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Dear Members

Greetings from Delhi Ophthalmological Society!

I wish to thank our members for showing wonderful zest and enthusiasm in receiving all the academic ventures of this executive. We stand humbled by the overwhelming acknowledgement received for our winter DOS 2015 last December, which saw unprecedented pre-registrations, conference attendance, ophthalmic industry participation, delegates’ interaction and scientific program in a new avatar.

The events of the ophthalmic calendar of 2015 – 2016 also featured a very successful DOS Teaching Programme - DOST 2016. I wish to thank all the postgraduate residents who came from far and near to make this programme a wonderful success and relish the realms of ophthalmic teaching at its greatest height.

We also look forward a successful programme of DESK II (DOS Enhanced Subspeciality Korner) scheduled for the 6th of March, 2016 in Glaucoma – Prevention of Blindness in Glaucoma: Current Best Practices which is covering an essentially new angle and focus in glaucoma management during the World Glaucoma Week celebrations.

The forthcoming 67th ANNUAL DOS Conference, now being launched with a new title of DOSCON 2016: OPHTHALMIC PANORAMA is promising to showcase the best academic content and ophthalmic exhibition of the season. This season’s Programme has an assembly of new introductions with sessions of DOS with several national societies’, breakfast sessions, Instruction courses, Symposiums in various subspecialities, Film Festival and Ophthalmic Photography apart from all the other regular features. In an attempt to serve our ophthalmic fraternity better, we also have introduced a new registration structure for the conference so as to minimize wastage of money and food. We sincerely hope that you would throng in large numbers to relish this academic extravaganza.

Be there to behold the rising of the DOSCON 2016: Ophthalmic Panorama, in April 15 – 17, 2016 at the Hotel Ashok, Chanakyapuri, New Delhi, India.

Dr. M. Vanathi
MD
Additional Prof. of Ophthalmology
Cornea, Cataract & Refractive Services
Dr R P Centre for Ophthalmic Sciences
All India Institute of Medical Sciences
New Delhi 110029, India
mvanathi.rpc@gmail.com

DOS Enhanced Speciality Korner – DESK II: GLAUCOMA
Prevention of Blindness in Glaucoma: Current Best Practices
India Habitat Centre, Basement Theatre
March 6, 2016, 8.30 am – 4.30 pm

Registration fee: Rs 500
Please send your details with payment (as cheque or cash) to the DOS OFFICE or
Log on to www.dosonline.org for online registration
Optical Coherence Tomography (OCT) has evolved over the past decade as one of the most important ancillary tests in ophthalmic practice and especially for Retina. It is a non-invasive imaging technique and provides high-resolution, cross-sectional images of the retina, the retinal nerve fiber layer (RNFL) and the optic nerve head. With axial resolution in the 5–7 μm range, it provides close to an in-vivo ‘optical biopsy’ of the retina. In 2006, the first commercially available spectral-domain (Fourier domain) OCT was introduced. SD-OCT now in use employs detection of the light echoes simultaneously by measuring the interference spectrum, using an interferometer with a high-speed spectrometer. This technique achieves scan rates of 20,000–52,000 A-scans per second and a resolution of 3-5 μm in tissue.

New innovations in SD-OCT hardware and software now allow for accurate choroidal thickness measurements. In addition, choroidal morphological changes on OCT are being appreciated. As a result, choroidal imaging is emerging as an emerging area of research. SD-OCT systems can image the choroid, using techniques such as image averaging and enhanced depth imaging (EDI). EDI involves setting the choroid adjacent to the zero delay line, which allows enhanced visualization of choroid up to the sclera. SS-OCT uses another form of Fourier domain detection to measure light echoes. It employs a tunable frequency swept laser light source, which sequentially emits various frequencies in time, and the interference spectrum is measured by photodetectors instead of a spectrometer. This increases the signal quality in deep tissue, thereby improving the visualization of the choroid. The choroid in healthy eyes is thinnest subfoveally and thins nasally more than temporally. In addition, a negative correlation exists between choroidal thickness and age. Margolis et al. and Manjunath et al. reported the mean subfoveal choroidal thickness of 287 ± 76 μm using the Spectralis OCT and 272 ± 81 μm using the Cirrus OCT device. Choroidal imaging is proving to be very helpful in eyes with AMD, CSCR, DME and inflammatory chorioretinal diseases etc.

A new OCT advancement, known as en-face imaging, allows the clinician to visualize three-dimensional data in a fundus projection. Using this technique, particular retinal and/or choroidal layers at a given depth are projected onto an en-face view. Microstructural changes and morphology of the retinal and choroidal vasculature are hard to evaluate using cross-sectional B-scans. This is expected to improve as en-face imaging provides further detail about the subtle pathological features in the retina and choroid in diseased states. A newer introduction is OCT Angiography, (popularly known as dyeless angiography), is a quick and non-invasive procedure which provides volumetric data with the clinical capability of specifically localizing and delineating pathology along with the ability to show both structural and blood flow information in tandem. Its current limitations include a relatively small field of view, inability to show leakage, and tendency for image artifact due to patient movement/ blinking. Published studies show OCTA's potential efficacy in the evaluation of common ophthalmologic diseases such age related macular degeneration (AMD), diabetic retinopathy, artery and vein occlusions, and easily detect the growth of abnormal blood vessels (neovascularization). At our centre, we find it useful in identifying choroidal neovascular membranes in post AMD and even myopic eyes with CNV which otherwise reveal absence of sub-retinal fluid on routine SD-OCT.

Another revolution comes in the form of real-time imaging provides feedback to surgeons to help guide surgical maneuvers via visualization of tissue-instrument interactions, thus came the intra-operative microscope integrated OCT. Use of intraoperative OCT during membrane peeling has been shown to provide crucial feedback, which helps with intraoperative surgical decision-making. Studies have shown that in 13% to 22% of cases, surgeons using intraoperative OCT identified residual membranes that required membrane peeling that would otherwise not have been identified. I also find it immensely useful for identifying ILM flaps in Macular Hole and Optic Nerve Pit - Maculopathy cases.

Thus major breakthroughs in OCT technology have provided scope for enhancement of the understanding, monitoring, progression and response to various treatment modalities employed in chorioretinal diseases. These advancements have revolutionized ophthalmic practice over the last decade.

Prof. Atul Kumar
MD, FAMS
Diplomate N.B.E. (Ophth.)
Vitreoretinal Fellow, University of Maryland (USA)
Hon., Consultant to Armed Forces
Awarded “Padma Shri” by the President
Chief & Professor
Dr R.P. Centre of Ophthalmic Sciences
All India Institute of Medical Sciences, New Delhi-29

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MESSAGE FROM THE PRESIDENT

Dear Colleagues and friends,

The year under review has been an eventful one. The ‘Avastin storm’ hit the ophthalmic fraternity hard. The alert notice by the Drug Controller General of India (DCGI) compelled us to stop using this wonderful drug for our patients. Representatives of AIOS, VRSI and an expert committee set up by the DCGI which included people from Roche, met the Joint DCGI on 8th February 2016. A great amount of time and effort went into preparing a strong and convincing case for withdrawing this notice. After sustained discussion and deliberation, it was decided that the notification would be withdrawn – permitting off label use of Avastin in Ophthalmology. Roche will reintroduce the Kezzler code which enables the genuine Avastin product to be confirmed and the spurious to be detected. Authorized distributors of Roche will be permitted to sell Avastin to ophthalmologists and a list of these will be made available. AIOS-VRSI guidelines have been drawn up for safe use of Avastin and submitted to the DCGI.

Our special thanks to Prof. Atul Kumar, Dr. Lalit Verma, Dr. Debashish Bhattacharya, Dr. Barun Nayak, Dr. D. Ramamurthy, Dr. Tarun Sharma, Dr. Vishali Gupta, Dr. Mahesh Shanmugan, Dr. Ajay Aurora and many others who worked tirelessly and selflessly to gather information, make a water-tight case and present it convincingly at the meeting. A pat on the back of the entire ophthalmic community which united and gave valuable inputs to the expert committee.

Hopefully we will be using Avastin again soon but we should wait for the official notification. This is also a time for us to reflect and resolve to observe self discipline and procure from reliable sources only, use the Kezzler code facility when available, follow guidelines for safe use and always keep medical ethics and the safety of our patients paramount.

Another draconian notification had appeared last year, making it mandatory for all ophthalmic B-scans to be registered under the PCPNDT act and maintain a log of each and every scan performed. We wrote to the Secretary Health, Government of Delhi protesting against this. The IMA had also challenged this legally and in a landmark judgment W.P. (c) 2721/2014: IMA vs Union of India, we have been able to get substantial relief. Non pelvic ultrasonologists are out of PCPNDT act with some riders. Ophthalmologists can also now breathe easy on this score.

For our society, this has been a year of consolidation of our systems and processes and progress on many fronts. The sub-committees have been active and I thank the Chairperson & members for their time and efforts. The Electoral process was reviewed, improvements suggested and loop holes plugged. As promised in the GBM, the Voter List was put online on the website. The Disciplinary & Ethics committee framed guidelines for graded punitive action in case of misconduct by a member. The monthly meeting format was also reviewed and the grading system refined. The monthly meetings have seen consistently good attendance and marked improvement in adherence to time. Thanks to the efforts of the Editor, the DJO is now indexed with Index Copernicus International. The online utilization of the Library has also been excellent, with the slightly unpleasant by product of the service provider wanting a steep hike in charges!

The DOS Annual in April is round the corner & the Secretary, Dr. Vanathi, & other members of the Executive have been working extremely hard to make it a great success. Plans are also underway to have an I-DOS in Sri Lanka around Christmas this year. In December 2017, the 4th World Congress of Pediatric Ophthalmology & Strabismus is slated to be held in conjunction with DOS at Hotel Ashok.

I would like to thank all of you for the privilege of being at the helm of our dynamic society this year. It has been a great learning experience for me, besides giving me the opportunity to visit my Alma Mater frequently. The DOS office staff have worked hard and uncomplainingly at all odd hours and a special thanks to them.

Last but certainly not the least, I thank all members of the Executive for their time, effort and co-operation in taking the society forward.

(Dr. Cyrus M. Shroff)

President
Delhi Ophthalmological Society

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Ocriplasmin (Jetrea; Thrombogenics, Leuven, Belgium), a recombinant truncated form of human plasmin with a molecular weight of 27.2 kDa. It is a laboratory harvested protease with activity against fibronectin and laminin, which are components of vitreoretinal interface. Its approval recently by the US Food and Drug Administration in the treatment of symptomatic vitreomacular adhesion/traction (VMA/VMT), has brought new attention to the field of pharmacologic vitreolysis, with gates open for its potential application in the treatment of macular hole (MH) and diabetic macular edema (DME). Until recently, the only treatment option available for VMT was vitrectomy. The surgical procedure carries the risk of complications, both intraoperative and postoperative, as well as a definite treatment burden for patients and their caregivers, hence limiting the indication for surgical vitreolysis until progression to significant visual loss. Till then, a period of watchful waiting was observed with untreated symptoms for spontaneous improvement, with equal probability at times of worsening enough not warranting favourable prognosis despite surgical intervention. As proved in phase II and III trials, comparing single intravitreal injection of ocriplasmin (0.125mg/0.1ml) with a placebo injection in patients with symptomatic VMA, resolution of VMA at day 28 was seen in 26.5% of test eyes compared to 10.1% of placebo injected eyes ($p<0.001$). Similarly, nonsurgical closure of MH was observed in 40.6% of ocriplasmin treated eyes compared to 10.6% of placebo treated eyes ($p<0.001$). Also noticed in phase 3 trials were certain adverse ocular events like vitreous floaters, photopsia, conjunctival haemorrhage, blurred vision amongst the ocriplasmin treated individuals, though labelled as “transient and mild in severity”. Hence, though proved beyond doubt the immense potential of ocriplasmin in non surgical treatment various vitreo-retinal interface disorders, judicious use, thorough pre treatment discussion, and careful follow up are needed in light of better understanding of the effectiveness and safety of new drugs to provide the best care to our patients. Post marketing surveillance data corroborate findings from the phase III trials, and provide additional insights into the characterization of the safety profile of this new treatment option.

The vitreoretinal interface consists of an adhesive interaction between matrix proteins such as laminin and fibronectin; these proteins act as a kind of glue, attaching the collagen fibrils of the posterior vitreous cortex to the internal limiting membrane (ILM). The natural ageing process results in liquefaction of the vitreous gel and its separation from the retina. Concurrent liquefaction of the vitreous gel and progressive posterior vitreous cortex separation from the retinal surface, ultimately leads to, in most eyes, non pathologic posterior vitreous detachment (PVD). In some cases, however, incomplete vitreo-retinal interface (VRI) separation can result in anomalous PVD with the potential for the development of pathologic features. As defined by the International Vitreo-macular Traction Study Group Classification System, anomalous PVD is a partial vitreous detachment with persistent attachment in the macular region, resulting in tractional deformation of retinal tissue. While, VMA refers to the stage in which there is peri-foveal vitreous detachment with foveal vitreous adherence of 3 mm radius of fovea, and no detectable change in retinal morphology, there are cases where course of disease is as follows:-

Hence, release of traction leads to spontaneous resolution of pseudocysts resulting in improvement in vision. Both VMA and VMT can be subclassified into either focal (<1,500 micro metre) or broad (>1,500 micro metre) adhesions based on attachment size. Focal areas of vitreous attachment with traction tend to distort the foveal surface, whereas broad areas of attachment with traction can cause generalized thickening of macula, vascular leakage on fluorescein angiography, macular schisis, and cystoid macular edema.

Excessive traction on inner retina may also result in the
Progression of PVD

- Periods of excessive traction on the macula lead to distortion of retinal architecture, termed as vitreomacular traction (VMT)
- Vitreo-macular traction (VMT)
- Result in intra-retinal pseudocyst formation, leading to elevation of the fovea from the retinal pigment epithelium (RPE), or a combination of both
- Results in reduced or distorted vision

development of a full thickness macular hole (FTMH), defined as an anatomic defect in the fovea featuring interruption of all neural retinal layers from the internal limiting membrane (ILM) to the RPE. FTMH can have either persistent VMT or complete release of vitreous which can be visualised with the help of an OCT based system, and hence, can help determine appropriate treatment options.

The standard treatment for VMT and MH is a three-port pars plana vitrectomy. Considering the risks and complexity of vitrectomy versus an injection, surgery is usually delayed until a significant visual impairment is detected. Intravitreous injection of vitreolytic compounds (being termed as pharmacologic vitreolysis), has been studied as an alternative treatment, aiming to cause vitreous liquefaction and a complete posterior vitreous separation from retina, as in PVD. In this review, we will be throwing light on the growing popularity of recently approved Ocriplasmin, after having gone through the outcomes of clinical trials judging its safety and efficacy in the management of symptomatic VMA.

Pharmacologic vitreolysis aims at promoting PVD by release of adhesion between vitreous and Internal limiting membrane (ILM)\(^ {14,15} \). It may be used in conjunction with vitrectomy or as a stand-alone therapy\(^ {16} \).

In combined approach with drug assisted surgical vitreolysis, it is aimed to liquefy vitreous along with weakening of vitreoretinal adhesions, hence making way for a faster and easier vitreous removal\(^ {16-21} \). So, it not only cut short the duration of surgery, but also makes it a more efficient one with a smaller gauge and lower aspiration rate approach, besides decreasing the risk of iatrogenic tears\(^ {22-25} \).

Vitreolytic agents can also be employed as a definitive treatment for active VMA-related diseases, such as early stage MH, VMT, and tractional cystoids macular edema\(^ {26} \). In these cases, complete PVD is related better anatomical and functional outcomes while reducing the incidence of progressive disease requiring surgery.

The connection between cortical vitreous collagen with the ILM surface is believed to be mediated by some form of extracellular matrix “glue”, including the proteins laminin and fibronectin\(^ {27-29} \). Uemera et al showed that intravitreous injection of plasmin dramatically increased the density of fibronectin and laminin, reducing the molecules to several fragments of low molecular weights at the ILM.

Apart from cleavage of fibronectin and laminin, plasmin has an additional ability to activate endogenous matrix metalloproteinases (MMPs). MMP2 (gelatnase A), which is normally found in vitreous has an affinity for various collagens, including basement membrane (type IV), and hence is thought to play an important role in vitreous liquefaction\(^ {30-32} \). Hermel et al described a 27% increase in vitreous removal through a 25 gauge vitrectomy system in rabbit eyes injected with plasmin compared to eyes with no preoperative injection.

Studies have demonstrated better visual outcomes with reduction in surgical time and iatrogenic retinal breaks in cases of tractional DME, when plasmin injection was given preceding surgery\(^ {33-36} \).

Successful anatomic outcomes were also described in paediatric patients with stage 5 retinopathy of prematurity, traumatic MH, and complex X-linked retinoschisis cases\(^ {37-39} \).

Ocriplasmin

Ocriplasmin, formerly known as microplasmin, is a bioengineered, recombinant, stable, truncated form of plasmin, a human serine protease, consists of only the catalytic domain from plasmin but has practically the same functional proteolytic activity against fibronectin and laminin\(^ {40,41,43} \). Ocriplasmin, being smaller than plasmin (27KDa versus 83 KDa), is supposedly having increased penetration of the tissues\(^ {42} \).

CLINICAL TRIALS AND THEIR RESULTS

There were a series of clinical trials sponsored by Thrombogenics to investigate the effect of ocriplasmin (earlier known as Microplasmin) in humans.

A phase I/IIa study – (Microplasmin Intravitreal Injection) MIVI-I, and two randomised controlled Phase II clinical trials – MIVI-IT and MIVI-III, were carried out to evaluate dose response along with safety and effectiveness of ocriplasmin. A dose of 125 μg was taken as safe and optimal efficient as patients receiving 125 μg of ocriplasmin were more likely to present PVD than controls. The results of MIVI-IT and MIVI-III indicated the potential of ocriplasmin as a nonsurgical alternative for the treatment of VMT\(^ {44} \).

Following the encouraging results from phase II trials, two phase III clinical trials were initiated by MIVI-TRUST
(Microplasmin for Intravitreal Injection- Traction Release Without Surgical Treatment) to evaluate use of Ocriplasmin for the treatment of symptomatic VMA, MIVI-006, conducted in the United States, and MIVI-007, conducted in both the United States and Europe, were practically identical multicentre, randomized, double masked, placebo-controlled studies designed to test the efficacy and safety of a single dose of 125 μg ocriplasmin injected intravitreally. As mentioned earlier, both the studies were sponsored, coordinated and designed by ThromboGenics.

The inclusion criteria were diagnosis of symptomatic VMA seen on OCT, and a best corrected visual acuity (BCVA) of 20/25 or worse. Exclusion criteria included high myopia, prior vitrectomy, prior macular photoagulation, MH greater than 400 μm, and other retinal pathologies that could affect visual function. There was no exclusion of patients with ERM. In total, 652 patients were enrolled: 464 patients were randomly assigned to receive 0.10 ml intravitreal injection of a solution containing 125 μg ocriplasmin, while 188 patients were randomly assigned to receive 0.10 ml intravitreal injection of a placebo solution containing the identical drug vehicle diluted with saline. The primary endpoint was nonsurgical VMA resolution at day 28, as determined by OCT at a formal reading centre. The main secondary endpoint was total posterior vitreous detachment at day 28, as determined by standardized B-scan ultrasonograms performed by the investigator. Nonsurgical closure of FTMH, BCVA improvement of three lines or more, change from baseline in BCVA, need for vitrectomy, and visual function questionnaire assessment at 6 months were also considered secondary endpoints.

Considering data from both trials, the primary endpoint of pharmacologic VMA resolution was achieved in 26.5% of patients in the ocriplasmin group versus (vs) 10.1% of patients in the placebo group (p<0.001). The VMA resolution rate was higher when VMA diameter was 1,500 μm or less (37.4% vs 14.6%; p<0.001) than in those with VMA diameter greater than 1,500 μm. In patients with ERM, ocriplasmin injection did not have such positive results (8.7% vs 1.5%; p<0.046) compared with VMA without ERM (37.4% vs 14.3%; p<0.001). In those patients with pure VMT at baseline, without ERM or MH, there was a success rate of 29.8% in the group treated with ocriplasmin vs 7.7% in the placebo. Considering FTMH, the closure rate in the ocriplasmin group was 40.6% at day 28 and remained as high at the end of the study at 6 months, whereas the placebo group had a 10.6% (p<0.001) closure rate at day 28 and 17% at 6 months (p=0.004). Closure rates varied according to the FTMH width at baseline.

Summarizing the outcomes from the phase III trials, a single injection of 125 μg ocriplasmin was shown to be safe and to significantly increase the rates of VMA resolution and FTMH closure when compared with placebo. It is relevant to note that the success rate appears higher for focal VMA less than 1,500 μm in diameter and small MH. A relevant drawback of this phase III study was the use of time-domain OCT, whereas spectral domain OCT was used only when available.

Kim et al recently published their initial outcomes on the use of intravitreal ocriplasmin in clinical practice. In a retrospective view of 19 patients with symptomatic VMA treated with ocriplasmin, they found a resolution of the VMA in 42.1% of non surgical cases. Patients with ERM at baseline had a lower success rate of VMA resolution (25%) than patients without baseline ERM (45.5%), which was comparable to the observations of clinical trial.

SAFETY PROFILE OF OCRIPLASMIN

In the pivotal phase III ocriplasmin clinical trials, the proportion of patients who had any ocular adverse event in the study eye was 68.4% in the ocriplasmin group and 53.5% in the vehicle group (p<0.001). Interestingly, this difference was driven primarily by adverse events known to be associated with vitreous detachment, as most common ocular adverse event in the study eye was vitreous floaters, reported by 16.8% of patients in ocriplasmin group and 7.5% of those in the vehicle group. Retinal tears or detachments were diagnosed in 1.9% of the patients given ocriplasmin compared to 4.3% of those given vehicle. Most retinal breaks in both groups occurred during or after vitrectomy; two (0.4%) retinal detachments in the ocriplasmin group and one (0.5%) retinal tear in the vehicle group occurred prior to any vitrectomy.

Most suspected treatment-related ocular adverse events were not serious, mild in severity, and occurred within 7 days after injection. Overall, most cases were transient with, as by the end of the 6 month study, reduced VA was reported in approximately 1% of patients in both the ocriplasmin and vehicle treated groups.

In addition, there were a total of 16 patients of dyschromatopsia (yellowish vision) and 10 patients with ERG changes, majority being reported from two uncontrolled open-label clinical studies (TG-MV-008 and TG-MV-010). Median time to onset was 1 day and median time to resolution was 3 months as far as dyschromatopsia was concerned, with resolution seen in 13 out of 16 cases. As far as dyschromatopsia was concerned, median time to onset was 1 week and median time to resolution being 6 months, with majority of patients showing a trend to recovery of the wave amplitude towards baseline and VA returning to baseline or better.

A recent case series reported changes in the ellipsoid zone following treatment with ocriplasmin, and the authors found that the rate of the ellipsoid zone (inner segment–outer segment zone) following treatment with ocriplasmin, with higher rate of ellipsoid zone loss seen in patients showing resolution of VMT (75% in responders vs 11% in non responders). It was also demonstrated in case series that out of 17 patients treated, ellipsoid zone loss was seen in 7 (41%), which was transient in all cases with mean time to resolution being 29 days.

Hence, periodic reporting plays a major role in the safety and assessment of a drug, as spontaneous reporting allows health authorities to gather safety information on newly available treatment options.

Cost factor:-

The cost of an ocriplasmin injection is £2500 (excluding VAT) (0.5 mg in 0.2 ml solution; MIMS, July 2013). Because repeat injections are not recommended, this is the cost for a full course of treatment. Also, costs may vary in different settings because of negotiated procurement discounts.

CONCLUSIONS

Ocriplasmin is a novel, efficacious treatment option for patients with VMA, as demonstrated by the results from the phase III trials, as well as recently published reports. Safety findings show that ocriplasmin is a well tolerated drug and most treatment related ocular adverse events were not serious, were mild in severity, and occurred within 7 days post injection. Further understanding is needed in some of the
observations that had not been reported in the phase III trials, such as ellipsoid zone changes, identifying which helps categorization of patients most likely to develop particular adverse effects in the treatment process. As, interestingly, ellipsoid zone changes may be a positive predictor of response as they tend to occur more frequently in patients showing VMA release post injection. Still, further studies, OASIS (Ocriplasmin for Treatment of Symptomatic Vitreomacular Adhesion including Macular Hole), ORBIT (Ocriplasmin Research to Better Inform Treatment), INJECT (The Investigation of JETREA in Patients with Confirmed Vitreomacular Traction), etc. are on their way to address the upcoming issues and will provide deeper insight into the treatment patterns.

Overall, till date, both the clinical trial data and the emerging real world experience show that ocriplasmin appears to be an effective and safe treatment option for various posterior segment pathologies, and, hence, provides a new armamentarium in the management of vitre-o-retinal interface disorders.

### REFERENCES

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Advances in the estimation of the eye’s wave aberration and the ability to precisely rectify it with adaptive optics have moved the wave aberration from an abstract academic concept to one of practical value to improve vision. The power of the wave aberration is that this single function completely describes the composite effects of the cornea and lens on light passing through the pupil. Understanding the wave aberration helps to create a perfect image on the retina by manipulating the light entering the pupil. Wavefront sensors have been built that capture as many as 65 components in the wave aberration.

SPHERICAL ABERRATION

Spherical aberration is one of the main wave aberrations that leads to decrease in contrast sensitivity. Thus, although cataract patients had been benefitting (from smaller incisions with phacoemulsification, from advanced instrumentation and techniques for axial length measurement, from appropriate use of advanced intraocular lens power calculations, and from astigmatism management) by attaining uncorrected visions of 20/20 in greater proportions and in shorter postop times, contrast sensitivity was found not to be improved. The need to manipulate spherical aberration derives from the demonstration of the improved clarity of vision (or contrast) under mesopic and photopic conditions, as well as the improved functional performance under night driving conditions.

WHAT IS Q VALUE

A spherical surface has a “Q value” of 0. A surface which is a parabola has the peripheral part of the lens relatively flatter than the centre and so bends the peripheral light rays less, eliminating this spherical aberration. Such a cornea has a negative Q value and has a prolate shape, whereas a parabola has a Q value of -0.5.

The human eye of a young person has a Q value of -0.5, which is made up of the cornea (Q=-0.25) and the lens of the eye (Q=-0.25) added up together. The over 40 age group has a rounding out of the lens, so its Q value becomes near 0. Hence older people have more natural spherical aberration as their Q value is only that of the cornea i.e. -0.5. A spherical surface has a “Q value” of 0. A surface which is a parabola has the peripheral part of the lens relatively flatter than the centre and so bends the peripheral light rays less, eliminating this spherical aberration. Such a cornea has a negative Q value and has a prolate shape. A parabola has a Q value of -0.5. The human eye of a young person has a Q value of -0.5, which is made up of the cornea (Q=-0.25) and the lens of the eye (Q=-0.25) added up together. The over 40 age group has a rounding out of the lens, so its Q value becomes near 0. Hence older people have more natural spherical aberration as their Q value is only that of the cornea i.e. -0.25. Central part of cornea has more curvature than the periphery.

CORNEAL SPHERICAL ABERRATION AND Q VALUE

Corneal spherical aberration and Q value are not the same thing. Spherical aberration describes how a wavefront deviates from the ideal after passing through a refracting surface. In actuality, it is a measure of the effect a surface has on light and is measured in microns. The Q value describes the refracting surface and is a measure of the shape of a surface; it has no units (Figure 1, 2). The shape of a surface does affect spherical aberration. An ideal spherical surface has a Q value of 0.00. A prolate surface has a negative Q value; a parabola is a prolate surface that eliminates all spherical aberration and has a Q value of -0.50. The human cornea has an average Q value of -0.26; it would require a value of -0.25 to eliminate all spherical aberration. The Q value of a young adult crystalline lens is -0.25; thus, the combined value for a young phakic eye results in elimination of spherical aberration. As the lens ages, the Q value changes, and after age 40 is 0.00.

Q value: Best fit ellipsoid (conic constant) to describe apical ratio of change

Spherical Aberration: Depends on the curvature of the surface
Same Q value, with different curvature will result in different visual benefits of correcting higher order aberration
amount of spherical aberration. Spherical aberrations predicts Contrast Sensitivity & is Inversely correlated. Q value and the change of Q value have no relationship with Visual Acuity and Contrast Sensitivity.

After a myopic PRK or LASIK, the Q value becomes positive with increased spherical aberration2. The cornea then has an oblate shape. No normal human cornea is oblate or has a positive Q value. However, all the modern lasers have “blend zones” that smooth off the mid-peripheral “knee” that has a high local Q value and this lessens the induced spherical aberration.

The wavefront characteristics of light can be described in mathematical terms using different systems, including Zernike polynomials and Fourier analysis. Using Zernike polynomials, sphere (defocus) and cylinder (astigmatism) describe the two higher-order aberrations (HOAs) that we measure with phoropters. These aberrations account for approximately 83% of the magnitude of the wavefront of light. Spherical aberration and coma are the next most significant HOAs. Spherical aberration describes the amount of bending that occurs as light passes through a refracting surface, such as the cornea, and compares the relative position of the focal points for the peripheral and central light beams. Positive spherical aberration occurs when the peripheral rays are focused in front of the central rays; this value is expressed in microns.

Spherical aberration is not really a problem with low myopic corrections but can be a problem with some patients having higher corrections e.g. about -5 D. The laser manufacturers are trying to improve the shape of the ablation profile to lessen this problem. All the “custom ablations” done by various lasers have totally “aspheric” profiles that have, in theory, no aberrations2,5,16. However, they can take off more tissue, which can again be a problem with higher corrections as there may not be much to spare.

Studies are suggesting that surgeons arm themselves not only with the Q value, a measurement of corneal shape from corneal topography, but also by measuring the amount of pre-op corneal spherical aberration at 6mm. This value helps to determine the best lens choice3,13,18. Corneal spherical aberration measurement is only available through topographic wavefront aberration analysis. Moreover, the abberrometer used must be able to separate corneal and lenticular aberrations.

With a perfect single refracting surface such as an ellipse, keratometry and Q value (asphericity measurement) could be used to calculate the spherical aberration of that surface. But Dr. Holladay warns against using eye’s keratometry reading and Q-value to derive the spherical aberration of cornea, because the model of an ellipse, which uses the q-value and a central corneal radius, doesn’t accurately match the shape of the real cornea. Cornea has a complex surface that is steeper centrally. If we were to take a -0.26 q-value, which is the average in the population, and a K-reading of 44, also the average in the population, we would come up with +0.18 μm as the cornea’s spherical aberration, which doesn’t match the true population average of +0.27 μm, underestimating it by 35 percent27,37.

The wavefront of the human eye can be measured using wavefront analyzers such as Shack-Hartmann systems and Tracey aberrometers (TRACE; Tracey Technologies, Corp.). Corneal topographers can measure the front surface of the cornea, and this data can be transformed to determine the HOAs of the cornea. By convention, corneal spherical aberration is measured at 6 mm.

**WHAT IS A WAVEFRONT?**

A wavefront is a physical representation of the optical quality of a light beam. The quality of a light beam can be degraded by any imperfect optical element, a lens, a piece of glass, and in the eye, a cornea for example. When the light beam or lens has spherical aberration, it is termed “aspheric” or “aspherical.” Spherical aberration accounts for approximately 83% of the total wavefront error and can cause a blurry vision. Spherical aberration is measured in microns and is typically between +0.15 μm and +0.30 μm for normal eyes.

**Physiologic Q Values for the Human Cornea**

<table>
<thead>
<tr>
<th>Q Value</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 0.05</td>
<td>Severe myopia</td>
</tr>
<tr>
<td>0.05 - 0.2</td>
<td>Mild myopia</td>
</tr>
<tr>
<td>0.2 - 0.3</td>
<td>Moderate myopia</td>
</tr>
<tr>
<td>&gt; 0.3</td>
<td>Severe hyperopia</td>
</tr>
</tbody>
</table>

**Figure 1: Prolate vs Oblate shapes of Cornea**

**Figure 2: Physiological Q values for Human Cornea**

Wavefront mapping is the technology that makes custom laser eye surgery possible by precisely measuring and diagramming the imperfections of an optical system, such as the eye. These imperfections are divided into lower-order aberrations, such as myopia, hyperopia, and astigmatism, and higher-order aberrations that affect night vision. Wavefront-guided technology is used in CustomVue laser eye surgery to correct refractive error and to improve night vision.

**HISTORY OF WAVEFRONT TECHNOLOGY**

Wavefront technology was originally developed for use in astronomy. In the 1900s, an astrophysicist named Johannes Hartmann devised a method of measuring the ray aberrations of mirrors and lenses. The Hartmann test used a metal disk in which regularly spaced holes had been drilled. The disk or screen was then placed over the mirror that was to be tested and a photographic plate was placed near the focus of the mirror. When exposed...
to light, a perfect mirror will produce an image of regularly spaced dots. If the mirror does not produce regularly spaced dots, the irregularities, or aberrations, of the mirror can be determined.

In the 1970s, Dr. Roland Shack and Dr. Ben Platt advanced the concept by replacing the screen with a sensor based on an array of tiny lenslets, thus creating the Hartmann-Shack sensor. In 1978, Dr. Josef Bille of Germany was the first person to use the Hartmann-Shack sensor in ophthalmology. Other wavefront pioneers include Dr. Junzhong Liang and Dr. David Williams who developed a wavefront device that could be used in a clinical setting.

In 1997, Drs. Liang and Williams presented a paper at the Association for Research in Vision and Ophthalmology that discussed the early clinical results attained with the wavefront device. At this time, ophthalmologists and major laser manufacturers, such as VISX, Bausch & Lomb, and Alcon, began to look at the possibilities of wavefront technology for correcting refractive error and to develop their own wavefront analyzers. In 2002, the FDA approved the first wavefront-guided custom LASIK application. Today, there are many integrated wavefront-guided LASIK systems that first generate a wavefront map of a patient’s unique optical imperfections, then send this information to an excimer laser that performs the custom LASIK procedure.

HOW ARE WAVEFRONTS MEASURED?

In ophthalmology, wavefronts are measured by devices called aberrometers. Aberrometers use wavefronts to objectively measure the overall refractive power error of the eye. They do this by mapping how light rays travel through the eye and by providing maps using color gradients to represent magnitudes of the refractive errors, which enables ophthalmologists to locate and possibly correct even obscure imperfections that cause vision defects. As the name indicates, an aberrometer measures aberrations, and an aberration is a vision defect that occurs when light rays are improperly bent (refracted) in the eye. An aberration may occur because of a flaw in the structure of the eye. There are lower order aberrations, sphere and cylinder; and there are higher order aberrations such as coma, trefoil and spherical aberration. Patients who complain of glare, halos, starbursts and poor night driving often have increased higher-order aberration.

WAVEFRONT ANALYSIS AND MAPPING

Wavefront analyzers are used to map aberrations in the eye. Several types of visual imperfections, referred to as lower and higher-order aberrations, exist within the eye and can affect both visual acuity and the quality of vision. Prior to wavefront technology, only lower-order aberrations such as myopia, hyperopia, and astigmatism could be measured and treated. However, these do not account for all potential vision imperfections. Higher-order aberrations can also have a significant impact on quality of vision and are often linked to glare and halos that may cause night vision problems. Wavefront analyzers use a Hartmann-Shack sensor, which maps both lower and higher-order aberrations by projecting waves of light into a patient’s eye and mapping the waves that bounce back through the pupil. A perfect wavefront would be completely flat. When light rays enter the eye and traverse the different refractive indices, the wavefront surface changes, taking on a shape unique to that eye. These variations are called wavefront aberrations. The aberration data is collected and then converted into a treatment formula by using Zernike polynomials, which are also called modes. Each mode describes a certain three-dimensional surface and the Zernike polynomials correspond with ocular aberrations. For instance, second-order Zernike polynomials represent the conventional aberrations such as defocus and astigmatism. Zernike polynomials above the second order represent the higher-order aberrations that are suspected of causing night glare and halos. Zernike polynomials help to simplify the wavefront technology by combining all aberrations into one simple map. This is called Zernike decomposition.

The wavefront analyzer software condenses the wavefront information into a conventional refraction in diopeters as well as in Zernike form. This map is then transferred to the laser, enabling treatment of the patient’s lower and higher order aberrations.

Why should wavefront aberrometry be an essential part of modern eye care practice?

Eye care has known a number of major technological innovations in recent years. Aberrometers have made it possible for the first time to measure higher order aberrations in the clinic. This breakthrough has been a runaway success with refractive surgeons from the outset, and now there is a growing realization in the market that the type of precision and detail offered by aberrometers is increasingly needed in the general practice as well. There is a growing number of new correcting elements based on information coming from aberrometry. Best known are Lasik and IOLs; aberrometers can help making a sharper prescription. Contact and spectacle lens manufacturers are under tremendous pressure to offer custom correction solutions. Aberrometers have been the key enablers for this trend. It is clear that aberrometers are an essential part of the forward-looking ophthalmic practice.

ADAPTIVE OPTICS

Adaptive optics (AO) is a technology used to improve the performance of optical systems by reducing the effect of wavefront distortions: it aims at correcting the deformations of an incoming wavefront by deforming a mirror in order to compensate for the distortion.

Dr. David Williams who directs the Center for Visual Science at the University of Rochester, is the world expert on the structure and function of the eye. Based on his pioneering work, it is possible to cut higher-order aberrations by a factor of 10 or 20, giving them sharper vision, especially night vision. At their best, Williams’ mirrors can correct vision to 20/10, the limit of normal human sight. (This limit is established by the density of cones and rods in the fovea. That density, combined with certain optical laws, means that human vision can’t get better than about 20/10 or 20/8. An eagle sees more sharply than we can because it has better optics and more densely packed cones.)

Let us go through some real case scenarios to understand the concepts.

CASE SCENARIOS

CASE 1 The patient had a previous myopic LASIK and presented with interest in cataract surgery. The iTrace revealed High (+) Spherical Aberration of value of 0.686. Because of the Qvalue of 0.76, IOL of choice for this surgeon will be either ALCON ACRYSOF® IQ offering - 0.17 of the maximum in (-) lenticular aberration correction. Therefore, because of the patients high (+) corneal SA at 6mm, the surgeon determined that the Tecnis will be the planned IOL for this.
CATARACT

Changes in the spherical aberration of mm) cataract surgery causes minimal changes. Small-incision (less than 2.8

In an aphakic eye, the anterior corneal surface and the lens; other sources are the posterior corneal surface and the retina.

SOME PEARLS

In the human eye, HOAs come primarily from the anterior corneal surface and the lens; other sources are the posterior corneal surface and the retina. In an aphakic eye, the anterior corneal surface accounts for 98% of wavefront changes. Small-incision (less than 2.8 mm) cataract surgery causes minimal changes in the spherical aberration of the eye and, for practical terms, can be considered to have no effect.

Measurements of spherical aberrations of the anterior corneal surface have found the average value to be 0.27 μm with a large standard deviation of 0.10 μm. Due to this variation, the value should be measured for each individual patient.

The presence of spherical aberrations can cause glare and halo around lights. The greater the degree of spherical aberration, the greater amount of halo that is induced.

In cataract surgery, targeting emmetropia has a greater effect on Snellen acuity outcome than manipulating spherical aberration. Thus, surgeons should first optimize their formulas for IOL power calculation before adjusting spherical aberration. Aspheric IOLs improve the quality of vision by providing greater contrast sensitivity, not by increasing Snellen acuity. An increase in spherical aberration away from 0.00 causes a decrease in contrast sensitivity.

Using aspheric IOLs improves driving safety due to improved contrast sensitivity. This is particularly evident on nighttime simulation testing, in which up to a 45-foot advantage in stopping distance at 55 mph (88.51 km/hr) can be achieved.

The impact of spherical aberration is dependent on pupil size. For practical purposes, spherical aberration comes into play when pupils are greater than 4 mm; thus, it has the most impact under mesopic or scotopic conditions and in younger patients. Older individuals may have large pupils, so pupils should be measured for each patient if aspheric IOLs are to be used.

The clearest image is provided when the total spherical aberration value for the eye is 0.00. Most of the effect of targeting this value is seen in nighttime lighting conditions.

Refractive error can compensate for residual spherical aberration. Positive spherical aberration causes a myopic shift, and negative spherical aberration causes a hyperopic shift in refraction. Although refractive error is independent of pupil size, spherical aberration is dependent on pupil size; for small pupils, it can be negligible, but for larger pupils it is significant in its effect. Thus, refractive error will compensate for spherical aberration at larger pupil sizes but will introduce defocus at smaller pupil sizes (Figure 4). This information can be used to customize results for individual patients based on the choice of aspheric IOL.

Incisional corneal surgery for astigmatism correction has minimal effect on spherical aberration.

Negative aspheric IOLs have a slightly higher power centrally. For a 20.00 D lens, this power can be 0.50 D greater and, thus, provides some pseudoaccommodative effect. This is one explanation for increased near vision in patients implanted with aspheric IOLs.

Tilt and decenteration affect the performance of aspheric IOLs. Aspheric lenses must be decentered more than 0.8 mm and tilted more than 10° before any effect is lost.

Leaving spherical aberration (positive or negative) in the optical system improves depth of focus, but at the cost of loss of contrast vision. Current strategies involve targeting up to -0.30 to -0.40 μm of spherical aberration in one eye, so as to increase depth of focus without significantly affecting Snellen acuity.

Our refractive surgical goal should be to eliminate or at least reduce all of the optical aberrations of the eye, including spherical aberration. We should remember, however, that it may take 6 to 12 months of neural adaptation for the patient to fully appreciate and exhibit improvement in subjective measures such as visual acuity and contrast sensitivity function. Older presbyopic patients may benefit from a small amount of residual negative spherical aberration in order to achieve better unaided near vision and depend less on readers—a similar compromise as with modern diffractive and refractive multifocal IOLs. Finally, there is no benefit to leaving any positive spherical aberration in the optical system, because it degrades the image and reduces patients’ near vision as their pupils constrict.

Light Adjustable Lens (LAL). LAL is a silicone lens with two C-PMMA haptics. The silicone macromers that are homogeneously distributed throughout the lens are photosensitive to the nearultraviolet wavelength of energy. As such, when energy of this wavelength strikes the lens in specific patterns, it changes the distribution of the macromers and, subsequently the refractive power of the lens. Customized aspheric treatment makes use of the near-ultraviolet illumination’s ability to fine-tune the shape of Lens & induce asphericity. With a specific irradiation
profile, we can induce aberrations in the lens, including spherical aberration,” and thus increase the depth of focus and, as a consequence, may get some intermediate or near vision.

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**Financial Interest:** The author does not have any financial interest in any procedure/product mentioned in this manuscript.
The most common cause of epiphora in children is congenital nasolacrimal duct obstruction (CNLDO). Usual presentation is watering, discharge and matting of eyelashes but dacyrocele (dilated lacrimal sac) formation or acute dacryocystitis can occur. Fortunately, most of the cases resolve by one year of age and lacrimal sac compression is usually advised for children less than a year age. For cases that persist, or those who have recurrent infections or need intraocular surgery, Irrigation and probing (I & P) of nasolacrimal duct (NLD) is done. Most clinicians prefer to wait till 9-12 months of age before considering child for I & P. However, the success rate of probing depends upon the type of CNLDO and other factors such as use of nasal endoscopic guidance (NEG). Nasal endoscopy guidance is important during pediatric NLD probing to identify and manage associated nasal abnormalities.

**CASE 1**

Five-year-old male child presented with persistent symptoms of CNLDO. He had undergone probing once earlier at the age of 15 months elsewhere. On examination, left eye had increased tear meniscus height, regurgitation on pressure over lacrimal sac (ROPLAS) was negative, and Fluorescein dye disappearance test (FDDT) was positive (Figure 1A). He was taken up for repeat I & P with NEG under general anesthesia. After upper punctum dilation Bowman lacrimal probe was passed. Rigid nasal endoscopy with 2.7 mm zero degree telescope, showed the probe lying submucosally along lateral nasal wall and its movement was appreciated beneath mucosa (Figure 1B). Such a variation of complex CNLDO is called “Buried probe.” It differs from usual thin membranous obstruction, which lies at the lower NLD opening. Buried probe identification and management needs special maneuver to bring the probe out of the mucosa under NEG (Figure 1C). Following this syringing was patent and the child is asymptomatic at 4 months follow up.

**CASE 2**

Twelve-month old male child presented with persistent watering and discharge after initial 3 failed attempts of probing elsewhere at the age of 6, 9 and 10 months. There was also history of acute dacryocystitis at the age of 5 months. Examination showed the positive FDDT and positive ROPLAS (Figure 2 A&B). He under went repeat probing with NEG. Nasal endoscopy showed the presence of a cyst at the opening of NLD without visualization of probe (Figure 2C). The findings were suggestive of intranasal cyst at the site of NLD opening. Sickle knife was then used to open the cyst (making two perpendicular incisions, i.e. cruciate incisions).
CASE 3

Eleven-month old male child had persistent CNLDO after initial failed probing twice at the age of 8 and 10 months elsewhere. On examination, tear meniscus was increased, ROPLAS and FDDT were positive on right side (Figure 3A). On nasal endoscopy, there was presence of lateralized inferior turbinate (Figure 3B). Spatula was then inserted beneath the inferior turbinate under NEG and turbinate was medialized (Figure 3C&D). Following this the probe was seen buried beneath the mucosa suggestive of buried probe. Probe was then externalized successfully (Figure 3E). Syringing was patent at the end of the procedure. Child is now asymptomatic at the follow up of 3 months.

DISCUSSION

Kushner divided CNLDO in two types: Simple and complex. Simple CNLDO has thin soft membranous obstruction at lower NLD end whereas complex CNLDO is a distinct variety and encompasses wide anatomical variations, nasal pathologies, or other abnormalities of lacrimal system (Table 1). Nasal endoscopic guidance (NEG) during I & P is important and now its role well established in literature. NEG allows identification and management of various subtypes of CNLDO and increases the success rate of the procedure.

Furthermore, various factors affect the success rate of I & P and poor prognosis is seen with: age > 36 months, bilateral affection, failed conservative treatment, earlier probing, dilated lacrimal sac, intraoperative firm obstruction, proximal obstructions, and physiologic causes.

Current cases highlight the role of NEG during probing. Previous probing failed in all cases because of the failure to
Financial Interest: The authors do not have any financial interest in any procedure/product mentioned in this manuscript.

Table 1: Etiology of Complex congenital nasolacrimal duct obstructions

1. **Bony obstruction** – Complete absence of NLD formation or NLD directed into lateral maxillary bone
2. **Craniofacial syndromes** – Association with Downs syndrome, Crouzon syndrome, Treacher Collins syndrome, Cleft lip/palate, hypertelorism
3. **Buried probe** – Probe lies submucosally along lateral wall of nose and fails to come out of the NLD opening
4. **Lateralized inferior turbinate** – Inferior turbinate lies in close approximation to the lateral nasal wall giving no access to the inferior meatus
5. **Dacryocele without intranasal cyst**
6. **Anlage duct (Lacrimal fistula)**
7. **Multiple blocks** – Stenosis at valvular sites in canaliculus, sac and NLD or sometimes diffuse stenosis of NLD can lead to multiple level NLD blocks
8. **Dacryocele with intranasal cyst** – Pressure inside Dacryocele can be transmitted to NLD and lead to its dilation and nasal cyst formation
9. **Atonic sac** – Longstanding CNLDO can lead to atonicity of lacrimal sac
10. **NLD upto the floor** – Variant in which NLD extends within the bone till nasal floor
11. **NLD into the inferior turbinate** – Caused by misdirection of inferior NLD
12. **Lateral nasal wall hypoplasia** – Lead to non-differentiation of lateral nasal wall structures such as inferior turbinate, inferior meatus or NLD opening

NLD-nasolacrimal duct, CNLDO- congenital nasolacrimal duct obstruction

recognize and treat significant findings. Case 1 had persistent CNLDO after probing because of the presence of buried probe. As mentioned earlier, probe is not visualized in such cases but its movement and mound is appreciated beneath the nasal mucosa on lateral wall. Gupta et al published largest series of buried probe involving 22 eyes and concluded that it is seen in older children and NEG is crucial for its management. They noted anatomical and functional success rate of 91% and 82% respectively with probing with NEG. Associated lacrimal anomalies were noted in 41% cases and included punctal agenesis, incomplete punctal canalisation, distal canalicular stenosis, atonic sac, mucocoele, impacted inferior turbinate etc. Therefore it is important in such cases not only to identify and treat buried probe but also associated anomalies.

In Case 2, there was presence of intranasal cyst on NEG. Leuder noted that intranasal abnormalities of distal NLD consisting of NLD cyst, redundant mucosal tissue at valve of Hasner and translucent flaccid membrane consistently in cases of CNLDO with mucocoele/acute dacryocystitis and in about 6-9% cases of complicated CNLDO. Furthermore they suggested that factors that may predict the presence of intranasal abnormalities are: (1) failure to palpate metal-on-metal contact between Bowman probes and spatula, (2) failure of fluid to irrigate easily after probing. These cases need removal of abnormal tissue at the site of cyst. Larger cysts are adequately managed with cruciate marsupialization. Case 3 showed the presence of lateralized inferior turbinate and buried probe. Inferior turbinate medialization is needed in such cases to expose the NLD opening, prevent false passage and identify other anomalies at distal NLD. It is preferable to label it as lateralized inferior turbinate as true impacted turbinate are quiet rare.

To conclude, Nasal endoscopy guidance is important during pediatric NLD probing to identify and manage associated nasal abnormalities. Its role is more important especially during repeat probing as discussed in the article.

Acknowledgements: Dr Javed Ali, Institute of Dacryology, LV Prasad Eye Institute, Hyderabad for all the teaching and innovations in the field.

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Simplified diagnostic criteria for Migraine in adults (International Headache Society)

Clinically, a syndrome is a collection of symptoms; Migraine is not a static condition; so there will be periods when patient will need relatively little attention and other periods when they will need close monitoring.

Repeated attacks (at least 5) of headache lasting 4 to 72 hours and with features-

a) Normal physical examination
b) No other reasonable cause for headache
c) At least two of-
   i) Unilateral pain
   ii) Throbbing or pulsating pain
   iii) Aggravation by movement like walking or climbing stairs
d) At least one of-
   i) Nausea/vomiting
   ii) Photo/Phonophobia

TRIGGERS
1) Less sleep
2) Food skipping
3) Stress
4) Smoking
5) Oral contraceptives
6) Food with tyramine (aged cheese, yoghurt, bananas, vinegar, beans and peanuts)
7) Tea, coffee, chocolates, fatty foods
8) Aspartame, MSG

Migraine is not a static condition; so there will be periods when patient will need relatively little attention and other periods when they will need close monitoring.

FOREWARNINGS OF MIGRAINE

Appear hours to days before headache event and include:-

- Photo/Phono/Osmophobia
- Lethargy
- Mood changes (Depression, Anger or Joy)
- Polyuria
- Soreness and stiffness of neck muscles
- Anorexia/aversion to food
- Diarrhoea/constipation

Aura - Aura is experienced by some migraineurs. Aura consists of focused symptoms that grow over 5 to 15 minutes and generally last about one hour. Mostly headache follows Aura.

a) Visual symptoms in Aura-
   i) Negative scotomata (Blurred/absent areas in vision fields, Tunnel vision or complete blindness)
   ii) Positive visual problems - Include Fortification Spectrum (Figure 1) and Visual Hallucinations. An absent arc or band of vision with a shimmering or glittering zig zag borders constitute the Fortification Spectrum.

   It is diagnostic of Classic Migraine. It is named so because jagged edges of hallucinated arc resemble a fortified town with bastions around it. It is often associated with photopsia, sensation of lights or sparks.

   b) Motor symptoms in Aura- Hemiparesis or Aphasia are less frequent

   i) Photophobia


<table>
<thead>
<tr>
<th>Difference between Tension Type headache and Migraine</th>
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</thead>
<tbody>
<tr>
<td>Tension Type Headache</td>
</tr>
<tr>
<td>Occurs without warning</td>
</tr>
<tr>
<td>Pain more likely to be all over</td>
</tr>
<tr>
<td>No throbbing</td>
</tr>
<tr>
<td>No nausea</td>
</tr>
<tr>
<td>No light or noise sensitivity</td>
</tr>
<tr>
<td>No visual disturbances</td>
</tr>
<tr>
<td>Rare to start during sleep</td>
</tr>
</tbody>
</table>
Classification of Migraine (International Classification for Headache Disorders III) (ICHD)

1) Migraine without aura
2) Migraine with Aura
   a) Migraine with typical aura
   B) Migraine with brainstem aura
   c) Hemiplegic migraine
   d) Retinal migraine
3) Chronic migraine
4) Complications of migraine
   a) Status migrainosus
   b) Persistent aura without infarction
   c) Migrainous infarction
   d) Migraine aura triggered seizures
5) Probable migraine
6) Episodic syndromes that may be associated with Migraine
   a) Recurrent Gastro intestinal Disturbance
   b) Benign Paroxysmal Vertigo
   c) Benign paroxysmal Torticollis

MANAGEMENT OF A PATIENT OF MIGRAINE –

- Counselling
- Prevention of attack of headache
- Management of acute attack of headache

Drugs approved for prevention of Migraine
- Flunarizine
- Propranolol and Metoprolol
- Valproate
- Amitriptyline
- Pizotifen
- Methylsurgide
- Topiramate

HOW TO START TREATMENT?

Stratified care - Physician determines the treatment in the beginning on the basis of likelihood of response

1) Non specific drugs - Start with Aspirin (900mg) or Paracetamol (1000mg) + Domperidone (10mg) / Metoclopramide (10mg)
   - Also can use Ibuprofen (400 to 800Mg) or Tolfenamic acid (200mg)

2) Ergotamine and Dihydroergotamine - They were the first anti migraine drugs to become available in 20th century. They are non specific
   - 5HT agonists and vasoconstrictors. They are now found to be less effective than Triptans in acute migraine and also have more side effects than Triptans.
   - Common side effects are nausea and vomiting (10% patients), diarrhoea, muscle cramps, numbness and tingling in extremities. Contraindicated in cardiovascular patients, uncontrolled hypertension, peripheral vascular disease, hyperthyroidism and pregnancy (teratogenic effects)
   - Serious/life threatening peripheral vasoconstriction (ERGOTISM) can occur if given with protease inhibitors (antiretroviral drugs, Anti Hepatitis C drugs), Macrolide antibiotics,
   - CYP3A4 inhibitors (Ketoconazole, Clarithromycin), OCPs, Beta Blockers.
   - Dosing – 1 to 2 mg, repeat after 30 minutes if required.
   - Maximum is 6mg/day or 12mg/week

3) Triptans - Triptans are now the preferred medicines for aborting the acute attack of Migraine.
   - They are 5 HT 1 Agonists (Tryptamine based drugs) leading to constriction of cranial blood vessels, inhibition of Neuropeptide release and reduced transmission of trigeminal pain pathways. They are highly effective in aborting the attack within 30 to 90 min in 70-80% of the patients.
   - Triptans available in Indian market are -
     i) Sumatriptan (25mg, 50mg,100mg tablets and 6mg inj)
     ii) Rizatriptan (5mg & 10 mg tablets)
     iii) Almotriptan (12.5mg & 25 mg Tablets)
     iv) Naratriptan (1mg & 2.5 mg Tablet)
     v) Zolmitriptan (5mg Nasal spray)

<table>
<thead>
<tr>
<th>Frequency of Headache /Month</th>
<th>Need of preventive treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 to 2 attacks</td>
<td>Not required</td>
</tr>
<tr>
<td>3 to 4 attacks</td>
<td>yes</td>
</tr>
<tr>
<td>5 or more than 5 attacks</td>
<td>yes</td>
</tr>
</tbody>
</table>

Name of medicine | Group | Dose | Side effects/Precautions |
<table>
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</thead>
<tbody>
<tr>
<td>Flunarizine</td>
<td>Calcium channel blocker</td>
<td>10 mg at bed time</td>
<td>Side effects – weight gain, somnolence, dry mouth; contraindicated in pregnancy/hypotension/congestive heart failure or arrhythmia</td>
</tr>
<tr>
<td>Propranolol</td>
<td>Beta Blocker</td>
<td>80 to 240 mg per day</td>
<td>contraindicated in patients with asthma/chronic obstructive pulmonary disease/diabetes mellitus/heart block or failure/ peripheral vascular disease/pregnancy</td>
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<tr>
<td>Valproate</td>
<td>Anticonvulsants</td>
<td>400 to 600mg per day</td>
<td>may cause neural tube defects and should not be given in pregnancy</td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>Antidepressants</td>
<td>25 to 75 mg per day</td>
<td>Contraindications include severe cardiac, kidney, liver, prostate and thyroid disease, glaucoma, hypotension, seizures</td>
</tr>
<tr>
<td>Pizotifen</td>
<td>Serotonergic agent</td>
<td>0.5 to 3 mg per day</td>
<td>Drowsiness, weight gain</td>
</tr>
<tr>
<td>Methylsurgide</td>
<td>Semisynthetic Ergot</td>
<td>1 to 6 mg per day</td>
<td>Risk of retroperitoneal and retro pleural fibrosis, drowsiness, leg cramps</td>
</tr>
<tr>
<td>Topiramate</td>
<td>Anti convulsants</td>
<td>25 to 200 mg per day</td>
<td>Confusion, weight loss</td>
</tr>
</tbody>
</table>
### Financial Interest

The author does not have any financial interest in any procedure/product mentioned in this manuscript.

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**Drugs available for management of acute attack of migraine**

<table>
<thead>
<tr>
<th>Non-specific drugs</th>
<th>Specific drugs</th>
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<tbody>
<tr>
<td>Aspirin</td>
<td>Ergotamine</td>
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<tr>
<td>Paracetamol</td>
<td>Dihydroergotamine</td>
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<tr>
<td>Naproxen</td>
<td>Triptans - Sumatriptan</td>
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<tr>
<td>Ibuprofen</td>
<td>Naratriptan</td>
</tr>
<tr>
<td>Tolfenamic acid</td>
<td>Rizatriptan</td>
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<td></td>
<td>Zolmitriptan</td>
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<tr>
<td></td>
<td>Almotriptan</td>
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**Clinical situation**

<table>
<thead>
<tr>
<th></th>
<th>Treatment options</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Failed Analgesics / NSAIDs</td>
<td>Sumatriptan (50mg)/Rizatriptan (10mg)/ Naratriptan (2.5mg) Ergotamine (1mg) Zolmitriptan nasal spray (5mg)</td>
</tr>
<tr>
<td>2) Headache recurrence</td>
<td>Ergotamine (2mg) perhaps most effective taken rectally, usually with caffeine</td>
</tr>
<tr>
<td>3) Very rapidly developing symptoms</td>
<td>Sumatriptan inj (6 mg subcutaneous)</td>
</tr>
<tr>
<td>4) Tolerating acute treatments poorly</td>
<td>Naratriptan (2.5mg)/Sumatriptan inj (6mg subcutaneous)</td>
</tr>
<tr>
<td>5) Menstrually related headache</td>
<td>Prevention—Ergotamine tablet taken at night, oestrogen patches Treatment—Triptans</td>
</tr>
</tbody>
</table>

**Side effects** are chest tightening, weakness, stomach ache, warmth, redness. Contraindicated in heart disease, stroke, Ischemic bowel disease, Hypertension, Hemiplegic or Basilar Migraine, liver disease.

Migraine in Children- 10% of School age kids suffer from migraine. 50% of all migraine sufferers have their first attack before 12 years of age.

- Migraine runs in families. If one parent is affected, chances of child getting affected are 40% and if both the parents are affected, chances of child to be affected are as high as 90%. It has been reported in child as young as 18 months.
- Before puberty, boys suffer from it more than girls. Mean age of onset is 7 years in boys and 11 years in girls.
- Kids may develop anticipatory anxiety that can disturb their schooling and social activities.

**Diagnosis of Migraine in Children** – It is difficult to diagnose migraine in kids as the presentation varies from adults

- Headache is less severe than other symptoms (Nausea, vomiting, abdominal pain, dizziness—these are called Migraine equivalents)
- Aura +/- (blurry vision/ flashing lights/coloured spots)
- Change in behaviour observed by parents before attack (mood swings, yawning, irritability, food craving, loss of appetite, lethargy, withdrawal)
- Child develops sensitivity to light/sound/touch/Smell; sleep walking, sleep talking, night terrors

Diagnosis is made by history, examination and ruling out other explanation for the symptoms (Blood, EEG, LP and neuroimaging).

**Treatment**

Education of the child and parents about migraine and its triggers is very important. A regular bed time and strict meal schedule should be maintained.

The child should not be overburdened with activities.

**Prophylaxis**– In case of frequent attacks (more than 1 headache per week) or more than 1 disabling headache a month (missing school or social activities), prophylaxis may be given. The approved medicines are Flunarizine (start with 5mg and then increase to 10mg after 1 month), Amitriptyline (1mg/kg/day). Propranolol and Methylsurgide.

**Acute attack of migraine** –

1. Advise the child to lie down in a cool, dark room and try to sleep
2. Rehydration and sedation (benzodiazepine)
3. Give simple analgesics (Acetaminophen 15mg/kg or Ibuprofen 7.5 to 10 mg/kg) with domperidone
4. In severe cases, only two Triptans are approved for Paediatric population.
   i) Almotriptan (for adolescents 12 to 17 years)
   ii) Rizatriptan (as young as 6 years)

**Migraine in pregnancy** – In about 55 to 90% of the women who suffer from migraine have found that their condition improves when they are pregnant. This can be attributed to the high levels of oestrogen during pregnancy. This also explains the worsening of migraine during the postpartum period due to rapid fall in hormone levels.

**Prophylaxis of migraine** – Rarely indicated. If required, the only drug that can be given is Propranolol.

**Acute attack of Migraine**–

1. Paracetamol (1000mg) is the drug of choice
2. Triptans and Ergotamine and Dihydroergotamine are contraindicated
3. For Nausea and Vomiting, Prochlorperazine and Metoclopramide (only in 2nd and 3rd trimester)

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**REFERENCES**

4. Ravishankar K. Headache pattern in India – A headache clinic analysis of 1000 patients. Cephalalgia 1997;17:316-17
Intracranial tumors may initially present with subtle ocular signs and symptoms due to increased intracranial pressure, cranial nerve involvement or brain compression before other neurological features become manifest. Early recognition of these symptoms along with relevant investigations may help us to diagnose these tumors at an early stage where they can be safely dealt with. The symptoms include decreased visual acuity; transient visual obscurations, visual field defect and diplopia that may be present for days to years. Ophthalmological review is, therefore, of paramount importance and ophthalmologist must have a high index of suspicion in these cases for early diagnosis.

CASE REPORT

A female aged 26 years presented in our department with the chief complaint of diplopia for last 2 weeks. She had a history of intermittent episodes of severe headache preceding diplopia for last 2 years for which she was prescribed oral medicines by a local practitioner. There was a history of oligomenorrhea for which she had been taking oral levothyroxine, norethisterone and metformin. Though she did consult ophthalmologist for diplopia before coming to us, she was told it was side-effect of drugs she was taking. On examination, her vision was 6/6 in both eyes, visual axis was parallel and ocular movements were full in all quadrants in right eye and slight restriction of abduction in left eye. She had binocular diplopia on charting and fundus examination revealed bilateral papilledema (Figure 1b). Rest of the ocular examination was normal. The visual field revealed bilateral homonymous hemianopia (Figure 2a and 2b). Her serum thyroxine, luteinizing hormone and follicle stimulating hormone level were normal at presentation. She was ordered an urgent contrast enhanced MRI (Figure 3a and 3b) that revealed a craniopharyngioma measuring 3.7 cm (AP), 2.7 cm (TR) and 2.1 cm (CC) in diameter. It was suprasellar, cystic in nature with specks of calcification compressing optic chiasma and third ventricle leading to dilated ventricles. She was referred to a neurosurgeon where she underwent right MPVP shunt followed by right pterional craniotomy with near total excision of tumor in two different surgeries. However, she developed primary optic atrophy of left eye and pallor of right disc. Now the patient faces lifetime morbidity of panhypopituitarism, necessitating life-long hormonal replacement therapy.

DISCUSSION

Craniopharyngiomas are the most common nonglial tumors in children and account for 3-5% of all pediatric brain tumors. Two types of craniopharyngiomas have been described in the literature: a childhood type, with frequent occurrence of cyst formation and calcification, an adamantinomatous microscopic pattern and generally poor prognosis and an adult type, generally without calcification or cyst formation, with papillary squamous epithelium and generally a good prognosis.

The mechanisms of craniopharyngioma formation remain unelucidated. The only fact is known for sure: these tumors are congenital and are most likely to be non-heritable. The reasons for growth initiation in different age groups are unclear. It is histologically benign. Various presentations include: progressive visual loss, transient obscuration of vision, delayed puberty, growth failure, weight gain and diabetes insipidus. Eventually, extension of the tumor into the hypothalamus, third ventricle and limbic system produces endocrine dysfunction. This patient also had oligomenorrhea.
and hypothyroidism with history of episodic headache for two years before presenting with diplopia. Treatment consists of either a gross total excision or a conservative approach to drain the cyst and resect non-adherent tumor and then, administer radiation therapy. Radiation therapy causes cognitive deficits and total surgical excision may be difficult as tumor may invade hypothalamus or the third ventricle and adhere to optic nerve or blood vessels. Focused treatment by

**Department of Ophthalmology DRPGMC Kangra at Tanda (Himachal Pradesh), India**

Dr. Avantika Dogra (MS)

Dr. Rajeev Tuli MS

Dr. R.K. Sharma MS

Dr. Gaurav Sharma MS

Dr. Mandeep Tomar MS
stereotactic radiosurgery like gamma knife can be advantageous in this regard\(^{10}\) and are used in surgically unresectable or recurrent tumors (recurrence rate being as high as 30 to 57\%)\(^{5}\).

**CONCLUSION**

Though this patient had in addition to headache, visual symptoms including diplopia, yet the patient was neither investigated nor referred to higher center for excluding possibility of a possible intracranial lesion. This highlights the importance of educating general practitioners and medical specialists about importance of basic eye examination like pupillary reaction and fundus examination, which should be a part of routine examination in patients of headache and can be carried out even by non ophthalmologists at PHC level. At tertiary level, investigations including visual fields are a must for these patients. These simple investigations can make a huge difference in preventing a great deal of morbidity and mortality in these patients with intracranial tumors by helping in an early diagnosis.

**REFERENCES**


Lichen Planus (LP) is a chronic inflammatory and immune mediated mucocutaneous disease of unknown aetiology. Cutaneous lichen planus (CLP) most commonly affects the flexor surfaces of the extremities, commonly presenting as small itchy violaceous papules in middle-aged adults. It is classically described as “Pruritic, Purple, Polygonal, Planar, Papules, and Plaques”. The conjunctival involvement is rare and isolated ocular LP, without cutaneous involvement is rarely reported.

CASE REPORT

A 58 years old male patient presented with symptoms of irritation, redness and severe photophobia in both the eyes since four years. He was diagnosed as dry eyes and was treated with multiple topical drugs including lubricants, steroids and cyclosporine 0.1%. He was refractory to the treatment. His best corrected visual acuity was 6/6 in both the eyes. The slit lamp biomicroscopic examination of both the eye revealed thickening of lid margins with punctal stenosis, conjunctiva revealed diffuse conjunctival hyperaemia with early symblepharon in the inferior fornix. Diffuse punctate epithelial erosions were noted on the cornea in both the eyes. The rest of the anterior segment was normal (Figure 1). Left eye additionally revealed corneal scarring with 360° superficial corneal vascularization in the periphery (Figure 2). Dry eye workup revealed decreased marginal tear film height in both the eyes with decreased tear film breakup time (TBUT) (6 seconds in the right eye, 4 seconds in the left eye). Shirmers test showed decreased values of 5mm in right eye and 3mm in the left eye. The systemic examination was normal. There was no evidence of any skin or oral lesions. Erythrocyte sedimentation rate (ESR) was raised to 116 mm/hour. Rheumatoid factor, Anti-nuclear antibody, Anti RO and Anti LA testing were normal.

Based on these clinical and laboratory findings, a presumptive diagnosis of ocular cicatral pemphigoid was made and the conjunctival biopsy was taken. The histopathological examination of the conjunctival biopsy showed showed acanthosis, focal thickening of the basement membrane, and a dense subepithelial mononuclear infiltrate (Figure 3). Immunofluoroscent study (IFS) revealed linear shaggy fibrinogen deposition along the basement membrane with no evidence of immunoreactant.
deposition in the epithelium Basal Membrane Zone. IFS was suggestive of Lichen Planus.

The patient was started on Tab Azathioprine 50 mg in two divided doses along with Tab Methyl prednisolone 4mg once daily. Topical treatment was continued with Cyclosporine 0.1% eye drops 4 times a day and preservative free artificial tears (CMC 0.5%) 6 times a day. The patient was symptomatically better with marked improvement in photophobia two months after initiation of treatment. The schirmer's test showed improved readings of 12 mm in the right eye and 10 mm in the left eye.

**DISCUSSION**

Although ocular involvement in CLP is well established, isolated conjunctival LP is very rare, with only 8 cases being reported till now as per our knowledge. Ocular LP presents as blepharitis in more than one-third of the patients. The present case and other conjunctival involvement of LP amusingly causes a diagnostic challenge. The immunofluorescent study is essential in differentiating these conditions, as many histopathological features in these diseases overlap. IFS in LP will reveal absence of immunoreactant deposition in the epithelium Basal Membrane Zone along with shaggy deposition of fibrinogen along the basement membrane.

Topical treatment with corticosteroids and cyclosporine remains the first line of treatment. However the patients not responding to topical therapy should be treated with systemic corticosteroids and other immunosuppressants like azathioprine, cyclosporine, or mycophenolate mofetil.

**CONCLUSION**

LP should be ruled out in chronic dry eyes and cicatricial conjunctivitis refractory to conventional treatment even if skin or mouth is not involved, so as to initiate appropriate therapy and prevent further morbidity. Diagnosis has to be established with histopathologic and immunofluorescence study. Lichen planus should be routinely included in the differential diagnosis of cicatricial conjunctivitis, as it has important therapeutic and prognostic implication. Systemic immunosuppression may be needed to control the inflammation.

**REFERENCES**

A 13-year-old boy, student in Delhi presented to our outpatient department with complaints of episodic discoloration of left half of face after exercise and drooping of right upper eyelid since 3 years of age as noticed by the parents. This drooping was non progressive and was not associated with any diurnal variation, diminution of vision, abnormal eye movements on chewing food or limitation of movements.

There was discoloration/ flushing/ sweating of left half of face after physical exercise which used to resolve on its own with rest and without any sequela.

There was neither history of systemic illness or trauma nor any medical or surgical intervention. Child was born full term through normal vaginal delivery at home. Perinatal course was uneventful. He was appropriately immunized till date.

Examination- General physical examination as well as central nervous system examination including sensation in trigeminal area was normal.

Ocular examination-Unaided visual acuity was 6/6 both eyes. Near vision was N/6 both eyes. Both Eye balls were normal. There was no limitation of extraocular movements. Exophthalmometry was 16 and 18 mm in right eye and left eye respectively with bar reading of 110mm. Right eye upper lid ptosis was present (Figure 1). Left eye examination was normal.

Lid crease was well developed with margin crease distance (MCD) 7mm, palpebral aperture in primary gaze was 7 mm and 10 mm, margin reflex distance (MRD1) 1 mm and 4 mm, MRD2 6 mm each, margin limbal distance (MLD) 6 mm each, levator palpebrae superioris (LPS) action 12 mm and 15 mm in right eye and left eye respectively suggesting moderate (3mm) amount of ptosis in right eye. Bells phenomenon was good. There was no jaw winking.

Iris showed heterochromia as right iris was lighter in colour as compared to left iris.

Pupil was circular with normal direct and consensual reaction. Anisocoria was present with pupil of right eye being smaller. Near reflex was normal in both eyes.

Anisocoria of 1 mm and 1.5 mm was observed in bright light and ambient light respectively. In the initial phase of dark light, anisocoria was maximum (2mm) decreasing to 1 mm later on which showed typical dilatation lag (Table 1, Figure 2).

Rest of the anterior segment examination as well as fundus was normal.

We subjected the patient to exercise in the form of climbing up the stairs and going downstairs for 10-15 minutes. We observed anhidrosis on right side and normal sweating and flushing of left side of face (Harlequin sign) (Figure 3). There was no change in lid position or pupil size of affected side in response to exercise whereas normal pupil dilated by 1 mm.

Patient was subjected to phenylephrine test by instillation of one drop of phenylephrine 2.5% in conjunctival sac. Result was negative.

We also observed diffuse greying of scalp hair. Histopathology showed patchy depigmentation of affected hair (Figure 4).

Chest X-ray, MRI and CT of brain and neck region didn’t show any abnormality. Carotid Doppler study was normal.

Autonomic functions tests were also performed and didn’t show any generalised dysfunction (Table 2).

So diagnosis of Idiopathic Congenital Horner Syndrome was made.

We counselled the parents about the condition and reassured them for anhidrosis. And we have planned Fasanella Servat surgery for ptosis of right upper lid for the patient.

**DISCUSSION**

Horner syndrome (Bernard-Horner’s syndrome or oculosympathetic palsy) is classically described as a triad of ipsilateral upper lid blepharoptosis, pupillary miosis and anhidrosis of face/body. The underlying pathology is interruption of oculosympathetic supply to eye between its origin in hypothalamus and eye.
oculosympathetic palsy) is classically described as a triad of ipsilateral upper lid blepharoptosis, pupillary miosis and anhidrosis of face/body. The underlying pathology is interruption of oculosympathetic supply to eye between its origin in hypothalamus and eye (Figure 5). Diagnosis of Horner syndrome is made clinically. Investigations are done to find the underlying cause if any. Acquired Horner syndrome is not so uncommon but congenital Horner syndrome is rare condition. Differentiation between acquired and congenital can be made by detailed history and presence of heterochromia. However, heterochromia can be present in long standing cases of acquired Horner syndrome.

Although pharmacological tests have been traditionally described for confirmation of diagnosis as well as localisation of level of lesion. These pharmacological tests are not done routinely for confirmation as diagnosis can be made clinically. Secondly, drugs

<table>
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<th>Table 2: Autonomic function tests</th>
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<tr>
<td><strong>Test</strong></td>
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<tr>
<td>1. Deep breathing test</td>
</tr>
<tr>
<td>a. Change in heart rate</td>
</tr>
<tr>
<td>b. E:I ratio</td>
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<tr>
<td>2. Valsalva manoeuvre test:</td>
</tr>
<tr>
<td>Valsalva ratio</td>
</tr>
<tr>
<td>3. Isometric exercise (hand grip test)</td>
</tr>
<tr>
<td>Rise in diastolic BP:</td>
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<tr>
<td>4. Cold pressor test:</td>
</tr>
<tr>
<td>Rise in diastolic BP</td>
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</tbody>
</table>

Figure 2: Pupillary size 2(a): ambient light; 2(b): bright light; 2(c): dark– 5 sec; 2(d): dark– 15 sec

Figure 3: Harlequin sign

Figure 4(a): Patchy pigmentation and depigmentation of same affected hair

Figure 4(b): 40X light microscopic view showing depigmentation

Figure 4(c): 40X light microscopic view of hair showing pigmentation
used for these tests are not available freely in the market. However, depending on the pattern of anhidrosis, localisation can be made. In the defect of first order neuron, there is hemihypohidrosis of entire half of affected side. In the defect of second order neuron, there is anhidrosis of the face and neck region of the affected side. While in third order neuron lesion, there is patchy anhidrosis of medial forehead and side of the nose.

Although, straightening of the scalp hair on affected side has been reported previously, we could not find any correlation or association between Horner syndrome and greying of scalp hair in literature\(^2\). It can be hypothesized that melanin in hair follicle may require intact sympathetic innervation like iris melanocytes but this needs to be further validated or it might be just coincidental finding which has no relation with Horner syndrome.

Imaging modalities are not indicated in congenital Horner syndrome until unless pathology is suspected clinically. These are required in acquired Horner to find the underlying case. However, we investigated the patient as history of discoloration was quite interesting and complaints were not present since birth and also as a part of academic exercise.

**CONCLUSION**

Horner syndrome can be misdiagnosed as a case of simple congenital ptosis. This case report highlights the importance of thorough clinical examination to reach the diagnosis and intervene accordingly.

**REFERENCES**


**Financial Interest:** The authors do not have any financial interest in any procedure/product mentioned in this manuscript.
A 50 year old male patient presented with irritation in his LE since few days. He was a known case of Marfan’s syndrome. He had undergone Descemet stripping endothelial keratoplasty, along with lensectomy and scleral fixated IOL implantation in his left eye elsewhere a month earlier. On examination his BCVA was 6/6 in the RE, 6/12 in LE. IOP was 12 mm Hg Hg by applanation tonometry in his RE and 30 mm Hg in his LE. RE was pseudophakic with
a clear cornea and anormal fundus. Optic disc was normal in colour with a cup disc ratio of 0.4. LE showed a clear graft (Figure 1) with pseudophakia. Fundus was normal with optic disc CD ratio of 0.4 and normal colour. Antiglaucoma medication was started in the LE and the topical steroids were continued for the corneal graft.

A month later base line visual field examination was done (Figure 2). To our surprise bitemporal hemianopia was seen. Immediately and MRI scan was done which showed a large pituitary macroadenoma. The patient underwent neurosurgery for the macroadenoma within a few days.

He was followed up regularly over 3 years. The LE IOP was monitored closely and serial visual fields were done. However, the LE showed progression of cupping (Figure 3 and 4). The visual fields also showed the field defect to be crossing the midline signifying that the glaucomatous component of optic neuropathy was worsening. RE visual field was improving as the compressive aspect of optic neuropathy was lessening (Figure 5).

This case report highlights the importance of visual field examination which is often overlooked as a diagnostic and prognostic tool in cases of primary and secondary glaucoma. Also, the fundus examination in this case was normal initially, so no suspicion of a brain tumour was entertained clinically. The baseline visual field was diagnostic of the pituitary adenoma and life saving for the patient.

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**Figure 4:** OCT RNFL of Le showing advanced cupping and loss of neuroretinal rim

**Figure 5:** Visual field of RE showing improvement in RE as compared to pre operative (macroadenoma debulking surgery) and progression of glaucomatous component of LE visual field

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Cornea Department ICARE Eye Hospital, NOIDA, U.P. India.

Dr. Aanchal Mehta MBBS  
Dr. Ravi Sharma DNB  
Dr. Uma Sridhar MS, DNB, FRCS

**Financial Interest:** The authors do not have any financial interest in any procedure/product mentioned in this manuscript.
Glaucoma is a blinding disorder often requiring definitive surgery for its control especially in developing countries where adherence to medical therapy is erratic for a multitude of reasons. The first surgical procedure described by Wecker in 1882 was named “filtering cicatrix”, due to generation of a bleb created by aqueous filtering out into the subconjunctival space. For almost seven decades free filtering surgeries ruled to be replaced by guarded filtering procedure namely trabeculectomy as described by Sugar and then Cairns in 1960s. Since then it has remained the most commonly performed glaucoma surgery worldwide. Over the past decade the surgery has declined partly due to concerns about bleb related complications. The major cause for these bleb related complications is antifibrotic agents like mitomycin C used to modify wound healing at both conjunctival and episcleral plane to ensure continual patency of the filtering cicatrix (bleb). Recent statistics reveal a 53% decrease in number of trabeculectomies with corresponding 18.4% increase in tube-shunt procedures in developed countries due to these bleb related complications.

BLEB CREATION

The outflow of aqueous through the full thickness inner sclerectomy and peripheral iridectomy around the superficial sclera creates an elevation of the overlying conjunctival flap to form a filtering bleb. Aqueous from this bleb is absorbed by conjunctival vessels and partly excreted through tears. Most filtering blebs contain loculations delimited by internal fibrous walls primarily at the sclerostomy site surrounded by peripheral smaller, loculations. Optimal wound healing of trabeculectomy envisages suboptimal healing around sclerostomy and filtering loculations with complete healing of outer walls of bleb and surrounding conjunctiva. This required dichotomy in never feasible and uniformity of suboptimal healing is the cause of bleb related problems like overfiltration, bleb leak and blebitis. On the other hand optimal healing is the genesis of subconjunctival scarring and bleb failure.

An ideal filter is low lying, diffuse, with reduced vascularity, cystic changes which maintains intraocular pressure (IOP) in low teens along with a formed anterior chamber. Any deviation from this norm is a cause of concern due to individual idiosyncrasies and surgical techniques no one norm of ideal bleb exists. Most classification eg IBAGS encompass more than one parameter in defining a functioning bleb. In addition the dynamic nature of healing process ensures ongoing bleb modulation and in order to label a bleb as suboptimal, both function and structure of it needs to be accounted. The following are some types of blebs deviant from the ideal.

LEAKING BLEB

This is usually seen in immediate post-operative period and manifests as hypotony (IOP in low single digits), absence of visible bleb, shallow anterior chamber with possible positive Siedel’s test localizing the leak (Figure 1a-c).

Siedel’s test: This is done by wetting ocular surface of expected bleb site area with 1% fluorescein dye and examining under cobalt blue filter on the slit lamp. A prerequisite is drying of the area examined. A leak can be diagnosed by a stream of unstained aqueous visible as blue rivulet in tear film stained green by the dye (Figure 1c). The area should be examined for a longer time since in some cases the leak is a mere ooze called as the “sweating bleb”. To corroborate a strong clinical suspicion in absence of a spontaneous leak, gentle pressure applied through the lower lid using tip of finger can unmask latent ooze.

The risk factors for bleb leak are first and foremost use of anti metabolite, avascular bleb, (Figure 1f) thin walled bleb, cystic overhanging bleb having both thick and thin areas with leak occurring at apex of a cyst or at interface between thick and thin areas (Figure 1e). Another vulnerable area is the most elevated part of bleb since it is prone to repeated micro-trauma.
from the blinking eyelid. This elevated part of the bleb often dries out due to inadequate tear resurfacing.

Bleb leak also occurs through the thin conjunctival tissue which becomes cystic with constant exposure to aqueous (Figure 1d). The five-year probability of developing a bleb leak after Mitomycin C (MMC) aided trabeculectomy has been estimated to be as high as 18%9.

**Management**

Early leaks usually occur along suture track, often due to inadequate bleb anchoring at the limbus (for fornix based conjunctival flaps) or through an inadvertent conjunctival buttonhole (more common with limbal based conjunctival flap). Lack of separation between conjunctiva and sclera due to absence of aqueous in the subconjunctival space causes adhesion and scarring between these two layers leading to early bleb failure.

a. Minor leaks often heal spontaneously by medical management including with holding or reducing topical steroid to hasten epithelial repair of the leak. Once the leak has sealed, standard full strength steroid regimen may be continued to minimize inflammation and scarring within the bleb. Conservative approach includes -

- Aqueous suppression by topical beta blocker/carbonic anhydrase inhibitors to prevent aqueous outflow which keeps the leak patent, thereby giving epithelial migration and sealing to occur.

- Prophylactic topical antibiotics to prevent blebitis or endophthalmitis. The antibiotic choice varies from nonepithelial toxic like fluoroquinolone to optimize epithelium sealing the leak from mild irritant stimulating proliferation of epithelial cells and hastening sealing.

- Lubricants to ensure adequate wetting of conjunctiva since dryness make the delicate conjunctiva more friable to shearing forces of blinking.

b. Intermediate leaks not insignificant enough to self-seal but not brisk enough to require suturing can be treated using a torpedo bandage (Figure 2) or large diameter soft bandage contact lens covering the leak. Torpedo bandage/pressure patch involves placement of a fusiform shaped cotton ball over upper lid in a location corresponding to leak over which a folded eye pad is placed below the brow. Second open eye pad is positioned and multiple strips of tape are applied with moderate tension over a period of 12-24 hours. A precaution taken is not to allow patient to close the eye while doing it as that would place torpedo over the cornea due to Bells phenomenon occurring instead of over desired superior conjunctiva. Instead the patient is asked to look down only and the torpedo is positioned on the superior conjunctival area.

A bandage contact lens helps in protects the bleb from trauma generated by the blinking upper lid and aids epithelial migration and repair.

c. Significant leaks due to gaping of wound, broken

Glaucoma Services, Guru Nanak Eye Centre, MAMC & Associate Hospitals, New Delhi

Dr. Kirti Singh MD, DNB, FRCS

Dr. Keerti Wali MS

Dr. Mainak Bhattacharyya MS
sutures require re-suturing with 8-0 nylon under antibiotic cover

The surgical modalities tried are: use of cyanoacrylate or fibrin tissue glue[10,11] or re-suturing (Figure 3a) Areas of weak conjunctiva are covered or reinforced with stronger tissue to halt leak, restore ocular integrity and preserve bleb function.

After bleb dissection to remove non viable tissue and the wound resutured without tension. Small leaks can also be repaired using pedicle flap of conjunctiva by making a horizontal, full thickness, relaxing incision in fornix, dissecting the conjunctiva from underlying Tenon’s layer and pulling the free tissue anteriorly over the bleb. For larger leak conjunctival advancement or rotational flap may be necessary and in situations of insufficient healthy adjacent conjunctiva to cover the bleb, free conjunctival graft from fellow eye can be taken. Double layered amniotic membrane grafts also can be sutured or glued on the bleb surface (Figure 3b). Quilting of the membrane with the underlying episcleral tissue must be done to prevent shearing off the entire amniotic membrane with blinking of the lid. Preserved pericardium or buccal mucosa from lower lip can also be used as a graft. A symblepharon ring or conformer can be used to ensure adequate retention of these tissues.

Prophylaxis
- Bleb anchoring sutures along with titration of bleb at end of surgery to ensure water tightness of conjunctival closure (Figure 4).
- Protective eye shield to prevent unconscious rubbing of eye during sleep.

OVERFILTERING BLEB

This complication is usually seen in the early operative period and presents with hypotony, shallow AC with a diffuse large / high bleb. Hypotonous maculopathy is a visual disabling sequel which may be associated with this condition. It is a result of loose scleral flap sutures, very thin superficial flap or excessively large inner sclerostomy not covered by the superficial sclera flap (Figure 5). Antimetabolite usage potentiates the hypotony by inhibiting the natural fibrotic response, thus reducing aqueous outflow resistance.

A usually self limiting condition it requires treatment in cases of persistent shallow anterior chamber beyond first 2 weeks of surgery and / or central iridocorneal touch. This condition is often associated with serous choroidal effusions.

Management: The frequency and dosage of topical corticosteroids is reduced to permit subconjunctival scarring sufficient to limit aqueous egress. This is combined with aggressive cycloplegia using atropine along with judicious use of aqueous suppressants to reduce outflow. Use of soft bandage contact lens or pressure patch / toric bandage can be effectively used in the early postoperative period. In cases unresponsive to conservative management surgical re-formation of anterior chamber is resorted to at air, balanced salt solution [BSS], viscoelastics, non expandable concentrations of gases like perfluorocarbon and sulfur hexafluoride[12]. Large serous choroidal effusions can require surgical drainage after an adequate trail of systemic and topical steroid therapy.

For chronic cases other alternative therapies include injection of autologous blood[13], compression sutures or surgical revision[14] (Figure 6a).

Autologous blood injection is performed under aseptic conditions in operating room environment. Since no anticoagulant is used, the procedure has to be performed quickly. An assistant withdraws blood into a 1 ml syringe from the cleaned antecubital fossa while surgeon changes needle into a 30 gauge. This fresh needle is inserted under observation into the bleb at least 5 mm away and 0.3 ml of blood is injected. Some blood may enter the AC during the procedure, which can be washed via a paracentesis if required.

Compression sutures of 80 or 90 nylon are used to create a narrow rectangle or an “X” on the bleb (Figure 6b). Anterior suture is passed through 50 percent depth of limbal cornea, and posterior aspect is fixated to fornix episclera. The two ends are tied creating a compression and flattening of the problematic bleb. These are left in place for 3-4 weeks and then removed.

FAILING BLEB

This is the commonest complication and can occur in early and late post operative period with differing etiologies and management.

Early post operative period (<1 month): Bleb failure manifests as reduced height or flat bleb and higher than expected IOP with formed anterior chamber (AC). This needs to be differentiated from shallow bleb due to bleb leak which is associated with hypotony and shallow AC. The causes of failed bleb are: tight scleral flap sutures, too thick superficial sclera flap and / or obstruction of sclerotomy ostium with fibrin / blood/iris. For the former cause separation of edges of sclera is attempted with digital massage[15]. Digital massage is done by pressing on sclera next to the flap with a sterile cotton tip applicator [Q tip] or using index finger to compress the globe through lower eyelid with patient looking upwards. The IOP, depth of the AC and height of bleb should be noted after the digital pressure. This massage is repeated 7-10 times in one sitting and 2-3 sittings are to be done per day. It is taught to the patient or care giver.

The second option is sequential removal of releasable suture or laser suturelysis. Releasable sutures allow tight closure of the scleral flap in immediate postoperative period and permit IOP reduction with sequential release during the later intermediate postoperative period. Only one suture is removed at a time (Figure 7).

Laser suturelysis may be performed using an argon or diode laser with Hoskins/ Ritch /Mandelkorn or Blumenthal lens[16-18]. These lenses compress conjunctiva and underlying tissue permitting visualization of underlying sclera suture. Laser settings of 400mW, 0.01 seconds and 50 μm are employed and procedure is done under topical anesthesia. Low energy and low exposures suffice since longer exposure can cause conjunctival coagulation and hole formation[19]. These methods are useful within a of 2-3 weeks period after trabeculectomy without antimetabolics. With adjunctive use of antimetabolites the window period for postoperative titration with suture removal is extended to one month.

Intermediate post operative period (1-3 months): If the bleb remains flat with raised IOP in spite of digital massage and suture release scarring at the episcleral surface is commonest cause for bleb failure. Internal sclerostomy block due to inadequate peripheral iridectomy or up-drawing of iris tissue must be ruled out as internal causes of failure before attributing it to subconjunctival fibrosis. Gonioscopy should thus be done for all cases of bleb failure to rule out blockage of internal sclerostomy.

Late post operative period (>3 months): Unsuccessfully inhibited wound
healing leading to subconjunctival and even sub scleral fibrosis is the commonest cause of bleb failure. Clinical signs of bleb failure include excessive conjunctival vascularization, (Figure 8a) corkscrewing of vessels, (Figure 8b) reduction in bleb height, (Figure 8c) ring of steel scar line forming in a limbal based conjunctival flap technique along with rising IOP. (Figure 8d).

Risk factors for bleb failure are: Chronic use of topical anti-glaucoma agent’s causes growth factors and chemotaxins to leak through dilated, permeable vessels and primes conjunctival and Tenon’s fibroblasts. Young age, prior conjunctival surgery and secondary glaucoma due to rubeosis, uveitis or ICE syndrome also predispose to subconjunctival scarring. Neovascular and uveitic glaucoma have a high risk of failure due to presence of angiogenic substances with partial fibroblastic growth actions. Aphakia is another risk factor due to preexisting scarring, vitreous factor stimulation of fibroblastic proliferation and rapid wound healing.

Management

Once bleb has reached the late proliferative phase or entered maturation phase, management is invasive. For a flat or small bleb laser suture lysis even several months postoperatively with MMC trabeculectomy can be tried.

Needling: This procedure is used to lyse subconjunctival adhesions, break down localized bleb and enlarge area of transconjunctival filtration. Performed with use of 26-30 guage needle it was originally described for encapsulated blebs, but has subsequently been found very useful in failing blebs. It evolved from scar tissue incision and manual spatula aided separation of conjunctiva from sclera by Ferrer in 1941. Addition of 5-fluorouracil (5 FU) improved success rate to 92%. It alters bleb architecture by making it lower or more diffuse, thus increasing patient comfort and improving filtration.

Needling is done with concurrent use of antifibrotics, either MMC (0.2mg/ml) or 5-FU (5 mg in 0.1cc). Mitomycin C has propensity for endothelial toxicity and can cause corneal edema, whereas 5-FU has higher epithelial toxicity and can cause severe punctuate to confluent epithelial erosions. Thus the drug must not leak out into the tear film.
Under topical anesthesia in aseptic OT setting, a syringe loaded with 5 FU solution is inserted 3-4 mm away from the demarcated bleb. Around 0.1-0.2 ml of fluid is injected, a subconjunctival bleb created under which the subconjunctival fibrosis demarcating the trabeculectomy bleb is lysed using to and fro and sweeping movements. In some cases, fibrosis extends under the scleral flap and an attempt has to be made to lift edge of scleral flap and needling done. Frequently, a change in bleb appearance is seen during or soon after needling with bleb size increasing and tenseness decreasing (Figure 9). Topical steroid antibiotic drops are used for 5-7 days. Needling done early on in late postoperative period is associated with higher success. Multiple sessions may be required. Complications include intrableb bleeding and leak due to conjunctival perforations.

**ENCAPSULATED BLEB / TENON’S CYST**

This complication occurs in 9-15% during 2-4th postoperative week and presents with a tense, “tightappearing” bleb with prominent blood vessels associated with high IOP and deep AC. The bleb is firm with few or no microcysts (Figure 10). Treatment involves use of aqueous suppressants, topical steroids, bleb needling, surgical bleb revision (partial/complete cyst excision) or repeat trabeculectomy in stepped up fashion.

**OVERHANGING BLEBS / BLEB DYSTHESIA**

A common association with use of antimetabolites, it can be associated with hypotony/ overfiltration, foreign body sensation (large bleb), (Figure 11a), dysesthesia due to interference with lid function and closure leading to corneal drying with dellen formation, (Figure 11b) unacceptable cosmesis, and visual compromise due to astigmatism. Initial treatment would be conservative with adequate lubrication, aqueous suppressants.

Surgical revision of bleb is indicated for intractable pain caused by dellen or fluctuating vision. Strategies include compression sutures, laser bleb reduction, partial excision of the overhanging corneal portion with conjunctival autologous patch graft, or conjunctival advancement, cryoapplication, application of trichloroacetic acid, and bleb excision with. Argon laser photoagulation can remodel and reduce a bleb’s size by means of protein denaturation and tissue shrinkage, but can induce a chronic leak especially in thin ischemic blebs. Cryoapplication and application of trichloroacetic acid is potentially destructive to corneal surface. Bleb excision with autologous graft or conjunctival advancement though technically difficult is a better option.

**BLEB RELATED INFECTIONS**

Bleb related infections can be blebitis or endophthalmitis occur more often in polycystic thin walled blebs. Infection usually starts in subconjunctival space as blebitis and spreads to AC and vitreous. The risk factors for bleb related infections include modulated surgeries with anti-fibrotics, bleb leakage, thin walled cystic blebs, inferiorly located blebs, axial myopia, prolonged use of antibiotics, seasons, history of prior intraocular surgeries.

Azuara-Branco, Katz and Greenfield divided bleb related infections into stages. Stage I blebitis presents with conjunctival hyperemia, discharge, foreign body sensation with a purulent yellowish bleb (Figure 12a) Stage II involves AC characterized by flare, cells and hypopyon (Figure 12b). Stage III includes vitritis confirmed on ultrasound B scan (Figure 12c). The source of bacteria is usually ocular flora with most common organisms being Streptococcus, Staphylococcus and Haemophilus influenza. Positive bacterial cultures carried a worse visual prognosis.

Blebitis usually responds to intensive antibiotic treatment both topical and systemic. A fluid tap should be taken and sent for microbiological examination in case of endophthalmitis. Fulblown endophthalmitis will need aggressive treatment in form of intravitreal injections and vitrectomy. The prognosis of stage I is generally good with retention of preoperative vision and IOP. Stage III patients have a poor prognosis with
final visual acuity of counting fingers or less, some of them even progressing to pthisis bulbi or may need to be enucleated.

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Financial Interest: The authors do not have any financial interest in any procedure/product mentioned in this manuscript.
Bleb leaks are not an uncommon complication of glaucoma filtration surgeries. Bleb leaks are known to occur both in early as well as late post-operative period. There is a wide variation in the reported incidence of bleb leaks, from 0% to 30%². With the advent of anti-metabolites as adjuncts to the filtration surgery there has been a noted increase in the incidence of both early and late leaks². Leaks are usually due to thin-walled blebs that are common with anti-metabolites supplemented surgeries. It is also more common with fornix based flaps than limbus based flaps³⁻⁴. A careful and complete pre-operative evaluation, a meticulous intra-operative technique and an appropriate and prompt post-operative management are necessary for a successful outcome. A close follow up during the early post-operative phase and long-term monitoring of the blebs is therefore essential to minimize the bleb-leak related complications, the most dreaded one being endophthalmitis⁵⁻⁶. One of the sequelae of an early bleb leak may also be failure of the bleb function⁷.

PREDISPOSING FACTORS

There are certain intra-operative as well as post-operative risk factors which predispose to a bleb leak (Figure 1-5).

Intra-operative factors:
• Tears or button holes in the conjunctiva⁸.
• Failure to achieve a water tight closure, especially in a fornix based flap.
• Inadvertent exposure of the cut edge of conjunctiva with anti-metabolite agent like mitomycin-C (MMC)⁹.
• Superficial sclera flap dissection leading to tearing of flap.
• Prolonged exposure time of the tissue with the anti-metabolites
• Inadequate suturing and failure to recognize leaking site on-table.

POST-OPERATIVE FACTORS

• Conjunctival retraction.
• Snapped suture
• Loosening of suture
• Rubbing of eyes or strenuous activity by patient in the early post-operative phase¹⁰.
• Post-operative 5-fluorouracil (5-FU) injections leading to thinning of bleb wall.
• Late necrosis of conjunctival edge.

HOW TO AVOID A LEAK?

• Good instrumentation and visualization intra-operatively¹¹.
• Gentle handling of the conjunctiva with non-toothed forceps.
• If a buttonhole is recognized on-table, it should be repaired using purse string sutures; either single or multiple mattress sutures using a 10-0 nylon suture on an atraumatic needle.
• Preventing exposure of conjunctival edge to anti-metabolite agent.
• Thorough irrigation of exposed area after removal of anti-metabolite soaked sponges.
• Exercising special care while fashioning sclera flap in buphthalmic and highly myopic eyes.
• Meticulous suturing to achieve water tight closure.
• Before concluding the surgery the site should be carefully inspected for any leaks.

WHEN TO SUSPECT A LEAK?

• Patient complaining of increased tearing, blurring of vision or discomfort.
• A thin bleb with avascular or cystic areas on slit lamp examination. The thinnest and most avascular areas as well as the junction of thick and thin areas are the most vulnerable areas.

Figure 1: Conjunctival edge necrosis due to intra-operative exposure to mitomycin C.
• **Alarming signs:** flat bleb, shallow anterior chamber (a/c), low intraocular pressure (IOP) or hypotony (IOP<6 mm Hg)

• **Ominous sign:** milky white bleb with loss of clarity and reaction in a/c or vitreous, which suggests blebitis and/or endophthalmitis resulting from a bleb leak.

• **HELP syndrome:** hypotony, endophthalmitis, leak and pain are the constellation of complications associated with anti-metabolites usage.

• Sometimes discovering a choroidal detachment may lead the surgeon to retrospectively examine for a leak. The Seidel’s test is used to localize the site of leakage. Fluorescein is applied over the bleb surface and suture line and bleb is examined under the cobalt blue filter at the slit-lamp. If there is a leak, unstained aqueous will be seen with a bright green boundary surrounded by a dark green background. In the absence of a spontaneous leak, a gentle pressure can be applied over the globe suspicious areas should be re-examined. This is known as the forced Seidel’s test.

**HOW TO PROCEED AND MANAGE A LEAK?**

The size and location of the leak, status of anterior chamber, intraocular pressure, time of onset of leak and notable decrease in visual acuity are all

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**Figure 2:** A Seidel’s positive frank leaking bleb.

**Figure 3:** A bleb leak which was repaired using tissue adhesive along with BCL placement.

**Figure 4 & 5:** A bleb with a frank leak, the same bleb under cobalt blue showing a Seidel’s positive test
crucial, in order to determine whether a spontaneous closure can be achieved with conservative, nonsurgical methods or early surgical intervention will be needed for the management. Certain situations like a flat anterior chamber with lens-cornea touch, hypotony maculopathy, tissue dehiscence or kissing choroidal detachments require immediate intervention to manage leak and may also require an urgent anterior chamber reformation (which can be achieved by using viscoelastics like healon).

**Conservative management:**

- **Aqueous suppressants** reduce aqueous flow through the leak and give a chance for the defect to heal. Beta blockers or carbonic anhydrase inhibitors can be used.
- **Antibiotics:** gentamycin is a mild irritant and can stimulate the epithelial cells to heal the defect. Some surgeons also use oral doxycycline to enhance closure of leak¹⁰⁻¹¹.
- **Discontinuation or tapering of steroids.**
- **Application of glaucoma tamponade shell or symblepharon ring.**
- **Adequate lubrication of bleb surface:** dryness of bleb surface can make it more friable and lubricants will help in keeping the surface moisturized.
- **Bandage contact lens (BCL):** protects the bleb from trauma and acts as a scaffold for epithelial healing. A standard sized soft BCL does not typically cover the filtering bleb and a large diameter lens should be preferred. The contact lens can be kept in place for about a week to allow for reepithelization. The IOP can be monitored through the lens by use of instruments like the tonopen.

**Tissue adhesives:** These are useful adjuncts to surgery in the management of late bleb leaks¹³⁻¹⁴. Slow low-flow leaks in old, well developed filtering blebs are suitable for closure with tissue adhesives. Both autologous fibrin glue as well as cyanoacrylate glue has been used to seal the leaking bleb. The fibrin glue is a mixture of fibrinogen and thrombin. Autologous fibrin glue is preferred over the commercially available types as this eliminates the risk of transmission of infections. This procedure requires a mobile conjunctiva to prevent retraction of the adhesive. Cyanoacrylate glue adheres to the tissue and can be used to close leaks that occur shortly after surgery. The leaking site is dried with a cotton tip applicator or weck-cel sponge just prior to application of the glue, 2-3 drops are focally applied using a 30 gauge needle. This is sufficient not only to cover the leaking area but also 1-2 mm of the surrounding tissue. Run off of the tissue adhesive should be avoided. Pressure bandage or BCL may be used as adjuncts. The BCL application prevents dislodging of the glue.

**Autologous blood injection¹⁵:** Intrableb and peribleb injection of autologous blood has been described for treating chronic, thin, friable blebs post-operative leaking bleb (Figure 6 & 7). About 2ml of whole blood is drawn from the vein of antecubital fossa with a 27 gauge needle, which is changed to a 30 gauge needle and about 0.5-0.75 ml of blood is immediately injected subconjunctivally adjacent to the bleb or into the bleb as per surgeon’s choice. As no anticoagulant is used the procedure must be done quickly. Some blood can enter the a/c during the procedure, which has to be washed through a paracentesis if required. Intrableb injection requires excellent patient co-operation to avoid bleb perforation and it may be very difficult if the leak is brisk enough to cause bleb flattening. Peribleb injection is considered a safer alternative in such situations. It has been hypothesized that crosslinking factors from the blood help in sealing the defect.

Remodelling of the leaking blebs can also be achieved by Nd:YAG Laser or Argon Laser. Thin areas should be avoided.

**Surgical repair¹⁶:** The goal should be primarily to restore integrity of the eye and secondarily the preservation of bleb function. The bleb function may not be retained after the tissue is reinforced to halt the leak.

- **Re-suturing the conjunctiva:** with or without conjunctival advancement to secure good wound apposition and achieving a water tight closure.
- **Conjunctival advancement:** may be needed to cover medium sized defects. It can only be possible if the adjacent conjunctiva is not scarred and sufficient healthy conjunctival tissue is available.
- **Free conjunctival autologous patch graft:** the conjunctival patch graft

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*Figure 6 & 7: The same bleb as in figure 5 after intrableb autologous blood injection, Seidel’s negative when examined under cobalt blue.*
can be secured to the recipient site with 10-0 nylon sutures. Limbal edge of the graft should be placed maintaining the limbal orientation. On follow up visits the graft should start vascularising from periphery towards the centre, which indicates a good graft uptake. Buccal mucosa or amniotic membrane can also be used as alternatives.

- **Pedicle flap of conjunctiva:** can be fashioned to close a small localized defect by making a horizontal incision through the conjunctiva and pulling it anteriorly to cover the defect.

- **Scleral patch graft:** can be used to repair necrotic areas of sclera flap along with suturing. This reinforces the friable tissue. If donor sclera is not available then other materials like donor cornea, pericardium or amniotic membrane can be used.

In conclusion, it is imperative to maintain individualized approach while assessing and managing post-trabeculectomy blebs and to follow up the patients closely in the immediate post-operative phase, in order to avoid potential vision threatening complications.

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**Financial Interest:** The authors do not have any financial interest in any procedure/product mentioned in this manuscript.

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Venus Surgitech

B-503, Plot No. 23, Sector-6, Dwarka, NEW DELHI-75
Ph.: 011-43557387, 9350257387
Email: pandeymanoj67@yahoo.co.in
Development may be impaired due to a variety of factors like maternal, genetic, perinatal, post-natal and social factors. Visual development is a highly complex maturation process involving structural and functional changes in both the eye and the CNS. The burden of visual handicap in childhood especially in a child with developmental delay is of enormous importance because of the life-long impact of the handicap on other areas of development. Early recognition of the problem may expedite treatment or other forms of management where the condition is not treatable who are recognized at an early age receive more developmental optimization and greater gains than those who are identified later in life. Early recognition of children with developmental problems is therefore important.

Developmental disability/delay (DD) is present when functional aspects of a child’s development in one or more domains (gross/fine motor, speech/language, cognition, social/personal, and activities of daily living) are significantly delayed compared to the expected level for age (≥25% from the expected rate or a discrepancy of 1.5 to 2 standard deviations from the norm). Development may be impaired due to a variety of factors like maternal, genetic, perinatal, post-natal and social factors. Visual development is a highly complex maturation process involving structural and functional changes in both the eye and the CNS. The burden of visual handicap in childhood especially in a child with developmental delay is of enormous importance because of the life-long impact of the handicap on other areas of development. Early recognition of the problem may expedite treatment or other forms of management where the condition is not treatable.

Developmental disability is estimated to occur in 5-10% of the population with enormous psychological, emotional, and economic impact on the affected individuals and society. Studies have shown that developmentally delayed children who are recognized at an early age receive more developmental optimization and greater gains than those who are identified later in life. Early recognition of children with developmental problems is therefore important.

Children with DD constitute a considerable proportion of patients seen at pediatric ophthalmology clinics. Knowledge of the prevalence of ocular manifestations is essential if these children are to be given optimal support for the overall development.

Herein, this review summarizes the ocular profile in 180 DD children belonging to 3 cohorts (Table 1) consisting of 100, 52, 28 children respectively, in cerebral palsy (CP), West Syndrome (WS) and autism spectrum disorders (ASD). (Figure 1) Comprehensive history with detailed antenatal, prenatal and postnatal events and complete ophthalmological examination was done.

West syndrome is a severe epilepsy syndrome composed of the triad of infantile spasms, an interictal electroencephalogram (EEG) pattern termed hypersrhythmia, and mental retardation, although the diagnosis can be made even if 1 of the 3 elements is missing (according to the international classification).
Table 1: Clinical description of three cohorts

<table>
<thead>
<tr>
<th>Developmental Disability</th>
<th>Description</th>
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<tbody>
<tr>
<td>Cerebral Palsy³</td>
<td>Early-onset non-progressive motor impairment with associated abnormalities in muscle tone</td>
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<tr>
<td>Autism³</td>
<td>Impairments in social skills, communication skills and restrictive / repetitive patterns of behavior</td>
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<tr>
<td>West Syndrome⁴</td>
<td>West syndrome is a severe epilepsy syndrome composed of the triad of infantile spasms, an interictal electroencephalogram (EEG) pattern termed hypsarrhythmia, and mental retardation, although the diagnosis can be made even if 1 of the 3 elements is missing (according to the international classification).</td>
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CEREBRAL PALSY

CP is defined as ‘A group of disorders of the development of movement and posture causing activity limitation, that are attributed to non-progressive disturbances in the developing fetal or infant brain’⁵. Children with CP have many other co-morbidities besides motor impairment that include disturbances of sensation, perception, cognition, speech and language behavior; epilepsy, feeding and swallowing dysfunction and as well as visual and hearing impairment and secondary musculoskeletal problems⁵. In developed countries, the overall estimated prevalence of CP is 2-2.5 cases per 1000 live births⁶. According to The Autism and Development and Disabilities Monitoring Network, the average prevalence of CP is approximately 3.3 per 1000 live births⁶. Perinatal cerebral hypoxia–ischemia remains a frequent cause of the chronic handicapping conditions of CP, mental retardation (MR), learning disability, and epilepsy⁷. 75% patients with CP present with associated deficits¹⁰,¹¹. These include the following

MR is a common association of CP up to an extent of 60%¹⁰. Visual impairments and disorders of ocular motility are common (28%) in children with CP¹¹. There is an increased presence of strabismus, amblyopia, nystagmus, optic atrophy, and refractive errors. Children whose CP is due to PVL are also more likely to have visual perceptual problems like weakness in visual object recognition, visuospatial skills, visual memory and oculomotor control¹². Hearing impairment occurs in approximately 12% of children with CP¹¹. Epilepsy is common in children with CP, 35% to 62% of children develop epilepsy. Speech and language is affected in CP due to bilateral corticobulbar and oromotor dysfunctions. Both receptive and expressive language deficits are common and go hand-in-hand with MR. Articulation disorders and impaired speech are present in 38% children with CP¹¹. Oro-motor problems with feeding difficulties, swallowing dysfunction and drooling are also present. Feeding problems are quite common in children with CP with 30-80%. Behavioral problems are also well documented, these patients are prone to have anxiety, depression, conduct disorders, hyperkinesia inattention and poor self esteem¹¹. Abnormalities of proprioception and tactile sensations are common in children with CP¹¹. Psychiatric

1. Department of Ophthalmology, Lady Hardinge Medical College & Associated Hospitals
2. PGIMER & Dr RML Hospital New Delhi.
disorders such as anxiety, depression, conduct disorders and hyperkinesia and inattention are seen as often as in 61% of 6-10 year-old children with CP\(^{15}\). The associated deficits may be itself be very incapacitating in children with CP.

Ocular disorders are very common in children with CP. Guzzetta et al\(^{13}\) (2001) hypothesized that disorders of visual function are most often due to damage of the central visual pathway. Hoyt et al\(^{14}\) (2003) hypothesized that perinatal hypoxic ischemia is the most cause of visually significant brain injury in CP and damage to any one or more of at least 5 separate visual systems (primary visual cortex, visual associative cortex area, optic radiations, optic nerves, and visual attention pathways) may account for the visual disability of children with brain injury.

**Ocular Profile in CP children**

We studied 100 children of CP with M: F: 57:43 with average age 6.85 ± 2.92 years (range 4–18 years) and found strabismus (58%) and refractive errors (92%) were found to be most common ocular abnormalities in children with CP. The variation in ocular alignment as horizontal strabismus with esotropia in 23 (39.66%) and exotropia in 30 (51.72%) and vertical strabismus as dissociated vertical strabismus in 5 (8.62%) CP children. Pattern strabismus was seen in 7 patients. The variations across refractive errors found to be with most patients had ametropia (83%) with hypermetropia (47%) as the most common refractive error followed by astigmatism (30%). Myopia was present in 6% patients. Anisometropia was present in 9% patients.

The other ocular abnormalities were amblyopia (12%), nystagmus (16%), gaze palsy (11%), anterior segment abnormalities include microcornea in 1%, congenital cataract in 1% and posterior segment abnormalities include fundal coloboma in 3%, optic disc abnormalities including optic disc pallor (11%), optic atrophy (1%), optic disc hypoplasia (1%) (Figure 2).

**WEST SYNDROME**

This is a catastrophic form of epileptic seizure, typically occur in infancy. The incidence- 2-4 per 10,000 live born, 60% being boys\(^{15}\).

1. Sudden axial muscle contraction occurring in clusters of muscles of neck, trunk & extremities. Unusual variants comprise subtle head nodding, shoulder shrugging, abdominal contractions, eye opening, eye rolling, grimacing, and yawning. Unusual variants like asymmetric spams should raise suspicion of an underlying cerebral lesion.

2. Diffuse paroxysmal activity on the EEG- hypsarrhythmia.

3. Developmental delay or deterioration (neuro-imaging is preferred though there is no characteristic finding for WS)

Visual and auditory defects are present in one-third to one-half of affected children. Mental retardation was observed in 71 to 90 percent of patients\(^{15}\). There is growing evidence that longer duration of spasms is associated with less neurodevelopmental outcomes\(^{15}\). A psychiatric disorder was diagnosed in 28% of patients and IS may play a significant role in the etiology of autism. Guzzetta F at al (2002) studied visual attention in WS and found maturation defect of fixation shift skills in WS children. In some cases, the impaired visual-attention abilities paralleled a cognitive deterioration, even months before the onset of spasms. During the acute stage of WS, infants lost the previously acquired visual and cognitive abilities, with a typical fluctuation of arousal. Usually at 2 years, there was a persistent defective visual attention detected with the fixation-shift test\(^{18}\).

**Ocular Profile in West Syndrome**

In our study, 52 children with diagnosis of WS with M: F: 34:18 with average age of onset of spasms approx. 5 months were studied for ophthalmic manifestations. The most common ocular findings include refractive errors in 98% (51) and strabismus in 71% (37). The variation in ocular alignment noted with horizontal strabismus as esotropia in 11 (21%) and exotropia in 26 (50%). The variation across refractive errors as astigmatism in 65% (35) hypermetropia in 31% (16) and myopia was present in 2% (1).

Eyelid anomalies in the form of epicanthus, telecanthus, mongoloid and anti-mongoloid slants, euryblepharon, and medial and lateral ectropion were present in 32/52 (61.5%) of these children points towards a possible dysmorphic phenotype that may aid early characterization of children with WS.

The other ocular findings include nystagmus in 13% (7), disc pallor in 15% (8), chorioretinal degeneration in 2% (1) (Figure 3).

**AUTISM**

Autism spectrum disorders (ASD) are a complex group of neurodevelopmental disorders characterized by impairments in communication skills, social skills, restricted range of interests along with stereotyped repetitive behaviors and mannerisms\(^{15}\). The exact etiology is unknown, but multiple causes have been implicated as etiological factors of ASD which include- insult to the brain stem early in embryogenesis, genetic and environmental factors\(^{20,21}\). Various medical disorders, such as tuberous sclerosis, fragile X syndrome, Down syndrome, neurofibromatosis-1, phenylketonuria and congenital rubella syndrome have been found to be associated with ASD in 10% of the cases\(^{22,23}\).

According to a study by Ikeda J et al (2013), ophthalmologic pathology was found in 40% of patients with autism or a related disorder with 29% having significant refractive errors, 21% demonstrating strabismus, and 10% having amblyopia\(^{24}\). Other common ocular findings reported with ASD include oculomotor dysfunction, atypical gaze or gaze avoidance, eye pressing, hand flicking, light gazing, side looking, visual inattention, poor visual awareness of surroundings, fascination with spinning objects, lights and
Table 2: Ocular Profile in Developmental Delay in 180 children

<table>
<thead>
<tr>
<th>Ocular Manifestations</th>
<th>CP</th>
<th>WS</th>
<th>ASD</th>
<th>Count</th>
<th>Percentages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refractive errors</td>
<td>92</td>
<td>51</td>
<td>25</td>
<td>168/180</td>
<td>93.3%</td>
</tr>
<tr>
<td>Squint</td>
<td>58</td>
<td>37</td>
<td>4</td>
<td>99/180</td>
<td>55%</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>16</td>
<td>7</td>
<td>4</td>
<td>27/180</td>
<td>15%</td>
</tr>
<tr>
<td>Eyelid abnormalities</td>
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<tr>
<td>Mongoloid slant (MS):</td>
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<tr>
<td>Epicanthal fold (EF):</td>
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<tr>
<td>Anti-Mongoloid slant (AMS):</td>
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<tr>
<td>Antimongoloid slant (AMS):</td>
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<tr>
<td>Euryblepharon:</td>
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<tr>
<td>Ectropion:</td>
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<tr>
<td>Anterior segment</td>
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<tr>
<td>abnormalities</td>
<td></td>
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<tr>
<td>Microcornea:</td>
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<td>Congenital cataract:</td>
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<td>Optic disc abnormalities</td>
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<tr>
<td>Optic disc pallor:</td>
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<tr>
<td>Optic atrophy:</td>
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<td>Optic disc hypoplasia:</td>
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<tr>
<td>Fundus abnormalities</td>
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<td>Fundus Coloboma:</td>
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<tr>
<td>Chorioretinal degeneration:</td>
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<td>Lattice degeneration:</td>
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<td>Others</td>
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<tr>
<td>Amblyopia:</td>
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<tr>
<td>Gaze palsy:</td>
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<tr>
<td>Abnormal visual</td>
<td></td>
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<tr>
<td>behaviors (see text)</td>
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</tbody>
</table>

Figure 4: Management of DD children by prescribing glasses for refractive and strabismus correction

shadows, and bright metallic objects.

Ocular Profile in Autism in the present study:

In our study 28 children diagnosed with ASD with M:F:18:10 with average age (4.7±2.0 yrs) and average age at which diagnosed as ASD was (3.5±1.3 yrs) were studied for ocular manifestations. Refractive errors (89%) formed the major group in 25 children among the ocular findings. The variation among refractive errors includes astigmatism in 43% (12), hypermetropia in 32% (9), anisometropia in 11% (3) and myopia in 3% (1).

Other ocular findings include strabismus in 14% (4), nystagmus in 14% (4), lattice degeneration in 3% (1), abnormal visual behaviors including poor eye contact (17), gaze avoidance (8), eye pressing (2), light gazing (3), side looking (4), visual inattention (6), poor awareness of visual surroundings (8), fascination with spinning objects, lights, shadows and bright metallic objects (11).

MANAGEMENT

The management of DD is multi-disciplinary. The goal is to improve the quality of life of the child by managing treatable co-morbidities, providing support and therapies aimed at skill building within the framework of existing individual strengths and weaknesses. With respect to the ocular manifestations, ocular misalignment can be managed with simple maneuvers like correction of refractory errors and patching for amblyopia. If the deviation does not change or partially changes with optimum refractive correction or the eyes do not demonstrate any refractive error and both eyes are capable of taking up alternate fixation (thus precluding gross amblyopia), strabismus surgery is indicated to correct that component of deviation which is not ameliorated by refractive correction (Figure 4) so as to restore back motor ocular alignment, important for optimum sensory rehabilitation.

Patients with CP have a high percentage of ocular disorders and irrespective of the level of impairment, deserve an early referral to a pediatric ophthalmologist for optimal rehabilitation, that may aid visually directed gross and fine motor functions subsequently. Objective assessment of refractive errors in children with ASD is likely to aid appropriate visual rehabilitation in these patients, which may indirectly aid visually directed behavior patterns. Also, children with WS were managed as per standards of practice for strabismus and amblyopia.

CONCLUSIONS

180 children with DD were studied for ophthalmic manifestations.

The most common ocular manifestations were refractive errors (93.3%), strabismus (55%).

Others include eyelid abnormalities (17.7%), nystagmus (15%), optic disc abnormalities (11.6%), fundus abnormalities (2.7%), amblyopia & gaze palsy (7%) and anterior segment abnormalities in 1%. Abnormal visual behaviors were observed in WS children (Table 2).
In view of high prevalence of visual impairment in these children, ophthalmic evaluation should be a part of early assessment of DD children so that appropriate management can be instituted at the earliest for optimum sensory rehabilitation for proper visual as well as other areas of development.

Children with multiple disabilities often undergo rehabilitation in motor system development, speech rehabilitation and other such tasks but often visual problems are overlooked in these children, which in turn can delay efforts at rehabilitation. Early diagnosis and appropriate management of ocular problems can help the child evolve to his maximum potential.

REFERENCES
3. Simon chiu. Basics to the approach of developmental delay
To understand the pathology, it is very important to know how the normal structures appear. The cornea, anterior chamber, posterior chamber, iris, ciliary body, and anterior lens surface can be easily identified. Normally, the iris has a roughly planar configuration with slight anterior bowing, and the anterior chamber angle is wide and clear (Figure 3).

**History**

UBM was developed by Dr. Charles Pavlin et al in 1989 in Toronto. They have developed probes -50, 80, 100 Mhz but due to limited depth of penetration, 80Mhz and 100 Mhz probes were discontinued.

**Instrumentation and Principle**

UBM is based on the principle of pulse-echo system similar to ultrasound B – scan (Figure 1).

**Technique**

It is usually done in supine position but can be done in sitting position also. After instillation of topical anaesthesia, a specially designed eye cup is used to separate the eyelids and create a water bath with normal saline (Figure 2a and 2b). A sufficient palpebral fissure width must be present to accommodate an eye cup. No undue pressure should be applied on the eye cup as it can distort the angle structures. To maximize the detection of the reflected signal, the transducer should be oriented so that the scanning ultrasound beam strikes the target surface perpendicularly.

**Normal Anatomy**

To understand the pathology, it is very important to know how the normal structures appear. The cornea, anterior chamber, posterior chamber, iris, ciliary body, and anterior lens surface can be easily identified. Normally, the iris has a roughly planar configuration with slight anterior bowing, and the anterior chamber angle is wide and clear (Figure 3).

A constant testing environment is critical for cross-sectional and longitudinal comparison as the morphologic
UNITED STATES ANATOMICUAL TISSUES

Table 1: Comparison between UBM and USG B-Scan

<table>
<thead>
<tr>
<th>UBM</th>
<th>USG B-Scan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Higher probe frequency (35.50 MHz)</td>
<td>Lower frequency (8-10 MHz)</td>
</tr>
<tr>
<td>Higher image resolution (approximately 25 microns of axial and 50 of lateral resolution)</td>
<td>Lower image resolution (approximately 150 microns of axial and 450 of lateral resolution)</td>
</tr>
<tr>
<td>Shallow tissue penetration (approximately 5 mm for a 50-MHz UBM instrument)</td>
<td>Deeper tissue penetration</td>
</tr>
<tr>
<td>Probe with transducer without covering, water bath needed</td>
<td>Covered probe</td>
</tr>
</tbody>
</table>

Table 2: Comparison between UBM and AS-OCT

<table>
<thead>
<tr>
<th>UBM</th>
<th>AS-OCT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uses acoustic waves</td>
<td>Uses near infrared light waves</td>
</tr>
<tr>
<td>Contact technique</td>
<td>Non-contact technique</td>
</tr>
<tr>
<td>Media clarity not essential</td>
<td>Needs clear media</td>
</tr>
<tr>
<td>Can visualize structures posterior to iris</td>
<td>Limited visualization post iris</td>
</tr>
<tr>
<td>Skilled operator needed</td>
<td>Faster examination</td>
</tr>
</tbody>
</table>

Figure 3: Normal anatomy on UBM

Figure 4: Ciliary body membranes

relationships among the anterior segment structures alter in response to a variety of physiologic stimuli (i.e., accommodative targets and light).

CLINICAL APPLICATION

UBM is helpful in various ocular conditions. The most important indications for this investigation can be grouped (with overlapping conditions) as follows:
- **Uvea**
- **Sclera**
- **Vitreoretina**
- **Trauma**
- **Tumours**
- **Glaucoma**

**UVEA**

1. Intermediate uveitis - UBM helps in diagnosis when the fundus evaluation is not possible due to complicated cataract or post-synechiae
   a) Active-low reflective mass over pars plana and ciliary body region and low reflective ciliary body thickening suggestive of oedema
   b) Inactive-high reflective pars plana membranes with / without traction on ciliary body and peripheral retina (Figure 4).

2. Hypotony-Persistent hypotony can be because of various reasons. UBM is helpful in diagnosing and differentiating between them.
   a. Ciliary body atrophy and reduction in number and blunting of ciliary processes (Figure 5).
   b. Supraciliary effusion/Choroidal effusion-echolucent space above ciliary body or choroid.
   c. Traction on ciliary body-membranes pulling the body.

3. Lens related uveitis-
   Optic and haptic locations can be assessed accurately by looking for a strong echo at their interface plane. The most peripheral portion of the haptic defines its position in the capsular bag, ciliary sulcus, or a dislocated point where it can be a cause of uveitis due to constant rubbing (Figure 6).

Retained cortical matter, which can be a cause for inflammation can be visualized on UBM (Figure 7).

4. Parasitic uveitis-
   Peripheral granulomas as in cases of toxocariasis can be detected.

**SCLERA**

The area for evaluation is limited by the size of eye cup and width of palpebral fissure. Typically, sclera appear as regular, high reflective tissue and episclera as relatively lower reflective tissue.

1. Episcleritis-Thickening of episcleral tissue only with a clear demarcation from scleral stroma (Figure 8).
Figure 5: Blunted ciliary process

Figure 6: IOL haptic rubbing against iris (red arrow)

Figure 7: Retained cortical matter (arrow)

Figure 8: Involvement of episcleral tissue with uniform, uninvolved sclera (arrow)

Figure 9: Loss of scleral architecture (arrow)

Figure 10: IOFB in angle (arrow)

Figure 11: IOFB in lens capsule (arrow)

Figure 12: Subluxated IOL

Figure 13: Angle Recession (arrow)
2. Scleritis-
   a. diffuse-mottled internal reflectivity (Figure 9).
   b. nodular-thickened episclera with localized nodule of lower reflectivity.
   c. necrotizing-scleral thinning present.

VITREORETINA
1. Penetrating Trauma-in cases of trauma, anterior site of vitreous/retinal incarceration can be identified. It helps in localization of anterior foreign bodies.
2. Surgical planning - UBM aids in selecting the site for instrument entry by assessing the position of pars plana and anterior retina.
3. Fibrovascular ingrowth at the sclerotomy site can be a cause for recurrent vitreous haemorrhage. This can be imaged on UBM and further management can be planned.
4. Unexplained hypotony after V.R. surgery-sclerotomy gape, ciliary effusion, ciliary membranes can be picked up on UBM.
5. To locate and confirm retinal dialysis

TRAUMA
Ocular trauma often limits the visibility of the ocular structure owing to the presence of hyphaema. Accurate assessment of the structural damage can be a challenging task when clear direct visualization is not achieved. In cases of trauma, UBM helps as follows:

1. Detection of intraocular foreign body (IOFB)
   - Detects small, metallic/nonmetallic, anteriorly located IOFB
   - IOFB appear as high reflective structures with posterior shadowing and reverberation (Figure 10, 11).

2. Assessment of lens dislocation and Zonular/Capsular integrity in cases of trauma
   - Change in zonular tension, loss of zonules – lens equator changes to rounded shape
   - Posterior capsule or posterior lenticulon can be seen in cases with shallow anterior chamber.
   - IOL tilt, decentration can be seen (Figure 12)

3. Evaluation of Hypotony
4. Detection of angle structures
   1. Angle recession- Ciliary body face is torn at iris insertion, resulting in a wide angle appearance (Figure 13)
   2. Cyclodialysis cleft- UBM is particularly important in situations when conventional gonioscopy is difficult like extreme hypotony, total hyphaema. Cyclodialysis appears as disinsertion of ciliary body from scleral spur with a direct communication between anterior chamber and suprachoroidal space (Figure 14)

TUMOURS AND CYSTS
UBM helps in cases of tumour and cysts as following:
• To identify the location
• To measure the size
• To estimate tumor margins and deeper involvement
• Assessment of tumor features like internal reflectivity (vascular/cystic areas)
• Preoperative planning
• Follow up

1. Ocular Surface Squamous Neoplasia (OSSN)
   UBM helps in identifying the extent and depth of penetration of intraepithelial and invasive squamous cell carcinoma of conjunctiva and cornea (Figure 15).

2. Iris cyst
   a. Primary neuroepithelial cyst are highly reflective, have smooth cyst wall with anechoic center with no solid component. (Figure 16)
   b. Implantation cyst have diffuse/dense internal echoes and site of origin can be noted.

3. Iris Naevus
   • Appear as a solid plaque-like lesion on the anterior iris surface and/or stroma.
   • Medium to high internal reflectivity
   • Does not extend past the iris root.
   • The posterior iris surface is flat or smoothly concave (Figure 17)
   • Minimal growth tendency

4. Iris Melanoma
   • Solid irregular mass
   • Variable internal reflectivity
   • Irregularity/convex bowing of posterior iris plane (Figure 18)
   • Low to medium internal reflectivity
   • Usually solid though sometimes cavitation may occur

5. Ciliary body melanoma
   • Oval shaped mass centered on ciliary body (Figure 19)
   • Low to medium internal reflectivity
   • Usually solid though sometimes cavitation may occur

6. Anterior Choroidal Melanoma
   Ciliary body /iris extension of peripheral choroidal melanoma can be noted. It appears as tissue of reduced reflectivity compared to surrounding ciliary body /iris

7. Retinoblastoma
   UBM is important in intraocular staging of disease in cases of advanced retinoblastoma

GLAUCOMA

Forces are generated to cause angle closure in four anatomic sites. Differentiating and localizing the affected site is essential to provide effective treatment. UBM is invaluable in localizing the site of block.

1. The iris (pupillary block)
2. The ciliary body (plateau iris)
3. The lens (phacomorphic glaucoma)
4. Behind the iris by a combination of various forces (malignant glaucoma and other posterior pushing glaucoma types)

1. Pupillary block
   • Shallow A.C.
   • Narrow angle recess
   • Forward bowing of iris(Figure 20)

2. Plateau iris
   • Angle closure without pupillary block
   • Prominent peripheral roll of iris
   • Anteriorly directed ciliary body

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**Figure 19: Ciliary body melanoma**

**Figure 20: Pupillary block causing angle narrowing**

**Figure 21: Plateau iris due to anteriorly directed ciliary body**

**Figure 22**

**Figure 23: Posterior bowing of iris causing rubbing if iris**
Absence of ciliary sulcus
• ‘double hump sign’ (Figure 21)

3. Malignant glaucoma
• Shallow /flat anterior chamber
• Anterior rotation of ciliary processes (Figure 22)
• Suprachoroidal effusion may or may not be present

OPEN ANGLE GLAUCOMA
The only type of open-angle glaucoma that shows characteristic findings on UBM is the pigment dispersion syndrome
1. Pigment dispersion syndrome
• widely opened angle
• an iris with slight concavity (bowing posteriorly) (Figure 23)
• increased iridolenticular contact

CONCLUSION
To conclude, Ultrasound biomicroscopy has revolutionized the evaluation of the anterior segment of the eye. The structures surrounding the posterior chamber that were difficult to examine clinically can be evaluated in detail. The qualitative and quantitative examination by UBM has enhanced our understanding of the pathophysiology of a variety of anterior segment disorders.

REFERENCES

Financial Interest: The author does not have any financial interest in any procedure/product mentioned in this manuscript.

CONGRATULATIONS
1. Dr. G. Mukherjee, Ocular Trauma Society of India, Guwahati was Awarded Gold Medal and Air Marshal Boparai oration on 31st October 2015.
2. Dr. Ajay Aurora received the BMJ 2015 South Asia Innovation award work on ANT (Aurora Needle Trocar) cannula System.
3. Dr. J.S. Bhalla, D.D.U. Hospital, was awarded the prestigious State Award for rendering meritorious service.
4. Dr. Bhavna Chawla was awarded to BMJ South Asia 2015, recognition for excellence in Medical Research on Retinoblastoma.
DOS Times Quiz 2015-16
Episode-5

Last date: completed responses to reach the DOS OFFICE by e-mail or mail before 5 pm on 8th April, 2016

Q1. An 8 months child, parents noted the lesion since birth. What is the Diagnosis and management?

Q2. A 5 year old child presented with the swelling since birth. What is the diagnosis and management?

Q3. All of the following characteristics of patients with Lowe’s syndrome EXCEPT:
   a. Autosomal dominant inheritance
   b. Renal tubular acidosis
   c. B/L congenital cataract
   d. Infantile cataract

Q4. Which one of the following statements regarding megalocornea is FALSE?
   a. This condition is defined as a clear normal appearing cornea with a diameter measuring >13mm.
   b. This condition is often associated with anterior megalophthalmos, an autosomal dominant disorder
   c. Simple form of megalocornea is usually a B/L condition
   d. Tearing and IOP are important factors in the workup

Q5. Identify the clinical condition in a 4 years old child.

Q6. A 32 years old female presented to emergency with acute onset swelling and pain, with no systemic illness.
   a. What is the differential diagnosis
   b. What is the management plan
   c. What is the final diagnosis after viewing CT scan
Q7. Which of the following is the least risk factor for developing ROP?
   a. Hyperoxia
   b. Low birth weight (1250g)
   c. Twins
   d. Gestational Age

Q8. Which one of the following is true regarding Coats Disease?
   a. AD inheritance with variable penetrance
   b. Usually B/L
   c. Males affected more frequently than females
   d. Usually diagnosed before 2yrs

Q9. The p value of a study is calculated to be p<0.03. What does this indicate?
   a. The incidence of the disease is 3%
   b. The likelihood of results occurring as a matter of chance is 3%
   c. There is a 3% confidence interval
   d. 3% of the data was biased

Q10. What is the anesthetic duration of proparacain?
   a. 5min
   b. 10min
   c. 15min
   d. 20min

Compiled by:

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

Dr. Dewang Angmo MD, DNB, FRCS, FICO
Senior Research Associate

DOS TIMES Quiz Rules

1. DOS TIMES QUIZ will now feature as 5 Episodes (Episode 1: July-August, Episode 2: September – October, Episode 3: November – December, Episode 4: January – February, Episode 5: March – April). Entries will have to be emailed before the last date mentioned in the contest questions form. Late entries will not be entertained.

2. Please email [as scanned PDF ONLY] completed responses for the quiz along with details of the contestant filled in and signed to dostimes10@gmail.com (with cc to dosrecords@gmail.com) or mail to DOS Times Quiz, Dr. M. Vanathi, Room No. 479, 4th Floor, Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi.

3. Nonmembers may also send in their entries but will be required to send along with their completed entries, the completed membership application (with the required documents) to enroll as member. Failing this their entries into the contest will not be considered.

4. Contestants are requested to attempt all the 5 episodes of the QUIZ contest and send in their applications within the date specified. No entries will be entertained after the last date. The scores of each contestant for all 5 episodes together will be compiled at the end of episode 5 and the winner will be announced in the DOS Annual Conference in April 2016. In the event of more than one winning contestants, a draw of lots will decide the winner. Winner of each episode will also be published in the next episode along with the previous episode answers.

5. Please write to dostimes10@gmail.com/dosrecords@gmail.com for further clarifications if any.
ACROSS
4. Persistent or recurrence of a visual image after the stimulus has been removed (10)
5. Consolidated Standards of Reporting Trials (7)
6. Custom wavefront ablation generally removes ____ tissue than a standard ablation in the same eye (4)
7. Principal supporting glial cells in retina (6)
10. Principle of stereotest used in Titmus fly test (12)
12. Preferred test for categorical data in hypothesis testing (12)
13. Optic nerve consists of ____ order neurons (6)
14. Oral agent used to treat multiple sclerosis with macular edema as side effect (10)

DOWN
1. Inlay indicated for intrastromal corneal implantation to improve near vision by extending the depth of focus in the non-dominant eye of phakic, presbyopic patients (5)
2. Commercial name for vegf trap (11)
3. Benign proliferation of apocrine or accessory lacrimal gland epithelium (10)
6. Whitish spot at posterior lens surface, remnant of hyaloid artery (10)
8. Dilates the pupil without cycloplegia (13)
9. Normal eye structure hyperintense on T2 (8)
11. Valved polypropylene glaucoma drainage device (5)
It is fascinating that there are many lines in the eye. Here are a few starting from conjunctiva to retina.

1. **Arlt’s line:** Conjunctival scar in sulcus subtarsalis in trachoma.
2. **Schwalbe’s line:** A thin white or irregularly pigmented line, seen in gonioscopy, represents the peripheral margin of Descemet’s membrane.
3. **Hudson stahli’s line:** Horizontal corneal epithelial line at inferior half of cornea, seen due to aging.
4. **Stocker’s line:** Corneal epithelial iron line seen at the edge of pterygium.
5. **Ferry’s line:** Corneal epithelial iron line seen at the edge of filtering bleb.
6. **Fingerprint lines:** Seen in map dot fingerprint dystrophy.
7. **Sampalaoesi’s line:** Increased pigmentation anterior to Schwalbe’s line, seen in pseudo exfoliation syndrome and pigment dispersion syndrome.
8. **Khodadaust’s line:** Corneal graft endothelial rejection line composed of inflammatory cells.
9. **Zentmayer’s line/Scheie’s line:** Pigment deposition on equatorial surface of lens, seen in pigment dispersion syndrome.
10. **Paton’s line:** Circumferential retinal folds seen in papilloedema.
11. **White lines of vogt:** Sheathed/sclerosed vessels seen in lattice degeneration.
12. **Rucker’s line:** Sclerosed vessels due to periphlebitis retinae seen in multiple sclerosis.
13. **Schlagel’s lines:** Multiple yellow lines at posterior pole and periphery, arranged in clumps or linear streaks in multifocal choroiditis.

**RINGS**

These are a few rings within the eye.

1. **Fleischer’s ring:** Ring of iron deposition seen in keratoconus patients at the base of cone at epithelial level.
2. **Pseudo fleischer’s ring:** Iron deposition seen in hyperopes.
3. **Corneal rust ring:** Small reddish-brown, circular opacity remained in the cornea after removal of iron foreign body.
4. **Coat’s ring:** Remnants of fine iron deposits on cornea.
5. **Kayser Fleischer’s ring:** Copper deposition seen at the level of Descemet’s membrane, seen in Wilson’s disease.
6. **Schwalbe’s ring:** Schwalbe’s line is sometime scale of Schwalbe’s ring.
7. **Soemmering’s ring:** A variant of early posterior capsular opacification.
8. **Vossious ring:** A ring of iris pigment deposits seen on anterior surface of lens, in concussion injury.
9. **Weiss ring:** Epipapillary glial tissue torn from optic disc, seen in posterior vitreous detachment.
10. **Double ring sign:** Seen in hypoplasia of optic disc, with the peripheral margin of the encircling ring corresponding to the border of normal sized optic disc.
11. **Limbal ring method:** An old method used in radiological localization of intraocular foreign body.
12. **Kera rings:** An intrastromal ring segment, designed for treating corneal ectasias and refractive errors.
DOST 2016
8th DOS Teaching Programme
January 23 – 24, 2016 (Saturday & Sunday)
Jawahar Lal Auditorium, AIIMS, New Delhi
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**DOST 2016**

8th DOS Teaching Programme

January 23 – 24, 2016 (Saturday & Sunday)

Jawahar Lal Auditorium, AIIMS, New Delhi
The DOS Clinical Monthly Meet – V was held at Safdarjung Hospital & Vardhaman Mahavir Medical College, New Delhi on November 22, 2016 from 11:00 A.M. to 1.00 P.M.

The DOS Clinical Monthly Meet – VI was held at DDU Hospital, Seminar Room, 3rd Floor, Hari Nagar, New Delhi on December 27, 2016 from 11:00 A.M. to 1.00 P.M.

MINI SYMPOSIUM: Optimising Visual Outcomes in Phacoemulsification Surgery  • Chair: Prof Kamlesh, Co-Chair: Dr MC Agarwal

Riddle of Dysphotopsia – Can it be Solved?: Dr J.S. Bhalla

Hydrodynamics of Surge - An Analytic Concept: Dr N.Z. Farooqui

Optimising Clinical Outcomes in Patients with Coexisting Cataract and ARMD: Dr Amit Mehtani
The DOS Clinical Monthly Meet – VIII was held at BHARTI EYE HOSPITAL E-52 Greater Kailash - 1, New Delhi, February 21, 2016.

**Case Presentations and Clinical Talk**

- Dr. Bhupesh Singh presenting on **CLINICAL CASE I**: So many Post Keratoplasty Complications!
- Dr. Neha Bharti presenting on **CLINICAL CASE II**: Unusual case of Exudative RD
- Clinical Talk by Dr. Sudhank Bharti on A to Z of Corneal Collagen Crosslinking

**MINI SYMPOSIUM : EMERGING TRENDS IN OPHTHALMOLOGY**

- Dr. Sudhank Bharti, Dr. Archana Gupta Mahajan, Dr. Mridula Mehta moderating the mini symposium
- Dr. Rajendra Prasad presenting on Terminal Chop
- Dr. Bhuvan Chanana presenting on OCT Angiography
### DOS Enhanced Speciality Korner – DESK II

**GLAUCOMA**

*India Habitat Centre, Basement Theatre*

**March 6, 2016, 8.30 am – 4.30 pm**

**Registration fee: Rs 500**

Please send your details with payment (as cheque or cash) to the DOS OFFICE or Log on to www.dosonline.org for online registration

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### Preventing blindness in Glaucoma: Current best practices

**9.00 am – 11.00 am**

<table>
<thead>
<tr>
<th>Topic</th>
<th>Speaker</th>
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<tbody>
<tr>
<td>Screening &amp; Early diagnosis</td>
<td>Reena Chaudhary</td>
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<tr>
<td>Chairs: HC Agarwal, R Sihota</td>
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<tr>
<td>Identifying a Glaucoma suspect in a comprehensive eye camp/ PHC</td>
<td>Sagarika Patyal</td>
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<td>Identifying a Glaucoma suspect in your OPD &amp; Fallacies in Optic nerve head examination for glaucoma</td>
<td>Esha Agarwal</td>
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<tr>
<td>Pitfalls of tonometry &amp; Diurnal phasing</td>
<td>Devindra Sood</td>
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<td>Easier gonioscopy</td>
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<tr>
<td>Definitive visual field defects in Glaucoma</td>
<td>Sunil Chaudhary</td>
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<tr>
<td>PAC – diagnosis &amp; management</td>
<td>Neha Midha</td>
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<tr>
<td>OHT / POAG suspect</td>
<td>Talvir Sidhu</td>
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<tr>
<td>Early diagnosis &amp; management of common secondary Glaucomas</td>
<td>Kirti Singh</td>
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**11.15 am – 1.30 pm**

### Preventing Glaucomatous optic neuropathy/its progression

**Chairs: JC Das, T Dada**

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<thead>
<tr>
<th>Topic</th>
<th>Speaker</th>
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<tbody>
<tr>
<td>Target IOP in different severities of different Glaucomas</td>
<td>Parul Sony</td>
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<tr>
<td>Patient education highlights</td>
<td>Usha Yadava</td>
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<tr>
<td>Detecting early progression in Glaucoma</td>
<td>Suneeta Dubey</td>
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<tr>
<td>Trabeculectomy – MMC dose &amp; duration</td>
<td>Viney Gupta</td>
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<td>Early postop management of a trabeculectomy</td>
<td>Taru Dewan</td>
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<td>Managing late complications of a trabeculectomy</td>
<td>Deven Tuli</td>
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<tr>
<td>Minimally invasive glaucoma surgeries</td>
<td>Tanuj Dada</td>
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**2.00 pm – 4.00 pm**

### Stabilizing Glaucoma in the long term

**Chairs: Harsh Kumar, Viney Gupta**

<table>
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<tr>
<th>Topic</th>
<th>Speaker</th>
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<tr>
<td>Long term studies of Glaucoma drainage devices</td>
<td>JC Das</td>
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<tr>
<td>Readjusting medical therapy over time</td>
<td>MD Singh</td>
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<tr>
<td>Repeat Glaucoma surgery</td>
<td>Harsh Kumar</td>
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<tr>
<td>Special precautions in Pediatric glaucoma</td>
<td>R Sihota</td>
</tr>
<tr>
<td>Laser trabecuoplasty</td>
<td>J K S Parihar</td>
</tr>
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**Panel discussion on common queries**

T Dada, V Gupta, MD Singh, Deven Tuli, Deepa

*Each Talk is for 10 minutes followed by 5 minutes of discussion*
UPDATE ON AVASTIN

Dear Members,

On behalf of the executive and all members of Delhi Ophthalmological Society, we convey our profound thanks and appreciation to all members of the group which met the DCGI on Monday 8th February. A great amount of time and effort went into preparing a strong and convincing case for withdrawing the alert notice which prohibited use of Avastin in ophthalmology. The committee comprised of representatives of VRSI, AIOS, experts from a numbers of major Govt. and Non-Govt. ophthalmic institutions and the Roche Company. It appears that the DCGI will soon issue a fresh notification permitting use of Avastin in ophthalmology – through off label. Roche will reintroduce the Kessler code which enables genuine Avastin product to be confirmed and spurious to be detected. Authorized distributors of Roche will be permitted to sell Avastin to ophthalmologists and a list of these will be made available. AIOS-VRSI guidelines will be do drawn up for safe use of Avastin.

Our special thanks to Prof. Atul Kumar, Dr. Lalit Verma, Dr. Debashish Bhattacharya, Dr. D. Ramamurthy, Dr. Tarun Sharma, Dr. Vishali Gupta, Dr. Mahesh Shanmugan, Dr. Ajay Aurora and many others who worked tirelessly and selflessly to gather information, make a water-tight case and present it convincingly at the meeting. A pat on the back of the entire ophthalmic community which united and put in a strong petition to the DCGI and gave valuable inputs to the select committee.

Hopefully we will be using Avastin again soon but we should wait for the official notification. This is also a time for us to reflect and resolve to observe self discipline and procure from reliable sources only, use the Kessler code facility when available, follow guidelines for safe use and always keep medical ethics and the safety of our patients paramount.

Dr. Cyrus M. Shroff
President - DOS

Dr. M. Vanathi
General Secretary - DOS

DOS ELECTION 2016

DOS Elections for the post of Vice President (1 post) and DOS Representatives to AIOS (2 posts) will be held on April 17, 2016 in Hotel Ashok, New Delhi during the Annual DOS Conference 2016 – DOSCON2016: Ophthalmic Panorama.

UPCOMING EVENTS

Glaucoma Walk from Lodhi Garden to India Habitat Centre, New Delhi March 6, 2016, 7:30 a.m.

DOS Enhanced Speciality Korner – Desk II Glaucoma India Habitat Centre, Basement Theatre March 6, 2016, 8.30 AM – 4.30 PM for more details login website: www.dosonline.org

RPC Update: Innovations in Ophthalmology: March, 12-13, 2016, 49th Foundation Day Dr. Rajendra Prasad Centre for Ophthalmic Sciences, J.L. Auditorium, AIIMS, New Delhi, registration free


Inflammatio: International Collaquium of Intra Ocular Inflammation March, 18-20, 2016 at Sankara Nethralaya, Chennai, India.