sclerotomies were created between 3.5 and 4 mm posterior to the limbus for pseudophakic and phakic patients respectively in the superonasal, superotemporal, and inferotemporal quadrants. Initially, the blade was inserted obliquely into the sclera at an angle of about 30^{o} to 45^{o} up to the cannula mark. Then, the direction of the blade was adjusted perpendicular to the sclera as it is inserted into the vitreous cavity. The biplanar incision not only holds the cannula in place but also prevents egress of fluid in the postoperative period.

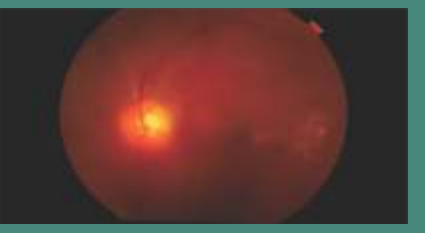

Figure 1. Vitreous hemorrhage in proliferative diabetic retinopathy

Figure 2. Figure showing the creation of biplanar incision using the Trocar Fixation plate

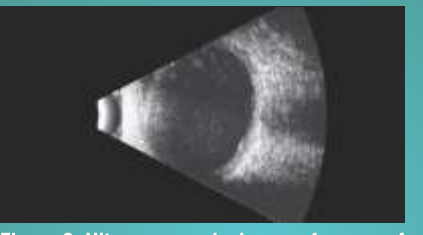

Figure 3. Ultrasonography image of a case of rebleed in a vitrectomised eye

Figure 4. Revision vitrectomy in a case of rebleed

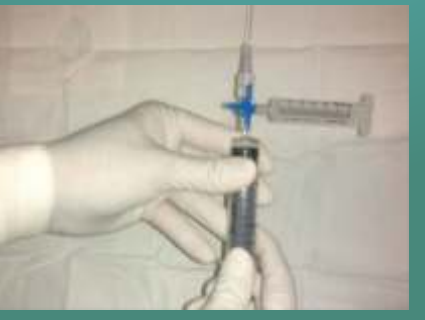

Figure 5. Technique of air blood exchange

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Chart 1. Incidence of rebleed in eyes with preparatory Avastin

Chart 2. Incidence of rebleed in eyes with preoperative PRP

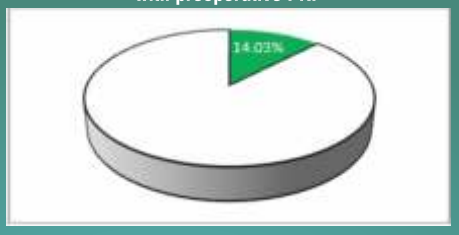

Table 1. Mean visual acuity following vitrectomy

	Based on 20 ft.	Logmar acuity
Preoperative	20/2000	2.0
1 Month Postoperative	20/320	1.2
6 Month Postoperative	20/120	0.78

Table 2. Interventions for rebleed

No of eyes	14
Vitrectomy and Endolaser PRP	7
Vitrectomy and Endolaser PRP with intravitreal Bevacizumab	1
Air blood exchange	6

Table 3. Underlying etiology in eyes with rebleed

Vitreous hemorrhage due to PDR	13 (92.85%)
Vitreous hemorrhage due to vein occlusion	1 (7.15%)

Table 4. Visual acuity following interventions in eyes with rebleed

	Preoperative	1 month postoperative	6 months postoperative (logmar)
Revision Vitrectomy (n= 8)	20/2000	20/320 (1.2)	20/200 (1.0)
Air blood exchange (n=6)	20/2000	20/400 (1.3)	20/225 (1.05)

After core vitrectomy, wherever indicated posterior vitreous detachment was induced followed by peripheral vitrectomy. The areas of active neovascularization were recognized and cauterized before dissection. In order to tackle the intraocular bleeding, IOP was raised instantly to control intraocular bleeding. If required, meticulous endodiathermy was carried out using the unipolar cautery. Supplementary laser panretinal photocoagulation extending beyond the level of the equator was performed.After the vitrectomy, the infusion pressure was lowered to 15 mm Hg and cannula plugged to prevent egress of fluid. The wound area was massaged with a blunt tip applicator for 10 to 15 seconds which allow a better sealing of the scleral fibers and prevents any inadvertent vitreous incarceration. A drop of povidone-iodine is then instilled followed by a subconjunctival antibiotic injection in the inferonasal quadrant.

All patients with recurrent or non-clearing vitreous haemorrhage on 1 month follow up underwent Ultrasound B scan to rule out a retinal detachment (Figure 3). Usually they were asked to follow up with conservative management for another month (2 months from surgery). At this stage intervention in the form of air blood exchange or re vitrectomy (Figure 4) was carried out in eyes with persisting haemorrhage. Eyes which showed low intensity uniform echoes on ultrasound suggestive of vitreous haemorrhage underwent air blood exchange while those where there were multiple dense echoes or any signs of proliferations or membranes were posted for revision vitrectomy.

Air blood exchange was done through the pars plana, done in the operating room using a 24 G needle attached to a three way cannula and 10 cc syringe (Figure 5). Air was first injected, followed by drainage of some fluid passively; this was repeated until the entire vitreous cavity is filled with air, at which the needle is withdrawn.

Snellen VAs were converted into logarithm of the minimum angle of resolution (logMAR). The paired t test was used to compare means with a statistical significance threshold at $P < 0.05$.

Results

A total of 119 eyes of 119 patients were identified that met the inclusion criteria. The mean age was 50 years, and 89 patients were male and 30 female. Vitreous hemorrhage due to proliferative diabetic retinopathy (95 eyes; 79.83%)(Figure 1), retinal vein occlusion (15 eyes; 12.60%)and vasculitis (9 eyes; 7.56%) were the three most common indications for surgery. Sixty four eyes (53.78%) had history of laser panretinal photocoagulation done previously, whereas preparatory intravitreal anti-VEGF injections were administered in 18 eyes (15.13%). Follow-up in all patients was for a minimum of 6 months with no patients lost during this period. Mean pre-operative visual acuity was 2.0 logMAR which improved postoperatively to 1.2 logMAR at 1 month and 0.78 logMAR at 6 months (Table 1). The improvement in VA at postoperative review was statistically significant ($P < 0.05$).

Rebleed was noted in seventeen patients of whom 3 cleared spontaneously within 2 months of surgery while 14 patients required intervention for non-resolving postoperative vitreous hemorrhage. Revision 23g TSV was performed in 8 eyes, while 6 patients underwent air blood exchange (Table 2). Of the 14 eyes with non-clearing vitreous hemorrhage (NCVH), the underlying etiology was proliferative diabetic retinopathy in 13 eyes (Table 3). 50 % of these had undergone panretinal photocoagulation previously (chart 1) and 3 patients had undergone preparatory anti-VEGF (chart 2).

All the patients had clear media and attached retina at the end of final follow up.

In eyes that underwent revision 23 gauge vitrectomy, the final postoperative VA was 1.0 logMAR at 6 months while those undergoing air blood exchange had a final VA of 1.05 (Table 4).

There were no instances of postoperative hypotony, endophthalmitis, rhegmatogenous retinal detachment and neovascular glaucoma in this series.

Discussion

Recurrent VH is one of the most common sequels of vitrectomy. Incidence rates of postoperative vitreous hemorrhage after vitrectomy for proliferative diabetic retinopathy vary substantially in the literature. Schachat et al and Novak et al reported incidence rates of immediate postoperative vitreous hemorrhage of 75 % and 63 %, respectively and recurrent vitreous hemorrhage of 29 % and 23 %, respectively[1,2]. However, both these reports were published more than 20 years ago and predate modern vitrectomy instrumentation and techniques.

A 10 year retrospective analysis of patients undergoing 20G pars plana vitrectomy from January 1999 to May 2010

by Gupta et al for tractional retinal detachment (TRD) and nonclearing vitreous hemorrhage (NCVH) secondary to PDR in 346 eyes had an incidence of 9.2 % patients requiring intervention for non-clearing post vitrectomy hemorrhage[3].

Ivastinovic et al in a randomized clinical trial compared 23-gauge and 20-gauge vitrectomy for diabetic vitreous hemorrhage (VH) regarding postoperative VH (PVH), re-operation rates, visual acuity (VA) and safety profiles[4]. Though the final visual acuity averaged 0.68 logmar in the 23-gauge group, the incidence of early (20%) and late (22.2%) postoperative vitreous hemorrhage was higher as compared to our study (11.76%). In their series postoperative hypotony was observed in 8.9% of cases and rhegmatogenous retinal detachment occurred in one eye.

Conclusion

The incidence of postoperative vitreous hemorrhage in our study was 11.76%, which appears to be comparable to reported incidence following 20 gauge vitrectomy.Recently with an advent of 23 & 25 gauge vitrectomy, there has been speculation amongst surgeons on whether the incidence of rebleeds is higher whilst using small gauge surgery and the concern is probably due to the higher reported incidence of post-operative hypotony in sutureless wounds[5]. In our series there was no case of hypotony noted and hence the overall rebleed incidence is not very different from the existing reports on 20 gauge vitrectomy. Proper valvular technique to place the cannulas at the beginning of surgery and ensuring an air/water tight closure at the end of surgery would reduce the incidence of hypotony which could indirectly reduce the rebleed. Of course a meticulous surgery with a good clean up of the haemorrhage and adequate endolaser remains the mainstay of the approach.

The resultant Vas of the patients in this study appears to be comparable to those of other recent reports. However, strict comparison of these studies is difficult because of different etiologies and variables with each of them.

REFERENCES

1. Schachat AP, Oyakawa RT, Michels RG, Rice TA. Complications of vitreous surgery for diabetic retinopathy. II. Postoperative complications. Ophthalmology. 1983 May; 90:522- 530.
2. Novak MA, Rice TA, Michels RG, Auer C. Vitreous hemorrhage after vitrectomy for diabetic retinopathy. Ophthalmology. 1984; 91:1485-1489.

   Gupta B
3. Wong R, Sivaprasad S, Williamson TH.Surgical and visual outcome following 20-gauge vitrectomy in proliferative diabetic retinopathy over a 10-year period, evidence for change in practice. Eye 2012
4. Ivastinovic D, Velikay M, Haas A, El-Shabrawi Y, Neuwirth R, Ardjomand N, Wedrich A. 23- gauge versus 20-gauge vitrectomy for diabetic vitreous hemorrhage.
5. Woo SJ, Park KH, Hwang JM, et al. Risk factors associated with sclerotomy leakage and postoperative hypotony after 23-gauge transconjunctival sutureless vitrectomy. Retina. 2009;29(4):456-63

UPCOMING CONFERENCES

ISOPT : The International Symposium on Ocular Pharmacology and Therapeutics
March 7-10, 2013
Paris, France
Conference Coordinator :
Dr. Lyat Shahal
Email: isopt@isopt.net
Website: http://www.isopt.net

2013 Annual Symposium and Congress of theAmerican Society of Cataract and Refractive Surgery (ASCRS)
19 - 23 April 2013
San Francisco, CA, USA

Dr Lingam Gopal

Coloboma of choroid: An Overview

Coloboma of choroid is a congenital anomaly that has wide spectrum of manifestations. It is caused by the defective closure of the embryonal fissure. Most cases have been seen to be sporadic and seem to be caused by an intra-uterine insult during the first trimester of pregnancy. Maternalmalnutrition, infections, exposure to x-rays etc can all be potentially responsible for the occurrence.

During embryogenesis, the optic vesicle developsthe foetal fissure ventrally as an invagination oriented anteroposterioly. The fissure permits entry of the mesenchyme that ultimately results in development of retinal vasculature. The fissure h normally closes by 6 week of gestation- supposedly starting from the centre and proceeding anteriorly and posteriorly. Depending on the timing and severity of the intra uterine insult, the severity of colobomatous defect can vary. While bilaterality is common- especially in the more severe varieties, coloboma of the fundus can manifest as an unilateral condition. The mildest manifestation is in the form of forme fruste lesions. These are inferred to be aborted colobomatous lesions by virtue of some characteristics.

1. Dipping of the ora serrata posteriorly in the infero nasal quadrant.

2. Patches of what look like chorio retinal atrophy in the infero nasal quadrant.

In most cases these lesions have been identified in the fellow eye when one eye has frank coloboma of the fundus or the same eye has coloboma of the disc and the periphery has the lesions described above. An optic pit that is located infero nasally on the disc and sometimes even temporally located could be seen in eyes with other features of fundal coloboma suggesting the aetiological relationship of the optic pit to defective foetal fissure closure.

Coloboma of the fundus can be associated with coloboma involving anterior segment structures such as the iris, ciliary body and the zonules. Although most severe fundus colobomata have associated iris coloboma, the association is not always present. The fundus coloboma itself presents several variations in the theme. Although embrologically the tissue first affected is the retinal pigment epithelium due to the defective closure of the fissure, secondarily the choroid and the neural retina are also affected. The sclera is also often found thin in this area and may be ectatic. In addition to the obvious and expected affect on the structure and function of the retina in the area of the coloboma, there is also a global affect on the development of the eye itself. Very often these eyes are microphthalmic. The patients also present with nystagmus – if the lesion is bilateral and severe.

Studies have shown that the normal retina continues as the intercalary membrane in the area of the coloboma. This transition can be sudden or gradual. At the junction, another important change that takes place is the split of the neural retina. The outer layer turns back and merges with the retinal pigment epithelium- an area termed 'locus minoris resistantiae' by Schubert. It is the inner that continues as the intercalary membrane. Mostly this is a fibrotic membrane that merges with the floor of the ectatic coloboma. However the part of the intercalary membrane close to the margin of the coloboma can have normal or near normal layering of the retina as seen in studies with optical coherence tomography.

From the functional standpoint, the involvement of the optic disc and more importantly the macula in the coloboma dictates the visual acuity. Eyes with large fundal coloboma but with the macula spared can have near normal visual acuity. Macula, although located outside the coloboma can sometimes be affected by chorioretinal atrophy and pigmentation and can result in subnormal vision. Vision can also be affected by amblyopia in unilateral or asymmetric cases since very often there can be considerable refractive error.

From the anterior segment surgeon's stand point, two features are noteworthy. The crystalline lens is disproportionately larger compared to the corneal diameter in the microphthalmic eyes with coloboma and they seem to develop fairly hard nuclear cataracts at an early age compelling one to perform extra capsular cataract extraction rather than phaco emulsification. While this is not always the case, the occurrence is common enough to be kept in mind. Hence, to deliver the nucleus, one may need to make a rather large limbal incision- on occasion greater than 180 degrees- since the circumference of the limbus is small in microphthalmic eyes and a smaller incision does not permit the delivery of the relatively large nucleus. One should also be cognizant of the fact that the scleral rigidity is affected by the thin sclera in the area of the coloboma and this can also complicate the surgery if lens delivery is by the extra capsular extraction. Extreme microphthalmos with cataract is a challenge to manage. Eyes with corneal diameter of 5-6 mm are difficult to operate upon by the anterior segment route. The often present peripheral corneal haze compounds the difficulties by reducing the area of visible anterior chamber.But these patients might be having navigational vision till the time of development of the cataract and could potentially benefit from removal of the cataract. Pars plana surgery may be a better option in such cases with extreme microphthalmos (using the fragmatome in case of harder nucleus).

UPCOMING CONFERENCE

World Ophthalmology Conference – APAO 2014 Congress
Period: April 2-6, 2014
Venue:
Tokyo International
Forum, Imperial Hotel, Tokyo

Organizing Societies
Host:
Japanese Ophthalmological Society (JOS)
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Asia-Pacific Academy of Ophthalmology (APAO)
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ARVO: 2013 Annual Meeting
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More often than not eyes with coloboma land up with the posterior segment surgeon due to the high incidence of retinal detachment (almost 40% life time risk). Retinal detachment in eyes with colobomata could be aetiologically related to the coloboma or an incidental occurrence. If the retinal detachment is due to peripheral break and the retinal detachment is not encroaching on to the coloboma, one can safely perform the usual management of the retinal detachment, ignoring the presence of the coloboma. However one should be fairly certain that the retinal detachment is not encroaching on to the coloboma. While this is easy to verify in eyes with good fixation and near normal size, it could be difficult to impossible in eyes that are significantly microphthalmic and with associated nystagmus, complicated cataract etc.

Eyes with retinal detachment attributable to the coloboma are best managed by vitreo retinal surgery where the majority of the decisions are taken intra operatively. Features unique to colobomatous eyes that dictate the progress of the vitreous surgery are:

1. The small eye and so the adjustment one needs to make in performing the sclerotomies (closer to limbus than usual)
2. Relatively rigid superior sphincter in eyes with iris coloboma hampering the visualization of the superior fundus. This can be managed by placing iris hooks to retract the superior pupil
3. Lens sacrifice very often may be needed –especially in relatively microphthalmic eyes.
4. Vitreous is often not detached and may need special effort to detach the same from the retina. Forceps peeling from the disc seems to work better than suction alone. This step is perhaps the most important and could dictate the success or failure of the surgery. It is these violent manoeuvres that can cause more and more communications to develop between the sub intercalary membrane space and sub retinal space at the 'locus minoris resistantiae'.
5. Management of any associated proliferative vitreo retinopathy is like in any other case of retinal detachment
6. Identification of breaks in the inter calary membrane is only of academic interest since the treatment is not directed to closing these.
7. Fluid air exchange with removal of vitreous fluid from the fundus (without endo drainage) can often elucidate the exact contribution of various factors to the occurrence of the retinal detachment. A ballooning of the retina around the coloboma indicates lack of communication between sub retinal space and vitreous cavity through the coloboma and should clearly indicate that the cause of the retinal detachment is elsewhere (peripheral break). Mere presence of peripheral break does not exclude the contribution of the coloboma in the causation of the retinal detachment. Where the coloboma is contributing to the retinal detachment, there is a break in the inter calary membrane and there is also a communication between the sub retinal space and sub inter calary membrane space by way of breaks in the locus minoris resistantiae. While the breaks in intercalary membrane are visible intra operatively (even if they are not visible preoperatively), the location of breaks in the locus minoris resistantiae are not clinically decipherable and are more of an inference based on the behaviour of the retina on fluid air exchange. In most of these cases on infusing air into the vitreous cavity and removing the vitreous fluid, the retina settles well since the sub retinal fluid is pushed out into vitreous cavity through the two communications described above.
8. Endolaser is done all along the border of the coloboma since one is never sure of the location of the breaks in the locus minoris resistantiae. Diode laser is preferred to reduce risk of nerve fibre layer damage, especially when the treatment has to go round the optic disc as in cases with disc coloboma and choroidal coloboma.
9. Silicone oil tamponade is preferred since a large area needs to be tamponaded. The success of reattaching detached retina in these eyes with coloboma has improved tremendously with the advent of vitreo retinal surgery compared to the era of scleral buckling. Most series have reported 80-90% results of reattachment of retina.

UPCOMING CONFERENCES

IIRSI – CHENNAI
6-7 JULY 2013

5th World Glaucoma Congress (WGC-2013)
July 17 - 20, 2013
Vancouver Convention Centre,
Vancouver, Canada
Phone: +31 (0)20 679 3411;
Fax:+31 (0)20 673 7306
wgc-2013-info@mci-group.com
http://www.worldglaucoma.org

31st Annual ASRS Scientific Meeting
August 24 - 28, 2013
Sheraton Centre Toronto
Toronto, Canada

13th EVRS Meeting
September 07-10, 2013
RODOS Palace Conference Center
RHODES – GREECE
INNOVATIONS AND
TECHNOLOGICAL ADVANCES IN RETINA






Dr. N S Muralidhar
Convener, Scientific Committee

Message from Convener Scientific Committee

Dear Friends,

Yet another VRSI newsletter is in your hands to coincide with the annual meeting of the VRSI in Guwahati. I congratulate Dr Natarajan and Dr Manish Nagpal and their team for this effort. The contents are very topical and I am glad to see the article on Prof Tewari, our teacher. Dr Harsha Bhattacharjee and Dr Satyen Deka are working hard to make the Guwahati meeting successful This year's annual meeting will see a different scientific program from the previous years. Come and participate in the meeting and enjoy the hospitality.

With regards,

DR N S Muralidhar

Dr. A. Giridhar
Honorary Secretary

Message from Honorary Secretary

Dear Colleagues,

Greetings from the Governing Council of Vitreo Retinal Society-India! We are bringing out the third newsletter for the year 2012.

We have successfully conducted one continuing medical education program this year at Patna which was organized by Dr Shalabh Sinha and it was a great success. VRS-I is very keen to hold such continuing medical education programmes in other parts of the country for General and Comprehensive Ophthalmologists to update them on the recent developments in the management of various vitreo retinal diseases and also to train Post Graduate students in basic diagnostic procedures in medical retina like fluorescein angiography, optical coherence tomography, etc. It will be nice if members come forward to organize such programmes in various parts of our country. This matter can also be discussed in the forthcoming annual conference of the VRS-I at Guwahati.

VRSI CME at Patna

The VRSI CME was attended by 70 ophthalmologists from Patna and neighbouring cities. The meeting was inaugurated By Shri Shatrughan Sinha MP Patna and senior ophthalmologist and President Patna Ophthalmological Society, Dr. P. C. Gupta. The guest faculty included Dr. Cyrus Shroff and Dr. Lalit Verma from Delhi and Dr. Saurav Sinha from Kolkata. Local faculty included Dr. Pooja Sinha, Dr. Nagendra Prasad, Dr. Subhash Prasad, Dr. Deepak Agarwal, Dr. Anshuman Sinha and Dr. Shalabh Sinha. It was an interesting meeting which had retinal detachment and vitreous hemmorhage as topics. The meeting was interactive and the local ophthalmologists were happy with such a close interaction with the stalwarts in retina. A positive feedback wasavailable from all quarters.
The meeting was held in Hotel Chanakya, Patna.

Dr. Dhananjay Shukla

Maculopathy secondary to optic disc pit: my experience

Dhananjay Shukla, MS, MAMS
Medical Director & Vitreoretinal Consultant, Centre for Sight, Ludhiana

What is OD pit and how does it affect macula

Optic disc (OD) pits are congenital excavations on the optic nerve head. They are typically sporadic, unilateraland temporal in location, which contrasts them with optic nerve colobomata occurring In line with embryonic fissure. Optic pits become symptomatic when maculopathy develops; temporal pits are most often associated with macular serous detachment or schisis (25-75% of cases)(Figure 1)[1].

Evolution of understanding about optic pit maculopathy

Retina specialists a decade back described optic pit maculopathy as central serous retinopathy, a term synonymous with macular serous detachment then, notwithstanding the brilliant description of macular schisisby Lincoff and colleagues, without the aid of optical coherence tomography (OCT)[2]. The macular schisis is now considered central to the development of maculopathy: the splitting starts at the macule edge of the pit, involves multiple retinal layers simultaneously, with some fluid gaining access to subretinal space. Though the origin of the fluid causing maculopathy is not established, an increasing majority of researchers, includingus, believe that synchitic vitreous enters the dysplastic area of pit and dissects through papillomacular retina. [5-7]Vitreomacular traction, both anteroposterior and tangential, appears to play a major role in the passage of fluid into macula [5,6,8].

Differential diagnosis

The appearance of a pit is generally characteristic. However, there are two potential "pitfalls:" pits can be acquired in high myopia[9] and in normal tension glaucoma[10,11] …and optic pits can sometimes be "occult", i.e. the resultant schisis is visible but the causative pit is not[12]. The high myopia is clinically obvious, but differentiation is tricky in the glaucomatous eyes: the key findings are normal-sized discs (the optic disc is enlarged in presence of congenital pit), and the location of pit along the vertical poles[10,11]. The occult pit can be revealed by the spectral domain OCT. The main importance of knowing about the occult pit is that if a case of central serous chorioretinopathy shows no obvious leakage, one should look at the OCT carefully. If schisis is more prominent than the detachment and begins nasally, an occult pit is the likely cause (Figure 2).

Natural history of optic pit maculopathy

Sobol & colleagues followed up 15 eyes of 15 patients with OD pit and serous detachment for an average period of 9 years. [13]They reported that 80% of the eyes end up with a vision of 20/200 or less - in spite of spontaneous resolution of fluid in 26% eyes - largely due to retinal pigment epithelial degeneration, inner retinal cysts, and formation of lamellar and full-thickness macular holes. The visual loss occurred within 6 months of the serous detachment. Other authorities confirm the poor visual prognosis in untreated optic pit maculopathy [1,5].

How and when to treat optic pit maculopathy

Though early treatment appears to be an intuitive choice from the poor prognosis, the issue becomes vexed when the patient is a child, the vision is excellent, and the duration of schisis is not known. The need or choice of treatment in such cases is not clear from the literature. Since the surgical prognosis is good (see below) in young-to-middle aged adults, who apparently have chronic maculopathy, I prefer to wait and follow up young children and adults with excellent visual acuity (6/6-6/9).

Once the treatment is indicated by progressive visual loss with corresponding increase in maculopathy, treatment options include barrage laser photocoagulation of the optic pit, pneumatic retinopexy, macular scleral buckling, and vitrectomy. Postel and colleagues compared the anatomical success of these procedures. [14]They found that laser demarcation was not much better than natural history; pneumoretinopexy succeeded in two-thirds of the cases; while buckling and vitrectomy were successful in nearly all the cases. Currently, small gauge vitrectomy is the default option for vitreoretinal surgeons due to relative difficulty and invasiveness of posterior scleral buckling. Vitrectomy is however performed in several combinations with gas tamponade, internal limiting membrane (ILM) peeling, and endolaser barrage. Since a comparative trial of all possible combinations is probably impractical, several authorities recommend a maximal approach for optimum results. [1,5,14]We agree with this rationale and have reported excellent

Figure 1 A: A 22-year old man with optic disc pit left eye and chronic macular schisis

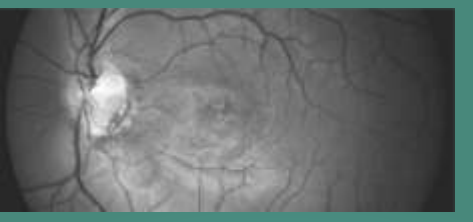

Figure 1 B: OCT of the same eye showing the pit *, macular schisis, & serous detachment

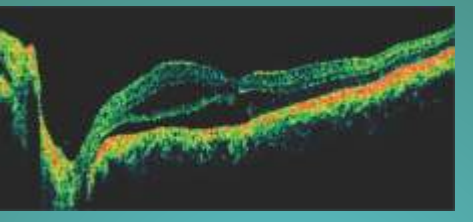

Figure 2A: Apparently typical central serous chorioretinopathy OD

Figure 2 B: OCT reveals an occult optic pit *, and macular schisis, more prominent nasally

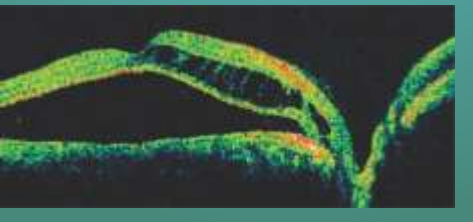

Figure 3A-D: Vitrectomy for OD pit with schisis

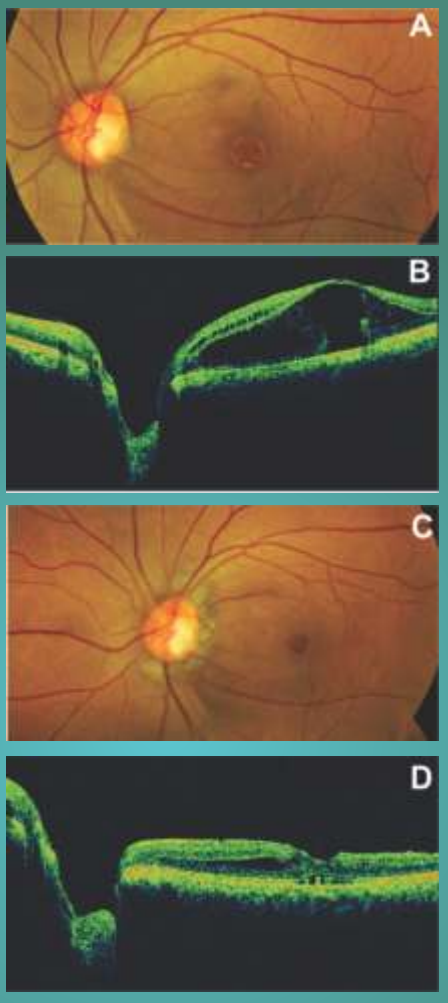

Figure 3A-D caption:

- (A) This 28-year-old woman had a pre-operative vision of 6/24 OS. A macular hole was clinically suspected.
- (B) OCT reveals a large optic pit & an outer lamellar hole with severe attenuation of foveal retina.
- (C) Two months postoperatively, best-corrected visual acuity improved to 6/9. Barrage to the disc margin was performed intraoperatively.
- (D) OCT shows considerable resolution of schisis and restoration of central foveal photoreceptor thickness. Note the compaction of retinal layers nasally due to barrage.

anatomical and visual outcomes in cases with poor prognosis by combining laser, ILM peeling and gas tamponade with vitrectomy. [15]Though there is a real risk of iatrogenic macular hole, especially in presence of attenuated central retina, ILM peeling is rarely the cause of hole formation; it rather helps in subsequent closure of the hole and improving the final outcome. [15]We peel ILM up to the edge of the disc, and drain fluid through only the pit, where possible; additional retinotomy for endo-drainage is redundant and potentially harmful[6,7,15].

An equally important aspect is to remember when not to treat optic pit maculopathy. Besides the young child, good vision scenario explained above, very chronic maculopathy, old patients (50-60 years), extensive chorioretinal degeneration, and full-thickness retinal cysts constitute relative contraindications forme.

Summary

Optic disc pit maculopathy is a relatively rare, unilateral, sporadic condition; there are no systemic associations. Acquired pits should be ruled out in presence of glaucoma; and an occult pit should be kept in mind when approaching a case of central serous chorioretinopathy with no clear-cut angiographic leakage. Treatment is required in all cases where visual decline is documented, though treatment is seldom urgent. Vitrectomy is the definitive treatment; maximal approach with laser, gas tamponade, and ILM peeling gives optimum results with minimal re-interventions.

Acknowledgements

The clinical case studies and personal publications quoted in this article are the derived from the author's tenure as a Professor & Consultant at the Retina-Vitreous Service of Aravind Eye Hospital & Postgraduate Institute, Madurai.

References

1. Brodsky MC. Congenital optic disk anomalies. SurvOpbtbalmol 1994;39:89-l 12.
2. Lincoff H, Lopez R, Kreissig I, et al. Retinoschisis associated with optic nerve pits. Arch Ophthalmol 1988;106:61–67.
3. Rutledge BK, Puliafito CA, Duker JS, et al. Optical coherence tomography of macular lesions associated with optic nerve head pits. Ophthalmology 1996;103:1047–1053.
4. Imamura Y, Zweifel SA, Fujiwara T, et al. High-resolution optical coherence tomography findings in optic pit maculopathy. Retina 2010;30:1104–1112.
5. Sadun AA. Optic disc pits and associated serous macular detachment. In: Ryan SJ, Schachat AP, eds. Retina. 4th ed. Vol 2. Philadelphia, PA: Elsevier Mosby; 2006:1883–1889.
6. Ehlers JP, Kernstine K, Farsiu S, Sarin N, Maldonado R, Toth CA. Analysis of pars plana vitrectomy for optic pit-related maculopathy with intraoperative optical coherence tomography: a possible connection with the vitreous cavity. Arch Ophthalmol. 2011;129(11):1483-1486.
7. Shukla D. Endodrainage of macular schisis thorough optic disc pit. Arch Ophthalmol 2012 Jun;130(6):808-9.
8. Besada E, Barr R, Schatz S, Brewer C. Vitreal pathogenic rolein optic pit foveolar retinoschisis and central serous retinopathy.ClinExpOptom 2003;86:390–398.
9. Ohno-Matsui K, Akiba M, Moriyama M, et al. Acquired optic nerve and peripapillary pits in pathologic myopia.Ophthalmology 2012;119:1685-92.
10. Radius RL, Maumenee AE, Green WR. Pit-like changes of the optic nerve head in open-angle glaucoma. Br J Ophthalmol. 1978;62:389-93.
11. Kiumehr S, Park SC, Syril D, Teng CC, et al. In vivoevaluation of focal lamina cribrosa defects in glaucoma. Arch Ophthalmol. 2012;130:552-9.
12. Spaide RF, Costa DL, Huang SJ. Macular schisis in a patientwithout an optic disk pit optical coherence tomographic findings.Retina 2003;23:238–40.
13. Sobol WM, Blodi CF, Folk JC, Weingeist TA. Long-term visual outcome in patients with optic nerve pit and serous retinal detachment of the macula. Ophthalmology 1990;97:1539–1542.
14. Postel EA, Pulido JS, McNamara JA, Johnson MW. The etiology and treatment of macular detachment associated with optic nerve pits and related anomalies. Trans Am Ophthalmol

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RETINA an insight
Vitreo-Retinal Society - India

Dr Rupesh Agrawal

Controversies in Ocular trauma classification

Dr Rupesh Agrawal, Dr Mehul Shah, Dr Sumita Sharma

Introduction: Ocular trauma is a subject of debate and controversy and there is nothing absolute in ocular trauma. Controversy persists over the optimal management of the injured eye. Ocular trauma classification & score is also notwithout any controversy and it's been challenged by numerous authors. Management of anterior segment trauma has its own share of debate related to patching of corneal abrasion, suture removal in cases with corneal laceration and timing of cataract surgery and intraocular implantation. There are unresolved controversies related to posterior segment ocular trauma. Almost half of the patients with posterior penetrating injuries are left with severe visual impairment and no light perception in significant proportion of patients. Debate persists over surgical repair of severely traumatized eyes with no light perception. There are varied opinions on use of prophylactic antibiotics either systemic or intracameral or intravitreal, prophylactic cryopexy and prophylactic scleral buckling. The role and timing of vitrectomy surgery in severe ocular trauma also remain topics of considerable debate, as does the role of vitrectomy in the management of magnetic intraocular foreign bodies. Management of orbital fractures and lid & adnexal trauma is also area of considerable debate. The international classification system considers ocular (globe) trauma and does not take into consideration lids or orbital trauma and hence one wonder if it should be revamped and renamed to ophthalmic trauma rather than ocular trauma. Role of observation, intravenous methylprednisolone and optic canal decompression in traumatic optic neuropathy is still controversial. Pediatric ocular trauma management also involves lot of unresolved debates. Clinicians are challenged about decision making in surgical repair of severely traumatized eyes with limited or nil visual prognosis in view of potential risk of sympathetic ophthalmia. Following article will dwell only with controversies pertaining to ocular trauma classification.

Ocular trauma classification controversies:-

Landmark for Zone III open globe injury: International Ocular trauma classification group have defined zone III injuries as those extending beyond 5 mm of limbus (Figure 1).[1] Based on the justification given by ocular trauma classification group, they have arbitrarily taken it as 5mm as injuries in that zone may not extend into pars plana and hence any injury not involving zone III or pars plana region are predicted to have favorable prognosis. However, based on literature search, the anteroposterior length of the ciliary body and hence the position of the pars plana varies considerably with the length of the eye. It is always longer on the temporal side and longest inferotemporally, the most expanded quadrant of the eye (Fig.2).[2] Temporally it is 5.6 to 6.3 mm in length, and nasally 4.6 to 5.2 mm.[2] Of importance surgically is the distance from the external corneoscleral limbus to the end of the ciliary body at the ora serrata which marks the beginning of the pars plana. This may be roughly estimated as 7 mm temporally and 6 mm nasally, since in 200 autopsy eyes the slightly shorter distance from Schwalbe's line internally to the end of the ciliary body at the ora serrata averaged 6.53 mm temporally, 5.73 mm nasally, 6.14 mm superiorly, and 6.20 mm inferiorly.[3] This distance of ora serrata from corneoscleral limbus almost coincides & overlaps with Spiral of Tillaux, the imaginary line along insertion of all rectus muscle (Figure 2).[3] Knyazer et al have identified high risk factors associated with posterior open globe injuries resulting in poor visual outcome.[4] The significant factor which was noted from the study done by us was the extent of wound affecting the final vision outcome and its correlation with insertion of the rectus muscle or 'spiral of Tillaux or annulus of Zinn'. The radial scleral lacerations extending beyond recti insertion were often associated with poorer outcome because of comorbid factors such as retinal detachment, vitreous traction and significant vitreous loss. We have also noted favourable outcome in cases with zone III injuries not extending beyond 'annulus of Zinn' as highlighted in Figure 1. So it will be more scientific & prudent to define landmark for zone III injury with reference to rectus insertion (Spiral of Tillaux) rather than conventional definition of injuries extending 5mm beyond limbus.

Preoperative visual acuity of no light perception and poor visual prognosis: Visual acuity can be profoundly impaired to the extent of no light perception (NLP) in presence of significant media opacity (e.g. corneal edema, hyphema, cataract, dense vitreous hemorrhage), retinal detachment, associated subretinal or subhyaloid hemorrhage, hemorrhagic choroidals and even psychological factors (e.g. hysteria). Assessment of light perception is a subjective measure and not a full proof test in the presence of severe media opacity secondary to dense vitreous hemorrhage, traumatic cataract, dense hyphema and corneal edema.[5,6] Assessment of light perception even with the bright light of an indirect ophthalmoscope can give false impression of NLP.[7] Ultrasonography is useful for assessment of posterior segment in the eyes with media opacity and to differentiate between retinal detachment and vitreous hemorrhage, but it is sometimes difficult to differentiate a detached retina from blood clots in the vitreous cavity or membranes.[8] Before deciding on enucleation in patients with NLP, reversible causes of vision loss should be excluded

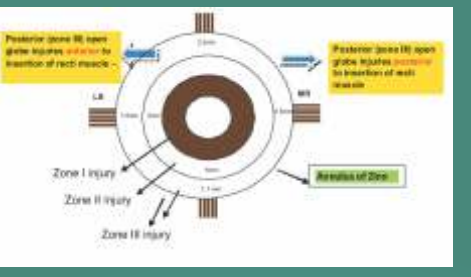

Figure 1: Diagramatic representation of extent of wound in relation to recti insertion

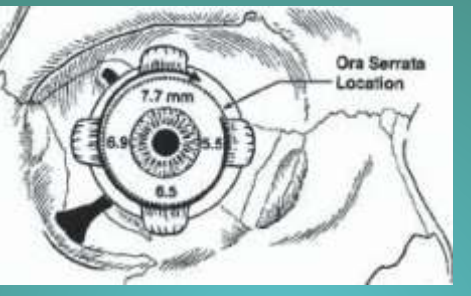

Figure 2: Anterior view of the right globe: Spiral of tillaux is shown with superimposed location of the ora serrata.

Adapted from Duanes Ophthalmology, Volume 2; Chapter 21: Orbital anatomy and its applications.

including psychological factors.[5,6] Even in situations in which enucleation seems inevitable, the ophthalmologist should discuss the possible options with the patient before making a final decision. Primary enucleation for severely traumatized eyes with NLP in view of risk of sympathetic ophthalmia was a controversial approach. Sympathetic ophthalmia with potential for bilateral blindness is a relative indication for enucleation of an injured eye.[5] Most reported cases (65%) occur between 2 weeks to 2 9 months after injury and is rare during the first 2 weeks after trauma. However the actual rate of post-traumatic sympathetic ophthalmia is not clear, and reported rates vary from 0.28% to 1.9%.[5,9] The use of modern immunosuppressives has also improved treatment and control of sympathetic ophthalmia. As such primary surgical repair should not be abandoned for the risk of sympathetic ophthalmia in eyes with NLP. Currently, most surgeons recommend globe salvaging procedure for eyes with severe trauma with no light perception vision at initial presentation.

Relative Afferent Pupillary Defect (RAPD): There is enough evidence that RAPD as an indicator of damage to the optic nerve or retina, may be falsely positive in the presence of severe hyphema or subretinal vitreous hemorrhage and may disappear after resorption or removal of the hemorrhage.[5,6,10] Based on this fact, presence of RAPD as a single poor prognostic factor may not be appropriate and as there is always a possibility of reversal of RAPD, it needs to be equally weighed with other preoperative variables following ocular trauma.

Conclusion: Despite all the advances in state of art surgery and understanding of ocular trauma, a variety of unresolved controversial issues remain in the management and treatment of open-globe injuries. Over last two decades, all these controversies remain standstill and there is no real progress in definitive management of ocular trauma in many aspects. Timing of vitrectomy and issues related to traumatic cataract seem to be never ending debate. Although a controlled, prospective clinical trial would be ideal, but in ocular trauma no two cases are alike and there are many confounding factors which can affect the final outcome. It is
difficult to control for the significant differences that occur with each individual injury. This makes it virtually impossible to independently assess potential risk factors or treatment variances for visual and anatomic outcome. Current management is based on surgeons experience and will continue to be based on retrospective review of accumulated data, and by personal preferences of the treating ophthalmologist. These types of management problems are dealt with on a case to case basis, and even most experienced ophthlamologists at some time or other will found themselves in dilemma in strategic planning of ocular trauma management. This report has attempted to provide comprehensive overview of most of the controversies pertaining to the classification of open globe injuries, and it has presented our observation and comments. This should aid ophthalmologists in the patient counseling and decision-making process regarding management of open-globe injuries involving anterior or posterior segment.

References:

1. Pieramici DJ, Sternber P Jr, Aaberg TM Sr, et al. A system for classifying mechanical injuries of the eye (globe). Am J Ophthalmol 1997;123;820-31.
2. Straatsma BR, Landers MB, Kreiger AE: The ora serrata in the adult human eye. Arch Ophthalmol 1968; 80:3.
3. Sherman DD & Lemke BN (1997): Orbital anatomy and its clinical applications. Tasman W Jaeger EA eds. In: Duane's Clinical Ophthalmology (Vol. 2). Philadelphia: Lippincott Raven. Chapter 21:13–20.
4. Knyazer B, Levy J, Rosen S, Belfair N, Klemperer I, Lifshitz T. Prognostic factors in posterior open globe injuries (zone-III injuries). Clin Experiment Ophthalmol. 2008 Dec; 36(9):836-41.
5. Morris R, Kuhn F, Witherspoon CD. Management of the opaque media eye with no light perception. In: Alfaro DV III, Liggett PE, eds. Vitreoretinal Surgery of the Injured Eye. Philadelphia: Lippincott-Raven; 1999:113–124.
6. Striph GG, Halperin LS, Stevens JL, et al. Afferent pupilary defect caused by hyphema [letters]. Am J Ophthalmol 1988;106:352–353.
7. Abrams GW, Kington RW. Falsely extinguished bright light flash electroretinogram. Its association with dense vitreous hemorrhage. Arch Ophthalmol 1984;100:1427–1429.
8. Rabinowitz R, Yagev R, Shoham A et al. Comparison between clinical and ultrasound findings in patients with vitreous hemorrhage. Eye 2004; 18: 253–256.
9. Makley TA Jr, Azar A. Sympathetic ophthalmia. A long term follow up. Arch Ophthalmol 1978; 96:257–262.
10. Tabatabaei SA, Soleimani M, Alizadeh M, Movasat M, Mansoori MR, Alami Z, Foroutan A, Joshaghani M, Safari S, Goldiz A. Predictive value of visual evoked

UPCOMING CONFERENCES

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CCH Hamburg, Germany
26 - 29 September 2013

XXXI Congress of the ESCRS
4 - 8 October 2013
Amsterdam RAI, Amsterdam, Netherlands

UP State Ophthalmological Society Annual Conference
25 - 27 October 2013
Jhansi(Orchha),
Uttar Pradesh
Chairman Scientific Committee UPSOS:
Dr D Nath
Geeta Netra Chikitsalaya
Sirsaganj, U P
Email: dr.dnath@yahoo.com
Local Organizing Chairperson Conference 2013:
Dr Prakash Gupta
Dr Jiya Lal Memorial Hospital Pvt Ltd
Opp Medical College Jhansi, U P

2013 AAO American Academy of Ophthalmology Annual Meeting
16 - 19 November 2013
Ernest N. Morial Convention Center – New Orleans, LA, USA

72nd Annual Conference of All India Ophthalmological Society
2014
Agra, Uttar Pradesh, India
Dr. SK Satsangi
Professor,
Department of Ophthalmology,
SN Medical College, Agra
Email: sksatsangi@gmail.com

A Tribute to a Living Legend : Prof. Dr. Hem Kumar Tewari

"Great men are not born great, they grow great . . ."
- Mario Puzo, The Godfather

Prof. Dr. H.K. Tewari - an outstanding Retina Surgeon born on 14th July 1942, who graduated from Maulana Azad Medical College, New Delhi in 1965. He did his Post Graduation from All India Institute of Medical Sciences, New Delhi in 1968. Thereafter he joined as a faculty at the AIIMS and worked in different capacities at Rajendra Prasad Center for Ophthalmic Sciences as: Registrar 1970- 73, Lecturer 1973-80, Assistant Professor 1980-1987, Additional Professor 1987- 1992 and Professor from 1992-2001. He is Senior consultant and Vitreo-retina surgeon at Sir Ganga Ram Hospital and Centre for Sight New Delhi.

He was the Chief of R.P.Centre from 2001 – 2004 and then Dean (Academics) at AIIMS from 2002-2004. He has also been the Honorary Adviser, Armed Forces Medical Services; Fellow of the National Academy of Medical Sciences; Diplomat National Board of Examinations; Director, WHO Collaborating Centre for Prevention of Blindness; President of Ophthalmic Research Association at Dr. Rajendra Prasad Centre for Ophthalmic Sciences; Vice Chairman, South Delhi District Blindness Control Society; Secretary General All India Ophthalmological Society; Vice President, All India Ophthalmological Society; President All India Ophthalmological Society. He has been Principal Investigator for Indo-US Project 'Development and Testing of Indian Visual Function Assessment Questionnaire' and ICMR Project "Prognostic Markers in Retinoblastoma immuno Histochemical Study" and Coordinating Investigator for the study titled "A Multicenter, Randomized, and Double-Masked Controlled Study to Evaluate the safety and Efficacy of an Intravitreal Fluocinolone Acetonide implant".

Prof. Tewari has been awarded Mohan Lal Wig Gold Medal in 1978 for best clinical research, WHO Fellowship for training in Intravitreal Surgery in USA in 1978, P. Siva Reddy Gold Medal Oration 1989, Indira Bai Khare Oration by IMA Nagpur 1989, C.N. Shroff Oration 1994, Sat Research Award by Nagri Eye Research Foundation Ahmedabad 1996, National Integration Award by Himotkarsh Selection Committee, Himachal Pradesh 1997, Excellence Award by Rotary International, Allahabad 1998, Jamshed Wania Oration held at Ahmedabad 1998, Dr. S.R.K. Malik Oration 1999, Distinguished Service Award during the 17th Asia Pacific Academy of Ophthalmology Congress held at Manila, Philippines 1999, Vitreo Retinal Oration Award by Vidharbha Ophthalmic Society 2001, Certificate of appreciation by Delhi Ophthalmological Society 2002.

He has over 175 scientific publications in reputed national and international journals i.e. in Orient. Arch. Ophthalmic, Acta Ophthalmologica, Afro Asian Journal of Ophthalmology East. Arch. Ophthal, Trans. Asia Pacific Acad. Ophthal, Indian Journal of Ophthalmology, Afro. Asian Journal of Ophthalmology, Annals of Ophthalmology, Australian & New Zealand Journal of Ophthalmology, Excerpta Medica, Ophthalmic Surgery, National Medical Journal of India, Medical Journal of Armed Forces, Indian Journal of Ophthalmology, Diabetologia, India Pediatrics, Delhi Journal of Ophthalmology etc.

Prof. Tewari has written textbooks on (i) Manual of Vitre-Retinal Surgery, (ii) Indirect Ophthalmoscopy, (iii) Lasers in Ophthalmology, (iv) Manual of Fluorescein Angiography and contributed Chapters in many books. He has organized and conducted many Workshops, Symposia, Updates and Instruction courses in various specialties of Ophthalmology especially in Retina in all parts of the country and a Community "Reach-in programme" was launched at Dr. R.P. Centre for patients from rural and urban slum areas for Cataract Surgery at Dr. Rajendra Prasad Centre. Moreover he has established a Retina Lab at Dr. Rajendra Prasad Centre for Ophthalmic Sciences AIIMS, New Delhi.

In view of his vast academic and clinical experience he has been invited to be a Guest Speaker/Speaker/Chief Guest/Chairman/ Co-Chairman/Panelist in various national and international Congress/ Conference/Updates and other Continuing Medical Education Programmes.

Convocation ceremony AIIMS

Taking Over as Chief RPC

with APJ KALAM

With DR Gholam Peyman

with Dr S S Hayreh

He has been awarded ·

- **ACHIEVEMENT AWARD by AMERICAN ACADEMY of OPTHALMOLOGY 2007**
- **LIFETIME ACHIEVEMENT AWARD by ALL INDIA OPHTHALMOLOGICAL SOCIETY 2008**
- **LIFETIME ACHIEVEMENT AWARD by SOUTH ASIAN ACADEMY OF OPHTHALMOLOGY 2008**
- **LIFETIME ACHIEVEMENT AWARD by VIDHARBA OPHTHALMIC SOCIETY 2009**

He is a life member of All India Ophthalmological Society, National Society for the Prevention of Blindness, Delhi Ophthalmological Society, Indian Medical Association, Delhi Medical Association and Member of American Academy of Ophthalmology, Indo-Japanese Federation, Nepal Ophthalmological Society, New York Academy of Sciences, and American Diabetes Association etc.

He has always been in the forefront in the specialties of Medical and Surgical Retina. He has trained specialists in his field who are heading some of the prestigious institutions in the country and abroad. He has special interest in Medical Education and has designed teaching curricula for Undergraduates and Postgraduates, Paramedics and Postdoctoral teaching. He is national cocoordinator of the Working Group on "Vision 2020" the right to Sight, Govt. of India.

Teaching for Prof.H.K.Tewari has always been equivalent to lighting the fire and not just filling the pail. Above all he is one of the most respected teachers in Ophthalmology who has always cared for the betterment of the students as a community. His students simply adore him.

Dr. S. Nataraja Pillai
(1900 - 1977)

NATARAJA PILLAI ORATION 2012

Govt. Ophthalmic Hospital, Madras (Estd. 1819)

The Governing Council of the VRS I is charged to select, every year, a Vitreoretinal surgeon of international repute, who has performed "An experimental or clinical work which gives a new contribution to the field of Vitreoretinal Surgery". This honour, the "Nataraja Pillai Oration", is awarded annually during the Annual Conference of the Vitreo Retinal Society - India.

Dr. Subramanya Nataraja Pillai was born on March 1st 1900 and studied at St. John's Coll ege, Palayamkottai, Tirunelveli District, Tamil Nadu,during the British rule in India. During the period 1938-1942, he was trained at the Govt. Ophthalmic Dr. Subramanya Nataraja Pillai was born on March 1st 1900 and studied at St. John's Coll ege, Palayamkottai, Tirunelveli District, Tamil Nadu,during the British rule in India.During the period 1938-1942, he was trained at the Govt. Ophthalmic Hospital (Estd. 1819), Madras, the 2nd oldest Eye Hospital in the world, second only to Moorfields Eye Hospital, London, UK, by none other than the legendary Lt. Col. R.E. Wright, an authority on Tropical Ophthalmology and the person who started the Museum in Ophthalmology at GOH, Chennai.

The hospital, now known as the Regional Institute of Ophthalmology - Govt. Ophthalmic Hospital (RIO-GOH), is also where his son – Dr. N. S. Sundaram, obtained his M.S.(Ophthalmology) and later went on to become the Director & Superintendent of RIO - GOH during the period Nov 1984 - Jan 1987. Later, true to family tradition, his Grandson – Dr. S. Natarajan too trained at RIO - GOH from 1982-84.

Dr. S. Nataraja Pillai pursued the L. M. P. Course at Tanjore Medical School and joined Govt. Medical Service as Sub assistant Surgeon in Tirunelveli in the earlier period. He had great interest in hunting and also represented the college hockey team. As per the then British laws, one had to serve 3 years ($1^1/_2$ years - jail duty and $1^1/_2$ years - Agency duty) and the person would be posted at his native place during the last five years of service. Therefore, Dr.S.Nataraja Pillai served all the three years of service as agency duty at Boipariguda in Koraput, Garjam, Dt.(Orissa); which was in the then Madras Presidency. After the Agency duty, he was posted as Sub - assistant Surgeon at GOH, Madras in 1938 as a Pathologist.

He studied and obtained Licentiate in Ophthalmology (L.O.) in 1939 and continued to serve in all the departments of GOH till 1943. He worked in Govt. Erskine Hospital, Madurai from 1948 to 1951 and then continued private practice till his demise at Madurai. An Ophthalmic surgeon par excellence, h e was one of the pioneers to conduct eye camps at Virudhun agar and Dindugal with the help of TVS family. He was a member of LIONS Club and worked as a Hon. Magistrate for two terms. He was blessed with two sons, two daughters and 14 grand children.

This year, the Governing Council of the VRSI unanimously decided to award the prestigious "Nataraja Pillai Oration 2011" to none other than world famous vitreoretinal surgeon, innovator, scientist, teacher, leader & visionary Dr. Carl Claes.

2001:
Dr. Neil E. Kelly,
California, USA.
Topic: "Genesis, Evolution & Revolution of Macular Hole Surgery."

2003:
Prof.Suresh Chandra,
Wisconsin, USA.
Topic:
"Update in Macular Degeneration."

2005:
Dr. John R Heckenlively
Topic:
"Autoimmune Retinopathy, CAR and MAR Syndromes."

2006:
Dr. Vinod Lakhanpal ,
Baltimore, USA
Topic:
"Prevention and Management of Massive Suprachoroidal Hemorrhage."

2007:
Dr. Baruch D. Kuppermann,
California, USA
Topic:
"Ocular Drug Delivery Systems and their Clinical implications."

2008:
Frank Koch M.D,
Frankfurt am Main, Germany
Topic: "Intrectomy - A true cost effective23 gauge sutureless PPV approach" & "Reality and virtual reality in modern Vitreo Retinal Diagnostics and Surgery."

2009:
Prof. Dr. med. Claus Eckardt, Germany
Topic:
"Advantages and Limits of Small Gauge Trans - conjunctival Vitrectomy."

2010:
Jerry A. Shields,
MD Frankfurt am Main, Germany
Topic:
"What's new and interesting in intraocular tumors."

2011:
Dr. Carl Claes, Belgium, Germany.
Topic:
Pediatric Vitrectomy

Dr. J K Ambati

Dr. J K Ambati

Dr. Ambati is Professor of Ophthalmology & Visual Sciences and Professor of Physiology at the University of Kentucky School of Medicine. He serves as Vice Chair of the Department of Ophthalmology & Visual Sciences and holds the Dr. E. Vernon Smith & Eloise C. Smith Endowed Chair in Macular Degeneration Research. Dr. Ambati was born and raised in India, the elder son of Muralimohan Rao, an Indian Institute of Technology (IIT)-educated mathematician, and Gomathi Rao, a scholar in Tamil literature. Dr. Ambati's group has made pioneering contributions in age-related macular degeneration and ocular angiogenesis. He was trained as an electrical engineer at The Johns Hopkins University and performed surgical and research retina fellowships at the Massachusetts Eye and Ear Infirmary. His laboratory has reported numerous seminal advances in journals such as Nature, Cell, Nature Medicine, New England Journal of Medicine, PNAS, and JCI, and collaborates extensively with his brother Bala at the University of Utah. He has been elected as a Fellow of the American Association for the Advancement of Science. He was the first ophthalmologist to win the Doris Duke Distinguished Clinical Scientist Award, the Burroughs Wellcome Fund Clinical Scientist Translational Research Award, and Ellison Medical Foundation Senior Scholar in Aging Award. He was also the first ophthalmologist to be elected to the Association of American Physicians and the American Society for Clinical Investigation. He has won the Cogan Award and the Camras Translational Award from ARVO, and the Roger Johnson Prize in Macular Degeneration. Research to Prevent Blindness has recognized him with its Senior Scientific Investigator Award, Lew Wasserman Merit Award, and Physician Scientist Award. He serves on the editorial boards of

Ophthalmology, IOVS and Translational Vision & Science Technology, and is a member of The Macula Society and Club Jules Gonin. He and his wife Kameshwari, an IIT-educated chemist, enjoy the scenic beauty of the Bluegrass in Lexington, KY with their daughters Meena and Divya.

VITREO RETINAL SOCIETY OF INDIA

VRSI2013

in Maharahstra

Lavasa- Convention Center – Pune, Maharahstra

Hosted by

Retina Conglomerate

Under the aegis of Poona Ophthalmological Society and Maharashtra ophthalmological society

Retina Conglomerate - Which is formed by 3 prestigious Retina Institutes of Western India, in alphabetic order

- Aditya Jyot Eye Hospital Pvt Ltd, Mumbai
- National Institute of Ophthalmology, Pune
- Retina Foundation, Ahmedabad

Aditya Jyot Eye Hospital was established in 1990 by **Prof. Dr. S. Natarajan** at Dadar, Mumbai. As part of its expansion the hospital shifted to a central location in Mumbai called Wadala. This 4 storey hospital which aims at being - A centre for total eye care under one roof is the biggest eye hospital in Mumbai. Aditya Jyot Eye Hospital strives to provide world class eye care service under one roof by innovative service through research Retina Foundation was established by **Dr Pran Nagpal** in 1976 at Ahmedabad, Gujarat to offer efficient and cost effective comprehensive eye care facilities. Over the past 33 years, the services provided at the Foundation have evolved to keep pace with the rapid developments in ophthalmology in general and in the specialty of Retina and Vitreous in particular. National Institute of Ophthalmology, located in the heart of Pune city, is a specialty eye hospital committed to the delivery of quality eye care. It was founded by **Dr. Shreekant Kelkar** and Mrs. Aruna Kelkar. NIO enjoys national and international recognition. Its state of the art technology, multidisciplinary super specialty approach and dedicated trained experienced personnel cater to complete patient care all under one roof.

The VRSI meet for 2013 would be held at Lavasa International Convention Centre (LICC), Lavasa, Pune, in Maharashtra from 12 -14 December 2013.

Organizing Secretary : Dr Aditya Kelkar, Dr Manish Nagpal and Dr Vinay Prasad.

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*For Management of Visual Impairment Secondary to the Macular Edema

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Treatment is given monthly and continued until maximum visual acuity is achieved, confirmed by stable visual acuity for three consecutive monthly assessments performed while on Lucentis® treatment. • Treatment is resumed with monthly injections when monitoring indicates a loss of visual acuity due to wet AMD, DME or macular edema secondary to RVO and continued until stable visual acuity is reached again for three consecutive monthly assessments. • Lucentis and laser photocoagulation in DME or in branch RVO: Lucentis has been used concomitantly with laser photocoagulation in clinical studies. When given on the same day, Lucentis should be administered at least 30 minutes after laser photocoagulation. Lucentis can be administered in patients who have received previous laser photocoagulation. • Lucentis must be administered by a qualified ophthalmologist using aseptic techniques. Broad-spectrum topical microbicide and anaesthetic should be administered prior to the injection. • The patient should be instructed to self-administer antimicrobial drops four times daily for 3 days before and after each injection. • Not recommended in children and adolescents. **Contraindications:** Hypersensitivity to ranibizumab or to any of the excipients, patients with active or suspected ocular or periocular infections, patients with active intraocular inflammation. **Precautions/Warnings:** • Intravitreous injections have been associated with endophthalmitis, intraocular inflammation, rhegmatogenous retinal detachment, retinal tear and iatrogenic traumatic cataract. Therefore proper aseptic injection techniques must be used. Patients should be monitored during the week following the injection to permit early treatment if an infection occurs. • Transient increases in intraocular pressure (IOP) have been seen within 60 minutes of injection of Lucentis. Sustained IOP increases have also been reported. Intraocular pressure and the perfusion of the optic nerve head must be monitored and managed appropriately. • There is a potential risk of arterial thromboembolic events following intravitreal use of VEGF inhibitors. A numerically higher stroke rate was observed in patients treated with ranibizumab 0.5 mg compared to ranibizumab 0.3 mg or control, however, the differences were not statistically significant. Patients with known risk factors for stroke, including history of prior stroke or transient ischemic attack should be carefully evaluated by their physicians as to whether Lucentis treatment is appropriate and the benefit outweighs the potential risk. • As with all therapeutic proteins, there is a potential for immunogenicity with Lucentis. • Lucentis has not been studied in patients with active systemic infections or in patients with concurrent eye conditions such as retinal detachment or macular hole. • There is limited experience with treatment of patients with prior episodes of RVO and of patients with ischemic branch RVO (BRVO) and central RVO (CRVO). In patients with RVO presenting with clinical signs of irreversible ischemic visual function loss, treatment is not recommended. • Should not be used during pregnancy unless the expected benefit outweighs the potential risk to the fetus. For women who wish to become pregnant and have been treated with ranibizumab, it is recommended to wait at least 3 months after the last dose of ranibizumab before conceiving a child; use of effective contraception recommended for women of child-bearing potential; breast-feeding not recommended. • Following treatment patients may develop transient visual disturbances that may interfere with their ability to drive or use machines. Patients should not drive or use machines as long as these symptoms persist. **Interactions:** No formal interaction studies have been performed. **Adverse reactions:** • **Very common adverse reactions are:** intraocular inflammation, vitritis, vitreous detachment, retinal hemorrhage, visual disturbance, eye pain, vitreous floaters, conjunctival hemorrhage, eye irritation, foreign body sensation in eyes, lacrimation increased, blepharitis, dry eye, ocular hyperemia, eye pruritus, intraocular pressure increased, nasopharyngitis, headache, arthralgia. • **Common adverse reactions are:** retinal degeneration, retinal disorder, retinal detachment, retinal tear, detachment of the retinal pigment epithelium, retinal pigment epithelium tear, visual acuity reduced, vitreous hemorrhage, vitreous disorder, uveitis, iritis, iridocyclitis, cataract, cataract subcapsular, posterior capsule opacification, punctuate keratitis, corneal abrasion, anterior chamber flare, vision blurred, injection site hemorrhage, eye hemorrhage, conjunctivitis, conjunctivitis allergic, eye discharge, photopsia, photophobia, ocular discomfort, eyelid edema, eyelid pain, conjunctival hyperemia, stroke, influenza, urinary tract infection*, anemia, anxiety, cough, nausea, allergic reactions (rash, pruritus, urticaria, erythema).**Uncommon** • **adversereactions are:** blindness, endophthalmitis, hypopyon, hyphema, keratopathy, iris adhesions, corneal deposits, corneal edema, corneal striae, injection site pain, injection site irritation, abnormal sensation in eye, eyelid irritation. • **Serious adverse events** related to intravitreal injections included endophthalmitis, rhegmatogenous retinal detachment, retinal tear and iatrogenic traumatic cataract. ** observed only in the DME population* **Packs:** Pack of 1 vial Before prescribing, please consult full prescribing information available from Novartis Healthcare Private Limited, Sandoz House, Dr. Annie Besant Road, Worli, Mumbai- 400 018, Tel: 022 2495 8888 For the use only of a registered medical practitioner or a hospital or a laboratory only. 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